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Insomnia After Stroke: Understanding its impact, assessment, and treatment.

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SUBMITTED IN FULFILMENT OF THE REQUIREMENTS FOR
THE DEGREE OF DOCTOR OF PHILOSOPHY

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Abstract

Background: Insomnia – broadly characterised by difficulties with initiating or maintaining sleep – is common after stroke, and is associated with poorer outcomes and greater risk of stroke recurrence. Yet, it has historically received less attention than other post-stroke sequelae. Consequently, current understandings of insomnia’s aetiology, impact, assessment, and treatment after stroke are limited. Seeking to address this, the present studies set out to answer four key questions:

1. What is the experience and impact of insomnia after stroke?
2. What are stroke survivors’ experiences and preferences relating to treatment?
3. How can we accurately identify which individuals are living with insomnia after stroke?
4. How can we best go about treating insomnia after stroke?

Methods: This thesis adopts a mixed-methods approach, and comprises three studies and one study protocol. Study 1 ($n = 10$) employed semi-structured interviews and interpretive phenomenological analysis to explore stroke survivors’ experiences of insomnia, and their preferences surrounding treatment. Building on the findings of this, Study 2 ($n = 180$) used receiver operating characteristic (ROC) analysis to assess the discriminant validity of a self-report measure of insomnia after stroke. Study 3 systematically reviewed 15 studies examining the efficacy of stimulus control and sleep restriction, independently and combined. Due to an absence of relevant studies within the context of stroke, older adults were selected as an appropriate adjacent population. Finally, Chapter 5 integrates the findings of all previous chapters, and presents a protocol for a single-case experimental design study evaluating the feasibility, acceptability, and efficacy of a behavioural intervention for insomnia, adapted to meet the needs of stroke survivors.

Results: Findings from Chapter 2 suggest that post-stroke insomnia arises from a complex network of psychological, physical, cognitive, and social factors, each deleteriously influencing one another. Participants described shortcomings in current clinical understanding and management of sleep after stroke, and expressed a strong preference for non-pharmacological approaches to treatment; largely due to scepticism over the safety and efficacy of hypnotic medications. Results of Chapter 3 demonstrate that the insomnia measure effectively distinguished between stroke survivors with and without insomnia, though the optimal dia-

gnostic cut-off (≤ 13) was lower than the conventional cut-off (≤ 16). Finally, stimulus control and sleep restriction significantly alleviated insomnia and depression symptoms, particularly when combined, with benefits observed in as few as two sessions. Collectively, these findings establish a robust point of departure for future research seeking to investigate sleep after stroke.

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Declaration

I declare that, except where explicit reference is made to the contribution of others, that this dissertation is the result of my own work and has not been submitted for any other degree at the University of Glasgow or any other institution

Mr Declan Simon McLaren McTague

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List of Abbreviations

- **AIE** - Attention-intention-effort
- **ARAS** - Ascending reticular activating system
- **AUC** - Area under the curve
- **BBTI** - Brief behavioural treatment for insomnia
- **BDI-ii** - Beck depression inventory
- **CBT-I** - Cognitive behavioural therapy for insomnia
- **CI** - Confidence interval
- **CT-I** - Cognitive therapy for chronic insomnia
- **DSM** - Diagnostic and statistical manual of mental disorders
- **EEG** - Electroencephalography
- **GDS** - Geriatric depression scale
- **GAD-7** - Generalised anxiety disorder scale
- **GET** - Group experiential theme
- **HPA** - Hypothalamic-pituitary-adrenal
- **IIS** - Insomnia impact scale
- **IPA** - Interpretive phenomenological analysis
- **ISI** - Insomnia severity index
- **PET** - Personal experiential theme
- **PHQ-9** - Patient health questionnaire
- **PSQI** - Pittsburgh sleep quality index
- **RCT** - Randomised controlled trial

- **REM** - Rapid eye movement
- **RCI** - Reliable change index
- **SD** - Sample standard deviation
- **SCI** - Sleep condition indicator
- **SCI-2** - Two-item variant of the SCI
- **SCN** - Suprachiasmatic nucleus
- **SE** - Sleep efficiency
- **SEM** - Standard error of measurement
- **SIS-SF** - Stroke impact scale - short form
- **SOL** - Sleep onset latency
- **STAI-Y** - State-trait-anxiety inventory
- **SQ** - Sleep quality
- **TIB** - Time in bed
- **TMS** - Transcranial magnetic stimulation
- **TST** - Total sleep time
- **VLPO** - Ventrolateral preoptic nucleus
- **WASO** - Wake after sleep onset
- $\bar{x}$ - Sample mean

Chapter 1

An introduction to insomnia after stroke

1.1 The physiological processes regulating sleep

Sleep is an essential physiological process, taking up approximately a third of each day [1, 2]. Grandner & Fernandez [3] describe sleep as a naturally occurring and reversible state – thus distinguishing it from anaesthetically induced sedation or a coma – marked by reduced consciousness and mobility relative to wakefulness, where one is perceptually disengaged; the onset and maintenance of which is regulated by homeostatic and circadian (circadian; from the Latin ‘*circa*’ and ‘*dies*’, meaning ‘about’ and ‘day’ respectively) processes. Although one may appear behaviourally inactive whilst asleep, sleep is by no means a passive process. While asleep, our bodies initiate and undergo myriad complex and organised processes vital for physiological and psychological well-being [2–4]. These include the clearance of neurological waste [5–7], metabolic processes [8], modulation of nociception (the perception of harmful stimuli) [9, 10], consolidation of memory and learning [11, 12], regulation of the immune system [13], and growth and repair of biological tissue [14, 15]. These processes occur throughout shifting phases – or stages – of sleep, which vary cyclically across the night. These are grouped broadly into rapid eye movement (REM) and non-rapid eye movement sleep (NREM), the latter of which is further broken down into more nuanced stages, corresponding with increasing ‘depth’ of sleep (for an overview see Carskadon & Dement [16]). Although a comprehensive understanding of the exact biological function of sleep remains elusive [4, 17], disruption to sleep impairs functioning across many domains [2, 3], and prolonged sleep loss results in death in animal models [18, 19]. Thus, sleep is considered a ‘non-negotiable biological state required for the maintenance of human life’ [3, p. 1].

1.1.1 The two-process model of sleep

In the latter half of the 20th century, findings from various animal models pointed towards the existence of an intrinsic, light-sensitive mechanism, underpinning regular variations in sleep-wake activity [20]. Building on these findings, Alexander Borbély put forth a theory suggesting that sleep was regulated by two oscillatory biological processes; the homeostatic sleep drive and the circadian rhythm [21, 22]. The former refers to the body’s biological drive for sleep homeostasis [22], which manifests as an increasing propensity for sleep with increasing duration of wakefulness [21, 23]. Conceptually akin to a ‘sleep economy’, the homeostatic sleep drive involves a system of accruing ‘sleep debt’ whilst awake, which is re-

paid during sleep [24, 25]. In early versions of this regulatory model, Borbély and colleagues hypothesised that neurochemical mechanisms may underpin this process [26]; a hypothesis later substantiated by research implicating adenosine, an inhibitory neurotransmitter, in the regulation of sleep homeostasis [27–32]. Adenosine, a waste product of energy metabolism in the brain [33], accumulates throughout periods of wakefulness [28, 29, 33–35], and decreases during sleep [28, 33, 35]. Molecules that bind antagonistically to adenosine receptors in the brain (i.e., blocking the action of adenosine), such as caffeine [28], promote wakefulness and reduce the propensity for sleep [28]; whereas adenosine agonists elicit the opposite effect [28].

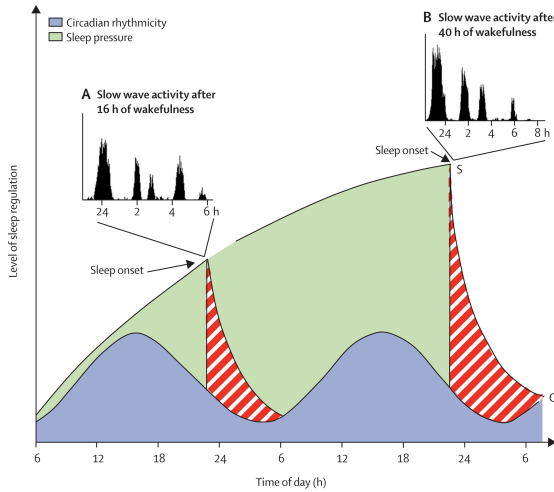


Figure 1.1: Two-process model of sleep regulation over a 48-hour period. Image from Riemann et al. [36], used with permission from Elsevier.¹

The circadian rhythm refers to a sinusoidal pattern in an organism’s propensity for sleep and wakefulness across a 24-hour period [21, 22]. These rhythms are first established in infancy [37], and vary across the lifespan [38–40]. In humans, the circadian process rhythmically follows an intrinsic cycle lasting approximately 24 hours [41], and is governed by a structure in the hypothalamus [42] known as the suprachiasmatic nucleus (SCN) [43–46]. This tiny bilateral structure, comprised of around 10,000 neurons, receives direct

input from photosensitive ganglion cells in the retina [42, 47]. This information allows the SCN to adjust the frequency of neuronal firing to correspond with light-exposure, resulting in variations in the expression of sleep- or wake-promoting signals [43]. This is part of a process known as ‘entrainment’ [48], which uses behavioural and environmental cues, known as ‘zeitgebers’ (time-givers) to maintain circadian rhythmicity [49]. While this can be influenced by many factors [50, 51], light is the primary zeitgeber in humans [49]. Corresponding with variations in light, the SCN increasingly elicits wake-promoting signals throughout the day, peaking in the early evening (shortly before one’s habitual bedtime), before switching to promote sleep (see Figure 1.1 for a graphical representation) [32, 49, 50, 52, 53].

¹The interaction between the homeostatic sleep drive (process S) and the circadian rhythm (process C) regulates the sleep-wake cycle. The red and white pattern represents the return of the sleep drive to homeostasis, following sleep. The green shaded area represents the homeostatic sleep drive in the absence of sleep. The blue shaded area represents the circadian process.

Although a limited degree of volitional control over one’s sleep does exist, the interplay of the homeostatic and circadian processes largely determines when we feel tired, the ease at which we fall asleep, and the architecture of our sleep across the night [49]. In Borbély’s model, one’s propensity for sleep is equal to the sum of the homeostatic sleep drive and the circadian rhythm, with healthy sleep being evidenced by alignment between these two systems [21, 54]. Under optimal conditions, levels of adenosine reach their peak in congruence with the upper threshold of the circadian process, triggering sleep. Adenosine is then cleared during sleep, and the individual awakens when the homeostatic sleep drive approaches the circadian rhythm’s lower threshold (Figure 1.1) [21, 23, 24]). Misalignment between these physiological mechanisms can lead to impaired sleep, often leading to sleep disorders such as insomnia [55], and may also increase the risk of cardiovascular disease [56].

1.1.2 The ‘flip-flop’ switch

Complementary to Borbély’s model is the ‘flip-flop’ switch model of sleep, proposed by Saper and colleagues [32, 57, 58]. The ‘flip-flop’ switch model of sleep regulation focuses on the transition between sleep and wake (and vice versa), hypothesising that distinct groups of neuronal cells regulate sleep and wakefulness via a bistable switch mechanism, with each half of this mechanism inhibiting the activity of the other. Neuronal cells in the hypothalamus, basal forebrain, and the ascending reticular activating system (ARAS) in the brain stem – which produce a signalling molecule known as orexin (or hypocretin) – comprise an arousal system which promotes wakefulness [32, 58, 59]. A comparatively smaller collection of neurons, found within the ventrolateral preoptic nucleus (VLPO), are thought to induce sleep, firing twice as quickly during sleep as they do during wakefulness [60, 61]. Rapid neuronal firing in the VLPO during sleep produces neurotransmitters which inhibit the activity of the arousal system; thereby disinhibiting their own activity. Conversely, increased activity of the ARAS – the arousal system – during wakefulness produces neurotransmitters which inhibit VLPO activity, thereby disinhibiting neuronal activity in the arousal system (for an overview see Arrigoni & Fuller [62]). This mutually inhibitory system is akin to circuits known as ‘flip-flop’ switches, common in electrical engineering [57, 63]. Bistable feedback loops such as these typically correspond with brief transitional periods, which are advantageous in the regulation of sleep as they prevent prolonged periods in an intermediate state, which would likely have been disadvantageous in our evolutionary history [32].

Consistent with Borbély’s two-process model, adenosine is thought to contribute to the disinhibition of VLPO neurons, thereby facilitating their promotion of sleep [62]. This potentially explains how the gradual potentiation of the homeostatic sleep drive can reach a threshold where the rapid onset of sleep occurs and illustrates the interplay between the two-process model and ‘flip-flop’ switch. Correspondingly, in response to emotional and cognitive inputs, wakefulness promoting cells in the arousal system may be able to temporarily override the action of the homeostatic and circadian regulatory systems [32, 64], thereby preventing the de-arousal necessary for sleep onset [25, 65]. While this response may have been evolutionarily adaptive, inappropriate activation of this process may be a mechanism underpinning sleep disorders such as insomnia [66].

1.2 Defining insomnia disorder

Insomnia is broadly defined as difficulty with the initiation or maintenance of sleep, or waking up too early [67–69]. To meet criteria for the classification of insomnia disorder, both the Diagnostic and Statistical Manual of Mental Disorders: 5th edition (DSM-5) [67] and the International Classification of Sleep Disorders: 3rd edition (ICSD-3) [68] stipulate that symptoms must occur at least three nights per week, persist for at least three months, and not be better explained by another sleep disorder, impaired sleep opportunity (e.g., purposefully curtailing the sleep window), or environmental circumstances (e.g., noise or light). The International Classification of Diseases for Mortality and Morbidity Statistics: 11th edition (ICD-11) [69] carries the same stipulation, except does not quantify a required weekly frequency of symptoms. While some variance in criteria does exist (see Table 1.1), all diagnostic manuals agree that symptoms must cause distress or impairment to daytime functioning – such as in academic, social, or vocational settings – for a diagnosis of insomnia disorder to be made. This final criterion extends the focus beyond nighttime symptomology, positioning insomnia as a 24-hour disorder [70], as reflected by associations between insomnia and reduced quality of life [71], impaired cognitive performance [72, 73], poorer mental health [74, 75], and increased suicide risk [76–79].

To those not familiar with the history of sleep research, the notion that poor sleep can precede, or arise independently of poor mental health is likely uncontroversial. Indeed, growing evidence does indicate a bi-directional relationship between sleep and psychiatric disorders [80–82]. Historically, however, this has

not been reflected in clinical approaches to insomnia care, where disturbed sleep was considered secondary to, or caused by a co-morbid psychiatric or somatic condition [83]. Skaer et al. [84] reviewed the data of over 3 million medical patients in the US from 1995 and 1996, and found that only 18.8% of people presenting with a primary complaint of insomnia received a diagnosis of insomnia disorder; the majority were diagnosed with non-sleep related conditions, such as depression. Ohayon’s 2002 landmark review [85] reported similar findings, with the summation of data from five European studies suggesting that a diagnosis of insomnia disorder was ascribed to only 35% of people meeting DSM-4 criteria for insomnia disorder. The remaining 65% received a diagnosis of another sleep disorder (5%), a diagnosis of a psychiatric condition (36%), or no diagnosis at all (24%). This perspective, of insomnia being secondary to existing psychiatric or somatic conditions, is reflected in earlier versions of the DSM which distinguished between ‘primary’ and ‘secondary’ insomnia. Thus, making the ontological assumption that insomnia occupied a different mode of existence depending on whether it manifested in isolation, or co-morbidly with another condition. This perspective implied that a fundamental difference in the nature of insomnia existed, depending on its relationship with other conditions. This hypothesis was not borne out of evidence, and mounting literature demonstrating the persistence of insomnia following successful treatment of co-morbid conditions [86, 87] called this distinction into question [70]. The DSM-5 heralded a shift away from this dichotomous view of insomnia’s pathology [67], establishing insomnia disorder as its own diagnostic category; followed sooner after by the ICSD-3 [68], and most recently by the ICD-11 [69].

Between-study heterogeneity in the assessment and definition of insomnia has complicated efforts to understand its epidemiology [85, 88–90]. In a review of population-based studies, Ohayon [85] demonstrated that estimates of insomnia’s prevalence varied considerably, from 5% to 50%, depending on which diagnostic criteria were employed. Studies without restrictive criteria for frequency or severity reported that insomnia symptoms were present in approximately 33% of participants, whereas studies employing stringent criteria in accordance with diagnostic manuals estimated prevalence to be around 6%. Factors such as older age and female sex are recognised risk-factors for insomnia [91]. Meta-analytic results suggest that insomnia is 1.4x more common in females than in males [92], and recent findings indicate that treatment responses may vary by sex [93]; although authors report that these are unlikely to be clinically significant, and do not advocate differing treatment approaches with regards to sex.

Table 1.1: Insomnia disorder according to DSM-5, ICSD-3, and ICD-11 criteria.

<i>DSM-5</i>	<i>ICSD-3</i>	<i>ICD-11</i>
A predominant complaint of dissatisfaction with sleep quantity or quality, associated with one or more of the following symptoms: Difficulty initiating sleep; Difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings; Early-morning awakening with inability to return to sleep.	The patient reports, or the caregiver observes, one or more of the following: Difficulty initiating sleep; Difficulty maintaining sleep; Waking up earlier than desired; Resistance to going to bed on appropriate schedule; Difficulty sleeping without parent or caregiver intervention.	A frequent and persistent difficulty initiating or maintaining sleep, resulting in general sleep dissatisfaction.

<i>DSM-5</i>	<i>ICSD-3</i>	<i>ICD-11</i>
The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, academic, behavioural, or other important areas of functioning.	The patient reports, or the caregiver observes, one or more of the following related to night-time sleep difficulty: Fatigue/malaise; Attention, concentration, or memory complaint; Impaired social, family, occupational, or academic performance; Mood disturbance, irritability; Daytime sleepiness; Behavioural problems (e.g., hyperactivity, impulsivity, aggression); Reduced motivation, energy, initiative; Proneness for errors/accidents; Concerns about or dissatisfaction with sleep.	Sleep disturbance causes some form of daytime impairment, such as: fatigue, depressed mood or irritability, general malaise, or cognitive impairment.
The sleep difficulty occurs at least 3 nights per week.	Sleep disturbance and associated daytime symptoms occur at least three times per week.	Sleep disturbance and associated daytime symptoms occur several times per week.
The sleep difficulty is present for at least 3 months.	Sleep disturbance and associated daytime symptoms have been present for at least three months.	Sleep disturbance and associated daytime symptoms have persisted for at least 3-months.

<i>DSM-5</i>	<i>ICSD-3</i>	<i>ICD-11</i>
The sleep difficulty occurs despite adequate opportunity for sleep.	The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e., enough time is allotted for sleep) or inadequate circumstances (i.e., the environment is safe, dark, quiet, and comfortable) for sleep.	Occurs despite adequate opportunity and circumstances for sleep.
The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing-related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia).	Sleep/wake difficulty is not better explained by another sleep disorder.	If the insomnia is due to another sleep-wake disorder, chronic insomnia should only be diagnosed if the insomnia is an independent focus of clinical attention.
The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).	Not applicable	If the insomnia is due to a substance or medication, chronic insomnia should only be diagnosed if the insomnia is an independent focus of clinical attention.
Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia.	Not applicable	If the insomnia is due to a mental disorder, chronic insomnia should only be diagnosed if the insomnia is an independent focus of clinical attention.

<i>DSM-5</i>	<i>ICSD-3</i>	<i>ICD-11</i>
Specify if insomnia presents with non-sleep disorder mental comorbidity, including substance use disorders, with other medical comorbidity, or with other sleep disorder.	Not applicable	Not applicable
Episodic: Symptoms last at least 1 month but less than 3 months. Persistent: Symptoms last 3 months or longer. Recurrent: Two or more episodes within the space of 1 year.	Not applicable	Some individuals with chronic insomnia may show a more episodic course, with recurrent episodes of sleep/wake difficulties lasting several weeks at a time over several years.
Acute and short-term insomnia (i.e., symptoms lasting less than 3 months but otherwise meeting all criteria) should be coded as another specified insomnia disorder.	Not applicable	Individuals who report sleep related symptoms in the absence of daytime impairment are not regarded as having an insomnia disorder.

With regard to age, Roth et al.[94] found that although sleep-related difficulties increase with age, reports of daytime impairment decrease, possibly due to lifestyle changes in later life. Thus, the increased focus on daytime symptoms in operational diagnostic criteria for insomnia may influence population-level estimates of insomnia in older adults. The variation in contemporary diagnostic criteria for insomnia, recent updates to these criteria, and heterogeneity in prevalence estimates, underscore the need for field-wide consensus on defining insomnia and for standardized assessment methods, validated in the general population and sub-populations. The present thesis aims to address this need within the context of stroke.

1.3 Approaches to treating insomnia disorder

Despite its prevalence, deleterious effects, and the perceived saliency of sleep as a pillar of health [95], insomnia is frequently inadequately managed [96, 97]. Contravening published guidelines [98–100], pharmacological interventions are commonly endorsed as first-line treatments for insomnia by primary care physicians [101]. One randomised retrospective chart review of patients from a medical centre at the University of Pennsylvania found that 51.5% were prescribed pharmacological interventions for sleep [97]. While effective in the short-term, hypnotic medications are associated with myriad unwanted side-effects [102, 103] – including an increased risk of stroke [104] – and should only be used for a short period (≤ 4 -weeks) due to the potential for dependence and tolerance [100, 105]. Moreover, evidence suggests that, given the choice, most individuals with insomnia would prefer a psychological intervention over a pharmacological one [106, 107]; in part due to stigma associated with hypnotic medication [95].

1.3.1 Cognitive behavioural therapy for insomnia (CBT-I)

Cognitive behavioural therapy for insomnia (CBT-I) is recommended as the first line treatment for insomnia disorder by the European Sleep Research Society, European Insomnia Network [100], the American Academy of Sleep Medicine [98, 108], the World Sleep Society [99], and the National Institute for Health and Care Excellence [109]. CBT-I is a non-pharmacological intervention targeting the cognitions, emotions, and behaviours that perpetuate insomnia disorder, which is typically delivered over 6-8 weekly sessions [110, 111]. Comprised of several

monotherapies, CBT-I is itself a multicomponent treatment, with its composition varying on a case-by-case basis. Core components include stimulus control [112], sleep restriction [113], sleep hygiene [114], and cognitive therapy [115]. More recently, ‘third-wave’ therapies such as mindfulness-based interventions, and acceptance and commitment therapy, have been explored as potential adjuncts [116, 117].

1.3.2 Stimulus control

Stimulus control [112, 118] operates within B.F. Skinner’s theory of behaviourism [119], assuming that sleep is an operant behaviour, the manifestation of which can be influenced by various behavioural, psychological, and environmental stimuli [118]. For those without sleep difficulties, items such as the bed or darkness may serve as discriminative stimuli for sleep, this relationship having been positively reinforced by the recognition of their presence historically preceding sleep. Whereas those with insomnia may have developed conditioned associations between the bed and sleeplessness, as well as sleep-inhibiting behaviours, such as watching T.V. or ruminating [25, 120]. A Pavlovian component also exists, where consistent sleeplessness leads to negative associations between the bed and frustration, and anxiety [120]. Bootzin [112] theorised that by controlling which stimuli an individual is exposed to within the context of sleep, associations harmful to sleep could be weakened – and those beneficial to sleep could be strengthened. Stimulus control was borne out of this hypothesis, and thus aims to establish the bed and bedroom as cues for sleep onset, weaken their associations with sleep-inhibitory behaviours, and to develop a regular pattern of sleep and wake [110].

The approach outlines precise and easy to follow instructions for patients [110, 120]:

1. ‘Lie down to go to sleep only when you are sleepy.’

This encourages patients to develop their awareness of their own physiological cues for sleep, rather than relying on potentially maladaptive methods, such as calculations of sleep duration [110]. The latter may lead to distress, further perpetuating negative associations between the bedroom and sleeplessness.

2. ‘Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward when you intend to go to sleep.’

This rule discourages participants from engaging in sleep-inhibitory behaviours in the bedroom, with the aim of weakening extant associations between the bedroom and sleeplessness and cognitive arousal [110]. Bootzin & Perlis [110] suggest that sexual activity is excepted in this instruction for practical reasons, though evidence does suggest that sexual activity may confer benefits to sleep [121–127]. Thus, engaging in sexual activity in bed may mediate the effects of stimulus control in establishing the bedroom as a place for restful sleep, although this is speculative at this stage, and investigation is indicated.

3. ‘If you find yourself unable to fall asleep, get up and go into another room. Stay up as long as you wish and then return to the bedroom to sleep.’

In carrying out rule 3, patients are encouraged get out of bed if they have been unable to fall asleep within approximately 10 minutes. Importantly, individuals are urged to avoid watching the clock, and to subjectively estimate this.

4. ‘If you still cannot fall asleep, repeat step 3. Do this as often as is necessary throughout the night.’

By encouraging the individual to leave their bed when they are not asleep, stimulus control limits the amount of time spent in bed awake, thereby weakening associations with wakefulness, strengthening associations with sleep, and potentially giving individuals a greater sense of perceived control over their insomnia [110].

5. ‘Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm.’

Encouraging the person with insomnia to maintain a regular sleep wake cycle is beneficial in maintaining circadian and homeostatic process alignment, in turn reinforcing the physiological cues for sleep.

6. ‘Do not nap during the day.’

The final rule aims to increase the pressure to sleep via the homeostatic drive, facilitating sleep onset. This further reinforces the bedroom as a restful place, and the participant’s perception of treatment success likely improves the likelihood of adherence.

Adhering to these instructions increases the likelihood that one will fall asleep with ease, and maintain sleep throughout the night, thereby reinforcing the bedroom as a discriminative stimulus for sleep, and loosening the grip of insomnia [110]. A recent systematic review and meta-analysis of the efficacy of stimulus control as a standalone treatment for insomnia found the intervention brought about significant improvements in sleep onset latency ($g = 0.85 - 0.87$) when compared to passive controls; although improvements were non-significant when compared to active controls [128]. Authors also remarked at the non-existence of research investigating the underlying mechanisms of stimulus control and recommended this be a priority in future research.

1.3.3 Sleep restriction

Sleep restriction is designed to exploit the body’s natural homeostatic sleep drive [120, 129]. Studies have shown it to be efficacious as a standalone treatment [130–132] – outperforming non-restrictive regulation of sleep and wake times [133] – and as part of a multi-component approach, where it has been demonstrated to reduce the night-to-night variability common to insomnia [134–136]. Sleep restriction is based on the observation that people with insomnia often spend excessive amounts of time in bed trying to sleep, which can exacerbate sleeplessness and impede the regularity of the sleep-wake cycle [120].

Spielman and colleagues [129] outline a contemporary approach to sleep restriction in the general population. To begin, clinicians establish an individual’s average total sleep time (TST), typical wake-time, and the portion of the night in which they find they sleep best. This information is ordinarily obtained via 7- to 14-day sleep diaries and clinical interviews, though objective measures are also occasionally employed. Clinicians and patients then collaborate to set a prescribed ‘sleep window’, restricting the time in bed (TIB) to the average estimated from the patients sleep diaries, with the minimum time in bed being 5 hours. Sleep and wake times are determined via examination of patterns in the individual’s nocturnal awakenings or sleep onset latency.

Patients continue to complete a sleep diary throughout the treatment, and the data are reviewed after each week. Adjustments to the sleep window are made each week, based on the individuals sleep efficiency (SE) – calculated as:

$$SE = \frac{TST}{TIB} \times 100 \quad (1.1)$$

If an individual's sleep efficiency is $\geq 90\%$, then the sleep window is increased by 15-30 minutes. If it is between 85% and $<90\%$, the sleep window is unaltered. If it falls below 85%, then the sleep window is shortened by 15-30 minutes. This titration process continues until sleep is sufficiently restricted to consolidate sleep and maintain a regular sleep-wake schedule, without significantly impairing daytime functioning [129].

However, patients often find sleep restriction difficult [137, 138], and it is not recommended for individuals whose vocations require optimal vigilance (e.g., bus drivers or air traffic controllers) or those with health conditions that would be exacerbated by reduced sleep (e.g., epilepsy or bipolar disorder) [129, 139]. Although results are inconsistent [140, 141], evidence suggests that adherence to sleep restriction protocols may influence treatment outcomes [140]. Thus, consideration of adaptations and alternatives in order to increase accessibility is indicated.

1.3.4 Sleep compression

Sleep compression [142] exists as a potentially more tolerable alternative, which targets the same mechanisms as sleep restriction, but operates in reverse; where sleep is incrementally curtailed over several weeks, rather than being immediately reduced to match time in bed. Resultantly, sleep compression avoids the temporary increase in daytime sleepiness [142] observed in sleep restriction [138], and thus may be a more acceptable treatment in individuals for whom the avoidance of daytime impairment is particularly warranted. A recent sub-group analysis of data from a randomised controlled trial (RCT) [143] found that both sleep compression and sleep restriction yielded similar improvements in objectively measured sleep parameters, though only sleep compression did so without any significant reduction in total sleep time; albeit it with a slower rate of change. However, baseline differences did exist between groups, which may have impacted results. Notably, recent evidence has shown poorer sleep at baseline to be associated with a greater magnitude of improvement following CBT-I [144].

In addition to enacting influences on sleep physiology [21], sleep restriction may also improve sleep via cognitive and emotional mechanisms. Altena et al. [145] posit that sleep restriction can be operationalised within Barlow's model of treating emotional disorders [146], which emphasises the importance of preventing emotional and behavioural avoidance. In sleep restriction, an individual is

exposed to their fears surrounding sleeplessness and its subsequent consequences, and learn that they are able to get through the day, even with less accumulated sleep [129]. Altema et al. postulate that this disrupts the cognitive-behavioural cycle of fear, anxiety, and avoidance that perpetuates insomnia [129, 145].

1.3.5 Sleep hygiene

Sleep hygiene [147] generally consists of psychoeducation around sleep, alongside recommendation of sleep-promoting behaviours and lifestyle changes [114, 120] - distinct from those recommended in stimulus control. As a standalone treatment, sleep hygiene has not been evidenced to be effective at treating the symptoms of insomnia [114, 148], and recent evidence suggests it may not carry any influence over treatment outcomes [148]. Nonetheless, it is generally thought to be useful in facilitating patients' understanding of more potent components such as stimulus control and sleep restriction [120]. Thus, while it may have historically played a larger role in treatment, today it is primarily valued as a potential mediator, and a possible means of preventing relapse [114]. Sleep hygiene recommendations tend to vary considerably [114, 120, 149], though common advice includes avoiding exercise in the late afternoon or evening, refraining from napping throughout the day, eating a light carbohydrate snack and avoiding liquids before bed, and limiting the consumption of caffeine, alcohol, and nicotine – particularly close to sleep. Additionally, establishing a quiet, dark, and comfortable environment, free of interruptions such as pets, and maintaining a regular sleep-wake schedule, is frequently recommended [114]. Generally, sleep hygiene is considered most useful when a person-centred approach is taken, and recommendations target specific sleep-inhibitory behaviours relevant to the individual [115].

1.3.6 Cognitive therapy

Cognitive therapy for insomnia can be categorised into two approaches [120]. The first, developed by Charles Morin [150], focuses on confronting dysfunctional beliefs relating to sleep. The second, put forth by Allison Harvey [151], builds on Morin's work by expanding the focus to five cognitive and behavioural processes thought to perpetuate insomnia: cognitive arousal (e.g., worry or rumination); selective attention to perceived sleep-related threats; erroneous perceptions about sleep and its impact on daytime functioning; dysfunctional beliefs pertaining to sleep; and a propensity to engage in maladaptive behaviours that perpetuate

these beliefs. Morin’s approach, also known as ‘Cognitive Restructuring’, aims to help individuals identify, confront, and adapt maladaptive or erroneous beliefs or attitudes relating to sleep, to reflect a more rational stance [150]. Harvey’s Cognitive therapy for chronic insomnia (CT-I) [151], based on a previously derived cognitive model of insomnia’s aetiology [152], aims to tackle cognitive factors that perpetuate insomnia throughout the day and night [151], while also planning for the prevention and management of potential relapse [120, 151]. To date, there is a paucity of research exploring cognitive therapy as a standalone treatment for insomnia [153–157]. One multi-centre RCT in North America comparing the efficacy of behavioural and cognitive treatments for insomnia in adults (≤ 25 years) found that while behavioural treatments were more efficacious in the short-term, cognitive therapy elicited slower but more robust improvements [158]. Nonetheless, CBT was the most efficacious approach overall. More recently, Furukawa et al. [148] performed a network meta-analysis examining the association of each CBT-I component with treatment outcomes. Results indicated that while CBT was the most likely approach to result in remission, cognitive restructuring was a critical component of this.

1.3.7 Issues surrounding CBT-I

The efficacy and effectiveness of CBT-I is well established, with numerous meta-analyses [159–177] confirming its potency in eliciting improvements in both daytime and nighttime symptoms of insomnia across various populations, with long-lasting results [178, 179]. Despite this, and discordant with the recommendation of CBT-I being the first-line treatment for insomnia [98–100, 161], widespread dissemination has been limited [180]. Sun and colleagues [97] found that only 5% of people presenting with insomnia to a University of Pennsylvania medical centre were referred for CBT-I. Similarly, Benca et al [95] reported that just 31% of primary care practitioners, and 45% of psychiatrists, reported recommending CBT-I ‘often’ or ‘always’ to patients with insomnia. The extent of this issue is considerable, such that a task force, comprised of members from The European Academy for Cognitive Behavioural Therapy for Insomnia and the European Insomnia Network, was established with the aim of furthering the dissemination of CBT-I throughout Europe [181]. In a narrative review, Koffel et al. [180] identify three main barriers to the dissemination of CBT-I: systemic, clinician, and patient.

1.3.7.1 Systemic barriers to CBT-I

Systemic barriers refer to the lack of availability of CBT-I, particularly in remote geographic regions. The scarcity of trained practitioners, as well as length treatment protocols (6-8 sessions) are significant contributors to this [181–183]. Estimates by Thomas et al.[184] suggest that that fewer than 800 behavioural sleep medicine providers exist worldwide, 88% of whom reside within the USA; 58% of those being concentrated within only 12 states. Hughes et al. [185] recommend broadening the provider base of CBT-I to include professionals beyond behavioural sleep specialists and clinical psychologists, in efforts to meet demands. Congruently, and with the same aim in mind, the European task force suggested implementing a stepped care approach, where CBT-I could be delivered by a range of professionals, with varying levels of expertise [181]. This approach would allow for the utilisation of non-sleep specialists, such as nurses and physician associates [186], as potentially viable vectors through which to alleviate the limited accessibility of CBT-I. While emerging evidence suggests this to be a potentially auspicious strategy [187–190], heterogeneity in protocols, practitioners, settings, and outcome measures limit ascertainment of efficacy in these trials [188].

Seeking to address challenges to access, Hughes et al. [185] also recommend expanding the mode of delivery to include group-based or online methods, as well as autonomous, self-directed, methods such as via apps or books; several of which appear efficacious [191–196]. Self-directed approaches appear accessible and effective for improving symptoms of insomnia [197, 198] and psychiatric distress [199], including in people with mild traumatic brain injury [200]. However, condition specific adaptations may be necessary in certain populations, such as caregivers of people with dementia [201, 202], and stroke survivors [203]. This thesis intends to explore the latter.

Another strategy may be to examine the feasibility, acceptability, and efficacy of CBT-I’s individual components. As the factors instigating and maintaining insomnia is likely to vary between individuals [25], so too may the optimal treatment strategies. Espie [25] argues that CBT-I should not be viewed as singular treatment, but as a treatment modality. By understanding the aetiological and perpetuating factors of an individual’s insomnia, and being armed with a versatile arsenal of treatment protocols, it becomes possible to examine which treatments work best for which patients [25]. Congruent with the concept of personalised medicine [204], Espie [25] outlines a customisable framework of CBT-I, targeting the physiological, cognitive, behavioural, and emotional factors contributing to

insomnia, that can be adapted to meet the needs of individual participants. A parsimonious approach, employing tailored interventions, may be helpful in addressing the systemic barriers to CBT-I's accessibility, and may contribute to the remediation of populations specific challenges (e.g., stroke [203]).

1.3.7.2 Clinician barriers to CBT-I

Koffel et al.[180] outline three main clinician barriers contributing to the difficulties surrounding the dissemination of CBT-I: lack of knowledge, treatment beliefs, and lack of motivation. Many clinicians are unfamiliar with CBT-I and are therefore hesitant to prescribe it, often prioritising other conditions such as depression or anxiety [180]. A recent survey of 126 primary care providers indicated how, cognisant of the scarcity of psychological treatment in general, clinicians were reluctant to prescribe cognitive behavioural therapy for 'a simple sleep disorder'; believing that this resource should be reserved for patients with more pressing needs [205]. The same study found that less than half (46%) of primary care providers surveyed knew where to refer patients for CBT-I, and many felt insufficiently trained in treating sleep problems or deprescribing hypnotic medications. Relatedly, clinicians may erroneously assume that patients are averse to CBT-I, preferring medication as a quicker solution [180, 206]. Benca et al. [95] found that 83% of primary care providers, and 86% of psychiatrists, believed that patients would struggle to adhere to non-pharmacological treatments, such as CBT-I. Compounding this further is the fact insomnia is not regularly screened for [180], and thus cases of insomnia are likely to be missed, even if CBT-I were available. One qualitative study of 24 general practitioners from Australia found that most did not routinely screen for insomnia [207]. Participants attributed this to already having an overwhelming number of things to screen for – stressing the importance of brief and easy to administer measures – but also because they felt as though they had nothing to offer patients for their sleep. Moreover, in a recent survey exploring current practices of post-stroke sleep management in occupational therapy [208], 84.7% of respondents reported assessing sleep in less than half of their patients - only 2.6% of respondents doing so using standardised scales - despite 98% believing that sleep is important for recovery.

Koffel and colleagues [180] argue that clinicians do express a desire to improve their understanding of insomnia, and advocate for interventions aiming to improve clinical education. In the same vein, Benca et al. [95] report that of 452 healthcare professionals surveyed, >93% expressed a desire to improve their

understanding of assessing and managing insomnia. While investigation of sleep-related educational interventions for clinicians is scarce, one Australian study found that a brief training session focusing on non-pharmacological treatment of insomnia corresponded with rates of benzodiazepine prescription as a first-line treatment reducing from 85% to 48% over a 7-month period [209].

1.3.7.3 Patient barriers to CBT-I

Several patient-driven barriers, as outlined by Koffel et al. [180], are indeed valid. Treatment adherence rates, for example, may be as low as 60% [210], and potentially lower for digital CBT-I [211]. In a systematic review, Mellor et al. [212] identify depression, support external from treatment, self-efficacy, and dysfunctional beliefs pertaining to sleep, as predictors of adherence to CBT-I protocols. However, a systematic review from Agnew et al. [213] reported that the ascertainment of reliable estimates of adherence rates in CBT-I was impeded by heterogeneity in how adherence was operationalised, advocating for a consensus on reliable measures; this is substantiated by findings in Mellor et al. [212]. Though speculative, it's possible that certain components of CBT-I may hinder accessibility, or efficacy, in certain populations [148, 214]. Thus, investigation of tailored and streamlined implementations is warranted.

Finally, contrary to clinician beliefs [207], evidence suggests that individuals with sleep difficulties are often unlikely to seek support [215, 216], often due to associated costs, believing medication was their only option, or feeling as though they should be able to handle their sleep without support [95, 216]. Perceived stigma is another barrier to support seeking [95, 180, 216], however, recent qualitative evidence suggests that societal perspectives are shifting, and people are becoming more comfortable with the concept of psychological support [217]. Notably however, over half of the participants in Benca et al. [95] reported feeling as though there was stigma associated with taking medication for sleep. Given the potential for stigma to influence treatment adherence [218], understanding patient perspectives and preferences pertaining to treatment for insomnia is critical for developing effective and acceptable interventions. This thesis aims to do so, within the context of stroke.

1.4 The aetiology of insomnia

Understanding the aetiology of insomnia is essential for determining the most appropriate treatment approaches [219]. Numerous aetiological models of insomnia disorder have been proposed [220–222], a comprehensive review of which would be outwith the scope of this thesis, and thus only those most relevant to the aims of this research will be discussed in detail. Nonetheless, the ubiquity of insomnia, and the diversity of its manifestations are exemplified through the variety of these models; each of which addresses distinct, yet often interrelated, facets of the disorder. Some models emphasise single-factor explanations of insomnia, while others adopt a multi-factorial approach, considering a variety of contributory aetiological factors.

Single-factor models have primarily focused on cognitive [152, 219, 223], behavioural [112, 224], or neurophysiological [221, 225–227] mechanisms. While each is comprehensive in its own right, the complexity and heterogeneity of insomnia’s various manifestations [228] - as demonstrated by the breadth of symptomology in diagnostic criteria [67–69] - likely necessitates a multi-component perspective [70, 222, 229]. Such models include, but are not limited to, the neurocognitive model [230], the hyperarousal model [225, 231], the psychobiological-inhibition model [25, 219, 223, 232], and psychophysiological [230], genetic [220], and neurobiological models [233]. Taken together, these models suggest insomnia to be aetiologically complex, likely involving multiple psychological, physiological, and neurobiological factors. Thus, positioning insomnia as a psychophysiological disorder [230, 231, 234].

1.4.1 Spielman’s three-factor model of insomnia

The broadest, most integrative, and arguably most influential of these models is Spielman’s three factor model; also known as the 3P behavioural model of insomnia, or the diathesis-stress model of insomnia [224]. This was the first fully articulated aetiological model of insomnia to be widely accepted [220], and has served as a conceptual framework for insomnia treatments - namely sleep restriction [113, 129] - as well as subsequent aetiological models. Spielman’s model posits that chronic insomnia manifests as a consequence of three interacting factors: predisposing factors, precipitating factors, and perpetuating factors (see Figure 1.2) [224].

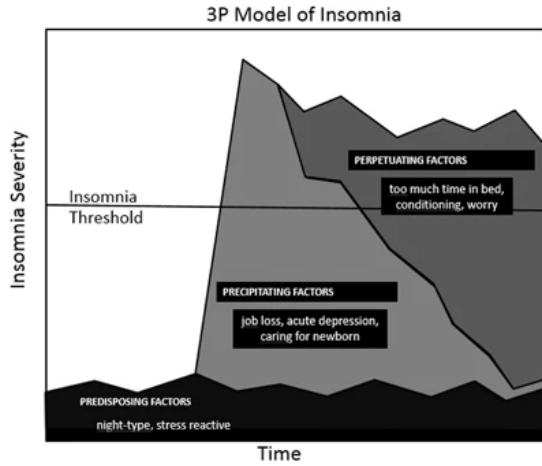


Figure 1.2: 3P Model of Insomnia – Image from Spielman & Glovinsky [235], used with permission from Springer Nature.

Several proposed models seek to expand on Spielman’s behavioural model by addressing these concepts. Frequently theorised is the role of cognition in the development and maintenance of insomnia disorder. A recent systematic review identified nine distinguishable models with a cognitive component, yielding a total of 39 cognitive factors and sub-factors relevant to insomnia’s aetiology [222]. Central to each model is the notion that arousal – cortical, cognitive, or

physiological – or an inability to de-arouse, as a result of cognitive or emotional factors, lies at the core of insomnia’s origins.

1.4.2 The neurocognitive model of insomnia

The neurocognitive model put forth by Perlis and colleagues [230] extends Spielman’s behavioural model [224] by suggesting that sleep-related stimuli can become classically conditioned triggers for cortical arousal, subsequently perpetuating insomnia [220, 230]. Perlis et al. [230] highlight evidence of high-frequency EEG activity around sleep-onset in individuals with insomnia, and postulate that this may render individuals more susceptible to environmental disruptions and less able to distinguish between sleep and wakefulness. This model further posits that this phenomenon may shed light on why individuals with insomnia commonly report discrepancies in objective and subjective measures of sleep [236].

1.4.3 The cognitive model of insomnia

Several other models [152, 237–241] posit that negative cognitive activity, such as worry or anxious thoughts, leads to physiological or cortical arousal which inhibits sleep onset [222]. Results from a recent systematic review [242] support this hypothesis, showing that pre-sleep cognitions in people with insomnia are more negatively toned than those without insomnia, and are contributory to prolonged sleep onset latency. Harvey’s cognitive model [152], one of the first to consider insomnia to be a 24-hour condition, posits that negative cognitions such as ex-

cessive worry or rumination prior to sleep onset are part of a cascading process, which ultimately leads to the maintenance of insomnia. The point of departure for the model is that people with insomnia, in response to this negative cognitive activity, will engage in maladaptive safety behaviours (e.g., extension of time in bed) intended to alleviate distress. However, elicitation of these behaviours prevents the individual from challenging their beliefs [146, 243], further perpetuating insomnia.

Together, negative cognitive activity and maladaptive safety behaviours trigger the sympathetic arm of the autonomic nervous system [152], the body's stress response [244]. Though useful for evading threats, the resultant arousal [245] is harmful to sleep, plunging the individual into an anxious state [152]. This anxiety is thought to contribute to sleep-related attentional bias, where individuals with insomnia may allocate excessive attention to perceived threats to sleep or phenomena that reinforce their sleep concerns [152, 246–248]. These can include internal cues (e.g., bodily sensations suggesting sleeplessness, or external cues (e.g., clock-watching) [152, 249]. Concurrently, several studies have demonstrated that people with insomnia attend to sleep-related words more than those without insomnia [246, 250, 251]. Harvey's model [152] further posits that this intensified attention, and the associated anxiety, leads individuals to misinterpret innocuous phenomena in a manner consistent with their concerns; an interpretive bias [252]. For example, one may interpret momentary irritability or difficulties with attention as evidence of insomnia's impact on their functioning. Recent meta-analytic findings implicate these biases as pertinent features in the development and maintenance of insomnia disorder [246]. Congruently, Harvey [152] posits that these biases, together with erroneous beliefs about sleep, and ex-consequentia reasoning – where an individual fallaciously interprets the existence of a threat as a result of their feeling anxious [253] - further exacerbate anxiety and cognitive arousal; thereby perpetuating insomnia.

1.4.4 The attention-intention-effort pathway

Espie and colleagues [223, 232] built on Harvey's cognitive model and Perlis' neurocognitive model to develop the Attention-Intention-Effort (AIE) pathway. This pathway embarks from the position of normal sleep; an automatic and involuntary process that can be disrupted by selectively attending to sleep-related cues or trying to control sleep volitionally [219] - specifically, sleep onset. Similar to Harvey's model [152], the AIE pathway puts forth that even after an ante-

cedent stressor has diminished, one’s attention may shift to the difficulty sleeping; which in turn becomes a stressor in its own right. Reinforced by historic conditioning with sleep-related cues, the associated arousal may contribute to a preoccupation with sleep. Consequently leading the individual to exert greater effort and intention to fall asleep. This notion is supported by a wealth of literature demonstrating evidence of attentional-biases towards sleep related cues in insomnia [246, 250, 251, 254]. Collectively, selective attention, intent, and volitional effort directed towards sleep are thought to underpin the cortical, cognitive, and physiological arousal associated with insomnia [223, 231], and serve as perpetuating factors for insomnia [223]. Moreover, attempts to exert volitional control over sleep may mediate the relationship between depression and anxiety [255], and predict treatment outcomes [256, 257]. Promisingly, recent evidence suggests this may be a viable target for mindfulness-based interventions aiming to alleviate insomnia and depression [258]. Broomfield and Espie [259] operationalised sleep effort via a self-report measure – the Glasgow Sleep Effort Scale (GSES) – which has been demonstrated to accurately distinguish between individuals with and without insomnia [259, 260]; emphasising the pertinence of sleep effort as a construct in the pathology of insomnia.

1.4.5 The hyperarousal model

The concept of hyperarousal is central to many contemporary aetiological models of insomnia [25, 225, 230, 231, 261]. Within this context, hyperarousal is defined as increased cognitive, cortical, emotional, or physiological arousal, present throughout the day and the night, which can act as a predisposing, precipitating, and/or a perpetuating factor for insomnia [231, 262]. The hyperarousal model corresponds with an integrative approach, attributing the manifestation and maintenance of insomnia to a nexus of physiological and psychological factors [231]. Concordant with the 3P model, the hyperarousal model posits that individuals may possess a premorbid disposition for heightened arousal and reactivity to stressors, which when coinciding with precipitating factors such as stressful life events, leads to acute insomnia [262]. This sleep disturbance may become a stressor in and of itself, becoming self-perpetuating, and leading to chronic insomnia [65, 262].

Proponents of the hyperarousal model [262] detail how the concept builds on the aforementioned models. Hyperarousal itself is thought to be sustained by classically conditioned wakefulness in response to sleep-related stimuli (e.g., the bed) [230], efforts to exert volitional control over one’s sleep [223, 232], and sleep-related anxiety [152]. This increased cognitive arousal and emotional distress – worsened by poor sleep [64] – contribute to (and are exacerbated by) attentional biases towards sleep-related phenomena [152, 246, 247] and maladaptive safety behaviours [152, 230]; thereby reinforcing a self-perpetuating cycle of arousal and sleeplessness.

1.4.6 The psychobiological inhibition model

Further developing the cognitive AIE pathway [223], Espie and colleagues have proposed the psychobiological-inhibition model of insomnia [25, 232]. This model is distinguishable from the hyperarousal model by its emphasis on an inability to de-arouse as the primary aetiological factor in insomnia, rather than hyperarousal itself. Espie [25] argues that while hyperarousal does occur in some individuals, insomnia universally involves an inability to de-arouse and inhibit wakefulness, as a result of the cognitive processes outlined in the AIE pathway: attending to sleep-related stimuli, possessing intent to sleep, and exerting effort to sleep [25, 223, 232]. Rather than facilitating sleep, these cognitions and beliefs inhibit cognitive and physiological de-arousal, preventing sleep onset, and perpetuating insomnia [25]. In fact, Espie [25] posits that a good sleeper is unlikely to think of sleep very much at all, and instead passively allows it to occur. To paraphrase John Rambo, the psychobiological inhibition model posits that, to a good sleeper, sleepin’ is as easy as breathin’.

1.4.7 Potential mechanisms underpinning existing models

More recent models, armed with contemporary evidence, have suggested potential mechanistic underpinnings to their own theories of insomnia’s aetiology. Both the hyperarousal [231, 262] and psychobiological inhibition [25, 219] models posit that cognitive and physiological arousal may interfere with actions of the ‘flip-flop’ switch – inhibiting its ability to govern the transitions between sleep and wake – potentially via neurochemical mechanisms [231, 262]; supporting the concept of insomnia being a psychophysiological disorder [231, 261]. For example, an individual with insomnia may have come to perceive sleeplessness, or sleep-related

stimuli, as predictable threats, thus exposure to these will activate the body's stress-response [65, 152, 245]. This, in turn, may override the pressure of the homeostatic sleep drive, inhibiting the de-arousal arm of the 'flip-flop' switch, and prevent the transition from wake to sleep [25, 219]. While this is certainly a useful adaptation in situations where nocturnal alertness is necessary (e.g., evading a predator, or handling an emergency), it can become maladaptive within the context of insomnia disorder.

Given the stress-related nature of insomnia – and its pertinence in the regulation of sleep and wake [245] - the hypothalamic-pituitary-adrenal (HPA) axis is implicated in its aetiology [36, 64, 65, 70, 245, 262]. Cortisol is the end product of HPA axis activity [245, 263]. Levels fluctuate in accordance with one's habitual sleep-wake cycle, rising rapidly and peaking shortly after waking, then gradually decreasing throughout the day, reaching a nadir in the early night [264]. A systematic review and meta-analysis of case-control studies found cortisol levels to be higher, throughout the day and night, in people with insomnia compared to controls [65]; supporting the notion of insomnia being a 24-hour disorder related to hyperarousal [231, 262]. Additional studies on other correlates of HPA axis activity, such as heart rate variability, and measures of cardiovascular and metabolic health, further support this association [265–268]. Chronic activation of the HPA axis, potentially via a self-perpetuating nexus of psychological and neurophysiological factors [65, 262, 269], may interfere with the regulatory processes of sleep via increasing cognitive, cortical, and physiological arousal [231, 262], or via preventing de-arousal across these domains [25]. Evidence also suggests that impairment to sleep may increase the sensitivity of the HPA axis, potentially via emotional and neurochemical mechanisms [270]. Thus, activation of the human stress response may be epiphenomenal to insomnia, while also acting as a predisposing and perpetuating factor [65].

Common across all models is a shared focus on the concept of arousal, framing insomnia as a psychobiological disorder. While different presentations of insomnia arise from varying factors, the consensus suggests that an interplay of psychological and physiological elements is an omnipresent feature in insomnia's aetiology. Rather than determining a single 'most appropriate' model, it is perhaps best to consider the relevance of each model to specific circumstances [220]. Relatedly, existing models have largely been informed using data from samples of participants from the general population, and their applicability to other populations (e.g., stroke survivors) is less understood. Thus, future research should explore cognitive, genetic, physiological, and neurological factors, across differing presentations and populations, to elucidate insomnia's multifaceted aetiology.

1.5 A brief overview of stroke

‘The healthy subject is taken with sudden pain; he immediately loses his speech and rattles his throat. His mouth gapes and if one calls him or stirs him, he only groans but understands nothing’

— Hippocrates [271]

1.5.1 Defining stroke

Stroke has been recognised as a medical phenomenon since antiquity, with the earliest accounts appearing in the writings of Hippocrates [271]. Hippocrates referred to the condition as *apoplexy*, meaning to ‘*strike down with violence*’ [271, 272]. This was the medically preferred term until the early 20th century, despite recorded use of the word ‘stroke’ as a lay term for apoplexy from as early as the 16th century [272]. Early phraseology appears influenced by religiosity and belief in the supernatural. Descriptions of stroke as ‘*a dreadful visitation*’, ‘*summons*’, ‘*the stroke of God’s hande*’, ‘*the mortal stroke*’, or the ‘*stroke of justice*’ [272] convey how the sudden and violent onset of symptoms was perceived as being indicative of divine judgement.

Contrary to such beliefs, stroke is a heterogenous condition, with upwards of 150 potential aetiologies [273]. As medical autopsies became popularised in the 17th century [274], Swiss born physician-anatomist Johan Jakob Wepfer first observed the causal link between apoplexy and cerebral haemorrhage [275–277]. A century later, Italian anatomist Giovanni Morgagni observed differences in autopsies of apoplexy patients [278], categorising cases as ‘*sanguineous apoplexy*’ (those containing blood) or ‘*serous apoplexy*’ (those containing only cerebral spinal fluid). This discovery established the basic distinction of cerebral haemorrhage and cerebral ischaemia, which has endured to the present day. ‘Sanguineous apoplexy’ became haemorrhagic stroke, and ‘serous apoplexy’ became ischaemic stroke [276]. The former describes a scenario where the blood supply to the brain is disrupted due to a ruptured blood vessel [279], resulting in bleeding within the brain itself (intracerebral haemorrhage), or between the membranes surrounding the brain (subarachnoid haemorrhage) [280]. Ischaemic stroke refers to disruption to the brain’s blood supply via a blockage, either arising within the affected vessel itself (thrombosis), or travelling there from elsewhere in the body (embolism) [281]. Ischaemic strokes account for around 80% of all strokes [282, 283],

and 70% of stroke-related fatalities [283]. The most recently updated definition of stroke maintains the distinction first observed by Giovoanni Morgagni, characterising it as 'an episode of acute neurological dysfunction presumed to be caused by ischemia or haemorrhage, persisting ≥ 24 hours or until death' [284, p. 2066].

1.5.2 Stroke epidemiology

Despite having been known to medicine for over two millennia, stroke remains the second leading cause of death worldwide [285, 286] - accounting for 11% of annual deaths [287] - and third-leading cause of burden of disease [285, 286]. Globally, it is estimated that there are 12.2 million incident strokes annually, and 101 million prevalent cases of stroke [285]. In the European Union, respective figures are 1.12 million and 9.53 million [288], and in the UK $>100,000$ [289] and 1.3 million [290]. In 2016, the global estimated lifetime risk of stroke after the age of 25 was approximately 24.9%; having increased from 22.8% in 1990 [291]. This risk is slightly higher in females (25.1%) [291], despite higher global annual incidence rates in males [292]. This disparity may simply reflect differences in life expectancy [292], though behavioural, biological, and medical inequalities may also play a role [293]. Results from the Global Burden of Disease Study indicate a 70% increase in the overall number of strokes over the past 30 years, with most stroke-related burden and death occurring predominantly in lower-income and lower-middle-income countries [285].

In high-income countries, public health efforts to prevent stroke have been effective, and *age-standardised* incidence of stroke continues to decline [283, 285, 291, 294, 295]; emphasising that stroke is preventable. A systematic review of longitudinal studies reported annual reductions in age-specific stroke incidence in high-income countries, of 1.0% to 1.5% over the preceding 30 years [295], and by 6% every five years within the UK. Nevertheless, increasing and ageing populations are expected to contribute to overall increases in the absolute incidence of stroke [283, 285, 288, 294–297]. Recent analysis of European data demonstrates that, despite marked reductions in age-standardised stroke incidence and prevalence between 2010 and 2019, absolute numbers have increased across Europe; particularly Western Europe [283]. Projected increases vary widely; Wafa et al. [288] estimate a 3% increase in incidence and a 27% increase in prevalence across Europe by 2047, assuming stable population sizes. Less conservatively, the Stroke Alliance for Europe projects a 34% increase in incidence by 2035 [297]. Specific to the UK, Li et al. [295] predict that, should incidence rates of age-specific stroke

remain stable, the absolute number of strokes would increase by 66% by 2045, and even if age-specific incidence continues to decline in line with current trends (6% every 5 years), the overall incidence of stroke will still increase by 13%. Estimates from King et al. [296] are much higher, with authors predicting a 59% increase in annual incidence and a 123% increase in prevalence between 2015 and 2035. However, these estimates are based on subjective predictions from consultations with stroke experts rather than extrapolation of trends in epidemiological data. Li and colleagues propose that for the absolute number of strokes in the UK to remain stable, efforts will be needed to increase the rate of decline in age-specific incidence to 8% every 5 years [295].

In addition to preventative campaigns targeting risk factors [298], timely and effective acute stroke care [299, 300] and prevention of recurrent strokes [301, 302] have significantly curtailed immediate stroke fatality rates [285, 294]. Consequently, the total number of deaths related to stroke in high-income countries has reduced by approximately 20% over the past two decades [287]. Analysis of population-based data from urban dwelling stroke survivors in South London suggested that mortality after ischaemic stroke decreased by 2.4% per year between 2000 and 2015 [303]. Whilst news of increased survival rates after stroke is very welcome, survivors commonly face ongoing care needs. Stroke is a leading cause of disability, accounting for 125 million disability adjusted life years globally [285]. Stroke survivors often experience impairments in communication [304], physical functioning [305–307], cognitive ability [308], and psychiatric well-being [309]. Taken together, concurrently increasing absolute numbers of incident strokes and survival rates will inevitably increase the overall societal impact of stroke. Wafa et al. [288] posit that if rates of post-stroke disability remain stable, increases in stroke incidence will correspond with a commensurate increase in demand for rehabilitation and long-term care. Older age, however, is a known risk factor for post-stroke disability [310]. Therefore, as older adults are expected to comprise a larger proportion of strokes in the future than at present [288], increases in the societal impact of stroke may in fact exceed increases in stroke incidence. Moreover, proportional increases in the retiree population mean that the economic burden of stroke must be shouldered by a proportionally shrinking working-age population, potentially straining the economy and healthcare system. King et al. [296] estimate a 194% increase in the societal cost of stroke in the UK over 20 years, from £26 billion in 2015 (1.4% of GDP) to £75 billion in 2035 (2.9% of GDP). However, given that these estimates are based on subjective predictions, they may be exaggerated. Nonetheless, significant challenges

lie ahead, and the focus of clinicians and researchers will likely continue to shift, from mortality and acute care to morbidity and rehabilitation. Future research on preventative and rehabilitative interventions could go far in improving the lives of stroke survivors and ameliorating the economic and societal costs of stroke.

1.6 Sleep after stroke

Historical accounts of stroke describe cynical and pessimistic prognoses for stroke survivors [272]. The frank nature may be reflective of the reality of the time, as without effective treatment or rehabilitation, the afflicted either ‘died, got better, or lingered on miserably’ [272, p. 336]. Stroke care and rehabilitation predominantly adopted a biomedical approach [311], primarily concerned with attending to the immediate somatic aspects, while often neglecting the long-term challenges faced by stroke survivors. Today, as a result of advancements in stroke rehabilitation and changing stroke epidemiology, the ethos has somewhat shifted towards ensuring people live well after stroke. One facet of life after stroke which is frequently impacted [312–317], but historically overlooked [317], is sleep.

1.6.1 The bi-directional relationship between sleep and stroke

Impairments to sleep are more prevalent after stroke than in the general population [318, 319], affecting up to 70% of stroke survivors [320], with varying severity throughout different phases of recovery [321]. While the present thesis focuses on insomnia, it is just one of six major categories of sleep disorders, as outlined in the ICSD-3: (i) insomnia disorders, (ii) sleep-related breathing disorders, (iii) central disorders of hypersomnolence, (iv) circadian rhythm sleep-wake disorders, (v) parasomnias, and (vi) sleep-related movement disorders [68]. Studies employing actigraphy and polysomnography (PSG) indicate difficulties with sleep initiation, sleep maintenance, and sleep efficiency in the weeks immediately following stroke, which often persist into the chronic phase [313, 322–324]. Sleep-related breathing disorders, particularly central (CSA) and obstructive sleep apnoea (OSA), are highly prevalent after stroke – affecting up to 70% of stroke survivors [321, 325, 326]. Hypersomnia – excessive daytime sleepiness – is also common, particularly in the acute stages of stroke [312], and most often following a thalamic stroke [327]. Circadian function may also be impaired, with results of a system-

atic review demonstrating reduced melatonin secretion in stroke survivors versus controls, as well as pre- to post-stroke changes in objective and subjective measures of circadian rhythms [318]. Other evidence highlights stroke’s influence on parasomnias [2], and suggests that sleep architecture [323], and dreaming [328, 329] may also be affected. For example, Bischoff & Bassetti [328] describe a case-report of a patient who experienced Charcot–Wilbrand syndrome – a total loss of dreaming – for three months after a bilateral occipital stroke, despite unimpaired REM sleep. Similarly, Sagnier et al. [329] describe the cases of two adults who experienced lucid dreaming – being consciously aware of the fact you are dreaming whilst you are asleep [330] – after thalamic strokes. Sleep-related movement disorders, such as restless-leg syndrome (RLS) and periodic limb movement disorder (PLMD) are also common after stroke. Characterised by intense sensations of urgently needing to move one’s legs whilst falling asleep, or involuntary jerks and movements of one’s limbs whilst asleep, PLMD and RLS are estimated to affect between 32% and 48%, and 10% and 14% of stroke survivors, respectively [321], and prevalence appears to trend upwards throughout recovery [321].

Emerging evidence suggests a bi-directional relationship between stroke and multiple categories of sleep disorders, with sleep disruption not only arising as a consequence of stroke, but also serving as a risk factor for stroke occurrence and recurrence [312, 314, 318, 331, 332]. Phenomena such as abnormal sleep durations, as well as conditions such as sleep disordered breathing, insomnia, and circadian rhythm disorders, have been linked to an elevated risk of stroke [318, 331, 333–343], and increased stroke severity [338]. While both short and long durations have been associated with increased stroke risk [314] and impaired recovery [2], the risk associated with extended sleep appears greater [344]. Sleep apnoea, in particular, is a commonly cited modifiable risk-factor for stroke [312]. Meta-analytic evidence suggests that OSA may be associated with a 2.1x greater risk of stroke [345]. However, treatment does appear effective in reducing this [346]. There is also strong evidence of circadian rhythmicity in the timing of stroke [314, 347], with incidence peaking in the morning – a phenomenon commonly referred to as ‘Wake-Up Strokes’ [348].

In addition the risk of first-time incidence of stroke, Hale et al. [331] report in a recent systematic review, that impaired sleep after stroke may increase one’s risk of stroke recurrence. Correspondingly, guidelines from the American Heart and American Stroke associations [349], along with a statement from a European task force of sleep and stroke experts [350], underline the importance of investigating sleep disorders in stroke care and emphasise sleep as a viable target for efforts in preventing recurrent cardiovascular events. Although the association between

sleep disorders and stroke is evident, the precise mechanisms remain unclear, and there remains a paucity in research exploring insomnia, restless leg syndrome, and periodic limb movement disorder, after stroke [350]; a paucity which this body of works seeks to begin to address.

Some authors [351, 352] have suggested that dysregulation of sleep and circadian processes may initiate harmful physiological mechanisms such as inflammation, increased cortisol secretion, and activation of the sympathetic branch of the autonomic nervous system (see 1.4.7), which in turn may contribute to increased risk of cardiovascular events. This theory is supported by the aforementioned findings by Dressle et al. [65] - indicating increased HPA axis activity and cortisol secretion in people with insomnia – and thus aligns with the hyperarousal perspective of insomnia. Moreover, observational studies have demonstrated circadian rhythmicity in the timings of stroke onset, with most occurring in the morning, a smaller peak in the evening, and a nadir during the night [347, 353, 354]; suggesting a possible role of the circadian process. Alternatively, the relationship between sleep and stroke may be explained by behavioural mechanisms [3]. Impairment to sleep is related to a greater propensity to engage in behaviours deleterious to cardiovascular health, such as poorer nutrition [355] and increased caloric intake [356], as well as sedentariness, smoking, consumption of alcohol, and lack of exercise [357].

1.6.2 Sleep and recovery after stroke

Not only does impaired sleep after stroke correlate with an increased risk of recurrence [331, 358], but evidence from animal and human models suggests it may also impair recovery. Experimental research has shown that rats deprived of sleep after stroke experience significant increases in lesion volume, cell damage, and the expression of genes that inhibit neuronal recovery [359, 360]. Conversely, animals who are prevented from sleeping prior to stroke, and hence sleep more afterwards, demonstrate lower lesion volumes [361, 362]. Additional research has demonstrated that the provision of GABA receptor agonists [363, 364] and melatonin [365] not only leads to better sleep, but also neural repair and reduced infarct volume in rats. Taken together, these findings suggest sleep may be neuroprotective after stroke, and thus impaired sleep may be inhibitory to recovery.

While experimental evidence is not available in humans for obvious reason, similar patterns emerge from observational studies. Findings highlight associations between impaired sleep and disability, impaired functional recovery, mortality, cognitive impairment, and psychological distress after stroke [366–372]. While the underlying processes are unclear, sleep is essential for neural regeneration and repair [373], processes which are critical for recovery after stroke [374]. The relationship between sleep, neuroplasticity, and stroke recovery implies that impaired sleep after stroke may impair recovery via compromises to sleep potentiated neuroplasticity [318, 375].

1.6.3 Insomnia after stroke: Prevalence and aetiology

Insomnia disorder is a particularly pernicious, yet under-researched [317, 350] outcome after stroke, associated with reduced quality of life [376], suicidality [377], and a 3.31- and 6.75-fold increased risk of anxiety and depression, respectively [369]. A recent cross-sectional study of over 46,000 participants in Canada suggested that the risk of developing insomnia was 53% greater in people with a history of stroke versus those without [378]. Meta-analytic findings [321, 379, 380] estimate that between 32.2% and 59.2% of stroke survivors experience insomnia, depending on which diagnostic criteria are employed [321, 379], and stage of recovery [321].

Insomnia’s aetiology after stroke remains unclear but may have neurological underpinnings. Hasan et al. [321] postulate that variation in insomnia prevalence across phases of recovery may be explained by neural repair and associated functional recovery, leading to the consolidation of sleep. Corroborating this speculation are myriad studies suggesting that lesion location may influence the likelihood of insomnia [314, 320, 375, 381–388]. The thalamus, in particular, is frequently implicated [320, 382–384, 387]. Helquist et al. [384] compared neuroimaging data from 12 case studies of individuals with insomnia after acquired brain injury with normative examples of stroke lesions. Results indicated that all 12 lesions were functionally connected to thalamus and demonstrated insomnia after stroke to be associated with significant alterations in thalamic functional connectivity – it should be noted however, that the quality and results of this study have yet to undergo peer-review. Earlier research suggests that damage to the thalamus may lead to impaired ability to produce sleep spindles [320], suggesting possible impairment to non-rapid eye movement (NREM) sleep [389]; further substantiated in findings by Mims & Kirsch [387]. Correspondingly, a

recent prospective study comparing stroke survivors (3-months post-stroke) with healthy controls reported that poor sleep efficiency after stroke was associated with comparative reductions in volumes of the thalamus, hippocampus, and amygdala [383].

Similar to insomnia in the general population however [390], studies exploring the neural correlates of insomnia in stroke return heterogeneous findings, and several other brain regions have also been implicated; namely the brainstem [314, 320, 375], basal ganglia [382, 385], and visual [388] and cognitive-emotional [385] processing centres. Reductions in levels of serotonin and norepinephrine [382], and reductions in functional connectivity [388] have also been demonstrated to correspond with post-stroke insomnia. However, beyond neurological damage, elements such as stroke severity, co-morbidities, environmental factors, pharmacology, and psychiatric wellbeing, have also been implicated in the development and maintenance of insomnia after stroke [2, 318–321, 347, 369, 375, 391, 392]. In keeping with the conceptualisation of insomnia as a psychobiological disorder, Wang et al. [388] posit that an interaction between anxiety and/or depression [74], and disrupted secretion of 5-hydroxytryptamine (serotonin) caused by localised lesions in regions such as the thalamus [382], leads to insomnia after stroke. However, this remains largely speculative, and much remains to be explored. In actuality, empirical understanding of insomnia’s aetiology after stroke remains in its infancy, and a multifactorial interaction comprised of functional and organic influences is most likely. While the aetiological models described earlier (see § 1.4) provide useful points of departure, their applicability after stroke remains to be confirmed; something it is hoped that this thesis can contribute to. Moreover, inferences about lesion location and insomnia related outcomes should be drawn carefully, as associated impairments to functionality or physiology are not necessarily a direct result of a focal lesion, but often disruption to functional connectivity and metabolism of structures and regions downstream of the damaged area; a phenomenon known as diaschisis [393]. Imaging studies employing both lesion-based and functional methods [394, 395], that also account for behavioural, psychiatric, and pharmacological factors, are necessary to derive comprehensive models of insomnia’s aetiology after stroke.

1.6.4 Approaches to treating insomnia after stroke

To date there are no guidelines for the treatment of insomnia that are specific to stroke [396]. While some studies do advocate for the use of pharmacological intervention [397–399], this contravenes general guidelines outlining CBT-I as the first line of treatment for insomnia [100]. Additionally, non-pharmacological intervention is generally preferred by patients [106, 107], although this has never been explored after stroke - a population gap that the present thesis aims to address. Moreover, the use of some hypnotic medications is thought to be associated with poorer functional outcomes, and increased mortality after stroke [400], although the latter is potentially explained by bias introduced from potential confounds [401].

Though CBT-I is known to be safe and efficacious in the general population [70, 83, 98, 100, 161], its utility after stroke, and in the context of acquired brain injury more broadly, is yet to be fully established. To date, seven studies – two RCTs, three pilot RCTs and two single case experimental design (SCED) trials – have examined the efficacy of CBT-I in samples of stroke survivors [402–408]. Five of these studies involved samples comprised exclusively of stroke survivors [402, 403, 405–407], while two included broader acquired brain injury populations, with stroke accounting for 65% of the sample in Ford et al. [404] and 57% in Ymer et al. [408]. Evidence suggests that CBT-I can yield short-term improvements in symptoms of insomnia after stroke, although with limited longevity [402–408], typically less than is observed in the general population [178, 179]. While preliminary results are promising, several studies suggest that stroke-related impairments may impede one’s ability to engage with CBT-I’s protocols [203, 403, 405], particularly with regard to alternative modes of delivery. Ford et al. [403, 404] and Fleming et al. [402] explored interventions delivered entirely, or primarily, online. Although demonstrating significant improvements in insomnia and mood, some issues did arise. Approximately 19% of participants in Fleming et al. withdrew from the study, predominantly those with elevated levels of psychiatric distress. Thus, alternative considerations may be indicated where depression and/or anxiety exist co-morbidly with insomnia after stroke.

Qualitative investigation of stroke survivors’ experiences of digital CBT-I found that participants felt their cognitive impairments contributed to difficulties engaging with the protocol [203]. Participants reported that, were it not for the support of their partners, they would have been unlikely to have been able to access the program, suggesting that the accessibility of digital CBT-I may be

limited in specific sub-groups of the stroke population, possibly due to proving too taxing for people living with cognitive impairment. Moreover, participants felt that aspects of the programme were not relevant, or not appropriate, to stroke survivors [203]. In particular, participants had concerns around stimulus control, and getting out of bed at night with mobility issues. Others found relaxation techniques, such as progressive muscle relaxation, challenging due to stroke related disabilities. Thus, stroke specific adaptations to the standard CBT-I protocol may be necessary to increase the accessibility of CBT-I to people living with impaired mobility or cognition after stroke. Ymer et al. [408] did previously adapt CBT-I for use after acquired brain injury, by increasing the structure of each session, using repetition and concrete concepts, providing greater support from clinicians, and including easy-to-read handouts. Treatment was delivered either in-person or via telehealth. Secondary data analysis revealed that treatment response to CBT-I in acquired brain injury was not moderated by the mode of delivery [409], suggesting that adapting treatment protocols to meet the needs of stroke survivors is feasible for in-person or digital CBT-I.

Purely behavioural interventions may prove to be more acceptable, but this is yet to be examined. Among the principal components of CBT-I, sleep restriction (see § 1.3.3) and stimulus control (see § 1.3.2) are generally considered the most potent [120, 148], and are explicitly behavioural in nature. While they generally are considered appropriate for use as monotherapies, they often require adaptation or increased support in populations with additional needs [98], such as stroke survivors [203]. Elements of each may appear counter-intuitive, overly burdensome, or even unsafe to people recovering from stroke. For instance, getting out of bed at night may be challenging or anxiety provoking for people with impaired mobility or vision after stroke [203]. Similarly, restricting time in bed may temporarily worsen fatigue [410], or conflict with individuals' expectations that sufficient rest is essential for recovery. Exploration of how these concerns can be addressed – in ways that preserve the therapeutic mechanisms driving change – is clearly indicated to increase the accessibility of behavioural interventions for insomnia after stroke.

Finally, several of the referenced studies [403–407] employed the Insomnia Severity Index (ISI) [150] and the Pittsburgh Sleep Quality Index (PSQI) [411]. While these measures were recently validated for use as screening tools after stroke [412], they were only done so in Chinese speaking samples, and have not been specifically validated as outcome measures. Moreover, while Youden's J values from Niu et al. [412] demonstrated optimal diagnostic threshold of the ISI to be the same after stroke as in the general population, the optimal threshold

for the PSQI was found to be ≥ 9 , not ≥ 5 , as is conventional. Studies employing the PSQI as an outcome measure after stroke, which adopt the conventional threshold, run the risk of misinterpreting results. Reductions in PSQI score that fall below nine, but not five, may erroneously be considered as clinically insignificant. Similarly, inappropriately using the lower threshold in a sample of stroke survivors may exaggerate estimates of sleep disruption at baseline. Resultantly, clinically insignificant changes (e.g., a score of 6 at baseline moving to a score of 4 at post-treatment) may be interpreted as significant, overestimating the effectiveness of the intervention. Thus, the need for studies investigating stroke specific adaptations, or more parsimonious approaches to CBT-I, which employ suitably validated screening and outcome measures is indicated. The present thesis will address these issues directly.

1.7 Thesis aims and objectives

Given the ubiquity of impaired sleep after stroke, and the corresponding deleterious consequences, our understanding of post-stroke insomnia must be expanded. Several key areas, outlined throughout this introduction, are of particular importance. Firstly, while quantitative investigations of insomnia's relationship with post-stroke outcomes are growing in number, there are no studies examining accounts of stroke survivors' subjective experiences. Such studies are necessary for understanding the personal impacts of insomnia after stroke, and to elucidate insights that are potentially inaccessible to positivist approaches [413]. Relatedly, knowledge of stroke survivors' experiences of insomnia may shed light on the applicability of existing aetiological models within the context of stroke. Secondly, insomnia's relationship with so many negative outcomes after stroke underlines the need to identify stroke survivors in need of support with sleep, and the inferential limitations in existing studies (discussed above) stress the necessity for doing so via tools validated for use in stroke. Finally, suggestions that existing treatments may be inaccessible, or too taxing, to some sub-groups of stroke survivors [203, 403, 405] emphasise a need to consider alternatives or adaptations. To that end, this thesis seeks to address four principal questions with regards to the impact, assessment, and management of insomnia after stroke:

1. What is the experience and impact of insomnia after stroke?
2. What are stroke survivors' experiences and preferences relating to treatment for insomnia?

3. How can we accurately identify which individuals are living with insomnia after stroke?
4. How can we best go about supporting people with insomnia after stroke?

1.8 Thesis outline

The above questions are addressed, in order, in the following chapters.

Chapter 2 addresses questions 1 and 2, exploring the experiences of stroke survivors living with insomnia, and investigating their experiences and preferences relating to treatment. Interpretive phenomenological analysis (IPA) [414] was employed to analyse data obtained from semi-structured interviews, seeking to garner insight into the meaning participants derive from their experiences. Establishing a foundation to build from, this study informed the development of subsequent chapters.

Chapter 3 evaluates the validity and reliability of the Sleep Condition Indicator [415], a brief measure of insomnia, in a sample of self-reported stroke survivors. Receiver operating characteristic analysis was used to determine the overall accuracy of the full 8-item and a short 2-item version of the scale, when discriminating between stroke survivors with and without DSM-5 insomnia disorder. This chapter was published in the *BMJ: Neurology Open* [416].

Chapter 4 presents a systematic review – published in the *Journal of Sleep Research* [417] - of studies examining the efficacy of stimulus control and sleep restriction (isolated and combined) in older adults. While we initially set out to examine these treatments in stroke, the literature doing so was deemed too sparse, and thus older adults were considered an appropriate adjacent population from which to embark.

Chapter 5 details a protocol for a single-case experimental design study seeking to elucidate the feasibility and acceptability of a short behavioural intervention for insomnia in stroke survivors, informed by the results of previous chapters.

Finally, Chapter 6 summarises and discusses the content and findings of all preceding chapters, considering the associated clinical implications, and suggesting directions for future research.

Chapter 2

The lived experiences and treatment preferences of stroke survivors living with insomnia: An interpretive phenomenological analysis

2.1 Abstract

Background: Insomnia is common after stroke, and associated with various negative outcomes, yet has historically been understudied. Understanding the experiences and treatment preferences of stroke survivors living with insomnia is important for guiding clinical practice and developing therapeutic interventions.

Aims: This study aimed to garner insight into the subjective experiences of insomnia after stroke, the meaning stroke survivors derive from these experiences, and their preferences pertaining to treatment.

Methods: Semi-structured interviews were conducted with 10 stroke survivors in the UK (aged 30-65 years; $\bar{x} = 50.90$, $SD = 10.71$). Screening forms assessed insomnia, mental health, and stroke severity. Interviews were transcribed verbatim, and analysed ideographically via Interpretive Phenomenological Analysis, generating *Personal* and *Group Experiential Themes*.

Results: Interviews naturally bifurcated into distinct discussions around experiences of insomnia after stroke, and experiences and preferences relating to treatment. Regarding the former, analysis derived two group experiential themes: (1) *Sleep is fluid, and changes throughout recovery*, and (2) *Insomnia makes everything harder*. Participants' sleep was impacted immediately after stroke – exacerbated by the hospital environment – and contributed to detriments in psychological, physical, social, and cognitive wellbeing; often in a bi-directional manner. Three group experiential themes were derived relating to treatment: (1) *Sleep isn't taken seriously after stroke*, (2) *Non-pharmacological intervention is preferred*, and (3) *Previous attempts at improving sleep have not worked*. Participants expressed dissatisfaction with the support they received for sleep, justified their aversions to pharmacological intervention, and lamented the loss of previously effective strategies for managing sleep.

Conclusions: This study is the first to qualitatively examine the lived experiences and treatment preferences of stroke survivors living with insomnia. Results paint a complex picture of post-stroke insomnia, which evolves throughout recovery and involves a nexus of varied and often bi-directional influences. Derivation of a potential model may inform research into insomnia's aetiology after

stroke. Participants' accounts highlight the considerable impact insomnia has on participants' wellbeing, identify shortcomings in clinical understanding and management of sleep after stroke, and emphasise the importance of affording people autonomy in their own care.

2.2 Introduction

Sleep is commonly disrupted following stroke [2, 312, 314, 315, 347, 358], yet has historically been overlooked in clinical and research contexts [317, 320, 350]. Emerging evidence suggests a bi-directional relationship [2, 312, 314, 315, 318, 382, 418], with impaired sleep presenting as both a risk-factor for, and a consequence of, stroke. Recent meta-analyses have indicated impaired sleep to not only be associated with an increased risk of first-ever stroke [341, 343, 419–421], but also recurrent stroke [331]. Moreover, quantitative findings indicate relationships between post-stroke sleep disruption and a range of various negative outcomes, including delirium [422], poorer functional recovery [367, 368, 375, 382, 391, 423], disability [368, 369], psychological distress [369, 424, 425], and cognitive impairment [321, 423, 426].

Particularly common, and deleterious, after stroke is insomnia disorder [321, 379, 380]. Broadly defined as difficulties initiating, maintaining, or returning to sleep, that impact an individual's daily functioning [67], insomnia disorder has an estimated point-prevalence in the general population of approximately 10% [85, 89]. Following stroke, however, estimates of prevalence are considerably higher, ranging from 32.21% to 59.16%; depending on which diagnostic criteria are employed, and phase of recovery [321, 379, 380]. Generally, insomnia appears more prevalent in the acute phase of stroke, declining slightly as recovery progresses [321, 380]; not, however, to levels commensurate with the general population [85, 89]. Despite quantitative research demonstrating its considerable impact (outlined in § 1.6), insomnia has received limited research focus relative to other sleep disorders after stroke [350]. There exists a particular paucity of subjective accounts from stroke survivors, of their experiences of post-stroke insomnia, and the personal significance this has for them. Qualitative explorations of these topics offer a means of discerning nuanced insights into insomnia after stroke, which are potentially inaccessible to other methods [413], and may shed light on potential underlying mechanisms and circumstances.

While our understanding of treatment approaches to insomnia in the general population is robust – with CBT-I considered the first-line of treatment [100] - the optimal approach following stroke remains to be confirmed [2, 427]. Early investigation of CBT-I suggests it to be acceptable, feasible, and efficacious after stroke [402–408]. However, the longevity of improvements remain unclear, and several studies have suggested that stroke-specific adaptations may be necessary [203, 403, 405]. Qualitative investigation of stroke survivors’ experiences of digital CBT-I [203] indicated that some elements of the treatment may be inappropriate, and suggested that stroke-related impairments may limit treatment accessibility.

Relatedly, people’s perceptions of their condition and associated treatments are known to influence treatment satisfaction, adherence rates, and treatment outcomes [428–433]. Negative perceptions may contribute to non-adherence [428]; not only reducing the cost-effectiveness of an intervention due to wasted resources, but potentially impeding clinical outcomes [434]. These findings emphasise the importance of considering patient preferences when developing and evaluating interventions [433]. To date, the preferences and concerns of stroke survivors pertaining to treatment for insomnia after stroke have never been explored, and qualitative investigations into the matter are indicated.

2.2.1 Aims and objectives

The current research aims to garner, by means of qualitative analysis, insight into the experiences of individuals living with insomnia after stroke, the personal significance of these experiences, and corresponding perspectives and preferences regarding treatment options. By elucidating the experiences and preferences of stroke survivors living with insomnia, alongside underlying beliefs and mechanisms, we hope to inform the development of tailored interventions for post-stroke insomnia in the future.

2.3 Methods

The present study is reported in accordance with the Standards for Reporting Qualitative Research framework [435].

2.3.1 Interpretive phenomenological analysis (IPA)

IPA [414] was considered the most appropriate means with which to address the present aims. Following an interpretivist research paradigm and underpinned by a critical realist epistemological stance, IPA allows researchers to derive insight into participants' experiences by moving beyond the descriptive nature of thematic analysis and encouraging researcher interpretation. The fundamental focus of IPA is on understanding how participants make sense of their experiences, and what meaning they derive from them [414, 436]. Hence why, in the present study, participants were regarded as experts on their own experiences. Analysis therefore remained grounded in the data to limit contamination by researchers' preconceptions, whilst remaining cognisant of the 'double hermeneutic'; the notion that the researcher is making sense of the participant's endeavours to make sense of their own experiences [414].

2.3.2 Recruitment and selection criteria

UK-based adults (≥ 18 years) with a self-reported history of stroke, and who met criteria for DSM-5 insomnia disorder [67] were recruited between February and October 2023. Participants were not excluded based on the time that had passed since their most recent stroke, nor the type or number of strokes they had experienced.

Participants were recruited via advertisements on social media (Twitter/X), and by contacting participants from a previous study, who had indicated interest in participating in future research and consented to being contacted. Each participant received a £6 Amazon voucher (or a donation of equivalent value made in their name to a charity of their choice) as a token of gratitude for their contribution.

Congruent with guidelines for sample sizes in IPA [436, 437] and Reid et al. [438]'s argument that 'less is more', we aimed to recruit ten participants. This was deemed sufficient to allow for a deep and interpretive analysis – as opposed to a shallower and more descriptive analysis being necessitated by a larger sample – whilst remaining broad enough to capture nuances within the population.

2.3.3 Materials and data collection

All potential participants were emailed a link to a screening form, hosted on Qualtrics (<https://www.qualtrics.com>), which gathered data pertaining to general demographics, and history of sleep and stroke. Embedded in screening forms was a comprehensive sleep history questionnaire [439], which was used to assess for insomnia disorder. All eligible participants were provided with a second online form, which supplemented the sleep history questionnaire with the Sleep Condition Indicator (SCI) [415], and included measures of anxiety, depression, and stroke severity, to characterise the sample.

Anxiety was measured via the Generalised Anxiety Disorder scale (GAD-7) [440], a 7-item self-report measure that quantifies anxiety on a continuous scale scored from 0-21, where higher scores denote greater severity of anxious symptoms. Although the validity of the GAD-7 after stroke has never been confirmed, the scale is widely used in primary care, and has been used in stroke populations previously [441, 442].

Depression was measured via the Patient Health Questionnaire (PHQ-9) [443], a 9-item self-report scale scored from 0-27, where greater scores denote greater severity of depressive symptoms. The PHQ-9 has been demonstrated to be a valid when discriminating between stroke survivors with and without depression [444].

Stroke severity was measured via the Stroke Impact Scale - Short Form (SIS-SF) [445], an 8-item self-report scale designed for use after stroke. Possible scores range from 5-40, with lower scores denoting greater severity of impact from stroke.

Secondary questionnaires also queried participants' preferred date/time and method of participation – Zoom, Microsoft Teams, or Telephone. Participants were encouraged to let researchers know if none of these options were suitable, and alternative arrangements could be made. An in-person interview was discussed with one participant, with whom contact was later lost. Interview invites were then sent via email, considering participants' preferences.

2.3.4 Procedure

In accordance with IPA guidelines [414], data were collected via semi-structured interviews. Eligible participants met individually with the lead author, a Post Graduate Researcher with formal training in IPA, via videoconferencing software (Zoom, Microsoft Teams). Videoconferencing is considered a viable method of collecting qualitative data [446], including among populations with acquired brain injury [447]. The lead author attended interviews from a private location – either an office within the School of Psychology & Neuroscience at the University of Glasgow, or from home while connected to the University VPN – to ensure participant confidentiality, and participants were encouraged to also join from a private location.

Interviews were guided by a broad interview schedule (Appendix A), which addressed participants’ experiences of sleep following stroke (e.g., how it changed, factors influencing it, and the impact it has on their lives), and their experiences and preferences relating to treatment (e.g., their experience of support for sleep after stroke, attempts they’ve made to improve sleep, and their opinions on treatment options). Deviations from the schedule were permitted, encouraging the natural evolution of conversation, and limiting the risk of the researcher imposing prior understanding or knowledge of post-stroke insomnia onto the participant’s narrative [436].

Participants were informed that interviews would be audio and video recorded, and that they were welcome to take a break or withdraw at any time. Interview length was not limited but generally lasted around one hour; allowing time for all questions to be exhausted without inflicting undue burden on participants. Participants were debriefed immediately following interviews, and were later emailed debrief forms.

Interview recordings were transcribed verbatim by the lead author using oTranscribe (<https://otranscribe.com>), and participants were given pseudonyms, selected arbitrarily. Transcripts were later inputted into an analysis template (Appendix B).

2.3.5 Analysis

All transcripts were analysed by DM, and two were also analysed independently by MG to validate the analytic process; disagreements were resolved via discussion. Following guidelines put forth by Smith et al. [414] analysis was conducted ideographically, and began with several in-depth readings of a transcript while listening to the corresponding recording, allowing the researcher to develop familiarity with the data. Preliminary interpretations and exploratory notes were made in the transcript margins, initially focusing on the semantic context of participants' data. Further readings involved descriptively coding objects of concern – phenomena, meaningful to participants, which are central to their interpretation of their experiences (e.g., relationships, events, values) [414] - and initial themes. Subsequent readings focused on linguistic elements of participants' data, and what interpretive significance they carry. Final readings allowed space for greater abstraction, granting researchers freedom to become more interpretive, and incorporate more conceptual coding of transcripts. Taking this level of abstraction further, exploratory notes and codes were examined chronologically and grouped to form Experiential Statements. These sought to reduce the amount of noise in the data, simplifying subsequent stages, while maintaining sufficient nuance and remaining true to the participant's words. Experiential statements were then examined outside the transcript, and similarities and contrasts amongst them were explored, leading to the development of Personal Experiential Themes (PET). The entire process was repeated for each transcript, and PETs were examined across case, to establish Group Experiential Themes (GET) [414].

This process was iterative, and later stages generally incorporated consideration of all levels of data. Transcripts were given equal weighting when developing themes, however, only the most relevant extracts are included in the results. In accordance with existing recommendations [414, 448], GETs were generally considered plausible if inhabited by at least half of the sample. Exceptions were made, however, where the lead author subjectively interpreted a theme as being especially pertinent to the research questions, or as representing the experiences of participants with characteristics underrepresented in the sample, but of unique importance to stroke (e.g., post-stroke fatigue or aphasia). In the interests of transparency, to demonstrate nuance between cases, and in compliance with recommendations put forth by Smith [448] and Smith et al. [414], details of which participants endorsed which themes are outlined in Table 2.2.

2.3.6 Ethical considerations

Ethical approval was granted by the University of Glasgow, College of Medical, Veterinary, and Life Sciences ethics committee (Approval # 200220039; see Appendix K). Consent was obtained electronically via screening forms. Participants were made aware their data would later be anonymised, and that their identity would only be shared outside the research group should they say anything indicating they may pose as a risk to themselves or others. Any identifiable information from interviews was redacted during the transcription process, and recorded audio and video data was destroyed afterwards. Any Information via which anonymisation could be reversed (e.g., email addresses) was stored securely via OneDrive for Business, accessible only to researchers directly involved in this study, and deleted after transcription. Any participants exhibiting signs of mood or anxiety disorders were provided with information signposting them towards support services during debriefing.

2.3.7 Reflexivity and positionality

IPA methodology encourages researchers to remain cognisant of the double hermeneutic [414]; a layered interpretive process where researchers conduct a second-order interpretation of participants' efforts to make sense of their own experiences. Thus, in order to fully comprehend how one arrives at the interpretation they do, researchers must understand, and remain reflexive, of their own positionality regarding topics and participants [414]. Within this context, the researcher's prior experience and understanding of stroke and insomnia, their research motivations and biases, as well as more general characteristics such as ethnicity, gender, age, class, education, language, and culture, will inevitably influence their perspectives and interpretations of the data [449]. Qualitative research is reliant upon a researcher's subjectivity [450], and where one's subjective perspectives are inherently intertwined into the methodological processes of the research, such as in IPA [414], neglecting to attend to this can undermine the validity of the findings. Therefore, the following reflexive statement from the lead author must be heeded when considering the findings of this study.

The lead author acknowledges that his understanding of stroke has primarily been derived from clinical and academic perspectives, as he has never experienced a stroke himself. Similarly, although the lead researcher has experienced transient episodes of sleeplessness, he has no personal experience with chronic insomnia. While this may have influenced interview direction and interpretation, it is facilitatory to researcher objectivity. Moreover, at the time of conducting the analysis for this chapter, the lead author acknowledges that he was also developing other chapters, specifically those relating to theoretical models of insomnia's aetiology. Being immersed in this literature may have influenced the interpretation and organisation of participants' data. Notably, while researching the aetiology of insomnia, the lead author was struck by the extent to which participants' accounts mapped onto existing models. This realisation contributed to the conceptualisation of a proposed aetiological model of insomnia after stroke (see Figure 2.2). The author recognises that this additional level of theorisation and abstraction may go beyond IPA's traditionally idiographic focus, and adopts a critical realist position [451] – one which assumes that participants' subjective accounts may be reflective of real underlying processes or mechanisms that are accessible via careful interpretation. While this interpretation and associated conceptual model are theory-informed, they remain grounded in participants' accounts of their own experiences, and are presented as one possible reflexive reading of the data. The lead author also notes that he shares a cultural and linguistic background with several Scottish participants, which may have enabled nuanced interpretations that were less accessible for other participants' narratives. Finally, the lead author was not involved in any aspects of participants' treatment or stroke rehabilitation prior to participation, nor is he associated with anyone who was. This was considered to facilitate an objective and unbiased perspective on participants' experiences with sleep support post-stroke.

2.4 Results

2.4.1 Participant Characteristics

Screening questionnaires were distributed to 33 potential participants and completed by 18. Contact was lost with five participants after they completed screening questionnaires despite multiple follow-up attempts, two participants were excluded as they did not meet diagnostic criteria for DSM-5 insomnia disorder [67], and one participant withdrew due to having another stroke prior to their scheduled interview. Details of participant recruitment can be found in Figure 2.1.

Ten participants (five male and five female) were invited to take part in interviews. The mean age of included participants was 50.9 years ($SD = 10.71$), ranging from 30 to 65. The mean time since stroke was 3 years and 9 months ($SD = 2$ years and 10 months), ranging from 9 months to 7 years and 11 months. Mean scores from psychometric measures are as follows: GAD-7 (anxiety) = 7.60 ($SD = 6.2$), PHQ-9 (depression) = 11.4 ($SD = 4.95$), SIS-SF (stroke severity) = 28.70 ($SD = 5.29$), and SCI (insomnia) = 7.00 ($SD = 3.33$). All interviews were conducted via Zoom or Microsoft Teams, and although breaks were offered, all were completed within one sitting. Interview durations averaged 62.1 minutes ($SD = 12.4$), ranging from 46.2 minutes to 80.4 minutes. Videoconferencing mostly proved successful, although two interviews were impacted by technical challenges. With one participant (Jermaine), audio delays occasionally interrupted the flow of conversation, and with another (Billy) visual data was lost mid-interview, although audio was not affected. In no cases were interviews impacted by stroke related impairments. Further sample characteristics can be found in Table 2.1, and a summary of derived group and personal experiential themes can be found in Table 2.2. The structure of conversations naturally split across participants' experiences of insomnia after stroke and their experiences and preferences relating to treatment, and results are presented accordingly.

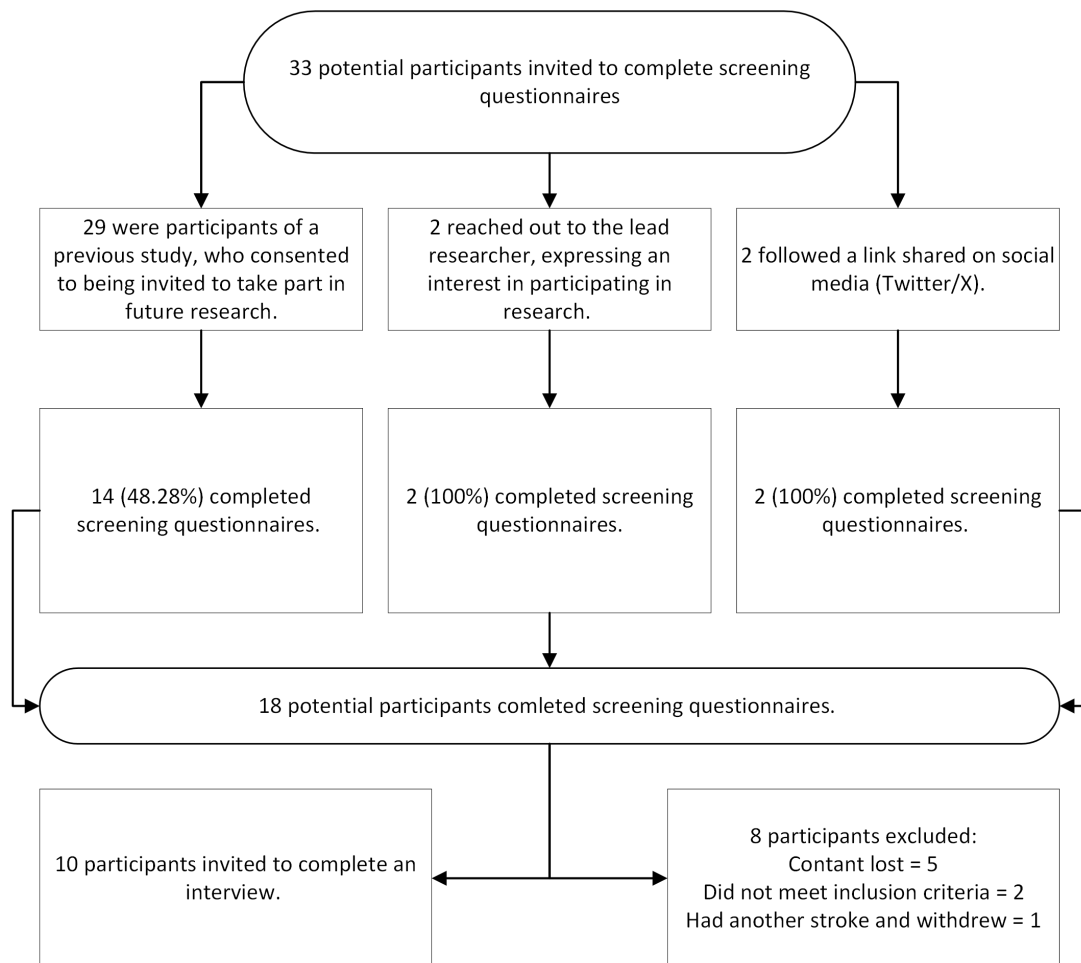


Figure 2.1: Participant Flowchart

Table 2.1: Sample characteristics¹

Pseudonym	Age	Gender	Time since stroke	GAD-7	PHQ-9	SIS-SF	SCI	Working status	Residential status
Alex	60	Male	1 year & 3 months	13	21	21	1	Not working (disabled)	Living at home with informal support
Amy	59	Female	11 months	8	12	28	7	Not working (disabled)	Living at home with informal support
Billy	55	Male	3 years & 3 months	2	8	25	9	Not working (disabled)	Living at home independently
Carla	49	Female	7 years & 6 months	3	14	27	4	Self-employed	Living at home with informal support
David	30	Male	7 years & 1 month	8	11	25	10	Not working (disabled)	Living at home with informal support
Jermaine	65	Male	4 years & 4 months	3	11	32	8	Not working (retired)	Living at home independently
Kay	52	Female	1 year & 6 months	12	14	32	3	Not working (disabled)	Living at home independently
Lorna	38	Female	7 years & 11 months	4	5	26	7	Not working (disabled)	Living at home with informal support
Michael	56	Male	3 years & 2 months	2	4	40	10	Working full-time	Living at home independently
Selina	45	Female	9 months	21	14	31	11	Working full-time	Living at home independently

¹GAD-7 Generalised Anxiety Disorder Questionnaire, PHQ-9 Patient Health Questionnaire, SIS-SF Stroke Impact Scale – Short Form, SCI Sleep Condition Indicator. Informal support refers to unpaid support from a spouse, partner, family member, or friend. Higher scores on the GAD-7 and PHQ-9, and lower scores on the SIS-SF and SCI, represent greater symptom severity.

Table 2.2: Derived Group Experiential Themes and Personal Experiential Themes.

Group experiential theme	Personal experiential theme	Participant endorsement
<i>The experience of insomnia after stroke</i>		
Sleep is fluid, and changes throughout recovery.	A hospital is not a restful place.	Billy, Kay, Lorna, Carla, Jermaine, Selina
	Sleep was impacted immediately but changed over time.	Kay, Alex, Billy, Jermaine, Michael, Amy, Lorna, Selina, Carla
Insomnia makes everything harder.	Insomnia and mental health after stroke: A bi-directional relationship	Kay, Alex, Billy, Jermaine, Michael, David, Amy, Lorna, Selina, Carla
	Insomnia and physical health after stroke.	Carla, Jermaine, Billy, Kay, Amy, Selina, Alex, David, Lorna
	Insomnia and cognitive health after stroke.	Alex, Selina, Carla, Michael, Amy, Kay, Jermaine
<i>Experiences and preferences relating to treatment for insomnia after stroke.</i>		
Sleep isn't taken seriously after stroke.	No one cares	Carla, Selina, Amy, Lorna, Jermaine, Michael, Kay, David, Alex, Billy
	Insomnia is not a priority after stroke.	Alex, Lorna, Carla, Michael, Billy
Non-pharmacological intervention is preferred.	Aversion to medication.	Alex, Jermaine, Lorna, Kay, David, Amy, Billy, Carla, Selina
	Attraction to behavioural treatments.	Billy, Lorna, Michael, Selina, Amy, Jermaine, Carla
Previous attempts at improving sleep have not worked.	Methods of distraction.	Alex, Amy, Michael, Selina, David, Billy
	Previous methods no longer work.	Michael, Jermaine, Amy, Selina, Alex

2.4.2 The experience of insomnia after stroke

2.4.2.1 Group experiential theme 1: *Sleep is fluid, and changes throughout recovery.*

Participants described how their sleep has changed and evolved following stroke. For most, sleep was impacted immediately after stroke – including while in hospital – and although it has improved slightly over time, it has never returned to the quality enjoyed before stroke. Keen to understand their sleep difficulties, participants draw comparisons between their current experiences with those prior to stroke and conceptualise their difficulties as consequent to stroke.

A hospital is not a restful place.

Participants unanimously reported poor sleep in hospital due to external factors. While these experiences of poor sleep so soon after stroke may not have been defined as insomnia disorder at that point, participants describe the distress they felt in great detail, and associate these experiences with ongoing sleep problems further down the line. Emphasising a perceived lack of control over one's sleep, Kay described the experience as akin to '*torture.*' Commonly, social disruptions, such as other patients, or noisy staff, were sources of sleep disruption:

'You couldnae get a decent sleep in hospital coz there was always people wakenin' up... It jist bursts yer heid' — Billy

Such experiences, during an already stressful time, led to considerable distress for participants. Kay, for example, was particularly disturbed by an incident where she felt that sleeplessness drove her to act out of character, becoming confrontational with another patient:

'I became a different person!... I didn't recognise myself. Y'know? I completely and utterly lost the plot, and it was sleep-deprivation... And it was devastating... That's not who I am.' — Kay

Participants also reported being awoken by care-related factors, such as nighttime medication rounds or medical observations, subsequently finding they ‘couldn’t get back to sleep again’ (Kay).

‘...through the night, you would get wakened up wie, y’know, somebody come in and doing yer blood pressure at two o’clock in the morning, or wakening ye up tae see if yer alive.’ — Billy

Environmental aspects, such as noise, temperature, and light, also contributed to sleep difficulties, leading participants to perceive the hospital ‘not a restful place’ (Selina):

‘I don’t sleep in hospital. It’s too warm, the beds arnae comfy ... it’s too noisy. I just don’t dae it.’ — Lorna.

‘It’s just, noise, noise, noise, noise, noise.’ — Carla.

Reflecting on their experiences, participants felt as though hospital staff don’t care or ‘realise how bad it is, in hospitals’ (Carla).

‘It was almost like they didn’t want me to sleep or something... I just felt nobody cared’ — Kay.

Kay links her experiences with sleep now, to her experiences in hospital; her words implying they still have a lingering effect on her sleep.

‘It is torturous lying there... It’s almost like something, somewhere is preventing me from sleeping, and that’s the sort of retriggering the experience that I had in hospital.’ — Kay

Sleep was impacted immediately but changed over time.

Excluding David, who felt that any initial sleep difficulties were masked by ‘strong medications’, participants reported that sleep was impaired immediately after stroke, persisting beyond discharge from hospital.

‘Immediately after the stroke and even coming home from hospital, erm, the biggest initial issue I had initially was, constantly waking up.’ — Amy

Sleep was said to have ‘gone through a lot of different phases’ (Jermaine), generally improving throughout recovery. However, for no-one had it returned to the quality and duration enjoyed before stroke. Nevertheless, even small improvements were taken ‘as a win’ (Kay), as participants rationalised them as indicative of recovery, or things ‘moving on’ (Kay).

‘... as my therapy progressed, well, I became sort of more comforted that I would sort of get my life back, and then my sleep got better.’ — Michael.

Sleep impairment was, thus, perceived as a reminder of stroke, and a marker of recovery. Anxious about the insomnia’s uncertain aetiology, and their apparent inability to recover their sleep participants desperately sought an explanation. Aware of the temporal relationship, participants contrasted their experiences of sleep before and after stroke, perceiving insomnia as consequent to their psychological response.

‘...the reason why I struggle to go to sleep is because I’m over thinking everything at the moment, whereas before... I could just switch off... can’t do that now. There’s always noise.’ — Alex.

Contrarily, Jermaine, the only participant to report poor sleep immediately prior to his stroke, questioned whether this disrupted sleep was a factor in his stroke’s aetiology:

‘...you look for things that might have caused the stroke, y’know? ... sleep is the only thing that I could come up with, that I know before wasn’t optimal.’ — Jermaine.

Permeating participants’ reflections was a pervasive sense of loss, as they recalled a time where sleep was effortless and restorative. Reflecting the significance of this loss, Alex lamented ‘I’m not living a life at the moment, I’m existing’, and emphasised his longing to ‘get some normality back’ with regards to sleep.

‘You’d swear at the alarm because it woke you up at 5 in the morning... I want those days back... You complain when they happen, but you miss them when they’re not there... I’m desperate to try anything at the moment. I just I want to try and get some normality back.’ — Alex.

2.4.2.2 Group experiential theme 2: *Insomnia makes everything harder.*

Participants primarily discussed insomnia through the lens of its impact on their mental, cognitive, physical, and social well-being. This impact appeared compounded by a complex nexus of interrelated factors, where insomnia exists as both a cause and consequence of broader challenges. A graphic model of this network is displayed in Figure 2.2.

Insomnia and mental health after stroke: A bi-directional relationship

Participants primarily described insomnia as being driven by negative emotions, anxiety, and rumination. For example, Lorna described her mind at night as like a *‘minefield’*. However, mental health also appeared profoundly affected by insomnia, leading to participants holding negative outlooks on life, and feeling as though they had less control over their emotions. Alex described how one factor leading to his depression was *‘the fact that [he] was so tired’*. This impact further eroded participants’ motivation to take on challenges, inherently believing that they would fail, and thus wanting to avoid any accompanying distress.

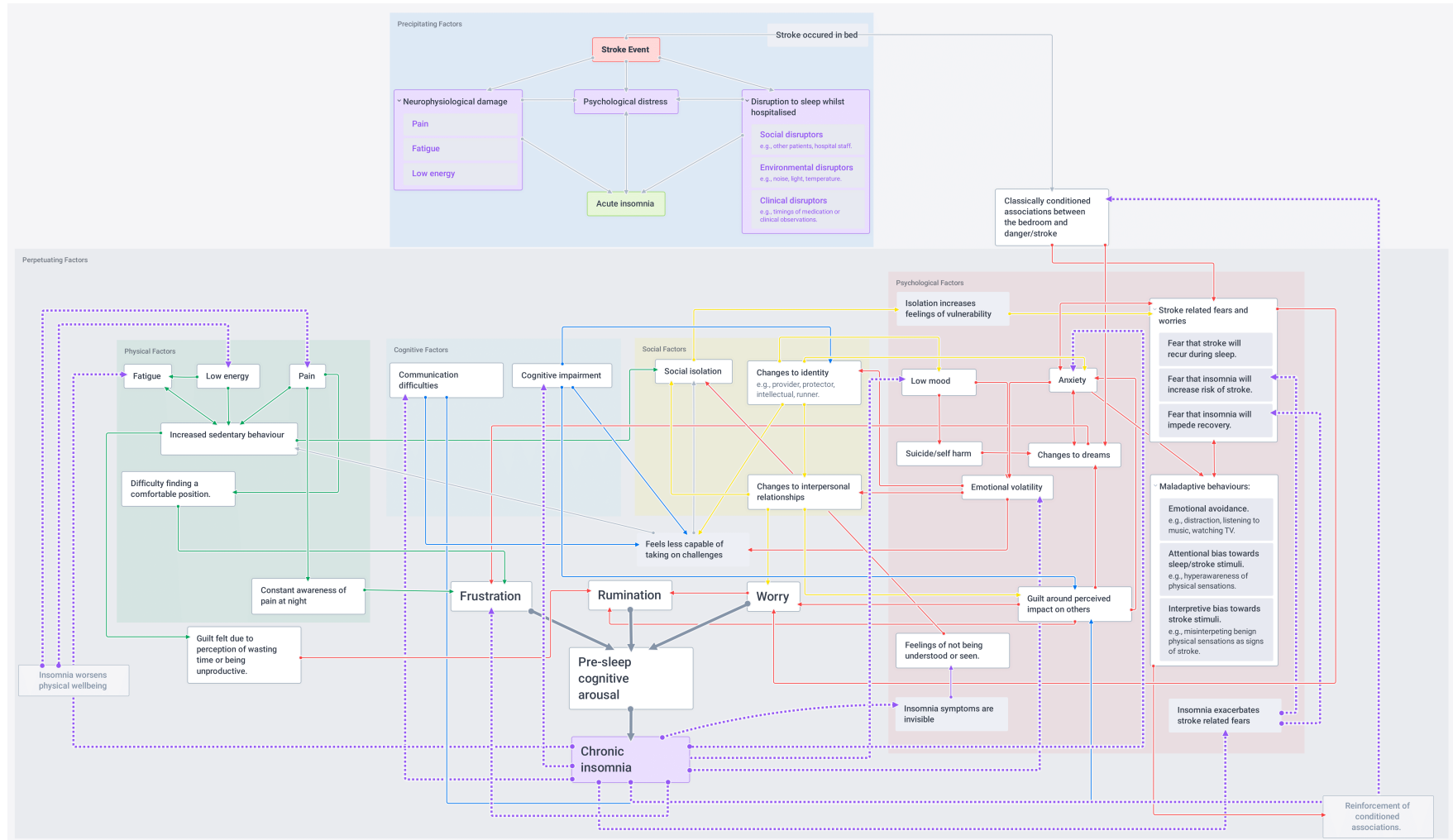
‘I’ll just end up in a heap of tears somewhere... I’m literally an unexploded bomb, basically, just waiting to go off.’ — Kay

Negative emotions such as anger and frustration – directed both, inwardly and outwardly – were frequently cited as impeding sleep. Lorna described indignation provoked by the belief that her stroke caused her sleeplessness. Resultantly, she would lambast her stroke: *‘Look at me! I cannae sleep properly now... Look! Look what you’ve done tae me!’*. Other times this anger can be internalised, as participants blamed themselves for both their insomnia, and their stroke.

‘I start tae get angry at myself and say “Oh for fuck’s sake, just go tae sleep! It’s no hard, you’re tired!”.’ — Lorna

'I think when I'm wakening up at four o'clock it's just like reliving that and saying: "if I'd acted quicker" ... I've worked in that field. I know the signs and symptoms. Why didn't I pick up on it myself until hours later?' — Amy

Figure 2.2: A conceptual model of insomnia after stroke.²



²Phenomena driving insomnia after stroke are grouped into their conceptual domains (cognitive, psychological, physical, social) and coloured accordingly. Relationships are colour-coded to denote their domain of origin. Relationships that are coloured grey have unclear or multiple origins. Relationships denoted by the broken purple line represent the influence of chronic insomnia on the network of factors, demonstrating where insomnia becomes self-perpetuating.

For others, worry and rumination were more pertinent issues. Routine concerns, such as finances or performance at work, were intensified by stroke, keeping participants awake. Kay, for example, described the stress of surviving on a reduced income as particularly stressful; something that *‘messes with your head’*. Cognitive impairments led others to worry about their colleagues’ perceptions, worrying that they were *‘holding them back’* (Selina).

Particularly impactful, were increases in health anxiety after stroke. Participants harboured an acute sense of vulnerability, and became hyper-vigilant of physical sensations, often misinterpreting benign phenomena such as a sore head, as potential harbingers of stroke recurrence. Aware of this tendency, participants avoided medical programmes prior to sleep, to avoid being reminded of their stroke, or prompted to lookout for another one.

‘I thought I was just sitting here like a ticking time bomb waiting to go... If I had a sore head, I didn’t know whether I wis having another stroke... Ye’ question every...single...thing! And that stops you sleeping.’ — Amy

Sleep took on a heightened importance, as participants feared that insomnia increased their *‘risk of having another stroke’* (Lorna) and impeded their chances of recovery. Alex, aware that neuroplasticity was necessary for neural repair, despaired that *‘it needs sleep to do that’*. This pressure and urgency around sleep only served to heighten cognitive arousal and further perpetuate insomnia.

‘I don’t understand how your brain is meant to heal if it’s not getting sleep... if your brain doesn’t switch off, how on earth can it heal?’ — Kay

At times these worries appeared exacerbated by participants’ interpretations of comments from healthcare professionals:

‘My consultant says, “you really need tae try and get sleep” and I think “well I a’ready try! Whit dae ye hink I’m dain? Paintin’ ma nails?!”’ — Lorna

Health anxiety was also exacerbated in those for whom, stroke occurred in bed, thereby associating the bedroom with danger and threat. Evidencing this, several participants reported awakening each night at times relevant to their stroke (e.g., the time they first recognised their symptoms, or times they had to take medication). Alex reported being *'scared to go to sleep for a couple of months'* due to have woken up *'up paralyzed on a Sunday morning'* and fearing *'could it happen again?'*

For four participants, sleep was also impaired by changes to the frequency and vividness of dreams, which appeared temporally related to stroke. Billy, who perceived these changes as positive, described how he will often awaken mid-dream, wanting to *'experience that dream again'*, and felt that *'frustration comes with that not being able to get back to sleep'*. Others viewed the changes to their oneiric experiences as frightening, impairing sleep by causing fear and anxiety.

'I was frightened to go to sleep. You didn't want I didn't want to go to sleep because you don't want to go to a place that's as frightening as that.' — Alex.

Selina described how, when awakening from a dream with heightened anxiety, *'you've automatically got that anxiety level which makes it even harder to get back to sleep'*, often instigating worry and rumination.

Nightmares related to stroke were highlighted; usually reflecting threats to identity. For Selina and Michael, nightmares allegorised stroke's impact on their cognitive ability, and the threat this posed to their vocational identities. Michael dreamt he had to go *'back to college to get a second PhD'*, as though believing he had to prove his competency. Selina had recurring nightmares of *'trying to get somewhere with a colleague... but everything is stopping [her] from getting there'*, reflecting her concerns of burdening her colleagues due to her cognitive difficulties. For Alex, nightmares often alluded to harming his family, and reflected his concerns about being unable to fulfil his role as a father or protect his family from the fallout of his stroke:

'The early day nightmares involved... They were quite graphic. They involved harming my family, which I love dearly, and I treasure dearly, and that's what worried me.' — Alex

The most distressing however, were one's that seemed to reflect the psychological consequence of his stroke happening in bed - a supposed safe-space:

, 'The nightmares that scared me the most were the ones that happened in familiar places... It was in this house... I've made this my safe space... But to make this house my safe place, but then have my nightmares invade the safe place...' — Alex.

Emphasising the severity of the relationship between sleep and mental health after stroke, Alex's dreams often alluded to self-harm. He and several others referred to intensely low mood and of suicide, at least partly attributed to insomnia. Lorna described having '*really bad thoughts*' of how she could '*sleep forever*'; her use of language demonstrating her conceptual connections between sleep and despair.

Compounding the relationship between sleep and mental health was a sense of social isolation and loneliness. Participants described how the invisible nature of insomnia often made it difficult for others to understand, or realise, '*how much of an impact it has*' (Selina).

'it's hard to describe my symptoms to people. By all appearances they'll say, "well you look fine". Well, I don't feel fine!' — Jermaine.

For David, familial relationships became strained as sleeplessness led to emotional volatility. Coupled with his beliefs that his family didn't understand or care about his difficulties, this often led to arguments. Resultantly, he feels there is now a '*clear gap*', as his family 'tend to keep a good, safe distance', leaving David feeling isolated, and concerned that he is '*a bit of a burden on them*'.

Isolation also intensified participants' feeling of vulnerability, and consequently, health anxiety. Positive relationships appeared protective however, particularly for those whose stroke occurred in bed. Amy, for example, finds it easier to sleep when her partner comes to bed, as there is '*comfort in knowing that somebody is there*' (Amy). For participants living alone however, the lack of immediate support may intensify their sense of vulnerability, further impeding sleep.

'[other stroke survivors] all talk about how much their husband, or their other half has helped them... how much their family has rallied round them... I haven't really got that. And it does make you realise how vulnerable you are, and that plays on my mind'. — Selina.

Insomnia and physical health after stroke.

Relationships were also described between sleep and physical aspects of well-being, such as energy, fatigue, and pain. Participants found that *‘extra physical activity during the day does help the sleep at night’* (Alex), as rather than feeling inclined to *‘languish in the negativity of the past’* (Amy) or chastise oneself for having *‘wasted a whole day of life... moping about’* (Amy), their cognitions were more positive, translating to better sleep.

‘See if I sit down and just give up, I can have a terrible night’s sleep... every day that you sit back and do nothing is a day lost. I’ll never get back the day that I had the stroke. I’ll never get back the three months after my stroke.’ — Amy

Participants found themselves in a quandary however, as insomnia compounded fatigue, depleting their energy reserves; likened, by Carla, to a *‘battery’* that is never fully recharged, which resulted in reduced physical activity. Emphasising this impact, they were explicit in distinguishing fatigue and tiredness, describing the former as *‘frightening’* and *‘debilitating’*, akin to being *‘stuck in a vat of treacle’* (Kay). Through fear of this, participants were hesitant about undertaking demanding tasks (e.g., socialising or exercising) when under-slept, as *‘that’s where the fatigue is waiting’* (Kay) - behind pushing on when resources are already diminished by insomnia. Even after a restful night, this hesitancy persisted, as participants worried about feeling inclined to *‘go for it’* and *‘do too much’* (Selina). This overexertion also led to fatigue, a phenomenon which Selina dubbed as the *‘Boom and bust’*.

Post-stroke pain was also an impediment to sleep. Participants were *‘constantly thinking about’* (Alex) their pain at night, *‘always aware’* (Billy) of the extra effort involved with trying to get comfortable. This was particularly distressing at night when there were no distractions. Alex remarked *‘some nights the pain was that bad’* that he asked his wife if she would *‘cut [his] leg off’*, because the pain *‘takes over everything’*. Carla described a constant aching, which not only interfered with sleep, but was thought to be more painful on days where she hadn’t slept well the night before:

‘It’s painful all the time. So, I have to find the right...angle, to go to sleep. My leg is pain. My leg; my foot; my toes. So, I have to get them in the right order, so I feel more comfortable to go to sleep.’ — Carla

Insomnia and cognitive functioning after stroke.

Insomnia was particularly impactful on participants' cognitive functioning, making routine tasks feel insurmountable. Selina, a young stroke survivor, described how this impacted her work, feeling as though she were *'thinking through mud'*; contributing to the anxiety she feels at night. Michael, a lecturer, worried about how the impact insomnia's worsening of his cognitive impairments had on his students. While able to get by due to the *'adrenaline'*, he feared that his classes were *'probably not [his] best'*. Participants often viewed the working week as an obstacle to *'get through'* (Selina), and organised demanding tasks (e.g., driving long distances, or meetings) earlier in the week to exploit any extra sleep obtained over the weekend.

'Anything big which I think is going to be...challenging on my brain... I try and get those for the beginning of the week... I know by the end of the week, I'm less able to cope.' — Selina

For others, insomnia impacted communication. Amy felt guilty about how this impacted her family, noting that she felt *'like a zombie'*, unable to focus on conversations. Similarly, Carla, who lives with aphasia, shared that she *'can't really have a conversation'* when insufficiently rested, and has to expend a great deal of effort to focus, otherwise her brain turns to *'gobbledygook'* and she *'can't understand them'*. Resultantly, Carla avoids travelling too far from home, worried that she may get lost and be unable to ask for help.

'The words doesn't. Everything gets slowed down: the words in my head if I'm having a conversation, if I got lost, I can't explain it. It's got my, my words disappeared... then if I got lost, how can I find to go back again?' — Carla

2.4.3 Experiences and preferences relating to treatment for insomnia after stroke.

Analysis of data pertaining to prior experiences of support for sleep – self-directed or supported – and preferences around treatment culminated in the derivation of three group experiential themes: *Sleep isn't taken seriously after stroke*; *Non-pharmacological intervention is preferred*; and *Previous attempts at improving sleep have not worked*. Corresponding PETs and details of which participants endorsed these themes can be found in Table 2.2.

2.4.3.1 Group experiential theme 3: Sleep isn't taken seriously after stroke

Participants' accounts suggest sleep difficulties after stroke to be largely considered normal, unremarkable, and often dismissed by healthcare professionals. They felt as though their concerns were largely ignored, leaving them feeling *'let down'* (Lorna) and abandoned after leaving hospital.

No one cares.

Participants were dismayed with the general lack of support for sleep after stroke, describing how the issue was rarely, if ever, discussed. For many, it was not mentioned until they sought support from third-sector organisations, yet still they remained bereft of practical strategies for tackling their difficulties.

'There's a thing on the Stroke Association about it, but it just basically says "your sleep can be disturbed after a stroke", it doesn't give you any ways in which you can deal with it' — Billy

Despite several having sought support, no participants had previously been formally assessed for, or diagnosed with, insomnia disorder. Conveying a common sense of abandonment, Carla remarked that *'the NHS is amazing. And then you leave'*.

'...it's very isolating a stroke. Because once you've had it and you're discharged that's it.' — Lorna

Many recounted feeling as though their concerns were minimised or normalised, and that clinicians didn't seem concerned, nor motivated to help. Jermaine described how, when seeking support for sleep, he felt as though his neurologist *'shooed'* him out of the door.

'I did tell my doctors that I wasn't sleeping well, and they said "Well, that's understandable. You just had a stroke".' — Michael

Lorna's doctors only raised her insomnia as an issue three years after her stroke, after it exacerbated her co-morbid conditions and led to hospitalisation. Even then, Lorna felt they were *'very dismissive about it'*, regarding it as *'an insignificant detail'*, and neglecting to offer any advice or support. To Lorna, this was the one area that *'the NHS failed a wee bit'*.

Clinicians' failure to earnestly consider their sleep problems led to participants normalising their own experiences. Juxtaposed to otherwise excellent support after stroke, Lorna assumed that her clinicians' lack of concern must have meant her insomnia was extraneous to stroke.

'I just thought, "this is daft. If [it] were stroke-related they would have told you" ... I never ever thought it was stroke, because as I say, they never mentioned it, and they mentioned everything else.' — Lorna

Participants began to view insomnia as something inevitable that they would *'just need tae cope with... just part of the stroke'* (Billy).

'it's coming up 8 years, it's part of my life... feels like almost normal. I know it's not normal but... This is my normal life, y'know? I can't sleep properly.' — Carla

'This is me now, my new sleep arrangements' — Lorna

Participants also felt that dismissal of their opinions continued in discussions around treatment, feeling as though clinicians *'just bulldoze... they don't actually listen to their patients'* (Kay). Denial of any autonomy in their care led to damaged relationships with healthcare professionals, as participants began to doubt their ability or motivation to help. Some felt pressured to take medication, despite not wanting to, and were disappointed to not be offered alternatives.

'... they just keep pushing a sleepin' pill on me. And that's not what I want' — Lorna

Contrarily, others described being *'very happy to take a sleeping pill every night'* (Michael) but were frustrated by their doctor's reluctance to prescribe.

'I would go find some heroin if it helps, y'know? Because it's easier to get than what they prescribed me.' — Jermaine.

For participants like Kay and Lorna, mere awareness that research into sleep after stroke is ongoing was comforting after all their experiences of feeling as though no one cared.

'I'm really grateful you're doing this research... I don't think anyone is really interested.' — Kay

'I was quite pleased that I got to meet you... I thought, this might be somebody that can help. Coz I'm about tae [laughter] just knock myself out! Gonnae sleep wie a brick and go "dunk!"' — Lorna

Insomnia is not a priority after stroke.

Within the wider context of stroke, sleep was low in most participants' priorities. Participants were initially *'overwhelmed... by all the stuff [they] had to work on'* (Michael) after stroke, but physical ailments generally took precedence. Alex, for example, felt that once he has *'got them sorted, then maybe we can look at things like the sleep'*.

'...my arm, fae my elbow down, tae my fingertips, see if I could get that tae work, life would be great... tae be able tae run, tae run again would be brilliant... And the sleeps a thing where... that's put tae the back o' my mind' — Billy

Even those motivated to improve their sleep appear primarily motivated to do so due to believing it may potentiate other facets of recovery.

'Any help with sleep would be good, because obviously it's the start. I'm hoping that if I can get better sleep, if I can manage this fatigue better, I can go out more.' — Alex

'...if I had a good night's sleep... I'd get a lot less symptoms, and a lot less deficits. So, I put it pretty high on my list of things I'd like to improve... I feel like I would be a lot better off if I slept better' — Jermaine.

2.4.3.2 Group experiential theme 4: Non-pharmacological treatment is preferred.

Willingness to accept pharmacological treatments varied widely across the sample, but most preferred non-pharmacological approaches (e.g., CBT-I). Prior experiences influenced an individual's proclivity for either option, but generally, medication was considered a *'last resort'* (Alex) in the event that one's *'health issues took a drastic decline'* (Lorna). This inclination was predominantly driven by an aversion to medication, rather than a preference for behavioural approaches, although the latter did play a role to a lesser extent.

Aversion to medication.

Aversion to medication was predominantly due to scepticism over safety and efficacy. Participants viewed it as unnatural – and by extension unhealthy – and thought that by *'forcing'* (Selina) one's brain to conform, they were only masking insomnia's symptoms, not addressing the cause.

'...they just want to give me sleeping tablets. And that's not dealing with... why I can't sleep.' — Kay

Many expressed concerns of side effects, feeling that they *'have enough trouble functioning'* (Kay), and that medication could exacerbate this. Common too, was fear of adding another medication into an already extensive regimen and intensifying the strain on their bodies, the belief being that it was *'not a good idea to mix them'* (Jermaine).

'I'm on enough medication. I really don't want anymore. I rattle enough as it is.' — Amy

'They've stuck me on 14 tablets a day... I'll no even have a paracetamol if I feel ill. Because I just think: "what my liver's doing for me now?" It's working hard tae get rid o' all that.' — Billy

Some would *'rather not take anything'* (Jermaine) as they felt they were uniquely vulnerable to side-effects by virtue of being a stroke survivor, often fearing that additional medication may increase their risk of another stroke.

'I do worry about other strokes; having other strokes. Likes' a' I don't take ibuprofen, or anythin' like that anymore... I'm really careful... about medication that I put in ma body.' — Lorna.

Conflict occasionally arose, between participants' concerns around medication, and their being *'pretty desperate'* (Jermaine) for a solution. Lorna's vehement aversion to more medication, for example, weakened when conceding that she would be forced to reconsider her stance were her health to deteriorate.

Also pervasive, were concerns around dependency, particularly for those already prescribed potentially addictive medication, or who had previous difficulties with dependency. David recounted going without *'sleep for four continuous days'* after abruptly stopping medication he was dependent on after his stroke, contributing to drug hesitancy now. Permeating the narrative around dependency was a desire to *'fight through it'* (Billy), as participants were reluctant to relegate control of their sleep to something external.

'I would be worried of becoming reliant on it, and the only way I could sleep is by taking the tablet... I was hoping that I could sort it myself.'
— Alex

Thus, to many, medication was considered a *'stop-gap'* (Jermaine), only to be used *'in a crisis'* (Selina) or while awaiting behavioural support. Partly due to the perception that any benefits would be lost the moment you cease to take the medication.

'Usually with drugs if you stop taking them, you come back to the same problem, whereas therapies tend to be longer term...' — Selina

Billy harboured a unique fear that medication carried a risk of suicide. Despite characterising himself as an inherently positive person, he noted rare occasions where he lamented his stroke, where he feared he would feel compelled to intentionally overdose on sleep medication were it available.

'That frightens me, that maybe the devil would say "Just take a hand fu' of them and you'll no need tae worry aboot stroke..." So, there's a fear there. A fear there for a drug.' — Billy.

Attraction to behavioural treatments.

Although scepticism of medication’s safety and efficacy appeared to be a major factor in participants’ preference for non-pharmacological approaches, some did display an affinity for behavioural approaches – particularly those that had benefited from them in the past.

‘CBT, I’m a big fan of that...I’m a real advocate for it, as I say, I really helped me.’ — Lorna.

However, perceived advantages of behavioural approaches were generally antitheses of medication’s disadvantages. For instance, the former was seen as more natural, with greater longevity of effects, and as offering insight into *why* one was suffering from insomnia in the first place.

‘...the more you learn about your brain and yourself, and the way it works, the more it helps.’ — Selina

Nevertheless, several barriers to treatment were identified. While most were typical to insomnia (e.g., concern over costs, or waiting lists), some were distinct to stroke. Billy, who lives with impaired mobility and muscle spasticity, felt the costs of aspects like stimulus control outweighed the benefits; that the *‘reward isnae worth... the palaver of trying tae get up’*. Others, such as Alex, who felt that CBT was *‘actually making [him] worse’*, were averse to CBT due to disappointing experiences in the past. However, they were generally receptive to aspects of CBT-I (e.g., stimulus control), when they were described independently to the term ‘CBT’.

No clear preference arose regarding the delivery of behavioural treatments (in-person vs online; individual vs group-based), and participants tended to prefer whichever methods were most familiar to them, though viewed all as acceptable. Due to her aphasia however, Carla displayed a strong preference for in-person, individual sessions. She described how communicating online was like *‘an extra layer’* on top of something already challenging, and that groups can be overwhelming.

‘...in a group, I’m not joking, for me it’s just gobbledegook after a while, I wouldn’t understand it properly.’ — Carla

2.4.3.3 Group experiential theme 5: Previous attempts at improving sleep have not worked.

Participants had gone to great lengths to try and alleviate their insomnia, becoming increasingly desperate with each failed attempt. Several persisted with techniques they considered to be *‘gimmicky’* (Lorna), despite doubting their effectiveness, due to fears that cessation may worsen their insomnia.

Methods of distraction.

In attempts to distract themselves from their anxieties at night, several participants listened to things such as the T.V., radio, audiobooks, or brown noise. This appeared effective in the short-term by allowing participants to get their *‘mind out of the zone’* (Amy), and distance themselves from their anxieties long enough to fall asleep.

‘When I listen to music, I can forget about the voices’ — Alex

Some found, however, that the effectiveness of this method decreased with repeated exposure.

Previous methods no longer work.

Many participants despairingly recounted how previously effective methods of facilitating sleep (e.g., meditative practices), or mitigating the impact of sleeplessness (e.g., coffee) were no longer accessible. Alex, for example, no longer consumes caffeine due to concerns around his blood pressure, thus cannot use the stimulant to stave off the effects of insomnia. Michael grieved the loss of memorised lists (e.g., the chronological order of Wimbledon champions and US presidents), which he would meditatively recite to *‘help [him] get back to sleep’* after waking up. He found these lists just *‘don’t work anymore after [his] stroke’*, and consequently his sleep is impacted by small worries, where it wouldn’t have been previously. This often reorientates Michael to thinking on what he has lost to his stroke.

‘My stroke affected a lot of things...I got back most of my mental faculties... But those, those memorised lists have gone... it reminds me that I lost that ability after my stroke... I aren’t able to get it back’
— Michael

Others who had previously benefited from similar practices, such as mindfulness, found stroke led to these practices being less effective, as they struggled to find the motivation and concentration necessary to engage.

‘It was kinda weird. I used to meditate every day, before my stroke. And then after my stroke it was like I just couldn’t get myself to sit down and do it’ — Jermaine.

As with Michael, not only are they deprived of a previously efficacious practice, but they are reminded of the cognitive consequences of their strokes, and what that means for them.

‘I just don’t know what to do, because all of my techniques that I’ve taught myself over the years just don’t work anymore... And I think it’s kind of adding to a bit of the paranoia of the brain injury has changed me.’ — Selina

2.5 Discussion

This is the first study to examine the lived experiences and treatment preferences of stroke survivors living with insomnia. Analysis of data gathered from stroke survivors in the UK resulted in the derivation of five Group Experiential Themes (GETs): two pertaining to the experience of insomnia after stroke – Sleep is fluid and changes of time, and Insomnia makes everything harder – and three pertaining to experiences and preferences relating to treatment – Sleep isn’t taken seriously after stroke, Non-pharmacological intervention is preferred, and Previous attempts at improving sleep have not worked. Key findings highlight the immediacy with which sleep is disrupted after stroke, how this is compounded by the hospital environment and persists throughout recovery, the resultant impact on well-being, the need to improve clinical understanding and approaches to sleep after stroke, and a preference for behavioural treatments.

2.5.1 The experience of insomnia after stroke

2.5.1.1 Summary of main findings

Participants' accounts paint a bleak picture of insomnia after stroke. Impairments to sleep appeared immediately after stroke, and though they do improve over time, evidence suggests a sustained chronicity, with several participants having lived with their difficulties for several years. Accounts of insomnia's negative impacts – through the lens of which participants predominantly described their experiences – were unequivocal, unanimous, and often bi-directional. Nowhere was this more obvious than in participants' descriptions of their mental health, which told of self-perpetuating cycles between anxiety, arousal, and sleeplessness.

2.5.1.2 Current findings within the context of the existing literature

Participants' accounts align with existing empirical evidence [321–323, 392, 452, 453] showing the immediate, and evolving nature of disturbed sleep after stroke, where hospital-related factors also appear to play a role. Objective reports suggest that approximately 21% of stroke survivors get less than 6 hours of sleep in the first fortnight after stroke [322], and reports of environmental, social, and care-related disturbances to sleep in hospital are echoed in results from stroke [454, 455] and non-stroke samples [456, 457]. Kenah et al. [455] suggest that disrupted sleep in stroke-rehabilitation contexts may exacerbate fatigue, lessening motivation to engage in therapeutic protocols. Whether this influences clinical outcomes is not fully known, but concerns pertaining to sleep and stroke recovery in hospital have been raised previously [347, 375], with recent observational research suggesting sleep disruption while hospitalised to be associated with slower functional recovery and worse motor outcomes in adults with acquired brain injury [367]. Thus, consideration of sleep as part of acute stroke management and rehabilitation protocols is clearly indicated.

Determining whether participants' experiences in hospital contributed to chronic insomnia further down the line is beyond the scope of this study, though some participants did imply this to be their belief. Though limited evidence does suggest that hospital admission is associated with the incidence of insomnia [458, 459], this has never been examined within the context of stroke. Within Spiel-

man’s behavioural model of insomnia [224](see § 1.4.1 for an overview), a stroke would certainly constitute a significant enough stressor to precipitate acute insomnia – the immediate impact on sleep certainly supports this – and modulation of this by negative experiences in hospital is not beyond the realms of possibility.

Indeed, the aetiology of their insomnia was a pertinent concern for participants, particularly as they conceptually linked it to their stroke and recovery. Participants considered sleep vital for recovery, and small improvements were believed to reflect psychological adjustment or neurological repair; implying that participants considered their sleep difficulties to be, at least partly, perpetuated by psychological factors or localised brain damage. The proposed model outlined in Figure 2.2 demonstrates the complex nature of insomnia after stroke, and outlines stroke specific factors which may contribute to its manifestation and origin.

The positioning of cognitive activity as an antecedent and perpetuator to chronic insomnia disorder is largely ubiquitous across existing aetiological models [25, 90, 152, 220, 224, 231] (see §1.4 for an overview), and is supported by results of a recent systematic review [242]. Several facets of participants’ accounts can be mapped onto these models. Harvey’s cognitive model [152] posits that, in response to distressing cognitions about sleep (e.g., sleep being vital for recovery after stroke), people with insomnia engage in safety behaviours, such as using distraction (e.g., listening to the T.V. or radio) or avoiding medical T.V. programmes before bed, as a means of emotional avoidance and preventing anxious arousal. While intended to facilitate sleep, these behaviours deny individuals an opportunity to challenge their beliefs [146, 243], and only serve to further perpetuate insomnia. Participants’ accounts of sleep onset being easier following physical activity, where one’s affect is more positive, strengthen the applicability of Harvey’s model to the current data, as does evidence indicating the presence of attentional biases, not only to sleep, but to pain, and potential portents of stroke. Such biases are common to insomnia [246, 250, 251], and Harvey [152] posits that concomitant intensification of anxiety can lead to interpretive biases [252]; misinterpretation of harmless phenomena in a manner consistent with one’s anxieties (e.g., perceiving benign bodily sensations as a sign of stroke, or attributing worsening health to insomnia). The co-occurrence of sleep-related anxiety and fears of impending stroke may be an example of *ex-consequentia reasoning* – the mistaken perception of a threat due to extant anxiety [253]; a phenomenon

also constituent to Harvey’s model. These fears appear intensified in participants whose stroke occurred in bed, and for whom the bedroom had thus become conditioned with danger and threat. Evidencing of this was most strongly reflected in Alex’s accounts of being ‘*scared to go to sleep*’ for fear of stroke occurring again.

One theoretical construct relevant to participants’ fears of stroke recurrence is Fear of Recurrence and Progression (FORP) – a trans-diagnostic mechanism characterised by elevated and persistent anxiety surrounding the return or worsening of a serious health condition [460]. Although FORP has mostly been explored in oncology [461–464], where it has been associated with behaviours such as health-related hyper-vigilance and elevated sensitivity to bodily sensations [461], emerging evidence suggests its relevance to cardiovascular and stroke populations [465–467]. In the present study, the fear of stroke recurrence was pervasive throughout participants’ accounts, as were descriptions of behaviours and cognitive processes seemingly motivated by this fear (e.g. rumination, hyper-vigilance, and attentional and interpretive biases). While recent evidence suggests that FORP may correspond with increased engagement in health-promoting behaviours among stroke-survivors [466, 468], participants’ narratives in the current study suggest that without guidance around sleep and insomnia, such behaviours can become maladaptive and lead to the perpetuation of sleep disruption. The potential influence of FORP appeared particularly salient among participants whose stroke occurred during sleep, possibly leading to a heightened association of the bed or bedroom with perceived danger or threat.

The possibility that insomnia arises as a consequence of localized damage to regions relevant to these systems is also plausible, and existing evidence supports this theory [314, 320, 321, 375, 381–388]. The brain does undergo varying degrees of neuronal regeneration (*restoration*) and functional reorganisation (*compensation*) after stroke [393], which Hasan et al. [321] hypothesise may explain observed temporal variation in the prevalence of sleep disorders after stroke. However, participants’ subjective reports are less informative for neuroanatomical considerations of insomnia than for cognitive ones, limiting the inferences that can be drawn from the present study in this regard.

Insomnia had a wide-reaching impact, which was overwhelmingly the basis for participants’ accounts of their experiences; a phenomenon also observed in non-stroke samples [469]. Analysis indicated a complex series of – potentially self-perpetuating – relationships between insomnia, socialisation, cognitive impairment, physical health, and mental health. While these findings align with those in the general population [82, 469–471], several nuances were unique to stroke,

such as reports of being kept awake by rumination and worry around stroke recurrence, insomnia's impact on recovery, and the impact of stroke on their lives. While some of these worries have been qualitatively identified in stroke previously [472], the present study is the first to outline their relevance to sleep.

Participants' accounts focused on mental-health, which not only drove sleeplessness – as discussed above – but was exacerbated by it. Psychological distress is common after stroke [473–475], and may impede rehabilitative efforts [476, 477], and increase mortality risk [478–481]. Many studies have linked poor mental health after stroke to sleep [313, 369, 379, 424, 482, 483]. The present results add to this by highlighting possible mechanisms underpinning this relationship, and detailing participants' descriptions of its bi-directional nature. Distress perpetuated insomnia, which in turn exacerbated participants' mental health the following day, establishing a self-perpetuating cycle of distress, arousal, and sleeplessness.

Moreover, while no participants reported suicidal thoughts at the time of participation, several had experienced them during their recovery period. In part, participants attributed this to insomnia, illustrated clearly by Lorna's reports of wanting to '*sleep forever*'. Existing evidence does suggest that insomnia after stroke may increase the risk of suicidality [377], potentially influenced by depression [482–485], social isolation [486] and feelings of desperation and helplessness [487]. Further investigation of these mechanisms is warranted, but present findings should alert clinicians and researchers to the importance of screening for sleep difficulties after stroke.

Physical factors, such as energy, fatigue, and pain, also contributed to, and were exacerbated by insomnia. Existing evidence suggests that poor sleep may predict fatigue in the chronic stage of stroke [488], however difficulties distinguishing between symptoms of sleep, fatigue, depression, and neurological sequelae of stroke may confound findings [347]. Participants considered physical and social activity as advantageous to sleep, but found that insomnia lowered the threshold for fatigue, and thus were reluctant to engage in health-promoting behaviours such as exercise and socialisation – corroborating studies linking post-stroke fatigue to increased sedentary behaviour [489, 490]. Aware that activity facilitated sleep, but fearful of the cycle between overexertion, exhaustion, and sleeplessness, participants found themselves in a quandary. Taken together with the existing literature, this may influence adherence to exercise or rehabilitative protocols [489, 490], and underlines the importance of person-centred approaches to rehabilitation [491].

Cognitive impairment, worsened by insomnia, contributed to difficulties in social and vocational contexts, often threatening their sense of identity and self-worth. Cognitive impairment is common after stroke [308, 492], and existing evidence does concur that it may be potentiated, and exacerbated, by insomnia [370, 423, 426]. Activities one engaged in prior to stroke (e.g., work) are important aspects of an individual's identity [493]. No longer being able to complete these as one could before can present a barrier to reconciliation and adjustment to a new concept of one's self [493, 494]. For participants in the current sample, this led to guilt over the perceived impact on others (e.g., colleagues, students), which kept them awake at night and contributed to a self-perpetuating cycle of distress, insomnia, and cognitive impairment. Maintaining, or re-establishing, an occupational identity after stroke is often challenging [495], and present findings suggest a role of insomnia in extrinsically exacerbating this by worsening cognitive impairment.

Cognitive impairments also led to challenges socialising and communicating with loved ones. Often, this was compounded by the nature of relationships with family members changing – a common occurrence after stroke [493, 496] – and by insomnia's invisible nature leading to feelings of being misunderstood, isolation, and loneliness [469, 493]. Social support is beneficial to recovery after stroke [497], and necessary for emotional adjustment [494]. Therefore, clinicians, researchers, and those supporting stroke survivors in the third sector should take heed of the potential impact insomnia may have on social support.

Changes to dreams after stroke have not previously been widely reported, although several case-studies have discussed short-term experiences of complete dream loss (Charcot-Wilbrand Syndrome) [328, 498, 499], nightmares [329, 500], and lucid dreaming [329], often linked to co-morbid conditions or medication. Participants' accounts of long-term changes to dreaming adds to these existing studies, and complements research demonstrating relationships between dreams and insomnia in the general population [501]. The content of participants' dreams mostly appeared allegorical of stroke's impact, particularly with regards to perceived loss of identity; a concern common in stroke [493]. Dreams and nightmares often led to a frustrated or anxious state upon awakening, which impeded participants returning to sleep, or made them fearful of going to sleep in the first place. The reasons for these changes are unclear, though Sagnier et al. [329] suggest a role of elevated distress, supported by present findings. Delage et al. [501] posit that behaviours immediately preceding sleep carry the greatest predictive value in anticipating the occurrence of a nightmare. During this time, participants described ruminating on stroke and its impact, often hypervigilant of any potential

threats indicating recurrence. Thus, it's not surprising that their dream content related to this. Stress [501–505], pre-sleep cognitive arousal [501, 506, 507], and an elevated perception of threats [508–511], have all been linked with an increased risk of nightmares in the general population, and there's no obvious reason why the same wouldn't be true after stroke. Moreover, both insomnia and nightmares have been identified as contributors to increased suicide risk [512]. Taken together with present findings emphasising the interrelationships between stroke, insomnia, mental health, and nightmares, this further emphasises the significance carried by sleep after stroke.

2.5.2 Experiences and preferences relating to treatment

2.5.2.1 Summary of main findings

Participants' accounts of their experiences and preferences pertaining to treatment for insomnia emphasised their dismay with the clinical support they received, their desperation to resolve their insomnia, and the extensive and ineffective efforts they have gone to previously. Participants displayed a clear preference for non-pharmacological approaches to treatment - in part driven by scepticism over the safety and efficacy of hypnotic medication - as well as a desire for autonomy over their care.

2.5.2.2 Current findings in the context of the existing literature

Accounts of clinicians' apparent lack of attention to sleep issues are echoed by previous findings both within [315, 317, 513], and beyond the context of stroke [469, 514]. Dyas et al. [469] suggests that having been felt dismissed by professionals in the past, people with insomnia may come to believe they have to emphasise insomnia's impact in order to be taken seriously; potentially explaining why participants' accounts in the current study focus so intensely on the impact of insomnia. Moreover, accounts suggest that feeling as though their sleep complaints were not taken seriously by authority figures (e.g., a neurologist or GP) – particularly when juxtaposed to otherwise excellent support – may have contributed towards participants questioning the legitimacy of their own concerns; normalising their insomnia, and assuming it unrelated to stroke.

Importantly, no participant had undergone a formal assessment for sleep, nor received a diagnosis of insomnia after their stroke, prior to enrolment in the study. For most, sleep was not discussed until years after their stroke or via third-sector support, if at all, echoing findings that sleep is rarely addressed adequately after brain injury [513, 514]. Khot & Morgenstern [315] attribute this shortcoming to poor clinical awareness of sleep after stroke, but lack of validated measures and clinical guidelines specific to stroke may also play a role [396]. This dearth of support meant that returning home from hospital was not indicative of progress in recovery but heralded the point at which support ended, leaving participants feeling isolated and abandoned; akin to the experiences of stroke survivors more broadly [472]. When support was offered, participants felt their preferences were ignored, and that they were pressured into accepting treatments they did not want – or denied those they did. Qualitative explorations of insomnia within primary care suggest that patients feel empowered when clinicians afford them autonomy over their own treatment [469]. Denial of this led to strained relationships between participants in the present sample, and their healthcare providers.

Echoing studies reporting the research priorities of clinicians, researchers, and stroke survivors [515–517], sleep was not seen as a priority for participants. Most participants instead prioritised physical aspects of their recovery, such as mobility or pain. Even those who did prioritise sleep, did so due to the extrinsic value they placed upon sleep as a means of improving other ailments, rather than the intrinsic value of sleep itself. For some, their perception of insomnia’s impact may not have been severe enough to motivate a significant response; a region-beta paradox. Admission that they would consider medication should their health deteriorate is testament to this. Congruently, a qualitative investigation of a behavioural intervention for sleep after stroke found that the severity of sleep difficulties influenced participants’ motivation to adhere to treatment protocols [203]. People’s perceptions of their condition and its treatment are known to influence treatment satisfaction, adherence, and outcomes [428–433], and thus are important factors when considering treatment approaches.

Relatedly, participants displayed a clear preference for behavioural treatments, as is commonly the case in insomnia [106, 518]. The ardency with which participants held this preference, however, appeared linked to insomnia’s perceived severity and impact, again, considering medication as a last resort. Echoing findings linking depression to a preference for behavioural insomnia treatments [106], those most willing to accept medication were generally those who focused more on the objective impact of insomnia (e.g., fatigue and cognition), and less on

the psychological impact. Aversion to medication was predominantly borne out of scepticism around safety and efficacy, and a reluctance to relinquish control over one's sleep to something external. The health locus of control [519] refers to one's perception of whether the agency over health lies internally (i.e., themselves) or externally (i.e., someone or something else). Loss of perceived control over one's health, as is common after stroke (Thompson, 1991), may influence mood and impede functional recovery [520, 521]. Sleep may represent a facet of wellbeing where control can potentially be reclaimed, and thus participants are particularly disinclined to relinquish any more. Thus, while not unique to stroke [469, 518], concerns around dependency are perhaps not surprising.

Prior experiences of dependency after stroke intensified participants' fears, as did anxieties around polypharmacy. Defined as regularly using ≥ 5 medications [522], polypharmacy is common after stroke [523], and is associated with poorer outcomes [524, 525]. Concerned about the toll their already extensive medication regimens were taking on their bodies, participants were reluctant to add any more for fear of side-effects (e.g., stroke recurrence), which they felt they were uniquely vulnerable to due to being stroke survivors. Indeed, hypnotic medications, particularly benzodiazepines, are associated with an increased risk of stroke [104, 526], though, associations are founded from observational studies, and a causality is not confirmed. As was observed in this study, concerns over the volume of medication intake, as well scepticism over a treatment's safety and efficacy, are known to influence adherence [428, 433, 527], and thus are important factors to consider when exploring treatment options.

Of all the perils participants associated with medication, concerns of intentional overdose were unique to one participant (Billy), and not something that has been qualitatively explored previously. Hypnotic medications are associated with increased suicide risk [528–530], as is insomnia after stroke [377]. That this phenomenon may play a role in treatment preferences should alert researchers and clinicians to the importance of incorporating stroke survivors in their own plan of care.

To a lesser extent, there was a specific preference for behavioural treatments such as CBT-I. This was not distinct from aversions to medication, however, and often arose from CBT-I's merits being considered antithetical to medications' demerits. For instance, participants thought behavioural treatments to be more natural – and by extension, healthier – than medication, and were attracted to greater longevity of effects – as opposed to benefits ceasing upon cessation of medication –, and the opportunity to gain insight into why their sleep was

impacted in the first place. Nevertheless, participants' affinity to behavioural approaches was not without caveats. Some concerns were typical to CBT-I (e.g., costs, accessibility, waiting times [180], although others were specific to stroke (e.g., difficulty mobilising at night). Corroborating existing findings [203, 405], stroke-related disability was a barrier to engaging with aspects of CBT-I such as stimulus control, and thus, stroke-specific adaptations to existing protocols may be necessary.

No clear preference arose with regards to the delivery of behavioural treatments. Participants were generally inclined towards options that were familiar to them, but mostly viewed all – online, in-person, individual, and group-based – as acceptable. Anomalous to this, Carla – the only participants to disclose living with aphasia – wished to manage her environment in order to mitigate the detriments of her language difficulties, and preferred individual in-person delivery. While the treatment preferences for insomnia in people living with language difficulties has never been explored, the adaptation of treatment for psychological distress is encouraged to ensure accessibility for people living with post-stroke aphasia [531]. However, connecting with others is valuable throughout stroke recovery [532], exerting positive influences on sleep and mental health [497]. Thus, the social aspects of group-based delivery may afford additional benefits where appropriate.

Particularly distressing for participants was the realisation that once-effective techniques for helping sleep, such as meditative practices, were no longer effective or accessible after stroke. This not only denied participants use of sleep-promoting strategies, but regularly reminded them of what they had lost to stroke. Recent research does suggest stroke-specific adaptations to standard mindfulness protocols, such as shortening sessions and simplifying language, can increase accessibility [533]. Additionally, group-based mindfulness – providing the opportunity for peer-support – also appears beneficial after stroke [534].

2.5.2.3 Clinical implications and future research

Current findings emphasise shortcomings in clinical knowledge and support for stroke survivors with insomnia. In light of participants' accounts, and known relationships between sleep and stroke [312, 332], we strongly encourage the optimisation of sleep in acute stroke settings; particularly regarding patients' exposure to light and noise [347, 375].

Findings elucidating insomnia’s impact on participants’ lives should alert clinicians and researchers to the need for routine screening of sleep after stroke. Building on present results, quantitative investigation of the frequency in which insomnia is discussed and/or formally assessed after stroke would inform recommendations for clinical practice, and align with recommendations to investigate sleep as potentially useful in preventative efforts to curtail recurrent stroke [349]. There does exist, however, a scarcity of insomnia measures validated for use after stroke, which must be addressed in order to facilitate timely detection and treatment of insomnia after stroke.

Ascertainment of factors predicting insomnia after stroke would also aid the development of aetiological models. Recent work by Mohandas et al. [319] goes some way to addressing this, but longitudinal studies investigating the potential role of hospital admission, neuroimaging studies employing a triangulation of lesion-based and correlational methods [395], and studies of physiological and cortical arousal, would be especially beneficial. The proposed aetiological model outlined in Figure 2.2 depicts a complex network of relationships which may drive insomnia after stroke. While it is likely that different people’s experiences may map onto this model differently, the information it provides may benefit clinicians working with stroke survivors who are having difficulty with their sleep. Potential precipitating and perpetuating factors of insomnia were identified across four conceptual domains (social, cognitive, psychological, and physical), which may present as viable targets for clinical intervention. Quantitative validation of these phenomena as potential predictors would be a welcome step in furthering our understanding of the mechanisms underpinning insomnia’s manifestation and perpetuation of insomnia in the context of stroke.

Feelings of isolation, and being misunderstood, may be viable targets for group-based therapies with individuals who share experiences and understanding [535]. However, current findings underline the importance of remaining cognisant of individual differences, and individual delivery may be more appropriate for people living with language difficulties. Thus, further investigation of the challenges faced by sub-populations of stroke survivors would be useful in furthering our understanding of which treatments are effective, and for whom.

Finally, some concerns and beliefs identified in participants' accounts, such as fears that insomnia increases one's risk of recurrent stroke and of stroke occurring in bed, are supported by evidence [56, 318, 347, 353, 358, 383, 536], and thus are not necessarily irrational or dysfunctional. Therefore, approaches that aim to changes one's relationship with their beliefs - rather than directly challenge these beliefs - such as mindfulness [116, 537], may be more appropriate, though should be adapted to meet the needs of stroke survivors [533].

2.5.3 Strengths and limitations

The present study addresses the dearth of research exploring stroke survivors' experiences of insomnia. Results from participants of varying age, educational background, and time since stroke, offer valuable insights into treatment preferences and insomnia's impact, and provide a strong point of departure for future research.

Nevertheless, participants self-selected to take part, which inherently carries a risk of bias [538]. Future ascertainment of population level reference values from large observational studies would be useful in understanding the generalisability of present findings. Similarly, stroke status was determined via participant self-reports, and not further verified. It is therefore possible that participants may have taken part, either knowingly or unknowingly, without a history of stroke. However, self-reports of stroke do show high specificity [539], and at no point throughout any interview did researchers raise concerns about the validity of a participants stroke status.

Additionally, the mean age of participants in the present study was 50.9 years, ranging from 30 to 65 years. While stroke can occur at any age, this is younger than would be considered typical in the general stroke population [540], and may limit the external validity of findings. This may reflect the self-selecting nature of recruitment and online approach to data collection, which may have favoured younger adults who may be more comfortable engaging with digital platforms [541]. Additionally, this approach may have inadvertently excluded those with more severe impairments, who are also more likely to be older [542]. Consequently, the generalisability of these findings to the broader stroke population should be interpreted with caution.

Finally, the present study was unable to garner recommendations for stroke-specific adaptations to behavioural treatments for insomnia. The inductive nature of IPA's methodology [414] precludes researchers from directing the discussion towards very specific questions. However, present results emphasise the importance of person-centred support after stroke, and it is likely that interventions for sleep will need to be flexible to the needs of individual stroke survivors.

2.5.4 Conclusion

This is the first study to qualitatively explore the experiences and treatment preferences of stroke survivors living with insomnia. Results emphasise the negative impact insomnia has on wellbeing after stroke, and position impaired sleep as part of a complex nexus of physical, psychological, cognitive, and social factors. This nexus formed the basis of a proposed aetiological model of insomnia after stroke, which may inform future investigations, and facilitate clinical understanding of the condition. Participants' accounts highlight the shortcomings of current clinical management of post-stroke insomnia and emphasise the importance of optimising sleep in acute-stroke settings, and of affording stroke survivors autonomy in their own care. Similarly, that no participant had received a formal diagnosis of insomnia disorder despite their persistent difficulties highlights the urgent need for clinical education and routine insomnia screening after stroke. Future research should focus on interventions for sleep which incorporate stroke-specific adaptations, including for those living with aphasia, and seek to explore the underlying mechanisms, aetiology, and long-term impact of post-stroke insomnia.

Chapter 3

Assessing insomnia after stroke: a diagnostic validation of the Sleep Condition Indicator in self-reported stroke survivors

The contents of this chapter have been published in the BMJ: Neurology Open, some minor changes have been completed prior to inclusion in this thesis.

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3.1 Abstract

Background: Insomnia is common after stroke and is associated with poorer recovery and greater risk of subsequent strokes. Yet, no insomnia measures have been validated in English-speaking individuals affected by stroke.

Aims: This prospective diagnostic validation study investigated the discriminatory validity and optimal diagnostic cut-off of the Sleep Condition Indicator when screening for DSM-5 insomnia disorder post-stroke.

Methods: A convenience sample of 180 (60.0% women, mean age = 49.61 ± 12.41 years) community-based, adult (≥ 18 years) self-reported stroke survivors completed an online questionnaire. Diagnosis of DSM-5 insomnia disorder was based on analysis of a detailed sleep history questionnaire. Statistical analyses explored discriminant validity, convergent validity, relationships with demographic and mood variables, and internal consistency. Receiver operating characteristic (ROC) curves were plotted to assess diagnostic accuracy.

Results: Data from the sleep history questionnaire suggested that 75 participants (41.67%) met criteria for DSM-5 insomnia disorder, 33 (18.33%) exhibited symptoms of insomnia but did not meet diagnostic criteria, and 72 (40.0%) had no insomnia symptoms at the time of assessment. The SCI demonstrated ‘excellent’ diagnostic accuracy in the detection of insomnia post-stroke, with an AUC of 0.86 (95% CI [0.81, 0.91]). The optimal cut-off was determined as being ≤ 13 , yielding a sensitivity of 88.0%, and a specificity of 71.43%.

Conclusions: The findings of this study demonstrate the SCI to be a valid and reliable method with which to screen for DSM-5 insomnia disorder and symptoms post-stroke. However, a lower threshold than is used in the general population may be necessary after stroke.

3.2 Introduction

A growing body of evidence indicates a bi-directional relationship between sleep and stroke [332]. Insomnia, broadly defined as difficulty with initiating, maintaining, or returning to sleep, which impacts daily functioning, is estimated to affect between 32.2% and 59.2% of people affected by stroke; although estimates vary depending on diagnostic criteria and phase of recovery [321, 379, 380]. Prior to an individual's first stroke, insomnia symptoms are associated with a 30% increased risk of stroke [337]; and post-stroke, are associated with poorer functional and cognitive outcomes [423], and psychological distress [369]. Correspondingly, clinical guidelines and stroke organisations encourage post-stroke sleep assessment [349, 543], to facilitate stroke management and reduce the risk of recurrence.

With respect to the assessment and diagnosis of insomnia disorder in the general population the use of clinical interviews investigating an individual's sleep history is recommended [100]. Psychometric tools commonly comprise part of this process, yet diagnostic validations of insomnia measures post-stroke have only recently been conducted. Chinese translations of the Insomnia Severity Index, Pittsburgh Sleep Quality Index, and Athens Insomnia Scale have demonstrated 'outstanding' [544] diagnostic accuracy post-stroke [412]. However, none of these measures sufficiently capture the diagnostic criteria for insomnia disorder detailed in the Diagnostic and Statistical Manual of Mental Disorders – 5th edition (DSM-5) [67].

A valid diagnosis of insomnia is dependent on specific diagnostic criteria [100], with which a scale intended for clinical use should be aligned. The Sleep Condition Indicator (SCI) [415] is a short self-report questionnaire, designed to screen for insomnia disorder in accordance with DSM-5 criteria [67]. A recent validation of the Indonesian translation of the SCI suggested the tool was valid for use post-stroke but recommended using a higher diagnostic threshold of ≤ 23 [545], rather than ≤ 16 as is used in the general population [415, 546]. However, existing validations of SCI translations do demonstrate heterogeneity in estimates of optimal cut-offs [547–550], suggesting possible confounds from translation and/or nuanced cultural differences.

Accurate diagnoses are inextricably linked to appropriate treatment. Thus, a scale intended for clinical, or research use must be able to discriminate accurately between individuals with and without the condition of interest. Therefore, this study investigated the discriminant validity and optimal threshold of the English

language SCI when detecting insomnia disorder, and symptoms, in a sample of self-reported stroke survivors. Secondary objectives involved assessing convergent validity, examining relationships between the SCI and variables relating to mood and general demographics, and assessing the internal consistency of the SCI after stroke. Additionally, we explored the discriminant validity, optimal threshold, and internal validity of the SCI-2 [551], a two-item version of the SCI comprised of items 3 and 7 from the full scale. The SCI was selected over alternative measures, such as the Insomnia Severity Index (ISI) [150], due to it being explicitly developed to align with DSM-5 criteria for insomnia disorder [415]. This alignment positions the SCI well for use as a screening tool in clinical and research contexts where an accurate reflection of diagnostic criteria is essential. Moreover, the SCI's brevity and ease of use make it a pragmatic choice in clinical contexts where time may be limited, and where cognitive impairments or fatigue may render longer assessments as impractical or burdensome.

3.3 Materials and methods

The pre-registered protocol for this cross-sectional prospective diagnostic validation study can be found at: <https://doi.org/10.17605/OSF.IO/4DGXW>.

3.3.1 Participants and recruitment

Using voluntary response sampling via social media (Twitter/X, Instagram, Facebook, Reddit), community-based adult (≥ 18 years) stroke survivors were recruited across the UK from June 2022 to October 2023. Participants experiencing jet lag (crossed ≥ 2 time zones in the week preceding participation), undergoing treatment(s) that may affect their sleep (e.g. cancer treatment), or working night-shifts were excluded. Participants were not asked to describe whether they had experienced a haemorrhagic or ischaemic stroke, to avoid the risk of individuals self-excluding due to not knowing or remembering this information. Consent was obtained electronically, and no compensation was provided for participation. Responses were anonymous, and participants had the option of sharing their email addresses for future contact by the research team. Email addresses could not be linked to survey responses.

3.3.2 Ethical favourable opinion

A favourable ethical opinion was obtained from the University of Glasgow College of Science and Engineering Research Ethics Committee (#300210126; see appendix L). Optional aphasia friendly materials were available to all participants via a link on the participant information page (see Appendix C).

3.3.3 Sample size estimation

An a priori power analysis was conducted using the ‘MKPower’ package [552]. Initial calculations were performed using parameters obtained from the existing literature. Expected prevalence was set to 32%, as estimated in Baylan et al. [379], and expected sensitivity was set to 89%, estimated by averaging the results of four existing validations of the Sleep Condition Indicator which used the conventional cut-off of ≤ 16 [415, 547, 553, 554]. To achieve a statistical power of 0.8, with $\alpha = 0.05$, and δ (delta) = 0.1, the required sample size was 316. Following data collection from the first 109 participants, sample size estimations were re-evaluated due to stroke specific parameters being made available in Hasan et al. [545]. Expected sensitivity was set to 94% [545] and expected prevalence to 40.37% (taken from the actual prevalence in the preliminary data). With all other parameters unchanged, the estimated sample size required to achieve 80% statistical power was 176.

3.3.4 Measures and materials

Participants completed an online survey comprising questionnaires pertaining to sleep, mental health, and stroke. Demographic information was also collected and comprised age, gender, time since most recent stroke, side of body affected by stroke, and total number of strokes. The index test being evaluated in this study was the SCI [415], total scores of which range from 0-32, with scores ≤ 16 considered indicative of insomnia disorder in the general population [415, 546]. The validity of a two-item variant (SCI-2) [551] was also examined. Reference standard classifications of DSM-5 ‘Insomnia disorder’, ‘Insomnia symptoms’, or ‘no insomnia’ were made following review of a comprehensive sleep history questionnaire [439], for use in people following acquired brain injury, adapted for use online by people after stroke (see Appendix D). Index and reference tests were

completed consecutively. Participants were classified as having ‘insomnia disorder’ if they reported difficulties with initiating or maintaining sleep, or early morning awakening, for at least three nights per week, which caused distress or impairment throughout the day, and symptoms had persisted for at least three months. Participants were classified as having ‘Insomnia symptoms’ if they displayed symptoms of insomnia but did not meet DSM-5 thresholds for severity, duration, or frequency, or their symptoms were likely to be better explained by another sleep disorder (see Table 1.1) [67].

Convergent validity was assessed by exploring correlations between the SCI and the Insomnia Severity Index (ISI) [150], a 7-item insomnia measure, scored from 0-28 with higher scores denoting greater insomnia severity. A Chinese translation of the ISI was recently validated for use after stroke [412].

Depression was measured via the Patient Health Questionnaire (PHQ-9) [443], a 9-item self-report scale scored from 0-27, where greater scores denote greater severity of depressive symptoms. Anxiety was measured via the Generalised Anxiety Disorder Scale (GAD-7) [440], a 7-item self-report measure scored from 0-21, where greater scores denote greater severity of anxious symptoms. Stroke severity was assessed via the Stroke Impact Scale Short Form (SIS-SF) [445], an 8-item self-report score scored from 8 to 40, where lower scores denote greater severity of impact from stroke.

Data were collected via an online survey platform (<https://www.qualtrics.com/>). Classifications were determined by two researchers (DM, MM) independently, blind to SCI scores, following calibration of scoring by MG on 5 responses. Percent agreement between assessor classifications was computed for the first 25 participants, with 100% agreement attained.

3.3.5 Statistical analyses

Statistical analyses were conducted using R (version 4.3.1). Forced responses prevented missing values on the sleep history questionnaire and the SCI. Missing data from other scales (GAD-7, PHQ-9, SIS-SF, ISI) or demographic information were omitted in their respective analyses. In instances where a participant par-

tially completed a scale, data for all items on that scale were omitted. As surveys could be completed across multiple sessions, mean durations greater than 1.5x the interquartile range ($n = 18$) were omitted when calculating summary statistics to curtail the impact of outlier effects.

Normality was assessed visually and with Anderson-Darling tests. Descriptive analysis of demographic and psychometric variables was conducted across classifications. Kruskal-Wallis tests were used for group comparisons of continuous variables, and Dunn’s tests with Bonferroni corrections used for pairwise comparisons. Group differences in categorical data were explored using Fisher’s exact test. Statistical significance was set at $p < 0.05$. Spearman’s correlations were used to quantify relationships between the SCI and continuous variables, and confidence intervals were computed via bootstrapping ($n = 1000$). The internal consistency of all psychometric scales was assessed via Cronbach’s α [555], and the commonly assumed threshold of ≥ 0.70 adopted to indicate acceptable reliability [556].

Receiver operating characteristic (ROC) curves were plotted using the ‘pROC’ package [557] to assess the SCI’s diagnostic accuracy. Youden’s J statistic [558] was calculated to determine the optimal diagnostic cut-off. The area under the curve (AUC) provided an estimate of overall accuracy. AUC values of 0.7 to 0.8 were considered ‘acceptable’, 0.8 to 0.9 ‘excellent’, and > 0.9 ‘outstanding’ diagnostic accuracy [544]. Confidence intervals were computed via bootstrapping ($n = 2000$).

3.4 Results

3.4.1 Participant characteristics

A total of 544 potential participants accessed the study platform (see Figure 3.1), of which 246 exited before providing consent, and 118 did not complete the SCI and/or sleep history. The remaining 180 provided consent and all met inclusion criteria. The mean survey duration was 24.48 minutes ($SD = 10.56$). Participant characteristics are summarised in Table 3.1.

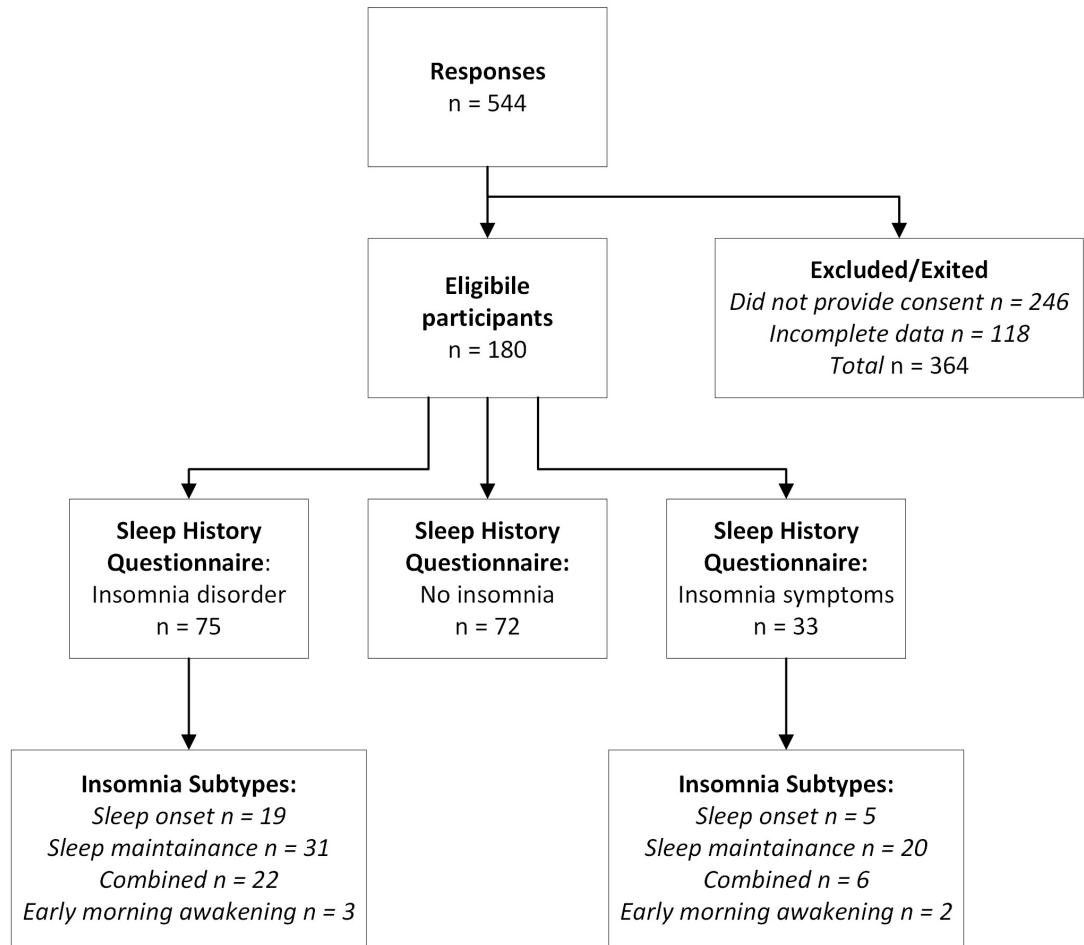


Figure 3.1: Participant flowchart.

After classification based on the sleep history, 75 (41.67%) participants met criteria for insomnia disorder, 33 (18.33%) exhibited insomnia symptoms without meeting diagnostic criteria, and 72 (40.0%) had no insomnia symptoms. A breakdown of participants by classification is available in Figure 3.1 (a confusion matrix of index versus reference standard classifications can be found in Appendix E).

Table 3.1: Participant characteristics¹

	Total (n = 180)	Insomnia Disorder (n = 75)	Insomnia Symptoms (n = 33)	No Insomnia (n = 72)	p value
<i>Variable</i>	<i>Mean (SD)</i>	<i>Mean (SD)</i>	<i>Mean (SD)</i>	<i>Mean (SD)</i>	<i>p value</i>
Age	49.61 (12.41)	49.24 (12.04)	51.82 (11.56)	48.99 (13.18)	.689
Depression (PHQ-9)	10.80 (7.31)	12.97 (7.36) *	11.48 (7.53)	8.20 (6.35) *	<.001
Anxiety (GAD-7)	7.92 (6.19)	9.83 (6.55) *	7.06 (5.94)	6.31 (5.40) *	.004
Stroke Severity (SIS-SF)	30.02 (6.40)	29.00 (5.99) *	28.97 (6.70)	31.60 (6.46) *	.018
Time Since Stroke (Years)	4.16 (4.26)	3.81 (3.55)	3.02 (3.72)	5.09 (5.02)	.069
SCI	13.74 (7.51)	8.47 (4.60)*+	13.52 (5.33) ^{+°}	19.35 (6.79) *°	<.001
ISI	13.40 (6.59)	17.68 (5.59)*°	13.76 (4.31) ^{°+}	8.78 (5.25) *+	<.001
<i>Variable</i>	<i>n (%)</i>	<i>n (%)</i>	<i>n (%)</i>	<i>n (%)</i>	<i>p value</i>
<i>Gender</i>					.087
Female	108 (60.00)	52 (69.33)	15 (45.45)	41 (56.94)	
Male	70 (38.89)	22 (29.33)	18 (54.55)	30 (41.67)	
Non-binary	2 (1.11)	1 (1.33)	0 (0)	1 (1.39)	
<i>Number of Strokes</i>					.336
1	152 (84.44)	62 (82.67)	32 (96.97)	58 (80.56)	
2	22 (12.22)	9 (12.00)	1 (3.03)	12 (16.67)	
3	2 (1.11)	1 (1.33)	0 (0)	1 (1.39)	
>3	4 (2.22)	3 (4.00)	0 (0)	1 (1.39)	
<i>Affected side</i>					.994
Don't remember	20 (11.11)	8 (10.67)	3 (9.09)	9 (12.50)	
Left	84 (46.67)	35 (46.67)	16 (48.48)	33 (45.83)	
Right	76 (42.22)	32 (42.67)	14 (42.42)	30 (41.67)	

¹n Number of participants. SCI Sleep Condition Indicator. ISI Insomnia Severity Index. SD Standard Deviation. Statistically significant differences exist between groups that are marked by the same symbol on one row. One participant was excluded from SIS-SF, PHQ-9 and GAD-7 analyses, and another from SIS-SF analyses, due to incomplete data. Both were classified as 'No Insomnia'.

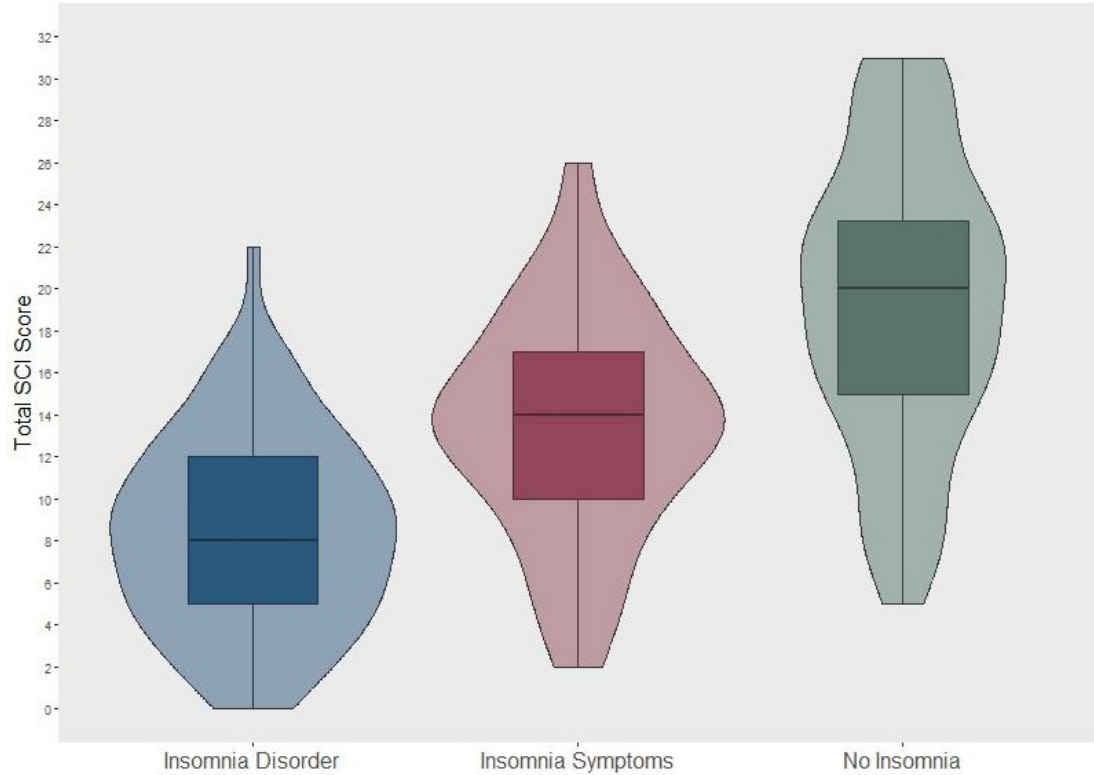


Figure 3.2: Violin and boxplot graphs displaying the distribution of total SCI scores by classification.²

The distribution of total SCI scores by classification is displayed in Figure 3.2. Skewness and kurtosis were 0.40 and 2.46, respectively. Visual inspection and Anderson-Darling tests ($p = .004$) indicated non-normal distribution of total SCI scores.

3.4.2 Sensitivity and specificity of the SCI

When detecting insomnia disorder post-stroke using the SCI, the AUC was 0.86 (95% CI [0.81, 0.91]; Figure 3.3), with an optimal cut-off of ≤ 13 , yielding sensitivity, specificity, and Youden's J values of 88.0%, 71.43%, and 0.59, respectively (see Table 3.2). When detecting insomnia symptoms, the AUC was 0.85 (95% CI [0.80, 0.91]; Figure 3.3), with an optimal cut-off of ≤ 14 , yielding sensitivity, specificity, and Youden's J values of 81.48%, 76.39% and 0.58, respectively (see Table 3.2). Both AUC curves are indicative of 'excellent' diagnostic accuracy [544]. Delong's test for two paired ROC curves found no statistical difference ($p = 0.91$) in the SCI's ability to detect insomnia disorder versus symptoms. Using

²Whiskers represent minima and maxima. The line dividing boxes represents the median.

the sensitivity observed when detecting insomnia disorder, with all other parameters unchanged, returns a power of 0.71. Sensitivity, specificity, and Youden's J values for all thresholds can be found in appendix F. Predictive values at a range of hypothetical prevalence rates can be found in appendix H.

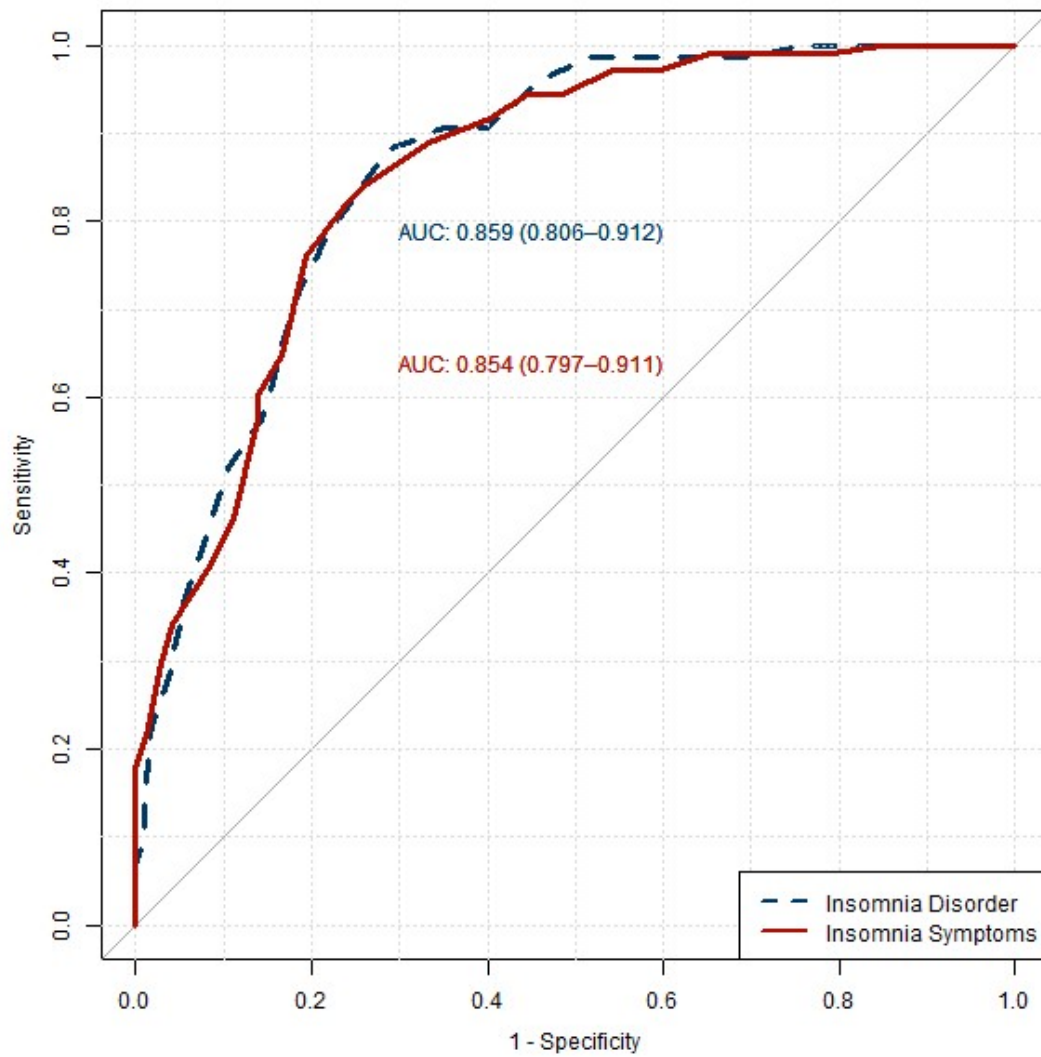


Figure 3.3: ROC curves displaying the accuracy of the SCI when detecting DSM-5 insomnia disorder and symptoms, including AUC values and 95% confidence intervals.

Table 3.2: Threshold values and associated specificity, sensitivity, and Youden's J when assessing Insomnia Disorder and Insomnia Disorder with Symptoms using the SCI.³

<i>Insomnia Disorder</i>				<i>Insomnia Disorder and Symptoms</i>			
<i>Threshold</i>	<i>Specificity (%)</i>	<i>Sensitivity (%)</i>	<i>Youden's J</i>	<i>Threshold</i>	<i>Specificity (%)</i>	<i>Sensitivity (%)</i>	<i>Youden's J</i>
8	89.52	52.00	0.42	8	91.67	40.74	0.32
9	85.71	57.33	0.43	9	88.89	46.30	0.35
10	81.90	70.67	0.53	10	86.11	57.41	0.44
11	80.95	73.33	0.54	11	86.11	60.19	0.46
12	78.10	78.67	0.57	12	83.33	64.81	0.48
13	71.43	88.00	0.59	13	80.56	75.93	0.56
14	64.76	90.67	0.55	14	76.39	81.48	0.58
15	60.00	90.67	0.51	15	73.61	84.26	0.58
16	54.29	96.00	0.50	16	66.67	88.89	0.56
17	48.57	98.67	0.47	17	59.72	91.67	0.51

³Optimal thresholds are displayed in bold. Youden's J - (J = sensitivity + specificity - 1)

3.4.3 Sensitivity and specificity of the SCI-2

ROC analysis was performed on the summed totals for items 3 and 7 of the SCI (Figure 3.4, forming the SCI-2 [551]). When detecting DSM-5 insomnia in stroke survivors, the AUC for the SCI-2 was 0.79 (95% CI [0.73, 0.85]), with an optimal cut-off of ≤ 2 . Corresponding with a specificity of 68.57%, sensitivity of 76.0%, and a Youden's J of 0.45. Delong's test provided statistical evidence ($p < .001$) that the SCI outperformed the SCI-2. Sensitivity and specificity values at all thresholds can be found in appendix G. The positive and negative predictive values of the SCI in this sample, using the optimal threshold of ≤ 13 , were 0.69 and 0.89, respectively.

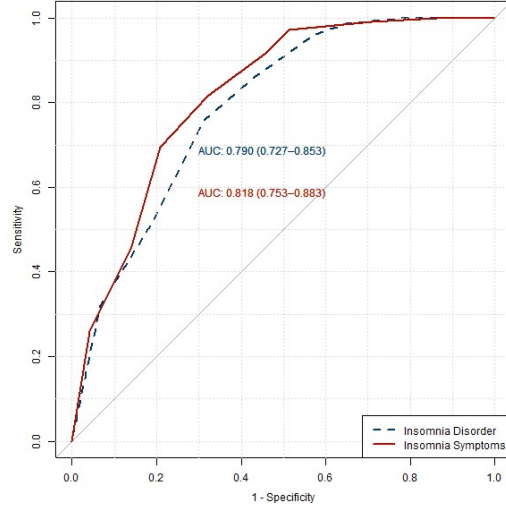


Figure 3.4: ROC curves displaying the accuracy of the SCI-2 when detecting DSM-5 insomnia disorder and symptoms, including AUC values and 95% confidence intervals.

3.4.4 Convergent validity

Spearman's rank correlation was computed to assess the relationship between total scores on the ISI and SCI, with ties handled by average ranks. Results revealed a large negative correlation between the two scales ($r(178) = -0.86$, 95% CI [-0.91, -0.80]; Figure 3.5).

3.5 Associations between the SCI, demographic, stroke, and mood variables.

Spearman's rho was computed to determine the strength of the relationship between total SCI scores and all other continuous variables. Results are presented in Table 3.3.

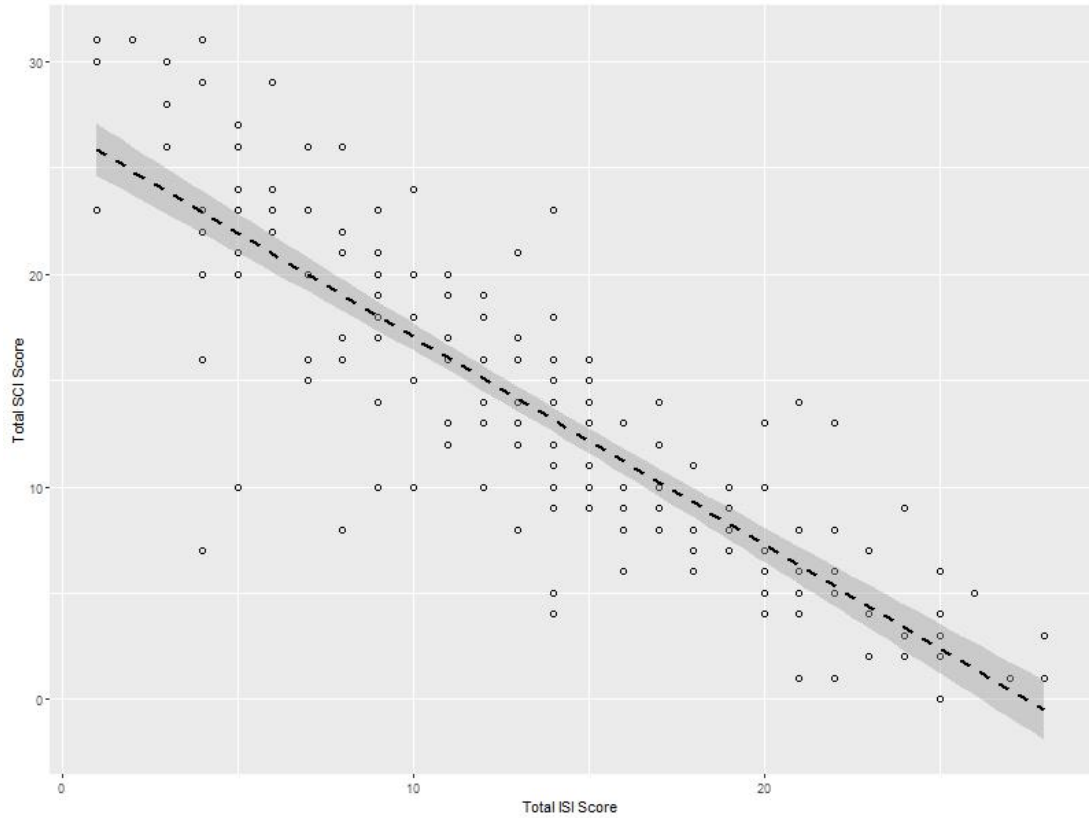


Figure 3.5: Scatter plot visualising the relationship between total SCI and ISI scores.

Table 3.3: Spearman's Correlation Coefficients, Confidence Intervals, and P values for Relationships Between the SCI and Select Demographic and Psychometric Variables⁴

Variable	r	95% CI	p value
Age	-0.07	(-0.20, 0.08)	.357
PHQ-9	-0.63	(-0.72, -0.52)	<.001
GAD-7	-0.47	(-0.57, -0.33)	<.001
SIS-SF	0.47	(0.34, 0.57)	<.001
Time Since Stroke	0.03	(-0.12, 0.18)	.713

3.6 Internal validity

The reliability of all included psychometric scales was assessed via Cronbach's α , and all deemed acceptable. For the SCI ($n = 180$), Cronbach's α was 0.84 (95% CI [0.80, 0.87]), for the SCI-2 ($n = 180$) α was 0.76 (95% CI [0.69, 0.82]), for the GAD-7 ($n = 179$) α was 0.91 (95% CI [0.89, 0.93]), for the PHQ-9 ($n = 179$), α was 0.89 (95% CI [0.87, 0.92]), and for the SIS-SF ($n = 178$) α was 0.83 (95% CI [0.78, 0.86]).

⁴CI - Confidence interval

3.7 Discussion

This is the first study examining the SCI’s validity and reliability, and that of any insomnia screening tool, in a sample of English-speaking stroke survivors. Results from 180 participants indicated that 41.7% met DSM-5 criteria for insomnia disorder, and 60.0% exhibited insomnia symptoms (including those classified as insomnia disorder), consistent with meta-analytic findings on post-stroke insomnia prevalence [321, 379, 380], and supporting the representativeness of the sample. The remaining 40% had no insomnia symptoms.

The SCI demonstrated ‘excellent’ [544] diagnostic accuracy when screening for DSM-5 insomnia disorder ($AUC = 0.86$) and/or symptoms ($AUC = 0.85$) post-stroke. Moreover, examination of internal consistency indicated acceptable reliability (Cronbach’s $\alpha = 0.84$). The SCI-2 demonstrated ‘acceptable’ accuracy, with an optimal cut-off of ≤ 2 ; paralleling validations in the general population [551]. However, the full SCI demonstrated significantly higher accuracy, and should be the preferred assessment method where possible.

Estimates of diagnostic accuracy in the current study are similar, albeit slightly lower, to recent validations in stroke populations of the SCI in Indonesian [545] and the Insomnia Severity Index, Pittsburgh Sleep Quality Index, and Athens Insomnia Index in Chinese [412]. Nonetheless, comparisons should be drawn cautiously. Existing validations of SCI translations demonstrate heterogeneity in estimates of optimal cut-offs [547–550], suggesting possible confounds from translation and/or nuanced cultural differences. Additionally, a recent meta-analysis demonstrated that condition prevalence may influence the sensitivity and specificity of binary diagnostic tests [559]. The prevalence of insomnia in the previous validations [412, 545] was 15.6% and 31.0%, respectively, both being lower than the current study (41.7%) and previous estimates of post-stroke insomnia prevalence [321, 379]. Thus, the variance in prevalence should be considered when comparing studies of diagnostic accuracy, and when considering a screening tool for clinical or research use.

The optimal cut-off for detecting insomnia disorder using the SCI in the current study was found to be ≤ 13 . This is lower than both the conventional cut-off for the general population of ≤ 16 [415, 546], and the optimal cut-off of ≤ 23 observed in the validation of the Indonesian translation of the SCI post-stroke [545]. Were the conventional cut-off adopted in the current sample, 96% of positive insomnia disorder cases would be correctly identified. However, approximately

45.7% of participants without insomnia disorder would be incorrectly classified as a positive case (i.e., having insomnia disorder). Depending on the intended use for the scale, there may be instances where clinicians or researchers wish to maximise sensitivity at the expense of specificity (or vice versa) but should do so with caution. Use of an erroneously high threshold on the SCI will lead to an increased false positive rate, potentially leading to inappropriate treatment and use of resources in clinical settings; and may cast doubt on the validity of findings in research.

Understanding the heterogeneity of optimal diagnostic thresholds is important for ensuring the SCI is appropriately used in different clinical samples, and future research should explore this further. One possibility is that poorer sleep post-stroke may necessitate a lower threshold. Mean SCI scores in the current study ($\bar{x} = 13.74$, $SD = 7.51$) are slightly lower, and display greater variance than results from a retrospective exploration of scores in 200,000 adults who completed the SCI ($\bar{x} = 14.97$, $SD = 5.93$) [546]. However, the results of the latter [546] may be biased towards people with sleep impairment. Assuming a normal distribution, these values suggest that the percentage of participants in Espie et al. [546] with probable insomnia (i.e., scoring 16 or less) is approximately 56.89%. This is considerably higher than would be expected in the general population [85], and thus true population mean SCI scores are likely higher.

Variation in reference standards likely also contribute to variability in optimal cut-offs, and ideally studies seeking to validate measures of insomnia should select reference standards in accordance with guidelines on the assessment and diagnosis of insomnia disorder [100]. In studies of diagnostic accuracy, the reference standard assumes infallibility. Yet, the index test may not always be solely responsible for the discrepancy [560]. The conventional cut-off of ≤ 16 [415, 546] was determined as the minimum score necessary for putative DSM-5 insomnia disorder, with convergent validity assessed against the ISI [150]. The ISI boasts commendable accuracy but standalone, is not a recommended tool for diagnosing DSM-5 insomnia disorder [100], and is less specific than clinical interviews. Thus, when comparing the optimal diagnostic cut-offs between studies, one should be cognisant of the possibility of differences in reference standards explaining some or all the variation in optimal cut-offs.

Spectrum bias, a phenomenon whereby accuracy of a diagnostic test may be confounded by symptom severity [561], may further explain discrepancies in optimal diagnostic thresholds between studies. Scores closer to the cut-off are more prone to misclassification, therefore, studies where index test scores demon-

strate a bimodal distribution have a greater risk of spectrum bias. Hasan et al. [545] excluded 164 participants with sleep or psychiatric disorders other than insomnia in their validation. Many of these participants would likely have scored lower on the SCI regardless of insomnia classification, as suggested by the relationships between SCI score and measures of mental health in the current study and corroborated by meta-analytic results from Hertenstein et al. [75]. Likely as a result of their inclusion criteria, mean SCI scores in Hasan et al. [545] for participants without insomnia were 29.55 (SD = 1.96), with little variance; compared to 19.33 (SD = 6.81) in the current study, which is more akin to previous research [313]. Such a skewed distribution of scores allows specificity to remain high at higher thresholds, contributing to potentially misleading estimates of accuracy and optimal cut-offs. Hasan and colleagues demonstrate that the Indonesian SCI can discriminate post-stroke insomnia disorder in the absence of potential confounds. The present study adds to this by validating the English SCI in self-reported stroke survivors and doing so whilst including participants with comorbid psychological or sleep disorders; thereby increasing generalisability of the findings. Results of the current study, when considered in the context of the existing literature, highlight the importance of considering spectrum bias in studies of diagnostic accuracy. When considering a screening tool, researchers and clinicians should consider whether a study's selection criteria are clinically meaningful to the population they intend to use the test in.

3.7.1 Strengths and limitations

This study has several strengths. A priori power analyses, a rarity in studies of diagnostic accuracy [562], ensured the study was suitably powered to obtain reliable estimates of test accuracy. Consecutive completion of index and reference tests reduced the risk of temporal changes in disease state. Finally, stratifying analyses by insomnia classification reduced the risk of spectrum bias and prevalence influencing estimates of accuracy [559].

Nonetheless, voluntary response sampling does carry a risk of response bias and may lead to over-representation of participants with an interest in sleep, or more severe sleep difficulties. However, prevalence in the current sample aligns with that of previous meta-analytic estimates [321, 379, 380]. Recruiting participants via social media may also have led to a younger than expected stroke sample (mean age = 49.61 ± 12.41 years). Future research may wish to consult older stroke survivors to explore methods of increasing the accessibility of online

stroke research to a wider age range. Nevertheless, stroke can affect people of any age, and research representative of younger stroke survivors is necessary, particularly to understand the challenges of improving life after stroke for working age individuals - perhaps with young families - compared to older adults. Results of a linear regression from a large cross-sectional study ($n = 200,000$) by Espie and colleagues [546] did demonstrate that SCI score was predicted to decrease by 0.057 points per year of life. Findings from the current study demonstrated no significant relationship between age, or gender, and total SCI score, however, it is likely that a much larger sample size would be needed to detect an effect as small as that described in Espie et al. [546]. Thus, although the modulating effect of demographic characteristics on the accuracy of the SCI was not explored, current findings provide no evidence to suggest that such an effect exists.

Due to the online nature of recruitment, participant self-reports of stroke were not verified by another means. Various efforts were made however, to minimise the risk of individuals without a history of stroke volunteering to take part. Firstly, recruitment adverts were shared explicitly by stroke researchers and third sector organisations supporting stroke survivors, targeting stroke peer support groups. Moreover, offering no incentive to participants removed the risk of individuals taking part without a history of stroke for financial gain. Furthermore, while the results of a systematic review by Woodfield et al. [539] demonstrate that the sensitivity and positive predictive value of self-reported stroke is low in samples with low prevalence, specificity (96 to 99.1%) and negative predictive value (88.2 to 99.9%) were consistently high. As one would expect, authors report that positive predictive values increased in line with prevalence. Thus, in samples with high prevalence of stroke, such as in the current study, the risk of false positives is likely to be small. We therefore believe self-reports of stroke in the present sample to be reliable. Nevertheless, where time and resources allow, future researchers may wish to verify self-reported stroke status by recruiting participants from stroke outpatient clinics or obtaining clinical records.

Additionally, the number of incomplete responses ($n = 118$) is worth noting. This may indicate systemic challenges related to conducting stroke research online, or difficulties faced by potential participants with language difficulties. Relatedly, the duration of the survey was relatively long, and future research may consider limiting survey length further to increase accessibility.

Although this study explores the psychometric properties of the SCI in a sample of stroke survivors, no exploration of whether individual items were interpreted as intended was conducted. Cognitive interviewing [563] – a means by which to ascertain whether respondents correctly comprehend survey content and instructions – is recommended by COSMIN (CONsensus-based Standards for the selection of health Measurement Instruments) guidelines [564] when applying a measurement tool in a population for which it was not originally developed. Due to the anonymous and online nature of data collection, cognitive interviewing was beyond the scope of the present study. Future research should consider incorporating cognitive interviewing techniques in a subsample of stroke survivors to ensure the SCI is interpreted as intended, thereby further validating the content validity of the scale in this population.

A further limitation to the current study concerns the reference standard employed. While the sleep history questionnaire [439] has not formally been validated for use after stroke, it was explicitly designed to mimic a structured clinical interview for DSM-5 insomnia disorder [67], allowing comprehensive differential classification. Perfect classification agreement was also observed in the subsample assessed by two independent researchers. Although the risk of misclassification via the reference standard cannot be ruled out, stratification by condition severity likely helped mitigate the risk of this influencing results. Moreover, if substantial misclassification had occurred would likely have resulted in the SCI demonstrating poor discriminant validity – contrary to the excellent diagnostic accuracy observed. Additionally, convergent validity was assessed via the ISI, which has been validated in traumatic brain injury [565] and Chinese-speaking stroke survivors [412]. This triangulation of methods strengthens confidence in the results. Nevertheless, a potential risk of misclassification does remain, the occurrence of which may have influenced estimates of sensitivity, specificity, and thus, optimal threshold. Future research may wish to further validate the discriminant validity and optimal threshold of the SCI after stroke by adopting clinician administered interviews when assessing insomnia.

Finally, participants were not asked which type of stroke they had. This was a methodological decision to avoid excluding participants who did not know or could not remember. Existing evidence does indicate potential relationships between insomnia and stroke type/location [2], therefore future researchers may wish to consider purposive sampling methods that allow exploration of whether these phenomena may influence insomnia presentation and the accuracy of diagnostic measures.

3.7.2 Conclusions

In summary, this study confirms the validity and reliability of the English version of the SCI when detecting DSM-5 insomnia in self-reported stroke survivors and indicates that a lower threshold than is traditionally used should be considered when using the SCI in the stroke population. At the optimal diagnostic threshold of ≤ 13 , the SCI demonstrates ‘excellent’ diagnostic accuracy. The brevity of the tool makes it attractive for clinical and research use and the SCI-2 does offer an acceptable shorter alternative. Nevertheless, the accuracy of the SCI-2 is outperformed by the full SCI, and use of the latter should be preferred where possible. In conclusion, the SCI should be considered a valid and reliable screening tool for DSM-5 insomnia disorder and insomnia symptoms post-stroke.

Chapter 4

The effectiveness of the behavioural components of cognitive behavioural therapy for insomnia in older adults: A systematic review

The contents of this chapter have been published as follows:

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Minor updates and expansion to some sections have been completed prior to inclusion in this thesis.

4.1 Abstract

Background: Insomnia is more prevalent in older adults (≥ 60 years) than in the general population. Cognitive behavioural therapy for insomnia is the gold-standard treatment; however, it may prove too cognitively taxing for some. This systematic review aimed to critically examine the literature exploring the effectiveness of explicitly behavioural interventions for insomnia in older adults, with secondary aims of investigating their effect on mood and daytime functioning.

Methods: Four electronic databases (MEDLINE – Ovid, Embase – Ovid, CINAHL, and PsycINFO) were searched. All experimental, quasi-experimental and pre-experimental studies were included, provided they: (a) were published in English; (b) recruited older adults with insomnia; (c) used sleep restriction and/or stimulus control; (d) reported outcomes pre-and-post intervention.

Results: Database searches returned 1689 articles; 15 studies, summarising the results of 498 older adults, were included – three focused on stimulus control, four on sleep restriction, and eight adopted multicomponent treatments comprised of both interventions. All interventions brought about significant improvements in one or more subjectively measured facets of sleep although, overall, multicomponent therapies demonstrated larger effects (median Hedge’s $g = 0.55$). Actigraphic or polysomnographic outcomes demonstrated smaller or no effects. Improvements in measures of depression were seen in multicomponent interventions, but no intervention demonstrated any statistically significant improvement in measures of anxiety. This corroborates with the existing consensus that multicomponent approaches confer the most benefit and adds to the literature by demonstrating this to be the case in brief, explicitly behavioural interventions.

Conclusions: This review guides future study of treatments for insomnia in populations where cognitive behavioural therapy for insomnia is not appropriate.

4.2 Introduction

4.2.1 Insomnia in older adults

Insomnia disorder is characterised in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) [67] as difficulties initiating or maintaining sleep, or early-morning awakening. These difficulties must occur despite sufficient opportunity for sleep, three or more times per week, for at least 3-months, and cause distress and/or impairment to daytime functioning. Although estimates of prevalence vary depending on which diagnostic measure is used (6%–48% in the general population) [85], it is widely accepted that the prevalence of insomnia and insomnia symptoms, specifically issues with sleep maintenance and early-morning awakening, are greater in older adults than in younger populations [85, 566–568]. USA-based survey reports using DSM-5 criteria estimate the point prevalence of insomnia in older adults to lie between 12% and 20% [94], approximately double that observed using the same criteria in the general population [85, 90]. Longitudinal observational research in the UK suggests that the prevalence of insomnia increases $1.1\times$ for every ten years of lifespan [569], and one study of 6800 adults over 65-years noted an incidence rate of 5% per year [570].

Age-related changes in lifestyle and physiology often translate to changes in sleep architecture, such as shorter and more fragmented sleep [571, 572], as well as less time spent in slow wave and rapid eye movement (REM) sleep [571]. Where elevated cognitive arousal is central to the pathophysiology of insomnia in healthy younger adults [25, 36, 231], age-related neurodegeneration and changes to circadian rhythms [573–576] are potential predisposing factors in older adults. Additionally, these age-related changes may influence beliefs about sleep, potentially leading to sleep-related anxiety in older adults, potentially exacerbating insomnia [568]. Correspondingly, variations to Spielman’s three factor model of insomnia [224] have been proposed to account for the role of age-related changes in sleep [577].

Insomnia carries an increased risk of many adverse effects in all populations, and should not be considered normal or benign. Individuals with chronic sleep disturbances experience a greater risk of psychological distress and disorders such as depression and/or anxiety [36, 74, 75, 81, 82, 470, 569, 578–582], often in a bi-directional manner [75, 81, 82, 470, 578, 582, 583]. In older adults, insomnia has been associated with an increased risk of frailty [584, 585], malnutrition [586],

and cardiovascular disease [343]. Evidence also links insomnia to an increased risk of suicidality in older adults [78], although research exploring this phenomenon is comparably less than other populations. Insomnia in later life has also been associated with disruption in subjective [73, 587] and objective [571, 588] reports of cognitive ability [589, 590], and cognitive decline [591–593]. Recent research has also demonstrated that sleep deprivation inhibits the clearance of waste products from human brains [5], and meta-analytic results demonstrate that insomnia is associated with a $1.53 \times$ increased risk of all-cause dementia [594]. Middle-aged reductions in total sleep time have been linked to the accumulation of beta-amyloid (A β) and tau proteins, which are believed to be related to Alzheimer’s disease, in later life [595]. Authors of a recent study exploring relationships between sleep and cognitive impairment after stroke [426] postulated that reduced time spent in non-REM sleep may impair the flow of cerebral spinal fluid [596], increasing tau pathology, and contributing to cognitive impairment in older adults [597]. Correspondingly, emerging research advocates for the investigation of sleep as a potential target in preventing dementia [593, 598].

Insomnia costs approximately £40bn per annum in the UK alone [599], and has been demonstrated to increase the overall utilisation of healthcare [600]. Wickwire et al. [600] found untreated insomnia to be associated with increased costs related to inpatient care, emergency care, and prescriptions. As the proportion of the world’s population over 60 years old continues to grow, the need for effective and easily disseminated insomnia treatment in older adults is abundantly apparent.

4.2.2 Interventions for insomnia in older adults

In its chronic manifestation, insomnia is unlikely to resolve without intervention [139], and evidence suggests chronicity of insomnia may be greater in older adults [601], for whom co-morbidities and extant medication use can complicate treatment implementation [602]. Treatment can be pharmacological or non-pharmacological. Pharmacological intervention is only recommended in people who do not respond sufficiently to non-pharmacological treatments [100, 108], and only for a short period of time, due to the risk of tolerance, dependence, and side effects [100, 105]. Physiological changes associated with ageing increase the risk of adverse side-effects from medications [568]. These can range from daytime sleepiness and cognitive impairment, to falls, dementia, and suicidality [102, 103, 603], contributing to increased costs for individuals and society [604]. Thus, non-

pharmacological interventions are recommended as the first line of treatment across population groups [91, 99, 100, 605], including older adults [605]. Despite this, older adults are more likely than younger populations to be prescribed hypnotic medication; and for longer [85, 606–608]. Recent analysis of electronic health record data from 163,748 individuals in England, of whom approximately 62.1% were over 55, found that 11% received a prescription for hypnotic medication, despite only 6% having a formal diagnosis of insomnia [609]. Studies from other high-income countries, focusing exclusively on participants aged 65 or older, estimate prescription rates of hypnotic medication to lie between 15%–30% [610–612]; apparently increasing with age [610].

Cognitive behavioural therapy for insomnia (CBT-I) is recommended as the first line of treatment for insomnia disorder [99, 100, 108]. CBT-I is comprised of several distinct components, including Stimulus Control, Sleep Restriction, Sleep Hygiene, Relaxation Training, and Cognitive Therapy [111, 120]. Despite being prescribed less frequently than hypnotic medications, CBT-I demonstrates efficacy comparable to or exceeding that of pharmacological interventions on insomnia symptoms [103], lasting up to a year post-treatment [179]. Recent meta-analytic evidence has demonstrated CBT-I to be safe and effective in older populations [164], and suggest it may also prevent incidence of depression in older adults with insomnia [613].

Nevertheless, some issues with CBT-I do exist. Non-response rates can be high [614], and in some clinical populations such as stroke survivors [402–407], the longevity of the effects of CBT-I may be low. One reason for this could be that the lengthy protocols and cognitive components of CBT-I may be too taxing for some [203]. Correspondingly, Hughes et al. [185] remarked on the clinical and functional heterogeneity among the older adult population, and recommended adapting aspects of CBT-I to accommodate potential barriers such as cognitive impairment, functional limitation and comorbidities. One recent study exploring CBT-I in geriatric primary care found that 33/70 participants either elected not to take part ($n = 28$) or withdrew during the intervention ($n = 5$), primarily due to the perceived level of effort involved [615]. Authors reported that older participants were more likely to decline participation. Relatedly, results from a recent qualitative exploration of an intervention coupling self-directed CBT-I and benzodiazepine discontinuation [616] found that older adults found the degree of commitment involved in CBT-I to be challenging; particularly when juxtaposed against the ease of taking medication. Additionally, CBT-I also requires adequately trained practitioners, of which there is currently a shortage [184], and limited dissemination of CBT-I has been identified as a principal systemic barrier

to guideline-concordant care [180]. Previous research has recommended shorter protocols or alternative methods of delivery to ameliorate these issues [180, 185, 617]. Moreover, the results of Chapter 2, § 2.4.2.2, identified several fears and worries relating to insomnia after stroke, which may not be viable targets for cognitive therapy. Thus, a comprehensive investigation of easy to disseminate and explicitly behavioural interventions for insomnia is indicated in several populations, and older adults serve as an excellent point of departure.

To do this, it is important to identify which components of CBT-I are critical, or most effective; of course, this is impossible when CBT-I is delivered as a multicomponent intervention. Elucidating which components of CBT-I contribute most potently to its efficacy - and in which populations they do so optimally - has recently enjoyed renewed interest, and can inform individual and population specific adaptations to the standard CBT-I protocol [25, 70, 145, 148]. Brief behavioural treatment for insomnia (BBTI) [618, 619] is a manualised treatment of insomnia which combines stimulus control and sleep restriction without any cognitive component. BBTI was developed as a shorter alternative to CBT-I, that could be delivered by a wider range of practitioners, thereby addressing several systemic barriers to CBT-I's dissemination [618]. The efficacy of BBTI has been demonstrated in a range of populations [620, 621], as well as via digital delivery [622]. One recent meta-analysis of RCTs demonstrated BBTI to be effective in alleviating symptoms of insomnia in older adults [623], suggesting that combined stimulus control and sleep restriction are feasible alternatives to CBT-I. However, isolated delivery of these treatments may offer a more personalised approach to treatment. Moreover, a more parsimonious approach may aid in elucidating the current gap between supply and demand of CBT-I. Several meta-analyses have found sleep restriction and stimulus control, delivered as monotherapies, to be effective at alleviating symptoms of insomnia in the general population [128, 131, 148, 624], however their utility in older adults is less clear. One recent component network meta-analysis [148], summarising the data of 31,452 participants, found behavioural and cognitive treatments to be similarly efficacious, although most effective when delivered within multicomponent CBT-I. More specifically, authors reported that sleep restriction was most effective at reducing wake after sleep onset and improving sleep efficiency. Whereas stimulus control appeared most potent at reducing sleep onset latency, implying that personalised approaches to treatment may be feasible and appropriate in some scenarios. However, participants were generally middle-aged and female, and whether these findings are applicable to older populations is not known.

Although the present thesis focuses on insomnia after stroke, the decision to instead focus this systematic review on older adults was driven by theoretical and practical considerations. At the time of writing, while several studies had investigated CBT-I after stroke [402–408], no research had explored specifically stimulus control and sleep restriction as treatments in this population, precluding a stroke-specific review. However, given the relationship between stroke-risk and age [540], as well as the overlap in pathophysiological, psychological, and functional morbidities across both populations [294, 625], older adults were considered an appropriate adjacent population to explore these interventions. Nevertheless, caution is warranted when generalising these findings to stroke populations. Rather than providing direct evidence for post-stroke interventions, this review intends to offer a theoretical and empirical foundation to guide future research within stroke contexts.

4.2.3 Aims and objectives

This review examined the efficacy of sleep restriction and stimulus control, in isolation and in tandem, in treating insomnia and insomnia symptoms in older adults. A secondary aim was to explore the effect of these interventions on mood-related and daily functioning outcomes. The results of this review should inform clinical practice and guide further research in other populations, and of other behavioural interventions.

4.3 Methods

The review protocol was registered with International Prospective Register of Systematic Reviews (PROSPERO), registration number CRD42020220663. Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) [626] guidance was used in the development of the research question, and in the selection and reporting of studies.

4.3.1 Search procedure and inclusion criteria

A systematic search of MEDLINE—Ovid, Embase—Ovid, CINAHL, and PsycINFO was conducted from inception to 3 December 2020. This was updated on the 6th of August 2024, to ensure the review was up to date. The search strategy was developed with support from a University of Glasgow Librarian, using keywords and Medical Subject Headings (MeSH), and was adapted for use across all bibliographic databases (the strategy for MEDLINE can be found in appendix I). The following inclusion criteria were employed.

4.3.1.1 Population

Common definitions of ‘Older Adult’ were explored. These have varied throughout the literature, with thresholds of 60 years or 65 years being used by different organisations [627, 628]. To include as many relevant studies as possible, the lower threshold of 60 years was adopted. When mixed ages were reported, only those where at least 80% of the sample were ≥ 60 years old were included. To determine this, a normal distribution was assumed, and the z-value for those > 60 years was calculated. Proportions were then derived by selecting the corresponding value from a Standard Normal Table, multiplying by 100, and subtracting from 100.

Studies were included whether insomnia was classified using recognised clinical diagnostic criteria (e.g., DSM, ICSD, ICD) or was self-reported. Studies where participants presented with sleep and/or circadian disorders distinct from insomnia or were undergoing treatment of severe medical conditions (where the individual was not in remission), for example active cancer treatment, were excluded.

4.3.1.2 Intervention

Only studies with an explicitly behavioural intervention, including sleep restriction and/or stimulus control as primary components, were included. Stimulus control and sleep restriction were permitted to be delivered individually or combined in a multicomponent intervention. Studies that included interventions containing different behavioural approaches, such as mindfulness, were excluded;

however, allowances were made for those including sleep hygiene [114], relaxation techniques, and sleep education – provided these were delivered alongside stimulus control or sleep restriction. Studies where the experimental groups were exposed to any type of cognitive intervention were excluded. That being any intervention where the mechanisms driving change were focused on an individual’s thoughts rather than manipulation of the external environment, behaviour change, or reduction of physiological arousal, meaning that employment of CBT-I was grounds for exclusion.

4.3.1.3

Studies with active, passive, or no control groups were included.

4.3.1.4 Outcomes

The primary outcome of this review was objective and/or subjective changes in sleep and/or insomnia-related measures, pre-to-post intervention. Secondary aims focused on outcomes related to psychological outcomes (depression, anxiety) and daytime functioning. Only studies where primary outcomes reported at pre-and-post intervention were included.

4.3.1.5 Study type

Any type of quantitative, pre-experimental, quasi-experimental or experimental study was included. Narrative, critical or systematic reviews, meta-analyses, commentaries without original results, studies with unpublished data, non-peer-reviewed studies, non-human animal studies and qualitative studies were all excluded. In studies with mixed designs, qualitative results were excluded. No minimum sample size was required.

4.3.2 Study selection and data extraction

Results of database searches were exported to EndNote X9 (version 3.3), and duplicates were deleted. References remaining after this step were uploaded to Rayyan QCRI [629] and a second round of de-duplication was conducted. Titles and abstracts were screened for inclusion by two independent researchers, optimising selection of appropriate studies [630]. The full texts of identified studies were then sourced and screened by the same researchers independently. Disagreements (on average 3%) were resolved by discussion, and by involvement of a third researcher if agreement was not met. Reference sections were also hand searched for studies fitting inclusion criteria.

A study-specific proforma was created, and two researchers independently extracted data from included studies. These included: citation details, design, control type, sample size, mean age, proportion female, treatment type, number of sessions, delivery method, insomnia definition, and objective and subjective outcome measures at all time points including follow-up. For outcome measures, numerical values for the mean ($\bar{X}$) and standard deviation (SD) were extracted where appropriate. Where studies reported standard error (SE), this was converted to SD by multiplying by the square root of the sample size. Where these data were not available, authors were contacted to request further information. Where authors provided data with missing pre-to-post data for individual participants, these participants were excluded.

4.3.3 Data synthesis and statistical analysis

All statistical analysis was carried out in R [631], version 4.0.2. Effect sizes for group x time interactions were determined using standardised mean difference [632], and small sample bias accounted for by calculating Hedges' g [633]. These values were computed for each study design using the R package 'esc' [634], using the formulae discussed in Lipsey & Wilson [635]. Within-group effect sizes were only calculated where necessary, to avoid regression to the mean biases. The results are presented as a narrative review, summarising findings, implications and risk of bias.

4.3.4 Risk of bias and quality assessment

Several tools were employed to assess study quality based on its design. Appraisals were carried out independently by two reviewers. Any disagreements were settled by discussion and, if necessary, the opinion of a third party.

For randomised-controlled trials (RCTs), version 2 of the Cochrane Risk of Bias Tool for Randomized Studies (RoB 2) [636] was used. Assessing bias across five domains: the randomisation process, deviations from the intended intervention, missing outcome data, measurement of the outcomes, and selection of the reported result.

Non-randomised trials were assessed using the Risk of Bias in Non-randomised Studies of Interventions tool (ROBINS-I) [637]. ROBINS-I assesses risk of bias across seven domains: confounding, selection of participants into the study, classification of interventions, deviations from intended interventions, missing data, measurement of outcomes, and selection of the reported result.

As the ROBINS-I is not appropriate for appraising single-armed studies, the Evidence Project Risk of Bias tool was used in these cases [638]. This assesses bias data, random assignment to intervention, random selection of participants, attrition rate, homogeneity of sociodemographic data, and homogeneity of baseline values of outcome measures. A template was created for use of this tool in this study (see appendix J).

Single-case experimental design studies were appraised with the Risk of Bias in N-of-1 Trials (RoBiNT) scale [639]. This assesses the internal and external validity of a study and gives a numerical score out of 14 and 16, respectively.

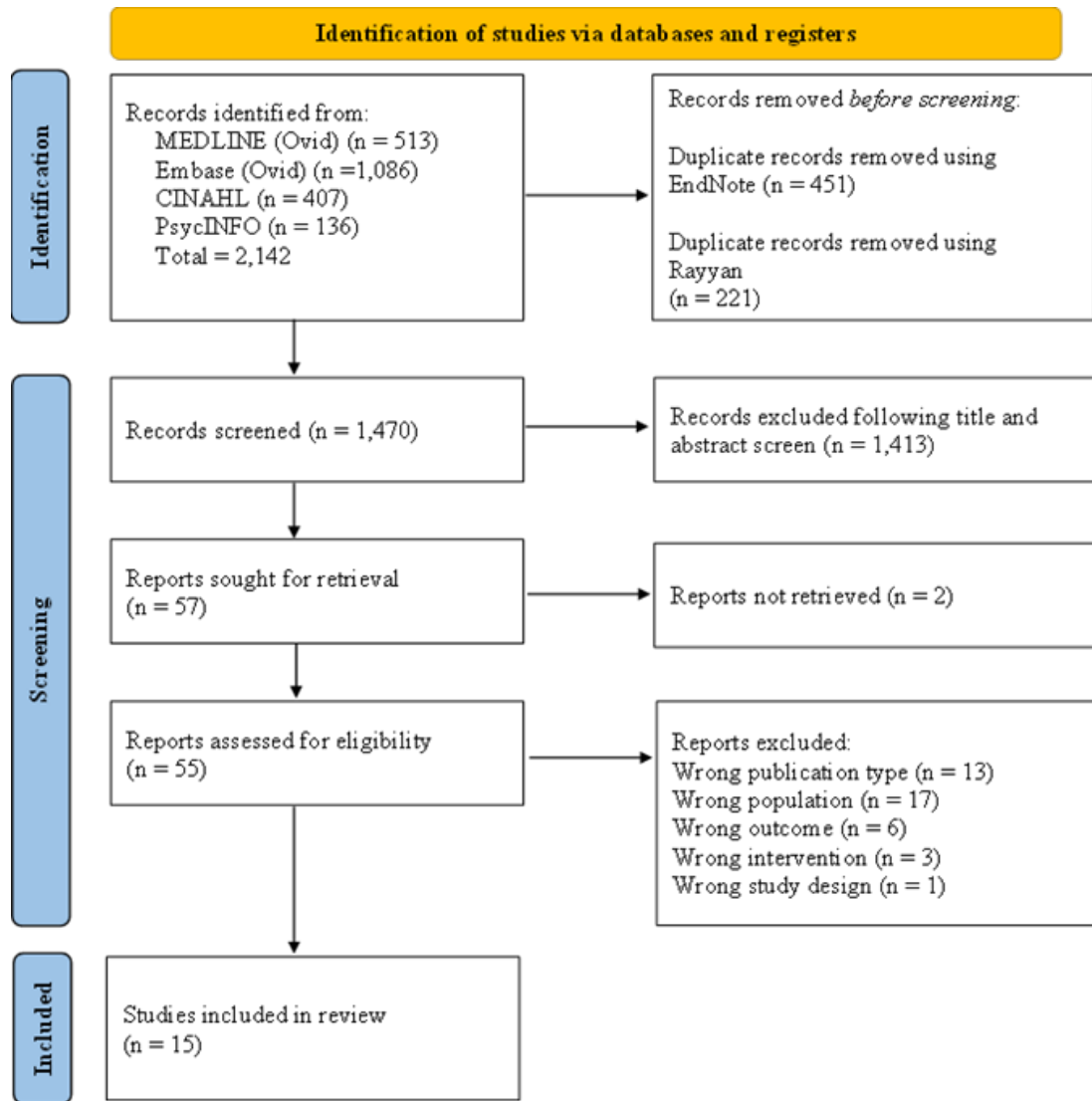


Figure 4.1: Preferred reporting items for systematic reviews and meta-analysis (PRISMA) flowchart

4.4 Results

4.4.1 PRISMA flowchart

Systematic database searches returned 2,142 articles. Following title, abstract and full screens, 15 studies were included in the present review. Details of those excluded can be seen in Figure 4.1. Data requests were submitted for two studies, of which only one responded [640].

4.4.2 Study characteristics

Table 4.1 provides details of all included studies. All were conducted in the USA, and publication dates spanned 36 years (1983–2019).

4.4.2.1 Population

The review summarised the data of 498 older adults, all from community samples, and recruited via voluntary response sampling. All studies recruited older adults from the general population, with the exception of Gebara et al. [640], who recruited military veterans with treatment resistant depression. The overall mean age, weighted for sample size, was 69.45 years (range: 65–77.2 years) and average sample size was 35.57. For most studies, insomnia classification was consistent with the most recent DSM or ICSD edition at time of publication. In some cases, author-defined definitions of insomnia were used (see Table 4.1).

4.4.2.2 Study design

The entire sample consisted of nine RCTs, one pilot RCT, two non-RCTs, two single case experimental designs (SCEDs) and one single-armed trial (see Table 4.1).

4.4.2.3 Intervention

Four studies [640–643] investigated Brief Behavioural Treatment for Insomnia (BBTI) [618, 619]. Tyagi et al. [643] was a secondary subgroup analysis of Buysse et al. [641], exploring nocturia’s impact on treatment outcome. A further four studies [644–647] were multicomponent studies studying variations of Sleep Restriction, Stimulus Control, Sleep Hygiene, and psychoeducation. Contradicting recent works by Chen et al. [623] and Kwon et al. [620], we considered the intervention described in Germain et al. [644] too distinct from published descriptions of BBTI [618, 619] to be considered as such, and it was thus classified as a multi-component intervention. Four studies looked at sleep restriction, but different methods of restricting time in bed were employed. Friedman et al. [648]

and Bliwise et al. [649] used conventional sleep restriction, where time in bed is abruptly curtailed to match total sleep time (TST) - plus a pre-determined time to allow for sleep onset - and slowly increased as sleep efficiency increases [113]. Whereas, Riedel & Lichstein [650] and Lichstein et al. [651] employed a protocol more akin to sleep compression [142], where time in bed is reduced incrementally. As these variations both target the same psychological mechanisms contributing to insomnia, and are broadly similar, we deemed it appropriate that they be combined for the narrative analysis. The remaining three studies [652–654] investigated stimulus control.

Three studies were delivered in groups [647, 648, 654]. The mean number of sessions was 4.53 (SD = 1.12), ranging from 2 to 6, group sizes ranged from 4 to 12. Generally, Stimulus Control and Sleep Restriction studies were published earlier than multicomponent studies.

4.4.2.4 Comparison

Comparison groups were employed in 80% of included studies, with three studies [645, 647, 650] having none. Details of individual comparisons can be seen in Table 4.1.

Table 4.1: Study characteristics¹

Study	Design	Control	Sample <i>N</i>	Mean Age (SD)	Female (%)	Treatment	Sessions	Delivery	Outcome Measures	Insomnia Definition
Puder et al., 1983	RCT	Delayed Treatment	16	67.06 (SD Not reported)	81.25	Stimulus Control	4	Group	SOL	SOL > 30 mins at least 1x in a week.
Morin & Azrin, 1988	RCT	Imagery Training, waitlist	27	67.4 (5.6)	62.96	Stimulus Control	6	Group	SOL, WASO, TST	WASO > 30 min/night, at least 3 nights per week, for ≥6 months, impacting daytime functioning.
Friedman et al., 1991	NRT	Relaxation	22	70.1 (10.1)	73.30	Sleep Restriction	4	Individual	SOL, WASO, TST, SE	Sleep efficiency <80% over two-week baseline.
Bliwise et al., 1995	NRT	Relaxation	32	68.7 (9.7)	68.75	Sleep Restriction	5	Individual	SOL, TST	Self-reported insomnia.
McCurry et al., 1996	SCED	No control	4	67.25 (6.08)	75	Multi-component	6	Group	PSQI, CES-D, HRSD, SOL, WASO, TST, SE	Self-reported sleep problems ≤3 nights per week.
Lichstein et al., 2000	RCT	No treatment	44	68.53 (6.55)	47.73	Stimulus Control	4	Individual	IIS, SOL, WASO, SE, TST, SQ, GDS, STAI-Y	ICSD-I
Reidel & Lichstein, 2001	Single Arm	No control	20	67.96 (7.07)	72.73	Sleep Compression	6	Individual	SOL, WASO, SE, TST, SQ, ESS	DSM-4
Lichstein et al., 2001	RCT	Relaxation, placebo	74	68.03 (7.04)	71.62	Sleep Compression	6	Individual	IIS, SOL, WASO, SE, TST, SQ, ESS, FSS	ICSD
Germain et al., 2006	RCT	Info only	35	70.2 (6.4)	71.43	Multi-component	2	Individual	SOL, WASO, SE, TST, PSQI, HARS, HRSD	DSM-4

Study	Design	Control	Sample, <i>N</i>	Mean Age (SD)	Female (%)	Treatment	Sessions	Delivery	Outcome Measures	Insomnia Definition
McCrae et al., 2006	SCED	No control	4	72.75 (4.86)	100	Multi-component	4	Individual	SOL, WASO, TST, SE, SQ, BDI-ii, GDS, STAI-Y	ICSD & DSM-4
McCrae et al., 2007	RCT	Sleep Hygiene	20	77.2 (8.0)	65	Multi-component	4	Individual	SOL, WASO, TST, SE	ICSD & DSM-4
Buyse et al., 2011	RCT	Info only	79	71.64 (7.24)	68.40	BBTI	4	Individual	SOL, WASO, TST, SE, SQ, PSQI, HARS, HRSD, ESS	ICSD & DSM-4
Tyagi et al., 2014	RCT	Info only	79	71.64 (7.24)	68.40	BBTI	4	Individual	SOL, WASO, TST, SE, SQ, PSQI, HRSD	ICSD & DSM-4
McCrae et al., 2018	RCT	Self-monitoring	62	69.45 (7.71)	67.74	BBTI	4	Individual	SOL, WASO, TST, SE, SQ, BDI-ii, GDS, STAI-Y	DSM-5
Gebara et al., 2019	RCT	Delayed Treatment	10	65 (4.01)	27.27	BBTI	4	Individual	ISI, SOL, WASO, SE, PHQ-9	DSM-5

¹BBTI, Brief Behavioural Treatment for Insomnia; BDI-ii, Beck Depression Inventory; CES-D, Centre for Epidemiologic Studies Depression Scale; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; DSM-V, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; ESS, Epworth sleepiness scale; FSS, Fatigue Severity Scale; GDS, Geriatric Depression Scale; HARS, Hamilton Anxiety Rating Scale; HDRS, Hamilton Depression Rating Scale; ICSD, International Classification of Sleep Disorders; IIS, Insomnia Impact Scale; NRT, Non-randomised trial; PSQI, Pittsburgh Sleep Quality Index; RCT, randomised-controlled trials; SCED, single-case experimental designs; SE, sleep efficiency; SOL, sleep-onset latency SQ, sleep quality; STAI-Y, State-Trait Anxiety Index; TST, total sleep time; WASO, wake after sleep onset.

4.4.2.5 Outcomes

Generic details of study-specific outcomes can be seen in Table 4.1. Sleep diaries were used in all studies. However, sleep-onset latency (SOL) was the only outcome to be reported in every trial. Actigraphy was used in three studies [641–643], and polysomnography also in three [641, 643, 651]. No treatment-related adverse outcomes were reported in any included studies.

4.4.3 Risk of bias

Risk of bias was assessed according to study design, using the appropriate tool for each study, as detailed in § 4.3.4.

4.4.3.1 Randomised controlled trials

Excluding Gebara et al. [640], the quality of RCTs generally improved as the publication date became more recent, likely down to changes in accepted standard across time. Only McCrae et al. [642] achieved a low risk of bias overall. Poorer scores were mostly due to participants or researchers not being blinded to group allocation. Often the design made this inevitable; however, measures were mostly self-report, thus limiting researcher bias. A traffic-light plot of risk of bias in RCTs can be seen in Figure 4.2.

		Risk of bias domains					
		D1	D2	D3	D4	D5	Overall
Study	Puder et al., 1983	-	X	X	-	-	X
	Morin & Azrin, 1988	X	+	+	+	X	X
	Lichstein, Wilson, & Johnson, 2000	-	+	+	+	X	X
	Lichstein et al., 2001	-	+	+	+	-	-
	Germain et al., 2006	+	+	+	+	-	-
	McCrae et al., 2007	-	-	+	+	+	-
	Buyse et al., 2011	+	+	-	+	+	-
	Tyagi et al., 2014	-	+	+	+	+	-
	McCrae et al., 2018	+	+	+	+	+	+
	Gebara et al., 2019	-	+	+	+	X	X

Domains:
D1: Bias arising from the randomization process.
D2: Bias due to deviations from intended intervention.
D3: Bias due to missing outcome data.
D4: Bias in measurement of the outcome.
D5: Bias in selection of the reported result.

Judgement
X High
- Some concerns
+ Low

Figure 4.2: Risk of bias in randomised controlled trials

4.4.3.2 Non-randomised controlled trials

Domain specific risk of bias for non-RCTs can be seen in Figure 4.3. Missing data, due to attrition and exclusion of participants on the grounds of missing data, resulted in a serious risk of bias in Friedman et al. [648]. All other domains for non-RCTs returned a low-moderate risk of bias.

		Risk of bias domains							
		D1	D2	D3	D4	D5	D6	D7	Overall
Study	Friedman et al, 1991								
	Bliwise et al, 1995								

Domains:

D1: Bias due to confounding.

D2: Bias due to selection of participants.

D3: Bias in classification of interventions.

D4: Bias due to deviations from intended interventions.

D5: Bias due to missing data.

D6: Bias in measurement of outcomes.

D7: Bias in selection of the reported result.

Judgement

 Serious

 Moderate

 Low

Figure 4.3: Risk of bias in non-randomised controlled trials

4.4.3.3 Single-armed trials

The study by Riedel & Lichstein was the only single-armed trial included in this review. This design undoubtedly introduces the greatest risk of bias overall. Nonetheless, in the interest of rigour, a simple appraisal categorising the presence or absence of specific criteria [638] was conducted. Many domains were not applicable due to study design, specifically those relating to comparisons of or assignment to groups. Riedel & Lichstein et al. [650] was a cohort analysis, with pre/post intervention data, and a follow-up rate >80%, suggesting a low risk of bias. However, the study had no comparison group and did not randomly select participants, suggesting a high risk of bias. An overview can be seen in Table 4.2.

Table 4.2: Risk of bias in single-armed studies²

Study	Domain	Response
Reidel & Lichstein, 2001	Cohort	Yes
	Control or comparison group	No
	Pre/post intervention data	Yes
	Random assignment of participants to the intervention	NA
	Random selection of participants for assessment	No
	Follow up rate of 80% or more	Yes
	Comparison groups equivalent on socio-demographics	NA
	Comparison groups equivalent at baseline on outcome measures	NA

²Note: A response of ‘yes’ suggests a low risk of bias; a response of ‘NA’ means this domain is not applicable to this study; a response of ‘No’ suggests a high risk of bias.

4.4.3.4 SCED trials

A high risk of bias was identified in both SCED studies, particularly pertaining to internal validity – specifically, the lack of statistical analysis, and instead presenting only summary data, and no randomisation in sequence of onset of phases between participants. Additionally, although study phases were adequate in length to provide detailed data with five or more data points, only aggregate data were reported (for a summary, see 4.3).

Table 4.3: Risk of bias in included SCED studies³

Internal Validity								External Validity								Internal Validity	External Validity	Total
Study	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15			
McCurry et al., 1996	1	0	0	0	1	0	0	1	0	1	2	1	0	0	0	2	5	7
McCrae et al., 2006	1	0	0	0	1	0	0	2	1	2	2	1	0	0	1	2	9	11

4.4.4 Synthesis of results

This review primarily aimed to explore the effectiveness of behavioural interventions to improve insomnia in older adults, and secondarily to investigate their impact on mood and daily functioning. Data were synthesised with studies grouped by intervention. Estimates of effect size and confidence intervals are presented in Tables 4.4 - 4.7, excluding for SCED studies – their design does not allow for the simple computation of effect sizes, and they are discussed separately. Initially it was intended that data of similar studies be pooled for meta-analysis; however, the heterogeneity of designs and outcomes prevented this. Studies varied across design (thus also effect size calculations), which outcomes were measured and how, whether participants were permitted to continue taking hypnotic medication, whether participants could have insomnia comorbid with another psychiatric or somatic condition, delivery methods, number of sessions, frequency of sessions, how insomnia was defined or whether difficulties were specifically with sleep onset or maintenance, and comparison groups. In some instances, such as in stimulus control, only studies with a high risk of bias could be pooled and, as this could

³Note: A lower value represents a greater risk of bias.

Item 1: Design with control; **Item 2:** Randomisation; **Item 3:** Sampling of behaviour; **Item 4:** Blinding of people involved in intervention; **Item 5:** Blinding of assessor(s); **Item 6:** Inter-rater agreement; **Item 7:** Treatment adherence; **Item 8:** Baseline characteristics; **Item 9:** Setting; **Item 10:** Dependent variable (target behaviour); **Item 11:** Independent variable (therapy/intervention); **Item 12:** Raw data record; **Item 13:** Data analysis; **Item 14:** Replication; **Item 15:** Generalisation.

lead to misleading results, it was deemed inappropriate by the researchers. Two studies appeared similar enough to be meta-analysed, but a meta-analysis of so few studies would only be appropriate if using a fixed-effects model. Due to variability between the studies, and the fact that we would intend that the findings be generalised beyond just these two studies, a fixed-effects model would not be appropriate to generate meaningful inferences in this case.

4.4.4.1 Stimulus control studies

Table 4.4: Effect size estimates for stimulus control studies.

Study	Experimental <i>n</i>	Comparison <i>n</i>	Outcome measure	Hedge's <i>g</i>	95% CI	Follow-up Hedge's <i>g</i>	Follow-up 95% CI	Design
Puder et al., 1983	9	7	SOL	0.92	[-1.16, 3.00]			RCT
Morin & Azrin, 1988 (Treatment vs imagery training)	9	8	SOL	0.15	[-1.76, 2.07]	0.49	[-1.56, 2.53]	RCT
			WASO	0.49	[-1.43, 2.41]	0.83	[-1.11, 2.77]	
			TST	-0.77	[-2.71, 1.17]	-0.80	[-2.74, 1.15]	
Morin & Azrin, 1988 (Treatment vs control)	9	10	SOL	-0.16	[-2.01, 1.69]			RCT
			WASO	0.98	[-0.87, 2.83]			
			TST	-0.96	[-2.80, 0.87]			
Lichstein et al., 2000	23	21	IIS	0.23	[-0.96, 1.41]	0.36	[-0.83, 1.54]	RCT
			SOL	0.12	[-1.11, 1.35]	0.45	[-0.78, 1.68]	
			WASO	0.45	[-0.74, 1.64]	0.43	[-0.77, 1.62]	
			SE	-0.48	[-1.67, 0.71]	-0.58	[-1.78, 0.63]	
			TST	-0.14	[-1.33, 1.04]	-0.30	[-1.49, 0.89]	
			SQ	-0.60	[-1.79, 0.59]			
			GDS	0.12	[-1.06, 1.31]	0.00	[-1.19, 1.19]	
			STAI-Y	0.43	[-0.77, 1.63]	0.36	[-0.83, 1.55]	

Effect estimates for stimulus control studies can be seen in Table 4.4. Studies varied in how outcomes were measured. SOL was recorded via sleep diaries in Puder et al. [654], and Lichstein et al. [652]. In Morin & Azrin [653], SOL and WASO were measured objectively using a 'Switch-Activated Clock'. The participant applied pressure to the switch when retiring to bed, as the participant fell asleep the pressure on the switch was released.

Puder et al., [654] and Morin & Azrin [653] focused on sleep-onset insomnia and sleep maintenance insomnia, respectively. Lichstein et al. [652] focused on ICSD-I insomnia comorbid with another medical or psychiatric condition.

³Note: Negative value denotes an increase over time.

CI, confidence interval; GDS, geriatric depression scale; IIS, insomnia severity index; RCT, randomised-controlled trial; SE, sleep efficiency; SOL, sleep-onset latency; SQ, sleep quality; STAI-Y, state-trait-anxiety inventory; TST, total sleep time; WASO, wake after sleep onset.

Subjective sleep-related outcomes

Stimulus control is designed to reduce the anxiety one may feel around the bed/bedroom, and train the individual to associate the act of going to bed with sleep; thus, primarily targeting SOL [110]. All stimulus control studies demonstrated significant effects on SOL, which were maintained at follow-up. However, between-group differences and a large effect size (albeit with a wide confidence interval) were only reported in Puder et al. [654]. In the others, comparison groups demonstrated similar changes, and effect sizes were indicative of a small or no effect.

Morin & Azrin [653] and Lichstein et al. [652] also reported significant improvements in WASO, both of which improved at follow-up. In the former, stimulus control outperformed a waitlist control and imagery training. In the latter, stimulus control did not result in significantly different results to the waitlist control. TST also improved at post-treatment but did not improve at a 12-month follow-up, and no significant between-group differences existed.

Mood-related outcomes

Pre-to-post values for measures of anxiety and depression were only reported in Lichstein et al. [652]; however, Morin & Azrin [653] do state that stimulus control elicited no significant effect on either outcome, measured by the STAI and BDI, respectively. Lichstein et al. [652] report the same, although effect sizes suggest a moderate treatment effect on anxiety at post-treatment and follow-up.

4.4.4.2 Sleep restriction studies

Table 4.5: Effect size estimates for sleep restriction studies.

Study	Experimental <i>n</i>	Comparison <i>n</i>	Outcome measure	Hedge's <i>g</i>	95% CI	Follow-up Hedge's <i>g</i>	Follow-up 95% CI	Design
Freidman et al., 1991	10	12	SOL	0.28	[-1.46, 2.02]	0.27	[-1.57, 2.10]	NRT
			WASO	0.50	[-1.27, 2.27]	0.21	[-1.49, 1.92]	
			SE	-1.26	[-3.02, 0.51]	-0.98	[-2.72, 0.77]	
Bliwise et al., 1995	16	16	TST	0.12	[-1.57, 1.82]	-0.41	[-2.10, 1.28]	NRT
			SOL	0.22	[-1.25, 1.70]	0.19	[-1.37, 1.75]	
Reidel & Lichstein, 2001 * +	20		TST	-0.18	[-1.59, 1.24]	-0.56	[-1.96, 0.84]	Single Arm
			SOL	0.51	[-0.11, 1.13]			
			WASO	0.88	[0.25, 1.52]			
			SE	-0.71	[-1.34, -0.09]			
			TST	0.08	[-0.52, 0.69]			
			SQ	-0.97	[-1.61, -0.33]			
			ESS	0.07	[-0.53, 0.68]			
Lichstein et al., 2001 (Treatment vs control) +	24	23	IIS	-0.09	[-1.23, 1.06]	-0.41	[-1.56, 0.75]	RCT
			SOL	0.04	[-1.14, 1.22]	0.49	[-0.69, 1.66]	
			WASO	0.06	[-1.09, 1.21]	0.45	[-0.70, 1.60]	
			SE	0.17	[-0.99, 1.33]	-0.29	[-1.43, 0.86]	
			TST	0.85	[-0.32, 2.03]	0.07	[-1.09, 1.22]	
			SQ	-0.41	[-1.56, 0.74]	-0.78	[-1.94, 0.38]	
			PSG SOL			-0.17	[-1.35, 1.01]	
			PSG WASO			0.23	[-0.92, 1.38]	
			PSG TST			-0.52	[-1.67, 0.64]	
			PSG SE			-0.47	[-1.62, 0.69]	
			ESS	-0.09	[-1.23, 1.06]	0.05	[-1.09, 1.20]	
			FSS	0.25	[-0.89, 1.40]	-0.06	[-1.21, 1.08]	
Lichstein et al., 2001 (Treatment vs relaxation training) +	24	27	IIS	-0.35	[-1.45, 0.75]	-0.34	[-1.44, 0.77]	RCT
			SOL	0.03	[-1.10, 1.16]	0.24	[-0.89, 1.36]	
			WASO	0.01	[-1.10, 1.12]	0.37	[-0.74, 1.48]	
			SE	0.10	[-1.01, 1.20]	-0.20	[-1.30, 0.90]	
			TST	0.85	[-0.27, 1.97]	0.30	[-0.80, 1.41]	
			SQ	0.07	[-1.03, 1.17]	-0.31	[-1.41, 0.80]	
			PSG SOL			-0.37	[-1.55, 0.81]	
			PSG WASO			0.25	[-0.86, 1.36]	
			PSG TST			-0.33	[-1.44, 0.77]	
			PSG SE			-0.17	[-1.27, 0.94]	
			ESS	-0.02	[-1.12, 1.08]	-0.09	[-1.20, 1.02]	
			FSS	-0.39	[-1.49, 0.71]	-0.20	[-1.30, 0.90]	

Effect estimates for sleep restriction studies can be seen in Table 4.5. Riedel & Lichstein [650] was single-armed, and thus effect sizes were calculated as within-group, and any comparisons should be drawn with this in mind. Studies differed in implementation of sleep restriction. Firstly, Riedel & Lichstein [650] and Lichstein et al. [651] used sleep compression rather than sleep restriction, where time in bed was reduced to baseline TST gradually, rather than immediately. Studies also varied in what sleep efficiency percentage should be regarded as the threshold at which time in bed is increased. Sleep compression studies opted for 90%, and sleep restriction studies 85%. Additionally, Friedman et al. [648] opted not to decrease time in bed if the sleep efficiency threshold was not met between sessions.

³Note: Negative value denotes an increase over time. Effect sizes for studies marked with '*' were calculated as within group; Studies marked with '+' used sleep compression as opposed to sleep restriction. Abbreviations: CI, confidence interval; FSS, fatigue severity scale; ESS, Epworth sleepiness scale; IIS, insomnia impact scale; NRT, non-randomised trial; PSG, polysomnography; RCT, randomised-controlled trial; SE, sleep efficiency; SOL, sleep-onset latency; SQ, sleep quality; TST, total sleep time; WASO, wake after sleep onset.

Subjective sleep-related outcomes

Sleep restriction studies [648, 649] demonstrated statistically significant reductions in SOL, which decreased further after 3 months. Between-group differences were not significant, and when compared against relaxation training comparisons, effect sizes were small. No significant effects were seen on TST at post-treatment. However, significant improvements were seen after 3 months, with sleep restriction outperforming relaxation in both studies.

As Riedel & Lichstein [650] was single-armed, direct comparisons of sleep compression studies could not be drawn; nevertheless, this study did report significant improvements in SOL, with a moderate effect size. However, in Lichstein et al. [651], where treatment was compared with relaxation and placebo groups, significant improvements were seen from baseline to post-treatment in all groups. Sleep compression did maintain improvements better after 12 months, demonstrating a moderate effect against the placebo, and a small effect against relaxation training. Sleep compression did not improve TST significantly in either study.

Objective sleep-related outcomes

Lichstein et al. [651] was the only Sleep Restriction/Compression study to report objective results, that being PSG data. This was recorded over 2 nights at baseline and follow-up. Authors elected to use data only from night two of PSG recording, due to results indicating that sleep was significantly worse on night 1. In comparison, sleep diary data were recorded over 14 nights. PSG data showed no significant change for any variable between baseline and a 12-month follow-up.

4.4.4.3 Multicomponent studies

Table 4.6: Effect size estimates for multicomponent studies.⁴

Study	Experimental <i>n</i>	Comparison <i>n</i>	Outcome measure	Hedge's <i>g</i>	95% CI	Design
McCrae et al., 2007	11	9	SOL	1.22	[-0.71, 3.14]	RCT
			WASO	0.98	[-0.85, 2.81]	
			TST	0.20	[-1.59, 1.99]	
			SE	-1.46	[-3.32, 0.39]	
Germain et al., 2006	17	18	SOL	0.83	[-0.62, 2.29]	RCT
			WASO	0.64	[-0.71, 1.98]	
			SE	-0.68	[-2.02, 0.66]	
			TST	0.18	[-1.14, 1.51]	
			PSQI	1.23	[-0.14, 2.60]	
			HARS	0.49	[-0.88, 1.85]	
			HRSD	0.86	[-0.50, 2.22]	

Effect size estimates for multicomponent studies can be found in 4.6, and specifically BBTI studies can be seen in Table 4.7. McCurry et al. [647] focused on dementia caregivers, McCrae et al. [645] on non-specific caregivers at different stages of care, McCrae et al. [646] solely on rural older adults, and Gebara et al. [640] on veterans with treatment-resistant depression. Upon receipt of the data for Gebara et al., it was established that authors had amalgamated the immediate and delayed treatment groups together in their final analysis, due to missing data for several participants in the delayed treatment group; this was confirmed by the authors. The decision was made to take the same approach in this study to avoid inflated effect sizes, and effect sizes for this paper were calculated as within-group. Other multicomponent studies consisted of older adults from the general population.

⁴*Note:* Negative value denotes an increase over time; Multicomponent Single Case Experimental Design Studies are not included in this table. .

Abbreviations: CI, confidence interval; HARS, Hamilton anxiety rating scale; HDRS, Hamilton depression rating scale; PSQI, Pittsburgh sleep quality index; RCT, randomised-controlled trial; SE, sleep efficiency; SOL, sleep-onset latency; SQ, sleep quality; TST, total sleep time; WASO, wake after sleep onset.

Table 4.7: Effect size estimates for BBTI studies.⁵

Study	Experimental <i>n</i>	Comparison <i>n</i>	Outcome measure	Hedge's <i>g</i>	95% CI	Follow-up Hedge's <i>g</i>	Follow-up 95% CI	Design
McCrae et al., 2018	32	30	SOL	0.48	[-0.53, 1.49]	0.41	[-0.60, 1.43]	RCT
			WASO	0.63	[-0.38, 1.63]	0.79	[-0.22, 1.80]	
			TST	0.15	[-0.85, 1.14]	-0.23	[-1.23, 0.77]	
			SE	-0.61	[-1.61, 0.39]	-0.69	[-1.69, 0.32]	
			SQ	-0.49	[-1.50, 0.51]	-0.69	[-1.70, 0.31]	
			Actigraphy SOL	0.40	[-0.61, 1.41]	0.01	[-0.98, 1.01]	
			Actigraphy WASO	0.58	[-0.42, 1.58]	0.43	[-0.57, 1.43]	
			Actigraphy TST	0.25	[-0.75, 1.25]	0.10	[-0.90, 1.10]	
			Actigraphy SE	-0.36	[-1.35, 0.64]	-0.29	[-1.29, 0.71]	
			BDI-II	-0.08	[-1.08, 0.92]	0.01	[-0.99, 1.00]	
			GDS	-0.13	[-1.18, 0.93]	-0.10	[-1.16, 0.96]	
			STAI-Y	-0.17	[-1.17, 0.83]	0.01	[-0.99, 1.01]	
Buysse et al., 2011 *	39	40	SOL	0.89	[-0.01, 1.80]			RCT
			WASO	0.62	[-0.27, 1.51]			
			TST	0.10	[-0.79, 0.98]			
			SE	-0.78	[-1.67, 0.11]			
			SQ	-0.68	[-1.57, 0.21]			
			Actigraphy SOL	0.55	[-0.38, 1.47]			
			Actigraphy WASO	0.34	[-0.55, 1.23]			
			Actigraphy TST	0.58	[-0.31, 1.47]			
			Actigraphy SE	-0.51	[-1.40, 0.37]			
			PSG SOL	-0.23	[-1.11, 0.65]			
			PSG WASO	0.35	[-0.53, 1.24]			
			PSG TST	0.13	[-0.75, 1.01]			
			PSG SE	-0.18	[-1.06, 0.70]			
			PSQI	1.00	[0.10, 1.89]			
			HARS	0.40	[-0.49, 1.29]			
			HRSD	0.69	[-0.20, 1.58]			
			ESS	-0.15	[-1.37, 1.07]			
Tyagi et al., 2014 (without nocturia) *	25	24	SOL	0.98	[-0.24, 2.20]			RCT
			WASO	0.47	[-0.66, 1.60]			
			TST	-0.04	[-1.16, 1.08]			
			SE	-0.91	[-2.07, 0.25]			
			SQ	-0.82	[-1.96, 0.32]			
			Actigraphy SOL	0.73	[-0.45, 1.90]			
			Actigraphy WASO	0.75	[-0.40, 1.89]			
			Actigraphy TST	0.81	[-0.33, 1.95]			
			Actigraphy SE	-0.65	[-2.49, 1.20]			
			PSG SOL	0.00	[-1.13, 1.13]			
			PSG WASO	-0.20	[-1.32, 0.92]			

Table 4.7: (Continued)

Study	Experimental <i>n</i>	Comparison <i>n</i>	Outcome measure	Hedge's <i>g</i>	95% CI	Follow-up Hedge's <i>g</i>	Follow-up 95% CI	Design
Tyagi et al., 2014 (with nocturia) *	14	16	PSG	-0.07	[-1.19, 1.06]			RCT
			TST					
			PSG	0.11	[-1.02, 1.23]			
			SE					
			PSQI	1.14	[0.00, 2.29]			
			HRSD	0.55	[-0.58, 1.68]			
			SOL	0.81	[-0.72, 2.34]			
			WASO	0.83	[-0.63, 2.28]			
			TST	0.32	[-1.12, 1.76]			
			SE	-0.73	[-2.19, 0.74]			
			SQ	-0.34	[-2.02, 1.34]			
			Actigraphy					
			SOL	0.57	[-0.97, 2.12]			
			Actigraphy					
			WASO	0.22	[-1.24, 1.68]			
			Actigraphy					
			TST	0.65	[-0.84, 2.14]			
			Actigraphy					
			SE	-0.71	[-2.17, 0.75]			
Gebara et al., 2019	5	5	PSG	-0.29	[-1.81, 1.23]			RCT
			SOL					
			PSG	1.20	[-0.28, 2.68]			
			WASO					
			PSG	0.60	[-0.87, 2.07]			
			TST					
			PSG	-0.43	[-1.87, 1.02]			
			SE					
			PSQI	0.74	[-0.71, 2.19]			
			HRSD	0.49	[-1.02, 2.00]			
			ISI	0.96	[-0.36, 2.28]			
			SOL	0.14	[-1.10, 1.38]			
			WASO	0.19	[-1.06, 1.43]			
			SE	-0.28	[-1.53, 0.97]			
			PHQ-9 - sleep	0.57	[-0.70, 1.84]			
			PHQ-9 + sleep	0.49	[-0.77, 1.75]			

Subjective sleep-related outcomes

Only SOL, WASO and sleep efficiency were reported in all multicomponent studies. Treatment brought about significant improvements, and outperformed comparisons, on SOL and WASO in all multicomponent studies. Effect sizes in RCTs ranged from moderate to large (SOL median Hedge's $g = 0.86$; WASO median Hedge's $g = 0.64$). Within-group effect sizes in Gebara et al. [640] were

⁵ *Note:* Negative values denote an increase over time; Studies marked with '*' used the same dataset.
Abbreviations: BDI-ii, Beck Depression Inventory; CI, confidence interval; ESS, Epworth sleepiness scale; GDS, Geriatric Depression Scale; HARS, Hamilton anxiety rating scale; HDRS, Hamilton depression rating scale; ISI, insomnia severity index; PSG, polysomnography; PHQ-9, patient health questionnaire; PSQI, Pittsburgh sleep quality Index; RCT, randomised-controlled trial; SE, sleep efficiency; SOL, sleep-onset latency; SQ, sleep quality; TST, total sleep time; WASO, wake after sleep onset; STAI-Y, State-Trait-Anxiety Inventory.

small for both outcomes (Hedge's $g = 0.14$ and 0.19 , respectively). Data from 3 months post-treatment [642] demonstrated that improvements in SOL and WASO were well maintained. SCED studies [645, 647] reported similar findings, with all participants demonstrating improvements in SOL and WASO at post-treatment. Sleep quality was reported in 5/8 multicomponent studies, and significant effects were seen in all. Sleep quality was assessed in various ways. The Pittsburgh Sleep Quality Index (PSQI) [411] was used in three studies: Buysse et al. [641], Germain et al. [644], and Tyagi et al. [643]. Germain et al. [644] used a 0-6 self-report scale, McCrae et al. [642] used a 0-5 self-report scale, and Tyagi et al. [643] also reported a 0-100 self-report scale. Interestingly, results from this were only significant in the group without nocturia.

Results from Tyagi et al. [643] suggest that the magnitude of the effect of BBTI on SOL was smaller in the group with nocturia, but still statistically significant. Contrastingly, the size of the effect on WASO was larger in those with nocturia than without. It should be noted that WASO was larger at baseline in people with nocturia (67.02 [SD = 34.17] min versus 44.32 [SD = 30.36] min) than those without.

Multicomponent therapy did not result in any significant effects of time, group or group $\times$ time on TST in any RCTs. Results were inconsistent in the SCED trials. Three (75%) participants in McCurry et al. [647] reported increases in TST, but only one participant (25%) in McCrae et al. [645] did so, whilst two reported decreases.

Objective sleep-related outcomes

BBTI brought about statistically significant improvements in actigraphic SOL and WASO in Buysse et al. [641], with moderate effect sizes. However, Tyagi et al. [643] demonstrated that these improvements were only seen in those without nocturia. McCrae et al. [642] was the only other multicomponent study to report actigraphic outcomes, and demonstrated no significant changes in any sleep diary domain.

In Tyagi et al. [643], participants with nocturia who received BBTI experienced a significant decrease in PSG WASO, where those with nocturia and controls experienced an increase. Note that BBTI participants with nocturia had elevated WASO at baseline (116.99 min, SD = 47.77) compared with those without nocturia (76.15 min, SD = 38.11). No other significant effects were seen in PSG outcomes.

Apart from McCrae et al. [642], all multicomponent studies that recorded depression, including both SCED studies, demonstrated significant improvements in depression outcomes across varying self-report and interview-based measures. In Tyagi et al. [643] statistically significant improvements in depression were only seen in participants who received BBTI and had nocturia. Measures of anxiety were reported in four multicomponent trials: Buysse et al. [641], Germain et al. [644], McCrae et al. [642, 645]. No statistically significant change was found, nor were the results in SCED studies suggestive of an effect.

4.5 Discussion

This systematic review is the first to investigate explicitly behavioural interventions, without any cognitive component, for insomnia in older adults. Specifically, this review aimed to examine the literature detailing the efficacy of sleep restriction and stimulus control therapies, both isolated and combined, at treating insomnia in older adults. The secondary aim was to explore the effect of these treatments on outcomes of mood and daytime functioning when these were reported. Our findings support the efficacy of these interventions, in alleviating the symptoms of insomnia in older adults. Albeit varying in the domains affected, and in the magnitude of effect, all interventions explored in the present review conferred some benefit to the participants.

4.5.1 Sleep-related outcomes

Given their importance in the nosology of insomnia, SOL and WASO are of particular interest. Improvements in subjective measures of these domains were demonstrated in all interventions. In isolation, stimulus control and sleep restriction both demonstrated an effect of time, which were maintained at 3-month follow-ups, but generally did not achieve significant between-group differences.

Where they did [650, 654], studies were older and contained a higher risk of bias. Contrastingly, multicomponent interventions demonstrated significant group $\times$ time effects on subjective SOL and WASO (median Hedge's $g = 0.62$), which were maintained 3 months after treatment ended; the one exception being Gebara et al.[640]. This effect on SOL and WASO was demonstrated to be the case even after just two sessions of a multicomponent intervention [644](Hedge's $g = 0.83$ and 0.64 , respectively). Effect sizes in studies using solely stimulus control or sleep restriction were generally smaller and notably more heterogenous. Inconsistency across all studies in what measures were used meant that TST was the only other sleep-related outcome for which data were sufficient to draw inferences. In stimulus control studies, TST increased, but group differences did not exist, nor did improvements continue at a 12-month follow-up. All studies with a sleep restriction component, isolated or combined with stimulus control, demonstrated small or no effects on TST immediately post-treatment, likely as a result simply of the nature of the therapy, initially reducing time in bed, and by extension TST. Where follow-ups are reported, TST does appear to lengthen, and the size of the effect appears to increase over time after treatment has ended.

Where they are available, objective measures tell a slightly different story than subjective ones. In Tyagi et al. [643], BBTI demonstrated a large statistically significant effect on PSG WASO in participants with nocturia. No other differential effects were witnessed in any other PSG-recorded outcomes from any study. Actigraphic data were available only from BBTI studies [641–643], and were generally more consistent with sleep diary data. Treatment brought about improvements in actigraphic SOL and WASO in all cases; however, these improvements were not statistically significant in McCrae et al. [642], despite outperforming the control group and demonstrating a moderate effect size in both outcomes. It is possible that moderate effect sizes may not be sufficient to reach statistical significance in groups of this size (n per group = 31). Note that the confidence intervals for these effect sizes are wide, suggesting a low degree of certainty to the true estimate. Results indicated no significant effect of treatment on actigraphic TST at post-treatment, except in BBTI participants without nocturia. However, like sleep diary data, actigraphic TST appears to begin to increase at 3-month follow-ups.

4.5.2 Mood and daytime functioning related outcomes

No measures of mood were reported in any isolated sleep restriction/compression study, and no effect was seen on any measure of daytime functioning. Stimulus control studies reported outcomes on depression, anxiety and measures of insomnia impact, but elicited no significant effect on any. When combined, stimulus control and sleep restriction appear effective at alleviating depression, but not anxiety. Despite a variety of different measures for both outcomes being employed across all studies, a clear pattern can be seen. Seven multicomponent studies provided data on measures of depression. Of these, six demonstrated significant improvements, with moderate effect sizes. The exception was McCrae et al. [642]; however, average BDI-II [655] and GDS [656] scores at baseline were in the normal range for both groups, thus treatment effect may not have been witnessed simply due to the absence of elevated levels of depression in the sample. Four multicomponent studies provided data on anxiety, of which none demonstrated any significant improvements.

4.5.3 Interpretation of results

The efficacy of CBT-I is well established, and in myriad different populations [582]. Consequently, it is recommended as the first line of treatment for insomnia by the American College of Physicians [161, 605], the American Academy of Sleep Medicine [98, 108], the European Sleep Research Society [100], the World Sleep Society [99], and the British Association for Psychopharmacology [657]. As a result, most contemporary research has focused on CBT-I, which though effective, can be both financially and temporally costly, and may not be best suited in some clinical populations. For example, two recent studies, one pilot RCT [407] and one case series [405] explored the utility of CBT-I following a stroke. Although both demonstrated improvements in sleep, mental health, quality of life and fatigue, improvements in insomnia and quality of life were not maintained at follow-up. Deficits in cognitive ability [492] and speech & language [304] are common following a stroke, and thus the cognitive demand of CBT-I and the six–eight sessions it is ordinarily delivered in may be too taxing. The renewed interest in unpicking the constituent components of CBT-I [25, 70], particularly without cognitive facets, may prove fruitful in improving treatment options in this domain. This review demonstrates that explicitly behavioural interventions can elicit moderate to large positive effects on outcomes critical to insomnia dis-

order. By extension, it has the potential to inform future research into developing interventions for insomnia following a stroke, or in similar populations, without the need for cognitive components. Moreover, by isolating the specific therapies that are advantageous in the treatment of insomnia, we can identify and evaluate the key mechanisms mediating clinical change and begin to explore when monotherapies such as stimulus control and sleep restriction could be more efficient alternatives [130, 131, 133].

In 2021, the American Academy of Sleep Medicine endeavoured to determine the ‘minimal characteristics’ of a CBT-I intervention, in the general population. The main conclusion was that CBT-I should be comprised of at least: sleep restriction therapy, stimulus control, and some cognitive component [98]. They noted that stimulus control and sleep restriction may be used as a single-component therapy on a conditional basis, but that these therapies may require adaptation or extra support in certain populations, such as older adults [98]. Hypnotic medications are recommended only for short-term use, in instances where CBT-I fails to work or is unavailable [70, 100], and non-pharmacological intervention is the preferred choice of most individuals [106, 107, 165], thus alternative behavioural options may increase treatment adherence. The results of the present review enhance our understanding of these options in older adults, and demonstrate that effective treatment can be delivered without cognitive therapy in as little as two sessions, with moderate to large effects [644].

The findings that treatment effects are generally largest when stimulus control and sleep restriction are combined, and that a multicomponent approach corresponds with improvements in mood related outcomes, are consistent with the existing literature in the general population [145, 148]. Altena et al. [145] suggest that, when combined, both treatments appear to help stabilise the inter-night variability in insomnia symptoms that is a hallmark of insomnia [134, 136], which may in turn contribute to reductions in the severity of depressive symptoms [135]. While the existence of a bi-directional relationship between sleep and psychiatric wellbeing is well-established [74, 470, 583], and CBT-I appears effective at preventing the onset of depression in older adults with insomnia [613], further investigation is required before inferences can be drawn about mechanisms underlying these phenomena.

The findings related to TST are consistent with existing CBT-I literature [140]. A diagnosis of insomnia is not contingent upon the duration of sleep, and thus TST is rarely a focus of treatment. Nonetheless, when long-term outcomes are explored, additional gains in SOL and WASO are infrequently reported; how-

ever, TST appears to increase somewhat linearly by about 30–40 min, 3–6 months after treatment [140]. In one study of 80 adults (>30 years old), after 6 weeks of CBT-I, only 45.4% reported a TST that exceeded that of baseline. By 24 months post-treatment, this had increased to 85.9%, without any additional clinical intervention [140]. Similar findings were observed in a synthesis of literature relating to BBTI in the general population [620]. Authors attribute the slower improvements in TST to initial restrictive mechanisms of sleep restriction therapy. The discrepancy between results of studies with and without a sleep restriction component in the current review support this hypothesis. Additionally, results from a recent randomised controlled trial [143] suggest that sleep compression elicits similar efficacy to sleep restriction (albeit at a slower rate), without the initial decrease in total sleep time. Heterogeneity of baseline characteristics may be an additional factor contributing to this phenomenon, however. One recent study demonstrates that greater severity of subjective sleep complaints at baseline correspond with greater increases in overall TST after CBT-I [144]. Whether or not participants benefit from increased TST remains unclear, however these results emphasise the importance of lengthy follow-ups to shed more light on the true effects of these interventions.

Similarly, discrepancies between subjective and objective measures, known as ‘paradoxical insomnia’ or ‘sleep state misperception’ are known phenomena in older adults [658, 659] and other populations [70]. PSG commonly results in less pronounced changes than self-report measures. For example, previous meta-analytic results reported average differences in PSG-recorded TST between people with insomnia and good sleepers of approximately 25 min, whereas subjectively recorded average differences are almost 120 min larger [660]. This remains to be a significant clinical and scientific challenge [70], although recent evidence suggests that this phenomenon does not influence treatment outcomes [661]. In all cases in this review, PSG data were collected over 2 nights and, in Lichstein et al. [651], the first night was ignored to account for ‘first-night-effect’ [662]. Of course, this provides less data points to average across than the 14 nights measured in sleep diaries and may be a potential explanation for the difference.

The contradictory results exhibited in Gebara et al. [640] are most likely a result of the study being a pilot, designed to assess feasibility, and not powered to detect differences between groups [663]. Pilot studies were included in this review to capture as much relevant literature as possible, but the limitations of their design should be considered, as well as the fact that effect sizes in Gebara et al. [640] were calculated as within-group.

4.5.4 Clinical implications and applications

These findings substantiate, add to the existing literature, and are of particular importance in guiding future investigations into treating insomnia in populations for whom cognitive therapy may not be appropriate or optimal. The evidence suggests that explicitly behavioural interventions, even brief ones, can be effective at alleviating the symptoms of insomnia, and consequently depression symptoms, in older adults, making a case for the dissemination of these interventions when CBT-I is unavailable or inappropriate. Moreover, Hughes et al.[185] emphasise that the heterogeneity of older adults as a population may necessitate that certain aspects of CBT-I may require adaptation if it is to be successfully disseminated widely. The findings of this study are facilitatory in deriving a deeper understanding of the utility of stimulus control and sleep restriction outside the context of CBT-I, providing an excellent point of departure for further exploration of for whom each component works most and least optimally. Rather surprisingly, all 15 included studies were conducted in the USA, concordant with findings in Kwon et al. [620] and Chen et al. [623]. This does limit the cross-cultural application of the findings and is perhaps partly explained by studies not published in English being excluded. However, considering insomnia prevalence and that the inclusion criteria were so broad, it was an unexpected finding, and does raise questions as to why more investigation of the behavioural components of CBT-I in older adults has not been conducted in other countries.

4.5.5 Strengths

Our review has several strengths. Firstly, the protocol for the review was pre-registered, promoting transparency in our procedures, reducing the potential for bias, and avoiding unintended duplication of research within the field. Additionally, the recently revived interest in unpicking the individual components of CBT-I to determine their individual utility [70], along with the current focus on improving our understanding of insomnia in older adults, as evidenced by the 300+ active studies listed on clinicaltrials.gov, highlights the timely nature of the present review, and its relevance to the current objectives of the field at large. Moreover, our broad inclusion criteria and comprehensive search strategy lessens the likelihood of relevant literature being missed out. Thus, we can be confident we have explored the entirety, or as close to as possible, of the existing evidence of the efficacy of behavioural interventions in older adults with insomnia.

4.5.6 Limitations

Some inevitable limitations to this review do exist. Firstly, only papers that were published in English were included. Meaning that any relevant papers published in other languages are therefore not addressed in this review. Moreover, although a great deal of planning and experimentation went into developing search terms for bibliographic database searches, it is of course possible that not all the terminology relevant to this research was used. Thus, any papers published by authors who have not used conventional terminology may have been missed. However, reference sections of included studies were searched, and detailed scoping searches were used to investigate the most appropriate terminology to use.

Two additional field-wide limitations also exist. Firstly, despite a relatively broad inclusion criteria, we were only able to identify 15 suitable studies. These studies differed widely with regards to insomnia classification, study design, delivery method, outcomes reported, comorbidities, nature of comparison and control groups, duration of treatment, number of therapeutic sessions, length of follow-up, and at times the interventions themselves. This phenomenon has been noted in previous meta-analyses exploring sleep restriction in general populations [131] and CBT-I [176, 664]. The field-wide lack of uniformity, particularly in selection of outcome measures, limits what inferences can be drawn. Specific domains where this issue is of particular concern are in the measurement of mood-related outcomes, and in sleep quality. Seven of the 15 studies did not measure sleep quality at all, those that did either used the PSQI [411] or a self-report rating scale – either 0–5, 0–6 or 0–100. The PSQI was also only adopted in multicomponent studies, making valid between-intervention comparisons for this outcome impossible. Moreover, variation in the design of studies included meant effect sizes were calculated differently for different studies, impairing the validity of comparisons between studies. The calculation of within-group effect sizes negates the benefit of having a control group and is sensitive to influence from pre-test variability [665]. Generally, this, along with concerns highlighted in risk of bias appraisals, identifies field-wide limitations, and the need for more high-quality RCTs. More specifically, the variation between studies meant that a meta-analysis was not possible as part of this review. Nonetheless, it did allow for an exploration offering new information on this topic, and the large amount of data extracted from the included studies allowed for the valid comparison of other outcomes.

Secondly, the sample sizes of many included studies were low. In general, the quality of studies tended to increase over time, as did sample size, and this is likely a result of improvements in our understanding of study design and statistical power in psychological research across time. Of course, given how few studies appear to have explored these interventions as monotherapies in older adults, expected effect sizes may not be available. In these circumstances, if we assume $d = 0.4$ to be an acceptable rough estimate of the smallest effect size of interest in psychological science [663], a simple comparison of two within-subjects conditions would require a minimum of 50 participants to achieve a power of 0.80, increasing into the hundreds as the complexity of the study increases [663]. Very infrequently do psychological researchers purposefully run a study that includes more than the minimum number of participants. This is seen as waste. However, the history of the field has demonstrated that this has at times resulted in many underpowered studies [663]. Kühberger et al. [666] demonstrated that the mean effect size found in studies with 50 participants is reliably larger than that in studies of 500 participants. Compounded by publication bias, this can then result in standardised effect sizes of published studies being too large when evidence of an effect is repeatedly demonstrated in small samples [667].

The vast majority of included studies (13/15) were largely made up of female participants. Sex differences in the prevalence of insomnia have been widely explored. One recent meta-analysis of 13 observational studies found a significantly higher prevalence of insomnia in females (odds ratio [OR] = 1.58; 95% confidence interval [CI] = 1.35, 1.85) [668]. This, accompanied with the primarily female samples included in this review, may limit the generalisability of the results. Relatedly, despite our broad inclusion criteria, the mean age of participants across included studies was 69.45 years, ranging from 65 to 77.2 years. While this goes some way towards addressing age-related bias in clinical research, the under-representation of the ‘oldest old’ remains a recognised and persistent issue in the wider literature [669, 670]. Commonly defined as adults aged 85 or older – thought no universal consensus exists on this threshold [671] – the oldest old are frequently excluded from or are under-recruited to clinical trials. Consequently, the included studies and the inferences drawn from their synthesis likely reflect the younger end of the older adult spectrum. This may potentially limit the applicability of findings to the oldest old, who may present with different clinical needs, experiences of insomnia, or responses to behavioural treatments. This reflects a broader issue across clinical research in ageing populations. Future studies may wish to consider more targeted methods of recruitment, and exploration of whether tailored, age-specific interventions are warranted.

Finally, due to heterogeneity in study design, intervention protocols, and outcome measures, as well as several studies containing a high risk of bias, we deemed it inappropriate to quantitatively synthesise the results of included studies. Studies exploring BBTI presented as the most viable targets for meta-analysis, however we elected not to do so for several reasons. Firstly, although Chen et al. [623] and Kwon et al. [620] considered Germain et al. [644] to employ BBTI, authors of the present study considered the intervention too distinct from the manualised BBTI protocol outlined in Troxel et al. [619] for this to be the case. Treatment duration and number of sessions were the primary reasons for this decision. Therefore, Germain et al. was included in the present review as a multicomponent intervention, but not BBTI. Secondly, Tyagi et al. [643] being a secondary data analysis of Buysse et al. [641] meant that inclusion of this data in a meta-analysis would have involved ‘double-counting’ of participants [672, 673], which may introduce bias into the model. Of the remaining three BBTI studies [640–642], one, Gebara et al. [640], was a pilot RCT. As outlined above, underpowered studies commonly overestimate the magnitude of treatment effects [663], and thus are typically not appropriate for inclusion in meta-analyses [674]. Moreover, it was confirmed that effect size estimates in Gebara et al. were derived following amalgamation of data from immediate and delayed treatment groups in the final analysis. Thus, inclusion of this would have meant comparing within- and between-group effect sizes. The remaining two studies [641, 642] were sufficiently similar to meta-analyse within a fixed-effects model, but too few for a random-effects model. Considering the between-study heterogeneity, and that we wish for these findings to be generalisable beyond the two included studies, we considered a narrative synthesis to be the most reliable analysis that could be performed with the data currently available.

4.5.7 Future directions

The present review highlighted several areas where the empirical literature is lacking in the investigation of behavioural interventions for insomnia in older adults. Firstly, studies employed primarily community-based samples, and it would be useful to investigate these interventions in other populations, such as those living in nursing homes. Moreover, results from Tyagi et al. [643] highlight how the outcomes of interventions can vary in different clinical populations. Future work should aim to delve deeper into this, in different samples. Studies outside of the USA as well as studies exploring the possibility of sex differences in treatment response would be particularly prudent. Additionally, this review focuses on the

effect of stimulus control and sleep restriction on outcomes related to sleep and mental health; however, the relationship between disrupted sleep and impaired cognitive function is well established [73, 426, 571, 587, 588, 675, 676]. A comprehensive examination of how behavioural interventions for insomnia may impact this relationship is indicated. In the case of multicomponent interventions including both sleep restriction/compression and stimulus control, researchers should consider experimental designs allowing for the incorporation of both ‘process’ and ‘outcome’ research. It is also important to consider measuring cognition at several points throughout treatment, to account for the possibility of sleep restriction impairing cognition in the short term. With the contemporary approach to healthcare of ‘precision medicine’ [677] comes a curiosity of how this notion can be applied to sleep medicine. Exploring different combinations of behavioural and cognitive methods to treat insomnia would be a prudent point of departure in determining which is optimal in different clinical populations. An extension to this should then be to determine in which groups, and how, these interventions can be implemented digitally to facilitate wider dissemination [678]. The feasibility and acceptability of internet-delivered CBT-I is already established [197, 679], but the efficacy of its individual components delivered in this way is yet to be established. Early studies exploring internet-delivered BBTI indicate the treatment to be feasible [622], however, randomised trials with much larger samples are necessary to ascertain estimates of treatment efficacy. Finally, the heterogeneity of outcome measures used across the studies emphasises the need for field wide consensus on the operationalisation of sleep parameters, in accordance with recommendations put forth by Hughes et al.[185].

Finally, although this review provides valuable insight into behavioural interventions for insomnia in older adults, its direct applicability beyond this population may be limited. Thus, this review should be considered a foundational point-of-departure for future research more closely aligned to the aims of this thesis – which may seek to investigate the acceptability, feasibility, and efficacy of sleep restriction and stimulus control in stroke survivors. While there are overlapping clinical features between older adults and stroke survivors [294, 625], challenges unique to stroke – as identified in Chapter 2 and in Smejka et al. [203] – may influence treatment feasibility and effectiveness. Thus, empirical evaluation of these interventions in samples of stroke survivors is warranted.

4.5.8 Conclusions

For some clinical populations, CBT-I may be cognitively taxing. By dissecting the individual components on CBT-I, and removing the cognitive aspects, we can evaluate the key behavioural mechanisms that drive clinical change. Thus, it is imperative that we develop a deeper understanding of how explicitly behavioural interventions elicit change in different clinical populations, allowing us to not only identify the optimal interventions for said groups, but also increase the ease with which effective, evidence-based treatment for insomnia can be disseminated to those who need it. This is the first systematic review to investigate focused behavioural interventions for insomnia in older adults. The results suggest that Stimulus Control and Sleep Restriction are both effective in alleviating insomnia symptoms in older adults, particularly when combined as a multicomponent therapy. Moreover, these interventions are demonstrated to elicit moderate to large effects on SOL and WASO, after only two therapeutic sessions. Impact on mood is less clear, but it does appear that when combined, stimulus control and sleep restriction can also elicit significant improvements on measures of depression, but not anxiety. These findings can inform future research into treating insomnia in populations where CBT-I may not be accessible or appropriate, such as stroke survivors.

Chapter 5

Brief Behavioural Treatment for Insomnia After a Stroke - A Protocol for a Single Case Experimental Design Study

5.1 Introduction

5.1.1 Background

Stroke is the second leading cause of death worldwide, accounting for 11% of annual mortality [287]. Strategic efforts to reduce the incidence of stroke by targeting known risk factors have reduced the incidence of *age-specific* stroke in high-income countries [283, 285, 291, 294, 295, 298], with estimated annual reductions of 1.0% to 1.5% over the last three decades [295]. Nevertheless, ageing populations are predicted to lead to future increases in the absolute numbers of incident stroke [283, 285, 288, 294–297]; although estimates of the magnitude of this increase vary considerably [288, 295, 296]. Advances in acute stroke care [299, 300, 680] and secondary stroke prevention [301, 302], have significantly curtailed incidence rates of immediate stroke fatality [285, 294]. While unquestionably welcome, necessarily associated with increasing incidence and survival rates is a corresponding increase in the overall impact of stroke and utilisation of healthcare [288, 294]. King et al. [296] predict that the societal cost of stroke in the UK will increase from £26 billion in 2015, to £75 billion in 2035, representing 1.5% and 2.9% of GDP respectively; however, one should remain cognisant of these estimates being based on subjective predictions, and thus potentially inaccurate. Regardless, clinicians and researchers are faced with many prospective challenges around improving the trajectory of stroke recovery, and alleviating the impact of stroke to the individual, their family, and society. Sleep has been identified as a possibly viable target through which to address some of these challenges, including the prevention of stroke recurrence [302, 349, 350].

Disturbances to sleep are common after stroke [313, 315, 318–324, 379, 380, 681], and appear bi-directional, acting as both a risk-factor and consequence of stroke [56, 312, 314, 315, 318, 319, 331–338, 340–343, 352, 418]. Insomnia disorder is particularly prevalent after stroke [321, 379, 380] - considerably more so than is typically observed in the general population [89] - and is associated with poorer outcomes [331, 369, 370, 376, 377, 381, 423, 424, 426]. Various studies have demonstrated that impaired sleep after stroke may impede aspects of recovery [359, 360, 366, 368, 375, 382, 391, 398, 423], but also that improving sleep be facilitatory to neurorehabilitation [361, 682, 683]. Thus, the importance of optimising sleep after stroke, and of ascertaining the optimal means of doing so, is indicated.

The results of Chapter 2 (see § 2.4.3.2), corroborate existing findings that non-pharmacological treatments are preferred by people with insomnia [106, 107]. Several such approaches have been trialled after stroke with varying degrees of success [684], including electroencephalography (EEG) biofeedback [685], bright light therapy [686], acupuncture [687, 688], transcranial magnetic stimulation (TMS) [689, 690], and Cognitive Behavioural Therapy for Insomnia (CBT- I) [402–408].

CBT-I is the first-line treatment for insomnia in the general population [83, 98–100, 605], although dissemination has been limited [180, 182, 184], and issues with adherence have been identified [210, 211, 691]. To date, seven studies have sought to explore CBT-I’s efficacy after stroke [402–408], and do show promising effects on sleep and mood, including when delivered digitally [402–404, 408, 409]. However, the longevity of effects appears diminished compared to that typically observed in the general population [178, 179], and that stroke-related impairments and disability may restrict engagement with treatment protocols [203, 403, 405]. Additionally, all investigations of CBT-I after stroke were conducted prior to any diagnostic validations of insomnia measures in stroke survivors [412, 545], including that outlined in Chapter 3. Consequently, several [402, 406–408] have employed cut-off scores on screening measures which may be suboptimal in stroke survivors, and comparisons and interpretations should be drawn with this in mind. Exploration of behavioural interventions which address stroke-specific barriers to treatment, and employ the optimal thresholds on validated measures, is indicated.

CBT-I itself is not a stand-alone treatment, but rather a treatment framework comprised of various monotherapies [25, 210]. Researchers and clinicians are increasingly advocating for a more parsimonious and targeted approach [25, 130]; calls which are supported by the results of Chapter 4. Such a shift would allow for individual elements to be adapted, added, or removed depending on the treatment needs of a specific population [25, 145]. Stripping CBT-I to its constituents not only streamlines treatments, potentially addressing the disparity between supply and demand, but also allows for more process focused research. This may elucidate the mechanisms underpinning treatment efficacy, and potentially uncover clinical scenarios, or populations, where different configurations of CBT-I confer the greatest benefit [25, 145, 148].

Stimulus control [112] and sleep restriction [224] are often considered the principal therapeutic components [120, 692] of CBT-I and are evidenced to be effective as monotherapies [128, 131, 624]. As highlighted in Chapter 4 (see § 4.4.4), these therapies are effective at alleviating symptoms of insomnia disorder, along with corresponding improvements in mood [145, 159, 199], and may help to stabilise night-to-night variability typical to insomnia [135, 145]. Although effective in isolation, these treatments appear most potent when delivered in tandem (see § 4.4.4), such as via Brief Behavioural Treatment for Insomnia (BBTI) [619].

5.1.2 Brief Behavioural Treatment for Insomnia (BBTI)

Comprised of psychoeducation, sleep restriction, and stimulus control, BBTI was developed with the limitations of CBT-I in mind. Hence, it is intended to be brief, acceptable, easy to deliver, and quickly effective [619]. The treatment is delivered over four sessions (two in-person, two via telephone), is explicitly behavioural in focus, and follows a manualised workbook, promoting treatment fidelity (for an overview and rationale see Troxel et al. [619]). BBTI's efficacy has been demonstrated in various clinical populations, particularly older adults [620, 621, 623] (see § 4.4.4.3), and appears non-inferior to CBT-I [693]. Given its brevity and evidenced acceptability and efficacy in older adults, BBTI may be a viable alternative to CBT-I for stroke survivors. With that in mind, several Patient Public Involvement (PPI) groups were conducted to garner insight into stroke survivors' views of BBTI and discuss possible stroke-specific adaptations to the standard protocol. PPI groups can aid efforts to enhance the quality and appropriateness of research [694], and can provide insight into issues relating to accessibility and representativeness that may not be obvious to people without specialist knowledge or lived experience. The findings of such groups have facilitated the development of the procedure and materials for the current protocol.

Importantly, BBTI is a multicomponent therapy, in which different elements may interact, influence multiple outcomes, and require individual tailoring to accommodate a participant's specific needs, preferences, and circumstances. Although it does follow a manualised workbook [619], limiting the risk of therapist effects [695], its flexibility and multiplicity can make complete standardisation difficult. As a result, BBTI qualifies as a 'complex intervention'—one in which variability in implementation and response must be carefully considered [696,

697]. To ensure rigorous evaluation, a study’s design must incorporate methods that account for these complexities, such as detailed process evaluations, multiple measurement points, and appropriate analytical techniques that can capture individual differences and treatment mechanisms [696, 697].

5.1.3 Single-case experimental designs (SCED)

SCEDs are useful for evaluating complex interventions, and can offer deeper insight into the efficacy, effectiveness, and experience of an intervention for an individual than standard group comparison designs [697–700]. Though prominent in the early years of psychological research, their use has fluctuated over the last several decades [698, 701]. Adolphe Quetelet’s discovery that statistical laws relevant to celestial phenomena were also applicable to human behaviour and characteristics – whereby the distribution of these phenomena typically forms a normal distribution with sufficient sampling [702] – shifted the psychological zeitgeist towards comparing groups, rather than studying individual organisms [698, 701, 703]. Furthered by eminent figures such as Francis Galton, Karl Pearson, and R.A. Fisher, group comparison – and RCTs in particular – became the predominant methodology in the 20th century [698, 701, 703].

More recently, the question arose as to whether the merits of RCTs were being overestimated, and if alternatives – such as SCEDs [700, 701] – may be preferable in certain circumstances [704, 705]. Of particular pertinence to applied researchers and clinicians is the applicability of aggregated group results to an individual [706]. In efforts to recruit the perfectly randomised and normally distributed sample, RCTs may exclude outliers, or complex cases, in effort to control between-subject variability [700, 701]. In doing so, researchers may inadvertently reduce the relevance of findings to an individual, as evidence of any nuanced differences between participants are lost in the aggregate [698, 700]. Clinicians are therefore often unable to determine whether an individual they aim to treat is more akin to those who benefited from the treatment, or those who did not [698]. Additionally, group comparison studies tend to focus on the outcome of an intervention, and less so on the events that transpire throughout the treatment process [698, 701]. Thus, their capability to elucidate therapeutic mechanisms, identify potential harms, and inform possible adaptations, is limited [698]. While RCTs are useful when determining whether a treatment works, they are less useful in ascertaining how it works, and for whom it works best [704–707].

SCEDs, in contrast, are well-positioned to address these questions [698–701]. Ranked by the Oxford Centre for Evidence-Based Medicine as level 1 evidence for treatment efficacy in individual cases [708] - equivalent to systematic reviews – SCEDs are enjoying a once again burgeoning interest in psychological research [699, 700, 709, 710]. These prospective studies use repeated measures to intensively study one or more participants before, during, and after intervention, analysing an individual’s data in its own right, thereby allowing participants to serve as their own control and providing insight into the processes and mechanisms underpinning treatment effect [698–701]. Moreover, SCEDs address several ethical and practical difficulties associated with RCTs, such as withholding potentially beneficial treatments from control groups, putting experimental groups at greater risk, and the temporal and financial costs of recruiting sufficiently sized samples [698, 711, 712]. These benefits are particularly advantageous in studies of complex and novel interventions, or in rare conditions where gathering a homogenous sample would be difficult [700]. Moreover, as SCEDs are able to generate reliable inferences from small samples, and statistical power is drawn from repeated measurements and not the number of participants, they are less prone to type-II errors [700].

5.1.4 Aims & objectives

Given the impediments caused by cognitive impairments outlined in the extant literature [203], and identified in Chapter 2, we postulate that removal of the cognitive components of CBT-I may increase accessibility for individuals living with post-stroke cognitive impairment or low cognitive engagement. To date however, no study has explored CBT-I’s behavioural components after stroke, in the absence of cognitive therapy. To that end, we aim to address this population gap by examining the feasibility and acceptability of BBTI, adapted to account for common difficulties after stroke, in a sample of stroke survivors with insomnia. Moreover, we aim to examine the efficacy of BBTI at alleviating symptoms of insomnia after stroke, and to explore whether these effects generalise to mental health and fatigue.

5.1.5 Hypotheses

We hypothesise that stroke adapted BBTI will be acceptable and feasible to people living with post-stroke insomnia. We also anticipate that the intervention will elicit reductions in both objective and subjective parameters of sleep that are relevant to the nosology of insomnia; specifically sleep onset latency and wake after sleep onset. Secondary to this, we predict that treatment effect will generalise to measures of fatigue and depression. However, guided by the results outlined in Chapter 4, we do not anticipate that treatment effect will generalise to anxiety.

5.2 Methods

The present study will be reported in accordance with the Single Case Reporting Guideline in Behavioural Interventions (SCRIBE) [713].

5.2.1 Study design

This study will employ a single-case experimental design, with multiple baselines across participants. Participants will enter baseline concurrently, but onset of the intervention will be staggered and randomised using www.randomizer.org; necessarily resulting in differing lengths of baseline phase. Participants will be blinded to details of the intervention until the beginning of the treatment phase. Evidence of any treatment effect will be gleaned from identifying that if change occurs, it only does so when the participant is exposed to the intervention; ensuring that the effects of the intervention are not confounded by environmental changes occurring contemporaneously [714]. Treatment effects will be evaluated across three participants and replicated in two subsequent samples.

5.2.2 Participants

Participants will be UK-based stroke-survivors (≥ 18 years old), meeting DSM-5 criteria for chronic insomnia disorder [67] (see Table 1.1), and have expressed interest in alleviating their difficulties with sleep. A history of stroke will be confirmed through correspondence with a participant's general practitioner or another relevant healthcare professional (e.g., neuropsychologist, neurologist), ensuring that diagnosis has been confirmed clinically or radiologically (e.g., CT or MRI scans). Selection criteria will require that participants are community-dwelling and demonstrate capacity to provide informed consent. A history of working nightshifts, existing enrolment in interventions for sleep or stroke, cognitive impairment sufficient to prevent engagement with study materials and tasks, a self-reported diagnosis of dementia, or the presence of any other sleep disorder will stand as grounds for exclusion. Following the recommendations of Troxel et al. [619], potential participants with a self-reported diagnosis of bi-polar disorder will also be excluded, due to the risks associated with sleep deprivation and mania. Furthermore, qualitative explorations of behavioural treatments for insomnia after stroke have identified sleep restriction as being particularly challenging to participants [203]. To avoid the risk of excessive sleep deprivation causing intolerable distress or impairment to participants, or impairing adherence to the protocol, and in concordance with recommendations put forth by Troxel et al. [619], participants who demonstrate a time in bed of < 6 hours pre-enrolment will also be excluded. Ongoing use of sleep medications will not preclude participation, provided that participants agree to make no changes to the medication or dosage for duration of the study unless indicated as necessary by their treating clinician. Sub-group analyses of BBTI in older adults established that no differences in magnitude or direction of treatment effect were evident between those participants taking medication and those not [641].

5.2.3 Recruitment

Recruitment will follow a community-based strategy, facilitated by third sector organisations such as the Stroke Association, and supplemented, if necessary, by sharing recruitment advertisements via social media. Gatekeepers will share details of the study with stroke survivors via peer support groups, encouraging those interested to contact the lead researcher via email. Potential participants will complete screening and consent forms via Qualtrics www.qualtrics.com. A

telephone consultation will follow, to confirm eligibility and afford potential participants an opportunity to ask any questions about the study, while making them aware that no advice relating to sleep, or specific details of intervention protocols, can be given until the intervention starts. Undecided participants will be encouraged to discuss the opportunity with their friends and family, and researchers will contact them 2-3 days later to discuss. Participants will be advised to discuss their participation with their GP.

5.2.4 Sample size

The intervention will be completed with three participants initially and replicated in two subsequent groups, giving a total of nine participants, allowing for three demonstrations of treatment effect, demonstrated three times, strengthening confidence that any change is not due to unaccounted for confounds or spontaneous recovery [639]. Recruitment will continue until nine people have completed the intervention.

5.2.5 Measures and materials

5.2.5.1 Screening and demographic measures

Demographic data will be collected at the screening stage, and will pertain to a participant's age, gender, ethnicity, living status, educational level, mental health, cognitive impairment, and stroke history. DSM-5 insomnia disorder will be assessed via a comprehensive sleep history questionnaire [439], designed to mimic a clinical interview. The Oxford Cognitive Screen (OCS) [715] will be used to screen for cognitive impairment prior to enrolment in the study. Scores lower than the 5th percentile (or higher than the 95th on reversed items) in comparison with normative data [715] will be considered indicative of cognitive impairment. Should this scenario arise, participants will be advised to discuss any cognitive difficulties with their GP, but will not be excluded provided they demonstrate sufficient capacity to understand, and retain information pertaining the study.

5.2.5.2 Outcome measures

Congruent with European Sleep Research Society guidance on the assessment of insomnia [100], sleep diaries will be used to gather subjective data pertaining to sleep onset latency (SOL), wake after sleep onset (WASO), number of awakenings (NWAK), total sleep time (TST), time in bed (TIB), sleep quality (SQ; measured on a 0-5 scale), and sleep efficiency (SE%). The Consensus Sleep Diary [716] will be completed each morning throughout all phases of the study. Results of the PPI group indicated that some stroke survivors may struggle to write with a pen after their stroke, while previous research [203] suggested that others may struggle to engage with online resources. Thus, participants will be permitted to complete sleep diaries using pen and paper, or via an online questionnaire; enhancing the accessibility of the intervention. Evidence suggests that subjective sleep parameters do not change with respect to whether they are completed digitally or via paper [717].

Objective measures of SOL, WASO, NWAK, TST, TIB, and SE% will be obtained using Axivity AX3 wrist mounted actigraphy devices (see axivity.com), which allows for triaxial recording of movement. Actigraphy devices are recommended for the measurement of physical activity after stroke and appear accurate in detecting sleep during acute stroke care [718], though have never been formally validated as a measure of post-stroke insomnia. Moreover, recent evidence suggests that, in the general population, actigraphy may not be accurate in distinguishing people with insomnia from good sleepers [719]. Hence, actigraphic data will be used to compliment sleep diary data, and to assess adherence to the intervention. Actigraphy devices will be worn on the participant's non-dominant wrist, unless movement in this wrist has been impacted by their stroke. In which case, participants will be asked to wear the device on whichever wrist was least impacted by their stroke. A sampling rate of 100hz will be adopted – allowing for up to 14 days of recording – and data will be averaged into 30 second epochs. Actigraphy data will be downloaded to a password protected laptop using the device manufacturer's software and analysed in R using the 'GGIR' package [720]. The Euclidian norm minus one method will be used to account for gravity [721], and the 'vanHees2015' algorithm will be used to detect periods of sleep and wake [722]; this algorithm has been validated against polysomnography. Actigraphy data will be recorded daily throughout all phases.

The Sleep Condition Indicator [415] will be used as a psychometric measure of insomnia symptoms and overall sleep quality. As outlined in Chapter 3, the SCI demonstrates ‘excellent’ [544] diagnostic accuracy when detecting insomnia disorder and symptoms after stroke, however, a threshold of ≤ 13 is considered indicative of insomnia disorder, as opposed to ≤ 16 in the general population [415, 546]. Thus, the lower threshold will be adopted in the present study, and improvement that cross this considered clinically significant. SCI data will be collected once at baseline, post-treatment, and at a 3-month follow-up.

5.2.5.3 Generalisation measures

As is clear from the results of Chapter 2 (see § 2.4.2.2), and corroborated by the existing literature [324, 369, 370, 376, 377, 423–426], insomnia’s impact after stroke reaches far beyond an individual’s sleep, affecting their mental, cognitive, social, and physical wellbeing. Thus, while it is important to examine a treatment’s efficacy with measures that are clinically relevant to the nosology of insomnia, it’s also important to consider what outcomes are relevant to the individual [723] and their life after stroke. Thus, we will include measures of behaviours relevant to insomnia, but also measures to assess the interventions’ ability to generalise to other areas of wellbeing. Generalisation measures are common in SCED studies, and useful for improving a study’s external validity. Assessing generalisation involves employing measures of untrained behaviours, in order to ascertain how well the intervention has performed at improving participants’ well-being or performance beyond what it directly targeted [700]. In the present study, this will include the Generalised Anxiety Disorder scale (GAD-7) [440], the Patient Health Questionnaire (PHQ-9) [443], and the Fatigue Severity Scale (FSS) [724]. Generalisation measures will be recorded once at baseline, in sessions 2 and 4, at post-treatment, and at a 3-month follow-up.

5.2.6 Procedure and intervention

All participants will go through four phases of data collection: baseline (8-12 days), treatment (28 days), post-treatment (10 days), and follow-up at 3-months (10 days). Baseline lengths will vary between 8, 10, and 12 days, ensuring at least seven days of sleep diary data, including at least one weekend [725], and a minimum of five data points for each participant [639].

The intervention itself will be delivered over four weeks by a suitably experienced researcher with formal training in the delivery of behavioural interventions for sleep, using a BBTI manual [619], adapted to meet the needs of stroke survivors and available to participants in print and digital formats. The manual includes the rationale behind the intervention and contains areas for researchers to include each participant's prescribed sleep and wake times, suggested changes to sleep related behaviours, and personalised adjustments to treatment.

BBTI is traditionally delivered over four sessions, with sessions 1 and 3 being delivered in-person, and 2 and 4 via telephone [619]. To facilitate data collection, sessions 2 and 4 will also be delivered in person in this study. Sessions will take place in participants' homes, once per week throughout the intervention phase. Participants will be requested to bring their actigraphy devices and sleep diaries to each session.

In session 1, the researcher will gather baseline data, work through the workbook with participants, and deliver psychoeducation around sleep. Any daytime behaviours that may impede sleep will be identified and discussed, along with baseline sleep diary data. The participant will then be introduced to the four instructions of BBTI: (a) Reduce your time in bed, (b) Wake up at the same time every day, no matter how poorly you slept the night before, (c) Do not go to bed unless you are sleepy, and (d) Do not stay in bed unless you are asleep [619]. Session 1 concludes with the researcher and participant collaborating to set the prescriptions for sleep related behaviour, such as sleep and wake times, in the subsequent week. Session 2 offers an opportunity to work through any challenges identified in the previous week, gather data, and discuss adherence to the protocol. In session 3, researchers perform a more in-depth examination of sleep diary data and provide information on how to titrate the time in bed constraints of sleep restriction. If a participant's subjective SOL and WASO are <30 minutes on most nights of the previous week, then they should add 15 minutes to one end of their sleep window – which end can be decided by the participant. Contrariwise, if the sleep parameters are >30 minutes on most nights, then 15 minutes should be subtracted, provided this does not take total time in bed below 6 hours. This algorithm is deliberately designed to be easier to follow than typical CBT-I protocols [619]. In the final session, participants are afforded a final opportunity to discuss any issues they may be facing. Again, data will be collected, including relevant psychometrics. The participant's progress will be reviewed and discussed, titration methods will be revisited, and relapse prevention will be discussed. The researcher will revisit the four instructions and work with the participants to strategise a course of actions to follow in periods of transient

sleep disruption. Following completion of treatment, participants will continue to record data for 10 days, after which, devices and sleep diaries will be returned to the lead researcher via courier. Three months after the end of treatment, sleep diaries and actigraphy devices will be sent to participants to collect a further 10 days of data.

A simple process evaluation will accompany the above procedures, to explore potential mechanisms driving treatment effect, to understand contextual influences on these, and to identify any barriers or facilitators to implementation. This will involve reviewing participants adherence to treatment (via reviewing sleep diary and actigraphy data), completion rates of all outcome measures, and any adverse events. All participants will also complete semi-structured interviews following the completion of all procedures. These will be conducted via Zoom or Microsoft Teams, and will explore participants' experience of the intervention, their feedback regarding the acceptability, and any perceived barriers to adhering to the protocol. This will inform the development of future research investigating widespread implementation of the intervention as part of routine post-stroke care.

5.2.7 Plan of statistical and visual analysis

How SCED studies are best analysed and interpreted has long been a point of contention [699]. Some argue that visual analysis alone should be sufficient, as if an effect is not evident on time series plots, then it is not of adequate magnitude to confer any practical application [680, 726]. Others argue that the inclusion of statistical analysis is necessary to limit bias and human error [699]. Consensus has largely moved towards advocating for the inclusion of both methods [700, 727–729]. Not least as this can produce standardised and comparable outcomes, allowing for meta-analyses [699]. Indeed, Kratochwill et al. [730] recommend that estimates of effect size from SCED studies should be comparable to those generated from group comparison studies (see Hedges et al. [731, 732]). Moreover, either method may account for the limitations of the other, and triangulation introduces an additional level of validation by confirming that the results of either approach corroborates the other [733].

Visual and quantitative analysis will be conducted on sleep diary and actigraphy data, and psychometric data will be discussed descriptively for each participant. The reliable change index (RCI) will be determined for each participant's pre- to post-treatment scores on all psychometric scales, in order to establish whether any change is greater than could be explained by measurement error [734]. The RCI is calculated as:

$$RCI = \frac{X_2 - X_1}{S_E}$$

where X_1 is the baseline score, X_2 is the post-treatment score, and S_E is the standard error of measurement (SEM). SEM will be calculated using published test-retest reliability parameters for each psychometric scale [440, 550, 735, 736]. Time series graphs will be plotted, and inspected visually in accordance with guidelines detailed by Lane & Gast [737]. Within and between condition visual analysis will examine trend (progress across time), level (the magnitude of the data), and stability (variability within the data), individually for each participant. Level and trend lines will be plotted on time series graphs to facilitate visual analysis, using the median to minimise the effect of outliers. For each participant, the stability envelope will be calculated for data in each condition using an 80%/20% ratio; where data within a condition will be deemed stable if 80% of the data points fall within a window $\pm 10\%$ of the median [737, 738]. Visual analysis will be facilitated by using online software designed for the analysis of SCED data (see www.manolov.shinyapps.io).

Manolov et al. [739] advocate for the use of statistical methods that: reflect the aims of the analysis, are easy to interpret and compute, align with the design and assumptions of the data, and are evidenced to perform adequately with SCED-type data. To that end, Tau-U [740, 741], will be used as a non-parametric means of determining the degree of non-overlapping data. Where visual analysis suggests the presence of any baseline trend, baseline-corrected Tau [742] will be employed, reducing the risk of spontaneous recovery, or other unaccounted for variables potentially confounding results [743]. Either approach will be computed using the same software used in visual analysis. Counts of data points where subjective and/or objective SOL or WASO is below 30 minutes – a threshold relevant to the nosology of DSM-5 insomnia disorder [67] - will be generated. The percentage of data points meeting these criteria, or Percentage of Goal Obtained (PoGo) [744], will be calculated in each phase. Between phase comparisons will determine whether the introduction of treatment results in clinically meaningful improvements in this regard.

Finally, sleep diary and actigraphic data will be analysed using multi-level modelling [745, 746]. A separate model will be conducted for each participant, and each outcome. This method uses hierarchical linear regression to quantify estimates of effect size for each participant. Multi-level modelling addresses the fact that SCEDs are limited in their ability to generalise to individuals outside the study sample, by allowing for the combination of data within, and between, SCED studies, enhancing generalisability (see MultiSCED).

5.2.8 Ethical considerations

This study received ethical approval from the University of Glasgow, College of Medical, Veterinary & Life Sciences ethics committee (Project # 200230214; see Appendix M). Informed consent will be attained during the screening phases, and participants will be provided with copies of the participant information, signed consent forms, and privacy notices. Should it arise at any point that a participant may be living with an undiagnosed cognitive impairment, or psychiatric disorder such as depression or anxiety, they will be signposted towards services that can support them. Participants will be made aware of how their data will be processed and assured that they will be identified via a participant ID number. Only the lead researcher will hold the information necessary to identify participants via these ID numbers, and this information will be deleted after data collection is complete. Participants will be made aware of their right to withdraw at any point, without needing to provide an explanation, up to the point that data collection is completed; as it may not be possible to identify their data beyond this point. By nature of a multiple baselines across participants design, some participants will be in the baseline phase for extended periods, posing a potential ethical dilemma should the treatment prove to be effective. To account for this, the length of time which participants will be in baseline for will not exceed 12 days. Due to having prior knowledge of the study design and procedures, individuals who took part in the PPI group will not be permitted to take part in the intervention. Should the intervention prove to be efficacious, their exclusion poses as an ethical dilemma. In such circumstances, these individuals will be provided a summary of the results of the study, and made aware that they can discuss the provision of BBTI with a suitably trained CBTI practitioner either via the NHS or privately. In the early stages of treatment, mild sleep restriction may result in sleepiness and fatigue through the day. It is not anticipated that this should cause severe distress or impairment, but should this arise, participants will be advised to avoid driving

long distances where possible. Adhering to treatment should remedy this, and it is a sign that participants are responding to treatment as expected. The lead researcher will meet bi-weekly with the supervisory team, ensuring support for researcher wellbeing will be available.

Chapter 6

General discussion

6.1 Thesis objectives and findings

Insomnia disorder is common after stroke [321, 379, 747], and is associated with many negative outcomes spanning cognitive, physical, and psychological domains [368–370, 382, 422–426]. Despite this, several methodological, measurement, and evidence gaps exist within the extant literature. This thesis sought to address these gaps by answering four key questions relating to the impact, assessment, and treatment of insomnia after stroke:

1. What is the experience and impact of insomnia after stroke?
2. What are stroke survivors' experiences and preferences relating to treatment?
3. How can we accurately identify which individuals are living with insomnia after stroke?
4. How can we best go about treating insomnia after stroke?

Table 6.1 outlines the primary objectives, corresponding questions, and key findings of each chapter. This discussion chapter will synthesise the evidence discussed in each of the preceding chapters and evaluate the success of the research in answering the questions outlined above. It will also critically appraise the strengths and limitations of each study and consider the findings within the context of the broader literature. Finally, the clinical and theoretical implications will also be explored, and potential avenues for future research endeavours seeking to advance our understanding of managing insomnia after stroke will be proposed.

Table 6.1: Thesis objectives and key findings

Question Chapter		Primary objective	Key findings
1, 2	2	To explore the experiences of individuals living with insomnia after stroke, understand the meaning they derive from these experiences, and elucidate preferences towards treatment.	Impairment to sleep after stroke is immediate, and appears exacerbated by factors encountered in hospital. Insomnia comprises part of a complex network of mental, cognitive, physical, and social factors after stroke. Participants were dissatisfied with the support they had received for sleep, and demonstrated a strong preference for non-pharmacological treatments. Insomnia after stroke aligns well with existing aetiological models and appeared primarily driven by cognitive factors.
3	3	To investigate the discriminant validity of the Sleep Condition Indicator (SCI), and a shorter two-item variant (SCI-2), when detecting insomnia disorder after stroke.	The SCI demonstrated ‘excellent’ diagnostic accuracy when discriminating between stroke-survivors with and without insomnia disorder or symptoms, and significantly outperformed the SCI-2. However, a diagnostic threshold of ≤ 13 , rather than the conventional ≤ 16 , should be adopted within stroke contexts to reduce false-positive rates.
4	4	To evaluate the efficacy of sleep restriction and stimulus control, delivered independently and combined, in treating insomnia disorder in older adults.	Both interventions brought about significant improvements in subjective sleep outcomes. Treatments were most potent when stimulus control and sleep restriction were combined, conferring benefits after as few as two therapeutic sessions.
4	5	To assess the feasibility, acceptability, and efficacy of a behavioural intervention for insomnia, adapted to meet the needs of stroke survivors.	Not yet assessed.

6.1.1 Questions 1: What is the experience and impact of insomnia after stroke?

6.1.1.1 Summary of findings

The first question this thesis sought to answer – ‘What is the experience and impact of insomnia after stroke?’ – is explored in Chapter 2. Through qualitative investigation using interpretive phenomenological analysis (IPA), we were able to derive nuanced insights into stroke survivors’ subjective experiences of insomnia, which would have likely been inaccessible to quantitative methods [413]. Analysis of data from semi-structured interviews with ten participants suggested a dynamic experience of sleep after stroke, where insomnia comprises part of a deleterious network of factors across social, cognitive, physical, and psychological domains.

Concurrent with prior findings [321–323, 392, 452–455], participants’ accounts describe an immediate onset of sleep disruption after stroke, which is exacerbated by social, environmental, and clinical phenomena whilst in hospital. Though impairment to sleep oscillates throughout recovery, and some improvement occurs over time – congruent with meta-analytic evidence [321] - for no participant did their sleep return to the quality and duration enjoyed prior to stroke. Instead, acute insomnia is perpetuated by a nexus of social, physical, cognitive, and psychological factors. Acute insomnia becomes chronic as these factors – such as health anxiety, cognitive impairment, and fatigue – compound one another, and establish a self-perpetuating cycle. The complexity of this nexus is demonstrated by Figure 2.2, emphasising the breadth of potential perpetuating factors for insomnia after stroke.

Central to this process was pre-sleep cognitive activity. Often this related to the fear of stroke recurrence during sleep – particularly in those for whom stroke occurred in bed previously – and the belief that insomnia will impede stroke recovery. The consequent anxiety inhibited sleep onset and sleep maintenance, often contributing to stroke-related nightmares. These nocturnal disturbances worsened fatigue, cognitive impairment, and emotional distress the following day, which, in turn contributed to sedentariness, social withdrawal and reduced vocational performance. All of which fed back into pre-sleep cognitive activity and anxiety, inhibiting sleep, and perpetuating the cycle.

Participants also described engaging in safety behaviours – such as using distraction to avoid distressing emotions – which prevent them from challenging their beliefs or fears around sleep. Attentional and interpretive biases towards stroke- and sleep-related stimuli compounded this, as maladaptive patterns of behaviour served only to strengthen conditioned associations between the bedroom/bed and psychological distress or danger.

6.1.1.2 Contribution to the literature

Chapter 2 provides the first qualitative investigation into the experiences of insomnia after stroke. In doing so, Chapter 2 addresses a methodological gap, and enriches the existing quantitative literature base by incorporating subjective accounts. The novel and nuanced insights garnered can inform the generation of testable hypotheses and provide a robust point of departure for future research.

Results emphasise the need to optimise sleep in acute stroke settings, and to incorporate the assessment of sleep into routine stroke care. Also apparent is the necessity of developing stroke-specific, and targeted treatments for insomnia after stroke. Though ascertaining a definitive aetiology of insomnia after stroke was not the aim of this study, such an endeavour may be informative where treatment is concerned, and findings do integrate with – and expand on – existing aetiological models [25, 152, 223, 224, 230, 231]:

- **Spielman’s 3P model**

- Working within the framework of Spielman’s stress-diathesis model [224], stroke itself and impaired sleep while hospitalised present as clear candidates for potential precipitators to insomnia disorder. Once established, acute insomnia becomes chronic through the bi-directional action of sleeplessness and various social, cognitive, physical, and psychological factors.

- **Harvey’s cognitive model**

- Harvey’s cognitive model [152] posits that attentional and interpretive biases towards sleep-related stimuli contributes to anxiety and pre-sleep cognitive arousal, thereby impairing sleep. Present findings extend this by implicating a health-specific dimension to cognitive arousal, which appeared intensified by stroke. Fears that stroke will recur during sleep, or that insomnia will impede recovery, led to participants fixating on innocuous physical sensations while in bed, misinterpreting them as portends of stroke. Additional to this was the anxiety and cognitive arousal resultant from concerns related to the impacts of stroke, such as cognitive impairment, particularly where this was perceived to have an impact on others (e.g., family members or colleagues).

- **The psychobiological inhibition model (PIM)**

- As a result of fearing that insomnia will impede stroke recovery, participants felt an increased importance around sleep, putting themselves under great pressure to fall asleep, for fear that failure to do so would be catastrophic to one’s recovery. Congruent with Espie’s PIM [25] and attention-intention-effort (AIE) pathway [223], this pressure led to participants attempting to exert volitional control over sleep. The AIE pathway argues that these efforts paradoxically serve to further perpetuate insomnia by reinforcing maladaptive conditioned associations, increasing pre-sleep cognitive arousal and anxiety.

These findings build on existing models by incorporating specific factors which contribute to the origin, evolution, and maintenance of insomnia after stroke. The identification of the nexus of drivers and consequences which post-stroke insomnia sits within corroborates the view that insomnia is a psychobiological condition [25, 230, 231, 234]. Whether this network of factors inhibits sleep via increased arousal [231, 262] or the inability to de-arouse [25] is not answerable with the information provided by the present study, but the outcome is ultimately the same – stress overcomes the homeostatic sleep drive and the transition from wake to sleep is inhibited.

Additionally, Chapter 2's answer to the opening question of this thesis emphasises the importance of identifying and supporting people living with insomnia after stroke. By identifying insomnia as a constituent part of a multifaceted network of factors, these results enhance our understanding of how insomnia can impede recovery and disadvantage quality of life and wellbeing after stroke.

6.1.2 Question 2: What are stroke survivors' experiences and preferences relating to treatment?

6.1.2.1 Summary of findings

This thesis' second key focus was to explore stroke survivors' experiences of seeking support for their sleep, and their preferences relating to treatment options. Despite their known influence on treatment adherence and outcomes [428–433], no study has previously explored patient perspectives and preferences where it relates to treatments for insomnia after stroke.

Chapter 2 addresses this knowledge gap, and results reveal shortcomings in current approaches to managing sleep after insomnia. Underscoring the lack of attention afforded to sleep after stroke, no participant had received a formal assessment of sleep or a sleep-related diagnosis. Prior research suggests that these shortcomings may be attributable to limited clinical awareness of sleep after stroke [315] and a paucity of validated measures and guidelines [396]. Participants' accounts demonstrate their profound dismay towards the lack of support they have received where sleep is concerned. Congruent with the existing literature [315, 317, 469, 513, 514], participants felt as though their clinician minimised, normalised, or outright dismissed their concerns and preferences regarding treatment. Bereft of support, participants felt isolated and abandoned, as though no one cared about their difficulties. Often this led to strained relationships between participants and their clinicians and may have contributed towards participants doubting the legitimacy of their concerns and normalising symptoms. Furthermore, sleep was not prioritised in recovery, ceding precedence to physical ailments, for clinicians and stroke survivors alike.

When discussing treatment options, participants expressed a clear preference for non-pharmacological approaches, a pattern consistent with the insomnia literature more broadly [106, 518]. This preference was largely driven by scepticism towards the safety and efficacy of hypnotic medications. Participants harboured concerns around side-effects, polypharmacy, and addiction or dependency; often intensified due to previous negative experience of pharmacological interventions. In contrast, desire to retain (or regain) agency over one's sleep contributed to participants proclivity for behavioural approaches. Such treatments were viewed as antithetical to pharmacological interventions, and were thought of as more 'natural', as having more robust and longer lasting effects, and as providing deeper insight into the origins of one's sleep difficulties.

Nevertheless, participants were not without reservations when it came to behavioural approaches; on the contrary, many concerns common to CBT-I generally – such as costs and availability [180] – arose in discussions. Other concerns were more distinct to stroke. For instance, stroke-related disabilities, such as cognitive or physical impairments, were seen as potential barriers to engaging with treatment protocols. Relatedly, participants expressed distress over the realisation that methods which were previously effective at facilitating sleep were no longer accessible or effective after stroke, contributing to their sense of desperation and helplessness.

6.1.2.2 Contribution to the literature

Chapter 2 presents the first study to explore the preferences stroke survivors hold with regards to insomnia treatment, and their experiences surrounding support for their sleep after stroke. Findings highlight significant shortcomings in current clinical practices and understanding of post-stroke insomnia, which corrective efforts must be directed towards, and reveals unique challenges faced by this population. Chapter 2 extends on prior evidence demonstrating inadequate clinical management of sleep after brain injury [513, 514] by elucidating the considerable personal impact this can have, and how those affected interpret and respond to their experiences. Particularly pertinent is the potential for clinicians' dismissive attitudes to influence patients' perspectives of their condition and experiences, which in turn may shape their perceptions and preferences around treatment [428–433].

Findings clearly indicate the treatment preferences of stroke survivors to echo those across the insomnia literature more broadly [106, 518]; that being for non-pharmacological approaches. Extending this however, present results compliment the work of Smejka et al. [203], by identifying potential stroke-specific obstacles to engagement. These obstacles present as obvious targets for researchers and clinicians seeking to optimise treatments for sleep after stroke. Consistent with calls for personalised approaches to sleep medicine [25, 748, 749], the heterogeneous nature of participants' experiences and concerns underlines the importance of adopting person-centred approaches when addressing post-stroke insomnia.

Though no unanimous consensus could be reached on preferred approaches to the delivery of behavioural interventions for insomnia, participants living with language difficulties expressed a preference for individual, in-person sessions, primarily to control their environment and mitigate its impact on their aphasia. This indicates the need to investigate specific treatment obstacles in different sub-groups of the stroke population. Relatedly, these findings align with existing models of health-related beliefs and their influence on treatment adherence and outcomes [428–433], further highlighting the importance of patient perspectives when designing and delivering interventions. Finally, Chapter 2 underscores the urgent need for validated measures of insomnia after stroke to facilitate the identification of individuals requiring support, and to improve clinical practice and reliable research.

6.1.3 Question 3: How can we accurately identify which individuals are living with insomnia after stroke?

6.1.3.1 Summary of findings

Addressing the need for improving post-stroke support for sleep identified in Chapter 2, Chapter 3 investigates how best to identify stroke survivors in need of support. Accurate assessment methods are critical for ensuring validity and reliability in clinician and research contexts. This has been hampered however, by an absence of insomnia measures validated for use after stroke, as well as stroke-specific clinical guidelines for the management of sleep and sleep disorders [396].

To address this measurement gap, an online questionnaire – comprised of the SCI [415], a sleep history questionnaire [439], and a battery of sleep, mental health, and stroke severity measures – gathered data from 180 stroke survivors. The accuracy of the SCI when discriminating between stroke survivors with and without DSM-5 insomnia disorder [67] was validated against classifications derived from the sleep history questionnaire.

The proportion of participants meeting criteria for DSM-5 insomnia disorder (41.7%), or demonstrating insomnia symptoms without meeting criteria for chronic insomnia disorder (60.0%) were consistent with meta-analytic estimates of prevalence after stroke [321, 379, 380]. Computation of Cronbach’s α found the SCI to demonstrate acceptable internal reliability after stroke ($\alpha = 0.84$). Receiver operating characteristic (ROC) analysis found the SCI to possess ‘excellent’ [544] diagnostic accuracy for both insomnia disorder (AUC = 0.86) and insomnia symptoms (AUC = 0.85). However, computation of Youden’s J [558] values yielded an optimal diagnostic threshold for DSM-5 insomnia disorder of ≤ 13 , not ≤ 16 as is conventional in the general population [415, 546]. Were the latter adopted in the current sample it would yield a false-positive rate of 45.7%. We therefore strongly advocate for the adoption of the lower threshold (≤ 13) when administering the SCI in stroke contexts.

The discriminant validity and reliability of a shorter, two-item version of the SCI – the SCI-2 [551] – was also explored. Demonstrating ‘acceptable’ [544] diagnostic accuracy, the SCI-2 may offer a more expedient alternative to the SCI in circumstances where time is constrained, or participant burden is high. However, Delong’s test evidenced that the full 8-item SCI significantly outperformed the SCI-2 ($p < 0.001$) and thus should be preferred where circumstances allow.

6.1.3.2 Contribution to the literature

Building on prior validations of Chinese [412] and Indonesian [545] translations of various insomnia measures after stroke, Chapter 3 presents the first validation of any insomnia measure in a sample of English-speaking stroke survivors; addressing a critical measurement gap in the literature. Findings establish stroke-specific recommendations when administering the SCI, enhancing the precision with which post-stroke insomnia can be identified, both in clinical and research contexts.

The reason why a lower threshold appears optimal after stroke is unclear and may have several potential explanations. Firstly, sleep may simply be poorer in general after stroke [313], or there may be a degree of crossover in symptoms of insomnia and common difficulties after stroke, and thus total SCI scores would potentially be reduced regardless of insomnia status. Mean SCI scores in Chapter 3 ($\bar{x} = 13.74$, $SD = 7.51$) are lower and demonstrate greater variance than those reported in values derived from a reference sample of 200,000 adults ($\bar{x} = 14.97$, $SD = 5.93$) [546]. However, assuming a normal distribution, these values suggest that the percentage of participants with probable insomnia (i.e., scoring 16 or less) is approximately 56.89%. This is considerably higher than prevalence estimates of insomnia [85] and insomnia symptoms [609] in the general population. Thus, it is possible that results in Espie et al. [546] may be biased towards people with insomnia, and the true population values are likely higher. Relatedly, ‘Gold Standard’ and infallible reference tests are not always available [560], and use of imperfect tests may introduce bias [750]. The SCI’s conventional threshold of ≤ 16 was determined by calculating the minimum score necessary on the scale necessary for putative DSM-5 insomnia disorder [415, 546]. Convergent validity was then assessed against the insomnia severity index [150]. While the ISI boasts commendable accuracy, it was not designed to detect DSM-5 insomnia disorder, and is less specific than clinical interviews. Thus, when comparing the optimal diagnostic cut-offs between studies, one should be cognisant of the possibility of differences in reference standards explaining some or all the variation in optimal cut-offs.

Moreover, our results expand on Hasan et al. [545]’s prior validation of the SCI in Indonesian, which reported greater accuracy, and recommended a higher diagnostic threshold (≤ 23). Existing validations of translated versions of the SCI do demonstrate heterogeneity in estimates of optimal cut-offs [547, 549, 554, 751], suggesting possible confounding effects of translation. Additionally, the prevalence of insomnia disorder in Hasan and colleague’s sample was 31%. This is notably lower than the 41.7% observed in the present works, which is more consistent with meta-analytic estimates of insomnia prevalence after stroke [321, 379, 380]. The accuracy of a binary diagnostic test is dependent upon prevalence [752], and though sensitivity and specificity are often thought not to be, recent meta-analytic results suggest the contrary [559]. If Murad et al. [559]’s findings are correct then the influence of prevalence on sensitivity and specificity will necessarily influence Youden’s J . Thus, variance in prevalence may explain, at least in part, discrepancies in accuracy and optimal thresholds.

Perhaps more likely however, is the indirect influence of prevalence on Youden's J , via the effects of differences in selection criteria. Spectrum bias is a phenomenon where the accuracy of a diagnostic test may be confounded by symptom severity [561, 753]. Proximal scores, closer to the threshold, are at a greater risk of misclassification. Therefore, studies where index test scores demonstrate a bimodal distribution have a greater risk of spectrum bias. Hasan et al. [545] excluded 164 participants with sleep or psychiatric disorders other than insomnia, many of whom would have undoubtedly scored lower on the SCI regardless of insomnia classification. This likely skewed the distribution of total SCI scores. Mean SCI scores in Hasan et al. for participants without insomnia disorder was 29.55 (SD = 1.96), compared to 19.35 (SD = 6.79) in the present work; more akin to prior investigations of sleep after stroke [313]. Having scores of people without insomnia stacked towards the top of scale allows specificity to remain high at higher thresholds, contributing to potentially misleading estimates of accuracy and optimal thresholds. The broader inclusion criteria employed in this thesis, as well as stratification by insomnia severity, reduce the risk of spectrum bias, and demonstrate the SCI's utility in a more representative and clinically meaningful sample.

6.1.4 Question 4: How can we best go about treating insomnia after stroke?

6.1.4.1 Summary of findings

Building on the results of the preceding chapters, Chapters 4 and 5 address the fourth and final question this thesis set out to answer: 'How can we best go about treating insomnia after stroke?'. Chapter 2 suggested that non-pharmacological approaches were the preferred choice of stroke survivors with insomnia. CBT-I is considered the first line treatment for insomnia in the general population [100], however, prior investigations of this after stroke suggest that the longevity of its effects may be curtailed after stroke [402–408]; potentially due to stroke related disabilities [203]. Moreover, some of the fears and beliefs identified in Chapter 2 – such as fear of stroke recurrence during sleep, or the belief that recovery would be impeded by sleeplessness – are supported by evidence [56, 318, 347, 353, 358, 383, 536]. Thus, these beliefs are arguably adaptive, not necessarily irrational or dysfunctional, and therefore potentially inappropriate targets for cognitive therapy

[152, 153, 156, 210]. Adding to this, given insomnia’s considerable impact on cognitive impairments highlighted in Chapter 2 an explicitly behavioural approach, comprised of stimulus control [112] and sleep restriction [113] was postulated to be optimal.

Though considered the primary behavioural components of CBT-I [120, 692], sleep researchers are returning to focus on the utility of these treatments as isolated monotherapies [25, 128, 130, 148, 624, 754, 755]. Due to an absence of studies in the extant literature examining stimulus control and sleep restriction independently in stroke, a systematic review was conducted which focused on their efficacy in an adjacent population: older adults.

Results are discussed in Chapter 4. In total, fifteen studies were included, summarising the data of 498 individual older adult participants, which assessed the efficacy of stimulus control and sleep restriction as stand-alone or combined interventions. All studies demonstrated improvements in subjective measures of sleep, which were maintained at 3-months post-treatment. However, evidence suggested that combined interventions brought about improvements of greater magnitude, and demonstrated improvements in outcomes relevant to the nosology of DSM-5 insomnia disorder [67] – sleep onset latency (SOL) and wake after sleep onset (WASO) – in as few as two therapeutic sessions. Notably, objective measures (e.g., polysomnography or actigraphy) were employed less frequently, and generally returned smaller, or no, effects.

Combined treatments also returned improvements in subjective measures of depression, but not anxiety. Isolated stimulus control did not appear to elicit any effect on mood, and no measures of mood were reported in studies employing sleep restriction as a monotherapy. The utility of CBT-I in alleviating symptoms of depression has been demonstrated previously [195, 199, 613, 756, 757], and concurring with prior research [755], current findings imply this may be driven primarily by sleep restriction. However, inconsistencies in measurement between studies, as well as risk of bias, limit what inferences can be drawn. Large scale comparative investigations of both interventions are necessary to elucidate this further.

6.1.4.2 Contribution to the literature

The findings of Chapter 4 expand understandings of insomnia treatment, suggesting that stimulus control and sleep restriction – particularly when combined – are suitable alternatives in populations for whom full CBT-I protocols may not be feasible or acceptable (e.g., stroke survivors).

Moreover, by stripping CBT-I to its constituent parts, this work may serve to inform investigations into personalised approaches to sleep medicine [25, 748, 749]. That the behavioural components of CBT-I remain effective when isolated from cognitive therapy can inform the development of interventions for insomnia which aim to circumvent the impediments to treatment common to stroke. Together with the findings of the previous chapters, this work moves us forward as a field, in better understanding how best to tackle the existing and growing issue of improving life after stroke; specifically, where it relates to sleep. Carrying forward the findings in Chapter 2, Chapter 4 establishes the point of departure for Chapter 5, where we outline plans for future research investigating stimulus control and sleep restriction, delivered in tandem and adapted to meet the needs of stroke survivors, as means of alleviating the symptoms of insomnia after stroke.

6.2 Thesis strengths and limitations

6.2.1 Thesis strengths

This body of work demonstrates several meritorious strengths, particularly with regards to its methodological rigour, innovative and novel contributions to the literature, and its clear potential for clinical applications.

Firstly, Chapters 3 and 4 were pre-registered, promoting transparency, integrity, and scientific rigour [758]. Relatedly, the consistent use of reporting guidelines across all chapters enhances methodological rigour and reproducibility, improving the thesis’ credibility and impact. Additionally, Chapter 3 included an *a priori* power analysis. Though a rarity in diagnostic validation studies [562], this is critical in ensuring we obtained a suitably powered sample, increasing the reliability of the findings, and confidence in our interpretations.

Moreover, the iterative nature of the thesis, with the results of each chapter informing those that followed, highlights a commitment to the principles of empiricism. Furthering this, methodological triangulation – incorporating interpretivist and positivist approaches – provides a multifaceted perspective towards insomnia after stroke. This mixed-methods approach is facilitatory in overcoming the limitations of singular approaches, enriching the analysis, and enhancing the overall depth and scope of the thesis [759]. Combining the precision and objectivity of quantitative methods with the richness and subjectivity of qualitative methods, this thesis bridges the historic divide between positivism and interpretivism, demonstrating that each approach can complement the other [413, 760, 761]. The use of IPA, in particular, enriches analysis by moving beyond a descriptive understanding, allowing researchers freedom to bring their own interpretations to the data, while remaining true to participants’ words [414, 436]. This methodological decision led to the revelation of a complex interplay between insomnia and well-being after stroke, which was instrumental in directing the research which followed and serves to enhance the development of future endeavours.

6.2.2 Thesis limitations

Despite the many strengths of this thesis, there are several limitations which should be addressed. Firstly, Chapter 2’s qualitative findings are based on a small, relatively homogenous sample, of self-selected stroke survivors. While small samples are typical in qualitative research, which does not necessarily seek to be generalisable in the same way positivist approaches do [762], the potential exclusion of underrepresented groups risks overlooking important nuanced perspectives. Evidencing this, the disparities identified in the experiences and preferences of participants with aphasia underscore the need for considering inclusivity in stroke research.

Relatedly, the quality of data generated from qualitative research is inherently reliant on a productive researcher-participant relationship, and thus necessarily will vary across cases. While each interview in Chapter 2 was conducted by the same researcher, removing the risk of between-researcher variation, technical difficulties arose in some interviews which impeded the natural flow of conversation, and potentially the richness of data. Similarly, it was apparent that the lead researcher shared a closer cultural and linguistic background with some participants more than others. This appeared beneficial in identifying linguistic

variations between topics, which may have advantaged the analysis of some transcripts over others. Ethnicity data was not collected, though future research may wish to explore cultural, clinical, or socio-economic influences on the experiences of life after stroke.

Due to contemporaneous public health restrictions due to Covid-19, all studies adopted online recruitment and data collection. Though this accelerated and geographically expanded recruitment, it largely limited participation to those willing and able to use video conferencing software (e.g., Zoom, Microsoft Teams). Efforts were made to address this, but nevertheless, data collection for Chapters 2 and 3 were predominantly via these channels. It therefore cannot be said that those unfamiliar or uncomfortable with using these platforms are represented in the data. Implementation of patient public involvement (PPI) groups did mitigate the impact that this had on subsequent chapters, however.

Finally, due to the absence of studies investigating stimulus control and sleep restriction after stroke, Chapter 4 investigated their efficacy in older adults. Due to shared challenges common to ageing and life after stroke (e.g., impairments to cognition and mobility) and associations between stroke-risk and age [763, 764], older adults were thought an appropriate proxy for the stroke population. Nevertheless, the veracity of this assumption remains to be tested.

6.3 Clinical implications and future directions

Across the body of work discussed throughout this thesis, various outcomes hold particular importance for clinical practice, or future avenues of research. Firstly, Chapter 2 illustrates the profound impacts of both nighttime and daytime symptoms of insomnia on the lives of stroke survivors. Pertinently, results highlight a complex series of bi-directional relationships between insomnia and cognitive, social, psychological, and physiological domains of well-being. These not only serve as potential targets for future interventions but serve also to make participants' accounts of the lack of support they received for their sleep all the more concerning. Discussions around shortcomings in current standards of clinical knowledge and approaches to sleep after stroke underscore the necessity of prioritising sleep as a clinical focus, particularly in acute stroke contexts. The findings of this thesis highlight that sleep disruption exists among the many post-stroke sequelae that are commonly overlooked, such as psychiatric distress and fatigue [765]. The

need for clinical guidelines for the assessment and management of insomnia – and indeed, sleep disruption more broadly – after stroke, is clearly indicated. To that end, we strongly advocate that clinicians and researchers seek to optimise sleep in acute stroke care – particularly where light exposure and noise are concerned [347, 375] – and assess sleep as part of routine care. Small changes, such as encouraging patients to turn off the lights, TV, and radio in their room or bed space later in the evening, using blinds to minimise disruption to via light, providing sleep masks or earplugs upon request, scheduling observations or medication rounds at times that minimise disruptions to sleep, or using quieter apparatus – such as soft closing bins or centralised buzzer systems – which may collectively enhance the experience of sleep in acute stroke settings. Moreover, investigation of the frequency that sleep after stroke is discussed or formally assessed would be beneficial in guiding clinical practice, and may inform efforts to explore sleep as a modifiable factor for reducing the risk of stroke recurrence [349, 350], or as a means of reducing the probability of mood disorders arising post-stroke.

Though ascertainment of insomnia’s aetiology after stroke was not an original objective of this thesis, increased understanding of it would likely benefit preventative and reactive efforts of management. Mohandas et al. [319]’s recent foray into predictors of sleep disturbance after stroke is an encouraging step towards elucidating this further. Expanding on this, longitudinal investigations of the role of hospital admission, comparative studies of physiological and cortical arousal, and studies triangulating correlational and lesion-based methods [395] to explore possible organic origins, would be particularly informative.

The recommendations borne out of Chapter 3 are directly applicable to clinical practice. However, while this thesis confirms the SCI’s validity as a screening measure, its usefulness as an outcome measure that is sensitive to change following interventions remains to be explored. Moreover, this thesis stands at the vanguard of online survey research in stroke survivors. By demonstrating this to be possible, Chapter 3 provides a model for validating self-report measures after stroke, which may extend to measures of sleep and beyond. The importance of recommending that the SCI’s diagnostic threshold be reduced to ≤ 13 should not be understated. Adoption of the conventional threshold in a sample of stroke survivors would likely lead to an inflated false positive rate, undermining confidence in research findings, and potentially leading to inappropriate diagnoses and use of resources in clinical contexts. In light of the absence of clinical guidelines on the assessment of sleep after stroke, we suggest that a systematic approach to screening for sleep disturbance should be integrated into existing post-stroke assessments and care pathways. Considering the immediacy with which sleep

impairment arises after stroke – discussed in Chapter 2 – screening should occur early during inpatient admission or rehabilitation. Brief screening measures such as the SCI or other validated measures of sleep can be administered by clinical (neuro)psychologists, medical, or nursing staff, as part of broader assessments. Given the dynamic nature of sleep throughout recovery after stroke [321], repeated assessments at early outpatient follow-up may be warranted. It is our hope that this easily actionable recommendation can facilitate the early detection and treatment of post-stroke insomnia. Future research should investigate not only the optimal means of intervention, but whether timely intervention corresponds with improved outcomes relating to one’s wellbeing and recovery after stroke. Relatedly, research from animal models suggests that sleep may be neuroprotective after stroke [359–361, 365]. Though experimental verification of this in humans is inherently unethical, investigation via observational methods is indicated. With the information at hand, we strongly recommend that sleep management be incorporated into rehabilitative protocols after stroke, and that the potential to improve stroke outcomes by optimising sleep be explored empirically. Whether treatment corresponds with improvements in the impacted domains outlined in Chapter 2 is also of interest.

With this in mind, the antepenultimate and penultimate chapters of this thesis seek to determine the optimal methods of addressing insomnia after stroke. The review discussed in Chapter 4 makes the case for explicitly behavioural interventions, stimulus control and sleep restriction. Not only does this suggest that these treatments offer efficacious alternatives where CBT-I may be unavailable, sub-optimal, or inappropriate, but also alternatives which be easier to disseminate, increasing accessibility to sleep medicine. Informed by the previous chapters, Chapter 5 outlines a proposal seeking to explore the feasibility, acceptability, and efficacy of these treatments after stroke. Clinical practice would benefit from not only the completion of this study, but from replicating the protocol in different subgroups of stroke survivors and expanding on it by developing similar approaches using additional designs. Additionally, exploration of alternative delivery methods (e.g., autonomous and/or digital delivery) would potentially be beneficial in addressing regional inequalities in access to sleep medicine [184].

6.4 Concluding remarks

This thesis aims to contribute towards the betterment of life after stroke for those living with insomnia. This body of work addresses several critical gaps in the existing literature concerning the impact, assessment, and treatment of insomnia after stroke. It is our hope that these efforts not only serve to further theoretical understandings of sleep after stroke, but also facilitates the development of improved clinical practice and outcomes, offering tangible benefits to people living with insomnia after their stroke.

Appendix A

Appendices

A Interview schedule

A.1 Experiences of sleep

1. How would you describe your sleep?

- (a) Can you tell me a bit more about this?
- (b) What is it like falling asleep?
- (c) Staying asleep?
- (d) Waking up?

2. Has your sleep changed since your stroke?

- (a) In what ways has your sleep been impacted?
- (b) Do you feel you sleep better or worse?
- (c) Has your sleep changed in duration?
- (d) Have you noticed any changes in how frequently you wake up?
- (e) How has the quality of your sleep changed?
- (f) Can you tell me a bit more about this? How has it changed overall?

3. Can you tell me what it is like to have a poor night's sleep?

- (a) Is there anything that impacts your sleep negatively?
- (b) What do you feel causes you to sleep poorly?
- (c) How often does this happen?
- (d) How does this affect you the next day?

4. **Can you tell me what it is like to have a good night's sleep?**
 - (a) What about this night makes it a good night's sleep?
 - (b) What is it like falling asleep and waking up?
5. **What do you think generally affects your sleep?**
 - (a) Is there anything you feel impacts your sleep the most?
 - (b) How does this effect you the next day?
 - (c) Do you feel that your health impacts your sleep? In what ways?
 - (d) Do you feel that your mental health impacts your sleep? In what ways?

A.2 Treatment views

1. Is there anything you have tried which helped you sleep better? This can include strategies that you use or treatments that you have been given.
 - (a) In what ways was this beneficial?
 - (b) Do you feel that it has resolved your sleep problem? In what ways?
 - (c) How did you find this technique?
 - (d) How do you feel about tablets/medication to help sleep?
 - (e) How do you feel about behavioural treatments?
 - (f) Zoom/phone/group/individual
2. Is there anything you have tried which did not help you sleep?
 - (a) What do you think wasn't helpful about it?
 - (b) Why do you think it didn't work for you?
3. Can you tell me how you would like your healthcare professional to improve your sleep?
 - (a) Do you feel you are given enough information about your sleep?
 - (b) Do you know where to access help from?
 - (c) Do you discuss your sleep with your doctor?

B Analysis Template

Emergent Themes/Experiential Statements		Transcript	Exploratory Coding/noting
	1 2 3 4 5 6 7 8 9 10 11 12		

C Aphasia Friendly Participant Information Sheet

Participant Information

What is the purpose of this research?

Overview

Who can take part?

Do I need to take part?

What do I have to do to take part?

What are the possible disadvantages and risks of taking part?

What are the possible benefits of taking part?

Will my taking part in this study be kept confidential?

What will happen to my data?

How long will my data be kept for?

What if I have questions about how my data will be handled?

Who is organising and funding this research?

Who has reviewed this research?

Contact for further information

Assessing the Diagnostic Validity of the Sleep Condition Indicator as a Diagnostic Tool for Insomnia After Stroke.

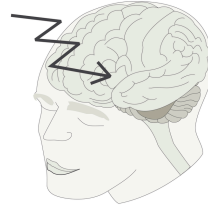
Mr Declan M. McLaren¹, Prof Jonathan Evans², Dr Satu Baylan², Dr Maria Gardani³

Participant Information

This is an easy-read version of the participant information sheet. Once you have read this document, please return to the previous page and navigate to the next part of the questionnaire using the arrow buttons. You do not need to re-read the information on the previous page.

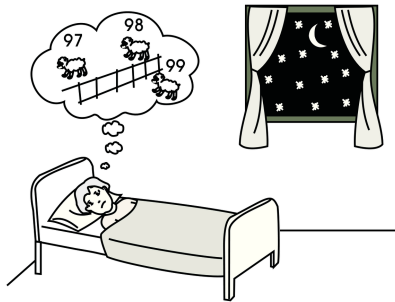
What is the purpose of this research?

This study aims to assess how well a questionnaire, the **Sleep Condition Indicator**, can identify **insomnia/sleep problems** in people after **stroke**.



Overview

Difficulties with sleep are common after a stroke.



The Sleep Condition Indicator is a questionnaire designed to assess sleep, but has never been tested in stroke survivors.



Who can take part?

You're invited to take part provided:

- You are aged **18 or older**



- You have experienced at least one **stroke**



- You can read, understand and write in English



- You are **not** currently working nightshift



- You are **not** currently jetlagged (disrupted sleep after travelling across more than two time zones in the last week).



- You are **not** currently undergoing treatment that may interfere with sleep, such as treatment for cancer.



Do I need to take part?

Whether or not you take part is completely **your decision**.

If you do wish to take part, please 'check' the appropriate box on the next page.

If you do take part, you are free to withdraw at any time, without giving reason, by not completing the survey. Should you choose to withdraw after starting the study, please note due to the study being anonymous, researchers cannot identify your data, and thus anything answers given before choosing to withdraw may be included in the study.

What do I have to do to take part?

Taking part involves **completing an online survey**. This survey gathers information about: - **Yourself** - Your **stroke** - Your **sleep and mental health** - Other **difficulties after your stroke**.

This will take around **20-25 minutes** to complete, but some may take less or more time.

You can take breaks, or you can return to the survey later if needed, but please use the same browser and do **not** clear your internet history.



We don't expect any of the questions should be upsetting or difficult, but you are welcome to seek support from a trusted friend or family member if required.

Additionally, if you'd like to take part but would prefer to do so in a different format, please contact Mr Declan M. McLaren (mailto:d.mclaren.2@research.gla.ac.uk?subject=SCI%20Validation%20Questionnaire%20Support)

What are the possible disadvantages and risks of taking part?

We don't anticipate any immediate risks or disadvantages to taking part. However, if you are not comfortable answering questions about your sleep, you may prefer not to take part.

What are the possible benefits of taking part?

There are no direct benefits to taking part, and you will not receive payment for participating.

The information you provide will help us improve how we diagnose sleep problems in people who have had a stroke.

We hope this will improve how quickly we can provide treatment to people having difficulty with sleep after stroke.

Will my taking part in this study be kept confidential?

All of the information you provide will be kept strictly confidential.

Participation is **completely anonymous**.

No information that could be used to identify you will be collected.

All electronic data will be stored securely on servers within the University of Glasgow.

No one outside the immediate research team or appropriate governance staff will have access.

What will happen to my data?

Any data collected will be anonymous and stored securely using OneDrive for Business.

Anonymous data will be deposited in the University of Glasgow's research data repository: Enlighten.

How long will my data be kept for?

In line with the University of Glasgow retention policy, data will be kept up to 10 years.

After this period, further retention may be agreed, or your data will be securely destroyed in accordance with the relevant standard procedures.

What if I have questions about how my data will be handled?

If you have any questions or comments about Research Data Management at Glasgow University, please email: research-datamanagement@gla.ac.uk (mailto:research-datamanagement@gla.ac.uk).

Findings from this study will be submitted for PhD thesis of the lead researcher and publication of the results will be in the form of academic papers, conference abstracts, and presentations, and will not contain any personal data or information that you may be identified with.

Who is organising and funding this research?

This research is being developed and conducted by the members of the research team (detailed above) at the **Universities of Glasgow & Edinburgh**, in collaboration with the Stroke Association, and is funded by the Scottish Graduate School of Social Science (ESRC and MRC).



Who has reviewed this research?

This research has ethical approval from the University of Glasgow, College of Science and Engineering Ethics Board.

Contact for further information

If you have any questions, please contact the lead researcher at

also be contacted in writing at:

Researchers can

Thank you for taking the time to read this participant information sheet. You may now **return to the previous window** and navigate to the next part of the questionnaire using the arrow button at the bottom of the page. Like the example below.



After reading this document, you do not need to read the information on the previous page.

To pursue any complaint about the conduct of the research: contact the College of Science Ethics Officer, Dr Julie Williamson, email: Julie.Williamson@glasgow.ac.uk (../Supplementary/Julie.Williamson@glasgow.ac.uk)

-
1. University Of Glasgow - School of Psychology↵
 2. University of Glasgow - Institute of Health and Wellbeing↵
 3. University of Edinburgh - School of Health in Social Science↵

D Sleep History Questionnaire

For the following questions, please give your answers as what has been the case for the past **two weeks**:

Sleep Routine

1. Is your opportunity to sleep affected by any external factors, such as caring responsibilities, noise, light, or pets?
 - (a) Yes
 - (b) No
 - i. (If Yes): Please provide details as to what is affecting your sleep
2. At what time do you usually get into bed? (Please answer to the closest 30 minutes)
3. At what time do you usually get out of bed in the morning? (Please answer to the closest 30 minutes)
4. How long after going to bed do you usually turn out the lights? (Please answer to the closest 5 minutes)
5. How long does it take you to fall asleep after you turn the lights off? (Please answer to the closest 5 minutes)
6. How many times do you wake up in a typical night?
 - (i) (If 6. $\neq$ 0): After waking up in the night, what is the total amount of time you typically spend awake?
For example, if you wake up 3 times and are awake for 10 minutes each time, answer '30 minutes'.
7. Do you usually feel refreshed after a typical night's sleep?
 - (a) Definitely not
 - (b) Probably not
 - (c) May or may not
 - (d) Probably yes
 - (e) Definitely yes

8. Do you nap during the day?

- (a) Never
- (b) Once a week
- (c) 2-3 times a week
- (d) 4-6 times a week
- (e) Every day
- (i) (If $\neq$ Never):
 - i. How long does a typical nap last? (Please answer to the closest 5 minutes)
 - ii. Do you usually feel refreshed after napping?
 - A. Definitely not
 - B. Probably not
 - C. May or may not
 - D. Probably yes
 - E. Definitely yes

9. How much time do you spend in your bedroom for activities other than sleep or sex?

- (a) None at all
- (b) A little
- (c) A moderate amount
- (d) A lot
- (e) A great deal

Sleep Status

10. Do you currently have problems with your sleep?

- (a) No
- (b) Yes
 - i. (If Yes): Please describe briefly the problems you are having with your sleep
 - ii. How often are you affected by your sleep problem?
 - A. Never

- B. Once a week
 - C. 2-3 times a week
 - D. 4-6 times a week
 - E. Every night
- iii. When did your sleep problem(s) start?
- A. Within the last two weeks
 - B. Within the last month
 - C. Within the last 3 months
 - D. Within the last 6 months
 - E. Within the last 12 months
 - F. More than a year ago
- iv. Were there any stressful events related to the onset of your sleep problem (e.g., divorce, exams, death of a loved one, retirement, medical or emotional problems)?
- A. No
 - B. Yes
- (i) (If yes:) Please give some more details of the event/circumstances.
11. Has your sleep changed since your most recent stroke?
- (a) No
 - (b) Yes
- i. (If Yes): Please describe briefly how your sleep has changed since your most recent stroke.

Sleep History

12. Have you had sleep problems in the past?
- (a) No
 - (b) Yes
- i. (If Yes:) When was this?
- ii. Please describe the past problem.
- iii. How long did these sleep problems last?

- A. 0–2 weeks
- B. 2–4 weeks
- C. 4–6 weeks
- D. 6 weeks–3 months
- E. 3–6 months
- F. 6 months–1 year
- G. More than a year

iv. Did this impact you throughout the day?

- A. No
- B. Yes
- C. (If Yes): Please describe the effect this had on you throughout the day.

13. Have you received a diagnosis of a sleep disorder from a medical professional? (e.g., Insomnia, Obstructive Sleep Apnoea, Narcolepsy, etc).

(a) No

(b) Yes

i. (If Yes): What sleep problem was diagnosed?

Insomnia Disorder

14. Do you find yourself worrying/ruminating before you go to sleep?

(a) No

(b) Yes

i. (If Yes): Please describe the nature of your worries.

15. Do you watch the clock and worry as to when you'll fall asleep?

(a) No

(b) Yes

16. Do you worry about the impact of not getting enough sleep?

(a) No

(b) Yes

i. (If Yes): How often does this cause you worry?

A. Never

B. Once a week

C. 2-3 times a week

D. 4-6 times a week

E. Daily

ii. On a scale of 1 to 10 (with 1 being least severe and 10 being most severe), how severe would you rate these worries as being?

iii. How long have you been experiencing worry about the impact of not getting enough sleep?

A. Less than 2 weeks.

B. 2-4 weeks.

C. 4-6 weeks.

D. 6 weeks to 3 months.

E. 3-6 months.

F. 6 months to a year.

G. More than a year.

Hypersomnia

17. Do you feel sleepy during the day?

(a) No

(b) Yes

i. (If Yes): Do you think you are sleepier than other people your age?

A. No

B. Yes

- ii. Do you have difficulty staying awake during the day?
 - A. No
 - B. Yes
- iii. Do you feel the need to take naps during the day?
 - A. No
 - B. Yes
- iv. Do you sleep for more than 9 hours during the night?
 - A. No
 - B. Yes
- v. Do you find it difficult to wake up in the morning?
 - A. No
 - B. Yes
 - C. (If Yes): How many times per week do you find it difficult waking up in the morning?
 - D. On a scale of 1 to 10 (with 1 being least severe and 10 being most severe), how severe would you rate the difficulty getting up in the morning as being?
 - E. On a scale of 1 to 10 (with 1 being least severe and 10 being most severe), how severe would you rate the difficulty getting up in the morning as being?

Narcolepsy

- 18. Do you find yourself falling asleep in situations where you need to be awake?
 - (a) Yes
 - (b) No
- 19. Do you experience sleep attacks (falling asleep suddenly and without warning)?
 - (a) Yes

(b) No

20. Do you experience sleep paralysis (a feeling of being awake but unable to move)?

(a) Yes

(b) No

21. Do you experience instances of imagining very realistic objects or events, usually visually, right before falling asleep (hypnagogic hallucinations)?

(a) Yes

(b) No

22. Do you experience sudden bouts of muscle weakness when awake (catalepsy), sometimes brought on by strong emotions or laughter?

(a) Yes

(b) No

i. (If Yes): How often do you experience sudden bouts of muscle weakness?

A. Less than once a month

B. Once a month

C. 2-3 times a week

D. 4-6 times a week

E. Daily

ii. How long have you been experiencing sudden bouts of muscle weakness brought on by strong emotions or laughter?

A. Less than 2 weeks

B. 2-4 weeks

C. 4-6 weeks

D. 6 weeks to 3 months

E. 3 -6 months

F. 6 months to a year

G. More than a year

Sleep Apnoea

23. Do you snore?

(a) No

(b) Yes

i. (If Yes:) Do you snore loudly?

ii. Do you snore irregularly?

24. Does your bed partner say that you snore?

(a) No

(b) Yes

(c) NA

25. Do you wake up with a sore head and/or dry mouth?

(a) No

(b) Yes

26. Do you (or your bed partner) notice that you sometimes stop breathing during your sleep?

(a) No

(b) Yes

i. (If Yes): On a scale of 1-10, how severe would you describe your stopping breathing during your sleep as being?

ii. How long have you been experiencing stopping breathing whilst asleep?

A. Less than two weeks

B. 2-4 weeks

C. 4-6 weeks

D. 6 weeks to 3 months

E. 3-6 months

F. 6-12 months

G. More than a year

PLMD

27. Do you experience leg twitches or jerks throughout the night?

(a) No

(b) Yes

- i. (If Yes): On a scale of 1-10, how severe would you describe these leg twitches/jerks as being?
- ii. How long have you been experiencing leg twitches and/or jerks throughout the night?
 - A. Less than two weeks
 - B. 2-4 weeks
 - C. 4-6 weeks
 - D. 6 weeks to 3 months
 - E. 3-6 months
 - F. 6-12 months
 - G. More than a year

RLS

28. Do you experience crawling or aching feelings in your legs, and an inability to keep legs still, whilst in bed?

(a) No

(b) Yes

- i. (If Yes): On a scale of 1-10, how severe would you rate these aching feelings or inability to keep your legs still as being?
- ii. How long have you been experiencing aching feelings or inability to keep your legs still whilst in bed?
 - A. Less than two weeks

- B. 2-4 weeks
- C. 4-6 weeks
- D. 6 weeks to 3 months
- E. 3-6 months
- F. 6-12 months
- G. More than a year

Parasomnias

Please indicate whether you experience any of the following, you may need to ask your bed partner if applicable. Do you experience nightmares (frightening or disturbing dreams that may wake you suddenly)?

29. (a) No
- (b) Yes
- i. (If Yes): On a scale of 1-10, how severe would you rate these nightmares as being?
 - ii. How long have you been experiencing nightmares?
 - A. Less than two weeks
 - B. 2-4 weeks
 - C. 4-6 weeks
 - D. 6 weeks to 3 months
 - E. 3-6 months
 - F. 6-12 months
 - G. More than a year
30. Do you experience night terrors (sudden bouts of fear, screaming, and flailing whilst asleep; from which you don't wake up and may be unlikely to remember)?
- (a) No

(b) Yes

i. (If Yes): On a scale of 1-10, how severe do you rate these night terrors as being?

ii. How long have you been experiencing night terrors?

A. Less than two weeks

B. 2-4 weeks

C. 4-6 weeks

D. 6 weeks to 3 months

E. 3-6 months

F. 6-12 months

G. More than a year

31. Do you every sleep-walk?

(a) No

(b) Yes

i. (If Yes): How often do you sleep walk?

A. Less than once a week

B. Once a week

C. 2-3 times a week

D. 4-6 times a week

E. Every night

ii. How long have you been sleep-walking for?

A. Less than 2 weeks

B. 2-4 weeks

C. 4-6 weeks

D. 6 weeks to 3 months

E. 6 months to a year

F. More than a year

32. Do you ever talk in your sleep?

(a) No

(b) Yes

i. (If Yes): How often do you talk in your sleep?

A. Less than once a week

B. Once a week

C. 2-3 times a week

D. 4-6 times a week

E. Every night

ii. For how long have you talked in your sleep?

A. Less than 2 weeks

B. 2-4 weeks

C. 4-6 weeks

D. 6 weeks to 3 months

E. 6 months to a year

F. More than a year

Circadian rhythm disorders

33. Would you consider yourself more of a morning or an evening person?

(a) Morning

(b) Evening

34. Around what time of day do you begin to feel sleepy (Please answer to the closest 30 minutes)?

35. If you were free to choose, what time of day would you naturally wake up (Please answer to the closest 30 minutes)?
36. If you were free to choose, what time of day would you go to bed (Please answer to the closest 30 minutes)?
37. If you do not go to bed or get up at your preferred times, do you find it difficult to function the next day?
- (a) No
 - (b) Yes
 - i. (If Yes): On a scale of 1-10, how difficult would you find it to function the following day if you didn't go to bed or get up at your preferred times?
 - ii. How often do you notice your daily functioning affected by not going to be, or waking up, at your preferred times?
 - A. Less than once a week.
 - B. Once a week.
 - C. 2-3 times per week.
 - D. 4-6 times per week.
 - E. Daily.

Consequences

38. Does your current sleep pattern affect your daily functioning?
- (a) No
 - (b) Yes
 - i. (If Yes): Please explain how your current sleep pattern affects your daily functioning?

E Confusion matrix of index vs reference test classifications.

Table A.1: Confusion matrix of index vs reference test classifications.¹

SCI Classification (≤ 13 cut-off)	Sleep history questionnaire classification		
		Negative	Positive
	Negative	75	9
	Positive	30	66

¹*Note:* The above matrix displays the classifications made via the clinical interview styled questionnaire, including detailed sleep history, versus classifications made via the Sleep Condition Indicator (SCI), using a diagnostic threshold of 13.

F Sensitivity, specificity, and Youden's J statistic for all thresholds on the SCI

Table A.2: Sensitivity, specificity, and Youden's J statistic for select thresholds on the SCI when detecting DSM-5 insomnia disorder and insomnia symptoms ²

Insomnia Disorder				Insomnia Symptoms			
Threshold	Specificity (%)	Sensitivity (%)	Youden's J	Threshold	Specificity (%)	Sensitivity (%)	Youden's J
0	100.00	1.33	0.01	0	100.00	0.93	0.01
1	100.00	6.67	0.07	1	100.00	4.63	0.05
2	99.05	9.33	0.08	2	100.00	7.41	0.07
3	99.05	14.67	0.14	3	100.00	11.11	0.11
4	98.10	22.67	0.21	4	100.00	17.59	0.18
5	96.19	28.00	0.24	5	98.61	22.22	0.21
6	94.29	37.33	0.32	6	97.22	29.63	0.27
7	92.38	42.67	0.35	7	95.83	34.26	0.30
8	89.52	52.00	0.42	8	91.67	40.74	0.32
9	85.71	57.33	0.43	9	88.89	46.30	0.35
10	81.90	70.67	0.53	10	86.11	57.41	0.44
11	80.95	73.33	0.54	11	86.11	60.19	0.46
12	78.10	78.67	0.57	12	83.33	64.81	0.48
13	71.43	88.00	0.59	13	80.56	75.93	0.56
14	64.76	90.67	0.55	14	76.39	81.48	0.58
15	60.00	90.67	0.51	15	73.61	84.26	0.58
16	54.29	96.00	0.50	16	66.67	88.89	0.56
17	48.57	98.67	0.47	17	59.72	91.67	0.51
18	42.86	98.67	0.42	18	55.56	94.44	0.50
19	40.00	98.67	0.39	19	51.39	94.44	0.46
20	33.33	98.67	0.32	20	45.83	97.22	0.43
21	29.52	98.67	0.28	21	40.28	97.22	0.38
22	24.76	100.00	0.25	22	34.72	99.07	0.34
23	18.10	100.00	0.18	23	25.00	99.07	0.24
24	15.24	100.00	0.15	24	20.83	99.07	0.20
25	15.24	100.00	0.15	25	20.83	99.07	0.20
26	10.48	100.00	0.10	26	15.28	100.00	0.15
27	9.52	100.00	0.10	27	13.89	100.00	0.14
28	8.57	100.00	0.09	28	12.50	100.00	0.12
29	5.71	100.00	0.06	29	8.33	100.00	0.08
30	3.81	100.00	0.04	30	5.56	100.00	0.06
31	0.00	100.00	0.00	31	0.00	100.00	0.00
32	0.00	100.00	0.00	32	0.00	100.00	0.00

²Optimal cut-off in each group is identified by bold text. Optimal cut-offs were determined via Youden's J ($J = \text{sensitivity} + \text{specificity} - 1$).

G Sensitivity, specificity, and Youden' J statistic for all thresholds on the SCI-2

Table A.3: Sensitivity, specificity, and Youden's J statistic for select thresholds on the SCI when detecting DSM-5 insomnia disorder and insomnia symptoms ³

Insomnia Disorder				Insomnia Symptoms			
Threshold	Specificity (%)	Sensitivity (%)	Youden's J	Threshold	Specificity (%)	Sensitivity (%)	Youden's J
0	93.33	32.00	0.25	0	95.83	25.93	0.22
1	80.95	52.00	0.33	1	86.11	45.37	0.31
2	68.57	76.00	0.45	2	79.17	69.44	0.49
3	56.19	86.67	0.43	3	68.06	81.48	0.50
4	42.86	96.00	0.39	4	54.17	91.67	0.46
5	35.24	98.67	0.34	5	48.61	97.22	0.46
6	20.95	100.00	0.21	6	29.17	99.07	0.28
7	9.52	100.00	0.10	7	13.89	100.00	0.14
8	0.00	100.00	0.00	8	0.00	100.00	0.00

³Optimal cut-off in each group is identified by bold text. Optimal cut-offs were determined via Youden's J ($J = \text{sensitivity} + \text{specificity} - 1$).

H Estimates of predictive values of the SCI when detecting DSM-5 insomnia disorder at various hypothetical prevalences

Table A.4: Estimates of predictive values of the SCI when detecting DSM-5 insomnia disorder at various hypothetical prevalences⁴

Prevalence	PPV	NPV
0.05	0.139	0.991
0.10	0.255	0.982
0.15	0.352	0.971
0.20	0.435	0.960
0.25	0.507	0.947
0.30	0.569	0.933
0.35	0.624	0.917
0.40	0.672	0.899
0.45	0.716	0.879
0.50	0.755	0.856
0.55	0.790	0.830
0.60	0.822	0.799
0.65	0.851	0.762
0.70	0.878	0.718
0.75	0.902	0.665
0.80	0.925	0.598
0.85	0.946	0.512
0.90	0.965	0.398
0.95	0.983	0.239

The positive and negative predictive values at the prevalence observed in the study (41.67%) were 0.688 and 0.893, respectively. Predictive values were calculated using the ‘MKmisc’ R package [766]. This package applies Bayes’ function to determine the positive and negative predictive values of a test, when given the sensitivity, specificity, and prevalence (actual or hypothetical). Bayes formula is useful for dealing with uncertainty, and can be used to determine conditional probability, including in the context of diagnostic assessment. See Webb & Sidebotham [767] for a comprehensive explanation.

⁴PPV, positive predictive value; NPV, negative predictive value.

Historically, sensitivity and specificity were considered independent from prevalence, while predictive values were known to be dependent upon prevalence [767]. However, recent meta-analytic evidence casts doubt on the former [559]. Given that sensitivity and specificity are parameters of the equations to compute positive and negative predictive values [767], one should consider the values in the above table for what they are: hypothetical estimates. And take heed of the possibility that the actual population values for sensitivity and specificity at varying prevalences may vary from those derived from the present study.

I Search strategy for Chapter 4

Ovid MEDLINE® Epub Ahead of Print, InProcess & Other Non-Indexed Citations, Ovid MEDLINE® Daily, Ovid MEDLINE and Versions®

1. exp "Sleep Initiation and Maintenance Disorders"/
2. insomnia*.tw.
3. 1 and 2
4. exp Sleep Deprivation/
5. (sleep adj3 (restrict* or depriv*)).tw.
6. (stimulus adj3 control*).tw.
7. exp Behavior Therapy/
8. (Behavio* treatment or Behavio* Therapy or Behavio* Intervention or BBTi or BBT-i).tw.
9. 4 or 5 or 6 or 7 or 8
10. exp Aged/
11. (elder* or (old* adj2 adult*) or geriatric).tw.
12. Geriatrics/
13. exp "Aged, 80 and over"/
14. 10 or 11 or 12 or 13
15. 3 and 9 and 14

Key

- 'exp' before an index/MeSH term indicates that the term was exploded.
- '*' at the end of a term indicates that this term has been truncated
- 'tw.' indicates a search for keywords in title and abstracts

The search strategy will be adapted, including associated MeSH and index terms, for use in other electronic databases (EMBASE, CINAHL, PsycINFO) as appropriate.

Figure A.1: Search strategy for Ovid Medline electronic database

J The Evidence Project risk-of-bias tool

Table A.5: The Evidence Project Risk of Bias Appraisal Tool Template

Risk of Bias Domain	Item	Description	Response
Study design	Cohort	Cohort analyses present data for a group of study participants followed over time. This may include pre-intervention to post-intervention analyses with or without a control or comparison group. If the study includes a cohort that was followed over time and included multiple assessments with the same people, this criterion is met. If the study did not conduct multiple assessments with a cohort of individuals over time, this criterion is not met. For example, a study that used a serial cross-sectional design with different individuals (even if they are from the same population) completing the assessments would not be considering as having a cohort design.	NA
Continued on next page			

Risk of Bias Domain	Item	Description	Response
	Control or comparison group	Control or comparison groups are defined as analyses that compare those who received the intervention to those who did not. They may also include those who received a more-intensive versus less-intensive intervention. These include analyses that compare intervention, control and/or comparison groups, and cross-sectional analyses that are stratified by whether participants did or did not receive the intervention. This item does not include before-after analyses without stratification. If the study included a control and/or comparison arm in addition to the intervention arm, this criterion is met. If the study only had an intervention arm, this criterion is not met.	NA
	Pre/post intervention data	Pre-post intervention outcome data is included in the risk of bias assessment, as it is common for studies to only assess outcome measures in the post-intervention catchments, especially for post hoc analyses and secondary study aims. Pre-post intervention data is present when the study presents outcome data for participants both before and after they receive the intervention. Such data may be presented at multiple time points either before or after the intervention. If the study presents data from both before (baseline) and after the intervention, this criterion is met. If data are only presented post-intervention, this criterion is not met. If data are only presented pre-intervention, the study would not meet the inclusion criteria of having post-intervention evaluation data.	NA
<i>Continued on next page</i>			

Risk of Bias Domain	Item	Description	Response
Participant representativeness	Random assignment of participants to the intervention	Random assignment to treatment groups assesses whether subjects were randomly assigned to treatment groups in multi-arm studies and includes group randomized designs. This criterion is nested within criterion for a control or comparison group in order to give added weight to designs which include randomization and control. In multi-arm study designs, if participants are randomly assigned to the intervention and control/comparison arm, this criterion is met. This is true for both individual and group randomized designs. If participants self-select into the intervention or if assignment to the intervention is not random, this criterion is not met. If the study only has an intervention arm, this criterion should be listed as not applicable.	NA
<i>Continued on next page</i>			

Risk of Bias Domain	Item	Description	Response
	Random selection of participants for assessment	Random selection of subjects for assessments is assessed to consider whether there was a selection bias in study enrolment. If authors use a probability sample to select participants (defined as a study in which the investigators pre-assess a sampling frame and randomly select groups or people from the specified population), this criterion is met. Similarly, if authors use a mixed sampling strategy but conducted random sampling for at least one part of that mixed strategy (for example, they have a non-probability sample of schools but then within schools randomly select students), then we consider this criterion as met because they randomly selected participants for assessment at some level, i.e., at one sampling frame. If authors used a non-probability sample (defined as a study in which the investigators use convenience or self-selected sampling strategies), then this criterion is not met.	NA
<i>Continued on next page</i>			

Risk of Bias Domain	Item	Description	Response
	Follow-up rate of 80% or more	Attrition of participants is measured at the final study follow-up. This is related to incomplete reporting, or loss-to-follow-up, that may introduce bias if participants who are retained are different than those who are not retained. One rule of thumb suggests that <5% loss leads to little bias, while >20% poses serious threats to validity [34]. This criterion is measured across the entire study population (all study arms). If the entire study group had a follow-up rate of 80% or more, this criterion is met. If the follow-up rate was less than 80% at the final assessment, this criterion is not met. For studies that are post-intervention only or serial cross-sectional in nature, this criterion should be listed as not applicable.	NA
Continued on next page			

Risk of Bias Domain	Item	Description	Response
Equivalence of comparison groups	Comparison groups equivalent on sociodemographics	Comparison group sociodemographic matching is assessed in multi-arm studies to determine if there are statistically significant differences in sociodemographic measures across arms at baseline. Sociodemographic measures may include characteristics such as age or gender, but should not include outcome measures. Study arms include intervention, control, or comparison groups. If the study arms are equivalent on sociodemographic characteristics, this criterion is met. If there are significant differences between one or more of the study arms on socio-demographic characteristics, this criterion is not met. If the study only has one study arm, this criterion should be listed as not applicable. If the study has multiple study arms but the authors do not report whether the arms were equivalent on sociodemographic characteristics, this criterion should be listed as not reported.	NA
Continued on next page			

Risk of Bias Domain	Item	Description	Response
	Comparison groups equivalent at baseline on outcome measures	Comparison group outcome matching is assessed in multi-arm studies to establish whether there were statistically significant baseline differences in study outcome measures. As above, study arms include intervention, control, or comparison groups. Outcome measures are those which the intervention is trying to change; they generally include things like knowledge, attitudes, behaviours, or biological outcomes. There may be one or more outcome measures in any given study. If the study arms are equivalent on outcome measures at baseline, this criterion is met. If there are statistically significant differences between one or more of the study arms on outcome measures at baseline, this criterion is not met. If the study only has one study arm, this criterion should be listed as not applicable. If the study has multiple study arms but the authors do not report whether the arms were equivalent on outcome measures at baseline, this criterion should be listed as not reported.	NA

K Ethical Approval Letter for Study 1



Ethical approval for:

Application Number: 200220039

Project Title: Experiences of Sleep Disturbances and Desired Treatment Preferences Post-Stroke

Lead Researcher: Dr Maria Gardani

This is to confirm that the College of Science and Engineering Ethics Committee has reviewed the above application and **approved** it. Please keep this letter for your records.

Please note that if your proposal involves **face-to-face research**, approval to carry out this research is only granted when one of the following two conditions has been met:

(a) You have performed a risk assessment of your research protocol in your research facility, had it approved by your Head of School / Director of Institute, and received permission to proceed with this specific research project, or

(b) The University has generally lifted its social distancing restrictions on face-to-face interaction, including research.

If any of the above is true, or if your research collects data in a format that does not require social contact (e.g., online research), you may begin data collection now. Approval for this project lasts for 6 months from the date you are allowed to proceed with data collection.

Also please download and read the Collated Comments associated with your proposal. This document contains all the reviews of your application and can be found below the approval letter on the Research Ethics System. These reviews may contain useful suggestions and observations about your research protocol for improving it. Good luck with your research.

Sincerely,

Dr Christoph Scheepers
Ethics Officer
College of MVLS
University of Glasgow

L Ethical Approval Letter for Study 2



Ethical approval for:

Glasgow, January 30, 2022

Application Number: 300210126

Project Title: Assessing the Diagnostic Validity of the Sleep Condition Indicator as a Diagnostic Tool for Insomnia After Stroke

Lead Researcher: Dr Maria Gardani

This is to confirm that the College of Science and Engineering Ethics Committee has reviewed the above application and **approved** it. Please keep this letter for your records.

Please note that if your proposal involves **face-to-face research**, approval to carry out this research is only granted when one of the following two conditions has been met:

(a) You have performed a risk assessment of your research protocol in your research facility, had it approved by your Head of School / Director of Institute, and received permission to proceed with this specific research project, or

(b) The University has generally lifted its social distancing restrictions on face-to-face interaction, including research.

If any of the above is true, or if your research collects data in a format that does not require social contact (e.g., online research), you may begin data collection now. Approval for this project lasts for 6 months from the date you are allowed to proceed with data collection.

Also please download and read the Collated Comments associated with your proposal. This document contains all the reviews of your application and can be found below the approval letter on the Research Ethics System. These reviews may contain useful suggestions and observations about your research protocol for improving it. Good luck with your research.

Sincerely,

Dr Christoph Scheepers
Ethics Officer
College of Science and Engineering
University of Glasgow

M Ethical Approval Letter for Study Protocol



29thh February 2024

Dear Prof Evans,

MVLS College Ethics Committee

Project Title: Brief Behavioural Treatment for Insomnia in Stroke Survivors: A Single Case Experimental Design
Project No: 200230214

The College Ethics Committee has reviewed your application and has agreed that there is no objection on ethical grounds to the proposed study. It is happy therefore to approve the project, subject to the following conditions:

- Project end date: End December 2024
- The data should be held securely for a period of ten years after the completion of the research project, or for longer if specified by the research funder or sponsor, in accordance with the University's Code of Good Practice in Research: https://www.gla.ac.uk/media/media_490311_en.pdf
- The research should be carried out only on the sites, and/or with the groups defined in the application.
- Any proposed changes in the protocol should be submitted for reassessment, except when it is necessary to change the protocol to eliminate hazard to the subjects or where the change involves only the administrative aspects of the project. The Ethics Committee should be informed of any such changes.
- You should submit a short end of study report to the Ethics Committee within 3 months of completion.
- For projects requiring the use of an online questionnaire, the University has an Online Surveys account for research. To request access, see the University's application procedure at <https://www.gla.ac.uk/research/strategy/ourpolicies/useofonlinesurveystoolforresearch/>.

Yours sincerely

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