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REVIEW ARTICLE

Mechanisms of cognitive behavioural therapy for insomnia

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Summary

Although much is known now about behavioural, cognitive and physiological consequences of insomnia, little is known about changes after cognitive behavioural therapy for insomnia on these particular factors. We here report baseline findings on each of these factors in insomnia, after which we address findings on their changes after cognitive behavioural therapy. Sleep restriction remains the strongest determinant of insomnia treatment success. Cognitive interventions addressing dysfunctional beliefs and attitudes about sleep, sleep-related selective attention, worry and rumination further drive effectiveness of cognitive behavioural therapy for insomnia. Future studies should focus on physiological changes after cognitive behavioural therapy for insomnia, such as changes in hyperarousal and brain activity, as literature on these changes is sparse. We introduce a detailed clinical research agenda on how to address this topic.

KEYWORDS
behavioural, cognitive, cognitive behavioural therapy for insomnia, functional magnetic resonance imaging, insomnia, physiological

1 | INTRODUCTION

Cognitive behavioural therapy for insomnia (CBT-I) is the recommended treatment for insomnia, and is more effective than medication alone, particularly on the long term (Edinger et al., 2001a; Edinger et al., 2001b; Edinger et al., 2021; Morin et al., 2009). Treatment adherence rates are estimated at approximately 60% for in-person CBT-I (Morin, 2006) and 50% for digital CBT-I (Horsch et al., 2015). The treatment typically includes sleep restriction, through which sleep pressure is increased and the positive association of the bed with sleeping restored. It also includes advice on using the bed for sleep and sex only, and informs patients about the influence of alcohol, bright light and late-night heavy meals on sleep. It can include relaxation techniques and, importantly, address dysfunctional thoughts and ideas about sleep while also treating repetitive thoughts, worries and rumination (Edinger et al., 2001a; Edinger et al., 2001b; Edinger et al., 2021; Morin et al., 2009). The main predictors of success for CBT-I have more recently been identified as being able to implement sleep restriction and other CBT-I requirements in daily life routines, but chronotype can also play a role (Faaland et al., 2022; Scott et al., 2022). Though many studies investigated adherence to treatment in insomnia, Mellor et al. conclude that the definition of adherence varies too much between CBT-I studies to define factors predicting adherence (Mellor et al., 2022). A predictor of treatment success on the long term is a reduction of scores on the Insomnia Severity Index (ISI), while other factors such as those assessing dysfunctional beliefs about sleep have weaker strength as long-term predictors of treatment success (Blom et al., 2021).

Given the large need for CBT-I and its limited accessibility, group therapy and digital forms of therapy have been shown to have similar

treatment efficacy as the classic in-person, one-to-one CBT. These new modes of CBT-I delivery are beneficial for those living more remotely and, for instance during pandemic-related restricted mobility, have more restricted access to healthcare (Cheng et al., 2021; Dekker et al., 2015; Hartley et al., 2022; Lancee et al., 2016; Leerssen et al., 2021; van Straten et al., 2009).

Insomnia is frequently associated with psychiatric comorbidity, in particular anxiety and depressive disorders (Freeman et al., 2020), with bidirectional relationships between chronic insomnia and mood disorders, including depression, generalised anxiety disorder, panic disorder, specific phobias and social anxiety (Breslau et al., 1996; Hertenstein et al., 2019; LeBlanc et al., 2009; Pigeon et al., 2017). There is also strong evidence that insomnia is a significant predictor of depression (Hertenstein et al., 2019; Pigeon et al., 2017), and it could be a likely predictor of anxiety disorders, bipolar disorder and suicide (Pigeon et al., 2017). Some authors recommend, in the event of proven comorbidity, to treat both insomnia and mental disorders simultaneously (Hertenstein et al., 2019). As insomnia is also highly comorbid with sleep apnea (Sweetman, Lack, Catcheside, et al., 2019), with the occurrence of both simultaneously being associated with a higher risk of mortality (Sweetman et al., 2022), CBT-I has been shown to be affective for both treating insomnia symptoms and the acceptance and observance of continuous positive airway pressure treatment (Sweetman, Lack, & Bastien, 2019).

It is particularly important to determine what is meant by "treatment" in the context of CBT-I, as this may have differing meanings between individuals with insomnia and the scientific and clinical communities (Araújo et al., 2017). For example, it has recently been shown that the reporting of a sleep complaint can be influenced by sociodemographic factors, such as years of education (Meadows et al., 2022). Most CBT-I treatment studies use changes in either global measures of sleep/insomnia, such as the ISI (Morin et al., 2011), or its main symptoms (e.g. sleep latency, wake after sleep onset [WASO], etc.) to determine treatment outcomes. Whilst these measures are reliable and valid indicators of change, how meaningful for the patient/participant are they? Although limited research has been undertaken in this area, qualitative research (Dyas et al., 2010) suggests that daytime impairment is the key complaint for many individuals with insomnia. As such, we should be mindful when examining the efficacy of any intervention, and the potential mechanisms underpinning it: it may be more pertinent to determine what is most relevant to the individual in terms of treatment outcome (Kjørstad et al., 2022). Insight into the mechanisms of CBT-I, and which factors of this multifactor treatment contribute most to its effectiveness, can help adapt treatment to individual needs. This can be done by investigating mediators, which can statistically explain the relationship between a dependent and independent variable (Kraemer et al., 2002; Nock, 2007), and has been useful to explain intervention effects in many different conditions, including insomnia (Schwartz & Carney, 2012; Sunnhed & Jansson-Fröjmark, 2015).

We will here report findings on insomnia mechanisms and how they are represented in those CBT-I mediators and factors contributing to treatment effectiveness. Insomnia mechanisms have been described referring to behavioural, cognitive and physiological factors, such as brain activity differences assessed through neuroimaging techniques.

2 | BEHAVIOURAL FACTORS OF INSOMNIA AND CBT-I

In a healthy sleep–wake cycle, prolonged wakefulness naturally leads to a build-up of the sleep drive, which then determines night-time sleep quantity and quality through increased sleep pressure (Borbély, 1982). An imbalance between sleep-inducing and wake-inducing mechanisms can lead to insomnia (Saper et al., 2005), which is often caused by sleep disruptive behaviour. Extended daytime napping and spending excessive time in bed, for instance, can disrupt homeostatic regulation and cause an enhancement of sleep difficulties (Schwartz & Carney, 2012). Other examples of sleep disruptive behaviour are maintaining irregular bedtimes, consuming heavy meals at nighttime, consuming alcohol, coffee or caffeine-containing soft drinks before going to bed or taking sleep disruptive drugs or medication (ecstacy, cocaine; Harvey et al., 2017) and remaining low levels of physical activity due to fatigue (Hartescu et al., 2015).

A method applied often as part of CBT-I interventions is sleep restriction. Based on actual sleep time estimates from the sleep diary and possibly actimetry, a time window is chosen during which the patient is allowed to stay in bed. Independent of whether they spent this time asleep, they are required to get up at the end of this time window (e.g. from 23:30 hours to 06:00 hours). No other sleep opportunities are allowed until the next sleep window. Sleep pressure increases as a consequence, which further increases the homeostatic sleep drive (Borbély, 1982). If this method is followed consistently, the time window will eventually be spent asleep, heightening sleep efficiency, reducing waketime, and improving sleep quantity and sleep quality. If sleep efficiency is high, 15–30 min can be added to the sleep window, until that period is also spent asleep, etc. It can be linked to Barlow's model (Barlow et al., 2016), which postulates that emotional avoidance is a central process in many psychopathological disorders. By using behavioural techniques such as restriction of time in bed, for example, patients are brought to confront what they fear, namely the fear of not sleeping (or of not sleeping enough) and the fear of the consequences from lack of sleep. Patients thus confront the emotions they dread and usually avoid by spending a lot of time in bed. This exhibition confronts them with their emotions and breaks the vicious circle of avoidance that perpetuates insomnia.

Effects of sleep restriction as part of CBT-I are, for instance, noticeable in a more rapid decline in electroencephalogram (EEG) delta power over the night, which is related to the therapeutic effect of CBT and to an increase in the homeostatic sleep drive (Krystal & Edinger, 2010). Parallel to this, as delta activity and sleep spindles density hold an inverse relationship (De Gennaro & Ferrara, 2003; Dijk et al., 1993), it has been shown that a lower spindle density after CBT-I is positively linked to lower sleep efficiency (Dang-Vu et al., 2017). Although this may not reflect sleep drive induced by

sleep restriction, it might still indicate that some EEG elements might be deficient before treatment is initiated, whichever mechanisms underlying a component of CBT-I these may be.

Next to ensuring that time in bed is spent asleep, sleep restriction also reverses the negative association patients with insomnia typically have of the bed with not-sleeping. A time window spent successfully asleep will motivate the patient to go to bed the next night and restore a positive relation of being in bed with sleeping. Sleep restriction can improve sleep continuity and reduce insomnia severity (Maurer, Ftouni, et al., 2020). Although some studies suggest that just stabilising bedtimes can also reduce bedtime variability and reduce insomnia severity (Sunnhed & Jansson-Fröjmark, 2015; Vestergaard et al., 2021), sleep restriction has been suggested to be superior to time in bed regularisation in improving objective and self-reported sleep variables and sleep-related quality of life (Maurer, Espie, et al., 2020). The 3-R model, by Maurer et al. (2018), proposed that restricting sleep leads to cognitive-behavioural reconditioning (such as a more positive bed–sleep association), and to physiological regularisation, such as improving the circadian sleep–wake cycle (Maurer et al., 2018). Furthermore, adding stimulus control to sleep restriction appears beneficial to decrease the well-known night-to-night variability in sleep difficulties typical of patients with insomnia (Sánchez-Ortuño et al., 2011; Vallières et al., 2005). In that regard, it has been shown that these two CBT-I components appear to act together to stabilise variability, and that the decrease in variability is paralleled by a decrease in depressive symptoms and insomnia severity (Suh et al., 2012).

3 | COGNITIVE AND EMOTIONAL PROCESSING FACTORS OF INSOMNIA AND CBT-I

Altered daytime functioning is part of the clinical diagnostic criteria for insomnia according to DSM-5, the most widely-accepted system of nosology for psychiatric disorders (*American Psychiatric Association diagnostic and statistical manual of mental disorders*, 2013), and according to ICSD (International Classification of Sleep Disorders, 3rd edn; Sateia, 2014). Insomnia-induced changes in daytime functioning can typically include memory problems, problems with multitasking and concentration. Several studies and meta-analyses have focused on verifying these subjective complaints with objectively impaired cognitive functioning in insomnia, including memory and working memory tasks as well as assessment of impairment in other executive functions than working memory (Backhaus et al., 2006; Bastien et al., 2003; Brownlow et al., 2020; Fernandez-Mendoza et al., 2010; Fortier-Brochu et al., 2012; Fortier-Brochu & Morin, 2014; Fulda & Schulz, 2001). Overall, effects are not consistent and subtle at best, with one meta-analysis describing more pronounced memory problems (Fortier-Brochu & Morin, 2014), while other find subtle effects on executive functioning (Altena, Ramautar, et al., 2010; Ballesio, Aquino, et al., 2019; Ferreira & de Almondes, 2014). Inconsistencies in insomnia diagnostic criteria and inconsistencies in the choice of cognitive tasks have been identified as factors that may reduce effect sizes examined in these studies.

According to the "cognitive model of insomnia" of Harvey (2002), cognitive aspects of insomnia include increased worry and rumination about both sleep-related and non-sleep-related issues, and overattributing insomnia consequences, such as the impact of sleep disturbance, on daytime functioning (Ballot et al., 2021). While the wake-to-sleep transition should normally occur naturally and automatically, the malfunctioning of this transition can lead to a dysfunctional tendency to consciously address this natural process by increasing attention and intention to sleep. This idea is represented in the "attention–intention–effort model" (A-I-E) of Espie et al. (2006), a component of Espie's (2002) broader psychobiological inhibition framework. The selective attention, explicit intention and efforts to sleep then disrupt the natural and automatic sleep process, which should then be restored by reducing these conscious efforts (Espie et al., 2006; Schwartz & Carney, 2012).

These cognitive models are supported by a limited number of behavioural and neuroimaging studies. Selective attention to sleep-related information and sleep-related interpretative biases have indeed been found to be increased in insomnia (Akram et al., 2017; Akram et al., 2016; Harvey & Greenall, 2003; Morin et al., 2002; Spiegelhalder et al., 2008; Woods et al., 2009), but this might depend on stimulus type presented (e.g. pictures instead of words; Spiegelhalder et al., 2018). When performing a cognitive task in functional magnetic resonance imaging (fMRI) or functional near-infrared spectroscopy (fNIRS), hypoactivity in those brain regions usually implicated in the task has been found, in particular in tasks typically activating prefrontal brain regions, requiring cognitive flexibility, planning or working memory (Altena et al., 2008; Drummond et al., 2013; Stoffers et al., 2014; Sun et al., 2017). Increased functional connectivity in a hippocampal–prefrontal network typically active during rumination, but silent during sleep, has been found to be related to insomnia severity (Leerssen et al., 2019).

Patients with insomnia show attentional bias when presented with sleep-related emotional stimuli (Barclay & Ellis, 2013; MacMahon et al., 2006; Lundh et al., 1997; for review, see Harris et al., 2015). Brain regions typically active when emotional stimuli are presented, such as the amygdala (Baglioni et al., 2014) and the brain reward network (Sanz-Arigita et al., 2021), show increased activity and connectivity in patients with insomnia compared with controls. These findings may, though based on a limited number of studies, offer neural support for increases of selective attention of sleep-related information, in the context of the opposite pattern of hypoactivity in prefrontal brain regions when presented with cognitive tasks.

Effective treatment can be achieved by cognitive restructuring, which focuses on increasing the patient's awareness of the ineffectiveness of dramatising thoughts and dysfunctional beliefs about lack of sleep and its consequences but also, importantly, on their aggravating influence on insomnia. The patient is then encouraged with the therapist to consider alternative thoughts about a lack of sleep and its possible consequences. Through psychoeducation, the therapist also informs the patient of scientific knowledge concerning the limited

impact of the consequences of lack of sleep. The patient is then asked to test the credibility of these alternative thoughts, for example by accepting the idea of sleeping less, possibly concomitant with sleep restriction. The resulting reduction of these conscious effects to sleep then favours the transition to the more natural and automatic sleep process.

Of those cognitive aspects associated with insomnia, changes in beliefs and attitudes about sleep as well as locus of control have been shown to be key aspects linked to effective CBT-I, and to diminished insomnia complaints as well as improvements in objective sleep (Chow et al., 2018; Edinger et al., 2001a; Thakral et al., 2020). In fact, a recent systematic review of mediators following CBT-I, with two-stage structural equation modelling, demonstrated that of several potential mediators only dysfunctional beliefs significantly mediated treatment outcome (Parsons et al., 2021). Because emotion regulation functions are typically not optimal during the night and less so during sleep, introducing a rumination moment during the day, as part of CBT-I interventions, can prevent night-time rumination (Tempesta et al., 2018). Decreased rumination was found to be a mediator in the beneficial effects of CBT-I on insomnia in one study (Ebert et al., 2015), while reducing both insomnia and depression symptoms in another study (Cheng et al., 2020). The more worrying, rumination and unhelpful beliefs about sleep are reduced, the more effective CBT-I is, reflected in reduced insomnia severity and improved daytime functioning (Sunnhed & Jansson-Fröjmark, 2015). Another study showed that particularly sleep-related worry and rumination were mediators for improved sleep efficiency, but not dysfunctional beliefs about sleep (Lancee et al., 2019).

Few neuroimaging studies focused on the effects of CBT-I in worry, rumination or selective attention, and those who did often have not included a well-defined control group or control condition. Conclusions based on these studies should be carefully proposed: many other explanatory factors such as time effects, habituation effects could play a role next to effects of CBT-I itself. Cortical hyperactivity in response to sleep-related sounds, indicative of selective attention to sleep-related stimuli, decreased after CBT-I in an fMRI study with 14 participants by Kim et al. (2019). Hyper-reactivity to other sleep-related stimuli also decreased after CBT-I in another study, which was related to less WASO (Kim et al., 2017). The same researchers showed that in the absence of performance or brain activity changes after CBT-I on the Stroop task, an inhibition task, decreased scores on the ISI were related to activity changes in the supramarginal gyrus, normally involved in Stroop performance (Hwang et al., 2019).

In a study that used a waitlist group as control before and after treatment, a typical pattern of hypoactivity in prefrontal regions during task performance has been shown to partially normalise after effective CBT (Altena et al., 2008). The same research group found that hypoactivity in task-related brain regions on a planning and working memory task, however, did not change after effective CBT-I (Stoffers et al., 2014). Effects of CBT-I might thus be brain region dependent, with more outspoken effects on the prefrontal cortex, although these findings remain to be replicated.

Very few studies investigated changes in brain structure after CBT. Changes in cortical thickness have been found in particularly the left orbitofrontal cortex after CBT-I as compared with a control group in insomnia comorbid with fibromyalgia (McCrae et al., 2018). Reductions of grey matter volume of the left orbitofrontal cortex in patients with insomnia compared with controls has previously been reported (Altena, Vrenken, et al., 2010; Stoffers et al., 2012; Yu et al., 2018), warranting more focused studies on this brain region, known for its role in emotional decision making and impulse regulation (Rudebeck & Rich, 2018), and how it relates to aspects of insomnia and CBT.

In summary, these results thus might indicate that increased selective attention for sleep-related stimuli decreases after CBT-I, while dysfunctional beliefs and attitudes about sleep, worry and rumination reduction are other effective cognitive elements of CBT-I. Brain activity related to cognitive tasks possibly normalises after CBT-I, but many of the neuroimaging results on changes of cognitive activity after CBT-I await replication and comparison with a well-defined control group.

4 | PHYSIOLOGICAL FACTORS OF INSOMNIA AND OF CBT-I

A characteristic symptom of insomnia is hyperarousal, which is a constantly increased level of arousal, independent of the presence of external stimuli (Bonnet & Arand, 1995, 1998; Perlis et al., 1997; Riemann et al., 2010), and may occur throughout the 24-hr cycle (Pérusse et al., 2013). Hyperarousal is typically divided in somatic hyperarousal and cortical hyperarousal (Perlis et al., 2001; Pigeon & Perlis, 2006; Cortoos et al., 2006). Somatic hyperarousal refers to increased heart rate, altered galvanic skin response and heart rate variability (Bonnet & Arand, 2010; Borbély, 1982; Lushington et al., 2000; Monroe, 1967; Perlis et al., 2005; Riemann et al., 2002; Vgontzas et al., 1998). Cortical arousal refers to increased evoked response potentials in the morning and evening (Bastien et al., 2008), and at the peri-onset of sleep, higher frequency EEG activity (Bastien et al., 2013; Fernandez-Mendoza et al., 2016; Turcotte et al., 2011) and increased EEG power (theta, gamma: Zhao et al., 2021).

During sleep, increased EEG power has been observed during rapid eye movement (REM) sleep (alpha, sigma) and non-rapid eye movement (NREM) sleep (theta, alpha, sigma). Sensory and information processing as well as long-term memory formation are thought to continue due to cortical hyperarousal, while they should slow down or be stopped to facilitate sleep onset. In fact, both somatic and cognitive hyperarousal can prevent wake to sleep transitioning (Bonnet & Arand, 2010) and are directly linked to sleep discontinuity (Perlis et al., 2017). Sleep fragmentation typically occurs during REM sleep in insomnia (Riemann et al., 2012), and patients with insomnia report having woken up more often than controls when woken from REM sleep, suggesting higher consciousness levels during REM sleep (Feige et al., 2018). To add to this body of empirical data, it has been shown that cerebral asymmetry is present in patients with insomnia compared with control, and that it also correlates to depressive and

anxious symptoms (St-Jean et al., 2012) or insomnia severity (Provencher et al., 2020). The hyperarousal concept of insomnia has been recently debated, however, particularly its occurrence in the absence of stress-provoking stimuli as well as its specificity for insomnia alone and not co-morbid disorders of insomnia (Bastien, 2020; Kalmbach et al., 2018; Vargas et al., 2020).

Next to EEG findings of enhanced states of arousal and vigilance (Losert et al., 2020; Oh et al., 2020), other physiological findings support the hyperarousal models of insomnia. fMRI findings point at insomnia-related hyperarousal and increased vigilance, in particular by changes in the insula network (Chen et al., 2014; for an overview, see also Kay & Buysse, 2017). Metabolism levels in several cortical regions are higher during sleep in insomnia, while prefrontal metabolism is in fact lower during wake, suggestive of an imbalance of brain activity during both sleep and wake in insomnia (Nofzinger et al., 2004; Van Someren, 2021). Altered thalamic connectivity in insomnia during both wake and NREM sleep further supports physiological hyperarousal models of insomnia, given the role of the thalamus in sleep–wake regulation (Zou et al., 2021). Elevated levels of the stress hormone cortisol in insomnia provide support of elevated somatic hyperarousal (Vargas et al., 2018), while levels of γ-aminobutyric acid (GABA), an inhibitory neurotransmitter with typical high levels during sleep, tend to be lower in patients with insomnia as compared with controls (Morgan et al., 2012; Winkelman et al., 2008).

Particular treatment components of CBT-I, such as applying relaxation techniques, aim to reduce hyperarousal. Evidence of reduced hyperarousal as a CBT-I mechanism is however limited. Decreased hyperarousal has been found as a result of effective CBT-I when measured by the Presleep State Arousal Scale (Rosen et al., 2000; Vincent & Lewycky, 2009; Wu et al., 2006). More specifically, Vincent and Lewycky (2009) found that reduced pre-sleep cognitive hyperarousal mediated the effect of computerised CBT on self-reported sleep data, as compared with a control condition that consisted of improving sleep schedule consistency only. Sleep restriction, a key element of CBT-I, had an effect on somatic hyperarousal in a study by Kalmbach et al. (2019), but somatic hyperarousal was more strongly affected by administration of complete CBT. More broadly, one study identified reductions in stress and anxiety as significant mediators of treatment success following CBT-I (Ubara et al., 2022). CBT-I has also been shown to decrease body temperature (Miller et al., 2015), although changes in other physiological measures such as heart rate variability (Jarrin et al., 2016) and cortisol (Miller et al., 2015) were in a contrary direction to those expected for reduced hyperarousal. Vallières et al. (2013) showed that following the first night of sleep restriction, decreases in beta-1 and -2 powers were observed, an indication that hyperarousal was also decreased during treatment.

Few neuroimaging studies however focus on hyperarousal-reducing effects of CBT-I. Most neuroimaging meta-analyses state that conclusions about pathophysiological brain mechanisms of insomnia are hampered by the large variability in diagnosis criteria, differences in magnetic resonance imaging (MRI) and task setups, analyses types and statistical thresholds applied, as well as the limited sample size of most studies (O'Byrne et al., 2014; Spiegelhalder et al., 2013; Spiegelhalder et al., 2015; Tahmasian et al., 2018). The lack of a well-defined control group or control condition (placebo, waitlist) is another weakness of several neuroimaging studies investigating CBT-I effects. Pursuing high-quality imaging studies based on consensus about diagnosis and imaging set-up is thus highly important, so meta-analyses are based on more studies, such as has been done in, for instance, depression (Goodkind et al., 2015). Further, it is vital to combine imaging data from large freely accessible databases to arrive at conclusions about insomnia mechanisms and intervention effects, such as is done in other psychiatric conditions (Kempton et al., 2011; Nichols et al., 2017).

During sleep, increased regional cerebral blood flow in the basal ganglia was observed after CBT-I intervention by Smith et al. (2004), as measured by SPECT in an exploratory study of four patients with insomnia without a control group. Functional connectivity changes in the thalamus, amygdala, caudate nucleus and orbitofrontal cortex after CBT-I were reported by Lee et al. (2017) in 14 patients with insomnia. Here again, however, no control group was included, while connectivity changes did not correspond to baseline differences between patients and controls. Increased connectivity between the default-mode network and the premotor/dorsolateral prefrontal cortex, correlating with sleep improvements after CBT-I, was found by Park et al. (2020) in 35 dialysis patients with insomnia, but this study also lacked a control group.

Taken together, some hyperarousal studies indicate an effect of CBT-I, such as on pre-sleep cognitive hyperarousal, body temperature changes, and reduced beta 1 and beta 2 power in EEG. Functional neuroimaging findings during daytime and nighttime resting state and task presentation showing changes after CBT however tend to point more at a partial restoration of the wake–sleep disbalance of psychophysiological processes than at hyperarousal reductions per se (Kay & Buysse, 2017). Furthermore, given the wide range of physiological measures that show indications of hyperarousal in insomnia as compared with those without insomnia before treatment, it is surprising that only few studies have focused on hyperarousal changes after CBT-I.

5 | DISCUSSION

The limited literature on mechanisms of CBT for insomnia and mediator effects indicates that changes in behavioural and cognitive factors, such as changing dysfunctional beliefs and attitudes about sleep, diminishing selective attention and reducing worry and rumination are stronger predictors of CBT-I effectiveness than other factors, such as hyperarousal. While sleep restriction is the strongest factor leading to diminished insomnia severity after treatment, particularly on the long term (Blom et al., 2015), cognitive interventions addressing dysfunctional beliefs and attitudes about sleep, worry, rumination and selective attention for sleep-related stimuli thus remain vital interventions that should be part of CBT-I interventions. Future studies should focus on increasing the limited knowledge about physiological mechanisms underlying CBT-I effects.

Given the importance of cognitive strategies as a mediator of CBT-I, next to dysfunctional beliefs and attitudes about sleep, it is important to focus on worries and rumination. Indeed, as worries and rumination are very active in insomniacs, and given the frequent psychiatric comorbidity, we can assume that the patient's concerns will relate to invasive themes or problems specific to her/him, and not related solely to sleep: related to their life history, personal situation, personality (Ellis et al., 2010). It is therefore important to carry out the cognitive restructuring by targeting beyond the sleep-related dysfunctional beliefs and by targeting, in addition, more general dysfunctional beliefs such as proposed by the treatment of early maladaptive schemas (EMS) by Young et al. (2003). Indeed, EMS are defined as pervasive themes for the individual concerning oneself, interindividual relationships or with the environment. They act as deep cognitive structures of the personality, rigidly and repetitively influencing perception and behaviour, leading to rumination, dysfunctional behaviour and emotional distress. Proposing questionnaires such as developed by Young (2005) to assess these schemas could be proposed in a therapeutic approach for those patients with insomnia presenting with rumination and dysfunctional beliefs about sleep. Treating such schemas is advisable as they may address different cognitive and emotion regulation aspects of insomnia at once (Young et al., 2003), particularly in groups at increased risk of psychiatric comorbidity. For instance, rumination has been shown to be related to impaired cognitive daytime function in insomnia, warranting its treatment (Ballesio, Aquino, et al., 2019; Ballesio, Ottaviani, & Lombardo, 2019). In future studies, effective CBT-I could thus result in lower scores on Young's Questionnaire maladaptive schema questionnaire.

Well-controlled studies focusing on changes after CBT in those physiological features typical of insomnia, such as differences in heart rates, cortisol levels and brain activity, would be of interest. Those effects of insomnia observable from group comparisons with healthy controls, such as hyperarousal before sleep and during sleep onset, local brain hypoactivity when performing cognitive tasks or local brain hyperactivity when presented with emotional stimuli, could be expected to (partially) normalise after successful insomnia treatment through CBT. Although studies investigating CBT-I effects using neuroimaging methods have often lacked a proper control group, some of the published findings, if replicated, may give insight into psychophysiological mechanisms underlying CBT-I.

Another area of research that has been overlooked so far is to document hyperarousal while subjects are awake at night. So far, sleep studies have mainly focused on hyperarousal at the peri-onset of sleep or during sleep. Considering that one of the main components of CBT-I is to restrain time in bed, it is to be noted that even if time awake after sleep onset decreases after treatment, some wake time is still often present during the night or during early awakening in the morning after CBT-I. So far, the mechanism from which hyperarousal might differ during awake time during the night before and after treatment remains unknown. Targeting wake time during the night during treatment might inform us of the state (level) of hyperarousal during that time and further enlighten us on treatment mechanisms.

Of course, lack of adherence has been identified as one of the most important reasons why CBT-I does not work instead of the mechanisms at work underlying each component. Patients may have difficulty believing that recommendations could provide a better sleep quality, and this can be shown either through a lack of motivation and/or confidence in integrating prescriptions/rules, or expressing that keeping status quo is reassuring because changing behaviours might be discomforting. One must also understand that keeping track of adherence is not an easy task. Still, measuring time out of bed during treatment could be a start or, also, it might be wise to adapt our language to our "clientele", as suggested by Muench et al. (2022). Of note that those individuals not responding to CBT-I might also be individuals for whom stimulus control, especially getting out of bed if not able to fall asleep, is not a prescription they can adhere to. In fact, what is really needed is to establish temporal stability and cognitive distraction. It has been shown that those individuals using counter-control instead of getting out of bed also spend less time awake during the night (Davies et al., 1986). CBT-I providers might thus rethink

TABLE 1 Research agenda to improve investigations into mechanisms of CBT-I

Topic of future research	Specific focus of topic
Daytime hyperousal changes after CBT-I	• Heartrate changes • Changes in cortisol levels • Brain activity changes during task or stimuli presentation • Brain activity changes during resting state (EEG, fMRI, NIRS)
Nighttime wake hyperarousal changes after CBT-I	• Heartrate changes • Changes in cortisol levels • Body temperature changes • Brain activity changes during resting state (EEG, fMRI, NIRS)
Changes in maladaptive cognitive schemas	• Changes in dysfunctional beliefs about sleep • Changes in rumination • Changes in cognitive task performance • Changes in individual-specific maladaptive schemas • Changes in general dysfunctional beliefs about role of self with environment
Adherence investigation	• Consistently define adherence in CBT-I • Investigate individual reasons and personal factors predicting non-adherence (motivation, resistance) • Investigate personalised means to improve adherence
Deconstruction studies	• Determine which components are related to which mechanisms, with a view to tailoring

Abbreviations: CBT-I, cognitive behavioural therapy for insomnia; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; NIRS, near-infrared spectroscopy.

the way they administer CBT-I to increase adherence and not take stimulus control rules at face-value. Bending the rule can sometimes be very fruitful. It is only in the context of comparing CBT-I adherents with non-adherents that we can also partly disentangle the underlying pathophysiological mechanisms of insomnia, and especially hyperarousal. Albeit measured through a decreased cognitive activity that correlates with cortical activation, decreased heart rate variability or less activity in the ARAS, at the end of the day, hyperarousal is and will remain one of the core components of insomnia. Related, more deconstruction studies are needed in order to determine "what works and for whom." In other words, are there some components more highly related to mechanistic or mediational outcomes, compared with others, and what is their relative importance to perceived treatment outcomes. Only then will we be able to refine and tailor CBT-I for vulnerable groups and circumstances (Table 1).

Finally, it is of interest to note that future studies will be based on more similar criteria when the latest diagnostic insomnia of DSM-6 and ICSD are applied, because they are more homogeneous, there is better comparability between studies. What is interesting is to note that so far some studies show that objective changes on hyperarousal after CBT-I intervention are obtained by CBT-I techniques that do not specifically target this aspect (e.g. relaxation) but by behavioural techniques (sleep restriction) and, above all, by complete CBT. This is in line with what is known of psychotherapeutic interventions which, when they produce improvements, have effects on annexed and broader psychological processes that were not directly targeted. These effects of interactions between different behavioural, cognitive and affective components, such as described in Lang's tripartite model of fear (Lang, 1968), have been widely studied in CBT for other conditions (Philippot, 2011). The value of taking them into consideration has been highlighted as they can provide useful information on the underlying therapeutic processes involved in the efficacy of treatments (Hartley et al., 2022; Turpin, 1991).

DATA AVAILABILITY STATEMENT

Data sharing not applicable - no new data generated.

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