Stockholm University

Cognitive therapy and behavioral therapy for insomnia disorder

efficacy, moderators and mediators

Rikard Sunnhed

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Abstract

Insomnia disorder is the second most prevalent mental disorder and the most prevalent sleep disorder. Cognitive Behavioral Therapy for Insomnia (CBT-I) is considered the treatment of choice with well-documented effects. Nevertheless, a significant proportion of patients fail to respond, and an even larger proportion fail to remit from the condition. In addition, very little is known about the effects of CBT-I's separate components or about what moderates and mediates their effect. Gaining knowledge about components, predictors, and mediators could be one route for optimizing and tailoring CBT-I and ultimately enhancing outcomes.

The overall aim of this thesis was to advance our theoretical and clinical knowledge about CBT-I by exploring Cognitive Therapy (CT) and Behavior Therapy's (BT) comparative efficacy and their potential moderators and mediators.

To pursue the study aims, one large randomized controlled trial was performed that involved 219 individuals with insomnia disorder randomized to CT, BT, or a waitlist control group. Study 1 examined CT and BT's comparative efficacy against a waitlist control on a broad range of outcomes. Study 2 examined theoretically derived constructs from both therapy models, and insomnia-associated correlates as potential predictors and moderators of outcome for the two therapies. Study 3 examined theoretically driven process variables from the cognitive model as mediators of outcome in both CT and BT.

Study I showed that both therapies outperformed the waitlist and turned out as comparably effective treatments on the majority of outcomes. BT was associated with significantly more adverse events, whereas CT received significantly more minutes of telephone support.

Study II showed that early morning waketime and bedtime variability moderated the effect of both CT and BT. Those experiencing lower early morning waketime and bedtime variability achieved greater insomnia severity reductions in CT. In contrast, those experiencing greater early morning waketime and bedtime variability achieved larger insomnia severity reductions in BT. The findings also showed that greater insomnia severity, waketime after sleep onset, and lower sleep efficiency at baseline predicted greater insomnia severity at posttreatment.

Study III provided evidence that reductions in dysfunctional beliefs and monitoring for sleep during treatment acted as drivers of the reduction in insomnia severity in CT. The results also indicated that reductions in safety behaviors and dysfunctional beliefs mediated reductions in insomnia severity in BT, although not as clear as the drivers of change for CT since they were also reciprocally predicted by reductions in insomnia severity.

Study I indicate that CT and BT achieve similar effects and that both therapies are effective as standalone therapies for insomnia disorder. Study II provided evidence that the two therapies in CBT-I can depend on different patient characteristics at baseline to be effective. The results from study II thus suggest that the therapies in CBT-I could be tailored based on patient's characteristics before treatment to optimize outcomes. Study III provided support for the role of cognitive processes as important routes to remediate insomnia and underscore the value of assessing and targeting dysfunctional beliefs, monitoring, and safety behaviors to achieve reductions in insomnia severity and emphasize the importance of these concepts in understanding insomnia.

Keywords: *Behavior Therapy, Cognitive Therapy, Insomnia, internet-delivered, efficacy, mediators, moderators, personalized medicine.*

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Contents

Abbreviations

BT	Behavioral Therapy
CBT-I	Cognitive Behavioral Therapy for Insomnia
CT	Cognitive Therapy
DSM-5	Diagnostic and statistical manual of mental disorders. 5th ed.
DSM-IV	Diagnostic and statistical manual of mental disorders. 4th ed.
ISI	Insomnia Severity Index
OR	Odds ratio

Abstract

Background
Insomnia disorder is the second most prevalent mental disorder (Wittchen et al., 2011) and the most prevalent sleep disorder (Morin & Benca, 2012). Cognitive Behavioral Therapy for Insomnia (CBT-I) is considered the treatment of choice for insomnia with well-documented effects. Although CBT-I is deemed to be an efficacious treatment, a significant proportion of patients fail to respond, and an even larger proportion fail to remit from the condition. In addition, very little is known about the effects of the separate components of the CBT-I package, or about what moderates and mediates their effect. Gaining knowledge about components, predictors, and mediators could be one way to identify areas for future optimization and tailoring of CBT-I components to insomnia subgroups, ultimately enhancing outcomes.

Study aims
The overall aim of this thesis was to advance our theoretical and clinical knowledge about CBT-I by exploring its main therapeutic models—Cognitive Therapy (CT) and Behavior Therapy (BT)—their comparative efficacy, what moderates their effect, as well as what mediates their effect.

Studies
To pursue the study aims, one large randomized controlled trial was performed that involved 219 individuals with insomnia disorder randomized to CT, BT, or a waitlist control. From this trial, three studies were derived. Study 1 examined the comparative efficacy of CT and BT against a waitlist control group on a broad range of insomnia-related outcomes. Study 2 examined theoretically derived constructs from both therapy models and insomnia-associated correlates as potential predictors and moderators of outcome for the two therapies. Study 3 examined theoretically driven process variables from the cognitive model as mediators of outcome in both CT and BT.

Results
The overall result from Study I was that both therapies outperformed the waitlist and turned out as comparably effective treatments for insomnia disorder on the majority of outcomes. BT was associated with significantly

more adverse events, whereas CT received significantly more minutes of telephone support.

Study II showed that early morning waketime and bedtime variability moderated the effect of both CT and BT. The results showed that those experiencing lower early morning waketime and bedtime variability achieved greater insomnia severity reductions in CT. In contrast, those experiencing greater early morning waketime and bedtime variability achieved larger insomnia severity reductions in BT. The findings also showed that greater insomnia severity and waketime after sleep onset at baseline predicted greater insomnia severity at posttreatment, and greater sleep efficiency predicted lower insomnia severity at posttreatment.

Study III provided evidence that reductions in dysfunctional beliefs and monitoring for sleep during treatment acted as drivers of the reduction in insomnia severity in CT. The results also indicated that reductions in safety behaviors and dysfunctional beliefs mediated reductions in insomnia severity in BT, although these were not such clear drivers of change as the finding for CT since they were also reciprocally predicted by reductions in insomnia severity.

Conclusions

The outcomes from Study I indicate that CT and BT achieve similar effects and that both CT and BT are effective as standalone therapies for insomnia disorder. Study II provided evidence that the two therapies in CBT-I can depend on different patient characteristics at baseline to be effective. This could suggest that BT may be more relevant when problems related to the night are more pronounced and CT when nighttime issues are less pronounced. The results from study II thus suggest that the therapies in CBT-I could be tailored based on the patient's characteristics before treatment to optimize outcomes. Study III provided support for the role of cognitive processes as important routes to remediate insomnia and underscore the value of assessing and targeting dysfunctional beliefs, monitoring, and safety behaviors to achieve reductions in insomnia severity, as well as emphasize the importance of these concepts in understanding insomnia.

Sammanfattning

Bakgrund

Insomni är den näst vanligaste psykiatriska störningen (Wittchen et al., 2011) och den vanligaste sömnstörningen (Morin & Benca, 2012). Kognitiv beteendeterapi för insomni (KBT-I) har väldokumenterade effekter och anses vara förstavals behandlingen för insomni. Även om KBT-I anses vara en effektiv behandling, så når fortfarande en betydande andel av patienterna inte en tillfredställande respons och en ännu större andel misslyckas med att bli fria från sina besvär. Dessutom är mycket lite känt om effekterna av de separata komponenterna i KBT-I-paketet, eller om vad som modererar eller medierar deras effekt. Att erhålla kunskap om komponenter, moderatorer och mediatorer kan vara ett sätt att identifiera relevanta områden för att optimera och anpassa komponenterna i KBT-I till specifika undergrupper av insomni, något som i slutändan kan förbättrar resultaten och hjälpa fler med insomni.

Studiens syfte

Det övergripande syftet med denna avhandling var att främja den teoretiska och kliniska kunskapen om KBT-I genom att utforska dess huvudsakliga terapeutiska modeller - kognitiv terapi (KT) och beteendeterapi (BT) - deras jämförbara effekt, vad som modererar, samt vad som medierar deras effekt.

Studier

För att realisera syftet utfördes en stor randomiserad kontrollerad studie som involverade 219 individer med insomni som randomiserades till CT, BT eller en väntelista. Från denna kliniska prövning härleddes tre studier. Studie 1 undersökte den jämförbara effekten av CT och BT gentemot en väntelista på ett brett spektrum av insomni relaterade utfall. Studie 2 undersökte teoretiskt härledda konstrukt från båda terapimodellerna och insomni relaterade konstrukt som potentiella prediktorer och moderatorer för utfallet av de två terapierna. Studie 3 undersökte teoretiska processvariabler från den kognitiva modellen som mediatorer för utfallet i både CT och BT.

Resultat

Det övergripande resultatet från studie I var att båda terapierna överträffade väntelistan och visade sig vara jämförbart effektiva behandlingar för insomni

på majoriteten av utfallen. BT var förknippat med betydligt fler negativa bieffekter, medan KT fick betydligt fler minuter av telefonstöd.

Studie II visade att mängden vakentid på morgonen och variationen i tiden för sänggående modererade effekten av både KT och BT. Resultaten visade att de som upplevde mindre ofrivillig vakentid på morgonen och hade mindre variation i tiden för när de gick och lade sig i sängen uppnådde större förbättringar av sin insomni i KT. Å andra sidan uppnådde de som hade större besvär med ofrivillig vakentid på morgonen och mer variation i tiden för sänggående större förbättringar av sin insomni i BT. Resultaten visade också att större grad av insomni och mer nattlig vakentid efter insomning innan behandlingen predicerade större grad av insomni efter behandlingen, samt att högre sömneffektivitet före behandling predicerade lägre grad av insomni efter behandling.

Studie III visade att en minskning av dysfunktionella antaganden och monitorering av sömn av sömnrelaterade hot under behandlingen medierade förbättring av insomni i KT. Resultaten visade också att minskade säkerhetsbeteenden och dysfunktionella antaganden under behandlingen medierade förbättring av insomni i BT, även om dessa inte var lika tydliga mediatorer som dem i KT eftersom förbättringar i dessa mediatorer också ömsesidigt predicerades av minskningar i insomni.

Slutsatser

Resultaten från studie I indikerar att KT och BT uppnår likvärdiga effekter samt att både KT och BT är effektiva som fristående behandlingar för insomni. Studie II gav stöd för att effekterna i de två terapierna i KBT-I kan bero på olika egenskaper hos patienten vid baslinjen. Resultaten från studie II tyder således på att behandlingarna i KBT-I skulle kunna skräddarsys utifrån patientens egenskaper före behandling för att optimera resultaten. Studie III gav stöd för kognitiva processer som viktiga faktorer att påverka för att minska insomni och understryker värdet av att mäta samt fokusera på att minska dysfunktionella antaganden, monitorering och säkerhetsbeteenden för att behandla insomni, samt betonar vikten av dessa begrepp i förståelsen av insomni.

Background

Although sleep is a vital part of life (Cirelli & Tononi, 2008), most, if not all, people have suffered from problems with sleeplessness in relation to stressful events, such as experiencing difficulties with falling asleep or maintaining sleep. However, for most people, these difficulties vanish when the stressor ends. But for a relatively large proportion of the population (10%), the sleeping difficulties persist and become an ongoing subject of concern that affects not only sleep but also daytime functioning with problems such as worry, distress, fatigue, concentration, and memory impairment.

These problems with sleep, referred to as insomnia, have been the subject of scientific scrutiny for decades. An effort that has provided evidence of how to both identify the problem diagnostically and its prevalence rates, as well as its broad range of associated negative consequences, such as functional impairments, absenteeism, higher sick-leave, and healthcare utilization. Besides such impairment, insomnia is also associated with an increased risk of developing comorbid health issues. Adverse effects that subsequently also have economic consequences for both the sufferer and the society. Together, underscoring the need for interventions that can remediate the condition and its negative consequences.

Today, CBT-I is the most empirically validated therapy and is considered the treatment of choice. Although an effective treatment, a large proportion of patients still do not respond, and an even larger proportion fail to remit. One reason for the insufficient effectiveness of CBT-I could be the lack of understanding of what components target what part of insomnia, what individual characteristics the treatment depends on to be effective, as well as how the observed change came about, i.e., what mechanism or process led to relief from insomnia.

To aid in advancing the clinical and theoretical understanding of CBT-I and optimize outcomes, this thesis aimed to address the abovementioned limitations by conducting a randomized controlled trial that sought to investigate the efficacy of the main therapies of CBT-I; CT & BT on a broad range of outcomes, as well as by assessing potential moderators and mediators derived from the theoretical models of insomnia.

Introduction

Insomnia

Around 37% of the population report sleep complaints, such as sleep being too short or too light, or a general dissatisfaction with sleep, and 34.5% report insomnia symptoms, such as difficulties in initiating, maintaining, or having non-restorative sleep (Morin & Benca, 2012; Ohayon & Reynolds, 2009). However, experiencing dissatisfaction and problems with initiating and maintaining sleep are still distinct from the 9.8% that, in association with insomnia symptoms and dissatisfaction with sleep, also experience significant distress, functional impairment, and daytime symptoms that meet the diagnostic criteria for insomnia disorder according to the Diagnostic and statistical manual of mental disorders (DSM-5; American Psychiatric Association, 2013). The definition of insomnia, according to DSM-5, one of the most widely used nosology systems, is defined as a predominant complaint with sleep duration or sleep quality that is associated with difficulties in initiating or maintaining sleep, or waking up too early with an inability to go back to sleep, and associated symptoms the following day. The difficulties occur despite adequate opportunities for sleep and are associated with clinically significant distress or impairment of daytime functioning, i.e., decreased energy, fatigue, problems with concentration, memory, and mood. For a diagnosis to be made, the nighttime difficulties must be present for at least three nights or more per week over at least three months (American Psychiatric Association, 2013).

The diagnosis can further be specified into either persistent, episodic, or acute insomnia (Morin & Benca, 2012), where the latter is a more transient condition, lasting from at least one month up to three months. Persistent insomnia, which is the default diagnostic category and the focus of this thesis, is, as mentioned above, defined as occurring for at least three months, and finally, recurrent insomnia refers to two or more episodes within a year. The definition of acute insomnia, which is the more common of the two subcategories, varies between nosologies, but usually involves it being shorter than persistent insomnia and more related to current stressors in life, in contrast to persistent, which is theorized to be more related to the current sleep situation (Ellis et al., 2012). The diagnosis can be further sub-specified with associated comorbidity using three categorizations; other non-sleep mental

comorbidities, medical comorbidity, and, finally, other sleep comorbidities (American Psychiatric Association, 2013).

Before concluding, it is also worth considering on the one hand that there are other widely used and recognized nosologies, such as the ICD-10 and ICSD-3 (Riemann et al., 2017). These consist of almost identical criteria for diagnosing insomnia as the DSM-5, but deviate slightly, for example, in that the ICD-10 requires only one month for a persistent insomnia diagnosis. Thus, there are several, although similar, nosologies. On the other hand, there is the fact that the criteria in nosologies tend to change over time. For example, both the ICSD-3 and the Diagnostic and statistical manual of mental disorders IV (DSM-IV) have adapted similarly to emerging findings on the lack of evidence that treating another somatic or psychiatric primary disorder relieves the insomnia syndrome, and have therefore moved away from the distinction of insomnia as primary or secondary to another disorder, and instead have adapted the umbrella term 'insomnia disorder' (Riemann et al., 2017; Seow et al., 2018). Furthermore, both diagnostic systems have deleted "non-restorative sleep" from the definition since this was not specific to insomnia, and have instead added the term 'sleep dissatisfaction', which in a better way describes the difficulties experienced in insomnia (Morin et al., 2015). Thus, the diagnostic systems used to identify insomnia are subject to changes in line with new research findings in the field.

Epidemiology of insomnia

The prevalence of insomnia ranges from 3.9% to 22.1% depending on the nosology used, which differ in severity and specificity as regards their criteria for diagnosing insomnia, but approximates 10% across nations when the DSM-IV is used (Morin et al., 2015; Ohayon & Reynolds, 2009; Roth et al., 2011). The condition is more prevalent in women than in men and in people with medical or psychiatric conditions (Bin et al., 2012; Ohayon, 2002; Pearson et al., 2006). For example, prevalence rates of 20% in Germany and 50% in Norway have been reported for patients consulting their general practitioner (Riemann et al., 2017). Furthermore, approximately 7-15% of the population are estimated to develop insomnia each year (LeBlanc et al., 2009; Morphy et al., 2007; Pillai et al., 2014), and although some of these cases are of a recurrent or of a situational art, it is often chronic with a median duration of three years, and around 56% to 74% still reporting insomnia at one-year follow-up. As many as 46% fulfilling criteria at three-year follow-up (Morin et al., 2009; Morphy et al., 2007; Pillai et al., 2015), and across ten years, one study found 44% still fulfilling criteria (Janson et al., 2001).

Insomnia-associated problems and consequences

Besides being a prevalent condition that tends to remain if untreated, insomnia also carries several associated consequences for both the sufferer and the society. For the individual, these consequences relate to an increased risk of developing other somatic and psychiatric conditions, functional impairments, and reduced quality of life. For society, the consequences are expressed as increases in healthcare consumption, higher frequencies of sick-leave, and absenteeism from work, all associated with economic consequences for both the individual and the society.

In terms of comorbidity, it has been estimated that individuals suffering from insomnia are more than five times as likely to present with anxiety or depression comorbidities and more than twice as likely to present with congestive heart failure as individuals without insomnia (Pearson et al., 2006). Furthermore, in a large epidemiological study (Ohayon & Reynolds, 2009), it has been shown that almost half of the sample that met the criteria for insomnia also met the criteria for another mental disorder. Of those, 22.6% met the criteria for an anxiety disorder, 8.3% major depressive disorder, and 8.4% a comorbid anxiety and mood disorder.

Besides being associated with comorbidities cross-sectionally, insomnia has also been prospectively associated with developing subsequent disorders. According to a recent meta-analysis (Hertenstein et al., 2019), suffering from insomnia disorder was significantly linked to an almost three-fold (OR 2.83) likelihood of developing depression, a three-fold (OR 3.23) likelihood of developing an anxiety disorder, a 35% (OR 1.35) likelihood of developing substance abuse, and a 28% (OR 1.28) percent likelihood of developing a psychotic disorder. Overall, these results indicate an increased risk of developing further psychiatric disorders. The same trend is evident for somatic conditions, where insomnia has turned out to be a significant risk factor for cardiovascular diseases in several meta-analyses (Li et al., 2014; Meng et al., 2013; Sofi et al., 2014). In more specific terms, suffering from insomnia has been linked to a significant risk for chronic heart failure, myocardial infarction, and arterial hypertension (Laugsand et al., 2011; Laugsand, Strand, Platou, et al., 2014; Palagini et al., 2013). Insomnia has also been identified as a risk factor for diabetes type 2 (Anothaisintawee et al., 2016), play a role in the development of cognitive impairments (Yaffe et al., 2014), been associated with cortical atrophy in older adults (Sexton et al., 2014), and involved in the general development of neurodegenerative diseases, particularly dementia (Osorio et al., 2011). Finally, those with persistent insomnia compared to those with recurrent and those without, have been identified to have an increased risk of mortality (Parthasarathy et al., 2015).

Finally, in terms of other associated consequences, those diagnosed with insomnia tend to display, as part of the diagnosis, some daytime functional impairments, such as difficulties with concentration and memory, fatigue,

disturbances in mood, tiredness, and reduced quality of life (American Psychiatric Association, 2013). Symptoms that together constitute a basis for impaired functioning in social, occupational, and leisure activities. Perhaps owing to such impairment, insomnia is also associated with a higher frequency of absenteeism, sick leave, lost productivity, accidents at work, and increased healthcare consumption (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009; Laugsand, Strand, Vatten, et al., 2014; Sivertsen, Øverland, Bjorvatn, et al., 2009; Sivertsen, Øverland, Pallesen, et al., 2009). These are side effects that besides impairing the individual, also carry significant direct and indirect economic consequences for both the individual and the society, as shown by studies estimating that insomnia accounts for 13.6% of all days out of role (Hajak et al., 2011), and 4.6% of all injuries demanding medical care (Kessler et al., 2012).

These are consequences that are further illustrated by numbers showing that direct and indirect costs associated with insomnia (Ozminkowski et al., 2007) have been estimated at 790 euro and 5.010 CA$ per year per person in Europe and Canada (Daley, Morin, LeBlanc, Grégoire, Savard, et al., 2009; Gustavsson et al., 2011). Costs of which up to 90% are estimated to be accounted for by the indirect costs of reduced productivity and work absenteeism due to insomnia (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009). Finally, in picturing the gross economic burden, insomnia has collectively been estimated to have a cost of 107.5 US$ billion per year (Léger & Bayon, 2010).

In summary, the persistent suffering of insomnia is a prevalent condition that places a heavy burden on both the individual and the society, which clearly points to the importance of society understanding its mechanisms and providing efficacious treatments.

The development and persistence of insomnia

One way of understanding both the development and persistence of insomnia, as well as ways to manage the condition, is the 3-P model depicted in Figure 1 (Spielman, Caruso, et al., 1987). According to this model, insomnia is viewed as the result of three factors: predisposing, precipitating, and perpetuating factors (the 3-Ps). Predisposing factors are preconditions or vulnerabilities that make an individual more or less susceptible to develop insomnia and can stem from the entire biopsychosocial spectrum such as genetic predispositions (Palagini et al., 2014), biological as in hyperarousal (Riemann et al., 2010) or psychological predispositions as in neuroticism, tendencies to worry or ruminate (Morin et al., 2015; Perlis et al., 2017). Precipitating factors, on the other hand, are usually life events that have triggered stress such as a separation, bereavement, the onset of a chronic illness, or more chronic daily struggles such as occupational or relationship stress that represent continuous ongoing stressors. For most people, such stressors can disturb and trigger acute insomnia at the time they are present, and for most people, sleep also normalizes when such precipitating events vanish or attenuate (Ellis et al., 2012; Espie, 2002). However, some continue to experience sleep disturbances that do not vanish over time, even though the precipitating events have been attenuated or have vanished, and the previous acute insomnia now turns into a persistent problem. According to the 3-P model, one explanation for this is the third P, the perpetuating factors. These are usually behaviors that individuals have enacted in an effort to try to handle the acute sleep problem with the goal of achieving sleep or compensating for lack of sleep. Examples of these are attempts to solve the problem by: worrying, minimizing daily activities to save energy, going to bed early, spending extra time in bed and taking daytime naps in order to catch up on insufficient sleep. Although these are reasonable attempts in trying to solve the sleeping problem, these behaviors are also proposed to prevent normal sleep from returning because they interfere with: keeping a normal circadian rhythm, relax and de-arouse during the evening, and building sleep drive for the evening.

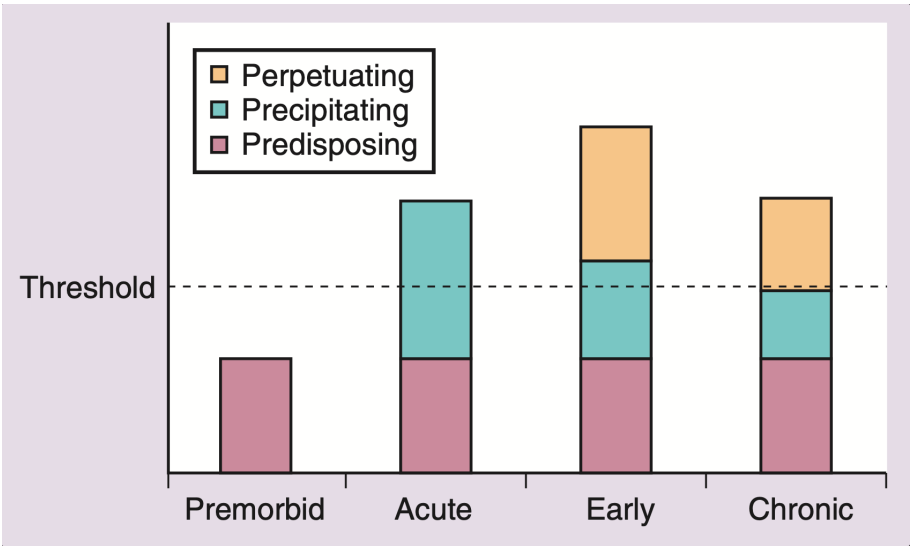


Figure 1. The 3-P Model

Overall, as depicted in Figure 1, although all three factors are of relevance at different time points during the development of insomnia, it is the perpetuating factors that are of most relevance in explaining why insomnia disorder has become persistent and how it can be managed at this time point. This is because, at this time point, the predisposing factors are hard to mold, and the precipitating factors have usually subsided.

Perhaps due to their role in identifying why people continue to suffer from persistent insomnia, there are several models that aim to describe how insomnia is maintained by outlining potential perpetuating factors. Most of these models are implicitly or explicitly based on the 3-P framework, and although some also describe predisposing and precipitating factors, the majority of them focus on the perpetuating factors and their role as targets for treatment (Perlis et al., 2017). Common to most perpetuating models is a focus on behaviors (i.e., spending excessive time in bed, going to bed early; Bootzin, 1972; A. A. Borbély, 1982; Morin, 1993; Perlis et al., 1997), cognitions (i.e., worry, attention, intention, effort; (Espie, 2002; Espie et al., 2006; Harvey, 2002; Lundh & Broman, 2000), hyperarousal (Riemann et al., 2010), or a combination of these, as perpetuating factors.

These abovementioned models of how insomnia becomes persistent thus all provide relevant explanations and thereby suggest suitable targets for treatment.

Treatments of insomnia disorder

A couple of options, spanning pharmacological, psychological, and alternative therapies (e.g., acupuncture, or herbs as in valerian root) have been provided in the search to find remedies for insomnia. However, alternative therapies are generally not recommended due to a lack of knowledge of both their potential benefits and associated risks (National Institutes of Health, 2005). Pharmacological and psychological (i.e., CBT-I) treatments have, on the other hand, gathered a solid empirical base and are both viewed as efficacious treatments (Morin et al., 2015; Riemann et al., 2017). For example, benzodiazepine receptor agonists, which are the most common pharmacological agent, have, in several meta-analyses been shown to be effective in enhancing sleep, but at the same time, they also carry a significant risk of side effects and has limited evidence for their long-term effect. Cognitive Behavioral Therapy for Insomnia (CBT-I), on the other hand, also has a solid evidence base, but in addition to pharmacological agents, also achieves improvements that are sustained over time (Morin et al., 2006) with minimal side effects, which is a clear advantage compared to pharmacological treatments. CBT-I is, in most cases, also considered the treatment of choice for insomnia (Morin et al., 2015).

Since CBT-I is the focus of this dissertation, this treatment will be further elaborated upon here. CBT for insomnia consists of sleep-focused cognitive and behavioral techniques that address the cognitive and behavioral processes (beliefs, worries, irregular sleep patterns) described by each theoretical model to perpetuate insomnia (Edinger & Carney, 2014; Morin & Espie, 2003). The treatment is usually delivered over approximately six sessions in an individual, group, or a self-help internet format. Another essential part of the treatment is to keep a daily sleep diary. Besides enabling evaluation of symptoms, sleep-schedules, and treatment progress, sleep diaries also engage the patient in the treatment process (Morin et al., 2015). This treatment for insomnia has, in several trials, using different delivery modes and diverse samples, including comorbid insomnia, demonstrated efficacy on a broad range of outcomes, including overall insomnia and insomnia symptoms, with small to moderate effect sizes (Riemann et al., 2017).

However, although CBT-I is an evidence-based psychological treatment considered the treatment of choice, with efficacy for a wide range of populations and comorbidities, a large proportion of patients (20-30%) still fail to achieve a clinically significant response to treatment, and an even larger proportion (60%) fail to remit from the condition (Morin et al., 2015). Furthermore, theoretical and clinical understanding about what it is in CBT-I that works, what the necessary components are, how change comes about, as well as how individual clinical characteristics affect the treatment outcome are still limited (Riemann et al., 2017; Schwartz & Carney, 2012). Thus, although an efficacious treatment, there is room for advancing both the clini-

cal and theoretical understanding of the treatment so that more patients among those not yet responding and remitting can benefit from CBT-I.

One way to advance our theoretical understanding and optimize the treatment to reach those that are not yet responding or remitting from CBT-I is to address the gaps in knowledge concerning what treatment components are necessary and sufficient for effect, what individual characteristics predict or moderate a favorable outcome, and what processes perpetuate insomnia, as well as how existing treatments bring about change (Kazdin, 2007; Kraemer et al., 2002; Schwartz & Carney, 2012). The first limitation concerns the fact that although CBT-I is composed of several components evolved from both cognitive and behavioral models of how insomnia is maintained, little is known about the comparable and separate effects of these components and hence the necessity or redundancy of each component in the efficacy of CBT-I. Second, and related to the abovementioned limitation, is the fact that very little is known about what individual insomnia—or demographic—characteristics the efficacy of CBT-I or any of its subcomponents depends on, and thus whether the treatment(s) is more effective for certain individuals. Third, and related to the abovementioned limitations, is the fact that even less is known about how CBT-I or any of its subcomponents leads to change, i.e., through what process the separate interventions in CBT-I bring about change. Filling these gaps in knowledge could provide more clarity to our theoretical and clinical understanding of insomnia and what therapy or therapy component is necessary and of most value for a specific individual. Also, understanding the processes of change that brings relief from insomnia could point to areas of the CBT-I package that need to be further elaborated, intensified, or discarded.

Thus, one way to advance our theoretical and clinical understanding of CBT-I is to explore the comparative efficacy of its separate components, what patient characteristics change depends on, as well as what processes are responsible for the observed change. Since CBT-I builds on two major therapies, CT, and BT, which stem from two separate and distinct theoretical models describing how insomnia is maintained, one reasonable start in trying to address the abovementioned limitations could be to examine these two therapy models in terms of their separate efficacy, how individual characteristics affect them differently, as well as their processes of change. Since this is the focus of the thesis, each theoretical model on which the therapy is based will be outlined below, by describing their proposed perpetuating factors of insomnia as well as how each treatment goes about addressing them, and their efficacy in doing so. After that, the existing evidence for the three areas of knowledge, i.e., the therapies' comparative efficacy, their separate predictors and moderators, and what processes lead to change, referred to as mediators, are summarized and reviewed as a way to provide the current state of knowledge on these issues and a background for the studies of this thesis.

The cognitive model and its treatment

The cognitive model presented by Harvey, 2002, builds on prior important cognitive models of insomnia and other mental disorders (Clark, 2015; Lundh & Broman, 2000; Morin, 1993; Wicklow & Espie, 2000). According to Harvey's model (see Figure 2 below), insomnia is viewed as the end result of a series of cognitive processes that are triggered or initiated by attempts of the individual to handle the sleep problem, such as engaging in excessive negatively toned cognitive activity (worry), monitoring for sleep, and use of safety behaviors applied to prevent a negative catastrophic belief (Salkovskis, 1991).

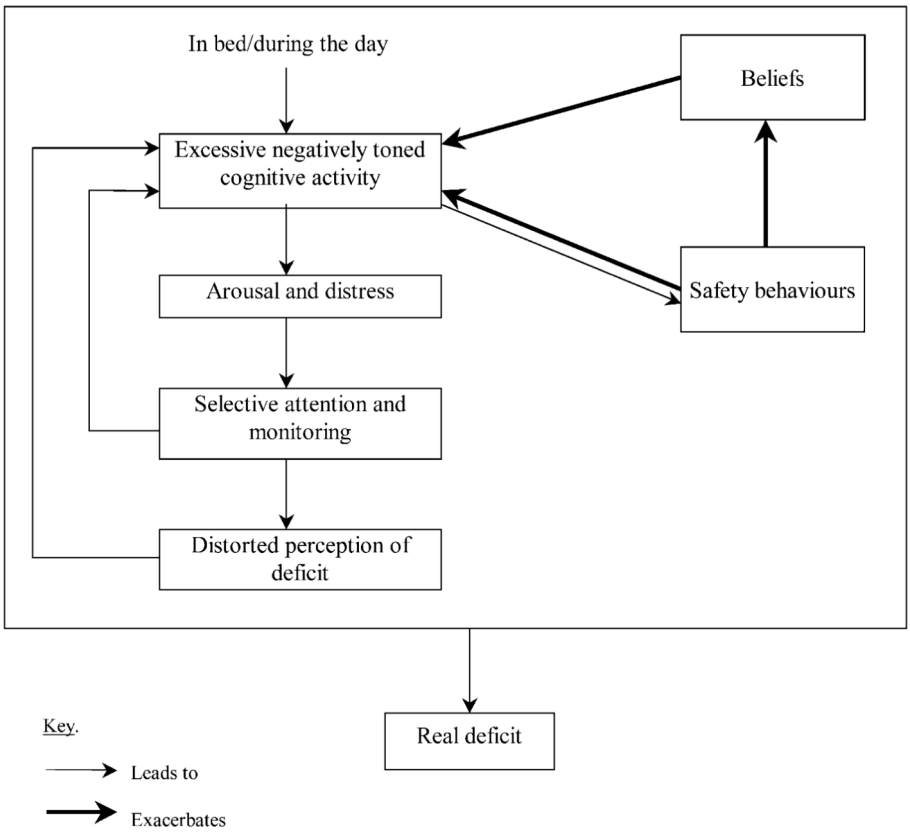


Figure 2. A cognitive model of the maintenance of insomnia.

The starting point for the subsequent cognitive processes that lead to insomnia is proposed to be an excessive negatively toned cognitive activity such as worry (Borkovec et al., 1998; Jansson-Fröjmark et al., 2011) or rumination (Carney et al., 2006). This occupation with worry or rumination about sleep is proposed to lead to or trigger emotional distress and autonom-

ic arousal due to its activation of the sympathetic nervous system —creating a state of alertness and anxiousness. This worry and arousal, in turn, initiate an increase in attention and focus on the threats inherent in the negative thought content as a way to try to handle the threats. This is suggested to prompt the individual to monitor (Semler & Harvey, 2004) both internal (physical sensations) and external cues (the environment) for threats to sleep, such as signs of not getting enough sleep and the effect of insufficient sleep on functioning during the day. The chance of detecting threats is, at this point, increased since selective attention to threats increases the odds of detecting otherwise meaningless cues as possible threats, and the already increased arousal means there are also plenty of bodily sensations to detect. Any subsequent detection of threat at this point then provides reasons for further concern, which feed into more worry and boost the subsequent processes in the model further. Together, this anxious state and the attentional process triggered by it is proposed to trick the individual into overestimating the extent of the perceived deficit, that is, that they obtained significantly less sleep than they did, or that their daytime performance was significantly worse than it actually was. This again feeds into and exacerbates the already elevated levels of worry (negatively toned cognitive activity), that further feed into the subsequent process in the model and increasing their combined negative effect on sleep. Besides the chain of events linking worry, arousal, monitoring, and misperception of sleep in a negative feedback loop, two additional processes are proposed to feed into and exacerbate the excessive negatively toned cognitive activity (worry). These are unhelpful beliefs about sleep (Morin, 1993) and the use of safety behaviors (Ree & Harvey, 2004a; Salkovskis, 1991) to handle the worrying content. Unhelpful beliefs are proposed to consist of faulty assumptions about how normal sleep proceeds and the consequences of poor sleep. Such beliefs feed into and provide a source and content for worry. Safety behaviors (Salkovskis, 1991), on the other hand, are enacted to solve or cope with the unhelpful beliefs and the worry content to achieve better sleep, but instead tend to reinforce existing beliefs and worry as well as prevent the possibility of disconfirming the unhelpful belief. In this way, dysfunctional beliefs and safety behaviors also fuel worry that feeds into the following processes. The result of all this is that the exacerbated worry and escalating arousal may culminate in a real deficit since distress and physiological arousal are states under which sleep is unlikely and daytime impairments probable.

Based on this model, CT aims to reverse or normalize the five processes proposed to perpetuate the model. CT achieves this by first educating the patient about sleep and daytime symptoms and then creating a cognitive conceptualization of the patient's day and nighttime problems. This conceptualization is then explored and critically evaluated by examining and questioning Negative Automatic Thoughts (Beck, 1995), as well as by designing

and testing behavioral experiments (Harvey, 2005; Harvey et al., 2007, 2014).

In detail, Socratic questioning and the identification of Negative Automatic Thoughts is applied initially in treatment to introduce and accustom patients to the idea that thoughts, assumptions, and beliefs that the patients hold regarding sleep and their insomnia may not be realistic. Hence, by investigating their Negative Automatic Thoughts about sleep, they start to elaborate their understanding of how their beliefs and thoughts about sleep might contribute to their insomnia, and in that process also restructures them towards more realistic beliefs and assumptions about sleep (Beck, 1995).

After getting acquainted with their beliefs and the potential unhelpful role of these in the cognitive model, behavioral experiments are introduced to more thoroughly challenge beliefs and assumptions about sleep and lack of sleep. A behavioral experiment is a planned experiential activity based on experimentation, which leads to new experiences (Ree & Harvey, 2004b). Some typical examples include the fear of poor sleep experiment, which has as its aim to gain new experiences of poor sleep (limited hours of sleep). Another experiment is the generating versus conserving energy experiment, which involves trying different strategies for coping with daytime fatigue. It is worth underscoring that since the aim is to reverse the specific individual cognitive processes, the experiments are designed directly based on the patient's cognitive conceptualization in order to obtain new and relevant information in relation to his or her conceptualization (Ree & Harvey, 2004b).

Both the cognitive model and the therapy that is devised to reverse the perpetuating cognitive factors of insomnia have received empirical attention and support. For the cognitive model, there have been experimental, cross-sectional, and clinical studies that overall provide some support for both how processes in the model affect or are related to symptoms of insomnia and how the pathways or relationship between each process are related to each other (Hiller et al., 2015). To mention some specific examples, experimentally induced worry has been shown to increase the time to fall asleep (Gross & Borkovec, 1982; Hall et al., 1996; Lichstein & Fanning, 1990), and interventions that decrease worry lead to a shorter time to fall asleep (Harvey & Payne, 2002; Haynes et al., 1981; Levey et al., 1991). Also, cross-sectional studies have shown that distress, anxiety, and catastrophic thoughts are more prevalent in people with insomnia, compared to good sleepers (Harvey & Greenall, 2003), and the greater the dysfunctional beliefs, the more frequent the occurrence of safety behaviors (Woodley & Smith, 2006). Finally, CT as based on the model has also gathered evidence as a therapy for managing insomnia. Although there is not as much evidence as for the model, one open trial and one randomized controlled trial have tested its effect compared to both BT and CBT, and found an overall similar effect as BT and a smaller effect compared to CBT (Harvey et al., 2007, 2014).

Overall, to some degree, there is empirical support for both the cognitive model of how insomnia is maintained and for its effect as a treatment of insomnia. However, little is still known about the relative effect of CT, what predicts and moderates this treatment, or through which processes CT brings about change in therapy

The behavioral model and its treatment

The behavioral model of insomnia, on which the behavioral therapy is based, rests on two biological models of sleep: the homeostatic and the circadian systems (Bootzin et al., 1991; Borbély, 1982; Spielman, Saskin, et al., 1987; Webb, 1988), as well as a theory of learning (Bootzin, 1972). Sleep is, according to the homeostatic system, regulated based on our time spent awake or asleep. The longer the time spent awake, the stronger the drive for sleep, and the accumulated sleep drive at bedtime determines the quality and quantity of sleep. This system interacts with but is independently regulated from the circadian system. The circadian system works as a biological pacemaker or clock that regulates the body's 24-hour sleep-wake pattern in interaction with time-cues (zeitgebers) from the environment (e.g., the dark-light cycle, sleep-habits, mealtimes). These zeitgebers thus aid in adjusting the biological 24-hour circadian rhythm to the living and environmental circumstances of a specific individual. In this way, the circadian system specifies an individual's 24-hour rhythm and, thereby, the individual's optimal window of sleep. When these two biological systems are synchronized, they feed into each other and support each other's drive for sleep or being awake (Dijk & Von Schantz, 2005). The learning theory described in the stimulus control model (Bootzin, 1972), on the other hand, rests on the theory of classical conditioning and refers to the notion that for sleep to occur, the bed and the bedroom need to be conditioned with or associated with a calm and relaxed state, ready for de-arousal and drowsiness, whereas the opposite would prevent or hinder optimal de-arousal and sleep.

Insomnia, according to the behavioral model, is proposed to be the result of coping behaviors that are engaged with, in an attempt to compensate for lack of sleep, that instead disturbs the normal workings of the homeostatic and the circadian system and re-conditions the bed with being awake instead of de-arousal and sleepiness.

Specifically, according to the behavioral model people with insomnia cope with their sleeping problem by the use of behaviors aimed at increasing their opportunity to get sleep, such as extending their time in bed by going to bed early or getting out of bed late or taking naps during the day. These behaviors usually become reinforced and continue, since they allow people to occasionally recover lost sleep and ameliorate the acute daytime effects of insufficient sleep. However, simultaneously this also results in less waketime

activity, which hinders homeostatic sleep pressure from sufficiently building up for the coming night, which in turn decreases the chances of having enough pressure for sleep the following night. Furthermore, the habits of going to bed early or sleeping in during the morning to catch up on sleep also create a constant day to day variability in the schedule for going to bed and rising. Although reasonable as a way to compensate for lack of sleep, this also prevents the endogenous circadian system from properly adjusting sleep according to a regular 24-hour cycle because no stability or consistency of the necessary zeitgeber is provided on a regular basis. This makes sleep less likely when attempted, since the circadian clock may not be set for sleep initiation if sleep is continuously irregularly initiated, for example, when going to bed early to get some extra sleep. Furthermore, the potential misalignment of the circadian system with the homeostatic system that these habits may cause may also prevent sleep, since their combined and synchronized drive for sleep is then missed out.

Finally, these efforts to compensate for lack of sleep by extending the time in bed often result in involuntarily waketime, usually in bed using non-sleep behaviors, such as remaining in bed either twisting and turning—trying to fall asleep or engaging in distracting activities such as reading. This leads to the continuous association of the bed and the bedtime with many things other than sleep, such as frustration with trying to sleep and associated arousal, in all providing a solid ground for conditioned arousal to the bed and bedtime, instead of sleep (Morin et al., 2015). This corresponds to the common report from insomniacs that just entering the bedroom made them suddenly feel wide awake, like a switch was turned from being sleepy to being wide awake (Perlis et al., 2017). Together with how the previously mentioned system hinders de-arousal, this is further proposed to prevent the de-arousal necessary for sleep.

The behavioral therapy (BT) for insomnia was built on the above-described models of sleep and is thus designed to optimize the processes proposed to perpetuate insomnia by regularizing the timing of sleep and sleeping ability (pressure) with opportunity. BT achieves this by applying two behavioral techniques, sleep restriction and stimulus control, and a more general technique called sleep hygiene.

Sleep restriction aims to both regulate the homeostatic system and entrain the circadian system by increasing the homeostatic sleep pressure before bedtime and regularizing the timing of the sleep period. This is achieved by limiting the time in bed to actual hours of sleep and establishing a fixed rise time from which the time for going to bed is calculated. Since insomniacs tend to underestimate their total sleep time (Means et al., 2003) and it is rare to sleep during all the time spent in bed (Baglioni et al., 2014; Spielman, Caruso, et al., 1987; Spielman, Saskin, et al., 1987), this initially creates a mild sleep deprivation that affects the homeostatic system by increasing the drive for sleep, which in turn increases the likelihood of better sleep quality

and quantity (Spielman, Saskin, et al., 1987). Since the restricted time in bed is also scheduled for the same time period every day, this also aids in entraining the circadian system.

Stimulus control, the other technique, targets the homeostatic and the circadian system as well, but also addresses the conditioned arousal around the bed and bedtime (Bootzin et al., 1991). Stimulus control achieves this by a set of behavioral instructions implemented to strengthen a consistent sleep-wake schedule, increase homeostatic sleep pressure, and strengthen the association between bedtime and the bedroom with sleep and rapid sleep onset. The instructions to achieve this are to: (a) go to bed only when sleepy; (b) get out of bed when unable to sleep; (c) only use the bedroom for sleep and sex (no reading or problem-solving in bed); (d) maintaining a fixed rise time every morning; (e) avoid napping (Bootzin et al., 1991). The fixed rise time and increased regularity of only using the bed for sleep, aid in providing clear zeitgebers and a consistent distinction between the night and the day that strengthen a consistent 24-hour rhythm of the circadian system (Perlis et al., 2017). Since these rules also prevent spending time awake in bed and using daytime naps, they also aid in increasing the homeostatic drive for sleep. Finally, these rules also aid in reconditioning the bed with sleep, since they prevent behaviors other than sleep in the bed, which allows for reconditioning of the bed with sleep instead of the bed being conditioned with trying to fall asleep.

The last component, sleep hygiene, which is usually a part of behavioral treatments for insomnia, has a less clear theoretical basis for its intervention, and is perhaps, therefore, viewed more as a way to remove obstacles for the other interventions to promote sleep and prevent relapse. Sleep hygiene consists of a set of general guidelines about health practices (substance use, exercise, diet) and setting of the sleep environment (temperature, noise, light) that have the potential to either promote or interfere with sleep (Peter J Hauri, 1991; Perlis et al., 2011), and it might also include some basic information about normal sleep and changes in sleep patterns with aging (Morin et al., 2015). The instructions include: (a) avoid caffeine, nicotine, and other stimulants several hours before bedtime, (b) avoid alcohol close to or around bedtime since it fragments sleep in the second half of the night, (c) exercise regularly, but not too close to bedtime, (d) avoid watching the clock, (e) keep the sleeping environment dark and quiet (Morin & Benca, 2012).

Worth mentioning in relation to the abovementioned models and treatment techniques is a more recently proposed model, called the triple-R model (Maurer et al., 2018), which outlines how the core component in BT, sleep restriction, comes to have an effect, not only on the two physiological systems, and the maladaptive conditioning as depicted above but also on how changes in these systems are subsequently associated with or lead to changes in processes described to maintain insomnia in other models, particularly the cognitive and the hyperarousal model of insomnia. The triple-R model, in

this way, suggests a reciprocal and simultaneous impact of sleep restriction on both cognitive behavioral and physiological processes when undergoing BT and sleep restriction.

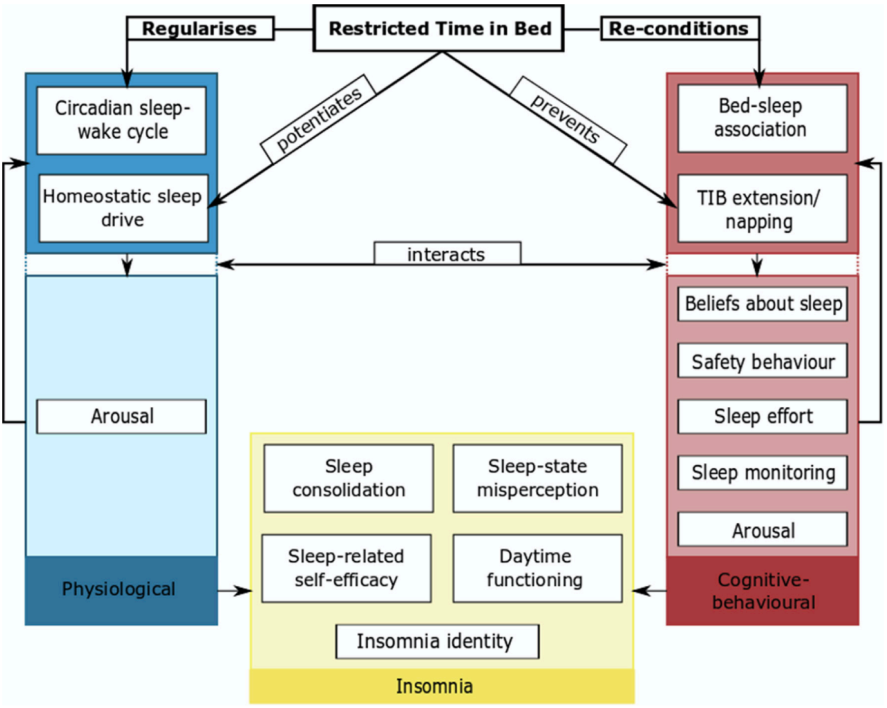


Figure 3. The Triple-R model

Specifically, as depicted in Figure 3, the triple-R model proposes that sleep restriction effectively treats insomnia primarily by restricting time in bed, regularizing the time in bed, and reconditioning the bed with sleep, all in line with the previous description of sleep restriction and stimulus control above. In addition to this, changes in these physiological systems are also proposed to slip over to and affect other important processes. For example, increased sleep pressure, caused by restriction, is proposed to decrease hyperarousal at bedtime, and limiting time in bed serves as a behavior experiment that challenges unhelpful beliefs about the need to extend time in bed and prevent the use of safety behavior which leads to new experiences and ultimately aids in restructuring the cognitive processes that maintain insomnia. This reciprocal and simultaneous change between both cognitive-behavioral and sleep physiological processes is proposed to also feedback onto cognitive processes during the day, such as monitoring, worry, and safety behaviors. The reason for this is partly that the new experiences of

more consolidated sleep and fewer problems with sleep initiation mean there is less drive or need for engaging in problem-solving such as worry, monitoring, and safety behavior during the day. Overall, the key point is that the model illustrates how several of the maintaining process described in several models of insomnia could all be affected by sleep restriction in BT and proposes how the processes in these models relate to and reciprocally interact with each other to reduce insomnia during treatment with sleep restriction. This proposal that processes from several models contribute to remediating insomnia during sleep restriction is interesting since it is a testable proposal that future trials can explore using sleep restriction and assessing change in, for example, cognitive processes.

These reviewed physiological and behavioral sleep-regulating models on which BT are built have received support for their roles in regulating normal sleep (Borbély et al., 2016). However, little attention and evidence has been provided for the BT proposal that insomnia develops due to dysregulation of the two sleep systems (Perlis et al., 2017), as the behavioral model suggests. Conversely, the treatment (BT) based on the model has gathered plenty of evidence and is considered an effective treatment for insomnia (Buysse et al., 2011; Morin et al., 2006).

For the proposed behavioral model on how insomnia is perpetuated, as mentioned, little attention has been paid to validating the model experimentally or cross-sectionally, and little or no evidence exists for the proposal that insomnia actually results from dysregulation of the homeostatic and the circadian systems due to extension of time in bed and variability of bed- and rise-times (Perlis et al., 2017). However, the interventions that are built on the model have received plenty of attention and gathered evidence for its efficacy, both used as separate components but more so as a package, delivered together.

Specifically, sleep restriction, stimulus control, and sleep hygiene used as separate components, have all received empirical attention, with small to moderate efficacy (Chung et al., 2018; Miller et al., 2014; Morin et al., 2006). However, it is their combination as used under the umbrella term ‘behavior therapy’ that is the most common way that these components are delivered as a treatment for insomnia. Although BT also comes in variations, sometimes including all three components and sometimes only sleep restriction and stimulus control (Morin et al., 2006), it is probably the most studied subcomponent of CBT-I and considered an empirically supported treatment of small to moderate efficacy (Buysse et al., 2011; Morin et al., 2006).

In all, there is solid support for the therapy but less direct evidence for its model and underlying theory. Furthermore, evidence regarding the relative effect of BT compared to CT and CBT, which variables that predict or moderate the effect of BT, and what processes that brings about change is also scarce.

Comparative efficacy of therapy components

Although there is evidence that both BT and CT are effective as monotherapies for insomnia (especially BT), less is known about their unique role in reducing the full spectrum of symptoms inherent in the diagnosis of insomnia (e.g., difficulties maintaining sleep, daytime functioning, worry). This is a problem since it implies that we do not fully understand how each subtherapy of CBT-I targets or ameliorates insomnia and the range of symptoms inherent in the diagnosis. Furthermore, this may also imply that we are intervening against symptoms already targeted by components of the same treatments or, in the worst case, delivering interventions with the potential to counteract each other. Overall, this holds the possibility that we may require more energy and effort than necessary to achieve a sufficient effect for those suffering.

Only one investigation is available, a randomized controlled trial that examined the relative effects of CT and BT compared to full CBT-I. This indicated no differences between CT and BT other than different trajectories of change from pretreatment to follow-up on insomnia severity, with BT having a stronger effect at posttreatment compared to CT, and deteriorating to follow-up, while CT had the opposite pattern of improving from posttreatment to follow-up (Harvey et al., 2014).

Although a very valuable contribution for disentangling the questions on the relative and comparative roles of CT and BT as part of CBT-I, there are still some unanswered questions remaining. First, since the study by Harvey et al., 2014, was the first comparing CT to BT in a randomized design, further replication is needed to validate the results. Second, since the previous study was delivered in a face-to-face format, there is no information on whether the relative effects of CT and BT hold across other ways of delivery or if they are unique to the face-to-face format. Thus, comparing CT and BT in other modes of delivery could add information on their relative efficacy across delivery modes. Third, the fact that there was no waitlist (WL) in the existing trial also opens up the risk of alternative explanations such as regressions to the mean, spontaneous recovery, or the effect of measurement to account for the positive effects. Therefore, adding a WL to future comparative studies could help to further disentangle the relative effect of CT and BT. Finally, to get a proper understanding of their relative and unique contribution as part of CBT-I in the amelioration of insomnia, it would be interesting not only to assess their impact on a broad range of measures related to the full spectrum of symptoms in the diagnosis, but also to assess constructs associated with the feasibility of each treatment, such as assessment of adverse effects, therapist support, treatment satisfaction, and the participants' perceptions of the two therapies (e.g., texts, exercises, and treatment workloads).

Gaining further knowledge from a broader range of measures could aid in providing the field with more clarity about the necessary and sufficient components, areas that are under- or over-targeted, and areas in need of further interventions.

Predictors and moderators

Although investigating the relative and comparative efficacy of the main therapies in CBT-I can provide the field with greater clarity about the necessary and sufficient ingredients for effect, it cannot provide a deeper understanding of whether and how the effect depends on the characteristics of those that undergo the treatment. Or in other words, whether CBT-I or its main sub-therapies work better for some individuals and worse for others, an area where still little is known.

Failing to understand the role of individual characteristics is a problem since such understanding could aid in explaining why some patients benefit from treatment while others do not, thereby enhancing the number of responders as well as isolating those in need of other interventions. Furthermore, understanding how the monotherapies in CBT-I (CT, BT) differ in terms of the characteristics they depend on to be effective, can, on the one hand, aid in tailoring CBT-I to the specific individual, while also providing knowledge on how the components in the treatment package target or suit different aspects of insomnia. This is knowledge that could lead to a better understanding of the variables that influence a treatment and the mechanism through which it works (Kraemer et al., 2002).

To further our understanding of what role individual characteristics play in CBT for insomnia, there are two common approaches that can be pursued (Wolitzky-Taylor et al., 2012). One is to identify individual baseline characteristics that have predictive properties on treatment response regardless of treatment and thus is dependent on individual characteristics rather than the treatment (referred to as a predictor). The other strategy goes one step further and tries to identify what baseline characteristics predict a response given a specific treatment. Such a variable thus interacts with and changes the effect of a specific treatment, dependent on the level of the predictor variable (referred to as a moderator), whereas a predictor has a main effect on the outcome above the treatment effect, i.e., it does not interact with treatment (Kraemer et al., 2002).

In trying to identify variables that have the potential to predict or moderate CT or BT, a couple of areas are of potential relevance. One of them is the insomnia symptoms inherent in the diagnosis, such as the global severity of the condition assessable with scales as the insomnia severity index (ISI: Bastien, Vallières, & Morin, 2001), or the nighttime symptoms assessable from a sleep diary or actigraphy (Buysse et al., 2006; Carney et al., 2012).

The ISI attempts to quantify the global severity of insomnia by assessing the severity of patients' nighttime problems, their satisfaction/dissatisfaction with sleep, their impairment due to the sleep complaint, their worry about sleep, and the degree to which the sleeping issue affects their quality of life. The nighttime symptoms attempt to quantify the severity of the nightly sleeping complaint. One of the three nighttime symptoms used for this is the difficulties with falling asleep, which in the sleep diary are defined and quantified as sleep onset latency, measured as the time it takes to fall asleep after switching the lights out. The second symptom is the difficulties with maintaining sleep, defined as wake after sleep onset, measured as the time spent involuntary awake after sleep onset. The third symptom is the problems with waking up too early in the morning, defined as early morning awakening, measured as the time spent involuntarily awake in the early morning before rising. Finally, there is a fourth associated nighttime issue, although not necessarily a symptom of insomnia, with gaining sufficient sleep defined as total sleep time, measured as the total time spent in bed subtracted by the total waketime in bed. One reason for putting effort into investigating insomnia symptoms as possible predictors or moderators is that, since the goal of treatment is to reduce such symptoms, their initial level of intensity could probably indicate something about the probability of the treatment succeeding. Another reason why symptoms are interesting to explore as potential predictors and moderators for the two sub-therapies is the fact that CT and BT address the day- and night-time symptoms of insomnia differently. If we, therefore, gain a better understanding of how different symptoms predict or moderate the two treatments, we can tailor CBT-I based on presenting symptoms and perhaps also identify symptoms that predict or moderate a negative outcome for both treatments, thus indicating a need for new interventions.

Another relevant area for identifying predictors and moderators concerns the clinically associated constructs that are related to insomnia, such as having comorbidities, reduced quality of life, functional impairments, use of hypnotics, and having symptoms of anxiety or depression. The reason they could predict or moderate the effect of CBT-I is that they, in adjunct to insomnia symptoms, also provide information on the overall status, function, and severity of the condition. Information that probably indicates something about the patient's ability to engage with and benefit from treatment.

A third relevant area for identifying predictor and moderator variables are the theoretical processes proposed to maintain insomnia according to the cognitive and behavioral models. Processes that each treatment also targets as their means to alleviate the condition. In CT, the relevant processes and their variables are the dysfunctional beliefs about sleep, as indexed by the Dysfunctional Beliefs and Attitude about Sleep scale (Morin et al., 2007). Monitoring for sleep-related threats, as indexed by the Sleep Associated Monitoring Index (Semler & Harvey, 2004). Worry, as indexed by the Anxi-

ety and Preoccupation about Sleep Questionnaire (Jansson-Fröjmark et al., 2011), and finally safety behaviors, as indexed by the Sleep-Related Behaviors Questionnaire (Ree & Harvey, 2004a). In BT, the relevant theoretical processes and their measures (Schwartz & Carney, 2012) are the variabilities in bed- and rise-times, as indexed by calculating the standard deviation of the weekly bed- and risetimes from the sleep diary. Time in bed, to capture changes in excessive time spent in bed, as indexed from the sleep diaries. Finally, sleep efficiency, defined as the total sleep time divided by the total time spent in bed, is also used to capture excessive time spent in bed. In following the theory, individuals with elevated baseline values on the processes in the CT or BT model should, if the theory is correct, probably be predictive of a favorable treatment outcome with that therapy, since these processes are what the treatment is targeting and aims to reduce. Understanding how CT and BT respond differently to high baseline values from the same and other theoretical models could thus provide further knowledge on how to tailor treatments based on presenting individual characteristics, as well as indirect validation for the theoretical models.

Below, the evidence on such variables as predictors and moderators is reviewed.

For insomnia symptoms as predictors and moderators, there are some previous findings showing that a total sleep time of six hours or more predicted a favorable response to BT or CBT in three studies (Bathgate et al., 2017; Bothelius et al., 2016; Troxel et al., 2013), while two, more recent studies, failed to find any predictive or moderated properties (Lovato et al., 2016; Rochefort et al., 2019). Finally, a recent examination found the opposite, a total sleep time < 6h to moderate a greater response (Galbiati et al., 2020). For insomnia severity, three studies have indicated that greater insomnia severity assessed by the Pittsburgh Sleep Quality Index (Backhaus et al., 2002), predicted greater reductions in severity at posttreatment (Morgan et al., 2003; Troxel et al., 2013; Van Houdenhove et al., 2011), and one study showed similar results using the insomnia severity index (ISI; Savard et al., 2016). However, there are also two studies that failed to find any predictive effects of ISI on: a reliable change index for ISI, sleep efficiency, reduced sleep onset latency or wake after sleep onset (Currie et al., 2002 & Lovato et al., 2013). In terms of the nighttime symptoms, longer sleep onset latency alone, and in combination with wake after sleep onset, have been shown to predict a favorable outcome using the same measures in two studies (Espie et al., 2001 & Troxel et al., 2013). However, wake after sleep onset alone was not predictive of effect in the latter study (Troxel et al., 2013). Finally, no studies analyzing early morning awakening as a predictor was identified. Overall, there is some empirical evidence for the symptoms of insomnia as predictors. Nevertheless, the results are mixed, and only a minority of the symptoms have been properly addressed in existing examinations.

For co-existing clinical constructs, such as functional impairment, comorbidities, and hypnotics analyzed as predictors and moderators, there is also some evidence available. Starting with depression symptoms, there are a number of trials available with mixed findings, where a couple of studies found no differences on a range of outcomes (insomnia severity, sleep efficiency, sleep onset latency, wake after sleep onset, total sleep time; Espie et al., 2008; Gagné & Morin, 2001; Jaap Lancee, van den Bout, van Straten, & Spoormaker, 2013; Manber et al., 2011). A corresponding number of studies founding depression symptoms to predict both a worse and better response on similar outcomes (ISI, wake after sleep onset, sleep onset latency, Pittsburgh Sleep Quality Index; Currie et al., 2002; Espie et al., 2001; Pruiksma et al., 2020; Troxel et al., 2013). Finally, a couple of studies also found depression symptoms to moderate a greater treatment effect on insomnia severity (Savard et al., 2016), as well as on sleep efficiency and sleep onset latency for CBT-I delivered over the internet with support, compared to no support (Lancee et al., 2014). The same pattern of findings is evident for symptoms of anxiety, where two studies found higher anxiety to predict lower insomnia severity (as indexed by Pittsburgh Sleep Quality Index) and wake after sleep onset, and lower anxiety to predict lower hypnotic use (Espie et al., 2001; Morgan et al., 2003; Troxel et al., 2013). In contrast, two studies found no predictive properties of anxiety on sleep efficiency (Espie et al., 2008; Gagné & Morin, 2001). The same pattern of mixed finding was true also for comorbidities (somatic or psychiatric), where a couple of studies could not find that comorbidities predicted or moderated outcome (Edinger et al., 2009; Espie et al., 2007; Troxel et al., 2013; Vincent et al., 2013), while others found psychiatric comorbidity to predict a worse outcome on insomnia severity (van de Laar et al., 2014), moderate the effect on fatigue (Vincent et al., 2013), and that somatic illnesses predicted higher sleep efficiency (Gagné & Morin, 2001). Finally, one study also found that those with comorbidities achieved a better response when they underwent full CBT-I as compared to undergoing CT and BT (Bélanger et al., 2016). For sleep aids, the evidence is limited and mixed, with a couple of studies showing neither a correlation with nor prediction of insomnia severity or sleep efficiency (Espie et al., 2001; Gagné & Morin, 2001; Troxel et al., 2013; Van Houdenhove et al., 2011), and one study showing that the use of sleep aids predicted an endpoint of sleep onset latency below 30 min (Currie et al., 2002). Finally, for the constructs referred to as functional impairment and quality of life, there is little evidence available, with one study examining both constructs, showing that higher daytime functioning at baseline predicted a better response on daytime functioning and on sleep quality, and lower levels of physical health-related quality of life predicting a better response on insomnia severity (Van Houdenhove et al., 2011).

For the theoretical processes studied as predictors or moderators, there is some existing evidence available, mostly regarding full CBT-I. Starting with

dysfunctional beliefs proposed by the cognitive model to maintain insomnia, there are a couple of studies reporting that those who experienced more dysfunctional beliefs before initiating full CBT achieved a better response to treatment (Edinger et al., 2008; Espie et al., 2001; Jansson-Fröjmark & Linton, 2008; Montserrat Sánchez-Ortuño & Edinger, 2010). Simultaneously, there are more recent studies with up-to-date statistical procedures that have failed to identify dysfunctional beliefs as either predictors or moderators (Lovato et al., 2013; Van Houdenhove et al., 2011). As regards worry, two studies have reported reductions during treatment with CT and CBT (Harvey et al., 2007; Lorenz et al., 2019), and a third found that higher baseline worry was associated with lower insomnia severity at posttreatment and that worry was the strongest variable in a composite predictor of sleep enjoyment (Espie et al., 2001). However, in a recent study, worry did not show any predictive properties (Lorenz et al., 2019). For safety behaviors in the cognitive model, there are two studies of CT & CBT that have identified reductions of safety behaviors from pre to posttreatment; however, the most recent one could not identify any predictive value of safety behaviors at baseline on the outcome of treatment (Harvey et al., 2007; Lorenz et al., 2019). For the perpetuating factor, attention and monitoring for threats to sound sleep in the cognitive model, little evidence is available, with one open study of CT, indicating a reduction of monitoring from pre to follow-up (Harvey et al., 2007). Overall, there seems to be some evidence for the cognitive processes as predictors, although very limited, with only pre to post reductions for two of the processes, and the two analyzed as predictors yielding equivocal results.

For the behavioral processes, there is little evidence available for their role as predictors or moderators. Bed- and rise-time variability has been shown to decrease in CBT relative to controls (Edinger et al., 2001, 2009), and greater adherence to recommended bed- and rise-times has been associated with improved outcomes (Tremblay et al., 2009). However, no studies have yet found bed- and rise-time variability to predict or moderate outcome. For time in bed, a similar pattern is evident, with significant pre to post decreases as well as larger reductions compared to controls. However, no studies investigating TIB as a predictor variable have, to our knowledge, been reported. Finally, higher sleep efficiency and variability in sleep efficiency at baseline have been shown to predict larger improvements in sleep efficiency at outcome (Chan et al., 2017; Espie et al., 2007). In summary, so far very little attention has been given to examining the processes from the behavioral model as predictors or moderators of treatment outcome.

In all, there are a broad array of studies that have provided the field with knowledge on a broad range of potential predictive and moderating variables for CBT-I, but in some instances, also for BT. Although this offers a valuable evidence base for our theoretical and clinical understanding about what predicts and moderates CBT-I, there are still many unresolved issues regard-

ing this evidence base, ranging from methodological problems in specific studies to a lack of consistency between studies that prevents any solid understanding of what predicts and moderates the effect of CBT on insomnia.

To mention a few of these limitations, starting with the specific studies, the majority have incorporated insufficient sample sizes, which reduces the possibility of finding predictive properties in the examined variables. Second, there are, in general, few studies that try to replicate previous findings by examining the same variables as potential predictors/moderators on the same outcome, and when they do, they usually treat the moderator variable as statistically different or use a statistical procedure that prevents a full comparison and firm conclusion to be made. Furthermore, since some of the outcomes are related to adherence to treatment or symptoms of insomnia (e.g., sleep efficiency, sleep onset latency, wake after sleep onset, total waketime or sleep time, sleep quality, bedtime variability) rather than being a clear measure of a global outcome as in insomnia severity, this prevents us from identifying what predicts reductions in insomnia severity. Third, the majority of studies investigating predictors usually incorporate only a small set of possible variables, which prevents alternative variables from being considered when explaining the variance in outcome. Furthermore, since the choice of included variables in studies using more than one variable also varies considerably between studies, this also prevents solid conclusions being drawn about the field. Fourth, the majority of existing studies have tended to incorporate statistical methods such as dichotomizing either the predictor and/or the outcome that by modern guidelines are known to introduce bias and produce misleading results (Hayes, 2013; MacCallum et al., 2002), since they, on the one hand, lead to arbitrary categorization of a clinical continuum, and on the other hand usually build on the characteristics of the sample which changes from study to study, thus preventing replication. Fifth, and finally, despite their relevance to the understanding of insomnia, very few studies have focused on the theoretical processes that each therapy model describes to maintain insomnia as predictors/moderators. Also, when studies have included theoretical variables as possible predictors, they have usually, with one or two exceptions, only included one of the variables from the specific theory, thus failing to assess the predictive value of the theory from other variables of the same theory in that sample.

Gaining a more solid and thorough understanding of the individual variables that indicate whether treatment will succeed and for whom a specific treatment will be most effective could thus aid in: identifying whether CT and BT are differentially effective dependent on the baseline characteristics of the patient; increasing the number of responders and remitters by tailoring treatments; and identifying and focusing clinical research on populations in need of further interventions and treatment refinement.

Mediators

Having a better understanding of the relative efficacy of CT and BT on insomnia disorder and the patient characteristics that they depend on to be effective will probably aid in enhancing the efficacy and, to some degree, also our theoretical understanding of CBT-I. However, understanding the relative efficacy and the predictors and moderators of effect cannot aid in understanding how change comes about, and what process should be targeted in treatment to bring about change. This is a problem, since knowing through which process change comes about during treatment can both advance our theoretical understanding of why insomnia persists and help in optimizing treatments by guiding them to target the process of most relevance. It can also spare the patients from exercises that target processes of less importance (Kazdin, 2007).

To gain further understanding of what the necessary process is to bring about change in CBT-I, a couple of the previously described models of insomnia could be of relevance as they provide suggestions for processes to examine. One model that has received a substantial amount of support, and that also delineates testable processes for which there also exist established measures, is the cognitive model described above (Harvey, 2002; Hiller et al., 2015). One reason for examining the processes in the cognitive model as mediators of change in both CT and BT is that given their role in perpetuating insomnia according to the cognitive model, if they also turn out to be relevant as mediators in BT, this adds further support for their role in insomnia. A second reason, touched upon above, is the suggestion of the triple-R model, which proposes that the cognitive process, in contrast to the cognitive model, is instead subsequently affected by and changes as a result of the effects exerted by sleep restriction in BT. The reason this model becomes relevant for this thesis, is that the role of cognitive processes can be tested both as drivers of change in CT and BT according to the cognitive model, and as affected by or subsequently reduced by sleep restriction according to the triple-R model. Thus, testing cognitive processes in CT and BT as monotherapies could provide knowledge of the two models' diverging ideas on the role of cognitive processes in insomnia.

Although the experimental studies reviewed under the presentation of the cognitive model above have provided relevant support for how the cognitive processes are related to insomnia, these studies usually assess only one process at a time. Assessing only one of the processes at a time is a problem since it means that the analysis then fails to grasp information from other processes and the dynamic interplay over time between the processes and insomnia severity, as the cognitive model suggests. Furthermore, existing experimental studies have also tended to incorporate a symptom of insomnia as an outcome rather than a measure of insomnia severity. Finally, these experiments have focused on only a short time frame, one to three days or

evenings, in contrast to patients who have insomnia in which the condition evolves around several weeks and where daytime and nighttime symptoms interact and affect each other.

One way to more thoroughly and realistically assess the cognitive model as well as the processes that lead to a reduction in insomnia during CT and BT is to examine and compare how cognitive processes change during both CT and BT, and how such changes subsequently relate to or explain reductions in insomnia severity at outcome, referred to as examining mediators (Baron & Kenny, 1986; Kazdin, 2007). Besides validating and furthering our knowledge on how processes in the model are related to insomnia, mediation analysis can also provide information on which processes are important to target in order to bring about necessary change in both CT and BT.

Although the majority of previous studies have failed to take account of the full model and the temporal interplay between processes and outcome, there are already a couple of studies that have investigated the cognitive processes as mediators in CBT-I. The process that has probably received the most attention is dysfunctional beliefs about sleep, where six studies found that it mediated outcome (Chow et al., 2018; Espie et al., 2014; Harvey et al., 2017; Lancee et al., 2015; Norell-Clarke et al., 2017; Sunnhed & Jansson-Fröjmark, 2015), and one found it did not mediate outcome (Okajima et al., 2014). The evidence for worry as a mediator is even more limited. One study has found that a decline in worry was associated with reductions in insomnia severity (Sunnhed & Jansson-Fröjmark, 2014). Two studies has found that a decline in worry mediated reductions in insomnia severity at the outcome (Harvey et al., 2017; Lancee et al., 2019). Furthermore, although with only one study each, both sleep-related safety behavior and monitoring for sleep-related threats have also been shown to mediate reductions in insomnia severity (Harvey et al., 2017; Lancee et al., 2015). Taken together, there are a number of studies that provide some support for the cognitive model and underscoring the importance of each process in ameliorating insomnia.

Although available studies have provided valuable information on how change comes about during CBT-I, a couple of questions remain. First, since the majority of existing investigations only examined pre- to post-changes in both mediator and outcome or failed to take temporality fully into account in the analysis when a mid-treatment assessment was available, there are still difficulties in outlining the temporal and causal links among treatment, process, and outcomes depicted in the cognitive model. Second, since most of the previous analyses, except one (Harvey et al., 2014), have examined cognitive and behavioral techniques in combination as one treatment. This makes it difficult to discern the treatment mechanism of a specific treatment, i.e., CT or BT. Overall, this makes it hard to separate what treatment mechanism a specific treatment works through and if a change in one of the cognitive processes preceded change in outcome and thus truly mediated the effect

of treatment on outcome, or if it happened the other way around. A final problem with existing research is that only one of the reviewed studies assessed more than three of the proposed processes from the cognitive model simultaneously, which thus prevents potential explanations from unassessed variables to participate in explaining the outcome.

Thus, gaining a more thorough understanding of how the change came about by identifying the processes that led to a reduction in the insomnia complaint could help both in enhancing our theoretical understanding as well as in developing and focusing interventions on the relevant process of change to achieve reductions in insomnia.

Summary

Insomnia is a prevalent condition that inflicts a heavy burden on both society and the individual. CBT-I is the treatment of choice for insomnia, and although effective, little is known on what the necessary components are for CBT-I to be effective, what patient characteristics CBT-I depends on to be effective, or through which processes the implemented interventions in CBT-I lead to change. Therefore, gaining a further theoretical and clinical understanding of these issues could both further our clinical and theoretical understanding of CBT-I and provide the means to enhance CBT-I to reach those not yet responding or remitting from treatment.

Aim of the thesis

Based on the described background, the overall aim of this thesis was to advance our theoretical and clinical understanding of CBT-I by examining the two theoretical models of CBT-I's comparative efficacy, their predictors and moderators, as well as the processes that bring about change for each of the two models. The specific research questions to achieve this overall aim were divided over three separate studies and are detailed below:

Study 1
To explore the comparative effect of CT and BT against a WL on a broad range of outcomes associated with insomnia disorder, such as co-existing clinical constructs and treatment-associated constructs, e.g., treatment satisfaction, credibility/expectancy, therapist support, time investment, etc.

Study 2
To explore what patient characteristics treatment depends on to be effective by examining a broad range of baseline characteristics as predictors and moderators of CT and BT.

Study 3
To examine processes from the cognitive model as mediators in CT and BT.

Overview of how the empirical studies address each aim

The three specific research questions were, as outlined above, explored in three separate studies. All three studies were performed under the scope of one large ethically approved (reference number 2016/856–31), pre-registered, randomized controlled trial (clinicaltrial.gov (NCT02984670)). This trial had three arms: CT, BT, and a waitlist control group. In Study I, the focus was on the outcomes of CT and BT compared to the waitlist, and the comparison of effects between CT and BT. In Study II, baseline variables were analyzed as predictors and moderators of insomnia severity. In Study III, bi-weekly cognitive theoretical processes from the 10-week treatment in the randomized controlled trial were analyzed as potential mediators of outcome in both therapies.

In the display of the three empirical investigations below, Study I will contain the broad and general methodology concerning the full trial, including recruitment, screening, randomization, power calculations, ethical considerations etc., as well as the specifics for Study I. In Studies II and III, the methodology section will instead be focused on the specifics of the statistical analysis of predictors, moderators, and mediators since the other relevant information regarding the trial has already been presented in Study 1.

Description of empirical studies

Study I: Comparing internet-delivered cognitive therapy and behavior therapy with telephone support for insomnia disorder: a randomized controlled trial

Aim

Based on the lack of knowledge on the comparative effectiveness of the main therapies in CBT-I, the aim was to compare the effects of CT and BT against a waitlist on a broad range of outcomes associated with insomnia and undergoing treatment, e.g., treatment satisfaction, credibility/expectancy, time investment, etc.

Methods

Procedure and participants

Participants for this trial were recruited by advertisements in the daily press and on social media. To be able to participate, participants had to fill in a web-based questionnaire available on the study webpage as part one of the three screening stages. Those who fulfilled DSM-5 diagnostic criteria for insomnia, had sufficient resources to participate in internet-delivered treatment and who were not suffering from severe depression or suicidal ideation proceeded to the next stage. Stage two consisted of a semi-structured telephone interview that sought to verify that the web-based registered insomnia was not primarily due to: other disorders (sleep, mental or somatic), sleep-disturbing medications or unstable use of sleep medications, shift work, coffee or alcohol consumption, or conditions in the environment. Those proceeding to stage three had as a final step to fill in a seven-day sleep diary and achieve a minimum of 30 minutes or more of involuntary waketime on at least three of the seven nights.

To achieve sufficient power for detecting effect sizes of small magnitude ($f = 0.1$), a total of 219 participants were included and randomized to CT ($n = 72$), BT ($n = 73$), and a waitlist (WL; $n = 74$). The mean age of the sample was 52.5 years, and 73.1% were females. Of the total sample, 16.4% fulfilled the criteria for a psychiatric disorder and 24.2% for a somatic disorder.

Finally, 42.5% reported the use of hypnotic medications, and 45.7% reported the use of medicines for a somatic condition.

Ethical considerations

Since the three studies were based on a randomized controlled trial that involved two active treatments compared to a waitlist and involved participants fulfilling diagnostic criteria for insomnia disorder, this also meant that some potential ethical issues were present.

Some of the main ethical issues that could arise were related to the fact that the trial involved treatment and thus expectancy of relief from the condition while simultaneously randomizing participants to one of three groups. This meant that, before gaining access to treatment, a large part of participants were asked to wait for ten weeks or was allocated to a non-preferred treatment. Another potential ethical issue was that participants were asked to share private information about their health status, which requires data management that secures participants' anonymity. Furthermore, in a clinical trial like this, despite a rigorous screening, there still exists the fact that participants may fail to respond, improve or even deteriorate and therefore may be in need of other types of treatments.

To minimize the chance for distress associated with these ethical concerns, the research group designed the trial in the following way.

Concerning potential ethical issues with being randomized to the waitlist or a non-preferred treatment, these issues are, on the one hand, an inevitable part of a randomized trial and were partly addressed by participants being informed and giving informed consent to participate under these conditions. But to further minimize the potential distress associated with it, the following actions were taken. To secure that the waitlist group also were offered treatment for their insomnia, they were informed that after the 10-week waiting period, they could choose their preferred treatment, BT or CT, which were then provided with the same therapist support as those initially randomized to CT or BT. Furthermore, to handle the potential disappointment of not receiving their preferred treatment, participants were informed that, if they requested, they were granted access to the self-help treatment material of the other therapy after the final follow-up assessment.

In relation to the ethical risk of participants deteriorating or needing other treatments, this was handled, on the one hand, by providing clear guidelines to therapists for how to proceed should this problem come up, consisting of suggesting or remitting those in need to more suitable care facilities. Furthermore, since all researchers and therapists in the project were licensed psychologists or a master student at the end of their clinical training, this meant that the relevant competence for these kinds of assessments and referrals were readily available for all personnel in contact with participants.

Finally, the fact that the trial prompted participants to share information about their health and their sleep problem meant that data needed to be

properly managed in a way that secured anonymity. In the project, this was secured by implementing a couple of methods for data management. Anonymity was handled by assigning each participant with a study code and allowing participants to choose a contact name. Furthermore, all assessments and data related to each participant were handled through a secure online platform that required a two-factor identification (Vlaescu et al., 2016), which ensured that anonymity and data were safely stored during the whole trial. Finally, all the abovementioned ethical concerns were also handled by the informed consent, where participants were fully informed about the trial and aspects of the trial that could affect them, and which they agreed to accept as part of their registration for the trial.

Treatments

Treatments were delivered in a self-help format via the internet with one module a week over ten weeks. The material was presented in PDF files that contained all the necessary information for participants to be able to apply the techniques by themselves. Besides the PDF files, participants also interacted with their therapist by registering their exercise practice and having a weekly 15-minute telephone call for feedback and problem-solving on exercises.

The BT arm consisted of sleep restriction, stimulus control, and sleep hygiene. These three components of BT were introduced sequentially during the 10-weeks of treatment in the presented order above. After their initial presentation, the application of each technique was assessed in terms of compliance and relation to outcome, and sleep restriction was adjusted based on the previous week's sleep efficiency. (For a treatment outline over the ten weeks, see Table S1 in supplemental materials for Study I).

The cognitive therapy (e.g., Harvey, 2005; Harvey et al., 2014, 2007) arm in this trial was based on the growing evidence that insomnia could result from the following maintaining processes: (1) unhelpful beliefs about sleep, (2) sleep-interfering or sleep-related worry, (3) attentional bias and monitoring for sleep-related threat, (4) misperception of sleep, and (5) safety behaviors. Hence, the aim of CT is to normalize these processes that have an maintaining influence on insomnia daytime and nighttime symptoms by cognitive restructuring, achieved by Socratic questioning of beliefs and behavioral experiments (Harvey, 2005; Harvey et al., 2007).

Measures and assessments

To assess the impact of the three arms on the insomnia condition, the ISI (Bastien, Vallières, & Morin, 2001; Morin, Belleville, Bélanger, & Ivers, 2011) was used as the primary outcome and was assessed bi-weekly from pre- to posttreatment. To further grasp the effect of treatments on patients with insomnia, a set of secondary measures was also included – assessing both nighttime and daytime symptoms, e.g., sleep onset latency, wake after

sleep onset, early morning awakening, functional impairment, quality of life, and symptoms of depression and anxiety, delivered pre- and posttreatment as well as at follow-up for the daytime symptoms.

Remission and response were defined based on the ISI as a score below eight points at posttreatment for remission, and a change of eight points during treatment for a response (Morin et al., 2011).

Statistical analyses

To investigate the study's aim, latent growth modeling with random effects (person-specific trajectories) was used to model individual change as a function of group (Bollen & Curran, 2006). Two primary outcome analyses were performed. The first sought to investigate the immediate effects of the two therapies compared to the WL during the active treatment phase, and the second to compare the two treatments on all measurement points to the six-month follow-up, including the sleep diary measures from pre- to posttreatment. Both analyses were fitted using full information maximum likelihood estimation with non-normality robust standard errors using Mplus vs. 7.4 (Muthén & Muthén, 2017). Rates of response and remission, as defined by ISI under measures section, were analyzed from pre- to posttreatment and from pretreatment to six-month follow-up, using logistic regression holding pretreatment scores on ISI constant.

Results

Both CT and BT outperformed the waitlist with moderate to large effect sizes on the majority of outcomes; see Figure 4 for a display of the changes in ISI between groups over the 10-week treatment. No significant differences emerged between CT and BT, except for sleep onset latency and adverse effects. Participants in BT had a six-minute shorter sleep onset at posttreatment compared to CT, and 43.2% in BT experienced adverse effects as a result of treatment, compared to 14.1% in CT.

Figure 5 shows the rates of response and remission at post and at follow-up for all three groups. Both treatments achieved a significantly larger proportion of treatment remitters and responders compared to the WL at posttreatment, and no significant differences emerged between the CT and BT.

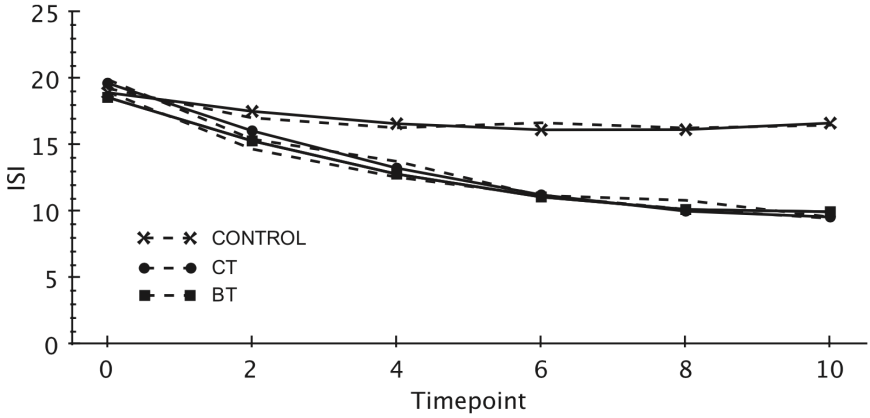


Figure 4. Observed and estimated means for the bi-weekly measurements on ISI.

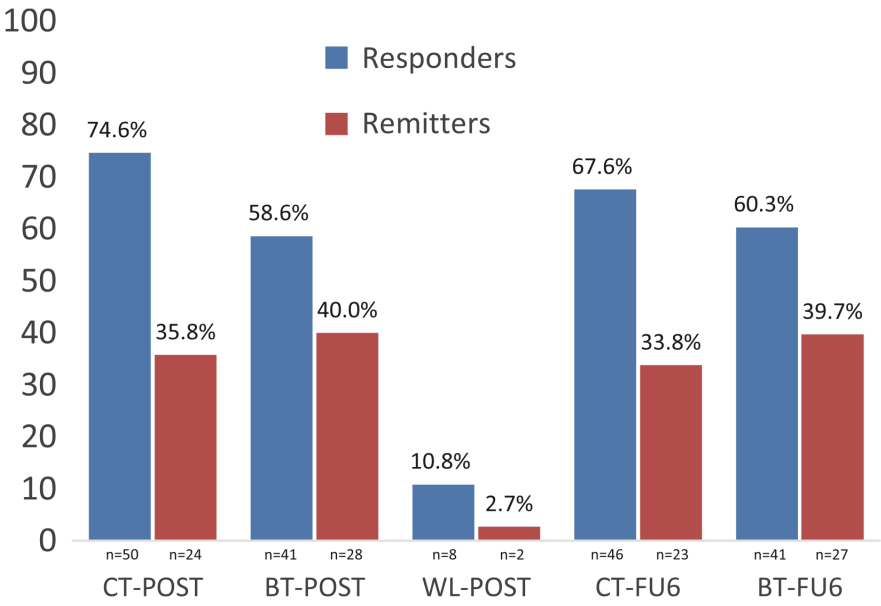


Figure 5. Percentage response and remission based on ISI (observed means).

There were also some significant differences regarding the participants' experience of their treatment, in that participants in BT stated that they invested more work in treatment, while participants in CT stated that they spent more time on treatment and that their treatment was more interesting. Finally, participants in CT received significantly more minutes of telephone support.

Conclusions

CT and BT produced comparable effects on the majority of outcomes and outperformed the WL. The results indicate that internet-delivered CT and BT are comparably effective as standalone therapies for insomnia disorder. The results highlight the need for future research to further examine what the necessary therapy and subcomponents are for achieving a sufficient effect on insomnia disorder.

Study II: Predictors and moderators of cognitive therapy and behavior therapy for insomnia

Aim

Based on the methodological limitations of previous research and the lack of consistency in both the design and results of previous examinations, and the almost non-existent examinations of predictors and moderators of CT and BT as monotherapies, our aim was to explore a broad range of insomnia-associated baseline characteristics as predictors and moderators of CT and BT.

Methods

Based on the data collected in the clinical trial described under Study I, this study employed multiple regression performed in a structural equation model framework using Mplus (Muthén & Muthén, 2017) to analyze proposed baseline variables as possible predictors or moderators of ISI.

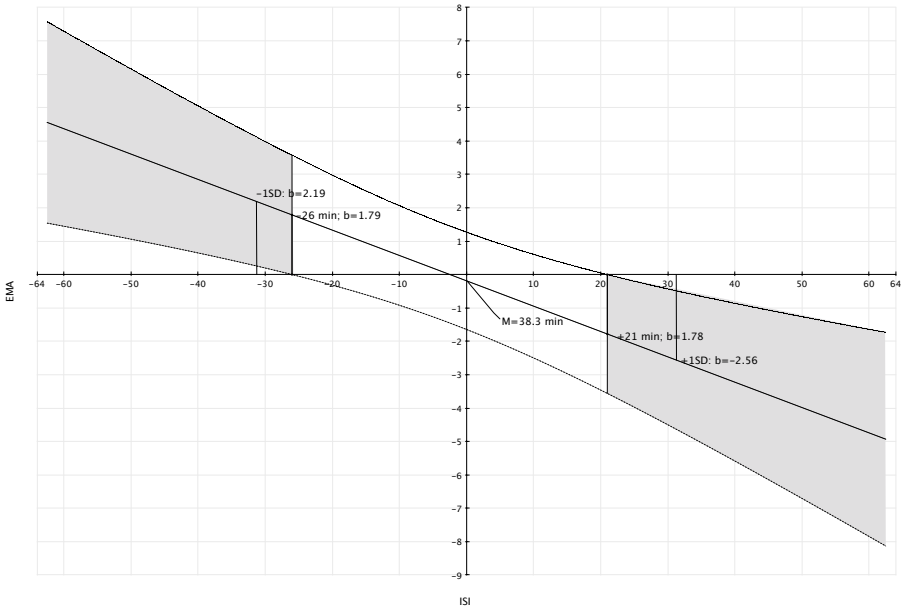
Potential predictor variables included insomnia symptoms (e.g., sleep onset latency, wake after sleep onset, early morning awakening, total sleep time), insomnia-associated constructs (comorbidities, hypnotics, quality of life, depression and anxiety symptoms), and theoretical process variables described by each therapy model to maintain insomnia (e.g., worry, bedtime variability, dysfunctional beliefs, etc.).

Each regression model contained ISI as the posttreatment outcome, and the following variables as predictors: a dummy coded group variable (CT=0, BT=1), one of the proposed baseline predictor variables (mean-centered), and the interaction term between the group and the proposed mean-centered predictor (e.g., the moderator). The mean-centered value of ISI at baseline was also included as a covariate to control for individual differences in outcome at baseline (Hayes, 2013).

Results

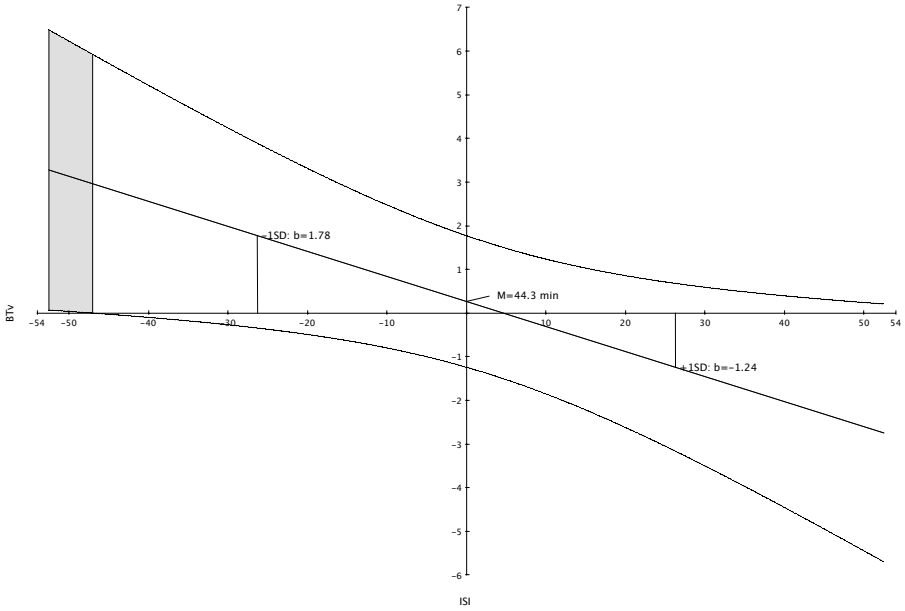
The results showed that both early morning awakening and bedtime variability moderated the effect of both therapies (see Figures 6 and 7 for details). The moderating effect demonstrated that BT was more effective when there was more involuntary waketime in the morning, and more variation in the time participants went to bed. However, the opposite was true for CT, which was more effective when there was little to no problem with waking up before risetime and little existing variation in the time participants went to bed at baseline.

Figure 6. Plot of simple slope indicating differences in outcome on ISI between CT and BT at different minutes of involuntarily early morning awakening (EMA), and regions of significance.



Note. When the slope is above zero on the y-axis, the difference in outcome is in favor of CT in that it symbolizes how much higher ISI participants scored in BT compared to CT at each level of the moderator. When the slope is below zero, the difference is in favor of BT in that it symbolizes how much lower ISI participants scored in BT compared to CT at each level of the moderator. Displayed at 1 SD below and above are the differences between CT and BT expressed as an unstandardized effect size in the original metric of the scale. The gray regions indicate a significant difference between the conditions (confidence intervals do not include zero).

Figure 7. Plot of simple slope indicating differences in outcome on ISI between CT and BT with different amounts of bedtime variability (BTv), and region of significance.



Note. When the slope is above zero on the y-axis, the difference in outcome is in favor of CT in that it symbolizes how much higher ISI participants scored in BT compared to CT at each level of the moderator. When the slope is below zero, the difference is in favor of BT in that it symbolizes how much lower ISI participants scored in BT compared to CT at each level of the moderator. Displayed at 1 SD below and above are the differences between CT and BT expressed as an unstandardized effect size in the original metric of the scale. The gray regions indicate a significant difference between the conditions (confidence intervals do not include zero).

Furthermore, longer waketime after sleep onset and higher insomnia severity at baseline predicted greater insomnia severity at posttreatment, while the opposite was true for sleep efficiency, where greater sleep efficiency at baseline predicted lower insomnia severity at posttreatment.

Conclusions

This study provided evidence of two variables as moderators of effect for both CT and BT, and three predictors of outcome for both therapies. The results indicate that therapies and perhaps components of CBT-I could be used to tailor insomnia treatments based on individual baseline characteristics, and thus match therapy to patient features in order to optimize outcomes.

Study III: Mediators of cognitive therapy and behavior therapy for insomnia disorder: a test of the processes in the cognitive model

Aims

The aim of Study III was to examine the role of processes from the cognitive model as mediators in both CT and BT.

Methods

Based on data collected in the clinical trial, processes from the cognitive model of insomnia (worry, dysfunctional beliefs, monitoring, and safety behaviors) were analyzed as mediators of treatment outcome (ISI) in CT and BT. To address important issues in prior mediation analysis, a rigorous test of mediation was set out by applying two statistical models that aimed to determine two criteria of mediation. Criterion (a) was evidence that CT or BT had an effect on one of the cognitive process, and that this effect also statistically accounted for some amount of the variation in ISI at outcome. This was determined by the use of parallel process growth modeling (Cheong et al., 2003). Criterion (b) was evidence that the within-subject change on one of the cognitive process systematically also predicted subsequent symptom change, rather than the other way around. This was determined by applying random intercept cross-lagged panel models (Hamaker et al., 2015). Together, these provided a more robust test of mediation.

Results

Based on criterion (a), dysfunctional beliefs, monitoring, and safety behaviors were identified as mediators of the treatment effect in both CT and BT, which thus indicated that they could account for the controlled effect of both treatments on the outcome. However, this was not evident for worry.

Based on the second criterion (b), it was confirmed that dysfunctional beliefs and monitoring (approaching sig.) predicted subsequent change in ISI for CT. For BT, a reversed association was found, in that ISI instead predicted subsequent changes in two processes (monitoring and worry), while for the remaining two, safety behaviors and dysfunctional beliefs, a reciprocal relationship was evident, in that outcome and process subsequently predicted each other. It is also worth mentioning that the predicting effect of safety behaviors on ISI, and the predicting effect of ISI on worry and monitoring, was greater in BT compared to CT. Indicating that safety behavior was a stronger or more important mediator in BT compared to CT, and ISI reduction to more strongly reduce worry and monitoring in BT compared to CT.

In comparison, the predicting effect of unhelpful beliefs and monitoring on ISI was not statistically different between the two therapies.

Conclusions

The strong test set out for examining cognitive processes as mediators in CT, and BT yielded support for dysfunctional beliefs and monitoring as drivers of change in CT. The findings also provided support for safety behavior as a mediator of BT, although this finding was not as clear cut, since there was a reciprocal pattern of prediction between process and outcome. Together, the findings underscore the importance of targeting dysfunctional beliefs, monitoring, and safety behaviors in the treatment of insomnia. The findings also highlight the value of these concepts in conceptualizing and understanding insomnia disorder.

General discussion

The aim of this thesis was to advance our theoretical and clinical knowledge on the treatment of insomnia with cognitive and behavioral techniques, with the ultimate goal of understanding the necessary components, their predictors of effect, as well as the process of change, to better tailor and prescribe treatment components with the highest likelihood of effect for a specific individual.

In this thesis, this aim was addressed by using one large randomized clinical trial consisting of three arms, which compared the main therapies in CBT-I, CT, and BT as separate treatments against a waitlist control. Study I focused on and examined the comparative efficacy of both therapies against the waitlist control. Study II explored whether the participant's baseline characteristics could predict or moderate the treatment outcome in CT and BT. Finally, Study III investigated processes of change during CT and BT by exploring cognitive processes assessed during treatment as mediators.

The overall result emerging from these analyses was that both CT and BT outperformed the waitlist and had comparable effects on the majority of outcomes, indicating that both CT and BT are effective as standalone therapies for insomnia disorder. It was also shown that the effects of CT and BT differed depending on the amount of early morning waketime and variability in the bedtime patients experienced at baseline. More specifically, those with more waketime and bedtime variability experienced a greater insomnia severity reduction in BT compared to CT, and the other way around, where those with less waketime before rising and bedtime variability experienced a greater reduction of insomnia severity in CT compared to BT. Finally, the results also revealed that reductions in unhelpful beliefs about sleep and monitoring for sleep acted as drivers of insomnia severity reductions in CT, whereas for BT, reductions in two processes, safety behavior and unhelpful beliefs, predicted reductions in insomnia severity, as well as the other way around.

The results add to existing knowledge and to the theoretical and clinical understanding of CBT-I by implying that both treatments could be used as separate therapies for insomnia and that clinicians could be more flexible when choosing which treatment to use. The findings also imply that the two therapies of CBT-I may suit certain individuals better than others and could perhaps, based on individual differences, be tailored in order to achieve an

optimal effect. Finally, the results highlight the value of targeting unhelpful beliefs, monitoring, and safety behaviors in order to reduce insomnia severity.

Answers to the research questions

Concerning the relative efficacy of therapy components

Study I was based on the lack of knowledge about the unique and comparative efficacy of the individual components of CBT-I. Its aim was, based on this gap, to further our understanding of the unique efficacy of CT and BT by comparing them as separate treatments against a waitlist on a broad range of outcomes in a randomized controlled trial. To our knowledge, this was the first study to compare CT and BT against a waitlist and the first to test CT delivered over the internet.

The main finding was that both therapies outperformed the waitlist and produced comparable effects on most of the assessed outcomes, with no significant differences between them, besides one notable exception on sleep onset latency. Two other significant findings of interest were that the two treatments differed in the amount of telephone support that patients received, with those in CT receiving more, and the number of adverse effects, with those in BT experiencing more.

The results are in line with previous research (Harvey et al., 2014), indicating that both BT and CT are comparably effective as standalone treatments, with effect sizes similar to larger than previous findings. In contrast to the previous study (Harvey et al., 2014), there were no differences between the treatments in the primary outcome between posttreatment and follow-up. A new finding was that participants in BT experienced significantly more adverse effects compared to CT, which could perhaps be understood from previous research showing that total sleep time (objective and subjective) decreases initially during sleep restriction in BT (Kyle et al., 2014). Also new was the finding that CT seems to demand more therapist support. An unexpected finding was that, overall, the two treatments achieved identical outcomes despite their differences in theory, techniques, and procedures.

The results add to previous research by indicating that CT and BT are comparably effective as standalone therapies when delivered over the internet and when compared to a waitlist. The results also add to previous research by indicating that the two treatments can differ in their effect on nighttime symptoms (although of small magnitude), their need for support, the number of adverse effects experienced, as well as in how participants perceive each treatment.

The overall answers from Study 1 to the thesis aim of furthering our understanding of the unique efficacy of CT and BT is that, so far, both therapies, overall, seem to be comparably effective in ameliorating insomnia severity, as well as the broader range of symptoms associated with the disorder. Thus, both therapies seem to be potent routes to treat insomnia, which raises the question of whether it is necessary to always provide both in treatment.

Concerning predictors and moderators

Study II grew from the lack of solid knowledge on how individual differences affect the outcome of either full CBT-I or its separate therapies. The aim was, therefore, to assess and explore a large number of baseline variables as possible predictors and moderators of treatment outcome in CT and BT delivered as separate therapies.

The main findings were that participants' waketime before rising, and their amount of bedtime variability moderated the effect of both treatments. The findings showed that CT was more effective when there was little bedtime variability and waketime before rising, and BT was more effective when the opposite was true.

Furthermore, insomnia severity, waketime after sleep onset, and sleep efficiency came out as significant predictors of insomnia severity reductions, showing that the greater the insomnia severity and waketime at pretreatment, the lower the reduction on insomnia severity at posttreatment, while the opposite was true for sleep efficiency, where greater sleep efficiency at baseline was associated with larger reductions in insomnia severity at posttreatment.

To our knowledge, neither early morning awakening nor bedtime variability had ever been examined as predictors or moderators, and so both the examination and the results for these constructs are new for the field. At the same time, it was somewhat surprising that none of the other nighttime symptoms or related constructs from the behavioral theory predicted or moderated the therapies.

One reason why early morning waketime was revealed as a moderator and not the other nighttime symptoms might be the high mean age of our sample, which has been shown to be more commonly linked to sleep maintenance problems rather than sleep onset problems (Pillai et al., 2015). A second reason might be that both therapies target sleep onset latency and wake after sleep onset by reducing worry and arousal, but only BT targets early morning awakening by its upward regulation of the homeostatic sleep pressure. This proposal is in line with the hyperarousal model (Riemann et al., 2010) of insomnia. This model suggests that insomnia is due to a 24-hour hyperarousal that is downregulated by homeostatic sleep pressure around bedtime, but as the pressure is attenuated with sleep during the night, the

hyperarousal again takes over in the early morning and makes a patient with insomnia wake up. However, since BT targets sleep pressure by restricting time in bed, this might be the mechanism that suppresses hyperarousal in the early morning and in so doing it reduces early morning awakening and insomnia severity.

A reason as to why bedtime variability turned out as a moderator and not risetime variability, might be, on the one hand, that BT more explicitly targets bedtime variability in comparison to CT, and, on the other hand, that risetime variability is generally harder to mold due to contextual factors (e.g., regular rise times due to work or small children). An unexpected result was that none of the cognitive processes turned out to be important for predicting outcome, given that CT explicitly targets them, and thus, according to theory, a high level of cognitive processes could hypothetically also indicate a probability of an effect with CT. One reason for this, in line with the triple-R and the cognitive model (Harvey, 2002; Maurer et al., 2018), might be that cognitive processes are effectively targeted by both therapies.

The finding that greater insomnia severity and waketime after sleep onset predicted greater insomnia severity at posttreatment was in contrast to previous studies that identified a reverse relationship, where greater severity predicted less severity at posttreatment (Morgan et al., 2003; Pruiksma et al., 2020; Savard et al., 2016; Troxel et al., 2013; Van Houdenhove et al., 2011). The reason why greater insomnia severity predicted more severity at posttreatment is hard to explain. One explanation could be that our relatively high baseline value of ISI, combined with the high mean age in our sample, might indicate that there are other comorbid concerns (e.g., somatic disorders) associated with insomnia that CT or BT does not target. The second finding that waketime after sleep onset turned out to be an overall predictor might be explained by the fact that both treatments are quite strong in targeting sleep onset, but neither of them is particularly potent when it comes to directly managing waketime after sleep onset, and thus, neither of the two therapies has enough efficacy to ameliorate this nighttime symptom. The third finding that greater sleep efficiency predicted lower insomnia severity was in line with one previous finding (Espie et al., 2007), but was unexpected from the behavioral theory that views low sleep efficiency as indicating excessive time spent in bed, which is a perpetuating factor that treatment aims to increase.

The results from Study II add to previous research by showing that the main therapies of CBT-I (BT and CT) can differ in their efficacy, depending on differences in the presenting complaint at baseline. These results shed light on the fact that CBT-I components may depend on different characteristics to be effective, and the results could, if replicated, provide a guide for the future tailoring of treatments.

The answer from Study II to the overall and specific research questions for this thesis is first that the therapies and perhaps components of CBT-I

can depend on different baseline characteristics to be optimally effective, and specifically, that CT seems to work better for patients with lower nighttime distress, whereas for BT the opposite seems to be true. This provides indications that the two treatments of CBT-I may suit subgroups of the insomnia populations differently, which, if valid, means that CBT-I could be tailored to individuals based on their presenting complaint, perhaps with an enhanced outcome as a result. Overall, this highlights the notion that variability in the presenting insomnia complaint perhaps indicates that an optimal treatment also needs to include various interventions for an optimal effect to occur.

Concerning mediators

Study III was conducted due to the lack of knowledge about how the change observed during treatment unfolds, i.e., what mechanism or process drives or is responsible for the observed change at the outcome after CBT-I. The aim was, therefore, to examine and test the role of the processes proposed to perpetuate insomnia in the cognitive model as mediators of outcome in both CT and BT.

The main findings from the parallel process growth model indicated that three of the four cognitive processes: dysfunctional beliefs, monitoring, and safety behaviors, could act as mediators in both CT and BT. For the cross-lagged panel model of mediation, the pattern of mediation differed between the two treatments, showing that unhelpful beliefs about sleep and monitoring were influencing insomnia improvements in CT, thus supporting the temporal order of change as depicted in the cognitive model. For BT, on the other hand, it was the other way around, with reductions in insomnia severity predicting reductions in worry and monitoring, thus supporting the view that these processes are not drivers of change in BT, but rather are reduced by previous reductions in insomnia severity. For the two remaining processes analyzed as mediators in BT, safety behaviors, and unhelpful beliefs, a reciprocal relationship emerged; thus, both insomnia severity and these processes predicted the subsequent change in each other. Furthermore, the influence of safety behavior on reductions in insomnia severity was stronger in BT compared to CT. The same was true for the effect of insomnia improvement on worry and monitoring in BT. However, as regards the influence of unhelpful beliefs about sleep and monitoring on insomnia improvement, this was not statistically different across the two therapies.

These results are in line with the previous research (Harvey et al., 2017; Lancee et al., 2015) although more detailed in terms of specificity and temporal relations, and, in general, support the hypothesis concerning how insomnia is maintained in the cognitive model. The support for the cognitive model was in specific true for the result that reductions in unhelpful beliefs and monitoring seem to drive the reductions in insomnia severity during CT,

and to some degree also the result that reductions in safety behavior and unhelpful beliefs seem to mediate reductions in insomnia severity during BT, although these relationships were reciprocal.

However, the result that worry did not turn out to be a mediator in either of the two analyses was surprising given both its central role in the cognitive model and prior research indicative of worry as a mediator (Harvey et al., 2017; Lancee et al., 2019). One reason might be that our assessment, which used a shorter version of the original Anxiety and Preoccupation about Sleep Questionnaire (APSQ; Jansson-Fröjmark & Sunnhed, 2020), failed to capture certain important domains of worry and thus became too similar to the items in ISI that assess worry. Another reason might be that worry in the cognitive model needs to be re-conceptualized as an epiphenomenon of poor sleep rather than as a driver, as suggested by the findings in Study III that improvement in insomnia severity predicted reductions in worry. A third reason is that worry could, instead of influencing a broad construct as the ISI, influence more specific symptoms of insomnia, such as sleep onset latency (Gross & Borkovec, 1982). The result that safety behavior exhibited a stronger effect on insomnia reduction in BT compared to CT was also somewhat surprising in relation to the role of safety behavior in the cognitive model, but overall, it adds to the overall importance of reducing safety behavior to ameliorate insomnia.

The answer from Study III to the overall and specific research questions in this thesis is that unhelpful beliefs, monitoring, and safety behavior, in line with the cognitive model, seem to be important processes, both as targets during treatment to achieve a reduction in insomnia, and conceptually in the understanding of insomnia.

Results in relation to theoretical frameworks

This thesis focused on exploring the two theoretical models of CBT-I by examining their separate efficacy, their predictors and moderators, as well as what processes or mechanisms that are responsible for change in them. The thesis has besides providing the field with further knowledge about their separate efficacy, potential predictors and moderators of success, and processes of change, also added information that might be of relevance for the refinement of the two theoretical models.

Starting with the cognitive model, the findings that CT yielded similar results on the majority of outcomes compared to BT and outperformed the waitlist provide indirect support for the validity of the cognitive model and thus indicate that focusing on reversing maladaptive cognitive processes also yields an effect, not only on insomnia severity and daytime-related symptoms but also on nighttime symptoms. However, the finding from Study II that CT achieved greater effects on insomnia severity when nighttime-related

symptoms and processes were minor indicates perhaps that this model has the most potency when insomnia is less characterized by nighttime difficulties. Finally, the results from Study III, that unhelpful beliefs and monitoring came out as mediators that acted as drivers of subsequent reductions in insomnia severity in CT, clearly support the proposals laid out by the cognitive model and indicate both the importance of targeting these constructs in treatment for effect and their role in insomnia maintenance. However, the finding that worry was not identified as a mediator in either of the two mediation analyses contrasts with the cognitive model, and perhaps indicates that the role of worry needs to be re-conceptualized in the model.

For the behavioral model, the significant effect in Study I provides indirect support for the behavioral model in general. Also, the indications of a stronger effect in BT on nighttime measures (sleep onset latency, wake after sleep onset, and early morning awakening) could be viewed as further support for the BT model. Since BT is the therapy that explicitly focuses on the nighttime symptoms of insomnia, by its strategy to manipulate the homeostatic system and the circadian system to optimize sleep drive and timing of sleep to achieve more consolidated sleep and less waketime during the night.

Further support for the behavioral model, and specifically for the workings of the homeostatic system, was to some degree also evident in the findings from Study II, which showed that individuals who exhibited greater levels of early morning waketime achieved larger reductions of insomnia when they underwent BT as compared to CT. This supported the notion in the BT model that sleep restriction targets dysregulated homeostatic sleep pressure and, in this way leads to more consolidated sleep and subsequent insomnia reductions in BT. The same study also provided support for the role of the circadian rhythm in insomnia as proposed by the behavioral model, in that those exhibiting greater variabilities in their bedtime achieved a greater reduction in insomnia severity when they underwent BT as compared to CT, thus, supporting the proposal that insomnia might result from a dysregulated circadian rhythm that BT explicitly targets. Finally, the fact that safety behavior as a mediator had a stronger predicting effect on subsequent change in insomnia severity in BT compared to CT might be viewed as in line with the behavioral model of insomnia that through its application of sleep restrictions and stimulus control more thoroughly and consistently reduces the use of safety behaviors compared to CT, implying that sleep-related safety behavior could be a relevant process of change in BT.

The results from the three studies are also supportive of and can partly be explained by the Triple-R model (Maurer et al., 2018), mentioned under the behavioral model in the introduction. Specifically, the results in Study I that BT yielded comparable effects on daytime symptoms as the cognitive therapy despite its focus on processes related to the night by optimizing the homeostatic and the circadian system (Bootzin et al., 1991; Spielman, Saskin, et al., 1987), compared to CT, which more directly targets daytime symp-

toms and processes (Harvey, 2005), indicate that BT indirectly also targets daytime symptoms. These results are in line with and could partly be explained by the notion put forward in the triple-R model, which proposes that sleep restriction in BT leads to changes in both day and nighttime cognitive processes. These cognitive changes are proposed to result on the one hand by sleep restriction also constituting a behavioral experiment that challenges unhelpful beliefs and prevents safety behaviors, and on the other hand, that subsequent improved nighttime symptoms due to sleep restriction lead to less daytime fatigue and, therefore, less incentive for engaging in daytime cognitive processes such as worry, monitoring, and safety behavior (Maurer et al., 2018). This is a proposal supported by both previous studies of BT (Harvey et al., 2014) and the results from Study I in this thesis. Overall, implying theoretically and empirically that although BT focuses on the night, this therapy model is also of relevance for the daytime processes associated with insomnia. Furthermore, the results in Study III that worry and monitoring were driven by reductions in ISI, and that the predictive effect of ISI on worry and monitoring was stronger in BT compared to CT, are also in support of the proposal in the triple-R model that sleep restriction drives subsequent changes in cognitive processes. The same is true, although not as clear cut, for unhelpful beliefs and safety behaviors in BT that were predicted by prior reductions in insomnia severity, as well as the other way around.

In summarizing the theoretical implications for the field, this trial has provided evidence for the validity of both the behavioral and the cognitive model. Simultaneously the moderator and mediator findings are indicating that the two theoretical models may be more or less valid for different phenotypes or subtypes of insomnia disorder depending on the way the symptoms and theoretical processes are expressed, e.g., more or less nighttime problems or high frequency of safety behaviors or dysfunctional beliefs.

Clinical implications

The findings in this thesis, of course, need to be replicated to establish firmer implications. However, having said that, there are a couple of possible clinical implications that could be drawn, from each separate study, as well as, from the three studies taken together.

In starting with implications from the single studies, the findings in study 1, that both CT and BT outperformed the WL and were overall comparably effective, adds support to a growing body of evidence that both CT and BT are effective as standalone therapies, and in clinical terms, suggests that therapists could, based on their or the patient's preferences, choose more flexibly between one of the two treatments when initiating treatment for insomnia. However, having said that, it is important to keep in mind, that there are still only two efficacy trials with somewhat mixed findings. Fur-

thermore, it is still unclear whether the two efficacy trials' results would remain in other contexts, such as in primary care or a psychiatric outpatient facility. Therefore, this and the previous trial's effects need to be replicated in other contexts to fully grasp their effectiveness outside the research context. Moreover, it is important to keep in mind the design of the two therapies in this trial. This trial delivered both CT and BT over the internet for ten weeks. Thus, it was BT and CT packaged as a self-help format, delivered over the internet for ten weeks, that yielded these results. Thus, the treatments were quite long, which perhaps makes their clinical implications less scalable. Further, whether CT and BT, composed differently, i.e., using other methods for delivering or sequencing the components in each therapy or other delivery formats with different lengths of treatments would yield the same results, is still unknown. Therefore, future studies are needed before more flexible implications of these studies' results are possible in the outpatient clinics.

The findings in Study II, that early morning awakening and bedtime variability moderated CT and BT differentially, could build on the implications in Study I by guiding therapists to tailor the focus of treatment based on the presenting clinical picture at baseline, such as focusing more on implementing behavioral techniques (i.e., sleep restriction and stimulus control) when the insomnia complaint is more characterized by problems with early morning awakening and large variabilities in bedtimes, and more on cognitive techniques when these bed and nighttime issues are small. Such tailoring could make the treatment more time-efficient for the single patient by reducing the patient and the therapist's workload while simultaneously maintaining efficacy by focusing on the most prevalent symptoms or the symptom of most relevance. Although these results are in line with the theoretical model, it is important for clinical implication to keep in mind that this is the first study identifying these moderating relationships.

Finally, the findings in Study III that unhelpful beliefs and monitoring seem to drive reductions in overall insomnia severity in CT, and safety behaviors as an important mediator of insomnia reduction in BT, point to the relevance of continuously assessing and targeting these processes using techniques in CT for reducing unhelpful beliefs and monitoring, and using BT techniques for reducing safety behaviors, to effectively reach the goal of reducing insomnia.

For the three studies taken together, the finding may implicate to clinicians that there are now increasing evidence for the validity of two theoretical models and therapies that could be used to understand and manage insomnia, suggesting a wider frame for understanding, conceptualizing, and selecting appropriate treatment components for a specific individual sleeping complaint.

The three studies together also indirectly validate a larger battery of interventions as important routes for managing and changing insomnia, as well as

important variables to keep track of before and during treatment, for tailoring as well as for making sure that the treatment is instigating the important process of change necessary for achieving insomnia reduction. Together this increases the flexibility of the clinician to tailor their interventions for the specific individual's complaint.

Methodological strengths and limitations

Although the findings in this thesis are of value for and contribute to our theoretical and clinical understanding of what makes CBT-I work, they also carry some methodological limitations that are important to keep in mind for a proper interpretation of the results and for understanding what further research is necessary to advance the field.

Regarding the validity of the clinical trial in general

Starting with issues general to the whole clinical trial and thus applicable to all three studies, the participants for this trial were recruited via social media and a daily newspaper and thus consisted of interested and motivated individuals willing to put in the necessary effort (Davidson et al., 2009). This probably made the participants in this trial different from patients seeking regular care. The extent to which this influenced the participant's involvement, compliance, and ultimately treatment outcomes is hard to say, but previous research has found that these samples differ demographically in terms of being characterized by a larger number of females, being more highly educated and having a higher socioeconomic status (Kazdin, 2003). This was similar to the participants in our study, who were characterized by having a higher age, and of being highly educated. Overall, this may pose limits concerning the generalization of the results from this trial to the broader population. However, it should also be noted that the sample showed severe and longstanding issues with insomnia, with 42.5% of the sample reporting use of hypnotics and having suffered a mean duration of 11.7 years of the complaints they sought treatment for.

Second, the treatment in this trial was delivered via the internet with support over the telephone. The internet-delivered format meant that all techniques and exercises needed for the therapy to become active in targeting insomnia were presented and delivered in text format. This text format probably demands greater effort and self-discipline compared to face-to-face formats, an issue that, together with our educated sample, may pose a further threat to the generalization of our findings to other populations. However, in regard to this, it is noteworthy that prior research has, to this point, not delivered any clear evidence of differences in outcome between formats of delivery (internet vs. face-to-face; Andersson, Titov, Dear, Rozental, & Carlbring,

2019; Zachariae, Lyby, Ritterband, & O'Toole, 2016), perhaps reducing the significance of this as a limitation.

Third, although the majority of the assessments in this trial were digitally administered and were not associated with the therapist contact, thus minimizing the risk for responder bias and social desirability (Kazdin, 2003), they were all based on subjective self-reports from the participants. Although relevant, since the insomnia diagnosis is based on subjective complaints, this, however, prevents the possibility of generalizing findings from this trial to objective measures of sleep (i.e., actigraphy or polysomnography). Thus, whether the two therapies are comparably effective on nighttime symptoms of insomnia assessed objectively is still an open question. Furthermore, the use of the Mini International Neuropsychiatric Interview (Sheehan et al., 1997) for assessing comorbidities could be further criticized for being a screening rather than a diagnostic instrument, which may limit the diagnostic validity of the sample. Also, it is important to keep in mind that our assessment batteries made use of multiple sources for assessing insomnia (i.e., questionnaire, interview, and sleep diary), and this, combined with the fact that the insomnia diagnosis is based on subjective complaints, perhaps makes these limitations less troublesome.

Fourth, there was no registration of compliance with the telephone support manual, which could open up for the possibility of treatment contamination and therapist drift. However, it is worth keeping in mind that these issues were also automatically handled by the fact that treatments were delivered in text form with telephone support. Furthermore, the support call only focused on problem-solving of available content and was guided by a therapist manual for handling treatment integrity and risk for contamination, with supervision on demand.

Fifth, 15% dropped out of the treatments but remained in the study. Although no differences emerged in terms of dropouts between the treatments, and the rate of the dropout was in line with previous findings for CBT for depression (17.5%; Cooper & Conklin, 2015), this attrition may still hinder the interpretation of outcomes in this trial. However, it is worth mentioning in relation to this that the primary statistical analysis for analyzing the outcomes produces accurate estimates under a lenient missing data assumption and is considered state of the art (Schafer & Graham, 2002).

Regarding the identification of relative effect and necessary components

In addressing the validity of the results from the outcome analysis in Study I, some issues related to the way treatments were delivered, such as the length of treatment, the therapist support, and the question of the dose-response relationship, are important to mention.

The fact that the cognitive and behavioral treatments in this trial were ten weeks in duration and internet-delivered with telephone support may seem

unreasonably long and resource-consuming compared to an average of six weeks for full CBT-I and to fully automated versions of CBT-I. This, overall, raises questions on both generalization and the necessity of using a 10-week therapy to achieve a treatment response. However, to evaluate this properly, there are a couple of other issues that need to be considered. First, CT, as used in this trial, is a more comprehensive therapy than the cognitive interventions usually incorporated in CBT-I, and includes six to 22 sessions, with an average of eight and 17 sessions respectively (Harvey et al., 2007, 2014). Second, it is also important to mention that although shorter therapies are of relevance for effective clinical management, the focus of this study was on comparing two theoretically distinct models on equal grounds to evaluate their individual efficacy. Furthermore, when speaking about the length of treatment and the optimal dose of treatment, it is important to keep in mind that this is still a relatively unaddressed issue which briefer therapies alone cannot answer. Furthermore, the fact that the participants continued to improve for all ten weeks (see Figure 4) also indicated that no floor effect was reached, thus contradicting the suggestion that the therapies in this trial were unnecessarily lengthy. Overall, our ten weeks of treatment with telephone support seemed to produce encouraging results for CT and BT as standalone therapies, but whether these results can also be generalized to more automated and shorter versions of CT and BT is still an unanswered question.

Another potential threat to the interpretation of the results was the differences that emerged between the two therapies, with participants in BT experiencing significantly more adverse effects compared to those in CT, and those in CT receiving significantly more minutes of telephone support. Although interesting for the study aim, this may also have affected outcomes by participants in CT being affected by the extra attention rather than the proposed active components, and participants in BT being less compliant with important techniques due to experiencing adverse events.

Regarding the identification of predictors and moderators of effect in CT and BT

Study II contained several methodological strengths in its aim of exploring what predicts or moderates the effect in CT and BT for insomnia. One of those was the inclusion of a broad range of possible baseline predictors, specifically the theoretical processes proposed to maintain insomnia in both therapy models. Another was the application of statistical procedures in line with modern guidelines (Hayes, 2013). A third was the design of the clinical trial that made it possible to identify unique predictors of the separate therapies of CBT-I.

Although there are many strengths, there are also a couple of limitations that need to be addressed for a proper interpretation of the findings. First, the fact that the sample was characterized by being highly educated and of high age may pose a threat for the generalization of the findings, since these characteristics may also mean that this sample displayed a limited heterogeneity of insomnia symptoms (e.g., more problems with early morning awakening and less with sleep onset latency; Pillai et al., 2015), which thus prevented other symptoms from ending up as predictors or moderators.

Second, in terms of assessments and analysis, there were no objective assessments of sleep, and the analysis focused only on the prediction of one outcome. While using only one outcome may also be considered a strength due to greater power and parsimony of understanding and interpreting the outcome, it also prevents the possible detection of variables in our trial that could predict other facets of insomnia, e.g., improvements on sleep onset latency, early morning awakening or worry. Furthermore, the absence of objective measures of sleep, analyzed as both outcome or predictors, further prevents us detecting objective variables as predictors or moderators of insomnia severity or as being predicted by the analyzed variables in this study. Thus, whether bedtime variability and early morning waketime still moderate the outcome when measured objectively is still an open question.

A third limitation is an inflated risk for type 1 error due to a large number of analyzed variables. Although an important limitation, this also needs to be judged against the overall aim of exploring a large set of possible baseline variables in a trial comparing both CT and BT as separate treatments, which makes it possible to explore how these therapies may be differentially affected by baseline variables.

Regarding the identification of the process of change in CT and BT

Study III contained some important strengths that were new for the field, such as the repeated assessments of outcome and proposed mediator that enabled analysis to explore the temporal order of change between the mediator and outcome, and the use of cross-lagged panel model in addition to a parallel process growth model. A further strength worth mentioning was the design, which enabled the analysis to address specificity between treatment, mediator, and outcome.

That being said, there are also a couple of limitations of value to illuminate when interpreting the findings. First, the fact that CT and BT involved several components that were disseminated sequentially throughout the ten weeks, combined with our bi-weekly assessment, might also have impacted the testing of temporal precedence. That is, the impact of a potentially important therapeutic ingredient may have vanished by the time of the next assessment.

Second, in terms of the constructs used for assessing the mediators, the shorter scales for assessing worry and monitoring might have meant that certain key aspects of the constructs were missed and imply that these constructs assessed differently may not have yielded the same results. Furthermore, although the scales used for assessing the processes in this trial are well established empirically-validated constructs, it is still unclear whether the results would hold with other assessment methods, such as objective measures of the same processes, i.e., selective attention and monitoring assessed by attention bias tests (Jansson-Fröjmark et al., 2013).

Third, the inclusion of several constructs from one theoretical model was a significant strength. However, it is worth mentioning that there are also several other potential mediators for insomnia reduction, which could have changed the effect of the mediators on the ISI in this study, if included, such as sleep medication or bedtime variability.

Fourth, the use of a complex analytical model with several, both manifest and latent variables, opens up the possibility of bias due to systematic measurement errors. Furthermore, the smaller sample sizes in the analysis of the subgroups might also have had an impact on the stability of parameter estimates and model fit measures in the fitting of the more complex models, such as the multigroup models. A final limitation to consider is that there might be overlaps between process questionnaires and the outcome measure. The most obvious risk for overlap is perhaps the one between APSQ-2 and ISI, since the latter contains an item that directly assesses worry.

Implications for future research

In order to build on and further advance the theoretical and clinical understanding of CBT-I, there are a couple of implications of relevance for future research.

First, in terms of examining the comparative efficacy of CT and BT, future research could build on the results from Study I by exploring the comparative efficacy of CT and BT in other patient groups (e.g., of younger age, recruited from regular care, and with different symptom presentations). Future studies could also advance the field by adding additional measures (e.g., actigraphy, objective assessment of daytime symptoms), as well as by examining the dose-response relationship, long-term effects, and cost-benefit ratio in both therapies. Finally, it would also be of interest to dismantle and disentangle the comparative efficacy of the separate components inherent in CT and BT, as well as what moderates or mediate their efficacy.

Second, in terms of predictors and moderators, although Study II contained many strengths, future studies should aim to replicate these by examining other patient groups or by using larger samples with enough power and heterogeneity in their presentation of insomnia, to allow for different patient

characteristics to be revealed as predictors. Future studies could also confirm the validity of the moderator findings by randomizing participants high and low on bedtime variability and early morning waketime to CT or BT. Also important for future studies would be to examine whether the allocation of patients to CT or BT based on and relevant for their presenting complaint (high or low on nighttime symptoms or theoretical process) would achieve the same outcomes as this trial on a lower dose, i.e., six weeks of treatments. Finally, since CT more directly targets daytime constructs, it would also be of interest to include a broader assessment of such constructs as predictors, e.g., objective measures of function and cognitive impairments.

Third, the examination of mediators in Study III contained many strengths in terms of both design (i.e., allowing specificity) and analytical procedures (i.e., employing two tests of mediation, including time-lagged prediction). However, future studies could build on these findings by assessing and analyzing different time lags during treatment (e.g., every other day, or weekly), assessing and analyzing other processes, such as rumination (Carney et al., 2013), or objective measures of cognitive processes, such as attention bias (Harris et al., 2015). Furthermore, the cognitive processes examined here should also be investigated in treatments using full CBT-I to explore whether the combination of CT and BT triggers differing patterns of mediation as well as CT's and BT´s mediating effect on other outcomes (e.g., sleep onset latency or fatigue). Finally, future studies with similar research designs should also attempt to examine behavioral processes as mediators in order to further elucidate the processes through which both treatments achieve their effect.

Concluding remarks

The overall aim of this thesis was to advance our theoretical and clinical understanding of CBT-I by examining the main therapies' (CT and BT) comparative efficacy, their predictors or moderators, as well as what processes mediate their efficacy.

The main result was that both therapies outperformed the waitlist with only one significant difference in outcome, and thus both were effective as standalone therapies. Furthermore, early morning awakening and bedtime variability at baseline turned out to moderate the two therapies, and thereby provided knowledge on how differences in baseline characteristics predict the effect of the two therapies. Finally, dysfunctional beliefs and monitoring acted as drivers of change in CT and safety behaviors, and dysfunctional beliefs instigated change in BT.

These findings indicate that clinicians can choose more flexibly between the two therapies when initiating treatment and suggest that BT may be the treatment of choice when bedtime variability and early morning awakening are more pronounced, while CT may be better when they are low. The findings also underscore the value of clinicians assessing and targeting dysfunctional beliefs, monitoring, and safety behavior throughout treatment as a means to ameliorate insomnia.

Theoretically, these findings provide new information on the way we understand insomnia by showing that the two therapies can be differentially effective depending on the baseline characteristics of the patient. The findings also provided support and illuminated the potentially different roles of cognitive processes in BT versus CT (drivers of insomnia reduction versus driven by insomnia reduction) and underscored the value of unhelpful beliefs, monitoring and safety behaviors in understanding insomnia maintenance.

These findings could be of relevance to both the society and the individual patient. The fact that patients and clinicians can choose more flexibly between the two therapies could reduce the workload of both patient and therapist and enable therapists to more flexibly choose based on individual preferences and levels of bedtime variability and early morning awakening. The support of unhelpful beliefs, monitoring, and safety behavior as important processes of change can aid in optimizing therapy and reducing the patient workload by guiding clinicians to focus on the most relevant processes.

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