UNDERSTANDING TREATMENT RESPONSE IN CBT-I: EXPERIENTIAL AND THEORETICAL PERSPECTIVES

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Abstract

Understanding Treatment Response in CBT-I: Experiential and Theoretical Perspectives

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Cognitive behavioural therapy for insomnia (CBT-I) is empirically supported as efficacious and is considered the first line treatment for insomnia. However, it is not clear which specific components of CBT-I are most strongly associated with most improved outcome, and for whom the treatment is most effective. The present study examined participants' experiences with CBT-I in terms of the treatment components that they found to be most helpful. Participants with chronic insomnia (N = 163) completed 4 sessions of in-person CBT-I in a clinical trial. At the end of treatment, participants completed "letters" to their future selves including reminders of which elements of treatment they found to be most helpful; these letters were analyzed from a middle range theory approach using qualitative analyses. Although some studies have investigated moderators and mediators of CBT-I, these studies are few and sparse, and there are a variety of methodological problems. In response to the demand for an investigation of treatment mechanisms in CBT-I, the current study investigated a number of presumed theoretical treatment mechanisms on CBT-I outcomes via mediational analyses. The relationship between pre- and posttreatment insomnia severity was expected to be mediated by a number variables based factors presumed to perpetuate insomnia in the long-term. Qualitative analysis yielded eleven themes of treatment components that participants found to be most helpful. The eleven

themes reflected the theoretical tenets of CBT-I, but also showed that self-efficacy, which is not currently prioritized in the CBT-I literature as a significant factor impacting treatment, to be important. Results from the quantitative analysis showed that sleep compression partially mediated prospectively measured insomnia severity at posttreatment, while changes in sleep-related safety behaviours mediated self-reported insomnia severity. Findings from this study show that from their own perspective, clients confirm that the theoretical treatment factors upon which CBT-I is based are indeed helpful, and also that additional, less widely studied variables related to CBT-I, therapy more generally, and broader client concerns are important. Implications from this research include important considerations for clinicians delivering CBT-I, and potential new avenues for future research directions.

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Dedication

I would like to dedicate my dissertation to my grandparents, and all those in my family who have sacrificed, worked, and dreamed, so that I might have the opportunity to access education and ultimately, study and work in this field. You are in everything I do.

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Introduction

Cognitive behavioural therapy for insomnia (CBT-I) is an empirically supported treatment (e.g., Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Espie et al., 2001; Morin et al., 1994; 2006; Harvey et al., 2014) and is the recommended intervention for insomnia disorder (Morgenthaler et al., 2006; Qaseem et al., 2016). While there is considerable support for the efficacy of CBT-I in the treatment of insomnia, there is little understanding as to *why* this intervention is successful, and for whom it is most effective. An important next step in the development of CBT-I is to better understand its nature as a treatment. What is it about CBT-I that leads to improved outcomes? Which components of the intervention are most effective in successfully treating insomnia?

We have a great deal of data to support the theoretical factors that are presumed to cause and perpetuate insomnia disorder, and it is upon these theoretical factors that CBT-I was based and developed as an intervention. That is, there are a number of variables that are targeted by CBT-I, and changes in these variables over the course of treatment are presumed to lead to improved outcomes. However, we know little about what processes of change are truly taking place for clients, and what they view to be the most important aspects of treatment that helped them to improve or cope with insomnia symptoms. This study thus posits the question: What do clients view to be the most essential, beneficial ingredients of the treatment? This study investigated factors reported by study participants who received the CBT-I intervention as most helpful, as well as a number of presumed (theoretical) treatment mechanisms in relation to posttreatment outcomes. The goal of this study was to distill the most important, effective components of the intervention, from both client and theoretical perspectives.

There is a growing call for mechanistic studies in the psychological treatment literature broadly, fueled at least in part by an increasing need to understand the key tenets of interventions that are associated with greater likelihood of improvement and recovery. A mechanism is considered to be a variable that explains the "processes or events that are responsible for the change" that occurs during an intervention (Kazdin, 2007, p.3). That is, mechanisms provide information about how and why the change occurred.

A 2012 review of the CBT-I literature showed that most treatment studies have yet to include analyses of mechanisms of treatment response, and suggested that researchers in the field focus their attention on examining mechanisms in both primary and secondary analyses of CBT-I treatment trials (Schwartz & Carney, 2012); this recommendation has more recently been echoed by other researchers in the field (e.g., Johnson et al., 2016; Vitiello, McCurry, & Rybarczyk, 2013). The present study addressed this gap in the literature by investigating a number of presumed mechanisms associated with insomnia and CBT-I as a treatment, and investigating the extent to which these factors relate to treatment outcomes. More specifically, presumed mechanisms of treatment response were examined by querying participants about what they found to be most helpful and analyzing responses via qualitative analysis, as well as through quantitative mediation analyses.

It is important to note that the variables presumed to function as mechanisms treatment response were not manipulated in the current study, and therefore influential factors cannot be interpreted as causal. In other words, participants were not randomized to different levels of the mediator variables. Nevertheless, the mediational analyses in the current study do provide improved understanding of variables that likely function as mechanisms of response compared to past research in that we not only examine the relationship between the intervention and

hypothesized mediator variables, but also assess the association between the mediators and therapeutic change by looking at change over time, per Kazdin's guidelines (2007). That is, whether the presumed mediating variables (M_k) mediated the relationship between pretreatment insomnia severity (independent variable *X*) and posttreatment insomnia severity (dependent variable *Y*) was assessed in the current study. Insomnia severity was evaluated in two different ways: total wake time, as assessed by prospective monitoring (the Consensus Sleep Diary; Carney et al., 2012), and self-reported retrospective monitoring (the Insomnia Severity Index; Morin, 1993). Although participants were not assessed on the variables of interest *continually* throughout the treatment (i.e., at each of the 4 treatment sessions), important change information was gleaned from the changes in their scores between pre- and posttreatment, providing an enhanced account of change over time in relation with treatment outcomes, as compared with static variables assessed at only one time point. That is, data from the present study emphasize the variables that most likely account for change over the course of CBT-I.

This research also provides insight into which treatment components clients found to be most helpful by analyzing open-ended letters written by study participants and reporting on this subject matter; these "experiential factors" thus illuminated other potential mechanisms of action responsible for outcomes in CBT-I. In sum, this study provided perspectives from participants regarding their treatment experiences, and began to untangle the theoretical, empirically supported elements of CBT-I that are associated with best response to treatment. Results from this study will help to provide treatment targets in order to further improve the already excellent efficacy of this intervention by clarifying differences and similarities between what we presume to be most important theoretically for CBT-I outcomes, versus what clients actually understand to be most helpful about the treatment. This increased understanding of processes of change

within CBT-I will also be informative for clinicians in terms of continued enhancement of posttreatment maintenance of gains and relapse prevention strategies.

Insomnia Disorder

Insomnia is a disorder of sleep that involves difficulty with initiation, maintenance, and/or subjective quality of sleep (Ohayon, 2002; Pigeon, 2010; Roth & Ancoli-Israel, 1999). It is notable that the most recent iteration of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5; American Psychiatric Association, 2013) includes the presence of distress or a complaint of impaired daytime functioning as a diagnostic criterion for insomnia disorder; insomnia is no longer considered to be a phenomenon specific to nighttime or a person's main sleep period, but is rather defined as a constellation of symptoms that cause distress or impairment throughout the 24-hour period. Insomnia is highly prevalent in the general population, more so than many other mental health concerns. Evidence from epidemiological studies suggests that the prevalence is between 19 and 30 percent of the adult population in North America (Leger, Guilleminault, Dreyfus, & Delhaye, 2000; Morin, LeBlanc, Daley, Gregoire, and Mérette, 2006; Roth et al., 2011). It is also a highly comorbid disorder, frequently co-occurring with medical difficulties (e.g., high blood pressure, chronic pain, cancer, heart disease; Roth et al., 2011; Stein, Belik, Jacobi, & Sareen, 2008; Suk, Yoshida, & Sugimori, 2003; Taylor et al., 2007) and other mental disorders (e.g., anxiety, depression, post-traumatic stress disorder; Mallon, Broman, & Hetta, 2002; Ohayon & Shapiro, 2000; Perlis, Giles, Buysse, Tu, & Kupfer, 1997; Pigeon et al., 2008).

Insomnia is associated with a variety of deleterious outcomes for individuals with the disorder, such as degraded quality of life and impaired functioning (Kyle, Morgan, & Espie, 2010; Ohayon, 2002). It is also burdensome from an economic perspective, causing strain for

healthcare systems. The combined direct and indirect annual costs associated with insomnia were found to be approximately $6.6 billion in the province of Québec a decade ago (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009), and have undoubtedly continued to rise since then. In the United States, the financial burden of insomnia has been calculated to be as much as $100 billion (Fullerton, 2006). Beyond insomnia specifically, the high prevalence and deleterious outcomes associated with sleep problems more generally have been labeled a serious public health epidemic that necessitates immediate attention (National Center for Chronic Disease and Prevention and Health Promotion, Division of Adult and Community Health, 2014). To mitigate these concerning consequences and costs, the continued development and refinement of effective treatments for insomnia is crucial.

Cognitive Behavioural Therapy for Insomnia

Cognitive Behavioural Therapy for Insomnia (CBT-I) was developed as an intervention that targets both the nighttime and daytime symptoms associated with insomnia (Edinger & Means, 2005; Edinger & Carney, 2008; 2015). It is considered to be the gold standard for nonpsychopharmacological intervention for insomnia. There is considerable evidence supporting its efficacy in the treatment of insomnia disorder (e.g., Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Espie et al., 2001; Johnson et al., 2016; Manber et al., 2008; Morin, Culbert, & Schwartz, 1994; Morin, Bootzin, Buysse, Edinger, Espie, & Lichstein, 2006). Indeed, compared to pharmacological intervention, research has accrued showing that CBT-I is just as efficacious in the short-term, and even more efficacious for maintaining gains in the long term (for review, see Riemann & Perlis, 2009).

Studies of CBT-I outcomes have included a variety of medical and psychiatric comorbidities, and the accumulated evidence suggests that CBT-I is also successful in treating

populations with more complex diagnostic profiles (for review, see Taylor & Pruiksma, 2014). This is critical for considering the potential effectiveness of a treatment in the "real world" (i.e., outside the lab environment), where clients rarely present with a single diagnosis or difficulty. For example, Manber and colleagues conducted an RCT examining the efficacy of CBT-I in a sample of individuals with comorbid depression, and results suggested significant improvement in insomnia symptoms at posttreatment (Manber et al., 2008). CBT-I has also been found to be efficacious in treating insomnia amongst individuals with a comorbid post-traumatic stress disorder diagnosis (Talbot et al., 2014) and comorbid chronic pain (Jungquist et al., 2010). A meta-analysis of randomized controlled trials (RCTs) of CBT-I in individuals who have recovered from cancer found large effect sizes for subjective insomnia severity at posttreatment; moreover, there was evidence that these gains were maintained over time (Johnson et al., 2016). Indeed, mounting evidence suggests that the improvements made in CBT-I intervention are longitudinally robust (e.g., Castronovoa et al., 2018; Espie, Inglis, & Harvey, 2001; Espie et al., 2008; Morin, Colecchi, Stone, & Sood, 1999; Sivertsen et al., 2006).

In addition to the benefits offered by CBT-I by way of efficacious insomnia treatment, it is also an intervention that may be administered in short-term treatments (e.g., 4 sessions) and delivered via a variety of modalities (e.g., online, in groups); it is therefore is a promising treatment in terms of cost-effectiveness (Johnson et al., 2016). In sum, the extant research has coalesced to show that CBT-I is an efficacious and cost-effective means of addressing insomnia, a prevalent health concern associated with many deleterious health, societal, and economic costs.

Although the efficacy of CBT-I is well established, it remains the case that we lack a clear understanding of the specific factors that determine the success of this treatment. Indeed, Vitiello and colleagues posited that continued studies examining the efficacy of CBT-I should no

longer be a priority for sleep researchers; rather, research focus should turn to an investigation of the most efficacious *components* of CBT-I, with the aim of potentially strengthening or refining these particular elements of the treatment in order to further increase the overall efficacy of the intervention (Vitiello et al, 2013).

There is a range of different techniques used in CBT-I, which are presumed to target the putative underlying factors that have been shown to perpetuate chronic insomnia in the long term. These factors can be understood from the theoretical framework advanced by Spielman and colleagues, who developed a model of insomnia based upon predisposing, precipitating, and perpetuating variables involved in the development and maintenance of insomnia (Spielman, Caruso, & Glovinsky, 1987). This diathesis-stress model describes factors presumed to relate to one's predisposition to develop insomnia (e.g., genetics, perfectionism, tendency to ruminate), onset of insomnia (e.g., loss of job, spousal separation), as well as variables that may be linked to the maintenance of the disorder in the long run. Cognitive behavioural therapy for insomnia was developed based on these latter perpetuating factors outlined by Spielman and colleagues. Targeting these theoretical perpetuating factors is the goal of CBT-I, and it is via changes on these indices that we presume positive treatment outcomes are achieved.

The key behavioural strategies recommended within the CBT-I protocol include stimulus control (refraining from attempting sleep until the body is sleepy, arising from bed if wakefulness is prolonged, using the bedroom or bed only for sleep and for sex) and sleep restriction (compressing the time period spent in bed to the average total amount of time spent sleeping, plus 30 minutes to account for normal wakefulness; avoidance of daytime napping). The main cognitive strategies include cognitive restructuring of inaccurate and maladaptive beliefs about sleep and daytime functioning, and developing strategies for coping with worry and

rumination about symptoms of insomnia. Sleep hygiene recommendations are also addressed as an educational component of the treatment, and are emphasized particularly when problematic behaviours (e.g., excessive caffeine or alcohol intake) may perpetuate insomnia symptoms. Additional optional components include implementation of daytime behavioural activation and relaxation strategies. While CBT-I as a whole has been shown to be efficacious, and many of these various individual components appear to have a positive impact on treatment outcomes and are preferred by clients (e.g., Harvey, Inglis, & Espie, 2002), the way in which the particular CBT-I components are tied to treatment response have not yet been untangled.

The American Academy of Sleep Medicine recommended a combination of the following treatment components as efficacious for insomnia intervention: stimulus control, sleep restriction, cognitive therapy, and relaxation training (Morgenthaler et al., 2006). An earlier study of client-reported use of 10 CBT-I components indicated that stimulus control/sleep restriction was the best predictor of improvement (shorter SOL and WASO indices), and found that cognitive restructuring was also important (Harvey et al., 2002). Interestingly, participants in this study endorsed relaxation strategies as the most used strategy at home; however, implementation of this treatment component was not associated with posttreatment gains in this sample.

In accordance with recent recommendations for research in this field (e.g., Johnson et al., 2016; Schwartz & Carney, 2012; Vitiello et al., 2013), the current study investigated whether CBT-I is efficacious in relation to its presumed mechanisms, and the relative impact of these presumed mechanisms on treatment response. The study also acquired important insight into client perspectives on key ingredients of the intervention, and examined those factors associated with improvement. The goal of this research is to develop a clearer understanding of the reasons

why CBT-I is effective at bringing about change in insomnia difficulty, and which of these helpful components are most essential for improving posttreatment outcomes. Results from this study may be harnessed to refine therapeutic techniques within CBT-I, as well as the treatment protocol more specifically. It will also provide critical information regarding pretreatment client characteristics that may be associated with better or poorer outcomes, thereby providing suggestions for future research to explore specific techniques or modifications tailored to subsets of individuals with insomnia disorder.

Variables Presumed to Impact Treatment Response

There are a number of theoretical mechanisms presumed to relate to treatment response in CBT-I, many of which were outlined in a recent review (Schwartz & Carney, 2012). As a protocol, CBT-I targets the factors that are presumed to underlie insomnia disorder. These factors are best understood within the context of the previously described theoretical model (Spielman et al., 1987), which is the basis for our understanding of the development and maintenance of insomnia. It is the perpetuating factors that CBT-I targets in order to affect symptom improvement; accordingly, these maintaining factors are expected to emerge as significant mediators of treatment response in the present study. The key theoretical mechanisms of CBT-I, outlined previously by Schwartz and Carney (2012), were examined in the present study, and are discussed below.

Theoretical perspectives. Based on the factors that are presumed to underlie the predisposing, precipitating, and perpetuating factors in insomnia disorder, there is a large body of research examining the theoretical mechanisms of CBT-I as a treatment. There are number of specific behavioural, cognitive, and state variables that have been examined in the literature, and

found to mediate CBT-I outcomes. These variables will be examined as likely mechanisms of treatment response in the current study via mediational analyses.

Behavioural variables. A number of behavioural variables are presumed to influence treatment response in CBT-I.

Time in bed. In an effort to retrieve sleep lost as a result of insomnia difficulties, many individuals augment their time spent in bed the next day. Other behaviours that fall under this behavioural category include napping during the day, going to bed early (i.e., even if sleepiness is not present), and sleeping in or lingering in bed in the morning past the normal rise time (Edinger & Means, 2005). Although these behaviours seem rational given the perceived sleep detriment, they are in fact actions that perpetuate insomnia difficulties in the long run by disrupting the body's natural sleep architecture (i.e., the structural organization of sleep, as it cycles through the various stages of sleep, including rapid eye movement and non rapid eye movement stages; Colten & Altevogt, 2006). For example, spending excess time in bed compared to how much time is spent asleep leads to an overall detriment to sleep quality, as the natural pattern of sleep cycles is disrupted.

To assess whether a CBT-I client is spending too much time in bed, the aforementioned behaviours can be monitored in daily sleep diaries and computed to attain a single estimate of an individual's total time in bed (TIB) over many nights. Increased TIB is strongly associated with insomnia maintenance, and is targeted by CBT-I (Carney, Edinger, Meyer, Lindman, & Istre, 2006; Edinger & Means, 2005). Specifically, TIB is addressed by the stimulus control and sleep restriction components of CBT-I, with the general goal of reducing wakefulness in bed and forcing consolidated sleep to occur during a specified, compressed sleep window.

A study examining client-reported compliance with a number of CBT-I components found that completion of the stimulus control and sleep restriction recommendations was the best predictor of improvement at posttreatment (Harvey et al., 2002). More recently, research has provided evidence that reduced TIB over the course of insomnia treatment is associated with improved outcomes (e.g., Krystal & Edinger, 2010; Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001; Rybarczyk, Lopez, Benson, Alsten, & Stepanski, 2002). Accordingly, it is hypothesized that changes in TIB from pre- to posttreatment will emerge as a mechanism of treatment response (i.e., greater reduction in TIB should be related to greater improvements at posttreatment).

Variability in rise time. Amongst other important functions, the human circadian system is responsible for regulating our sleep-wake cycles (Webb, 1988). Because highly variable routines disrupt the body's natural, 24-hour circadian rhythm, variable bed and rise times have been advanced as factors that perpetuate chronic insomnia (Bootzin, 1972), and are therefore targeted by CBT-I. The human circadian system governs many functions in the human body, including sleep. In the brain, the central circadian pacemaker that is responsible for these rhythms is the suprachiasmatic nucleus (Keefe & Turek, 1985; Klerman, 2005). The circadian system functions in cycles of approximately 24 hours in duration, and disruption to the circadian sleep system can occur through a variety of changes to one's diurnal patterns (e.g., timing of meals, social activities, and exercise; light exposure). With regard to sleep, circadian disruption can occur when there is disturbance to the internal timing system, or misalignment between the timing of sleep and an individual's daytime activities (Barion & Zee, 2007; Klerman, 2005). Disruption of the circadian system can lead to sleep difficulties because this system determines our general sleep window (e.g., tending towards morningness or eveningness) and also is

responsible for the timely secretion of hormones and chemicals associated with sleep onset, such as melatonin (Lack & Lushington, 1996). Therefore disrupted circadian functioning can lead to atypical patterns of sleepiness and wakefulness, and this can in turn lead individuals to engage in behaviours that perpetuate insomnia symptoms (e.g., going to bed earlier and rising later, napping during the day), as previously discussed.

During treatment, the client's average total sleep time is assessed; from this number, a sleep schedule that is agreed upon by both client and therapist is prescribed (Edinger & Carney, 2008; 2015). Past research found that pretreatment sleep schedule variability was a significant predictor of adherence to the prescribed bed time (BT) and rise time (RT) during treatment, which presumably impacts client response (Maich, Lachowski, Burnett, & Carney, 2015). Further, past research has showed that higher sleep schedule variability mediates CBT-I outcomes (e.g., Edinger et al., 2009), and increased sleep schedule consistency is related to improved posttreatment outcomes (Tremblay, Savard, & Ivers, 2009). It was hypothesized that reduced RT variability over the course of treatment (i.e., between pre- and posttreatment) would mediate treatment response in the present study. Although past research has found significant associations between BT adherence and variability (e.g., Maich et al., 2015), and reducing sleep schedule variability is targeted by CBT-I (and inherently involves consistency in BT and RT), there are notable difficulties in analyzing BT as a mediator or mechanism. We only investigated RT variability in the current study, and not BT variability, for reasons discussed below.

Although CBT-I clients are given a sleep schedule prescription that includes both "earliest bedtime" and "latest rise time," it is unclear how best to examine BT as a variable related to either treatment adherence or treatment response. Within the CBT-I protocol, a sleep schedule is decided upon based on data from an initial (pretreatment) sleep diary. Specifically,

clients' average total sleep time (TST) over 2 weeks is calculated, and the TIB window is determined by adding 30 minutes to this average TST (to account for normal wakefulness in bed, for example during the sleep onset period). Once the total number of hours spent in bed is established, client and therapist discuss a sleep schedule that is feasible and reasonable, with consideration for the client's daytime schedule (e.g., work and family commitments) and chronotype (i.e., within the circadian system, tendency towards a preference for and alertness during either morning or evening hours, or in more pronounced cases, an advanced (morning "larks") or delayed ("night owls") circadian rhythm (McCany & Lee, 2000).

After the sleep schedule has been prescribed, it is then recommended that clients adhere to this sleep schedule by attempting sleep *no earlier* than the assigned bedtime, and rising from bed (typically, through use of an alarm clock) *no later* than the prescribed rise time. These instructions are consistent with stimulus control rules, which involve a dissociation of the bed from wakefulness. That is, to properly follow stimulus control guidelines, if a client attempted sleep at their assigned bedtime and sleep was not produced within a reasonable time window (i.e., approximately 20 minutes), the recommendation within CBT-I is that the client arise from bed and engage in a pleasurable, nonarousing activity (e.g., knitting, watching a movie) until they become sleepy. Similarly, if a client were not sleepy at their assigned bedtime, stimulus control rules would indicate that the individual should not attempt sleep at that time, in order to avoid unnecessary wakefulness in bed. In this instance, the adherent client would remain awake, enjoying relaxing activities outside of the bedroom, until sleepiness arose. Herein lies the main predicament in developing a suitable method for evaluating BT as a treatment mechanism: a reduction in BT variability may in some instances actually suggest that the client is *not* adhering to stimulus control recommendations, if they are consistently going to bed at the prescribed BT,

but do not feel sleepy when doing so. In this case, if the client continued with this BT pattern over the course of CBT-I treatment, we would not necessarily expect to see improvement in their insomnia symptoms at posttreatment. On the other hand, an increase in variability may not necessarily be indicative of treatment adherence; for example, an individual might sometimes be waiting until their prescribed bedtime before attempting sleep, while on other evenings attempting sleep much earlier than recommended. Thus it is virtually impossible to determine whether BT variability, and changes in BT variability, is indicative of adherence or nonadherence to CBT-I's stimulus control recommendations. Results from such analyses would be difficult to untangle without a day-by-day sleep diary assessment of stimulus control adherence over the course of the 8-week treatment. In sum, BT variability is a poor indicator of adherence due to its inherent lack of clarity as it pertains to stimulus control, a key intervention targeting putative treatment mechanisms. It was therefore deemed not to be a useful variable to incorporate into the present study's analyses, as results from such analyses would be inherently convoluted for the aforementioned reasons and impossible to interpret in a meaningful way. For these reasons, BT variability was not included as a presumed factor impacting treatment response in the current study.

Rise time variability, however, is a putative treatment mechanism that can be analyzed statistically and interpreted with relative confidence. A reduction in RT variability is a key aim in CBT-I that targets a number of factors that are presumed to maintain insomnia in the long-term. Specifically, adherence to a consistent rise time is a treatment goal that targets: prolonged time in bed by eliminating oversleeping in the morning, feelings of fatigue during the day that may be related lower levels of daytime activity, and finally, disruption of the circadian rhythm that occurs when the sleep schedule varies greatly (e.g., between weekday and weekend

mornings). It could be argued that the RT variability factor might be problematic for reasons similar to BT variability; for example, an individual may be consistent in their RT, but in fact be consistently rising from bed at a time later than the agreed upon prescription (e.g., setting the alarm later than the prescribed RT; setting a second alarm).

To address such a situation, it is helpful to consider an average client, most of whom have a relatively consistent home schedule, whether this schedule relates to their own employment, education, or responsibilities; a partner or spouse's routine; or the routine of children, parents or housemates with whom the client lives. Indeed, the CBT-I protocol in its original format is not recommended for administration to individuals who work on rotating shifts, such as emergency healthcare providers, and those engaged in rotating shifts are not eligible for the present study as a result of their unpredictable schedules. In other words, the vast majority of study participants must awaken at a set hour in the morning some or most days during the week; it is generally based on these morning commitments that the original sleep schedule prescription is derived during CBT-I. A participant's RT, then, is likely to be relatively consistent (e.g., variable within approximately 1 hour) on those mornings when they are obligated to fulfil various commitments (e.g., being at work by 9 a.m.). Variability in RT, which is indicative of nonadherence and presumed to be related to poorer outcomes at posttreatment, is thus likely to arise on days when the individual has no or fewer obligations (e.g., weekends, holidays) or days when sleep has been particularly poor. Such a scenario would, of course, involve greater average RT variability over the course of a two-week monitoring period, whereas low RT variability would suggest that the participant was adhering to RT recommendations even when outside obligations did not require adherence. Further, the non-CBT-I commitments that most participants have would make nonadherent RT consistency less tenable. In other words, in order to consistently rise from bed

much later than the sleep schedule prescription, a participant would have to accept being consistently late for outside commitments, such as being on time for their work. In summary, we can assume that decreased RT variability is indicative of adherence to CBT-I recommendations, and accordingly, related to improved outcomes at posttreatment. RT variability was anticipated to be much less problematic than BT variability for analysis and interpretation in the present study.

Safety behaviours. Maladaptive coping behaviour is another presumed factor that perpetuates insomnia chronically. Sleep related safety behaviours are generally considered to be unhelpful overt or covert coping behaviours performed by individuals in order to try to control their sleep, or to prevent poor sleep and its negative consequences from occurring (Hood, Carney, & Harris, 2011). Within Harvey's cognitive model of insomnia, rumination about the negative consequences of a poor night of sleep (see "tendency to ruminate," below) is thought to lead to the adoption of safety behaviours aimed at improving sleep (Harvey, 2002), which in turn serve to further entrench maladaptive beliefs about sleep. For example, an individual with difficulty falling asleep might begin to use alcohol as a means by which to decrease sleep onset latency (i.e., fall asleep faster), by increasing nighttime drowsiness. However, this strategy would backfire in the long-run, as evidence shows that alcohol interferes with sleep quality over the course of the night, promoting middle-of-the night awakenings and disrupting one's natural sleep rhythms (Roehrs & Roth, 2001). Another example would be relying on sleep medications to ensure sleep onset at bedtime, which might lead to heightened cognitive arousal and difficulty sleeping without medication use. Individuals often continue to engage in sleep related safety behaviours because they are helpful for reducing their short term distress about insomnia and its consequences. However, these safety behaviours function to maintain maladaptive beliefs and

behaviours that perpetuate insomnia in the long term, presumably by reinforcing threat based appraisals of how catastrophic and how likely poor sleep and the negative consequences of a poor sleep would be (Harvey, 2002; Hood et al., 2011).

Since Harvey's (2002) model of safety behaviours was advanced, the extant research has supported the notion that the implementation of coping or "safety" behaviours is a factor that maintains insomnia (for review, see Harvey, 2005). In addition, greater insomnia severity is significantly related to higher reported need to employ safety behaviours (i.e., perceived utility of such behaviours) by individuals with insomnia (Hood et al., 2011). We expected that change in reported safety behaviour usage from pre- to post-CBT-I treatment would mediate insomnia outcome (i.e., reduction in safety behaviours would mediate improved insomnia severity at posttreatment).

Cognitive variables.

Sleep effort. Sleep effort, a construct that involves an endeavour to control one's sleep (Broomfield & Espie, 2004), and has been posited as a cognitive factor that may maintain insomnia in the long-term (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). This effort may manifest behaviourally (e.g., delaying bedtime to avoid possible insomnia experiences) as well cognitively (e.g., becoming anxious at bedtime, worrying about the consequences of insomnia), (Ree & Harvey, 2004). This increased effort to achieve sleep makes sense given that sleep is necessary to maintain human life, and acquiring sleep on a regular basis is important for optimal daytime functioning; thus loss of sleep or a perceived inability to produce sleep may be deemed a threatening prospect (Broomfield & Espie, 2004; Harvey, 2002). However, increased sleep effort tends to increase arousal and vigilance, given that the possibility of failing to obtain sleep is considered threatening, which in turn makes sleep production less

likely. Furthermore, sleep is not a voluntary process. It is largely involuntary, and as such efforts to control sleep are generally unsuccessful (Broomfield & Espie, 2004). Thus, sleep effort is considered to be an important factor that may perpetuate chronic insomnia, and is targeted in CBT-I treatment.

Given the likely importance of sleep effort in the maintenance of insomnia difficulties, it is a viable mediator of treatment response. Indeed, changes in sleep effort have been observed post-CBT-I treatment (Ong, Shapiro, & Manber, 2008) as well as after the administration of a sleep restriction-only intervention (Kyle, Morgan, Spiegelhalder, & Espie, 2011). One study also noted that increased scores on a measure of sleep effort after completion of CBT-I increased risk of insomnia relapse at 1-year posttreatment (Ong et al., 2008). Although these preliminary results show promising support for the importance of targeting sleep effort in CBT-I, additional research examining the extent to which sleep effort functions as a mechanism of treatment response is recommended (Schwartz & Carney, 2012). Accordingly, in the current study changes in sleep effort over the course of CBT-I intervention were hypothesized to function as mechanisms of response on indices of insomnia severity.

Tendency to ruminate. Individuals with insomnia tend to ruminate about their symptoms during the day (Carney et al., 2010; Kohn & Espie, 2005); CBT-I targets this ruminative process in order to reduce distress about insomnia symptoms during the day. Past research has shown that pretreatment ruminative tendency moderates treatment response; specifically, greater pretreatment ruminative tendency predicted improvements on sleep indices at posttreatment (Edinger, Carney, & Wohlgemuth, 2008; Maich, Bogouslavsky, & Carney, 2016). If the tendency to ruminate functions as a pretreatment moderator of treatment outcome, it may also be a viable mediator of response. It makes sense that an individual may engage in increased

rumination about their sleep if their insomnia severity is heightened, and that reduced rumination during CBT-I treatment may lead to reduced symptom severity. The current study investigated the extent to which ruminative tendency functions as a treatment mechanism. That is, we explored whether changes (i.e., reductions) in the tendency to ruminate about insomnia symptoms were associated with improvements in posttreatment outcomes.

State variables.

Fatigue severity. Fatigue during the daytime is considered to be a feeling of low energy, and is a common complaint amongst individuals with insomnia disorder (Hossain et al., 2005; Riedel & Lichstein, 2000). However, poor sleep is only one of many factors that may contribute to feelings of fatigue during the day. Other contributors may include anxiety and depression (Greenberg, 2002), lack of or over-activity (e.g., Puetz, O'Connor, & Dishman, 2006), boredom (Grandjean, 1979), or iron deficiency (anemia; Sobrero et al., 2001), amongst others. However, research has shown that individuals with sleep difficulty often consider fatigue to be exclusively caused by poor sleep the previous night (e.g., Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). Such misattribution of the causes of fatigue may function to perpetuate insomnia in the long run by increasing the sense of anxiety about, and pressure to improve, one's sleep quality, thereby creating or augmenting cognitive arousal in anticipation of the sleep period (e.g., Espie, 2002; Harvey, 2002). Targeting fatigue over the course of CBT-I may improve sleep-related misattributions of fatigue (as found by Harris et al., 2012), and therefore improving self-rated fatigue may function as a mechanism via which insomnia improves. In the present study, change in fatigue severity between pre- and posttreatment was expected to mediate CBT-I treatment outcome.

Depression and anxiety. Research has long supported the association between insomnia and symptoms of depression and anxiety (e.g., Kraus & Rabin, 2012; Roy-Byrne, Uhde, & Post, 1986). Insomnia and depression are highly comorbid disorders (Franzen & Buysse, 2008; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005); indeed, difficulty with sleep is a criterion for major depressive disorder (American Psychiatric Association, 2013). It appears that changes in insomnia severity may mediate depression outcomes (e.g., Staner, 2010), but it is not clear whether the inverse relationship exists. That is, do changes in depression over the course of CBT-I treatment function as mechanisms of treatment response? Recent research suggests that for individuals with comorbid insomnia and depression, CBT-I improves outcomes for both symptom clusters (e.g., Ashworth et al., 2015; Manber et al., 2008). From a theoretical perspective, it was hypothesized that depression would mediate clinical outcomes in CBT-I.

Like depression, anxiety symptomatology frequently co-occurs with insomnia presentations (Taylor et al., 2005) and the relationship between the two disorders appears to be bidirectional (Johnson, Roth, & Breslau, 2006). That is, anxiety disorders frequently precede the onset of insomnia disorder, but the opposite course of development is also prevalent. Within insomnia populations, individuals also tend to experience increased nonpathological worry in a variety of areas (e.g., higher incidence of health-focused anxiety), with a specific anxiety content focused on sleep-related threats (Harvey, 2002). Within CBT-I, relaxation strategies may be delivered to address symptoms of hyperarousal and anxiety. Thus changes in the severity of anxiety and worry may co-occur with changes in sleep over the course of CBT-I treatment. The present study investigated the extent to which changes in anxiety between pre- and posttreatment mediate outcomes.

Experiential perspectives. Although the aforementioned factors are presumed to function as key components of CBT-I treatment, we know little about clients' understanding of and experiences with the treatment, and with what they found to be most helpful. Increasingly, studies in the sleep field have begun to use qualitative methods in examining client experiences with insomnia (e.g., Maich, 2015; for review see Araujo, Jarrin, Leanza, Vallieres, & Morin, 2017). Yet, few have examined factors related to the specific processes underlying and factors associated with change. Those who have incorporated qualitative analyses into studies of insomnia have tended to focus on client experiences with insomnia proper (e.g., Kleinman et al., 2013) as well as client and health care practitioners' perspectives on treatment options and barriers (e.g., Cheung et al., 2014; Davy, Middlemass, & Siriwardena, 2015; Henry, Rosenthal, Dedrick, & Taylor, 2013), as opposed to analysis of specific *treatment components* that clients view as most clinically beneficial. It is critical that client voices are included in studies of factors that impact treatment response, in particular because it is most likely these are the precise factors that clients will recall and find most appealing and feasible to re-implement once treatment is complete. That is, it is very probable that these are the mechanisms of treatment response that affect long-term insomnia outcomes. To better understand the maintenance of gains as well as factors involved in relapse, it is crucial to understand clients' perspectives in order to improve our understanding of mechanisms of treatment response in CBT-I.

Study Hypotheses

Regarding participants' views on the aspects of treatment they considered more or less helpful, it was expected that a number of themes would emerge from the qualitative analysis of the participant response data. Based on the extant CBT-I literature, clinical experiences, and anecdotal evidence from the Sleep and Depression lab at Ryerson University, it was anticipated

that participant responses would echo at least some of the main treatment recommendations outlined in the CBT-I protocol (e.g., sleep compression, stimulus control). That is, it was hypothesized that the theoretical factors presumed to function as mechanisms of the treatment would emerge in the participant response. Specifically, it was anticipated that six key CBT-I "ingredients" would be supported by participant opinions: stimulus control, sleep compression, circadian rhythm regulation, behavioural activation, cognitive restructuring, and focus of attention, effort, and sleep-related safety behaviours. To acknowledge these factors that were expected to emerge in participant responses *a priori* to qualitative analysis, a template for reviewing participant responses was developed in advance of analyses (please refer to *Analyses* section of this dissertation for review of the qualitative interpretive process; see Appendix B for Template).

It was nevertheless also anticipated that some participants would report preference for treatment components that are generally not considered 'essential' in the efficacy literature (e.g., relaxation exercises, implementing a 'wind-down' period before bed, psychoeducation about sleep architecture and norms, having a therapist to help them maintain accountability with regard to their sleep schedule). Indeed, a past investigation of clients' reported use of the various CBT-I components yielded relaxation exercises as the most frequently used (and as such, likely most favoured) component of treatment (Harvey et al., 2000). In order to reduce investigator bias as much as possible during analysis, and to remain as open as possible to thematic patterns observable in the qualitative data, these nonessential treatment components were not included in the *a priori* template. Participant responses from the current investigation provided more nuanced understanding of the treatment components that are not only most *used* during

treatment, but also which components were found to be most *helpful* in improving participants' insomnia symptoms.

In terms of the quantitative component of this study, it was expected that a number of variables would impact clinical insomnia severity at post-CBT-I treatment. Based on the existing literature, the keystone presumed factors underlying the development and maintenance of insomnia, and clinical experience, the variables expected to have the largest mediating effect were: change in TIB, change in RT variability, and change in sleep effort. It was also expected that pretreatment variability RT, tendency to ruminate, and sleep effort would moderate the strength of the relationship between pre- and posttreatment insomnia severity in the secondary regression analyses.

Methodology

Research Design

The present study was a mixed-methods design–specifically, a triangulation design using the convergence model of analysis (Creswell, 1999; Creswell, Plano Clark, Gutmann, & Hanson, 2003). The purpose of this design is to acquire complementary qualitative and quantitative data in order to elucidate our current understanding of a singular topic (Morse, 1991), in this case, mechanisms of treatment response in CBT-I. Qualitative and quantitative data were collected and analyzed simultaneously in a single phase. Upon completion of analyses, results were considered in comparison, in order to better interpret theoretical factors integral to treatment response and understand those factors that are likely to function as treatment mechanisms.

Participants' perception of factors believed to most improve their insomnia complaint was the main outcome of interest in this study. To assess participant perceptions, qualitative data were derived from handwritten statements (Letter to Self; LTS), which were completed by

participants between their third and fourth (i.e., final) CBT-I sessions. Participants were directed by trial therapists to complete the LTS by describing the strategies and tools that they found to be most helpful for improving their sleep and daytime insomnia complaint. Data obtained through the LTS were then examined using qualitative analysis to pinpoint patterns within participants' responses. Prior to analysis, an *a priori* template of presupposed themes was developed based on theoretical factors. This template was used as a guide during qualitative data analysis, which also incorporated immersion/crystallization in order to modify, refine, and add emergent themes not included in the *a priori* template (i.e., factors independent from the presumed CBT-I mechanisms). The qualitative analysis yielded a final list of treatment components that clients reported to be most helpful for improving their sleep.

In addition to acquiring participant perspectives on what worked best for them during their treatment, we wanted to examine theoretical factors that are presumed, based on the research literature, to function as mechanisms of CBT-I treatment. These factors were analyzed quantitatively. First, to assess treatment response, changes in key insomnia indices (total wake time; TWT) and self-reported insomnia severity (scores on the ISI) from pre- to post-CBT-I intervention were measured. Variables expected to impact posttreatment outcomes from a quantitative perspective included pretreatment and pre- to posttreatment change variables. Specifically, the hypothesized mediating variables included behavioural indices (time in bed, rise time variability, sleep related safety behaviours); measures assessing mood state (fatigue severity; anxiety and depression subscales of the DASS-21); and cognitive factors (tendency to ruminate, sleep effort).

Participants and Recruitment

Participants were individuals with insomnia between age 18 and 79 years who participated in a larger ongoing clinical trial of CBT-I (Canadian Institutes of Health Research Operating Grant: *Longitudinal Assessment of Cognitive Reactivity in Insomnia;* Principal Investigator [PI]: Carney). Recruitment methods for the parent trial included media advertisements throughout the Ryerson University campus and the larger Toronto community, as well as referrals from external sleep clinics (e.g., the Sleep and Alertness Clinic) and hospitals (e.g., St. Michael's Hospital Psychology Training Clinic). Complete details of the clinical trial procedures are reported in Figure 1 below (p. 49), while procedures relevant to the present study can be found beginning on page 45. Details about the sample for the current study are reported below (see participant demographics, p. 63).

Inclusion criteria included meeting Research Diagnostic Criteria (RDC-I; Edinger et al., 2004) for Insomnia Disorder (ID). Specifically, participants must have endorsed the presence of insomnia symptoms at least three times per week over the previous 3 months, in accordance with the most recent iteration of the Diagnostic and Statistical Manual for Mental Disorders (DSM 5; American Psychiatric Association, 2013). Eligible participants completed one week of daily Consensus Sleep Diary (CSD; Carney et al., 2012) monitoring to further assess the presence and severity of insomnia complaint. Estimates of sleep efficiency (SE) derived from the CSD were required to be below 85 percent for inclusion. Participants were also asked to complete a retrospective measure assessing subjective insomnia severity, the Insomnia Severity Index (ISI; Morin, 1993) as part of a larger questionnaire battery. To be eligible for the study, participants were required to obtain a score greater than 14 on the ISI. Participants were excluded from the study if they indicated: an immediate psychiatric (e.g., suicidality) or medical crisis; a recent

(previous 6 months) suicide attempt; a medical condition that caused sleep disruption; a comorbid psychiatric disorder (e.g., schizophrenia, borderline personality disorder) that may excessively impact their sleep, ability to participate, or in-lab behaviour; recurring travel across time zones; rotating night shift employment; met criteria for another underlying sleep disorder for which CBT-I would be unhelpful (e.g., circadian rhythm disorder, periodic limb movement disorder, apnea); or there was a significant history of alcohol, benzodiazepine, narcotic, or other substance abuse or dependence in the past six months prior to the screening assessment. Because individuals with insomnia are frequently treated with sleep medications as a first-line treatment at primary care facilities or by their general practitioners, participants were permitted to use various prescription and nonprescription sleep aid medications throughout the course of the study. This inclusion criterion was developed with the goal of broadening the relevance of study results to the broader population of individuals with insomnia. However, individuals were excluded from participating if hypnotic dependence was indicated (based on the criteria delineated in the Duke Structured Interview for Sleep Disorders; see *Measures*). If diagnostic criteria for hypnotic dependence were not met, participants were asked to maintain consistency with regard to their medication dosage and timing throughout the course of the study.

Compensation received by participants in the stages of the trial relevant to the present research was free cognitive behavioural treatment for insomnia (i.e., CBT-I); as part of the larger parent trial, participants were eligible to receive up to $75 if they completed all aspects of the study (that is, including one year of monthly follow up telephone calls and a one-year posttreatment appointment). All participant data were protected, and identifying information (e.g., contact information, signed consent forms) was kept separate from study data in a locked filing cabinet. Participant information was otherwise identified only by study number, and all

other data were stored in password-protected computers at the SAD Lab at Ryerson. Only individuals involved in the study in some capacity (including the PI, graduate therapists, lab coordinator, and research assistants and volunteers) were permitted access to the aforementioned materials. All data pertaining to this study will be destroyed 10 years after the present study is published (other related data will be destroyed 10 years after publication of results from the parent trial).

Measures

Structured Clinical Interview for DSM-IV Axis I and Axis II Disorders. The Structured Clinical Interview for DSM-IV Axis I and Axis II Disorders (SCID-I/SCID-II; First, Spitzer, Gibbon, & Williams, 2002) are semistructured diagnostic interviews that are administered to assess for and establish DSM-IV Axis I and Axis II diagnoses. There is good evidence for the psychometric validity and reliability of the SCID, and in practice, it is a tool that widely used to establish psychiatric diagnoses in clinical psychological assessment. For the present study, the SCID was employed to assess for the presence of potential exclusionary psychiatric diagnoses. Specifically, the SCID-I was administered to assess for and exclude individuals who met lifetime criteria for bipolar disorder, schizophrenia, or other psychotic disorders, while the SCID-II was used to exclude individuals who met lifetime criteria for borderline personality disorder or antisocial personality disorder, which were also exclusionary. Criteria for insomnia disorder diagnosis were updated to reflect changes in the 5th edition of the DSM (i.e., insomnia complaint at least 3 nights per week over the past 3 months; American Psychiatric Association, 2013).

Letter to Self. Data for the qualitative component of this study were gathered through the Letter to Self (LTS; see Appendix A). Developed for the current study, the LTS is a tool

designed to encourage participants to reflect back on their CBT-I treatment progress and think about what worked best to help them with their sleeping difficulty. The LTS is a largely open-ended form upon which participants were asked to note strategies and skills that they developed over the past several weeks (i.e., since beginning treatment) that they personally found to be most helpful in improving their insomnia symptoms and/or distress. The LTS was provided by therapists at the end of the third treatment session with instruction to participants to consider a hypothetical future period when insomnia symptoms return, and to use this scenario to instruct their 'future self' on how to once again improve their symptoms. Specifically, participants were asked to consider what they found most helpful about CBT-I, and remind themselves of these strategies on the LTS. Therapists were asked to emphasize that although the letter was to be discussed in the final treatment session, participants should write the letter in any manner that they believe would be most helpful for them to read in future (e.g., point-form vs. narrative format), as opposed to attempting to reiterate all of the rules and recommendations delineated in the treatment protocol. In other words, the key rationale for the LTS was for participants to provide a personalized record that they could use to recall and re-implement those particular factors that they found to be most helpful in reducing their insomnia symptoms and/or associated distress as a means by which to incorporate relapse prevention planning into the final CBT-I session. Theoretically, the LTS was expected yield an array of responses, including both presumed factors targeted by CBT-I treatment (i.e., perpetuating factors) as well as potential components of the intervention or therapy more broadly that have not yet been explored in the CBT-I treatment literature.

Within the CBT-I protocol, therapists also provided participants with a summary of the treatment and their progress; however, there is general consensus within the field of psychology

that there is utility in having clients generate their *own* perceptions of the treatment, helpful strategies, and changes they have noticed, as a method of increasing collaboration, learning, and recall (as opposed to passive receipt of psychoeducation from the therapist). Studies of the long-term efficacy across psychopathological interventions have examined a variety of strategies for preventing or minimizing the likelihood of relapse (e.g., Bockting, Hollon, Jarrett, Kuyken, & Dobson, 2015; Lin et al., 2003; Ludman et al., 2000; Marlatt & Witkiewitz, 2008). Most of this research points to the importance of client self-efficacy and confidence in maintaining gains; 'reminders' that clients can return to in future, such as the LTS, are important aspects of long-term self-efficacy, and are thus included in a variety of treatment protocols and clinical practice (e.g., Boersma, Hakanson, Salomonsson, & Johansson, 2015; Pielech, Sieberg, & Simons, 2014; Rohde, Stice, Shaw, & Gau, 2016).

The motivation behind requesting the LTS at the third treatment session was threefold: a) it provided the therapist and participant with an opportunity to reflect together on the participant's progress over the course of treatment at the final session; b) the therapist could supplement any notable missing information and reinforce key strategies at the final session; and c) the participant did not review the therapist's summary letter until after they have written their own LTS, thereby reducing the likelihood that the responses on the LTS would merely reiterate the main CBT-I treatment protocol components (as opposed to the participant's own perceptions).

Consensus Sleep Diary. Sleep diaries are recommended tools for assessing insomnia symptoms over one or two weeks, and also for monitoring symptom change over the course of treatment (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). Diaries are the gold standard tools for insomnia because insomnia is a subjective disorder, requiring self-reported

sleep difficulty as well as a daytime complaint (i.e., impairment or distress; American Psychiatric Association, 2013). The Core Consensus Sleep Diary (CSD; Carney et al., 2012) is a prospective tool that was developed by a consensus group of sleep experts with the goal of standardizing sleep diary monitoring and improving ease of cross-study comparison. It is considered the gold standard measure for subjective assessment of insomnia. There is strong psychometric evidence supporting the clinical utility, validity, and usability of the CSD for use in insomnia assessment and treatment (Maich, Lachowski, & Carney, 2016). It also shows good utility in differentiating normal sleepers from those with insomnia disorder (Maich, Lachowski, Harris, & Carney, 2013).

Administration of the CSD requires nightly completion of a self-report record form that queries individuals on specific sleep indices, such as bed time, rise time, estimated time to fall asleep, and awakenings during the night. Data obtained from this measure are entered into a mathematical algorithm that yields estimates of the individual's average nightly total wake time (TWT), total time in bed (TIB) including naps, variability in rise time (RT), as well as other key sleep related information such as sleep schedule variability and sleep-wake patterns (Buysse et al., 2006; Carney et al., 2012).

Duke Structured Interview for Sleep Disorders. The Duke Structured Interview for Sleep Disorders (DSISD; Edinger et al., 2004) is a measure that was developed to aid in the diagnosis of sleep disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000). There is evidence indicating that the DSISD has adequate validity and reliability (Edinger et al., 2009). The DSISD will be used in the current study during participants' initial assessment interview to determine the presence of insomnia disorder, and assess for the presence of other exclusionary diagnoses such as sleep

apnea, Circadian Rhythm Disorders, Periodic Limb Movement Disorder (PLMD), or physiological dependence on prescription hypnotic medication.

Insomnia Severity Index. The Insomnia Severity Index (ISI; Morin, 1993) is a 6-item self-report scale that queries participants on the perceived severity of their sleep difficulties, including difficulty falling asleep, maintaining asleep, waking up too early in the morning, and distress about their sleep and its perceived impact on daytime functioning. Participants are asked to rate each item on a Likert-type scale ranging from 0 (*no sleep difficulty*) to 4 (*severe sleep difficulty*). These ratings are then summed to acquire a composite score (between 0 and 28). The recommended cutoff is 14; scores 15 and above indicate the presence of moderate (scores 15-21) or severe (scores 22-28) insomnia (Buysse et al., 2006). Psychometric evidence yields strong support for the validity and reliability of the ISI in the assessment of subjective insomnia severity (Bastien, Vallieres, & Morin, 2001; Morin, Belleville, Belanger, & Ivers, 2001). In the current study, ISI score will be used as one of two measures of treatment response.

Sleep Related Behaviors Questionnaire. The Sleep Related Behaviors Questionnaire (SRBQ; Ree & Harvey, 2004) is a 32-item measure developed to assess both nighttime (e.g., worrying about the negative consequences of lack of sleep on the following day) and daytime (e.g., engaging in napping or morning lingering behaviours to recover lost sleep; reducing or avoiding social activities to conserve energy) safety behaviours related to insomnia. There is preliminary psychometric evidence supporting the SRBQ's reliability and ability to discriminate between good sleepers and those with insomnia (Jansson-Frojmark, Harvey, Norell-Clarke, & Linton, 2012; Ree & Harvey, 2004). Each item is scored on a Likert-type scale ranging from 0 (*almost never)* to 4 (*almost always*). In addition to providing these frequency ratings, participants were asked to rate each item on this measure in terms of *necessity* on a 0 (not at all

necessary) to 10 (absolutely necessary) scale, and *anticipated distress* expected if unable to use the coping strategy on a 0 (no distress at all) to 10 (extremely distress) scale. For the purposes of the present study, only the initial Likert-type ratings related to frequency of the safety behaviour were used in analyses.

Depression Anxiety Stress Scales. The Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995) combine three subscales into a single, 21-item measure to assess subjective presence of the scale's eponymous symptoms (i.e., depression, anxiety, and stress). Each item is rated on a 4-point scale that ranges from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*). To score the measure, the seven items that address each individual subscale are summed. Because the DASS-21 is a shortened version of the original 42-item measure (DASS-42), subscale scores are multiplied by 2 prior to interpretation, in order to ease comparison between the measures. There is adequate support for the validity and reliability of the DASS-21 (Antony, Bieling, Cox, Enns, & Swingson, 1998; Brown, Chorpita, Korotitsch, & Barlow, 1997). The DASS-21 has also been used extensively in insomnia research (e.g., Harris, Lack, Wright, Gradisar, & Brooks, 2007; Smith, Kozak, & Sullivan, 2010). For the purposes of the current study, only the anxiety and depression subscales were examined in analyses.

Fatigue Severity Scale. The Fatigue Severity Scale (FSS; Krupp et al., 1989) is a 9-item self-report questionnaire that assesses fatigue symptoms. Items are scored on a 7-point Likert-type scale from 1 (*strongly disagree*) to 7 (*strongly agree*). To score the FSS, items are summed and the average is obtained. Higher scores are suggestive of greater levels of subjective fatigue. There is support for the validity and reliability of the FSS; it has excellent internal consistency

(Cronbach's alpha = .94) and good test-retest reliability (Krupp, LaRocca, Muir-Nash, & Steinberg, 1989; Valko, Bassetti, Bloch, Held, & Baumann, 2008).

Ruminative tendency. To assess the tendency to ruminate, the Daytime Insomnia Symptom Response Scale (DISRS; Carney, Harris, Moss, & Edinger, 2010; Carney, Harris, Falco, & Edinger, 2013) was administered. The DISRS is a 20-item self-report measure that assesses individual response to insomnia symptoms experienced during the day, including the tendency to ruminate about daytime symptoms and consequences of insomnia (e.g., irritability, fatigue, difficulty concentrating). Participants are asked to score each item on a four-point scale ranging from 1 (*almost never*) to 4 (*almost always*). To score the measure, items are summed to obtain a composite score; higher total scores represent a greater tendency to ruminate. There is evidence supporting the internal consistency and validity of the DISRS (Carney et al., 2013).

Glasgow Sleep Effort Scale. The Glasgow Sleep Effort Scale (GSES; Broomfield & Espie, 2005) is a 7-item measure designed to assess effort to control sleep over the past week. Each item is rated on a three-point scale (*very much*, *to some extent*, or *not at all*). To score the questionnaire, each response is assigned a numeric value (very much, 2; to some extent, 1; not at all, 0) and summed to obtain a total score. Higher total scores are indicative of greater sleep effort. The GSES has been shown to have adequate internal consistency and good ability to discriminate between those with insomnia and those with good sleep (Broomfield & Espie, 2005).

Study Procedures

Data were collected over a 5 year period at the Sleep and Depression Laboratory at Ryerson University in Toronto, Canada. Participants were part of a larger parent trial examining the longitudinal efficacy of CBT-I in individuals with insomnia (CIHR Operating Grant:

Longitudinal Assessment of Cognitive Reactivity in Insomnia; PI: Carney). Data acquired for the qualitative component of this study were analyzed over a 1.5 year period, until saturation was reached. To determine preliminary eligibility for enrolment in the parent trial, participants first completed a brief (approximately 10 minutes) telephone screening interview. The telephone interview assessed whether individuals experienced current insomnia, had past insomnia experience, or were good sleepers; other preliminary eligibility criteria included age between 18 and 79 and nonpresence of a major psychiatric disorder or concern (e.g., current psychosis, Borderline Personality Disorder). If participants met initial eligibility criteria, they were asked to complete one weeks' worth of sleep logs (i.e., the CSD) prior to attending a two-hour full eligibility assessment at the SAD Lab at Ryerson University. Prior to administering the in-person interview, the assessor described participation requirements and expectations and participants provided written informed consent to participate in the study. In addition to establishing the presence of insomnia diagnosis, other comorbid sleep difficulties (e.g., sleep apnea, circadian rhythm disorder), medical issues, and psychological disorders were assessed using the SCID and DSISD interviews. Upon completion of the interview, participants completed a battery of questionnaires, including measures assessing a number of the mechanisms of interest for the present study.

If deemed eligible for the treatment trial, participants were then scheduled to complete four biweekly sessions of CBT-I. For the purposes of the present study, data were gathered on an ongoing basis. Treatment sessions were scheduled two weeks apart in order to acquire two-week sample of sleep diary data between treatment sessions, as per recommendations for nightly sleep monitoring required to establish a stable picture of an individual's sleep (Wohlgemuth, Edinger, Fins, & Sullivan, 1999). Each two-week CSD yielded sleep variables that were used to

examine treatment mechanisms in the present study (e.g., the outcome variable TWT was calculated from CSD data). Additionally, participants completed a retrospective, self-reported assessment of their insomnia severity (i.e., the ISI) before each of the four treatment sessions.

The CBT-I intervention that was delivered to eligible participants in the current study was developed based on a manualized protocol (Edinger & Means, 2005; Edinger & Carney, 2008, 2015), and was designed to be delivered to participants over the course of 4 biweekly sessions. The focus of the first session was on providing participants with psychoeducation information about sleep and insomnia broadly, as well as explaining the rationale for a number of evidence based recommendations for changes specific to improving their sleep. The first session was focused on behavioural changes, including assignment of a collaboratively agreed upon sleep schedule, stimulus control instructions, and eliminating naps. However, components of cognitive therapy could also be incorporated into this first session, as time allowed (e.g., beginning to challenge beliefs about sleep needs). The second session was more cognitively focused, with the introduction of thought records, worry time, the concept of presleep cognitive arousal (or "insomnia brain"), as well as in session Socratic questioning, troubleshooting any behavioural difficulties encountered over the previous two weeks, and adjusting the sleep schedule as necessary. The final two sessions were chiefly focused on these latter targets (i.e., troubleshooting, adjusting recommendations based on outcomes), as well as discussion of relapse prevention and maintenance of gains.

The LTS forms were provided to participants at their third treatment session, with instruction to write down what factors they found to be most helpful in improving their insomnia problem over the course of their treatment. Participants were asked to return the completed LTS to their therapist at session four, at which point the letters were photocopied for the purposes of

this research. The trial therapist and participant then reviewed the LTS together in session. Sample size (n = 27) for the qualitative component of the present study was considered adequate once saturation is achieved; that is, qualitative analyses were discontinued when no new or relevant concepts appeared to be emerging from the collection of additional data (i.e., there were diminishing returns and therefore no additional benefit to continued sampling).

After the final (fourth) CBT-I session, participants were scheduled for a posttreatment appointment (approximately 1 hour in duration) two weeks later. Battery questionnaires containing the additional measures assessing putative mechanisms were administered at the posttreatment appointment, as well as the ISI and the CSD.

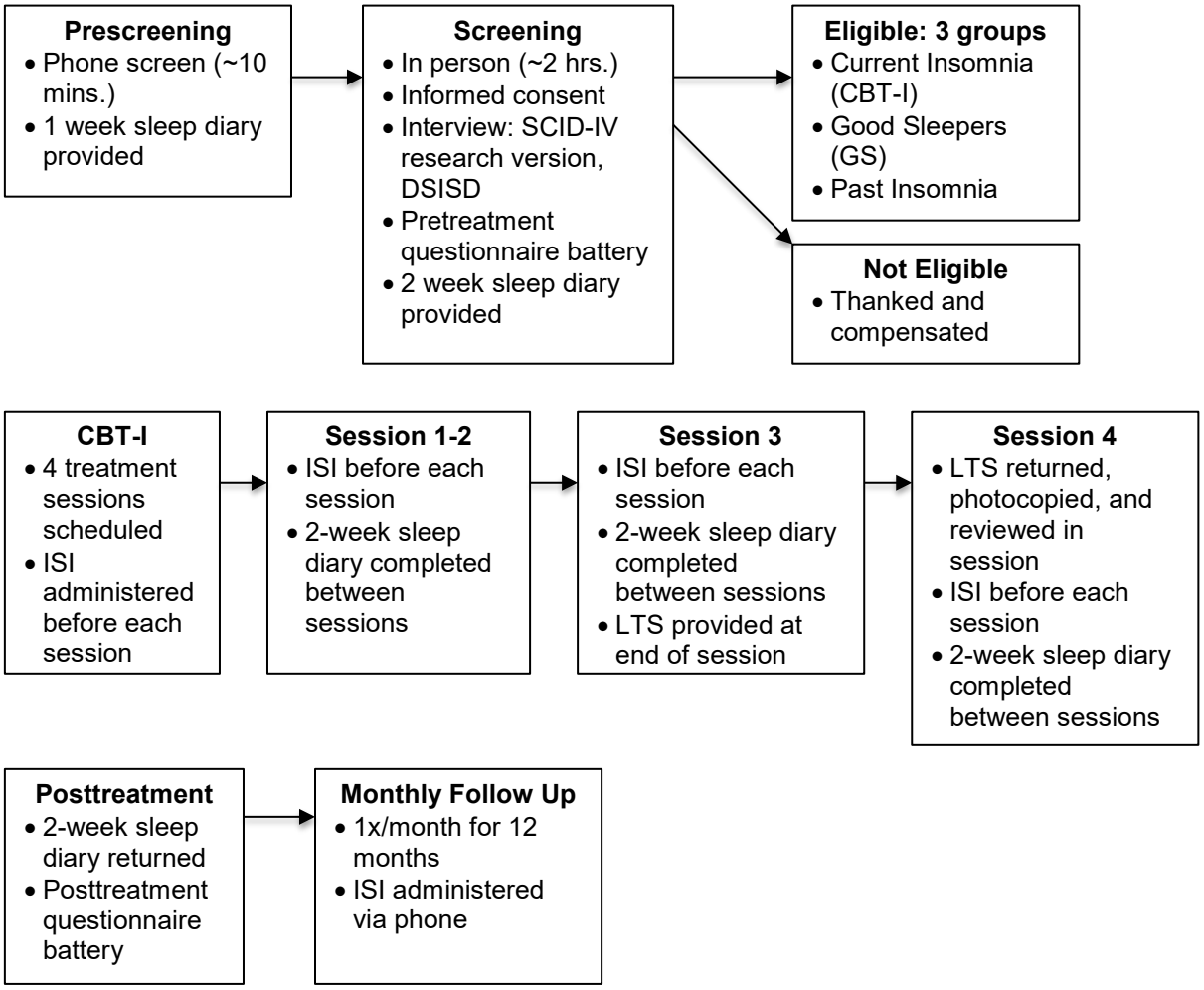

Figure 1. Procedures of the clinical trial of CBT-I for the current study.

Analyses

Qualitative analyses were completed using a combination of the template approach and the immersion/crystallization technique. This methodology is described below. All quantitative statistical analyses were conducted using IBM SPSS software (Versions 19.0 and 21.0; SPSS Inc., Chicago, IL, USA).

Qualitative analysis. To understand participant perspectives on helpful treatment components, data from the Letter to Self (LTS) were analyzed qualitatively. In general, qualitative analysis is a method for developing hypotheses about a phenomenon or process (Creswell & Clark, 2007). It is typically used to acquire subjective data from individuals about their experiences and perspectives on a certain topic. In the present study, data obtained through the LTS were examined using qualitative methods to pinpoint patterns within participants' responses regarding helpful treatment components.

Theoretical approach. The data were approached from a middle range theoretical perspective, meaning that the analysis involved empirical inquiry that was both theory-oriented and also open to emergent themes not previously identified in the insomnia literature (Merton & Merton, 1968; Pawson, 2000; Soltani et al., 2014). Middle range theories are those that are neither specific, working hypotheses, nor broader unified theories (Merton & Merton, 1968); that is, middle range theory attempts to "consolidate otherwise segregated hypotheses and empirical regularities" (Merton, 1957, p.280). Importantly, middle range theory is relatively wide in terms of scope, while simultaneously involves description of a set of phenomena (Soltani et al., 2014).

Thus the middle range theoretical approach to qualitative analysis was uniquely appropriate for analyzing the data derived from the LTS in the current study as it is constructed via integration of presupposed theoretical factors with empirical data, which made sense given

the considerable research base upon which CBT-I is based; however middle range theory also allows for a more open approach to analysis, wherein the data may be expected to reflect some concepts suggested by empirical literature, while also allowing for the possibility that other, unanticipated factors may emerge from the data. Middle range theory is therefore more structured than other theoretical perspectives, however maintains a nonrestrictive stance in allowing that nontheoretical themes may be present in the data. Thus middle range theory balances a more structured, empirically based framework with a nonrigid open approach to data analysis. For the purposes of the present study, it was assumed likely that certain themes would be apparent in the data; it was also acknowledged that not everything is known about the mechanisms via which CBT-I works to improve client outcomes. As such, qualitative analysis was conducted with the presumption that six putative CBT-I treatment components would be visible themes in participant responses on the LTS, and also that unanticipated, nontheoretical factors would also likely emerge.

Presupposed theoretical factors. Given the breadth of research on factors presumed to cause improvement in insomnia symptoms following a therapeutic course of CBT-I, responses on the LTS were expected to reflect at least the key elements of the treatment. These components were drawn from the CBT-I treatment manual, but originated in the theoretical sleep literature (e.g., Spielman, 1987). Since posited as factors involved in the maintenance of insomnia, these theoretical factors have been supported by extensive research evidence (e.g., Bootzin et al., 1991; Edinger & Means, 2005; Espie et al., 2006; Harvey, 2002; Wohlgemuth & Edinger, 2000). Template themes included the following six theory-based tenets of CBT-I: elements of stimulus control; sleep compression strategies; regulation of factors impacting the circadian rhythm; daytime behavioural activation; cognitive restructuring strategies; and focus of

attention, effort, and behaviours around sleep themes. These presupposed themes are summarized in Appendix B, along with specific examples of how each of these treatment components would be enacted in practice (e.g., one way in which "sleep compression" is attained in CBT-I is through client adherence to a restricted time in bed window).

Approach to coding. From a middle range theory perspective, a template style was used to organize the data. The template approach to qualitative data analysis involves the development of a template coding manual containing presupposed themes in advance of text analysis as part of the overall process of interpretation (Crabtree & Miller, 1999). Templates are based on *a priori* hypotheses or areas of interest related to the general topic (Meadows, Verdi, & Crabtree, 2003). In keeping with middle range theory, in the current study there were a number of presumed mechanisms of CBT-I treatment response, which were also used to inform the quantitative mediation analyses. For the qualitative analyses, these same factors were considered in conjunction with the CBT-I protocol and the empirical research upon which CBT-I was based in order to develop a template of themes *a priori*, as a framework via which to approach analysis of the LTS data (refer to Appendix B to see the template list of presupposed theoretical factors used for coding the data). That is, those theoretical factors expected to influence CBT-I treatment response, previously described in the introduction section, were used to populate the template with themes included from the middle range theory. During the first cycle of text analysis, participant contributions via the LTS were culled for themes from the template, in order to ascertain whether responses supported the empirically based theoretical factors that are the foundation of CBT-I.

A template approach is a method of organizing text that allows for efficient identification of expected themes, but also involves refinement, alteration, and addition to the list of themes

after the first cycle of the data review has been completed (Crabtree & Miller, 1999). This approach also involves a process of connecting themes, wherein relationships and patterns in the data may be detected and merged, as the candidate themes are refined. An important advantage of the template approach is that it facilitates connection between theory and participant views in this study (Crabtree & Miller, 1999). Although it has been suggested that information not anticipated in advance to emerge from the data could be more likely to be missed using a template style, the use of a template notably does not preclude the researcher from making additions, modifications, and revisions to template themes (Crabtree & Miller, 1999). Rather, given the more open-ended middle range theory from which these data were approached, it was expected that new, unanticipated themes (i.e., those not included on the template) would emerge from the data. That is, we anticipated that participants in the current research would generate important insights that are not currently viewed as important components of CBT-I from the perspective of the research literature.

The data were therefore reviewed in several cycles via a dynamic iterative process; latter cycles of data analysis included immersion in the LTS responses, crystallization of new, unanticipated themes, and modification of the *a priori* template themes. For example, some themes were merged into a single theme, and conversely, single themes spliced into multiple themes, through the iterative process. Following application of the template, immersion/crystallization (I/C) was applied to the data in order to increase the likelihood of discovering new themes that might have been present in participant responses.

Immersion/crystallization is a process that is widely used in qualitative research, particularly when theories or hypotheses about expected themes in the data are present (DiCicco-Bloom & Crabtree, 2006). I/C involves a "concentrated textual review of the data, with

concerned reflection and intuitive insights, until reportable interpretations become apparent" (Borkan, 1999, p. 622). Given this definition, the I/C technique for analyzing the data in this study was uniquely suited to this project, given the PI's involvement as a CBT-I therapist and history of research in this field. Immersion/crystallization as an approach allows for the presence of ideas that might guide or influence data coding, but importantly, allows for the investigator to remain open to the emergence of new themes. Essentially, I/C is an iterative process that involves repeated, cyclic review of the data until the point of saturation is reached, after which no new themes are expected to emerge. In the current study, "immersion" was the first step following application of the template, wherein the PI became immersed in the collected data by reading all of the responses to the interview questions in detail. Following immersion was crystallization, wherein the PI suspended immersion (i.e., refrained from reading the original data responses) and articulated candidate theories about new factors that emerged from the data set. That is, in this stage of data analysis, emergent themes were generated and, if appropriate, presupposed template themes were adapted or refined. The cycles of immersion, crystallization, and refinement of the template were repeated until saturation was reached and emergent themes were narrowed down and clarified. This latter process of refinement included alteration or merging of themes where conceptual overlap existed, and deletion of extraneous/redundant themes. To help manage the influence of the inherent bias of the PI during the analyses, committee member and co-supervisor on this project Dr. Kelly McShane, whose research area is outside of the field of sleep and who has expertise in qualitative research methods, was consulted and provided feedback at integral stages of the project development (e.g., methodological development, *a priori* template item development).

Organizing the data in the current study by applying an *a priori* template, as well as using I/C to analyze the data, therefore incorporated both a more open I/C approach (Crabtree & Miller, 1999), to facilitate understanding of client experiences and discover, as well as the more deductive and structured template approach, which preserved and accounted for the fact that the PI entered into the qualitative analyses with a strong understanding of the theoretical factors presumed to influence treatment response in this study, as well as the fact that there is a substantial body of research evidence pointing towards a number of critical CBT-I treatment components. In other words, given that this study included an investigation of a number of presumed treatment mechanisms, and these factors were hypothesized to emerge in the participants' LTS responses, it made sense to incorporate this *a priori* knowledge into the data organization using a template developed in advance. Indeed, Crabtree and Miller (1999) suggest that it is important to consider the extent to which knowledge and understanding of the subject matter exist prior to analysis, clarifying that it may be indicated to use a template approach in particular if a) there is a body of existing literature on the subject; b) prior research provides insight on themes that could be anticipated to emerge; and c) there is a substantial amount of existing theory. In the case of the current project, all three of these criteria were met. To approach the data using solely I/C as an organizational method would therefore overlook the extant putative factors supported by decades of research, the same factors upon which the treatment provided to participants in this project was based. However, it was also the goal of this research to better *understand* participant experiences of CBT-I. Given that it was expected that participants may have alternative, or at least additional, perspectives on the treatment components that were most helpful to them, it was necessary explore the data via a more open, heuristic interpretative process: I/C.

In summary, to analyze participant responses regarding what they found most helpful in CBT-I, a template of themes derived from theory and the CBT-I treatment literature was applied to the LTS data. Following application of the template, the data were reviewed in cycles of immersion and crystallization, with continual revisions to the original template, until saturation was reached with regard to themes evident in the responses. It was expected participant responses would reflect at least some of the themes from the template, and that additional, unexpected factors would also emerge during analysis.

Quantitative analyses. Quantitative analyses included the primary mediation analyses and the secondary moderation analyses, in addition to preliminary analyses, which included demographics, Spearman's Rho correlations, and descriptive statistics of the variables of interest.

Primary analyses: Mediation. Hypothesized mediating variables were compared to posttreatment outcomes (i.e., scores on the ISI and TWT on the CSD) using multiple mediation analysis (PROCESS, Hayes, 2018; Preacher & Hayes, 2008). The objective of mediation analysis is to examine the degree to which a predictor variable (X) influences the outcome variable (Y) via multiple mediators (Hayes, 2012). From a conceptual perspective, chronic insomnia disorder (a criterion for inclusion in the current study's clinical trial) would presumably cause worsening in each of the areas assessed by the mediator variables (i.e., increased TIB, RT variability, sleep-related safety behaviours, sleep effort, rumination about the consequences of a poor night's sleep, and worsened mood state [increased fatigue, anxiety, and depression symptoms]). However, it is presumed that these relationships are bidirectional; that is, considering Spielman and colleagues' model of insomnia (1987), the onset of insomnia symptoms following a stressful precipitating event causes some individuals to adopt rational, yet maladaptive behaviours and thinking processes in an effort to cope with their sleep difficulty;

and it is these changes that we believe to perpetuate insomnia chronically. That is, changes on these indices (the mediating variables), are presumed to cause worsened insomnia symptoms. In CBT-I treatment, then, we assume that the change in insomnia severity between pre- and posttreatment is attributable to changes on these mediating variables (i.e., changes to maladaptive thinking styles and behavioural coping strategies).

Mediation analyses allow for testing of hypothesized mechanisms through which a presumed causal effect operates (Hayes, 2017, p. 76). Multiple mediation analysis advances the simple mediation model by allowing examination of multiple hypothesized mechanisms simultaneously, within a single model (Hayes, 2013). Please refer to Figure 2 for a visual depiction of a parallel mediator model (adapted from Hayes, 2018).

Preliminary mediation analyses used scores from pretreatment variables, which were entered as mediators into the analysis. Parallel multiple mediation with pretreatment variables was conducted as a first step to promote understanding of the intervening variables that may point to underlying mechanisms of treatment response. As outlined by Kazdin (2007), such preliminary analyses are considered an important first step prior to understanding the mechanisms of action via which the effect of treatment occurs (i.e., process of change over the course of treatment). We nevertheless expected that different mediators might emerge from the primary analyses where change variables were entered as mediators, as compared to the preliminary analyses involving pretreatment scores entered as mediators, given that the change mediators were expected to represent change over the course of CBT-I treatment, whereas the pretreatment variables were merely representative of participants' behaviours, cognitions, and mood states prior to CBT-I treatment.

Mediation is an analytical process that approximates the sampling distribution of the indirect effect by resampling the data many times in order to form a new distribution, rather than requiring that the original data be normally distributed (Hayes, 2009; 2013). Though the data may be resampled anywhere between 1,000 to 50,000 times, Hayes (2013) recommends 10,000 resamples for most analyses. Once the data has been adequately resampled, the new distribution is then used to generate the indirect mediational effect and also estimate confidence intervals. Unlike previous iterations of mediational analyses (e.g., Sobel mediation), bootstrapping is a nonparametric test and is therefore more flexible for use with data and indirect effects that may be nonnormal, which tends to be the case for many sleep indices and related variables (e.g., sleep efficiency always violates the assumption of normality because its mean in normal populations is 85 percent). In addition to the advantage of being a nonparametric test, bootstrapping also yields low Type 1 error rates and high power compared to other mediation analytic models (Hayes, 2009).

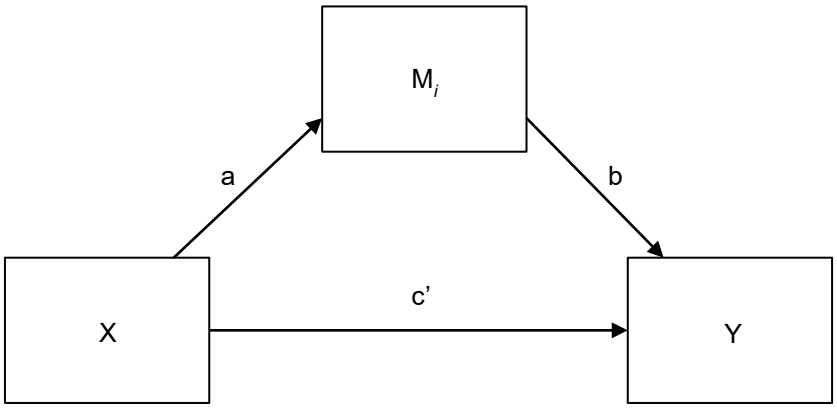

Figure 2: Parallel multiple mediator model (adapted from Hayes, 2018). The association between *X* and *Y* is expected to be partially accounted for by the indirect effects of M_i mediating variables. The c pathway represents the total effect of *X* on *Y*. The c' pathway shows the direct effect of *X* on *Y* (i.e., the degree to which *X* differs as a function of *Y*, controlling for M_i mediating variables. The *a* pathway shows the total effect of *X* on M_i, and pathway *b* shows the total effect of M_i on *Y*.

In the primary analysis, parallel multiple mediation (PROCESS; Hayes, 2017) was again conducted to assess the extent to which changes in the presumed mechanisms accounted for changes in outcome. In the primary analysis, the change scores (i.e., between pre- and posttreatment) of the presumed mechanisms of interest entered as mediators. This method of analysis was designed with the goal of addressing a problem identified in the mechanisms literature, namely that: "even when a timeline is established, mediation does not necessarily suggest the mechanism of action" (Kazdin, 2007, p. 9). Specifically, the variables presumed to function as mechanisms were assessed via self-report measures at pre- and posttreatment time points. The change scores of the mediating variables were compared to treatment outcomes in the multiple mediation analysis. This allowed for a more thorough assessment of those processes (i.e., changes or progressions) that occur over time (specifically, prior to treatment vs. at the initial posttreatment follow-up session), and how these changes relate to insomnia symptom change by the end of treatment. Thus, this analysis used change scores in order to better understand processes of change that may be responsible or partly responsible for bringing about changes in symptoms; that is, the use of change variables facilitated better understanding of variables that may represent mechanisms of therapeutic change in CBT-I.

Secondary analyses: Moderation. In order to determine whether the variables of interest influenced the strength of the relationship between the predictor and outcome variables, multiple linear regression analyses were conducted to assess for moderation effects (see Figure 3 for visual depiction of the multiple moderator model, adapted from Hayes, 2018). Variables that were expected to moderate the strength of the relationship between pre- (predictor variable) and posttreatment (outcome variable) insomnia severity were entered into multiple hierarchical linear regression analyses in SPSS. The predictor variable (ISI score or CSD TWT at pretreatment) was entered in the first step, with expected moderating variables entered into the second step. Hypothesized continuous variables expected to show a moderating effect included pretreatment behavioural indices (TIB; RT variability; sleep-related behaviours), state variables (depression; anxiety; fatigue), and cognitive variables (tendency to ruminate; sleep effort). Variables that emerged as significant predictors of outcome were considered moderators of CBT-I treatment response (i.e., moderators of the relationship between pre- and post-CBT-I treatment insomnia severity).

Multiple regression is an analysis that is robust even when data is nonnormally distributed (Field, 2009; Sullivan & D'Agostino, 2002), which is an advantage for the present research as sleep data is frequently nonparametric, as discussed above.

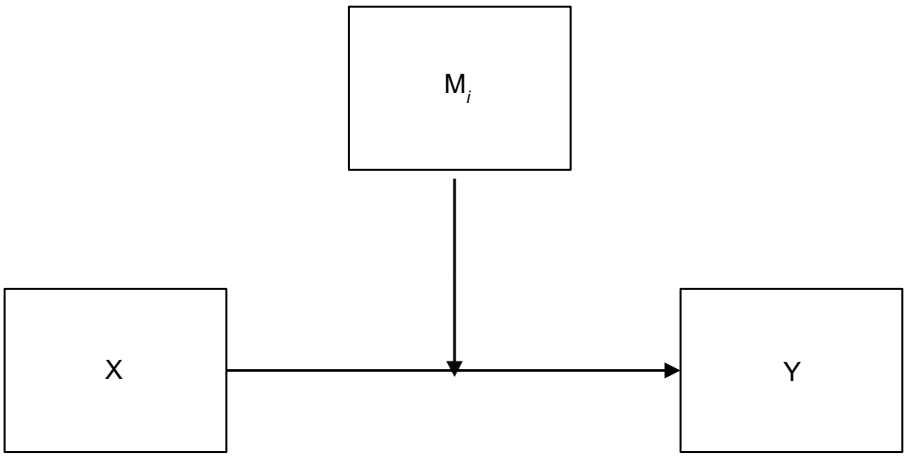

Figure 3: Multiple moderation model (adapted from Hayes, 2018). Moderators (M_i) are expected to influence the strength of the association between X and Y.

Results

Participant Demographics

The sample consisted of 163 adults between ages 19 and 77 years ($M = 46.7$, $SD = 14.9$). The majority of the participants were female ($n = 115$, 70.6%). Additional demographic information for the sample is presented in Table 1.

Table 1

Sociodemographic Characteristics of Study Participants

Characteristic	*n* (%)
Gender	
Female	115 (70.6)
Male	47 (28.8)
Other/Do not identify/Chose not to say	1 (0.6)
Ethnic Background	
Aboriginal Canadian	1 (0.6)
African Canadian	1 (0.6)
Caribbean	2 (1.2)
East/Southeast Asian	1 (0.6)
European Canadian	106 (65.0)
Latin/Central/South American	9 (5.5)
South Asian	10 (6.1)
West Asian/Arab Canadian	2 (1.2)
Other	29 (16.0)
Missing / Declined to answer	4 (2.5)
Living Arrangement	
Living alone	49 (30.1)
With spouse/partner	50 (30.7)
With spouse/partner and children	37 (22.7)
With friend(s)/roommate(s)	14 (8.6)
With family member(s)	10 (6.1)
Other	1 (0.6)
Missing / Declined to answer	2 (1.2)
Relationship Status	
Married/common-law	84 (51.5)
Single	57 (35.0)
Divorced	7 (4.3)
Widowed	6 (3.7)

Live-in partner (less than 2 years)	5 (3.1)
Separated	3 (1.8)
Missing / Declined to answer	1 (0.6)
Employment Status	
Full-time	92 (56.4)
Part-time	30 (18.4)
Not working	40 (24.5)
Missing / Declined to answer	1 (0.6)

Preliminary Analyses

Prior to conducting preliminary statistical analyses, missing data were labeled and coded appropriately in the master quantitative database (i.e., missing data labeled "999"), such that these data points would not influence analyses (i.e., they were excluded from analyses). The distributions of the variables of interest were examined for violations of assumptions related to normality, multicollinearity, and homoscedasticity. Spearman's Rho correlational analyses were conducted on the pretreatment and change score variables of interest, in comparison with the predictor and outcome variables (see Tables 2 and 3). For analytic purposes, only participants who completed posttreatment were included, in order to capture true pre- to posttreatment changes.

Tests of assumptions. The distributions of the mean pretreatment and change scores of presumed mediating variables (i.e., TIB, RT, SRBQ, DISRS, GSES, FSS, and the DASS anxiety and depression subscales) and the predictor and outcome variables (i.e., ISI and CSD TWT at pretreatment and posttreatment) were visually examined for normality. Data were also screened for outliers and influential cases. Initial inspection showed several outliers with data that were impossible given the scoring limits of the measures completed (e.g., ISI item score of 44.0, when the item scale was 0-5), suggesting data entry error (i.e., typo). These items were verified on the original questionnaires and corrected in the database. Two additional influential outlier cases were removed from the sample, as their removal substantially improved the distributions of the sleep indices. Following these corrections, skew and kurtosis values were examined. All variables of interest showed skew and kurtosis within the acceptable limits of |2| and |7|, respectively (West, Finch, & Curran, 1995), suggesting that for the most part, the data did not depart substantially from normality. For all mediating variables of interest (i.e., pretreatment and

change values for the mediating variables), skew values ranged from -0.92 to 1.41 and kurtosis values from -.03 to 4.07. For the predictor (pretreatment) and outcome (posttreatment) variables of insomnia severity (ISI) and CSD TWT, skew values ranged from 0.05 to 1.83, and kurtosis values from -0.41 to 4.40. The most extreme values largely occurred within the distributions of the sleep variables at posttreatment, which is to be expected given presumed improvement on these indices by the end of CBT-I treatment. For example, CSD TWT would be expected to have a positive skew at posttreatment, as mean total wake time would be expected to cluster closer to 0 compared to total wake time at pretreatment.

In accordance with guidelines for assessing multicollinearity (Hair, Anderson, Thatham, & Black, 1995; Tabachnick & Fidell, 2001), Variance Inflation Factor (VIF) and tolerance values of the predictor and mediating variables were examined. Multicollinearity was assumed if VIF values were greater than 10 (Myers, 1990), or tolerance values were less than 0.1 (Menard, 1995). VIF values ranged from 1.14 to 3.22, and tolerance values ranged from 0.31 to 0.88 for all models, suggesting that the assumption nonmulticollinearity was not violated.

Although the VIF and tolerance values did not violate the aforementioned absolute cut-off thresholds for multicollinearity, it appeared possible that the variables could nonetheless be highly correlated, given the strong convergence of these indices as constructs. For example, it was reasonable to expect that TIB and TWT would be quite highly associated. Because the pathways from each mediating variable to the outcome variable are estimated while controlling for all other mediators in the model in mediation analyses, very high correlations (≥ 0.8) between mediators can veil significant effects when the model involves multiple mediators (Field, 2009; Hayes, 2013). More specifically, strong correlations between mediators can influence the rate of sampling variance and in so doing, might broaden the width of the confidence intervals; if such

were to occur in mediation analyses, it would become more difficult to detect significance. The assumption of nonmulticollinearity amongst independent variables was confirmed by examining bivariate correlation coefficients (see Tables 2 and 3). As previously noted, multicollinearity was considered as potentially problematic if correlation values were 0.80 or greater (Field, 2009); no correlations met this criterion. It is important to note that a constraint of the parallel multiple mediation model, as is used in the current mediation analyses, is that no mediator should be modeled as influencing another mediator (Hayes, 2018). However, although mediators should not causally influence each other, mediators are nevertheless *not* presumed to be independent from one another (Hayes, 2013). Indeed, Hayes notes that it is likely and acceptable that mediators will be correlated to some degree. He furthermore posits that conducting a parallel multiple mediation with k mediating variables when the mediators are correlated with Y could be more advantageous than performing k separate mediation analyses, as a single multiple mediation may increase the power of testing for indirect effects. This further buttresses the argument to conduct a parallel multiple mediation in the current model. Returning to the question of multicollinearity, given that the mediators in the present study did not exceed $\rho = 0.57$, it was reasonable to consider the association between variables acceptable and proceed with the multiple mediation analyses.

Finally, data were examined for possible heteroscedasticity, the presence of which would suggest that the assumption that the variance of the dependent variables is consistent at each level of the predictor variables was violated (Field, 2009, 2013). The assumption of homoscedasticity iterates that the regression errors variance must be constant (i.e., not heteroscedastic), (Hayes & Cai, 2007). Initial inspection of standardized residuals suggested that there might have been deviation from homoscedasticity. To determine whether the data were

homoscedastic, the Breusch-Pagan test for heteroscedasticity (Breusch & Pagan, 1979) was conducted in SPSS, using a macro available online (Daryanto, 2018). The Breusch-Pagan tests the null hypothesis that the error variances are equal. A significant result indicates that the null hypothesis should be rejected, suggesting heteroscedasticity in the data. The results of the test were significant ($X^2 = 63.32$, $p < .001$.), confirming that the assumption of homoscedasticity was violated. To correct for heteroscedastic data, a heteroscedasticity-consistent standard error estimator (HC3; Davidson & MacKinnon, 1993) was applied in the mediation analyses, per recommended guidelines for PROCESS mediation (Hayes & Cai, 2007).

Correlations. Nonparametric Spearman's Rho (ρ) correlation coefficients were calculated to assess for significant associations between the variables of interest. Predictor (BL ISI, BL TWT) and outcome (post ISI, post TWT) variables were compared with pretreatment and change score mediator variables (TIB, RT, SRBQ, FSS, DASS-A and –D, DISRS, and GSES). See Table 2 for change variables of interest, and Table 3 for pretreatment variables of interest.

Table 2

Spearman's Rho Correlation Coefficients of Predictor, Outcome, and Change Variables of Interest

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
1. BL TWT	1.00											
2. Post TWT	.41***	1.00										
3. BL ISI	.28***	.28**	1.00									
4. Post ISI	.05	.45***	.29***	1.00								
5. Δ TIB	.44***	-.03	.05	-.05	1.00							
6. Δ RT	.01	-.13	.04	-.04	.11	1.00						
7. Δ SRBQ	.18*	-.13	.19*	-.47***	.18*	.14	1.00					
8. Δ FSS	-.03	-.29***	.08	-.52***	.06	.08	.54***	1.00				
9. Δ DASS-A	.04	-.03	.15	-.18*	.08	-.10	.34***	.26**	1.00			
10. Δ DASS-D	-.01	-.14	.10	-.32***	.05	.00	.37***	.34***	.43***	1.00		
11. Δ DISRS	-.07	-.12	.12	-.44***	-.01	.06	.49***	.47***	.40***	.42***	1.00	
12. Δ GSES	-.06	-.23**	.09	-.53***	.06	.10	.52***	.52***	.30***	.32***	.48***	1.00
13. Age	.14	.03	-.24**	-.06	.02	-.18*	-.08	-.14	-.26**	-.06	-.16*	-.22**

Note. Δ = Change. Predictor variables = pretreatment TWT and ISI. Outcome variables = posttreatment TWT and ISI. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21.

Presumed mediating variables = pre- to posttreatment mean change scores of total time in bed (TIB), rise time variability (RT), sleep-related behaviours (SRBQ), ruminative tendency (DISRS), sleep effort (GSES), fatigue (FSS), anxiety, and depression (DASS-A, -D). TWT = sleep diary-measured Total Wake Time. ISI = Insomnia Severity Index. BL = pretreatment (pretreatment), Post = posttreatment.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 3

Spearman's Rho Correlation Coefficients of Predictor, Outcome, and Pretreatment Variables of Interest

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
1. BL TWT	1.00											
2. Post TWT	.41***	1.00										
3. BL ISI	.28***	.28**	1.00									
4. Post ISI	.05	.45***	.29***	1.00								
5. BL TIB	.30***	.02	-.04	-.13	1.00							
6. BL RT	-.07	-.01	.06	.09	.02	1.00						
7. BL SRBQ	.18*	.23**	.44***	.20*	.06	.21*	1.00					
8. BL FSS	.11	.06	.35***	.13	.11	.18*	.46***	1.00				
9. BL DASS-A	.06	-.01	.28***	.15	.03	.12	.40***	.21**	1.00			
10.BL DASS-D	.06	.02	.29***	.08	-.02	.12	.46***	.43***	.45***	1.00		
11.BL DISRS	-.05	.06	.42***	.15	.08	.24**	.55***	.57***	.42***	.54***	1.00	
12.BL GSES	-.08	.01	.32***	.06	-.03	.21**	.42***	.24**	.39***	.27**	.41***	1.00
13.BL Age	.14	.03	-.24**	-.06	.01	-.38***	-.17*	-.25**	-.30***	-.30***	-.38***	-.39***

Note. BL = pretreatment. Predictor variables = pretreatment TWT and ISI. Outcome variables = posttreatment TWT and ISI. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21. Presumed mediating variables = pre- to posttreatment mean change scores of total time in bed (TIB), rise time variability (RT), sleep-related behaviours (SRBQ), ruminative tendency (DISRS), sleep effort (GSES), fatigue (FSS), anxiety, and depression (DASS-A, -D). TWT = sleep diary-measured Total Wake Time. ISI = Insomnia Severity Index. BL = pretreatment (pretreatment), Post = posttreatment. * = $p < .05$, ** = $p < .01$, *** = $p < .001$

Descriptive statistics. The means and standard deviations of all predictor, outcome, and mediating variables, as well as possible covariate variables, can be found below (Table 4).

Table 4

Descriptive Characteristics of Study Variables

Variables	Mean	SD	Minimum	Maximum	Range
Outcome					
Post TWT	1.2	0.7	0.1	4.2	4.1
Post ISI	9.9	5.9	0	25.0	25.0
Predictor					
BL TWT	2.9	1.3	0.8	7.6	6.8
BL ISI	19.6	3.5	11.0	28.0	17.0
BL TIB	8.7	1.1	6.2	12.5	6.3
BL RT	2.5	1.5	0	7.5	7.5
BL SRBQ	55.5	16.0	0	99.0	99.0
BL DASS-A	7.4	7.7	0	38.0	38.0
BL DASS-D	10.8	9.6	0	42.0	42.0
BL FSS	4.6	1.4	0	7.0	7.0
BL DISRS	49.6	12.8	0	78.0	78.0
BL GSES	9.0	3.5	0	14.0	14.0
Post TIB	7.4	0.9	5.1	10.1	5.0
Post RT	2.2	1.3	0	7.5	7.5
Post SRBQ	39.7	17.5	0	89.0	89.0
Post DASS-A	4.2	5.6	0	32.0	32.0
Post DASS-D	7.0	7.8	0	34.0	34.0
Post FSS	3.6	1.5	0	6.78	6.78
Post DISRS	40.8	13.2	0	80.0	80.0
Post GSES	5.4	3.0	0	13.0	13.0
Age	46.7	14.9	19.0	77.0	58.0

Note. Post = posttreatment, BL = pretreatment, change = pre- to posttreatment change score, TWT= Total Wake Time, ISI = Insomnia Severity Index, RT = variability in rise time, TIB = total time in bed (including naps), SRBQ = Sleep Related Behaviours Questionnaire, DASS = Depression, Anxiety, Stress Scales, DASS-A = Anxiety subscale of the DASS, DASS-D = Depression subscale of the DASS, FSS = Fatigue Severity Scale, DISRS = Daytime Insomnia Symptom Response Scale, GSES = Glasgow Sleep Effort Scale.

Tests of pre- to posttreatment differences. To assess whether participants' insomnia severity was significantly different between pre- and posttreatment, paired *t*-tests were conducted on the two main measures of insomnia severity (i.e., retrospective self-report on the ISI, prospectively monitored CSD TWT), comparing pre- and posttreatment means. When insomnia severity was measured using prospective monitoring (CSD TWT), participants showed a decrease in TWT between pretreatment ($M = 2.9$, $SD = 1.3$) and posttreatment ($M = 1.2$, $SD = 0.7$), and this decrease was statistically significant, $t(149) = 18.6$, $p < .0001$. When participants self-rated their insomnia severity (ISI score), they also showed decreases in their scores on the ISI between pretreatment ($M = 19.6$, $SD = 3.5$) and posttreatment ($M = 9.9$, $SD = 5.9$), and the difference was significant $t(159) = 22.0$, $p < .0001$.

Preliminary mediation analysis. To investigate whether pretreatment variables of interest mediated treatment outcomes, parallel multiple mediation analysis was performed in SPSS (PROCESS; Hayes, 2018). Total, direct, and indirect effects were considered significant at the .05 level. As previously described, all hypothesized mediating variables (TIB, RT, SRBQ, FSS, DASS-D, DASS-A, GSES, and DISRS) were analyzed simultaneously (i.e., parallel to one another) within a single integrated multiple mediation using PROCESS macro (Hayes 2018). Confidence intervals (CIs) were derived from 10,000 resamples of the sampling distribution. If the bounds of the 95% bias-corrected CIs did not contain zero, indirect effects were considered significant (i.e., the null hypothesis that the effects equalled 0 was rejected). Total and direct effects were considered significant if $p < .05$.

Results from the multiple parallel mediation analysis with pretreatment variables entered as hypothesized mediators are displayed in Table 5 (TWT as predictor and outcome variables)

and Table 6 (ISI as predictor and outcome variables). None of the pretreatment variables mediated treatment response.

Table 5

Total, Direct, and Indirect Effects of a Parallel Multiple Mediation Model Predicting Posttreatment TWT from Pretreatment TWT, with Hypothesized Mediator Variables at Pretreatment

Path	Coefficient	SE(HC3)+	*95% CI*		*t*	*p*
			Lower	*Upper*		
Total effect (c)*	.2416	.0527	.1376	.3457	4.5890	<.0001
Direct effect (c')*	.2520	.0580	.1373	.3667	4.3447	<.0001
Indirect effects						
Total	-.0103	.0234	-.0556	.0378		
a_1b_1 (TIB)	-.0122	.0113	-.0341	.0112		
a_2b_2 (RT)	-.0005	.0041	-.0065	.0104		
a_3b_3 (SRBQ)	.0077	.0135	-.0161	.0389		
a_4b_4 (DISRS)	-.0019	.0100	-.0237	.0196		
a_5b_5 (GSES)	.0005	.0062	-.0112	.0153		
a_6b_6 (FSS)	-.0016	.0068	-.0174	.0113		
a_7b_7 (DASS-A)	-.014	.0077	-.0226	.0107		
a_8b_8 (DASS-D)	-.0009	.0047	-.0131	.0066		

Note. TWT = total wake time. TIB = time in bed. RT = variation in rise time. SRBQ = Sleep Related Behaviours Questionnaire. DISRS = the Daytime Insomnia Symptom Response Scale. GSES = Glasgow Sleep Effort Scale. FSS = Fatigue Severity Scale. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21. CI = confidence interval.

* = $p < .0001$, or the CI does not straddle 0 (for indirect effects).

+ HC3 is a heteroscedasticity consistent estimator (Davidson & MacKinnon, 1993) that was applied within the PROCESS macro to account for heteroscedasticity in the data, as previously described.

Table 6

Total, Direct, and Indirect Effects of a Parallel Multiple Mediation Model Predicting Posttreatment ISI from Pretreatment ISI, with Hypothesized Mediator Variables at Pretreatment

Path	Effect (*B*)	SE	*95% CI*		*t*	*p*
			Lower	*Upper*		
Total effect (c)***	.5755	.1301	.3184	.8326	4.4232	<.0001
Direct effect (c')*	.4545	.1569	.1443	.7647	2.8960	.0044
Indirect effects						
Total	.1211	.1010	-.0757	.3231		
a_1b_1 (TIB)	.0082	.0182	-.0254	.0501		
a_2b_2 (RT)	.0210	.0279	-.0153	.0935		
a_3b_3 (SRBQ)	.1073	.0778	-.0366	.2713		
a_4b_4 (DISRS)	.0071	.0877	-.1669	.1813		
a_5b_5 (GSES)	-.0630	.0556	-.1830	.0425		
a_6b_6 (FSS)	.0505	.0685	-.0855	.1877		
a_7b_7 (DASS-A)	.0207	.0542	-.0860	.1335		
a_8b_8 (DASS-D)	-.0306	.0505	-.1371	.0673		

Note. ISI = Insomnia Severity Index. TIB = time in bed. RT = variation in rise time. SRBQ = Sleep Related Behaviours Questionnaire. DISRS = the Daytime Insomnia Symptom Response Scale. GSES = Glasgow Sleep Effort Scale. FSS = Fatigue Severity Scale. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21. CI = confidence interval.

*** = $p < .0001$. * = $p < .005$, or the CI does not straddle 0 (for indirect effects).

Primary Analyses

Qualitative. Per middle range theory, participant responses (n = 27) on the LTS were initially analyzed using direct application of the template. In this cycle of analysis, the PI purposefully searched for the presence of the presumed theoretical mechanisms of treatment (i.e., the expected 'ingredients' of CBT-I) in the data, using language from the *a priori* template during review of the LTS responses. Please refer to Appendix B for review of the template. During this cycle, LTS responses were read neither closely nor thoughtfully by the PI, nor were inferences about language and intent were made. Rather, responses were reviewed with continual reference to the template, with purposive searching for reference to the six *a priori* themes. In this cycle, participant responses showed frequent reference to the template themes, supporting *a priori* theory derived from the research literature.

After the template was applied to the data in the first cycle, the LTS responses were revisited using an I/C approach. During this cycle, the PI became immersed in the data. That is, the PI read each LTS carefully and thoroughly once, without noting common emergent themes or documenting themes that were reflected in the *a priori* template. After reading each LTS, the PI allowed time (i.e., 1-5 minutes) to process and consider each individual participant's reflections upon their treatment experiences and their descriptions of what they found to be most helpful, and of what they would like to remind themselves in future should their sleep begin to again interfere with their functioning/quality of life, or cause them heightened distress.

After this cycle of immersion, the third review cycle took place, and entailed re-immersion in the LTS documents with simultaneous written notation of candidate themes that were not reflected in the template, or may have aligned with template themes but did not directly support them. Notation of new and modified candidate themes was not limited in scope and

involved little attempt at thematic organization; instead, all possible themes were noted in this cycle. The PI documented possible codes in general clusters (e.g., "acceptance" and "mindfulness" were written on a different area of the page than "stimulus control" and "sleep hygiene"), however these clusters became amorphous over the course of this cycle, and the draft categories were significantly altered in later cycles. During this third cycle, nontemplate candidate themes were identified on the physical photocopies of the LTS using a system of colourful prompts (i.e., coloured sticky notes), with different colours representing different potential codes. Potential new themes were identified with the same colour, and the PI noted possible themes that each coloured sticky might represent; however the different coloured sticky notes were not necessarily bound to any singular theme, and many participant notes acquired several potential colours (i.e., the participant response could pertain to several candidate themes, and also support themes from the template). It was during this cycle that new candidate themes were posited as reflecting possible patterns in the data (one example of a theme note included: "coping skills? Self-efficacy, soothing / confidence? Themes of acceptance"). Data were reviewed in this cycle until saturation was reached; that is, until no additional information relevant to the template and candidate themes were observable.

During the fourth cycle, each individual participant LTS document (i.e., the LTS responses) was transcribed to a table in Microsoft Word, following guidelines for performing qualitative research on computers (Hanh, 2011). During this cycle, thematic clusters were subjected to the first stage of refinement. Specifically, refinement included the merging of themes/clusters that were quite clearly similar or overlapped (e.g., "managing alcohol consumption," and "managing caffeine intake," were collapsed into a single *nutrition/health* theme), and separation of themes that appeared to represent more than one theme (e.g.,

"acceptance or mindfulness" became two discrete codes). Possible new themes not previously identified were also noted during this cycle. For example, *words of encouragement to oneself* ("You can do it!"), which had previously been subsumed by other themes such as s*elf-efficacy* or *confidence*, emerged as a new theme: *optimism*. Finally, during this stage erroneous information unsupported by our CBT-I protocol and the CBT-I literature more broadly, or information that was not included at all within the treatment protocol, were also amalgamated under the theme "*contradictory to empirical evidence*."

The fifth cycle involved refinement of each theme, including making decisions about the wording of each theme, with the goal of facilitating illustrative, well-defined thematic categories. In this cycle, for example, the template theme "cognitive restructuring strategies" and emergent theme "coping with worry" were subsumed under the more explanatory "counterarousal" theme. The final codes were again compared with the raw participant data to ensure no patterns were overlooked. After again confirming that saturation had been reached, any remaining overlap between themes was examined and themes were dissected to evaluate whether revision might increase clarity; for example, the *a priori* theme "focus of attention, effort, and behaviours around themes of sleep" was parsed into "confidence" (for responses reflecting "thinking like a good sleeper" themes) and "safety behaviours" (e.g., refraining from reliance on sleep medications).

A final sixth cycle involved review of the emergent themes with the PI's research supervisor, Dr. Colleen Carney. After discussion and review of the CBT-I literature, it was agreed that participant responses containing language around broad themes of confidence, optimism, and acceptance of one's sleep represented a single unified theme, ultimately subsumed under the thematic label "self-efficacy." That is, after discussion with an expert in the field of

insomnia, reflection on the emergent themes, and re-immersion in the participant data, the theme of self-efficacy crystallized as a single theme subsuming the candidate themes of confidence, acceptance, and optimism.

Emergent Treatment Component Themes. After application of the template to LTS data and cycles of I/C, modification, and refinement, a total of 11 themes in participant responses were observed. The 11 themes comprised both expected theoretical factors (i.e., those components upon which CBT-I is based) and unanticipated CBT-I themes. The 11 identified themes are presented in Table 7 with brief descriptions.

Table 7

Emergent Treatment Component Themes, with Descriptions and Examples from Participant Responses on the LTS

Theme	Description	Participant Examples
1. Sleep schedule consistency	Sleep routine Reduced variability in RT	"Developing a regular sleep schedule (aprox. 11:30PM-7:30AM)" (70982) "Keep a more or less same bed & rise time" (70841) "Structure bedtime + up time as regularly as possible" (70909) "Unless there are extraordinary circumstances, it is most effective to stick to the schedule 11pm-6am." (70899)
2. Sleep compression	Restricted TIB window Earliest BT, latest RT No naps	"What has worked for you was a 6.5 hour sleep window. Going to bed at 11 30pm or later and waking up at 6am ensured proper sleep dirve [sic] would build the next day and you're not spending too much time in bed." (70828) "Get out of bed once you wake up to build up sleep drive." (71061) "Getting up early even after a late night" (70933) "shorten sleep hours to average time + have conistiant going to bed + getting up times" (70988)
3. Stimulus control	Conditioning: The bed/bedroom is only for sleeping (and sex), including: Remove wakeful activities (e.g. television, reading, work) Get out of bed if sleep is not coming; return only after sleepiness comes Only attempt sleep once sleepy	"getting out of bed when I cannot sleep is helpful and less frustrating than laying in bed." (71045) "Reducing stimuli in bed" (70982) "get out of bed if not tired" (70919)

4. Counterarousal	Strategies for coping with arousal, distress: Challenging maladaptive thoughts about sleep Scheduling worry time Buffer zone / wind down time pre-sleep Relaxation techniques e.g. deep breathing, progressive muscle relaxation Soothing techniques e.g. self-care, hugging Mindfulness, meditation Refrain from watching clock, calculating hours remaining during night Journaling	"Not put pressure or think about how many hours of sleep I should get, or even think about sleep at all." (70858) "Do not think or worry about things in bed – focus on breath if need to relax." (70841) "Setting aside a separate worry time during the day takes discipline, but can be useful during times of stress." (70899) "mindfulness, meditation, Being Present" (70988)
5. Psychoeducation	Normalizing sleep difficulties Sleep needs differ between people Sleep need differs depending on daytime activities Quality of sleep matters more than quantity Impact of circadian rhythm Automatic compensatory mechanism in the body (making up for lost sleep)	"Each and every person's need for sleep is different" (70992) "It is ok not to have an 8 hr sleep." (71061) "Not everyone needs 8 hours of sleep a night" (70841
6. Daytime behavioural activation	Complete typical or planned daytime activities Avoid avoidance Do not isolate – continue to socialize Exercise Sleep drive (builds over the day with increased activity, deteriorated by naps/sedentary)	"Activities to look forward to. Live life as normal/usual as possible" (70988) "Exercise is always effective at re-energizing, even after a bad night." (70899) "Activities to look forward to. Live life as normal/usual as possible" (70988)
7. Sleep hygiene	Bedroom quiet (e.g., ear plugs, white noise) Bedroom environment dark (e.g. sleep mask, blinds) Exercise during the day Limit caffeine, alcohol	"Don't's: use caffeine – especially after noon.. Drink alcohol. Limit to 2 servings (max!) per day Neglect good exercise. It promotes good quality sleep" (71070) "reduce caffeine intake to no later

		than 3PM wear a mask to reduce ambient light disturbance" (70909)
8. Reduced safety behaviours	Reduced sleep effort Reduced sleep-related safety behaviours (e.g., bedtime routines needed, medications required)	[Don't] "Rely on medications for sleep – kick them." (71070) "Not taking sleeping peels [sic] also opened my eyes that medication actually didn't make it better at all." (70962)
9. Monitoring sleep	Using a sleep diary to track sleep pattern	"Keeping the sleep log to track hours" (70933) "Keep sleep diary every morning (71070)
10. Sleep self-efficacy	Confidence in oneself Confidence that sleep will improve, or gains will be maintained Knowledge that one has the tools to return to good sleep Belief in the ability to sleep well Resilience of self, body Trusting one's body to sleep Thinking like a good sleeper Acceptance of sleep difficulties as part of life Acceptance of shorter sleep duration than expected Belief in one's ability to function during the day Belief that the future is bright Positive self-statements/beliefs about self, future, sleep	"You will be fine, I promise." (70867) "Honour and accept my body's natural rhythm" (70988) "All will be well. All is well." (70841) "But most importantly – don't stress over it! One or two nights of bad sleep will not ruin your life. You've got this!" (70982)
11. Contradictory to empirical evidence	Statements about strategies not supported by the empirical evidence, extant research Suggestions that are not reflective of CBT-I protocol; thereby reflecting misunderstanding, misinformation from elsewhere, or therapist error in information conveyed Nonempirically supported strategy that client found to be helpful	"no night eating" (70898) "Thinking of benefits of sleep (not feeling guilty for sleeping)" (70933)

Primary mediation analysis. To examine the primary hypothesis that pre- to posttreatment change variables mediated treatment response, parallel multiple mediation analysis was conducted using PROCESS in SPSS (Hayes, 2018). Bootstrapping analytic procedures replicated those conducted with pretreatment variables in the preliminary analyses. To reiterate, 10,000 bootstrapping resamples were performed to derive confidence intervals (CIs; bias-corrected at 95%). Indirect effects were considered significant if the upper and lower bounds of the CIs did not straddle zero. Total and direct effects were considered significant at the $p < .05$ level. As previously described, hypothesized mediating variables (pre- to posttreatment change in TIB, RT, SRBQ, FSS, DASS-D, DASS-A, GSES, and DISRS) were analyzed in parallel.

In the mediation analysis predicting posttreatment TWT, TIB emerged as the only mediator variable that had a significant indirect effect on treatment response (lower CI: -.0865, upper CI: -.0040). Complete results from the mediation analysis predicting posttreatment TWT from pretreatment TWT are presented in Table 8. Figure 4 shows the pathway coefficients of the model. In the analysis predicting posttreatment ISI, SRBQ emerged as the only mediator variable that had a significant indirect effect on treatment response (lower CI: -.1831, upper CI: -.0094). Complete results from the mediation analysis predicting posttreatment ISI from pretreatment ISI are presented in Table 9. Figure 5 shows the pathway coefficients of the model.

Table 8

Total, Direct, and Indirect Effects of a Parallel Multiple Mediation Model Predicting Posttreatment TWT from Pretreatment TWT, with Hypothesized Change Variables Entered as Mediators

Path	Coefficient	SE(HC3)+	*95% CI*		*t*	*p*
			Lower	*Upper*		
Total effect (c)*	.2552	.0537	.1491	.3613	4.7536	<.0001
Direct effect (c')*	.2864	.0562	.1752	.3975	5.0940	<.0001
Indirect effects						
Total	-.0312	.0325	-.1009	.0261		
a_1b_1 (TIB)*	-.0402	.0212	-.0865	-.0040		
a_2b_2 (RT)	.0010	.0042	-.0066	.0117		
a3b_3 (SRBQ)	-.0048	.0095	-.0300	.0083		
a_4b_4 (DISRS)	-.0056	.0082	-.0256	.0070		
a_5b_5 (GSES)	.0079	.0088	-.0047	.0291		
a_6b_6 (FSS)	.0077	.0105	-.0116	.0307		
a_7b_7 (DASS-A)	.0018	.0044	-.0068	.0120		
a_8b_8 (DASS-D)	.0010	.0040	-.0061	.0108		

Note. a_1 = TIB, a_2 = RT, a_3 = SRBQ, a_4 = DISRS, a_5 = GSES, a_6 = FSS, a_7 = DASS-A, a_8 = DASS-D. TWT = total wake time. TIB = time in bed. RT = variation in rise time. SRBQ = Sleep Related Behaviours Questionnaire. DISRS = the Daytime Insomnia Symptom Response Scale. GSES = Glasgow Sleep Effort Scale. FSS = Fatigue Severity Scale. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21. CI = confidence interval.

* = $p < .0001$, or the CI does not straddle 0 (for indirect effects).

+ HC3 is a heteroscedasticity-consistent estimator (Davidson & MacKinnon, 1993) that was applied within the PROCESS macro to account for heteroscedasticity in the data, as previously described.

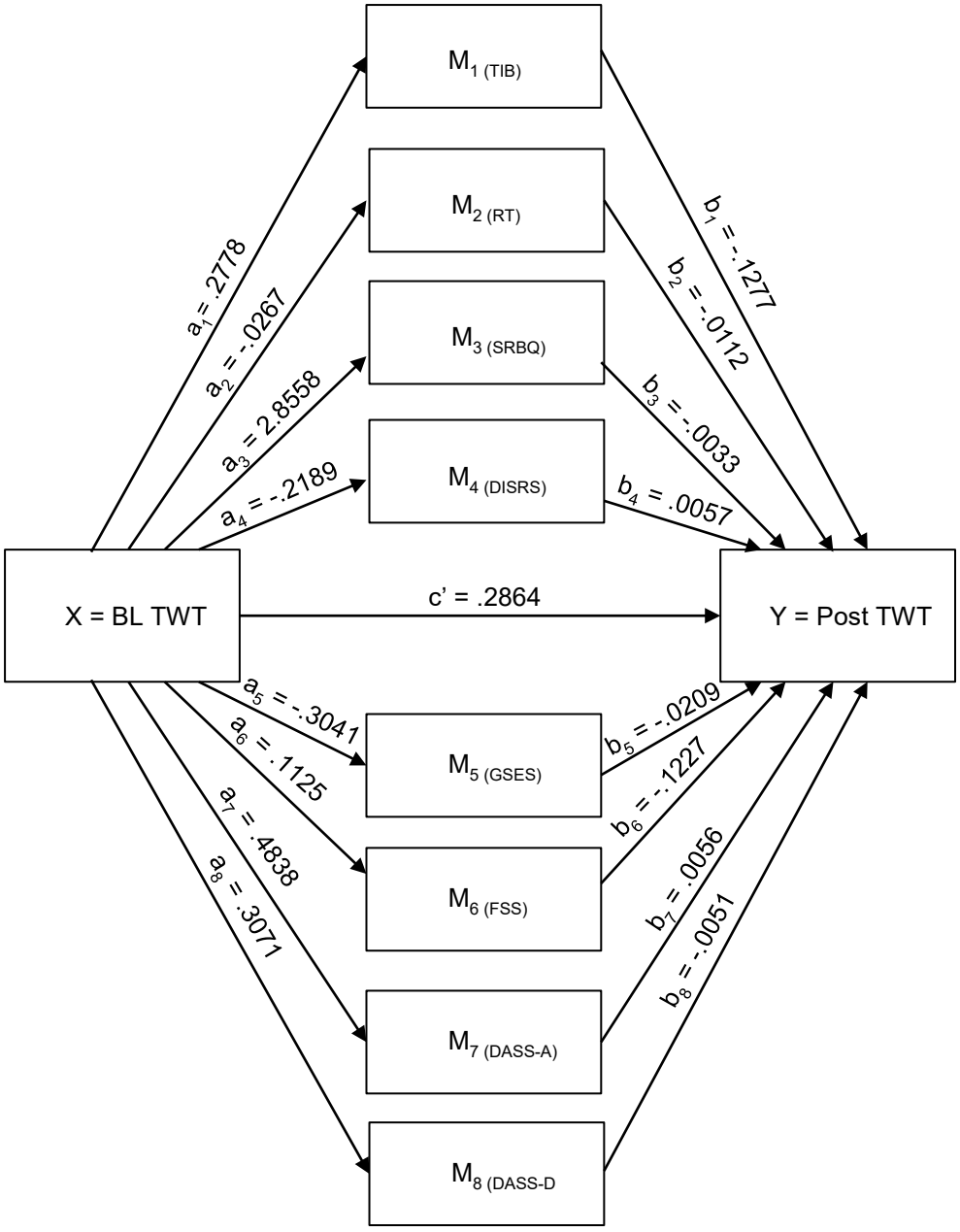

Figure 4. Parallel multiple mediation model predicting posttreatment TWT ("Post TWT) from pretreatment TWT ("BL TWT"), including coefficients for the hypothesized mediator pathways (*a* and *b* pathways), and the direct effect of X on Y, controlling for the mediators (*c'* pathway).

Table 9

Total, Direct, and Indirect Effects of a Parallel Multiple Mediation Model Predicting Posttreatment ISI from Pretreatment ISI, with Hypothesized Change Variables Entered as Mediators

Path	Coefficient	SE	*95% CI*		*t*	*p*
			Lower	*Upper*		
Total effect (c)*	.6079	.1349	.3412	.8746	4.5052	<.0001
Direct effect (c')*	-.0312	.1005	.5816	.9792	7.7628	<.0001
Indirect effects						
Total	-.1725	.0994	-.3751	.0195		
a_1b_1 (TIB)	-.0011	.0113	-.0253	.0241		
a_2b_2 (RT)	.0073	.0148	-.0208	.0410		
a_3b_3 (SRBQ)*	-.0786	.0451	-.1831	-.0094		
a_4b_4 (DISRS)	-.0396	.0345	-.1252	.0073		
a_5b_5 (GSES)	-.0340	.0341	-.1051	.0311		
a_6b_6 (FSS)	-.0455	.0368	-.1285	.0154		
a_7b_7 (DASS-A)	.0435	.0302	-.0045	.1114		
a_8b_8 (DASS-D)	-.0244	.0235	-.0777	.0145		

Note. ISI = Insomnia Severity Index. TWT = total wake time. TIB = time in bed. RT = variation in rise time. SRBQ = Sleep Related Behaviours Questionnaire. DISRS = the Daytime Insomnia Symptom Response Scale. GSES = Glasgow Sleep Effort Scale. FSS = Fatigue Severity Scale. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21. CI = confidence interval. ** = $p < .0001$, or the CI does not straddle 0 (for indirect effects).

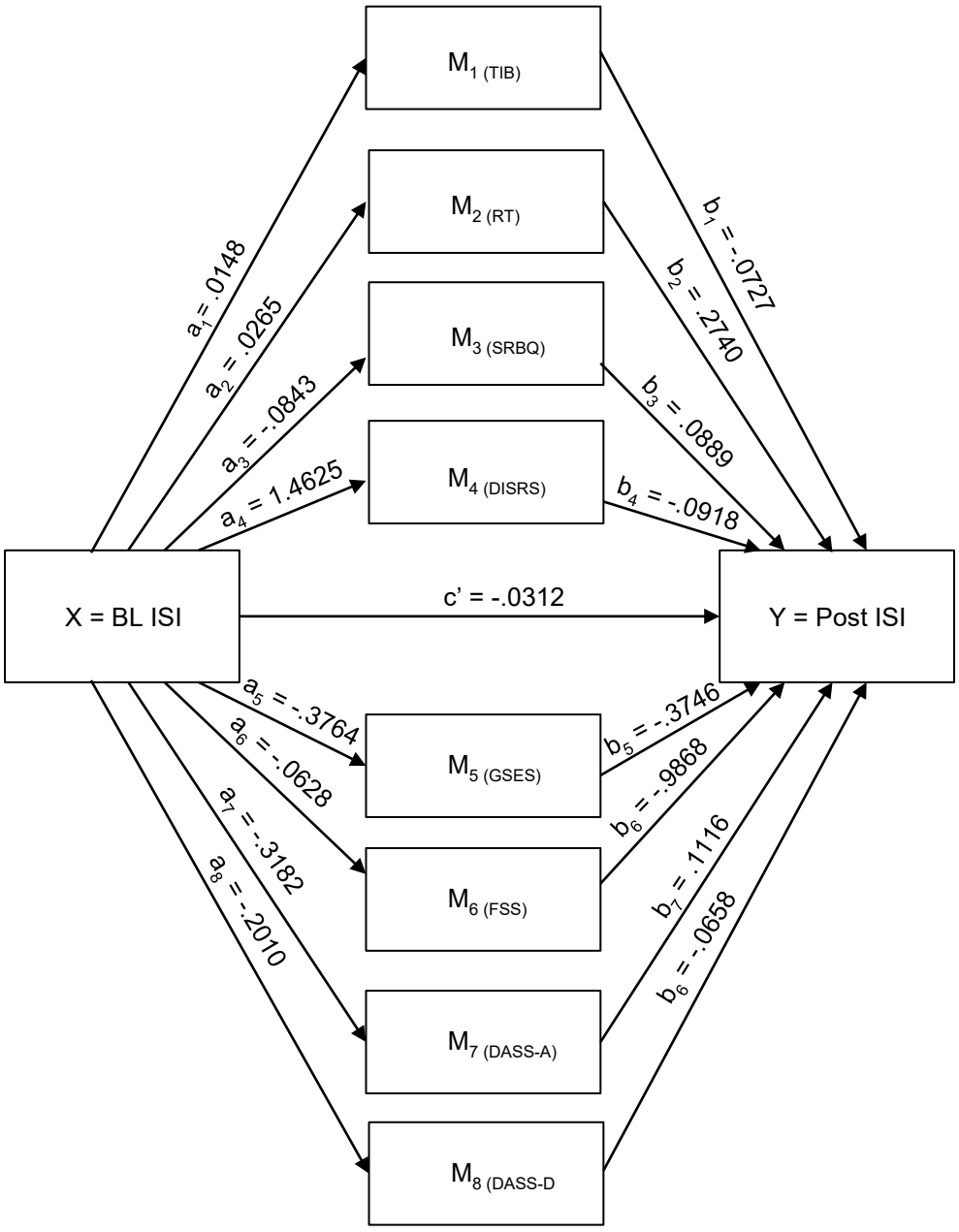

Figure 5. Parallel multiple mediation model predicting posttreatment ISI ("Post ISI") from pretreatment ISI ("BL ISI"), including coefficients for the hypothesized mediator pathways (*a* and *b* pathways), and the direct effect of X on Y, controlling for the mediators (*c'* pathway).

Secondary Analyses

Moderation. Two hierarchical linear regression analyses were conducted to assess the extent to which the variables of interest predicted posttreatment outcome on the two key measures of treatment response (i.e., posttreatment TWT and ISI). That is, we wanted to understand the way in which participants' pretreatment characteristics impacted their response to CBT-I treatment (i.e., their insomnia severity following treatment). For both analyses, the outcome variable was posttreatment insomnia severity (as assessed by TWT or ISI); pretreatment insomnia severity (pretreatment TWT or ISI) was entered in the first step in each analysis respectively. Interaction terms for the hypothesized moderating variables (i.e., TIB, RT, SRBQ, DISRS, GSES, FSS, DASS-A, and DASS-D at pretreatment) were computed in SPSS by multiplying each individual variable by the predictor variable in each respective analysis (i.e., pretreatment TWT, pretreatment ISI) to yield interaction (product) terms. The eight computed interaction terms were entered in the second step for each regression analysis.

In the regression analysis that used TWT as predictor, the model was significantly predictive of posttreatment response (TWT) in the first step, adjusted $R^2 = .21$, $F(1,145) = 40.77$, $p < .0001$. The addition of the interaction terms significantly improved the predictive value of the model, adjusted $R^2 = .28$, $F(8,137) = 2.63$, $p = .01$. The Durbin-Watson test yielded a value of 1.99, suggesting that the assumption of independent errors was met per guidelines that values close to 2 are indicative of the independence of errors, while values less than 1 or greater than 3 suggest this assumption may be violated (Field, 2009). See Table 10 for beta, standard error, and partial correlation values. For a graphical depiction of the interaction effect, please refer to Figure 6.

Table 10

Hierarchical Linear Regression Analysis Predicting Posttreatment TWT from Pretreatment TWT, with Hypothesized Moderating Variables Entered as Interaction Terms in the Second Step

Variables		β^{+}	*SE*	*t*	*p*	Partial *r*
Step 1	BL TWT***	.493	.042	6.422	< .001	.481
Step 2	BL TIB x BL TWT	.038	.054	.493	.623	.042
	BL RT x BL TWT**	.225	.052	2.993	.003	.248
	BL SRBQ x BL TWT	-.052	.058	-.577	.565	-.049
	BL DISRS x BL TWT	-.012	.076	-.107	.915	-.009
	BL GSES x BL TWT	-.069	.058	-.800	.425	-.068
	BL FSS x BL TWT	-.006	.061	-.068	.946	-.006
	BL DASS-A x BL TWT	-.232	.054	-2.871	.005	-.238
	BL DASS-D x BL TWT	.186	.075	1.935	.055	.163

Note. ISI = Insomnia Severity Index. TWT = total wake time. TIB = time in bed. RT = variation in rise time. SRBQ = Sleep Related Behaviours Questionnaire. DISRS = the Daytime Insomnia Symptom Response Scale. GSES = Glasgow Sleep Effort Scale. FSS = Fatigue Severity Scale. DASS-A = Anxiety subscale of the DASS-21. DASS-D = Depression subscale of the DASS-21.

+Standardized regression coefficients are estimates from the regression model.

*** = $p < .001$, ** = $p < .005$, * = $p < .01$

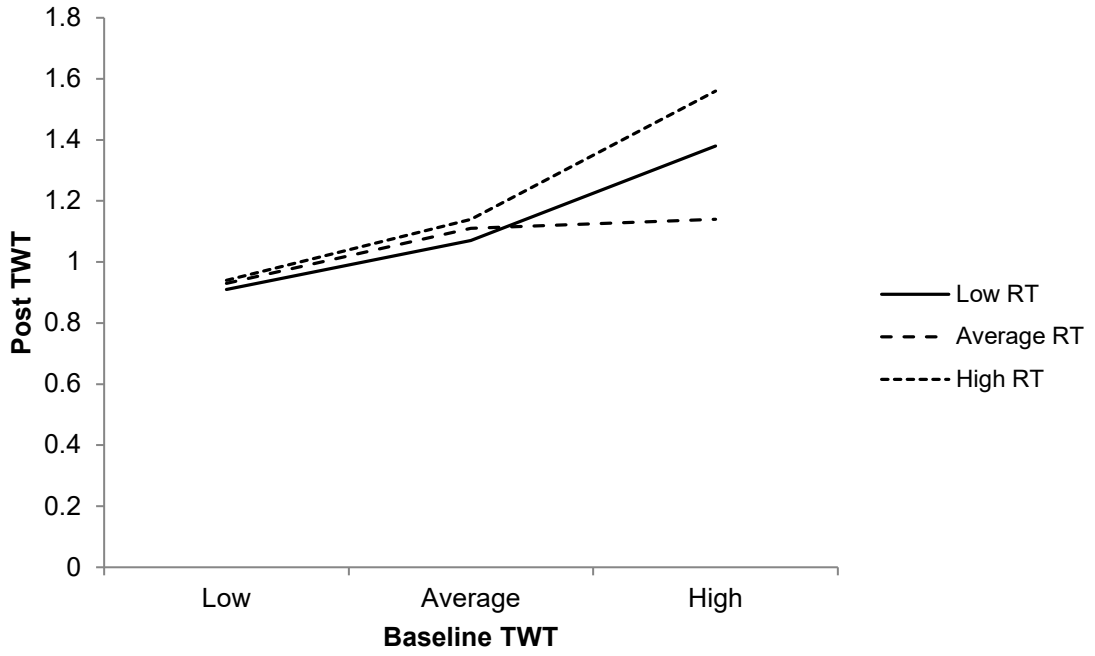

Figure 6. Graphical depiction of the interaction effect between TWT at pretreatment and the interaction term (product of pretreatment TWT and pretreatment RT).
Note. RT = variation in rise time. TWT = total wake time.

In the second analysis, the hierarchical linear regression analysis showed that ISI at pretreatment was significantly predictive of response (on the ISI) at post treatment in the first step, adjusted $R^2 = .11$, $F(1,152) = 19.69$, $p < .0001$. The addition of the interaction terms did not significantly improve the predictive value of the model, adjusted $R^2 = .12$, $F(8,144) = 1.19$, $p = .31$. The Durbin-Watson test yielded a value of 2.15, suggesting that the assumption of independent errors was met. See Table 11 for beta, standard error, and partial correlation values.

Table 11

Hierarchical Linear Regression Analysis Predicting Posttreatment ISI from Pretreatment ISI, with Hypothesized Moderating Variables Entered as Interaction Terms in the Second Step

Variables		β^{+}	*SE*	*t*	*p*	Partial *r*
Step 1	BL ISI***	.370	.140	4.490	< .0001	.350
Step 2	BL TIB x BL ISI	.159	.565	1.913	.058	.157
	BL RT x BL ISI	.052	.458	.640	.523	.053
	BL SRBQ x BL ISI	.133	.585	1.219	.225	.101
	BL DISRS x BL ISI	.088	.706	.659	.511	.055
	BL GSES x BL ISI	-.034	.548	-.357	.721	-.030
	BL FSS x BL ISI	-.005	.662	-.043	.966	-.004
	BL DASS-A x BL ISI	-.046	.623	-.384	.702	-.032
	BL DASS-D x BL ISI	.015	.711	.111	.912	.009

+Standardized regression coefficients are estimates from the regression model.

** = p < .01, * = p < .05

Posthoc Analyses

At pretreatment (i.e., the screening interview), 43.36% of participants were using a prescribed medication; at posttreatment, a number of individuals had discontinued or tapered off their medications, with only 29.41% of participants reporting medication use at that time point.

To assess between groups differences on posttreatment insomnia severity, independent samples *t*-tests were conducted in SPSS. In terms of prospectively monitored posttreatment insomnia severity (i.e., TWT on the CSD), there were no differences between those who reported medication use ($M = 1.26$) and those who did not ($M = 1.12$) at the screening interview, $t(100) = -0.97$, $p = .34$. When posttreatment insomnia severity was assessed via retrospective self-report (i.e., ISI score), there were no differences between those who reported medication use ($M = 10.67$) and those who did not ($M = 9.55$) at the screening interview, $t(106) = 0.93$, $p = .34$.

To examine whether comorbid psychiatric concerns impacted treatment outcomes, independent *t*-tests were conducted. The only between groups differences on posttreatment insomnia severity were those with symptoms consistent with major depressive disorder (MDD) according to the pretreatment screening interview ($M = 13.12$) had significantly higher self-reported posttreatment insomnia severity (i.e., ISI score) compared to those without MDD ($M = 9.65$), $t(127) = 2.87$, $p < .01$. There were no between groups differences in terms of depression when posttreatment insomnia severity was measured by TWT on the CSD. Results from analyses of other comorbidity groups are summarized in Table 12.

Table 12

T-Tests of Between Groups Differences on Posttreatment Insomnia Severity Across Psychiatric Comorbidities

Comorbidity	Frequency (%)	Posttreatment insomnia severity	*t*(df)	*p*
MDD	21.1	TWT	1.58	.12
		ISI	2.87*	.005
GAD	26.7	TWT	0.23	.82
		ISI	0.65	.52
SAD	13.0	TWT	-0.23	.82
		ISI	0.32	.75
Panic disorder	20.5	TWT	0.25	.81
		ISI	-0.28	.78
Agoraphobia	3.7	TWT	-0.37	.71
		ISI	0.21	.84
PTSD	1.9	TWT	1.12	.26
		ISI	-0.03	.97

Note. MDD = major depressive disorder. GAD = generalized anxiety disorder. SAD = social anxiety disorder. PTSD = posttraumatic stress disorder. TWT = total wake time. ISI = Insomnia Severity Index. * = $p < .01$

In summary, the results of the primary analyses showed that participants reported themes consistent with the presumed theoretical factors underlying CBT-I to be helpful for improving their sleep. Participants also indicated that increasing their sense of self-efficacy was important for them during treatment. Results from the primary mediation analyses showed that change in TIB significantly mediated treatment response when insomnia severity was measured via CSD TWT. When measured through self-reported ISI score, change in sleep-related safety behaviours (i.e., change in SRBQ) mediated treatment response. Results from the moderation analyses showed that pretreatment RT variability was a significant moderator of treatment response when insomnia severity was assessed via CSD TWT. When assessed by ISI score, none of the moderator terms emerged as significant.

Discussion

Participant perspectives on the aspects of treatment that were most helpful supported the theoretical factors upon which CBT-I was developed, and as hypothesized, also yielded several themes that were not hypothesized in the current study, and are not explicitly considered to be mechanisms of treatment response in the CBT-I literature. Total time spent in bed and sleep-related safety behaviours mediated pre- to posttreatment response differentially, depending on whether outcomes were measured via prospective monitoring (CSD TWT) versus retrospective self-report (ISI score). Specifically, those with longer TWT durations at pretreatment showed significantly decreased TWT at posttreatment via the mediating variable of change in TIB. Contrary to hypotheses and research evidence, reduction in sleep-related safety behaviours did not appear to mediate the relationship between retrospective, self-reported insomnia severity (i.e., ISI score) in the expected direction. Those with poorer self-reported insomnia severity (i.e., higher distress) at pretreatment tended to make fewer changes to sleep-related safety behaviours, but nonetheless showed decreased self-reported insomnia severity at posttreatment. The other

variables expected to influence treatment response in the mediation analyses (i.e., change in rise time variability, change in sleep effort) did not emerge as significant mediators.

Although none of the pretreatment variables mediated outcomes in the preliminary analysis, it was nevertheless considered important to proceed to examination of the change variables as potential mediators of treatment outcomes, as we expected that results might change when the mediator variables represented change over the course of treatment as opposed to static, pretreatment scores on measures administered at the screening interview prior to the CBT-I intervention. As aforementioned, this assumption was supported by the significant mediators that emerged in the primary analyses. In terms of the secondary analyses, pretreatment rise time variability appeared to have the strongest impact on the strength of the relationship between pretreatment and posttreatment insomnia severity, as measured by TWT on the sleep diary.

Results from the qualitative component of this study both support and extend the existing literature on presumed mechanisms of treatment response in CBT-I. Key ingredients of CBT-I considered most important for insomnia improvement were supported by participant responses on the LTS, including: sleep compression, stimulus control, sleep schedule consistency, countering safety behaviours, and counterarousal. That is, those theoretical factors upon which CBT-I was developed were found to be important to participants in this study. Factors included in the treatment protocol that were expected to be neither critical to treatment response nor considered imperative by clients also appeared in LTS responses, such as psychoeducation about sleep, daytime behavioural activation, sleep hygiene, and simply monitoring one's sleep using the sleep diary. Participants also resoundingly reported themes of self-efficacy to be beneficial to their sleep over the course of treatment. Finally, there was a category of responses that

emerged from the data that actually contradict the empirical evidence upon which CBT-I is based, but which participants reported to be helpful on the LTS.

Self-efficacy was not hypothesized to emerge as a theme reported by participants to be a helpful treatment component in the current study. The CBT-I protocol does not explicitly direct therapists to review self-efficacy with clients, nor does it explicitly state that fostering a sense of self-efficacy in clients is key to their symptom improvement. Further, self-efficacy about sleep was not generally considered to be a perpetuating factor of chronic insomnia by Spielman and colleagues in the early theoretical literature (1987). However, self-efficacy about sleep – including acceptance, confidence, optimism, "thinking like a good sleeper," and trust in one's body to perform sleep – is in many ways a tacit objective of CBT-I. Although the CBT-I protocol does not explicitly direct therapists to focus on building self-efficacy during therapy sessions, it can be argued that in fact the overarching goal of CBT-I is to improve clients' trust in their body's ability to perform sleep, *without* them meddling in the process by adjusting their sleep schedules, avoiding daytime activities, adding safety behaviours, or using cognitive energy focusing on their symptoms in a maladaptive fashion.

Individuals with "good," or normal sleep, from a population perspective, tended not to engage in such behaviours. Considering the perpetuating factors mapped out by Spielman and his colleagues (1987), it follows that individuals who do *not* engage in behaviours that maintain insomnia symptoms in the long term most likely possess as a sense of self-efficacy and confidence about their sleep. For example, after experiencing a precipitant such as loss of one's job, after which one's sleep deteriorates and insomnia symptoms arise in the short-term, an individual theoretically may possess as a sense of self-efficacy about their ability to once again return to good quality sleep in future. Such an individual may believe fluctuations in sleep

quality to be normative depending on life stressors, or have an inherent sense of confidence in their body's ability to meet their sleep needs in the longer-term. This individual would likely notice their symptoms improve over time, and insomnia would likely remain acute and not become chronic as a result of these beliefs.

Alternatively, this same individual may believe their insomnia symptoms to be representative of permanent change, or some inherent "problem" with their body. Individuals with low self-efficacy regarding their ability to produce normative sleep, or return to good quality sleep again in future, might lead to them opting to engage in those behaviours considered to be perpetuating in the long run (e.g., sleeping in, worrying about the sleep difficulty and its impact, napping, using alcohol to induce drowsiness). These perpetuating behaviours tend to lead to more chronic insomnia, and over time these are the individuals who tend to be seen by CBT-I therapists.

Thus, it makes sense that boosting one's overall sense of self-efficacy about one's sleep would likely lead to improved sleep, via the pathway of adjustment of those theoretical factors presupposed to function as mechanisms of change in CBT-I. In other words, client self-efficacy is an inherent goal of CBT-I insofar as it is a therapy that offers strategies for improving sleep that are based on those theoretical factors that are frequently employed by those with chronic insomnia due to fear about their ability to produce sleep. Adjustment of these maladaptive cognitions and behaviours clearly leads to symptom improvement, and in so doing, likely augment clients' belief in their ability to sleep. Indeed, research has shown that CBT-I improves sleep-related self-efficacy, in addition to insomnia symptoms and maladaptive beliefs about sleep (Edinger & Sampson, 2003). Another study found that adherence to CBT-I recommendations

was associated with participant perceptions about their self-efficacy related to sleep (Bouchard, Bastien, & Morin, 2003).

It is also possible that self-efficacy is targeted by the psychoeducation provided; for example, the normalization of sleep duration and the rationale for reducing maladaptive behaviours may cause clients to realize that their bodies knew how to acquire good quality sleep all along, thereby implanting a sense of self-efficacy that leads to confident implementation of the CBT-I recommendations. Regardless of the way in which self-efficacy manifests in clients over the course of CBT-I, it makes sense that those with increased self-efficacy about their ability to sleep report this to be a helpful component of treatment. Insomnia is a highly subjective disorder; an individual with a strong belief, and confidence, in their ability to obtain good quality sleep would be much less likely to meet DSM-5 criteria for insomnia disorder compared to an individual with a low sleep-related self-efficacy. That participants in the current study emphasized the importance of self-efficacy in improving their sleep following a course of CBT-I is therefore unsurprising, despite the fact that this was not posited as factor hypothesized to be reported as helpful to participants in the current study.

With regard to the finding that some participants endorsed factors that are not supported by the empirical insomnia literature, there are several possibilities. It is possible that during treatment sessions, study therapists provided, subtly enforced, or failed to correct participants' incorrect assumptions about how to improve their sleep. Alternatively, it may have been the case that some participants entered this trial having already performed substantial research into insomnia treatments and remedies, which may have included components not supported by the literature (e.g., naturopathic remedies, use of guided audio tapes during sleep, napping), and these beliefs failed to shift substantially over the course of treatment. Anecdotal evidence

suggests that many clients reported remedies suggested by friends, colleagues, or others in their lives who offered solutions for sleep difficulties which would not be indicated by the current research evidence, and for some clients, first-person "case study" evidence may hold more weight than recommendations from the research, and it may also be the case that some participants held on to these remedies recommended by (nonsleep experts) in their lives as other possible solutions to their sleep difficulty.

It is also possible that participants in the current study began this informal research into sleep remedies during the course of CBT-I, increasing their focus on sleep-related matters during the time they were involved with the intervention, and as such the details of CBT-I recommendations were confused with suggestions from other sources. To fully untangle the source of nonempirically supported information noted to be helpful by participants in the current study would likely require lengthy exit interviews at the end of treatment, subsequent to analysis of which treatment components they found to be helpful. Regardless of the source, it is interesting that some participants found these nonempirically supported to be helpful during treatment, and suggests that correcting or eliminating such factors may not be of foremost importance during treatment.

It is important to note that while many participants reported these nonevidence based factors to be helpful, they also included CBT-I treatment components such as sleep schedule consistency, sleep hygiene recommendations, and sleep compression to be helpful; that is, although there was some report of nonrecommended strategies for improving their sleep, these clients also tended to also recall evidence-based components of CBT-I that they did find to be helpful. It is possible, then, that the factors that were reported that were not empirically-based may have appeared helpful to clients *in conjunction with* their adherence to the other, empirically

supported aspects of treatment that they found to be beneficial. That is, it may be that some factors functioned as something of placebos in terms of efficacy, while the true mechanisms of change were actually the empirically based tenets of CBT-I.

That the main treatment components of CBT-I emerged as themes in participant responses on the LTS, as hypothesized, supports the idea that clients recognize that these aspects of treatment truly are beneficial for improving their sleep. Many clients of CBT-I find some of the keystone CBT-I recommendations to be difficult to implement (e.g., stimulus control, sleep compression) and can often seem counterintuitive (i.e., shortening the sleep period in order to "get better sleep"). That participants nonetheless identified these themes as beneficial to their insomnia treatment is significant. It suggests that while these same clients may have had doubts or hesitation about implementing some of the recommended strategies, after four sessions of CBT-I they likely subjectively noticed their own symptom improvement and realized that the improvement resulted from these often difficult-to-implement behavioural strategies.

It could be argued that participants merely noted the same CBT-I recommendations offered to them in the readings and literature provided to them over the treatment sessions; however the LTS responses were all notably idiosyncratic, and none appeared to directly reflect every CBT-I recommendation. Indeed, while some participants noted only one or two aspects of treatment that they found helpful, others wrote narrative "letters" to their future selves, which often included themes of encouragement and self-efficacy. While some letters included additions to the LTS that appeared to be added during the fourth and final CBT-I session (i.e., upon reflection of their treatment progress with their therapist), no LTS summarized the CBT-I recommendations completely, accurately, and concisely. Given that the responses appeared to be entirely self-generated in this respect, it is most parsimonious to infer that participants who

endorsed the theoretical factors of CBT-I to be helpful indeed consider these strategies beneficial, and also plan to re-implement these strategies in future should symptoms re-emerge. These results suggest that clients find the main tenets of CBT-I to be helpful, and also that a 4-session CBT-I protocol is adequate for emphasizing these main strategies such that at least some of the key strategies may be recalled spontaneously by clients by the end of treatment.

With regard to the mediation analyses, when assessing treatment response via sleep diary measured index of sleep (i.e., CSD TWT), TIB was the only variable that significantly mediated the relationship between pre- and posttreatment insomnia severity (i.e., TWT), and the indirect effect was negative. Participants who differed by one unit on pretreatment TWT were estimated to differ by -.04 units in their posttreatment TWT, via their change in TIB. That is, those with higher pretreatment TWT had lower posttreatment TWT (as indicated by the negative indirect effect) as a result of the tendency for those with higher pretreatment TWT to have a larger change in TIB (as indicated by the positive path between the predictor variable pretreatment TWT and mediator TIB change), which in turn was associated with a decrease in posttreatment TWT (as indicated by the negative pathway between the mediator TIB change and outcome variable posttreatment TWT). More simply put, individuals who had more severe insomnia (as measured by lengthier total wake times) before treatment showed better improvement in their insomnia severity at posttreatment due to significantly decreased time spent in bed. These results support the hypothesis in the current study that change in TIB would mediate treatment outcome, as well as more broadly supports the theory-based, central tenet of CBT-I that compression of the window of time spent in bed is a key ingredient of the intervention's success.

For subjective insomnia severity (i.e., score on the self-report ISI), SRBQ emerged as the only significant mediator of treatment response. Participants who differed by one unit on

pretreatment ISI were estimated to differ by -.08 units in posttreatment ISI through changes in SRBQ. Specifically, those with higher ISI at pretreatment had lower scores on the ISI at posttreatment (indicated by the negative indirect effect) as a result of the tendency for those with higher pretreatment ISI scores to have a smaller change in SRBQ (indicated by the positive path between pretreatment ISI and change in SRBQ), which in turn was associated with decreased posttreatment ISI (demonstrated by the negative path between SRBQ change and posttreatment ISI).

The inverse relationship between pretreatment ISI and SRBQ changes suggests that those who rated their insomnia to be more severe prior to treatment may have been less adherent to recommendations about reducing their safety behaviours related to sleep; interestingly, the posttreatment reduction in insomnia severity (i.e., decreased ISI) also suggests that these individuals nonetheless self-rated their insomnia to have improved significantly over the course of treatment. This particular mediational finding suggests a number of possibilities. It appears that four sessions of CBT-I functions to improve participants' own view of the severity of their insomnia symptoms, *regardless* of whether they eliminate or significantly reduce their sleep-related safety behaviours.

It also may suggest that those who believe their insomnia symptoms to be more severe are perhaps more reluctant to relinquish those behaviours that had previously increased their sense of control or aided in short-term reduction of sleep-related anxiety (i.e., safety behaviours). Indeed, it is imaginable that a short-term treatment for insomnia might in fact increase one's reliance on safety behaviours in the short term, if the focus on insomnia also caused anxiety about sleep to increase. We see a similar process unfold for some individuals in anxiety disorders treatments; that is, it is often the case that in the early sessions of an anxiety protocol

(e.g., for generalized anxiety disorder, social anxiety disorder), anxiety worsens, before eventually improving. This makes sense, given that during such interventions individuals are focusing on symptoms, thoughts, and behaviours that they had perhaps previously managed via avoidance.

Similarly, in CBT-I it may be that the increased focus on sleep inherent to the intervention could cause sleep-related anxiety to worsen, thereby augmenting these clients' reliance on tools at their disposal that had previously proven to be efficacious, at least in the short-term. The fact that the duration of the CBT-I treatment protocol administered in this study is only 4 sessions, clients would have significantly not have the time to habituate, and/or to learn to manage anxiety over the course of treatment, as would occur in, for example, a 12-session protocol. Should this be the case, such concerns could likely be addressed in a shorter time frame than 12 sessions, however it may be indicated to target safety behaviours and their function (i.e., coping with anxiety about sleep) earlier in treatment, in order to improve outcomes for this subset of clients. For example, graduated exposure therapy could be more explicitly discussed in therapy sessions, and in particular safety behaviours could be addressed in accordance with exposure guidelines (i.e., specific, feasible, predictable, timely, etc.). This could be achieved through increasing the emphasis placed on safety behaviours during treatment sessions, or through the addition of a safety-behaviour focused session.

With regard to the finding that participants also reported significantly improved severity of symptoms following treatment, it may be that for this sample, the cognitively focused components of CBT-I had a relatively greater impact on their perception of symptom severity, despite the fact that they did not significantly alter many or some of their safety behaviours. It is also true that the SRBQ questionnaire, which assessed for sleep-related safety behaviours in this

study, contains a number of questions about behaviours that are either cognitively-focused (e.g., "I try to stop all thinking when trying to get to sleep," "I try to stop all thinking when trying to get to sleep") or involve daytime activities (e.g., "I miss or cancel appointments (daytime or evening)." While CBT-I is indeed a treatment that targets daytime functioning, given the updated criterion that captures deteriorated daytime functioning as a symptom of insomnia disorder (American Psychiatric Association, 2013), it is also true that the focal point of the intervention is sleep schedule compression plus stimulus control. That is, although daytime activation is important for increasing sleep drive, thereby augmenting overall sleep quality during the main sleep period at night, treatment focuses mainly on development of a sleep schedule "prescription" and any difficulties adhering to this schedule (in particular, to rise time consistency), as well as on disassociating the bedroom environment from wakeful activities (e.g., work, television, rumination/worry). As such, it is feasible that the individuals who showed subjective improvement following CBT-I despite somewhat increased engagement with safety behaviours were simultaneously adhering to the "broad strokes" of treatment, for example maintaining a consistent sleep schedule or adhering to stimulus control rules, despite continued engagement with certain sleep-focused behaviours.

Alternatively, it is possible that these individuals significantly decrease more "obvious" sleep focused safety behaviours (such as "I figure out how I will catch up on my sleep later on"), but maintain more subtle safety behaviours (such as "I stay in the background in social situations") that are less frequently targeted during treatment, thereby maintaining a moderate-to-high score on the SRBQ. This possibility makes sense given that CBT-I involves focused attention on insomnia symptoms, and as such the level of cognitive attention to sleep-related issues may increase over the course of four treatment sessions. Theoretically we would expect

this to decrease in the longer-term, and it would be interesting to examine outcomes on the SRBQ at posttreatment compared to 6- or 12-months later.

While the use of safety behaviours are generally considered to interfere with clinical improvement in the anxiety disorders literature (e.g., Forsyth et al., 2006), some recent research has suggested that prudent employment of safety behaviours may actually be helpful for symptom improvement and lead to better treatment outcomes (e.g., Blakey & Abramowitz, 2016; Deacon, Sy, Lickel, & Nelson, 2010; Levy & Radomsky, 2016; Milosevic & Radomsky, 2012). These recent studies suggest that when associated with improved outcomes, safety behaviours may function to augment clients' feeling of control in feared situations and make exposure exercises more palatable or acceptable to the client. Taking these findings into account, it may be that safety behaviours in the current study mediated the relationship between pretreatment and posttreatment insomnia severity by helping participants to make the recommended changes to their sleep schedule while maintaining a sense of control and acceptability of the changes.

In addition to the aforementioned possibilities about why the SRBQ mediated the relationship between pre- and posttreatment insomnia disorder in an unexpected way, it is notable that certain items on the SRBQ may be interpreted in different ways. For example, "I spend time considering ways to improve my sleep" theoretically should be endorsed by all individuals who actively participate in CBT-I, given that they are spending at minimum 1 hour per week discussing how to improve their sleep with a therapist, and likely far more than that when considering the amount of attention required by some of the CBT-I recommendations, or even merely the fact that in the present trial, participants were offered four chapters (Carney & Manber, 2013).

It is also possible that those who had smaller changes on the SRBQ over the course of treatment actually entered treatment with low SRBQ scores, and as such had little room to change. It may be that these individuals adhered to recommended changes but were already engaging in few safety behaviours, and therefore change to the SRBQ was small. This possibility would also make sense given that those with more severe perceptions of their insomnia at pretreatment showed smaller changes in their sleep-related safety behaviours but nonetheless showed significantly reduced self-rated insomnia severity following treatment, because this suggests that those who already refrain from engaging in sleep-related safety behaviours prior to treatment perceive that they do better in treatment. That is, if one enters treatment already avoiding safety behaviours, their insomnia symptoms may remit more quickly compared to those who rely on safety behaviours.

Contrary to hypotheses, change in RT variability and change in sleep effort did not significantly mediate outcomes in the analyses. For the mediation analysis that examining treatment response as measured by TWT at pretreatment and posttreatment, it may be that we could have expected the most significant mediators to reflect behavioural changes, since a decrease in total wake time at posttreatment suggests that changes were made to the sleep schedule that resulted in less time spent away in bed. It makes sense then that change in TIB was a significant mediator in this analysis, and it may be that change in RT variability did not emerge as significant due to TIB change being a more important factor overall for TWT changes at posttreatment. That is, while decreased RT variability may aid an individual in decreasing their TWT, it makes sense that compressing their TIB window is more important overall on this index of insomnia improvement. With regard to sleep effort (measured by score on the GSES), it is likely that this variable would have had more impact on subjectively measured insomnia severity

(i.e., on the analysis that assessed treatment response by measuring ISI at pretreatment and posttreatment).

That change in GSES did not emerge as a significant mediator of subjective treatment responses suggests that sleep effort may have a more subtle impact on symptom severity, or may function differently within the intervention. For example, it may be that reduced sleep effort improves a client's ability to adhere to the behavioural recommendations, but that little change in sleep effort is required for larger changes in the behavioural variables; were this the case, it makes sense that behavioural variables emerged as mediators, but not cognitive variables such as change in GSES. With both analyses, it is also likely the case that many or most of eight cognitive, behavioural, and state variables entered into the analysis as hypothesized mediators had a small, nonsignificant mediating effect on treatment response. That is, it may be that many factors influence treatment response, but there are major treatment components that are the most important mediators of response, such as changing one's time in bed.

In the secondary analyses, to test the hypothesis that posttreatment insomnia severity is a function of a number of CBT-I treatment components, the pretreatment variables of interest in this study were entered into hierarchical linear regression analyses. Contrary to hypotheses, no variables were shown to moderate the relationship between pre- and posttreatment insomnia severity when measured via retrospective self-report (i.e., ISI score). When insomnia severity was measured through prospective monitoring (i.e., CSD TWT), RT variability had a significant moderating effect on the relationship between pre- and posttreatment insomnia severity. However, counter to hypotheses, sleep effort (GSES) and tendency to ruminate (DISRS) were not significant moderators. It is interesting that the cognitive variables did not moderate treatment response in the current study, particularly when insomnia severity was subjectively

measured. It may be that CBT-I results in improvement for most *regardless* of their pretreatment state and cognitive profiles, and that the behavioural components of CBT-I are most important for treatment response. It is also possible, as aforementioned, that cognitive factors may shift less, but have a greater impact on improving clients' ability or desire to adhere to the *behavioural* recommendations, and it is in turn these behavioural components of treatment that yield the greatest benefit for improving insomnia symptoms over the course of treatment.

Results from the secondary analyses supported the hypothesis that the interaction between pretreatment rise time variability and total wake time was a significant moderator of treatment response when insomnia severity was measured prospectively on the sleep diary (i.e., predicting posttreatment TWT from pretreatment TWT). In the second step of the multiple linear regression analysis, 28% of the variance in total wake time at posttreatment was accounted for by all of the moderating variables. Examination of the standardized beta coefficients showed that the interaction term between pretreatment rise time variability and pretreatment total wake time accounted for the majority of this variance (22.5%) in posttreatment total wake time. Examination of the interaction plot showed an enhancing effect, wherein as pretreatment TWT and pretreatment RT variability increased, posttreatment TWT lengthened. At average levels of pretreatment TWT, posttreatment TWT was similar for those with low, average, and high RT variability. However, those with high pretreatment TWT and average pretreatment RT variability tended to show greater improvement at posttreatment (i.e., shorter TWT values). Those with high RT variability and high TWT at pretreatment had the worst posttreatment outcomes in terms of TWT. Interestingly, those with high TWT and average RT variability at pretreatment showed the greatest improvement in posttreatment TWT.

It makes sense that individuals beginning treatment with both long durations of wakefulness in bed and greater rise time variability improve less over the course of treatment. Larger variability in rise time may make treatment recommendations more difficult to adhere to if the client is accustomed to following a different schedule on weekends, for example, as compared with weekdays. Indeed, large variability in rise time may point to circadian rhythm concerns; although sleep schedule recommendations in CBT-I may help to improve a client's circadian rhythm maladjustment within the treatment (e.g., recommendations around light exposure in the morning, rise time consistency), CBT-I is not a treatment designed to target circadian rhythm disorders and we would not expect such concerns to improve significantly following the intervention. It is possible, then, that these individuals with high RT and high TWT at pretreatment are those who have subthreshold circadian rhythm concerns (since those who met full criteria were screened out at the pretreatment screening interview).

Alternatively, it may be that those with greater variability in RT at pretreatment also have less motivation to change their rise time schedule. Since change was measured between pre- and posttreatment in the current study, it is possible that improvement was made over the course of treatment, but then clients decided and/or discussed with their therapist during relapse prevention planning that they would prefer a somewhat more dysregulated rise time, even with the knowledge that this would result in elevated TWTs. Insomnia is a subjective disorder; as such, participants in the current study may have improved significantly on measures of insomnia severity but nevertheless maintained certain habits that would not be recommended as components of CBT-I. Indeed, many individuals with good or normal sleep engage in nonCBT-I practices (e.g., napping, sleeping in on the weekends), and do not meet criteria for a sleep disorder. Considering the impairment and distress criterion of a diagnosis, it is also important to

consider that those who show smaller improvements on TWT at posttreatment may nevertheless have seen a significant improvement in the degree to which they are distressed about their sleep, and the amount of interference in their daytime functioning that results.

It is important to note that for the sample as a whole, TWT significantly decreased between pretreatment and posttreatment. It is clear that over the course of CBT-I, participants had significant improvement in when insomnia severity was assessed using a prospective measure (i.e., CSD TWT), suggesting improvement in treatment response. Results from the moderation analysis showing that those with greater RT variability and greater TWT at pretreatment have higher TWT at posttreatment may suggest that for these individuals, greater time in CBT-I sessions might be spent developing client-specific strategies for targeting rise time variability. For example, for some individuals for whom rise time consistency is particularly difficult, it might be helpful to relax the recommendation about rise time to allow for a 1-hour window of variability across the week. Those for whom consistent rise times are more difficult or less palatable may find the recommendations easier and/or more reasonable for their lifestyle if there is allowance for some degree of variability.

It may be that if a highly consistent rise time seems unreasonable or unattainable for some clients, that they ignore the recommendation entirely, instead of making adjustments that improve the variability. It might also be considered that the client and therapist work collaboratively to develop a rise time range that is acceptable to the client, but nevertheless moves them towards less variability in their schedule (e.g., 2 hours of variability, versus 5). However, it is also true that as a whole, TWT decreased significantly between pre- and posttreatment; it therefore may be the case that participants were satisfied with their symptom improvement and saw no need to alter their variable rise times at posttreatment. Specifically, in

considering their lifestyle and most desired sleep schedule going forward after treatment, clients who tended to have higher RT variability and also higher TWT at pretreatment saw significant improvements to their sleep concerns and as such, opted not to adhere as closely to recommendations by the end of treatment.

In terms of the posthoc analyses, although medication use was not expected to impact treatment outcomes in the present study, we wanted to ensure that there were no between groups differences for those using versus not using medication during the course of their treatment, given that recently published study found that individuals with insomnia treated with only CBT-I, and no psychopharmacological medications, tended to show greater improvement in the long term (Castronovo et al., 2018). It was unsurprising that medication use did not impact treatment outcomes, and these results replicate past studies that have shown that non-dependent medication use does not impact CBT-I outcomes.

We also wanted to examine whether there were any differences in outcomes depending on other mental health disorder comorbidities. There were no differences in posttreatment insomnia severity, as assessed by self-reported ISI score or by CSD TWT, for individuals with symptoms that met DSM-IV-TR (research version; American Psychiatric Association, 2013) criteria for generalized anxiety disorder (GAD), social anxiety disorder, panic disorder, agoraphobia, or post-traumatic stress disorder (PTSD) compared to those who did not. However, individuals who met criteria for current major depressive disorder (MDD) at pretreatment reported significantly higher distress about insomnia symptoms at posttreatment compared to those without MDD, as assessed by their score on the ISI. Interestingly, those with MDD were not significantly different on posttreatment insomnia severity when severity was assessed via prospectively monitored CSD TWT. These findings suggest that those who begin CBT-I with a

concurrent depressive disorder in addition to insomnia may perceive their symptoms to be more problematic or distressing after treatment, while those without MDD report greater improvement. That these groups did not differ on posttreatment TWT suggests that the *perception* of their insomnia severity is poorer at posttreatment, while their actual wake time while in bed is not significantly different from those who reported improved insomnia severity at post-treatment.

These posthoc findings suggest that it may be helpful for clinicians to target distress about insomnia specifically, for clients who present with comorbid depressive symptoms. It is important to note, however, that although those with MDD reported higher posttreatment distress (i.e., greater ISI score), the mean score was nevertheless below the clinical cutoff for insomnia disorder on this measure. That is, those who entered CBT-I with symptoms consistent with MDD nevertheless reported distress about insomnia at posttreatment that was below the threshold for clinical insomnia diagnosis. This suggests that those with MDD perceived improvement over the course of treatment, however somewhat less so compared to those without MDD.

It is also notable that participants in the current trial were screened for *possible* diagnoses, and these diagnoses were not conveyed to participants. The purpose of assessing participants for potential comorbid psychiatric disorders was to evaluate whether they met inclusion/exclusion criteria for the trial, but we did not administer supporting questionnaires relevant to these various other psychiatric concerns (e.g., no measure of post-traumatic stress symptoms was provided). In other words, while participants were assessed for the aforementioned psychiatric disorders, these posthoc analyses were conducted with the assumption that participants had symptoms consistent with these disorders, and not necessarily assuming that they met criteria for a specific diagnosis. Thus results from these analyses should be interpreted with caution, and future

researchers may want to consider the possibility of completing full diagnostic assessments in future studies of CBT-I, to better establish diagnostic clarity. It would be interesting to see whether the results from the current study that individuals with symptoms consistent with MDD reported greater distress at posttreatment compared to those without MDD would be replicated in a sample of individuals who met full criteria for diagnosis of MDD.

Limitations

There are several limitations of the current study. First, the design of the parent trial focused on longitudinal outcomes after CBT-I, and therefore included acquisition of data from participants during follow up phone calls on a monthly basis and a final 1-year follow up interview. As such, the variables of interest in the present study were not assessed at each of the four CBT-I sessions, but rather were only assessed at pretreatment and posttreatment. Even if these variables could be assessed more frequently over the course of treatment and compared with symptom change at each individual time point (such as might be possible via path analysis or structural equation modeling), it would likely remain difficult to pinpoint the process of change and account for all possible variables that could account for symptom change at each time point (Kazdin, 2007). That is, while variables accounting for therapeutic change in such an analysis may be significant mediators of treatment response, it may also be true that the construct measured by the variable may not fully explain the way in which the change occurred (i.e., there might be a more global construct that includes many different variables/treatment components), or there may be additional variables unexamined in such analyses that might better account for therapeutic change. Therefore, although results from the current study do not firmly establish mechanisms of therapeutic change in CBT-I, the mediators and moderators found to influence

treatment response nonetheless provide increased clarity as to how symptom change may come about between pre- and post-CBT-I treatment.

Another limitation of the current study is that there may be other variables that function as mechanisms of symptom improvement in CBT-I that were not assessed in the current study. For example, maladaptive beliefs and attitudes about sleep likely perpetuate symptoms for those with chronic insomnia (e.g., Edinger et al., 2009; Jansson & Linton, 2005), and while this factor was assessed at pretreatment in the current study using the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin, 1993), it was not administered at posttreatment and as such could not be assessed as change variable that mediated treatment outcomes in the current study. Although the present study investigated the majority of theoretical factors presumed to relate to change in insomnia symptoms over the course of CBT-I, other factors such as maladaptive beliefs and attitudes about sleep have yet to be explored in conjunction with the other presumed mechanisms.

The variables examined as potential mediators in the present research nevertheless included those the main theoretical factors presumed to perpetuate insomnia, as outlined by Spielman and colleagues (1987) and upon which CBT-I was developed as an intervention (Edinger & Means, 2005; Edinger & Carney, 2008, 2015). While it may be true that there are additional variables that account for changes in insomnia symptoms over the course of CBT-I, given the expanse of existing research on the theoretical factors investigated in the current study, it is likely these factors account for the greatest changes in insomnia severity, or represent the overarching constructs that account for change in CBT-I.

Indeed, one of the main strengths of the current study is that the majority of the presumed mechanisms of treatment response in CBT-I known to researchers to date were investigated;

almost every mediator described in a recent review of CBT-I mediators (Schwartz & Carney, 2012) was explored in the current study. Further, these factors presumed to be causally related to treatment response in CBT-I were investigated simultaneously in the present study; that is, in the primary quantitative analyses, all variables of interest were entered into multiple mediation analyses, which provided important insight about the relative importance of each individual factor in relation to the other presumed mechanisms. This was replicated in the secondary analyses, with all moderator terms entered in the second step of the multiple hierarchical linear regression analyses. It is rare for mediation and moderation analyses to be adequately powered such that eight mediator/moderator terms can be examined in a single analysis, and the ability to examine this many variables simultaneously provides a great deal of value in the current study. That is, while mediation and moderation analyses used with clinical trial data may yield important insights about treatment response when single mediator/moderators are examined, the analyses performed in the current study are an important step forward in the field, as the comparative weight of each individual variable operating simultaneously could be explored.

Furthermore, the fact that data from the current study included data from a clinical trial that spanned several years and included over 150 participants in the insomnia disorder sample allowed for such in-depth analyses, and also suggests that results from the current study are likely more generalizable to the broader population of individuals with insomnia who undergo a course of CBT-I.

Perhaps most importantly, the present research focused on client perspectives on the most essential components of the treatment, that is, what aspects of CBT-I they felt were most beneficial to them. While many studies have examined the influence of theoretical factors on treatment outcome through statistical analyses, few have used qualitative methods to better

understand the way in which clients make sense of CBT-I and what factors they believe had the most impact on their symptom improvement. The focus on client perspectives in the current study is an important step in the progression of the insomnia treatment literature.

Implications and Future Directions

Findings from the present study provide critical information about the treatment components that are most essential to insomnia symptom improvement, from both the perspective of the participants themselves as well as through quantitative analysis of the theoretical factors presumed to function as mechanisms of change in CBT-I. While we as clinicians and researchers have a great deal of data to support our notions of the most essential aspects of CBT-I, the current findings suggest that cultivation of sleep-related self-efficacy is also critically important to clients. As clinicians, there are a number of components of the CBT-I protocol that are considered imperative to convey with regard to explaining the rationale, troubleshooting any difficulties, and working with clients to improve adherence. Yet, participant perspectives on the treatment suggest that fostering a sense of self-efficacy is essential for treatment outcomes. These results suggest self-efficacy might be considered as a more explicit goal of CBT-I; that is, while the development of self-efficacy is an inherent goal of the treatment, any apparent difficulties with the development of self-efficacy in treatment ought to be targeted by therapists.

Future research should begin to disentangle the elements of self-efficacy versus the key presupposed theoretical factors upon which CBT-I was based, that is, the behavioural and cognitive CBT-I strategies to better understand the respective contribution of these various factors on treatment response. While it may be the case that self-efficacy is developed as a result of the changes clients make over the course of treatment, it may nevertheless provide important

insights to examine this factor as a potential mediator of treatment response. Furthermore, future studies should investigate the components of CBT-I examined in the current study, as well as those which emerged from the client responses on the LTS, via longitudinal mediational models, as others have suggested (Garland, Zhou, Gonzalez, & Rodriguez, 2016). Such longitudinal analysis will be critical for better understanding the temporal relation between potential mechanisms and symptom improvement in the longer term.

In addition, results from the present study indicate that CBT-I clients agree with the extant sleep research with regard to the essential "ingredients" of the treatment; that is, that what accounts for symptom change includes those theoretical factors upon which CBT-I was based. In other words, participants' report of the treatment components they found to be most beneficial mirrored the theoretical factors investigated as mediators of treatment outcome in the current study. This confirms client uptake of the information and recommendations provided over the course of CBT-I, and also indicates that clients share the perspective of researchers and clinicians about the importance of these factors in contributing to their symptom improvement.

That client perspectives provided important additional and confirming information about presumed mechanisms of treatment response in CBT-I suggests that future researchers should incorporate qualitative analyses to better understand client perspectives. Future research could investigate the way in which clients develop and understand those treatment components that they found to be helpful during the CBT-I intervention. Clients' understanding of the components they found to be helpful in treatment could be examined in the therapeutic setting by recording each therapy session (with participant consent), and during data analysis, coding each recorded interview for the presence of, for example, language related confidence, optimism, efficacy, and other broad themes suggestive of self-efficacy enhancement. Researchers could

then rank the degree to which each emergent treatment component was a focus in CBT-I sessions (for example, by including "counts" of occurrence of language pertaining to each treatment component in therapy sessions), and use qualitative as well as quantitative multiple mediator models of to better understand the relationship of these components to treatment outcomes.

To better understand the source of nonempirically supported information identified to be helpful by participants in the current study, future research would likely need to administer lengthy exit interviews at the end of treatment, subsequent to analysis of which treatment components they found to be helpful. Future qualitative research about client perspectives on treatment mechanisms may want to consider interviewing clients after review of the LTS, and analyzing these interviews, in order to better understand the source of information provided by clients in their LTS responses, as well as how, when, and why they found these components to be helpful. Acquiring examples from clients for each of the components endorsed on the LTS would provide helpful information about the way in which these factors may have played a role in clients' symptom improvement.

Future research investigating the process of change in CBT-I should also include qualitative interviews at *many time points* throughout the intervention; for example, at pretreatment, prior to or immediately following each treatment session, and at posttreatment. For further longitudinal information, similar short-form interviews could be administered during monthly follow-ups, to acquire an understanding of which factors were most helpful for clients in the longer-term (as opposed to only within the 8-week treatment window). For feasibility, a study such as this that incorporated many qualitative interviews could create a semistructured, short form, clinician administered interview that queries participants in an open ended manner; results from the present research would be critically valuable in designing initial drafts of such

an interview. Gathering data at multiple time points over the course of treatment, at multiple time points, in a manner temporal with the time points during which clients' symptoms are presumably shifting, would provide crucial information about the process via which change occurs in CBT-I, and would advance the field by incorporating elements reported by participants in the current study as potential mechanisms.

Results from the statistical analyses in the present study confirm the well-established data supporting CBT-I as an efficacious treatment, and emphasize the two factors that are generally considered to be the cornerstone elements of the treatment: sleep compression and stimulus control. Although there are many avenues for future exploration in relation to the mechanisms of treatment response in CBT-I and how the treatment might be refined in general, as well as tailored in consideration of varying client presentations, the present results provide support for the efficacy of these principle underlying tenets of CBT-I. In other words, adherence to these most essential recommendations is likely to be sufficient for clients to notice a significant improvement in their insomnia symptoms following the intervention.

From a quantitative perspective, future studies should focus on whether sleep-related safety behaviours decrease significantly in the longer term, and compare this to insomnia severity longitudinally. Given that the current study found that small changes on the SRBQ mediate treatment response in terms of self-reported symptom severity, it may be that larger changes in safety behaviours tend to occur over time, as clients become accustomed to their new sleep routines and as confidence, trust in their bodies to perform sleep, and a sense of self-efficacy about their ability to sleep well consolidates. That is, for the participants in the current study who made small changes to their sleep-related safety behaviours, they may have maintained some degree of safety behaviour use which allowed them to engage more fully in the

CBT-I treatment recommendations, which in turn led to significant subjective symptom improvement at posttreatment. It may be that for these individuals, changes to safety behaviours may continue to grow (i.e., reduced reliance on the behaviours) in the weeks and months subsequent to fourth session of CBT-I, such that clients either experiment with dropping safety behaviours or a decrease in safety behaviours continues over time until engagement with safety behaviours is negligible. If this is the trajectory of change for some individuals who maintain safety behaviours over the course of treatment, it may be that these individuals continue to make changes over time; future longitudinal mediational analyses could investigate the extent to which reduced safety behaviours is responsible for symptom improvement in the longer term.

Regarding safety behaviour use, it would also be important to extend recent psychological research that suggests that maintenance of some degree of safety behaviours may actually be helpful for treatment outcomes, by comparing safety behaviour use to outcomes in CBT-I. To better understand the way in which safety behaviours might operate to improve adherence and/or treatment response in CBT-I, researchers could develop an experiment wherein participants are randomly assigned to conditions with different levels of safety behaviour usage (e.g., none, few, many), while controlling for safety behaviours already used by participants. Alternatively, a nonmanipulated study could involve continual assessment of safety behaviour usage over the course of treatment (i.e., at multiple time points), and compare usage to associated symptom improvement throughout treatment as well as at posttreatment.

Conclusions

Findings from this study suggest that client perspectives on the components of CBT-I that were most helpful for improving their insomnia symptoms generally correspond with the theoretical factors presumed to function as mechanisms of the treatment. However, these results

also suggest that other factors considered less critical to the treatment, such as psychoeducation and normalization, self-monitoring, and sleep hygiene information, are also provide clients with important information that they associate with improved symptoms. Results also suggest that aspects of CBT-I that are more implicit, such as adherence to sleep hygiene recommendations and development of clients' sense of self-efficacy, were beneficial to clients. These latter results indicate that emphasis on augmenting clients' sense that they have the ability and tools to achieve good sleep, and on increasing their sense of acceptance of normal sleep may be an important explicit treatment target. That is, while these aspects of CBT-I are inherent goals of treatment, it may be valuable for clinicians to focus on these components more explicitly in therapy, for example troubleshooting any difficulties with self-efficacy that appear to arise over the course of treatment.

Results also emphasize the importance of sleep compression to symptom improvement. Given that time in bed emerged as a mediator of prospectively measured treatment response (i.e., CSD TWT), continued focus on sleep compression and related elements of treatment (e.g., stimulus control, sleep schedule consistency) is supported by results from the present study. For those for whom safety behaviours represent a problem coming in to treatment, results from the present study suggest that a) these clients may experience significant symptom improvement by posttreatment *even if* they make few changes to their safety behaviours, and b) it is possible that a targeting these clients' perceptions of their insomnia severity (e.g., cognitive focus to treatment, with emphasis on the function of safety behaviours) or focusing on behavioural experiments that involve dropping safety behaviours and assessing the outcome could have a greater impact on improved treatment response. It also may be that those who engage in few safety behaviours at the beginning of treatment and also rate their insomnia to be more severe,

tend to experience subjectively greater treatment response compared to those with either less severe subjective insomnia or more safety behaviours at pretreatment. Finally, results from this study suggest that those clients with both long total wake time and high variability in rise time at pretreatment tend to improve less by posttreatment. This suggests that for these clients, greater emphasis on reducing variability in rise time may improve outcomes. Nevertheless, results from this study indicate that prospectively monitored wakefulness in bed improves significantly over the course of treatment, suggesting that even for those with greater difficulties with rise time consistency and wakefulness in bed, they experience significant symptom improvement following a course of CBT-I.

In summary, although not all presumed mechanisms of treatment response emerged as significant mediators in the present study, it is clear that clients understand these treatment components to be important. Further, results confirm that most essential ingredients of CBT-I are related to behavioural changes made during the course of treatment; that is, reduced time spent in bed. It is also likely that changes to rise time variability and reductions in sleep-related safety behaviours have a significant impact on treatment response, and future research should focus on further disentangling these constructs in relation to symptom improvement across therapy sessions. It is clear that CBT-I is a highly efficacious treatment for most individuals with insomnia. To refine and tailor this treatment to the best of our ability as researchers and clinicians, future studies need to continue to focus on better understanding the processes by which change is brought about from a temporal perspective by assessing potential mediators, including those posited by clients themselves, continually and longitudinally.

Appendices

Appendix A

ID _____ Treatment # when provided __
Treatment # when received __

Letter to Self

Many patients find it helpful to remind themselves of the treatment strategies that they found to be most helpful for improving sleep during cognitive-behavioural therapy for insomnia. You may return to this letter in future, should insomnia symptoms return. This letter may be as comprehensive or brief as you like; the purpose is to make notes in a way that will remind you of the helpful strategies you can re-implement to improve your sleep.

Dear _____,

Remember….. _____

_____

_____

_____

_____

_____

_____

_____

_____

Appendix B

A Priori Template Themes for Coding the LTS[1]

1) Stimulus control elements[2]
- Not attempting sleep until body is sleepy
- Arising from bed, leaving the bedroom if wakefulness is prolonged
- Using the bed/bedroom only for sleep and sex to avoid conditioned association with wakeful activities (e.g., no television, reading, work)

2) Sleep compression strategies[3]
- Restricted TIB window (TST+30 minutes)
- Sleep schedule: Scheduling earliest bedtime/latest rise time
- Avoidance of daytime napping

3) Regulation of factors impacting circadian rhythm[4]
- Increasing consistency, routine of sleep schedule
- Reducing variability of RT, BT
- Nonsleep-specific circadian elements (e.g., regular meals, exercise)

4) Daytime behavioural activation[5]
- Engaging in scheduled daytime activities
- Increasing daytime activity in order to augment sleep drive
- Avoiding avoidance
- Do not isolate/withdraw

5) Cognitive restructuring strategies[6]
- Challenging maladaptive thoughts about sleep
- Challenging maladaptive thoughts about daytime functioning/ consequences of a bad night
- Using tools for managing rumination and worry (e.g., scheduling constructive worry time, thought records)
- Counterarousal techniques (e.g., relaxation, mindfulness)

6) Focus of attention, effort, and behaviours around themes of sleep[7]
- Thinking like a "good sleeper"
- Reducing sleep-related safety behaviours
- Reducing effort to sleep

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[1] All template themes were derived from the research evidence, summarized in the CBT-I protocol administered in the current clinical trial (Edinger & Carney, 2008; 2015; Edinger & Means, 2005), and described extensively in the introduction to this dissertation (beginning on p.15).
[2] Bootzin, Epstein, & Wood, 1991
[3] Spielman et al., 1987; Wohlgemuth & Edinger, 2000
[4] Bootzin, 1972
[5] Ibid.
[6] Harvey, 2002, 2005; Harvey et al., 2007
[7] Harvey, 2002; Espie et al., 2006

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