AN EXAMINATION OF A BRIEF INTERVENTION TARGETING CAUSAL

ATTRIBUTIONS FOR DAYTIME FATIGUE

by

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Abstract

An Examination of a Brief Intervention Targeting Causal Attributions for Daytime Fatigue

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Research has shown that poor sleepers focus primarily on their sleep as a cause of daytime fatigue rather than the multitude of other possible causes of fatigue. This can create sleep-related anxiety and further perpetuate the insomnia. In order to lessen the increased focus on sleep, the present study investigated whether people could learn to consider other attributions for fatigue via an information-based intervention, and whether this cognitive change would have implications for relevant mood states. Participants were randomized to receive either "causes of fatigue" information (FI), or generic sleep-information (control), and were tested pre- and post-intervention. FI participants were significantly more likely to consider non-sleep-related attributions for fatigue at post-intervention, relative to control participants. There were no significant group differences on relevant mood states. These results demonstrate that attributions for fatigue are amenable to change via an information-based intervention; thus, this research explores one avenue toward refining insomnia treatments.

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AN EXAMINATION OF A BRIEF INTERVENTION TARGETING CAUSAL

ATTRIBUTIONS FOR DAYTIME FATIGUE

Insomnia is a devastating disorder which is diagnosed based on a subjective complaint of difficulty falling asleep, maintaining sleep, waking up too early, or feeling poorly rested despite adequate sleep. Impaired daytime performance, such as fatigue, difficulty concentrating and poor social or vocational functioning, are also frequent complaints among individuals with insomnia. (American Psychiatric Association, 2000). Epidemiological studies suggest that insomnia is a highly prevalent disorder affecting approximately 10 – 15% of the adult population (Ford & Kamerow, 1989; Ohayon, 2002). This condition is also associated with a host of significant personal and societal costs for Canadians (Daley, Morin, LeBlanc, Gregoire & Savard, 2009). Specifically, insomnia has been linked to reduced quality of life, increased healthcare utilization, increased use of prescription and over the-counter medication, increased absenteeism, decreased work productivity and increased traffic and work-related accidents (Edinger & Wohlgemuth, 1999; Roth & Ancoli-Israel, 1999). With respect to economic burden, annual costs for Canadians associated with insomnia-related absenteeism and decreased work productivity are estimated to be $970.6 million and $5.0 billion respectively (Daley et al., 2009). Further, insomnia often presents as comorbid with other psychiatric disorders, especially depression and anxiety (Ohayon, Caulet, & Lemoine, 1998), and these comorbid groups are particularly challenging to treat (Smith, Huang, & Manber, 2005).

The development of chronic insomnia is primarily dependent on three factors: predisposing factors, precipitating events, and perpetuating factors (Spielman & Glovinsky, 1991). Predisposing factors are specific individual characteristics such as sensitivity to light (Chesson et al., 1999) or propensity to worry (Lichstein & Rosenthal, 1980) that can increase

vulnerability for developing insomnia. When an individual with such predisposing characteristics comes into contact with precipitating factors (e.g., stressful life events), they may then develop an acute sleep disturbance (Healy, Kales, Monroe, Bixler, Chamberlin, & Soldatos, 1981; Morgan & Clarke, 1997). Given the discomfort associated with this sleep difficulty, people may begin to engage in maladaptive thought processes and behaviours in order to help cope with their temporary sleep loss, such as increased sleep-related anxiety and poor bedtime habits. It is these cognitive and behavioural perpetuating factors that will ultimately turn their acute sleep disturbance into chronic insomnia. Furthermore, these perpetuating factors have adverse impacts on the sleep regulatory processes (Bootzin, 1972; Harvey, 2002), and it is the interaction between such perpetuating factors and sleep regulatory processes which works to maintain the insomnia.

There are three primary processes which regulate sleep: the homeostatic, circadian, and arousal systems (Saper, Cano, & Scammell, 2005; Webb, 1988). The homeostat controls the propensity for sleep (i.e., sleep drive) by building "pressure" to sleep with increasing hours of wakefulness (i.e., sleep debt); this pressure is then released during sleep. Behaviours such as napping, going to bed earlier than usual, sleeping-in, or being sedentary, all result in lowered homeostatic drive for sleep, and thus more disturbed sleep. The circadian system affects the timing of our sleep-wake schedule in the 24-hour day. Poor bedtime habits, such as varying bed times and rise times, disrupt the circadian system in the same way that jet lag taxes our system. Finally, while the circadian and homeostatic systems work to regulate sleep, both systems can be trumped by the arousal system. Even during periods of high homeostatic drive (e.g., periods of sleep deprivation) or optimal timing for sleep (i.e., during the night), if the body perceives a threat, the arousal system allows people to stay awake. In this sense, repeated pairings of sleep

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disturbances with the bedroom unintentionally conditions the bed to be an alerting/wakeful stimulus, and thus, the arousal system is an important factor in insomnia. In sum, while predisposing and precipitating factors work to create the sleep disturbance, it is the perpetuating factors, and their interaction with the sleep regulatory processes, which contribute to the development and the maintenance of clinical insomnia. As such, in order to better understand, prevent and treat insomnia, identification of these perpetuating factors needs further exploration in both the research and treatment literatures.

There is extensive evidence that both behavioural and cognitive processes are key factors in the maintenance and perpetuation of insomnia. With respect to behavioural factors, many people with insomnia problems have poor bedtime habits and do not maintain consistent sleep-wake schedules (Bootzin, 1972). For example, insomnia sufferers often attempt to catch-up on sleep by napping during the day or sleeping-in (Morin, 1993). As explained above, while these may seem to be effective strategies, they interfere with the body's homeostatic sleep drive, which itself is designed to compensate for accumulated sleep loss by increasing our ability to sleep (Feinberg, March, Floyd, Jimison, Bossom-Demitrack, & Katz, 1985; Webb, 1988). Other behavioural routines, such as remaining inactive during the daytime (Morgan, 2003; Sherrill, Kotchou, & Quan, 1998) or engaging in mentally demanding activities too close to bedtime (Broman & Hetta, 1994), may also prolong sleep onset. Over time, these sleep disruptive habits come to perpetuate one another, and ultimately can lead to the inception of chronic insomnia.

An important question is how do people come to develop such disruptive sleep habits? The answer may lie in the thinking processes and general cognitive styles of those with insomnia. Such cognitive processes include maladaptive beliefs and misperceptions about sleep (Morin, Stone, Trinkle, Mercer & Remsberg, 1993), as well as heightened anxiety and worry

regarding the consequences of sleep loss (Lichstein & Rosenthal, 1980; Wicklow & Espie, 2000). These cognitive factors may underlie the behavioural processes which are thought to lead to sleep deficits. For example, some individuals believe they require a certain number of hours of sleep each night in order to function the following day. If they do not meet this specific requirement, they may then decide to sleep-in or nap in order to compensate for this sleep debt. However, as discussed above, oversleeping can disrupt the body's homeostatic mechanism, which can then make it more difficult to fall asleep at night. As another example, people who worry about their sleep, particularly at night, are more likely than their non anxious counterparts to have sleep-onset difficulties (Hall, Buysse, Reynolds, Kupfer & Baum, 1996; Haynes, Adams & Franzen, 1981). Ultimately, it appears that the cognitions support and maintain the sleep-disruptive habits, which in turn perpetuate the insomnia.

Some may notice that many normal sleepers adopt the abovementioned maladaptive behaviours, such as napping during the day, and do not develop any sleep disturbances. There are several reasons why this might occur. As discussed in the first section, people with insomnia may have specific predisposing factors which enhance their susceptibility to developing a chronic sleep problem. In particular, the homeostatic mechanism known to standardize our sleep-wake patterns has been found to be dysregulated in those with insomnia (Pigeon & Perlis, 2006). As such, a daytime nap for some individuals (i.e., non-insomniacs) may not be sufficient to produce sleep-onset difficulties, whereas for those with a more sensitive homeostat (i.e., those with insomnia), this may result in deleterious consequences for sleep. In addition, what may be more important than the behaviour itself, such as a nap, is the significance and meaning assigned to the particular event. Take the following analogy as an example. For most people, eating a single chocolate cupcake does not have a significant impact on their lives. However, for someone

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with diabetes, such a treat would present a health risk to the individual and thus he or she would be more inclined to monitor for and stay away from such health-related threats. In this vein, if a given individual does not have a cognitive orientation to attribute meaning to sleep loss, then he or she is less likely to consider the event threatening, and as a result there are no functional consequences for sleep.

Given the instrumental function of cognitive processes in the development and maintenance of insomnia, recent theoretical models have outlined how these mechanisms work to perpetuate the disorder. In particular, Harvey's (2002) contemporary cognitive model of insomnia describes these cognitive processes in detail. Briefly, Harvey's model states that individuals with sleep difficulties often experience negatively toned mental activity regarding their sleep and subsequent daytime functioning. This cognitive activity usually stems from thoughts about not getting enough sleep and about the impact that sleep disturbance will have on daytime functioning and performance. These negative thoughts can trigger a state of increased arousal and distress, which is associated with an attentional bias towards threat, whereby poor sleepers focus their attentional resources on distressing sleep-related cues (i.e., threats). Given their negative thoughts about sleep and attentional bias, they selectively monitor for sleep-related threats both internally (within themselves) and externally (in the environment). Together, the increased sleep-related anxiety and bias toward sleep threat can actually make it more difficult to fall asleep, as research has shown that the optimal conditions for sleep-onset include minimal cognitive activity and effort in the pre-sleep period (Espie, 2002; Kohn & Espie, 2005). This lack of sleep during the night can also lead to impaired daytime performance, which is then often exclusively attributed to the previous night's poor sleep. In general, the model suggests that the

aforementioned cognitive processes work to trap the individual in an ongoing cycle, wherein they become more absorbed by and anxious about their sleep difficulty.

In addition to Harvey's model, there are other theories that suggest cognitive processes play a central role in the maintenance of insomnia. For example, Espie's (2002) psychobiological inhibition model posits that intrusive thinking in the pre-sleep period inhibits the normal reductions in arousal necessary for sleep to unfold. Similarly, Lundh and Broman's (2000) theory of sleep-interfering and sleep-interpreting processes suggests that the interaction of stressful life events, arousal, and negative appraisal of sleep conspire to produce insomnia. Taken together, in the context of both Harvey's (2002) and these other models, it is cognitive arousal and interpreting or attributional processes which operate to maintain insomnia, and as such, are important targets of research.

Maladaptive Sleep-Related Beliefs and Attributions in Insomnia

Harvey (2002) acknowledges additional factors can further maintain the negatively toned cognitive activity, and thus the insomnia. These are known as unhelpful beliefs about sleep. An example of such a belief is the common notion that one must obtain a minimum of eight hours of sleep in order to function the following day. In fact, sleep quantity is a relatively unimportant predictor of daytime functioning in comparison to sleep quality, and as such, many people function adequately with less than eight hours of sleep (Pilcher, Ginter, & Sadowsky, 1997). Although many people strictly adhere to this "golden rule" of needing a minimum of eight hours of sleep, it is the strength of this belief, or the conviction with which it is believed to be true, that differentiates between good and poor sleepers (Morin et al., 1993). For example, someone with insomnia would likely be quite concerned if they received seven or nine hours of sleep due to their inflexibility regarding such sleep beliefs, whereas a good sleeper who holds the same belief

may be more adaptable in this situation. That is, a good sleeper knows that there are a range of possibilities with respect to the link between daytime functioning and sleep. One can obtain eight hours of sleep and feel sluggish during the day, or obtain six hours and feel great. According to Harvey's model, holding these maladaptive, rigid beliefs combined with the reduced capacity to produce eight hours of sleep per night can create anxiety within the individual and continue to fuel the negative cognitive activity. Thus, unhelpful beliefs about sleep are additional exacerbating factors that work to further perpetuate the insomnia. Indeed, research has found that poor sleepers tend to endorse stronger unhelpful beliefs and attitudes about sleep than do good sleepers (Morin et al., 1993).

Attributions are a specific type of belief implicated in sleep disturbance and insomnia (Morin et al., 1993). As an example of a sleep-related attribution, people with insomnia may *attribute* their sleep disturbance to a specific type of medication, or perhaps to a difficult week at work. As another example, poor sleepers may *attribute* a bad day to the fact that they did not sleep well the night before. While such attributions may be true, inaccurate attributions can have deleterious consequences for poor sleepers. In the latter case above, if negative daytime experiences are exclusively, or incorrectly attributed to poor sleep, it increases pressure to sleep well (i.e., to avoid further negative experiences during the day). In support of this idea are studies showing that similar to maladaptive beliefs about sleep, faulty attributions are significant precursors of heightened cognitive arousal and thus play an influential role in perpetuating the insomnia (Espie, 2002; Lundh & Broman, 2000; Morin, 1993). However, while general unhelpful beliefs about sleep have been subject to a number of empirical tests in the literature (Carney, Edinger, Manber, Garson, & Segal, 2007; Edinger, Carney & Wolgemuth, 2008; Morin

et al., 1993), and are outlined as exacerbating factors in Harvey's (2002) model, misattributions related to insomnia have not received as much attention in the literature.

Nevertheless, several early studies have demonstrated the importance of attributions in cognitive models of insomnia. For example, Storms and Nesbitt (1970) found that after instructing a group of insomnia patients that a placebo pill would have an arousing affect, they had shorter sleep onset latency compared to those who were told the pill would have a relaxing effect. The authors suggested that the participants' faster sleep onset was a result of attributing their arousal to the pill rather than to processes within themselves. This attribution decreased their anxiety, thus enabling them to fall asleep more quickly. In a similar study, people with insomnia received treatment including both behavioural therapy as well as sleep medication (Davison, Tsujimoto, & Glaros, 1973). Following the drug therapy, half of the participants were told that they received an optimal dose of the drug whereas the other half was notified that the dosage was not sufficient to produce any noticeable change. As expected, the group informed that they received a suboptimal dose achieved greater maintenance of their improvements, as they did not attribute their success to the drug. The group that was told they received an adequate dose, however, did attribute their improvement to the drug, and were not able to maintain these improvements in the long term. These manipulation studies demonstrate that attributing sleep loss to internal versus external factors can have a significant influence on whether their sleep improves as a result.

While the aforementioned studies examined different types of attributional tendencies within insomnia sufferers, the question remains whether the content of attributions differ among those with and without insomnia. To answer this question, Van Egeren, Haynes, Franzen and Hamilton (1983) examined the content of sleep-related attributions among individuals with

varying degrees of sleep-onset insomnia. The attribution measure used in this study consisted of a list of commonly perceived causes of sleep loss which were rated along three dimensions: location (internal-external cause), stability (transient-stable cause), and intentionality (degree of perceived control over the event). The results indicated that the most important predictor of sleep disturbance were attributions over which participants had no control, suggesting that individuals with sleep-onset insomnia perceive their sleep problem as uncontrollable.

Daytime Fatigue and its Causes

In addition to sleep-related cognitions which operate during the night, research has begun to consider daytime experiences in insomnia, most notably fatigue. Fatigue is a feeling of low energy during the daytime that is a common complaint among people with insomnia. Today, the research literature agrees that insomnia is both a nighttime and a daytime disorder (Buysse et al., 2007; Harvey, 2002; Moul, Nofzinger, Pilkonis, Houck, Miewald, & Buysse, 2002), and as a result, cognitive models of insomnia (i.e., Harvey, 2002) can be applied to both nighttime and daytime symptoms. In this sense, poor sleepers' heightened arousal and awareness of sleep-related stimuli during the night tends to mirror their increased attention to sleep-related cues during the daytime, which typically manifests as fatigue. For example, during the night, people may notice their heart pounding and thoughts racing, preventing them from falling asleep, whereas during the day, they may notice fatigue or inability to concentrate, precluding their ability to function effectively. In either case, this type of monitoring and detection of threatening cues continues to fuel sleep-related anxiety and worry, and thus the insomnia (Harvey, 2002).

It is important to note, however, that daytime experiences of insomnia (e.g., fatigue) may in fact be a byproduct of the increased attentional bias toward sleep-related threats during the daytime. In this sense, if poor sleepers monitor for sleep threats within their body and in their

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environment, they are more likely to detect instances of fatigue compared to those who do not monitor. Indeed, research has found that people with insomnia have an increased attentional bias toward sleep-related stimuli, suggesting that they are more emotionally and cognitively impacted by such stimuli (Spiegelhalder, Espie, Nissen, & Riemann, 2008). As such, it is possible that daytime fatigue in those with insomnia is a reflection of a sleep-related attentional bias rather than objective fatigue.

Nevertheless, daytime fatigue is a very frequent complaint among those with insomnia, (Roth & Ancoli-Israel, 1999; Ustinov et al., 2010) and as such, warrants research attention. Indeed, recent research found support for the notion that poor sleepers are highly concerned by the prospect of fatigue (Harris & Carney, 2009). This study explored the concept "fear of fatigue", which states that poor sleepers have an increased aversion to the experience of fatigue and will attempt to avoid this experience if possible. Indeed, results of this study indicated that poor sleepers view fatigue as threatening, and engage in behaviours to avoid this experience. Essentially, these findings demonstrate that poor sleepers have a pre-occupation with fatigue, which, similar to their increased focus with sleep-related cues, can contribute to their sleep-related anxiety and further perpetuate the insomnia.

There are many possible causes of fatigue. Whereas insomnia sufferers tend to assume that lack of sleep is always the most likely cause of daytime fatigue, several research studies have demonstrated that there are a multitude of causes of fatigue, many of which are unrelated to sleep. Some particularly common examples include: 1) a natural daily mid-afternoon dip in core body temperature and alertness controlled by the circadian pacemaker (Hayashi, Watanabe, & Hori, 1999); 2) boredom and low stimulation (Grandjean, 1979); 3) physical under- or over-activity (Puetz, O'Connor, & Dishman, 2006); 4) illnesses such as a virus or anemia (Sobrero et

al., 2001); 5) depression and anxiety (Greenberg, 2002); and 6) caffeine withdrawal (Juliano & Griffiths, 2004).

Indeed, many physical and psychological factors, such as low systolic blood pressure, increased heart rate, sleepiness, high levels of anxiety and worry, depressed mood, and unhealthy lifestyle are significant predictors of fatigue (Wijesuriya, Tran & Craig, 2007). Similarly, research exploring fatigue in cancer patients revealed that weight loss, muscle abnormalities, pain, circadian rhythms, depression, and stress are all correlates of cancer-related fatigue (Ancoli-Israel, Moore, & Jones, 2001; Stone, Richards & Hardy, 1998). While sleep disruption is also a common complaint among cancer patients, to date, there is no evidence that daytime fatigue and sleep difficulties are causally related in this patient population (Ancoli-Israel et al., 2001). Further, in Chronic Fatigue Syndrome, a condition whose primary symptom is daytime fatigue, sleep disturbance is not considered to be a fundamental causal factor (White, 2004). As such, the primary causes of daytime fatigue may be more multifaceted than one would expect.

Attributions of Daytime Fatigue

Given the burdensome nature and high prevalence of daytime fatigue (Roth & Ancoli-Israel, 1999), recent research has begun to examine people's causal attributions for this daytime impairment. As demonstrated above, there are many possible causes of daytime fatigue. Nevertheless, research has shown that poor sleepers tend to focus on sleep as an explanation of their fatigue and ignore other equally likely causes. For example, Morin and colleagues (1993) found that individuals with insomnia made stronger attributions of mood disturbance and low energy to poor sleep than did good sleepers. Further, they found that good sleepers disagreed with the statement, "one can hardly function during the day without a good night's sleep" more strongly relative to poor sleepers. Other researchers agree that people with insomnia tend to

misattribute daytime symptoms to sleep more often than do those without sleep disturbances (Carney & Edinger, 2006; Espie, 2002).

The psychobiological inhibition model of insomnia presents a parsimonious rationale for the misattribution of daytime impairment to poor sleep (Espie, 2002). This model posits that insomnia arises from the disruption, or inhibition, of one or more processes which contributes to normal sleep in good sleepers. As such, Espie argues that good sleepers have more accurate sleep-wake attributions, such that they are less likely to attribute fatigue and other daytime impairments to the preceding night's sleep. Instead, he explains, they are more likely to associate fatigue with corresponding life events (e.g., work stress) rather than a maladaptive sleep pattern. According to this model, it is the disruption of the accurate sleep-wake attribution system which contributes to misattributions of fatigue among poor sleepers.

Consequences of Misattributions of Fatigue

When considering these research findings, it is important to understand why people with insomnia are more likely to attribute fatigue to poor sleep, and more importantly, what the consequences are of these mis- or over-attributions. As previously mentioned with regard to Harvey's (2002) model, those with insomnia tend to focus their attention on sleep as a result of negative cognitive activity and subsequent arousal. At this point, they become pre-occupied with sleep and monitor internally and externally for sleep-related threats. In this sense, fatigue can be considered a sleep-related threat that occurs during the daytime. Given their preoccupation with sleep, the fatigue will likely be attributed to poor sleep, rather than the other myriad of possibilities. This process of attributing fatigue solely to a poor night's sleep will again lead to negative cognitive activity related to sleep. Thus, according to the model, attributing fatigue

solely to poor sleep can perpetuate the insomnia cycle via increased sleep-related anxiety and monitoring for sleep threats.

Misattributing fatigue exclusively to poor sleep can also affect people's behaviours in the pre-sleep period. Along with the increased negative cognitive activity and sleep-related anxiety, insomnia sufferers begin to feel pressured to sleep better in order to overcome their daytime fatigue problems (Harvey, 2002). As such, they often go to bed at night with the intention to fall asleep. However, sleep is one of the few things humans do wherein increased effort and pressure actually increases the likelihood of the problem persisting (Broomfield & Espie, 2005; Espie, 2002). Indeed, research has found that putting forth effort to fall asleep can effectively discriminate good and poor sleepers (Kohn & Espie, 2005). Further, Ansfield, Wegner and Bowser (1996) provided experimental evidence that sleep effort is a maintaining factor of insomnia. The authors instructed good sleepers to either fall asleep quickly or whenever they desired, under high or low mental load conditions. Participants who were instructed to fall asleep immediately in the high mental load condition had the longest sleep-onset latency, as this was the most cognitively demanding condition. Given that insomnia sufferers are prone to increased cognitive activity (i.e., high mental load) in the pre-sleep period, these findings confirm that putting forth effort to sleep will result in poor sleep outcomes. In sum, over-attributing fatigue to poor sleep can lead to increased sleep effort at night, which can make it even more difficult to bring on sleep, thus perpetuating the insomnia.

Implications for Treatments of Insomnia

Given the substantial research findings and theory supporting the problematic outcome of attributing fatigue solely to poor sleep, it might be important for poor sleepers to learn to consider other potential attributions for daytime fatigue. Doing so may reduce anxiety about

sleep and decrease the likelihood of further maintaining the insomnia. Specifically, it would be interesting to see whether a brief cognitive intervention can reduce insomnia sufferers' over-attribution tendency and allow them to consider other plausible reasons for feeling tired. Indeed, such an intervention may be an important addition to current treatments for insomnia.

There are currently several treatment options for individuals with insomnia, including Cognitive Behavioural Therapy (CBT). CBT is a multi-component treatment which is comprised of cognitive and behavioural interventions for treating insomnia, such as sleep restriction, stimulus control, and cognitive therapy. Its theoretical underpinnings are supported by empirical research findings which demonstrate that cognitive and behavioural changes leads to symptom improvement in individuals with insomnia. CBT is currently the first-line treatment for insomnia, given its well-documented empirical support in insomnia populations (Morin et al., 2006). CBT has been found to be efficacious when compared to control groups (e.g., Lichstein, Wilson, & Johnson, 2000), placebo treatments (e.g., Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001), stand-alone behavioural interventions (e.g., Edinger, Wohlgemuth, Radtke, Marsh & Quillian, 2001), and pharmacotherapy treatments (e.g., Jacobs, Pace-Schott, Stickgold, & Otto, 2004). More recently, research has provided evidence for the efficacy of CBT in the treatment of comorbid disorders (e.g., Edinger et al., 2009; Rybarczyk, Lopez, Alsten, Benson & Stepanski, 2002), and the clinical effectiveness of CBT in primary care settings (e.g., Espie, Inglis, Tessier, & Harvey, 2001). These research findings are consistent with the results of two meta-analyses on non-pharmacological treatments of insomnia (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995), wherein CBT had effect sizes in the large range for most outcome variables (Cohen, 1988).

Despite the overwhelming empirical support for CBT in insomnia populations, there continues to be room for improvement. For example, the overall average improvement was found to be only 50-60% (Morin et al., 1994) and there are a subset of insomnia patients for whom CBT does not work at all (Harvey & Tang, 2003; Morin et al.,1994). Further, many individuals who do improve continue to have residual sleep disturbance, which may pose a risk for future relapse (Morin et al., 2006). As such, research aimed at improving treatment outcomes for this highly burdensome disorder is urgently needed.

Importantly, while many research studies have provided evidence for behavioural strategies for treating insomnia (Morin et al., 1994; 2006), research examining cognitive treatments is still in its infancy. Nonetheless, as discussed above, it is the cognitive processes which are often hypothesized to be the driving force behind the sleep-disruptive behaviours, and which ultimately maintain the insomnia. Indeed, recent research suggests that targeting maladaptive cognitions in treatment may have important implications for improving response rates. For example, people with insomnia who benefitted most from CBT were the patients who had the highest degree of cognitive change with respect to their maladaptive sleep beliefs (Edinger et al., 2008). Similarly, an open trial of cognitive therapy for insomnia found improvements in both nighttime and daytime measures of sleep impairment during treatment and up to 12-month follow-up (Harvey Sharpley, Ree, Stinson, & Clark, 2007). Taken together, it appears that exploring the value of adding cognitive strategies into CBT is a worthwhile research endeavor.

Although some studies have examined maladaptive sleep beliefs and other components of cognitive therapy for insomnia, no studies to date have directly explored whether attributional biases can be effectively modified and whether this would have implications for improving mood

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or anxiety. Specifically, Harvey and colleagues (2007) targeted fatigue attributions in their treatment, but it is not clear if the sleep improvements were due to changes in attributions or other treatment targets in the intervention. As such, the goal of the present study was to fill this gap in the literature. By examining whether people could change their sleep-focused tendency with a minimal attribution-targeted intervention, this study investigated a new and unexplored avenue toward refining insomnia treatments.

The Present Study

The present study examined people's attributions for fatigue. The results of Storms and Nesbitt (1970) indicated that when participants were given an alternative explanation for their arousal before bed (the pill), they were able to fall asleep more quickly, as they no longer attributed their arousal to internal factors that were under their control. Similarly, when individuals are fatigued during the day, it is likely that they would over-attribute, or perhaps misattribute that sleepy state to insufficient sleep. Indeed, as Harvey's (2002) model suggests, poor sleepers have a tendency to attend to and scan their body for sleep-related threats, thus increasing the likelihood that they would attribute their fatigue to poor sleep. However, if alternative explanations were provided, they may be more likely to attribute their fatigue to something else, thereby reducing their sleep-related anxiety. As such, the purpose of this study was to determine whether presenting alternative explanations for fatigue would increase the likelihood that participants would attribute their fatigue to reasons unrelated to sleep, and whether this cognitive change would have implications for relevant mood states.

To answer this research question, participants were randomized to either a Fatigue-Information (FI) group or a Control-Information (control) group. Participants in the FI group were given information related to possible causes of fatigue whereas the control group was

provided with information related to sleep but unrelated to the causes of fatigue. Cognitive change was determined via pre- and post-intervention outcome measures designed for the purpose of this study, which were as follows: 1) a participant-generated list of attributions (LAT) that account for them feeling tired, 2) a ranking of the frequency (i.e., in accounting for their fatigue) of the participant-generated attributions, 3) the participants' rating of the likelihood that each of these participant -generated factors contributes to their fatigue, and 4) unipolar Visual Analog Scales (VAS) to measure fatigue, positive mood and worry. Using this design, this study examined whether fatigue-related information was helpful in orienting participants to the other possibilities for the causes of their fatigue.

While both good and poor sleepers were eligible to participate in the study, only participants who scored above the clinical cutoff of 3.8 (Carney et al., 2010) on the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16: Morin, Vallieres, & Ivers, 2007) were included in the analyses. Individuals who scored above this cutoff have a high degree of maladaptive sleep beliefs and an increased cognitive vulnerability to insomnia (Carney et al., 2010; Morin et al., 1993). There are several reasons as to why participants were selected based on their presumed cognitive vulnerability on the DBAS-16. First, given that this study was testing a cognitive intervention, it made intuitive sense to use a cognitively vulnerable subset of participants. Second, such participants were selected so not to exclude participants who indicated that they did not have insomnia at the time of their testing (i.e., if their insomnia was not activated), or if they had an acute sleep disturbance as a result of one difficult week. Indeed, insomnia can be a transient phenomenon which may be influenced by the time of year, physical well-being and several other factors for undergraduate students (Jensen, 2003; Lack, 1986), and it was important to ensure that we would capture an enduring measure of insomnia susceptibility

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as opposed to a temporary assessment of sleep disturbance. Finally, selecting a vulnerable sample enabled us to get a sense of whether this intervention would be effective for the prevention of insomnia in future studies.

In order to determine whether those vulnerable to insomnia could adopt other, non-sleep-related attributions for fatigue via a brief information intervention, the following predictions were proposed:

Primary Hypotheses:

- Hypothesis 1: the number of generated sleep-related attributions for feeling tired during the day, relative to non-sleep-related attributions, in the list of attributions task, would decrease for those in the FI condition only.

- Hypothesis 2: the rank and proportion allotted to sleep-related attributions, relative to non-sleep-related attributions, in the generated list would decrease after FI.

Secondary Hypothesis:

- Hypothesis 3: the VAS fatigue and worry ratings would decrease, and the positive mood rating would increase for those in the FI condition.

Method

Design

This study used a randomized 2x2 experimental design with one between-subjects variable representing group with two levels (FI and control) and one within-subjects variable representing time with two levels (pre- and post-intervention). In this design, an interaction between condition and time was expected. That is, the study hypotheses predicted that cognitive and mood change on the dependent variables would occur between pre- and post- intervention for the FI group, but not for the control group. The study procedures were completed within a one-hour testing session.

Participants

Participants were undergraduate students enrolled in an introductory psychology course at Ryerson University. The students were recruited via SONA, the psychology department's online recruitment system. Students who were interested in this study volunteered to participate in partial fulfillment of their introductory psychology course requirement. They were not financially compensated.

A total of 93 undergraduate students participated in this study. The sleep of people approaching age 18 may be different from those aged 18 or older because of circadian influences that relate to puberty and brain development; thus, two participants under the age of 18 were excluded from the analyses. Another three participants did not complete all outcome measures at pre- and post- intervention and were not included in the analyses. Thus, there were 88 participants available for analyses between the ages of 18 and 39 ($M = 20.00$, $SD = 4.30$). Participant demographics are presented in Table 1.

Table 1

Participant Demographics and Self-Report Measure Psychometrics for Total Sample

Variables	Proportion (%)	
Sex		
Female	81.8	
Male	18.2	
Ethnicity		
Caucasian	34.1	
Asian	31.8	
Black	9.1	
Middle-Eastern	3.4	
Aboriginal	2.3	
Other	19.3	
Self-Report Measures	Mean (SD)	Alpha
[a]ISI	9.97 (4.71)	.80
MFI	57.42 (12.79)	.89
DBAS-16	4.91 (1.60)	.85
BDI-II	14.69 (9.31)	.89

Note. ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep Scale, BDI-II = Beck Depression Inventory.
[a]73.8% above clinical cutoff of ISI > 7 (Bastien et al., 2001).

Self-Report Measures

      The Insomnia Severity Index (ISI: Morin, 1993). The ISI is a recommended self-report measure for assessing insomnia (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). It is a 7-item scale which measures the severity of insomnia symptoms as well as degree of dissatisfaction, daytime interference, noticeability of impairment, and distress caused by the sleep disturbance. Each item is measured on a 5-point Likert scale ranging from 0 ("not at all") to 4 ("extremely"). Total scores range from 0 to 28, with higher scores suggesting increased insomnia severity. The recommended interpretation guidelines are as follows: scores of 0 – 7 suggest no clinical insomnia, scores of 8 – 14 suggest sub-threshold insomnia, scores of 15 – 21 suggest moderate insomnia, and scores of 22 – 28 suggest severe insomnia (Bastien, Vallieres, & Morin, 2001). The ISI has been found to have good internal consistency (Chronbach's alpha = .91) (Sierra, Guillén-Serrano, & Santos-Iglesias, 2008) and good concurrent validity, as it correlates with sleep diary measures and polysomnography (Bastien et al., 2001). See Table 1 for the psychometric properties of each of the self-report measures in the present sample.

      The Multidimensional Fatigue Inventory (MFI: Smets, Garssen, Bonke, & DeHaes, 1995). The MFI is a 20-item scale that assesses various dimensions of fatigue, including: general, physical, mental, reduced motivation, and reduced activity. These five dimensions represent distinct subscales of the MFI, each of which contains four items. Responses range on a 5-point scale from "yes that is true" to "no that is not true". The MFI has good internal validity (Chronbach's alpha = .84) and adequate convergent validity, as was demonstrated by correlations between the MFI and Visual Analog Scales measuring fatigue (Smets et al., 1995). The MFI has been tested in insomnia populations with breast cancer (Quesnel, Savard, Simard, Ivers, &

Morin, 2003) and with comorbid alcohol dependence (Arnedt, Conroy, Rutt, Aloia, Brower, & Armitage, 2007).

The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16: Morin et al., 2007). The DBAS-16 is a self-report measure assessing unhelpful sleep-related beliefs. The DBAS-16 is an abbreviated version derived from the original 30-item DBAS. The measure assesses beliefs about the consequences of insomnia, worry about sleep, sleep expectations, and causal attributions for insomnia. The individual rates his/her level of agreement with each statement on a 10-point scale ranging from 0 ("strongly disagree") to 10 ("strongly agree"). The total score is obtained via a mean item score, with higher mean scores representing more maladaptive sleep beliefs. The DBAS-16 demonstrates adequate internal consistency (Chronbach's alpha = .79) and has appropriate convergent validity with the ISI, sleep diaries and polysomnography (Morin et al., 2007). It can also effectively discriminate between those who do and do not have clinical levels of unhelpful sleep beliefs via a cutoff score of 3.8, which maximized both sensitivity (80%) and specificity (76%) based on an ROC curve (Carney et al., 2010). In the present study, only those above this suggested cutoff score of 3.8 were selected for the main analyses.

The Beck Depression Inventory, Second Edition (BDI-II: Beck, Steer & Brown, 1996). The BDI-II is a 21-item measure that assesses common depressive symptoms, such as depressed mood, hopelessness, suicidal ideation, sleep disturbance, and appetite change. Total scores range from 0 to 63, with higher scores representing greater levels of depression. The recommended interpretation guidelines are as follows: scores of 0 – 13 suggest no depression, scores of 14 – 19 suggest mild depression, scores of 20 - 28 suggest moderate depression, and scores of 29 or above suggest severe depression. The BDI-II has very good internal consistency (split half

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Pearson = .93) and is correlated with similar measures of depression, such as the Hamilton Rating Scale for Depression ($r = .71$; Beck et al., 1996). It also has well established content validity and is good at differentiating between depressed and non-depressed individuals (Beck et al., 1996; Richter, Werner, Heerlein, Kraus, & Sauer, 1998). The BDI-II has been used and validated in insomnia patients (Carney, Ulmer, Edinger, Krystal, & Knauss, 2009).

Outcome Measures

List of Attributions Task (LAT) (see Appendix A). Participants were instructed to fill in a chart representing their attributions for fatigue, which was developed for the purposes of this study. In the first column, participants were asked to list factors that could account for them feeling tired during the day. The purpose of this list was to determine whether the attributions were related or unrelated to sleep. To this end, the data were coded according to sleep-related versus non-sleep-related attribution categories. For example, items such as "didn't get enough sleep", "up late at night studying", and "waking up too early" were coded as sleep-related items. Table 2 presents the most frequent categories and examples of responses which were coded as non-sleep-related. In order to come up with a single aggregate score for each participant, the number of attributions in each category (i.e., sleep-related and non-sleep-related) were added together for both pre- and post- intervention. The score was computed by dividing the number of sleep attributions by the total number of attributions, producing a score which represents the proportion of sleep-related attributions. This score will be subsequently referred to as NumberAttributionScore.

In the second column, participants rank ordered each listed attribution in order of frequency of occurrence, by assigning a rank of 1 to the factor that accounts for their fatigue most *often*. The rankings were scored in such a way that each rank would get a score from 1 to

Table 2

Frequency of Non-Sleep-Related Attribution Categories

Category	Examples	Frequency
School/Work	"Too much school work" "Studying all the time" "Working many hours"	105
Food/Nutrition	"Poor diet" "Ate too much" "No proper breakfast"	69
Anxiety/Stress	"Stressed out" "Worrying about the future" "Nervous"	50
Exercise	"Overexerted myself" "Not active enough" "Too much exercise"	40
Physical Illness	"Headaches" "Low iron" "Not feeling well"	19

Note. Frequency = Number of times this attribution category appeared in the dataset.

10, with a score of 10 representing the highest ranked attribution and a score of 1 representing the lowest ranked attributions. Specifically, a rank of 1 was given a score of 10, a rank of 2 was given a score of 9, a rank of 3 was given a score of 8…and a rank of 10 or higher was given a score of 1. This scoring system allowed for sufficient variability of scores, as most participants did not list more than 10 attributions. The resulting scores for the sleep-related attributions were then added together to compute an aggregate score representing the frequency with which sleep-related attributions account for their fatigue. As an example, if a participant listed two sleep attributions and assigned them ranks of '1' and '3' from their total list of attributions, their aggregate score would be 18 (10 + 8). This variable will be referred to as FrequencyScore.

In the third column, participants were asked to indicate the likelihood that each factor accounts for their fatigue. To this end, participants were able to allocate a specific proportion (out of 100%) to each attribution, with allocating a greater proportion to those factors that were considered to be more *important* or more *likely* in accounting for their fatigue. For example, a factor that was considered to be most likely to account for their fatigue may be allocated a proportion of 90%, whereas an attribution that was perceived to be less likely to contribute to their fatigue may be given a proportion of 25%. The aggregate score was computed in the same manner as was done in the first column: The total proportion for sleep-related items was divided by the total proportion for all attributions in order to retrieve a single score, which will be referred to as ImportanceScore.

Visual Analog Scales (VAS) (see Appendix B). The VAS queried current states of fatigue, positive mood and worry. Participants were instructed to mark an "X" on a 100 millimetre line which represented their current state. The VAS was a unipolar scale, whereby the responses ranged from "not at all" to "extremely". The VAS was scored with a ruler, whereby

the millimetre distance from the left edge of the line to the centre of the X was the score given for that particular VAS.

Intervention Materials

Fatigue Information (FI) Intervention (see Appendix C). The FI intervention consisted of a double-sided sheet containing information regarding common factors that can explain feeling fatigued during the day. This information was obtained from several studies throughout the literature which examine common causes of fatigue (e.g., Chaudhuri & Behan, 2004; Grandjean, 1979; Greenberg, 2002; Hayashi, Watanabe, & Hori, 1999; Juliano & Griffiths, 2004; Puetz et al., 2006; Resnick, Carter, Aloia, & Phillips, 2006; Sobrero et al., 2001; Wijesuriya, Tran & Craig, 2007). Some examples include caffeine rebound, poor nutrition, physical inactivity, and post-lunch changes in body temperature. Along with each listed factor was a short blurb explaining how fatigue can be caused by that particular factor.

Sleep Control (Control) Intervention (see Appendix D). The control intervention consisted of a comparable amount of information to the FI condition; however the information was about sleep, and unrelated to the causes of fatigue. For example this information session included information regarding sleep stages and tips to improve sleep. The control condition was necessary to control for the amount of time and sleep-related information inherent in the FI condition, but there should not have been any reason for the control group to alter their subsequent fatigue attributions or VAS ratings on the basis of the sleep information.

Procedure

Participants completed this study in a one-hour session in the Sleep and Depression Laboratory at 105 Bond Street, Ryerson University. The study candidates were first informed about the purpose and description of the study as well as the risks and benefits associated with

participation, after which they were required to provide written consent in order to participate (see Appendix E). To determine which information session they would receive, a Microsoft Excel random number generator was used to randomize participants into the two conditions. In the first part of the study, all participants completed a booklet of baseline measures, including the self-report questionnaires (ISI, MFI, DBAS-16, BDI-II) and outcome measures of the study (LAT and VAS). This took approximately 30 minutes.

After completing these measures, the participants either received information about reasons for fatigue (FI condition) or generic sleep information (control condition). The study investigator read the information sheet alongside each participant individually, which took approximately 5 minutes. All of the participants once again filled out the outcome measures (LAT and VAS), which took approximately 10 minutes. Once the participants completed the study, they were verbally debriefed, given a written debriefing form (see Appendix F), and had the opportunity to ask the study investigator any further questions regarding the study. Given that we asked participants about depression via the BDI-II, they were given another debriefing form which provided additional information regarding the signs and symptoms of depression, as well as resources for where to seek help (see Appendix G).

The written responses on the LAT were coded into sleep-related versus non-sleep-related attributions by a lab volunteer blinded to the study hypotheses. The study investigator then reviewed the categories while being blind to participants' random assignment to ensure that there was agreement. Responses on which there was discrepancy between the lab volunteer and the study investigator were flagged and were discussed. The majority of such items were responses which identified environmental issues related to sleep, such as "uncomfortable bed" or "loud roommate during the night". It was decided that these responses would fall under the category of

"sleep-related attributions", given that they would interfere with sleep. In total, only 12 responses out of 870 were changed, representing 1.38% of all item responses in the database. Once the attributions were coded into the appropriate categories, aggregate scores for the dependent variables on the LAT were created in order to test the study hypotheses, which were described in the preceding section.

Analyses

To ensure that the study sample was valid and comparable to other samples reported in the literature, Cronbach's alphas, means, and standard deviations were reported for the self-report measures (see Table 1).

As discussed above, only those participants who scored above the clinical cutoff of 3.8 on the DBAS-16 were selected for the main analyses. Independent t-tests were conducted to determine whether those high and low on the DBAS-16 were significantly different on subjective measures of insomnia (ISI) and fatigue (MFI). Independent t-tests and chi-square analyses were used to assess for any pre-existing group differences between those in the FI and the control group. For the main analyses, mixed within-between analyses of variance (ANOVAs) were conducted to determine whether participants' attributions for fatigue, based on the LAT, changed from pre- to post-intervention for those in FI relative to the control group. These same analyses were used to determine whether VAS ratings of fatigue, positive mood, and worry changed significantly, depending on group assignment. In each of the main analyses, the hypothesis was that there would be a significant group x time interaction; that is, that the FI group would alter their post-treatment cognitive and mood responses in the hypothesized direction, whereas the control group would not change their responses significantly.

Results

Preliminary Analyses

Prior to conducting the primary analyses, the data were screened for violations of the normality assumption. An inspection of the distribution of the mean scores on the ISI, MFI, DBAS-16, and BDI-II suggest that the distributions were approximately normal and the skewness and kurtosis values were within the normal ranges of |2| and |7| respectively (West, Finch, & Curran, 1995).

Participant demographic characteristics as well as mean scores and Cronbach's alpha values for the self-report questionnaires are displayed in Table 1. The correlations among these self-report measures are reported in Table 3. All of the questionnaires were significantly correlated with one another ($p < .01$).

Validity check for DBAS-16 cutoff score. To understand the characteristics of those participants selected for cognitive vulnerability for the main analyses, group differences were assessed (i.e., those above and below the DBAS-16 cutoff) on measures of insomnia and fatigue. Participants who were above the DBAS-16 cutoff had significantly higher levels of self-reported insomnia ($M = 11.05$, $SD = 4.78$) compared to those below the cutoff ($M = 6.96$, $SD = 2.90$), t (86) = 3.85, $p < .01$. Group differences were also found on the MFI, with those above the DBAS-16 cutoff scoring higher ($M = 60.97$, $SD = 11.59$) than those below ($M = 47.39$, $SD = 10.69$), t (86) = 4.92, $p < .01$.

The remaining analyses are based on the cognitively vulnerable subset of 65 participants. Mean scores and standard deviations for the self-report measures for each group of this selected sample are displayed in Table 4. As in the total sample, there were no violations of the normality assumption and the self-report measures were all significantly correlated with one another.

Table 3

Correlations among Self-Report Measures for the Total Sample

Measure	*1*	*2*	*3*	*4*
1. ISI	--	.38*	.49*	.46*
2. MFI		--	.52*	.55*
3. DBAS-16			--	.47*
4. BDI-II				--

Note. ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep Scale, BDI-II = Beck Depression Inventory.
*$p < .01$.

Table 4

Means and Standard Deviations for Pre-Intervention Scores on Self-Report Measures

Self-Report Measures	FI (n = 35)		Control (n = 30)	
	M	*SD*	*M*	*SD*
ISI	11.31	5.17	10.73	4.34
MFI	60.06	11.17	62.03	12.16
DBAS-16	5.46	1.13	5.85	1.11
BDI-II	18.62	10.01	16.03	7.65

Note. FI = Fatigue Information, ISI = Insomnia Severity Index, MFI = Multidimensional Fatigue Inventory, DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep Scale, BDI-II = Beck Depression Inventory.

Pre-existing group differences. The FI and control groups did not differ significantly with respect to age: $t(63) = -.79$, $p = .43$, sex: $\chi^2(1) = .12$, $p = .76$, or ethnicity: $\chi^2(1) = .21$, $p = .90$. As well, no significant pre-intervention group differences were found for any of the self-report measures [ISI: $t(63) = .49$, $p = .63$; MFI: $t(63) = .68$, $p = .50$; DBAS-16: $t(63) = -1.39$, $p = .17$; BDI-II: $t(63) = 1.16$, $p = .25$]. There were also no significant pre-existing differences on the LAT outcome measures [NumberAttributionScore: $t(63) = .97$, $p = .33$; FrequencyScore: $t(63) = .58$, $p = .57$; ImportanceScore: $t(63) = .40$, $p = .67$]. Finally, there were no significant pre-existing group differences on the VAS [Fatigue: $t(63) = .12$, $p = .90$; Positive Mood: $t(63) = .52$, $p = .61$; Worry: $t(61.75) = -.34$, $p = .74$].

Main Analyses

The mixed between-within subjects ANOVA results are as follows. For NumberAttributionScore, there was a significant interaction between group and time, $F(1, 63) = 7.06$, $p = .01$, $\eta^2 = .10$, such that the number of sleep attributions relative to the total number of attributions decreased for those in FI (pre: $M = .32$, $SD = .23$; post: $M = .22$, $SD = .23$), but not for those in the control group (pre: $M = .27$, $SD = .19$; post: $M = .31$, $SD = .24$) (see Figure 1). There was no main effect of group, $F(1, 63) = .11$, $p = .75$, $\eta^2 = .00$, nor was there a main effect of time, $F(1, 63) = 1.61$, $p = .21$, $\eta^2 = .03$.

With regard to FrequencyScore, there was a significant interaction between group and time, $F(1, 63) = 7.85$, $p = .007$, $\eta^2 = .11$. Thus, the rankings of how frequently each sleep-related attribution contributes to daytime fatigue significantly decreased for the FI group (pre: $M = 13.77$, $SD = 9.56$; post: $M = 8.66$, $SD = 7.87$), whereas no significant changes were found in the control group (pre: $M = 12.43$, $SD = 9.07$; post: $M = 12.50$, $SD = 9.75$) (see Figure 2). There was no main effect for group, $F(1, 63) = .37$, $p = .55$, $\eta^2 = .01$. However, there was also a significant
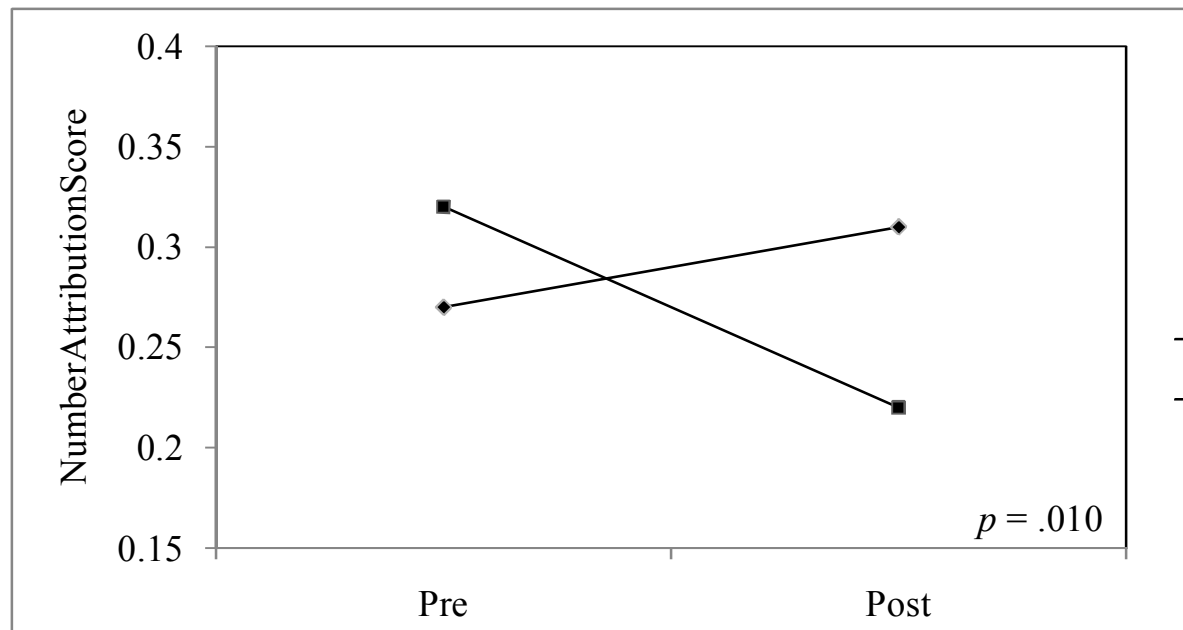
Figure 1. Number of sleep-related attributions relative to the total number of attributions for FI and control at pre- and post- intervention.
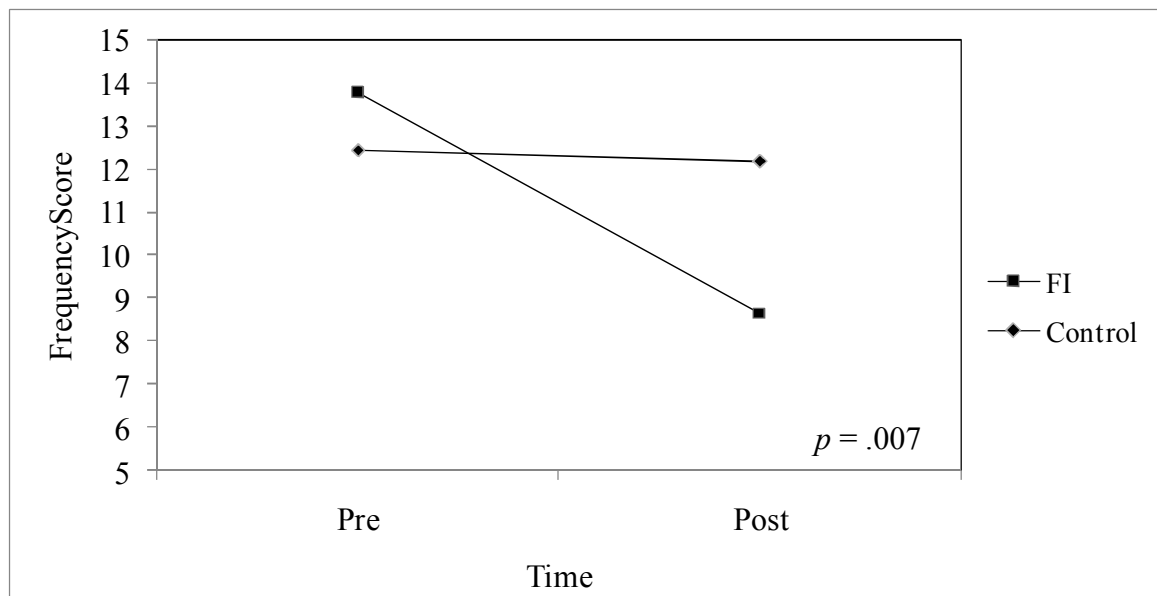
Figure 2. Rank scores of sleep-related attributions for FI and control at pre- and post-intervention.

main effect of time, $F (1, 63) = 7.45$, $p = .008$, $\eta^2 = .11$.

Finally, a significant interaction was found for ImportanceScore, $F (1, 63) = 5.10$, $p = .027$, $\eta^2 = .08$, such that compared to the control group (pre: $M = .35$, $SD = .25$; post: $M = .36$, $SD = .28$), the proportion of importance allotted to sleep-related attributions relative to the total proportion significantly decreased at post-intervention for those in the FI group (pre: $M = .38$, $SD = .27$; post: $M = .27$, $SD = .27$) (see Figure 3). There was no significant main effect for group, $F (1, 63) = .29$, $p = .60$, $\eta^2 = .01$, or time, $F (1, 63) = 3.85$, $p = .054$, $\eta^2 = .06$.

The means and standard deviations for the VAS outcome measure are presented in Table 5. There was no significant interaction on VAS fatigue ratings, $F (1, 63) = .69$, $p = .41$, $\eta^2 = .01$, nor was there a significant main effect of group, $F (1, 63) = .21$, $p = .65$, $\eta^2 = .00$. However, there was a main effect of time, $F (1, 63) = 4.54$, $p = .04$, $\eta^2 = .07$, such that levels of fatigue decreased between pre- ($M = 63.61$, $SD = 19.69$) and post- ($M = 59.65$, $SD = 21.58$) intervention, regardless of group assignment. For VAS ratings of positive mood, there was no significant interaction between group and time, $F (1, 63) = .00$, $p = .98$, $\eta^2 = .00$. There were also no significant main effect of group, $F (1, 63) = .31$, $p = .58$, $\eta^2 = .01$, or time, $F (1, 63) = 1.35$, $p = .25$, $\eta^2 = .02$. Finally, there was no significant interaction for self-rated worry, $F (1, 63) = 1.12$, $p = .29$, $\eta^2 = .02$, and no main effect of group, $F (1, 63) = .00$, $p = .98$, $\eta^2 = .00$. However, there was a significant main effect of time, such that self-rated worry did decrease significantly from pre- ($M = 75.64$, $SD = 30.55$) to post- ($M = 64.92$, $SD = 32.71$) intervention for both FI and control conditions, $F (1, 63) = 18.89$, $p < .01$, $\eta^2 = .23$.
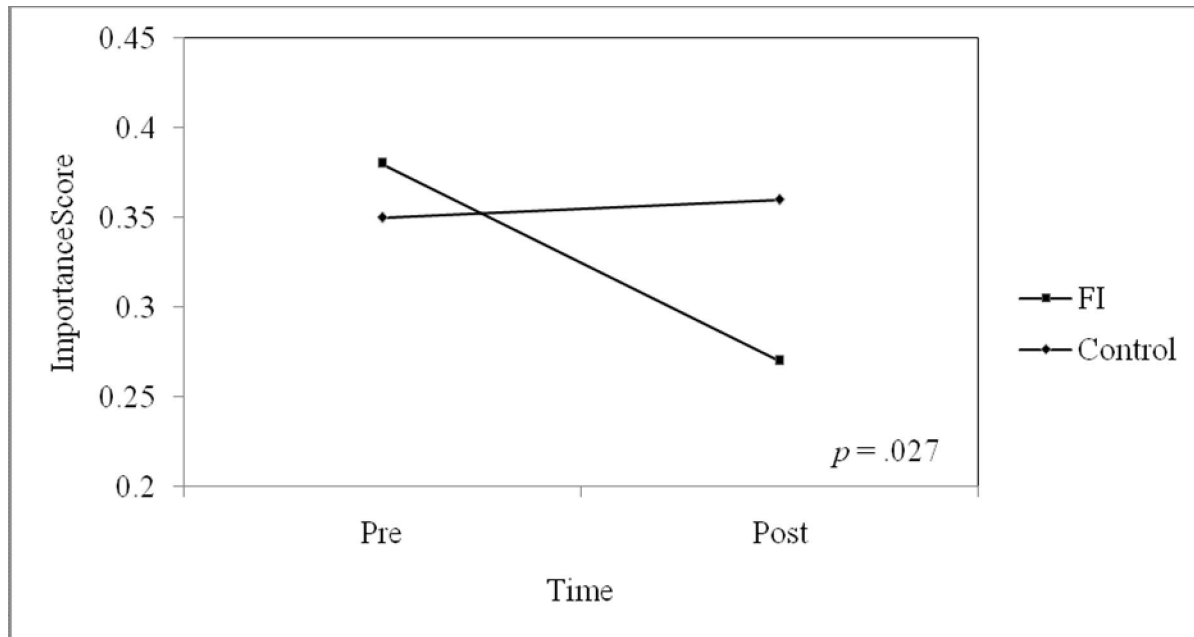
Figure 3. Proportion of importance allotted to sleep-related attributions relative to total proportion for FI and control at pre- and post- intervention.

Table 5

Means and Standard Deviations for VAS Pre- and Post-Intervention

	Pre-Intervention		Post-Intervention	
VAS	FI	Control	FI	Control
Fatigue	63.88 (21.45)	63.28 (17.79)	61.38 (23.47)	57.63 (19.33)
Positive Mood	55.83 (21.93)	53.25 (17.36)	57.97 (21.76)	55.48 (16.49)
Worry	58.19 (27.02)	60.16 (19.98)	51.73 (30.31)	49.53 (19.01)

Note. FI = Fatigue Information, VAS = Visual Analogue Scales.

Discussion

In this test of a brief psycho-educational intervention for those with a cognitive vulnerability to insomnia, the results revealed significant changes in cognitive attributions for fatigue. Specifically, the findings demonstrated that educating people about the many causes of daytime fatigue was successful in broadening their scope of non-sleep-related fatigue attributions. However, the intervention did not produce significant improvements in fatigue, positive mood, or worry, relative to those who did not receive this educational information.

With respect to the primary hypotheses regarding cognitive change, the results demonstrated that attributing fatigue to poor sleep was indeed amenable to change for people who have a cognitive vulnerability to insomnia. In particular, with regard to the number of attributions reported, the proportion of sleep-related attributions decreased significantly for the group provided with information about the many causes of fatigue (i.e., FI) relative to those who were provided with generic sleep information (i.e., control). This finding suggests that participants in FI were less likely to attribute fatigue back to poor sleep, and more likely to consider additional non-sleep-related attributions post-intervention. Thus, as predicted, the number of sleep attributions decreased in the group who was informed about the contribution of factors other than sleep in producing daytime fatigue.

The expected results were also found for the perceived frequency and importance of the respective attributions. Specifically, the rank scores allotted to sleep-related attributions significantly decreased post-intervention for those in FI, suggesting that the sleep-related factors were perceived to cause their fatigue less often than they were pre-intervention. The significant time x group interaction suggests that those who received information about the causes of fatigue were less likely to consider sleep-related attributions as the more frequent causes of fatigue.

Likewise, the proportions of importance allotted to sleep-related attributions relative to the total proportion allotted to attributions also decreased post-intervention for FI participants. This finding suggests that the perceived importance (i.e., the likelihood that the fatigue is caused by the attribution) of these sleep-related attributions declined over time for those who received information regarding the many possible causes of fatigue.

Taken together, the expected findings for the primary hypotheses suggest that the minimal cognitive intervention used in this study was sufficient to create a change in people's reported attributions for their daytime fatigue. More importantly, this change was in the expected direction, as those who received the relevant fatigue information emphasized sleep-related attributions to a lesser degree, and were more likely to consider non-sleep-related factors as potential causes of their fatigue at post-intervention. Finally, in addition to these statistically significant findings, the effect sizes for the time x group interactions were all within the medium to large range (Cohen, 1988).

The secondary hypothesized decrease of negative mood states (i.e., as a result of cognitive change), was not supported in this sample. There was no interaction between time and the type of information received (i.e., group) for fatigue, positive mood, or worry. One possible explanation is that, while the study intervention had an effect on participants' reported attributions for fatigue, it may not have been sufficiently potent to bring about change in relevant mood states. Specifically, the cognitive intervention used in this study was akin to a psycho-educational intervention. Thus, this intervention should be regarded as quite minimal in that it consisted of a two-page sheet of information read by the study investigator to the participants. In order to ensure that each participant received the same information, the study investigator did not elaborate on the information provided in the session, nor was the information personalized for

each individual participant. As a result, the participants were quite passive during the

information sessions. This differs markedly from traditional cognitive therapy, where the patients

play an active role in session and the information can be discussed at length and individualized

for specific cases. Given the well-established efficacy of several of these cognitive-based

treatments (Beck, Hollon, Young, Bedrosian, & Budenz, 1985; DeRubeis & Crits-Christoph,

1998; Hollon, Thase, & Markowitz, 2002), perhaps a longer and more interactive cognitive

intervention would be needed in order to see improvements in fatigue, mood and worry. In sum,

the information session used in this study was a minimal intervention, which may not have

contained enough active ingredients to produce state change.

Despite the non-significant VAS time x group interactions, there were significant time

effects for both self-rated fatigue and worry; thus levels of fatigue and worry decreased post-

intervention regardless of the type of information received. While these results were not

anticipated (i.e., in the absence of a significant interaction effect), there are several possibilities

as to why these effects may have occurred. Although each group received different information

sessions, it may be that the information provided in both groups was helpful in alleviating

feelings of fatigue and anxiety. For example, it is possible that learning novel information,

regardless of the content, may have increased mental stimulation, thus reducing fatigue levels. In

a similar vein, perhaps learning about relevant sleep information, such as tips for improving

bedtime habits in the control condition, was able to relieve sleep-related anxiety, thus reducing

VAS ratings of worry.

In addition to receiving information, both the FI and control conditions were instructed to

write down their attributions for fatigue before and after the interventions. Research has shown

that the simple act of writing can have a profound effect on mood and psychological health

(Baikie & Wilhelm, 2005; Pennebaker, 1993). As such, although the written component of the LAT was relatively minimal, it is possible that generating written responses was sufficient to alleviate anxiety. Further, given that writing is a mentally stimulating activity, it could have also brought about minimal improvements in levels of fatigue.

In many respects, the study sample was comparable to other samples in the literature. Cronbach's alphas for each of the self-report questionnaires (ISI, MFI, DBAS-16 and BDI-II) were good and similar to those reported in the literature (Bastien et al., 2001; Beck et al., 1996; Morin et al., 2007; Sierra et al., 2008; Smets et al., 1995). Correlational analyses revealed significant relationships between insomnia (ISI), fatigue (MFI), maladaptive sleep beliefs (DBAS-16) and depression (BDI-II). These correlations were all similarly correlated with one another (*r*'s range from .38 to .55) and suggest that while these measures do share some common variance (14 – 30%), they are indeed measuring distinct constructs. These findings are comparable to previous examinations in the literature which too have documented significant relationships among insomnia, depression, and fatigue (Ferentinos et al., 2009; Greenberg, 2002; Moul et al., 2002; Ustinov et al., 2010). More recent studies have documented similar associations between maladaptive sleep beliefs (measured by DBAS-16) and insomnia (measured by the ISI), as well as depression (measured by the BDI-II) (Carney et al., 2007; Morin et al., 2007). However, this is the first study to date establishing a correlation between maladaptive sleep beliefs and daytime fatigue, which is important to note given that improvements in maladaptive sleep beliefs may have implications for associated improvements in fatigue. Taken together, these results demonstrate that this study sample appears to be valid and comparable to previous samples found in the literature.

Given that the purpose of this study was to test a cognitive intervention, there are several notable treatment implications which can be drawn from the study findings. As discussed in the introduction, while CBT currently addresses maladaptive sleep beliefs, unhelpful attributions for daytime fatigue is not a current target of treatment. However, the study findings demonstrated that misattributions of fatigue to poor sleep are amenable to change with a minimal psycho-educational intervention. In light of the findings in the literature which suggest that over-attributing fatigue to poor sleep can increase sleep-related anxiety and further perpetuate the insomnia cycle (Espie, 2002; Harvey, 2002; Lundh & Broman, 2000), this type of intervention may prove to be a helpful adjunct to CBT. Indeed, as there is little known about effective cognitive interventions for insomnia (Morin et al., 2006), these promising results provide one possible avenue toward successfully targeting and altering maladaptive cognitions in poor sleepers.

The lack of support for the secondary hypothesis also has important implications for the treatment of insomnia. Early research on insomnia treatments focused primarily on behavioural interventions, while cognitive therapy was kept on the sidelines, with the idea being that psycho-education was sufficient to bring about cognitive change in insomnia patients (see Harvey, 2005). Indeed, until recently, the only type of cognitive intervention included in CBT was in the format of education, whereby one session was used to alter maladaptive beliefs about sleep via educational information regarding sleep needs, the biological clock, and the effects of sleep loss on daily functioning (e.g., Edinger et al., 2001). However the non-significant interactions for the VAS ratings suggest that psycho-education (i.e., the information session used in this study), while may be effective in altering reported attributions and beliefs about sleep, was not sufficient to produce change in levels of fatigue, mood, and worry, relative to those who did not receive the

fatigue information. This is particularly problematic given that the modification of such mood states is an important secondary goal of many therapies.

In order to improve the intervention as it currently stands, additional treatment components are needed to supplement the psycho-education piece. Indeed, Harvey (2005) proposed that essential components of successful cognitive therapy for insomnia include Socratic questioning, guided discovery, behavioural experiments, assigning homework, and eliciting feedback. For example, behavioural experiments allow participants to actually test their maladaptive cognitions and associated predictions via real life behavioural trials. Likewise, doing homework provides patients with the opportunity to apply the strategies that they learn in session to their everyday lives. Both cognitive therapy techniques allow patients to challenge their cognitions in real-life situations, which is often more effective than challenging them verbally in session, as was done in the present study. Further, Harvey, along with others (e.g., Beck, 1995), states that the cornerstone of effective cognitive treatment is the therapeutic alliance, which is characterized by an active collaboration in therapy, including input from both the therapist and the client. Understandably, the brevity of the information session used in the present study precluded the opportunity to develop such a therapeutic alliance.

For optimal results, future research should expand this psycho-education information session in order to incorporate these other well-established cognitive treatment components, such as Socratic questioning, guided discovery and behavioural experiments. As an example of a behavioural experiment, participants can test whether exercise has an influence on their energy levels by rating their fatigue levels after exercise versus no exercise. Indeed, it will be interesting to see whether having participants playing a more active role, and catering this information to

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each individual participant, may increase the likelihood that the intervention produces changes in relevant mood states.

There are several strengths and limitations to this study. One notable strength of this study is its well-controlled experimental design. Given that participants were randomized to two groups, and no pre-existing differences were found, the most plausible explanation for post-treatment differences between each group was the information condition to which they were assigned. As such, the differences at post-intervention between each condition are likely directly attributable to the type of information that they received.

Another strength of this study is that participants had the opportunity to generate their own attributions for fatigue in the LAT. Whereas many self-report based studies require participants to select a response from several options, generating their own responses does not allow participants to be influenced by the responses provided on the page. It also compels participants to think about the question at hand rather than simply recognizing attributions that seem to align with their perceptions of their fatigue.

One could argue that attributions are simply an epiphenomenon of insomnia, such that these maladaptive beliefs are simple a consequence of the sleep problem and do not warrant treatment of their own. If this was the case, a study such as this might not be clinically useful, as targeting the sleep disturbance only should resolve any insomnia. However, there is some evidence in the literature that suggests that this may not be the case. Specifically, previous studies have demonstrated that maladaptive sleep beliefs improve with belief-targeted CBT for insomnia to a significantly greater extent than they do with pharmacotherapy (Morin, Blais, & Savard, 2002) or non-belief-targeted behavioural therapy (Carney & Edinger, 2006). Further, in the former study, both pharmacotherapy and CBT for insomnia produced equivalent

improvements in other indices of sleep quality, suggesting that improved sleep itself is alone not sufficient to produce changes in maladaptive sleep beliefs. In addition, both studies revealed that decreases in maladaptive sleep beliefs from pre- to post- treatment were associated with clinically relevant improvements in other sleep indices. Taken together, these findings suggest that unhelpful beliefs about sleep are likely not simply a byproduct of poor sleep, but warrant their own attention with respect to both research and treatment.

Admittedly, this study is somewhat limited in that it used a non-clinical sample of participants, consisting of undergraduate students. The use of an analog sample limits the generalizability of the study findings, as undergraduate students have specific features that distinguish themselves from that of clinical insomnia populations. For example, the gender distribution for clinical populations is estimated to be approximately 60% female (Hale et al., 2009; Zhang & Wing. 2006), whereas females in the current study comprised approximately 85% of the total sample. As well, clinical insomnia is a problem across the lifespan (Roth, 2007), and the use of undergraduate students precludes generalization of the results to older adults. However, as discussed above, this sample is comparable to other samples with respect to the internal consistency and the correlations among the self-report measures of insomnia, fatigue, depression and maladaptive sleep beliefs. Furthermore, the mean scores for the ISI, BDI-II and DBAS-16 in this sample were above the mild clinical cutoffs as per the guidelines suggested in the literature (Bastien et al., 2001; Beck et al., 1996; Carney et al., 2010). As such, while the demographic characteristics of the study sample may differ from that of a clinical population, the clinical features of this sample are somewhat similar to those found in clinical insomnia.

Given that the study sample consisted of an analog population, no formal diagnostic assessment of insomnia was used to determine whether study participants met criteria for clinical

insomnia. However, while this sample was not a clinical insomnia population per se, undergraduate students are a uniquely vulnerable population, particularly to the development of insomnia (Coren, 1994; Jensen, 2003). Studies have shown that increased stress levels among college students coupled with their highly irregular schedules can have a negative impact on sleep (Carney, Edinger, Meyer, Lindman, & Istre, 2006; Verlander, Benedict, & Hanson, 1999). Indeed, undergraduate students' sleep habits are among the first daily habits to change after beginning college (Pilcher et al., 1997). Furthermore, while there was no formal assessment of insomnia, we selected only those above a clinical cutoff on an insomnia beliefs questionnaire (DBAS-16). As such, the participants selected for the main analyses in this study were particularly vulnerable to the development of sleep disturbances. Indeed, previous studies have shown that scores on the DBAS-16 are able to distinguish good from poor sleepers (e.g., Morin et al., 1993). Finally, a large proportion of the sample (i.e., 74%) had ISI scores above the recommended clinical cutoff, which suggests that this sample was particularly prone to sleep disturbances. Nevertheless, while efforts were taken to select a study sample with increased vulnerability to insomnia, future research should test subsequent attribution-based interventions on insomnia-diagnosed clinical populations.

Insomnia is highly comorbid with other disorders, such as depression, anxiety and substance abuse (Ohayon, Caulet, & Lemoine, 1998), with estimates of psychiatric comorbidity at approximately 40% (Ford & Kamerow, 1989; Roth, 2007). Thus, the fact that there was no formal assessment of comorbid psychiatric disorders could be construed as a potential limitation. In this sense, it is difficult to know the extent to which these results would generalize to those with or without comorbid psychiatric problems, because the distribution of comorbid psychiatric issues in this sample is unknown. Likewise, there was also no formal diagnostic assessment of

other comorbid sleep disorders. Thus it is not known whether an occult sleep disorder, such as a circadian rhythm disorder, could account for the findings. However, research has shown that insomnia is the most frequent sleep disorder found among college students (Giesecke, 1987; Jensen, 2003). Indeed, a study on the prevalence of sleep disorders among young adults found that the rate of insomnia was substantially higher than hypersomnia, a disorder characterized by daytime sleepiness (Breslau, Roth, Rosenthal, & Andreski, 1999). Other research has found that the prevalence of disorders such as obstructive sleep apnea and certain circadian rhythm disorders are more common in middle-age groups relative to college populations (Guilleminault & Bassiri, 2005; Sharma & Feinsliver, 2009). As such, sleep disturbances found among college students are more likely due to insomnia than they are to these other occult sleep disorders.

Nevertheless, future studies should include a more formal diagnostic assessment for sleep disorders and comorbid conditions to determine the extent to which these results would generalize to complex clinical groups. Specifically, with the use of formal assessment tools such as the Duke Structured Interview for Sleep Disorders (DSISD: Edinger, Lineberger, Loiselle, Wohlgemuth, & Means, 2004) and the Structured Clinical Interview for the DSM Axis I Disorders (SCID-I: Spitzer, Williams, Gibbons, & First, 1996), patients can be tested pre- and post-intervention to determine changes in both clinical insomnia as well as comorbid Axis I disorders.

While the use of an undergraduate population can be construed as a potential limitation to this study, there are several reasons why an undergraduate sample was indeed suitable for this type of research. It was appropriate to enlist an analog population given that this study is the first step in a series of untested research questions. In this sense, this was the first study to test an attribution-based cognitive intervention, and thus it was important to see whether sleep-related

attributions were, at minimum, amenable to change, before testing this intervention within insomnia populations. Indeed, the first step of research is often not to establish external validity, but instead to determine whether something *can* happen (Mook, 1983). To this end, establishing that reports of fatigue attributions can change with psycho-education will allow future research to move beyond this first stage, and examine whether this can be replicated in clinical populations.

Another potential limitation of this study was the brevity of the experiment. Given that this study was completed in a one-hour session, there was only a short span of time between completing the pre- and post-intervention outcome measures. Perhaps we would have received different results if people were tested across a longer span of time. In this sense, a longer testing interval may have permitted greater consolidation of the material. The brief nature of the study also precluded the use of a more inclusive information session, which could have included other cognitive therapy components. Finally, given that an integral goal of this research is to reduce insomnia symptoms, a longer testing interval would allow for assessment of pre- to post-changes in relevant sleep indices.

In a similar vein, some may think that the short time span may have increased the likelihood of participants remembering the responses they originally indicated in the pre-intervention LAT and VAS, or the information contained in FI. While this may be the case, the outcome measures used in this study were designed in a manner in which would decrease the likelihood of participants remembering exactly what they wrote pre-intervention. For example, VAS's were chosen as the measure of mood change because they instructed participants to place an X along a continuum. This type of scale would make it less likely that they would remember their exact placement of the X in the pre-information VAS, as compared to selecting a number

from 1 to 10 or 1 to 100. Similarly, the LAT had three different components, whereby participants were instructed to list possible attributions for their fatigue, rank the frequency of the attributions, and indicate the likelihood that each of them accounts for their fatigue. Given that this task was relatively complex and incorporated multiple components, it is unlikely that participants were able to recall their exact responses from the pre-intervention measures.

It is also possible that demand characteristics played a role in FI participants' post-intervention responses on the LAT. During this study, FI participants first generated reasons for their fatigue during the day, they were then educated about the non-sleep-related attributions, and finally their causal attributions for fatigue were re-tested. Given the nature of this pre-post intervention design, participant responses may have been influenced by their perception of what the study investigator was looking for. That is, they may have speculated that the purpose of the study was to identify an increase in non-sleep-related attributions after being taught them in the intervention. Indeed, previous studies examining levels of maladaptive sleep beliefs before and after psycho-education also acknowledged the possibility that demand characteristics played a role in participants decrease in such beliefs (e.g., Carney & Edinger, 2006). With regard to demand characteristics and remembering information in the FI, it is important to note that the LAT specifically instructed the participants to focus on themselves when listing their fatigue attributions. That is, the participants were told to list possible attributions related to their personal experiences with fatigue, rather than people's fatigue in general. Furthermore, participants were also asked to rank the frequency and rate how likely each factor accounts for their fatigue, further personalizing the LAT measure. Looking at this issue from a different perspective, the primary objective of the intervention was to inform participants of the many causes of fatigue, and have participants consider these factors as potential causes of *their* daytime fatigue. As such,

participants who did in fact use the causes contained in the information session as their own attributions may have been internalizing these factors as causes of their own fatigue. Indeed, increased consideration for these non-sleep-related causes of fatigue was the ultimate objective of the intervention.

Taken together, future research should consider running this experiment over a longer span of time, to allow for deeper consolidation of the material. A longitudinal study would also allow for the development of a more potent and inclusive cognitive intervention with the addition of the supplementary cognitive techniques listed above. Indeed, an important end-goal of this research is to establish whether this intervention can produce both cognitive and mood change, as well as improve sleep, in the long-term.

Certainly, future studies should examine whether targeting attributions via cognitive strategies would be effective in clinical insomnia populations. Given that this intervention is geared to individuals with clinical insomnia, further research needs to establish whether this intervention can, in fact, produce significant changes within such a clinical sample. Ultimately, cognitive change should produce sleep improvement, so future studies could move beyond the self-report outcome measures used in this study and prospectively monitor sleep to see whether there are pre- to post-intervention improvements in relevant sleep indices.

Given that insomnia is the number one rated health problem facing people (Canals, Domenech, Carbajo & Blade, 1997), maximizing the effectiveness of interventions is critically important work. With respect to future research in this area, it would be helpful to know if an attribution-based intervention could increase treatment response rates if it were incorporated into the more general cognitive therapy for insomnia. As noted previously, while CBT for insomnia is efficacious (Morin et al., 2006), there is room for improvement. As such, establishing whether

the inclusion of attribution-targeted interventions into CBT can improve treatment response rates is an important and long-term goal of this research. To this end, the promising findings of the present study revealed that people can learn to broaden their scope of fatigue attributions via a minimal cognitive-based intervention. Given such encouraging findings, it is exciting to consider the improvements that could be made with a more expanded and inclusive cognitive intervention. In establishing that these cognitions are amenable to change, this study's findings are both important and necessary in order to inform future research whose end-goal is to improve treatment response and refine our treatments for insomnia.

List of Attributions Task (LAT)

What tends to account for you feeling tired? First, list below all factors that you think could account for you feeling tired. Please do not list "other." Next, please rank these factors in order of frequency, with #1 indicating the factor that most often accounts for your fatigue. Finally, next to each factor, please indicate the likelihood out of 100% that you think each factor accounts for your fatigue, with allocating a greater proportion to those factors that are considered to be more important in contributing to your fatigue.

For example, Factor A accounts for my fatigue most often so it is rated #1 and the likelihood that it accounts for my fatigue is 80%. Factor B occurs less often so I rank it #2 and its likelihood is 60%.

Example:

What factors account for my feeling tired?	Rank in order of frequency	Likelihood (0 – 100%) that this factor accounts for you feeling tired?
Factor A	*#1*	*80%*
Factor B	*#2*	*60%*

Please fill in your response below:

What factors account for my feeling tired?	Rank in order of frequency	Likelihood (0 – 100%) that this factor accounts for you feeling tired?

Visual Analogue Scales (VAS)

Please mark an "X" on the line below to indicate how you are currently feeling. Use the labels below the line to help you in your judgment.

1.   How fatigued do you feel?

_____

    Not at all   Extremely
     fatigued   fatigued

2.   How positive is your mood?

_____

    Not at all   Extremely
     positive   positive

3.   How worried do you feel?

_____

    Not at all   Extremely
     worried   worried

Fatigue Information (FI) Intervention

There are many reasons other than poor sleep that account for feeling tired. The list below contains the most common factors implicated in feeling tired. Please note that these factors may or may not apply to you.

- Caffeine rebound – Sometimes caffeine can have the opposite effect and cause you to be more fatigued. While a caffeinated beverage like an energy drink, pop, tea or coffee may initially make you feel alert, it will subsequently lead to a drop in energy and in some cases, an experience described as a "crash".

- Post lunch changes in body temperature – Your level of alertness is controlled in part by our biological clock. Your body temperature naturally rises and falls over the course of a 24-hour day and you are most sleepy when temperatures are falling at night. One other time the body temperature falls is during a brief dip in temperature usually sometime between 12 PM and 3 PM. It is temporary but people experience a dip in their energy levels during this time everyday.

- Inactivity – When you engage in physical activity, your metabolism speeds up and your body releases endorphins, both of which give you more energy. As such while exercising is a strenuous activity, it makes you feel less tired in the long run.

- Overactivity/physical exertion – Fatigue can also result from overextending yourself and engaging in too much physical activity. This can lead to feelings of exhaustion and as a result your body will not have sufficient resources to carry on with your day.

- Diet – We need appropriate nutrients from foods in order to maintain energy in our bodies. Following certain diets, such as those low in carbohydrates, can deprive your body of vital nutrients and fuel that you need to get through the day.

- Dehydration— Given that our bodies are more water than anything else, we need to keep them replenished by drinking water. Not replenishing your body's fluids can reduce your energy level, causing you to feel fatigued.

- Depression/Low Mood – Fatigue and low energy are common symptoms of depression. When you feel down, you are less activated and thus more likely to feel fatigued.

- Pain – The physical and emotional energy your body uses to deal with pain can cause you to feel fatigued.

- Anxiety/Stress – People who are overly anxious or stressed keep their bodies in overdrive. The body often uses adrenaline to deal with anxiety and stresses, causing fatigue to set in.

- Boredom – Engaging in mundane activities can lead to fatigue, as your brain is not being stimulated. For example, staring at a computer screen or doing repetitive work without frequent breaks can cause eye strain, mental and physical fatigue.

- Constipation – When you are constipated, toxins in your body are not being eliminated properly. The toxins can thus build up and demand more of your body's energy to process and store.

- Iron levels (anemia) – Anemia often results in decrease oxygen delivered to the heart and other vital organs throughout the body. This can drain your energy and thus cause fatigue.

- Infections – Fatigue can be brought on by various infections such as the flu, HIV infections and food allergies. Your body uses a lot of energy to fight these infections, thus leaving you feeling fatigued.

- Hypothyroidism - An underactive thyroid can cause fatigue, as the thyroid controls your metabolism, the speed at which your body operates.

Appendix D

Sleep Control Information Intervention

The following is some information about sleep

- Purpose of sleep – While we know that sleep is important, the exact purpose of sleep is not known. Some possible reasons why sleep is needed is so our bodies can restore and repair themselves or so that they can maintain a constant body temperature.

- Stage 1 sleep – This is a very light stage of sleep that takes up only about 5% of the night's sleep. If you were awakened from this stage of sleep, you would likely believe that you were not asleep at all. This stage is a segue to the deeper stages of sleep

- Stage 2 sleep – Stage 2 is somewhat deeper than stage 1 and takes up about half of a night's sleep. It is more difficult to wake a person from this stage of sleep and people tend to report being asleep when they are awoken.

- Delta sleep – This is the deepest stage of sleep, which takes up approximately 10 – 20% of your night's sleep. During this stage, the body is restoring and rebuilding itself. It is most difficult to wake a person from delta sleep.

- Rapid Eye Movement (REM) – REM sleep takes up about 20 – 25% of the night's sleep and is the stage during which most dreaming takes place. There is also increased bloodflow to the brain during this stage, and this activity is very similar to that seen during wakefulness. As a result this is a very light stage of sleep.

- Sleep Architecture – Represents the structure of sleep throughout the night. Sleep cycles progresses through light sleep, followed by deep sleep and finally a REM period. The sleep cycle takes approximately 90 minutes, and we usually have 4 – 6 cycles per night. Most deep sleep occurs in the first cycle. REM sleep tends to predominate towards the second half of the night. This is why some dreams may seem very vivid when people wake up.

- Prevalence of Sleep Problems – Sleep problems are quite common. Approximately 9 – 10% of people in Canada have problems falling or staying asleep. Very few of these people get treatment for their sleep.

Some basic good habits for improving sleep

- Bedtime snack – Having a light snack before bed may be beneficial for sleep. Foods such as milk, cheese or peanut butter contain chemicals that your body uses to bring on sleep.

- Sleep environment – In order to have a good night sleep, try to ensure your bedroom is quiet and dark.

- Temperature - Make sure the temperature in your bedroom is comfortable, preferably not much higher than 75 degrees Fahrenheit.

Consent Agreement

Title of Study: Attributions for Fatigue

You are being asked to participate in a research study. Before you give your consent to be a research volunteer, it is important that you read the following information to be sure that you understand what you will be asked to do.

<u>Investigators:</u>
Andrea Harris, B.A. Graduate Student (Supervisor: Dr. Carney), Department of Psychology, Ryerson University
Colleen Carney, Ph.D. Assistant Professor, Department of Psychology, Ryerson University

<u>Purpose of the Study:</u>
Insomnia refers to a difficulty falling asleep, staying asleep or waking too early from sleep. In order to better understand and help people with insomnia, we are conducting an investigation of the experience of feeling tired, or fatigued, during the day. In particular, we are interested in understanding what you believe causes you to feel tired during the day. A maximum of 300 people will be asked to participate for this study. Participants will be volunteers from undergraduate introductory psychology courses at Ryerson University.

<u>Description of the Study</u>:
You will be asked to complete a study consisting of questionnaires and information regarding sleep. The completion of the study will take approximately 1 hour. The procedures in this study will include:
1. Completing questionnaires that ask about your sleep habits, fatigue and mood
2. Filling out a form that asks you about what causes your fatigue
3. Reading information regarding the daytime and nighttime experience of sleep
4. Answering some more questions about fatigue and mood
5. Reading a Study Conclusion form that contains a thank you for participating, a summary of information about the study and contact information if you have any further questions

<u>What is Experimental in this Study:</u>
We are not testing any interventions--the only experimental aspect of this study is whether or not people's perspective change based on reading information about the daytime and nighttime experience of sleep.

<u>Risks or Discomforts:</u>
One risk of this study is that some of the measures you will be asked to complete might be perceived as revealing "personal" information, so you could feel uncomfortable. However, these brief measures ask about basic symptoms and behaviors, not emotionally sensitive or personally relevant history. If you feel uncomfortable at any point throughout the study, you may discontinue participation and still get the credit. Also, all studies carry the risk of an accidental breach of confidentiality. We have a number or procedures in place to protect you from such an occurrence. Instead of using your name or any identifying information, we will assign a study number so that your data is not identifiable (i.e., not linked to your name).

<u>Benefits of the Study:</u>

Participating in this study will give you a chance to see what is involved with clinical research. This research will contribute to science and society as it will allow us to identify potential factors associated with insomnia, thereby enabling us to better understand and treat the disorder.

Confidentiality:
In order to protect your confidentiality, we will assign you a distinct research code number and use this code number rather than your name on study-related data. When your survey responses are transferred to an electronic database for use in the planned analyses, these data sets will include only your research code numbers as identifiers. The database will be password protected. No names or other unique identifiers will be included in any of the data sets used in the analyses of this project. Confidentiality will be maintained to the extent of the law for 10 years.

Incentives to Participate:
You will be completing this study in partial fulfillment of your course requirement for introductory psychology, as you will receive one percent toward your final grade. If you decide not to complete the study, you will still be granted the credit. No financial compensation will be offered to participate in this study.

Voluntary Nature of Participation:
Participation in this study is voluntary. Your choice of whether or not to participate will not influence your future relations with Ryerson University. If you decide to participate, you are free to withdraw your consent and to stop your participation at any time without penalty or loss of benefits to which you are allowed.

At any particular point in the study, you may refuse to answer any particular question or stop participation altogether. If you decide not to answer a particular question, you can simply leave it blank.

Questions about the Study:
If you have questions about the research, you may contact:

Dr. Colleen Carney (416) 979-5000 ext. 2177

If you have questions regarding your rights as a human subject and participant in this study, you may contact the Ryerson University Research Ethics Board for information.

Alexander Karabanow
Research Ethics Board
c/o Office of the Vice President, Research and Innovation
350 Victoria Street, Ryerson University
Toronto, ON M5B 2K3
416-979-5000 Ext. 7112
Email: alex.karabanow@ryerson.ca

Agreement:

Your signature below indicates that you have read the information in this agreement and have had a chance to ask any questions you have about the study. Your signature also indicates that you agree to be in the study and have been told that you can change your mind and withdraw your consent to participate at any time. You have been given a copy of this agreement.

You have been told that by signing this consent agreement you are not giving up any of your legal rights.

_____
Name of Participant (please print)

_____   _____
Signature of Participant   Date

_____   _____
Signature of Investigator   Date

Appendix F

Debriefing Form

Thank you very much for participating in our study. We are interested in what people think about fatigue and what causes fatigue. People who sleep poorly tend to focus solely on poor sleep as an explanation for their fatigue when there are actually many other possible causes of fatigue, such as low mood, boredom and lack of physical activity. If a person thinks that the only reason they feel tired during the day is because they slept poorly, they begin to feel increasingly pressured to sleep well. The more pressure there is to sleep, the more likely it is that the idea of sleep will produce anxiety or tension (Broomfield & Espie, 2005; Harvey, 2002)

The purpose of this study is to determine if providing information about fatigue would increase the likelihood that non-sleep related fatigue factors would be considered as an explanation for fatigue and if this would decrease anxiety. Half of the people participating in this study received information about fatigue and the other half received information about sleep. We will examine if the fatigue-information group changed their mind about the importance of other causal factors for fatigue or whether their opinions and mood remained the same. For those in the fatigue-information group, please note that the information about fatigue contained a variety of possible explanations for being tired, which may or may not apply to you.

Once again, we would like to thank you very much for your participation. If you are interested in further information, you are encouraged to take a look at the references provided below. Finally, please feel free to contact us if you have any further questions pertaining to this research.

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Appendix G

Depression Debriefing Form

Sometimes people who have difficulty with sleeping also have difficulties with depression. Please note that our study methods do not provide diagnostic information regarding depression. However, if you would like more information about depression, we have included this information below as well as resources for where to find help.

Here are some common depression signs:
ð   You feel sad, empty, down, agitated, angry on most days for at least 2 weeks
ð   Over the past few weeks, you have had difficulty enjoying things that you normally like to do
ð   Over the past few weeks, you have had trouble getting interested in things, or persistent troubles with motivation

If any of these problems have been present most of the last two weeks or more, you should be evaluated for depression.   Below are some other "signs" of depression that often accompany one or more of the symptoms described above (e.g., low mood, difficulty enjoying things or motivational problems):
ð   You have been withdrawing from people you would normally enjoy being around
ð   You feel tired much of the time on most days
ð   You are feeling bad about yourself, thinking that you are worthless, struggling with poor self-esteem or having self-critical thoughts that are more than usual
ð   You are struggling with guilty thoughts, or thoughts relating to feeling "punished" in some way
ð   You have had an increase or decrease either in your appetite or weight. Any decreases in your weight should not be due to a weight loss program (e.g., the weight loss should be unintentional).
ð   People notice that you are either: 1) moving/talking unusually slow, or 2) moving/talking unusually fast.
ð   You have more difficulty making decision than usual
ð   You have greater difficulty concentrating or remembering things than usual

If these signs of depression apply to you and/or you would like a referral for evaluation, please do not hesitate to contact Dr. Colleen Carney (416) 979-5000 ext. 2177 for assistance.

If you would prefer to arrange an evaluation privately, please consider the Ryerson's Centre for Development and Counseling. They are located on the Lower Ground of Jorgenson Hall in JOR-07C and can be reached by telephone at (416) 979-5195 during their regular office hours, or a message can be left after hours and a receptionist will return your call on the next business day. Regular operating hours throughout the year are Monday through Friday from 9:00am to 5:00pm. There are no fees for their service.

If you are having any thoughts or images relating to harming yourself, for example, "My family would be better off if I were dead," or picturing yourself driving off the road, you should immediately present for evaluation. It is important to seek help in this situation to protect yourself from harm. If you are having thoughts of harming yourself, call 911 or go to the nearest emergency room and ask to speak with the psychiatrist on call.

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