AN EXAMINATION OF SOCIAL RHYTHMS IN A CLINICAL INSOMNIA POPULATION AND GOOD SLEEPER COMPARISON GROUP

by

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Abstract

An Examination of Social Rhythms in a Clinical Insomnia Population and Good Sleeper Comparison Group

Doctor of Philosophy, Fall 2014

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Insomnia had generally been conceptualized as a nighttime disorder, while the daytime experience of insomnia has been largely ignored. However, there are several lines of research suggesting daytime experiences as well as daytime behaviours are equally important. For example, daily behavioural routines commonly referred to as social rhythms (e.g., exercise, attendance of school or work, recreation, engagement in social activities) have been identified as potential zeitgebers (i.e., time cues that help to regulate the biological clock). Previous research has shown that regulating behavioural zeitgebers may have promising benefits for sleep. As such, this study examined the daytime activities in a clinical insomnia population and a good sleeper comparison group. Participants ($N = 69$) prospectively monitored their sleep and daily activities for a two-week period, while wearing a wrist actiwatch. Those with insomnia appear to engage in activities characterized by significantly less regularity than good sleepers. However, those with insomnia were found to engage in similar levels of daily activities compared to good sleepers. Findings from this study highlight the relative importance of daytime activities on this supposed nighttime process. Accordingly, future research would benefit from testing treatment components that focus on regulating daytime activities, which would likely improve treatment outcomes.

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Dedication

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CHAPTER ONE

INTRODUCTION

1.1 Statement of Problem

Insomnia is a prevalent public health concern (Ford & Kamerow, 1989; Morin, LeBlanc, Daley, Gregoire, & Merette, 2006a; Ohayon, 2002) and, although the consequences of insomnia have been previously under recognized, it is now known that it is a disorder that is associated with substantial personal and societal costs (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009). On a personal level, these costs include reduced quality of life, increased healthcare utilization, increased use of medications, increased absenteeism, decreased work productivity and increased traffic and work-related accidents (Ozminkowski, Wang, & Walsh, 2007; Stoller, 1994). Furthermore, even after controlling for age, gender, the use of prescription medication, depression, and visual or mobility impairments, insomnia is linked to serious falls in older adults (Brassington, King, & Bliwise, 2000). The annual economic cost of insomnia for Canadians is estimated to be $970.6 million for insomnia-related absenteeism and $5.0 billion for decreased worked productivity (Daley et al., 2009).

Insomnia negatively affects the functioning, health status, and quality of lives of millions of individuals worldwide (Edinger & Means, 2005; Zammit, Weiner, Damato, Sillup, & McMillan, 1999). It is a condition that is characterized by complaints of poor sleep quality, insufficient sleep quantity, or nonrestorative sleep, accompanied by daytime impairment that is attributed to the sleep difficulty (American Psychiatric Association, 2000). The development of chronic insomnia is largely dependent on predisposing, precipitating, and perpetuating factors (Spielman, Caruso, & Glovinsky, 1987). A predisposing factor is a factor that increases an individual's vulnerability to sleep difficulties. Once predisposed to this difficulty, a precipitating

factor can initiate the onset of acute insomnia. Perpetuating factors, such as coping strategies that develop in response to the sleep difficulty, can contribute to the development of chronic insomnia. The perpetuating factors can have negative consequences for the sleep regulatory processes (Bootzin, 1972a; Harvey, 2002), which work to maintain the insomnia. These sleep regulatory processes include the following: 1) the homeostatic process; 2) the circadian process; and 3) the arousal system (Borbély & Achermann, 1999; Saper, Cano, & Scammell, 2005).

Given that insomnia is a prevalent disorder with large personal and societal consequences, it warrants timely, effective, and enduring treatment. Currently, Cognitive Behavioural Therapy for insomnia (CBT-I) is the recommended gold-standard treatment in the management of this disorder (Morgenthaler et al., 2006; Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Efficacy studies suggest that approximately 70-80% of insomnia patients achieve a therapeutic response with CBT-I and about 40% achieve full clinical remission (Morin, 2010; Morin et al., 2006b; Morin et al., 1999). However, there is a small subset of insomnia patients for whom CBT-I does not work (Harvey & Tang, 2003; Morin, Culbert, & Schwartz, 1994). Accordingly, research aimed at improving treatment outcomes for this disorder is needed.

Insomnia has largely been conceptualized as a nighttime disorder. As such, insomnia treatment mainly focuses on modifying nighttime behaviours (e.g., restricting time in bed, asking clients to get out of bed when unable to sleep). Despite the inclusion of daytime sequelae as part of research diagnostic criteria (e.g., Edinger et al., 2004), to date, the daytime experience of insomnia has been largely ignored. However, it should be noted that this conceptualization has begun to shift and the Sleep Disorders section in the new Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) has been renamed Sleep-Wake Disorders. Understanding and incorporating daytime treatment targets may be one way to

improve our current treatments. More specifically, it may be helpful to gain a deeper understanding of the way in which daytime environmental and behavioural factors interact with the sleep regulatory system. For example, it is well documented that consistent and high quality sleep is contingent on a strong endogenous circadian rhythm (Monk, Reynolds, Buysse, DeGrazia, & Kupfer, 2003; Reid & Zee, 2005) and that environmental zeitgebers (i.e., cues that helps to regulate the biological clock, such as daylight and darkness) influence circadian regulation and entrainment (Czeisler, Buxton, & Khalsa, 2005; Monk, Flaherty, Frank, Hoskinson, & Kupfer, 1990). Daily behavioural routines, referred to as "social rhythms," such as exercise, attendance of school or work, recreation, and engagement in social activities have been identified as potential zeitgebers (Carney, Edinger, Meyer, Lindman, & Istre, 2006a; Monk et al., 1990; Monk, Kupfer, Frank, & Ritenour, 1991; Monk, Petrie, Hayes, & Kupfer, 1994; Monk, Reynolds, Machen, & Kupfer, 1992; Monk et al., 2003). Optimally, these social rhythms would act in synchrony with environmental and physiologic zeitgebers to ensure appropriate circadian entrainment and sufficient sleep. However, these social rhythms can conflict with, and disrupt, the regular circadian entrainment, which can lead to disturbed sleep. To date, social rhythms have been investigated in analog good and poor sleepers. Poor sleepers exhibited a lower frequency and less regularity of daily activities relative to good sleepers, with good sleepers participating in more regular activities with increased active social engagement (Carney et al., 2006a). Accordingly, these findings demonstrate that regulating zeitgebers and engaging in activities with other people may have promising benefits for sleep.

1.2 Purpose of the Present Study

Previous findings have suggested that frequent and consistent social rhythms have a beneficial effect on sleep in an undergraduate sample (Carney et al., 2006a). However, it is

important to examine these daytime activities in a clinical population with a wider age range, as these findings may not generalize across the age range and to those with more severe levels of insomnia. More importantly, those with clinical levels of insomnia are the individuals who would benefit from potential enhancements of CBT-I. The current study examined the influence of overall activity levels (including the amount, timing, and regularity) on sleep in a clinical insomnia population, and in a good sleeper comparison group. All participants prospectively monitored their sleep and daily activities for a two-week period, in addition to wearing a wrist actiwatch, a tool that provides an indirect but objective measure of sleep/wake activity. It was hypothesized that people with insomnia would engage in fewer activities and have more irregular daytime schedules, compared to good sleepers. This study provides sleep researchers and clinicians with information on the relative importance of daytime activities on this supposed nighttime process. Accordingly, based on these findings, CBT-I could test treatment components that focus on increasing and regulating daytime activities. Such enhancements may improve treatment outcomes, and decrease the overall burden of insomnia on society.

CHAPTER TWO

LITERATURE REVIEW

2.1 A Review of the Problem

Insomnia, a chronic and reoccurring condition (Hohagen et al., 1994), is a highly prevalent disorder affecting approximately 10 – 15% of the adult population (Ford & Kamerow, 1989; Morin et al., 2006a; Ohayon, 2002), with approximately 30% of the population suffering from insomnia symptoms at any given moment (Morin et al., 2006a). However, these figures are likely an underestimate of the actual prevalence. The diagnostic criteria for Insomnia Disorder require that a sleep complaint (e.g., sleep onset or maintenance difficulties, or nonrestorative sleep) occurs a minimum of three nights per week and is present for at least three months (American Psychiatric Association, 2013). However, there are no conventional cutoffs determined for how to quantify disrupted sleep in order to receive this diagnosis. A commonly used convention is that the time spent awake at the beginning of the sleep period or in the middle of the night is greater than 31 minutes and that these onset or maintenance difficulties occurs three or more nights per week (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). However, the DSM-5 (American Psychiatric Association, 2013) has not yet adopted these specific guidelines. Perhaps it is difficult to quantity insomnia as there is wide variability in insomnia complaints and, also, wide variation in the relation between quantitative values and other indices of morbidity (Lineberger, Carney, Edinger, & Means, 2006). For example, Lineberger et al. (2006) found that individuals with one night of severe sleep disturbance per week (e.g., spending three hours awake in bed once per week), approximates the impact of someone with more frequent but mild quantitative sleep disturbance (e.g., taking only 30 minutes to fall asleep nightly). Dissimilar to other sleep disorders (e.g., sleep-disordered breathing, periodic limb

movement disorder), insomnia is a subjective condition, in which a diagnosis is made from clinical interview and self-reported tools (American Sleep Disorders Association, 1995).

Aside from a subjective sleep complaint, the diagnostic criteria requires the disturbance to cause clinically significant distress or impairment in social, occupational, or other areas of functioning. This criterion generally includes one or more of the daytime symptoms of insomnia listed in the Research Diagnostic Criteria for Insomnia (Edinger et al., 2004a), including fatigue/malaise, attention/concentration problems, negative mood, social/vocational dysfunction or poor school performance, somatic symptoms such as tension headaches and/or gastrointestinal symptoms in response to sleep loss, motivation/energy/initiative reduction, daytime sleepiness, and/or worry about sleep. Thus, even though insomnia is often conceptualized as a nighttime disorder, it is in fact best characterized as a 24-hour problem.

Finally, diagnostic exclusions include another sleep disorder (e.g., circadian rhythm disorder, breathing-related sleep disorder), or another mental disorder (e.g., major depressive disorder) that could account for the insomnia. Additionally, the insomnia cannot be due to the physiologic effect of a substance (e.g., medication or illicit drugs) or a general medical condition. An insomnia diagnosis can and should be made in the presence of another mental, medical, or sleep disorder, if there is a prominent insomnia complaint that causes distress or impaired functioning.

Considering that insomnia is listed as symptom for over 20 conditions in the DSM-IV-TR (American Psychiatric Association, 2000), it is frequently viewed as a symptom, rather than a comorbid condition. For example, in a survey of general practitioners, the most common symptom used to identify major depressive disorder (MDD) was the presence of a sleep complaint or insomnia (Krupinski & Tiller, 2001). However, when reassessed, only 28% of those

diagnosed with MDD actually met DSM-IV-TR criteria for this condition; the remaining patients had insomnia and were incorrectly diagnosed. Consequently, insomnia continues to be under diagnosed, which contributes to an underestimation of prevalence rates. Furthermore, insomnia complaints are often viewed as trivial and incorrectly believed to dissipate after the primary condition is treated, despite studies that contradict the belief. For example, over half of those with comorbid insomnia still have insomnia after successful treatment of the so-called primary disorder (i.e., depression; Carney, Harris, Friedman, & Segal, 2011a; Carney, Segal, Edinger, & Krystal, 2007); thus insomnia is more than a mere symptom.

Insomnia most often presents as comorbid disorder, particularly with mood and anxiety disorders. Comorbidity in insomnia appears to be the rule rather than the exception, as one study found that only one-quarter of individuals with insomnia did not meet criteria for a comorbid psychiatric disorder (Bixler, Vgontzas, Lin, Vela-Bueno, & Kales, 2002). Moreover, insomnia symptoms often precede the onset of another psychiatric condition and can be a risk factor for the later development of another psychiatric disorder (Harvey, 2001). Ford and Kamerow (1989) found that individuals with insomnia symptoms were 3 times more likely to develop an anxiety disorder and 35 times more likely to develop major depression in the following year, compared to good sleepers. Thus, not only is insomnia a disorder that needs to be recognized, it could be a marker for the future onset of psychiatric disorders, which has substantial clinical implications in terms of prevention.

2.2 Sleep Processes

In order to understand insomnia, it is helpful to consider how sleep is regulated in a non-disease context. There are three distinct processes that underlie sleep regulation. Borbély and colleagues (Achermann & Borbély, 2003; Borbély & Tobler, 1985) have proposed a model for

sleep regulation that includes a homeostatic process (sleep drive, process S) and a circadian drive (process C). The homeostatic process accumulates during wakefulness and diminishes during sleep. Simultaneously, a circadian process takes place, which is a clock-like mechanism that defines the interchange of periods with high and low sleep propensity. Borbély et al. (1999; 1985) hypothesized that the circadian propensity for sleep increases during the sleep state, ensuring sustained sleep, despite a lessoning of the homeostatic need for it near the end of the sleep cycle. Conversely, the circadian waking drive accumulates throughout the day and is at its highest during the hours just prior to sleep, when the homeostatic sleep drive is nearing its peak. Finally, whereas the circadian and homeostatic systems regulate sleep, these 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 (Borbély & Achermann, 1999; Saper et al., 2005). Considering the importance of these sleep regulatory processes, they will now be reviewed in detail.

2.2.1 Homeostatic Regulation.

Sleep homeostasis refers to the sleep/wake dependent component of sleep regulation in which homeostatic mechanisms work to stabilize deviations from a typical reference level of sleep. This process reflects the propensity for sleep (i.e., sleep drive) when sleep is reduced or lacking by building "pressure" to sleep, which accumulates throughout wakefulness (i.e., sleep debt) and in reaction to a surplus of sleep, this process acts to decrease sleep propensity (Achermann, 2004; Taillard, Philip, Coste, Sagaspe, & Bioulac, 2003). Thus, this sleep drive rises during wakefulness and declines during sleep. Sleep consists of two distinct phases: rapid eye movement (REM) sleep and nonREM (NREM) sleep, both of which can be identified using

polysomnography (Dijk, 2009). Rechtschaffen and Kales (1968) classified NREM sleep into stages 1, 2, 3, and 4. Stage 1 and 2 are considered light sleep and stages 3 and 4 are considered deep sleep, also referred to as slow wave sleep (SWS; Dijk, 2009). During NREM sleep, sleep spindles with changes in amplitude of 12-14 Hz oscillations can be observed on the electroencephalogram (EEG). Additionally, K complexes, lasting a minimum of 0.5 seconds and consisting of a negative sharp wave followed by a positive component and slow waves or delta waves, with slow frequency (< 2 Hz) and high amplitude (> 75 μV) can also be observed on the EEG (Dijk, 2009). Sleep spindles are present in sleep stages 2-4, but are more prevalent in stage 2 in contrast to stage 3 and 4 in which slow waves dominate in the EEG (Dijk, Hayes, & Czeisler, 1993). Accordingly, slow waves in the EEG acts as a physiological marker of sleep homeostasis in NREM sleep. Researchers have observed a high prevalence of slow waves in the early part of sleep, with a gradual decline throughout the night (Blake & Gerard, 1937; Dement & Kleitman, 1957; Webb & Agnew, 1971). These findings support the homeostatic properties of the sleep drive, such that at the beginning of the night, when the drive is the highest, slow waves dominate the sleep period and these slow waves diminish as the drive is fulfilled.

SWS is considered to be the most restorative sleep stage, associated with both sleep quality and sleep maintenance (Dijk, 2006). SWS and slow wave activity are associated with variations in endocrine and cardiovascular parameters (Dijk, 2009). For instance, the rate of human growth hormone secretion is at its highest during SWS (Van Cauter et al., 2004) and the autonomic nervous system shifts from a sympathetic to a parasympathetic dominance during SWS (Brandenberger, Ehrhart, Piquard, & Simon, 2001; Viola, James, Archer, & Dijk, 2008). Experimental disruption of SWS leads to an increase in shallow sleep and sleep fragmentation, intensifies daytime sleep propensity, and may interfere with daytime functioning (Dijk, 2009).

Sleep deprivation studies have demonstrated that homeostatic regulatory mechanisms are involved in the organization of a sleep episode. Specifically, partial or total sleep deprivation has been found to increase SWS in the recovery night (Borbély, Baumann, Brandeis, Strauch, & Lehmann, 1981; Williams, Hammack, Daly, Dement, & Lubin, 1964). Similarly, studies of sleep during the daytime (i.e., naps) have shown that more SWS is present in afternoon naps relative to morning naps (Karacan, Finley, Williams, & Hursch, 1970a). Consequently, morning naps minimally affect nocturnal sleep, whereas an afternoon nap can reduce the SWS for the following night (Feinberg et al., 1985; Karacan, Williams, Finley, & Hursch, 1970b). The normal amount of SWS can be reduced by naps and enhanced by sleep deprivation, providing further support of the compensatory nature of sleep homeostasis. Thus, behaviours such as napping, going to bed earlier than usual, sleeping-in, or being sedentary, can all result in a lowered homeostatic drive for sleep and, consequently, more disturbed sleep.

2.2.2 Circadian Rhythms.

A salient characteristic of human sleep is its daily rhythmicity. The circadian rhythm (process C) is an endogenously driven, approximately 24-hour, cycle that depends on circadian time cues (i.e., zeitgebers) to remain properly oriented to an individual's environment and preferred routine (Moore, 1997, 1999). The circadian propensity for sleep increases during the sleep state ensuring continued sleep, despite the diminishing homeostatic need for it near the end of the sleep cycle (Achermann & Borbély, 2003; Borbély & Tobler, 1985; Saper et al., 2005). Conversely, the circadian waking drive builds up throughout the day and it is at its greatest during the hours just before sleep, when the homeostatic sleep drive is nearing its peak. This was first demonstrated by Kleitman (1963) who examined human circadian rhythms by shielding participants from periodic environmental cues. He studied two participants living on non-24 hour

sleep/wake, light/dark, and meal schedules, while living in a cave, shielded from the earth's 24-hour day. Findings from this study demonstrate that the physiological rhythm can oscillate in the absence of periodic changes in the environment and at a period different from that of behavioral cyclicity. Therefore, this was one of the first studies providing support for the endogenous and physiologic nature of human circadian rhythms (Czeisler & Gooley, 2007).

Dement and Kleitman (1957) were the first to report that the duration of REM episodes increased throughout the duration of the night. Studies have confirmed an endogenous circadian rhythm of REM sleep propensity, which is closely associated with the body temperature rhythm (Czeisler, Zimmerman, Ronda, Moore-Ede, & Weitzman, 1980). Specifically, REM sleep is highest near the low point of the circadian temperature cycle and it at its lowest during the highest point of the cycle (Endo et al., 1981). Whereas the amount of SWS is dependent on the amount of prior wakefulness, the amount of REM sleep is generally dependent on the time of day (Endo et al., 1981) and it increases throughout the course of the night (Dement & Kleitman, 1957). In nap studies after a regular night's sleep, a larger amount of REM sleep is typically observed in the morning naps, in comparison to the afternoon and evening naps, as a consequence of the increase of SWS (e.g. Endo et al., 1981; Karacan et al., 1970a). Moreover, the REM pressure during the peak of the circadian rhythm is strong enough to overwhelm the tendency towards SWS, even after extended prior wakefulness (Endo et al., 1981). Although REM has a large circadian component, deprivation of REM sleep can lead to REM rebound, an alteration of the REM stage in the recovery night, such that on the recovery night the onset latency of REM is reduced and an increased duration of REM sleep is observed (Weitzman, Kripke, Goldmacher, McGregor, & Nogeire, 1970).

The circadian system has 3 components: 1) a pacemaker located in the suprachiasmatic nucleus (SCN), which generates a rhythm of approximately 24 hours; 2) stimuli that synchronize the pacemaker with the external environment; and 3) behavioural and physiological rhythms that are regulated by the pacemaker (Benloucif et al., 2005). In the absence of external cues, the circadian system in mammals depends on the SCN, the "master clock," located in the anterior hypothalamus (Richter, 1967). This master clock acts as the central neural pacemaker for the initiation and/or synchronization of circadian rhythms (Klein et al., 1993; Ralph, Foster, Davis, & Menaker, 1990; Welsh, Logothetis, Meister, & Reppert, 1995). Generally, circadian rhythms are entrained to the solar day on earth, which ensures that behavioral, physiologic, and genetic rhythms are timed properly with daily environmental changes (Czeisler & Gooley, 2007). The SCN clock is also regulated by other factors, such as melatonin secreted from the pineal gland (Arendt, 2003; Liu et al., 1997; McArthur, Gillette, & Prosser, 1991; McArthur, Lewy, & Sack, 1996; Zimmerman & Menaker, 1975), signals from the retina encoding environmental light levels (Moore & Lenn, 1972), and core body temperature (Benloucif et al., 2005).

The circadian pattern of melatonin release is controlled by the SCN. In addition to its effects on the circadian clock, melatonin has been implicated as a sleep promoting hormone, as it can induce sedation and lower core body temperature (Dijk & Cajochen, 1997; Krauchi & Wirz-Justice, 2001; Lavie, 1997; Zhdanova et al., 1995). However, since melatonin is secreted during the dark phase of the circadian cycle in nocturnal animals (Kennaway & Wright, 2002), it is not considered a universal sleep-inducing hormone, but rather acts as a hormonal indication of darkness (Bartness & Goldman, 1989). In humans, the administration of exogenous melatonin in the early evening advances the phase of circadian rhythms, whereas administration of melatonin in the early morning delays the phase (Lewy, Ahmed, Jackson, & Sack, 1992). However,

exogenous melatonin administered at night before bedtime does not improve sleep due to the endogenous melatonin already present in the body (Wyatt, Dijk, Ritz-de Cecco, Ronda, & Czeisler, 2006).

Without environmental signals (i.e., light input from the retina), the SCN preserves a circadian rhythm with a period either marginally shorter or longer than the 24 hour. In such a situation, the organism is considered to be free running and the rest-activity/sleep-wake cycles progressively become out of phase with the 24-hour day/night cycle. Individuals who are totally blind exhibit these free-running circadian rhythms (Arendt, Aldhous, Wright, 1988; Sack et al., 1992), which highlight the importance of light in human entrainment. However, the role of nonphotic time cues (i.e., daily activities) cannot be ruled out entirely. In a subset of blind individuals, daily activities associated with the 24-hour day are able to entrain the circadian pacemaker (Mistlberger & Skene, 2005). It may be that daily activities cues exert their force on the circadian system indirectly through light exposure and these activities may mask or alter a circadian rhythm, but daily activities are not thought to be the main entraining agent.

When exposed to typical daylight cycles, the circadian rhythm is entrained to the 24 hours of the sun. This entrainment is mediated, in part, by light signals received by the retina and relayed to the SCN (Klein, Moore, & Reppert, 1991). The expression of specific genes in the SCN (i.e., clock genes) determines the phase of the circadian clock and can be affected by nocturnal exposure to light. Thus, bright light exposure in the evening leads to a phase delay in the circadian clock and exposure in the late night causes a phase advance (Lowrey & Takahashi, 2000). Since the SCN clock regulates pineal melatonin release, these phase shifts can lead to noticeable changes in the timing of melatonin secretion. Accordingly, melatonin levels are one of

the most consistent measures of the phase of the circadian clock in humans (Arendt, 2003; Lewy & Sack, 1997).

Similar to melatonin, core body temperature rhythms (temperature of the brain and abdominal cavity, including inner organs) are generated by the SCN and is determined though the balance between heat production and heat loss (Cagnacci, Krauchi, Wirz-Justice, & Volpe, 1997). The nocturnal decline of core body temperature begins four to five hours prior to sleep (Cagnacci, Elliott, & Yen, 1992) and self-selected bedtimes ensue at the time of the maximal rate of decline of core body temperature (Campbell & Broughton, 1994). This process is opposite to the nocturnal rise of melatonin, such that peak melatonin levels are associated with the core body temperature nadir and the subsequent decline of melatonin is instantly followed by a rise in core body temperature (Cagnacci et al., 1997). This association has been observed in individuals entrained in a typical day night period, in addition to those in a light-induced phase shift (Shanahan & Czeisler, 1991).

Behavioural circadian rhythms associated with the timing of daily activities appear to take place, analogous to that of physiological circadian rhythms (i.e., core body temperature and plasma hormone concentrations; Monk, Frank, Potts, & Kupfer, 2002). Human circadian rhythms are measured via intrusive physiologic assays, such as 24-hour core body temperature and salivary or 24-hour catheter-extracted plasma melatonin levels; however, measuring behavioural rhythms associated with the timing of sleep, meals, work, and social interactions may be just as important to assess as physiological assays (Monk, 2010). Although measuring physical zeitgebers has become more common (Czeisler et al., 1986), social time cues (i.e., behavioural social rhythms) were previously recognized as the main zeitgebers (Wever, 1979). Early investigations into circadian rhythms involved participants' tracking their activities to

allow for these events to be plotted out in relation to sequential sleep/wake cycles (Wever, 1979). Frequently, the gap in timing between meals was predictive of the subjective day length as a whole, in free-running conditions (Aschoff, von Goetz, Wildgruber, & Wever, 1986). Unfortunately, the majority of the circadian rhythms research to date has focused on the physiological variables, rather than the behavioural ones (Monk et al., 2002), even though the behavioural rhythms are the ones most noticeable to the individual themselves (Monk, 2010). In fact, further support for the validity of behavioural markers comes from a study that found participant bed and rise times on the Social Rhythm Metric (SRM; Monk et al., 1990) to be associated with their endogenous circadian temperature rhythm (Monk et al., 1994).

It is important to clarify that these behavioural circadian rhythms are not only circadian rhythms in their own right, but also act as gatekeepers to the zeitgebers that affect the individual and act to entrain his or her circadian rhythm to a 24-hour period (Monk, 2010). Although photic zeitgebers (i.e., ones that occur from sleeping in the dark) are considered to be the most influential (Moore, 1997), there is evidence to suggest that nonphotic zeitgebers (e.g., exercise) are also zeitgebers (Mistlberger & Skene, 2004). Several studies have shown that subjective sleep quality is related to daily activity levels, such that poor sleep quality arises from inactivity or proneness toward sedentary lifestyles (Morgan, 2003; Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001; Sherrill, Kotchou, & Quan, 1998). Similarly, studies have demonstrated that irregular daily routines are linked to poor sleep quality (Carney et al., 2006a; Monk et al., 1994; Monk et al., 1992; Monk et al., 2003). Consequently, the majority of behavioural treatments of insomnia include targeting the regularity of sleep time (Bootzin, 1972b; Morin, 2004; Morin et al., 1994; Morin et al., 1999) by prescribing a rise-time, which ultimately boosts the power of the patient's behavioural circadian rhythm. Finally, social engagement may also play an important

role in sleep quality, such that various studies have demonstrated that sleep complaints are relatively common among those who are socially alienated or disappointed with their social relations (Edinger, Stout, & Hoelscher, 1988; Marchini, Coates, Magistad, & Waldum, 1983; Ohayon et al., 2001).

2.2.3 Arousal.

Whereas the circadian and homeostatic systems work to regulate sleep, both systems can be trumped by the arousal system, which is best characterized as a state of excitation or activation. Anxiety, a type of activation, could be considered physiological, behavioural and/or cognitive in response to threatening stimuli. Anxiety appears to serve a protective function, as it will alert and mobilize the organism away from danger through the activation of the sympathetic nervous system (Hoehn-Saric & McLeod, 1988). However, this state of alertness is not conducive for sleep, as sleep requires a passive disengagement from the environment. Thus, this sleep system could be disruptive to the sleep onset period.

Prevailing etiological theories of insomnia typically focus on physiological (Bonnet & Arand, 1995), emotional, or cognitive hyperarousal (Morin, Rodrigue, & Ivers, 2003) as predisposing, precipitating, or perpetuating factors (Spielman et al., 1987). Arousal has demonstrated negative consequences for sleep onset in insomnia. For example, Waters and colleagues (1993) found that negative emotion, stress, and attention variables accounted for approximately 41% of the variance in sleep variables, providing support for the link between emotional arousal and sleep. Other researchers have focused primarily on physiological arousal and most insomnia treatments target a reduction in physiological arousal (Bootzin, 1972a; Morin et al., 1999). Although only one study (Monroe, 1967) provides empirical support for the hypothesis that poor sleepers are more physiologically aroused (i.e., greater autonomic activity

pre-sleep and during sleep, higher mean heart and pulse rates, rectal body temperatures, and phasic vasoconstriction) in the pre-sleep period in comparison to good sleepers, there is evidence that those with insomnia experience greater overall hyperarousal in a 24-hour period (Bonnet & Arand, 1995, 1997; Nofzinger et al., 2004). Specifically, in comparison to good sleepers, those with insomnia have demonstrated increased metabolic rate (Bonnet & Arand, 1995), increased sympathetic nervous system activity (Bootzin, 1972a), and increased cortical arousal, regardless of the time of day (Nofzinger et al., 2004).

Similarly, cognitive arousal appears to play a prominent role in insomnia (Lichstein & Rosenthal, 1980). For instance, Gross and Borkovec (1982) demonstrated that even among good sleepers, inducing cognitive arousal by telling participants they would have to give a speech in the morning, significantly increased the sleep onset latency. However, other studies have failed to find a link between cognitive arousal and sleep. Thus, some researchers have concluded that cognitive arousal may be a consequence, rather than a cause of insomnia (Freedman & Sattler, 1982). This could occur after repeated pairings of sleep disturbances with the bedroom, which could unintentionally lead to conditioned arousal, in which the bed becomes an alerting/wakeful stimulus (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Despite mixed findings, even during periods of high homeostatic drive (e.g., periods of sleep deprivation) or optimal timing for sleep (i.e., during the night), the arousal system can prevent people from sleeping, thus, this system appears to have some importance in insomnia.

2.3 Models of insomnia

Various models purport to clarify the onset and maintenance cycle of chronic insomnia. The 3P behavioural model, also referred to as the Spielman model, three-factor model, or the behavioural model (Spielman et al., 1987), is a widely invoked diathesis-stress model. The 3P

model suggests that the development of chronic insomnia is largely dependent on three factors, namely, predisposing, precipitating, and perpetuating factors (Spielman et al., 1987). A predisposing factor (e.g., anxiety sensitivity) is a factor that increases an individual's vulnerability to sleep difficulties. These predisposing factors are not a direct cause of insomnia, however, they increase the likelihood that an individual could develop a sleep problem when encountering a stressor. Once predisposed to sleep difficulties, a precipitating factor (e.g., stressful event) can trigger the onset of acute insomnia. Thus, an individual with a predisposition to insomnia would only develop an episode of acute insomnia in response to this precipitating event. Although for some individuals this acute insomnia episode may be temporary and could normalize after the stressor had been removed, this is not the case for everyone. Perpetuating factors, such as maladaptive psychological or behavioural coping strategies, develop in response to the sleep difficulty as a means to compensate for, or cope with, fatigue. For example, individuals may begin to spend excessive amounts of time in bed in order to increase the opportunity for sleep (Perlis et al., 1997). Unfortunately, this increased time in bed can interfere with ability to produce enough of a sleep drive to maintain uninterrupted sleep throughout the night, leading to long periods of wakefulness while in bed. As a result, an association between ones sleep environment and wakefulness may be fostered (i.e., conditioned arousal), in which the bed and bedroom can lead to arousal and apprehension, as opposed to sleep and sleepiness (Perlis et al., 1997), which further interferes with the ability to produce sleep.

In response to the discomfort associated with acute sleep difficulties, Harvey's (2002) cognitive model of insomnia posits that individuals may engage in maladaptive cognitive processes to cope with this sleep loss, leading to increased sleep-related anxiety. This model suggests that a cognitive process that functions both during the day and night maintains the

insomnia. Specifically, these processes include worrying about not being able to sleep, accompanied by physiological arousal and distress; selective attention and monitoring of internal (e.g., bodily sensations) and external (e.g., environmental noise) threats to sleep; misperception of sleep and daytime functioning deficits; maladaptive safety behaviours (e.g., drinking coffee to stay awake); and dysfunctional beliefs about sleep (e.g., belief that you need at least eight hours of sleep; Harvey, 2002). In particular, those with insomnia are more likely to hold these dysfunctional beliefs about sleep relative to good sleepers and these beliefs are considered to play a significant role in the maintenance of the disorder (Carney & Edinger, 2006b; Carney et al., 2011a; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001).

Accordingly, both cognitive and behavioural perpetuating factors can turn an acute sleep disturbance into chronic insomnia. These perpetuating factors can negatively impact the sleep regulatory processes (Bootzin, 1972a; Harvey, 2002) and it is this interaction between the perpetuating factors and sleep regulatory processes that work to maintain the insomnia. Morin (1993) developed the cognitive behavioural model of insomnia in order to explain the factors hypothesized to play a role in insomnia. This model incorporates empirically supported psychotherapeutic interventions, which encourages poor sleepers to behave and think like good sleepers. The main objective of cognitive behavioural therapy for insomnia (CBT-I) is to target the cognitive and behavioural factors that serve to maintain the insomnia by modifying poor sleep habits, altering maladaptive beliefs about sleep, and developing more adaptive coping skills (Morin, 2004, 2010).

Both meta-analyses (Morin et al., 1994; Smith et al., 2002) and systematic reviews (Irwin, Cole, & Nicassio, 2006; Morin et al., 2006b; Morin et al., 1999) have summarized the efficacy data from clinical trials of CBT-I in adults. These reviews suggest that CBT yields reliable

changes in several sleep parameters, including sleep onset latency, wakefulness after sleep onset, total sleep time, and sleep quality. Specifically, this data suggests that approximately 70-80% of insomnia patients achieve a therapeutic response with CBT-I and about 40% achieve clinical remission (Morin, 2010; Morin et al., 2006b; Morin et al., 1999). Moreover, the efficacy data demonstrates that these sleep improvements are maintained over time, with almost 50% of treated patients remaining in remission up to 24 months after treatment completion. However, follow-up data must be interpreted with caution, as only limited studies report this data and attrition rates tend to increase over time. Nevertheless, CBT-I has been endorsed as the front-line treatment for insomnia by the National Institute of Health and the British Association for Psychopharmacology (National Institutes of Health, 2005; Wilson et al., 2010) and is recommended as the initial treatment in the management of chronic insomnia (Morgenthaler et al., 2006; Schutte-Rodin et al., 2008).

The main components of CBT-I include stimulus control, sleep restriction, sleep hygiene, and cognitive therapy. The main objective of stimulus control (Bootzin, 1972b) is to break this conditioned arousal that arises after repeated unsuccessful sleep attempts (Perlis et al., 1997). Weakening of this conditioned arousal is achieved by focusing on re-associating the bed and bedroom with successful sleep attempts by instructing the client to: 1) only go to bed only when sleepy; 2) get out of bed whenever awake for long periods; and 3) avoid reading, watching television, eating, worrying and other sleep-incompatible behaviours in the bed and bedroom (Bootzin, 1972b). Thus, stimulus control mainly targets arousal sleep system by decreasing hyperarousal; the circadian system by enforcing a rise time, and the homeostat by decreasing the time in bed (See Figure 1; Edinger & Means, 2005).

Sleep restriction, another main behavioural component of CBT-I, is a method designed to shorten the amount of time in bed to match the client's total sleep time (Morin et al., 2006b). This is determined by evaluating the clients' two-week sleep diary and calculating the total sleep time and adding an additional 30 minutes to allow time to fall asleep. Together with the client, the therapist would determine an earliest rise time, which would be consistent each day, and work backwards to calculate an earliest bedtime. Throughout treatment, the therapist would make adjustments to this sleep schedule, based on the clients' ability to fill-up the time in bed with sleep (i.e., sleep efficiency). By restricting sleep time, this component of CBT-I directly targets the homeostatic regulation system by increasing ones sleep debt, in addition to the circadian system by regulating the daily rise time. Finally, throughout the course of CBT-I, clients are educated on healthy sleep behaviours and sleep promoting environmental conditions. These include the elimination or reduction of caffeine, alcohol, and nicotine; engaging in exercise; eating a light bedtime snack with tryptophan (an amino acid that promotes sleepiness); and creating a sleeping environment that is quiet, dark, and comfortable.

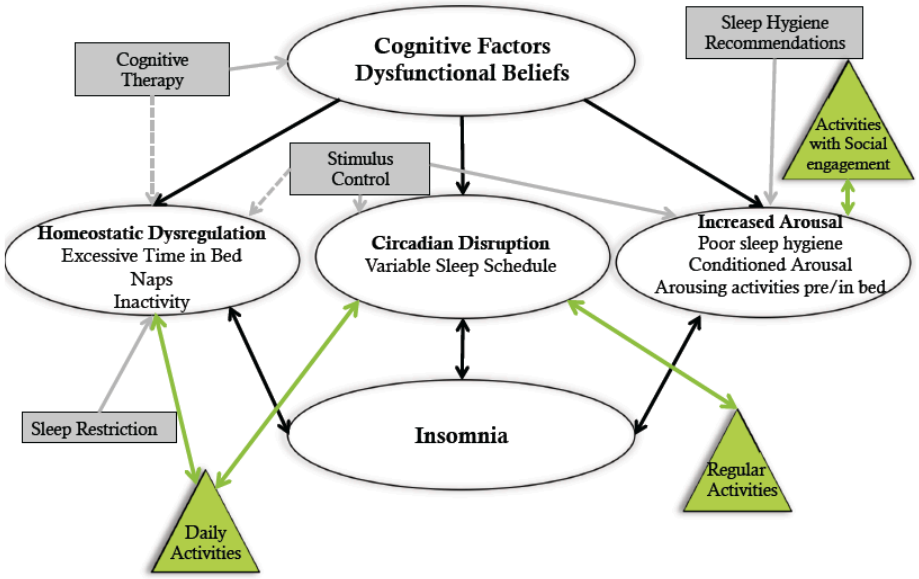


Figure 1. Interaction of cognitive and sleep regulatory processes in the development and maintenance of Primary Insomnia; CBT-I treatment component targets and the potential role of daily activities. Adapted from "Cognitive-behavioral therapy for primary insomnia" by J. D. Edinger and M. K. Means, 2005, *Clinical Psychology Review, 25*, 539-558.

Cognitive therapy is a psychological method aimed at challenging and adapting sleep misconceptions and beliefs about insomnia and the perceived daytime consequences of sleep loss (Morin et al., 2006b). Cognitive techniques used in CBT-I involve teaching the client to identify, realistically evaluate, and modify their sleep related thoughts (Harvey, 2002) e.g., "if I do not get eight hours of sleep, I will not be able to get through the day tomorrow," or "I need to nap to catch up on lost sleep." Many of these misconceptions can lead to hyperarousal (i.e., anxiety about not being able to fall asleep and the consequences) and maladaptive compensatory coping strategies (e.g., daytime napping, increased time in bed). Thus, the cognitive component targets

all components of the sleep system by decreasing arousal and behaviours that would interfere with the homeostat and circadian system.

Evidently, CBT-I works by exerting influence on the sleep system, however this treatment would likely be further improved by focusing on additional daytime behaviours that disrupt these systems. For example, targeting behavioural zeitgebers, mainly the frequency and regularity of activities, that presumably help to entrain the circadian clock, enhance the homeostat, and decrease arousal, may be beneficial. Further research that examines the behavioural factors that serve to maintain the insomnia is necessary in order to increase efficacy rates of CBT-I and reduce the risk of recurrent episodes of insomnia in the long-term.

2.4 Social Rhythms

Empirical investigations of daily routines and sleep have been investigated in various populations. For example, Carney and colleagues (2006a) used prospective monitoring of daily activities to examine the effect of social rhythms on sleep quality in undergraduate students, ranging in age from 18 to 39 years old. Undergraduate students retrospectively reported their activities for a two week period, using the Social Rhythm Metric (SRM; Monk et al., 1990) and then completed the Pittsburg Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Based on the PSQI cutoff scores, participants were divided into good and poor sleepers and compared on regularity, frequency, timing, and extent of social engagement during activities. Relative to good sleepers, poor sleepers demonstrated a lower frequency and less regularity of social rhythms, even after controlling for depression. Furthermore, good sleepers engaged in more regular activities with active social engagement. Thus, these findings suggest that regulating behavioural zeitgebers is beneficial for sleep and, although conclusions about

causality cannot be made, there appears to be an association between engaging in activities with other people and regularity.

Similarly, this relationship of sleep quality and daily activities has also been investigated in the elderly, as this is a population in which insomnia symptoms are prevalent (Foley et al., 1995). Buysse et al. (2010) compared older adults with and without chronic insomnia, using the short version of the SRM, and failed to find a significant group difference. In contrast, Zinsberg and colleagues (Zisberg, Gur-Yaish, & Shochat, 2010) examined lifestyle regularity in a sample of elderly Israelis, living in a retirement community. Regularity was assessed via the Scale of Older Adults Routine (SOAR; Zisberg, Young, & Schepp, 2009) at three time points, and a subsample of participants completed the SRM. Similar to Carney et al. (2006a), this study used the PSQI to divide the sample into good and poor sleepers. Regression analyses demonstrated that increased stability, assessed via the SOAR and the SRM, was predictive of shorter sleep onset latency, higher sleep efficiency, and improved sleep quality, beyond functional status, comorbidities, and age. Considering the limited research examining social rhythms and the mixed findings, it remains unclear whether regularity in daily routines is beneficial for sleep in the elderly.

Finally, social rhythms have been examined in a general adult population. Similar to the aforementioned studies, Monk and colleagues (2003) also examined lifestyle regularity (based on SRM scores) and sleep quality (assessed via the PSQI). As expected, there was a significant negative correlation between lifestyle regularity and sleep quality, such that those with increased levels of regularity, reported fewer sleep difficulties. Furthermore, in a categorical analysis, the proportion of poor sleepers in the irregular routine group was double that of the regular routine group. Accordingly, it appears as though sleep quality is affected by the frequency and regularity

of daily activities. However, other than one study looking an older adult population (Buysse et al., 2010), these findings are based on sleep quality, rather than people with insomnia and good sleeper comparisons.

2.4.1 ***Social Rhythms and Sleep.***

Optimally, social rhythms would act in synchrony with zeitgebers in order to ensure circadian entrainment. However, societal determination of work and social schedules can interfere with this alignment (Wittmann, Dinich, Merrow, & Roenneberg, 2006). For example, those with late chronotypes (i.e., preference for later bed and rise times) experience a large sleep debt over the week. In order to compensate for this, these individuals often sleep for longer durations on the weekend (Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002; Taillard et al., 2003). Consequently, this misalignment between the biological and social clock (i.e., social jetlag) can lead to increased mental exhaustion (i.e., fatigue), increased sleepiness, decreased mood and cognitive performance, and insomnia, even with an adjustment of the sleep schedule by as little as two hours (Taylor, Wright, & Lack, 2008; Wittmann et al., 2006; Yang & Spielman, 2001; Yang et al., 2001). Essentially, people can experience the same symptoms that are experienced when traveling to a different time zone, without actually travelling. Aside from these observable disturbances, delaying rise-times on the weekend can be measured by physiological indices. For example, a delay as minimal as three hours can result in a delay of dim light melatonin onset by 42 minutes, as measured by salivary samples (Taylor et al., 2008). Irregular zeitgebers can produce daily jetlag symptoms including disturbed sleep and other common daytime symptoms of insomnia.

It is possible that those engaged in more frequent activities would be more likely to leave the house, thus increasing the likelihood of sunlight exposure, which helps to entrain these

biological rhythms. Similarly, those engaged in a more regular lifestyle (i.e. consistent meal times and bedtimes), would presumably have an internal clock that is more entrained (Aschoff et al., 1986; Morin, 2004). In contrast, those with reduced activities may remain in the home, limiting chances of sunlight exposure, or live a variable lifestyle, providing the clock with limited zeitgebers. As such, it is plausible that social rhythms could conflict with and disrupt the regular circadian entrainment, which may contribute to disturbed sleep.

The homeostatic process would presumably also be affected by social rhythms. For instance, those with sleep disturbances attempt to engage in compensatory coping behaviours, such as sleeping-in, napping, and living an overall sedentary lifestyle. These behaviours can decrease ones sleep drive, resulting in both sleep onset and maintenance problems. In addition, daytime behaviours, such as napping, have been linked to less slow-wave restorative sleep (Feinberg et al., 1985; Karacan et al., 1970a; Karacan et al., 1970b), which could continue the cycle of a sedentary lifestyle. Thus, these behaviours could be both precipitating and perpetuating factors of insomnia. Increased daily activities may contribute to a higher sleep drive by eliminating these behaviours that interfere with this sleep process.

Finally, and perhaps to a less likely degree, the arousal system may be affected by social rhythms, such that decreased social engagement and activities could increase negatively valenced emotions and arousal, thus having negative consequences on sleep (Mistlberger & Skene, 2005). Additionally, those with reduced activities may spend more time in bed, thus conditioning the bed with wakefulness (i.e., conditioned arousal; Perlis et al., 1997), which is not conducive to sleep. Research examining the relationship between social rhythms and sleep in a clinical population would provide further insight to the contributing role of daily activities in insomnia.

2.5 The Current Study

The current study extends the existing body of literature that suggests frequent and consistent social rhythms may have beneficial effects for sleep (e.g., Carney et al., 2006a; Monk et al., 2003; Zisberg et al., 2010). Specifically, this study examined sleep and overall activity levels, such as the amount, timing, and regularity, of social rhythms and the degree to which daily routines entail social engagement in both those with clinical insomnia and those with good sleep. In order to improve upon previous studies that used retrospective self-report data, the current study utilized prospective measures of sleep, including sleep diaries, the gold-standard in sleep research, and actiwatches, which provide an objective and prospective measure of daily activities and sleep.

This study design allowed for improved understanding of the role of social rhythms in sleep. While it is beyond the scope of the current study, results that provide empirical support for the beneficial effect of activity and regularity on sleep could have important clinical implications for those suffering from insomnia. Since the first line treatment of insomnia (i.e., CBT-I) has a 70-80% response rate (Morin, 2011), room for improvement remains. Although, CBT-I targets the regularity of bed and rise-times, it does not address the regularity of other activities. Accordingly, this research could potentially decrease the risk of insomnia and increase treatment response by targeting daytime activities (frequency and regularity) as an insomnia treatment target, as increased daily regularity would likely reduce jetlag symptoms. Finally, since insomnia can increase the risk, worsen, and interfere with the treatment of other mental health disorders (e.g., MDD), a reduction in insomnia would have mental health benefits that extend beyond insomnia.

Consistent with previous literature on poor sleep and social rhythms (Carney, Edinger, Meyer, Lindman, & Istre, 2006a; Monk et al., 1990; Monk, Kupfer, Frank, & Ritenour, 1991; Monk, Petrie, Hayes, & Kupfer, 1994; Monk, Reynolds, Machen, & Kupfer, 1992; Monk et al., 2003), those with insomnia disorder (ID) were expected to engage in fewer habitual activities, have a more irregular daily schedule, and have less social engagement, compared to normal sleepers (NS). Furthermore, it was anticipated that findings from this study would demonstrate a direct relationship between sleep and psychomotor daily activity level among ID, such that the daily psychomotor activity level of ID is related to the previous night's sleep, which would likely not be the case for NS.

2.6 Hypotheses

- *Primary hypothesis:* activities on the SRM will significantly differ between sleeper status (i.e., NS vs. ID). Specifically, it is hypothesized that relative to NS, ID will: 1) engage in fewer daily activities 2) engage in social rhythms characterized by less regularity and 3) be involved in fewer activities with active social engagement.
- S*econdary hypothesis:* Among those with insomnia, frequency of daily psychomotor activities will significantly predict the subsequent night's sleep ('total wake time') on the actiwatch. Likewise, it is expected the previous night's sleep will significantly predict the daily psychomotor activity level on the actiwatch among the ID, such that following a poor night's sleep, ID will engage in fewer daytime psychomotor activities, whereas after a good night's sleep, they will engage in more daytime psychomotor activities. In contrast, it is hypothesized that NS daytime psychomotor activities and the previous night's sleep will not be significantly related.

- *Tertiary hypothesis:* Given that thoughts, feelings, and behaviours, are related (Beck, Rush, Shaw, & Emery, 1979) and that rigid sleep beliefs are common among those with insomnia (Carney & Edinger, 2006b; Carney et al., 2011a; Edinger et al., 2001), a closer examination of dysfunctional beliefs about sleep and whether they contribute to daily activity levels will be conducted. Beliefs about sleep are hypothesized to mediate the relationship between sleep and daily activities. Specifically, it is hypothesized that maladaptive beliefs about sleep (e.g., "after a poor night's sleep, I know it will interfere with my activities the next day") contribute to those with insomnia engaging in fewer daytime activities. In contrast, good sleepers are less likely to hold these unhelpful sleep beliefs and their daytime activities are likely unaffected by sleep loss.

CHAPTER THREE

METHOD

3.1 Participants

Study participants (N = 80) were recruited between December 2011 and March 2013 and included people with Insomnia Disorder (ID; Edinger et al., 2004a) and Normal Sleeper (NS; Edinger et al., 2004a) volunteers from the Greater Toronto Area. Participants were recruited via advertisements posted in the community and online (e.g., Kijiji and Craigslist). Additionally, medical professionals in the community referred a proportion of the ID participants to the Sleep and Depression Lab at Ryerson. After participating in a brief phone screen, 107 potential participants partook in the screening interview in order to determine eligibility. In order to be eligible for participation, the ID volunteers were required to meet Research Diagnostic Criteria (RDC; Edinger et al., 2004a) for Insomnia Disorder, whereas the Normal Sleeper were required to meet RDC for NS. Given that insomnia often presents as comorbid with other Axis I disorders, those with comorbid disorders that would not confound our results and were permitted to participate, thus making our findings more generalizable. *Eligibility* criteria for all participants are as follows: 1) between 18 and 74 years old, 2) fluency in English. *Exclusion* criteria are as follows: 1) meets criteria for a mental health disorder that could interfere with sleep, such as bipolar disorder, substance dependence, any psychotic disorder, or endorses current suicidal ideation, as per the Structured Clinical Interview for DSM-IV-TR Disorders (SCID-IV-TR; First, Spitzer, Robert, Gibbon, & Williams, 2002), 2) Meets criteria for a sleep disorder that could confound results, such as moderate to severe apnea, hypersomnia, or a circadian rhythm disorder, as per the validated Duke Structured Interview for Sleep Disorders (DSISD; Edinger et al., 2004b). After this screening interview, 18 potential participants did not meet study eligibility

requirements and, thus, did not partake in the study. Moreover, nine participants that met eligibility requirements declined to participate.

3.2 Measures

3.2.1 Screening measures.

Duke Structured Interview for Sleep Disorders (DSISD; Edinger et al., 2004b).

The DSISD is a structured diagnostic interview designed to assist in determining sleep disorders in accordance with both the DSM-IV-TR and the International Classification of Sleep Disorders (ICSD-2; American Sleep Disorders Association, 1996) sleep disorder nosologies. It also permits assessment of whether someone meets RDC criteria for ID or NS, which was required for participation. A clinical structured interview is recommended to assure consistency with RDC criteria (Edinger et al., 2004a). This diagnostic interview was used to determine volunteers met ID criteria and to rule out apnea, hypersomnia, or a circadian rhythm disorder. The DSISD is divided into four modules: 1) sleep disorders associated with insomnia complaints; 2) sleep disorders associated with complaints of excessive daytime sleepiness-hypersomnia; 3) circadian rhythm sleep disorders; and 4) sleep disorders associated with parasomnias. The DSISD has both acceptable inter-rater reliability for an insomnia diagnosis (r = .46 across DSM-IV-TR & ICSD-2 categories; Carney, Edinger, Olsen, Stechuchak, & Krystal, 2008) and discriminant validity (Carney, Ulmer, Edinger, Krystal, & Knauss, 2009). Likewise, this measure has moderate to good inter-rater reliability for the exclusion criteria apnea (r =.74) and a circadian rhythm disorder (r =.44; Carney et al., 2008). Moreover, the DSISD is a commonly used assessment interview in sleep research (e.g., Carney, Moss, Harris, Edinger, & Krystal, 2011b; Edinger et al., 2009; Talbot et al., 2011).

Structured Clinical Interview for DSM-IV Disorders (SCID; First, Spitzer, Robert, Gibbon, & Williams, 2002).

The SCID is a semi-structured interview used for making the major DSM-IV-TR Axis I diagnoses. This instrument is considered the gold standard semi-structured interview for the assessment of clinical disorders (Lobbestael, Leurgans, & Arntz, 2011). The SCID was used to exclude participants with an Axis I disorder that could interfere with sleep, such as bipolar disorder, substance use disorder, psychotic disorder, or current suicidal ideation that could interfere with safety of the participant. In a mixed sample of inpatients, outpatients, and non-patient controls, Lobbestael and colleagues (2011) assessed the inter-rater reliability of the SCID-IV-TR and demonstrated fair to excellent inter-rater agreement of Axis I disorders, with kappa values ranging from .61 to .83. Relevant to the current study exclusion criteria, these authors reported good kappa values for substance use disorder (.65 to .77). Given the wide use of SCID methodology in various research applications over the past several decades, the current version of the SCID has a strong history of being utilized in research, which further supports its reliability, validity, and utility for screening and diagnosing patients.

3.2.2 Standard battery

Insomnia Severity Index (ISI; Morin, 1993).

The ISI (see Appendix B) is a 7-item self-report questionnaire that provides an index of the global severity of insomnia by assessing the nature, severity, and impact of insomnia (Bastien, Vallieres, & Morin, 2001; Morin, Belleville, Belanger, & Ivers, 2011). This measure was used to evaluate the severity of sleep onset, sleep maintenance, early morning awakening problems, sleep dissatisfaction, interference of sleep difficulties with daytime functioning, noticeability of sleep problems by others, and distress caused by the sleep difficulties. Each item is rated on a 5-

point Likert scale, ranging from "0 = no problem" to "4 = very severe problem," yielding a total score between 0 and 28. A cutoff score of 10 is optimal for detecting insomnia in a community sample (Morin et al., 2011). The ISI is increasingly being used as a measurement of treatment response in clinical research (Morin et al., 2011).

The ISI has been found to be a reliable and valid instrument to measure self-reported insomnia severity (Bastien et al., 2001; Morin et al., 2011). Bastien and colleagues (2001) investigated the psychometric properties of the ISI in a clinical sample of young and older adults and found adequate internal consistency (Cronbach $\alpha = 0.74$) and significant correlation coefficients (at the 0.01 level) between the ISI individual items and corresponding variables on the sleep diary. Likewise, Morin and colleagues (2011) further investigated the internal consistency of the ISI in both a community and clinical sample and found high internal consistency for the ISI in both samples (Cronbach $\alpha = 0.90$ and 0.91).

Additionally, the ISI has been found to be a valid and sensitive measure to detect changes in self-reported sleep problems across treatment, as demonstrated by significantly lower ISI total scores at post-treatment, relative to baseline, which parallels improvements obtained from sleep diary and PSG. Furthermore, the internal reliability coefficients remained stable from baseline (.76) to follow-up (.78; Bastien et al., 2001). Thus, these finding provide support that the ISI is a reliable and valid measure to identify insomnia in the population and is sensitive to treatment response in clinical patients.

Depression and Anxiety Stress Scale (DASS-21; Lovibond & Lovibond, 1995).

The DASS-21 (see Appendix C) is a shortened version of the original DASS-42. This measure is a 21-item self-report questionnaire that assesses three negative states, mainly depression, anxiety, and stress from the past week. This measure was used to primarily evaluate

depression severity, as this score was entered into the model in the statistical analysis of the primary hypothesis. The Depression scale includes item that measures symptoms often related to dysphoric mood (e.g., sadness or worthlessness). The Anxiety scale includes items that are primarily associated with symptoms of physical arousal, panic attacks, and fear (e.g., trembling). Finally, the Stress scale includes items that assess for symptoms such as tension, irritability, and a tendency to overreact to stressful events (Antony, Bieling, Cox, Enns, & Swinson, 1998). Each item is rated on a 4-point scale ranging from 0 = "Did not apply to me at all" to 3 = "Applied to me very much or most of the time." Scores from each subscale are summed and multiplied by two, which yields a total subscale score ranging from 0 to 42. The following are optimal cutoff scores for detecting the degree of depression: 10-13 mild, 14-20 moderate, 21-27 severe, and 28+ extremely severe on the Depression subscale. The following are optimal cutoff scores for detecting the degree of anxiety: 8-9 mild, 10-14 moderate, 15-19 severe, and 28+ extremely severe on the Anxiety subscale. The following are optimal cutoff scores for detecting the degree of stress: 15-18 mild, 19-25 moderate, 26-33 severe, and 34 extremely severe stress on the Stress subscale.

Psychometric properties of the full version of the DASS scale suggest that it has excellent internal consistency and temporal stability and provides a better distinction between the features of depression and anxiety than other existing measures (Brown, Chorpita, Korotitsch, & Barlow, 1997; Lovibond & Lovibond, 1995). In a sample of nonclinical volunteers and patients diagnosed with mood and anxiety disorders, Antony et al. (1998) assessed the internal consistency of the DASS-21 and reported high Cronbach's alphas for the Depression, Anxiety, and Stress subscales (.94, .87, .91, respectively). Similarly, Henry et al. (2005) reported high Cronbach's alphas for the Depression, Anxiety, and Stress subscales (.88, .82, .90, respectively)

in a non-clinical population. The concurrent validity of the DASS-21 with other measures of depression and anxiety has also been investigated in a clinical sample of outpatients diagnosed with an anxiety disorder or major depressive disorder (Antony et al., 1998). The Stress scale was found to correlate moderately with other measures of depression and anxiety (.69-.70); the Depression scale correlated most highly with other measures of depression (.79) and moderately with the anxiety measures (.51-.71); and the anxiety scale correlated most highly with the anxiety scale (.85). Thus, the DASS-21 appears to be a reliable and valid measures of depression, anxiety, and stress, and offers several advantages to the original 42-item version, including fewer items, a cleaner factor structure, and smaller interfactor correlations (Antony et al., 1998).

The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16; Morin, Vallieres, & Ivers, 2007). The DBAS-16 (see Appendix D) is a self-report measure that assesses unhelpful beliefs about sleep and is a 16-item abbreviated version of the original 30-item scale. The DBAS was used to determine the degree of unhelpful beliefs held among participants and was used to analyze whether these beliefs influenced daily activity levels (hypothesis three). The DBAS was also used in posthoc analyses to test whether these beliefs indirectly affect the relationship between regularity and insomnia severity. The DBAS includes item subsets or subscales designed to measures four discrete cognitive themes, including 1) sleep-related worry and helplessness; 2) beliefs about sleep medications; 3) expectations about sleep need; and 4) beliefs about the consequences of insomnia. Each item is rated 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 greater maladaptive beliefs about sleep.

The DBAS-16 had demonstrated adequate internal consistency for both clinical and research insomnia samples (Cronbach's alpha = .77 and .79 respectively) in addition to

appropriate convergent validity with the ISI, sleep diaries and polysomnography (Morin et al., 2007). The DBAS can distinguish between good sleepers and those with insomnia and scores have been found to decrease with treatment that specifically targets these beliefs (i.e., CBT-I; Carney & Edinger, 2006b). In a large multi-study investigation, Carney and colleagues (2010) demonstrated that the DBAS-16 is a reliable and valid tool. These researchers reported that the DBAS can 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). The DBAS is the most commonly used tool for assessing maladaptive beliefs in insomnia (Carney et al., 2010; Edinger et al., 2001).

3.2.3 At home monitoring.

Social Rhythm Metric (SRM; Monk et al., 1990).

The SRM (see Appendix E) is a daily monitoring diary that is used to quantify social rhythms. The SRM is the primary outcome measure and was used to calculate the frequency and regularity of participants' daily activities (i.e., social rhythms) and was included in the analyses of all three hypotheses. Typically, the diary includes 17 activities, of which 15 are specified and are assumed to be common zeitgebers. However, the SRM was modified for this study to reflect more current activities involving technologies (e.g., internet usage). The 17 activities are as follows: 1) out of bed; 2) first contact with a person; 3) first beverage; 4) breakfast; 5) first time outside; 6) start work/school; 7) lunch; 8) nap; 9) dinner; 10) physical exercise 11) evening snack/beverage; 12) watch/read/listen to the news; 13) watch television (not news); 14) use phone for the first time 15) use computer for the first time (not to obtain news) 16) return home; and 17) get into bed. The remaining activity is personalized; such that each participant included

an activity that they engage in on a regular basis (e.g., caring for a pet) and was required to monitor this activity throughout the observation period. Various indices can be computed from the SRM, including the amount of overall activities (ALI; frequency count of the total number of activities) and the number of activities completed with active (ALI-A; rated as a 2), minimal (ALI-N; rated as a 1), or no (ALI-N; rated as a 0) social engagement.

The regularity of activities (SRM) was computed using a validated algorithm (Monk et al., 1990; Monk et al., 1991), which determines a habitual time for each activity. Observations that fall outside 1.5 SD of the mean time for a specific individual were omitted, and a habitual time was computed that excludes outlier data. Afterwards, all the observations, including the outliers, were merged together to calculate the amount of "hits," which are the number of activities that take place within 45 minutes of the habitual time. Lastly, the total amount of hits for activities that take place three or more times a week was divided by the amount of activities occurring three or more time per week. This calculation yields an index between 0 (i.e., no regularity) and 7 (i.e., regular). Thus, this index represents an individuals' level of daily lifestyle regularity for that week, with a higher number representing more regularity. The regularity of activities with active social engagement (SRM-A) was calculated by choosing only those activities where social interaction was rated a 'two' and then only those events would be included in the SRM algorithm. Similarly, the regularity of activities with no or minimal social engagement (SRM-N) were calculated by selecting activities where interaction was minimal, rated as 1, or the activity was performed alone, rated as 0, and only those activities were included in the SRM algorithm.

The SRM has been used to examine lifestyle regularity in a variety of populations. These populations include healthy young adults (Monk et al., 1994), older adults (Monk et al., 1992), both depressed and non-depressed elderly bereaved persons (Brown et al., 1996; Prigerson et al.,

1996), depressed individuals in remission (Monk et al., 1991), individuals with an anxiety disorder (Shear et al., 1994), and new parents (Monk, Essex, Smider, Klein, & Kupfer, 1996). The reliability and validity of this measure has been documented in previous studies (Monk et al., 1990; Monk et al., 1991; Monk et al., 1994). Validation research suggests the reported bed and rise times on the SRM are usually within 10 – 20 minutes of the objective, actigraphy estimates of bed and rise time (Monk et al., 1994). Furthermore, comparisons have found that the SRM index relates to the endogenous circadian temperature rhythm, such that increased SRM regularity is associated with a larger nocturnal dip in core body temperature (Monk et al., 1994).

Although a shorter version of the 'gold standard' SRM exists (i.e., SRM-5), which has acceptable values of kappa (0.69), sensitivity (74%) and specificity (95%; Monk et al., 2002), the current study used the full version (i.e., the SRM-17). The SRM-17 provides more information relevant to the implications of the current study. For example, whereas the SRM-17 provides details on breakfast, lunch, and dinner, the SRM-5 is limited to one meal (i.e., dinner). Given that regulating daily activities such as meals could be a target for insomnia treatment, only having information on one meal would be disadvantageous. Moreover, the SRM-5 does not provide information on whether the activities involved active social engagement or not, which is pertinent to the research questions. Finally, the SRM-17 is the gold-standard daily diary and has been frequently used in previous studies; thus, it was the most appropriate measure for this study.

Consensus Sleep Diary.

Completion of a sleep diary (see Appendix F) is the gold standard assessment tool in insomnia research (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). In fact, this assessment tool is regarded as the "gold-standard" for subjective sleep assessment (Carney et al., 2012). Participants were asked to complete a two-week expanded version of the Consensus Sleep

Diary (Carney et al., 2012) and report their subjective estimates of sleep patterns and sleep quality. This specific sleep diary has been developed by a panel of insomnia experts and was revised based on qualitative input from patients. Information collected on this sleep diary was used to confirm a diagnosis of insomnia. This sleep diary includes information surrounding nighttime bed habits, such as the length of time it takes to fall asleep, number and length of nocturnal awakenings, time of final awaking, and rise time. All information was recorded daily upon wakening, over the two-week assessment period of the study.

Taken together, the information derived from these sleep logs was as follows: total sleep time, sleep onset latency, time spent awake after initially falling asleep or "wakefulness after sleep onset", the percentage of time in bed spent sleeping or "sleep efficiency", a 10-point Likert-scale rating of sleep quality and a rating of restfulness upon awakening. These daily morning estimates of sleep parameters are common subjective sleep indices recommended as per the established guidelines in the literature (Buysse et al., 2006). The diaries yield a reliable and valid index of insomnia symptoms, despite not reflecting absolute values obtained from PSG. Those with insomnia typically overestimate sleep onset latency and wakefulness after sleep onset, while underestimating total sleep time, compared to PSG (Means, Edinger, Glenn, & Fins, 2003). However, sleep diaries are more likely to provide information on night-to-night variability (Wohlgemuth, Edinger, Fins, & Sullivan, 1999), which typically characterizes chronic insomnia. Accordingly, these diaries may in fact provide a more representative sample of an individuals sleep than one or two night of PSG (Buysse et al., 2006).

Wrist Actigraph.

A wrist actigraph is a portable device, worn like a wristwatch, that provides an indirect but objective measure of sleep/wake activity from the presence or absence of limb movements

(American Sleep Disorders Association, 1996; Ancoli-Israel et al., 2003; Lichstein et al., 2006; Littner et al., 2003; Sadeh & Acebo, 2002; Sadeh, Hauri, Kripke, & Lavie, 1995). The device can be worn continuously day and night for periods longer than one week (Hauri & Wisbey, 1992). Actigraphy yields typical sleep-pattern estimates, including sleep onset latency, wakefulness after sleep onset, and total sleep time (Lichstein et al., 2006). Over the past 30 years, the relationship between actigraphy and polysomnography have yielded correlation coefficients ranging from .89 to .98 (Jean-Louis et al., 1997; Kripke, Mullaney, Messin, & Wyborney, 1978; Mullaney, Kripke, & Messin, 1980; Sadeh et al., 1995). Accordingly, wrist actigraphy is considered a valid, objective instrument for the measurement of sleep/wake activity, with a potential decrement in accuracy when significant insomnia is present. The Standards of Practice Committee of the American Academy of Sleep Medicine recommend a minimum of three consecutive 24-hour periods (Littner et al. 2003; Lichstein et al., 2006). Rowe and colleagues (2008) recommend using two weeks of monitoring if variability of the sleep parameter is of interest.

Participants wore a Mini-Mitter Actiwatch Score model actigraphs (Mini-Mitter Co., Inc., Sun River, Or.) on their non-dominant wrist for 14 consecutive nights. The Actiwatch model contains a calibrated accelerometer and 32 K memory storage apparatus housed in a casing that, in size and shape, resembles a wristwatch. The Actiwatch is designed to interface with a PC computer via a specially designed Reader/Interface unit. PC Windows-style software accompanying the Actiwatch is used to program the recording unit, download data into storage, and engage a scoring algorithm that provides estimates of various sleep parameters. The epoch length (i.e., the sampling interval) for this study was set to one minute. The analysis interval was set to a period of 24 hours. Thus, total activity counts (TAC) include all one-minute epochs in a

24-hour period. Actiwatch software scores all epochs as either sleep or wake as determined by comparing activity counts for each given epoch, and those immediately surrounding it, to a set sensitivity threshold. For example, for a 1-minute epoch the activity counts of the surrounding 2 epochs are multiplied by 1/5, and the activity counts of the 2 epochs beyond those are multiplied by 1/25. These values are then added to the activity counts of the given epoch for a total count value. For the present study, activity counts that exceeded a threshold of 20 (Lichstein et al., 2006) were scored as activity. Activity counts less than or equal to the threshold were scored as sleep.

Participants were instructed to push the event-marker button on the device to mark occurrences such as bedtime and rise time. For those participants who failed to indicate these times, the sleep period was scored based on self-reported bed time and/or rise times on participant sleep diaries, which were recorded concurrently with the actiwatch period. The sleep variables used for analyses were total wake time (TWT), which was calculated by combining the sleep onset latency and wakefulness after sleep onset values and subtracting this from the time in bed value (i.e., TIB – [WASO + SOL]). Given our interest in mean daily activity levels, mean total activity (mean of the sum of all valid physical activity counts for all one minute epochs from start time to end time of a given interval) was also included.

3.3 Procedure

All participants took part in a screening assessment to assess for study eligibility. This screening interview took approximately one-hour in duration at the Sleep and Depression Lab, Ryerson University. During this one-hour session, participants were first informed of 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. After consent had

been obtained, trained MA- and PhD-level clinical psychology graduate students administered the SCID and DSISD in order to determine whether the participant met RDC and demographic eligibility requirements to partake in the study. If so, they were immediately invited to participate.

Next, participants completed a battery of self-report measures, including a demographics form (see Appendix B), ISI, DBAS and the DASS-21. Afterwards, they received training on how to complete the at-home monitoring portion of the study, including the SRM, sleep diary, and actiwatch. During this training for the SRM, each activity was reviewed and operational definitions for each activity was provided. Additionally, the sleep diary and actiwatch instructions were reviewed and the participants had an opportunity to ask questions. They were then provided with 14 days worth of SRMs and sleep diaries, as well as an actiwatch, to use over a two-week monitoring period. During this period, participants were asked to wear the actiwatch for fourteen days, complete the sleep diary every morning upon awakening, and complete the SRM every night before bed. After this two-week monitoring period, participants were asked to return to 105 Bond Street and surrender their actiwatch, completed sleep diary, and SRM. At that time, they were debriefed and compensated $50 for their time.

CHAPTER FOUR

RESULTS

4.1 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 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). The distribution of the ALI mean scores on the SRM did not fall within these parameters and was transformed using a log transformation. The distribution was re-checked to ensure that the distribution approximated normal.

4.2 Demographic Characteristics of Study Sample

The study population consisted of 80 participants. However, due to inconsistent and/or incomplete reporting on the SRM diary, 11 participants were removed from the final analyses. This final sample ($N = 69$) included ($n = 33$) those meeting RDC criteria for insomnia disorder and ($n = 36$) normal sleeper comparisons. The demographic and clinical characteristics of the sample are presented in Table 1. A Chi Square analyses was conducted in order to examine demographic group variables. The ID and NS did not differ significantly with respect to sex: χ^2 (1) = .07, p = .79, ethnicity: χ^2 (8) = 13.7, p = .09, marital status: χ^2 (5) = 4.4, p = .49, living arrangement: χ^2 (4) = 2.0, p = .73, or employment status: χ^2 (2) = 3.1, p = .22. An independent samples t-test revealed significant group differences for age: t (68) = -5.1, p = .001. The means of the self-report and actigraphy indices are presented in Table 2. As expected, groups differed on the DASS-D, ISI, DBAS, TWT, TST, and sleep efficiency (SE). The correlations among the sleep indices are reported in Table 3.

Table 1.

Participant Demographics

	Insomnia Disorder (n = 33)	Normal Sleeper (n = 36)
Sex (% female)	76	73
Age (*M, SD)*	47 (12)	32 (13)
Ethnicity (%)		
Aboriginal	3	0
African Canadian	3	8
Caribbean Canadian	3	3
European Canadian	79	52
Latin/Central/South American	0	6
East/South East Asian	6	3
South Asian	3	25
West Asian	3	0
Pacific Islander	0	3
Marital Status (%)		
Single	34	46
Married/common-law	44	38
Live-in Partner (< 2 years)	10	13
Divorced/Separated	9	0
Widowed	3	3
Living Arrangement (%)		
Alone	19	24

With spouse/partner	31	38
With spouse/partner & children	19	8
With family members	15	16
With friends/roommate	16	14
Employment Status (%)		
Unemployed	28	22
Part-time	18	38
Full-time	54	40

Table 2.

Group Comparison of Self-report and Actigraph Outcome Variables

	Normal Sleeper		Insomnia Disorder		Statistics	
Activity	Mean	*SD*	Mean	*SD*	*t*-statistic	*p*-value
DASS-D	1.38	3.09	8.81	8.48	-4.68	.001*
DBAS	2.36	1.25	6.29	1.67	-10.72	.001*
ISI	1.69	1.80	20.84	3.62	-25.99	.001*
TAC	346 904.94	89 778.88	317 046.82	93 907.45	1.16	.25
SE_{act} (%)	67.92	9.77	55.89	14.23	3.55	.001*
TWT_{act} (min)	163.86	58.29	223.61	71.63	-3.28	.002*
TST_{act} (min)	336.84	52.12	284.65	82.45	2.73	.009*
SE_{diary} (%)	92.43	0.04	70.86	10.55	10.42	.001*
TWT_{diary} (min)	37.20	23.40	147.00	53.40	-10.31	.001*
TST_{diary} (min)	447.60	37.80	343.20	54.00	8.62	.001*

Note. DASS-D = Depression subscale of the Depression Anxiety Stress Scale-21; DBAS = Dysfunctional Beliefs about Sleep; ISI = Insomnia Severity Index; TAC = total activity counts per day on the actiwatch; SE_{act} = actiwatch Sleep Efficiency; TWT_{act} = actiwatch total wake time; TST = actiwatch TST_{act}; SE_{diary} = sleep diary Sleep Efficiency; TWT_{diary} = sleep diary total wake time; TST = sleep diary TST_{diary}; *p > .001.

Table 3.

Correlations among Sleep Indices on Total Sample

Sleep Indices	*1*	*2*	*3*	*4*	*5*	*6*	*7*
1. ISI	--	.45**	-.38*	-.47**	.35*	-.72**	-.85**
2. TWT_{act}		--	-.76**	-.96**	.18	-.37*	-.36*
3. TST_{act}			--	.90**	-.12	.37*	.17
4. SE_{act}				--	-.21	.31**	.32*
5. TWT_{diary}					--	-.25	-.39**
6. TST_{diary}						--	.84**
7. SE_{diary}							--

Note. ISI = Insomnia Severity Index; TWT_{act} = actiwatch total wake time; TST = actiwatch TST_{act}; SE_{act} = actiwatch Sleep Efficiency; TWT_{diary} = sleep diary total wake time; TST = sleep diary TST_{diary}; SE_{diary} = sleep diary Sleep Efficiency; *$p > .01$, **$p > .001$.

4.3 Group Differences on the Social Rhythm Metric Indices

A multivariate analysis of covariance (MANCOVA) tested whether ID and NS differed on the three regularity-dependent measures (total SRM, SRM-A [with active social engagement], SRM-N [minimal or no social engagement]) resulting from the SRM diaries, while covarying for age (see Figure 2). Using Roy's largest root, there was a significant group effect on regularity [$F(3,50) = 3.21$, $p = .002$]. Follow-up ANCOVAs revealed a significantly lower total SRM score [$F(1,67) = 9.12$, $p = .004$] and SRM-N [$F(1,67) = 11.01$, $p = .001$] in the ID, relative to the NS (with lower scores indicative of increased irregularity). However, there was no significant group differences on the SRM-A [$F(1,52) = 0.92$, $p = .34$]. According to Cohen's (1977) classification of effect sizes, the effect size for the SRM total and SRM-N were large ($d = 0.74$ and $d = 0.82$, respectively). A MANCOVA that tested for group differences on the three activity level dependent measures (total ALI, ALI-A, and ALI-N) was not statistically significant [$F(2,55) = 0.82$, $p = .45$; see Figure 3]. Because the omnibus test was not significant, no follow-up ANCOVAs were conducted.

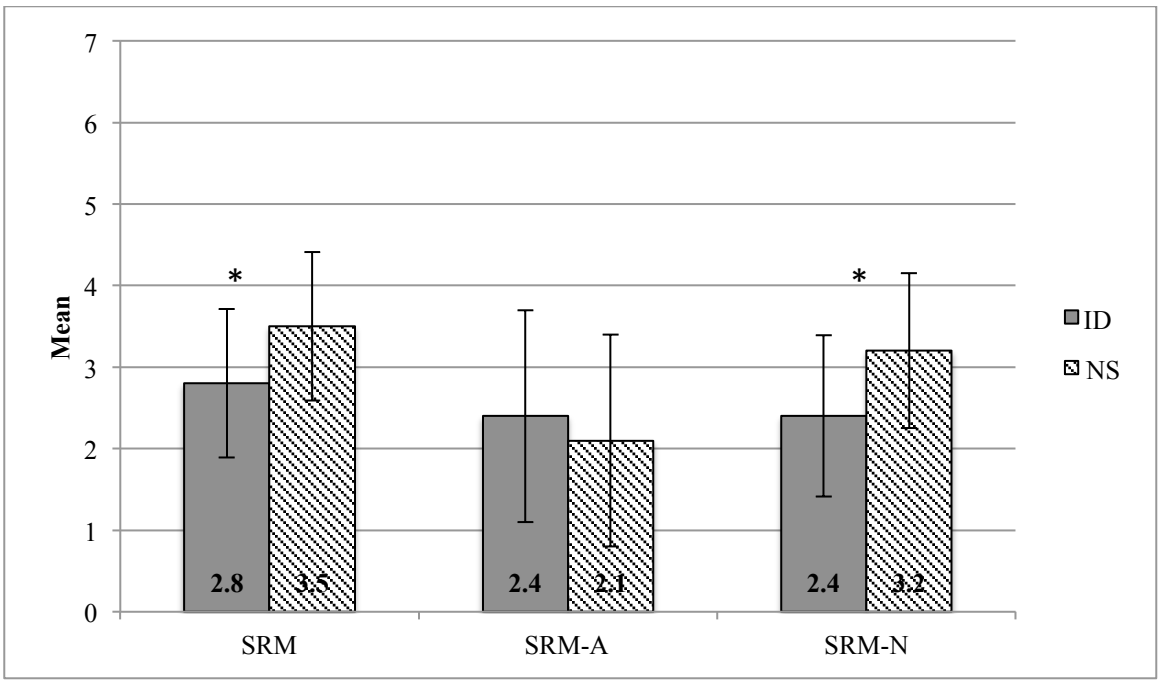


Figure 2. Group differences on mean SRM indices, while controlling for the effect of age; *$p < 0.01$, error bars represent one standard deviation above and below the mean.

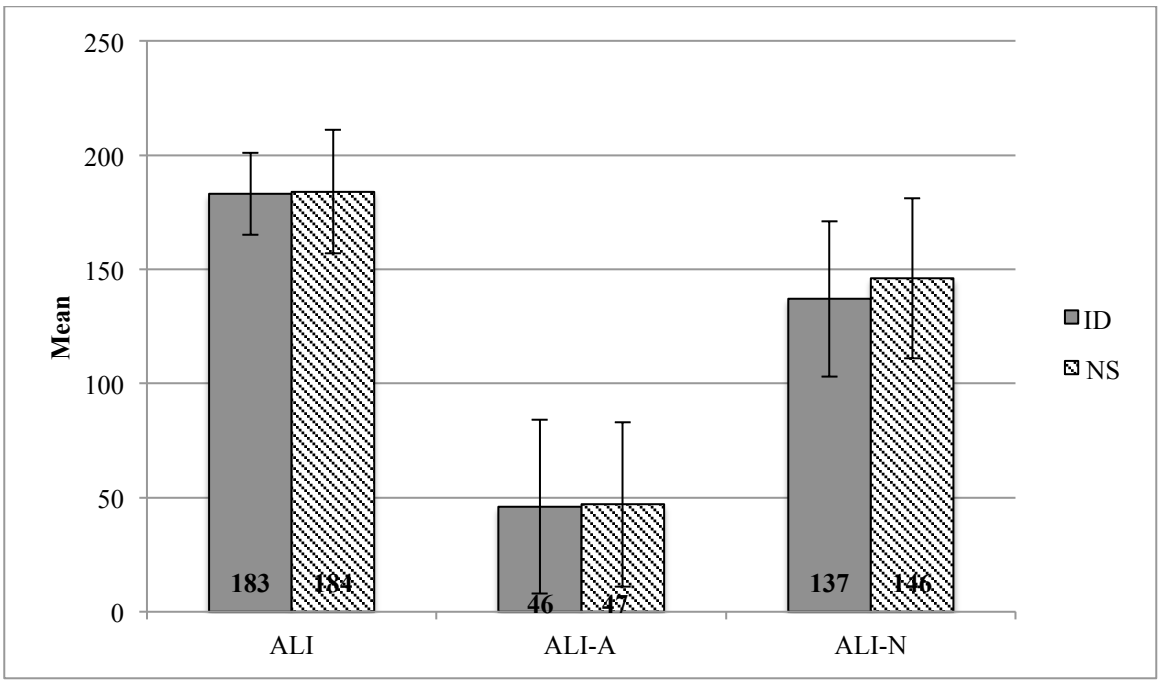


Figure 3. Group differences on mean ALI indices, holding age constant; Differences non-significant; error bars represent one standard deviation above and below the mean.

4.4 Group Differences on the Social Rhythm Metric Indices While Assessing for Dysphoria

In order to understand the potential influence of mood on daily regularity, dysphoria was included in a post-hoc hierarchical regression analysis. Given that depression has a high degree of overlapping symptomatology with insomnia (Carney et al., 2009), it is difficult to find a way to assess for the overlap of depression and insomnia without altering the variables to the point at which they are no longer meaningful. Miller and Chapman (2001) provide a particularly good exploration of the statistical issues in this type of situation. When using a quasi-experimental study design, such as the present study, the groups of interest (i.e. ID and NS) are naturally occurring groups and thus not randomly assigned. Because there is no random assignment, one cannot expect the covariate to be randomly distributed across the groups; indeed this is the issue, such that we would expect dysphoric symptoms to be higher in the ID group because of the overlap. Given that dysphoria and insomnia share variance, removing the variance associated with dysphoria (by entering the DASS-D subscale as a covariate in a MANCOVA) would not provide meaningful results because it alters the group in an artificial way. Picture a venn diagram with overlapping circles and imagine cutting out the overlap between the two, what is left is a portion of the variable; missing is a potentially important part of the variable. Thus, it is convention to refrain from the use of a covariate in such situations (Miller & Chapman, 2001) so as not to alter the variable by removing the overlap in quasi-experimental designs. Statistically valid alternatives to using an ANCOVA include assessing for interaction effects, in order to understand the potential role of dysphoria and its interaction with daily activity regularity in these groups. Thus, to test whether dysphoria contributes to insomnia symptom severity (ISI total), above and beyond that of daily activities and social rhythms, hierarchical linear multiple regression analyses were conducted. All participants (N = 64) were included in this analysis,

such that they were not separated by diagnostic categories (i.e., ID and NS). For all variables in each model, the Tolerance values were > 0.10 and the Variance Inflation Factor (VIF) values were < 10.0, which excluded multicollinearity (Myers, 1990).

The SRM total was entered in the first step of the hierarchical linear regression analysis, and the DASS-D total score was entered on the second step. As expected, the first step was significant (p = .002); thus, SRM significantly predicts insomnia symptom severity (ß = -3.63, *p* = .002). In step 2, dysphoria (as measured by the DASS-D) added to the prediction of insomnia (F change = 16.03; *p* = .001); the model accounted for 30% of the variance (DASS-D: ß = 0.68, *p* = .001). To determine if the sleep and regularity relationship depended on the level of dysphoria, an interaction term was calculated by multiplying the SRM and DASS-D. The interaction term for the SRM and DASS-D (i.e., the test of moderation) was entered on the third step. The interaction term in the final step was not statistically significant (*p* = .07; see Table 4).

Table 4.

Hierarchical Multiple Regression Analyses Predicting Insomnia Severity on the ISI

			Model Outcome		
Predictor	ß	*p*	Adjusted R^2	*F* change	*p*
Step 1			0.13	10.24	.002*
SRM	-.3.62	.002*			
Step 2			0.30	16.03*	.001*
SRM	-0.91	0.46			
DASS-D	0.68	.001*			
Step 3			0.33	3.29	.074
SRM	-2.07	0.13			
DASS-D	0.16	0.65			
DASS-DxSRM	0.22	0.07			

Note. SRM = Social Rhythm Metric Total; DASS-D = Depression subscale of the Depression Anxiety Stress Scale -21; *p < .05.

4.5 Group Differences on the Mean Variability in the Timing of Daily Activities

In order to investigate whether the variability in the timing of social rhythms was important for SRM activities, intra-subject standard deviations for the time of day that each activity took place over the two-week monitoring period were calculated. Consistent with previous regularity research (Carney et al., 2006; Monk et al., 2003), an a priori decision was made to exclude activities that seldom occurred (i.e., reported by less than two-thirds participants), as these were unlikely to influence sleep. Using this cutoff, slightly less than one standard deviation, permits exploration of regularity (i.e., by excluding activities that occurred less often) and is a good starting point for a less developed area of research. This resulted in the exclusion of "Afternoon Nap" (done at least three time per week by only 9% of participants), "Exercise" (done at least three time per week by only 53% of participants), "Watch News Program" (done at least three time per week by only 64% of participants), and activity A, as this was an idiosyncratic activity that varied for each participant. Visual inspections of the distribution histograms and Shapiro-Wilk tests revealed that this data was not normally distributed. Thus, this data was transformed using the log transformation, which was successful in normalizing the distributions.

The transformed variables were analyzed using a MANCOVA to investigate the intra-subject variability of daily activities between groups, while controlling for the effect of age. A MANCOVA tested whether ID and NS differed on the variability in timing of daily activities resulting from the SRM diaries (see Table 5 for means and standard deviations). Using Roy's largest root, there was a significant group effect on variability of these activities [$F(1,48) = 2.29$, $p = .02$]. Follow-up ANCOVAs revealed significantly lower variability in timing of getting out of bed [$F(1,69) = 14.97$, $p = .001$], the first beverage [$F(1,69) = 20.80$, $p = .001$], breakfast

[$F(1,69) = 20.25$, $p = .001$], lunch [$F(1,69) = 4.64$, $p = .03$], evening snack [$F(1,69) = 9.08$, $p = .004$], watching television [$F(1,69) = 5.55$, $p = .02$], and getting into bed [$F(1,69) = 4.93$, $p = .03$] in the NS group, relative to the ID. However, there were no observed significant group differences in timing variability of going outside for the first time [$F(1,69) = 3.83$, $p = .05$], first contact [$F(1,69) = 3.67$, $p = .06$], start work [$F(1,69) = 1.63$, $p = .21$], use the phone [$F(1,69) = 3.34$, $p = .07$], use the computer [$F(1,69) = 0.71$, $p = .40$], return home [$F(1,69) = 1.95$, $p = .17$],or dinner [$F(1,69) = 0.01$, $p = .91$]. A MANCOVA tested whether ID and NS differed on mean timing of daily activities resulting from the SRM diaries [$F(1,52) = 1.10$, $p = .38$]. No follow-up ANCOVAs were conducted, since this test was non-significant (see Table 6 for means and standard deviations). Thus, overall, those in the ID group exhibited increased variability in a variety of daily activities in comparison to NS, but the average timing of daily activities did not significantly differ.

Table 5.

Group Comparison on Intra-Subject Variability of Daily Activities

Activity	Normal Sleeper		Insomnia Disorder		*F* statistic	*P* value
	Mean	SD	Mean	SD		
Out of Bed	1:02	0:33	1:26	0:48	14.97	.001*
First Contact	1:22	0:51	2:00	1:21	3.67	.06
Morning Beverage	1:08	0:35	1:38	1:03	20.80	.001*
Breakfast	1:06	0:28	1:44	1:07	20.25	.001*
Outside	2:08	1:06	2:22	1:16	3.83	.05
Start Work	1:39	1:15	1:57	1:04	1.63	.21
Phone	2:08	1:31	3:02	1:09	3.34	.07
Lunch	1:11	0:43	3:01	7:45	4.64	.03*
Return Home	2:49	1:14	2:45	1:10	1.95	.17
Dinner	1:18	0:46	1:33	1:27	0.01	.91
Computer	3:16	1:27	3:34	1:10	0.71	.40
Watch television	3:02	1:44	3:48	2:38	5.55	.02*
Evening Snack	1:44	1:26	2:55	3:13	9.08	.004*
Into Bed	0:57	0:31	1:17	0:57	4.93	.03*

Note. SD = standard deviation; means are the mean intra-subject standard deviations and the SD is the standard deviation of the mean values; *$p < 0.05$.

Table 6.

Mean Time for Activities by Sleeper Status

Activity	Normal Sleepers		Insomnia Disorder		
	Mean	SD	Mean	SD	*p*-value
Out of Bed	8:15	1:14	8:29	1:36	.76
First Contact	9:07	1:16	9:11	1:44	.96
Morning Beverage	8:49	1:18	9:01	1:18	.98
Breakfast	9:19	1:18	9:34	1:22	.89
Start Work	11:03	2:14	10:20	1:49	.74
Outside first time	9:55	1:23	10:51	2:10	.19
Lunch	13:25	0:40	14:22	3:18	.37
Phone	10:25	2:51	11:20	2:16	.57
Return Home	19:17	2:04	19:48	2:17	.21
Watch television	17:04	3:55	16:35	3:52	.55
Computer	11:44	3:00	13:02	3:39	.12
Evening Snack	20:49	1:39	19:49	2:21	.05
Into Bed	23:23	1:01	23:36	1:29	.69

Note. Means are the mean time the activity was performed over 14 days; SD = standard deviation.

4.6 Examination of Actigraph Sleep Indices and Daily Activities

Of the original sample, 73% of participants had utilizable actigraphy data (i.e., data that could be scored), resulting in a total of $N = 50$ individuals in the final actigraphy analyses (NS: $n = 27$; ID: $n = 23$). In order to test the secondary hypothesis, specifically, the proposed reciprocal relationship between daily activities and sleep among those with those with insomnia (see Figure 4), a Multilevel Modeling (MLM) technique, also referred to as HLM (Bryk & Raudenbush, 1992), was employed. Given the hierarchical nature of the data (i.e., 14 consecutive days of monitoring nested within participants), this data analytic technique was appropriate as it is used to address a nested data structure. Moreover, this approach has the ability to capture the fluctuations across time in sleep and activity patterns. MLM can also handle missing data, which is important when dealing with monitoring across several days. MLM is an extension of the general linear model and does not require observations to be independent. Accordingly, this is a flexible approach and appropriate for daily data observations nested within each participant (McCrae et al., 2008; Willett, Singer, & Martin, 1998; Zautra, Johnson, & Davis, 2005).

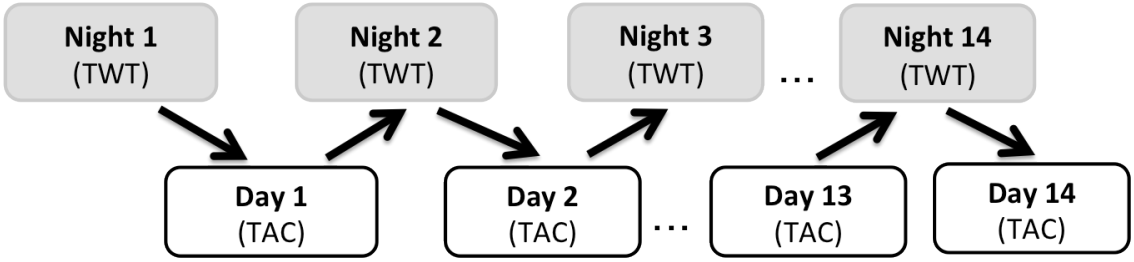


Figure 4. Proposed reciprocal relationship between daily total activity counts (TAC) and total wake time (TWT) across 14-day monitoring period.

Objective actigraphy indices were utilized using this MLM approach, using the MIXED Procedure in SAS (version 9.3). All models were estimated using the Residual Maximum

Likelihood (REML) method. The capability of a model to predict the dependent variable (i.e., TWT or TAC) superior to a baseline (null) model was used as an index of Goodness of Fit. The proportional reduction of within- and between-person residual variances were compared to this baseline model in order in order to determine whether improvements in predictability were made (Bryk & Raudenbush, 1992). A reduction in residual variances represents a proportional decrease of the prediction error, which is equivalent to R^2, and used as an estimate of effect size. The effect of different error structure specifications on model fit was tested and the different error structures had little effect on the fixed and random parameter estimates or their pattern of significance (Singer & Willett, 2003).

4.6.1 Creating predictor variables.

Prior to testing the hypotheses of this study, all variables were person-mean centered. In order to person-mean center TAC and TWT, a mean of each variable was calculated for each participant. Each participant's mean on a particular variable was then subtracted from all of his or her daily ratings on that specific variable. The resulting person-mean centered variables then had a meaningful zero point, such that when the variable is equal to 0, the value for that day is equal to their overall average score across the 14 day period. By performing this person-mean centering procedure, both the person-mean centered predictors (TWT or TAC) and the person mean (TWT or TAC) could be included in each model. Specifically, in these models, the person means of each variable represent each person's mean amount of TWT or TAC for the 14 days, while the person-mean centered variables represented each participant's daily fluctuation around his or her own mean TWT or TAC. As person-mean centered predictors and person means are orthogonal, including both types of predictors in the model allows the within-person and between-person effects to be disentangled more simply.

4.6.2 Creating models to evaluate hypotheses.

In order to test the hypothesis regarding the reciprocal relationship between night (i.e., TWT) and day (i.e., TAC), multilevel models were estimated using both within- (level 1) and between- (level 2) individuals. In this model, Level 1 consists of within-person predictors (i.e., predictors that varied within persons and for which there was a new value at each occasion of measurement). The predictor was each person's centered daily score (e.g., daily TWT score – person's TWT mean), which signified each person's daily deviation from their average value on the predictor. At this level, the dependent variable TAC was modeled as function of person-mean centered TWT and the interaction between sleeper status and person-mean centered TWT. Level 1 analyses addressed questions such as: 'On nights in which a person reports a total wake time above their specific average total wake time, does this person also engage in fewer total psychomotor activities the following day?'

Level 2 consists of between-person predictors only for which there was only one value per person (i.e., for the variables of TWT, the Level 2 predictor was each person's mean TWT across the 14 days). These predictors do not vary within a given individual but vary across individuals. At this level, the mean level of the dependent variable TAC for an individual was modeled as a function of person-mean amount of TWT, sleeper status, and their interaction (mean TWT by sleeper status). Level 2 analyses examined questions such as: 'Do people who are generally awake more of the night report lower levels of daily psychomotor activities?'

In terms of building the model to be tested, the first step included the null model (i.e., with no predictors), which outlined the variance to be explained and functioned as the baseline against which subsequent models would be used to calculate variance explained and improvement in fit. Next, potential baseline and demographic variables covariates (i.e., age,

DASS-D, and DBAS) were entered in the model as predictors of TAC. Also, the effect of time (i.e., Day) was entered into the model in order to control for any overall temporal trends in the data. However, among these variables, Day (i.e., Day of monitoring 1 – 14) was the only significant predictor of TAC and, thus, the only variable included in later models. Therefore, in the next step, TWT was estimated without the non-significant baseline and demographic variables (i.e., age, DASS-D, and DBAS). A Level 1 effect (i.e. the association between participants' day-to-day deviations from their mean level of TWT and their day-to-day variation activity level) and a Level 2 effect (i.e. whether there were significant between-person differences in the strength of the day-to-day sleep and activities relationship) were estimated. Lastly, the association between a participant's mean level of TWT and TAC, in addition to sleeper status and mean TAC, were estimated.

Using the empty model with no predictors, the intraclass correlation coefficient (ICC), which provides an index of within- and between-person variability to be explained, was 0.66. The ICC indicates that 34% of the overall variability in activity counts is a within person phenomenon and 66% is a between person occurrence. Accordingly, an MLM analytical framework, which separates within- and between-person variance components, appears to be warranted.

In the final model, the following effects were included as predictors of TAC: intercept, Day, TWT centered, TWT mean, sleeper status, and the interaction between TWT centered and sleeper status. TWT was not a significant predictor at the within-person level (Level 1); however, Day was a significant predictor (β = 3510.92, t (590) = 2.86, p = .004), such that a linear temporal trend in TAC exists (i.e. TAC increases throughout the two-week monitoring period). At the between-group level (Level 2), TWT and the interaction between TWT and sleeper status

were not significant predictors of TAC. See Table 7 for a total listing of predictor estimates, significance levels, variance explained, and model parameters for the final MLM.

Table 7

Results from the Analyses Modeling Daytime Activities as a Function of Sleep

Fixed Effect	**B**	**SE**	**df**	***t***	***p***
Intercept	336705.00	38780.00	48	8.68	<.0001
Day	3510.92	1228.26	590	2.86	0.004*
TWT centered	130.50	90.26	590	1.45	0.15
TWT mean	-93.82	203.57	48	-0.46	0.65
Sleeper status	-22365.00	28686.00	48	-0.78	0.44
Sleeper status by TWT centered	-183.89	124.11	590	-1.48	0.14
Random Effect		**Estimate**	***SE***	***z***	***p***
Random Intercept Variance		7.3678E9	1.7463E9	4.22	<.0001
Level-1 Residual Variance		1.436E10	8.3631E8	17.17	<.0001

Note. TWT = actiwatch total wake time; Sleeper status by TWT centered = the interaction between sleeper status and average TWT; *$p < .05$.

Similarly, the reciprocal research question was tested, such that Level 1 determined: 'On days in which a person reports fewer psychomotor activities than they generally participate in, does this person experience increased total wake time that night?' Level 2 analyses examined questions such as: 'Do those who are generally more active during the day report better sleep (i.e., decreased TWT)?' The levels were the same as the aforementioned analysis, such that Level 1 effects were estimated using predictors that varied within persons, and for which there was a new value at each occasion of measurement. Thus, the predictor was each person's centered daily score (i.e. daily TAC score – person's TAC mean). The Level 2 effects were estimated using predictors, which varied between persons, and for which there was only one value per person (i.e., for the variables of TAC, the Level 2 predictor was each person's mean TAC). The first step of the model included the null model (i.e., no predictors). Next, the effect of TAC was estimated. A Level 1 effect (i.e. the association between participants' day-to-day deviations from their mean level of TWT and their day-to-day variation activity level) and a Level 2 effect (i.e. the association between a participant's mean level of TAC and TWT and sleeper status and TWT) were estimated.

The intraclass correlation coefficient (ICC) of the empty model with no predictors was 0.63. Thus, the ICC indicates that 37% of the overall variability in activity counts is a within person phenomenon and 63% is a between person occurrence. None of the baseline demographic variables or Day were significant and, thus, not included in the subsequent model. In the final model, the following effects were included as predictors of TWT: intercept, TAC centered, TAC mean, sleeper status, and the interaction between TAC centered and sleeper status. TAC was not a significant predictor at the within-person level (Level 1). At the between-group level (Level 2), TAC was not a significant predictor of TWT, however, sleeper status was a

significant predictor of TWT. The interaction between TAC and sleeper status was also non-significant. See Table 8 for a total listing of predictor estimates, significance levels, variance explained, and model parameters for the final MLM.

Although it was hypothesized that increased TWT on night one would directly influence the following days' TAC and that days' TAC would directly influence that nights' TWT in those with ID, this finding was not confirmed. A relationship between TWT and subsequent TAC was not expected in the NS group and, indeed, this was confirmed. The only observed significant predictor of activity counts on the actiwatch was Day, such that activity levels increased throughout the two-week monitoring period.

Table 8

Results from the Analyses Modeling Sleep as a Function of Daytime Activities

Fixed Effect	**B**	**SE**	**df**	***t***	***p***
Intercept	190.89	39.37	48	4.85	<.0001
TAC centered	-0.000009	.00003	530	-0.27	0.79
TAC mean	-0.00007	0.0001	48	-0.68	0.50
Sleeper status	57.03	19.29	48	2.96	0.005*
Sleeper status by TAC	-0.00007	0.00006	530	-1.16	0.25
Random Effect		**Estimate**	***SE***	***z***	***p***
Random Intercept Variance		3929.62	918.76	4.28	<.0001
Level-1 Residual Variance		6235.21	382.01	16.28	<.0001

Note. TAC = actiwatch total activity count; Sleeper status by TAC centered; the interaction between sleeper status and average TAC; *$p < .05$.

4.7 Examination of Beliefs as Potential Mediator of Sleep and Daily Activities

A bootstrapping mediation analysis (Hayes, 2009) was employed in order to determine whether beliefs about sleep indirectly account for the relationship between sleep and daytime activities on the SRM. This analysis is one of the more valid and powerful methods for testing intervening, or indirect variable effects (Mackinnon, Lockwood, & Williams, 2004; Williams & Mackinnon, 2008) and was conducted separately between diagnostic sleep groups. Sleep (TWT on the actiwatch) was entered as the predictor variable and total activities on the actiwatch (TAC) were entered as the dependent variable. The indirect effect of dysfunctional beliefs about sleep (DBAS) on the relation between sleep and activity was tested using a confidence level of 95 resampled 10, 000 times. Although it was originally hypothesized that dysfunctional beliefs about sleep would significantly mediate the relationship between sleep and daily activities in the ID, but not the NS group, this relationship was not found for either sleep group. Specifically, an indirect effect of the DBAS on the relation between TWT and TAC was not observed in the ID (bias corrected confidence interval = -.282.7 to 80; standard error = 88.7) or the NS groups (bias corrected confidence interval = -69.2 to 675.43; standard error =164.68).

4.8 Post-hoc Analyses

Although not a stated objective of the current study, an exploratory post-hoc test specific to the SRM was employed. A bootstrapping mediation analysis (Hayes, 2009) was utilized in order to determine whether beliefs about sleep indirectly account for the relationship between daytime regularity and insomnia severity. Regularity (SRM total from the SRM diary) was entered as the predictor variable and insomnia severity (ISI) was entered as the dependent variable. The indirect effect of dysfunctional beliefs about sleep (DBAS) on the relation between regularity and insomnia severity was tested using a confidence level of 95 resampled 10 000

times. An indirect effect of the DBAS on the relation between SRM and ISI was observed (bias corrected confidence interval = -5.30 to -1.99; standard error = .84; p =.0002; see Figure 5), such that these beliefs were found to indirectly account for the relationship between daytime regularity and insomnia severity. Thus, those individuals with increased regularity appear to be less rigid in their sleep beliefs and perceive their sleep to be less disrupted on a retrospective questionnaire inquiring about insomnia severity.

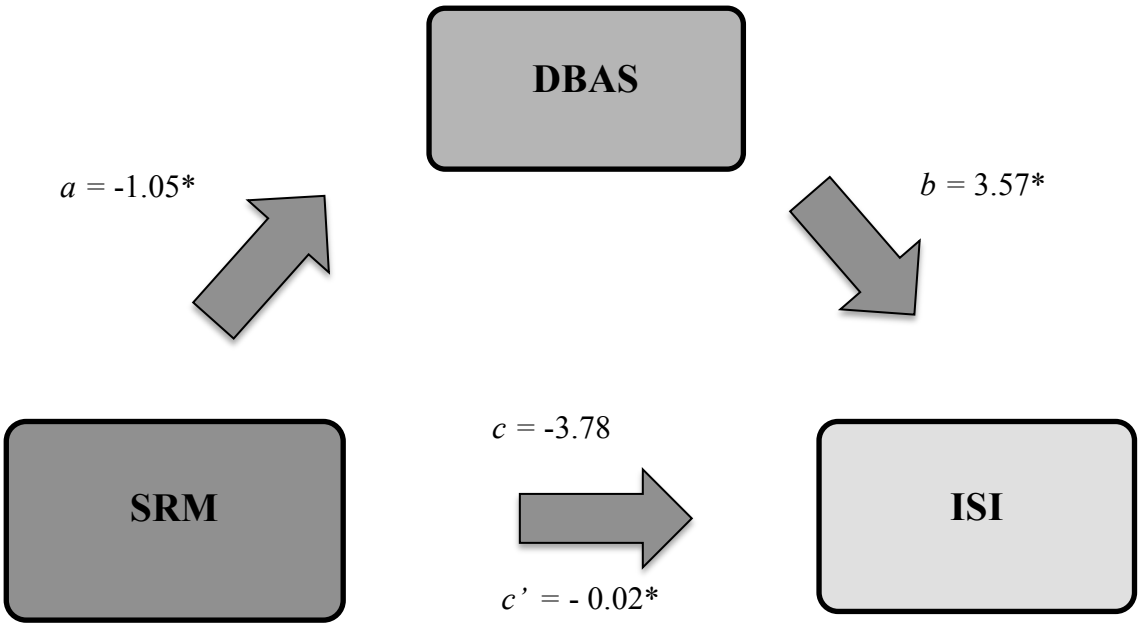


Figure 5. Indirect effect of DBAS on the relation between SRM and ISI. Coefficients for each path listed beside corresponding arrow; *p < .001. DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; SRM = Social Rhythm Metric; ISI = Insomnia Severity Index.

CHAPTER FIVE

DISCUSSION

5.1 Synthesis of the Literature and Findings from Current Study

The current study investigated whether social rhythms are related to sleep. Specifically, this study compared presumably reliable daytime activities (i.e., amount, timing and regularity) in those with and without insomnia. This study was the first in the area of social rhythms and sleep to utilize a clinical insomnia population and have participants prospectively monitor their sleep and daily activities for a two-week period, using both self-report and objectives measures. The main hypothesis, that those with insomnia would exhibit decreased regularity in comparison to the normal sleeper group, was confirmed. The specific results, limitations of the current study, and clinical implications will be discussed in detail.

Social rhythms have previously been investigated for their role in mood in various populations, including healthy young adults (Monk et al., 1994), older adults (Monk et al., 1992), general adult population (2003), both depressed and non-depressed elderly bereaved persons (Brown et al., 1996; Prigerson et al., 1996), depressed individuals in remission (Monk et al., 1991), individuals with an anxiety disorder (Shear et al., 1994), and new parents (Monk et al., 1996). However, in this study, social rhythms were presumed to be important for sleep. Indeed, in the current study, in contrast to normal sleepers, those with clinical levels of insomnia showed disrupted social rhythms (i.e., the timing and engagement of everyday activities was less regular). This finding parallels the extant literature on the relationship between regularity and sleep quality, in which good sleepers demonstrated increased regularity in comparison to poor sleepers (Carney et al., 2006a; Monk et al., 2003). Additionally, research focused on manipulations of daily routines has also demonstrated changes in circadian rhythmicity. For example, Naylor and

colleagues (2000) enforced a schedule of structured social and light physical activity among elderly residents of continued-care retirement facility. Compared to the control group, those exposed to structured activities exhibited increased amounts of slow-wave sleep (i.e., deep sleep) and demonstrated improvements in memory-oriented tasks after the enforced schedule intervention. Other researchers have looked at the effects of Ramadan on circadian rhythms, as this is a month characterized by daytime fasting, a delay and shortening of night-time sleep, and changes in behaviour and social habits (Al-Hadramy, Zawawi, & Abdelwahab, 1987; Bogdan, Bouchareb, & Touitou, 2001). During this period, participating individuals demonstrate changes in the rhythmic pattern of various hormones, including decreased amplitude of melatonin (Bogdan et al., 2001). As such, regularity of daily routines appear to have important implications for the circadian system, as it has the ability to differentiate between those with and without insomnia and can be measured via physiological assays. These combined findings provide further support for the importance of social zeitgebers in daily life.

Several studies have shown a positive association between daily activity level and sleep, such that that poor sleep can be a consequence of inactivity or a sedentary lifestyle (Carney et al., 2006a; Morgan, 2003; Ohayon et al., 2001; Sherrill et al., 1998). Thus, it was hypothesized that in the current study, those with insomnia would engage in fewer daily activities than normal sleepers, which was not confirmed. This non-significant finding was unexpected. Overall, both those with and without insomnia in the current study participated in a larger number of daily activities than in previous investigations, which may be linked to the wider age range and fewer academic-related obligations (i.e., this was not an academic sample). However, it is important to recognize that within the literature, sleep studies have used a variety of activity definitions ranging from exercise participation (Sherrill et al., 1998) to occupational (Ohayon et al., 2001),

recreational (Janson, Lindberg, Gislason, Elmasry, & Boman, 2001), social (Habte-Gabr et al., 1991), and personal maintenance (Newman, Enright, Manolio, Haponik, & Wahl, 1997) activities. Given that these activities can vary in energy cost and physiological impact, one cannot assume that their impact on sleep represents a collective physiological mechanism (Morgan, 2003). In looking at those with major depressive disorder, a clinical group who has generally been found to exhibit decreased activity levels (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011; Martell, Dimidjian, & Herman-Dunn, 2010), a recent study reported similar levels of regular daily activities between those with and without depression (Stetler, Dickerson, & Miller, 2004). This study also measured daily activities using the SRM, which further suggests that the SRM may not always be able to capture group differences. Thus, although based on the current findings one may be tempted to conclude that daily activity level is not integral for good sleep, it is essential to consider that recording the number of daily activities using the SRM may not be appropriate; perhaps the use of other methodologies could provide a better estimate of this construct, which may have found differences between those with and without insomnia.

The current study also suggests that whether there was social engagement in daily activities is less important for both the number and regularity of activities. Increased regularity was found to hold true for normal sleepers, even for those activities with no active social engagement (e.g., eating breakfast alone, watching television while someone else is home). When alone, regularity appears to be preserved for normal sleepers, which is not the case for those with insomnia. This finding is disparate to previous research, which found that, in comparison to poor sleepers, good sleepers displayed more overall regularity and increased regularity with active social engagement (Carney et al., 2006a). It may be that amongst an undergraduate population, generally a time period characterized by irregularity, activities with

others serve to anchor students' daily routines. One would expect others to help keep naturally "irregular individuals" on a more socially acceptable schedule. For example, it would be unconventional to meet others for dinner at 11:00 pm in North America and, therefore, someone with a delayed chronotype who may be inclined to eat dinner later in the evening (i.e., someone whose day it typically delayed in comparison to the norm), would eat at a more "acceptable" time. However, the current study did not find this to be true. Aside from social interaction acting as an anchor to regulate daily schedules, social engagement could influence sleep via the arousal system. Conceivably, decreased social engagement and activities could increase negatively valenced emotions and arousal, thus having negative consequences on sleep (Mistlberger & Skene, 2005). Scheduling social activities in the elderly has been linked to improved sleep (Naylor et al., 2000), but again, it is unclear whether this was a result of the social interaction or the regular schedule. Countless studies have found a link between sleep complaints and those who are socially isolated or dissatisfied with their relationship (Edinger et al., 1988; Marchini et al., 1983; Ohayon et al., 2001). However, further research into this relationship is necessary, as it is unclear at this point whether this is a result of the arousal or circadian system, or if this is related to other mechanisms (i.e., overall mood). Findings from the current study, using the SRM, suggest that social engagement is less important.

In summary, the regularity of daily activities, rather than the number of activities, appears to be the most influential for sleep. Thus, further attention was paid to the variability in the timing of social rhythms. Data from the current study reveal that daily activities occur more regularly among normal sleepers compared to those with insomnia. Specifically, normal sleepers have less variability in the timing of their rise and bedtimes, their morning beverage and breakfast, evening snack, lunch, and time of watching television. This finding parallels previous

research that found normal sleepers to have less variability in rise and bedtimes, in addition to their morning beverage, when compared to poor sleepers (Carney et al., 2006a). In this current study, there was approximately a 20-minute difference in variability of both rise and bed times between normal sleepers and those with insomnia. The insomnia group had over an hour of daily variability for both the rise and bedtimes, which can contribute to social jetlag and can produce daytime symptoms comparable to those caused from insomnia (e.g., fatigue, irritability, etc.; Taylor et al., 2008; Wittmann et al., 2006; Yang & Spielman, 2001; Yang et al., 2001). Is it well established within the literature that those with insomnia have more variability of rise times and bedtimes and, in fact, regulating rise times is already a treatment target included in CBT-I (Bootzin, 1972b; Morin, 2004; Morin et al., 1994; Morin et al., 1999). Although it is difficult to set a regular bedtime as it could exacerbate the insomnia to have people going to bed prior to feeling sleepy, part of CBT-I includes setting a regular earliest bedtime (i.e., the earliest time the individual can get into bed). Findings from the current study further highlight this essential CBT-I target of scheduling into and out of bed times. Although the current study design does not provide information on causality, one could speculate that less variable rise and bedtimes, even as minimal as 20 minutes, could have sleep benefits and should be considered for future research.

Those with insomnia in the present study also displayed increased variability of meal times in comparison to normal sleepers. Specifically, those with insomnia were more variable with respect to the timing of the morning beverage, breakfast, lunch, and evening snack. Meals are an important zeitgeber for the circadian rhythm. In fact, research conducted over a quarter century ago highlighted the relative importance of meals. Specifically, Aschoff et al. (1986) found that the gap in timing between meals was predictive of the subjective day length as a whole in free-running conditions (e.g., conditions without light cues). While those with insomnia

in the current study displayed increased variability in the timing of their meals, lunch was the most variable meal (i.e., three hours), with over two hours more variability than normal sleepers. It may be that those with insomnia have a difficult time identifying hunger cues due to their overall irregularity and disruption in the circadian system or that their more variable rise time leads to more uncertain times of feeling famished. Regardless, this finding highlights the potential importance of regular mealtimes, something that is not currently a treatment target in CBT-I. One could theorize that incorporating a regular meal schedule would help to entrain the internal clock, thus, contributing to healthier sleep.

A final noteworthy finding is that those with insomnia in the current study had evening routines characterized by more irregularity. For example, their evening snack had a range of almost three hours and almost four hours for watching television. Aside from the potential role of food on entraining the circadian system, it would be interesting to know what type of food the normal sleepers were consuming as foods high in the amino acid tryptophan (TRP; e.g., milk, turkey, peanuts, cheese, yogurt) can help to facilitate sleep (Southwell, Evans, & Hunt, 1972). This would be of particular interest given that the normal sleepers ate their evening snack, on average, one hour later than those with insomnia (i.e., closer proximity to bedtime). Consuming foods with TRP is often encouraged in CBT-I as part of healthy sleep hygiene (King, Dudley, Melvin, Pallant, & Morawetz, 2001; Stepanski & Wyatt, 2003).

Given that rigid sleep beliefs are common among those with insomnia and, in fact, are considered to play a significant role in the maintenance of the disorder (Carney & Edinger, 2006b; Carney et al., 2011a; Edinger et al., 2001), a closer examination of dysfunctional beliefs about sleep and whether they contribute to daily regularity was employed. These beliefs were found to indirectly account for the relationship between daytime regularity and insomnia severity.

Specifically, those individuals with increased regularity appear to be less rigid in their sleep beliefs and perceive their sleep to be less disrupted on a retrospective questionnaire inquiring about insomnia severity. For example, if an individual endorses the belief, "When I don't get the proper amount of sleep on a given night, I need to catch up the next day by napping or the next night by sleeping longer," this could result in them sleeping in and, consequently, disrupt their daily routine. This finding is noteworthy, as it further highlights that importance of beliefs in insomnia and suggests that cognitive factors are also important in this behavioural process of social rhythms. Clinically, this finding is important as maladaptive beliefs about sleep are already targeted as part of CBT-I (Morin, 2004, 2010). Given the aforementioned findings about the importance of regularity, it appears as though both regulating daily activities and targeting dysfunctional beliefs about sleep (e.g., via thought records, a common cognitive tool used to challenge beliefs) are important sleep targets for CBT-I.

Outside of the insomnia literature, social rhythms have received considerable attention in the area of mood disorders (Ehlers, Frank, & Kupfer, 1988; Harvey, 2008; Haynes, Ancoli-Israel, & McQuaid, 2005; Murray & Harvey, 2010). In fact, the etiology of depression has been tied to social zeitgebers, such that major life events are hypothesized to disrupt zeitgebers, which can dysregulate the circadian system and precipitate depressive episodes in vulnerable individuals (Ehlers et al., 1988). Given that insomnia is a disorder that commonly occurs alongside major depressive disorder (Buysse et al., 1994; Giannotti et al., 2002), further analyses were employed to determine whether the above findings are purely a result of the dysphoria, rather than the insomnia. Both insomnia severity and dysphoria were found to be predictors of regularity, but dysphoria accounts for a larger proportion of the variance. There was no interaction effect; thus, the relationship between insomnia and social rhythms is not dependent on the level of dysphoria.

It appears as though regularity is an important predictor of insomnia severity, regardless of dysphoria. However, these findings confirm the existing literature, such that dysphoria largely contributes to disturbed sleep. Further research into understanding the potential role of dysphoria and its interaction with social rhythms in insomnia is required before conclusions can be made. This finding highlights the potential importance of regularity as a treatment target among those individuals with insomnia, even amongst those without a co-occurring mood disorder.

It was also hypothesized that there would be a negative reciprocal relationship among actiwatch-measured sleep and actiwatch-measured activity counts (i.e., poor sleep on night one would directly influence the following days' activity levels and that days' activity levels would directly influence that nights' sleep) in those with insomnia. However, this finding was not confirmed. A relationship between sleep and subsequent daytime activity level was not expected in the normal sleepers and, indeed, this was confirmed. The only observed significant predictor of activity counts on the actiwatch was Day, such that psychomotor activity levels increased throughout the two-week monitoring period. It may be that when participants are mindful of monitoring their activities, it affects their activity level such that it increases throughout the monitoring period (Nelson & Hayes, 1981). The only observed significant predictor of wakefulness was sleeper status, such that those with insomnia experience more wakefulness throughout the night when compared to normal sleepers, a finding already well-established in the literature. Essentially, a reciprocal relationship between total daily activity level and level of sleep disruption, as measured by the actigraph, does not appear to exist. It seems as though, in general, total activity level is less important than the regularity of activities. However, as will be discussed in detail below, the actigraph is not without its limitations and these results should be interpreted with caution. Finally, although maladaptive beliefs such as, "After a poor night's

sleep, I know it will interfere with my activities the next day," were hypothesized to mediate the supposed relationship between sleep and daily total activities in those with insomnia, this was, in fact, not the case. As such, beliefs about sleep do not contribute to actiwatch-measured total activity counts. Given that daily activity level, as measured by these particular self-report and objective methods, do not appear to be as important for sleep as initially hypothesized, this finding that beliefs do not play a role is less important.

5.2 Limitations & Future Directions

It is important to consider the findings of this study in light of several possible limitations. In regards to the study sample, this was a mostly female sample (over 70%). Although insomnia often afflicts women more than men (Zang & Wing, 2006), the larger proportion of women may limit the generalizability of the findings for men. Those with and without insomnia differed significantly on age, such that the average age of the insomnia group was 47 years old compared to 32 years old for the normal sleeper group. Regularity, as measured by the full version of the SRM, has consistently been found to increase with age (Monk et al., 2006; Monk et al., 1997; Monk et al., 1992). Despite having an older insomnia disorder group (i.e., a group known to be more regular), decreased regularity was still observed in those with insomnia on two out of the three regularity indices, further highlighting this robust finding. Perhaps in groups with comparable age ranges, all three indices would have been significant, although one can only speculate at this time. Given that older adults are generally phase-advanced, such that timing of their daily activities generally occur earlier (e.g, meal and bedtimes; Bonnet & Arand, 1997; Lewy & Sack, 1989), this could have also influenced the mean timing of activities and may account for the similar timing in daily activities across sleeper status groups. Future studies

would benefit from including a wider age range in both the insomnia and control group, in order to rule out possible effects of age on the circadian system.

Another potential limitation of the methodology includes possible constraints of the SRM diary, a validated and commonly used measure for social rhythms. Firstly, the SRM could be considered a burdensome measure (Monk et al., 2006; Monk et al., 2002) as it requires participants to record 18 daily activities, including the timing and whether social engagement was involved, across a two-week monitoring period. Although this does not require a sizeable time commitment (i.e., five minutes per day), some participants indicated that they found this to be a cumbersome process. The developer of the SRM acknowledges this limitation and created a reliable and valid five item version (Monk et al., 2002). This shorter version of the SRM is recommended for use when the SRM is not the prime area of interest. Given that regularity was the main outcome variable in the current study, the original SRM provided richer data, which would not have been possible to obtain via the shortened measure. Nonetheless, the arduous monitoring required may have led to errors and intentionally skipped questions, thus, impacting the data collected.

A second potential drawback of this measure is that, despite participants being trained on how to monitor daily activities using the SRM, some may have been confused as to how to use this measure accurately. In particular, participants may have been unclear on how to record whether social engagement was involved, as this was left blank for several participants and may have influenced the social aspect of the results. Considering it is unrealistic to expect participants to carry the monitoring diary with them throughout the day, they were likely relying on memory for the timing of various activities, which is subject to errors. Future studies would benefit from more thorough training and perhaps a check-in after the first day to confirm participants

understand the monitoring procedures. The use of monitoring using mobile devices may be a promising alternative to pen and paper monitoring (Collins, Kashdan, & Gollnisch, 2003). Monitoring daily activities using technology (e.g., app on a smartphone) would likely be advantageous, as it would ensure that the timing is correct via a time stamp, data entry would be instant and less vulnerable to inaccurate reporting, and this method is easy to integrate into daily life (Collins et al., 2003).

Finally, using the SRM diary, participants in the current study recorded if they engaged in 17 predetermined activities, plus one idiosyncratic activity. The total frequency count (i.e. ALI score) includes the total number of activities across the two-weeks. However, the highest score a participant can obtain would be 252 (18 activities across 14 days). As such, the ALI would not capture data from those individuals who engage in more daily activities than listed on the sheet, or engage in listed activities several times (e.g., watch television before work and after work). Although the ALI has been validated as a measure of the overall level of activity, this measure may not adequately capture the desired construct. For example, is it important to determine the number of activities, the degree of energy expenditure, or the duration? Again, these are common issues in the sleep literature and definitions of daily activities vary amongst studies (Morgan, 2003). These construct issues remain unresolved. Thus, in this study, although a validated measure was chosen, there are arguably more germane constructs. These construct issues need to be delineated in future studies, but it may be that the absolute number of activities is not actually essential information. The findings from the current study that suggest that the number of daily activities are not important for sleep should be interpreted with the caveat in mind that ALI may not able to fully capture this. Again, using momentary assessment methods may prove to be a viable option to overcome this and would allow participants to record all

activities they partake in, as they occur. Moreover, resolving these construct issues in future studies would help to clarify this relationship.

Similar to the construct issues of activity discussed above, it can be difficult to quantify regularity. Regularity, as measured by the SRM (i.e., SRM total), essentially includes regularity of the first onset of an activity. For example, the first time someone uses the computer or consumes a snack. It may be that the timing of first onset for an activity is all that is important for the circadian system, such as is the case for physiological measures of the circadian clock (e.g., dim light melatonin onset; Lewy & Sack, 1989). However, these constructs and methodologies employed to measure this will have to continue to evolve with time. Nonetheless, the SRM is the most commonly cited tool within the social rhythms literature and has been validated in a variety of populations (Brown et al., 1996; Monk et al., 1996; Monk et al., 1991; Monk et al., 1994; Monk et al., 1992; Prigerson et al., 1996; Shear et al., 1994).

Aside from self-report measures, this study utilized actigraphy data in order to gain objective information on daily psychomotor activities and sleep. Although not unusual in sleep research (e.g., (McCall & McCall, 2012), the low correspondence between the actigraph and sleep diary estimates of total wake time warrants further comment. Prospective sleep diaries, polysomnography, and actigraphy, measure different aspects of sleep and, at times, they do not correlate, as one would presume. Expert consensus recommendations for the assessment of insomnia (e.g., Buysse et al., 2006) acknowledge that the inconsistencies between insomnia questionnaires, sleep diary, polysomnography, and actigraphy, are unavoidable and that these indices are to be considered complementary. It is plausible that participants' underreport their sleep on the sleep diary or that miscalculations were made on the sleep diary. For example, early morning awakenings could have been incorrectly reported on the diary, as participants can find it

difficult to report when that period began if they are drifting in and out of sleep in the morning hours. Consequently, early morning awakenings would not be captured in this situation, leading to a lower diary total wake time value. Alternatively, the algorithm for this actiwatch may have been too sensitive and erroneously miscalculated movement as time awake, thus, increasing the total wake time on the actigraph. Despite this lack of convergence, it is encouraging that the total wake time values obtained via both methods (i.e., actigraphy and self-report) were significantly different between those with and without insomnia, with those with insomnia exhibiting increased wakefulness, as one would expect.

Although actigraph total activity counts is a validated objective index of activity that correlates with energy expenditure (Chen et al., 2003), it may not be the best or most relevant measure for capturing daily activities. Arm movement alone, sampled infrequently, may not accurately assess the degree of activity. For example, it may be difficult to distinguish between an individual who stays home and watches television for most of the day versus someone who has a desk job, which requires little physical movement. This is an important distinction as total activity counts as an outcome variable may not be able to capture total daily activity levels, as initially planned. In regards to the homeostat (i.e., the sleep drive), it may not matter whether an individual is sitting at a desk versus watching television, as a sedentary lifestyle in general has been linked to decreased sleep quality (Morgan, 2003; Ohayon et al., 2001; Sherrill et al., 1998). This information could, however, have implications for the circadian system via the amount of daylight exposure, as someone who works outside the home versus someone who stays home watching television may have reduced exposure to natural light. Clinically, it is important to understand the nature (e.g., inside versus outside the home) and number of activities, in order to gain insight into whether daily activities are important for sleep. Also, there are certain physical

activities in which participants were unable to wear the watch, for example, activities that involved the wrist (e.g., volleyball) or any activities with water or steam (e.g., swimming, hot yoga). Moreover, while the monitoring period (two weeks) provided a wealth of information, in order to obtain this rich data, the epoch length (i.e., the sampling interval) for this study had to be set to 1 minute (as opposed to 30 seconds for one week monitoring period). Thus, certain movements may not have been included in the total activity count, which could have affected the overall activity value. Since sleep and activities are quite variable, this two-week monitoring period was an essential piece of the study design.

Although the actigraph can reliably predict energy expenditure from activity (Chen et al., 2003) in certain study designs (e.g., studies manipulating exercise), it is possible that this particular actigraph index is not appropriate to detect daily activity levels in a relatively non-active population and, as such, the results should be interpreted cautiously. It may be that it is more important to consider the type or vigor of the activity instead of the mean daily minutes during which the actigraph detects movement. Future studies would benefit from a complement of activity indices, including prospective momentary assessment to better understand this relationship. Moreover, given the focus on circadian rhythms, it would be interesting for future studies to include an actiwatch equipped with a light sensor in order to provide information on photic input. Since entrainment of the circadian clock is mediated, in part, by light signals (Klein et al., 1991), this light sensor would provide useful information on the amount and timing of light. Previous research that looked at light input found a link between decreased habitual behaviours and exposure to lower average levels of light (Haynes et al., 2005). Also, future studies may benefit from tracking planned daily activities and whether these activities were delayed or cancelled in response to perceived sleep quality the previous night. For example, after

a sleep disrupted night, does an individual cancel their dinner plans because they are feeling "too tired" or would they keep these plans, despite feeling tired? It is likely that an individual with insomnia would change their plans in response to perceived insufficient sleep, whereas a good sleeper would not. However, this research question cannot be addressed from the current study design via the actigraph. A combination of the above suggestions may provide more fruitful information on the relationship between sleep and daily activities.

The current study is the first in the field to prospectively investigate social rhythms in a clinical insomnia population and a normal sleeper comparison group. Although these findings reveal important information about daily regularity, these findings do not speak to causality. It is unclear whether those with insomnia develop dysregulated rhythms in response to insomnia or if it is this dysregulation that actually contributes to the development of insomnia. Although this directionality may be less important from a treatment standpoint, this information can have significant implication in terms of preventing chronic insomnia. In order to address questions of causality, future studies would benefit from a longitudinal study design that measures regularity and sleep at various time points.

5.3 Clinical Implications

Findings from the current study highlight the importance of the daytime in insomnia. Individuals with insomnia appear to have dysregulated social rhythms, in comparison to normal sleepers. This finding has the potential to enhance current evidence-based treatments of insomnia. CBT-I is the recommended gold-standard treatment in the management of insomnia (Morgenthaler et al., 2006; Schutte-Rodin et al., 2008), with 70-80% of insomnia patients achieving a therapeutic response and approximately 40% achieving full clinical remission (Morin, 2010; Morin et al., 2006b; Morin et al., 1999). Nonetheless, there remains a subset of

insomnia patients for whom CBT-I does not work (Harvey & Tang, 2003; Morin et al., 1994). Although insomnia has previously been conceptualized as a nighttime disorder, with treatment mainly focuses on modifying nighttime behaviours (e.g., asking clients to get out of bed when unable to sleep), the daytime experience of insomnia has been largely overlooked. The main objective of this study was to gain a deeper understanding of the way in which daytime environmental and behavioural factors interact with the sleep regulatory system. Findings from the current study suggest that the daytime is an essential component to sleep and that incorporating daytime treatment targets would be a worthwhile addition to CBT-I

Thus far, sleep restriction, a main behavioural component of CBT-I, focuses on setting a consistent earliest bedtime and standard rise time, which is thought to aid in entraining the circadian clock. However, attention to regularity within a 24-hour period is lacking. Findings from the current study do suggest that integrating daytime treatment targets would certainly be valuable. It seems as though the frequency of activities, and whether social engagement is involved, is less important than a daily routine, in regards to sleep benefits. Regulating meals, particularly in the first part of the day, appears to be most essential. Currently, CBT-I treatments place more emphasis on the morning time. This is because it is more within our control to wake-up (i.e., with an alarm) than to initiate sleep. Thus, adding a scheduled breakfast time in close proximity to the regular rise time, which is already incorporated into the treatment, would easily compliment CBT-I. Also, this may increase regular exposure to sunlight, which helps to entrain the clock (Klein et al., 1991). This morning sunlight exposure is especially important in a time when people spend a significant time indoors. Although frequency of activities was not found to be as important as regularity in the current study, increasing the number of positive activities is still an essential target for overall mood (Dimidjian et al., 2011; Martell et al., 2010), which

would likely have sleep benefits. As such, these findings are not meant to persuade clinicians from increasing frequency of activities; rather, these findings are intended to emphasize the importance of daytime targets in sleep treatment, with regularity being a focus.

Interpersonal and Social Rhythm Therapy (IPSRT; Frank et al., 2005) is a common evidence-based treatment for Bipolar Disorder. One treatment target of IPSRT includes the maintenance of regular daily rhythms. Some of the techniques used in IPSRT would be a helpful adjunct to CBT-I. Specifically, IPSRT therapists use the SRM (Monk et al., 1990) to obtain a baseline (typically three to four weeks worth) of their patient's typical routines and highlight areas that appear to be unstable (e.g., extreme variations between the weekend and weekday, extreme variability in meal times, etc.). Once these are identified, the therapist educates the patient on the importance of regularity and the consequence of irregularity. At this point, the therapist and patient would then collaboratively generate specific short (e.g., adhere to eating breakfast at 8 am for 7 days), intermediate (e.g., adhere to eating breakfast at 8 am and lunch between 11 and 1 for month), and long-term (e.g., eat 3 regular meals daily) regularity goals. These goals are closely monitored and modified throughout treatment and potential environmental stressors that could disrupt the social rhythms are highlighted and discussed (Frank, Swartz, & Kupfer, 2000). These behavioural strategies could easily being incorporated into CBT-I treatment. Also, given that beliefs about sleep were shown to play an important role in regularity, these beliefs should be targeted via cognitive strategies (e.g., thought records), which is already included in traditional CBT-I. The addition of daytime targets to CBT-I has the potential to increase treatment success and decrease relapse rates, above that of standard CBT-I.

5.4 Conclusion

This investigation has extended the existing body of literature that suggests frequent and consistent social rhythms have a beneficial effect on sleep (e.g., Carney et al., 2006a; Monk et al., 2003; Zisberg et al., 2010). This study design was the first of its kind to utilize a clinical insomnia population and good sleeper comparison and to employ both objective and subjective prospective measures of daily activities and sleep across a two-week monitoring period. Findings from this study highlight the important role of regularity in daily routines, as regularity scores on the SRM can distinguish between those with and without clinical insomnia. Moreover, these findings are generalizable beyond the study population as exclusion criteria were not very stringent.

Given that insomnia is a prevalent disorder with large personal and societal consequences, enhancing treatment outcomes is warranted. Insomnia has generally been conceptualized as a nighttime disorder, although this has already begun to shift in the literature. For example, the daytime sequelae has been included as part of research diagnostic criteria of insomnia (e.g., Edinger et al., 2004) and the Sleep Disorders section in the new Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5; American Psychiatric Association, 2013) has been renamed Sleep-Wake Disorders. Accordingly, if the conceptualization of insomnia is now shifting and is considered to be a 24-hour disorder, as opposed to just a nighttime issue, insomnia treatments will have to be modified to reflect this.

Current CBT-I mainly focuses on modifying nighttime behaviours, thus, largely ignoring the daytime. In other disorders characterized by dysregulation of social rhythms (Taylor et al., 2008; Yang & Spielman, 2001), daytime targets focused on increasing (i.e., behavioural activation; Dimidjian et al., 2011; Martell et al., 2010) and regulating (Frank et al., 2005) daily

activities are a large part of the treatment. Which begs the question, why has this not been expanded to insomnia treatment? Findings from the current study further highlight the importance of the daytime in sleep. Including daytime treatment target in CBT-I and utilizing techniques from IPSRT that focus on regulating social rhythms would likely be a beneficial addition to this already efficacious treatment. Behavioural rhythms (e.g., feeling hungry) are the ones most noticeable to the individual themselves (versus melatonin levels or core body temperature; Monk, 2010) and may prove to be more useful in having people understand their own body clock. Moreover, these additional treatment targets would be consistent with the behavioural approach to insomnia and would be a feasible addition. Spielman's (1987) 3P model of insomnia suggests that chronic insomnia develops as a result of predisposing, precipitating, and perpetuating factors. Although it is unclear at this point whether irregularity is a precipitating or perpetuating factor, regularity certainly has a place in insomnia treatment. Future studies would likely benefit from including participants with a larger age range, obtaining more information on the type, frequency, and vigor, of daily activities, using momentary assessment methods, and utilizing a longitudinal design. With continued research into social rhythms and its relationship with insomnia, there is potential to enhance existing conceptual models and CBT treatments of insomnia and, thus, decrease the overall burden of insomnia on society.

Appendix A

Demographic Information

AGE: _____

SEX (check one): __ F __ M

ETHNIC BACKGROUND (check one):
___ Aboriginal Canadian
___ African Canadian
___Caribbean Canadian
___East/Southeast Canadian
___European Canadian
___Latin/Central/South Canadian
___Oceanic Canadian
___South Asian Canadian
___West Asian/Arab Canadian
___ Pacific Islander Canadian
___ Other: _____

MARITAL STATUS (check one):
___ Single
___ Married/common-law
___ Live-in Partner (less than 2 years)
___ Divorced
___ Separated
___ Widowed

LIVING ARRANGEMENT (check one):
___ Living Alone
___ With Spouse/Partner and Children
___ With Friend(s)/Roommate(s)
___ With Spouse or Partner
___ With Family Member(s)
___ Other

CHECKLIST OF MEDICAL CONDITIONS & SYMPTOMS

Have you ever had or do you currently have (i.e. past month) any of the following disorders or medical symptoms? Please check the appropriate box (current or past) for all that apply.

CURRENT (past month)	**PAST**	**DISORDER/SYMPTOMS**
		Heart Disease: Examples: Angina; Cardiac Arrhythmias (irregular heart beats); Congestive Heart Failure; Myocardial Infarction (heart attack)
		Pulmonary Disease: Examples: COPD; Emphysema; Cystic Fibrosis; Asthma
		Gastrointestinal Disorders: Examples: Stomach or Duodenal Ulcers; Reflux Disease (GERD)
		Neurologic Disorders: Examples: Huntington's Disease; Seizures; Spastic Torticollis; Blepharospasm; Dystonias; Parkinson's Disease; Alzheimer's Disease; Dementia; Sleep-disruptive Headaches
		Head Trauma: Examples: Blow to the head with loss of consciousness; Concussion
		Chronic Pain Disorders: Examples: Fibrositis/Fibromyalgia; Arthritis; Muscle Cramps; Chronic Low Back Pain
		Endocrine Disorders such as Thyroid Disease
		Metabolic Disorders such as Diabetes
		Kidney Disease: Examples: Kidney Failure with Dialysis; Kidney Stones
		Autoimmune Disorders such as Lupus
		Cancer
		HIV/AIDS
		Headaches
		Other. Please specify: _____

CHECKLIST OF MEDICATION, DRUGS, AND OTHER SUBSTANCES

Have you ever used or do you currently use (i.e. past month) any of the following medications or substances? Please check the appropriate box (current or past) for all that apply.

PRESCRIPTION MEDICATIONS

CURRENT (past month)	PAST	MEDICATIONS/SUBSTANCES
		Antidepressants Examples – Elavil, Trazodone, Prozac, Zoloft
		Anti-anxiety drugs Examples – Valium, Xanax, Buspar
		Anti-psychotics Examples – Thorazine, Haldol, Risperidone
		Antihypertensives Examples – beta blockers, Clonidin
		Thyroid medication Examples – Thyroxin
		Antiasthmatics Examples – Theophyllin, Clebuterol
		Anti-Parkinson drugs Examples – L-Dopa, Sinemet, Requip
		Anticonvulsants Examples – Dilantin, Tegretol, Phenobarbital
		Headache medicines Examples – Cafergot, Imitrex
		Stimulants Examples – Ritalin, Cylert
		Sleep medicines Examples – Ambien, Trazodone, Amitriptyline, Benadryl
		Other – Specify _____

OTHER DRUGS AND SUBSTANCES

CURRENT (past month)	PAST	DRUGS/SUBSTANCES
		Cannabis
		Marijuana
		Hashish
		Cocaine/Crack
		Heroin
		LSD
		Amphetamines
		Glue
		Diet pills
		Other – Specify: _____

COMMON BEVERAGES

CURRENT (past month)	PAST	DRUGS/SUBSTANCES
		Alcohol (used in large quantities or on a daily basis)
		Caffeinated beverages (coffee, tea, soft drinks) 4 or more cups per day or 2 or more cups after 6:00 p.m.

EXPOSURE TO:

Have you ever been or are you currently (i.e. past month) exposed to any of these substances? Please check the appropriate box (current or past) for all that apply.

CURRENT (past month)	PAST	SUBSTANCES
		Lead
		Arsenic
		Mercury
		Copper
		Other poisonous substances Please specify: _____

FOOD ALLERGIES:

Have you ever had or do you currently (i.e. past month) have allergies to any of these foods or beverages? Please check the appropriate box (current or past) for all that apply.

CURRENT (past month)	PAST	FOODS/BEVERAGES
		Milk
		Wheat
		Shellfish
		Eggs
		Other foods or beverages: Please specify: _____

CHECKLIST OF UNUSUAL EVENTS AND BEHAVIORS OCCURRING DURING SLEEP

Have you ever had or do you currently have (i.e. past month) any of the following symptoms? Please check the appropriate box (current or past) for all that apply.

CURRENT (past month)	PAST	SYMPTOMS
		Recurrent disturbing dreams
		Night terrors (abrupt awakening with feelings of fright and confusion)
		Sleepwalking
		Strange sensory experiences upon awakening or falling asleep
		Memory changes or bizarre behaviors during the night
		Confusion and difficulty coming to your senses when awakened from sleep
		Painful leg cramps during sleep
		Paralysis or inability to move while in bed
		Behaviors that are aggressive to others or dangerous to yourself during sleep
		Acting out your dreams
		Grinding your teeth during sleep
		Eating/drinking during sleep
		Groaning during sleep
		Loud noises in your head upon awakening or falling asleep
		Bedwetting

LIST OF MEDICATIONS

IF YES, CHECK [√]	NAME OF THE MEDICATION	ALSO CALLED	DOSE (IF KNOWN)	DATES TAKEN (IF KNOWN)	REASON FOR STOPPING
	Amitriptyline	Elavil			
	Amoxapine	Asendin			
	Bupropion	Wellbutrin			
	Citalopram	Celexa			
	Clomipramine	Anafranil			
	Desipramine	Norpramin, Pertofrane			
	Doxepin	Triadapin, Adapine, Sinequan			
	Duloxetine	Cymbalta			
	Escitalopram	Lexapro, Ciprolex			
	Fluvoxamine	Luvox			
	Fluoxetine	Prozac			
	Imipramine	Tofranil			
	Isocarboxazid	Marplan			
	Maprotiline	Ludiomil			
	Mirtazapine	Remeron			
	Nefazodone	Serzone			
	Nortriptyline	Aventyl			
	Paroxetine	Paxil			
	Phenelzine	Nardil			
	Protriptyline	Triptil, Vivactil			
	Sertraline	Zoloft			
	Tranylcypromine	Parnate			
	Trazodone	Desyrel			
	Trimipramine	Surmontil			
	Venlafaxine	Effexor			
	Others (?)	_____			
	_____	_____			

Appendix B

Insomnia Severity Index

1. Please rate the current severity of your insomnia problem(s):

	None	Mild	Mod.	Severe	Very Severe
Difficulty falling asleep	0	1	2	3	4
Difficulty staying asleep	0	1	2	3	4
Problem waking up too early	0	1	2	3	4

2. How satisfied/dissatisfied are you with your current sleep pattern?

Very Satisfied		Moderately Satisfied		Very Dissatisfied
0	1	2	3	4

3. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g., daytime functioning, ability to function at work/daily chores, concentration, memory, mood, etc.)?

Not at All	A little	Somewhat	Much	Very much
0	1	2	3	4

4. How NOTICEABLE to others do you think your sleeping problem is in terms of impairing the quality of your life?

Not at All	A little	Somewhat	Much	Very much
0	1	2	3	4

5. How WORRIED/distressed are you about your current sleep problem?

Not at All	A little	Somewhat	Much	Very much
0	1	2	3	4

After a poor night's sleep, which of the following problems do you experience the next day?

Circle all those that apply.

a. Daytime fatigue: tired, exhausted, washed out, sleepy.
b. Difficulty functioning: performance impairment at work/daily chores, difficulty concentrating, memory problems.

c. Mood problems: irritable, tense, nervous, groggy, depressed, anxious, grouchy, hostile, angry, confused.
d. Physical symptoms: muscle aches/pain, light-headed, headache, nausea, heartburn, muscle tension.
e. None.

Appendix C

Depression Anxiety Stress Scale-21

DASS21

Name: *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:
0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

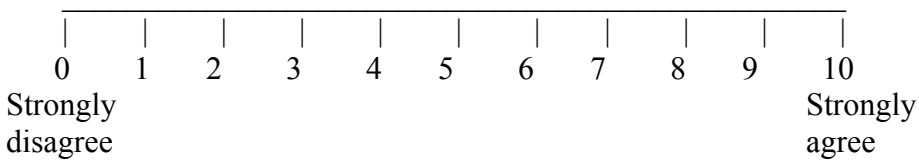
1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Appendix D

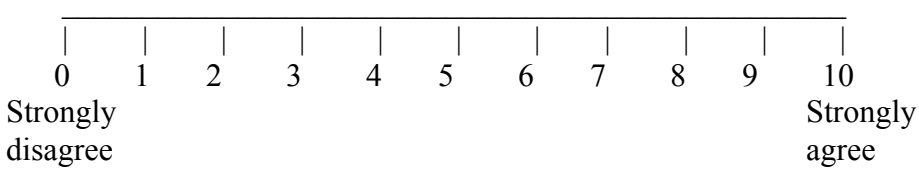
Dysfunctional Beliefs and Attitudes About Sleep Scale

Several statements reflecting people's beliefs and attitudes about sleep are listed below. Please indicate to what extent you personally agree or disagree with each statement. There is no right or wrong answer. For each statement, circle the number that corresponds to your own personal belief. Please respond to all items even though some may not directly apply to your situation.

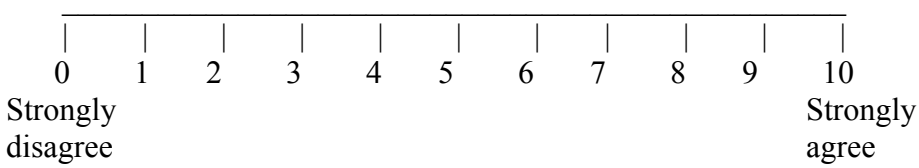
1. I need 8 hours of sleep to feel refreshed and function well during the day.



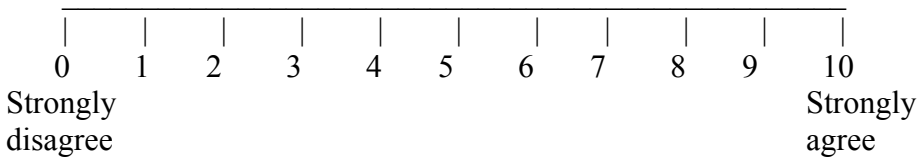
2. When I don't get the proper amount of sleep on a given night, I need to catch up the next day by napping or the next night by sleeping longer.



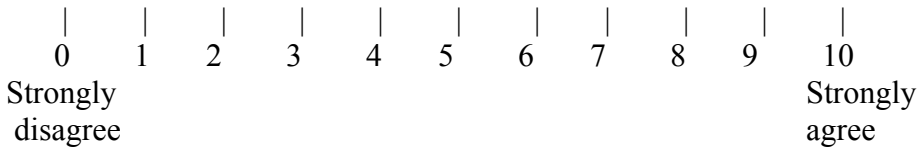
3. I am concerned that chronic insomnia may have serious consequences on my physical health.



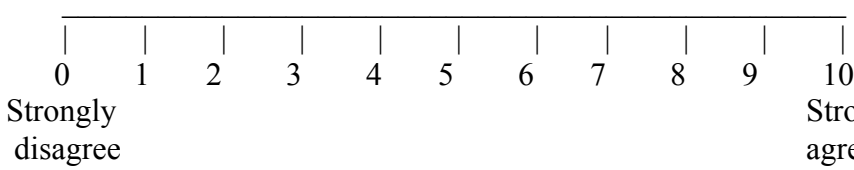
4. I am worried that I may lose control over my abilities to sleep.



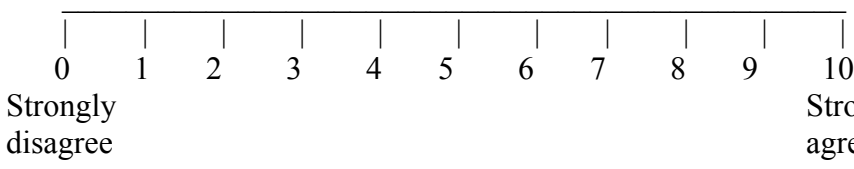
5. After a poor night's sleep, I know it will interfere with my activities the next day.



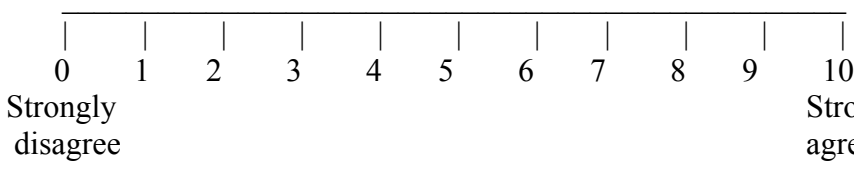
6. To be alert and function well during the day, I believe I would be better off taking a sleeping pill rather than having a poor night's sleep.



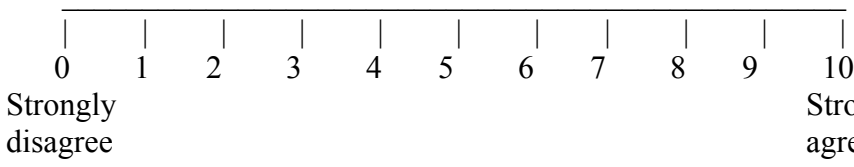
7. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before.



8. When I sleep poorly one night, I know it will disturb my sleep schedule for the whole week.

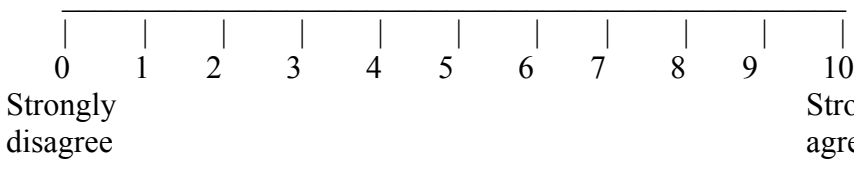


9. Without an adequate night's sleep, I can hardly function the next day.

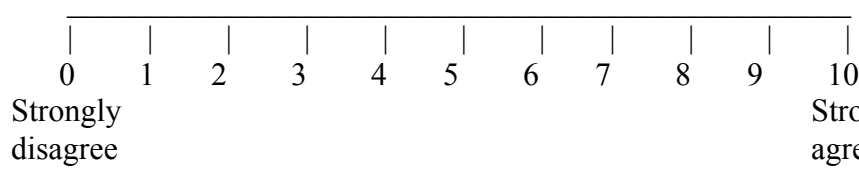


10. I can't ever predict whether I'll have a good or poor night's sleep.

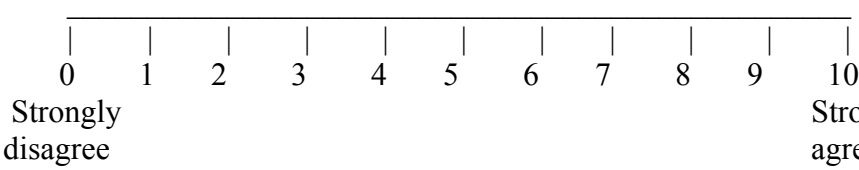
11. I have little ability to manage the negative consequences of disturbed sleep.



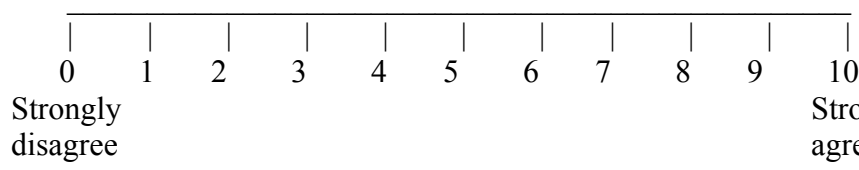
12. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before.



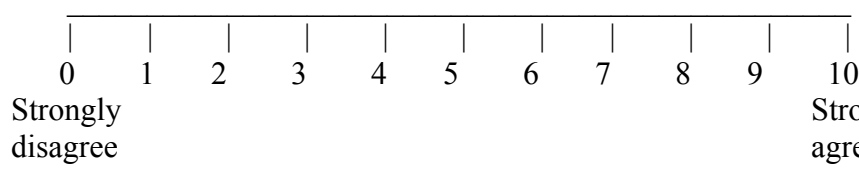
13. I believe insomnia is essentially the result of a chemical imbalance.



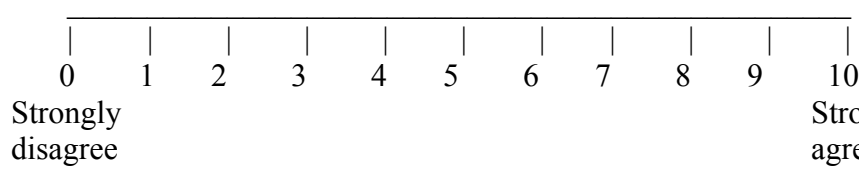
14. I feel insomnia is ruining my ability to enjoy life and prevents me from doing what I want.



15. Medication is probably the only solution to sleeplessness.



16. I avoid or cancel obligations (social, family) after a poor night's sleep.



Appendix E

Social Rhythm Metric

A) Activity Diary ***(Please complete in the evening, daily)***

This measure evaluates daily routines and schedules, what they do, at what time, with whom, and to what degree other people are involved. There are no "good" or "bad" schedules. Some people have set schedules, while others do not; it's different for everybody. Thus, we do not have any expectations concerning your daily schedules, nor are we asking you alter your schedule in any way.

Day of the Week: _____ Date: _____ (month/day/year)

ACTIVITY Please indicate the time at which you FIRST did any of the behaviours below. Check DID NOT DO, if you did not engage in this behavior today. Use Activity A to capture any behavior not listed below that you do most days. For example, if you have a hobby such as playing piano, write PIANO next to Activity A and complete the columns to the right for playing piano.	Check if you DID NOT DO	TIME			Check if	PEOPLE 1 = Just present 2 = Actively Involved			
		Clock Time	A.M. √	P.M. √		Spouse/ Partner	Children	Other Family	Other Person(s)
SAMPLE ACTIVITY		6:20 am	√			2			
Out of bed: Time you actually got out of bed to start day									
First Contact with Another Person: first interaction (e.g., telephone)									
Morning Beverage: When did you drink your first drink of the day?									
Breakfast: At what time did you first eat food? It does not have to be the morning.									
Go Outside for First Time									
Start employment, schoolwork, housework, or childcare: Time at which you started your primary goal-directed activity for the day									
Lunch: At what time did you eat your second meal?									
Nap: record when you first lay down to try nap									
Dinner: At what time did you last eat a meal? If you eat only two meals, write the first for breakfast and the second for dinner.									
Physical Exercise: refers to the first time you "break a sweat"									
Evening Snack/Drink: includes a drink or snack after dinner (not dessert)									
Watch/Read/Listen to News: This can include using the phone or computer to obtain news									
Watch TV (not news): Use item above if it was news									
Use phone for first time									
Use computer for first time (not to obtain news)									
Return Home for Last Time: If you did not leave the house today, enter the last time you went outside, even if it is to pick up mail, put out the trash etc.									
Activity A: Any regular activity you engage in _____									
Get into Bed: time you got into bed, not the time you fell asleep									

Appendix F

Sleep Diary

B) Sleep Diary (Please complete upon awakening)

Day of the Week: _____ **Date:** _____ **(month/day/year)**

	Sample	
Today's Date	4/5/08	
1. What time did you get into bed?	10:15 p.m.	
2. What time did you try to go to sleep?	11:30 p.m.	
3. How long did it take you to fall asleep?	55 min.	
4. How many times did you wake up, not counting your final awakening?	6 times	
5. In total, how long did these awakenings last?	2 hours 5 min.	
6a. What time was your final awakening?	6:35 a.m.	
6b. After your final awakening, how long did you spend in bed trying to sleep?	45 min.	
6c. Did you wake up earlier than you planned?	☑ Yes ☐ No	☐ Yes ☐ No
6d. If yes, how much earlier?	1 hour	
7. What time did you get out of bed for the day?	7:20 a.m.	
8. In total, how long did you sleep?	4 hours	
9. How would you rate the quality of your sleep?	☐ Very poor ☑ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good
10. How rested or refreshed did you feel when you woke-up for the day?	☐ Not at all rested ☑ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested
11a. How many times did you nap or doze?	2 times	
11b. In total, how long did you nap or doze?	1 hour 10 min.	
12a. How many drinks containing alcohol did you have?	3 drinks	
12b. What time was your last drink?	9 :20 p.m.	
13a. How many caffeinated drinks (coffee, tea, soda, energy drinks) did you have?	2 drinks	
13b. What time was your last drink?	3 :00 p.m.	
14. Did you take any over-the-counter or prescription medication(s) to help you sleep?	☑ Yes ☐ No	☐Yes ☐No
If so, list medication(s), dose, and time taken	Medication(s): Relaxo-Herb Dose: 50 mg Time(s) taken: 11 pm	Medication(s): Dose: Time(s) taken:
15. Comments (if applicable)	I have a cold	

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