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Sleep Behaviors and Thoughts as Links Between Social Rhythmicity and Mental Health Outcomes

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SLEEP BEHAVIORS AND THOUGHTS AS LINKS BETWEEN SOCIAL RHYHMICITY
AND MENTAL HEALTH OUTCOMES

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science
at Virginia Commonwealth University.

by

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Abstract

SLEEP BEHAVIORS AND THOUGHTS AS LINKS BETWEEN SOCIAL RHYHMICITY AND MENTAL HEALTH OUTCOMES

By Sahar M. Sabet, M.A.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Social rhythmicity refers to the regularity with which one engages in social and lifestyle activities. Considerable research has recognized that social rhythms have important implications for health and well-being. For example, disturbances in social rhythms are one potential factor that may contribute to the development of anxiety and depressive disorders. Existing research on social rhythm irregularity has heavily focused on its link with bipolar disorder and, to a lesser extent, with depression. Less is known about the association of social rhythms with anxiety or with subclinical levels of depression. Furthermore, it is unclear how social rhythms are tied to mental health outcomes. Sleep shows potential as an underlying link, however, no studies have investigated sleep behaviors and sleep thoughts as potential links between social rhythmicity and mental health outcomes. The current study used a large sample of 3,284 adults to examine the role of sleep thoughts and behaviors as mediators of the association between social rhythms and mental health for clinical and subclinical levels of depression and anxiety. Moderated parallel mediation models revealed that greater social rhythmicity is directly associated with fewer depressive and anxiety symptoms, and that sleep behaviors and thoughts mediate this association. Further, sleep health was found to be a particularly stronger link for clinically depressed adults. The findings highlight the critical role lifestyle regularity plays in anxious and

depressive symptom pathology. Results also highlight the importance of sleep behaviors and thoughts by demonstrating their concurrent ties to mental health, as well as their link between regularity and mental health. Future work is needed to further disentangle the impact of these constructs on mental health. Study implications and possible explanations of the findings are discussed.

Keywords: sleep, social rhythmicity, lifestyle regularity, depression, anxiety, self-efficacy

Sleep Behaviors and Thoughts as Links Between Social Rhythmicity and Mental Health

Anxiety disorders are consistently found to be the most common mental illness worldwide (Kessler et al., 2009) and are highly comorbid with depression, the leading cause of disability worldwide ("World Health Organization," 2018). According to the National Institute of Mental Health (NIMH), an estimated 19.1% of U.S. adults had an anxiety disorder in 2017, and an estimated 31.1% of U.S. adults experience an anxiety disorder at some point in their lives (National Institute of Mental Health, 2017). Moreover, an estimated 7.1% of adults in the U.S. had at least one major depressive episode (National Institute of Mental Health, 2017). Globally, an estimated 284 million people reported having an anxiety disorder, and 264 million people reported a depression diagnosis (World Health Organization, 2018). Given the high prevalence of anxiety and depression, there remains a need to better understand the etiology of these two disorders in order to contribute to effective prevention and treatment efforts.

Disturbances in social rhythms, the regularity of daily activities, is one potential factor that may contribute to the development of anxiety and depressive disorders. It has been theorized that disturbances in social rhythms may disrupt circadian rhythms which, in turn, promote the onset of affective and somatic symptoms in vulnerable individuals (Ashman et al., 1999; Ehlers, 1988; Grandin et al., 2006). However, the bulk of research in this area has examined the link between social rhythmicity and bipolar disorder, specifically mania. It is less understood how social rhythmicity contributes to depression, and studies examining its relation with anxiety are even more limited. A common thread among existing research on social rhythmicity and mental health outcomes is the importance of sleep. Regular social rhythms are a predictor of healthy sleep (Monk et al., 2003) and it has long been recognized that sleep disturbances are prominent in virtually all major psychiatric disorders. Researchers have consistently established high

comorbidity rates between sleep disturbance, and depression and anxiety disorders (Alvaro et al., 2013). Previous research, however, that has investigated sleep's association with social rhythms and psychiatric disorders has studied sleep via a narrow lens by merely focusing on sleep disorders (i.e., insomnia) and sleep deficiency (i.e., poor sleep quality). Whereas *disordered sleep* exists only in some individuals, the continuum of *sleep health* can be measured and applied to every individual (Buysse, 2014). Thus, there is a need to extend our understanding of sleep in relation to social rhythms and mental health outcomes by investigating sleep across a continuum to reflect varied experiences of sleep health. Specifically, there is limited research examining how healthy sleep behaviors and sleep-related thoughts are related to social rhythms and symptoms of anxiety and depression.

To date, no studies have investigated sleep behaviors (e.g. sleep health) and sleep thoughts (e.g. sleep self-efficacy) as potential links between social rhythmicity and mental health outcomes. As such, the current study aims to examine the importance and role of sleep thoughts and behaviors as mediators of the association between social rhythms and anxiety and depressive symptoms. Social rhythms, sleep thoughts, and sleep behaviors are universal factors that could have a profound impact on mental health given their daily reoccurrence. Also, as these factors are significant, they are modifiable and could be targeted to potentially reduce anxious and depressive symptoms. The current study seeks to investigate these associations in individuals reporting subclinical and clinical levels of anxiety and depression.

Social and Circadian Rhythms

Social rhythmicity refers to the regularity with which one engages in social and lifestyle activities. Such activities include the timing of meals, bedtimes, and social interactions (Bullock et al., 2011). The timing of these activities is partially driven by the body's internal biological

clock, the circadian system (Grandin et al., 2006). In humans, this circadian system is endogenously determined and self-sustaining, but can be influenced and synchronized by exogenous factors (or external cues) such as light, temperature, or social interactions known as "zeitgeibers" (Schimitt et al., 2010). The ability of an external cue (e.g. the light-dark cycle) to influence a circadian rhythm by causing it to align to a new schedule is referred to as "entrainment" (Mistlberger & Skene, 2004). In order to examine the influences of the regularity of social rhythms on behaviors (e.g. sleep) and mental health (e.g. depression), it is necessary to first describe the system that plays a vital role in regulating many human functions such as the sleep/wake cycle – the circadian system.

Researchers have found that in free-running environments, circadian rhythms (e.g. the sleep-wake cycle) have a period of approximately 25 hours (Aschoff et al., 1971; Grandin et al., 2006). Thus, circadian rhythms need to be resynchronized regularly to compensate for these slightly longer than 24-hour cycles. Under the influence of zeitgebers, this cycle is reduced to approximately 24 hours (Ehlers, 1988; Schimitt et al., 2010). Light is the primary, most dominant zeitgeber influencing circadian rhythms (Mistlberger & Skene, 2004); when natural changes of light are unable to unfold (e.g., constant dark or constant light), many social rhythms will desynchronize and circadian time periods will be lost (Ehlers, 1988). Such occurrences have major implications for mood disorders (Bullock et al., 2011; Grandin et al., 2006). Recently, however, interest has shifted to other external factors that influence circadian systems. Social zeitgebers, for instance, are also powerful agents for entraining circadian rhythms (Shen et al., 2008).

It is hypothesized that circadian rhythms in some species may be able to be reset in response to interactions with other members of the species and to other social stimuli 5/14/2020

3:20:00 PM. Researchers have begun to focus on social cues for regulating our lives by studying social zeitgebers and their effect on circadian systems, as well as the regularity of social rhythms. The regularity of social rhythms, for instance, has been associated with better sleep (Carney et al., 2006). Theoretical models, such as Ehler's (1988) *social zeitgeber theory*, have been put forth to explain the link between these social and biological rhythms.

The Social Zeitgeber Theory

According to the social zeitgeber theory (see Figure 1), changes in mood arise as a consequence of life events that disturb social zeitgebers which, in turn, derail social and biological rhythms (Ehlers, 1988). As mentioned earlier, the term "zeitgeber" is used to describe the environmental, or external, time cues that set, synchronize, and maintain daily rhythms. Specifically, life events (whether positive or negative) can affect social zeitgebers. After being involved in an accident, for example, an individual may decide to stay home and not leave the house or interact with others. Changes in social zeitgebers, in turn, can result in a disruption of social rhythms (e.g., bedtimes, socialization, and mealtimes;(Ashman et al., 1999). Social rhythms are theorized to entrain biological rhythms (e.g. sleep); therefore, the disruption of social rhythms could subsequently impact biological rhythms, which often manifests itself in somatic symptoms (e.g. poor sleep). Finally, affective episodes are theorized to result from the disruption of biological rhythms (Boland et al., 2012). In other words, life events may disturb social zeitgebers, impacting social rhythms, which then, consequently, disrupt biological rhythms.
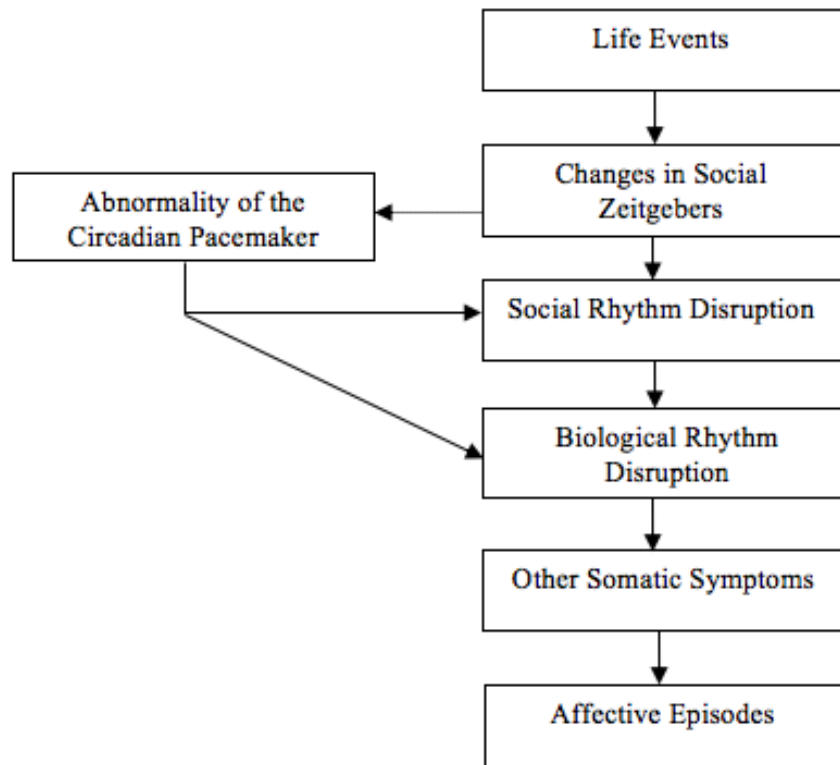
Figure 1. A model of the Social Zeitgeber Theory.

Though zeitgebers are often derived from social contact with other individuals, activities that one engages in solitude can also be considered as zeitgebers for the circadian clock. For example, activities such as eating meals, commuting to work, or going to bed may occur alone or with others. Through the integration of both biological and psychosocial perspectives, this theory thus proposes that social rhythm instability may cause disrupted biological rhythms (e.g., sleep-wake cycle, hormonal levels).

Social Rhythmicity and Mental Health

The social zeitgeber theory offers a model by which social rhythms may influence mood and mental health. The association between social rhythm instability and disrupted biological rhythms (e.g. sleep-wake cycle) often results in the manifestation of affective (e.g., anxiety, depression) and somatic symptoms (e.g., poor sleep, decreased appetite), which can then lead to

depressive and/or manic episodes (Boland et al., 2012). If the instability in rhythms is considerably severe and other risk factors are at play, an individual is ultimately at risk for developing a major mood disorder (Ashman et al., 1999). Consequently, it has been theorized that low or irregular social rhythmicity is a critical contributor to negative mental health and pathology (e.g. the etiology of affective symptoms), particularly bipolar disorder.

Social rhythms have been empirically studied in relation to bipolar disorder, depression, and anxiety. However, the bulk of the literature on social rhythmicity has focused on bipolar disorder. As such, there is substantial research highlighting how disruptions to social rhythms have been found among individuals with bipolar disorder (Ashman et al., 1999; Boland et al., 2012; Bullock et al., 2011; Ehlers, 1988; Grandin et al., 2006; Margraf et al., 2016; Monk et al., 1991). For instance, lower and more irregular social rhythmicity is a commonly reported feature of bipolar disorder (Bullock et al., 2011). Longitudinal research has illustrated that social rhythm irregularity is linked to quicker onset of depressive and manic episodes in individuals with bipolar disorder (Shen et al., 2008). Additionally, another study found that individuals with bipolar disorder displayed significantly greater levels of social rhythm disruption and sleep loss compared to normal, healthy controls following life events (major positive, major negative, minor positive, minor negative; (Boland et al., 2012). By exhibiting more social rhythm and sleep disruption following all life events, including those that are considered "minor" and "low-impact," this research suggests that individuals with bipolar are highly susceptible to irregular and disrupted social rhythms following (any) life events (Boland et al., 2012). Further, there is evidence that life events characterized by social rhythm disruptions are associated with the onset of manic (not depressive) episodes within bipolar disorder (Malkoff-Schwartz et al., 1998); this finding was replicated in a later study (Malkoff-Schwartz et al., 2000).

As further evidence to support the important role of social rhythms for mood disorders, one of the most widely used and well-known innovative treatments for bipolar disorder is Interpersonal and Social Rhythm Therapy (IPSRT). This treatment, which involves a psychosocial intervention, was specifically designed to regularize patients' social rhythms, as well as to identify and manage potential precipitants of rhythm dysregulation (Frank et al., 1997). The central tenet of IPSRT, similar to the social zeitgeber theory, is that the dysregulation of one's social and circadian rhythms are the underlying mechanisms in bipolar disorder (Grandin et al., 2006). Although the proponents of IPRST acknowledge that there are biological factors underlying bipolar disorder, they have found that establishing the regularity of daily routines (i.e., social rhythms) can be effective for regulating circadian rhythms and, consequently, improving sleep (Frank et al., 1997).

Though the association between social rhythmicity and bipolar disorder has received considerable attention, there is also a large body of literature that has examined social rhythmicity's link with depression. It has been well-established that individuals diagnosed with depression display significant disturbances in their biological rhythms, particularly in their sleep-wake cycles and in their neuroendocrine systems (Ashman et al., 1999; Boland et al., 2012; Ehlers, 1988; Grandin et al., 2006). In addition to irregular biological rhythms, research also points to the presence of irregular social rhythms within individuals diagnosed with depression. Patients with unipolar depression demonstrated more intraindividual variability in their patterns of social rhythms compared to a healthy control group (Monk et al., 1991). Similarly, individuals with major depressive disorder also displayed significantly lower levels of social rhythm regularity compared to non-depressed controls (Brown et al., 1996). Social rhythm regularity was inversely linked to the severity of depressive symptoms, as well as both objective and sleep

quality, suggesting that as stability of social rhythms decreases, both depression and sleep impairment increases (Brown et al., 1996).

Conversely, little research has investigated the link between social rhythmicity and anxiety. Existing research suggests that similar to individuals diagnosed with depression, individuals diagnosed with anxiety disorders also exhibit irregular social rhythms (Margraf et al., 2016; Shear et al., 1994). Specifically, individuals diagnosed with an anxiety disorder had significantly lower regularity of social rhythms compared to healthy controls. Moreover, regularity was shown to be associated with levels of anxiety. Shear and colleagues (1994) suggested that individuals with anxiety disorders might have a heightened sensitivity to events that disrupt social rhythms, and that lower regularity in daily routine may contribute to feelings of unpredictability and uncontrollability that is characteristic of these disorders.

Social Rhythmicity and Sleep

Sleep disturbance is increasingly recognized as an important, but understudied, mechanism associated with social rhythm disruption in several ways. As mentioned above, Ehlers et al. (1988) posited that the "primary path" of the social zeitgeber theory consisted of a chain of events in which disruption of social rhythms can lead to an instability in biological rhythms, particularly sleep. Stressful life events may disrupt one's social rhythms, resulting in a cascade of circadian and sleep disruptions. These events can, in turn, precipitate the onset of mania in bipolar patients (Wehr, 1987), lead to a major depressive episode (Grandin et al., 2006), or anxiety (Shear et al., 1994). For example, it is possible that social rhythm disruption may cause sleep loss, resulting in elevated mood, which may ignite a manic episode. Further, it has long been recognized that a well entrained circadian rhythm promotes sleep quality (Monk et al., 2003). One major function of the circadian system is to prepare an individual for obtaining a

restful night of quality sleep. Consequently, a disrupted rhythm will make it more challenging for consistent, high quality sleep to be obtained. Indeed, many techniques to aid poor sleep quality (e.g., regularity in getting into and out of bed) lead to such stable and robust circadian rhythms. Thus, there appears to be an important relationship between sleep quality and lifestyle regularity.

Sleep

Sleep is a universal behavior and multidimensional concept that consists of endogenous and recurring behavioral states (Buysse, 2014). Many definitions of sleep have been put forth, including one offered by (Carskadon & Dement, n.d.): "Sleep is a recurring, reversible neuro-behavioral state of relative perceptual disengagement from and unresponsiveness to the environment" (p. 1). Due to its restorative processes and countless benefits, sleep is undeniably critical to health and well-being. For example, sleep increases mood and performance (Dinges et al., 1997), optimizes emotion regulation (Walker, 2010), and makes an important contribution to processes of learning, memory, and brain plasticity (Walker, 2008, p. 2). Conversely, insufficient sleep triggers a complex set of bidirectional changes in brain activity and connectivity (Krause et al., 2017). For example, cortisol levels rise, cognitive and emotional abilities are markedly impacted, and immune regulation and metabolic control are impaired (Walker & Stickgold, 2006).

Though there is an abundance of research pointing to the importance and necessity of sleep for an individual's health and well-being, millions of people suffer from poor sleep quality and do not get enough sleep. For example, insomnia is one of the most prevalent disorders and the most common sleep disorder, causing severe distress as well as social, interpersonal, and occupational impairment (Harvey, 2002). According to the National Sleep Foundation's (NSF) Sleep Health Index, forty-five percent of Americans report that poor or insufficient sleep affected

their daily activities at least once in the past seven days ("National Sleep Foundation," 2014). The NSF's 2018 Sleep Health Index finds that only 27% of Americans are getting the recommended sleep time of 7 to 9 hours per night, with 65% getting less than 7 hours ("National Sleep Foundation," 2018). Much of these sleep-related issues are left undiagnosed and untreated.

A majority of American adults (65%) think sleep contributes to next day effectiveness. Despite its importance, sleep is not on the top of the list for most people when prioritizing their daily activities. An overwhelming 90% of Americans do not list sleep as their top priority compared with other items; instead, it is ranked as important as socializing (National Sleep Foundation, 2018). Moreover, sleep health is a strong predictor of feeling that one is a more effective person. Among the best sleepers, 89% rate themselves as extremely effective when it comes to getting things done each day, with the worst sleepers rating themselves at only 46% effective. Overall, Americans are aware of this association: 65% say they are more effective when they get enough sleep (National Sleep Foundation, 2018).

Sleep is a multifaceted construct that encompasses many dimensions and can be measured in a variety of ways. The assessment of sleep is an essential component for evaluating one's health. Sleep can be measured objectively and subjectively along multiple dimensions and can be characterized across self-report, behavioral, physiological, circuit, cellular, and genetic levels of analysis (Buysse, 2014), with the three most common methods being physiological, behavioral, and self-report. For instance, polysomnography (PSG) is considered the 'gold standard' method for assessing sleep as it measures sleep continuity, sleep architecture, and rapid eye movement (REM) sleep (Lockley et al., 1999). However, PSG is not always a practical option since it needs to be used in the sleep laboratory with continuous attendance. Sleep behaviors can also be measured indirectly with the use of actigraphy. The most widely used

methods are ones that require minimal supervision and are both convenient and time efficient. For this reason, subjective self-report assessments, such as sleep diaries, logs, or questionnaires, are highly popular.

Sleep Health. Similar to other medical disciplines, the area of sleep medicine for much of its history has focused on (sleep) disorders and treatment. More recently, sleep health has entered the discussion. Though sleep is critical to optimal health, far fewer studies have examined the positive role of sleep and the potential benefits of good sleep. In contrast to the competing construct of "sleep deficiency," "sleep health" is both infrequently used and rarely defined in the literature. Buysse (2014) provides the following definition: "Good sleep is characterized by subjective satisfaction, appropriate timing, adequate duration, high efficiency, and sustained alertness during waking hours" (p. 12). This definition and others identify sleep health as a positive attribute that should be sought and maintained. Although it is important to identify and treat disorders and deficits, sleep health is not simply their absence (Buysse, 2014). Rather, it is more useful to examine sleep along a broader continuum that is inclusive of all individuals.

The proposed definition of sleep health identifies and incorporates several key elements that can be viewed as vague and challenging to quantify. For this reason, Buysse (2014) proposed the RU-SATED metric for quantifying sleep health where each letter stands for a critical component of sleep health. The metric is comprised of six crucial dimensions of sleep: regularity, satisfaction with sleep, alertness during waking hours, timing of sleep, sleep efficiency, and sleep duration. These dimensions of sleep are present to some degree in every person (Buysse, 2014) and represent the individual dimensions of sleep, as well as their interaction as a whole.

(Sleep) Self-Efficacy. In addition to the importance of prioritizing healthy sleep behaviors, beliefs and attitudes about sleep can promote or hinder sleep quality. Sleep self-efficacy refers to attitudes and beliefs about one's ability to obtain healthy sleep (Lacks, 1987). This construct builds off of Albert Bandura's self-efficacy component of his social-cognitive theory, which many scholars believe to be a critically important theoretical contribution to the study of motivation, learning, and achievement. Since Bandura published his 1977 paper, "Self-Efficacy: Toward a Unifying Theory of Behavioral Change," self-efficacy has become one of the most studied and influential topics in psychology.

Self-efficacy refers to an individual's belief in his or her capacity to execute behaviors necessary to produce specific performance attainments (Bandura, 1977). According to Bandura, self-efficacy beliefs lie at the core of human functioning. It is not enough to merely possess the knowledge and skills to perform a task; rather, one must also have the conviction that they can successfully execute and perform the behavior appropriately under both typical and challenging circumstances. This level of confidence encompasses the ability to exert control over one's own motivation, behavior, and social environment. Further, given that people guide their lives by their beliefs and attitudes, self-efficacy is described as a major basis of action (Bandura, 1997). It is believed to affect an individual's choice of activities, effort, and persistence. People who have low self-efficacy for accomplishing a specific task or carrying out a specific behavior may avoid it, while those who believe they are capable are more likely to engage. Moreover, individuals who feel efficacious are hypothesized to expend more effort and persist longer, which points to the self-limiting process that inevitably comes with low self-efficacy (Bandura, 1977, 1986, 1997).

The concept of self-efficacy in general has been studied minimally in relation to sleep dysfunction. Generally, applying self-efficacy theory within the health domain suggests that self-efficacy is the belief people have in their ability to participate in certain behaviors needed to promote their health (Bandura, 1997). Thus, it would be beneficial to examine how one's self-efficacy in relation to sleep (e.g. how confident a person is that they can obtain a restful night of sleep) may play a role in sleep dysfunction, whether clinical (e.g. insomnia) or subclinical (e.g. poor sleep quality). For example, high sleep self-efficacy may predict readiness to undertake behavioral change, whereas low self-efficacy points to a lack of readiness to take action.

Healthy sleep requires that an individual be motivated and actively involved in prioritizing sleep, as well as have self-efficacy for undertaking and sustaining behavioral change. Researchers have identified high (sleep) self-efficacy for undertaking sleep-improving behavior as one of the most significant predictors of acceptance of behavioral treatments for insomnia (Bluestein et al., 2010, 2011) with a strong association between low self-efficacy for sleep and insomnia severity (Bluestein, Healey, & Rutledge, 2011).

Similar findings emerged from another study that sought to identify factors tied to sleep self-efficacy in a group of primary care patients diagnosed with insomnia (Rutledge et al., 2013). Self-efficacy for sleep was found to be correlated with insomnia severity, health status, depressive symptoms, dysfunctional beliefs about sleep, and insomnia treatment acceptability for behavioral approaches. For example, depression was found to be linked to poor self-efficacy for sleep. Overall, researchers suggest that improving self-efficacy for sleep can improve acceptance of behavioral treatment for insomnia, as well as reduce insomnia severity and, in turn, the resulting impairment of other related health outcomes (i.e., mood, anxiety; (Bluestein et al., 2010, 2011; Rutledge et al., 2013).

Although a small body of work has examined the construct of sleep self-efficacy, it is connected to and based on a larger body of work that emphasizes the role of sleep-related thoughts, beliefs, and cognitive patterns. Historically, the psychological factors underlying sleep dysfunction have been less understood; however, the intersection of poor sleep quality and psychology is a growing area of interest. The following cognitive processes have been suggested to play a role across various models of insomnia and sleep dysfunction: rumination, worry, dysfunctional beliefs, arousal, selective attention and monitoring, and safety behaviors (Harvey, 2002; Hiller et al., 2015; Norell-Clarke et al., 2014).

One of the most widely cited and accepted models in the field is Harvey's (2002) cognitive model of insomnia. This model suggests that several mechanisms are at play that perpetuate insomnia and trap an individual into becoming progressively more absorbed by and anxious about sleep and issues to pertaining to sleep (Harvey, 2002). Essentially, these (negatively toned) cognitive processes trigger both autonomic arousal and emotional distress. In turn, the experience of this arousal is the result of the sympathetic nervous system activation, or the body's preparation for fight or flight. Thus, the individual finds him or herself in an anxious state. The unfortunate consequence of this sequence of events is that the increasing anxiety often leads to a real sleep deficit, as conditions such as physiological arousal and high levels of distress cause sleep onset to be unlikely. Moreover, an essential feature of this model is that daytime processes are emphasized to be of equal importance to the maintenance of insomnia as the nighttime processes. Harvey (2002) suggests that erroneous beliefs about sleep fuel excessive cognitive activity during the day and that people with insomnia are more focused on monitoring their body sensations during the day for signs of fatigue. Across many studies researchers

generally agree that repetitive thought processes such as rumination play a significant role in sleep disturbances (Carney et al., 2010; Hiller et al., 2015; Takano et al., 2012).

Towards an Integrative Conceptual Model of Self-Efficacy and Mood. Similarly, sleep self-efficacy can be viewed as a branch of this body of work. Since it has been suggested that cognitive processes such as rumination and worry are an important maintaining factor in sleep dysfunction and contribute to symptoms of anxiety, self-efficacy can be viewed as a connecting piece given that it is rooted in one's thoughts and beliefs. For example, the more a person ruminates about sleep the more likely they are to have lower sleep self-efficacy and higher symptoms of anxiety, which consequently reinforces the sleep dysfunction. Thus, in addition to sleep behaviors (i.e., sleep health) that may impact mood symptoms, one's beliefs and attitudes (i.e., self-efficacy) about how confident they feel in their ability to carry out these behaviors could also be associated with mood outcomes.

Sleep and Mental Health

It has long been recognized that sleep disruption is highly comorbid with many, if not most, psychiatric disorders (Benca et al., 1992; Harvey et al., 2011). The *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013) lists sleep disturbance as a symptom of many psychiatric disorders (Harvey et al., 2011). More specifically, sleep disturbance is a recognized and common symptom of all anxiety disorders (Papadimitriou & Linkowski, 2005), which have consistently found to be the most common mental illnesses worldwide (Kessler et al., 2009) and are highly comorbid with depression. In fact, sleep disturbance is present across the full axis of major anxiety disorders, including generalized anxiety disorder, social anxiety disorder, and post-traumatic stress disorder, suggesting trans-

diagnostic applicability. Moreover, sleep disruption has been linked to the development, progression, and maintenance of anxiety and mood disorders (Neckelmann et al., 2007).

There is also a very strong association between sleep disturbance and major depression, as major depression commonly co-occurs with symptoms of sleep disturbance (Alvaro et al., 2013; Benca et al., 1992). For instance, disturbed sleep is a highly distressing symptom that significantly impacts the quality of life in individuals who are depressed (Nutt et al., 2008). Individuals diagnosed with insomnia have a twofold risk of developing depression compared to individuals with no sleep difficulties (Baglioni et al., 2011). The close link between depression and insomnia suggests that these conditions are not merely randomly associated. In fact, insomnia is now considered not only a symptom of but also a possible predictor of depression (Baglioni et al., 2011). There is also evidence to suggest that chronically disrupted and restricted sleep may lead to changes in neurobiological and neuroendocrine systems that contribute to stress-related disorders such as depression (Novati et al., 2008). Though there is a substantial amount of research that has pointed to the high comorbidity rates between sleep disturbances, depression, and anxiety, the etiological associations between these conditions remains unclear (Alvaro et al., 2013).

Our understanding of the role of sleep disturbance in both depression and anxiety has yielded important insights into the development, maintenance, and treatment of these disorders. Although the association between sleep disturbance and both anxiety and depression is well demonstrated, there is much less research examining how constructs of sleep self-efficacy and sleep health may contribute to anxiety and depression. Disordered sleep is important to address; however, it does not represent the full continuum of sleep health. Furthermore, given the strong cognitive components of anxiety and depressive symptoms, sleep self-efficacy could be an

important risk or protective factor for these symptoms. As sleep health and sleep self-efficacy can be seen as important pillars of sleep behaviors, beliefs, and attitudes, it would be beneficial to explore the relations between these constructs.

Summary and Purpose of Current Study

Social rhythmicity refers to the regularity with which one engages in social and lifestyle activities throughout the week. It has long been recognized that social rhythms (e.g., daily activities such as getting into or out of bed, eating, and adhering to a work schedule) have important implications for health (i.e., sleep) and well-being (Carney et al., 2006). The human circadian rhythm is endogenously determined and synchronized by exogenous factors, or zeitgebers, such as light and temperature. In humans particularly, social zeitgebers are important entraining factors for the circadian system. One of the leading theories of the etiology of mood symptoms is the social zeitgeber theory (Ehlers, 1988), which suggests that life events disrupt social zeitgebers, which impact daily social rhythms, which in turn disrupt biological rhythms, resulting in affective and somatic symptoms (Boland et al., 2012). It is theorized that certain life events could cause changes in social zeitgebers of vulnerable individuals, altering social rhythms and consequently biological rhythms, resulting in affective and somatic symptoms, such as mood disorders, in a cascade effect (Schimitt et al., 2010).

One major role of the circadian system is to prepare the individual for a restful night of sleep. Sleep disturbance is increasingly recognized as an important, but understudied, mechanism associated with social rhythm disruption in several ways. For example, the disruption of one's social rhythms leads to the disruption of circadian entrainment, often resulting in the consequential disturbance of sleep. Testament to this link, properly entrained and robust circadian rhythms lead to better sleep (Monk et al., 2003). Social zeitgebers are important

behavioral entraining factors for circadian control of the sleep/wake rhythm (Ehlers, 1988) and it is theorized that sleep is an integral link by which social rhythm disruption influences mood. Further, sleep and circadian rhythm disruption are frequently observed in mood disorders, particularly bipolar disorder and depression.

Though considerable research has highlighted the detrimental impact social rhythm irregularity has on various mental health outcomes, much of the literature has focused on the link with bipolar disorder and, to a lesser extent, with depression. Very little research has examined how the regularity of one's routines are associated with anxiety or with subclinical symptoms of depression. Similarly, there is limited research studying how sleep behaviors (i.e., sleep health) and sleep-related thoughts (i.e., sleep self-efficacy) may be contributing to symptoms of anxiety and depression. Though sleep is critical to optimal health, far fewer studies have examined the positive role of sleep and the potential benefits of good sleep. Although it is important to identify and treat sleep disorders, a growing body of literature points to the need to promote sleep health. Furthermore, given that it is widely acknowledged that cognitive processes (i.e., rumination, dysfunctional beliefs, one's sense of efficacy) play a central role in the cause and maintenance of anxiety and depression, it would be beneficial to add to the body of literature by also examining constructs such as sleep self-efficacy.

It is essential to understand the mechanisms involved in the development and maintenance of anxiety and depressive symptoms for prevention and treatment strategies. For example, how low or irregular social rhythmicity predicts poor sleep health and self-efficacy, which in turn predicts an individual's subclinical symptoms of anxiety and/or depression remains unclear. Although previous research has examined the association between poor sleep and anxiety, sleep health and sleep self-efficacy have yet to be examined as potential pathways by

which social rhythmicity may predict fewer anxiety and depressive symptoms. Lastly, although there is mounting evidence that highlights the link between social regularity, sleep, and mood disorders, research examining the role of regularity in subclinical populations is more limited. Although the associations between social regularity, sleep, and mood symptoms are likely bidirectional and/or reciprocal, this study will explore the role of social regularity as a predictor of mood symptoms and the role of sleep as an underlying mechanism.

Building on this evidence, the current study aimed to characterize the associations between social rhythmicity, sleep health, and sleep self-efficacy in individuals reporting subclinical and clinical symptoms of anxiety and depression. As such, this study had two aims: (1) to examine the association between social rhythmicity and anxiety/depressive symptoms, and (2) to investigate sleep health and sleep self-efficacy as mediators of the social rhythmicity and anxiety/depressive symptoms associations in adults (see Figure 2). Specifically:

Aim 1. The first aim of the study was to examine whether (a) social rhythmicity (also referred to as lifestyle regularity; regularity) predicted symptoms of anxiety and depression and (b) this association differed between clinical (self-reported diagnosis of anxiety or depression) and subclinical samples (no self-reported diagnosis of anxiety or depression). Based on a review of the literature, I hypothesized that:

> (1) Greater social rhythmicity (i.e., higher regularity) would be associated with fewer
>
> symptoms of anxiety and depression after adjusting for select covariates and that this
>
> association would be stronger for the clinical sample.

Aim 2. The second aim of the study was to investigate whether (a) sleep health and sleep self-efficacy mediated the association between social rhythmicity and symptoms of anxiety and depression and (b) whether sleep health and sleep self-efficacy was a stronger mediator for

clinical (self-reported diagnosis of anxiety or depression) versus subclinical samples (no self-reported diagnosis of anxiety or depression). Based on existing literature, I hypothesized that:

(1) Greater social rhythmicity would be associated with better sleep health and greater sleep self-efficacy.

(2) Better sleep health and greater sleep self-efficacy would be associated with fewer symptoms of anxiety and depression.

(3) Sleep health and sleep self-efficacy would mediate the social rhythmicity and anxiety and depressive symptoms associations.

(4) The above hypothesized associations and mediation would be stronger for the clinical sample compared to the subclinical sample.
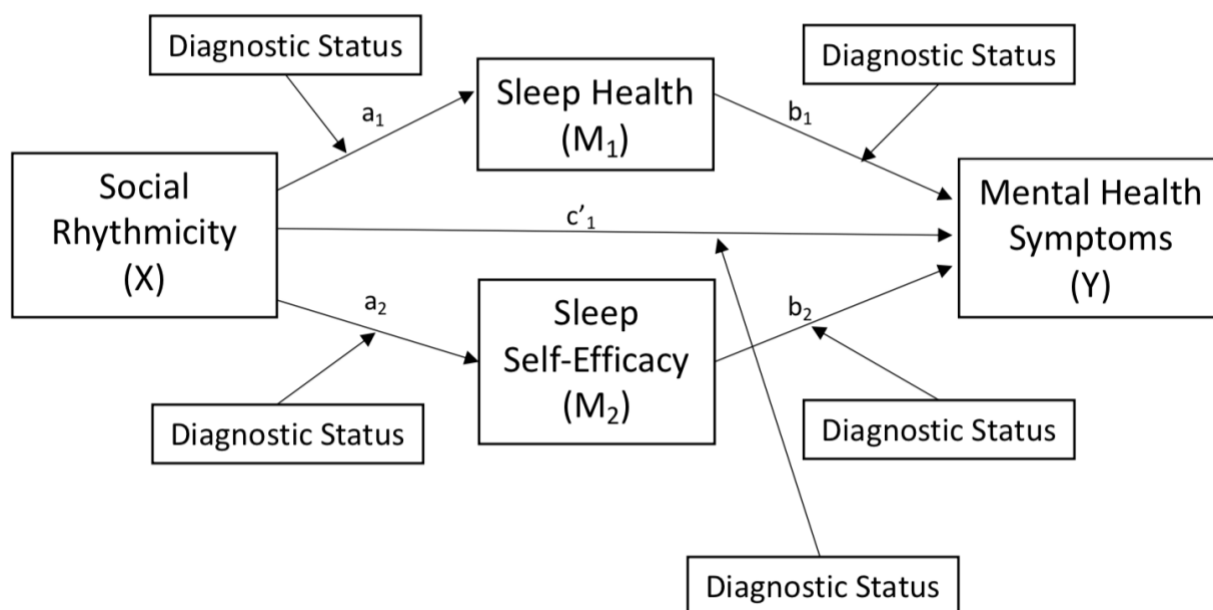
Figure 2. Example of parallel mediation model with moderated pathways.

Method

Participants

This project involved an archival analysis of a dataset from an online study investigating sleep longitudinally across normal development (ISLAND Study). This work was funded by the National Institute on Aging (K23AG049955, PI: Dzierzewski). In order to be eligible, participants had to be 18 years or older. Data collection for Wave 1 of the ISLAND Study began February 2018 and ended July 2018. The sample consisted of 3,284 participants (49% female) aged 18+. A description of methods pertinent to the present study are provided here.

Procedure

Participants were recruited through MTurk and instructed to open a Qualtrics link to access a survey if they wished to participate. Upon clicking on this link, participants were informed about the purpose of the study and gave consent. In order to participate, access to a computer, tablet, or phone was required. Participants then completed a series of behavioral and psychological measures through self-report questionnaires. Participants received $0.25 in exchange for participating in the study, which lasted approximately 25 minutes.

Measures

A subset of the behavioral and psychological measures included in the ISLAND Study dataset was selected for analysis in the current study. The following sections detail the measures used in the current study.

The Brief Social Rhythm Scale (BSRS): Participants completed a 10-item self-report questionnaire that assesses the regularity with which they engage in basic daily activities during the weekdays and weekend in general. The BSRS is a shorter version of the Social Rhythm Metric (SRM; (Monk et al., 1991), a daily monitoring diary designed to assess social rhythms.

The SRM is a widely-used measure to quantify an individual's daily social rhythms. Participants were asked to rate the general regularity of each activity (i.e., eating and meeting other people) in their lives in general using a scale ranging from 1 (*very regularly*) to 6 (*very irregularly*), with high mean scores indicating high irregularity. This measure can be administered at a single time point, rather than requiring a week of daily data to score. Summary scores are the average across all 10 items. The original version of this scale, the SRM, has shown good test-retest reliability and construct validity, as well as adequate criterion validity for adults (Monk et al., 1991, 2002; Monk et al., 2003).

RU Sated Scale: Participants completed a 6-item self-report questionnaire (Buysse, 2014) that is used to assess six key dimensions of sleep health (regularity, satisfaction with sleep, alertness during waking hours, timing of sleep, sleep efficiency, and sleep duration) that have been consistently tied to physical and mental health outcomes. They were asked to indicate the frequency with which they experience or engage in each of the 6 sleep-wake behaviors or characteristics in general. Individual items are scored on a scale from 1 (*rarely/never*) to 3 (*usually/always*). An example item includes "Do you go to bed and get out of bed at the same time (within one hour) every day?" Item scores are totaled. A total score of "6" represents poor sleep health, and a score of "18" represents good sleep health. Preliminary evidence using a European sample of adults suggests that the scale is a valid and reliable measure for the assessment of sleep health indicators (Becker et al., 2018).

Self-Efficacy of Sleep Scale (**SES**): Participants completed a 9-item scale used to assess the sense of self-efficacy one has in regard to sleep (Lacks, 1987). On this measure, they were asked to indicate their level of confidence when performing various sleep-related behaviors on a 5-point scale. Response options ranged from 1 (*not confident at all*) to 5 (*very confident*). The

sum of confidence ratings provides an overall score of sleep self-efficacy that can range from 9 to 45, with higher scores reflecting more confidence. Example items included "lie in bed feeling physically relaxed" and "fall asleep in under 30 minutes." A previous study which used this measure with adults (aged 18+) demonstrated strong internal reliability ($\alpha = .85$) as well as good concurrent validity and test-retest reliability (Bluestein et al., 2010).

Generalized Anxiety Disorder Scale (2 Items) (GAD-2): The GAD-2 is a short screening tool for generalized anxiety disorder that consists of the first two questions of the original GAD-7 scale 5/14/2020 3:20:00 PM. Participants were asked how often, during the last 2 weeks, they were bothered by the following two symptoms: 1) "feeling nervous, anxious, or on edge", and 2) "not being able to stop or control worrying." Response options were "*not at all*," "*several days*," "*more than half the days*," and "nearly every day," scored as 0, 1, 2, and 3, respectively. A total sum score was derived by adding the responses and was used in analyses. A score of 3 points is the preferred cut-off for identifying probable cases of GAD and in which further diagnostic evaluation is warranted. The original measure demonstrated excellent internal reliability ($\alpha = .92$), as well as good criterion, construct, and procedural validity in a sample of adults aged 18+ (Spitzer et al., 2006).

Patient Health Questionnaire (2 items) (PHQ-2): The PHQ-2 consists of the first two questions of the PHQ-9—the depression module from the full PHQ (Spitzer et al., 1999). Participants were asked to choose how often they experienced two depressive symptoms ("little interest or pleasure in doing things," and "feeling down, depressed or hopeless") over the past 2 weeks. There were four response options ranging from *not at all* to *nearly every day*. A PHQ-2 score ranges from 0 to 6 and a total score was used in analyses by adding the responses. A score of 3 is the cut-off point when screening for depression with scores of 3 or greater indicating a

likelihood that major depression is present. There is strong evidence for the reliability and validity (i.e. construct and criterion) of both the PHQ-9 and PHQ-2 (Kroenke et al., 2003).

Mental Health Symptoms: The variable *mental health symptoms* was derived by combining the sum scores of the GAD-2 and PHQ-2. This variable is used throughout the remainder of the paper to classify the combination of both depression and anxiety symptoms.

Diagnostic Status: Participants were asked to select from a list of disorders whether they have a current diagnosis for (1) any anxiety disorder and/or (2) any depressive disorder by answering the following prompt: "Do you have or are you being treated for any of the following (select all that apply)."

Data Analytic Plan

Data Analyses

To assess all five hypotheses, SPSS PROCESS macro (Hayes, 2013) was used to run three moderated regressions and multiple parallel mediation models to test the mediating roles of sleep health and sleep self-efficacy in the association between social rhythmicity and symptoms of depression and anxiety. A two-mediator model can be either a serial or parallel mediator model, with the former having a path between the two mediators and the latter not (Hayes, 2013). Specifically, in a parallel mediation model the independent variable is predicting the dependent variable via concurrent mediations, whereas a serial mediation assumes a causal chain linking the mediators, with a specified direction of causal flow. In the present study, as sleep health and sleep self-efficacy are estimated to operate concurrently but independently, a parallel mediation model was employed. All parallel mediation models controlled for identified covariates (i.e., age and gender). Using the PROCESS macro, the indirect effects of sleep health and sleep self-efficacy were tested using a non-parametric, bias-corrected bootstrapping

procedure that provided an empirical approximation of the sampling distribution of the product of the estimated coefficients in the indirect paths using 5,000 resamples from the dataset. Additionally, the potential moderating effects of clinical diagnosis were explored via testing moderators of all pathways in each model by diagnosis status (e.g., no diagnosis versus self-reported anxiety or depression for the overall mental health symptoms model, no diagnosis versus self-reported anxiety for the model with anxiety as an outcome, and no diagnosis versus self-reported depression for the model with depression as the outcome). Additionally, an index of moderated mediation, a test of equality of the conditional indirect effect for moderators, was provided for each model to test for the potential moderating effects for the overall mediated models.

In order to examine these associations closely and thoroughly, three separate models were developed and tested for all possible outcomes: combined mental health symptoms (depression and anxiety), depressive symptoms, and anxiety symptoms. In addition, three moderators were used across all three models for diagnostic status: combined diagnostic status (depression and/or anxiety), depression diagnosis, and anxiety diagnosis.

Specifically, parallel mediation models were used to assess whether sleep health (M_1) and sleep self-efficacy (M_2) mediate the association between social rhythmicity (X) and anxiety and depressive symptoms (Y; see Figure 2). In addition, the direct effect of: 1) social rhythmicity (X) predicting anxiety and depressive symptoms (Y; c'_1); 2) the direct effect of social rhythmicity (X) predicting sleep health (M_1) and sleep self-efficacy (M_2; a_1, a_2); and 3) the direct effects of sleep health (M_1) and sleep self-efficacy (M_2) predicting anxiety and depressive symptoms (Y; b_1, b_2) were assessed. All models controlled for selected covariates of age and gender given known associations between an individual's age and gender and social rhythmicity, sleep

behaviors and mental health (Alvaro et al., 2013; Harvey, 2002; Monk et al., 2002; "National Institute of Mental Health," 2017; "World Health Organization," 2018).

<div align="center">**Results**</div>

Data preparation and data cleaning

SPSS 26.0 was used for all data analyses. Data was cleaned and descriptive statistics (means, standard deviations, and frequencies) were calculated to verify that data met the assumptions of the planned analyses. A review was conducted to assess skewness, kurtosis, and outliers for all main variables and covariates of interest. Skewness and kurtosis values for mean social rhythmicity, sleep health, sleep self-efficacy, anxiety symptoms, depressive symptoms and diagnostic status were close to or below an absolute value of 1, indicating that they were approximately normally distributed. In addition to a review of skewness, kurtosis, and outliers, assumptions of independence, normality, multicollinearity, and homoscedasticity were assessed. All values were sufficiently met.

Power calculations using G*Power (Faul et al., 2009) suggest that for a mediation analysis with ten predictors, a sample size of least 64 participants is needed to detect a small effect at an alpha level of .05, with a power of .80. In the current study, assuming a small effect size, 4,388 participants will be sufficient to detect an effect. After reviewing the validation checks and removing participants who did not accurately respond to either of the two checks, the final overall sample size for the present study was 3,284 participants (original $N = 4,388$ participants).

Descriptive and correlational results

First, sociodemographic and mental health characteristics were examined (Table 1). On average, participants were 43.48 years old ($SD = 16.68$), primarily White (80.1%) and employed

(61.9%), with over 50% of participants having a 4-year Bachelor's degree or greater. Self-reported diagnoses of any anxiety disorder in the current sample (17.7%) are fairly representative of the national sample of U.S. adults in 2017 (i.e., 19.1%; National Institute of Mental Health, 2017). However, rates of depression are significantly higher in the current sample (15.7%) compared to the prevalence within a national sample of adults (i.e., 7.1%; National Institute of Mental Health, 2017). An independent-samples t-test was conducted to examine gender differences among our variables of interest. The results indicate that there was a significant difference in self-reported diagnoses between males and females for both depression, t (3009.51) = 5.86, $p < .001$ and for anxiety, t (2993.13) = 6.93, $p < .001$. Specifically, women reported higher rates of both self-reported diagnoses of depression ($M = .18$, $SD = .38$) and anxiety ($M = .20$, $SD = .40$). Similarly, women also reported higher symptoms of depression ($M = 1.55$, $SD = 1.73$) and anxiety ($M = 1.79$, $SD = 1.90$). Pearson correlations were conducted to examine bivariate associations between all main variables of interest (i.e., social rhythmicity, sleep health, sleep self-efficacy, anxiety symptoms, depressive symptoms and diagnostic status) and all covariates as presented in Table 2.

Table 1

Participant Sociodemographic and Health Characteristics (M, SD)

Variable	*N*	Percentage (%)
Age (43.48, 16.68)		
18 - 44 years	1799	54.8
45 - 64 years	978	29.8
65+ years	507	15.4
Gender		
Male	1479	45.0
Female	1594	48.5
Other	211	6.4
Race		
White/Caucasian	2652	80.8
Black/African American	263	8.0
Hispanic/Latino	216	6.6
Asian American	208	6.3
Native American/Pacific Islander	67	2.1
Self-Reported Diagnosis		
Depression	517	15.7
Anxiety	580	17.7
Employment Status		
Employed for wages	2034	61.9
Unable to work or disabled	111	3.4
Unemployed	266	8.1
Retired	459	13.9
Student	281	8.6
Homemaker	212	6.5
Education		
Doctorate Degree	147	4.5
Master's Degree	372	11.3
4-Year Bachelor Degree	1171	35.7
2-Year Associate Degree	387	11.8
Some college	853	26.0
GED or high school equivalent	96	2.9
High school	238	7.2
Less than high school	20	0.6

Note. More than one option may be endorsed for categories of race and employment status.

Table 2 *Pearson Correlation Coefficients among Social Rhythmicity, Sleep Health, Sleep Self-Efficacy, PHQ, GAD, Diagnostic Status, and Covariate Variables*

Variable	M (SD)	1	2	3	4	5	6	7	C_1	C_2
1. Social Rhythmicity	28.72 (9.91)	-								
2. Sleep Health	7.59 (2.69)	-.406	-							
3. Sleep Self-Efficacy	29.32 (8.13)	-.365	.617	-						
4. PHQ	1.59 (1.74)	.331	-.397	-.482	-					
5. GAD	1.73 (1.86)	.283	-.366	-.490	.730	-				
6. Depression	.16 (.36)	.132	-.139	-.198	.307	.261	-			
7. Anxiety	.18 (.38)	.119	-.134	-.212	.260	.329	.556	-		
C_1 Age	43.48 (16.68)	-.056	.145	.130	-.204	-.240	-.045*	-.110	-	

Note. All data are significant at the $p < .001$ level except *$p < .05$.
C_1 indicates covariate.

The direct association between social rhythmicity and mental health outcomes, depressive symptoms, and anxiety symptoms

Accounting for covariates of age and gender, social rhythmicity was a significant predictor of mental health symptoms (i.e., depressive and anxiety symptoms; $b = 0.095$, $SE = 0.006$, $p = .006$) in general, as well as both depressive symptoms ($b = 0.050$, $SE = 0.003$, $p < 0.001$), and anxiety symptoms ($b = 0.045$, $SE = 0.003$, $p < 0.001$). Higher irregularity predicted more symptoms overall as well as more symptoms of depression and anxiety individually. The overall model for mental health symptoms, after controlling for age and gender was significant, $F(6, 913) = 13.257$, $p < .001$, $R^2 = 0.080$. Similarly, the overall model for depressive symptoms alone was significant, $F(5, 3278) = 178.973$, $p < .001$, $R^2 = 0.214$, as well as the overall model for anxiety symptom alone, $F(5, 3278) = 179.161$, $p < .001$, $R^2 = 0.215$. However, in the investigation of whether these associations would differ by clinical diagnosis, no significant moderated effects were found across all 3 models. Consequently, the direct associations between social rhythmicity and mental health symptoms, including both depressive and anxiety symptoms, appeared to hold regardless of whether participants reported a clinical diagnosis of

depression and/or anxiety. Results from the three moderated regression models for all three outcomes are presented in Table 3.
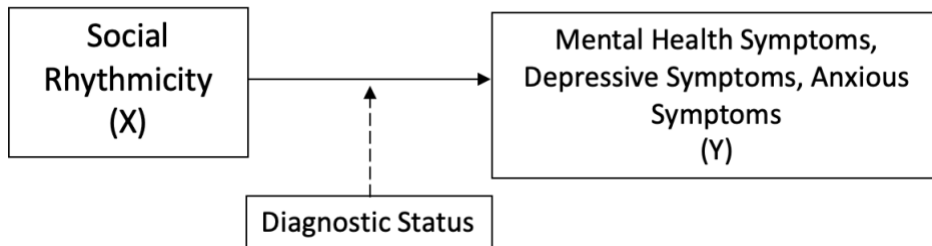
Figure 3. Visual representation of moderated regression model results for all outcomes. Solid line indicates significant path and dashed line indicates non-significant path.

The mediating roles of sleep health and sleep self-efficacy

A parallel mediation model was run to assess the indirect associations of social rhythmicity and mental health symptoms via the parallel mediators of sleep health and sleep self-efficacy. First, the direct association between social rhythmicity and sleep health and sleep self-efficacy was investigated in the model examining overall mental health symptoms (Table 4). After controlling for selected covariates, social rhythmicity was significantly associated with sleep health 95% CIs [–0.110, –0.091] and sleep self-efficacy [–0.301, –0.243]. Similarly, both sleep health [–0.132, –0.034] and sleep self-efficacy [–0.156, –0.124] were significantly associated with mental health symptoms. Lastly, both sleep health [0.008, 0.019] and sleep self-efficacy [0.044, 0.058] significantly mediated the association between social rhythmicity and mental health symptoms. Greater social rhythmicity predicted better sleep health and sleep self-efficacy. Additionally, sleep health and sleep self-efficacy were associated with fewer symptoms of mental health. The relation between social rhythmicity and mental health symptoms was significantly mediated by both sleep behaviors and sleep thoughts such that greater social

rhythmicity appears to be tied to fewer mental health symptoms via better sleep health and sleep self-efficacy.

Next, for the model examining depressive symptoms, the direct association between social rhythmicity and sleep health and sleep self-efficacy was investigated (Table 5). After controlling for selected covariates, social rhythmicity was significantly associated with sleep health 95% CIs [–0.110, –0.092] and sleep self-efficacy [–0.300, –0.244]. Similarly, both sleep health [–0.079, –0.027] and sleep self-efficacy [–0.071, –0.054] were significantly associated with depressive symptoms. Sleep health and sleep self-efficacy were then investigated as indirect pathways linking social rhythmicity to symptoms of depression. Both sleep health [0.005, 0.011] and sleep self-efficacy [0.019, 0.026] were found to be significant mediators of the social rhythmicity and depressive symptoms associations. Greater social rhythmicity predicted better sleep health and sleep self-efficacy, which in turn, predicted fewer symptoms of depression. Both sleep behaviors and sleep thoughts significantly mediated the association between social rhythmicity and symptoms of depression.

Finally, the direct association between social rhythmicity and sleep health and sleep self-efficacy was investigated in the model examining anxiety symptoms (Table 6). After controlling for selected covariates, social rhythmicity was significantly associated with sleep health 95% CIs [–0.112, –0.093] and sleep self-efficacy [–0.304, –0.247]. Similarly, both sleep health [–0.060, –0.004] and sleep self-efficacy [–0.088, –0.070] were significantly associated with anxiety symptoms. Social rhythmicity was linked to anxiety symptoms via sleep health [0.002, 0.009] and sleep self-efficacy [0.024, 0.032] as significant mediators. Once again, greater sleep health and sleep self-efficacy were associated with fewer anxiety symptoms, and significantly mediated

the social rhythmicity – anxiety symptoms relation. Results from the three parallel mediation models for all three outcomes are presented in Figure 4.
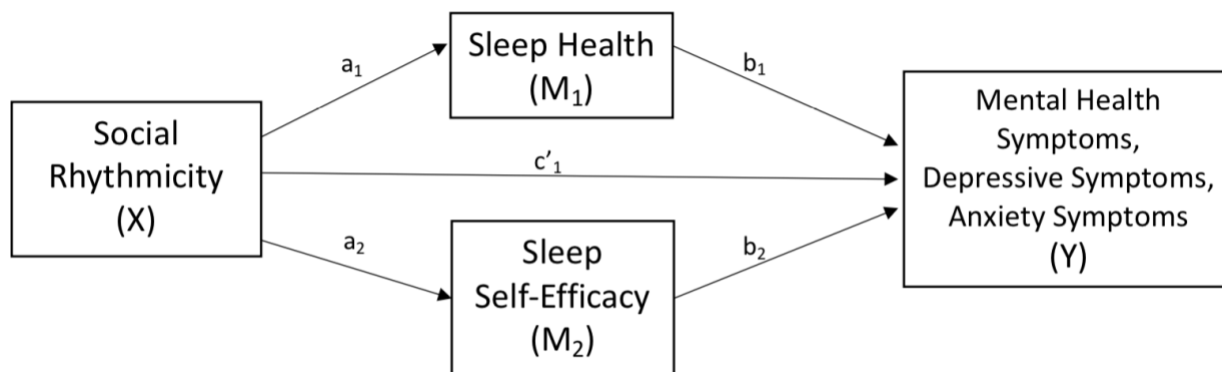
Figure 4. Visual representation of parallel mediation model results for all outcomes. Solid line indicates significant path.

Moderated Mediations

Lastly, the index of moderated mediation was examined for each model. When investigating the model examining overall mental health symptoms, neither of the indices were significant for sleep health [–0.002, 0.023] and sleep self-efficacy [–0.004, 0.030], suggesting there were no differences in this mediation model by diagnostic status. Next, for the model examining depressive symptoms, the index of moderated mediation was significant indicating a significant difference between diagnostic groups for the strength of the mediation for sleep health ($\beta = 0.012$, $SE = 0.005$, 95% CIs [0.002, 0.022]). Specifically, sleep health was found to be a particularly stronger link for individuals with a depression diagnosis ($\beta = .0017$, $SE = 0.005$, [0.007, 0.027]). However, the index was not significant for sleep self-efficacy [–0.002, 0.019], suggesting that sleep-related thoughts mediates the association regardless of diagnostic status. Finally, for the model examining anxiety symptoms, neither of the indices were significant for sleep health [–0.002, 0.016] and sleep self-efficacy [–0.008, 0.012], suggesting there were no differences in this mediation model by diagnostic status.

Table 3 *Moderated Regressions for all Three Outcomes*

	Mental Health Symptoms (Y)		Depressive Symptoms (Y)		Anxiety Symptoms (Y)	
	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI
Social Rhythmicity (X)	0.095*** (0.006)	0.083, 0.106	0.050*** (0.003)	0.044, 0.056	0.045*** (0.003)	0.039, 0.051
Diagnostic Status (W)	1.639(0.247)	1.155, 2.123	1.244*** (0.239)	0.775, 1.713	1.437*** (0.242)	0.962, 1.912
X x W	–0.007 (0.008)	–0.021, 0.008	–0.001 (0.007)	–0.016, 0.013	–0.005 (0.008)	–0.019, 0.009
Age (U1)	–0.038*** (.003)	–0.044, –0.032	–0.018*** (0.002)	–0.021, –0.015	–0.021*** (0.002)	–0.024, 0.018
Gender (U2)	0.421*** (0.086)	0.252, 0.589	0.174*** (0.045)	0.085, 0.264	0.283*** (0.049)	0.187, 0.378
	$R^2 = 0.080$		$R^2 = 0.214$		$R^2 = 0.215$	
	$F(6, 913) = 13.257, p < .001$		$F(5, 3278) = 178.973, p < .001$		$F(5, 3278) = 179.161, p < .001$	

Note. U_1 and U_2 specify covariates. $*p < .05, **p < .01, ***p < .001$.

Table 4 *Moderated Parallel Mediation Models for Combined Mental Health Symptoms as Outcome*

	Sleep Health (M1)		Sleep Self-Efficacy (M2)		Mental Health Symptoms (Y)	
	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI
Social Rhythmicity (X)	–0.101*** (0.005)	–0.110, –0.091	–0.272*** (0.015)	–0.301, –0.243	0.048*** (0.006)	0.037, 0.060
Sleep Health (M1)	--	--	--	--	–0.083*** (0.025)	–0.132, –0.034
Sleep Self-Efficacy (M2)	--	--	--	--	0.140*** (0.008)	–0.156, –0.124
Diagnostic Status (W)	0.014 (0.201)	–0.390, .0418	–1.987** (0.626)	–3.214, –0.761	2.566*** (0.438)	1.708, 3.424
X x W	–0.011 (0.006)	–0.024, 0.001	0.001 (0.019)	–0.037, 0.038	–0.020** (0.008)	–0.035, –0.005
M1 x W	--	--	--	--	–0.073* (0.034)	–0.140, –0.005
M2 x W	--	--	--	--	–0.013 (0.011)	–0.035, –0.010
Age (U1)	0.018*** (0.003)	0.013, 0.023	0.043*** (0.008)	0.027, 0.058	–0.030*** (0.003)	–0.036, –0.025
Gender (U2)	–0.151* (0.072)	–0.292, –0.010	–1.245*** (0.218)	–1.672, –0.817	0.225** (0.079)	0.071, 0.379
	$R^2 = 0.190$		$R^2 = 0.184$		$R^2 = 0.381$	
	$F(5, 3278) = 153.414, p < .001$		$F(5, 3278) = 147.689, p < .001$		$F(9, 3274) = 223.774, p < .001$	

Note. M_1 and M_2 indicate mediators. U_1 and U_2 specify covariates. *$p < .05$, **$p < .01$, ***$p < .001$.

Table 5 *Moderated Parallel Mediation Models for Depressive Symptoms as Outcome*

	Sleep Health (M1)		Sleep Self-Efficacy (M2)		Depressive Symptoms (Y)	
	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI
Social Rhythmicity (X)	–0.101*** (0.005)	–0.110, –0.092	–0.272*** (0.014)	–0.300, –0.244	0.028*** (0.003)	0.022, 0.034
Sleep Health (M1)	--	--	--	--	–0.053*** (0.013)	–0.079, –0.027
Sleep Self-Efficacy (M2)	--	--	--	--	–0.063*** (0.004)	–0.071, –0.054
Diagnostic Status (W)	0.185 (0.376)	–0.551, 0.922	–2.438* (1.145)	–4.684, –0.193	2.802*** (0.432)	1.954, 3.649
X x W	–0.024* (0.012)	–0.047, –0.002	–0.017 (0.035)	–0.086, 0.052	–0.021** (0.008)	–0.036, –0.006
M1 x W	--	--	--	--	–0.084** (0.034)	–0.150, –0.018
M2 x W	--	--	--	--	–0.023* (0.011)	–0.044, –0.001
Age (U1)	0.019*** (0.003)	0.014, 0.024	0.046*** (0.008)	0.031, 0.061	–0.013*** (0.002)	–0.016, –0.010
Gender (U2)	–0.166* (0.071)	–0.306, –0.026	–1.363*** (0.218)	–1.790, –0.936	0.072 (0.042)	–0.010, 0.155
	$R^2 = 0.189$		$R^2 = 0.177$		$R^2 = 0.332$	
	$F(5, 3278) = 152.875, p < .001$		$F(5, 3278) = 141.013, p < .001$		$F(9, 3274) = 180.675, p < .001$	

Note. M_1 and M_2 indicate mediators. U_1 and U_2 specify covariates. *$p < .05$, **$p < .01$, ***$p < .001$.

Table 6 *Moderated Parallel Mediation Models for Anxiety Symptoms as Outcome*

	Sleep Health (M1)		Sleep Self-Efficacy (M2)		Anxiety Symptoms (Y)	
	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI	Coefficient (SE)	0.95% CI
Social Rhythmicity (X)	–0.103*** (0.005)	–0.112, –0.093	–0.276*** (0.015)	–0.304, –0.247	0.020*** (0.003)	0.014, 0.027
Sleep Health (M1)	--	--	--	--	–0.032* (0.014)	–0.060, –0.004
Sleep Self-Efficacy (M2)	--	--	--	--	–0.078*** (0.005)	–0.088, –0.070
Diagnostic Status (W)	0.006 (0.356)	–0.693, 0.705	–3.025** (1.084)	–5.150, –0.900	1.943*** (0.434)	1.092, 2.793
X x W	–0.016 (0.011)	–0.037, 0.006	–0.002 (0.034)	–0.068, 0.064	–0.014 (0.008)	–0.029, 0.002
M1 x W	--	--	--	--	–0.052 (0.035)	–0.120, 0.016
M2 x W	--	--	--	--	–0.005 (0.011)	–0.028, 0.017
Age (U1)	0.018*** (0.003)	0.013, 0.023	0.041*** (0.008)	0.026, 0.057	–0.017*** (0.002)	–0.020, –0.014
Gender (U2)	–0.167* (0.072)	–0.308, –0.026	–1.302*** (0.218)	–1.730, –0.874	0.171*** (0.045)	0.082, 0.259
	$R^2 = 0.187$		$R^2 = 0.180$		$R^2 = 0.335$	
	$F_{(5, 3278)} = 151.063, p < .001$		$F_{(5, 3278)} = 143.62, p < .001$		$F_{(9, 3274)} = 182.987, p < .001$	

Note. M_1 and M_2 indicate mediators. U_1 and U_2 specify covariates. *$p < .05$, **$p < .01$, ***$p < .001$.

Discussion

The purpose of the present study was to characterize the associations between social rhythmicity, sleep health, sleep self-efficacy, and mental health in individuals reporting subclinical and clinical levels of depression and anxiety. In order to examine these associations closely and thoroughly, three separate models were developed for all possible depressive and anxiety outcomes and diagnostic statuses. By testing three distinct models, the hope was to tease apart the differences, if any, between these associations for both of these symptoms/disorders. For instance, are these relations specific to depression, anxiety, or a combination of both? This study found evidence that social rhythmicity is directly associated with depressive and anxiety symptoms, and that sleep behaviors and thoughts mediate this association. Additionally, differences in these associations for individuals with clinical or subclinical levels of depression and anxiety were only partially supported.

The findings from the current study add to the literature supporting the important roles of regularity, sleep, and mental health for both clinical and subclinical symptoms of depression and anxiety. Specifically, the first aim of the study highlighted the critically contributing role lifestyle irregularity plays in anxious and depressive symptom pathology. These findings align with previous studies that report associations between social rhythmicity and depression (Brown et al., 1996; Monk et al., 1991), as well as anxiety (Margraf et al., 2016; Shear et al., 1994). As previously mentioned, there is a much smaller body of literature that has linked social rhythmicity to anxiety. Additionally, research examining the role of lifestyle regularity in subclinical populations of depression is also more limited. The current study's findings add to the small but growing body of research in both of these areas and suggest that social rhythmicity

is tied to anxiety and depressive mental health functioning in a continuum of individuals ranging from no symptomatology to severe.

Beyond tying social rhythmicity to mental health outcomes, a major aim of this study was to try to understand *how* social rhythmicity is related to mental health. Building on a common thread among existing research on social rhythmicity and mental health outcomes that recognizes the importance of sleep, the current study found further support for the importance of sleep by using more diverse and unique constructs. Specifically, we examined sleep behaviors (sleep health) and sleep thoughts (sleep self-efficacy) as potential underlying processes within the social rhythmicity and mental health associations. Though an abundance of research has illustrated associations between sleep dysfunction and deprivation and both depression and anxiety, there is much less research examining how constructs of sleep health and sleep self-efficacy may also be contributing to these mental health disorders and symptoms, whether in clinical or subclinical levels or both. Individual components of sleep health (i.e. sleep duration) have been tied to both depression and anxiety (Alvaro et al., 2013; Benca et al., 1992), as well as to social rhythms (Monk et al., 2003). In addition, sleep self-efficacy has been examined in relation to insomnia severity, health status, and insomnia treatment acceptability for behavioral approaches (Bluestein et al., 2010, 2011; Rutledge et al., 2013). However, sleep self-efficacy has not been examined in relation to mental health outcomes or social rhythmicity and no studies to date have examined sleep health and sleep self-efficacy as mediators.

Our finding that sleep health and sleep self-efficacy mediated the association between social rhythmicity and mental health outcomes is notable for several reasons. First, the examination of sleep health in relation to social rhythmicity or mental health outcomes is a novel addition to the literature. Upon first glance, this finding is not a surprise as good sleep is essential

to good mental health, and research highlighting the link between sleep and depression has been well-demonstrated (Alvaro et al., 2013; Novati et al., 2008). For example, sleep dysfunction (including insomnia) is now considered not only a symptom of but also a possible predictor of depression (Baglioni et al., 2011). As mentioned above, the six characteristics of sleep health proposed by Buysse (2014) have each been studied separately quite heavily, with clear associations with physical, mental, and neurobehavioral health. For instance, sleep satisfaction/quality and efficiency (two of the dimensions of sleep health) have been previously linked to depression (Baglioni et al., 2011). However, the construct of sleep health in its entirety has been studied minimally in relation to mental health symptoms. Our finding suggests that overall, the six dimensions of sleep health—regularity, satisfaction with sleep, alertness during waking hours, timing of sleep, sleep efficiency, and sleep duration—are linked to both social rhythm regularity and mental health outcomes. Sleep health indicates how *well* an individual is doing holistically in terms of their sleep practices. The expansion of our understanding of the sleep-mental health association to include sleep health has significant implications for clinical treatment and interventions targeting sleep in depression. The measure of sleep health provides concrete targets for health promotion and prevention; therefore, researchers and clinicians may benefit from routinely assessing sleep health among clinically depressed adults, providing sleep health treatment recommendations, and exploring sleep health promotion as a preventive intervention.

Second, our findings highlight the importance of *both* sleep behaviors and thoughts by demonstrating their ties to mental health, as well as their link between regularity and mental health. Though these are two entirely distinct constructs of sleep, they appear to be complementing each other by operating in a similar manner and producing similar effects for

depressive and anxiety symptoms. Additionally, the fact that both of these constructs remained significant when entered in parallel in the model suggests that they are contributing uniquely and additively to the social rhythmicity-mental health associations. The notion that thoughts and behaviors impact mental health is not new. For instance, it is widely acknowledged that cognitive processes play an essential role in the development, maintenance, and treatment of various disorders, from depression and anxiety disorders to insomnia (Harvey, 2002; Hiller et al., 2015). Similarly, behavioral activation, which aims to increase engagement in adaptive activities, is a popular evidence-based treatment for depression. As an example, these two pieces are collectively highlighted and acknowledged through the widely-used Cognitive-Behavior Therapy (CBT), which places equal importance on the roles of thoughts and behaviors as agents of change. However, this is the first study to find that lifestyle regularity is linked to mental health via sleep behaviors and thoughts, as well as the first to examine both of these constructs together. The findings suggest that how we think and behave in regards to sleep are underlying mechanisms in this association, which sheds light on the connection between each of these constructs. In addition to existing research which suggests social rhythmicity can predict better health outcomes (Boland et al., 2012; Shen et al., 2008), our findings suggest that regular daily activities may promote mental health by facilitating healthy sleep behaviors and thoughts. For example, perhaps by maintaining a regular sleep/wake schedule and starting work at the same time, it is easier to obtain sufficient regular sleep (behaviors) and avoid catastrophizing and engaging in safety behaviors as the result of a poor night's sleep (thoughts). Although the cross-sectional study design precludes making assumptions about directionality, the mediation results stress the need to extend our understanding of sleep to include sleep-related behaviors and thoughts, as they are both playing significant roles in relation to social rhythms and mental

health. An investigation into how social rhythmicity may contribute to healthy sleep behaviors and thoughts, is also warranted.

Lastly, the present study illustrated that sleep health is a particularly stronger link within the social rhythmicity—depressive symptoms association for individuals who hold a diagnosis of a depressive disorder. Consequently, it appears that it is even more key to have better sleep health for individuals who are depressed. Also, greater lifestyle regularity appears to be more strongly associated with sleep health for this subsample. Specifically, regularity was also found to be a significant predictor of sleep health for only clinically depressed individuals. Overall, our findings suggest that the behavioral component of sleep is as or even more important than the cognitive component for those reporting a depression diagnosis. A potential explanation of this finding can be found in research on cognitive and behavioral approaches to treating depression. Cognitive therapy has been the most extensively studied psychological treatment for depression, with numerous studies documenting its efficacy (Dimidjian et al., 2006; Hollon et al., 2002). Its rise over the past decades has overshadowed more behavioral approaches; however, a landmark study revealed that behavioral activation produced as much change in depressive symptoms as cognitive therapy (Jacobson et al., 1996). Though the current literature in this area is equivocal (Dobson et al., 2008; Lorenzo-Luaces & Dobson, 2019), numerous studies have continued to support this finding. Among more severely depressed patients, behavioral activation has been found to be as enduring as cognitive therapy (Dobson et al., 2008), as well as comparable to antidepressant medication, significantly outperforming cognitive therapy (Dimidjian et al., 2006). Thus, our study provides further support in including behavioral components, such as sleep health, when delivering treatments for clinical depression and recognizing the importance of sleep behaviors for understanding how regularity can promote fewer depressive symptoms.

More work is needed to investigate why sleep behaviors are particularly critical in this association for this population.

     In contrast to the above finding, sleep behaviors and thoughts underlay the association between a regular lifestyle and mental health symptoms, specifically anxiety symptoms, regardless of one's diagnostic status. The results suggest that the regularity of social rhythms, sleep behaviors and thoughts, and mental health symptoms are important for all, regardless of whether the reported depressive and anxiety symptoms are clinical or subclinical. Each of these constructs, including mental health, are critical facets of daily functioning in adults. Given that these are all daily behaviors, our study reveals the potential mental health benefits associated with making small changes on a daily basis in universally performed activities (i.e. the time in which one gets into and out of bed or thoughts about sleep). Further, as these constructs have not yet been examined together, our study sheds light on the significance they each hold as they each exist within every individual. This offers researchers and clinicians multiple points to intervene to better target positive health outcomes, such as decreased mood and anxiety symptoms. Therefore, more work is needed to continue to investigate the possible implications of addressing these daily behaviors and thoughts for treatment recommendations and prevention strategies.

Study Implications

     This study utilized empirically strong measures with a large sample of adults, strengthening previous work in this subject area. Though the current study cannot speak to the bidirectionality amongst our variables of interest, it nonetheless highlighted important connections between lifestyle regularity, sleep behaviors and thoughts, and mental health outcomes. In addition, the current study expanded on previous research's conceptualization of sleep to include important pillars of sleep behaviors and sleep-related thoughts, attitudes, and

beliefs. Previous work has not exclusively examined how the constructs of sleep self-efficacy and sleep health may contribute to depression and anxiety. The current study was able to extend knowledge and provide information regarding the association between social regularity, sleep, mood and anxiety symptoms. Future work should examine the benefits of addressing sleep behaviors, attitudes, and thoughts versus solely focusing on objective sleep measures or single markers of dysfunction such as sleep duration. Additionally, the present results inform future research addressing whether sleep behaviors and thoughts should be addressed concurrently or sequentially and whether addressing both is advantageous or if addressing only one of the two is sufficient.

Additionally, although a substantial amount of research has highlighted the detrimental impact of social rhythm irregularity on various mental health outcomes, these associations have primarily been limited to its link with bipolar disorder (Bullock et al., 2011; Shen et al., 2008) and, to a lesser extent, with depression (Ehlers, 1988; Grandin et al., 2006).This is important as very little research has examined how the regularity of one's routines are associated with anxiety or with subclinical symptoms of depression; further, these links are not fully understood. Consequently, another implication of the current study is that social rhythmicity, sleep health, and sleep self-efficacy appear to be relevant across a continuum of mental health functioning from good to poor.

Furthermore, the associations between these constructs have potentially important implications for treatment development and implementation. As previous researchers have pointed to the bidirectional nature of these relationships, treatment of either problems has the potential to impact the other. For example, interventions such as progressive muscle relaxation and guided imagery are empirically supported methods to reduce arousal and are often employed

for both heightened mood and anxiety symptoms, as well as sleep disruptions (Coles et al., 2015). Interpersonal and Social Rhythm Therapy (IPSRT) is also another possible intervention, as it is targeted to regularize patients' social rhythms (Frank et al., 1997). Furthering our understanding of the factors that contribute to depression and anxiety will allow us to improve our interventions. One such factor is sleep, as evidenced by the general literature as well as this study.

It may be worthwhile for clinicians to provide individuals with psychoeducation regarding implications of lifestyle regularity as well as the associations between regularity, sleep, and mood/anxiety. Clinicians and researchers can also extend their conceptualization of sleep to also include behaviors and thoughts. Finally, it is important to note that the variables of interest in this study are each universal factors that are modifiable and could be targeted to potentially reduce mood and anxiety symptoms. For example, interventions targeted at increasing lifestyle regularity, sleep health, and/or sleep self-efficacy may prove to be effective ways to improve mental health and mood more broadly.

Strengths, Limitations, and Future Directions

There are several limitations in the current study which must be addressed. First is the overall lack of racial and ethnic diversity in the current sample. As a large majority of the sample is White, the homogeneity of the sample ultimately limits the generalizability of the overall study findings. Therefore, similar investigations should take place in more diverse samples (i.e. with respect to race/ethnicity) before any broader claims can be made about the findings. It is also important to note that the rate of clinical depression in our sample was higher than the national population, which further limits the generalizability of findings specific to clinical levels of depression. Furthermore, because the data is cross-sectional, we were unable to assess

directionality of the associations of interest by the current analyses. For example, it is possible that those with fewer mental health symptoms are more able to engage in a more regular lifestyle. Future research using prospective designs would be beneficial for investigating the direct association between social rhythmicity and mental health outcomes as well as the potential influence of sleep as a pathway underlying this association. Specifically, repeated concurrent assessment of daily routines, sleep behaviors and thoughts, and mental health symptoms would be particularly useful. In addition, though the PHQ-2 and GAD-2 are both reliable and valid measures, they are brief and may not capture the full range of depressive and anxiety symptoms (or lack thereof) an individual experiences daily. Thus, incorporating more clinical and variable levels of mental health would be important to investigate in future work. The current study assessed mental health symptoms retrospectively and was not able to examine daily fluctuation of depressive and anxiety symptoms. More research is needed to further understand precisely how changes in sleep behaviors and thoughts are tied to mental health outcomes on a daily basis.

Additionally, the diagnostic status variable used in the study solely was based upon self-reported diagnoses of depression and anxiety. Though there are many advantages to self-reported data, it is possible that this question did not fully capture what we intended. For example, the variable assessed for *current* diagnostic status (i.e., "do you have," "are you being treated") and participants did not endorse past diagnoses. Due to various factors such as social-desirability bias, response bias, or health illiteracy, participants may not have accurately responded to this question (e.g. over or underreporting diagnostic status). Thus, the present study may not have accurately represented clinical levels of depression and anxiety by this question. Similarly, incorporating objective measures of sleep (i.e. actigraphy) would complement existing self-report measures of sleep behaviors, attitudes, and beliefs. As individuals age, the discrepancy

between subjectively reported sleep (i.e. via a self-report questionnaire) and objectively reported sleep (i.e. via wrist actigraphy) increases (Kay et al., 2015). Therefore, obtaining the most complete assessment of sleep requires the utilization of more than one measure of sleep. Daily sleep diaries could also be administered via tablets and uploaded each night to avoid retrospectively reporting data. Alternatively, future studies could implement ecological momentary assessment multiple times throughout the day to assess mood and anxiety symptoms, regularity of routines, and other variables of interest. This could help tease apart some of the temporal associations between these constructs, as well as better capture the daily fluctuations of mood and anxiety.

Although the study does contain limitations, there are several strengths. First, the data represents a national sample of adults. Although limited in racial diversity, through this data we examined a broad range of adults living in the US. Of particular note, the sample included a wide age-range of adults, with 15% of participants aged 65+ years. Second, the diagnoses of an anxiety disorder in the sample were fairly representative of the national sample of U.S. adults, albeit the self-reported depression diagnoses were significantly higher; this allowed for broad comparisons to be made for adults with clinical levels of anxiety symptoms and disorders. Third, this study extended the study of sleep by examining more than one aspect of sleep. Fourth, we used well-validated measures of mental health symptoms, regularity of social rhythms, and sleep behaviors/thoughts. Lastly, by developing three separate models to test our hypotheses, we were able to tease apart differences amongst depression and anxiety and clearly differentiate the unique unfolding of relations per each outcome and diagnostic status. Despite the aforementioned limitations, the current study was able to highlight future areas of work, which

could help to extend understanding of how sleep is associated with lifestyle regularity and mental health outcomes in adults.

Conclusions

The current study presented evidence that both higher symptoms of depression and anxiety are associated with disruptions in social rhythms, as well as poorer sleep health and lower sleep self-efficacy. Additionally, social rhythmicity was associated with both depressive and anxiety symptoms via sleep health and sleep self-efficacy. Though differences in these associations for clinical and subclinical levels of depression and anxiety were only partially supported, our study shed light on sleep health being particularly important within depressed individuals. Social rhythms, sleep behaviors, sleep behaviors, and mental health are critical facets of daily functioning in adults. Therefore, continued research efforts are needed to extend our understanding of their profound impact on health given their daily reoccurrence.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing.

Alvaro, P. K., Roberts, R. M., & Harris, J. K. (2013). A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. *Sleep*, *36*(7), 1059–1068. https://doi.org/10.5665/sleep.2810

Aschoff, J., Fatranská, M., Giedke, H., Doerr, P., Stamm, D., & Wisser, H. (1971). Human circadian rhythms in continuous darkness: Entrainment by social cues. *Science*, *171*(3967), 213–215. Retrieved from JSTOR.

Ashman, S. B., Monk, T. H., Kupfer, D. J., Clark, C. H., Myers, F. S., Frank, E., & Leibenluft, E. (1999). Relationship between social rhythms and mood in patients with rapid cycling bipolar disorder. *Psychiatry Research*, *86*(1), 1–8. https://doi.org/10.1016/S0165-1781(99)00019-0

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., Lombardo, C., & Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, *135*(1–3), 10–19. https://doi.org/10.1016/j.jad.2011.01.011

Bandura, A. (1977). Self-efficacy: Toward a Unifying Theory of Behavioral Change. *Psychological Review*, *84*, 191–215.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs.

Bandura, A. (1997). *Self-Efficacy: The Exercise of Control*. W. H. Freeman Company.

Benca, R. M., Obermeyer, W. H., Thisted, R. A., & Gillin, J. C. (1992). Sleep and psychiatric disorders: a meta-analysis. *Archives of General Psychiatry*, *49*(8), 651–668.

Bluestein, D., Healey, A. C., & Rutledge, C. M. (2011). Acceptability of behavioral treatments for insomnia. *The Journal of the American Board of Family Medicine*, *24*(3), 272–280. https://doi.org/10.3122/jabfm.2011.03.100246

Bluestein, D., Rutledge, C. M., & Healey, A. C. (2010). Psychosocial correlates of insomnia severity in primary care. *The Journal of the American Board of Family Medicine*, *23*(2), 204–211. https://doi.org/10.3122/jabfm.2010.02.090179

Boland, E. M., Bender, R. E., Alloy, L. B., Conner, B. T., LaBelle, D. R., & Abramson, L. Y. (2012). Life events and social rhythms in bipolar spectrum disorders: An examination of social rhythm sensitivity. *Journal of Affective Disorders*, *139*(3), 264–272. https://doi.org/10.1016/j.jad.2012.01.038

Brandolim Becker, N., Martins, R. I. S., Jesus, S. de N., Chiodelli, R., & Stephen Rieber, M. (2018). Sleep health assessment: A scale validation. *Psychiatry Research*, *259*, 51–55. https://doi.org/10.1016/j.psychres.2017.10.014

Brown, L. F., Reynolds, C. F., Monk, T. H., Prigerson, H. G., Dew, M. A., Houck, P. R., Mazumdar, S., Buysse, D. J., Hoch, C. C., & Kupfer, D. J. (1996). Social rhythm stability following late-life spousal bereavement: Associations with depression and sleep impairment. *Psychiatry Research*, *62*(2), 161–169. https://doi.org/10.1016/0165-1781(96)02914-9

Bullock, B., Judd, F., & Murray, G. (2011). Social rhythms and vulnerability to bipolar disorder. *Journal of Affective Disorders*, *135*(1–3), 384–388. https://doi.org/10.1016/j.jad.2011.06.006

Buysse, D. J. (2014). Sleep health: Can we define it? Does it matter? *Sleep*, *37*(1), 9–17.

    https://doi.org/10.5665/sleep.3298

Carney, C. E., Edinger, J. D., Meyer, B., Lindman, L., & Istre, T. (2006). Daily activities and

    sleep quality in college students. *Chronobiology International*, *23*(3), 623–637.

    https://doi.org/10.1080/07420520600650695

Carney, C. E., Harris, A. L., Moss, T. G., & Edinger, J. D. (2010). Distinguishing rumination

    from worry in clinical insomnia. *Behaviour Research and Therapy*, *48*(6), 540–546.

    https://doi.org/10.1016/j.brat.2010.03.004

Carskadon, M. A., & Dement, W. C. (2005). Normal human sleep: an overview. Principles and

    practice of sleep medicine, 4, 13-23.

Coles, M. E., Schubert, J. R., & Nota, J. A. (2015). Sleep, Circadian Rhythms, and Anxious

    Traits. *Current Psychiatry Reports*, *17*(9), 73. https://doi.org/10.1007/s11920-015-0613-x

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E.,

    Gallop, R., McGlinchey, J. B., Markley, D. K., Gollan, J. K., Atkins, D. C., Dunner, D.

    L., & Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive

    therapy, and antidepressant medication in the acute treatment of adults with major

    depression. *Journal of Consulting and Clinical Psychology*, *74*(4), 658–670.

    https://doi.org/10.1037/0022-006X.74.4.658

Dinges, D. F., Pack, F. P., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., … Pack, A. I.

    (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance

    performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep*.

    https://doi.org/10.1093/sleep/20.4.267

Dobson, K. S., Hollon, S. D., Dimidjian, S., Schmaling, K. B., Kohlenberg, R. J., Gallop, R. J., Rizvi, S. L., Gollan, J. K., Dunner, D. L., & Jacobson, N. S. (2008). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *Journal of Consulting and Clinical Psychology*, *76*(3), 468. https://doi.org/10.1037/0022-006X.76.3.468

Ehlers, C. L. (1988). Social zeitgebers and biological rhythms: A unified approach to understanding the etiology of depression. *Archives of General Psychiatry*, *45*(10), 948. https://doi.org/10.1001/archpsyc.1988.01800340076012

Frank, E., Hlastala, S., Ritenour, A., Houck, P., Tu, X. M., Monk, T. H., Mallinger, A. G., & Kupfer, D. J. (1997). Inducing lifestyle regularity in recovering bipolar disorder patients: Results from the maintenance therapies in bipolar disorder protocol. *Biological Psychiatry*, *41*(12), 1165–1173. https://doi.org/10.1016/S0006-3223(96)00241-7

Grandin, L. D., Alloy, L. B., & Abramson, L. Y. (2006). The social zeitgeber theory, circadian rhythms, and mood disorders: Review and evaluation. *Clinical Psychology Review*, *26*(6), 679–694. https://doi.org/10.1016/j.cpr.2006.07.001

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, *40*(8), 869–893. https://doi.org/10.1016/S0005-7967(01)00061-4

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review*, *31*(2), 225–235. https://doi.org/10.1016/j.cpr.2010.04.003

Hayes, A. F. (2013). *Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach.* Guilford Press.

Hiller, R. M., Johnston, A., Dohnt, H., Lovato, N., & Gradisar, M. (2015). Assessing cognitive

processes related to insomnia: A review and measurement guide for Harvey's cognitive

model for the maintenance of insomnia. *Sleep Medicine Reviews*, *23*, 46–53.

https://doi.org/10.1016/j.smrv.2014.11.006

Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and Prevention of

Depression. *Psychological Science in the Public Interest*, *3*(2), 39–77.

https://doi.org/10.1111/1529-1006.00008

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., Gortner,

E., & Prince, S. E. (1996). *A Component Analysis of Cognitive-Behavioral Treatment for

Depression*. 10.

Kay, D. B., Buysse, D. J., Germain, A., Hall, M., & Monk, T. H. (2015). Subjective–objective

sleep discrepancy among older adults: Associations with insomnia diagnosis and

insomnia treatment. *Journal of Sleep Research*, *24*(1), 32–39.

https://doi.org/10.1111/jsr.12220

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Chatterji, S., Lee, S., Ormel, J., Üstün, T. B., &

Wang, P. S. (2009). The global burden of mental disorders: An update from the WHO

World Mental Health (WMH) Surveys. *Epidemiologia e Psichiatria Sociale*, *18*(1), 23–

33.

Krause, A. J., Simon, E. B., Mander, B. A., Greer, S. M., Saletin, J. M., Goldstein-Piekarski, A.

N., & Walker, M. P. (2017). The sleep-deprived human brain. *Nature Reviews

Neuroscience*, *18*(7), 404–418. https://doi.org/10.1038/nrn.2017.55

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (n.d.). *The Patient Health Questionnaire-2:

validity of a two-item depression screener*. *41*(11), 10.

Lacks, P. (1987). *Behavioral treatment for persistent insomnia*. Pergamon Press.

Lockley, S. W., Skene, D. J., & Arendt, J. (1999). Comparison between subjective and

actigraphic measurement of sleep and sleep rhythms. *Journal of Sleep Research*, *8*(3),

175–183. https://doi.org/10.1046/j.1365-2869.1999.00155.x

Lorenzo-Luaces, L., & Dobson, K. S. (2019). Is Behavioral Activation (BA) More Effective than

Cognitive Therapy (CT) in Severe Depression? A Reanalysis of a Landmark Trial.

International Journal of Cognitive Therapy, *12*(2), 73–82.

https://doi.org/10.1007/s41811-019-00044-8

Malkoff-Schwartz, S., Frank, E., Anderson, B. P., Hlastala, S. A., Luther, J. F., Sherrill, J. T.,

Houck, P. R., & Kupfer, D. J. (2000). Social rhythm disruption and stressful life events in

the onset of bipolar and unipolar episodes. *Psychological Medicine*, *30*(5), 1005–1016.

https://doi.org/10.1017/S0033291799002706

Malkoff-Schwartz, S., Frank, E., Anderson, B., Sherrill, J. T., Siegel, L., Patterson, D., &

Kupfer, D. J. (1998). Stressful life events and social rhythm disruption in the onset of

manic and depressive bipolar episodes: a preliminary investigation. *Archives of General*

Psychiatry, *55*(8), 702. https://doi.org/10.1001/archpsyc.55.8.702

Margraf, J., Lavallee, K., Zhang, X., & Schneider, S. (2016). Social rhythm and mental health: a

cross-cultural comparison. *PLOS ONE*, *11*(3), e0150312.

https://doi.org/10.1371/journal.pone.0150312

Mistlberger, R. E., & Skene, D. J. (2004). Social influences on mammalian circadian rhythms:

Animal and human studies. *Biological Reviews*, *79*(3), 533–556.

https://doi.org/10.1017/S1464793103006353

Monk, T. H., Frank, E., Potts, J. M., & Kupfer, D. J. (2002). A simple way to measure daily lifestyle regularity. *Journal of Sleep Research*, *11*(3), 183–190. https://doi.org/10.1046/j.1365-2869.2002.00300.x

Monk, T. H., Kupfer, D. J., Frank, E., & Ritenour, A. M. (1991). The social rhythm metric (SRM): Measuring daily social rhythms over 12 weeks. *Psychiatry Research*, *36*(2), 195–207. https://doi.org/10.1016/0165-1781(91)90131-8

Monk, T. H., Reynolds, C. F., Buysse, D. J., DeGrazia, J. M., & Kupfer, D. J. (2003). The relationship between lifestyle regularity and subjective sleep quality. *Chronobiology International*, *20*(1), 97–107. https://doi.org/10.1081/CBI-120017812

National Institute of Mental Health. (2017). *Mental Health Information: Statistics*. https://www.nimh.nih.gov/health/statistics/any-anxiety-disorder.shtml

National Sleep Foundation. (2014). *Inaugural Sleep Health Index*, Washington, D.C.

National Sleep Foundation. (2018). *Sleep in America Poll - Summary of Findings.*, Washington, D.C.

Neckelmann, D., Mykletun, A., & Dahl, A. A. (2007). Chronic insomnia as a risk factor for developing anxiety and depression. *Sleep*, *30*(7), 873–880.

Norell-Clarke, A., Jansson-Fröjmark, M., Tillfors, M., Harvey, A. G., & Linton, S. J. (2014). Cognitive processes and their association with persistence and remission of insomnia: Findings from a longitudinal study in the general population. *Behaviour Research and Therapy*, *54*, 38–48. https://doi.org/10.1016/j.brat.2014.01.002

Novati, A., Roman, V., Cetin, T., Hagewoud, R., den Boer, J. A., Luiten, P. G. M., & Meerlo, P. (2008). Chronically restricted sleep leads to depression-like changes in neurotransmitter

receptor sensitivity and neuroendocrine stress reactivity in rats. *Sleep*, *31*(11), 1579–1585. https://doi.org/10.1093/sleep/31.11.1579

Nutt, D., Wilson, S., & Paterson, L. (2008). Sleep disorders as core symptoms of depression. *Dialogues in Clinical Neuroscience*, *10*(3), 329–336.

Papadimitriou, G. N., & Linkowski, P. (2005). Sleep disturbance in anxiety disorders. *International Review of Psychiatry*, *17*(4), 229–236. https://doi.org/10.1080/09540260500104524

Rutledge, C. M., Guardia, A. C. L., & Bluestein, D. (2013). Predictors of self-efficacy for sleep in primary care. *Journal of Clinical Nursing*, *22*(9–10), 1254–1261. https://doi.org/10.1111/jocn.12005

Schimitt, R. L., Zanetti, T., Mayer, M., Koplin, C., Guarienti, F., & Hidalgo, M. P. (2010). Psychometric properties of Social Rhythm Metric in regular shift employees. *Revista Brasileira de Psiquiatria*, *32*(1), 47–55. https://doi.org/10.1590/S1516-44462010000100010

Shear, M. K., Randall, J., Monk, T. H., Ritenour, A., Tu, X., Frank, E., Reynolds, C., & Kupfer, D. J. (1994). Social rhythm in anxiety disorder patients. *Anxiety*, *1*(2), 90–95.

Shen, G. H., Alloy, L. B., Abramson, L. Y., & Sylvia, L. G. (2008). Social rhythm regularity and the onset of affective episodes in bipolar spectrum individuals. *Bipolar Disorders*, *10*(4), 520–529. https://doi.org/10.1111/j.1399-5618.2008.00583.x

Spitzer, R. L., Kroenke, K., & Williams, J. B. W. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary care study. *JAMA*, *282*(18), 1737. https://doi.org/10.1001/jama.282.18.1737

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A brief measure for

    sssessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine*,

    166(10), 1092. https://doi.org/10.1001/archinte.166.10.1092

Takano, K., Iijima, Y., & Tanno, Y. (2012). Repetitive thought and self-reported sleep

    disturbance. *Behavior Therapy*, *43*(4), 779–789.

    https://doi.org/10.1016/j.beth.2012.04.002

Walker, M. P. (2008). Cognitive consequences of sleep and sleep loss. *Sleep Medicine*, *9*, S29–

    S34. https://doi.org/10.1016/S1389-9457(08)70014-5

Walker, M. P. (2010). Sleep, memory and emotion. In *Progress in Brain Research* (Vol. 185, pp.

    49–68). Elsevier. https://doi.org/10.1016/B978-0-444-53702-7.00004-X

Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual Review of*

    Psychology, *57*(1), 139–166. https://doi.org/10.1146/annurev.psych.56.091103.070307

Wehr, A. (1987). Sleep reduction as a final common pathway in the genesis of mania. *Am J*

    Psychiatry, 4.

World Health Organization. (2018). *Mental Disorders: Key Facts*. https://www.who.int/news-

    room/fact-sheets/detail/mental-disorders

Appendix A: Brief Social Rhythm Scale

The following statements are related to your life rhythm. You will find a series of statements about various every day activities. Please indicate how regularly you perform each of these activities.

	Very regularly (1)	Quite regularly (2)	Somewhat regularly (3)	Somewhat irregularly (4)	Quite irregularly (5)	Very irregularly (6)
Going to bed Mondays through Fridays	○	○	○	○	○	○
Going to bed on the weekend	○	○	○	○	○	○
Getting out of bed Mondays through Fridays	○	○	○	○	○	○
Getting out of bed on the weekend	○	○	○	○	○	○
Meeting other people at school or work Mondays through Fridays	○	○	○	○	○	○

Appendix B: RU SATED Scale (Sleep Health)

The following questions are related to sleep. Please respond by marking <u>one</u> box per row.

	Rarely/Never (1)	Sometimes (2)	Usually/Always (3)
Do you go to bed and get out of bed at the same time (within one hour) every day?	○	○	○
Are you satisfied with your sleep?	○	○	○
Do you stay awake all day without dozing?	○	○	○
Are you asleep (or trying to sleep) between 2:00 a.m. and 4:00 a.m.?	○	○	○
Do you spend less than 30 minutes awake at night? (This includes the time it takes to fall asleep plus awakenings during sleep.)	○	○	○
Do you sleep between 7 and 9 hours per day?	○	○	○

Appendix C: The Self-Efficacy for Sleep Scale

Please answer the following questions about your sleep.
For the following nine questions, rate your ability to carry out each behavior. If you feel able to accomplish a behavior some of the time but not always, you should indicate a lower level of confidence.

	Not confident at all				Very confident
	1	2	3	4	5
Lie in bed, feeling physically relaxed	○	○	○	○	○
Lie in bed, feeling mentally relaxed	○	○	○	○	○
Lie in bed with your thoughts "turned off"	○	○	○	○	○
Fall asleep in under 30 minutes	○	○	○	○	○
Wake up at night fewer than 3 times	○	○	○	○	○
Go back to sleep within 15 minutes of waking in the night	○	○	○	○	○
Feel refreshed upon waking in the morning	○	○	○	○	○
Wake after a poor night's sleep without feeling upset about it	○	○	○	○	○
Not allow a poor night's sleep to interfere with daily activities	○	○	○	○	○

Appendix D: PHQ-2 and GAD-2

Over the past 2 WEEKS, how often have you been bothered by any of the following problems?

	Not at all (1)	Several days (2)	More than half the days (3)	Nearly every day (4)
Little interest or pleasure in doing things (1)	○	○	○	○
Feeling down, depressed, or hopeless (2)	○	○	○	○
Feeling nervous, anxious, or on edge (11)	○	○	○	○
Not being able to stop or control worrying (7)	○	○	○	○

Vita

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