THE EFFECT OF SHORT-TERM EXERCISE ON SLEEP AND DAYTIME IMPAIRMENT IN ADULTS WITH INSOMNIA

by

Andrew George Steven Kubala

B.S. Exercise Science, Slippery Rock University, 2015

M.S. Health Physical Activity and Chronic Disease, University of Pittsburgh, 2017

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This dissertation was presented

by

Andrew George Steven Kubala

It was defended on

October 4, 2021

and approved by

Bethany Barone Gibbs PhD, Associate Professor, Department of Health and Human Development, University of Pittsburgh

Daniel J. Buysse MD, UPMC Endowed Chair in Sleep Medicine and Professor, Department of Psychiatry, University of Pittsburgh

John M. Jakicic PhD, Senior Investigator, Director of Population Science, AdventHealth – Translational Research Institute

Dissertation Director: Christopher E. Kline PhD, Associate Professor, Department of Health and Human Development, University of Pittsburgh

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Andrew George Steven Kubala, M.S

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Evidence suggests acute exercise is related to improved sleep quality, though research is largely limited to normal sleepers. Whether acute exercise impacts sleep and daytime impairment in samples of adults with insomnia disorder remains unclear. **Methods:** Following a baseline sleep and physical activity monitoring week, 24 participants with insomnia (70.8% white, 70.8% female, age: 33.7±9.8 y, body mass index: 25.8±4.0 kg/m^2) were randomized to one of two experimental conditions that took place on three non-consecutive days over a week-long observation period: 1) moderate-intensity exercise (i.e., 30-minute walks performed at 50% of heart rate reserve); or 2) quiet rest (i.e., watching documentaries while seated). Daily sleep behavior was collected using a diary and wrist-worn actigraphy. Subjective pre-sleep arousal was measured in the diary (i.e., Pre-sleep Arousal Scale (PSAS)). Insomnia severity, daytime impairment, and sleep quality were assessed at the end of the baseline and experimental weeks with the Insomnia Severity Index, PROMIS Sleep-related Impairment scale, and PROMIS Sleep Disturbance scale, respectively. Exercise sessions were monitored with a heart rate monitor and self-report; quiet rest sessions were monitored via video calls. Repeated measures analysis of variance (ANOVA) models were used to examine mean-level differences in sleep between weeks with interactions by condition. Linear mixed effect models examined day-level associations between PSAS and sleep and the difference in sleep behavior on nights following exercise versus non-exercise days. **Results:** Both experimental groups showed reduced insomnia severity and daytime impairment between weeks

($p<0.01$), with no interactions by condition over time ($p>0.35$). Inconsistent within-group improvements in sleep behavior were found, with no condition x time interactions observed ($p>0.086$). Day-level analyses found an association between pre-sleep arousal and sleep (actigraphy-based sleep efficiency [mean±standard error]: B=-0.3±0.1%, p=0.030; actigraphy-based wake after sleep onset: B=1.6±0.5 min, p=0.048; sleep quality B=-0.1±0.2, p=0.001). Sleep parameters were significantly improved following exercise versus non-exercise days (actigraphy-based total sleep time: B=0.9±0.4 h, p= 0.032; diary-based total sleep time: B=0.8±0.4 h, p= 0.049; diary-based morning restedness: B=0.5±0.2, p=0.019). **Conclusions:** In participants with insomnia, acute exposure to walking exercise may not elicit global improvements in sleep and daytime function, though sleep may improve the night after exercise sessions.

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Preface

I owe *every one* of my successes in part to my parents. I would not be where I am today without leaning against the unwavering support of my mother Suzanne MacMurdo. She is strong, thoughtful, understanding, analytical, and practical. She helped guide me and keep me going even when I didn't know I could. My father, Michael MacMurdo, is the hardest working man I know. He does not quit, he does not complain, and he has moved mountains for me. I am grateful for their guidance.

I owe gratitude to my partner Alex Inman. We met at the beginning of my training, and he stuck around long enough to see it through into the next chapter of our lives. He sacrificed a lot for me, and I only hope I can return the favor. He reminds me that sometimes life is simpler than people make it out to be and that sometimes we are the ones in our own way. I am grateful for his love.

I owe my other family and friends a huge thank you as well. I have loved ones to call, ask for wild favors, study with, cry with, laugh with, contemplate existence with, and honestly just complain. It is one of my privileges that I know others may not have. I am grateful for their support.

I owe Dr. Christopher Kline gratitude for guiding me, being patient with me, expecting more from me, giving me various opportunities, and ultimately making sure I can be proud of what I do. I am also thankful for my dissertation committee—Dr. Bethany Barone Gibbs, Dr. John Jakicic, and Dr. Daniel Buysse—who have each contributed to my development long before the start of this project. Lastly, I am extremely grateful for the training from faculty and mentors within the University of Pittsburgh Center for Sleep and Circadian Science during my time at the University of Pittsburgh and look forward to future collaborations!

1.0 INTRODUCTION

1.1 BACKGROUND

Sleep is a unique physiological state that is present across all living species and critical for optimal health. However, despite the necessity of sleep, poor and/or insufficient sleep is common in society. Sleep-related disorders such as sleep apnea, restless legs syndrome, and insomnia are prevalent in adults.[1,2] Insomnia is the most prevalent sleep disorder and characterized as a dissatisfaction with sleep and an inability to fall asleep or stay asleep in conjunction with daytime impairment due to poor sleep. In the adult population, 10-20% meet diagnostic criteria and up to 30% experience symptoms of insomnia.[3] Insomnia understandably has a major impact on daytime functioning, but it is also associated with a variety of poor health outcomes. Insomnia has been linked to an increased risk of cardiovascular disease, lower quality of life, and increased health care utilization.[4-6] As a result, insomnia is now recognized as a significant public health concern. Additional understanding into its pathophysiology, association with health outcomes, and treatment options is greatly needed.[7]

Insomnia is primarily treated with cognitive-behavioral therapy for insomnia (CBT-I) or hypnotic medication. While both have shown to be effective at improving sleep and reducing insomnia severity, each of these treatments have limitations. Due to its long-term effectiveness and minimal risks, CBT-I is the gold-standard treatment strategy for insomnia.[8] However, CBT-I is an intensive program that traditionally relies upon in-person meetings with trained providers and high levels of participant engagement to garner success.[9] CBT-I may also not be readily covered

by insurance providers, further limiting access to the treatment.[10] Despite the emergence of CBT-I as the recommended first-line treatment for insomnia, hypnotic medications remain commonly prescribed in the United States.[9-11] Unfortunately, hypnotic medications have potential side effects such as morning sedation, dependence, and increased risk of falls and fractures in the elderly.[12,13] These limitations of current treatments warrant investigation into identifying accessible non-pharmacological treatments to improve sleep and reduce daytime impairment. Exercise has been proposed as a non-pharmacological treatment for poor sleep. Both single exercise bouts (i.e., acute) and chronic exercise training (i.e., 4-24 weeks) have been shown to improve objective and subjective domains of sleep, including duration, continuity, and quality.[14] However, most studies have utilized samples of adults who report no or minimal sleep complaints.[14,15]

Limited experimental research has explored the effects of chronic exercise training (> 1 week of exercise) on sleep and daytime impairment in samples of adults who meet diagnostic criteria for insomnia. In samples of adults with insomnia disorder, most randomized controlled trials (RCTs) that have used exercise training found improvements in subjective sleep quality and reductions in insomnia symptoms, however, not all have found a beneficial impact of aerobic exercise on sleep.[16,17,18] Non-randomized experimental studies have found modest improvements in actigraphic and polysomnographic sleep in addition to improvements in self-reported sleep quality in samples with insomnia disorder.[19-21] These mixed results, albeit from small sample sizes, potentially indicate a modest effect of exercise on objective measures of sleep.

In contrast, only a small number of acute exercise studies (i.e., 1 bout of exercise with subsequent sleep measurement) have been conducted to better understand any immediate effects on sleep in adults with insomnia. Investigations into the acute effect of exercise are important for improving the clinical message of how exercise adoption may impact sleep in patients with

insomnia. Acute studies are not only important to understand the biological mechanisms in which exercise may improve sleep but may also distinguish any immediate treatment effects due to exercise and inform the potential utility of prescribing exercise in insomnia treatment. To date, there is only one acute exercise study in a sample of adults who meet diagnostic criteria for insomnia.[22] Two other acute studies of similar methodology sampled participants with various degrees of insomnia symptoms.[23,24] In general, these studies have found that acute moderate-intensity aerobic exercise improved sleep as measured by actigraphy and polysomnography. Although beneficial effects of acute exercise have been found, a secondary analysis of an exercise training RCT found that exercise bout duration was not associated with the subsequent night's sleep; however, better sleep the previous night was related to longer exercise bout duration the next day.[25] In contrast to the other experimental studies, this study suggests that one exercise bout may not elicit an effect on sleep in adults with insomnia.[26]

In response to the current literature, we aimed to better understand the short-term clinical benefit of adopting exercise training among adults with insomnia. Including multiple bouts of exercise in a short time span while measuring sleep with objective and self-reported measures may be more reflective of capturing acute effects of exercise implementation on the sleep of those with insomnia. We proposed to conduct a randomized parallel group trial to evaluate the effects of short-term moderate-intensity aerobic exercise on insomnia severity and subjective and objective measures of sleep in a sample of adults who met diagnostic criteria for insomnia. This project has addressed prior limitations in this area of research by utilizing a sample with diagnosed insomnia, assessing multiple nights of sleep, and implementing multiple bouts of exercise stimuli within an acute time period. This project examined the following aims:

1.2 SPECIFIC AIMS

Aim I: To examine whether 1 week of moderate-intensity aerobic exercise improves objective and subjective sleep compared to 1 week without exercise in a sample of adults who meet diagnostic criteria for insomnia.

Hypothesis: *The week consisting of aerobic exercise will improve sleep compared to a week without exercise (primary outcome: actigraphy-assessed sleep efficiency; secondary outcome: PROMIS Sleep Disturbance questionnaire).*

Aim II: To examine whether 1 week of moderate-intensity aerobic exercise reduces daytime impairment compared to 1 week without exercise in a sample of adults who meet diagnostic criteria for insomnia.

Hypothesis: *The week consisting of aerobic exercise will reduce daytime impairment compared to a week without exercise (primary outcome: Insomnia Severity Index; secondary outcome: PROMIS Sleep-Related Impairment questionnaire).*

Exploratory Aim I: To explore whether pre-sleep arousal (*measure: Pre-Sleep Arousal Scale*) is associated with that night's objective and subjective sleep and whether this association is different between the exercise and control conditions *(primary outcome: actigraphy-assessed sleep efficiency).*

Exploratory Aim II: To explore whether sleep differs following a day in which there is structured exercise in comparison to a non-exercise day.

Exploratory Aim III: To explore whether the effect of exercise on the subsequent night's sleep differs from the first to the third exercise session among those allocated to the exercise condition.

1.3 THEORETICAL FRAMEWORK AND CLINICAL SIGNIFICANCE

Multiple mechanisms have been proposed to explain how exercise improves sleep characteristics. Yet, many of these remain speculative hypotheses and lack experimental evidence, particularly in humans. Additionally, the studies that do explore mechanisms lack investigation into pathways that may be specific to insomnia. **Figure 1** summarizes how acute and chronic exercise could improve sleep in adults with and without insomnia. Further explanation of the biological mechanisms between exercise and improved sleep is found in **Section 2.3.2**.

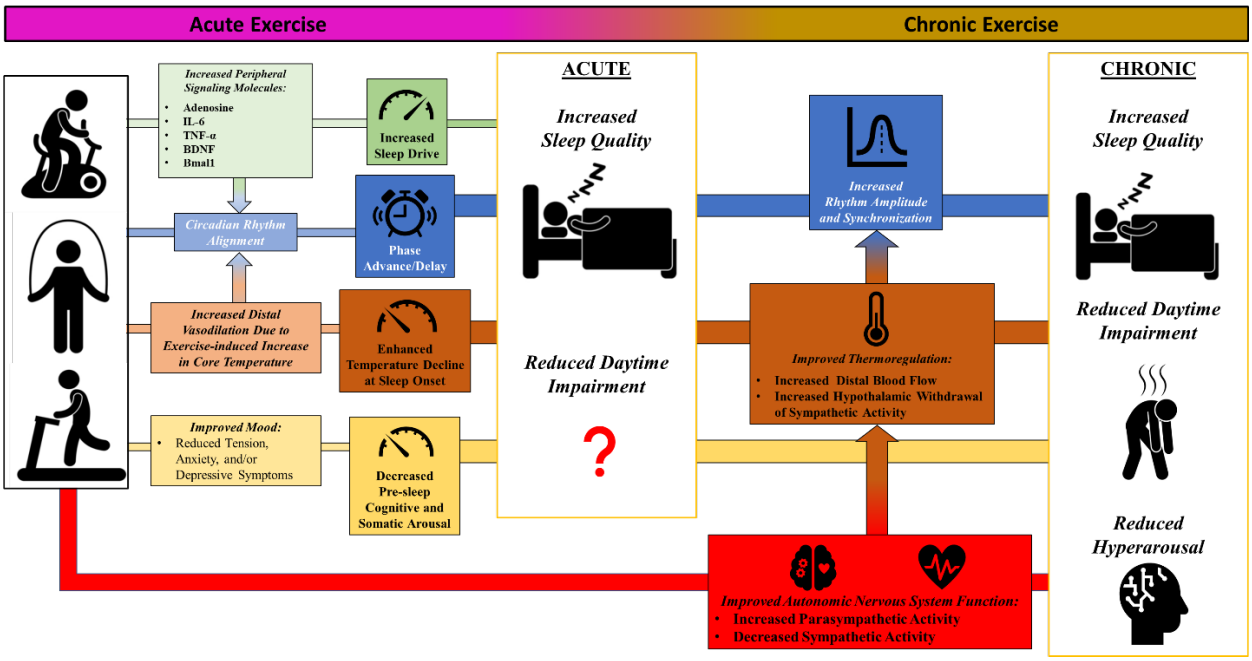


Figure 1. Postulated mechanisms of the effect of exercise on sleep and impairment

Insomnia is often characterized as a disorder of hyperarousal through which somatic, cognitive, or neuronal body systems are more active than necessary, resulting in difficulty promoting adequate sleep.[13,27] Cognitive hyperarousal encompasses feelings of sleep-related worry or anxiousness (e.g., attention to sleep-related threats such as noises, ruminating thoughts),

while somatic hyperarousal encompasses physiology such as tension, core and distal body temperature, and autonomic nervous system responses. Acute exercise may improve cognitive or somatic hyperarousal through mechanisms that improve mood state (cognitive arousal) or through improved temperature reduction prior to bedtime (somatic arousal), respectively.[28,29] Additionally, non-arousal pathways, such as increased sleep drive from the accumulation of extracellular adenosine or positive changes to the timing and strength of circadian rhythms, may also be implicated in explaining how acute exercise improves sleep in adults with insomnia.

Insomnia is often linked with sleep-related worry and anxiousness near bedtime.[30,31] Acute exercise has anxiolytic effects; as a result, it may reduce pre-sleep cognitive arousal in people with insomnia.[32-34] As a potential marker of pre-sleep anxiety and physiological arousal, this proposed study will be the first to investigate whether bouts of aerobic exercise improve self-reported pre-sleep arousal in adults with insomnia.

Although this mechanism has not been tested in humans, acute exercise in mice leads to increased adenosine concentration in the whole brain. Increased accumulation of adenosine is a marker of increased sleep drive and thus could represent a pathway through which acute exercise improves sleep.[35] Additionally, peripheral signaling molecules such as the cytokines interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α) have been associated with sleep initiation, and moderate-intensity exercise has shown to result in modest transient increases in IL-1, IL-6, and TNF-α. Thus, exercise may beneficially impact sleep by inducing the release of these sleep-promoting cytokines.[36]

Adults with insomnia may also have higher 24-hour core body temperatures and a reduced ability to dissipate heat at bedtime.[37,38] This is important as body cooling prior to bedtime has been associated with more rapid initiation of sleep.[38] Evening exercise has been proposed to help the

body cool by increasing core body temperature which subsequently initiates the decrease in temperature via distal vasodilation prior to sleep.[37]

Although direct scientific testing of biological mechanisms did not occur, this study addresses critical research gaps that will help inform sleep medicine on how exercise may impact sleep among adults with insomnia. This is the second study to explore the effect of acute exercise in a sample that meets diagnostic criteria for insomnia disorder. This study was also the first to utilize multiple bouts of acute exercise distributed throughout a week to explore the effect of short-term aerobic exercise on sleep and daytime impairment. This study may inform clinicians whether patients with insomnia may experience immediate benefit in their sleep and daytime functioning after the short-term adoption of an aerobic exercise regimen. This study may also inform future research. From these data, future research can investigate the extent to which acute exercise implementation varies from chronic exercise training and investigate the effects on habitual (>1 week) and daily measures of hyperarousal, objective sleep, and daytime impairment in insomnia. Understanding the time course of changes in sleep and daytime function following adoption of an exercise regimen by adults with insomnia will help clinicians better calibrate insomnia patients' expectations to treatment with exercise.

2.0 REVIEW OF THE LITERATURE

2.1 EPIDEMIOLOGY AND PATHOPHYSIOLOGY OF INSOMNIA

2.1.1 Definition and Prevalence of Insomnia

Insomnia disorder is defined as a dissatisfaction with sleep quality or quantity that is concurrent with one or more of the following sleep-related complaints: difficulty initiating sleep, inability to maintain sleep with frequent nighttime awakenings, or an inability to fall back asleep after early morning awakenings.[31,39] The dissatisfaction and symptomatology of insomnia are present even with adequate opportunity for sleep and must also be associated with subsequent sleep-related physical, cognitive, social, or psychological domains of daytime impairment. To meet DSM-5 diagnostic criteria for insomnia disorder, these sleep-related complaints must be present ≥ 3 nights per week and occur for ≥ 3 months.[31,39] Insomnia can occur on its own or co-morbid with other health conditions.[13,31] It is recommended that official diagnosis of insomnia occur through clinical interviews with detailed patient histories. There are no biological assays for insomnia, and objective measurement of sleep via polysomnography is not clinically recommended except to rule out other sleep-related disorders such as sleep apnea or periodic limb movements.[13,40]

The prevalence of insomnia is difficult to precisely determine due to the variable definition of insomnia in epidemiological studies. When loosely defined as experiencing insomnia symptoms, the worldwide adult prevalence is around 30-35%.[3,31] Utilizing Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) criteria, insomnia prevalence estimates are

10-23%.[3,41] Some epidemiological studies determined estimates of insomnia that are comorbid with another medical or psychiatric condition and found a prevalence of 4.4-6.4%.[3] Furthermore, data analyzed from the American Insomnia Survey of over 6,000 respondents found differences in the prevalence of experiencing certain types of insomnia symptoms.[42] Specifically, difficulty maintaining sleep (61.0%) and early morning awakenings (52.2%) are more common than difficulty initiating sleep (37.7%) or nocturnal dissatisfaction with sleep (25.5%).[42] However, experiencing multiple symptoms has been found to be more common than only experiencing a single symptom.[43]

Certain demographic characteristics are associated with a higher prevalence of insomnia symptoms and diagnosis. Compared to males, women are more likely to have insomnia symptoms, experience daytime impairment, and meet diagnostic criteria for insomnia disorder.[3,44] Increasing age has also been associated with increased risk of insomnia diagnosis and presence of insomnia symptoms such as frequent nighttime awakenings.[3,31,41] However, the estimated prevalence of insomnia disorder in younger to older adults remains similar due to a lack of difference in reports of daytime impairment despite increases in nocturnal sleep problems.[41] Although further large scale multi-ethnic studies are needed, some studies indicate a greater prevalence of insomnia in Hispanics and African Americans compared to Caucasians.[45-49] Lastly, lower socioeconomic status has been predictive of increased risk for insomnia symptoms.[45,46,48]

Future epidemiological work on the prevalence of insomnia needs to be clear in identifying those who meet diagnostic criteria and delineating those samples from others who only identify the presence of insomnia symptoms. Additionally, better understanding of the demographic correlates of the disorder could better inform the development of tailored treatment options. The high prevalence of this disorder is an important public health concern that has sparked exploration

into the etiology and pathophysiology of the disorder. An overview of the hallmark pathophysiology and health risk associated with insomnia is provided below.

2.1.2 Pathophysiology: 3-P Model of Insomnia

Insomnia is a heterogenous disorder that lacks a universally accepted model of etiology and pathophysiology. Subsequently, there are multiple models of the pathophysiology of insomnia that co-occur and focus on different components of insomnia across behavioral, cognitive, and neurobiological targets.[50] One overarching stress-diathesis model of insomnia globally focuses on predisposing, precipitating, and perpetuating factors that contribute to acute and chronic insomnia through non-modifiable and behavioral mechanisms.[51] Predisposing factors are generally non-modifiable and can be physiological and psychological contributors to the risk of insomnia development. Hallmark predisposing factors include genetic influences, personality traits such as a propensity for worry or rumination, female gender, increasing age, and societal restraints on preferred sleep schedules. Precipitating factors are acute occurrences that trigger the onset of insomnia, such as stressful life events or onset of illness. Lastly, perpetuating factors are maladaptive behaviors that are developed to cope with sleep loss (e.g., increased time in bed, napping) but in turn contribute to and perpetuate the insomnia. This "3-P" model of insomnia is complementary to the stimulus control model of insomnia, proposed by Bootzin and colleagues.[52] The stimulus control model suggests that what was once sleep-promoting stimuli, such as the sleep environment and pre-sleep routines, become stimuli that promote anxiety, wakefulness, and hyperarousal through classical conditioning.[31,51] Both of these models of insomnia are practical,

broad, and efficacious models through which chronic insomnia is currently treated. However, they do not address the potential physiological and neurobiological causes of insomnia.

2.1.3 Pathophysiology: Hyperarousal in Insomnia

Insomnia is commonly characterized as a disorder of hyperarousal. Hyperarousal can be defined as increased sensitivity and subsequent response to stimuli which disrupt the natural ability to sleep.[27,31,53] Hyperarousal is a somewhat nebulous term that has been in common reference to multiple physiological characteristics that are considered heightened in insomnia compared to healthy sleepers, with these characteristics spanning across somatic, cognitive, and neuronal systems depending on the specific pathophysiological model of insomnia.[50] Specifically, Levenson and colleagues provide a comprehensive schematic of the pathophysiology of insomnia framing the contributing factors that may lead to general hyperarousal in insomnia through psychological and neurophysiological body systems (**Figure 2**).[53]

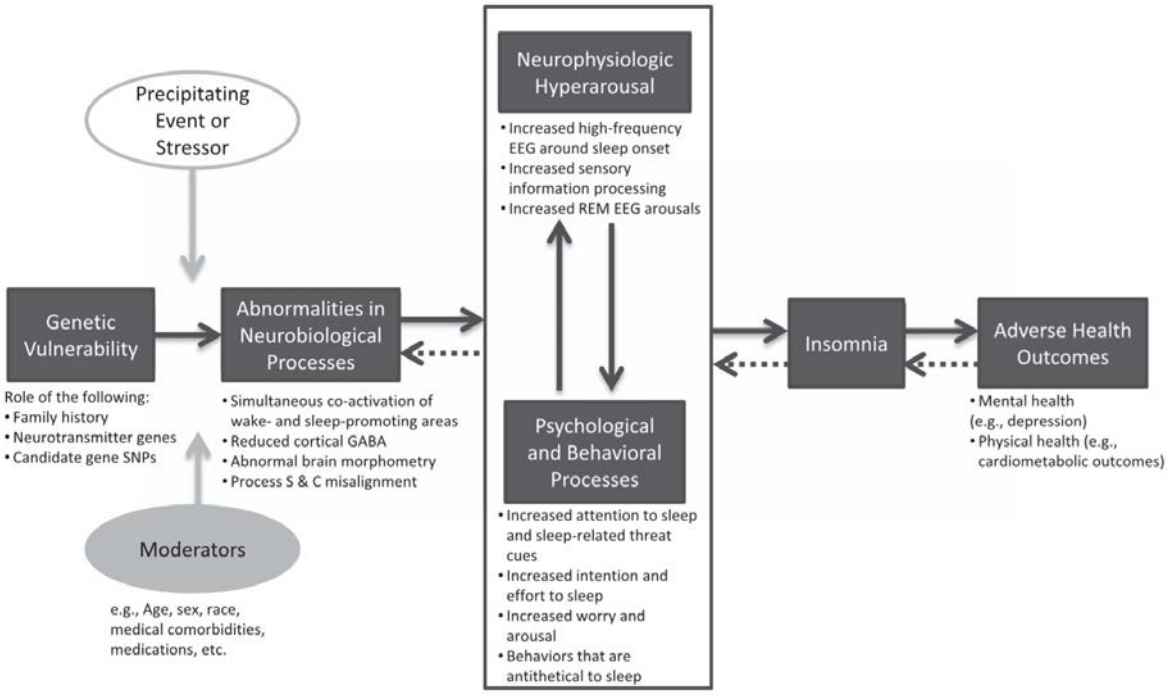


Figure 2. Pathophysiology of insomnia proposed by Levenson and colleagues[53]

Psychological and behavioral processes contribute to cognitive hyperarousal in which sleep-related worry increases attention towards threats to sleep. As proposed by cognitive models of insomnia,[54] these threats may include environmental cues (e.g., light, noise, temperature), attention to the passage of time, and internal threats such as feelings of alertness and wakefulness.[30,53,55,56] These negative cognitive cues lead to sleep-compensatory behaviors and an increased attention to sleep-related daytime impairment.[51,53] Coupled with cognitive arousal, there is a documented sleep state discrepancy in some adults with insomnia where they may have physiologically normal sleep according to polysomnography, but report the experience of wakefulness, poor sleep quality, and arousal.[57] This mismatch is a potential downstream symptom explained by the neurobiological model of insomnia proposed by Buysse and colleagues.[58]

The neurobiological model highlights the neuronal sleep-initiating and wake-inhibiting processes of the brain and suggests that the "sleep switch" is dysregulated in insomnia, which leads to simultaneous sleep-wake features as displayed by electroencephalography (EEG) of cortical brain regions during non-rapid eye movement (NREM) sleep.[53,58] Studies have found greater high-frequency EEG activity in people with insomnia compared to controls.[53,58,59] Furthermore, when comparing brain glucose metabolism in various brain regions in people with insomnia compared to controls, studies document greater glucose metabolism in specific brain regions among people with insomnia transitioning from wake to NREM sleep compared to controls.[60,61] This suggests a dysregulation of sleep-promoting and wake-inhibiting brain regions in insomnia.

In addition to cognitive and neurobiological arousal, somatic arousal has been documented in insomnia. Specifically, indices of autonomic and central nervous system dysregulation such as heart rate and body temperature, respectively, have been shown to be elevated in people with insomnia compared to normal sleepers.[27,62] Nighttime indices of heart rate variability have been shown to be lower in people with insomnia compared to normal sleepers, indicating a reduced withdrawal of sympathetic nervous system activity and lower level of parasympathetic activity.[63] In addition, cardiovascular measures of sympathetic nervous system withdrawal from the transition from wakefulness to sleep have shown to be blunted in adults with insomnia compared to controls.[64] Indices of systemic arousal such as higher body temperature and increased basal metabolic rate have been documented in insomnia compared to normal sleeping counterparts as well.[65,66] Increased activation of the hypothalamic-pituitary-adrenal (HPA) axis, as represented by circulatory and urinary cortisol levels, has also been documented.[27,67] Compared to healthy controls, studies have shown higher levels of 24-hour urinary cortisol as well as higher

serum levels of cortisol and adrenocorticotropic hormone prior to sleep and during the first half of the night among adults with insomnia.[27,67] These physiological markers may be interpreted as indicative of hyperarousal, yet it is not exactly clear whether these markers are causal or a downstream effect of insomnia. It has also been proposed that subjects with insomnia and shorter sleep duration have greater markers of hyperarousal than their normal sleep duration counterparts, exemplified by studies that show longer multiple sleep latency tests and increased vigilance in subjects with insomnia and short sleep duration.[68] However, this literature does not discount the possibility that hyperarousal is present in those with insomnia and normal sleep duration. It is also important to note that other studies have not found differences in somatic markers in insomnia, which may attest to the heterogeneity of this disorder.[69-71]

In summary, physiological and psychological hyperarousal is a hallmark feature of the pathophysiology of insomnia across cognitive, behavioral, and neurobiological models. These pathophysiological mechanisms are the target of treatment strategies to ultimately reduce insomnia severity and minimize its health and daytime consequences. Below the common health outcomes associated with insomnia are outlined.

2.2 HEALTH IMPACT AND TREATMENT OF INSOMNIA

2.2.1 Health Consequences Associated with Insomnia

In addition to its high prevalence, insomnia is associated with poor health outcomes and significant economic costs. There is strong evidence linking insomnia as a predictor or consequence of

psychiatric disorders. Insomnia is commonly co-morbid with psychiatric conditions such as anxiety and depression; two-thirds of people with depression complain of sleeping difficulties while sleep onset and maintenance insomnia are common in those with anxiety disorder.[72,73] Insomnia may also be a risk factor for the development of mental health disorders or exacerbate symptoms of certain psychiatric disorders. Specifically, a meta-analysis determined insomnia to be a significant predictor of depression, anxiety, psychosis, and alcohol abuse, with odds ratios greater than 2.0 for depression and anxiety.[74] Individuals with bipolar disorder and insomnia have been shown to have greater thoughts of suicide and emotional impulsivity than participants with bipolar disorder but without insomnia.[75]

In addition to mental health, prospective evidence has found associations between insomnia and cardiovascular and metabolic disease risk. A meta-analysis of 13 prospective studies found a 45% increased risk of cardiovascular morbidity and mortality when insomnia symptoms were present.[6] An additional prospective study found that insomnia, when coupled with objective short sleep duration (< 6 hours), was related to a significantly greater risk of cardiovascular disease compared to insomnia or short sleep duration in isolation.[76] This highlights that phenotypes of insomnia, such as samples of insomnia with short sleep duration, may be more at risk for cardiovascular disease than others.[68] In addition to cardiovascular disease, difficulty initiating sleep and taking hypnotic medications have also been associated with a 52% increased odds of diabetes development in men.[77] A meta-analysis of prospective studies found an increased relative risk of diabetes with the presence of difficulty initiating or maintaining sleep.[78]

In addition to chronic disease risk, insomnia has been related to lower quality of life, lower overall health status, and greater feelings of pain.[79] Utilizing a nationally representative sample, insomnia was associated with a greater reduction in quality of life years than any other health

condition examined, which included hypertension, arthritis, and depression.[5] In a multi-site survey of healthcare beneficiaries, the presence of insomnia was related to a greater frequency of emergency room visits even after adjustment for comorbid conditions and demographic factors.[4] Insomnia also affects productivity and societal function. In a sample of over 2,000 US-based company workers, insomnia symptoms were related to lower presenteeism at work.[80] The most recent estimate of the economic burden of insomnia found its direct costs to be $21.8 billion annually in the United States.[81] Lastly, healthcare utilization for individuals with insomnia has been shown to be greater for inpatient and emergency department usage when compared to normal sleepers.[82,83] Insomnia is a disorder that is related to chronic disease, psychiatric disorders, as well as high economic and quality of life burden. Thus, multiple treatment options have been developed to reduce these consequences of insomnia. Outlined below are two common treatment strategies for insomnia.

2.2.2 Cognitive-Behavioral Therapy and Pharmacotherapy for Insomnia

Cognitive-behavioral therapy for insomnia (CBT-I) is considered the gold standard treatment for insomnia.[8] As an intensive intervention typically lasting 6-12 weeks, CBT-I is often guided by a medical provider (e.g., sleep medicine physician, clinical psychologist) and consists of implementing strategies related to altering the precipitating and perpetuating cognitive and environmental factors that contribute to the insomnia.[13] CBT-I comprises a set of behavioral and cognitive interventions that include restricting time in bed, optimizing the bedroom for sleep, addressing sleep-related anxiety, and reducing maladaptive behaviors that are typically seen in insomnia such as excessive caffeine intake and daytime napping.[13,84] CBT-I is considered the

initial treatment strategy for insomnia as it has shown to be efficacious as a short- and long-term strategy for insomnia severity reduction and improved sleep. Meta-analyses have shown CBT-I to improve objective measures of sleep onset latency, wake after sleep onset, and sleep quality.[9,10] Importantly, when compared to pharmacological treatments for insomnia, CBT-I has been shown to be at least as effective as hypnotic medication for short-term sleep improvement, but superior for long-term treatment at 6 months or more of follow-up.[10] Despite these advantages, CBT-I has limitations. Because it is typically administered by trained medical professionals, there is a disproportionate number of individuals in need of CBT-I and an insufficient supply of qualified practitioners to administer the treatment.[85] Clinician-level barriers have typically involved a general lack of familiarity with CBT-I and knowledge of its superiority over hypnotic medications as a treatment strategy.[85] These barriers have elicited exploration into online platforms for CBT-I administration as well as brief versions of CBT-I.[86,87] Although efficacious, additional investigation into adherence to CBT-I as well as other potential adjunct therapies is warranted.[84]

Medications are also a frontline treatment strategy for insomnia, and they are often administered alone or in combination with CBT-I.[13,31] Common hypnotic medications are benzodiazepines and benzodiazepine receptor agonists, as well as drugs not originally intended for treatment of insomnia such as anxiolytics, antidepressants, antipsychotics, and anticonvulsants.[12,13] Hypnotics have been shown to have small to moderate effects on subjective sleep quality and beneficial effects on objectively measured total sleep time, sleep continuity, and sleep onset latency.[12] Although medications are a viable short-term solution, long-term use is not generally as favorable as the use of CBT-I.[10,12,31] Additionally, sleep medications have potential side effects such as excessive morning sedation, amnesia, parasomnias (e.g., sleep walking and driving), and dependence and addiction.[12] In addition, increased mortality and cancer risk has been

found with taking even low dosages of hypnotic medications.[88] Despite these potential side effects and risks, a study utilizing the National Health and Nutrition Examination Survey dataset found that 3% of adults used a medication commonly prescribed for insomnia in the past month, with 55% of those respondents indicating taking at least one additional sedating medication.[11] Additionally, from 2011 to 2018, the percentage of patients with insomnia who were dispensed low-dose trazodone increased from 8.7% to 14.5%, while the percentage of adults dispensed zolpidem remained high but decreased from 33.7% to 22.6%.[89]

Current treatment strategies for insomnia, although beneficial, have inherent limitations that warrant additional investigation into other adjunct or alternative treatment strategies. These hypothesized strategies must improve access to care, have minimal patient burden, and lack significant health risks and side effects. The next section of this review will critically evaluate how exercise may be a viable non-pharmacological treatment for insomnia that addresses the current treatment limitations and warrants further investigation into its efficacy and utility.

2.3 PHYSICAL ACTIVITY AND INSOMNIA

2.3.1 Physical Activity and Sleep

Before exploring how exercise may directly impact insomnia, a brief review of the literature that surrounds the effects of physical activity on sleep in non-clinical populations is necessary. Broadly, five meta-analyses of experimental studies[14,90-93] and two systematic reviews[15,94] have been conducted on the effect of exercise on sleep (excluding studies involving sleep-related disorders);

each have concluded that exercise has a beneficial impact on sleep. Specifically, in a meta-analysis from Kredlow and colleagues, acute exercise (as defined as < 1 week of exercise) and chronic exercise (exercise training of ≥ 1 week) each result in small to modest improvements in sleep.[14] Acute exercise has been shown to elicit increases in sleep duration, sleep efficiency, and slow wave sleep as well as reductions in wake after sleep onset and sleep onset latency.[14] Many of these acute studies utilized polysomnography or actigraphy as an objective measure of sleep. However, most studies involving chronic exercise training assessed sleep quality with the Pittsburgh Sleep Quality Index (PSQI); pooled analyses of these studies indicate large beneficial effects of exercise training on sleep quality.[14,92] In a meta-analysis restricted to randomized controlled exercise trials involving middle-aged women, exercise significantly improved subjective sleep quality, with the significant effect being driven from trials that featured moderate-intensity physical activity, not light-intensity activity (e.g., yoga).[92]

Although these studies found beneficial effects of exercise on sleep, they are inherently limited. Most of the experimental studies featured samples of normal sleepers or adults with mild sleep difficulties assessed by questionnaires. This greatly limits the generalizability of the effect of exercise on populations with clinically relevant sleep disorders such as insomnia. Additionally, most studies lack exploration into considerations for exercise prescription such as the timing or mode of exercise. Many studies focus on aerobic activities such as walking and cycling with minimal investigation into resistance training. A recent systematic review of resistance training exercise studies concluded that chronic resistance training has a moderate to large beneficial effect on sleep quality; however, no chronic resistance training studies obtained objective measures of sleep and were heterogenous in design, sleep measurement, and resistance intensity.[95] Of the small number of studies exploring the acute effect of resistance exercise on sleep, few have used

objective measures; based on the available research, there are mixed findings as to whether objective sleep indices (e.g., arousal index, slow-wave sleep) are improved with acute resistance exercise.[95] The lack of consensus regarding the impact of acute resistance training on sleep warrants further investigation with experimental designs. Exercise timing has also been sparsely examined; most of the available research has focused on the impact of late-night exercise on sleep. In contrast to the common admonition to avoid exercise close to bedtime, a recent meta-analysis of crossover studies found that evening exercise led to beneficial effects on sleep as measured by polysomnography.[93] Similar to the limitations already mentioned, these studies exploring the impact of late-night exercise were conducted in healthy participants without sleep complaints. It is currently not well understood how timing or mode of exercise impacts adults with sleep disorders or significant sleep complaints such as insomnia. Yet, one recent experimental study in a small sample of participants with insomnia found highly variable responses to late-night exercise, including severe sleep impairment for a small number of the participants. These findings provide mixed support to the belief that late-night exercise may negatively impact sleep in people with insomnia.[96]

2.3.2 Biological Mechanisms Underlying the Effect of Exercise on Sleep

A variety of mechanisms of physiological and psychological origins have been proposed to explain how exercise beneficially impacts sleep. However, many of the potential mechanisms are not fully understood. Additionally, there is a lack of formal testing of these mechanisms in those with insomnia. However, when considering insomnia as a disorder characterized by cognitive or

somatic hyperarousal, acute and chronic exercise could provide reduction in hyperarousal that may directly benefit adults with insomnia or directly impact sleep-wake systems.

One hallmark hypothesis explaining the benefit of acute exercise on sleep is its effects on body temperature regulation. Body temperature reduction and the dissipation of heat from the core have been associated with the initiation of sleep and deeper sleep.[28,37] Body temperature reduction is controlled by the cascade of sympathetic withdrawal by internal warm-sensing neurons in the preoptic/anterior hypothalamus and information from peripheral temperature-detecting neurons which then subsequently triggers peripheral vasodilation and body cooling.[38,97] Blunted temperature reduction has been observed in adults with insomnia prior to bedtime.[38,98] When performed in the afternoon or evening, acute exercise may increase the body's core temperature and trigger temperature reduction and subsequent body cooling prior to bedtime.[37] In turn, this may improve sleep in people with insomnia. In addition to the impact of an acute bout of exercise, chronic exercise training may improve distal blood flow, temperature dissipation, and body cooling at bedtime from hypothalamic withdrawal of sympathetic activity and thus optimize the capacity for the body to reduce its temperature at bedtime.[99]

In addition to temperature, exercise may influence the timing and consistency of the circadian system. The circadian system is critically important in regulating sleep and wake patterns. An advanced or delayed sleep/wake rhythm may compete with behaviorally necessary sleep patterns (e.g., bedtimes and waketimes for work), thereby contributing to a difficulty falling or staying asleep.[100] A proposed marker of this misalignment is a mismatch between the circadian timing of the pre-sleep reduction of body temperature, resulting in a higher core body temperature while attempting to sleep, which could be contributing to sleep onset or maintenance insomnia symptoms.[65] Exercise has been implicated as a potential synchronizer of the circadian system,

although how exercise may cause alterations in circadian timing is not well understood.[101] A review by Yamanaka and colleagues suggests that daily bouts of physical exercise could facilitate phase advances (moving of rhythm earlier) and increased amplitude of the circadian rhythm, though there are mixed results regarding the directionality of circadian timing shifts.[102,103] A recent study in inactive young adults found that 5 consecutive days of moderate-intensity aerobic exercise at different times of day altered circadian timing, as measured by dim light melatonin onset, and the direction of the shifts were dependent on individual chronotype.[104] Evidence of exercise as a circadian zeitgeber has been found in studies where wheel running increased expression of circadian clock gene Per2 in mice and upregulated expression of the circadian genes *Cry1*, *Per2,* and *Bmal1* in the quadriceps muscle *after multiple repetitions of leg extension exercise in human subjects.[105,106] This indicates a potential peripheral regulation of the circadian system through muscle activation. In general, these studies highlight that acute and chronic exercise may impact circadian signaling mechanisms and, as a result, potentially improve sleep.*

In addition to its thermogenic and circadian effects, both acute and chronic exercise may improve mood through reductions in anxiety and depression. This may be especially relevant among adults with insomnia, as anxiety and depression are often tightly linked with insomnia complaints.[29,73] Reductions in state anxiety have been observed after acute exercise, while chronic exercise training has been shown to reduce trait-like anxiety symptoms.[34,107] Exercise has anti-depressant effects; as a result, exercise could benefit sleep through a reduction in depressive symptoms.[108] Exercise training, when used as an adjunct to pharmacotherapy, has been shown to reduce insomnia symptoms in people with depression.[109] Additionally, in a sample with diagnosed insomnia, exercise training reduced tension and anxiety, depression, and total mood disturbance.[20] In general, reductions in anxiety and depression through exercise is an intriguing pathway for

reductions in pre-sleep cognitive arousal (i.e., sleep-related worry, anxiety about the sleep environment). More research is needed into the impact of acute and chronic exercise on mood prior to sleep in adults with insomnia.[28,29]

Exercise training is also closely linked to adaptations to the autonomic nervous system. Exercise training has been shown to improve autonomic nervous system function, resulting in lower heart rate and higher heart rate variability.[110] Subsequently, chronic exercise may improve sleep in adults with insomnia by increasing parasympathetic nervous system activity, as measured by increased heart rate variability.[28,29,63] This positive impact on sleep is only a mechanism of chronic exercise training, as acute exercise may result in a transient increase in nocturnal heart rate and no alteration in heart rate variability.[28]

Acute exercise has also been used to investigate the impact exercise may have on homeostatic sleep drive through increased production of sleep-promoting signaling molecules.[111] Specifically, adenosine is a neurotransmitter that has been implicated in the homeostatic regulation of sleep.[112] A study in rats found that high-intensity exercise increased adenosine concentrations in the whole brain compared to a no-exercise control.[35] A potential increase in serum or cellular adenosine after exercise has not yet been studied in human subjects in relation to sleep, although this pathway could explain exercise effects on sleep drive and explain why exercise has been found to increase slow-wave sleep (a marker of sleep drive) in healthy samples. In addition to adenosine, pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α) have been related to sleep initiation and are at their peak concentrations during the nighttime.[36] Specifically, exogenous administration of IL-1 and TNF-α increase NREM sleep and slow-wave sleep.[36,101] Interestingly, acute moderate-intensity exercise results in modest transient increases in IL-1, IL-6, and TNF-α; this magnitude of increase has been suggested to be

the most beneficial to sleep, since high concentrations of these cytokines have been associated with sleep disruption.[36,101,113]

Increased brain-derived neurotropic factor (BDNF) after exercise is another proposed signaling protein mechanism. BDNF regulates neuronal development and function within the brain.[114] Animal studies have found that injecting BDNF in the cortex results in greater slow-wave sleep during the subsequent nocturnal period.[111] It is still unclear how exercise may increase BDNF, but acute exercise has been shown to increase BDNF levels in the blood in a dose-response manner.[115] Yet, there is currently a limited understanding of how much bloodborne BDNF may cross the blood-brain barrier. In animal models, prolonged exercise has been shown to increase BDNF gene expression in the brain directly through production of the ketone body D-β-hydroxybutyrate.[116]. Lastly, neuronal activity itself increases BDNF production and exercise-related motor neuron activity may directly increase BDNF expression.[117] Although only preliminary data exist, exercise may increase BDNF and subsequently result in better sleep.

Peroxisome proliferator-activated receptor gamma coactivator (PGC-1α) is a protein that increases gene expression of energy metabolites and has been shown to increase with concurrent increases in exercise intensity.[118] PGC-1α is a potentially important peripheral signaling molecule because it promotes the expression of Bmal1, a core circadian clock gene.[119] In mice models, restoring skeletal Bmal1 production after a temporary neural block improved slow-wave activity during sleep. It is still unclear whether expression of Bmal1 through skeletal muscle activation is a direct pathway for sleep improvement, but future studies are warranted to explore this mechanism.[111]

Looking at these biological mechanisms globally suggests that exercise—either acute or chronic—may reduce somatic (e.g., body temperature regulation, autonomic function, relaxation)

and cognitive (e.g., anxiety) markers of arousal and have direct impacts on sleep-promoting neurotransmitters, cytokines, and the circadian system.[111] Further investigation into how exercise impacts each of these pathways is warranted as they are largely unexplored, especially in samples of adults with insomnia.

2.3.3 Epidemiologic Associations Between Physical Activity and Insomnia

Several cross-sectional and prospective observational studies have explored the relationship between physical activity and insomnia. In general, these studies have found that physical activity participation is related to fewer insomnia symptoms and a lower risk of developing insomnia.[120,121] In a large-scale epidemiological cohort of adults in Japan (N = 10,211), participation in 5 or more days/week of walking was cross-sectionally related to lower odds of difficulty initiating sleep (DIS) (OR = 0.75) and difficulty maintaining sleep (DMS) (OR = 0.72).[122] Additionally, frequent participation in recreational/sporting activities of (i.e., ≥ 5 days/week) was related to lower odds of DIS (OR = 0.86) and use of hypnotics (OR = 0.82).[122]

In a longitudinal study of women, leisure-time physical activity was used to predict insomnia incidence (defined as having a symptom of insomnia combined with daytime sleepiness or fatigue).[123] They found that women who maintained medium or high levels of physical activity and those who increased their level of activity over the 10-year follow up period were protected from insomnia.[123] Another study explored the cross-sectional and longitudinal relationships between different physical activity domains and insomnia. They found that recent and historical lifestyle-based activity (e.g., activities of daily living, caregiving) were not related to insomnia risk; in contrast, greater levels of sports and exercise were linked to lower insomnia risk.[124] These

results seem to suggest that, although all types of activity may be beneficial to sleep, purposeful physical activity may be the most beneficial to reductions in insomnia risk.

More recent observational research (N = 12,718) by Chen and colleagues found that, when excluding for multiple sleep-related disorders other than insomnia as well as health-related and socioeconomic covariates, being physically inactive was related to increased risk of clinically diagnosed insomnia (OR = 1.22).[125] In general, other cohorts have found similar results and corroborate the beneficial relationship between physical activity participation and reduced insomnia risk and severity.[126-128]

2.3.4 Exercise Training and Insomnia

Although experimental research on the effects of chronic exercise (i.e., >1 week) on sleep is accumulating, there is currently limited evidence exploring how exercise impacts sleep and daytime impairment in adults who meet diagnostic criteria for insomnia. Most of the existing research has used proxies in place of clinical insomnia diagnosis (e.g., PSQI > 5, Insomnia Severity Index (ISI) ≥ 10) or have relied upon of the self-report of various combinations of insomnia symptoms. **Table 1** summarizes the RCTs and single-group experimental studies that have explored the effects of chronic aerobic or resistance exercise on insomnia severity, sleep quality, and various aspects of daytime impairment related to insomnia. Of note, some studies have explored the effects of tai chi and yoga on insomnia symptoms with evidence of beneficial effects; however, effects may be mechanistically different than that of traditional (i.e., aerobic and/or resistance) exercise. As a result, these studies will not be further discussed in the context of this literature review.[129-132]

To date, three RCTs have recruited samples of adults who meet diagnostic criteria for insomnia, as determined by DSM-4 or -5 criteria or the Research Diagnostic Criteria for Insomnia.[133] Two of the three RCTs found improvements in subjective sleep quality after moderate-intensity exercise training compared to no-exercise control groups. Reid and colleagues found a significant improvement in PSQI-assessed sleep quality, while Hartescu and colleagues found improvements in ISI.[16,17] This is in contrast to the RCT from Guilleminault and colleagues, which found no changes in a single-item question of self-reported sleep quality.[18] These differences in results could be due to the measure used, as the PSQI and ISI are more comprehensive measures of sleep quality than single-item questions. In addition, the study by Guilleminault and colleagues did not strictly sample inactive participants, while the other studies did. Including participants with potentially higher habitual physical activity levels could have reduced the potential impact of exercise on sleep.

Additional support of the positive impact of exercise on sleep in insomnia comes from other experimental studies, described in **Table 1**, that found both moderate-intensity resistance and aerobic exercise training significantly reduced PSQI scores.[19,20] These studies also obtained objective measurement of sleep via polysomnography[20] or actigraphy[19] and found significant reductions in sleep onset latency and wake after sleep onset. However, cautious interpretations may be necessary, as the study by Passos and colleagues[20,21] lacked a control condition and the study by D'Aurea and colleagues[19] compared moderate-intensity resistance training to a non-randomized control group. In general, these studies prompt the necessity for future investigations that explore different exercise prescriptions in terms of type, duration, intensity, volume, and timing of exercise. To date, Passos and colleagues have published the only chronic exercise study to explore the potential impact of exercise timing (i.e., morning vs. afternoon) on sleep in adults

with insomnia; they found that most effects on sleep were not dependent on exercise timing.[20] No RCTs have directly compared chronic resistance training and aerobic activity in adults with insomnia.

Table 1 also provides a summary of the available research exploring the impact of exercise training on indices of daytime impairment in samples with insomnia. Across multiple studies, a significant reduction in depressive symptoms has been observed following exercise training.[17,20,21] Anxiety symptoms also seem to be reduced after exercise training.[16,20] In some studies, reductions in depression and anxiety were significantly correlated with improvements in subjective sleep quality, potentially indicating that these may be pathways through which exercise reduces insomnia symptoms.[17,20]

Although improvements in mood seem to occur after exercise training, improvements in physical impairment do not seem to be as uniform across studies in samples with insomnia. Hartescu and colleagues, utilizing the Fatigue Severity Scale, found no significant changes in fatigue after six months of moderate-intensity aerobic exercise.[16] Similarly, D'Aurea and colleagues failed to observe changes in health-related quality of life (as assessed by the Short-form 36-item Health Survey [SF-36]) after resistance training compared to the stretching and no-exercise control conditions.[19] This is contrary to other studies that found improvements in the SF-36 vitality[17] and general health perception[20] subscales after exercise training. Potentially, exercise mode has a differential effect on impairment in insomnia; resistance exercise did not elicit improvements in physical impairment while aerobic exercise did. Ultimately, the small amount of literature using very diverse measures of daytime function and quality of life make it difficult to draw strong conclusions.

Together, this small batch of studies indicates that chronic exercise has a beneficial effect on daytime mood and subjective reports of sleep quality, and mixed findings on objective improvements in sleep, in samples of adults with insomnia. Although the number of RCTs using exercise training are a fraction of those conducted using CBT-I, improvements in actigraphy-assessed sleep are somewhat comparable to that of CBT-I, while improvements in PSQI and diary-assessed sleep are somewhat lower in magnitude.[134,135] However, further exploration into the potential mechanisms by which these improvements occur will assist in targeting interventions that improve those mechanisms. Hartescu and colleagues propose that chronic exercise has a dampening effect on hyperarousal. In addition to a significant reduction in insomnia severity, they found that objective measures of reaction time worsened following exercise training compared to the control group.[136] In terms of cognitive hyperarousal, people with insomnia may perform better on simple reaction type tests due to greater neurocognitive vigilance compared to their normal-sleeping counterparts.[137] Thus, after an intervention that successfully reduced insomnia severity, reductions in reaction time performance may represent reduced hyperarousal in insomnia.[136] Acute exercise could potentially elicit similar effects, albeit smaller in magnitude, on other indices of hyperarousal in insomnia, yet no acute exercise studies have explored this in samples of adults with insomnia.

Table 1. Chronic exercise studies in samples who meet diagnostic criteria for insomnia

Author/Year	Design	Sample	Intervention	Results: Sleep	Results: Impairment
D'Aurea 2019 [19]	EXP	28 inactive adults (mean: 45 y, BMI=30)	MRE (n=10), STR (n=10), CON (n =8); 50 minutes, 3/w, 4 months	MRE and STR reduced PSQI scores compared to CON; Improved ACT SOL, WASO, SE; no change in PSG sleep in MRE or STR	Reduced tension-anxiety in STR compared to CON post-intervention
Guilleminault 1995 [18]	RCT	30 adults (mean: 45 y)	SH (n =10), SH+BL (n=10), SH+MAE (n=10); 45 minutes, 7/w, 1 month	No change in ACT sleep after SH+MAE compared to SH; No change in self-reported sleep complaints	No change in self-reported fatigue or alertness in SH+MAE group at post-intervention
Hartescu 2015 [16]	RCT	41 inactive adults (30 females, mean: 60 y, BMI=26, ISI=16)	MAE (n=17), CON (n=18); 30 minutes, 5/w, 6 months	MAE training reduced ISI	MAE training reduced depression and trait anxiety compared to CON post-intervention; No change in fatigue severity between groups
Hartescu 2019 [136]	RCT	41 inactive adults (30 females, mean: 60 y, BMI=26, ISI=16)	MAE (n=17), CON (n=18); 30 minutes, 5/w, 6 months	MAE training reduced ISI	MAE training increased (i.e., worsened) reaction time compared to CON post-intervention
Passos 2014 [21]	EXP	21 inactive adults (16 females, mean: 45 y, BMI=25, PSQI=11)	MAE (n=21); 50 minutes, 3/w, 4 months	Reduced PSQI; Improved PSG SOL, WASO, TST, SE, REM sleep	Reduced depression; Reductions in plasma cortisol and improved markers of immune function
Passos 2011 [20]	EXP	19 inactive adults (15 females, mean: 45 y, BMI=25, PSQI=11)	MMAE (n=10), AMAE (n=9); 50 minutes, 3/w, 4 months	MMAE and AMAE had improvements in PSG SOL, WASO, Stage 2 latency, REM latency; MMAE had larger improvements in self-reported sleep quality compared to AMAE	Similar significant reductions in tension-anxiety, depression, anger-hostility, and total mood disturbance in MMAE and AMAE over time
Reid 2010 [17]	RCT	17 inactive older adults (16 females, mean: 61 y, BMI= 27)	SH+HAE (n=10), SH+REC (n=7); 40 minutes, 4/w, 4 months	SH+HAE reduced PSQI score compared to no change in SH+REC	Reduced sleepiness and depression and increased vitality in SH+HAE group compared to SH+REC

Abbreviations: ACT: actigraphy; AMAE: afternoon moderate-intensity aerobic exercise; BMI: body mass index (kg/m^2); BL: bright light; CON: no-exercise control group; EXP: experimental study; HAE: high-intensity aerobic exercise; ISI: Insomnia Severity Index; MAE: moderate-intensity aerobic exercise; MMAE: morning moderate-intensity aerobic exercise; MRE: moderate-intensity resistance exercise; PSG: polysomnography; PSQI: Pittsburgh Sleep Quality Index; RCT: randomized controlled trial; REC: recreational activities; REM: rapid eye movement; SE: sleep efficiency; SH: sleep hygiene; STR: stretching control; SOL: sleep onset latency; TST: total sleep time; WASO: wake after sleep onset.

2.3.5 Acute Exercise and Insomnia

Table 2 summarizes the small number of experimental and observational studies that have explored the effects of an acute bout of aerobic or resistance exercise on subsequent sleep in samples with insomnia. Each of these studies examined inactive middle- to older-aged adults who met clinical diagnostic criteria for insomnia or reported a combination of insomnia symptoms. Many studies included objective assessment of sleep in the form of polysomnography or actigraphy.

Most acute exercise studies (i.e., <1 week) have found improvements in objectively measured sleep via polysomnography or actigraphy. Specifically, Passos and colleagues found that evening moderate-intensity aerobic exercise increased total sleep time and sleep efficiency, while reducing sleep onset latency and total wake time.[22] A similar study by Morita and colleagues also found improvements in sleep continuity and reductions in night-time arousals regardless of insomnia phenotype (i.e., early morning awakening vs. difficulty initiating sleep) after participating in morning step exercise.[23] In contrast, Youngstedt and colleagues tested the impact of late-night (i.e., completed 2 h before bedtime) moderate-intensity aerobic and resistance exercise compared to quiet reading in a sample of young adults with insomnia.[96] They found no significant differences in polysomnography- or diary-assessed sleep after exercise compared to quiet reading. Yet, there were two participants out of sixteen who did experience substantially worse sleep. This may lend support to caution with prescribing late-night exercise to individuals

with insomnia, although more research is necessary to explore why some individuals may respond poorly to late-night exercise.

In addition to studies that utilized polysomnography, Chen and colleagues assessed sleep with actigraphy for two nights before and after a single bout of moderate-intensity aerobic exercise; increased sleep efficiency and reduced sleep onset latency were observed following exercise relative to control.[24] Although not an experimental study testing the effect of exercise on sleep, these improvements in sleep after a single exercise bout were not corroborated by an observational study by Baron and colleagues. Using multilevel linear modeling of daily data from a 16-week exercise intervention previously published by Reid and colleagues,[17] they found that the duration of an acute exercise bout was not related to any metric of actigraphic sleep or subjective sleep quality the same night, while sleep onset latency the previous night predicted exercise bout duration the following day.[25] Although some acute studies suggest small immediate improvements in sleep following an acute bout of exercise, these null results in conjunction with the findings of Youngstedt and colleagues may suggest that a single exercise bout may not be enough to elicit beneficial effects on sleep in adults with insomnia.

The current literature is limited regarding the potential effects of acute exercise on subjective sleep quality or metrics of daytime consequences of insomnia. Morita and colleagues assessed subjective sleep quality and found no improvements after exercise, while the secondary analyses from Baron and colleagues found that exercise duration was not related to sleep quality in the subsequent evening.[23] These results from the acute exercise literature contrast the chronic exercise studies previously discussed that found beneficial impacts on sleep quality.[16,17,19,22] Unfortunately, the other acute studies that found beneficial improvements in objectively measured sleep did not measure self-reported sleep quality.[22,24]

In regard to the daytime consequences of insomnia, Passos and colleagues assessed changes in state anxiety following a variety of acute exercise conditions and found that only the moderate-intensity aerobic exercise group significantly reduced anxiety; neither resistance nor high-intensity aerobic exercise led to significant improvements in anxiety.[22] Morita and colleagues found no changes in self-reported fatigue or sleepiness after morning or evening moderate-intensity step exercise.[23] Other acute exercise studies did not assess indices of daytime impairment in insomnia. The lack of investigation into whether acute exercise impacts daytime impairment remains a space for further inquiry.

Overall, these studies indicate the need for further investigation into whether acute exercise improves the different characteristics of sleep across objective and subjective indices and measures of daytime impairment. Additionally, there should be inquiry into how different exercise prescriptions (e.g., timing, mode, duration) impact sleep. These studies also highlight methodological considerations that have not yet been explored, such as whether multiple bouts of exercise across a short time frame impact sleep and daytime impairment in insomnia. This is especially important as people with insomnia present with high night-to-night variability in sleep;[26,138] as a result, implementing multiple bouts of exercise over a short time frame may better elucidate the effect of acute exercise in those with this disorder.

Table 2. Acute exercise studies in samples with insomnia disorder or symptoms

Author/Year	Design	Sample	Conditions	Analysis/Results
Baron 2013 [25]	Observational secondary analysis of RCT	11 inactive women who met diagnostic criteria for insomnia disorder (mean: 61 y, BMI=27)	MAE (n=11); 30 minutes of walking exercise at 55% HRM	Hierarchical linear modeling found that daily acute exercise duration was not related to same-night SQ or actigraphic SOL, TST, WASO, SE, yet actigraphic SOL was related to next-day duration of exercise.
Chen 2019 [24]	RCT	40 inactive adult women who scored > 5 on the Athens Insomnia Scale[139] (mean: 61 y, BMI=24)	MAE (n =20), CON (n=20); 50 minutes of walking at 45-55% HRM	2x2 repeated measures ANOVA revealed that walking exercise reduced actigraphic SOL and WASO and increased SE compared to CON. SQ was not assessed.
Morita 2017 [23]	Within-subject repeated measures design	40 inactive adults experiencing insomnia symptoms (25 females, mean: 59 y, BMI=21)	NCON (n=13), DIS (n=12), EMA (n=15); Completed morning and evening moderate-intensity (59% HRR) step exercise	Two-way repeated measures ANOVA found that morning exercise resulted in a reduction in PSG-assessed stage shifts as well as reduction in arousal index in the second half of the night compared to baseline. No improvements in SQ.
Passos 2010 [22]	Randomized parallel design	48 inactive adults who met diagnostic criteria for insomnia (38 females, mean: 45 y, BMI=25)	MAE (n=12), MRE (n=12), CON (n=12), HAE (n=12); 50 min of either MAE (1st VT) or three 10-min bouts of HAE (2nd VT) with alternating 10 min of rest, or 50 min of MRE	Repeated measures ANOVA revealed that MAE decreased PSG SOL and WASO and increased TST and SE. Diary-assessed SOL decreased and TST increased. MAE reduced pre-sleep anxiety levels. SQ was not assessed.
Youngstedt 2021 [96]	Within-subject repeated measures design	12 inactive adults who met diagnostic criteria for insomnia (mean: 27 y, ISI: 18.1)	Late-night walking/running exercise at 60-70% HRM + resistance exercise, or a sedentary reading condition (ending 2 h before bedtime).	Paired t-tests found no differences in PSG- or diary-assessed sleep between reading and late-night exercise. Pre-sleep anxiety after exercise was related to PSG sleep.

Abbreviations: ANOVA: analysis of variance; BMI: body mass index (kg/m^2); CON: control group; DIS: difficulty initiating sleep; EMA: early morning awakening; HAE: high-intensity aerobic exercise; HRM: heart rate maximum; HRR: heart rate reserve; ISI: insomnia severity index; MAE: moderate-intensity aerobic exercise; MRE: moderate-intensity resistance exercise; NCON: normal sleeping controls; PGI-C: patient global impression of change scale; PSG: polysomnography; PSQI: Pittsburgh Sleep Quality Index; RCT: randomized controlled trial; SE: sleep efficiency; SOL: sleep onset latency; SQ: sleep quality; TST: total sleep time; VT: ventilatory threshold; WASO: wake after sleep onset

2.3.6 Summary and Future Directions

Insomnia is a prevalent sleep disorder that impacts the health and wellbeing of those it afflicts. When exploring the current literature, exercise shows promise as an accessible, inexpensive, and effective strategy to improve sleep. Yet, the scant amount of well-designed, comprehensive, and insomnia-focused research with acute exercise sparks questions that will be addressed in the current proposal. Specifically, only 3 out of 5 studies sampled individuals with insomnia who met clinical diagnostic guidelines. This distinction in sampling criteria is important as results must be generalizable to patients with diagnosed insomnia and inherently more useful to clinicians who treat these patients. In addition to inadequate sampling, the acute exercise literature lacks broad assessment of sleep with objective and subjective measures. Specifically, there is a lack of concurrent global measures of sleep quality or insomnia severity as is recommended in the study of insomnia.[140] The current proposal will utilize metrics of insomnia severity and sleep quality to better understand how acute exercise impacts key measures of insomnia. Lastly, previous acute studies have only explored how one bout of exercise impacts subsequent sleep. Some literature indicated that one bout of exercise may not be enough to elicit beneficial effects on sleep in insomnia; thus, studying multiple exercise bouts over a short time frame could better answer how acute initiation of exercise affects sleep and insomnia severity. Assessing the impact of exercise bouts across multiple days of sleep assessment is also more methodologically suited to address the significant night-to-night variability in sleep among adults with insomnia and may globally capture effects that may not be seen with one day of sampling or experimentation.[26,138,141]

3.0 METHODS

This project was proposed and implemented in 2020 during the COVID-19 pandemic. Due to the various public health constraints (e.g., research facility closures) that were implemented to combat the pandemic, this project's protocol was altered prior to data collection to effectively implement the research while maintaining scientific rigor. Originally this was intended to be an experimental study that would require in-person visits to our research facility for the initial screening, anthropometric assessments, sleep interviews, and subsequent experimental sessions. Specifically, this lab-based strategy would have allowed for the greatest level of staff oversight to precisely monitor experimental session completion. Due to the ongoing pandemic, the protocol was modified to have multiple face-to-face study visits occur over video conference calls and all experimental visits to occur remotely (i.e., walking exercise and quiet rest sessions). Although this protocol change reduced the precision and experimental control over the participants' experience, it did allow for the implementation of a study with strong ecological validity which is lacking in the experimental literature in the sleep and exercise field to date.

3.1 STUDY DESIGN

Using a parallel group design, participants who met diagnostic criteria for insomnia were randomized to either an aerobic exercise condition or a quiet rest condition (**Figure 3**). Prior to randomization in a 1:1 allocation ratio stratified by gender, eligible participants completed the

following assessments in sequential order: (1) a consent visit conducted via videoconference; (2) an online-based questionnaire battery; (3) a sleep interview conducted via videoconference; (4) an at-home assessment of anthropometrics and resting heart rate; and (5) a 8-day baseline assessment using ambulatory wrist-worn sleep and hip-worn physical activity monitoring with daily diary assessment of sleep and daytime behaviors (**Figure 3**). Following the baseline assessment, participants were randomized to their experimental condition and repeated the device and diary monitoring for an additional 8 days. In both experimental conditions, participants completed 3 experimental sessions on non-consecutive days consisting of either walking exercise or quiet rest.

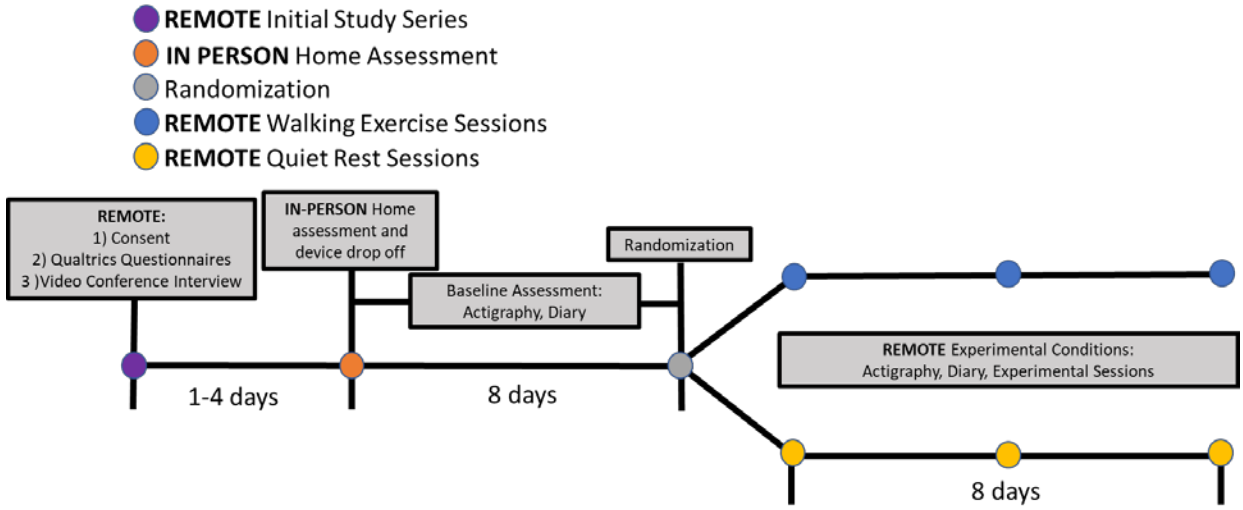


Figure 3. Study flow diagram

3.2 SAMPLE POPULATION

The target population for this study was generally healthy and physically inactive (< 60 min/week of aerobic exercise over the past three months) adults between 18-55 years with an Insomnia Severity Index (ISI) of ≥ 10 who met Diagnostic and Statistical Manual of Mental Disorders

(DSM-5) criteria for insomnia disorder.[39,142] An ISI inclusion of ≥ 10 was chosen as it indicates a sensitive and specific threshold for insomnia.[142] Individuals were excluded based on: (1) self-reported presence of uncontrolled acute or chronic medical conditions (i.e., central nervous system disorders [e.g., head injury, seizure disorder, multiple sclerosis, tumor], cardiovascular or hemodynamically significant cardiac disease, renal failure, diabetes); (2) untreated major psychiatric disorder (e.g., bipolar disorder, panic disorder, obsessive compulsive disorder); (3) current co-morbid sleep disorder (e.g., narcolepsy, restless legs syndrome) or being categorized as high risk for sleep-disordered breathing (≥ 5 on the STOP-Bang questionnaire);[143] (4) musculoskeletal injuries or considerations that may contraindicate exercise participation (e.g., endorsing ≥ 1 item on the Physical Activity Readiness Questionnaire [PAR-Q+]);[144] (4) obesity (body mass index [BMI; kg/m^2] ≥ 30); (5) current indication of moderate/severe depressive or anxiety symptoms (i.e., Patient Health Questionnaire-9 [PHQ-9] score ≥ 10 or Generalized Anxiety Disorder Questionnaire [GAD-7] score ≥ 10, respectively); (6) current treatment for insomnia; (7) suspected circadian rhythm disorder (delayed sleep phase: habitual bedtime ≥ 2:00 am or waketime ≥ 10:00 am; advanced sleep phase: habitual bedtime ≤ 9:00 pm or waketime ≤ 5:00 am)[100] or nocturnal shiftwork (i.e., working between 12:00 am and 6:00 am); or (8) current pregnancy or intention to become pregnant in the next 3 months. Individuals with well-controlled health conditions that did not affect sleep or well-being (e.g., asthma, high blood pressure, diabetes, or ulcers) or subclinical or controlled depression or anxiety were not excluded.

3.3 PARTICIPANT SCREENING AND INFORMED CONSENT

Participants were recruited through the University of Pittsburgh Research Participant Registry (i.e., Pitt+Me), direct emails sent to prior participant contact lists available from the dissertation advisor, direct emails sent to University of Pittsburgh faculty, staff, and students, and flyers distributed around the University of Pittsburgh campus **(Appendix A)**. Interested participants contacted study staff and were sent an online Qualtrics survey or completed a phone screen based on the prospective participant's preference. The screening took approximately 10-15 minutes to complete and contained questions relevant to the exclusion criteria such as age, level of physical activity participation, and brief questionnaires such as the ISI, PAR-Q+, and the STOP-Bang.[142-144] The screening questionnaire is found in **Appendix B**. Participants who met initial eligibility requirements were sent an informed consent document through Qualtrics and then contacted by phone or videoconference call to receive a detailed explanation of the study protocol prior to providing their electronic signature.

After electronic informed consent was obtained, participants were scheduled for a video conference sleep interview and sent a questionnaire battery through Qualtrics that included: (1) a demographic characteristics questionnaire; (2) a medical history survey; (3) the State-Trait Anxiety Inventory (Form Y2), a measure of trait anxiety;[145] (4) the Hyperarousal Scale, a measure of general hyperarousal;[146] (5) the Composite Scale of Morningness, a measure of chronotype;[147] (6) the Ford Insomnia Response to Stress Test, a measure of stress-related sleep reactivity;[148] and (7) PHQ-9 and GAD-7, measures of depressive and anxiety symptoms, respectively (**Appendix C**).[149,150] Scores of ≥ 10 on the PHQ-9 and the GAD-7 indicate moderate/severe levels of depression or anxiety, respectively.[149,150] If prospective participants presented with moderate to

severe depression and anxiety symptoms they were given reference material, guidance to treatment services, and helplines available for immediate contact. Participants completed these questionnaires at least a day prior to their sleep interview.

The presence of comorbid sleep disorders was assessed using the STOP-Bang and the video conference sleep interview. As 21-39% of adults with insomnia have OSA,[151,152] the STOP-Bang questionnaire was chosen as a screening tool because it has low participant burden compared to other objective assessments and is recommended as a simple strategy with the ability to predict and rule out OSA with reasonable accuracy.[153,154] An exclusion score of ≥ 5 on the STOP-Bang or 2 STOP questions + either male sex or large neck circumference (i.e., > 17 in [men], > 16 in [women]) was chosen as it indicates high risk for sleep apnea. Over a HIPAA-compliant videoconference call, the Structured Clinical Interview for DSM-5 Sleep Disorders (Sleep SCID) Module was administered by a study staff member trained in the administration of this interview.[155] This brief interview, which asked 20-51 questions regarding nine major sleep-related disorders, is a gold standard methodology used to identify clinically relevant insomnia while also ruling out additional comorbid sleep-related conditions.

3.3.1 Home Assessment

Following confirmation of eligibility during the sleep interview, study staff conducted a 30-minute at-home assessment the day prior to starting the baseline sleep and physical activity monitoring period. Participant height and weight were measured with a portable stadiometer and scale (Seca*;* Chino, CA). Measurements were taken until two measures were verified to be within 0.5 centimeters (cm) and 0.2 kilograms (kg) for height and weight, respectively. Following height and

weight assessment, waist and neck circumference were measured with a Gulick tape measure at the level of the iliac crest and the cricothyroid cartilage, respectively. Self-reported neck circumferences reported during the screening questionnaire for OSA risk assessment were verified with the measured circumference obtained at this visit. Measurements were taken until two measures were verified to be within 1 cm. Following the waist and neck circumferences, participants had their resting heart rate assessed by wearing a Polar H10 heart rate monitor (Polar Electro; Kempele, Finland) and sitting for 5 minutes in a seated position with their back supported and feet on the floor. After 5 minutes, the heart rate was recorded every 30 seconds until three consecutive heart rates within 5 bpm occurred. The three measures were then averaged to obtain a resting heart rate. Staff then described the sleep diary and ambulatory devices so the participants could begin their 8-day baseline assessment.

3.3.2 Baseline Assessment

Starting immediately after the at-home assessment, participants began the baseline assessment of their sleep and daytime behaviors. This baseline period provided an assessment of the participants' typical sleep patterns and served as a pre-randomization check that participants could be adherent to the protocol. Details regarding the assessments are provided in **Section 3.5** below.

Participants were considered adherent if they: 1) completed ≥ 5 days of the daily sleep diary; 2) wore the sleep monitoring watch for ≥ 5 days with valid data; and 3) wore the physical activity monitoring device for ≥ 4 days. If a participant did not meet these requirements, they were asked to repeat the baseline assessment; if they refused, they were excluded from further study participation. Participants were also excluded from the study if they obtained ≥ 150 min of

moderate- to vigorous-intensity physical activity based on waist-worn accelerometry (described below).

3.4 EXPERIMENTAL CONDITIONS

3.4.1 Randomization

Eligible subjects were randomly assigned to one of the two experimental conditions in a 1:1 ratio. Assignments were performed according to a computer-generated random schedule in blocks of two within gender strata (male and female). Experimental condition assignments were handled by a research staff member who placed them in sequentially numbered, opaque, sealed envelopes and monitored diligently. Participant ID was written on the envelope prior to opening, immediately time-stamped post-allocation, and filed for quality assurance.

To establish equipoise in expectations between experimental conditions, study staff strictly avoided the use of the word “control” regarding the quiet rest condition during the consent process and throughout the study. Following randomization, participants completed a 3-item questionnaire indicating their expectations for improvement in their insomnia, sleep quality, or daytime impairment (**Appendix D**).

3.4.2 Experimental Sessions and Procedures

The 8-day experimental condition began within 7 days after randomization for all participants except for 1 participant who began 14 days after randomization due to a minor injury not related

to the protocol. Participants received a new diary and the same wrist- and hip-worn devices that were worn during the baseline assessment. The experimental sessions occurred on Monday, Wednesday, and Friday. The day of the week for the first experimental session was counterbalanced across participants (i.e., one-third of participants began on Monday, one-third began on Wednesday, and one-third began on Friday). Experimental sessions occurred in the morning (i.e., between 7:00 am and 12:00 pm) and at least 48 hours apart from one another. Pre-sleep arousal was assessed in the diary on the three days in which the experimental sessions occurred using the Pre-Sleep Arousal Scale (PSAS), described further in **Section 3.5.4.** At the conclusion of the experimental condition, participants completed insomnia severity and self-reported sleep quality measures described in **Section 3.5.2**.

3.4.3 Walking Exercise Group

For participants randomized to the exercise group, staff calculated the participant's age-predicted maximum heart rate (APMHR) with an equation developed by Tanaka and colleagues (208 – [0.7 *age]).[156] Maximal heart rate was calculated with estimating equations as participant burden would be too high to utilize a graded exercise test to objectively determine the maximal heart rate for exercise prescription. Participants were then prescribed to walk at a target heart rate that corresponded to moderate intensity: 50% of their heart rate reserve (HRR) with an acceptable range of fluctuation in HR of ±5% (i.e., 50% HRR = [(APMHR-RHR) x 0.50] + RHR). This method of prescribing exercise intensity, which incorporates both the individual's maximal heart rate and their resting heart rate, is recommended by the American College of Sports Medicine (ACSM) because relying on maximum heart rate alone has been shown to poorly identify the

appropriate intensity.[157] Participants selected an outdoor route for their walk that was deemed safe and could be performed at a moderate intensity. Participants were instructed to walk at the prescribed intensity for 30 minutes during each session; in addition, they were instructed to complete a 5-minute warm-up and 5-minute cool-down immediately before and after the exercise bout that consisted of gradually increasing or decreasing their walking speed, respectively. Thus, each exercise session lasted approximately 40 minutes. Participants wore a Polar H10 heart rate monitor and M200 smartwatch (Polar Electro; Kempele, Finland) to continuously monitor their exercise intensity during the session. As the unsupervised exercise prescription relied on estimation equations, a Rating of Perceived Exertion (RPE) scale was described to participants as a secondary method for participants to titrate their walking intensity as a safety precaution. Using the Borg scale (i.e., 6-20 rating system), participants were informed that the prescribed heart rate range should coincide with an intensity of 12-14 on the RPE scale (i.e., somewhat hard), but if they were within the heart rate range and at an RPE below 12, they could increase their walking intensity up to an RPE of 14 and disregard the heart rate range. Conversely, if the prescribed heart rate range was eliciting an RPE of ≥ 15 (i.e., hard exercise) participants were instructed to reduce the walking speed until they could maintain an RPE of 12-14.[158] Participants self-reported the details of their exercise sessions (i.e., time and duration of the walk, average RPE, comments) with the Exercise Session Data Sheet (**Appendix E**) to act as a quality check for adherence and in the case of objective HR monitor data loss. Participants were instructed to not alter other lifestyle factors outside of the exercise sessions. To increase adherence, the study PI would text or call participants to remind them of their scheduled walk and was available to troubleshoot any monitor or exercise session issues. The PI also monitored weather forecasts and adjusted start times and

end times of the participants' exercise sessions to achieve full compliance within the preselected time window.

3.4.4 Quiet Rest (Control) Group

Participants randomized to the quiet rest group logged on to a video conference call with the study PI for each 40-minute session and were instructed not to change their typical physical activity or lifestyle behaviors. During each session, participants were streamed "Our Planet", a Netflix nature documentary that consisted of various informational clips focused on animals and their habitats. The first 40 minutes of three different episodes (i.e., "One Planet", "Coastal Seas", and "The High Seas") were played. During each video, participants wore a Polar H10 heart rate monitor and M200 smartwatch (Polar Electro; Kempele, Finland) to record their heart rate for the duration of the session to mimic the experience of the exercise condition. Participants were not permitted to complete homework or work during the allotted time to reduce the chance of unintended stimuli. They were able to use the restroom freely, in which case the monitor and video were paused and resumed upon their return. The study PI used the Quiet Rest Data Sheet (**Appendix F**) to record adherence to the protocol.

3.5 MEASUREMENTS

3.5.1 Daily Assessment of Sleep

Sleep was objectively assessed on a daily basis with the Actiwatch (AW) Spectrum Classic (Philips Respironics; Murrysville, PA), which is a widely used wrist-worn device for monitoring sleep in free-living conditions..[159,160] The AW records accelerometry, ambient light, and user inputs to estimate rest and sleep intervals. Thus, it was the optimal device to measure sleep domains such as efficiency (primary outcome of Specific Aim I), wake after sleep onset, total sleep time, and measures of sleep regularity such as the standard deviation of sleep midpoint. Data were collected in 30-second epochs and edited using Actiware software (version 6.0.9).

Participants were instructed to press an event marker when they get into bed each night with the intention of going to sleep and when they stopped attempting sleep in the morning. Rest intervals were manually established by a trained technician who was blinded to the participants' randomization conditions. The technician followed a standardized approach that incorporates the following inputs, ranked in order of importance: event marker, light intensity, sleep diary, and activity counts.[161] Once rest intervals were established, sleep/wake status for each epoch was determined with the Actiware algorithm to calculate sleep measures using optimal settings suggested by a recent paper by te Lindert and colleagues (sleep onset: 10 min; sleep offset: 0 min; wake threshold: 40).[162]

Sleep was also subjectively assessed on a daily basis with a modified Pittsburgh Sleep Diary (PSD, **Appendix G)**.[163] Participants reported their bedtimes and waketimes and estimated their sleep onset latency and wake after sleep onset. Participants also reported the quality of their

sleep each night of on a rating scale of 0 (very poor) to 4 (very good) as well as the degree that they felt rested or refreshed on a scale of 0 (not at all) to 4 (very well-rested). The use of sleep diaries in tandem with objective measures such as actigraphy is recommended in the study of insomnia as the perceptions of a subject's sleep is distinct and complementary to its objective assessment.[140] The diary reports of bedtime and waketime were also used to edit actigraphy records.

Lastly, participants also utilized visual analog scales in which they placed a check mark across a 100-mm long horizontal line with word anchors of "Not at all" and "Extremely" at each end to express their degree of feeling certain symptoms of arousal (i.e., having an overactive mind, feeling worried, feeling anxious, feeling tense; **Appendix G**). Visual analog scales are suited for daily assessment of symptoms due to their reliability and simplicity.[164]

Actigraphy and sleep diaries were chosen to assess sleep because people with insomnia experience large night-to-night variability in sleep that may not be adequately assessed by a single night of polysomnography.[26,140] Actigraphy and sleep diaries are recommended in studies involving participants with insomnia, while polysomnography is typically implemented as a screening tool for other comorbid sleep-related disorders.[140] Thus, it would be less informative to utilize one night of polysomnography as an outcome measure for this investigation that aims to understand short-term exercise over multiple nights in adults with insomnia.

3.5.2 Weekly Assessment of Sleep and Daytime Impairment

At the conclusion of the 8-day baseline and experimental assessment periods, global assessments of the preceding week's insomnia severity, sleep quality, and daytime impairment were completed.

To assess insomnia severity, participants completed the Insomnia Severity Index (ISI; primary outcome of Specific Aim II; **Appendix H**). The ISI is composed of 7 items that address the severity of sleep-related insomnia symptoms, satisfaction with current sleep patterns, and the impact of insomnia on daytime functioning. The total score ranges from 0-28, with greater scores indicating greater insomnia severity.[142] The ISI is a widely used assessment tool in insomnia research, and has been shown to have concurrent validity with metrics of daytime impairment such as fatigue, anxiety, and quality of life in addition to its reliability and sensitivity to assess changes in perceived sleep difficulties.[142,165] The ISI implemented for this study used a 7-day recall timeframe in contrast to the typical standard time frame of the past 2 weeks.

Although not directly developed to assess impairment resulting from insomnia disorder, the Patient-Reported Outcomes Measurement Information System Sleep-Related Impairment (PROMIS-SRI) short-form questionnaire is a valid assessment of sleep-related impairment (secondary outcome of Specific Aim II, **Appendix I**).[166] The PROMIS-SRI is an 8-item scale in which participants indicate the severity of a variety of potential daytime complaints related to sleep (e.g., "I felt irritable because of poor sleep"), ranging from 1 (not at all) to 5 (very much). One of the eight items ("I felt alert when I woke up") is reverse-scored. Items were summed and then converted into a T-score for analyses with a free online service (HealthMeasures Scoring Service, powered by Assessment Center[SM]), with higher scores indicating worse impairment. Unlike many other typical measures of daytime impairment (e.g., Functional Outcomes of Sleep Questionnaire, PSQI), the PROMIS-SRI has a recall period of 7 days.[166] The Epworth Sleepiness Scale (ESS), a popular measure of daytime sleepiness, was not chosen since increased sleepiness has not been consistently shown in adults with insomnia.[140] However, the PROMIS-SRI has been shown to have convergent validity with the ESS as well as the PSQI.[166] We also assessed short-term changes to

subjective sleep health as measured with the RU-SATED questionnaire,[167] which assesses six different sleep health dimensions: regularity (getting in and out of bed at similar times each day), satisfaction (feeling satisfied with one's sleep), alertness (ability to stay awake during the day without dozing), timing (sleeping between 2:00 a.m. and 4:00 a.m.), efficiency (being awake for less than 30 min each night after trying to fall asleep), and duration (obtaining between 6 and 8 h of sleep per night). Participants indicated how often over the past week they met the criteria for each dimension (0 = rarely/never, 1 = sometimes, 2 = usually/always). Individual items were summed to provide a total score (range 0–12); higher scores indicated better sleep health (**Appendix J**).[168]

Lastly, the PROMIS Sleep Disturbance (PROMIS-SD) short-form questionnaire was used to assess sleep quality (secondary outcome of Specific Aim I) over the past 7 days.[166] The scale is composed of eight items rated on a 5-point scale ranging from 1 (not at all, very poor, or never) to 5 (very much, always, or very good), with four items reverse-scored (**Appendix K**). Items were summed and then converted into a T-score for analyses with a free online service (HealthMeasures Scoring Service, powered by Assessment Center^{SM}), with higher scores indicating greater sleep disturbance. The PROMIS-SD is valid and convergent with the PSQI.[166] The short forms of the PROMIS-SD and PROMIS-SRI have been shown to highly correlate with the originally developed PROMIS-SD and PROMIS-SRI item banks.[169]

3.5.3 Assessment of Daytime Behaviors

In addition to collecting information regarding sleep, participants used the PSD each day to report the time they ate breakfast, lunch, and dinner, and indicate how many alcoholic drinks (one drink

defined as one 12 oz beer, 5 oz wine, or 1.5 oz liquor) and caffeinated drinks (coffee and tea, one drink = 6-8 oz; for caffeinated soda, one drink = 12 oz) they consumed.

To monitor physical activity during the baseline assessment and experimental conditions, the participants wore a physical activity monitor. The ActiGraph GT9X (ActiGraph Corp, Pensacola, FL) is a widely used accelerometer that assesses sedentary activity (SED), light physical activity (LPA), and moderate and vigorous physical activity (MVPA). Participants were instructed to only remove the monitor during non-wake periods, bathing, or swimming. The monitor was placed on the right side of the hip with an elastic waistband, as shown to be optimal for measuring indices of physical activity.[170] Data were considered valid for measurement if worn for $\geq$ 10 hours on $\geq$ 4 days.[171] Time spent in each intensity category of activity was determined by using Freedson cut-point thresholds for adults of $\leq$ 100 counts per minute (cpm; SED), 101-1951 cpm (LPA), and $\geq$ 1952 cpm (MVPA).[171-173]

3.5.4 Assessment of Hyperarousal Symptoms

The 16-item Pre-Sleep Arousal Scale (PSAS; **Appendix L**) was used to assess arousal prior to bedtime on selected nights. This self-reported measure was developed to quantify somatic and cognitive symptoms of arousal that may occur prior to sleep.[32] The scale contains eight items that assess cognitive arousal (e.g., mental alertness) and eight items that assess perceived somatic arousal (e.g., cold hands, jittery) experienced at bedtime. Participants indicated the severity of each item using a range from 1 (not at all) to 5 (extremely). Cognitive and somatic arousal items are summed separately. A total score from 8 to 40 is computed for each subscale, with higher scores indicating higher arousal. A total PSAS score is calculated by summing both subscales

together.[174,175] The combined scale has been shown to be highly internally consistent (Cronbach's alpha = .91)[174,175] with high test–retest reliability.[32] The cognitive subscale of the PSAS has shown to be correlated with other measures of anxiety and depression (e.g., Center for Epidemiologic Studies Depression Scale), while the somatic subscale has been correlated with other measures of somatic arousal (e.g., Cognitive Somatic Anxiety Scale).[32] The cognitive and somatic subscales of the PSAS are also strongly correlated with self-reported sleep onset latency and nighttime awakenings.[32]

As pre-sleep arousal has been shown to be elevated in adults with insomnia compared to healthy sleepers, the PSAS was chosen as a novel measure in the exercise-related literature that could be directly related to the experience of hyperarousal in insomnia and potentially impacted by exercise training.[176] The PSAS was administered on Monday, Wednesday, and Friday during the baseline assessment and following the experimental sessions on Monday, Wednesday, and Friday of the experimental week. Participants were directed to complete the PSAS ≤ 20 minutes prior to going to sleep, with adherence assessed by a self-reported time stamp.

3.5.5 Assessment of Exercise Self-Efficacy

Following the conclusion of the experimental conditions, participants in both experimental groups were administered the Self-Efficacy of Exercise Scale (SEES) to assess whether participating in exercise increased self-efficacy toward future exercise participation. The SEES contains 9 items that assess the confidence a person has that they could complete exercise 3 times per week for 20 minutes under various conditions (e.g., "If you felt tired…", "If you felt stressed…"). Scores range from 0-90, with higher scores indicating greater self-efficacy for exercise participation (**Appendix**

M). This scale has shown to be highly internally consistent and reliable.[177] Measures of self-efficacy have been shown to be strong predictors of future participation in physical activity and exercise adoption.[178,179]

3.6 POWER ANALYSIS

Sample size requirements were estimated using G*Power (Version 3.1.9.4). A required total sample size of 24 was determined by using an effect size of Cohen's $d = 0.53$ (Cohen's $f = 0.265$) derived from a recent study that examined the impact of acute moderate-intensity aerobic exercise on sleep efficiency in a sample of adults with insomnia.[24] Additionally, 80% power was assumed with a two-sided $\alpha=0.05$ and a within-subject correlation of 0.63 for the sample size calculation. As previous literature does not inform a potential within-subject correlation, r=.63 was assumed. Other correlations were tested to explore the impact of different within-subject correlations on sample size requirements, and we found that within-subject correlations of r=.50 and .70 altered the recommended sample size to 30 and 20, respectively.

3.7 STATISTICAL ANALYSIS

Statistical analyses were performed using IBM SPSS Statistics (v. 23; IBM, Chicago, IL). Descriptive statistics characterized the sample, including demographics, BMI, unsupervised physical activity (i.e., MVPA), sleep quality, and insomnia severity. One-way ANOVA or independent t-tests were utilized to explore differences in demographic factors and expectations

in experimental effects. Repeated measures analysis of variance (ANOVA) models were used to examine the adherence to the experimental sessions and whether there were any differences in experimental session characteristics between the experimental groups.

To evaluate Specific Aims I and II, repeated measures analysis of variance (ANOVA) models were used to examine mean-level differences in actigraphic sleep efficiency (Aim I) and ISI (Aim II) between baseline and experimental conditions (i.e., weeks) as well as other secondary subjective and objectively measured dimensions of sleep and sleep timing. Partial eta squared effect sizes were calculated indicating small ($\eta_p^2 = 0.01$), medium ($\eta p^2 = 0.06$), or large effects ($\eta_p^2 = 0.14$).[180] Repeated measures ANOVA models were also used to examine whether meal timing (mean weekly timing of meals: breakfast, lunch, dinner) and caffeine and alcohol consumption (mean number of daily beverages consumed) were similar across baseline and experimental conditions for both groups.

To analyze Exploratory Aim I, mixed effect models accounting for the within-subject correlations between repeated measures was used to explore the association between pre-sleep arousal and the subsequent night's sleep and whether the magnitude of that relationship was modified by the experimental condition (interaction term: experimental group*pre-sleep arousal). In addition to the primary outcome of actigraphic sleep efficiency, all other actigraphy- and diary-assessed sleep variables were explored in individual models. If a significant or trending interaction was present, individual models were stratified by experimental group to explore the relationships.

For Exploratory Aim II, mixed effects models were used to explore whether sleep differed on nights following experimental sessions versus non-experimental session days. The model was restricted to the experimental week and explored the within-subject effect of experimental day (i.e., walking exercise or quiet rest) versus non-experimental day (reference). A between-subject

effect (day type*condition) was included to explore any differences in day type (experimental day vs. non-experimental day) by experimental condition (walking exercise vs. quiet rest).

Lastly, for Exploratory Aim III, a mixed effects model was restricted to the participants within the walking exercise condition and explored whether the relationship between exercise session day and sleep differed between the first, second, or third exercise session. The first exercise session was treated as the reference session.

4.0 RESULTS

The purpose of this experimental study was to investigate the impact of short-term exercise participation on sleep quality and insomnia severity in a sample of participants who met diagnostic criteria for insomnia. The results are presented below and begin with a description of the study sample and participant adherence. The subsequent results are then organized by specific aims.

4.1 STUDY PARTICIPANTS

4.1.1 Recruitment and Enrollment

A CONSORT flow diagram in **Figure 4** details the participant retention through the study. Overall, 248 individuals were contacted and screened for eligibility; of these, 205 individuals were excluded after initial screening for meeting exclusion criteria (e.g., BMI $\geq$ 30, ISI < 10). A total of 43 individuals consented to participate in the study. Four individuals were lost to follow up after consenting. Of those participants who completed the Qualtrics questionnaires and videoconference sleep interview, 4 were excluded due to evidence of a comorbid sleep disorder (e.g., restless legs syndrome), 4 were excluded for excessive anxiety or depressive symptoms (i.e., GAD-7 or PHQ-9 $\geq$ 10), 3 excluded themselves due to relocation or being out of region, and 1 was excluded due to a preexisting musculoskeletal injury in which walking exercise was contraindicated. Twenty-seven participants completed the home assessment and baseline sleep and physical activity monitoring period; following these baseline assessments, 3 were excluded for not wanting to

continue participation (n=1), non-compliance to the protocol (n=1), or BMI ≥ 30 (n=1). All 24 participants who were randomized to an experimental condition (n=12) to walking exercise group; n=12 to quiet rest group) completed the study.

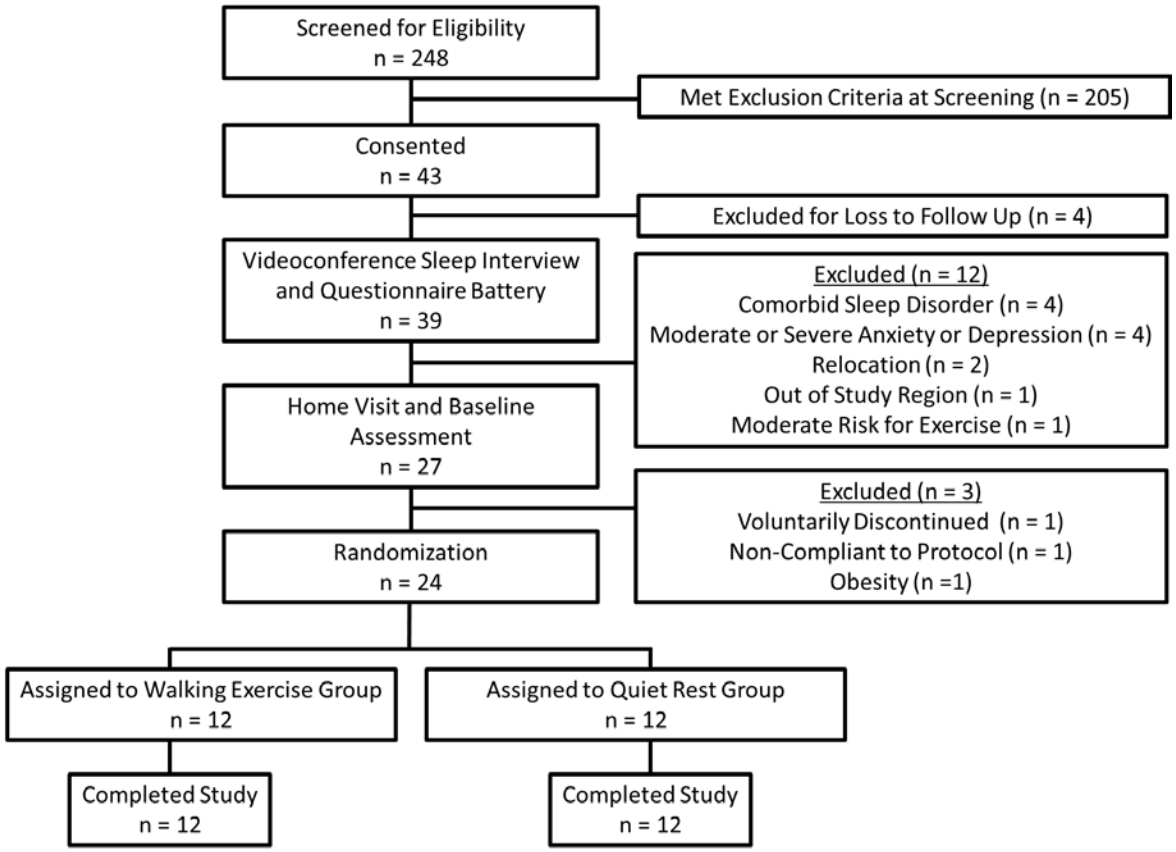


Figure 4. CONSORT flow diagram

4.1.2 Participant Characteristics

Table 3 summarizes the characteristics of the 24 participants who completed the study. On average, participants were 33.7±9.8 years old and had moderate to severe insomnia based on the ISI. In addition, 20 of 24 participants had Hyperarousal Scale values that were similar or greater

than prior studies of adults with insomnia[181], and 9 of the 24 participants had high-risk Ford Insomnia Response to Stress Test scores.[148] Participants reported mild anxiety and depression symptoms; only 4 participants had clinically diagnosed depression and/or anxiety, each of which was currently well controlled with either medication or counseling. No participants were participating in structured physical activity, and none met physical activity guidelines based on accelerometry.[182] There were no significant differences in demographic characteristics between those randomized to the walking exercise or quiet rest condition.

Table 3. Participant baseline characteristics

Characteristic	**Full Sample**		**Exercise Group**		**Quiet Rest Group**		**Difference**
Mean (SD)	N=24		n=12		n=12		p-value
Age (years)	33.7	(9.8)	33.3	(9.0)	34.0	(10.9)	0.87
Anxiety Symptoms (GAD-7; 0-21)	3.5	(2.4)	3.5	(2.6)	3.4	(2.3)	0.94
Depression Symptoms (PHQ-9; 0-27)	4.4	(3.3)	3.7	(2.9)	5.2	(3.7)	0.28
Hyperarousal (HS; 0-78)	37.5	(9.0)	38.2	(9.8)	36.9	(8.4)	0.74
Sleep Reactivity (FIRST; 9-36)	25.4	(6.1)	25.8	(7.0)	25.0	(5.2)	0.75
Chronotype (CSM; 13-54)	33.2	(7.8)	33.4	(9.8)	32.9	(5.6)	0.88
Insomnia Severity (ISI; 0-28)	15.6	(3.8)	14.4	(4.2)	16.8	(3.1)	0.14
Total MVPA (min/week) *	37.6	(38.9)	46.5	(46.7)	29.4	(8.5)	0.30
Body Mass Index (kg/m^2)	25.8	(4.0)	26.1	(3.1)	25.5	(4.9)	0.76
n (%)							
Female sex, n (%)	17	(70.8)	9	(75.0)	8	(66.7)	0.67
White race, n (%)	17	(70.8)	9	(75.0)	8	(66.7)	0.67
Diagnosed Depressive Disorder, n (%)	3	(12.5)	1	(8.3)	2	(16.7)	0.56
Diagnosed Anxiety Disorder, n (%)	3	(12.5)	2	(16.7)	1	(8.3)	0.56

*measured using ActiGraph GT9X accelerometer. Abbreviations: CSM: Composite Scale of Morningness; FIRST: Ford Insomnia Response to Stress Test; GAD-7: Generalized Anxiety Disorder-7; HS: Hyperarousal Scale; ISI: Insomnia Severity Index; MVPA: moderate- to vigorous-intensity physical activity; PHQ-9: Patient Health Questionnaire-9; SD: standard deviation.

4.1.3 Participant Expectations

Following randomization, but before beginning the experimental week, participants answered a brief three-item questionnaire that inquired about their expectations regarding changes to sleep and daytime function as a result of study participation. As displayed in **Figure 5**, no differences in expectations were found between those randomized to the walking exercise condition compared to the quiet rest condition (daytime function: 2.92±0.67 [exercise] vs. 2.50±0.52 [quiet rest], t=1.70, p=0.10; insomnia severity: 2.75±0.62 [exercise] vs. 2.58±0.51 [quiet rest], t=0.72, p=0.48; sleep quality: 2.83±0.84 [exercise] vs. 2.42±0.67 [quiet rest], t=1.35, p=0.19). Across both randomized groups, most participants indicated they expected to feel the same or somewhat better after the experimental week.

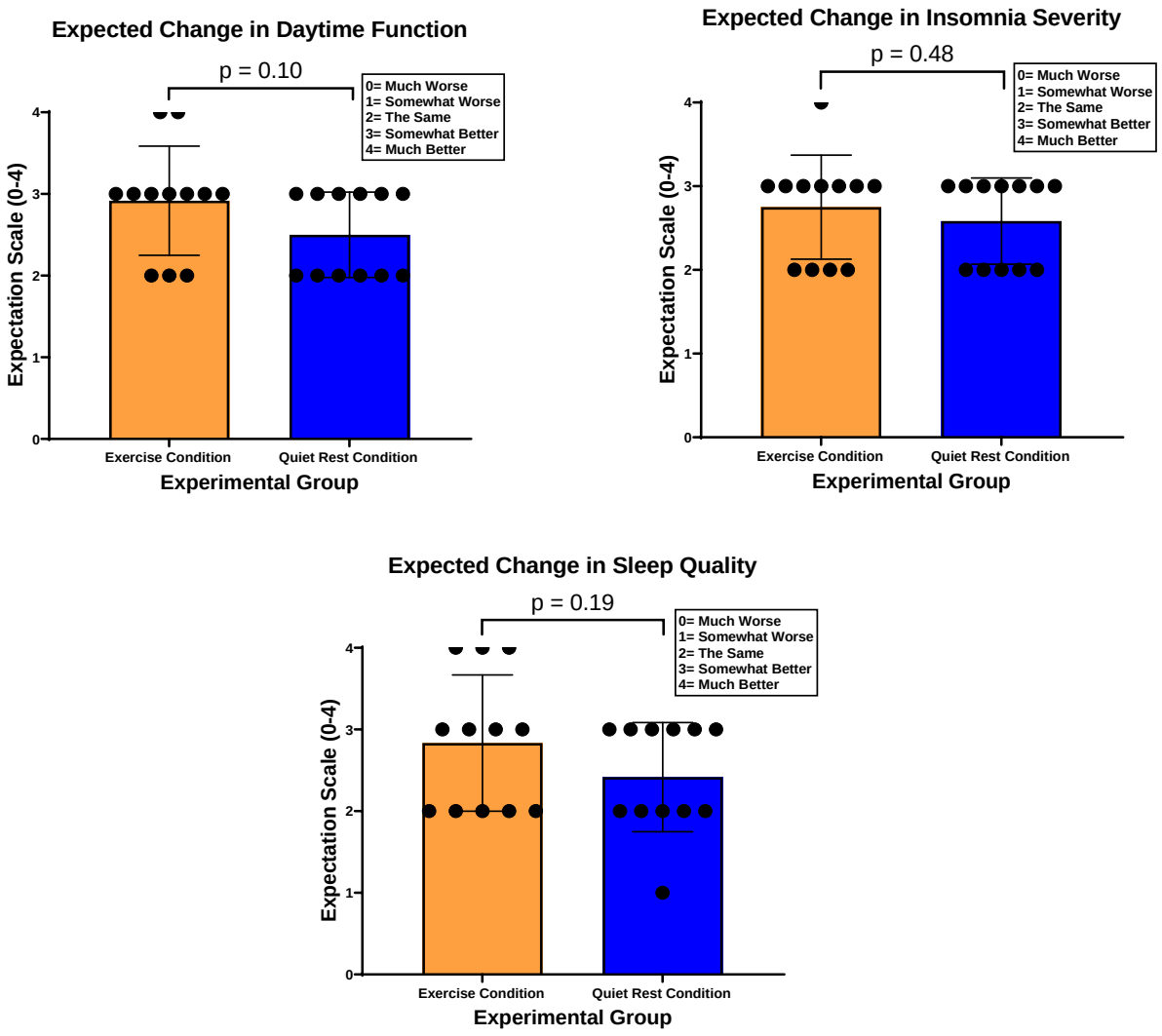


Figure 5. Participant expectations according to experimental condition.

4.1.4 Participant Adherence to the Protocol

Table 4 describes the session characteristics for the three experimental days for both randomized conditions. Session duration and time of day were derived from the Polar H10 heart rate monitor and M200 watch, while MVPA on session days was derived from the GT9X accelerometer. All sessions across experimental conditions occurred within the prescribed time interval (7:00 am-

12:00 pm) except for one walking exercise participant who completed their second walk at 3:00 pm.

Overall, objective data for 58 of the 72 experimental sessions were available for analysis. The missing data can be attributed to Polar H10 monitor failure due to participant error (e.g., quiet rest participants not starting the watch when prompted before video viewing, exercise participants improperly wearing the devices for prescribed walks, or inadequate charging prior to sessions resulting in missed session data). Although the missing data prevented us from obtaining objective verification of session completion across all experimental days, participants completed all experimental sessions as verified by interview and self-reported data.

During the experimental week, there were no significant differences between the characteristics of the three individual experimental sessions across either experimental condition ($p \geq 0.51$). Additionally, experimental sessions were similar in duration and timing between the experimental groups, respectively ($p=0.36$, $p=0.43$). Participants in the quiet rest condition had less MVPA across experimental session days compared to their walking exercise counterparts ($p=0.01$). Additionally, mean MVPA was significantly greater on days in which experimental sessions occurred compared to non-experimental days, with a significantly larger difference between day type in the walking exercise group (walking exercise group: 60.6±26.8 min [exercise days] vs. 5.6±9.7 min [non-experimental days]; quiet rest group: 21.0±23.2 min [rest session days] vs. 5.1±5.7 min [non-experimental days]; $p<0.001$). Mean % HRR is negative for quiet rest sessions due to the resting HR of the experimental sessions being lower than the resting HR ascertained at the home assessment. On average, the walking exercise participants met the prescribed heart rate goal of 50% HRR.

Table 4. Experimental session characteristics

Characteristic	Session 1		Session 2		Session 3		Session Effect	Condition Effect	Interaction Effect
Mean (SD)							p-value	p-value	p-value
% HRR achieved*[1]							0.652	**<0.001**	0.714
Exercise Condition	52.8	(13.2)	52.2	(9.9)	51.8	(5.2)			
Quiet Rest Condition	-1.0	(11.0)	-5.9	(12.0)	-1.6	(6.7)			
GT9X MVPA (min)*[2]							0.480	**0.010**	0.563
Exercise Condition	60.4	(23.5)	48.6	(27.7)	51.9	(22.9)			
Quiet Rest Condition	21.5	(25.1)	20.5	(28.3)	21.0	(28.4)			
Session Duration (min)[3]							0.938	0.355	0.227
Exercise Condition	38.3	(3.7)	40.4	(0.9)	38.9	(3.7)			
Quiet Rest Condition	34.9	(14.3)	33.6	(13.8)	34.9	(14.3)			
Time of Day (hh:mm)[4]							0.451	0.430	0.718
Exercise Condition	9:23	(2:43)	9:57	(3:15)	9:03	(1:55)			
Quiet Rest Condition	9:06	(2:03)	8:52	(1:52)	8:33	(1:41)			

*Bouted MVPA. GT9X: ActiGraph accelerometer; % HRR: percent heart rate reserve ([Mean session HR – RHR]/ [APMHR – RHR] * 100)); MVPA: moderate- to vigorous-intensity physical activity; SD: standard deviation.

(1) Exercise Condition: n=8
Quiet Rest Condition: n=7

(2) Exercise Condition: n=7
Quiet Rest Condition: n=10

(3) Exercise Condition: n=8
Quiet Rest Condition: n=8

(4) Exercise Condition: n=10
Quiet Rest Condition: n=12

Within the walking exercise group specifically, adherence to the exercise prescription was high. **Figure 6** describes the total exercise session duration and the duration of each exercise session that was within or above the prescribed heart rate range. On average, participants spent between 26.8 and 29.2 min at or above the prescribed heart rate range across all sessions compared to the goal of 30 minutes.

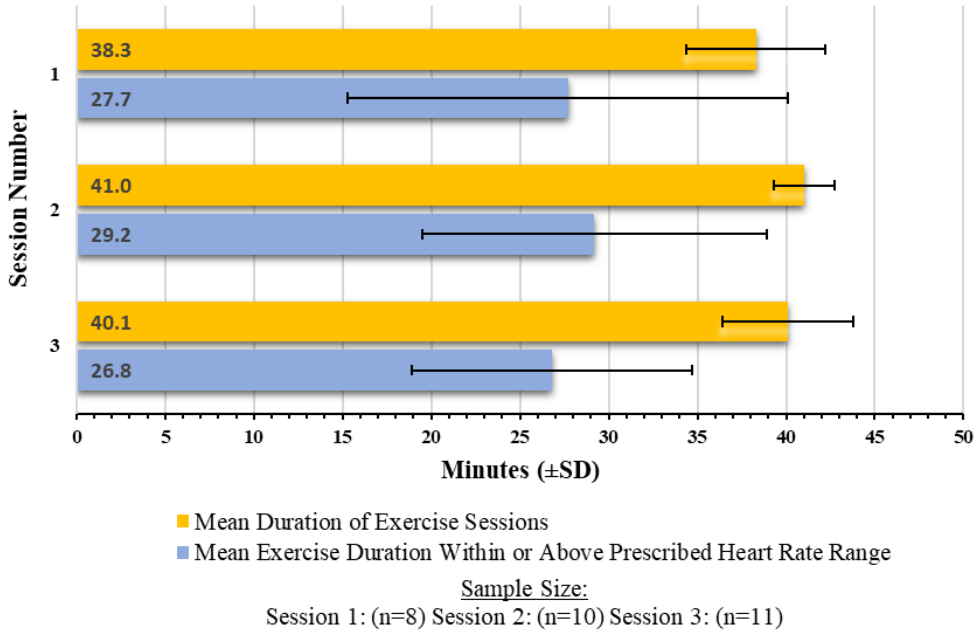


Figure 6. Exercise duration in prescribed heart rate zone

We also explored whether common lifestyle behaviors that may impact sleep differed from baseline to experimental assessments and whether these changes differed across experimental groups. As displayed in **Table 5**, we examined the timing of breakfast, lunch, and dinner as well as daily caffeine and alcohol consumption. None of the lifestyle behaviors changed from baseline to the experimental week ($p \geq 0.16$) except for breakfast timing; breakfast occurred approximately 20 minutes earlier in both conditions during the experimental week (main effect of experimental week; $p=0.021$). In addition, none of the lifestyle behaviors differed in their pattern of change across assessment weeks between the experimental conditions ($p \geq 0.17$).

Table 5. Maintenance of common lifestyle behaviors between experimental weeks

	Baseline Week Mean (SD)		Experimental Week Mean (SD)		Main Effect (Week)		Interaction (Week*Condition)	
					p	η_p^2	***p***	η_p^2
Breakfast (hh:mm)					**0.021**	0.345	0.238	0.105
Exercise Condition (n=8)	9:23	(1:11)	9:11	(1:07)				
Quiet Rest Condition (n=7)	9:57	(1:22)	9:25	(1:06)				
Lunch (hh:mm)					0.757	0.006	0.869	0.002
Exercise Condition (n=11)	13:13	(00:54)	13:07	(00:49)				
Quiet Rest Condition (n=7)	13:34	(1:14)	13:32	(00:58)				
Dinner (hh:mm)					0.618	0.012	0.174	0.086
Exercise Condition (n=12)	18:48	(00:55)	18:58	(00:57)				
Quiet Rest Condition (n=11)	19:21	(1:11)	19:00	(00:54)				
Caffeine (Servings/day)					0.165	0.086	0.286	0.051
Exercise Condition (n=12)	1.1	(1.2)	1.2	(1.0)				
Quiet Rest Condition (n=12)	1.3	(1.4)	2.1	(3.3)				
Alcohol (Servings/day)					0.851	0.002	0.814	0.003
Exercise Condition (n=12)	0.8	(0.9)	0.8	(0.7)				
Quiet Rest Condition (n=12)	0.4	(0.3)	0.4	(0.7)				

Missing data points are due to less than 4 days of available data for either assessment week. Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; *p*: p-value; SD: standard deviation.

4.2 SPECIFIC AIM I: Actigraphy- and Diary-Assessed Sleep

Table 6 describes the changes in sleep outcomes between experimental weeks in the walking exercise and quiet rest conditions. The primary outcome of Specific Aim I was actigraphy-assessed sleep efficiency. The change in actigraphy-assessed SE from baseline to experimental week was similar across experimental conditions ($p=0.52$, $\eta_p^2=0.019$). Following this pattern, the change in diary-assessed sleep efficiency from baseline to experimental week was also similar across experimental conditions ($p=0.88$, $\eta_p^2=0.001$). Although there was a significant reduction in diary-assessed sleep onset latency and actigraphy-assessed wake after sleep onset between weeks (SOL: $p=.004$, $\eta_p^2=0.314$; WASO: $p=0.038$, $\eta_p^2=0.181$), the reduction in each variable did not

differ between experimental groups (SOL: $p=0.73$, $\eta_p^2=0.006$; WASO: $p=0.29$, $\eta_p^2=0.050$). Across other measured actigraphy- and diary-assessed sleep variables, there were no significant differences in treatment effect between either experimental condition ($p\geq0.16$). However, a trending interaction effect with actigraphy-assessed sleep onset latency suggested a reduction in sleep onset latency between baseline and experimental weeks in the walking exercise condition while sleep onset latency increased in the quiet rest condition ($p=0.087$, $\eta_p^2=0.127$). **Figure 7** and **Figure 8** detail the main effects of the intervention across experimental conditions while highlighting the large amount of variability in mean change in sleep variables.

Table 6. Changes in actigraphy- and diary-assessed sleep variables across experimental weeks

	Baseline Week Mean (SD)		**Experimental Week** Mean (SD)		**Main Effect** (Week)		**Interaction** (Week*Condition)	
					p	η_p^2	***p***	η_p^2
Actigraphy SlpE (%)					0.193	0.076	0.524	0.019
Exercise Condition	87.9	(5.9)	89.0	(3.8)				
Quiet Rest Condition	84.7	(6.5)	85.1	(5.7)				
Diary SlpE (%)					*0.069*	0.142	0.877	0.001
Exercise Condition	85.5	(9.0)	88.5	(8.5)				
Quiet Rest Condition	84.3	(11.1)	86.8	(10.7)				
Actigraphy TIB (h)					*0.054*	0.158	0.359	0.038
Exercise Condition	8.2	(0.8)	8.1	(0.7)				
Quiet Rest Condition	8.0	(1.1)	7.6	(1.1)				
Diary TIB (h)					*0.091*	0.125	0.165	0.086
Exercise Condition	8.5	(0.7)	8.4	(0.8)				
Quiet Rest Condition	8.1	(0.9)	7.6	(1.1)				
Actigraphy TST (h)					0.161	0.087	0.202	0.073
Exercise Condition	7.2	(1.0)	7.2	(0.7)				
Quiet Rest Condition	6.8	(1.0)	6.4	(0.7)				
Diary TST (h)					0.937	0.000	0.389	0.034
Exercise Condition	7.3	(1.1)	7.5	(1.0)				
Quiet Rest Condition	6.8	(1.3)	6.6	(1.3)				
Actigraphy SOL (min)					0.708	0.007	*0.087*	0.127
Exercise Condition	12.2	(11.2)	9.3	(4.5)				
Quiet Rest Condition	15.6	(18.4)	20.2	(28.0)				
Diary SOL (min)					**0.004**	0.314	0.727	0.006
Exercise Condition	38.7	(28.8)	30.4	(27.1)				
Quiet Rest Condition	36.9	(31.2)	30.3	(25.5)				
Actigraphy WASO (min)					**0.038**	0.181	0.292	0.050
Exercise Condition	45.7	(16.5)	43.2	(15.5)				
Quiet Rest Condition	58.6	(20.9)	51.4	(16.3)				
Diary WASO (min)					0.154	0.090	0.732	0.005
Exercise Condition	32.3	(30.3)	25.6	(23.3)				
Quiet Rest Condition	39.9	(33.2)	29.2	(29.4)				
Actigraphy Bedtime (h)					0.936	0.000	0.975	0.000
Exercise Condition	23.9	(1.5)	23.9	(1.8)				
Quiet Rest Condition	24.4	(1.3)	24.4	(1.0)				
Diary Bedtime (h)					0.958	0.000	0.172	0.083
Exercise Condition	23.9	(1.4)	23.7	(1.3)				
Quiet Rest Condition	24.3	(1.1)	24.5	(1.1)				
Actigraphy Waketime (h)					0.190	0.077	0.536	0.018
Exercise Condition	8.1	(1.6)	8.0	(1.6)				
Quiet Rest Condition	8.4	(1.6)	8.0	(1.3)				
Diary Waketime (h)					0.149	0.092	0.826	0.002
Exercise Condition	8.4	(1.4)	8.1	(1.4)				
Quiet Rest Condition	8.3	(1.6)	8.1	(1.4)				
Actigraphy SMP (h)					0.537	0.018	0.890	0.001
Exercise Condition	4.1	(1.5)	4.0	(1.7)				
Quiet Rest Condition	4.5	(1.2)	4.4	(1.1)				

Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; *p*: p-value; SD: standard deviation; SlpE: sleep efficiency; SMP: sleep midpoint; SOL: sleep onset latency; TIB: time in bed; TST: total sleep time; WASO: wake after sleep onset.

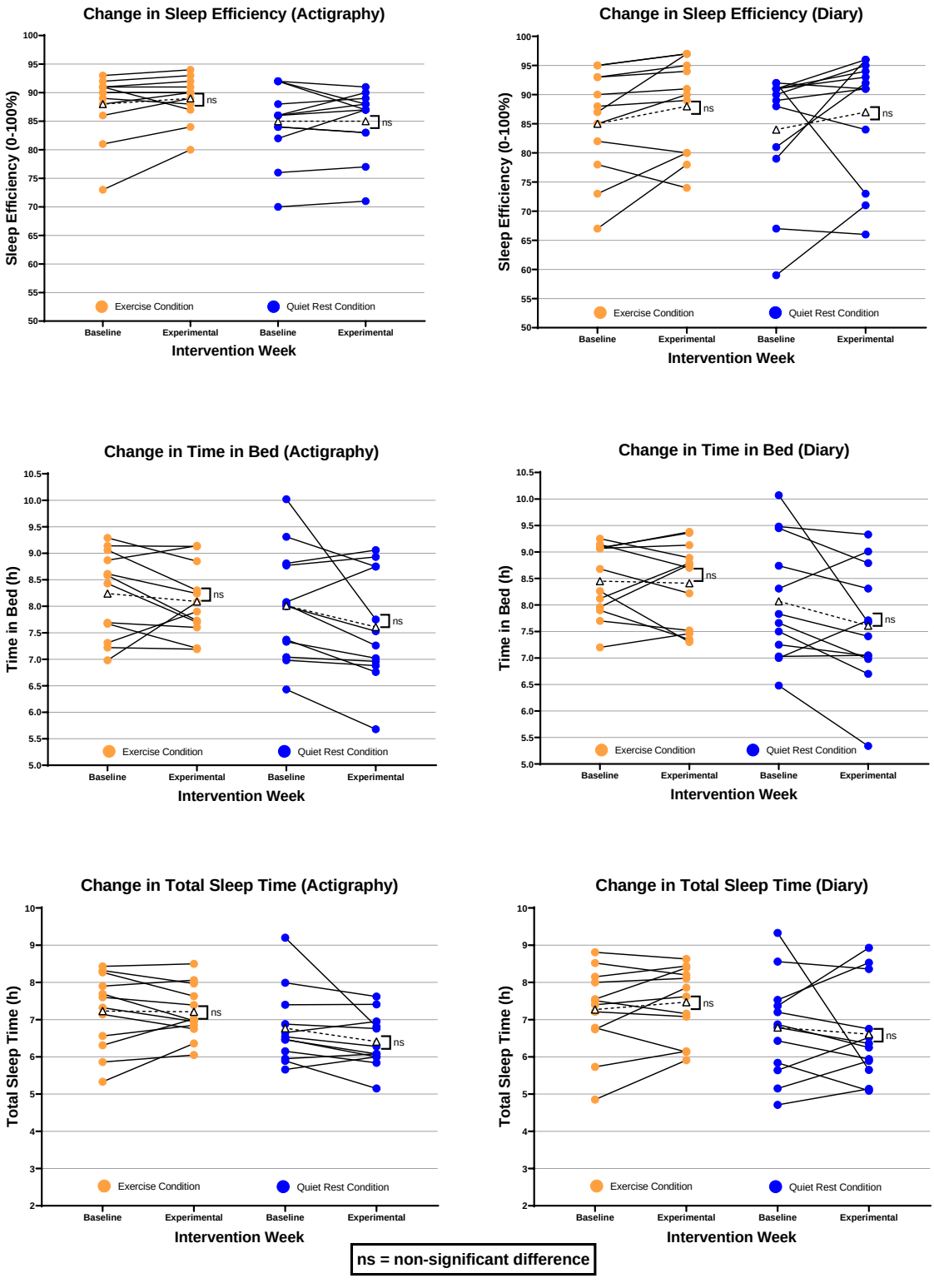


Figure 7. Changes in actigraphy- and diary-assessed SlpE, TIB, and TST

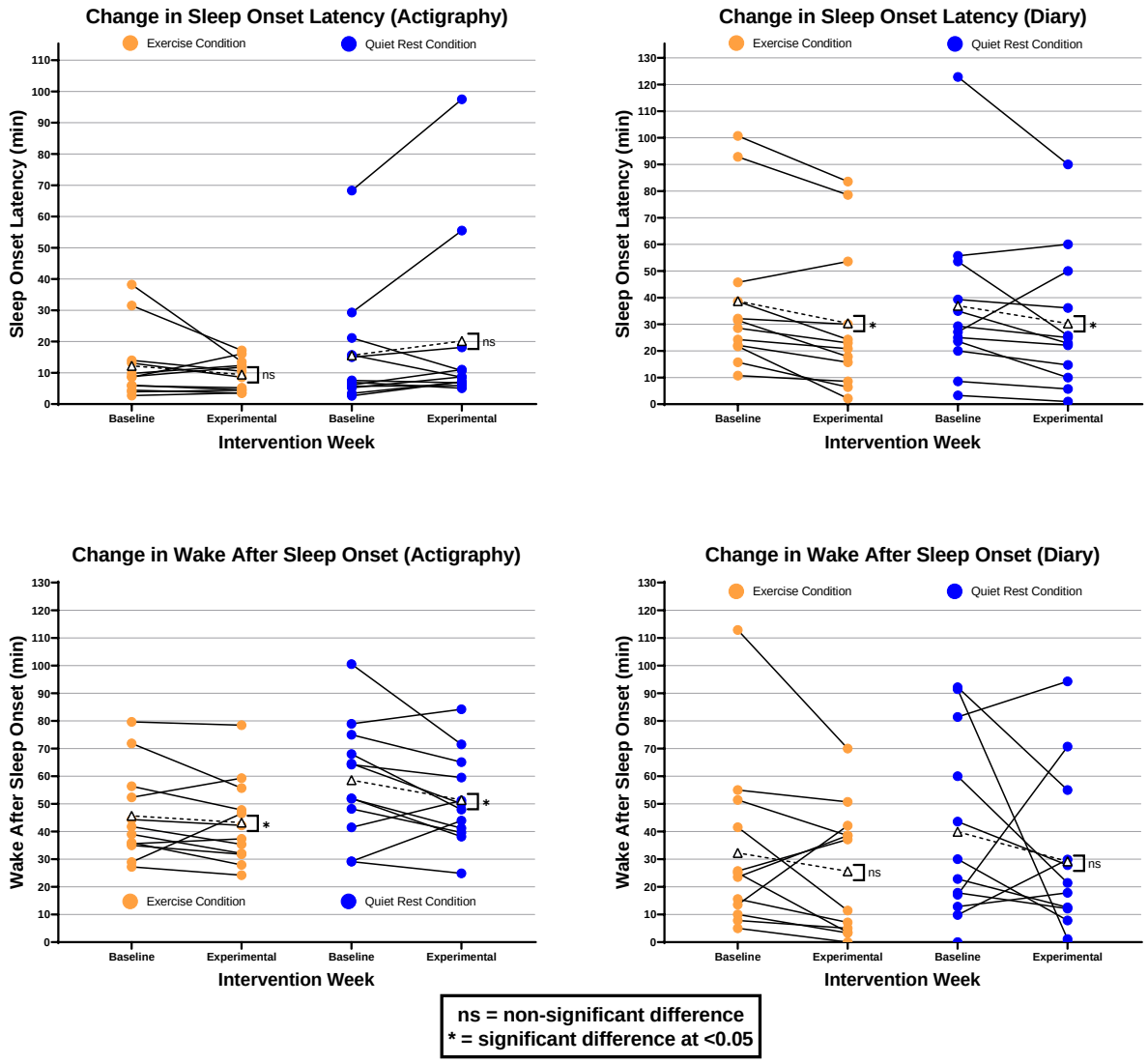


Figure 8. Changes in actigraphy- and diary-assessed SOL and WASO

The secondary outcome of Specific Aim I was self-reported sleep disturbance measured with the PROMIS Sleep Disturbance scale (PROMIS-SD; **Table 7)**. The change in sleep disturbance between the baseline and experimental weeks was similar across experimental conditions ($p=0.59$, $\eta_p^2=0.014$). Likewise, the change in sleep health from baseline to experimental

week was not different between conditions (p=0.54, η_p^2=0.018). **Figure 9** displays the participant-level change in sleep disturbance and sleep health within experimental conditions.

Table 7. Changes in sleep disturbance and sleep health across experimental weeks

	Baseline Week Mean (SD)		**Experimental Week** Mean (SD)		**Main Effect** (Week)		**Interaction** (Week*Condition)	
					p	η_p^2	***p***	η_p^2
PROMIS-SD (28.9-76.5)					0.147	0.093	0.587	0.014
Exercise Condition	57.9	(6.2)	56.8	(8.5)				
Quiet Rest Condition	60.9	(3.4)	58.5	(8.2)				
RU-SATED Score (0-12)					0.535	0.018	0.535	0.018
Exercise Condition	6.7	(1.9)	7.3	(2.4)				
Quiet Rest Condition	6.0	(2.3)	6.0	(2.1)				

Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; *p*: p-value; PROMIS-SD: PROMIS Sleep Disturbance short-form scale; RU-SATED: sleep health questionnaire; SD: standard deviation.

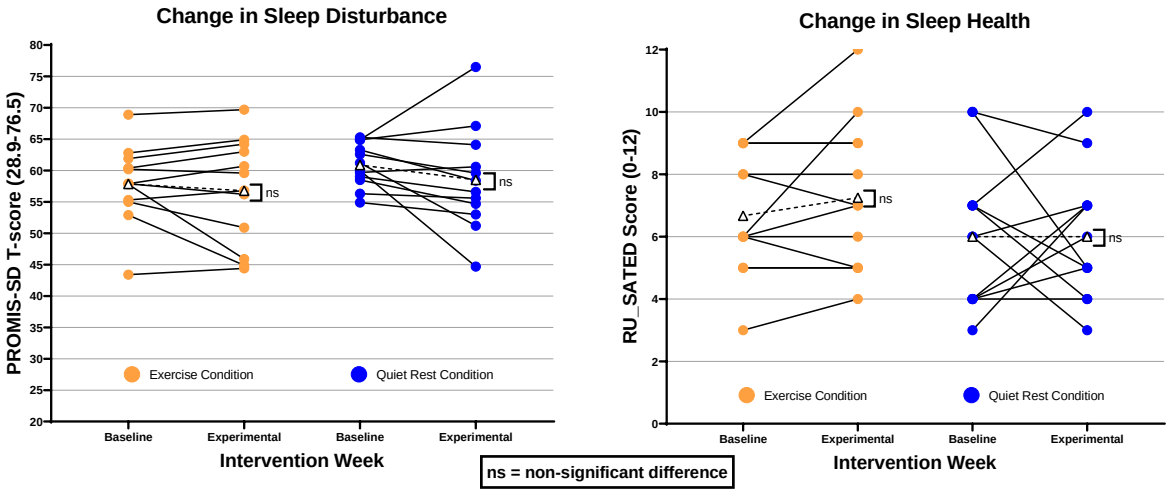


Figure 9. Changes in sleep disturbance and sleep health

4.3 SPECIFIC AIM II: Daytime Impairment

4.3.1 Primary and Secondary Outcomes

Table 8 describes the change in global self-reported measures of daytime impairment between experimental weeks in the walking exercise and quiet rest conditions. The primary outcome of Specific Aim II was insomnia severity measured with the Insomnia Severity Index (ISI). Although there was a significant reduction in insomnia severity from baseline to experimental week ($p=0.002$, $\eta_p^2=0.359$), the reduction in severity did not differ between experimental conditions ($p=1.0$, $\eta_p^2=0.000$). The secondary outcome for Specific Aim II was daytime impairment measured with the PROMIS Sleep-Related Impairment scale (PROMIS-SRI). There was a significant reduction in daytime impairment from baseline to experimental week ($p=<0.001$, $\eta_p^2=0.443$); however, the reduction in daytime impairment did not differ between experimental conditions ($p=0.35$, $\eta_p^2=0.039$). **Figure 10** details the participant-level change in insomnia severity and daytime impairment across assessment weeks according to experimental group.

Table 8. Changes in insomnia severity and daytime impairment across experimental weeks

	Baseline Week Mean (SD)		**Experimental Week** Mean (SD)		**Main Effect** (Week)		**Interaction** (Week*Condition)	
					p	η_p^2	***p***	η_p^2
ISI (0-28)					**0.002**	0.359	1.0	0.000
Exercise Condition	14.4	(4.2)	11.9	(5.1)				
Quiet Rest Condition	16.8	(3.1)	14.3	(5.2)				
PROMIS-SRI (30.0-80.0)					**< 0.001**	0.443	0.354	0.039
Exercise Condition	55.0	(7.1)	52.3	(9.0)				
Quiet Rest Condition	62.2	(6.4)	57.9	(7.1)				

Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; ISI: Insomnia Severity Index; *p*: p-value; PROMIS-SRI: PROMIS Sleep-Related Impairment short-form scale; SD: standard deviation.

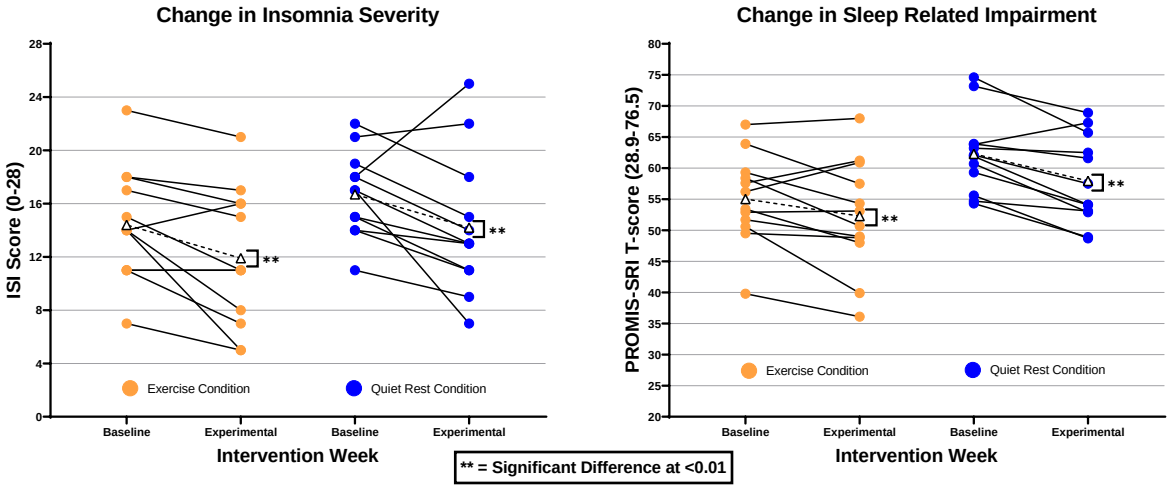


Figure 10. Changes in insomnia severity and daytime impairment

4.3.2 Additional Diary Sleep Quality and Pre-sleep Arousal Assessments

Table 9 describes the changes in additional diary-based measures of sleep quality and arousal between conditions. No significant change from baseline to experimental week was found in most variables. Although there was a significant reduction in experiencing an overactive mind prior to bedtime from baseline to experimental week ($p=0.034$, $\eta_p^2=0.189$), the reduction did not differ between experimental conditions ($p=0.63$, $\eta_p^2=0.010$). **Table 10** describes the change in pre-sleep arousal (PSAS) between conditions. Although there was a significant reduction in total pre-sleep arousal and pre-sleep cognitive arousal (Total PSAS: $p=0.002$, $\eta_p^2=0.366$; PSAS-Cognitive: $p=0.001$, $\eta_p^2=0.413$), the reduction in either variable did not differ between experimental conditions (Total PSAS: $p=0.64$, $\eta_p^2=0.010$; PSAS-Cognitive: $p=0.42$, $\eta_p^2=0.029$). While there was a trend toward a reduction in pre-sleep somatic arousal from the baseline to experimental week, the change did not differ by experimental condition ($p=0.752$, $\eta_p^2=0.005$). **Figure 11** details

the participant-level change in pre-sleep arousal across assessment weeks within experimental conditions.

Table 9. Changes in additional sleep diary variables across experimental weeks

	Baseline Week Mean (SD)		**Experimental Week** Mean (SD)		**Main Effect** (Week)		**Interaction** (Week*Condition)	
					p	η_p^2	***p***	η_p^2
Sleep Quality (0-5)					0.457	0.025	0.291	0.051
Exercise Condition	1.8	(0.5)	2.0	(0.6)				
Quiet Rest Condition	1.8	(0.4)	1.8	(0.6)				
Feeling Rested (0-5)					*0.083*	0.130	0.551	0.016
Exercise Condition	1.6	(0.5)	1.9	(0.7)				
Quiet Rest Condition	1.3	(0.6)	1.4	(0.7)				
Feeling Anxious (VAS)					0.208	0.071	0.933	0.000
Exercise Condition	50.0	(20.5)	35.5	(18.8)				
Quiet Rest Condition	37.0	(22.2)	32.3	(23.7)				
Feeling Worried (VAS)					0.147	0.093	0.939	0.000
Exercise Condition	37.3	(18.2)	32.3	(17.7)				
Quiet Rest Condition	35.5	(21.3)	29.9	(23.3)				
Overactive Mind (VAS)					**0.034**	0.189	0.634	0.010
Exercise Condition	50.8	(19.6)	42.0	(18.1)				
Quiet Rest Condition	47.1	(24.8)	41.4	(26.1)				
Feeling Tense (VAS)					0.615	0.012	0.920	0.000
Exercise Condition	28.7	(21.3)	27.4	(21.2)				
Quiet Rest Condition	32.5	(20.0)	30.5	(25.1)				

Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; *p*: p-value; SD: standard deviation; VAS: visual analog scales

Table 10. Changes in pre-sleep arousal across experimental weeks

	Baseline Week Mean (SD)		**Experimental Week** Mean (SD)		**Main Effect** (Week)		**Interaction** (Week*Condition)	
					p	η_p^2	***p***	η_p^2
PSAS-Total (16-80)					**0.002**	0.366	0.643	0.010
Exercise Condition	31.9	(7.9)	27.9	(6.4)				
Quiet Rest Condition	30.2	(5.2)	27.1	(6.3)				
PSAS-Cognitive (8-40)					**0.001**	0.413	0.422	0.029
Exercise Condition	20.5	(5.2)	17.0	(4.2)				
Quiet Rest Condition	19.5	(4.5)	17.3	(5.5)				
PSAS-Somatic (8-40)					*0.083*	0.130	0.752	0.005
Exercise Condition	11.5	(3.0)	10.9	(2.7)				
Quiet Rest Condition	10.7	(1.7)	9.8	(1.3)				

Significant values are bolded. Trending values are italicized. Abbreviations: η_p^2: partial eta-squared effect size; PSAS: pre-sleep arousal scale; SD: standard deviation

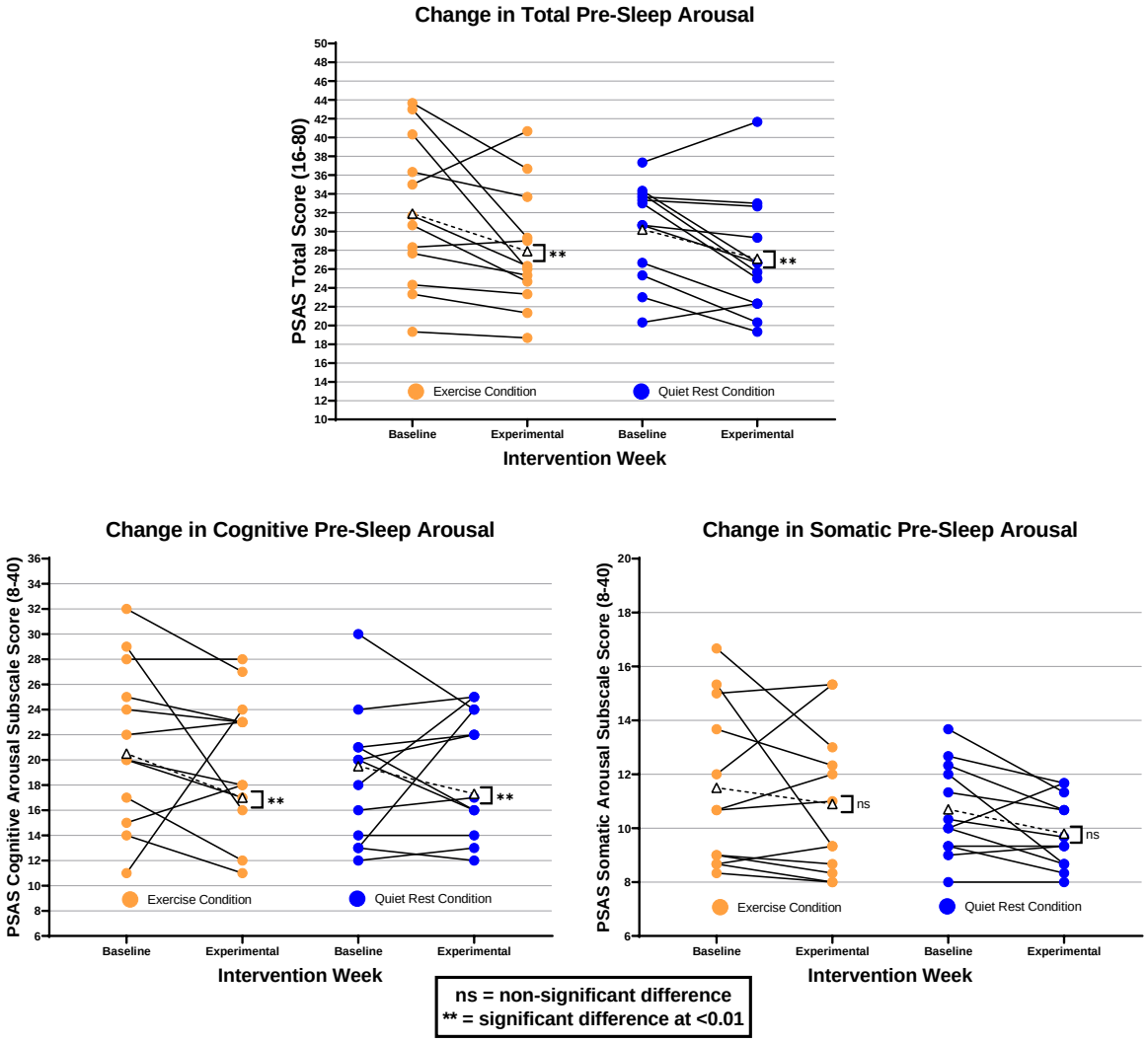


Figure 11. Changes in pre-sleep arousal across experimental weeks

4.4 EXPLORATORY AIMS

4.4.1 Exploratory Aim I

Table 11 describes the associations between pre-sleep arousal and subsequent actigraphy- and diary-assessed sleep during the experimental week. Greater pre-sleep arousal was associated with lower actigraphy-assessed sleep efficiency (B=-0.3, SE=0.1, p=0.030), lower diary-assessed sleep efficiency (B=-0.8, SE=0.2, p=0.001), greater diary-assessed sleep onset latency (B=2.6, SE=0.7, p=<0.001), greater actigraphy-assessed wake after sleep onset (B=1.6, SE=0.5, p=0.048), lower sleep quality (B=-0.1, SE=0.2, p=0.001), and lower feelings of restedness (B=-0.1, SE=0.2, p=0.003). We also found that the association between pre-sleep arousal and diary-assessed sleep onset latency and actigraphy-assessed wake after sleep onset differed according to the experimental condition, respectively (p=0.092; p=0.002).

In stratified analyses summarized in **Table 12**, the association between pre-sleep arousal and diary-assessed sleep onset latency was stronger in the quiet rest condition (walking exercise: B=-1.0, SE=0.7, p=0.185; quiet rest: B=2.2, SE=0.5, p<0.001). The association between pre-sleep arousal and higher actigraphy-assessed wake after sleep onset was also stronger in the quiet rest condition (walking exercise: B=0.4, SE=0.3, p=0.208; quiet rest: B=1.5, SE=0.6, p=0.015).

Table 11. Association between pre-sleep arousal and dependent sleep outcomes

Sleep Outcome				**Interaction** (Condition*PSAS)
	B (SE)*		p-value	p-value
Actigraphy SlpE (%)	-0.3	(0.1)	**0.030**	0.108
Diary SlpE (%)	-0.8	(0.2)	**0.001**	0.311
Actigraphy TIB (min)	-2.2	(2.5)	0.381	0.473
Diary TIB (min)	-0.40	(2.0)	0.866	0.603
Actigraphy TST (min)	-2.8	(2.1)	0.200	0.468
Diary TST (min)	-4.2	(2.8)	0.134	0.242
Actigraphy SOL (min)	0.5	(0.2)	*0.068*	0.315
Diary SOL (min)	2.6	(0.7)	**<0.001**	*0.092*
Actigraphy WASO (min)	1.6	(0.5)	**0.048**	**0.002**
Diary WASO (min)	1.3	(0.8)	0.125	0.525
Actigraphy Bedtime (min)	-1.6	(2.8)	0.582	0.323
Diary Bedtime (min)	-0.8	(2.5)	0.735	0.381
Actigraphy Waketime (min)	-3.2	(2.8)	0.261	0.824
Diary Waketime (min)	0.2	(2.8)	0.948	0.284
Actigraphy SMP (min)	-2.6	(2.5)	0.317	0.598
Sleep Quality (0-5)	-0.1	(0.02)	**0.001**	0.877
Feeling Rested (0-5)	-0.1	(0.02)	**0.003**	0.625

Significant values are bolded. Trending values are italicized. B (SE)*: represents the change in the dependent variable for every 1-unit increase in total PSAS score. Abbreviations: B: beta coefficient; PSAS: Pre-Sleep Arousal Scale; SE: standard error; SlpE: sleep efficiency; SMP sleep midpoint; SOL: sleep onset latency; TIB: time in bed; TST: total sleep time; WASO: wake after sleep onset

Table 12. Relationship between pre-sleep arousal and selected sleep outcomes within experimental condition

Sleep Outcome	B (SE)*		p-value
Actigraphy WASO (min)			
Exercise Condition (n=12)	0.4	(0.3)	0.208
Quiet Rest Condition (n=12)	1.5	(0.6)	**0.015**
Diary SOL (min)			
Exercise Condition (n=12)	-1.0	(0.7)	0.185
Quiet Rest Condition (n=12)	2.2	(0.5)	**<0.001**

Significant values are bolded. Trending values are italicized. β (SE)*: represents the change in the dependent variable for every 1-unit increase in total PSAS score. Abbreviations: B: beta coefficient; PSAS: Pre-Sleep Arousal Scale; SE: standard error; SOL: sleep onset latency; WASO: wake after sleep onset

4.4.2 Exploratory Aim II

Table 13 displays the differences in sleep between experimental (i.e., quiet rest or exercise session) days and non-experimental days (i.e., typical days without experimental manipulation) and whether those differences varied between experimental condition. Based on interaction analyses, actigraphy- and diary-assessed total sleep time increased on exercise days compared to non-experimental days in the walking exercise condition to a greater extent than the differences observed on quiet rest days vs. non-experimental days in the quiet rest group (actigraphy: B=0.9, SE=0.4, p=0.032; diary: B=0.8, SE=0.4, p=0.049). A similar pattern was observed for time in bed, though the interaction effects only trended toward significance (actigraphy: B=0.8, SE=0.4, p=0.080; diary: B=0.6, SE=0.4, p=0.094).

The exercise group also reported feeling significantly more rested and refreshed after a night following exercise compared to non-experimental days while the quiet rest group's feeling of refreshment was stable across day types (walking exercise: 2.1±0.2 vs. 1.7±0.2; quiet rest: 1.4±0.2 vs. 1.4±0.2; interaction: B=0.5, SE=0.2, p=0.019). A similar pattern was observed for sleep quality, though the interaction effect only trended toward significance (walking exercise: 2.3±0.2 vs. 1.8±0.2; quiet rest: 1.7±0.2 vs. 1.7±0.2; interaction: B=0.4, SE=0.2, p=0.076). No other sleep variables demonstrated differences between experimental and non-experimental days that differed between walking exercise and quiet rest (each interaction $p \geq 0.113$).

Table 13. Differences in sleep outcomes between experimental and non-experimental days

	Exercise Condition		Quiet Rest Condition				
Sleep Outcome	**Exercise Days** Mean (SE)	**Non-Exp Days** Mean (SE)	**Quiet Rest Days** Mean (SE)	**Non-Exp Days** Mean (SE)	**Day Effect** p-value	**Condition Effect** p-value	**Interaction Effect** B (SE)* p-value
Actigraphy SlpE (%)	90.1 (1.2)	89.2 (1.2)	85.6 (1.2)	86.6 (1.2)	0.902	**0.036**	1.8 (1.3) 0.151
Diary SlpE (%)	90.1 (2.7)	88.6 (2.7)	87.4 (3.0)	87.2 (3.0)	0.268	0.525	2.0 (2.1) 0.341
Actigraphy TIB (h)	8.4 (0.3)	8.0 (0.3)	7.4 (0.3)	7.7 (0.3)	0.941	0.114	0.8 (0.4) *0.080*
Diary TIB (h)	8.6 (0.3)	8.2 (0.3)	7.3 (0.3)	7.6 (0.3)	0.544	*0.045*	0.6 (0.4) *0.094*
Actigraphy TST (h)	7.5 (0.3)	7.0 (0.3)	6.2 (0.2)	6.6 (0.2)	0.952	**0.015**	0.9 (0.4) **0.032**
Diary TST (h)	7.8 (0.4)	7.2 (0.4)	6.4 (0.4)	6.6 (0.4)	0.273	*0.065*	0.8 (0.4) **0.049**
Actigraphy SOL (min)	6.1 (2.3)	7.4 (2.3)	12.3 (2.3)	9.7 (2.2)	0.731	0.106	-3.9 (3.9) 0.326
Diary SOL (min)	27.2 (7.2)	29.8 (7.3)	28.1 (7.2)	31.6 (7.2)	0.225	0.899	0.8 (4.9) 0.875
Actigraphy WASO (min)	41.3 (4.8)	42.0 (5.0)	49.2 (4.8)	54.7 (5.0)	0.290	0.114	4.8 (5.8) 0.413
Diary WASO (min)	17.8 (7.4)	21.8 (7.4)	26.3 (7.4)	23.8 (7.3)	0.811	0.594	-6.4 (6.5) 0.324
Actigraphy Bedtime (h)	23.9 (0.4)	23.7 (0.4)	24.8 (0.4)	24.0 (0.4)	**0.002**	0.309	-0.6 (0.3) 0.113
Diary Bedtime (h)	23.7 (0.4)	23.6 (0.4)	24.9 (0.4)	24.4 (0.4)	**0.004**	*0.086*	-0.5 (0.3) *0.085*
Actigraphy Waketime (h)	8.2 (0.4)	7.9 (0.5)	8.0 (0.4)	7.9 (0.5)	0.142	0.915	0.2 (0.3) 0.601
Diary Waketime (h)	8.1 (0.4)	7.7 (0.4)	8.0 (0.4)	7.7 (0.4)	**0.013**	0.936	-0.02 (0.3) 0.947
Actigraphy SMP (h)	4.1 (0.4)	3.9 (0.4)	4.6 (0.4)	4.0 (0.4)	**0.004**	0.551	-0.4 (0.3) 0.204
Sleep Quality (0-5)	2.3 (0.2)	1.8 (0.2)	1.7 (0.2)	1.7 (0.2)	*0.078*	0.230	0.4 (0.2) *0.076*
Feeling Rested (0-5)	2.1 (0.2)	1.7 (0.2)	1.4 (0.2)	1.4 (0.2)	*0.060*	0.106	0.5 (0.2) **0.019**
Feeling Anxious (VAS)	29.5 (6.5)	39.0 (6.6)	29.7 (6.6)	33.8 (6.6)	**0.016**	0.776	-5.4 (5.5) 0.336
Feeling Worried (VAS)	29.4 (6.3)	34.6 (6.3)	28.9 (6.3)	30.4 (6.2)	0.215	0.788	-3.8 (5.3) 0.484
Overactive Mind (VAS)	41.9 (7.4)	46.7 (7.3)	42.1 (7.4)	39.3 (7.2)	0.731	0.725	-7.6 (5.9) 0.207
Feeling Tense (VAS)	26.3 (7.2)	30.1 (7.1)	27.8 (7.2)	31.1 (7.1)	0.162	0.895	-0.5 (5.0) 0.919

Significant values are bolded. Trending values are italicized. B (SE)*: Represents the difference in the difference between experimental and non-experimental days comparing the quiet rest condition versus the walking exercise condition (n=3 experimental days; n=4 non-experimental days). Abbreviations: B: beta coefficient; SE: standard error; SlpE: sleep efficiency; SMP: sleep midpoint; SOL: sleep onset latency; TIB: time in bed; TST: total sleep time; VAS: visual analog scale; WASO: wake after sleep onset

4.4.3 Exploratory Aim III

Exploratory Aim III investigated whether the effect of exercise on sleep outcomes was similar across the three experimental sessions. Compared to sleep following the first exercise session, there were no differences in sleep across the second and third exercise sessions (**Table 14** and **Table 15**), except for actigraphy-assessed sleep onset latency. Participants had significantly greater sleep onset latency on the night following the third exercise session compared to the night following their first exercise session (B=4.7, SE=2.0, p=0.031).

Table 14. Sleep timing following each exercise session

Sleep Outcome (n=12)	Mean (SE)	B (SE)	p-value
Actigraphy Bedtime (h)			
1st Session	23.8 (0.6)	---	---
2nd Session	23.7 (0.6)	-0.1 (0.4)	0.802
3rd Session	24.3 (0.6)	0.4 (0.4)	0.409
Diary Bedtime (h)			
1st Session	23.8 (0.4)	---	---
2nd Session	23.6 (0.4)	-0.2 (0.3)	0.659
3rd Session	23.7 (0.4)	-0.1 (0.3)	0.403
Actigraphy Waketime (h)			
1st Session	8.5 (0.6)	---	---
2nd Session	7.8 (0.6)	-0.7 (0.5)	0.227
3rd Session	8.3 (0.6)	-0.1 (0.5)	0.792
Diary Waketime (h)			
1st Session	8.7 (0.6)	---	
2nd Session	7.9 (0.6)	-0.1 (0.5)	0.117
3rd Session	8.6 (0.6)	-0.8 (0.5)	0.822
Actigraphy SMP (h)			
1st Session	4.2 (0.6)	---	---
2nd Session	3.8 (0.5)	-0.4 (0.4)	0.402
3rd Session	4.4 (0.5)	0.2 (0.4)	0.669

Values represent the comparison between sleep following that exercise session versus sleep following the 1st session. Significant values are bolded. Trending values are italicized. Abbreviations: B: beta coefficient; SE: standard error; SMP: sleep midpoint

Table 15. Sleep outcomes following each exercise session

Sleep Outcome (n=12)	Mean (SE)		B (SE)		P-Value
Actigraphy SlpE (%)					
1st Session	90.7	(1.1)	---		---
2nd Session	89.2	(1.1)	-1.5	(0.9)	0.147
3rd Session	89.8	(1.1)	-0.8	(0.9)	0.405
Diary SlpE (%)					
1st Session	90.5	(2.9)	---		---
2nd Session	89.1	(2.9)	-1.4	3.6	0.698
3rd Session	91.4	(2.9)	0.9	3.6	0.810
Actigraphy TIB (h)					
1st Session	8.7	(0.5)	---		---
2nd Session	8.1	(0.4)	-0.6	0.6	0.323
3rd Session	8.1	(0.4)	-0.6	0.6	0.311
Diary TIB (h)					
1st Session	8.9	(0.4)	---		---
2nd Session	8.3	(0.4)	-0.6	(0.5)	0.252
3rd Session	8.9	(0.4)	0.02	(0.5)	0.973
Actigraphy TST (h)					
1st Session	7.9	(0.5)	---		---
2nd Session	7.2	(0.5)	-0.7	(0.5)	0.249
3rd Session	7.3	(0.5)	-0.6	(0.5)	0.321
Diary TST (h)					
1st Session	8.1	(0.5)	---		---
2nd Session	7.4	(0.5)	-0.7	(0.6)	0.247
3rd Session	8.1	(0.5)	0.02	(0.6)	0.969
Actigraphy SOL (min)					
1st Session	4.5	(1.8)	---		---
2nd Session	7.5	(1.8)	3.0	(2.0)	0.159
3rd Session	9.3	(1.8)	4.7	(2.0)	**0.031**
Diary SOL (min)					
1st Session	31.3	(10.4)	---		
2nd Session	34.3	(10.4)	3.0	(10.0)	0.767
3rd Session	23.8	(10.4)	-7.5	(10.0)	0.461
Actigraphy WASO (min)					
1st Session	42.5	(4.1)	---		---
2nd Session	44.1	(4.0)	1.7	(3.8)	0.664
3rd Session	36.6	(4.0)	-5.8	(3.8)	0.138
Diary WASO (min)					
1st Session	13.7	(7.9)	---		---
2nd Session	19.6	(7.9)	5.8	(10.1)	0.567
3rd Session	20.8	(7.9)	7.1	(10.1)	0.488

Significant values are bolded. Trending values are italicized. Abbreviations: B: beta coefficient; SE: standard error; SlpE: sleep efficiency; SOL: sleep onset latency; TIB: time in bed; TST: total sleep time; WASO: wake after sleep onset

5.0 DISCUSSION

5.1 SUMMARY OF FINDINGS

This study aimed to better understand the short-term impact of exercise on a comprehensive battery of sleep and daytime impairment measures in a sample who meet clinical criteria for insomnia. Previous research suggests that sustained exercise training may elicit meaningful improvements in objectively and subjectively measured sleep characteristics and moderate improvements in insomnia severity and indices of daytime impairment (i.e., anxiety, depression, insomnia severity). Meanwhile, the acute exercise literature suggests mild to moderate improvements in sleep characteristics assessed with polysomnography and actigraphy after aerobic exercise with limited concordant findings in subjectively assessed sleep quality or diary-assessed sleep. Additionally, the current acute literature lacks comprehensive assessment of changes in daytime impairment and lacks sampling of individuals who meet clinical criteria for insomnia disorder or account for the high night-to-night variability of sleep-in insomnia. To address these research gaps, we conducted a randomized controlled trial to evaluate the effects of short-term moderate-intensity aerobic walking exercise on measures of sleep, insomnia severity, and daytime impairment in a sample of adults who meet diagnostic criteria for insomnia.

We found no significant differences in treatment effect between participants who performed three days of walking exercise compared to participants who completed a quiet rest control experience. Both experimental groups experienced improvements in their insomnia severity and their global experience of daytime impairment from the baseline to the experimental

week, with inconsistent within-group improvements in actigraphy- or subjectively assessed sleep. In addition, exploratory analyses provided several observations, including: (1) significant associations between pre-sleep arousal and sleep characteristics; (2) significant improvements in multiple sleep parameters following exercise sessions compared to days without exercise; and (3) similar effects of exercise on sleep from the first to third walking exercise sessions. Overall, this study suggests that incorporating three walking exercise sessions within a week may not elicit immediate global improvements in sleep and daytime function in a sample of participants with insomnia. However, there were consistent day-level improvements in sleep after exercise sessions compared to non-experimental days which, in conjunction with the current acute literature, may support the hypothesis that incorporating walking exercise has acute benefits to sleep in those experiencing insomnia.

5.2 SPECIFIC AIM I: Actigraphy-assessed and Self-reported Sleep Variables

Aim I examined whether one week of moderate-intensity aerobic exercise improves objective and subjective sleep parameters compared to one week without exercise in a sample of adults who meet diagnostic criteria for insomnia. We hypothesized that three moderate-intensity walking exercise sessions within a week would improve sleep, particularly actigraphy-assessed sleep efficiency, compared to a week without exercise. Our results did not support this hypothesis, as there were no significant between-group differences in the change from baseline to experimental week in sleep efficiency or other sleep parameters that assess timing (bedtime, waketime, midpoint) or duration of sleep (total sleep time, time in bed).

Unlike the current acute exercise literature in samples with insomnia or insomnia symptoms, this study incorporated three experimental sessions within a 1-wk monitoring period to explore potential global changes in sleep in addition to any day-level effects (discussed later in Section 5.4). We are unaware of any other studies using a 1-week protocol in the context of acute exercise implementation in samples with insomnia; as a result, there are no directly comparable studies for aims I and II of this investigation. Yet, of the few studies that have investigated a single bout of aerobic exercise, most of these studies found improvements in objectively measured sleep (i.e., increased total sleep time and sleep efficiency, decreased wake after sleep onset and sleep onset latency) with a lack of concurrent change in subjectively assessed sleep.[22-24]

Most similar to the current study, Chen and colleagues enrolled a sample of inactive women who reported insomnia symptoms and randomized them to complete either one 30-minute bout of light-intensity (45-55% of age-predicted maximal heart rate) walking exercise or quietly read.[24] Sleep was measured using a wrist-worn actigraph for two days pre- and post-experimental session day. They found that post-experiment sleep efficiency increased by 3.8% following the walking exercise session and decreased by 1.3% in the reading group. Additionally, sleep onset latency decreased by 3.3 minutes after the walking exercise day while it increased by 1.6 minutes following the reading condition. Similarly, Passos and colleagues found polysomnography- and diary-assessed sleep onset latency and total wake time decreased and sleep efficiency and total sleep time increased after moderate-intensity aerobic exercise compared to a baseline night.[22] Lastly, Morita and colleagues used a repeated measures design to explore the impact of a single bout of step exercise on subsequent sleep in a sample of adults with insomnia symptoms and found a reduction in polysomnography-assessed arousals and wake stages after morning exercise.[23]

When exploring the entire week of sleep, the results from the current study corroborate some, but not most, of the results of these previous studies. In alignment with previous research, we found that actigraphy-assessed sleep onset latency decreased over time in the walking exercise condition but increased in the quiet rest condition; however, this interaction was only trending toward statistical significance. Regardless, results using sleep onset latency should be interpreted with caution as it is the least reliable sleep characteristic measured by actigraphy.[183] Contrary to this previous research, the current investigation found no significant within-group improvements in sleep efficiency or many other sleep characteristics for either experimental condition.

In corroboration with our week-level findings, additional studies in samples with insomnia or poor sleep have not found acute improvements in objectively or subjectively assessed sleep after one bout of aerobic exercise. Youngstedt and colleagues explored the impact of late-evening aerobic and resistance exercise in a sample who met diagnostic criteria for insomnia and found that a single bout of exercise did not improve sleep compared to a night of quiet rest.[96] Additionally, Brupbacher and colleagues conducted a randomized controlled trial to investigate whether a single bout of aerobic cycling exercise improved sleep in a sample of adults with depression.[184] No improvements in sleep were found after moderate-intensity cycling exercise compared to a quietly resting control group.[184] The results from these experimental studies, which utilized similar designs as the previous studies mentioned, suggest that an acute bout of exercise may not elicit changes in sleep.

Importantly, our methodology of incorporating three bouts of exercise within a week is fundamentally different than the current body of acute literature that has explored a single bout of exercise. Although acute exercise bouts have benefits to the subsequent night of sleep as seen in some of the previous literature, dispersing acute exercise bouts throughout the week may not

subsequently improve the experience of sleep across a short monitoring period in samples with insomnia as hypothesized. Further discussion of how the current acute literature compares to our day-level analyses is provided in Section 5.4.2.

5.3 SPECIFIC AIM II: Daytime Impairment and Sleep Quality

Aim II examined whether one week of moderate-intensity aerobic exercise reduces daytime impairment and insomnia severity compared to one week without exercise in a sample of adults who meet diagnostic criteria for insomnia. We hypothesized that three moderate-intensity walking exercise sessions within a week would reduce insomnia severity and sleep-related daytime impairment, as measured by the Insomnia Severity Index and PROMIS Sleep-Related Impairment Scale, respectively. Our results did not support this hypothesis; although we observed significant improvements in insomnia severity and daytime impairment for both experimental groups compared to baseline levels, no between-group differences were observed. Secondary analyses also explored whether there were improvements in pre-sleep arousal as measured by the Pre-Sleep Arousal Scale (i.e., total score, cognitive subscale, somatic subscale) and visual analog scales of feeling worried, anxious, tense, or having an overactive mind before bedtime. Again, while we observed significant within-group reductions in total and cognitive pre-sleep arousal for each experimental group compared to baseline, no between-group differences were found.

In two recently published systematic reviews, long term (> 3 weeks) exercise training was found to elicit moderate-sized improvements in insomnia severity and sleep quality in samples with insomnia or insomnia symptoms.[135,185] Yet, to our knowledge, this is the first evaluation of

the acute effect of moderate-intensity aerobic exercise on indices of daytime impairment and insomnia severity. Although minimal research has examined the impact of acute exercise on general daytime impairment in samples of adults with insomnia, several studies have examined mood and anxiety. Specifically, Passos and colleagues found that an acute bout of moderate-intensity aerobic exercise led to a significant reduction in pre-sleep anxiety in contrast to no change in the control, resistance exercise, or high-intensity aerobic exercise groups.[22] Additionally, Youngstedt and collegues found that although evening exercise and quietly reading both reduced pre-sleep anxiety, the change in pre-sleep anxiety following exercise was significantly correlated with that night's total sleep time and wake after sleep onset while the change after quietly reading was not.[96]

Our results partially corroborate these findings as our week-level analyses and day-level exploratory analyses (described in Section 5.4) found significant within-group (i.e., walking exercise and quiet rest) improvements in pre-sleep arousal, and feeling anxious prior to bedtime, respectively. Our lack of between-condition differences compared to Pasoss and collegues may be due to a few reasons. First, the Pre-Sleep Arousal Scale used in the current study has somatic and cognitive domains that do not specifically assess "anxiety" as a construct; in contrast, the studies mentioned above used the Spielberger State-Trait Anxiety Inventory to evaluate the state change in pre-sleep anxiety after exercise. This explaination is corroborated by findings from a recent randomized controlled trial from Brupbacher and colleagues where they found no changes in cognitive or somatic pre-sleep arousal following acute moderate-intensity cycling exercise as measured by the Pre-sleep Arousal Scale.[186] This is interesting as their sample also had unipolar depression and were thus hypothesized to experience improvements to pre-sleep arousal due to acute exercise.[186] This highlights that the impact of acute exercise on pre-sleep mental health may

be more intricate and construct specific as previously speculated. Additionally, the different effects on mood within the study from Passos and collegues could also be partially attributed to their exercise prescription being of a greater intensity.[22] Passos and colleagues had the advantage of conducting a graded maximal exercise test with respiratory gas measurement to establish exercise intensity; for their study, moderate-intensity aerobic exercise was operationalized as the treadmill speed and incline that elicited the first ventilatory threshold.[22] This is a more rigorous method of identifying moderate-intensity aerobic exercise compared to the estimation technique used in the current study; as a result, participants in the current study may have exercised at a lower intensity than the participants in the study by Passos and colleagues.[157,187] Although the evidence regarding the importance of exercise intensity on mood is not conclusive, recent reviews found that higher-intensity exercise interventions resulted in greater reductions in anxiety than lower-intensity exercise.[188,189] Lastly, the quiet rest control group in the current study may have experienced an increase in relaxation during their session which may have contributed to the reduction in pre-sleep arousal,[190] which mirrors the reduction in pre-sleep anxiety found in the 'quiet reading' control night in the study from Youngstedt and colleagues.[96]

This study highlights the need to comprehensively assess daytime impairment in studies involving participants with insomnia disorder in future research. We assessed insomnia severity and a measure of daytime impairment other than mood, but additional measures could be useful in future investigations. Primarily, day-level or weekly-level assessment of fatigue and alertness may be useful. Measures such as the Daily Fatigue Impact Scale may show day-level changes that have otherwise gone overlooked in the acute and chronic literature involving exercise and insomnia.[191] Additionally, alertness measures such as the Karolinska Sleepiness Scale[192] could be useful at the daily level as reductions in alertness during the day may actually support acute reduction in

hyperarousal, similar to how Hartescu and colleagues concluded that chronic exercise has a dampening effect on hyperarousal in their sample with insomnia disorder.[136,193,194]

In summary, although we observed significant improvements in insomnia severity and daytime impairment for both experimental groups compared to baseline levels, no between-group differences were observed. Our results do not suggest that incorporating three days of moderate-intensity walking exercise within a week improves insomnia severity or sleep-related impairment compared to quietly resting alone. Yet, the within-subject effects warrant future comprehensive consideration of measures of impairment in acute exercise studies in samples with insomnia disorder.

5.4 EXPLORATORY AIMS

This study also investigated three distinct exploratory aims related to the daily associations between experimental sessions and subsequent sleep characteristics: 1) Exploratory Aim I explored whether pre-sleep arousal following experimental sessions was associated with the subsequent night's objective and subjective sleep and whether the association was different between experimental conditions; 2) Exploratory Aim 2 explored whether sleep differed on days where there was an experimental session in comparison to a non-experimental session day; 3) Exploratory Aim 3 explored whether the effect of exercise on the subsequent night's sleep differed from the first to the third exercise session in those allocated to the exercise condition.

5.4.1 Pre-Sleep Arousal and Sleep Characteristics Between Experimental Conditions

We found that greater pre-sleep arousal was related to significantly worse diary- and actigraphy-assessed sleep efficiency, greater diary-assessed sleep onset latency and wake after sleep onset, and lower diary-assessed sleep quality and feeling rested in the morning. It has been established that people with insomnia have higher self-reported pre-sleep arousal than their healthy-sleeping counterparts.[195,196] However, less research has directly linked pre-sleep arousal measures to subsequent sleep characteristics in samples with insomnia. Our results corroborate those from a study by Wicklow and colleagues, who found that greater cognitive pre-sleep arousal was related to significantly lower diary-assessed sleep efficiency and greater sleep onset latency.[197] Although not explicitly assessing the construct of arousal, Youngstedt and colleagues found that improvements in pre-sleep anxiety were related to sleep after late-night exercise, but not after quietly resting;[96] in addition, a similar study by Passos and colleagues found that improvements in pre-sleep anxiety were not related to changes in sleep after aerobic exercise.[22] In contrast to Youngstedt and colleagues, we found that greater pre-sleep arousal was related to greater wake after sleep onset, but only in those who participated in a quiet rest session that day. Our finding may suggest that the beneficial relationship between acute exercise and sleep may not be explained by a reduction in pre-sleep arousal as a biological mechanism. Although speculative, acute exercise may increase homeostatic sleep drive through other mechanisms such as increased peripheral circulatory cytokines, adenosine, or skeletal muscle expression of circadian clock genes.[111] These mechanisms may blunt or override any impact of pre-sleep arousal on subsequent sleep.

5.4.2 Sleep on Experimental Days vs. Non-Experimental Days

For our second exploratory aim, we aimed to explore whether sleep differed following days in which experimental sessions occurred compared to non-experimental days and whether these differences were unique to the exercise condition. Bedtime (diary- and actigraphy-assessed) and sleep midpoint were both significantly later following experimental days compared to other non-experimental days, but these changes did not differ by experimental condition. However, total sleep time and self-reported morning restedness demonstrated differences between experimental and non-experimental days that varied by experimental condition. Specifically, diary- and actigraphy- assessed total sleep time was approximately 30 min longer on nights following walking exercise sessions compared to non-experimental days, while total sleep time was shorter following days when quiet rest sessions occurred compared to other days. Participants also felt more rested after nights following walking exercise compared to non-exercise days in the exercise condition, while morning restedness was stable between experimental and other days in the quiet rest condition. Although only trending toward statistical significance, time in bed and sleep quality followed a similar pattern as total sleep time and restedness.

To compare similar literature, a recent systematic review and meta-analysis compiled studies that evaluated the day-to-day associations between physical activity and sleep using experimental and observational data.[198] The results between individual studies were mixed with positive, negative, and null relationships between physical activity and subsequent sleep, but the meta-analyses across sleep variables (i.e., sleep efficiency, sleep onset latency, sleep quality, wake after sleep onset) found that only high total physical activity was associated with low total sleep time the following night within individuals with a small effect size.[198] The meta-analyses contained

studies with diverse methodological approaches regarding measures of sleep and physical activity, study designs, and analytic plans. Additionally, most of the studies included in the review did not examine samples with poor sleep (e.g., insomnia). The focus on good sleepers in these studies may partially explain the discrepancy between the review's results and the current study.

Similar to the current study, Baron and colleagues explored the daily bi-directional relationship between exercise duration and sleep using data from a randomized controlled intervention of 11 older adult women with insomnia.[25] Baron and colleagues fit a hierarchical linear regression in which they explored whether increases in exercise duration from the individual mean was related to better or worse sleep the subsequent night. They found that exercise duration was not related to actigraphy-assessed sleep onset latency, total sleep time, wake after sleep onset, sleep efficiency, or subjective ratings of sleep quality. These results suggested that daily deviations in exercise duration during a chronic exercise intervention were not related to subsequent deviations in sleep. These results do not coincide with the current investigation; several potential reason may explain this discrepancy. First, the way in which Baron and colleagues conceptualized the analyses was different from the current investigation. Their data utilized mean-centered variables of self-reported exercise duration and actigraphy-assessed sleep across the 16-week exercise intervention, while our predictor was whether an experimental session was performed across a week-long time frame. This is an important difference, as our observable acute day-level effects may not concur with findings from Baron and colleagues due to being "washed out" by inclusion of weeks after the acute effects of exercise become more chronic. This is plausible as the 16-week intervention they sampled from resulted in significantly better sleep quality at post-intervention.[17]

In contrast to Baron and colleagues, our day-level analyses coincide with much of the single-bout literature in samples with insomnia and insomnia symptoms. As previously mentioned in Section 5.2, the majority of acute exercise studies have found improvements in objectively measured sleep (e.g., increased total sleep time and sleep efficiency, decreased wake after sleep onset and sleep onset latency) with a lack of concurrent change in subjectively assessed sleep.[22-24] Similar to our current study, Passos and colleagues was the only other study with a parallel design that found improvements in total sleep time the night following exercise; however, they did not assess subjective sleep quality.[22] These findings from the current investigation add to the evidence that various sleep characteristics such as total sleep time and sleep quality may improve on nights following exercise in the acute time frame in samples with insomnia or insomnia symptoms.

Importantly, these day-level findings across acute experimental studies are found despite somewhat incongruent methodological considerations. Particularly, the current study sampled both men and women while others only sampled only female participants.[24,25] A meta-analysis by Kredlow and colleagues found that sex did not significantly moderate the beneficial effect of acute exercise on most sleep parameters across all studies, yet some individual studies found the effect of exercise was blunted in women compared to men.[14] Additionally, secondary analyses of a 4-week randomized trial by Bisson and colleagues found that the relationship between physical activity and sleep quality was stronger in females than males.[199] These mixed findings suggest that targeted research on sex differences is warranted in samples with insomnia. Additionally, the previous studies included samples of an older age than the current investigation. Chen and colleagues specifically sampled older adults while the current study's sample consisted predominantly of younger adults.[24] Many chronic and acute exercise studies have sampled older adults and found beneficial effects of exercise on sleep typically resulting in larger effect sizes

than younger samples.[200] Older adults often have worse sleep than their younger counterparts[201] and, as a result, have greater room for improvement after exercise.[200] Although the current study also found acute improvements in sleep, studies utilizing older adults may be more likely to observe improvements in sleep following exercise at a lighter intensity than the current study. Finally, the current sample met self-reported and interview assessed diagnostic criteria for insomnia, but were better sleepers than most of the studies focused on acute exercise and sleep.[22,24,96] Compared to the other study samples, this may contribute to our lack of consistent improvements in other indices of sleep such as sleep efficiency or sleep onset latency.

In summary, the growing amount of research devoted to acute exercise and insomnia suggests that acute morning moderate-intensity exercise may confer mild to moderate benefits to sleep on the night following a bout of exercise. Yet, in conjunction with the week-level results of our study, the acute literature may now more strongly suggest that the effects of acute exercise do not seem to result in noticeable improvements in sleep within the context of the short duration of a week any more than performing a consistent quiet rest period.

5.4.3 Sleep Across the Walking Exercise Sessions

As mentioned previously, there is evidence to suggest that the impact of exercise on sleep may accumulate over time, demonstrated by the small effect sizes in the acute literature compared to larger and broader effects after chronic exercise training.[135] Thus, we aimed to explore whether there was an additive effect of exercise bouts on sleep in the context of our acute study. In summary, we found that sleep did not differ across the three exercise sessions. To our knowledge, the potential compounding effect of multiple exercise bouts on sleep or sleep-related outcomes has

not been experimentally explored. In the meta-analyses by Kredlow and colleagues, the number of exercise bouts per week during long-term exercise interventions did not moderate the relationship between exercise and sleep.[14] Other physiological outcomes (e.g., glucose tolerance, oxidative stress, hormone changes) have shown different post-exercise patterns based on the number of exercise bouts, so it is plausible that there may be an identifiable time-course or compounding improvement in sleep outcomes after multiple exercise sessions.[202-204] Although we found no preliminary evidence of an additive improvement in sleep during acute implementation of exercise, when considering the conclusions from the day-level analyses in Section 5.4.2, future studies are warranted to better understand at what timepoint acute exercise effects may become chronic effects (i.e., better sleep even on non-exercising days) or more explicit investigations into the trajectory of sleep improvement across long-term exercise interventions (i.e., comparing the acute effects of exercise across each experimental week of an intervention). Future observational and experimental studies would benefit from a more detailed exploration of the time course of sleep improvement due to exercise; this may be especially relevant to explore in samples with insomnia disorder, who would benefit from better understanding how exercise participation may impact their sleep experience.

5.5 STRENGTHS AND LIMITATIONS

Compared to the current literature on the impact of acute exercise on sleep, this study has high ecological validity. Including non-supervised outdoor walking exercise allowed us to examine in a more realistic scenario how inactive people with insomnia might increase their physical activity

and attempt to improve their sleep. Outdoor activities were recently named the fourth most popular fitness trend in America for 2021 and, because walking exercise is a free and relatively easy activity to adopt, our investigation is timely and clinically digestible.[205] Although we could not monitor each session in person, we assessed heart rate to gauge exercise intensity, provided one-on-one explanation of ratings of perceived exertion and their heart rate prescription, and required session logs to verify adherence. In addition to the ecological validity of the exercise condition, we also included a control group which allowed for rigorous comparison of exercise to a typical behavior (quietly resting). Our study design included multiple days of sleep monitoring, which allowed for exploration of week-level changes in sleep parameters as well as exploration into day-level associations between physical activity and sleep. As people with insomnia report greater night-to-night variability in their sleep, including analyses of the weekly change in sleep acknowledges this variability and provides clinically relevant results.[26,30,138] Our study also utilized actigraphy, which is the recommended method for objectively characterizing sleep in people with insomnia instead of polysomnography.[12,26,30,138] Due to the week-long assessment periods used in this study, we were also able to assess potential exercise-induced changes in insomnia severity and subsequent daytime impairment which, to our knowledge, has not been well examined to date.

This study also has several limitations. Although external validity was high, the variability in exercise environment and lack of laboratory control over other behaviors and environment throughout the experimental week negatively influenced the study's internal validity. Our method of estimating the appropriate heart rate range for moderate-intensity exercise is commonly utilized in clinical and research settings; however, we were unable to perform a graded maximal exercise test, which is the most accurate way to identify the heart rate range associated with moderate-intensity exercise. Additionally, although we used validated screening tools for

other common sleep disorders, we did not utilize polysomnography or limited-channel home sleep testing devices to objectively confirm that the participants were free from apnea or periodic limb movements. Adults with insomnia and comorbid sleep disorders may have a different biological response to exercise compared to an individual with only insomnia. For example, if participants were to have co-morbid obstructive sleep apnea, exercise may have improved their sleep via symptom reduction that was not assessed and different biological mechanisms that are specific to those disorders could not be generalized to insomnia disorder (e.g., rostral-fluid shift).[28,101,206]

Additionally, this study did not utilize eligibility criteria based on diary- or actigraphy-assessed sleep measures after the baseline assessment, which has been utilized to confirm insomnia in other studies.[17,207-209] Common measures that are confirmed with diary or actigraphy are > 30 minutes wake after sleep onset or sleep onset latency and/or < 85% sleep efficiency on three or more nights. Although we lacked an objective screening cutoff, participants met DSM-5 diagnostic criteria for insomnia disorder and reported at least mild insomnia severity based upon the Insomnia Severity Index. Additionally, our sample presented with self-reported hyperarousal and sleep reactivity that were similar to other samples with insomnia.[148,181] Yet, the actigraphy- and diary-assessed sleep of our sample were more similar to estimates observed in normal sleeping samples[201] than adults with insomnia. Although Insomnia Severity Index scores were relatively high, the generally mild sleep disturbance observed with the diary and actigraphy data in this sample may have caused ceiling effects and reduced the likelihood that exercise could further improve sleep in this sample, particularly for our primary outcome of sleep efficiency.[210]

When summarizing the week-level findings between the experimental conditions, the parallel results in the exercise and quiet rest condition may suggest a potential placebo effect. A meta-analysis and systematic review from Yeung and colleagues compiled randomized controlled

trials using pharmacological or behavioral interventions that used a full deception placebo group and a no-treatment group to calculate the placebo effect size on insomnia symptoms.[211] They found statistically significant placebo effects on sleep onset latency, total sleep time, and global sleep quality. This is relevant to the current study, as the quiet rest condition was implemented as a control for the walking exercise condition, and we established equal expectations for a treatment effect across groups. Although we did not have a no-treatment group to explicitly test this effect, there may have been a placebo effect in the quiet rest group that may account for the improvements in sleep and insomnia severity. Yeung and colleagues mention that these placebo effects may mask an active treatment effect because any placebo effect within our walking exercise group may not have been additive to the treatment effects. If possible, future acute and chronic exercise studies may benefit from a no-treatment group to help uncover these potential effects in insomnia as they are present across pharmacological and behavioral interventions.[211]

Although our study analyzed whether other daytime behaviors (e.g., meal timing, caffeine consumption) differed between baseline and experimental weeks within and between experimental groups, we could not control potential changes in behavior during the study; this, too, may have contributed to the null results. This study referenced various biological mechanisms which may explain how acute exercise may benefit sleep, yet the design of the study did not allow for exploration into testing these mechanistic pathways. Importantly, we powered the study for the primary outcome of actigraphy-assessed sleep efficiency based on estimates from available literature, we performed analyses across many sleep characteristics and were unable to power the study across other measures; this may have contributed to null results (i.e., interaction effects).

5.6 CONCLUSIONS AND FUTURE DIRECTIONS

In conclusion, the results of the current investigation suggest that inactive adults with insomnia may not experience noticeable improvements in week-level sleep and daytime function after adopting three days of walking exercise compared to participating in three quiet rest sessions but exercising may lead to improved sleep the night after exercising.

Although we investigated the impact of acute exercise on sleep and daytime impairment in a sample of adults with insomnia, future research remains needed to advance our understanding of this association. Particularly, this study joins only a handful of published experimental studies that have explored the acute impact of exercise on sleep in adults with insomnia or insomnia symptoms; notably, it is the only investigation to explore the acute impact of exercise on common measures of daytime impairment other than anxiety. These types of investigations are clinically important and impactful as they can be leveraged to explore biological correlates and mechanisms in which exercise may improve sleep in insomnia and elucidate the time course of treatment effects due to exercise. The gold-standard treatment for insomnia, CBT-I, takes time to elicit improvements in insomnia and due to its breadth of research that information can be effectively and sufficiently conveyed to patients. The same needs to be evident for exercise participation so clinicians can communicate the utility of exercise and how to behaviorally integrate it into treatment.

If more acute exercise studies are conducted in samples with insomnia, they should include global, but most importantly *daily* indices of impairment that may be clinically meaningful (e.g., fatigue, physical function, cognitive performance, alertness). Additionally, exercise studies should explore the biological mechanisms that may explain any acute and chronic benefit of exercise, as most hypothesized mechanisms remain speculative and without experimental interrogation. Such

research would provide clarity into how exercise improves sleep in those with insomnia and potentially translate into its inclusion as an alternative or adjunct treatment option for insomnia.

Although the current study suggests moderate-intensity walking exercise in the morning elicits acute day-level improvements in some sleep characteristics, the current literature lacks experimental investigations that identify the optimal exercise prescription (i.e., timing, duration, and type, and intensity) for people with insomnia disorder. This is unfortunate, as unsubstantiated statements regarding the optimal exercise type, timing, and/or duration for insomnia often appear in the lay literature.[212] For instance, very few studies have directly evaluated the impact of exercise timing on sleep in adults with insomnia;[20,23] the existing evidence provides no consensus regarding optimal exercise timing. Moreover, although studies have evaluated common types of exercise such as resistance training, aerobic exercise, yoga, and tai chi, [15,135] minimal well-powered randomized controlled trials have compared exercise types in samples with insomnia or insomnia symptoms.[22] In general, future studies should directly compare exercise prescription components and their effects on sleep in well-defined samples with insomnia disorder. The experience of insomnia, although standardized for research purposes, can vary greatly between individuals; as a result, future investigations into the demographic, psychosocial, and physiological characteristics that differentiate 'responders' from 'non-responders' regarding exercise and insomnia would also enhance our understanding of who will benefit from exercise and what exercise prescriptions may be of the most benefit. The long-term goal is to understand how exercise can be used safely and effectively as a stand-alone treatment, or more appropriately, in conjunction with other methods of insomnia treatment such as CBT-I or pharmacology.

Appendix A: Recruitment Items

A.1 Prior Participant Email

STUDY19050174 PRIOR PARTICIPANT EMAIL

To be sent from email account associated with STUDY19050174 (sleepheartstudy@pitt.edu):

Dear [insert name],

You previously expressed interest in participating in the Insomnia and Cardiovascular Health Study. Because of this, I wanted to let you know about a new study that may be of interest to you!

The new study examines whether participating in 1 week of aerobic exercise compared to not participating in exercise has beneficial effects on sleep and daytime consequences in people with insomnia. The study lasts about 3 weeks, is conducted remotely, and includes:

- Questionnaires for you to complete (sent over email)
- 1 video conference interview about your sleep
- 1 home visit (following COVID-19 safety guidelines) to assess your height, weight, waist and neck girth, and resting heart rate
- A 7-day home baseline assessment of your sleep and physical activity;
- An additional 7-day sleep assessment period in which you will be randomly assigned to participate in either a walking exercise group or a quiet rest group. During this assessment period you will either complete 3 sessions of walking exercise at home or 3 quiet rest sessions at home monitored over video conferencing.

More information about this study can be found at this link: https://pitt.co1.qualtrics.com/jfe/form/SV_5nnmSHOi9fUid9P. At that site, you can also complete a screening questionnaire to see if you'd be a good fit for the study.

If you are interested in learning more about the study, feel free to reply to this email or contact Andrew Kubala, the study's coordinator, at 724-590-2299 or AndrewKubala@pitt.edu.

Thank you again for your interest in our research!

Best regards,

Chris

Christopher E. Kline, PhD
Primary Investigator, Insomnia and Cardiovascular Health Study
Department of Health and Human Development
University of Pittsburgh
Phone: 412-383-4031
E-mail: sleepheartstudy@pitt.edu

A.2 Read Green Email

READ GREEN

Email Subject Line: Insomni-Ex Study

Email Description:

Do you suffer from insomnia and are currently inactive??

Researchers at the University of Pittsburgh are conducting a study to explore whether 1 week of aerobic exercise benefits sleep and improves how one feels during the day compared to not participating in exercise in people with insomnia.

We think that exercise may help people with insomnia, but we do not know whether these effects are noticeable soon after someone with insomnia begins an exercise program.

This study is recruiting participants with insomnia who:

- Are 18 and 55 years of age
- Have trouble falling asleep or staying asleep regularly
- Are not currently physically active
- Have no physical health or untreated mental health problems

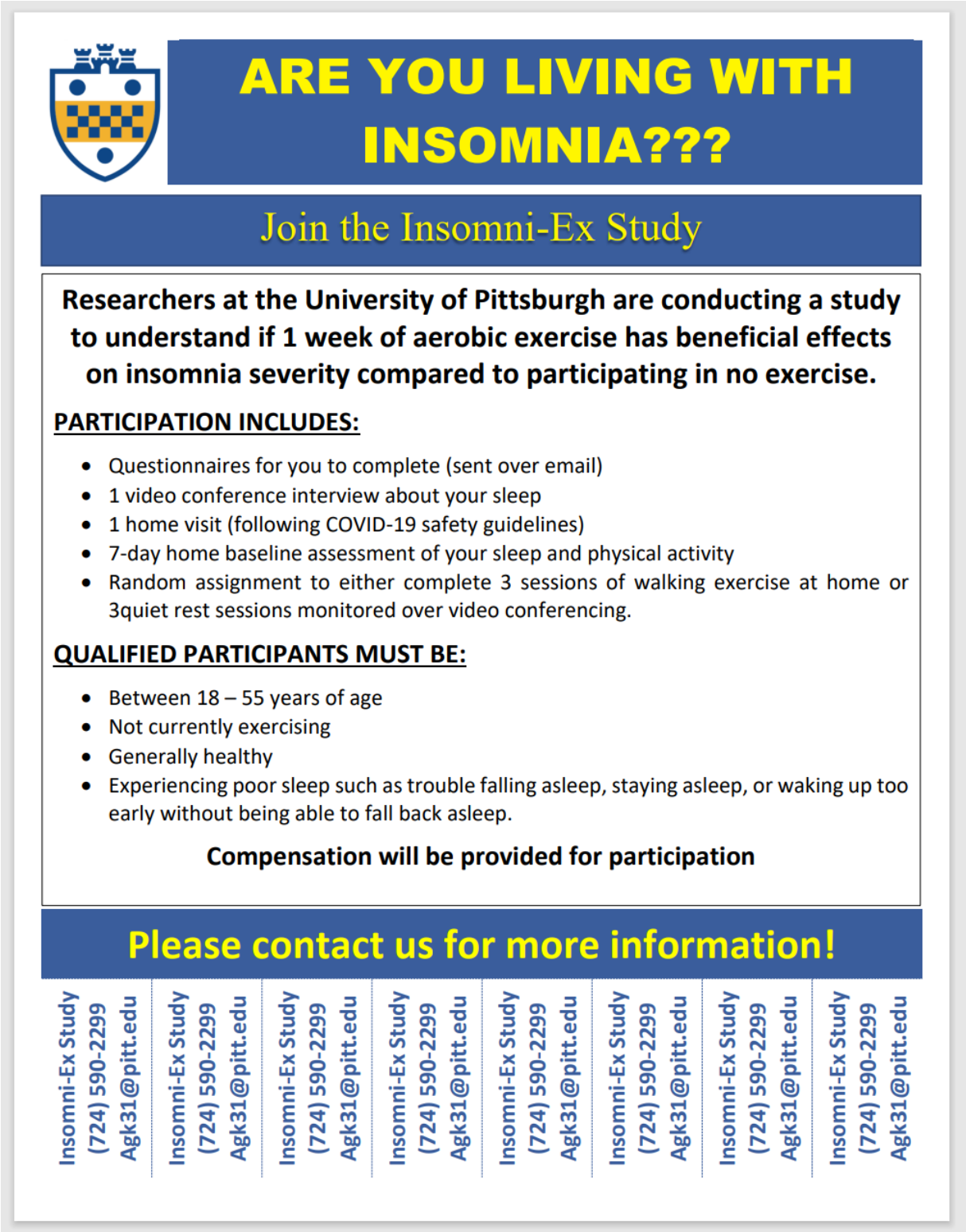
The study lasts about 3 weeks, is conducted remotely, and includes:

- Questionnaires for you to complete (sent over email)
- 1 video conference interview about your sleep
- 1 home visit (following COVID-19 safety guidelines) to assess your height, weight, waist and neck girth, and resting heart rate
- A 7-day home baseline assessment of your sleep and physical activity;
- An additional 7-day sleep assessment period in which you will be randomly assigned to participate in either a walking exercise group or a quiet rest group. During this assessment period, you will either complete 3 walking exercise sessions at home or 3 quiet rest sessions at home monitored over video conferencing.

Interested in learning more about the study? Contact us at 724-590-2299 or email AndrewKubala@pitt.edu

From Address Display Name:
Read Green from the Insomni-Ex Study

A.3 Recruitment Flyer



ARE YOU LIVING WITH INSOMNIA???

Join the Insomni-Ex Study

Researchers at the University of Pittsburgh are conducting a study to understand if 1 week of aerobic exercise has beneficial effects on insomnia severity compared to participating in no exercise.

PARTICIPATION INCLUDES:

- Questionnaires for you to complete (sent over email)
- 1 video conference interview about your sleep
- 1 home visit (following COVID-19 safety guidelines)
- 7-day home baseline assessment of your sleep and physical activity
- Random assignment to either complete 3 sessions of walking exercise at home or 3quiet rest sessions monitored over video conferencing.

QUALIFIED PARTICIPANTS MUST BE:

- Between 18 – 55 years of age
- Not currently exercising
- Generally healthy
- Experiencing poor sleep such as trouble falling asleep, staying asleep, or waking up too early without being able to fall back asleep.

Compensation will be provided for participation

Please contact us for more information!

Insomni-Ex Study (724) 590-2299 Agk31@pitt.edu

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Appendix B: Phone Screen

Insomni-Ex Study

PHONE SCREENING

Hi, my name is [*insert name*], and I am calling on behalf of the Insomni-Ex Study. You previously contacted us to learn more about the study. Do you have a few minutes for me to describe it to you?

If YES, continue with the phone script.
If NO, ask them when would be a better time to reach them: _____
Date Time

We are conducting this study to explore whether short-term participation in 1 week of aerobic exercise benefits sleep and reduces daytime consequences of insomnia compared to not participating in exercise. We think that exercise may help people with insomnia, but we do not know whether there are immediate benefits to sleep after short-term exercise participation. We are inviting you to participate in this study because you have poor sleep and are physically able to participate in exercise but are currently not active. To be eligible to participate, you need to be between 18 and 55 years of age and not have any physical heath or untreated mental health problems that would prevent you from participating. We will enroll 24 adults for this study.

Participation in this research study involves the following four steps:

1. You will complete a screening questionnaire that should take about 15 minutes to complete. You can complete this over the phone or we can give you the website address to answer these questions on the computer. We will ask you about your background, your physical and emotional health, and your sleep.

2. We will the send you the consent document over email and schedule a phone call to discuss the study. We will then send you an online version of the consent document to provide your consent to participate. After your consent is obtained, we will schedule a video call that lasts about 45 minutes and send you two online surveys to complete prior to the video call. During this call, we will ask you questions about your sleep and schedule an in-person visit.

3. Using COVID-19 safety procedures, a research technician will arrive at your home to obtain your height, weight, waist and neck girth, as well as your resting heart rate. We will then provide you with a diary as well as a wrist and hip monitor to wear for 7 days before you begin the experimental condition.

4. After you wear the monitors and complete the diary you will be randomly assigned to an experimental condition that lasts for 7 days. The two experimental conditions are walking exercise or a quiet-rest condition. You will complete 3 experimental sessions separated by at least 24 hours each. Depending on the condition you were randomized into, the sessions will consist of either 40 minutes of walking exercise on your own between 7:00am – 12:00pm or 40 minutes of a quiet rest monitored through a video call. During these 7 days, you will complete a diary again and wear monitors on your wrist and hip.

The total time commitment for participating in this study is about 3 weeks. Compensation is $20 for completing step 2 and 3, and $100 for completing step 4, totaling $120 if the participant completes all steps of the study.

I've just given you a lot of information—did you have any questions?

If you would like to think more about whether this is something you'd like to participate in, you can call me back at a later date. You can also complete an online screening questionnaire to

determine whether you may be a good fit for the study, or we can conduct the screening questionnaire over the phone right now—it would take about 15 minutes.

Would you like to complete the phone interview today? YES NO

If YES, continue. If NO, thank him/her for his/her time and encourage them to visit the website and call back if they have any questions or would like to complete the phone interview at a different time.

The first step toward participating in the study is determining whether you may be eligible. To see whether you are eligible, we will ask you questions about your background, your physical and emotional health, and your sleep. It will take approximately 15 minutes to complete these questions.

Your participation in this phone screen is voluntary. It is possible that some of the questions may make you uncomfortable or distressed; if so, you may refuse to answer any of the questions asked. There is also some risk of a breach of confidentiality in providing this screening information. This risk is greatly minimized by software that encrypts the data and having the information stored on secure servers.

Your responses to these questions are confidential. Any information you provide will be kept indefinitely for the purpose of this study, but your identity will never be revealed.

I will let you know at the end of the interview whether or not your answers tell me that you are likely a good fit for the study. If it seems like you may be a good fit, I will ask you for your contact information so we can set up a visit to our facility.

Before we begin, do I have your permission to ask you these questions? YES NO

Record the caller's consent at the top of the next page, and sign your name.

Phone Screen Interview

The caller gives verbal permission to conduct the Phone Screen: YES NO

Verbal Consent was given to: _____
Research Staff Member Signature

Date: _____ *Time:* _____

1. How did you hear about our research study? *(Open-ended; Select all that apply.)*
   - Participant Registry
   - Brochure/flyer
   - Email
   - Other (please specify:_____

2. What is your age? _____ *Check if < 18 or > 55* ☐

3. What is your gender? MALE FEMALE

4. What is your height? _____ ft & in

5. What is your current weight? _____ lb

INTERVIEWER: Calculate their BMI by using the online BMI calculator (www.nhlbi.gov/health/educational/lose_wt/BMI/bmicalc.htm):

BMI:_____ *Check if BMI ≥ 30* ☐

6. Are you currently pregnant or plan to become pregnant in the next three months? YES NO *Check if YES.* ☐

7. Has a doctor ever said that you have a heart condition? YES NO *Check if YES.* ☐

8. Do you have a chronic medical condition that would make it unsafe to exercise (for example: uncontrolled diabetes, uncontrolled high blood pressure)?

YES NO

INTERVIEWER: If the participant says YES to Q8 ask:

Would you please describe the medical condition?

_____
_____

9.In the last 6 months, have you been diagnosed with depression or anxiety ?

YES NO

INTERVIEWER: If the participant says YES to Q9 ask:

Are you currently being treated for depression and anxiety?

_____

_____

10. In the last 6 months, have you been diagnosed with any other mental health problem (for example: post-traumatic stress disorder, bipolar disorder, substance use disorder)?

YES NO

INTERVIEWER: If the participant says YES to Q10 ask:

Would you please describe the mental health problem?

_____

_____

11. What time is your usual bedtime (for example: 10:15 pm):_____ *Check if before 9:00 pm or after 2:00 am.* ☐

12. What time do you usually get out of bed for good in the morning? _____ *Check if before 5:00 am or after 10:00 am.* ☐

These next seven questions refer to problems with your CURRENT sleep, which you can think of **as the last 2 weeks**.

13. How would you rate the severity of your DIFFICULTY FALLING ASLEEP?
    - None 0
    - Mild 1
    - Moderate 2
    - Severe 3
    - Very Severe 4

14. How would you rate the severity of your DIFFICULTY STAYING ASLEEP?
    - None 0
    - Mild 1
    - Moderate 2
    - Severe 3
    - Very Severe 4

15. How would you rate the severity of your PROBLEMS WAKING UP TOO EARLY?
    - None 0
    - Mild 1
    - Moderate 2
    - Severe 3
    - Very Severe 4

16. How SATISFIED or DISSATISFIED are you with your CURRENT sleep pattern?
    - Very Satisfied 0
    - Satisfied 1
    - Moderately Satisfied 2
    - Dissatisfied 3
    - Very Dissatisfied 4

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

Not At All Noticeable	0
A Little	1
Somewhat	2
Much	3
Very Much Noticeable	4

18. How WORRIED or DISTRESSED are you about your current sleep problem?

Not At All Worried	0
A Little	1
Somewhat	2
Much	3
Very Much Worried	4

19. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (for example, daytime fatigue, mood, ability to function at work or with daily chores, concentration, memory, etc.) CURRENTLY?

Not At All Interfering	0
A Little	1
Somewhat	2
Much	3
Very Much Interfering	4

INTERVIEWER: Add up the scores associated with the responses to questions 13 through 19. The minimum score is 0 and the maximum score is 28. The score needs to be at least 10.

Check if < 10. ☐

20. Are you receiving any treatment for your sleep problem? YES NO *Check if 'Yes'.* ☐

INTERVIEWER: If the participant says YES to Q20 ask:

Would you please describe the treatment?

_____

_____

21. Have you been diagnosed with or are you being treated for sleep apnea? YES NO *Check if 'Yes'.* ☐

22. Do you SNORE LOUDLY, as in loud enough to be heard through closed doors, or your bedpartner elbows you for snoring at night? YES NO

23. Do you often feel TIRED, FATIGUED, or SLEEPY during the daytime, such as falling asleep during driving? YES NO

24. Has anyone OBSERVED you stop BREATHING or CHOKING or GASPING during your sleep? YES NO

25. Do you have or are being treated for High Blood Pressure ? YES NO

26. Age older than 50? YES NO

27. *Ask if Male:* Is your shirt collar 17 inches / 43cm or larger? YES NO UNKNOWN

Ask if Female: Is your shirt collar 16 inches / 41cm or larger? YES NO UNKNOWN

INTERVIEWER: Add up the NUMBER OF YES responses from questions 22 through 27. The minimum score is 0 and the maximum score is 6.

Check if:
> 5;
≥2 (Q:22-27) + Male;
≥2 (Q:22-27) + BMI > 35;
≥2 (Q:22-27) + Neck Circumference (YES).

28. Do you currently work at your job at any time between midnight and 6 am—in other words, night shifts? YES NO

Check if 'Yes'.

29. In the past 3 months, how many times per week did you usually do 20 minutes or more of vigorous-intensity physical activity that makes you sweat, puff or pant (for example: fast bicycling, sports, jogging, aerobics, heavy lifting)?

None

1 to 2 times per week

3 or more times per week

30. In the past 3 months, how many times per week did you usually do 30 minutes or more of moderate-intensity physical activity or walking that increases your heart rate or makes you breathe harder than normal? (for example: bicycling at a regular pace, doubles tennis)?

None

1 to 2 times per week

3-4 times per week

5 or more times per week

Check if Q27 or Q28 >2.

31. Are you willing and able to quietly rest at home or complete walks outside? You would need to complete the sessions at a consistent time that you choose between the hours of 7:00am-12:00pm on a Monday, Wednesday, and Friday.

Yes

No

Check if NO

32. Do you have access to the internet and materials (e.g., phone or computer webcam) to complete free video calls?

Yes

No

Check if NO ☐

END OF INTERVIEW

ELIGIBILITY UNCLEAR

If you need time to ask a question or are not sure whether the individual is eligible, end the call by saying:

Thank you for your time in answering these questions. We will have the person that reviews these questions get back with you as soon as possible. In the meantime, if you have any questions, do not hesitate to call us.

INELIGIBLE

If participant is not eligible based upon his or her responses, say the following:

Thank you very much for your information. I am sorry to say that you do not seem like a good fit for the study. In the future, though, we may have a study that better suits you—would you like us to collect your contact information and contact you in the future if a suitable study comes around?

Collect their contact information for future studies? YES NO

*If YES, collect contact information on next page. If NO, **do not** collect contact information.*

ELIGIBLE

If participant seems like he or she may be eligible, say the following:

Thank you for your time in answering these questions. Based on your responses, it appears that you may be a good fit for the study. The next step involves setting up a meeting with study staff to review the details of the study and to further evaluate your eligibility. I just need to obtain your contact information so our study coordinator can set up this appointment.

INTERVIEWER: Collect contact information on next page.

Appendix C: GAD-7 & PHQ-9

C.1 GAD-7

INSOMNI-EX
GAD-7

Participant ID: _____ _____
Date : _____ / _____ / _____
Research Staff:_____

INSTRUCTIONS: ***For each question, please CIRCLE the number that best describes your answer.***

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

	Not at all	Several days	More than half the days	Nearly every day
1. Feeling nervous, anxious or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Feeling bad about yourself — or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3

C.2 PHQ-9

INSOMNI-EX
PHQ-9

Participant ID: ____ _____
Date : _____ / _____ / _____
Research Staff:_____

INSTRUCTIONS: ***For each question, please CIRCLE the number that best describes your answer.***

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

	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself — or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed? Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

Appendix D: Expectations Questionnaire

INSOMNI-EX
Expectations Questionnaire

Participant ID: _____
Date of Visit: ____ / ____ / _____
Research Staff: _____

1. As a result of my participation in this study, I expect that my **insomnia** will become:
   - _____ Much worse
   - _____ Somewhat worse
   - _____ The same
   - _____ Somewhat better
   - _____ Much better

2. As a result of my participation in this study, I expect that my **overall sleep quality** will become:
   - _____ Much worse
   - _____ Somewhat worse
   - _____ The same
   - _____ Somewhat better
   - _____ Much better

3. As a result of my participation in this study, I expect that **my daytime functioning (for example, my mood, concentration, sleepiness)** will become:
   - _____ Much worse
   - _____ Somewhat worse
   - _____ The same
   - _____ Somewhat better
   - _____ Much better

INSOMNI-EX
Exercise Session Data Sheet

Participant ID: _____
Watch/HRM #: _____/_____
Target HR: _____
Target HR Range: _____

Session #1	Was the walk performed and if not why?	Walk Duration	Average RPE	Comments:
Day:				
Date:				
Time:				
Session #2	**Was the walk performed and if not why?**	**Walk Duration**	**Average RPE**	**Comments:**
Day:				
Date:				
Time:				
Session #3	**Was the walk performed and if not why?**	**Walk Duration**	**Average RPE**	**Comments:**
Day:				
Date:				
Time:				

INSOMNI-EX
Quiet Rest Session Data Sheet

Participant ID: _____
Watch/HRM #: _____/_____

Session #1	Session Duration	Did the Participant Attend?	Did the participant wear their monitor?	Visit Setting	Comments:
Day:		Yes No	Yes No		
Date:					
Time:					
Session #2	Session Duration	Did the Participant Attend?	Did the participant wear their monitor?	Visit Setting	Comments:
Day:		Yes No	Yes No		
Date:					
Time:					
Session #3	Session Duration	Did the Participant Attend?	Did the participant wear their monitor?	Visit Setting	Comments:
Day:		Yes No	Yes No		
Date:					
Time:					

Appendix G: Weekly Diary

G.1 Baseline Week Diary Sample Pages

ID#_____

Please fill out this page at **BEDTIME**

Tonight is (*circle one*): Sun Mon Tue Wed Thur Fri Sat Today's date is:_____

Today, I had my first contact (in person or by phone) with another person at: _____:_____ AM PM

Today, I started work, school, housework, volunteer activities, child or family care at: _____:_____ AM PM

Today, I ate my meals at: *(if none, write 'none')*

Breakfast _____:_____ AM PM

Lunch _____:_____ AM PM

Dinner _____:_____ AM PM

NAPPING

Today, I took _____ naps *(if none, write '0')*. Naps include any sleep out of bed, however brief.

This also includes any time you fell asleep in the evening before going to bed. My nap times were:

START	END	START	END
1. ____:____ AM PM	____:____ AM PM	2. ____:____ AM PM	____:____ AM PM
3. ____:____ AM PM	____:____ AM PM	4. ____:____ AM PM	____:____ AM PM

IMPORTANT: *At nap start and end, hold the button on the left side of the watch until a circular animation is displayed*

DAYTIME SITTING & ACTIVITY

(*Refer to pg 3 for sitting time & activity definitions; If you did not do this type of activity today, write '0'*)

How many hours today did you spend **SITTING**? _____ hours (*round to the nearest half-hour*)

How many minutes did you spend today performing each of the following types of **PHYSICAL** activity?

Light activity: _____ minutes; Moderate activity: _____ minutes; Vigorous activity: _____ minutes

---

CAFFEINE AND ALCOHOL CONSUMPTION

During the course of today, I had the following amount in each time period *(if none, write '0')*:

	before or with breakfast	after breakfast & before or with lunch	after lunch & before or with dinner	after dinner
Caffeinated drinks				
Drinks containing alcohol				

DEVICE REMOVAL

Did you take off the hip monitor during the day other than before going to sleep? Yes No

If yes, when did you take it off and put it back on?

OFF	ON	REASON
1. ____:____ AM PM	____:____ AM PM	_____
2. ____:____ AM PM	____:____ AM PM	_____

When did you take off the hip monitor before officially going to sleep? _____:_____ AM PM

If this is a Monday, Wednesday, or Friday evening, please complete a PSAS questionnaire at the end of this diary before going to bed

ID#_____

Please fill out this page in the **MORNING**

Today is (*circle one*): Sun Mon Tue Wed Thur Fri Sat Today's date is:_____

IMPORTANT: Hold the button on the left side of the watch until a circular animation is displayed

Last night I got into bed at _____:_____ PM AM

I actually tried to go to sleep at _____:_____ PM AM

I think it took me about _____ minutes to fall asleep

I woke up _____ times, not counting my final awakening.

(Number of times I woke up between when I first fell asleep and my final awakening.)

In total, these awakenings lasted _____ hours _____ minutes.

(The TOTAL time I was awake between the time I first fell asleep and finally awakened.)

This morning, I finally woke at _____:_____ AM PM

I actually got out of bed to start my day at _____:_____ AM PM

IMPORTANT: Hold the button on the left side of the watch until a circular animation is displayed

My final awakening this morning was caused by (*check one*):

Alarm clock/radio ❒ Someone woke me ❒ Noises ❒ I just woke up ❒

When did you put on the hip monitor for the day? _____:_____ AM PM

---

*For these 4 items, please place an **"X"** on the following lines where it best describes your feelings:*

Last night, how active was your mind?

Not at All _____ *Extremely*

Last night, how worried were you?

Not at All _____ *Extremely*

Last night, how anxious did you feel?

Not at All _____ *Extremely*

Last night, how physically tense did you feel?

Not at All _____ *Extremely*

*For these two items, please **circle** one response:*

How would you rate the quality of your sleep last night?

Very Poor Poor Fair Good Very Good

How rested or refreshed do you feel after waking up for the day?

Not At All Slightly Somewhat Well-Rested Very Well-Rested

G.2 Experimental Week Diary Sample Pages

ID#_____

Please fill out this page at **BEDTIME**

Tonight is (*circle one*): Sun Mon Tue Wed Thur Fri Sat Today's date is:_____

Today, I had my first contact (in person or by phone) with another person at: _____:_____ AM PM

Today, I started work, school, housework, volunteer activities, child or family care at: _____:_____ AM PM

Today, I ate my meals at: *(if none, write 'none')*

Breakfast _____:_____ AM PM

Lunch _____:_____ AM PM

Dinner _____:_____ AM PM

NAPPING

Today, I took _____ naps *(if none, write '0')*. Naps include any sleep out of bed, however brief.

This also includes any time you fell asleep in the evening before going to bed. My nap times were:

START	END	START	END
1. ____:____ AM PM	____:____ AM PM	2. ____:____ AM PM	____:____ AM PM
3. ____:____ AM PM	____:____ AM PM	4. ____:____ AM PM	____:____ AM PM

IMPORTANT: *At nap start and end, hold the button on the left side of the watch until a circular animation is displayed*

DAYTIME SITTING & ACTIVITY

(*Refer to pg 3 for sitting time & activity definitions; If you did not do this type of activity today, write '0'*)

How many hours today did you spend **SITTING**? _____ hours (*round to the nearest half-hour*)

How many minutes did you spend today performing each of the following types of **PHYSICAL** activity?

Light activity: _____ minutes; Moderate activity: _____ minutes; Vigorous activity: _____ minutes

---

CAFFEINE AND ALCOHOL CONSUMPTION

During the course of today, I had the following amount in each time period *(if none, write '0')*:

	before or with breakfast	after breakfast & before or with lunch	after lunch & before or with dinner	after dinner
Caffeinated drinks				
Drinks containing alcohol				

DEVICE REMOVAL

Did you take off the hip monitor during the day other than before going to sleep? Yes No

If yes, when did you take it off and put it back on?

OFF	ON	REASON
1. ____:____ AM PM	____:____ AM PM	_____
2. ____:____ AM PM	____:____ AM PM	_____

When did you take off the hip monitor before officially going to sleep? _____:_____ AM PM

If you completed an experimental session today, fill out a PSAS questionnaire at the end of this diary.

ID#_____

Please fill out this page in the **MORNING**

Today is (*circle one*): Sun Mon Tue Wed Thur Fri Sat Today's date is:_____

IMPORTANT: Hold the button on the left side of the watch until a circular animation is displayed

Last night I got into bed at _____:_____ PM AM

I actually tried to go to sleep at _____:_____ PM AM

I think it took me about _____ minutes to fall asleep

I woke up _____ times, not counting my final awakening.

(Number of times I woke up between when I first fell asleep and my final awakening.)

In total, these awakenings lasted _____ hours _____ minutes.

(The TOTAL time I was awake between the time I first fell asleep and finally awakened.)

This morning, I finally woke at _____:_____ AM PM

I actually got out of bed to start my day at _____:_____ AM PM

IMPORTANT: Hold the button on the left side of the watch until a circular animation is displayed

My final awakening this morning was caused by (*check one*):

Alarm clock/radio ❒ Someone woke me ❒ Noises ❒ I just woke up ❒

When did you put on the hip monitor for the day? _____:_____ AM PM

---

*For these 4 items, please place an **"X"** on the following lines where it best describes your feelings:*

Last night, how active was your mind?

Not at All _____ *Extremely*

Last night, how worried were you?

Not at All _____ *Extremely*

Last night, how anxious did you feel?

Not at All _____ *Extremely*

Last night, how physically tense did you feel?

Not at All _____ *Extremely*

*For these two items, please **circle** one response:*

How would you rate the quality of your sleep last night?

Very Poor Poor Fair Good Very Good

How rested or refreshed do you feel after waking up for the day?

Not At All Slightly Somewhat Well-Rested Very Well-Rested

Appendix H: Insomnia Severity Index (ISI)

INSOMNI-EX
ISI

Participant ID: _____
Date : _____ / _____ / _____
Timepoint (circle): Baseline Intervention

COMPLETE WHEN YOU HAVE FINISHED THE DIARY

INSTRUCTIONS: ***For each question, please CIRCLE the number that best describes your answer.***

1. Please rate the current (i.e., last 7 days) **SEVERITY** of your insomnia problem(s).

	None	Mild	Moderate	Severe	Very Severe
a. Difficulty falling asleep	0	1	2	3	4
b. Difficulty staying asleep	0	1	2	3	4
c. Problems waking up too early	0	1	2	3	4

2. How **SATISFIED/DISSATISFIED** are you with your **CURRENT** sleep pattern?

Very Satisfied	Satisfied	Moderately Satisfied	Dissatisfied	Very Dissatisfied
0	1	2	3	4

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

Not at all Noticeable	Barely	Somewhat	Much	Very Much Noticeable
0	1	2	3	4

4. How **WORRIED/DISTRESSED** are you about your current sleep problem?

Not at all Worried	A Little	Somewhat	Much	Very Much Worried
0	1	2	3	4

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

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering
0	1	2	3	4

Appendix I: PROMIS Sleep-Related Impairment Scale (PROMIS-SRI)

INSOMNI-EX
PROMIS-SRI

Participant ID: _____
Date : _____ / _____ / _____
Timepoint (circle): Baseline Intervention

COMPLETE WHEN YOU HAVE FINISHED THE DIARY

INSTRUCTIONS: ***Please respond by marking one box per row.***

IN THE PAST 7 DAYS…

	Not at all	A little bit	Somewhat	Quite a bit	Very much
I had a hard time getting things done because I was sleepy…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I felt alert when I woke up…	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
I felt tired…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I had problems during the day because of poor sleep…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I had a hard time concentrating because of poor sleep…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I felt irritable because of poor sleep…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I was sleepy during the daytime…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I had trouble staying awake during the day…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

Appendix J: RUSATED Questionnaire

INSOMNI-EX
RUSATED

Participant ID: _____
Date : _____ / _____ / _____
Timepoint (circle): Baseline Intervention

COMPLETE WHEN YOU HAVE FINISHED THE DIARY

INSTRUCTIONS: ***Please indicate your sleep health over the past seven days in each of these categories from rarely/never to usually/always achieving that behavior. Respond by marking one box per row.***

		Rarely/ Never (0)	Sometimes (1)	Usually/ Always (2)
Regu**l**arity	Did you go to bed and get out of bed at about the same time (within one hour) every day?			
Satisfaction	Were you satisfied with your sleep?			
Alertness	Did you stay awake all day without dozing?			
Timing	Were you asleep (or trying to sleep) between 2:00 a.m. and 4:00 a.m.?			
Efficiency	Did you spend less than 30 minutes awake at night? This includes the time it takes to fall asleep plus awakenings during sleep.			
Duration	Did you sleep between 6 and 8 hours per day?			

Appendix K: PROMIS Sleep Disturbance Scale (PROMIS- SD)

INSOMNI-EX
PROMIS-SD

Participant ID: _____
Date : _____ / _____ / _____
Timepoint (circle): Baseline Intervention

COMPLETE WHEN YOU HAVE FINISHED THE DIARY

INSTRUCTIONS: ***Please respond by marking one box per row.***

IN THE PAST 7 DAYS…

	Not at all	A little bit	Somewhat	Quite a bit	Very much
My sleep was restless…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I was satisfied with my sleep…	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
My sleep was refreshing…	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
I had difficulty falling asleep…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

	Never	Rarely	Sometimes	Often	Always
I had trouble staying asleep…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I had trouble sleeping…	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
I got enough sleep…	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1

	Very poor	Poor	Fair	Good	Very good
My sleep quality was…	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1

Appendix L: Pre-Sleep Arousal Scale (PSAS)

INSOMNI-EX
PSAS

Participant ID: _____
Date : _____ / _____ / _____
In-person session # (circle) : 1 2 3
Time survey is taken: _____:_____

ONLY COMPLETE ON A NIGHT AFTER YOU HAVE HAD AN IN-PERSON SESSION

INSTRUCTIONS:

The questions on this scale ask you about your thoughts and feelings you may have when you go to sleep at night.

Please indicate how intensely you experience each statement below. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to complete each statement fairly quickly. Again, we are interested in how you feel when you go to sleep tonight.

	1 Not at all	2 Slightly	3 Moderately	4 A Lot	5 Extremely
1. Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas, etc.).					
2. Being mentally alert, active.					
3. Feeling distracted by sounds, noise in the environment (e.g., noises in the laboratory, ticking of clock).					
4. A tight, tense feeling in your muscles.					
5. Thoughts keep running through your head.					
6. Cold feeling in your hands, feet, or your body in general.					
7. A jittery nervous feeling in your body.					
8. Worry about problems other than sleep.					
9. Perspiration in palms of your hands or other parts of your body.					
10. Depressing or anxious thoughts.					
11. Dry feeling in mouth or throat.					
12. Worry about falling asleep.					
13. Shortness of breath or labored breathing.					
14. Can't shut off your thoughts.					
15. Heart racing, pounding, or beating irregularly.					
16. Review or ponder events of the day.					

Appendix M: Self-Efficacy for Exercise Scale (SEES)

INSOMNI-EX
SEES

Participant ID: _____
Date : _____ / _____ / _____
Timepoint (circle): Baseline Intervention

COMPLETE WHEN YOU HAVE FINISHED THE DIARY

INSTRUCTIONS: ***For each question, please CIRCLE the number that best describes your answer.***

How confident are you right now that you could exercise three times per week for 20 minutes if:

	Not Confident										Very Confident
1. the weather was bothering you	0	1	2	3	4	5	6	7	8	9	10
2. you were bored by the program or activity	0	1	2	3	4	5	6	7	8	9	10
3. you felt pain when exercising	0	1	2	3	4	5	6	7	8	9	10
4. you had to exercise alone	0	1	2	3	4	5	6	7	8	9	10
5. you did not enjoy it	0	1	2	3	4	5	6	7	8	9	10
6. you were too busy with other activities	0	1	2	3	4	5	6	7	8	9	10
7. you felt tired	0	1	2	3	4	5	6	7	8	9	10
8. you felt stressed	0	1	2	3	4	5	6	7	8	9	10
9. you felt depressed	0	1	2	3	4	5	6	7	8	9	10

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