Exercise to Improve Sleep in Insomnia: Exploration of the Bidirectional Effects

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Background: Exercise improves sleep quality, mood, and quality of life among older adults with insomnia. The purpose of the study was to evaluate the daily bidirectional relationships between exercise and sleep in a sample of women with insomnia.

Methods: Participants included 11 women (age $M = 61.27$, $SD = 4.15$) with insomnia who engaged in 30 min of aerobic exercise 3 times per week. Self-reported sleep quality was assessed at baseline and at 16 weeks. Sleep and exercise logs and wrist activity were collected continuously. Sleep variables included subjective sleep quality and objective measures recorded via wrist actigraphy (sleep onset latency [SOL], total sleep time [TST], sleep efficiency [SE], wake after sleep onset [WASO], and fragmentation index [FI]). Age, subjective sleep quality, TST, SOL, and physical fitness at baseline were tested as moderators of the daily effects.

Results: TST, SE, and self-reported global sleep quality improved from baseline to 16 weeks (p values < 0.05). Baseline

ratings of sleepiness were negatively correlated with exercise session duration ($p < 0.05$). Daily exercise was not associated with subjective or objective sleep variables during the corresponding night. However, participants had shorter exercise duration following nights with longer SOL ($p < 0.05$). TST at baseline moderated the daily relationship between TST and next day exercise duration ($p < 0.05$). The relationship between shorter TST and shorter next day exercise was stronger in participants who had shorter TST at baseline.

Conclusion: Results suggest that sleep influences next day exercise rather than exercise influencing sleep. The relationship between TST and next day exercise was stronger for those with shorter TST at baseline. These results suggest that improving sleep may encourage exercise participation.

Keywords: Insomnia, physical activity, sleep

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Insomnia is characterized by difficult initiating and/or maintaining sleep or non-restorative sleep which causes at least one area of impairment, such as depressed or irritable mood, decreased concentration, daytime sleepiness, or physical malaise.¹ Chronic insomnia is present in 10% to 15% of the population. Insomnia is more prevalent in women and prevalence increases with age.²⁻⁴ It is estimated that up to 35% of older adults have insomnia.⁵ Consequences of insomnia among older adults include decreased quality of life, lower cognitive function, and risk for hip fractures.⁶⁻⁹ Non-pharmacological treatments are often preferred by patients and physicians due to concern over side effects and increased mortality in hypnotic users.^{10,11}

Aerobic exercise has been tested in multiple studies as a non-pharmacological intervention for sleep in older adults that has general health benefits and is readily accessible to most individuals.¹² The benefits of exercise on insomnia symptoms are most consistent for self-reported sleep quality and sleep diary based measures. A systematic review of 6 randomized trials of exercise in older adults (with and without insomnia) demonstrated improvements of self-reported global sleep quality, decreased self-reported sleep latency, and decreased sleep medication use.¹³ Only a few studies have reported objective sleep data. In a study of older adults, King and colleagues¹⁴ demonstrated that 12 months of moderate intensity aerobic activity led to improvements in self-rated and diary-based measures of sleep quality, as well as modest improvement in some polysomno-

BRIEF SUMMARY

Current Knowledge/Study Rationale: Multiple studies have demonstrated large improvements in subjective sleep quality and moderate improvements in objective sleep parameters with exercise interventions. However, few studies have evaluated the acute effects of exercise on sleep.

Study Impact: This study demonstrates a day to day relationship between sleep and next day exercise. Patients with insomnia should be encouraged to evaluate the effectiveness of exercise on sleep over time rather than day to day. Poor sleep may decrease exercise participation.

graphic measures (such as fewer awakenings in the first third of the night) in a sample of older adults with poor sleep quality. A recent study conducted in Brazil demonstrated improvements in both self-reported sleep and PSG measures in a sample of middle-aged adults with insomnia.¹⁵ In this study, 6 months of aerobic exercise (50 min, 3 times/week) led to objective and subjective improvements, including decreased sleep onset latency on polysomnography and sleep diary, decreased wake after sleep onset, increased sleep efficiency (SE), and increased ratings of sleep quality and feeling rested.

There have been fewer investigations into the acute effects of exercise in patients with insomnia. In clinical settings, patients often have the expectation that exercising—particularly vigorous exercise—will lead to rapid improvement in their sleep that night (e.g., “I exercised until I was exhausted and I still couldn’t sleep”). Although the relationship between acute

exercise and sleep has been studied extensively in laboratory studies of healthy populations,¹⁶ the relationship between acute exercise and sleep has been investigated in only two prior studies in participants with sleep disturbance. King and colleagues¹⁷ found no correlation between polysomnographic variables and exercise when evaluating whether the assessment was conducted on a night that followed or did not follow exercise in older adults with sleep complaints. However, one study demonstrated acute improvements in polysomnographic measures among adults with insomnia.¹⁸ Compared with a control night without exercise, one session of moderate aerobic exercise lead to a reduction in sleep onset latency (SOL) and total wake time, and increases in total sleep time (TST) and SE during the corresponding night. There were no significant improvements in sleep during the night following high-intensity exercise or moderate-intensity resistance training.

The prior studies of acute exercise for sleep disturbance have only evaluated one or two nights of exercise and sleep. This is potentially problematic in insomnia because the disorder itself is characterized by variability in sleep pattern.¹⁹ Therefore, one or two nights may not adequately capture the sleep of persons with insomnia. The use of wrist actigraphy allows for the evaluation of estimated sleep patterns in the home environment for a longer period of time with minimal participant burden. The goal of the present study is to explore the acute relationship between exercise and sleep through 16 weeks of daily data (daily sleep diary, logs, and actigraphy) collected in women enrolled in an exercise intervention for insomnia. The structure of the data (daily values over 16 weeks) allows us to evaluate the directionality of the effect. We previously reported improvements in self-reported sleep quality, quality of life, daytime sleepiness, TST, and SE associated with 16 weeks of aerobic exercise in this sample.²⁰ This analysis extends these findings by evaluating the day-to-day relationships between exercise and sleep.

METHODS

Data from this study were drawn from a larger study, which tested the effects of a 16-week exercise and sleep hygiene intervention versus 16 weeks of non-physical activity and sleep hygiene intervention on sleep, daytime function, and metabolism in older adults with insomnia. Data from the exercise group alone was used for these analyses. This study was approved by the Northwestern University Institutional Review Board and all participants provided written informed consent.

Additional details of the study are listed elsewhere.²⁰ Participants included in this analysis were 11 healthy, community-dwelling sedentary older adults (≥ 55 years) with sleep disturbance ≥ 3 months. All participants met DSM-IV criteria for primary insomnia.²¹ In addition, this study included the following subjective and objective criteria: Pittsburgh Sleep Quality Index Score ≥ 5 , SE $< 80\%$ and/or waking earlier than desired if before 06:00, and TST ≤ 6.5 h demonstrated on 7 days of wrist actigraphy. Sedentary behavior was defined as participation in mild to moderate intensity exercise < 30 min per day on < 2 days per week. Inclusion criteria also included the minimal status exam (MMSE) > 26 .

Exclusionary criteria included the following: (a) significant comorbid sleep disorders documented on screening polysom-

nography (apnea index > 10 , periodic leg movement arousal index > 15 , REM behavior disorder); (b) history of cognitive or other neurological disorders; (c) history of DSM-IV criteria for any major psychiatric disorder, including mania or alcohol or substance abuse; (d) significant depressive symptoms as assessed by the Center for Epidemiological Studies Depression Scale (CES-D score > 22); (e) unstable or serious medical conditions or cardiopulmonary disease that contraindicate exercise; (f) current use or use within the past month, of psychoactive, hypnotic, stimulant, or analgesic medications; (g) shift work or other types of self-imposed irregular sleep schedules; (h) BMI > 35 kg/m²; (i) history of habitual smoking (≥ 3 cigarettes per week) or caffeine consumption (> 300 mg).

After initial eligibility was determined through telephone screening, participants were screened with overnight polysomnography, screening questionnaires (MMSE, CES-D, PSQI), and 7 days of activity monitoring using wrist actigraphy and a sleep log. If participants met inclusion criteria, they were randomized to treatment groups—exercise plus sleep hygiene or a mental (non-physical) activity and sleep hygiene. At Baseline, participants were admitted to the clinical research unit (CRU) for a 4-day baseline admission which involved exercise testing, questionnaires, polysomnography, health measures (e.g., oral glucose tolerance test), and performance testing. Seven days of baseline actigraphy was also completed prior to beginning the intervention. At the end of the 16-week intervention period, participants completed another 4-day CRU admission for a similar battery of tests and questionnaires. Participants completed 7 days of actigraphy at the end of the intervention.

Interventions

Sleep Hygiene

Participants received sleep hygiene education which consisted of a sleep hygiene appointment with a sleep specialist, in which they were provided with verbal and written sleep hygiene instructions according to materials published by the American Academy of Sleep Medicine. Patients were encouraged by research staff to continue practicing sleep hygiene instructions during study visits every 2 weeks.

Exercise Intervention

Baseline exercise testing was performed to determine exercise capacity and tailor the exercise intervention to each participant. The exercise testing protocol used the modified Bruce protocol and was designed for participants to reach their anaerobic threshold (a marker of oxygen demand exceeding the supply, indicated by increasing CO₂ levels in the airway) no sooner than 5 and not longer than 15 minutes.²² The anaerobic threshold was determined by measurements of oxygen consumption and end-tidal CO₂. A symptom-limited maximal ergometer test with a 10 to 40 watt/minute-step protocol was used to measure VO₂ max for each subject.

The conditioning period (week 1-6) was conducted under the supervision of an exercise physiologist. The conditioning protocol included exercise sessions 4 times per week with the following specifications: (Week 1) 10-15 min/day at 55% max heart rate (HR) as measured with a heart rate monitor (Protrainer, Polar Electro Inc., Port Washington, NY); (Week 2) 15-20

min/day at 60% max HR; (Week 3) 20-25 min/day at 65% max HR; (Week 4) 25-30 min/day at 70% max HR; (Week 5-6) attaining 75% of max HR for 30-40 min.

After completion of the conditioning period, participants were asked to exercise for either two 20-min sessions or one 30- to 40-min session at 75% of their maximum HR 4 times per week for the duration of the study. Exercise sessions were conducted in the afternoon or evening (13:00-19:00), and participants were required to miss no more than 1 exercise session per week. Participants engaged in ≥ 2 of 3 aerobic activities (walking, stationary bicycle, or treadmill) and engaged in each activity at a similar level of exertion, as measured by the BORG scale of Perceived Exertion and heart rate monitor.

Measures

Subjective sleep quality was measured by The Pittsburgh Sleep Quality Index (PSQI).²³ This 19-item measure assessed self-reported sleep quality and disturbances over a 1-month time interval. There are 7 component scores, which are scaled from 0 to 3. The PSQI global score is the sum of the component scores (range, 0-21). A higher PSQI global score indicates greater sleep disturbance. Scores ≥ 5 are associated with clinically significant sleep disturbance.²³ Although not specific to insomnia, this measure has been designed for use in clinical populations and validated in older adults.²⁴

Self-reported sleepiness was measured using the Epworth Sleepiness Scale (ESS).²⁵ On this 8-item questionnaire, participants rated the likelihood of dozing off in daily situations, such as sitting and reading, watching TV, as a passenger in a car for an hour without a break, and laying down in the afternoon if circumstances permit, from 0 (not at all likely) to 3 (very likely). Scores range from 0-24, with higher scores indicating greater sleepiness. Adequate reliability and validity have been reported for this measure.²⁵⁻²⁷

Participants completed daily sleep logs and turned them in to study staff for review and to assist in scoring actigraphy at 2 week intervals. Participants recorded bedtime, get up time, number of awakenings during the night, and daily subjective rating of sleep quality from 1 (excellent) to 4 (poor).

Daily rest-activity rhythms were assessed via wrist actigraphy during the duration of the study (AW-64 Actiwatch, Mini Mitter Co. Inc., Bend, OR). Actiwatchers were set with 30-sec epoch length and medium sensitivity. Sleep onset was scored as the first epoch with 10 min of inactivity. Sleep onset time, sleep offset time, minutes of wake after sleep onset (WASO), TST, sleep efficiency, and fragmentation index were calculated from actigraphy recordings using Actiware-Sleep 3.4 software (Mini Mitter Co. Inc., Bend, OR). Sleep diary based measures of bedtime and rise time were manually entered and used for calculation of sleep onset latency and sleep efficiency. Periods where the watch was clearly removed or reported as removed (bathing, swimming, etc.) were not included in analysis, and a day was not considered valid if there was any off-wrist time reported during the rest interval. All valid days were utilized in the analyses. The 2 weeks prior to the beginning of the exercise participation were considered the baseline portion of the study; weeks 1-16 were considered the exercise portion of the study.

Adherence to the exercise intervention was evaluated using self-reported exercise logs in which the participants recorded

the following variables for each exercise session: start time, duration, perceived exertion, and type of exercise. Due to incomplete data for perceived exertion and limited range for exercise type (e.g., most were aerobic and treadmill walking), analyses were limited to exercise duration.

Fitness was defined by VO_2 max, determined by treadmill exercise testing conducted at baseline and 16 weeks.

Data Analysis

Descriptive analysis, correlations, and t-tests were conducted using SPSS v. 20. We also conducted bivariate correlations between baseline sleep variables, change in sleep variables, and exercise duration.

Day-to-day analyses of the relationship between exercise and sleep variables were conducted using hierarchical linear modeling (HLM).²⁸ The hierarchical structure of the data (diary and actigraphy days nested in individuals) allowed us to test both within (level 1) and between (level 2) subject effects. In all models, diary day was entered as an un-centered variable, with day 1 scaled as zero. All other level 1 variables were centered around each individual's mean, referred to as "person centered."²⁹ This method of centering allowed us to ask the question "Do individuals report better quality sleep or higher TST on nights following days with more than their own average exercise duration," as well as "Do individuals better quality sleep or higher TST following days with higher than their own average exercise?" In models with significant variability in the day to day within-person effects (level 1), moderators, age, baseline PSQI global score, and habitual sleep variables measured at baseline (SOL and TST) were entered at level 2 to test between-group differences in the daily relationships between exercise and sleep. In the first set of models, we tested the question, "Does exercise duration predict sleep variables during the corresponding night." In the second set of models, we created a time lagged variable to test "Do sleep variables predict exercise duration during the next day?" Finally, due to build-up of homeostatic sleep pressure after a poor night of sleep, we created 2 daytime lagged variables to test if sleep 2 nights prior predicts exercise.³⁰ Estimates reported are unstandardized coefficients and can be interpreted similar to B coefficients in multiple regression analyses. Statistical significance was set at p-values < 0.05 using 2-tailed tests.

RESULTS

Participant Characteristics and Sleep Variables (Table 1)

Of the 23 participants who were eligible for the study, this analysis includes the 11 participants who were randomized to the exercise group. There were no dropouts from this group, but data from one participant were censored at 12 weeks due to a stressful life event that affected sleep and mood. All participants in this analysis were female, and average age was 61 years (SD 4.4). Participants completed an average of 54.4 (SD 14.4) exercise sessions over the 16 weeks. Average duration of exercise was 32.5 (SD 3.8) min. Treadmill was the most common type, comprising 65% of sessions. Average BMI was 26.7 (4.9) kg/m^2 . Average ESS was 9.2 (SD 5.3). Sleep-wake estimates measured via actigraphy as well as self-

Table 1—Participant characteristics and sleep data (n = 11)

Variable	Baseline M (SD)	
Age	61.4 (4.4) years	
Baseline BMI	26.7 (4.9) kg/m ²	
Baseline VO ₂ max	24.8 (5.3) L/kg	
Baseline ESS	9.2 (5.3)	
Number of exercise sessions attended	54.4 (14.4) sessions	
Average duration of exercise session	32.5 (3.8) min	
Sleep Data	Baseline M (SD)	16 weeks [‡]
Sleep Onset Time	23:28 (0:42)	23:21 (0:50)
Sleep Offset Time	6:19 (1:03)	6:42 (0:32)
Time in Bed (hh:mm)	7:30 (1:02)	7:50 (0:29)
Sleep Latency (hh:mm)	0:18 (0:20)	0:26 (0:22)
Total Sleep Time** (hh:mm)	5:54 (0:36)	6:40 (0:44)
Wake After Sleep Onset [†] (hh:mm)	1:00 (0:30)	0:46 (0:19)
Sleep Efficiency* (hh:mm)	84.1 (5.98)	87.8 (5.32)
Fragmentation Index	35.3 (11.06)	32.9 (8.82)
Subjective Daily Rating (Diary)	2.42 (0.60)	2.58 (0.67)
PSQI Global Score***	10.2 (2.1)	5.1 (1.4)

ESS, Epworth Sleepiness Score. PSQI and actigraphy scores have been previously reported: *p < 0.05, **p < 0.01, ***p < 0.001, [†]p < 0.10. [‡]Data at 16 weeks are available for 10 participants.

reported sleep quality ratings are listed in **Table 1**. Participants demonstrated significant increases in TST (p < 0.01) and sleep efficiency (p < 0.05), and decreases in global ratings of sleep quality on the PSQI (p < 0.001). There was a trend for a reduction in WASO (p < 0.10). Timing of sleep onset, offset, time in bed, sleep latency, fragmentation index, and daily diary based ratings of sleep quality did not significantly change from baseline to 16 weeks.

Correlations Between Baseline Sleep Variables and Exercise Duration

Participants with higher self-reported sleepiness on the ESS at baseline reported shorter average duration of their exercise sessions (r = -0.67, p = 0.03). Exercise duration was not correlated with baseline actigraphy or self-reported sleep variables or VO₂max.

Daily Exercise and Sleep During the Corresponding Night

In the first set of multilevel models, we tested exercise duration as a predictor of sleep during the corresponding night. Results demonstrated that exercise was not associated with SOL, TST, WASO, SE, or subjective ratings of sleep quality. There was not significant variability in level 1 effects, therefore moderators could not be tested in level 2 of the model. Exercise also did not predict sleep variables in 2-day lag models.

Sleep and Next Day Exercise

The next set of models tested sleep as a predictor of next day exercise. SOL was negatively associated with next day ex-

Figure 1—Daily relationship between sleep onset latency and next day exercise duration, individual trajectories

ercise (b = -2.30, standard error = 0.90, p = 0.029, **Figure 1**). There was positive association between TST and next day exercise (b = 1.41, standard error = 0.66, p = 0.06), but this did not reach statistical significance. Variability in level 1 effects of this model allowed us to test moderators in level 2. Baseline TST was a significant moderator of the within subject effects of daily TST and next day exercise (b = -2.66, standard error = 0.93, p = 0.02; **Figure 2**). Participants with shorter baseline TST had a stronger daily relationship between TST at night and next day exercise duration. SE, WASO, FI, and daily ratings of subjective sleep quality were not associated with next day exercise. Sleep variables did not predict exercise 2 days later. There was significant variability for in the 2-day lag model for SOL (p = 0.03), but none of the moderator variables tested in level 2 explained this variability.

DISCUSSION

Results of this study provide new insight into the relationship between exercise and sleep. In this study, we used the structure of the daily actigraphy, sleep, and exercise log data that were collected over 16 weeks for a unique perspective on the daily relationships between these variables in participants' home environment over many nights. We found that exercise during the day was not associated with sleep during the corresponding night. However, sleep at night did predict next day exercise. Specifically, coefficients from the HLM model indicate that for every 30-minute increase in sleep onset latency above the individual's own average value, there was a one-minute decrease in next day exercise duration. We also found an interaction between habitual TST and the daily relationship between TST and next day exercise. For shorter sleepers, there was a stronger relationship between poor sleep and decreased next day exercise duration.

Our finding that exercise did not correlate with sleep during the corresponding night is consistent with data reported by

King and colleagues, who also evaluated the acute effects of exercise on sleep in a 12-month study conducted in older adults with sleep complaints.¹⁷ In this study, King and colleagues did not find a difference in sleep measured by home polysomnography on 2 consecutive nights if one night followed exercise and one night did not. In a laboratory study, Passos and colleagues¹⁵ found that only moderate-intensity aerobic exercise, not strenuous aerobic exercise or resistance training improved PSG measures of sleep in a sample of middle-aged adults with insomnia. There are far more studies of effects of acute exercise in healthy populations without insomnia. Results from a meta-analysis of 38 laboratory studies of acute exercise in healthy participants demonstrated that acute exercise improved polysomnographic measures of sleep including reducing sleep onset latency and increasing TST.¹⁶ This suggests that understanding the influence of variables such as age, presence of an insomnia diagnosis, and exercise type is important to understanding the acute effects of exercise on sleep. For example, among studies conducted in healthy populations, age has been associated with a larger effect size for exercise on polysomnographic measures of sleep.³¹

In our study, individuals with shorter habitual TST were more responsive to the daily effects of each night of sleep. This finding is interesting, given that our sample were all patients with insomnia and short TST (≤ 6.5 h). Research has demonstrated that sleep loss affects exercise tolerance, motivation, and mood. Laboratory studies conducted in young, healthy samples have demonstrated that sleep deprivation increases perceived exertion and time to exhaustion in exercise testing.^{32,33} In addition, the combination of exercise and sleep loss may further affect mood and motivation. In a laboratory study that combined exercise with 30 hours of sleep loss compared to sleep loss alone, those assigned to sleep loss and exercise demonstrated greater decreases in vigor, as well as greater increases in depression and fatigue.³⁴ Although 30 hours of sleep deprivation is different from insomnia, this does suggest that the combination of sleep loss and exercise may have additive effects. To date, there are no studies evaluating whether improving insomnia or extending sleep among healthy individuals will increase exercise. However, increasing time in bed for college basketball players improved sprint times and free throw accuracy as well as decreased fatigue and increased vigor.³⁵

There are several plausible mechanisms that could link better sleep to increased exercise, including decreased HPA activation, inflammation, improved metabolism, greater energy conservation. One study demonstrated that inflammatory response to physical exercise was greater after partial sleep deprivation.³⁶ Sleep deprivation has also been related to increased pain ratings.^{37,38} In a daily questionnaire study, sleep at night was predictive of next day pain ratings.³⁹ Thus, disrupted sleep may lead to decreased desire to exercise and increased pain, which decreases next day exercise.

In correlations between baseline variables and exercise adherence over the duration of the study, we also found that participants with higher self-rated sleepiness at baseline had shorter average duration of their exercise sessions. This suggests that the feeling of sleepiness may interfere with exercise participation. Furthermore, in the setting of insomnia, the effect of sleep loss and dysregulated affective control may magnify the effects of sleep loss on motivation to exercise.⁴⁰

Figure 2—Total sleep time at baseline moderates the relationship between daily total sleep time and next day exercise duration

Although there is no consensus as to how to calculate statistical power in multilevel models, it is likely that our small sample limited power to discern and contributed to many marginal findings.⁴¹ Furthermore, our results may not be generalizable to younger age groups, adults without insomnia, or men. It is also important to note that exercise timing, frequency, and duration were prescribed by the protocol to be conducted for at least 30 min in the afternoon, 3-4 times per week. This is an important point because acute effects of exercise may greatly differ based on time of day,¹⁶ and the effect size may be larger in participants who were not monitored on compliance. In addition, this study only evaluated exercise duration, and there may be other effects of exercise intensity and type of exercise. Strengths of this study are the use of daily data continuously monitored over 16 weeks, which included over 100 observations per participant. In addition, use of a monitored exercise protocol using actigraphy allowed us to control the delivery and at the same time observe these relationships over a long duration in a real-world setting.

In conclusion, results suggest despite many patients' expectations that exercise will immediately improve sleep, we found that sleep affects exercise participation. Data demonstrate that aerobic exercise is an effective intervention that improves objective and self-rated sleep in older women with insomnia. However, the duration of exercise was unrelated to sleep during the corresponding night. Patients with insomnia should be encouraged to exercise regularly and monitor improvement in sleep over longer periods of time rather than focusing on daily improvement. Understanding the daily relationship between exercise and sleep may help inform the development of behavioral interventions for insomnia and identify those at risk for poor adherence to exercise interventions.

ABBREVIATIONS

SOL, sleep onset latency
TST, total sleep time
WASO, wake after sleep onset

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