EFFECTS OF RESISTANCE EXERCISE TIMING ON SLEEP ARCHITECTURE

A Thesis
by
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Abstract

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Short sleep duration and poor quality of sleep have been associated with health risks including cardiovascular disease, diabetes, and obesity. Prior research has suggested that regular aerobic exercise improves quality of sleep; however, less is known regarding resistance exercise (RE) and how RE may affect sleep architecture. The purpose of this study was to investigate the acute effects of timing of RE on sleep architecture. College-aged subjects engaged in 5 laboratory visits. Visits 1 (C) and 2 provided a non-RE control day and established the 10-repetition maximum (10RM) on each of nine RE machines, respectively. During visits 3-5, subjects reported at 7 a.m. (7A), 1 p.m. (1P), and 7 p.m. (7P) in a randomized order to perform 30 minutes of RE. An ambulatory sleep-monitoring headband was worn during sleep following C, 7A, 1P, and 7P. Time to fall asleep was significantly different between RE conditions 7A and 1P ($P = 0.03$) and 7A and 7P ($P = 0.02$). All exercise conditions exhibited significantly fewer times woken than the non-RE control day ($P = 0.04$), with 7P resulting in significantly less time awake after initially falling asleep (WASO) as compared to C ($P = 0.01$). While timing of RE does not appear to impact

sleep stages, these data indicate that engaging in RE at any time of day may improve quality of sleep as compared to no RE. Resistance exercise may offer additional benefits regarding the ability to fall asleep and stay asleep to populations with osteoporosis, sarcopenia, anxiety, or depression.

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Foreword

The contents of this thesis will be submitted to the *American Journal of Physiology – Regulatory, Integrative and Comparative Physiology*, an international peer-reviewed journal published by the American Physiological Society; it has been formatted according to the style guidelines for this journal.

Effects of Resistance Exercise Timing on Sleep Architecture

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Running Head: Resistance Exercise and Sleep

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ABSTRACT

Short sleep duration and poor quality of sleep have been associated with health risks including cardiovascular disease, diabetes, and obesity. Prior research has suggested that regular aerobic exercise improves quality of sleep; however, less is known regarding resistance exercise (RE) and how RE may affect sleep architecture. The purpose of this study was to investigate the acute effects of timing of RE on sleep architecture. College-aged subjects engaged in 5 laboratory visits. Visits 1 (C) and 2 provided a non-RE control day and established the 10-repetition maximum (10RM) on each of nine RE machines, respectively. During visits 3-5, subjects reported at 7 a.m. (7A), 1 p.m. (1P), and 7 p.m. (7P) in a randomized order to perform 30 minutes of RE. An ambulatory sleep-monitoring headband was worn during sleep following C, 7A, 1P, and 7P. Time to fall asleep was significantly different between RE conditions 7A and 1P ($P = 0.03$) and 7A and 7P ($P = 0.02$). All exercise conditions exhibited significantly fewer times woken than the non-RE control day ($P = 0.04$), with 7P resulting in significantly less time awake after initially falling asleep (WASO) as compared to C ($P = 0.01$). While timing of RE does not appear to impact sleep stages, these data indicate that engaging in RE at any time of day may improve quality of sleep as compared to no RE. Resistance exercise may offer additional benefits regarding the ability to fall asleep and stay asleep to populations with osteoporosis, sarcopenia, anxiety, or depression.

Key Words: Sleep quality, resistance training, exercise timing

INTRODUCTION

Short sleep duration is associated with an increased prevalence of adverse cardiovascular events including stroke, myocardial infarction, and congestive heart failure (1) as well as conditions of impaired glucose tolerance (18, 40), increased cortisol levels (40), elevated blood pressure (22, 28, 43), and an increase in systemic inflammation (19, 28, 48), all of which may have long term negative effects on health and contribute to the development of diseases such as diabetes, obesity, and hypertension. Depression and anxiety have also been linked to insufficient sleep (5, 26, 27, 31, 42). Prior research has suggested that six to eight hours of sleep per night is optimal for health (1, 4, 14) and that individuals who consistently experience shorter sleep duration have a higher risk of all-cause mortality (4, 13).

While amount of sleep is critical for health, quality of sleep is also important. Sleep phases are typically differentiated by patterns of brain wave activity as measured on an electroencephalogram (EEG) and can be divided into rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. Non-rapid eye movement sleep is comprised of three stages: N1, N2, and N3 (formerly divided into stages 3 and 4) (36). Light sleep includes N1 and N2 and occurs between wakefulness and deep sleep, or N3. A typical night of sleep for a young adult usually consists of 10-25% of total sleep as deep sleep, which is also commonly referred to as slow wave sleep, and 18-25% of total sleep as REM sleep (7). During a normal night of sleep, the body cycles through approximately five 90-minute successions of REM and NREM sleep with the amount of deep sleep decreasing in each subsequent cycle (50). Conversely, REM sleep stages increase in duration and intensity as the end of the sleeping period approaches (36). In addition to EEG activity that resembles that which occurs during

wakefulness, REM sleep is characterized by atonia and movement of the eyes, and most memorable dreaming occurs during this stage. Both REM and deep sleep appear essential to memory (36), and research suggests that deep sleep plays a major role in physiological restoration, especially in relation to cardiovascular and endocrine function (7, 12). Specifically, secretion of human growth hormone is highest during deep sleep (47), and the parasympathetic branch of the autonomic nervous system dominates during this sleep stage, which may contribute to the relationship between deep sleep disruption and glucose intolerance (3, 7, 41, 44). Investigations that demonstrate alterations in sleep architecture concurrent with diseases such as obesity, diabetes, and hypertension support the importance of normal sleep architecture for health (12, 21, 35).

It is generally accepted that engaging in regular aerobic exercise can decrease the risk of cardiovascular disease (CVD) as well as improve quality of sleep (9, 49). Mind-body practices such as yoga, Tai Chi, and traditional Chinese exercise have also been shown to have beneficial effects on sleep (6, 23); however, the additional benefits of performing resistance exercise (RE) may render this mode a higher priority for certain populations, such as individuals with sarcopenia or osteoporosis. Resistance exercise may be used to attenuate the advance of sarcopenia by increasing muscle mass and strength in older adults, thus improving functional capabilities and delaying disability associated with the disease (29). Because of its potential to positively modify bone mineral density, resistance training has also been included in recent exercise recommendations for osteoporotic populations (16). In addition, orthopedic limitations may prevent aerobic exercise participation and make RE a more realistic option for certain individuals.

Resistance training interventions have previously been reported to alleviate anxiety and depression (2), both of which are common in college populations (20). According to a 2010 review (20) of mental health problems among college students, 15% of college students are diagnosed with depression, but fewer than a quarter of those diagnosed receive treatment. Anxiety disorders, including panic or generalized anxiety, are also prevalent in this population, with 10-12% of college students screening positive but less than 20% of those diagnosed receiving treatment (20). Disturbed sleep is a common affliction of depressed individuals (51). Furthermore, the presence of depressive symptoms concurrent with sleep disturbances is likely to lead to increased anxiety and impairments (33). The anxiolytic and antidepressant effects of both anaerobic and aerobic exercise are well-documented (2). Ten weeks of RE was previously found to improve depression as well as subjective sleep quality in older adults (39), suggesting that RE could provide a non-pharmacological means to reduce multiple associated complaints, especially for individuals who do not seek other forms of treatment. In addition to improving subjective sleep quality, RE has been found to positively alter sleep architecture (49). Compared to aerobic exercise, RE bouts take less time to complete, suggesting that this mode of exercise may be more compatible with the busy schedules of college students. For example, Ferris and colleagues (11) found that a light workout consisting of only one set of five exercises improved subjective sleep quality over three months.

Although different modes of exercise may result in similarly favorable alterations, timing of exercise is important. While at least one study has shown that vigorous late-night aerobic exercise does not impair sleep in highly trained athletes (52), growing evidence suggests that aerobic exercise performed late in the evening may negatively impact

subsequent sleep in untrained individuals (9, 10, 37, 46, 53). Previous research suggests that aerobically fit individuals, defined in the literature as those who engage in aerobic exercise for at least 20 minutes three times per week or have peak oxygen consumption (VO_{2peak}) values of at least 40 and 50 $mL \cdot kg^{-1} \cdot min^{-1}$ for females and males, respectively (53), exhibit more optimal sleep patterns than their sedentary counterparts (9) and that differences in training regimes influence sleep architecture (45). Fitness has been proposed to modulate the influence of exercise on sleep by shortening time required to recover from sympathetic nervous system arousal associated with exercise, although the evidence to support this theory is inconsistent (53). Nevertheless, untrained subjects should be examined to elucidate the acute effects of a bout of RE on sleep architecture. Presently, few investigations have studied the effects of RE on sleep in sedentary to recreationally active subjects, and very little information is available as to how timing of RE may influence sleep. Although RE has been shown to enhance sleep similarly to aerobic exercise (11, 39, 49), RE is commonly performed in the afternoon as opposed to early in the morning. Despite this, most studies examining the effects of RE on sleep have included interventions performed only in the morning (11, 49) or have not revealed the timing of the intervention (39), thus the literature regarding the effects of timing of RE on sleep in previously untrained individuals is limited. Because it is currently unknown how the timing of RE may affect objective quality of sleep, the purpose of this study was to examine the acute effects of timing of RE on sleep architecture in healthy college students. Based on previous research from our lab regarding aerobic exercise, we hypothesized that subjects engaging in RE at 7 a.m. would experience the most optimal sleep architecture as signified by decreased sleep onset latency (SOL), more time spent in REM and deep sleep, and less wake time after sleep onset (WASO).

METHODS

Participants. Normotensive to pre-hypertensive nonsmokers between the ages of 18-25 years were recruited from the student population at Appalachian State University. Subjects were sedentary to recreationally active, as defined by participation in no more than 150 minutes of any mode of planned exercise per week. To participate in the study, individuals had to meet additional inclusion criteria that included no orthopedic limitations to exercise, no history of CVD, and not taking any blood pressure or sleeping medications or aspirin therapy throughout the duration of the study. All study procedures were approved by the Appalachian State University Institutional Review Board, and all subjects gave their written informed consent prior to any involvement in the study.

Experimental protocol. Twenty-four participants attended all five visits required to complete the study, which lasted approximately 2 wk. The first visit was a non-RE control day (C) that consisted of anthropometric and blood pressure measurements and a treadmill maximal graded exercise test (GXT). Subjects were instructed to arrive for this visit well-hydrated and to eat as they normally would prior to exercise. After completing the health history questionnaire, blood pressure was measured manually using a standard stethoscope and sphygmomanometer after the subject had been seated for five minutes. Height and weight were then recorded without shoes or socks, and body fat percentage was determined using a foot-to-foot bioelectrical impedance analysis system (Model TBF-300A Body Composition Analyzer, Tanita Corporation of America, Inc., Arlington Heights, IL, USA). The GXT involved a modified Balke protocol to determine cardiorespiratory fitness. Briefly, after a 5-minute warm-up at 1.5 mph and 0% incline, subjects walked at 3.3 mph for 1-minute stages with grade increasing by 1% each stage. If 25% incline was reached, speed

then increased by 0.2 mph each stage. Subjects were encouraged throughout the test to continue the exercise until maximum exertion was reached, and the test ended when volitional exhaustion was attained. Gas exchange was measured throughout the GXT using a metabolic measurement system (TrueOne® 2400, Parvo Medics, Sandy, UT, USA), and heart rate was recorded telemetrically using a Polar heart rate monitor (Polar Electro Inc., Lake Success, NY, USA). Peak oxygen consumption and maximum heart rate (HR_{max}) were defined as the highest respective values obtained during the test using the 15-second averaging analysis setting.

During the second testing session, participants established a ten repetition maximum (10RM) on each of the nine RE machines to be used during the following visits. Exercises were performed on standard double-leg press, leg extension, hamstring curl, calf raise, abdominal crunch, triceps extension, biceps curl, lat pulldown, and chest press exercise machines. Determination of 10RM involved a warm-up set of ten repetitions at a self-selected weight followed by progressively heavier sets of ten repetitions separated by 2 minutes of rest. A 10RM was accepted as the weight at which the subject could perform ten but not more than ten repetitions.

Visits 3, 4, and 5 were performed at 7 a.m. (7A), 1 p.m., (1P), and 7 p.m. (7P), in a randomized, counterbalanced order. During each of these sessions, three sets of ten repetitions were performed at 65% of the individual's 10RM on each respective exercise machine. Each workout lasted approximately 30 minutes and was supervised by the researchers. Repetitions were counted to maintain consistent timing of concentric and eccentric phases of each exercise across participants. Thirty seconds to one minute of rest

were allotted between sets. At least 60 hours separated each visit 2, 3, and 4 from the subsequent visit.

An ambulatory wireless sleep-monitoring headband (Zeo Sleep Manager™, Zeo, Inc., Newton, MA, USA) was worn during sleep following C, 7A, 1P, and 7P. Use of this device, which was chosen due to its advantage of requiring minimal alterations to subjects' sleeping environments, has previously been validated in a healthy population (38). Participants were provided instructions for use at the conclusion of visit C, and sleep architecture data were collected the day following each session. To limit confounding influences, participants were instructed to avoid consumption of alcohol or caffeine on days of C, 7A, 1P, and 7P and to maintain their normal sleep-wake rhythm throughout the study. Caffeine and alcohol, which are both common drugs used by college students, have previously been shown to disturb sleep even when consumed several hours prior to habitual bedtime (8, 15). Participants were also asked not to take naps and to avoid participating in any other organized exercise on days of the study visits.

Statistical analysis. Sleep architecture data collected included SOL, number of times woken, total sleep time, time in REM, light, and deep sleep, and WASO. A 1 (group) × 4 (time) repeated measures analysis of variance (ANOVA) was conducted to detect significant differences between visits. If significance was detected, an appropriate post hoc comparison was then performed to determine where the differences occurred. Significance was set at $P < 0.05$ for all statistical analyses, and all data are presented as means ± SE. Analyses were completed using statistical software (IBM® SPSS® Statistics version 19, IBM Corporation, Armonk, NY, USA).

RESULTS

Physiological characteristics of subjects are presented in Table 1.

Analysis of sleep data revealed significant alterations in SOL, times woken, and WASO. Subjects experienced shorter time to fall asleep after 7A as compared to 1P (36 ± 5.2 min v. 57 ± 7.1 min, respectively) and 7P (71 ± 13.1 min; Figure 1A). All RE visits resulted in significantly fewer times woken during the sleep bout as compared to C (7A: 3 ± 0.5 times woken; 1P: 2 ± 0.5 times woken; 7P: 2 ± 0.5 times woken; C: 4 ± 0.8 times woken; Figure 1B), with 7P significantly decreasing WASO (5 ± 1.4 min v. 16 ± 4.1 min; Figure 1C). No significant differences in total, light, REM, or deep sleep were observed between visits.

DISCUSSION

Since the timing of exercise may influence sleep architecture, it is important to investigate differential exercise modes. This study was the first to examine the effects of performing a RE bout at various times of day on sleep architecture in a normotensive to pre-hypertensive population of college students. The main finding, that timing of RE did not significantly affect total, light, REM, or deep sleep, was contrary to our hypothesis; however, our results suggest that performing RE at any time of day enhances the ability to stay asleep as compared to not performing this mode of exercise. We also found evidence that lifting weights at 7 a.m. may be superior to other times of day in regard to diminishing SOL but that 7 p.m. may result in a subsequent bout of sleep with the least amount of time spent awake after initially falling asleep.

The finding that exercise timing did not affect sleep stages is interesting in comparison to previous literature that has investigated aerobic exercise timing in relation to sleep. Fairbrother and colleagues (10) recently demonstrated that aerobic exercise performed at 7 a.m. resulted in the highest sleep quality as determined by more deep sleep and less REM sleep. In a meta-analysis, researchers (53) reported moderate effects of acute aerobic exercise on deep, REM, and total sleep with exercise increasing, decreasing, and increasing these respective variables. However, the discrepancies between our results and the meta-analysis could arise from differences in exercise mode, as the only other study to assess objectively sleep quality following RE in healthy, untrained individuals saw a significant change in light sleep alone, with less light sleep occurring following the exercise intervention (49). Although we did not observe significant alterations in light sleep in the present study, our results are concurrent with the findings of the previous investigation in that RE did not

affect total, deep, or REM sleep as compared to a non-exercise control day. Some evidence suggests that total sleep duration is most related to exercise duration and that exercise bouts less than an hour produce negligible effects on total sleep duration (51, 53). Although aerobic exercise has been studied more extensively than RE in this regard, this finding could explain the absence of any changes in total sleep in the present study. It also calls into question the applicability of attempting to use RE to improve total sleep time in the general population.

When compared to the control visit, all exercise days resulted in significantly fewer times woken during the night. Similarly, Viana and colleagues (49) reported a lower arousal index in subjects who performed RE. Two studies that investigated the effects of resistance training on subjective sleep quality also found that sleep was improved following the exercise intervention (11, 39); however, it should be noted that each of these studies (11, 39, 49) included older adults and that older adults have been shown to exhibit longer SOL periods and more awakenings as compared to young adults (50). The inclusion of a depressed population (39) also limits the comparison of the current study with earlier research. Though many individuals regularly perform aerobic exercise, those who do not regularly engage in aerobic exercise because of health or other limitations could improve their ability to maintain sleep and concomitantly limit the risk of adverse health conditions such as CVD, diabetes, and depression, which have all been associated with poor or insufficient sleep, by incorporating RE into their weekly routines, thus supporting the role of exercise as an effective prophylactic health measure.

Morning exercise (7A) significantly improved time to fall asleep. This finding is in agreement with an earlier study in which subjects who exercised in the morning reported less

trouble falling asleep compared to evening exercisers (46). Aerobic exercise in the morning as compared to 1 p.m. and 7 p.m. was also found to diminish SOL when measured objectively (10). Although we attempted to minimize any alterations in the subjects' normal sleep-wake habits, the study design necessitated that subjects awakened prior to 7 a.m. for the 7A visit. As we studied college students, it is possible that this wake time was earlier than some of the subjects' habitual wake times, which could have contributed to the decreased SOL reported following 7A. One of the most popular theories offered to explain regulation of sleep includes the collaboration of a homeostatic drive for sleep and the circadian modulation of sleep (30). This model proposes that the homeostatic drive to sleep, or sleep pressure, increases directly with time spent in wakefulness and decreases during sleep and that this sleep pressure combined with the circadian sleep drive promotes maintenance of sleep during the biological night and wakefulness during the day. Based on this theory, it is possible that results would differ in participants who regularly awakened before 7 a.m. It is also important to note that, although C was not significantly different from any of the RE visits, SOL following each RE visit was less than SOL following C.

Evening exercise (7P) significantly reduced WASO. A previous review of the current literature (51) has also reported variations in SOL and WASO dependent on exercise timing, with the most beneficial effects occurring after subjects engaged in exercise 4 to 8 h prior to bedtime. However, these researchers also found that activities performed within 4 h of bedtime generally decreased WASO and slightly increased SOL. Our results for WASO are consistent with this review. Although not significant, we also saw increased SOL in the latest exercise time as compared to 7A and 1P. In addition, similar to our results for SOL,

we observed non-significant beneficial effects of RE on WASO, as each RE visit resulted in less WASO than C.

A major strength of the present study was our ability to evaluate sleep architecture within the subjects' usual home sleeping environments as opposed to in a laboratory setting. The use of a healthy, untrained population with no previously diagnosed sleep disorders also eliminated possible confounding factors, although the use of good sleepers may have limited the effects of exercise on sleep architecture. Chronic exercise training is thought to influence sleep architecture (9), so one could speculate that the multiple exercise sessions may have led to a training effect evident on the latter visit(s); however, we do not consider this to be a limitation of the current study due to the randomized order that visits were performed and the previous finding that the influence of training on sleep is not evident before eight weeks of training (37). Although we imposed controls by requiring subjects to abstain from caffeine and alcohol consumption, a limitation of the study design was that we did not assess compliance with these instructions. In addition, the order of visits, with C always occurring first, may have influenced our results. Specifically, this order effect may have been responsible for the large standard errors produced by the sleep architecture variables during the control visit (Figure 1); although preliminary testing by the research personnel revealed that the sleep-monitoring headband was comfortable and did not interfere with normal sleep, it is possible that some subjects experienced difficulty sleeping due to lack of previous familiarization with this device. This variation could have masked differences that would have otherwise been significant.

Mechanisms to explain the influence of exercise and exercise timing on sleep have included tissue restoration, energy conservation, temperature downregulation, and alterations

in secretion of endogenous compounds such as hormones or cytokines. Trinder and colleagues (45) provided evidence against the ideas that amounts of total and deep sleep are proportional to tissue restoration requirements and that sleep duration reflects daily energy expenditure, although the temperature downregulation hypothesis, which suggests that elevating body temperature through exercise prior to the onset of sleep facilitates sleep through the loss of heat and the associated mechanisms that affect sleep architecture, particularly deep sleep (9), remains a viable explanation (51). Fairbrother and colleagues (10) recently demonstrated that early morning aerobic exercise resulted in the highest sleep quality, possibly as a result of regulating the secretion of leptin or other hormones. Additional researchers have postulated that adding exercise improves sleep via increasing exposure to bright light, producing shifts in circadian rhythm as well as enhancing sleep through an antidepressant mechanism (9); however, this theory may be more relevant to aerobic exercise, which is more commonly completed outdoors, where lux values typically exceed 2500 (53), than to RE. It has also been suggested that cytokines and growth factors mediate sleep (9, 37). Specifically, interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α) have been implicated in sleep regulation because of their modulatory effects on body temperature and calcium release and observations that slightly elevated concentrations of these pro-inflammatory cytokines, as occurring acutely after low to moderate intensity exercise, promote drowsiness, while much higher levels of IL-6, such as the 100-fold increase seen after marathon completion, have the opposite effect and are associated with wakefulness (37). A recent comparison (25) of moderate versus high intensity cycling matched for external workload found significantly elevated plasma IL-6 following the high intensity bout both immediately (~10 v. ~ 7 pg/mL) and 1.5 h (~8 v. ~ 7

pg/mL) after the exercise. An investigation (34) of IL-6 levels in response to a moderate intensity RE protocol similar to that utilized in the present study showed an immediate average elevation to only 5.1 pg/mL in healthy sedentary subjects. Due to the similarities between protocols and given that IL-6 responses depend largely on exercise intensity and duration, the muscle mass recruited, and the fitness level of the individual (32), it is plausible to suggest that subjects in the present study may have demonstrated comparable IL-6 elevations. Furthermore, it is possible that a reduction in conditions that negatively affect sleep, such as obesity, depression, or anxiety, is somewhat responsible for the positive influence of exercise on sleep (37). Depression and anxiety are common mental health problems among college and college-aged populations (20), although we cannot make any assumptions to this regard about the students who volunteered for our study as we did not assess these conditions within the current study. At least one prior investigation of the effects of resistance training on quality of sleep speculated an increase in growth hormone or growth hormone-releasing hormone secretion could be a potential cause for the improvements they found, although these researchers did not measure levels of either hormone in the respective study (11). Growth hormone-releasing hormone has been identified as meeting all the criteria required to be classified as a regulating substance for NREM sleep (54), but the influence of growth hormone administration on sleep in humans is still inconclusive (17). Specific hormonal responses to exercise depend on factors such as intensity, total work, and rest periods, so without any relevant measures in the present study it is not possible to determine the magnitude of the hormonal response. However, prior research suggests that it is unlikely that the lower intensity of the protocol used would have dramatically increased growth hormone or modified the acute leptin response (24). It is

likely that some combination of the aforementioned conditions may explain the sleep improvements following RE reported in the literature and the alterations observed in this study, although the variables measured herein require that any discussion of causation remains speculative.

The present study has provided additional support for the potential of exercise, specifically RE, to improve sleep. A ceiling effect has been proposed in which individuals who are good sleepers experience minimal sleep architecture alterations in response to an intervention (9, 51); therefore, we selected to investigate the influence of RE on a population at-risk for depression and anxiety disorders but with no previously diagnosed sleep disorders in order to isolate the effects of RE while simultaneously acknowledging that the same intervention could produce a magnified response in individuals who suffer from some combination of sleep, depression, and anxiety disorders but are otherwise similar to the sample under study. These findings within a college-aged population warrant future investigations into the use of resistance training as a non-pharmacological means of enhancing sleep quality in groups that may suffer from both disturbed sleep and other conditions which may be alleviated by participation in a progressive RE intervention, such as osteoporosis, sarcopenia, depression, or anxiety. Further improvements or alterations in sleep architecture may become apparent only when participants are not good sleepers (9, 51), thus there is reason to believe that timing of RE could have a significant impact on sleep stages in a different population, possibly elucidating the optimal time of day to perform RE to facilitate restorative sleep.

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TABLES

Table 1. *Subject Characteristics*

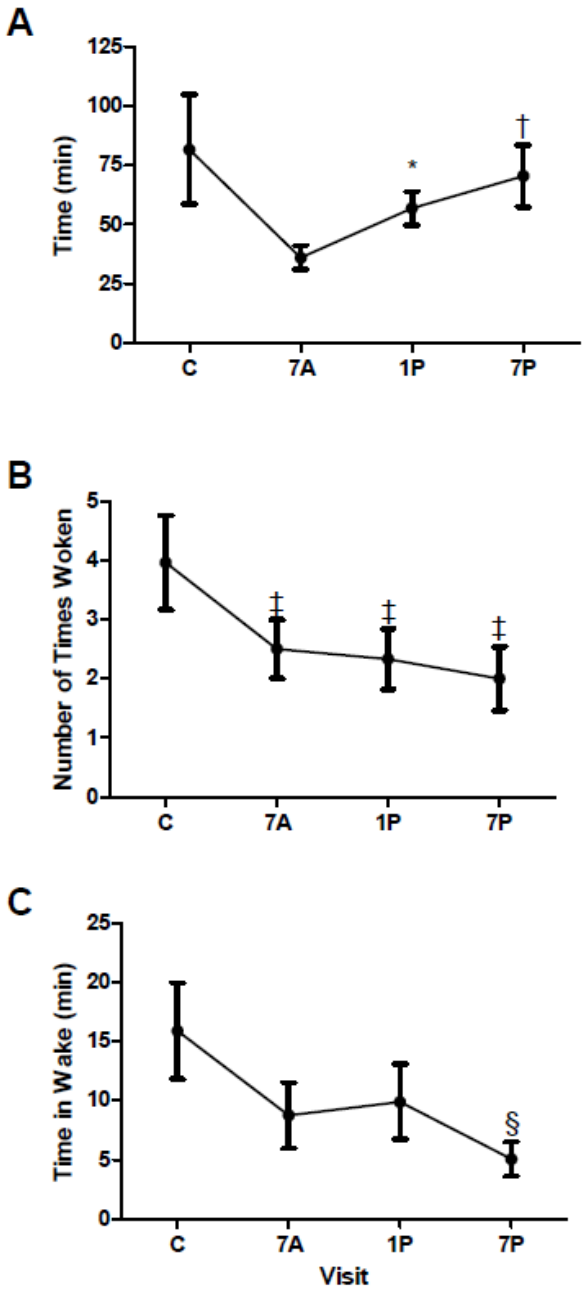
Variable	Mean	SE
Age (yr)	20	0.3
Height (cm)	171	2.1
Weight (kg)	72.7	3.2
BIA (%)	22.4	1.9
SBP (mm Hg)	118	1.7
DBP (mm Hg)	72	1.9
VO_{2peak} ($mL \cdot kg^{-1} \cdot min^{-1}$)	38.44	1.4
HR_{max} (bpm)	187	2.5

Data are from 24 healthy college students (n = 12 males). SE defines ± SE. BIA, bioelectrical impedance analysis; SBP, systolic blood pressure; DBP, diastolic blood pressure; VO_{2peak}, peak oxygen consumption; HR_{max}, maximum heart rate obtained during graded exercise test.

FIGURE CAPTIONS

Figure 1. Sleep onset latency (SOL; *A*), number of times woken (*B*), and wake after sleep onset (WASO; *C*) following control visit (C) and resistance exercise at 7 a.m. (7A), 1 p.m. (1P), and 7 p.m. (7P). **P* = 0.03, significant difference from 7A. †*P* = 0.02, significant difference from 7A. ‡*P* = 0.04, significant difference from C. §*P* = 0.01, significant difference from C.

Figure 1.


A
125
100
75
50
25
0
Time (min)
*
†
C
7A
1P
7P
B
Number of Times Woken
5
4
3
2
1
0
‡
‡
‡
C
7A
1P
7P
C
Time in Wake (min)
25
20
15
10
5
0
§
C
7A
1P
7P
Visit

Vita

Jessica Renee Alley was born in 1990 to Kay and Graylen Alley and grew up in New Castle, VA. She graduated from Craig County High School in June 2008 and entered Longwood University the following August. She received her Bachelor of Science in Kinesiology with a concentration in Exercise Science and a minor in Health Education in May 2012. In the fall of 2012, she accepted a research assistantship within the Exercise Science program at Appalachian State University and began working towards a Master of Science degree. In May 2012, Jessica graduated from Appalachian State University with an M.S. in Exercise Science with a concentration in Research.

Jessica is a member of Phi Kappa Phi and the American College of Sports Medicine. She plans to continue in academia after hiking the Appalachian Trail.