Effects Of Resistance Exercise Timing On Sleep Architecture And Nocturnal Blood Pressure

By: JESSICA R. ALLEY, JOHN W. MAZZOCHI, **CAROLINE J. SMITH**, DAVID M. MORRIS, AND SCOTT R. COLLIER

Abstract

Short sleep duration and poor quality of sleep have been associated with health risks including cardiovascu- lar disease, diabetes, and obesity. Prior research has sug- gested that regular aerobic exercise improves the 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 and nocturnal blood pressure. 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 on each of 9 RE machines, respectively. During visits 3–5, the subjects reported at 0700 hours (7A), 1300 hours (1P), and 1900 hours (7P) in a randomized order to perform 30 minutes of RE. Ambulatory blood pressure and sleep-monitoring devices were worn during sleep after C, 7A, 1P, and 7P. Time to fall asleep was significantly different between RE conditions 7A and 1P and between 7A and 7P. All exercise conditions exhibited significantly fewer times woken than the non-RE control day, with 7P resulting in signif- icantly less time awake after initially falling asleep as compared with C. Although timing of RE does not seem to statistically impact sleep stages or nocturnal blood pressure, these data indicate that engaging in RE at any time of the day may improve quality of sleep as compared with 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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Jessica R. Alley, John W. Mazzochi, Caroline J. Smith, David M. Morris, and Scott R. Collier

Vascular Biology and Autonomic Studies Laboratory, Department of Health and Exercise Science, Appalachian State University, Boone, North Carolina

Abstract

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Key Words sleep quality, resistance training, exercise biorhythms

Address correspondence to Scott R. Collier, colliersr@appstate.edu.

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Introduction

Short sleep duration is associated with an increased prevalence of adverse cardiovascular events including stroke, myocardial infarction, and congestive heart failure (1) and conditions of impaired glucose tolerance (32), increased cortisol levels (32), elevated blood pressure (21,33), and an increase in systemic inflammation (21,37), 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 (4,20). Prior research has suggested that 6–8 hours of sleep per night is optimal for health (1,3) and that individuals who consistently experience shorter sleep duration have a higher risk of all-cause mortality (3,11).

Although the 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 and can be divided into rapid eye movement (REM) and nonrapid eye movement (NREM) sleep. 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 (39). Conversely, REM sleep stages increase in duration and intensity as the end of the sleeping period approaches. Research suggests that deep sleep plays a major role in physiological restoration, especially in relation to cardiovascular and endocrine function (5,10). Specifi secre- tion of human growth hormone is the highest during deep sleep (36), and the parasympathetic branch of the autonomic nervous system dominates during this sleep stage (31). Nocturnal blood pressure dipping, which is classifi as a 10–20% reduction in blood pressure from daytime levels, also occurs primarily during deep sleep (31); however, this phenomenon is often absent in hypertensive individuals, a condition that is associated with an increased risk of developing serious cardiovascular complications (25). 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 (10,15,27).

It is generally accepted that engaging in regular aerobic exercise can decrease the risk of developing cardiovascular

disease and improve quality of sleep (7,38); however, the additional benefits of performing resistance exercise (RE) may render this mode a higher priority for certain populations, such as college students. Resistance training interventions have previously been reported to alleviate anxiety and depression (2), both of which are common in college populations (14). According to a 2010 review (14) 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 ,20% of those diagnosed receiving treatment (14). Disturbed sleep is a common affliction of depressed individuals (40). Further, the presence of depressive symptoms concurrent with sleep disturbances is likely to lead to increased anxiety and impairments (24). 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 and subjective sleep quality in older adults (30), suggesting that RE could provide a nonpharmacological 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 (38). Compared with 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 et al. (9) found that a light workout consisting of only 1 set of 5 exercises improved subjective sleep quality over 3 months.

Although different modes of exercise may result in similarly favorable alterations, timing of exercise is important. Although at least 1 study has shown that vigorous late-night aerobic exercise does not impair sleep in highly trained athletes (41), growing evidence suggests that aerobic exercise performed late in the evening may negatively impact subsequent sleep in untrained individuals (7,8,28,35,42). Previous research suggests that aerobically fit individuals, defined in the literature as those who engage in aerobic exercise for at least 20 minutes 3 times per week or have peak oxygen consumption ($\dot{V}O_2$peak) values of at least 40 and 50 ml\$kg^{21}\$min^{21} for women and men, respectively (42), exhibit more optimal sleep patterns than do their sedentary counterparts (7) and that differences in training regimes influence sleep architecture (34). Fitness has been proposed to modulate the influence of exercise on sleep by shortening the time required to recover from sympathetic nervous system arousal associated with exercise, although the evidence to support this theory is inconsistent (42). 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 (9,30,38), 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 (9,38) or have not revealed the timing of the intervention (30); thus, the literature regarding the effects of timing of RE on sleep in previously untrained individuals is limited. In addition, the blood pressure dipping response characteristic of restorative deep sleep may vary depending on the time of the day that the exercise is performed. It has previously been reported that the hypotensive response to aerobic exercise is greater after exercise in the evening as compared with that during the morning hours (16), although, to our knowledge, no previous study has examined the response of nocturnal blood pressure to RE performed at different times of the day. Because it is currently unknown how the timing of RE may affect both the objective quality of sleep and nocturnal blood pressure, the purpose of this study was twofold: to examine the acute effects of timing of RE on sleep architecture in healthy college students while simultaneously determining the effects on nocturnal blood pressure. Based on previous research from our laboratory regarding aerobic exercise, we hypothesized that subjects engaging in RE at 0700 hours 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), and that there would be differences in the nocturnal blood pressure corresponding to exercise timing.

Methods

Experimental Approach to the Problem

The study design included 5 visits and lasted approximately 2 weeks. Visits included a first testing session to establish baseline anthropometric and cardiovascular parameters and to familiarize participants with equipment, a second testing session to establish the load for the exercise during the remaining visits, and 3 testing sessions during which subjects arrived for exercise at 3 different times of day. Sleep architecture and nocturnal blood pressure data were collected after these visits to test the corresponding hypotheses.

Subjects

Normotensive to prehypertensive nonsmokers between the ages of 18 and 25 years were recruited from the student population at the Appalachian State University (Table 1). The subjects were sedentary to recreationally active, as defined by participation in not .150 minutes of any mode of structured 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

TABLE 1. Subject characteristics.*†

Variable	Mean	*SE*
Age (yrs)	20	0.3
Height (cm)	171	2.1
Weight (kg)	72.7	3.2
BIA (%)	22.4	1.9
Resting SBP (mm$Hg)	118	1.7
Resting DBP (mm$Hg)	72	1.9
$\dot{V}O_2$peak (mlkg^{21}min^{21})	38.44	1.4
HRmax (b$min^{21})	187	2.5

*BIA = bioelectrical impedance analysis; SBP = systolic blood pressure; DBP = diastolic blood pressure; $\dot{V}O_2$peak = peak oxygen consumption; HRmax = maximum heart rate obtained during the graded exercise test.

†Data are from 24 healthy college students. *SE* defines 6*SE*.

Appalachian State University Institutional Review Board, and all the subjects gave their written informed consent before any involvement in the study. Twenty-four partici- pants completed the study (n = 12 men).

Procedures

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). The subjects were instructed to arrive for this visit well hydrated and to eat as they normally would before exercise. After completing the health history questionnaire, resting blood pressure was measured manually using a standard stethoscope and sphygmomanometer after the subject had been seated quietly for 5 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 2.41 kph and 0% incline, the subjects walked at 5.31 kph for 1-minute stages with grade increasing by 1% each stage. If a 25% incline was reached, speed was then increased by 0.2 mph each stage. The 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 (HRmax) were defined as the highest respective values obtained during the test using the 15-second averaging analysis setting.

During the second testing session, the participants established a 10-repetition maximum (10RM) on each of the 9 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 10 repetitions at a self-selected weight followed by progressively heavier sets of 10 repetitions separated by 2 minutes of rest. A 10RM was accepted as the weight at which the subject could perform 10 but not .10 repetitions.

Visits 3–5 were performed at 0700 hours (7A), 1300 hours (1P), and 1900 hours (7P) in a randomized, counterbalanced order. During each of these sessions, 3 sets of 10 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 1 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 after C, 7A, 1P, and 7P. The use of this device, which was chosen because of its advantage of requiring minimal alterations to subjects' sleeping environ- ments, has previously been validated in a healthy population (29). Nocturnal blood pressure was recorded with an Oscar 2 ambulatory blood pressure monitor (SunTech Medical, Morrisville, NC, USA) that subjects also wore during sleep after C, 7A, 1P, and 7P. The use of this device has also been previously validated (17). The participants were provided instructions for the use of both devices at the conclusion of visit C, and sleep architecture and blood pressure data were collected the day after each session. To limit confounding infl , the 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 before habitual bedtime (6,12). The participants were also asked not to take naps and to avoid participating in any other organized exercise on the days of the study visits.

Statistical Analyses

Sample size calculations were performed using data from a previous study conducted in our laboratory (8). The a priori power analysis (G*Power 3, Heinrich Heine University Dü sseldorf, Dü sseldorf, Germany) determined that 24 subjects were needed to reach signifi ance with a power of 0.8 at an alpha level of 0.05. Descriptive statistics were determined by univariate calculations. Sleep architecture

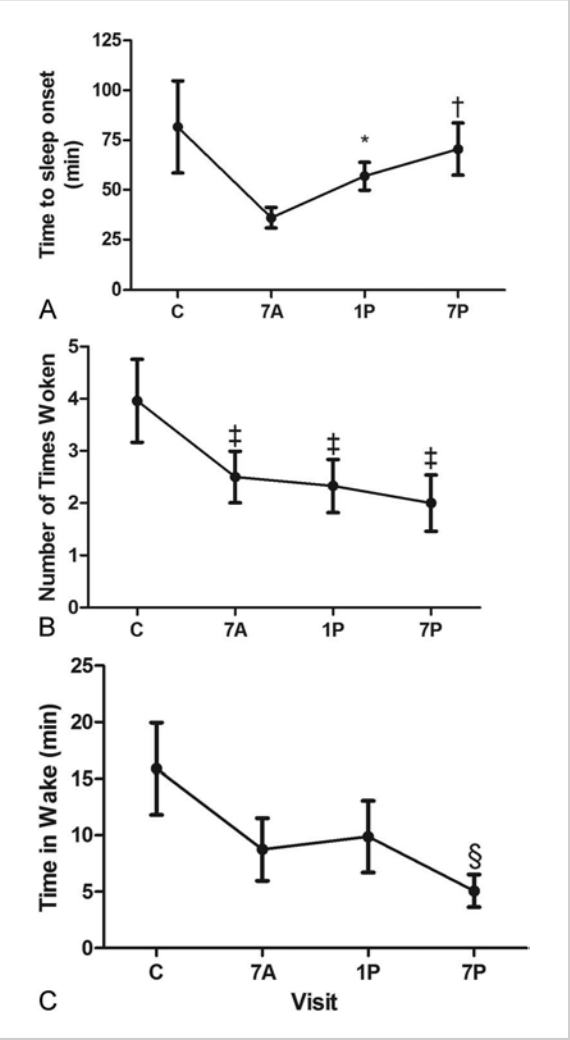

Figure 1. Sleep onset latency (SOL; A), number of times woken (B), and wake after sleep onset (WASO; C) after Control visit (C) and resistance exercise at 0700 hours (7A), resistance exercise at 1300 hours (1P), and resistance exercise at 1900 hours (7P). *p = 0.03, Significant difference from 7A. †p = 0.02, Significant difference from 7A. ‡p = 0.04, Significant difference from (C) §p = 0.01, Significant difference from (C).

data collected included SOL, number of times woken, total sleep time, time in REM, light, and deep sleep, and WASO. Blood pressure data analyzed included nocturnal means for systolic blood pressure (SBP) and diastolic blood pressure (DBP). A 1 (group) 3 4 (time) repeated measures analysis of variance was conducted to detect significant differences between visits. If significance was detected, an appropriate post hoc comparison was then employed to determine where the differences occurred. Significance was set at $p \# 0.05$ for all statistical analyses, and all data are presented as mean 6 *SE*. Analyses were completed using statistical software (IBM SPSS, Statistics version 19, IBM Corp., Armonk, NY, USA).

TABLE 2. Mean nocturnal blood pressure.*†

	SBP (mm$Hg)		DBP (mm$Hg)	
Visit	Mean	*SE*	Mean	*SE*
C	115	2.7	59	1.4
7A	119	3.3	60	1.9
1P	117	3.6	58	1.5
7P	116	3.1	59	1.2

*SBP = systolic blood pressure; DBP = diastolic blood pressure; C = control visit with no resistance exercise; 7A = resistance exercise at 0700 h; 1P = resistance exercise at 1300 h; 7P = resistance exercise at 1900 h.
†Data are from 23 healthy college students. *SE* defines 6*SE*.

RESULTS

Analysis of sleep data revealed significant alterations in SOL, times woken, and WASO. The subjects experienced shorter time to fall asleep after 7A as compared with that after 1P (36 6 5.2 minutes vs. 57 6 7.1 minutes, respectively) and 7P (71 6 13.1 minutes; Figure 1A). All RE visits resulted in significantly fewer times woken during the sleep bout as compared with that for C (7A: 3 6 0.5 times woken; 1P: 2 6 0.5 times woken; 7P: 2 6 0.5 times woken; C: 4 6 0.8 times woken; Figure 1B), with 7P significantly decreasing WASO (5 6 1.4 minutes vs. 16 6 4.1 minutes; Figure 1C). No significant differences in total, light, REM, or deep sleep were observed between visits.

Analysis of nocturnal blood pressure means revealed no significant differences between any of the visits for SBP or DBP, although both SBP and DBP were elevated after 7A as compared with those for the other visits (Table 2). One subject was excluded from the blood pressure analyses because of lack of data.

DISCUSSION

Because 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 an RE bout at various times of the day on sleep architecture and nocturnal blood pressure in a normotensive to prehypertensive population of college students. The main finding, that the timing of RE did not significantly affect either mean nocturnal blood pressure or total, light, REM, or deep sleep, was contrary to our hypothesis; however, our results suggest that performing RE at any time of the day enhances the ability to stay asleep as compared with not performing this mode of exercise. We also found evidence that lifting weights at 0700 hours may be superior to lifting weights during other times of the day in regard to diminishing SOL but that the same exercise at 1900 hours 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 with the findings in the literature that has investigated aerobic exercise timing in relation to sleep. Fairbrother et al. (8) recently demonstrated that aerobic exercise performed at 0700 hours resulted in the highest sleep quality as determined by more deep sleep and less REM sleep. In a meta-analysis, researchers (42) reported moderate effects of acute aerobic exercise on deep, REM, and total sleep with exercise increasing, decreasing, and increasing these respective variables. However, the discrep- ancies between our results and those of the meta-analysis could arise from differences in exercise mode, because the only other study to objectively assess sleep quality after RE in healthy, untrained individuals saw a significant change in light sleep alone, with less light sleep occurring after the exercise intervention (38). Although we did not observe significant alterations in light sleep in this 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 with a nonexercise 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 (40,42). Although aerobic exercise has been studied more extensively than RE in this regard, this finding could explain the absence of any changes in the total sleep in this study.

When compared with the control visit, all exercise days resulted in significantly fewer times woken during the night. Similarly, Viana et al. (38) 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 after the exercise intervention (9,30); however, it should be noted that each of these studies (9,30,38) included older adults and that older adults have been shown to exhibit longer SOL periods and more awakenings as compared with that exhibited by young adults (39). The inclusion of a depressed population (30) also limits the comparison of this study with earlier research.

Morning exercise (7A) significantly improved time to fall asleep. This finding is in agreement with that of an earlier study in which subjects who exercised in the morning reported less trouble falling asleep compared with evening exercisers (35). Aerobic exercise in the morning as compared with 1300 and 1900 hours was also found to diminish SOL when measured objectively (8). Although we attempted to minimize any alterations in the subjects' normal sleep–wake habits, the study design necessitated that subjects awakened before 0700 hours for the 7A visit. Because 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 after 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 (22). 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 0700 hours.

Evening exercise (7P) significantly reduced WASO. A previous review of the current literature (40) has also reported variations in SOL and WASO dependent on exercise timing, with the most beneficial effects occurring after subjects engaged in exercise 4–8 hours before bedtime. However, these researchers also found that activities performed within 4 hours of bedtime generally decreased WASO and slightly increased SOL. Our results for WASO are consistent with the findings of this review. Although not significant, we also saw increased SOL in the latest exercise time as compared with 7A and 1P. In addition, similar to our results for SOL, we observed nonsignificant beneficial effects of RE on WASO, because each RE visit resulted in less WASO than C.

A major strength of this 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 (7), 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 this study because of the randomized order that visits were performed and the previous finding that the influence of training on sleep is not evident before 8 weeks of training (28). 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 because of lack of previous familiarization with the monitoring devices. This variation could have masked differences that would have otherwise been significant.

In regard to mean nocturnal blood pressure assessments in this study, it should be noted that although there were no significant differences in SBP or DBP after any of the visits, both mean nocturnal SBP and mean nocturnal DBP were

the highest after the 7A exercise condition, suggesting a trend toward higher nocturnal blood pressures after RE in the early morning hours. Park et al. (25) reported a greater drop in nocturnal SBP in nondippers as compared with that in dippers after evening (1700–1900 hours) aerobic exercise. A limitation of the blood pressure data collected in this study includes our failure to measure ambulatory blood pressure throughout the day. This information would have allowed us to validate the dipping status of the normotensive to prehypertensive individuals included in the study and to evaluate the influence of the time of the day of RE on dippers as compared with nondippers. Further research should also investigate the possibility of deleterious blood pressure effects of RE performed in the early morning when values are known to be elevated.

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 et al. (34) 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 before the onset of sleep facilitates sleep through the loss of heat and the associated mechanisms that affect sleep architecture, particularly deep sleep (7), remains a viable explanation (40). Fairbrother et al. (8) 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, and produces shifts in circadian rhythm and enhances sleep through an antidepressant mechanism (7); however, this theory may be more relevant to aerobic exercise, which is more commonly completed outdoors, where lux values typically are .2500 (42), than to RE. It has also been suggested that cytokines and growth factors mediate sleep (7,28). Specifically, interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha 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 proinflammatory cytokines, as occurring acutely after low- to moderate-intensity exercise, promote drowsiness, whereas 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 (28). A recent comparison (19) of moderate- vs. high-intensity cycling matched for external workload found significantly elevated plasma IL-6 after the high-intensity bout both immediately (;10 vs. ;7 pg$ml^{21}) and 1.5 hours (;8 vs. ;7 pg$ml^{21}) after the exercise. An investigation (26) of IL-6 levels in response to a moderate-intensity RE protocol similar to that used in this study showed an immediate average elevation to only 5.1 pg$ml^{21} in healthy sedentary subjects. Because of 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 (23), it is plausible to suggest that subjects in this study may have demonstrated comparable IL-6 elevations. Further, 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 (28). Depression and anxiety are common mental health problems among college and college-aged populations (14), although we cannot make any assumptions in this regard about the students who volunteered for our study because we did not assess these conditions within this study. At least 1 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 (9). Growth hormone–releasing hormone has been identified as meeting all the criteria required to be classified as a regulating substance for NREM sleep (43), but the influence of growth hormone administration on sleep in humans is still inconclusive (13). Specific hormonal responses to exercise depend on factors such as intensity, total work, and rest periods, so without any relevant measures in this 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 (18). It is likely that some combination of the aforemen- tioned conditions may explain the sleep improvements after RE reported in the literature and the alterations observed in this study, although the variables measured here require that any discussion of causation remains speculative.

This 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 (7,40); therefore, we selected to investigate the influence of RE on a population prone to depression and anxiety disorders but with no previously diagnosed sleep, depression, or anxiety disorders to isolate the effects of RE while 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 nonpharmacological means of enhancing sleep quality in groups that may suffer from both disturbed sleep and other conditions that may be alleviated by participation in a progressive RE intervention, such as

osteoporosis, sarcopenia, depression, or anxiety. In addition, orthopedic limitations may prevent aerobic exercise participation and make RE a more realistic option for certain older individuals. Further improvements or alterations in sleep architecture may become apparent only when participants are not good sleepers (7,40); 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 opti- mal time of day to perform RE to facilitate restorative sleep.

Practical Applications

By measuring objective nocturnal sleep quality after RE performed at various times of the day, we have demonstrated that RE sessions at certain times of the day may assist in sleep goals such as shortening time to fall asleep or decreasing the amount of time spent awake during the night. Therefore, practitioners should urge clients who struggle to fall asleep to work out earlier in the morning, whereas those who struggle to stay asleep may benefit more from evening strength training sessions. Stressing the sleep-enhancing advantages of RE is also critical. Although 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 developing 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; this thus supports the role of exercise as an effective prophylactic health measure. Although future research is needed to determine if nocturnal blood pressure is detrimentally elevated after early morning RE compared with RE at other times of the day, practitioners may choose to advise prehypertensive individuals against performing this type of exercise early in the day as lower nocturnal blood pressures have been shown to contribute to better cardiovascular health.

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