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Exercise Training and Sleep Quality in Young Adults from the Training Interventions and Genetics of Exercise Response (TIGER) Study

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Exercise Training and Sleep Quality in Young Adults from the Training Interventions and Genetics of Exercise Response (TIGER) Study

by

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Report

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Dedication

I dedicate this work to my brother, Isaiah, who has been a source of constant support, love, and wisdom in my life. I could not have survived the ups and downs of life without your unwavering faith and love.
Acknowledgements

I acknowledge my advisor and role model, Molly Bray for her support and never accepting less than perfect work.
Abstract

Exercise Training and Sleep Quality in Young Adults from the Training Interventions and Genetics of Exercise Response (TIGER) Study

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Study Objectives. Sleep is regulated by internal mechanisms that respond to environmental cues. Physical activity is one external cue that can affect sleep. It has been suggested that exercise affects sleep in a variety of ways, including influencing neurotransmitter levels and altering circadian rhythms. The purpose of this study was to examine self-reported sleep quality both before and after a well-defined exercise protocol.

Design, Setting, and Participants. The TIGER study involves a 15-week aerobic exercise intervention conducted in young adults (n=2,027, mean age 21.8 ± 5 y). Participants were required to engage in 30 minutes of aerobic exercise at 65-85% maximal heart rate reserve three times/week. Multivariate regression was used to identify factors associated with sleep quality and duration.
Measurements and Results. Multiple measures of body size/composition, heart rate (HR), and blood pressure (BP) were obtained on all participants. Sleep quality and duration were accessed via a condensed sleep quality profile (SQP). Prior to exercise, age (p<0.001), gender (p<0.008) and overweight/obesity status (p<0.001), but not race/ethnicity, were all significantly associated with SQP score. Age (p<0.002), and race/ethnicity (p<0.05) were significantly associated with sleep duration, with African Americans and Hispanics having significantly shorter sleep times compared to non-Hispanic whites. SQP score was not significantly different following chronic exercise training.

Conclusions. Although overweight/obesity groups had significantly different sleep quality scores before and after exercise, sleep quality did not change for subjects after 15 weeks of aerobic exercise intervention.

Key Words: circadian, clock, aerobic exercise, anthropometrics, rhythm
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Introduction

Sleep is a compulsory human function for tissue repair and metabolism. Health can be negatively impacted if sleep is asynchronous with daily light/dark cycles or if sleep efficiency/quality is impaired\(^1\). In our modern 24-hour lifestyle, poor and inadequate sleep is commonplace, particularly among college students, shift workers, or the diseased state\(^2\)\(^3\)\(^-\)\(^8\). Specific physiological conditions can contribute to poor sleep quality or be exacerbated by decreased sleep\(^3\)\(^-\)\(^8\). Overall sleep quality can be affected by disease, age, health, sex, and fitness level\(^6\),\(^7\),\(^9\)\(^-\)\(^12\).

Sleep quality is regulated by external and internal control mechanisms. External mechanisms include photic and non-photic entrainment of the central and peripheral molecular circadian machinery. Internal control is driven by intrinsic molecular circadian clock mechanisms that are found in virtually every cell in the body and regulate many metabolic circadian rhythms. If external or internal stimuli are altered by disease, irregular behavioral schedules, medication, stress, or other factors, poor sleep quality can result. Sub-optimal sleep quality may, in turn, lead to disease and/or physiological impairment\(^13\),\(^14\).

Although improvement of sleep with exercise has been observed in several studies\(^15\), some studies show the opposite or no effect\(^16\). Improvement in sleep quality has been
shown when exercise occurs late in the day or is of high intensity\textsuperscript{15}. Sleep quality is less influenced by exercise for fit individuals habituated to exercise and for males compared to females\textsuperscript{15}. Frequently discussed reasons for discrepancies in the literature regarding the effects of exercise on sleep include variability in time of day and type of exercise, relative physical fitness of the individual, medication and caffeine usage, psychological stress, external environment and morningness/eveningness profile\textsuperscript{3, 13, 17-20}. Most studies have investigated the effect of acute exercise (48hrs-14days) on sleep quality\textsuperscript{21}. It is currently unknown how longer durations of exercise impact sleep quality. The purpose of this study is to examine factors that influence sleep quality both before and after 15 weeks of exercise training in young adults.
Methods

Participants. Participants in this study were drawn from the Training and Interventions and Genetics of Exercise Response (TIGER) Study, a prospective cohort study designed to examine the effects of genetic variation on exercise response and adherence to aerobic exercise. The target subjects for the TIGER study included healthy sedentary men and women, 18-35 years of age, who had not been regularly physically active for at least 30 days prior to participation in the study. Participants were excluded from the study if they were pregnant, lactating, and/or had metabolic disease known to influence body composition.

All participants were exposed to 30 minutes of aerobic exercise three times a week for up to 15 weeks as part of a course for college credit. Participants chose from a variety of aerobic exercise options, including arm ergometry, elliptical stepping, rowing, running, stair stepping, stationary cycling, track running/walking, or treadmill stationary cycling. Participants were required to engage in 30 min of exercise at their age- and gender-specific target heart rate of 65%-85% of maximal heart rate reserve (HRR). The TIGER Study has been approved by the Institutional Review Boards of the University of Texas at Austin, the University of Alabama at Birmingham, the University of Houston, and Baylor College of Medicine. All participants completed written informed consent prior to participation.
**Subject Measures.** At the beginning and the end of each 15 week study period, subjects underwent a brief physical exam, which included body composition, anthropometrics, venipuncture, and resting blood pressure and heart rate measurements. Dual energy x-ray absorptiometry (DXA) was utilized to estimate bone mineral content, lean mass, and fat mass (Hologic Delphia-A unit adult whole-body software v.11.2; and Hologic Discovery W Instrument adult whole-body, software QDR v.12.3). A portable stadiometer (Seca Road Rod) was used to measure height and a digital scale (Seca 770, Hanover, MD) was utilized to record body weight. Males had skinfold measures recorded at the chest, abdomen, and thigh, and females had recordings at the triceps, iliac crest, and thigh (Beta Technology, Santa Cruz, CA or Lafayette Instruments, Lafayette, LA) using established protocols. Three measurements were taken for each body site and the average was used for data analysis. Three measurements were also taken and the last two measures averaged for blood pressure and resting heart rate, measured using digital blood pressure monitors (Omron HEM-907XL, Bannockburn, IL). At the beginning of the study and after 15 weeks of the intervention, 10 ml of whole blood samples were taken by trained phlebotomists for the purpose of blood and DNA analysis. A battery of self-report questionnaires was administered to all subjects at the beginning and end of the 15-week exercise period. The measures included a complete medical history, eating behavior, dietary intake, weight history, exercise efficacy and motivation, sleep quality, and other measures related to exercise response and adherence.
**Exercise Measures.** To quantify total exercise dose, duration for each session was adjusted for average exercise intensity (percent HRR) to create a measure of intensity-minutes for each workout, which were summed over all exercise sessions to formulate the heart rate physical activity score (HRPAS)\(^\text{22}\). Because total available sessions for each cohort varied due to differences in semester length and weather-related university closures, a normalized HRPAS, adjusted for the different numbers of possible workouts in each cohort, was also calculated.\(^\text{22}\). Previously we have shown that HRPAS is correlated with significant alterations in multiple physiological measures following aerobic exercise training\(^\text{22}\).

**Sleep measures.** Sleep quality is generally defined by feelings of restfulness or restoration upon waking and alertness throughout the day\(^\text{23}\). Sleep quality can be assessed via measures of sleep duration, sleep latency, number of arousals and depth of sleep\(^\text{24}\). Sleep quality and quantity were assessed for all subjects using a short online questionnaire developed for use in the TIGER Study. The TIGER Sleep Quality Profile (SQP) included a total of ten questions related to general components of sleep quality such as sleep impairments due to limb movement or disordered breathing. The SQP also evaluated self-reported time spent sleeping on weekends and weekdays (see Appendix 1). Each question was given a score of 0 or 1, corresponding to no or yes respectively. The weighted average of weekday and weekend sleep values was calculated and scored
on a scale from 0 to 3. The scores were summed to provide an index of sleep quality ranging from 0 to 12, with a higher score representative of greater sleep impairment.

In order to validate the SQP, a subset of the cohort completed the Pittsburg Sleep Quality Index (PSQI), a 19 question self-rated questionnaire, originally developed by Bussey et al., which has been widely used to document sleep quality for a broad range of individuals. The PSQI measures components related to sleep quality and duration over the past month by subject recall. Both the SQP and PSQI were administered via online questionnaires. The PSQI consists of 19 self-rated questions, which resulted in seven component scores measuring sleep quality, sleep latency, sleep duration, habitual sleep efficiency, use of sleeping medications, sleep disturbances, and daytime dysfunction. These component scores were summed to give an overall global score ranging from 0 to 21. The PSQI has been proven to be both stable (Cronbach’s alpha = 0.82-0.89) and valid (80-100% relatable to clinical diagnosis of insomnia), and thus serves as a gold standard criterion measure for validation of the SQP. For both questionnaires, the weighted average of weekday and weekend sleep values was calculated and scored according to the PSQI scoring protocol.

**Statistical Analysis**

All statistical analyses were performed using STATA version 13 (Statacorp Inc., College Station, Texas), and all analyses were limited to non-Hispanic white, Hispanic, African
American, and Asian subjects. Histograms, normal probability plots, central tendency and variability measures were used to examine the distributions and create descriptive statistics for sleep quality measures, anthropometric measures, blood pressure, resting heart rate, percent body fat, and fasting glucose. In order to examine the internal validity of the SQP, Cronbach’s alpha was calculated for SQP and PSQI scores, and linear regression was used to examine the relationship between SQP score and both total and component scores of the PSQI.

Using SQP score as the dependent variable, multivariate regression was used to examine the association between baseline sleep quality and physiological outcomes, including BMI, percent body fat (%Fat), waist and hip circumferences, resting systolic (SBP) and diastolic blood pressure (DBP), resting heart rate, estimated aerobic capacity, and fasting glucose, adjusted for age, gender, and race/ethnicity. Repeated measures analysis of variance was used to examine the associations between change in sleep quality and exercise frequency, intensity, and total exercise dose (HRPAS), adjusted for age, gender, race/ethnicity, and baseline value of the SQP score. Likelihood ratio tests were used to evaluate the significance of full versus reduced models, in order to identify the most parsimonious set of predictors of SQP. Post hoc analyses, stratified by gender and overweight/obesity status, were calculated for models in which these variables were significant predictors. For all tests, statistical significance was set at p<0.05.
Results

Subject sample. A total of 2,027 participants completed the SQP at baseline, and descriptive statistics for this subject sample are included in Table 1. The distribution of the subject sample by race/ethnicity was 971 non-Hispanic whites, 248 Hispanic, 646 African Americans, and 162 Asians. The mean age of participants was 21.8 ± 5.0 y.

Validation of the SQP. The total scores of SQP at baseline ranged from 0-12 (mean: 3.1 ± 2.0). SQP scores were significantly correlated to PSQI scores ($F(1, 469)=632.9$, $r^2=0.57$). Stepwise regression was used to examine whether additional measures would increase the amount of variance in SQP accounted for by PSQI scores; only race/ethnicity was found to increase the significance of the prediction model ($\chi^2=11.9$, $p<0.007$). Cronbach’s alpha for the total SQP and total PSQI scale was 0.93, indicating strong agreement between the two measures. Because the SQP is designed to capture a summary measure of total sleep quality, Cronbach’s alpha for the SQP and each of the seven sub-scales (i.e. component scores) of the PSQI were lower, ranging from 0.03 for “habitual sleep efficiency” to 0.55 for “sleep duration.”

Predictors of baseline sleep quality. Based on the published literature, age, gender, race/ethnicity, and obesity status have all been reported to be associated with sleep quality. Thus, we first examined these factors for association to SQP in our subject sample using hierarchical stepwise forward multivariate regression. Among all subjects,
age (p<0.001), gender (p<0.008) and overweight/obesity status (p<0.001), but not race/ethnicity, were significantly associated with SQP score.

Poorer sleep quality has been reported for those with higher body weight compared to lower body weight and for females compared to males, which may be driven by hormonal differences. Using stepwise regression followed by multivariate regression, the analyses were stratified by gender (Table 2). For both males and females, age and DBP were significantly associated with SQP score. For males, additional predictors of sleep quality included BMI and hip circumference. For females, height and skinfold thickness were also significantly predictive of SQP score.

Both sleep duration (p<0.03) and SQP score (p<0.001) were significantly associated with obesity status (Figure 1), with obese individuals having the poorest sleep quality (SQP score for normal weight - 2.81 ± 2.09, overweight - 3.13 ± 2.23, obese - 3.46 ± 2.33) and the shortest sleep duration (sleep duration [hrs] for normal weight – 7.26 ± 1.2; overweight - 7.20 ± 1.2, obese - 7.1 ± 1.3). In analyses stratified by overweight/obesity status for all subjects (Table 3), no single variable was associated with SQP score in all overweight/obesity groups. Age was associated with SQP score in normal and overweight subjects, height was associated with SQP score in overweight and obese subjects, and skinfold thickness was associated with sleep quality in normal and obese subjects but not in overweight subjects. Hip circumference was uniquely associated with SQP in normal weight subjects only, while DBP was associated with SQP in obese
Figure 2 depicts mean SQP score by gender and overweight/obesity status. In females but not males, mean SQP score was significantly higher (p<0.001) in obese participants (3.6 ± 2.3) compared to normal weight (2.9 ± 2.1) or overweight (3.2 ± 2.3) individuals. Analyses stratified by both gender and overweight/obesity status are shown in Table 4. Skinfold thickness was the most consistent predictor of sleep quality across all gender and obesity status groups, with the exception of overweight females. Age, hip circumference, and skinfold thickness were significantly associated with sleep quality in normal weight males. Only percent body fat and skinfold thickness were significantly related to SQP score in overweight males, while WHR, percent body fat, and skinfold thickness were significantly related to SQP score in obese males. For normal weight females, glucose and percent body fat were inversely correlated to SQP score, while skinfold thickness was positively associated with sleep quality. In overweight female subjects, age and BMI were significantly associated with SQP score, while in obese female subjects, DBP and skinfold thickness were significantly associated with SQP score.

Predictors of baseline sleep duration

As expected, SQP score was significantly related to sleep duration (p<0.001), with sleep duration explaining approximately 25% of the variation in SQP scores. In the same
manner as analyzing sleep quality (SQP score), the potential relationship of sleep duration to age, gender, race/ethnicity, and overweight/obesity status was examined. In multivariate regression, the model \( F(6,2078) = 10.82, p<0.001 \) showed that age (\( p<0.002 \)), and race/ethnicity (\( p<0.05 \)) were significantly related to sleep duration, while gender and overweight/obesity status were non-significant. Mean sleep duration by race/ethnicity is shown in Figure 3. Average sleep duration for African Americans (6.97 ± 1.25 h) and Hispanics (7.06 ± 1.09 h) was significantly lower (\( p<0.001 \)) than for non-Hispanic whites (7.36 ± 1.22 h) but not different from Asians (7.18 ± 1.08 h). These results were independent of gender or overweight/obesity status.

**Predictors of change in sleep quality**

Following aerobic exercise training, only overweight/obesity status was significantly associated with SQP score (\( p<0.002 \)), with obese individuals having the poorest sleep quality score (3.3 ± 2.4) compared to normal weight (2.7 ± 2.1) or overweight (2.9 ± 2.3) subjects. For sleep duration, age (\( p<0.04 \)) and race/ethnicity (\( p<0.001 \)) remained significantly associated following exercise training. African Americans had significantly shorter sleep times (6.8 ± 1.1 h) compared to all other racial/ethnic groups (7.3 ± 1.1, 7.2 ± 1.1, and 7.4 ± 1.0 for non-Hispanic whites, Hispanics, and Asians, respectively) following aerobic exercise training.

Sleep quality change was quantified as the difference in SQP score before and after the
exercise intervention, while sleep duration change was defined as the difference in hours of sleep before and after exercise training. The mean difference pre- and post-exercise training in SQP score was 0.05 ± 1.87, with approximately 64% of participants remaining within 1 point of their baseline sleep score, 21.5% showing improved sleep quality, and 14.3% reporting poorer sleep quality following exercise. Repeated measures analysis of variance in all subjects indicated that sleep quality scores and sleep duration remained stable over the 15-week exercise intervention. Groups formulated based upon improvement in sleep score (SQP score more than 2 points lower following exercise), no change in sleep score (post-exercise SQP score within 1 point of pre-exercise score), or worsening of sleep score (SQP score increasing more than 2 points following exercise training) over time were not significantly different in terms of total exercise dose, average duration of workouts, or average intensity of workouts. Nevertheless, the difference in total sleep duration before and after exercise was significantly different by SQP change group (p<0.001). Those whose sleep quality improved had a mean increase in sleep time of 0.9 ± 1.2 hours of sleep per night, while those who experienced worse sleep quality following exercise had an average decrease of 0.7 ± 1.2 hours of sleep per night (Figure 4). Sleep duration remained unchanged for those whose SQP score remained with 1 point of their starting score; thus, change in sleep quality appears to be highly associated with changes in total sleep duration. In linear regression analysis, controlling for age, gender, baseline value of SQP score, and total exercise dose, Hispanic ethnicity and percent change in BMI were both associated with pre/post-
exercise differences in SQP scores. Because gender and race/ethnicity differences for sleep quality have been established in the scientific literature\textsuperscript{28}, we examined sleep quality change by these parameters. In females, but not males, overweight/obesity status at baseline and percent change in BMI were significantly associated with SQP change (p<0.05), controlling for baseline measures of SQP score, age, and race/ethnicity. Hispanic ethnicity was a significant predictor of SQP score change in males (p<0.03) but not in females. In Hispanics, overweight/obesity status at baseline (p<0.02) was a significant predictor of SQP score change, while in non-Hispanic whites, percent change in BMI was significantly associated (p<0.002) with change in SQP score following exercise training.
Discussion

The overall goal of this study was to identify which physiological factors were related to sleep quality in young adults. This study also examined whether the long-term exercise intervention in the TIGER study would influence sleep quality in young adults and what phenotypic measurements, if any, might moderate change in sleep quality and duration following aerobic exercise training. In this study of young adult males and females of multi-ethnic background, sleep quality was significantly associated with a number of physiological factors that are indicative of health or weight status. Since the literature is inconsistent regarding possible sleep differences for males and females, analyses were stratified for each gender. Additionally, analyses were stratified by obesity/overweight status for all groups in order to compare our results to the body of literature that points to BMI or weight status as being correlated with poor sleep quality.

We first examined the joint effects of gender, age, overweight/obesity status and race/ethnicity, as the most commonly reported factors influencing sleep quality and duration. In our study, age, gender, and overweight/obesity were all significantly related to sleep quality, while age and race/ethnicity were related to sleep duration. No other factors were predictive of sleep quality and/or duration in all subjects combined.

Analyses were then stratified by overweight/obesity status and by gender for all
subjects. Groups were stratified into different categories by BMI (normal, overweight, obese) based standard definitions. Interestingly, skinfold thickness was significantly related to sleep quality at baseline for all groups stratified by gender and BMI status, with the exception of overweight females. Skinfold thickness is an indirect measure of subcutaneous body fat that is highly correlated with total body fat. Increasing skinfold thickness may be related to poor sleep quality because excess adiposity has been associated with sleep disturbances. Tworoger et al. have reported both BMI and percent body fat to be associated with actinographic sleep measures in men and women\textsuperscript{34}. Percent body fat was also significantly related to sleep quality for male overweight and obese subjects, as well as normal females\textsuperscript{34}. Similarly, a study of white, middle-aged men with metabolic syndrome also found significant correlations for PSQI sleep quality score with BMI and body fat percentage\textsuperscript{7}. Waist circumference was found to be negatively correlated with sleep in males, while this study found hip, but not waist, circumference to be negatively correlated with sleep for males in our study\textsuperscript{7}. Based on the large number of studies for which skinfold thickness and BMI were significantly associated with SQP, there is strong evidence that poor sleep quality is more often associated with higher adiposity and body size\textsuperscript{34}.

One factor that may link adiposity to sleep quality is the hormone orexin/hypocretin, which is responsible for both food intake and arousal. Hypocretin is actively secreted by
the lateral hypothalamus during periods of wakefulness and food needs, an area of the brain that also contains the master circadian clock mechanism. Studies have shown that hypocretin is secreted during times of forced wakefulness\textsuperscript{35}. When individuals force themselves to be awake, orexin levels increase. Increased orexin levels stimulate an increase in feeding and are associated with obesity and alterations in glucose metabolism. Experimentally-induced sleep deprivation and short sleep times also increases ghrelin and decreases leptin\textsuperscript{29}. These hormones, in conjunction with orexin, are associated with food intake, and increases in these hormones are associated with obesity and shortened sleep\textsuperscript{29}.

Sleep suppresses catecholamines and cortisol, which suppress blood pressure and glucose tolerance. Thus it could be hypothesized that poor sleep quality or self imposed sleep deprivation leads to increased BMI, body fat percentage, diastolic blood pressure and skinfold thickness rather than these phenotypic qualifiers of overweight or obesity status being the reason for poor sleep quality. But the only factors in our study that were uniquely associated with poor sleep in overweight or obese individuals were WHR (obese males) and diastolic blood pressure (all obese/obese females). When stratifying by gender percent body fat was also associated with sleep quality in overweight/obese males. Interestingly this effect of body fat had a different directional quality. Higher body fat was associated with worse sleep quality in overweight males and better sleep quality in obese males.
Diastolic blood pressure was the next most frequent significant correlate of sleep score at baseline in subjects not regularly exposed to exercise. Diastolic blood pressure was positively correlated with SQP score, indicative of poor sleep quality in males, obese subjects, and obese males and approached significance for females. Bansil, et. al, found that short sleep, poor sleep, and sleep disorders were associated with hypertension. In fact, oneway analysis of our data showed that SQP score was significantly (p=0.025) different for those individuals with hypertensive diastolic BP versus those with normal diastolic blood pressure (<90 Hg). Shittu et al, also found that a high diastolic blood pressure (>90 Hg) was associated with a poor sleep score as measured by PSQI. In general, sleep plays a role in decreasing blood pressure by decreasing catecholamines and cortisol, thus these results are consistent with higher BP being associated with less sleep time or quality. The link between sleep quality and blood pressure could be due to its common control in the superchiasmatic nucleus, ablation of which has been found to affect blood pressure circadian rhythms without affecting the total sleep time (sleep/wake cycle). Variability of sleep quality affects neuroendocrine control of blood pressure and heart rate; our study found these cardiac functions to be significantly related to sleep quality and sleep duration in multiple groups.

The research literature often reports discrepancies in sleep quality for males and females. In univariate models, sleep quality score was not significantly different between males and females either before ((p=0.06) or after exercise (p=0.08) in our
study, although average sleep quality scores were consistently higher for females (3.1 ± 2.2 pre-exercise, 3.0 ± 2.2 post-exercise) compared to males (2.9 ± 2.2 pre-exercise, 2.7 ± 2.3 post-exercise).

Sleep duration was most significantly related to race/ethnicity. Similar to our findings, African Americans and Hispanics have shown to sleep less compared to whites in other studies\(^\text{40, 41}\). When examining other factors that were related to sleep quality, as BMI increased for overweight and obese individuals, sleep duration decreased. In our study, sleep duration was not associated with overweight/obesity status either before or after exercise training. This is in contrast to a study that found BMI was significantly predictive of sleep duration and predictive of being overweight for males but not for females\(^\text{33}\). It is possible that exercise counteracts the metabolic effects associated with a higher BMI in decreasing sleep duration.

The research literature reports improvements in obesity outcomes, including those on sleep duration and quality, associated with exercise. Exercise may improve physiological outcomes such as glucose score, body fat, BMI, weight, and blood pressure. Changes in these physiological factors will result in improvement in other areas such as insulin response, inflammation levels and hormonal signaling. These factors could improve sleep directly or indirectly through effects on internally controlled circadian rhythms. In fact, for all individuals after exercise intervention, skinfold thickness (p<0.001) changed
significantly over time, as did diastolic blood pressure (p=0.01), percent body fat (p=0.004), weight (p<0.004), and glucose score (p=0.05).

Although a number of factors changed with exercise, only change in BMI and Hispanic ethnicity were associated with the difference in SQP score from the beginning of exercise intervention to the end of this study. This research found that females of a higher weight status who had a change in BMI were more likely to have a change in SQP score. Research points to the female sex having sleep affected by a greater number of physiological and environmental factors\textsuperscript{27}. The female neurological system is affected by greater levels of estradiol\textsuperscript{27} and in female rats have shown greater effects of the orexin/hypocretin system\textsuperscript{42}. Hispanic males had a greater effect of change in SQP score than other races/gender. Also Hispanics with a higher weight status were more likely to have a significant change in SQP score with exercise. On the other hand, non-Hispanic whites who had a change in BMI were more likely to have a change in sleep quality as well. This and the apparent sleep duration differences between races points to race discrepancies in sleep and response of sleep to exercise.
Conclusion

Research appears to indicate that exercise will improve sleep quality\textsuperscript{43}. Much of this research is done on individuals who already have poor sleep quality\textsuperscript{44} or who have a disease such as obstructive sleep apnea,\textsuperscript{45} rather than the general population. Additionally, a great deal of this research focuses on elderly individuals who are less active than young adults and in general have poorer sleep quality than a younger population\textsuperscript{46-48}. In an older population, six months of exercise did not improve self-reported sleep quality consistency, but a modest improvement was seen after 12 months of moderate endurance exercise\textsuperscript{49}. Other studies have reported improved sleep quality on individuals exposed to large doses of exercise in a short period of time\textsuperscript{50}. It may be that this effect of improved sleep or circadian phase shift\textsuperscript{51} is seen as a byproduct of fatigue or acute response rather than being the result of long term physiological changes that serve to regulate circadian rhythmicity or other phenotypic factors indicative of poor metabolic function. Although some research points to exercise as improving sleep\textsuperscript{15}, intervention methods and subjects vary in experimental protocols. Other studies point to little to no benefit of exercise on sleep\textsuperscript{16}. For example very fit subjects have demonstrated increased sleep when exposed to exercise in the heat but not in cool conditions\textsuperscript{52}, this and other studies have implied that the effects
observed of acute exercise on improvement of sleep is a by-product of increased thermal load on individuals rather than directly due to exercise itself. Other attributable factors that may affect sleep and are affected by exercise are anxiety, circadian phase shifting, neurotransmitter levels, and cytokine concentration. But the relative changes in these factors all depend on total exercise dose, duration, intensity, time, and environment, as well as if the exercise is chronic or acute. Because of this variability in research methods, it is difficult to definitively determine if sleep quality is improved by chronic exercise similar to what people would engage in on their own, over time.

This study found that the majority of participants did not have changes in their sleep quality after 15 weeks of exercise. Consistent with this are reports of lack of perceived sleep improvement after four months of exercise by Elavsky et al. As opposed to factors related to sleep quality before exercise, only overweight/obesity status was a predictor of poor sleep quality. This is congruent with literature reports of higher weight status individuals having decreased sleep quality or duration.

Body fat percentage as measured by DXA score was a significant predictor of change in SQP score representing a change in sleep quality for all subjects not stratified by overweight/obesity status or gender. We found a greater percent change in sleep score was correlated with decreasing body fat percentage (p<0.021) which represents a correlation with a decrease in SQP or improvement in sleep quality. Although we
question if exercise will improve sleep for all individuals, it is clear that exercise will
decrease fat levels, and sleep improved in those individuals who had a decrease in fat
(n=566). Although non-significant (p=0.07) a relationship was clear that those individuals
who had a decrease in skinfold thickness also had a greater improvement in sleep
quality (decrease in SQP). A t-test also showed that mean skinfold thickness decreased
with exercise as did a t-test for the mean of all subject’s weight before exercise
intervention compared to after. Percent change in SQP score was also significantly
correlated with relative change in weight (p=0.035), although percent change in SQP
was not significantly related to individuals who gained weight or who lost weight. This is
not worrisome as often individuals gain weight with increased physical fitness as lean
body mass replaces fat tissue. One could speculate that it is not sleep loss as the
initiator of increased body fat, skinfold thickness, weight, and glucose score but rather
poor nutritional status that leads to dysregulation of metabolic systems, insulin
resistance, hormonal changes. These factors will then decrease good quality sleep or
appropriate sleep duration. In fact, glucose regulation changes throughout the day and
night, with poorer glucose control at night, due to decreased glucose utilization and
decrease insulin sensitivity. Since the body operates primarily on feedback loops poorer
glucose control could be responsible for decreased sleep. Similarly a poor Western diet
with a positive energy balance will lead changes in orexigenic and anorexigenic factors
that arise in diabetes, obesity, and metabolic syndrome. It is known that some of these

factors, like orexin and leptin, respectively, affect or are affected by sleep\textsuperscript{55}. It could be that a decrease in sleep duration/ poor sleep is concomitant with the development of metabolic disease rather than the initiating factor, or even alleviating factor for that matter. Although sleep loss has been shown to alter the neuroendocrine control of appetite\textsuperscript{55}, it may be that changes in the neuroendocrine system from a positive energy balance diet can dysregulate sleep. Thus, when exercise improves sleep, it does so indirectly by improving factors correlated with metabolic disease.

Additionally, circadian rhythmicity of hormones and vasoactive peptides of the hypothalamic-pituitary-adrenal, renin-angiotensin-aldosterone, and the hypothalamic-pituitary-thyroid systems are affected by changes in sleep duration, pattern, and quality. These systems affect body phenotype in terms of weight status, body fat percentage, glucose response and BMI. Thus it logical to measure phenotypic quantities that will vacillate as a result of changes to these key biological systems. Exercise is also a mediator of these phenotypic qualities directly by hormonal and vasoactive mechanisms. It may be hypothesized that exercise is able to improve circadian rhythmicity of these bioactive compounds in order to affect improvements in phenotype and that sometimes, improvement in phenotype can cause changes in sleep quality.

Strengths of the study include a large population, a well-controlled exercise protocol, and multiple measures of sleep quality. A limitation of this study is that post-exercise
measures of sleep quality were evaluated during final exams at the end of the semester. This is a time of additional duress and decreased sleep and thus could have increased SQP score indicating poorer sleep quality than may have occurred during normal times of moderate stress. This could have downplayed the effect of exercise on sleep quality. Additional limitations of this study are lack of direct physiologic measurement by polysomnographic instruments of sleep duration and quality as well as self reports of sleep quality and duration. Taking such measurements would have been extremely difficult in a study of this size. An additional strength of this study is the long period of 15 weeks for exercise intervention. Most studies that examine the effects of exercise on sleep monitor the immediate response of sleep to exercise on a daily or weekly basis. This study takes long-term physiological effects into account as changes in habits, hormones, or circadian rhythms are more probable over many months of exercise.
Table 1: Means and Standard Deviations for Baseline Body Measurements

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-Hispanic White (n =394)</td>
<td>Hispanic (n =107)</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Age (years)</td>
<td>22</td>
<td>3.2</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>26.7</td>
<td>5.2</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>86.7</td>
<td>13.5</td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>102.3</td>
<td>10</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.84</td>
<td>0.07</td>
</tr>
<tr>
<td>Heart rate</td>
<td>73.77</td>
<td>12.8</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>121.9</td>
<td>10.6</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>69.4</td>
<td>9.6</td>
</tr>
<tr>
<td>Percent fat</td>
<td>17.4</td>
<td>7.9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>117.1</td>
<td>7.1</td>
</tr>
<tr>
<td>Weight (lbs)</td>
<td>184.76</td>
<td>40.6</td>
</tr>
</tbody>
</table>

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Table 2: Predictors of sleep quality stratified by gender before exercise intervention

<table>
<thead>
<tr>
<th>Variable</th>
<th>Males (n=684)</th>
<th>Females (n=1218)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Regression coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.055</td>
<td>0.01</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>0.089</td>
<td>0.008</td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>-0.04</td>
<td>0.018</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>0.026</td>
<td>0.008</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>-0.02</td>
<td>0.028</td>
</tr>
<tr>
<td>Skinfold Thickness</td>
<td>0.012</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Final Model</td>
<td>$R^2=0.04$</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Table 3: Predictors of sleep quality stratified by overweight/obesity status before exercise intervention

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Subjects Normal (n=962)</th>
<th>All Subjects Overweight (n=618)</th>
<th>All Subjects Obese (n=386)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>0.7</td>
<td>0.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>-0.04</td>
<td>0.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td></td>
<td>0.03</td>
<td>0.01</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>-0.32</td>
<td>0.04</td>
<td>-0.027</td>
</tr>
<tr>
<td>Skinfold Thickness</td>
<td>0.016</td>
<td>&lt;0.001</td>
<td>0.009</td>
</tr>
<tr>
<td>Final Model</td>
<td>$R^2=0.02$</td>
<td>0.0003</td>
<td>$R^2=0.04$</td>
</tr>
</tbody>
</table>

*normal, overweight, and obese status classified on BMI measurements
*significance level for linear regression set at p=0.1
Table 4: Predictors of sleep quality score stratified by gender and overweight/obesity status

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Male Subjects Normal (n=288)</th>
<th>All Subjects Male Overweight (n=278)</th>
<th>All Male Subjects Obese (n=137)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression coefficient</td>
<td>p-value</td>
<td>Regression coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.14</td>
<td>0.041</td>
<td></td>
</tr>
<tr>
<td>Hip Circumference (cm)</td>
<td>-0.08</td>
<td>0.031</td>
<td></td>
</tr>
<tr>
<td>WHR</td>
<td></td>
<td>8.6</td>
<td>0.01</td>
</tr>
<tr>
<td>Percent Body Fat</td>
<td></td>
<td>0.77</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Skinfold Thickness</td>
<td>0.019</td>
<td>0.005</td>
<td>-0.19</td>
</tr>
<tr>
<td>Final Model</td>
<td>R²= 0.08</td>
<td>0.0005</td>
<td>R²= 0.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R²= 0.11</td>
<td>0.005</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Female Subjects Normal (n=667)</th>
<th>All Subjects Female Overweight (n=330)</th>
<th>All Female Subjects Obese (n=249)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression coefficient</td>
<td>p-value</td>
<td>Regression coefficient</td>
<td>p-value</td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.13</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>-1.61</td>
<td>0.064</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>-0.014</td>
<td>0.069</td>
<td></td>
</tr>
<tr>
<td>Diastolic BP</td>
<td></td>
<td>0.035</td>
<td>0.024</td>
</tr>
<tr>
<td>Percent Body Fat</td>
<td>-0.04</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Skinfold Thickness</td>
<td>0.02</td>
<td>0.011</td>
<td>0.014</td>
</tr>
<tr>
<td>Final Model</td>
<td>R²=0.013</td>
<td>0.03</td>
<td>R²=0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R²=0.056</td>
<td>0.0009</td>
</tr>
</tbody>
</table>
Figure 1: Sleep Quality Score Stratified by Gender and Overweight/Obesity Status Prior to Exercise Training
Figure 2: Sleep Quality Score Stratified by Gender and Overweight/Obesity Status Prior to Exercise Training
Figure 3: Average Sleep Duration by Race/Ethnicity Prior to Exercise Training

![Bar chart showing average sleep duration by race/ethnicity.](chart.png)
Figure 4: Change in Sleep Duration by SQP Score Change Category Following Exercise
References


31. Taheri S. The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity. Archives of disease in childhood 2006;91:881-4.


43. Reid KJ, Baron KG, Lu B, Naylor E, Wolfe L, Zee PC. Aerobic exercise improves self-reported sleep and quality of life in older adults with insomnia. Sleep


50. Wang X, Youngstedt SD. Sleep quality improved following a single session of moderate-intensity aerobic exercise in older women: Results from a pilot study. Journal of Sport and Health Science.


