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A single night of sleep restriction impairs recovery from heavy exercise

John D. Chase
James Madison University

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A SINGLE NIGHT OF SLEEP RESTRICTION IMPAIRS
RECOVERY FROM HEAVY EXERCISE

John D. Chase

A thesis submitted to the Graduate Faculty of
JAMES MADISON UNIVERSITY

In

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FACULTY COMMITTEE:

Committee Chair: Dr. Nick Luden

Committee Members/ Readers:

Dr. Trent Hargens

Dr. Mike Saunders

Dr. Chris Womack

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ABSTRACT

We examined the effects of one night of sleep restriction (*Sleep-*; 2.5 hrs) on subsequent 3-km cycling performance and skeletal muscle recovery from heavy exercise compared to a full night of rest (*Sleep+*; 7 hrs). Seven recreational cyclists (n=6 male, n=1 female; age = 24.4 ± 7 yrs; height = 170 ± 10 cm; weight = 68 ± 13 kg $VO_{2max} = 61.5 \pm 4.4$ ml/kg/min) completed four simulated 3-km time trials (TT) and six peak isokinetic torque trials at different speeds (30 and 120°/sec) under both conditions. The first exercise trial (EX1) consisted of baseline testing followed by heavy exercise (60 min interval session + resistance exercise) in the evening, while follow-up testing occurring in the morning of the next two days (EX2 and EX3). TT performance and peak torque were assessed on the evening of EX1 between 3-5pm and between 8-10am the following morning (EX2), while only peak torque was assessed during the same morning time on EX3. Magnitude-based inferences were used to evaluate all variables. *Sleep-* clearly impaired average power output ($-12.7 \pm 1\%$) and overall time ($-3.5 \pm 0.39\%$) of TT performance compared to *Sleep+*, while there was no clear impact of sleep on peak torque at either speed. The current study demonstrates the importance of sleep on recovery from heavy exercise with potential differences in exercise types, and warrants further research on the topic.

CHAPTER ONE

INTRODUCTION

Sleep is a primordial necessity of human life. The absence of sleep is associated with negative effects on motor and cognitive performance, as well as decreased metabolic, hormonal and immunological function (13). The National Sleep Foundation (NSF) recommends that adults sleep between 7 and 9 hours per night (19). However, many Americans fall short of these guidelines. A large-scale (n = 1.1 million Americans; aged ≥ 30 y) sleep study conducted by the American Cancer Society reported that that over half (52%) of the sample population slept less than 7.5 hrs per night, 20 percent slept less than 6.5 hrs per night, and 4 percent slept less than 5.5 hrs per night. Altogether, 76 percent were obtaining less than 8 hrs of sleep. Similarly, a more recent report indicates that subjects self-reported sleeping 6.8 hrs on weekdays, and 7.4 hrs on weekends (17). Additionally, the Center for Disease Control and Prevention (CDC) reported that $\geq 30\%$ of adults received an average of less than 6 hrs per night in 2005-2007 (42). These studies indicate that average sleep duration has decreased over the past few decades and that Americans are not meeting NSF recommendations. The lack of sleep among Americans is the result of various factors such as medical conditions, light pollution, work conditions, anxiety, caffeine intake, and the environment in which one sleeps (3, 9). Regardless of the cause, inadequate sleep has a host of behavioral and physiological ramifications for all individuals. The effects of insufficient sleep for athletes are of particular interest because of the physiological demands of heavy training and peak performance. Moreover, this population is particularly susceptible to sleep disruption because of their training and competition habits. For instance, athletes have reported

difficulty falling asleep, early waking patterns, and increased stress levels during training and near competition (12, 17).

Sleep disruption can be dissected into two categories: sleep deprivation and sleep restriction. Sleep deprivation is defined as the absence of sleep, while sleep restriction is characterized by intermittent waking, early waking and delayed onset of sleep. Several studies have detailed the impact that sleep deprivation and restriction have on overall athletic performance (5, 28, 30, 31, 35, 37, 39, 44). Although both forms of sleep disruption impair performance during periods of rest or light training, the effects are more prominent with sleep deprivation. Additionally, the effect of sleep disruption on recovery from heavy bouts of exercise has not been well-documented.

Most of what is known about sleep disruption has been gathered in the context of sleep deprivation and the literature clearly indicates that it can impair performance. Initial work, in the early 1980's, demonstrated that 36 hrs of sleep deprivation reduced VO_{2max} by 11% (30). Subsequent research has similarly concluded that sleep deprivation can compromise peak physical capacity in activities ranging from sprint trials to several hour time trials. For example, 30 hrs of sleep deprivation reduced 30 min run performance by 2.9 percent (35). Additionally, between 30-36 hrs of sleep deprivation reduced Wingate and sprint trial performance in separate studies, suggesting that anaerobic function is altered following extensive sleep loss (43, 44). However, some studies have shown no relationship between sleep deprivation and anaerobic performance (46–48). Collectively, these findings suggest that sleep deprivation may negatively influence athletic performance, and anaerobic performance may be less affected.

Though less is known about sleep restriction, there is evidence that it can negatively influence various aspects of physiology and athletic performance. While little is known about the effects of sleep restriction on aerobic performance, several studies reported decreased anaerobic (1, 45) and strength-related components (37, 39). In an early study, distance covered in cycle ergometer fixed intensity (75% $\text{VO}_{2\text{max}}$) tests was not different after 3 hrs of sleep onset restriction, compared to a full night of rest (34). Some studies reported impaired Wingate performance following approximately 4 hrs of sleep restriction (2, 45), while other studies with similar amount of restriction did not (32, 45). Additionally, maximal and sub-maximal weight lifting performance decreased following 3 consecutive nights of 3 hrs of sleep (39). However, it is important to note that all studies where Wingate performance was negatively affected, sleep was restricted at the end of the night, while no changes were seen when sleep onset was delayed. Souissi and colleagues confirmed this difference by comparing both conditions (45). Their results indicated decreased muscular strength and power when sleep was restricted at the end of the night, but no differences from baseline when sleep onset was delayed. Therefore, athletic performance is likely inhibited following a night of sleep restriction, and timing of sleep restriction may be of importance.

In addition to physical capacity, sleep alteration perturbs a variety of physiological variables including growth hormone, testosterone, cortisol, and lactate. These variables are widely known to be elevated in the blood following heavy exercise and can be affected by sleep deprivation or sleep restriction. As little as 3 hrs of sleep restriction led to higher blood lactate levels during a 20-min submaximal exercise, compared to a full night of sleep (34). The authors speculated that this was the result of

sleep loss, increased metabolic stress from heavy exercise, or a combination of the two conditions. However, other results have indicated that preceding sleep restriction has no effect on blood lactate levels during exercise (29, 44). It was later reported that sleep restriction also reduces cortisol levels when sleep is restricted at the end of the night, suggesting that timing of sleep restriction plays a role in neuroendocrine function (33). Additionally, testosterone and growth hormone were increased after 4.5 hrs of sleep and subsequent sprint trials when compared to baseline sleep and exercise, while plasma cortisol was not significantly different (2). Further research must be done on these markers to determine the effect that sleep deprivation and sleep restriction in combination with exercise has on these markers.

Collectively, it appears that both sleep deprivation and sleep disruption can negatively influence performance. It is important to note that all of the aforementioned work examined how sleep disruption influences performance and/or physiology after several days of rest/light physical activity in preparation for the performance session. Virtually nothing is known about how sleep affects recovery from heavy exercise. Skein and colleagues examined performance and glycogen levels in athletes after a full night of rest compared with 30 hours of sleep deprivation, having subjects perform heavy exercise on two consecutive days separated by either sleep condition (43). Maximal voluntary contraction of the quadriceps, sprint trials, and muscle glycogen restoration were reduced following sleep deprivation, suggesting that recovery processes are sensitive to sleeping patterns. Whether or not more practical sleep disruption (i.e. sleep restriction) influences recovery from heavy exercise has not been examined. Further, nothing is known about whether or not sleep restriction influences muscle soreness ratings. Therefore, the

purpose of this study is to determine the effects that sleep restriction has on subsequent performance and recovery from heavy exercise.

Aims and Hypotheses

Aim 1: To determine the effects that sleep restriction has on subsequent time trial performance and muscle power following heavy exercise, compared to a full night of rest.

Hypothesis 1: Sleep restriction will impair subsequent time trial performance and muscle power following heavy exercise, compared to a full night of rest.

Aim 2: To determine the effects that sleep restriction has on perceived muscle soreness following heavy exercise, compared to a full night of rest.

Hypothesis 2: Sleep restriction will increase muscle soreness following heavy exercise, compared to a full night of rest.

Significance of the Study

Several studies have detailed the impact that sleep deprivation and restriction have on overall athletic performance. Yet little is known about how sleep restriction may impact recovery from heavy exercise.

To our knowledge, only two studies have examined the effects of sleep disruption on recovery from exercise. Although this information is valuable for our understanding of the important role that sleep has during recovery from exercise, it is of limited practical relevance when considering typical sleep patterns. Since athletes are susceptible to disrupted sleep patterns, examining the effects that sleep restriction has on recovery from heavy exercise is more practical. Discovering these effects may provide coaches and athletes the necessary information to properly time heavy exercise, and determine the importance of sleep on muscle recovery. This information may also be beneficial to the general population in order to properly plan heavy exercise around days when a full night of sleep is more likely. Doing so may prevent muscle soreness, which can lead to discomfort and in turn decreased subsequent exercise during recovery days. Thus, the effects of sleep restriction with subsequent heavy exercise on muscle soreness should also be examined. Regardless of the question being answered, these questions have practical training importance.

CHAPTER TWO

METHODS

Subjects

Approximately 10-15 male cyclists, age 18-25, were recruited from James Madison University (JMU) and the surrounding area. Recruitment efforts were focused on the JMU Triathlon and Cycling Club teams and local cycling shops. Subjects were required to have a VO_{2max} of ≥ 50 ml/kg/min or 4 L/min. Additionally, subjects who reported injuries or disordered sleeping, according to the Pittsburgh Sleep Quality Index (PSQI), were disqualified. Subjects were provided verbal and written information regarding procedures, and a subsequent written informed consent was obtained. All procedures were approved by the JMU Institutional Review Board prior to data collection.

Experimental Design

This protocol was designed to examine the influence of sleep restriction on recovery from heavy exercise. Subjects completed a preliminary trial, a familiarization phase, and 2 experimental phases, each separated by approximately 7 days. The familiarization phase and 2 experimental phases consisted of three exercise sessions performed on consecutive days (EX1, EX2, and EX3). EX1 included baseline performance testing followed by an exercise protocol designed to elicit fatigue. EX2 and EX3 were performed the following two mornings and were used to assess recovery from EX1. EX1 and EX2 performed during the experimental phases were separated by either a full night of sleep (*Sleep+*) or a night of sleep restriction (*Sleep-*). Order of subject assignment to *Sleep+* and *Sleep-* was determined using a randomized crossover design.

Preliminary Trial

Following height (cm) and body weight (kg) measurements, maximal oxygen consumption (VO_{2max}) and peak power (W_{max}) were obtained on an electronically braked cycle ergometer (Velotron, RacerMate, Inc.; Seattle, WA, USA). Following the VO_{2max} test, subjects performed a one-repetition maximal effort test on a leg press machine (Cybex International, Inc.; Medway, MA). Subjects performed a self-selected warm-up for 5 minutes and began the test at an intensity corresponding to a comfortable 60-min ride. Intensity was increased by 25 W every 2 minutes until voluntary termination or until RPM drop below 50. W_{max} was used to determine workloads during the EX trials. Breath samples were analyzed for oxygen uptake (VO_2), ventilation (VE), and respiratory exchange ratio (RER) (Moxus; Pittsburgh, PA, USA) and heart rate was measured using a Polar heart rate monitor (Lake Success, NY, USA). Subjects indicated rate of perceived exertion by pointing to a Borg (6-20) RPE scale at the end of each stage.

Familiarization Phase

The familiarization phase was identical to the EX phases (detailed below), and was used to ensure that subjects can complete EX protocols at the pre-determined intensity, and to minimize learning associated error variance during EX trials. Sleep was monitored, but not controlled, during this phase.

Exercise Trial 1 (EX1)

Subjects arrived at the human performance lab between 3-5pm, not having consumed alcohol, tobacco or caffeine 24 hrs prior to testing. Following 15 min of seated

rest, muscle soreness was assessed, as detailed below. Subjects then warmed up on a treadmill for 5 min at 3.5 mph and 0% grade. Upon completion of the warm-up, subjects performed single-leg extension peak isokinetic dynamometer tests at 120 and 30 deg/sec (BioDex, Shirley, NY, USA). Following a 10-min rest, subjects performed a 20-min warm-up (10 min at 50% W_{max} followed by 10 min at 60% W_{max}) on the cycle ergometer. Following the 20 min warm-up, subjects began a 3-km self-paced time trial. Subjects were verbally encouraged to give maximal effort prior to beginning the trial. After 10 min of rest, subjects performed a 60-min sprint interval session, previously detailed by Goh et al. (15). Sprint intervals lasted 2 min at 95% W_{max} followed by 2 min at 50% W_{max} , at a cadence of ≥ 60 rpm. When subjects failed to maintain cadence at 95% W_{max} , intensity was reduced by 10% in subsequent sprints. Following a 10-min rest period, subjects performed 3 sets of 10 repetitions on a leg press at a weight corresponding to 80% of their one-repetition max (Cybex International, Inc.; Medway, MA).

Experimental Trial 2 (EX2)

Subjects arrived at the human performance lab between 7-10am, not having consumed alcohol, tobacco or caffeine 24 hrs prior to testing. Following 15 min of seated rest, muscle soreness was assessed, using the same protocol as EX1. Subjects then warmed up on a treadmill for 5 min at 0% grade. Upon completion of the warm-up, subjects performed the same peak isokinetic torque protocol used in EX1. Following 10 min rest, subjects performed the same warm-up and 3-km protocol used in EX1.

Experimental Trial 3 (EX3)

Subjects arrived at the human performance lab between 7:00-10:00am, not having consumed alcohol, tobacco or caffeine 24 hrs prior to testing. Following 15 min of seated rest, muscle soreness was assessed using the same protocol as EX1 and EX2. Subjects then warmed up on a treadmill for 5 min at 0% grade. Upon completion of the warm-up, subjects performed the same peak isokinetic torque protocol used in EX1 and EX2.

Sleep Restriction

Subjects underwent the first set of EX trials separated by either *Sleep+* or *Sleep-*. Subjects were then assigned the alternate sleep condition 7 days later. Subjects attempted onset of sleep set between 10:00 pm-12:00 am for both conditions, replicating the same onset time in both experimental phases. Onset of sleep was measured using both an Actigraph accelerometer and the Sleep Cycle smartphone application, which uses motion detection to determine approximate sleep onset and wake time. Subjects were instructed to set wake time 8 hrs following sleep onset on *Sleep+*, and 3 hrs following sleep onset on *Sleep-*.

3-km Time Trial Performance

3-km cycling time trials were performed on the Velotron ergometer mentioned above. 3-km finishing times and average power output were used as the performance criterion.

Muscle Recovery

Muscle Function

Peak isokinetic torque was determined using the BioDex dynamometer mentioned above (120° and 30°/sec), at the aforementioned times during EX phases.

Muscle Soreness

Muscle soreness ratings were obtained using a visual analog scale from 0-100 mm, with 0 mm indicating no muscle soreness and 100 mm indicating impaired movement due to muscle soreness, as detailed by Saunders et al (41). Soreness ratings were obtained upon arrival on EX1, EX2, and EX3 sessions while walking up and down a flight of stairs.

Cardiorespiratory Measures

Heart Rate & Rate of Perceived Exertion (HR & RPE)

During EX trials, HR and RPE were recorded after reaching steady-state during min 10 and min 20 of the 20-min warm-up preceding the 3-km time trial.

Oxygen Consumption, Ventilation, & Respiratory Exchange Ratio (VO₂, VE & RER)

VO₂, VE, and RER were assessed during EX phases using a Moxus metabolic cart (Pittsburgh, PA, USA). Breath samples were obtained throughout the 20 min warm-up preceding the 3 km time trials. Minutes 9-10 and 19-20 were averaged and recorded.

Physical Activity & Dietary Control

Subjects recorded all food and beverage intake for 24 hrs preceding EX1. After the initial EX phase, subjects were provided with copies of their dietary records and instructed to replicate their dietary habits following EX1 for the second EX phase. Subjects reported to all testing after a >4 hr fast. Within 1 hour of completing EX1, subjects consumed a predetermined amount of Ensure Shake corresponding to 20-25% of daily energy expenditure using the Harris-Benedict equation. Additionally, subjects were instructed to refrain from consuming any other macronutrients during the 2 hrs following EX1. Subjects recorded all physical activity 72 hrs prior to EX1 in both phases. Subjects were instructed to avoid physical activity between EX1 and EX2, and to keep physical activity habits consistent between EX phases.

Statistics

Total time and mean power output (Watts) from each 3-km time trial were used as the performance measures. All data was log transformed to diminish the effects of non-uniformity. Magnitude-based inferences about the data were derived using methods described by Hopkins and colleagues (20). For performance, a previously established 'smallest worthwhile change' in performance was used as the threshold value for a condition effect (EX1 time trial vs. EX2 time trial) (21). The smallest worthwhile change in performance was defined as 0.3 x the within subject variability of select groups of elite cyclists across repeated time trials (CV = 1.5% for time and estimated 4.5% for power) (21). For all other variables, the threshold value for a substantial treatment effect was defined as 0.2 x within-subject standard deviation, under resting conditions.

All data were log transformed to diminish the effects of non-uniformity. Magnitude-based inferences about the data were derived using methods described by Hopkins and colleagues (20). For performance, a previously established ‘smallest worthwhile change’ in performance was used as the threshold value for a condition effect (EX1 time trial vs. EX2 time trial) (21). The smallest worthwhile change in performance was defined as 0.3 x the within subject variability of select groups of elite cyclists across repeated time trials (CV = 1.5% for time and estimated 4.5% for power) (21). For all other variables, the threshold value for a substantial treatment effect was defined as 0.2 x within-subject standard deviation, under the *Sleep+* condition.

Published spreadsheets (Hopkins, 2006a and b) were then used to determine the likelihood of the true treatment effect (of the population) reaching the substantial change threshold; these percent likelihoods were classified as: < 1% almost certainly no chance, 1-5% = very unlikely, 5-25% = unlikely, 25-75% = possible, 75-95% = likely, 95-99% = very likely, and > 99% = almost certain. Clinical inference criteria were used to classify the effects of both conditions on performance. Specifically, if the percent chance of the effect reaching the substantial change threshold was < 25% and the effect is clear, it was classified as ‘trivial’. If the percent chance of the effect reaching the substantial change threshold for benefit exceeded 25%, but the chance for harm was > 0.5%, the effect was classified as unclear. An exception to the 0.5% chance of harm criterion was made if the benefit/harm odds ratio was > 66, in which case the effect was interpreted as clear and an inference was assigned.

Following individual condition analysis, treatment comparison (*Sleep-* vs *Sleep+*) outcomes were assessed using the aforementioned published spreadsheets (8). The

classification system detailed above was applied, but mechanistic criteria were used. If 90% confidence intervals include values that exceeded the substantial change threshold for both a positive and negative effect, effects were classified as unclear (> 5% chance of reaching the substantial threshold for both a positive and negative effect).

CHAPTER THREE

MANUSCRIPT

INTRODUCTION

Sleep is a primordial necessity of human life. The absence of sleep is associated with negative effects on motor and cognitive performance, as well as decreased metabolic, hormonal and immunological function (13). The National Sleep Foundation recommends adults obtain between 7-9 hours of sleep per night (19). However, a large-scale sleep study (n=1.1million adults, ≥ 30 years) conducted by the American Cancer Society reported that approximately three quarters of Americans ($\sim 76\%$) obtain ≤ 0.5 hours per night (25). Notwithstanding the causes of sleep loss, inadequate sleep is accompanied with a host of behavioral and physiological ramifications.

The effects of insufficient sleep for athletes are of particular interest because of the psychological and physiological demands of heavy training and peak performance. Moreover, this population is particularly susceptible to sleep disruption due to their training and competition habits. For instance, athletes have reported difficulty falling asleep, early waking patterns, and increased stress levels during training and near competition (12, 17).

Sleep disruption can be separated into two categories: sleep deprivation (SDEP) and sleep restriction (SR). SDEP is defined as the absence of sleep, while SR is characterized by intermittent waking, early waking or delayed onset of sleep. Most of what is known about sleep disruption and exercise has been gathered in the context of SDEP, and the literature clearly indicates that it can impair performance (30, 35, 43, 44).

Although less is known about the effects of SR on performance, SR appears to have a negative impact on anaerobic (1, 45) and strength-related performance (37, 39). The effect of SR on aerobic performance is unclear due to limited investigation and conflicting results. One study reported a detrimental effect on total distance covered in an incremental running test to exhaustion (31), while two others showed no difference in distance covered during running and cycling to exhaustion (34, 37).

Collectively, it appears that SDEP and SR can impair performance, but the physiological mechanisms are poorly understood. It is important to note that all aforementioned work examined how sleep disruption influences performance after several days of rest/light physical activity in preparation for the performance session. Virtually nothing is known about the impact of sleep disturbance on recovery from a heavy bout of exercise. Skein and colleagues examined performance and glycogen levels in athletes after a full night of rest compared to 30 hrs of SDEP, having subjects perform heavy exercise separated by either sleep condition (43). Performance and muscle glycogen restoration were reduced following SDEP, suggesting that recovery processes are sensitive to sleeping patterns. Whether or not more practical sleep disruption (i.e. SR) influences recovery from heavy exercise has not been examined in the context of aerobic performance. Further, nothing is known about the impact of SR on perceived muscle soreness. Therefore, the purpose of this study is to determine the effects that SR has on subsequent aerobic performance and recovery from heavy exercise.

METHODS

Subjects

17 male (n=14) and female (n=3) recreationally trained cyclists, age 18-40, were enrolled in the study. However, only 8 subjects (n=6 male, n=2 female; age = 24.4 yrs; $VO_{2max} = 61.5 \pm 4.4$ ml/kg/min) completed the study, and data from one female subject was removed due to sleep compliance issues. The remaining subjects were not retained due to scheduling conflicts, training conflicts, and injury (non-study related). Female subjects' experimental trials were planned to coincide with the follicular phase of their menstrual cycles by completing trials within days 1-13. Subjects were also screened for disordered sleeping in accordance with the Pittsburgh Sleep Quality Index (PSQI). Subjects were provided with verbal and written information regarding procedures, and subsequent written informed consent was obtained. All procedures were approved by the JMU Institutional Review Board prior to data collection.

Experimental Design

This protocol was designed to examine the influence of sleep restriction on recovery from heavy exercise. Subjects completed a preliminary trial, a familiarization phase, and 2 experimental phases, each separated by 11 ± 8 days. The familiarization phase and 2 experimental phases consisted of three exercise sessions performed on consecutive days (EX1, EX2, and EX3). EX1 consisted of baseline performance testing followed by an exercise protocol designed to elicit fatigue. EX2 and EX3 were performed the following two mornings and were used to assess recovery from EX1. EX1 and EX2 performed during the experimental phases were separated by either a full night of sleep

(*Sleep+*) or a night of sleep restriction (*Sleep-*). Order of subject assignment to *Sleep+* and *Sleep-* was determined using a randomized crossover design.

Preliminary Trial

Following height (cm) and body weight (kg) measurements, maximal oxygen consumption (VO_{2max}) and peak power (W_{max}) were obtained on an electronically braked cycle ergometer (Velotron, RacerMate, Inc.; Seattle, WA, USA). Following the VO_{2max} test, subjects performed a one-repetition maximum test on a leg press machine (Cybex International, Inc.; Medway, MA). The VO_{2max} test consisted of a self-selected warm-up for 5 minutes and began at an intensity corresponding to a comfortable 60-min ride. Intensity was increased by 25 W every 2 minutes until voluntary termination or until RPM fell below 50. W_{max} was used to determine workloads during the EX trials. Breath samples were analyzed for oxygen uptake (VO_2), ventilation (VE), and respiratory exchange ratio (RER) (Moxus; Pittsburgh, PA, USA) and heart rate was measured using a Polar heart rate monitor (Lake Success, NY, USA).

Familiarization Phase

The familiarization phase was identical to the EX phases (detailed below), and was used to ensure that subjects could complete EX protocols at the pre-determined intensity, and to minimize learning error variance during the experimental trials. Sleep was monitored, but not controlled, during this phase.

Exercise Trial 1 (EX1)

Subjects arrived at the human performance lab between 3:00 and 5:00 pm, not having consumed alcohol, tobacco or caffeine 24 hrs prior to testing. Following 15 min of seated rest, heart rate was recorded and muscle soreness was assessed, as detailed below. Subjects then warmed up on a treadmill for 5 min at 3.5 mph and 0% grade. Upon completion of the warm-up, subjects performed peak isokinetic dynamometer leg extension tests at 120° and 30°/sec (BioDex, Shirley, NY, USA). Following a 10-min rest, subjects performed a 20-min warm-up (10 min at 50% W_{\max} followed by 10 min at 60% W_{\max}) on the cycle ergometer. Dependent measurements obtained during EX1 are described below. Following the 20 min warm-up, subjects began a 3-km self-paced time trial. Subjects were verbally encouraged to give maximal effort prior to beginning the trial. After 10 min of rest following the 3-km time trial, subjects performed a 60-min sprint interval session, as previously detailed by Goh et al. (15). Sprint intervals consisted of 2 min at 95% W_{\max} followed by 2 min at 50% W_{\max} , at a cadence of ≥ 60 rpm. When subjects failed to maintain cadence at 95% W_{\max} , intensity was reduced by 10% in subsequent sprints. Following a 10-min rest period, subjects performed 3 sets of 10 repetitions on a leg press at a weight corresponding to 80% of their one-repetition max (Cybex International, Inc.; Medway, MA).

Experimental Trial 2 (EX2)

Subjects arrived at the human performance lab between 7:00 and 10:00am, not having consumed alcohol, tobacco or caffeine 36 hrs prior to testing. Following 15 min of seated rest, heart rate was recorded and muscle soreness was assessed. Subjects then

performed the same peak isokinetic torque protocol, warm-up, and 3-km time trial detailed in EX1.

Experimental Trial 3 (EX3)

Subjects arrived at the human performance lab between 7:00 and 10:00 am, not having consumed alcohol, tobacco or caffeine 60 hrs prior to testing. Subjects then warmed up on a treadmill for 5 min at 0% grade following muscle soreness assessment. Subjects then performed the same peak isokinetic torque protocol used in EX1 and EX2.

Sleep Restriction

Subjects underwent the first set of experimental trials separated by a randomly-assigned *Sleep+* or *Sleep-*. Subjects were then assigned the alternate sleep condition 7 days later. Subjects attempted to initiate sleep between 10:00 pm and 12:00 am for both conditions, replicating the same onset time in both experimental phases. Mean sleep onset and wake times were 10:51 pm (8:40 pm – 12:25 am) and 6:28 am (4 am – 8 am), respectively, during *Sleep+*. Mean onset and wake times for *Sleep-* were 11:19 pm (10:10 pm – 12:46 am) and 2:00 am (12:38 am – 3:41 am) during *Sleep-*. Onset of sleep was measured using both an Actigraph accelerometer (Pensacola, FL) and the Sleep Cycle smartphone application, which uses motion detection to determine approximate sleep onset and wake time. Subjects were instructed to set wake time 8 hrs following sleep onset on *Sleep+*, and 3 hrs following sleep onset on *Sleep-*. Following awakening on *Sleep-*, subjects immediately reported to the laboratory whereupon an investigator accompanied them until testing commenced. From the time at which subjects arrived

until the beginning of EX2 (between 8:00-10:00am), subjects were permitted to engage in any sedentary activity that kept them awake. Activities included playing video games, watching movies, and reading books. Sleep was not monitored from EX2 to EX3.

Mean sleep time during *Sleep+* was 6.86 ± 0.5 hrs, and 2.47 ± 0.3 hrs during *Sleep-*. Mean efficiency (sleep time/time in-bed) was $91.3 \pm 7\%$ during *Sleep+* and $90.2 \pm 7\%$ during *Sleep-*. Subjects awakened 16.4 ± 12 times during *Sleep+* and 5 ± 4 times during *Sleep-*.

3-km Time Trial Performance

3-km cycling time trials were performed on the cycle ergometer described previously. 3-km finishing times and average power output were used as the performance criterion.

Muscle Recovery

Muscle Function

Peak isokinetic torque for single leg extension was determined using the BioDex dynamometer described above (120° and $30^\circ/\text{sec}$), at the aforementioned times during EX phases.

Muscle Soreness

Muscle soreness ratings were obtained using a visual analog scale from 0-100 mm, with 0 mm indicating no muscle soreness and 100 mm indicating impaired movement due to muscle soreness, as detailed by Saunders et al (41). Soreness ratings

were obtained upon arrival on EX1, EX2, and EX3 sessions while walking up and down a flight of stairs.

Cardiorespiratory Measures

Heart Rate & Rate of Perceived Exertion (HR & RPE)

During EX trials, HR and RPE were recorded at the end of each 10-min stage of the 20-min warm-up preceding the 3-km time trial.

Oxygen Consumption, Ventilation, & Respiratory Exchange Ratio (VO_2 , VE & RER)

VO_2 , VE , and RER were assessed during EX phases using a Moxus metabolic cart (Pittsburgh, PA, USA). Breath samples were obtained throughout the 20 min warm-up preceding the 3-km time trials. Minutes 9-10 and 19-20 were averaged and recorded.

Physical Activity & Dietary Control

Subjects recorded all food and beverage intake for 24 hrs preceding EX1. After the initial EX phase, subjects were provided with copies of their dietary records and instructed to replicate their dietary habits following EX1 for the second EX phase. Subjects reported to all testing after a > 4 hr fast. Within 1 hour of completing EX1, subjects consumed a predetermined amount of Ensure Shake (Abbott Nutrition; Columbus, OH) corresponding to 20-25% of daily energy expenditure using the Harris-Benedict equation. Additionally, subjects were instructed to refrain from consuming any other macronutrients during the 2 hrs following EX1. Subjects recorded all physical activity 72 hrs prior to EX1 in both phases. Subjects were instructed to avoid physical

activity between EX1 and EX2, and to keep physical activity habits consistent between EX phases.

Statistics

All data were log transformed to diminish the effects of non-uniformity. Magnitude-based inferences about the data were derived using methods described by Hopkins and colleagues (20). For performance, a previously established ‘smallest worthwhile change’ in performance was used as the threshold value for a condition effect (EX1 time trial vs. EX2 time trial) (21). The smallest worthwhile change in performance was defined as 0.3 x the within subject variability of select groups of elite cyclists across repeated time trials (CV = 1.5% for time and estimated 4.5% for power) (21). For all other variables, the threshold value for a substantial treatment effect was defined as 0.2 x within-subject standard deviation, under the *Sleep+* condition.

Published spreadsheets (Hopkins, 2006a and b) were then used to determine the likelihood of the true treatment effect (of the population) reaching the substantial change threshold; these percent likelihoods were classified as: < 1% almost certainly no chance, 1-5% = very unlikely, 5-25% = unlikely, 25-75% = possible, 75-95% = likely, 95-99% = very likely, and > 99% = almost certain. Clinical inference criteria were used to classify the effects of both conditions on performance. Specifically, if the percent chance of the effect reaching the substantial change threshold was < 25% and the effect is clear, it was classified as ‘trivial’. If the percent chance of the effect reaching the substantial change threshold for benefit exceeded 25%, but the chance for harm was > 0.5%, the effect was classified as unclear. An exception to the 0.5% chance of harm criterion was made if the

benefit/harm odds ratio was > 66 , in which case the effect was interpreted as clear and an inference was assigned.

Following individual condition analysis, treatment comparison (*Sleep-* vs *Sleep+*) outcomes were assessed using the aforementioned published spreadsheets (8). The classification system detailed above was applied, but mechanistic criteria were used. If 90% confidence intervals include values that exceeded the substantial change threshold for both a positive and negative effect, effects were classified as unclear ($> 5\%$ chance of reaching the substantial threshold for both a positive and negative effect).

RESULTS

3-km Time Trial Performance

Sleep- was detrimental for recovery of 3-km TT performance compared to *Sleep+* (finishing time – ‘*very likely*’; mean power output – ‘*likely*’). *Sleep+* ‘*possibly*’ impaired 3-km TT time (EX1 vs. EX2, $0.5 \pm 2.3\%$), whereas *Sleep-* ‘*very likely*’ impaired TT time ($4.0 \pm 2.2\%$). Likewise, *Sleep+* ‘*possibly*’ impaired average power output ($-1.8 \pm 6.0\%$), while *Sleep-* ‘*very likely*’ impaired average power output ($-14.5 \pm 11.1\%$). Mean time trial performance times are displayed in Fig.1. Mean power outputs, with are displayed in 0.5-km increments for EX2 of *Sleep+* and *Sleep-* in Fig. 2.

Muscle Recovery

Muscle Function

EX1 vs. EX2:

The effects of sleep condition on peak isokinetic torque at both contraction speeds (30°/sec and 120°/sec) were ‘*likely trivial*’. *Sleep+* had a ‘*most likely trivial*’ effect on torque at 30°/sec ($-4.0 \pm 1.5\%$), while *Sleep-* ‘*possibly*’ decreased torque ($-8.1 \pm 6.3\%$). Both sleep conditions had a ‘*likely trivial*’ effect on torque at 120°/sec (*Sleep+*, $-8.1 \pm 4.0\%$; *Sleep-*, $-7.5 \pm 4.7\%$). Raw data for all muscle recovery variables is displayed in *Table 1*.

EX1 vs. EX3:

Sleep condition had a ‘*possible*’ effect on peak isokinetic torque at both contraction speeds. Both *Sleep+* and *Sleep-* had a ‘*likely trivial*’ effect on torque at

30°/sec (*Sleep+*, $-2.3 \pm 7.2\%$; *Sleep-*, $2.2 \pm 7.9\%$). *Sleep+* ‘likely’ impaired peak torque at 120°/sec (EX1 vs EX3, $-7.1 \pm 5.1\%$), whereas *Sleep-* had a ‘possibly trivial’ effect ($-2.7 \pm 7.4\%$).

Muscle Soreness

EX1 vs. EX2:

Sleep- increased muscle soreness rating compared to *Sleep+* (soreness – ‘very likely’). *Sleep+* ‘likely’ decreased muscle soreness ($24.8 \pm 16.7\%$), whereas *Sleep-* ‘possibly’ increased soreness ($13.6 \pm 21.0\%$).

Cardiorespiratory Measures

Heart Rate and Rate of Perceived Exertion (HR & RPE)

Sleep condition had a ‘possible’ effect on resting HR (EX1 vs EX2 – ‘possibly’; EX1 vs EX3 – ‘possibly’). *Sleep+* ‘possibly’ decreased resting HR from EX1 to EX2 ($4.7 \pm 6.1\%$), whereas *Sleep-* had a ‘possibly trivial’ effect on resting HR ($0.5 \pm 10.7\%$). Resting heart rate ‘likely’ decreased under both conditions from EX1 to EX3 (*Sleep+*, $-9.2 \pm 11.3\%$; *Sleep-*, $-11.5 \pm 10.1\%$).

Sleep condition had a ‘possible’ effect on both submaximal- and 3-km TT heart rates (Submaximal 20:00 – ‘possible’; 2-km – ‘possible’; 3-km – ‘possible’).

Submaximal HR was ‘likely’ lower *Sleep+* (EX1 vs EX2, $-2.5 \pm 2.7\%$), whereas *Sleep-* had a ‘possibly trivial’ effect ($-0.8 \pm 3.6\%$). *Sleep+* ‘likely’ decreased 2-km HR ($-3.2 \pm 4\%$), while *Sleep-* ‘very likely’ decreased 2-km HR ($-4.2 \pm 1.5\%$). Similarly, *Sleep+*

'likely' decreased 3-km HR ($-1.8 \pm 1.8\%$), and *Sleep-* 'very likely' decreased 3-km HR ($-3.4 \pm 1.9\%$).

RPE during the 20-min warm-up was 'possibly' affected by sleep condition. *Sleep+* had a 'possibly trivial' effect on RPE (EX1 vs EX2, $-0.2 \pm 3.4\%$), and *Sleep-* 'possibly' increased RPE ($2.5 \pm 5.8\%$).

Oxygen Consumption, Ventilation, & Respiratory Exchange Ratio (VO₂, VE & RER):
EX1 vs. EX2

Sleep condition had no clear meaningful effects on *VO₂*, *VE* or *RER* (*VO₂* – 'possibly trivial'; *VE* – 'possibly trivial'; *RER* – 'possibly'). Both conditions 'likely' increased *VO₂* (*Sleep+*, $4.8 \pm 4.2\%$; *Sleep-*, $5 \pm 5.6\%$) and had a 'possibly trivial' effect on *VE* (*Sleep+*, $0.9 \pm 4.7\%$; *Sleep-*, $1.3 \pm 5.5\%$). *Sleep+* 'most likely' decreased *RER* by $4.7 \pm 1.8\%$, whereas *Sleep-* 'very likely' decreased *RER* by $3.4 \pm 2.1\%$. Raw data for all cardiorespiratory variables is displayed in *Table 2*.

Table 1. Raw data for muscle recovery variables under *Sleep+* and *Sleep-*

Variable	Sleep	EX1	EX2	EX3
Peak Torque 30°/sec (N*m)	+	195.5 ± 74.6	187.7 ± 71.6 ^b	193.6 ± 85.0
	-	196.7 ± 77.0	183.8 ± 76.3 ^b	203.5 ± 83.6 ^c
Peak Torque 120°/sec (N*m)	+	172.3 ± 47.0	157.0 ± 43.0 ^b	159.2 ± 49.6
	-	168.0 ± 46.3	157.0 ± 49.6 ^b	165.1 ± 51.0 ^c
Muscle Soreness (mm)	+	9.9 ± 8.0	30.5 ± 17.0	24.9 ± 17.1
	-	11.5 ± 9.3	31.7 ± 21.5	34.5 ± 20.9 ^a

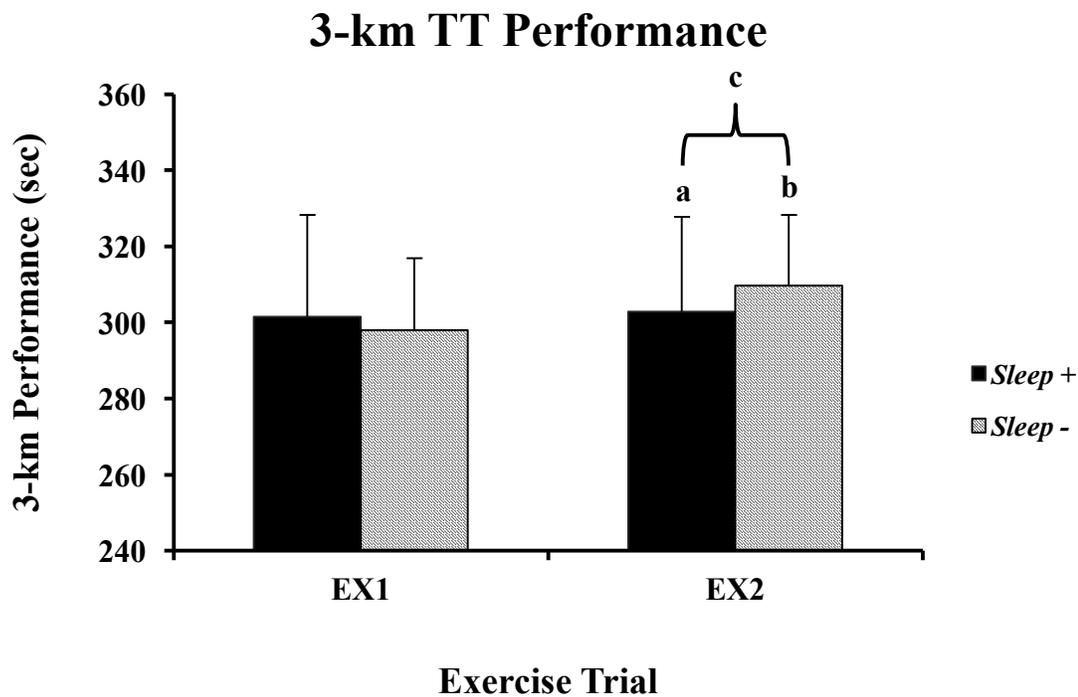
Average (\pm standard deviation) of highest two recorded values of peak isokinetic torque at 30°/sec and 120°/sec during EX1, EX2, and EX3; Muscle soreness taken while walking up and down stairs prior to exercise. Within treatment effects are not included, but are reported in results. (a) ‘*Very likely*’ increased vs *Sleep+*; (b) ‘*Likely trivial*’ effect between conditions; (c) ‘*Possibly*’ increased vs *Sleep+*.

Table 2. Raw data for cardiorespiratory variables under *Sleep+* and *Sleep-*

Variable	Sleep	EX1	EX2	EX3
HR_{rest}	+	58 ± 9	60 ± 6	52 ± 5
	-	61 ± 15	57 ± 2 ^a	53 ± 7 ^b
HR_{20-min}	+	149 ± 11	146 ± 11	-
	-	151 ± 11	149 ± 9 ^b	-
HR_{2-km}	+	177 ± 14	171 ± 8	-
	-	178 ± 12	170 ± 13 ^c	-
HR_{3-km}	+	183 ± 10	179 ± 10	-
	-	182 ± 9	176 ± 10 ^a	-
VO_{2 20-min}	+	2.64 ± 0.4	2.77 ± 0.4 ^c	-
	-	2.57 ± 0.4	2.69 ± 0.4 ^c	-
VE_{20-min}	+	64.83 ± 9.7	65.68 ± 8.9 ^c	-
	-	64.97 ± 10.1	66.16 ± 8.9 ^c	-
RER	+	0.89 ± 0.04	0.85 ± 0.03	-
	-	0.90 ± 0.05	0.87 ± 0.05 ^b	-

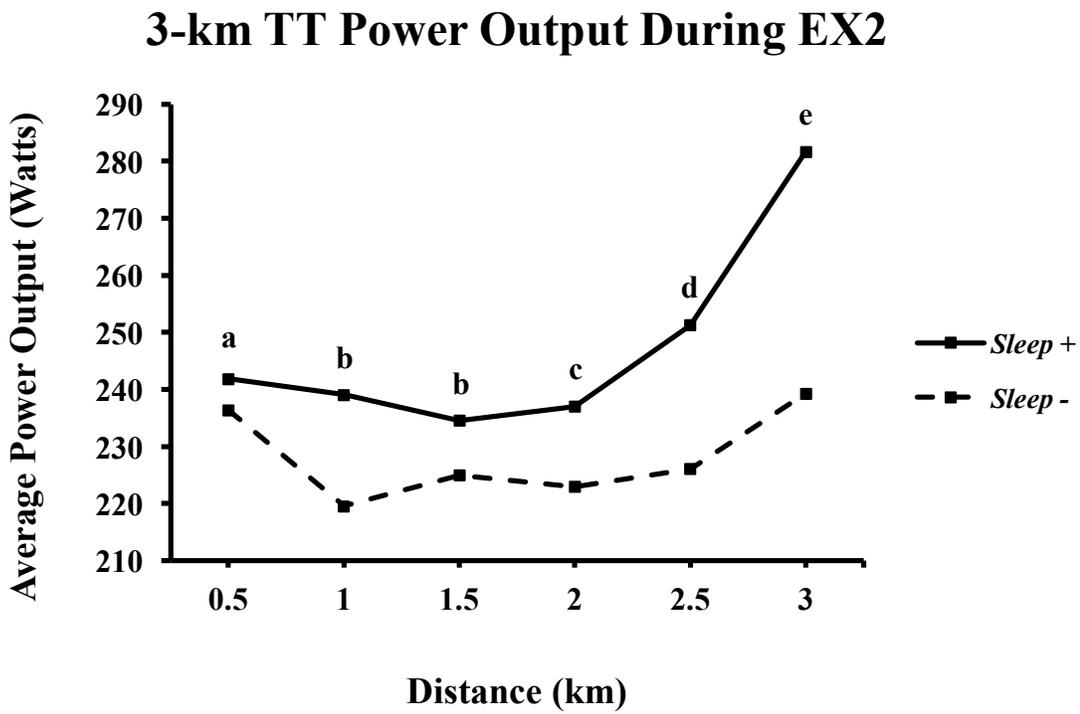
Average (\pm standard deviation) of cardiorespiratory variables at rest, during 20-minute warm-up (20-min), and during 3-km TT (2-km, 3-km): Heart Rate (HR) – beats per minute, Oxygen Consumption (VO₂) – L/min, Ventilation (VE) – L/min, and Respiratory Exchange Ratio (RER). Within treatment effects are not included, but are reported in results. (a) ‘Possibly’ decreased vs *Sleep+*; (b) ‘Possibly’ increased vs *Sleep+*; (c) ‘Possibly trivial’ effect between conditions.

Figure 1. Average 3-km time trial completion (sec) during EX1 and EX2 for *Sleep+* and *Sleep-*



(a) 'Possible' impairment in performance from EX1 to EX2 under *Sleep+*. (b) 'Very likely' impairment in performance from EX1 to EX2 under *Sleep-*. (c) 'Very likely' impairment in performance under *Sleep-* compared to *Sleep+*.

Figure 2. 3-km average power output (Watts) during EX2 in 0.5-km increments for *Sleep+* and *Sleep-*



(a) 'Likely' improvement in EX2 power output vs *Sleep-*. (b) 'Likely' improvement in EX2 power output vs *Sleep-*. (c) 'Possible' improvement in EX2 power output vs *Sleep-*. (d) 'Very likely' improvement in EX2 power output vs *Sleep-*. (e) 'Most likely' improvement in EX2 power output vs *Sleep-*.

DISCUSSION

The purpose of this study was to determine the effects of one night of sleep restriction on subsequent 3-km cycling performance and skeletal muscle recovery from heavy exercise compared to a full night of rest. The primary finding was that a single night of sleep restriction impaired recovery of 3-km cycling performance. This was also accompanied by possibly greater skeletal muscle soreness, possibly decreased 2-km and 3-km HR, and possibly higher RPE during submaximal exercise. Although these variables were potentially altered by sleep restriction, the small magnitude of change in these variables suggests that other unknown factors may be responsible for the detriment in performance.

To our knowledge, this is the first study to assess the impact of a single night of sleep restriction following heavy exercise. Others have documented the effects of sleep restriction on aerobic performance without a previous session of heavy exercise (34, 38), with only one finding a reduction in performance (31). The same study reported decreased time to exhaustion when sleep was restricted at the beginning of the night, as well as a greater detriment to performance when sleep was restricted at the end of the night. Accordingly, the specific type of sleep restriction may explain the mixed outcomes in previous literature. Sleep restriction at the end of the night, similar to the current study, appears to decrease performance (31), whereas sleep restriction in the middle of the night has no clear effect on performance (34, 37). While it is apparent that a single night of sleep restriction, particularly early waking, can impair performance the next morning, very little is known about the interaction between sleep and recovery from

heavy exercise. Previous work on this topic has been exclusive to sleep *deprivation* and impact on strength-related components following sleep *restriction*.

In the context of recovery from heavy exercise, sleep *deprivation* has been associated with decreased aerobic performance during a prolonged self-selected pace sprint interval session (43). Athletes performed three consecutive days of high-intensity intervals accompanied by sleep *deprivation* lasting 30 hrs. Less distance was covered during the final 10-min of sprints, and slower mean sprint times were observed, compared to a full night of sleep. Impaired performance was also accompanied by negative mood states with no increase in perceived effort, which the authors suspect was a result of the self-selected exercise intensity (i.e. lower intensity begets lower RPE). Sleep restriction at the beginning of the night for 3 consecutive nights has also been associated with declined maximal and submaximal weight-lifting performance in the afternoon following the third night, indicating impaired recovery from cumulative sleep loss (39). In agreement with the current study, peak strength remained unaltered following the first night, suggesting that consecutive nights of sleep loss may be more detrimental to maximal strength. However, the exercise protocols and timing of exercise drastically varied between the two studies, as well as the form of sleep restriction. Independent of recovery, sleep *deprivation* appears to have a clearly detrimental effect on aerobic performance (5, 28, 30, 35). Sleep *deprivation* and *restriction* have potentially negative effects on maximal strength and anaerobic performance without recovery from heavy exercise, although the impact is less clear (44, 46–48). Additionally, both sleep *deprivation* and *restriction* have clearly negative psychological implications in a previously rested state.

The detrimental effects of sleep restriction at the beginning and end of the night, as well as chronic restriction (i.e. 2.5-5 hrs per night for 5 days), on neurocognitive performance, motor performance, memory, decision making, and overall mood have been well-documented (4, 6, 10, 18, 36, 39). However, the underlying physiological mechanisms behind the negative implications of sleep restriction are intrinsically complex, particularly as they relate to sport. It is proposed that cerebral metabolism in several regions of the brain is slowed following sleep loss, thereby directly affecting cognitive performance (50, 51). Several studies have detailed the negative implications this may have on exercise performances ranging from combat simulation to weight lifting (11, 26, 36, 39, 53). These implications include, but are not limited to: increased fatigue, reaction time, perceived effort, and decreased vigor. Our results indicate a '*possible*' but subtle increase in RPE (n=2 increased RPE). Similarly, the only other known study to observe impaired aerobic performance after sleep restriction reported no change in RPE during an incremental test to exhaustion (31). However, it is possible that a more precise measurement of fatigue, such as the more extensive Profile of Mood States (POMS) questionnaire, may be necessary to detect changes following sleep restriction. A more comprehensive questionnaire may have revealed whether or not impaired performance was affected by potentially negative performance expectations associated with subject knowledge of sleep restriction.

In addition to psychological stress, sleep restriction may also negatively impact physiological responses to exercise. Typical exercise responses to sleep restriction include, but are not limited to increased heart rate, minute ventilation, and plasma lactate concentration (34). These responses are attributed to the atypical responses to increased

metabolic demand and an augmented catecholamine release in response to the stress of sleep restriction (7, 14, 30). In contrast, we found no changes in submaximal VO_2 , ventilation, or RER. Furthermore, we found a '*possible*' decrease in TT heart rate, which is in agreement with a recent study indicating increased running time to exhaustion with no change in heart rate or perceived exertion (31). It is plausible that the '*possibly*' decreased heart rate was due to effect of heart rate variation from morning to evening, since exercising heart rate is decreased in the morning (49, 56). Since these findings conflict typical responses, even when accompanied by decreased absolute workload, other mechanisms may provide a better explanation for impaired performance following sleep loss than changes in VO_2 , ventilation or RER.

Although not measured in the current study, impaired glycogen replenishment conceivably explains the effect of sleep restriction on performance. In the aforementioned study regarding recovery from 30 hrs sleep *deprivation*, Skein et al. reported decreased muscle glycogen content compared to a full night of sleep (43). Considering sleep deprivation also inhibited sprint performance and pacing strategies during the interval session, negative alterations in muscle metabolism may lead to decreased performance. Training with decreased muscle glycogen content has been associated with decreased RER, suggesting decreased glucose metabolism as a possible explanation for impaired repeated bouts of high-intensity exercise (23). Furthermore, several studies have indicated impaired brain and skeletal muscle glucose metabolism following sleep loss (54, 55, 57). The direct effect of impaired glucose metabolism on exercise has not been studied in the context of sleep *restriction*. However, the clear negative consequences

associated with glucose metabolism impairment could potentially explain impaired performance in the current study.

Our results indicated a '*very likely*' effect of sleep restriction on muscle soreness. While increased muscle soreness may contribute to impaired TT performance, soreness did not impact peak strength. Sleep restriction appears to have a '*trivial*' effect on peak isokinetic torque. While some studies have indicated impaired strength-related components (39), others are in agreement with our findings (16, 37). Interestingly, maximal strength performances appear to be less affected by sleep restriction than submaximal exercises (39). Collectively, these data suggest athletes may be able to overcome detrimental effects of sleep restriction during high-intensity exercises lasting only a few seconds.

In summary, the results of our study indicate impaired 3-km time trial performance following sleep restriction. Although the physiological variables measured in the current study did not explain the detriment to performance, the apparent increase in subjective muscle soreness and '*possible*' increase in perceived exertion suggest neural fatigue as a plausible source of negative consequences of sleep restriction. While the effect of sleep restriction on perceived exertion is unclear, other methods, such as the POMS questionnaire, should be considered in further research. Furthermore, future research on the effects of glycogen depletion and impaired glucose metabolism following sleep restriction may provide better insight on detriments to performance.

PERSPECTIVES

Due to the inherent potential for sleep loss leading up to competition, athletes should aim to minimize external stressors and distractions that may inhibit sleep duration and quality. For instance, athletes may consider arriving to the location of competition several days prior to their event, especially when traveling across time zones.

Additionally, athletes should attempt to minimize noise and light pollution before sleep onset, as well as occupational work when possible. Furthermore, athletes should consider scheduling heavy training sessions around nights in which greater sleep duration and quality are most likely in attempt to optimize recovery from training. Future research is needed to confirm the detrimental effects of sleep restriction on varying aerobic performances and the underlying physiological consequences. Strategies to overcome the detrimental effects of sleep impairment should also be investigated.

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APPENDICES

IRB

James Madison University
Human Research Review Request**FOR IRB USE ONLY:**

Exempt:	<input type="checkbox"/>	Protocol Number:	1 st Review: _____	Reviewer: _____
Expedited:	<input checked="" type="checkbox"/>	IRB: <u>16-0082</u>	2 nd Review: _____	Reviewer: _____
Full Board:	<input type="checkbox"/>	Received: _____	3 rd Review: _____	

Project Title:	<u>The Effects of Sleep Restriction on Recovery from Heavy Exercise</u>		
Project Dates: (Not to exceed 1 year minus 1 day)	From: <u>9/1/15</u> MM/DD/YY	To: <u>8/15/16</u> MM/DD/YY	
Minimum # of Participants:	<u>8</u>		
Maximum # of Participants:	<u>15</u>		
Funding:	External Funding:	Yes: <input type="checkbox"/> No: <input checked="" type="checkbox"/>	If yes, Sponsor: _____
	Internal Funding:	Yes: <input type="checkbox"/> No: <input checked="" type="checkbox"/>	If yes, Sponsor: _____
	Independently:	Yes: <input type="checkbox"/> No: <input checked="" type="checkbox"/>	
Incentives:	Will monetary incentives be offered? Yes: <input checked="" type="checkbox"/> No: <input type="checkbox"/> If yes: How much per recipient? <u>Drawing for \$150 and \$75</u> In what form? <u>Payment Vouchers</u>		
Must follow JMU Financial Policy:	http://www.jmu.edu/financemanual/procedures/4205.shtml#_394IRBApprovedResearchSubjects		
Responsible Researcher(s):	<u>Nicholas Luden</u> <u>Christopher Womack</u> <u>John Chase</u> ludennd@jmu.edu womackcx@jmu.edu chase2jd@dukes.jmu.edu		
E-mail Address:			
Telephone:	<u>540-568-4069</u>		
Department:	<u>Kinesiology</u>		
Address (MSC):	<u>2302</u>		
Please Select:	<input checked="" type="checkbox"/> Faculty	<input type="checkbox"/> Undergraduate Student	
	<input type="checkbox"/> Administrator/Staff Member	<input checked="" type="checkbox"/> Graduate Student	
(if Applicable):			
Research Advisor:	_____		
E-mail Address:	_____		
Telephone:	_____		

Department: _____

Address (MSC): _____

Investigator: Please respond to the questions below. The IRB will utilize your responses to evaluate your protocol submission.

1. **YES** **NO** Does the James Madison University Institutional Review Board define the project as *research*?

The James Madison University IRB defines "research" as a "systematic investigation designed to develop or contribute to generalizable knowledge." All research involving human participants conducted by James Madison University faculty and staff and students is subject to IRB review.

2. **YES** **NO** Are the human participants in your study *living* individuals?

"Individuals whose physiologic or behavioral characteristics and responses are the object of study in a research project. Under the federal regulations, human subjects are defined as: living individual(s) about whom an investigator conducting research obtains:

(1) data through intervention or interaction with the individual; or (2) identifiable private information."

3. **YES** **NO** Will you obtain data through *intervention* or *interaction* with these individuals?

"Intervention" includes both physical procedures by which data are gathered (e.g., measurement of heart rate or venipuncture) and manipulations of the participant or the participant's environment that are performed for research purposes. "Interaction" includes communication or interpersonal contact between the investigator and participant (e.g., surveying or interviewing).

4. **YES** **NO** Will you obtain *identifiable private information* about these individuals?

"Private information" includes information about behavior that occurs in a context in which an individual can reasonably expect that no observation or recording is taking place, or information provided for specific purposes which the individual can reasonably expect will not be made public (e.g., a medical record or student record). "Identifiable" means that the identity of the participant may be ascertained by the investigator or associated with the information (e.g., by name, code number, pattern of answers, etc.).

5. **YES** **NO** Does the study present *more than minimal risk* to the participants?

"Minimal risk" means that the risks of harm or discomfort anticipated in the proposed research are not greater, considering probability and magnitude, than those ordinarily encountered in daily life or during performance of routine physical or psychological examinations or tests. Note that the concept of risk goes beyond physical risk and includes psychological, emotional, or behavioral risk as well as risks to employability, economic well being, social standing, and risks of civil and criminal liability.

CERTIFICATIONS:

For James Madison University to obtain a Federal Wide Assurance (FWA) with the Office of Human Research Protection (OHRP), U.S. Department of Health & Human Services, **all** research staff working with human participants must sign this form and receive training in ethical guidelines and regulations. "Research staff" is defined as persons who have direct and substantive involvement in proposing, performing, reviewing, or reporting research and includes students fulfilling these roles as well as their faculty advisors. The Office of Research Integrity maintains a roster of all researchers who have completed training within the past three years.

Test module at ORI website <http://www.jmu.edu/researchintegrity/irb/irbtraining.shtml>

Name of Researcher(s) and Research Advisor	Training Completion Date
Nicholas Luden	6/25/15
Christopher Womack	4/15/15
John Chase	7/13/15

For additional training interests, or to access a Spanish version, visit the National Institutes of Health Protecting Human Research Participants (PHRP) Course at: <http://phrp.nihtraining.com/users/login.php>.

By signing below, the Responsible Researcher(s), and the Faculty Advisor (if applicable), certifies that he/she is familiar with the

ethical guidelines and regulations regarding the protection of human research participants from research risks. In addition, he/she agrees to abide by all sponsor and university policies and procedures in conducting the research. He/she further certifies that he/she has completed training regarding human participant research ethics within the last three years.

 Principal Investigator Signature

 Date

 Principal Investigator Signature

 Date

 Principal Investigator Signature

 Date

 Faculty Advisor Signature

 Date

Purpose and Objectives

Sleep is a primordial necessity of human life. The National Sleep Foundation recommends that adults sleep between 7 and 9 hours per night (58). However, many Americans fall short of these guidelines. A large-scale sleep study conducted by the American Cancer Society reported that that over half (52%) of the subjects slept less than 7.5 hrs and 76 percent were obtaining less than 8 hrs of sleep (17). Inadequate sleep has a host of behavior and physiological ramifications. Sleep loss can be categorized as either sleep deprivation (SDEP) or sleep restriction (SR). SDEP is the complete absence of sleep, while SR involves interrupted sleep cycles through delayed sleep onset, intermittent waking, or early waking.

SDEP and SR can decrease glucose metabolism in the brain, increased blood pressure, and increase sympathetic nervous system activity (24, 40, 52) – conditions that can impair athletic performance. The effects of insufficient sleep for athletes are of particular interest because of the physiological demands of heavy training and peak performance. Moreover, athletes are particularly susceptible to sleep disruption because of their training and competition habits. This includes both difficulty falling asleep and early waking patterns, perhaps secondary to elevated stress levels (17).

Several studies have demonstrated the negative effects of SDEP and SR using different measures of aerobic, anaerobic, and strength performance (2, 27, 30, 32, 35, 37, 39, 43–45, 48). In addition to physical capacity, sleep disruption perturbs a variety of physiological variables including growth hormone, testosterone, cortisol, and lactate. Collectively, it appears that sleep disruption can negatively influence performance. Relevant to the proposed investigation, the aforementioned work examined how sleep disruption influences performance and/or physiology after several days of light physical activity prior to the performance session. Whether or not more practical sleep disruption (i.e. SR) influences recovery from heavy exercise has not been examined. Therefore, the

purpose of this study is to determine the effects that SR has on subsequent performance and recovery from heavy exercise.

Procedures/Research Design/Methodology

Participants:

8-15 recreationally trained male cyclists will be recruited from James Madison University and the greater Harrisonburg/Rockingham County area. Testing will occur in the Human Performance Lab in Godwin Hall at James Madison University. Participants will be recruited by word-of-mouth, email, and social media. Recruitment of subjects will be limited by inclusion criteria based on responses to general health and habits questionnaires (see Appendix C-E).

Inclusion Criteria

To be eligible for study participation, the subject must meet the following criteria:

- 1) Age: 18-45 years
- 2) Sex: Male
- 3) Recreational cyclists: cyclists are considered “recreational” if cycling consists of a minimum of 30 minutes of cycling exercise, 1-2 day per week, consistently over the past 3 months.
- 4) Subjects are willing and able to give written informed consent, and to understand, participate and comply with the study requirements.
- 5) Do not currently smoke cigarettes.
- 6) Health: Characterized as “low risk” for exercise complications using criteria from the American College of Sports Medicine’s Guidelines for Exercise Testing and Prescription (9th Ed., ACSM, 2014; see Appendix B). Low risk is characterized by the ACSM as individuals “who are asymptomatic and meet no more than one risk factor threshold” from the list below:
 - a) Age: Males \geq 45 yrs
 - b) Family History: Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or other male first-degree relative, or before 65 years of age in mother or other female first-degree relative.
 - c) Cigarette Smoking: Current cigarette smoker or those who quit within the previous 6 months.
 - d) Sedentary Lifestyle: Persons not participating in a regular exercise program or not meeting the minimal physical activity recommendations from the U.S. Surgeon General’s Report.
 - e) Obesity: Body mass index $>$ 30 kg/m².
 - f) Hypertension: Systolic blood pressure \geq 140 mmHg or diastolic \geq 90 mmHg, confirmed by measurements on at least two separate occasions, or on antihypertensive medication.

- g) Dyslipidemia: Low-density lipoprotein (LDL) cholesterol > 130 mg/dl or high-density lipoprotein (HDL) < 40 mg/dl, or on lipid-lowering medication. If total serum cholesterol is all that is available use > 200 mg/dl rather than LDL > 130 mg/dl.
- h) Prediabetes: Impaired fasting glucose = fasting plasma glucose ≥ 100 mg/dL confirmed by measurements on at least two separate occasions.
- i) High-serum HDL Cholesterol: This is considered a “negative risk factor”, so 1 of the above risk factors can be subtracted if HDL > 60 mg/dl.

As recommended by the ACSM, we will use the AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire to identify the above criteria (ACSM, 2010, p. 28; see Appendix B). Before testing is initiated, subjects will be given consent forms to read and sign that provide a comprehensive description of the study, the risks and benefits associated with the study, and the ways in which confidentiality will be maintained (see Informed Consent – Appendix A).

Experimental Design:

This protocol was designed to examine the influence of sleep restriction on recovery from heavy exercise. Subjects will complete a preliminary trial, a familiarization phase, and 2 experimental phases, each separated by approximately 7 days. The familiarization phase and 2 experimental phases will consist of two exercise sessions performed on consecutive days (EX1 and EX2). EX 1 will include baseline performance testing followed by an exercise protocol designed to elicit fatigue. EX 2 will be performed the following morning and will be used to assess recovery from EX 1. EX1 and EX2 performed during the experimental phases will be separated by either a full night of sleep (+) or a night of sleep restriction (-). Accordingly, EX1 and EX2 sessions separated by a full night of sleep will be referred to as EX1+ and EX2+, whereas EX1 and EX2 sessions separated a night of sleep restriction will be referred to as EX1- and EX2-. Order of subject assignment to *Sleep+* and *Sleep-* will be determined using a randomized crossover design. A questionnaire will also be distributed to screen for health history (Appendix E), physical activity habits (Appendix D), and dietary habits (Appendix C). The total time commitment should not exceed 12 hours.

Preliminary Trial (n=1, 60 min):

Informed Consent – Before testing is initiated, subjects will be given consent forms to read and sign that provide a comprehensive description of the study, the risks and benefits associated with the study, and the ways in which confidentiality will be maintained (see Informed Consent).

Body Mass and Height - Subjects will have their body weight measured to the nearest 0.5 kg, and height measured to the nearest 0.5 cm.

VO_{2max} - During this assessment, subjects will perform a graded exercise test to determine their maximal oxygen uptake (VO_{2max}). Subjects will ride a cycle ergometer at a self-selected workload estimated as “a comfortable, but not easy pace for a 1-hour ride”. Workload will be increased by 25 W every 2 minutes until subjects voluntarily request to stop due to fatigue or are unable to continue at a cadence >50 rpm. Oxygen uptake will be assessed at each stage during this test. VO_{2max} will be assessed directly from data obtained during the test and used as a descriptive characteristic.

Familiarization Phase (n=2 visits on consecutive days, 120 min and 60 min, respectively):

The familiarization phase will consist of two consecutive days of exercise trials. Procedures will be the same as the experimental trials detailed below. Exceptions include that no blood samples will be obtained and subjects will not be assigned to a sleep condition.

Experimental Phase

Exercise Trial 1 (n=2, 120 min each)

Subjects will arrive at the human performance lab between 3-5pm, not having consumed alcohol, tobacco or caffeine 24 hrs prior to testing.

Skeletal Muscle Function – Subjects will rest in a seated position for 15 minutes, after which perceived muscle soreness ratings will be obtained using a 0-100mm analog scale (taken after walking up and down stairs). Subjects will warm-up for 5 minutes on a treadmill (3.5 mph) after which they will perform peak muscle function testing. Peak isokinetic concentric muscle force will be assessed following a standardized treadmill warm-up (3.5 mph; 5 min). This test will be conducted using a Biodex muscle function device (Biodex Medical Systems Inc., Shirley NY). Muscle function will be assessed by having subjects push as hard as possible against a shin pad that is connected to an electronic device that controls speed of movement through the leg-extension. Isokinetic leg dynamometry will consist of two warm-up repetitions followed by two maximal exertion isokinetic peak torque measurements at 120 deg/sec. One repetition consists of knee extension immediately followed by knee flexion, with 30 seconds of rest between each maximal exertion repetitions.

3-km Cycling Time Trial – Immediately following skeletal muscle function testing, subjects will perform a 20-min warm-up on a stationary cycle ergometer (Velotron) (10 min at 60% W_{max} followed by 10 min at 70% W_{max}). During the warm-up, various physiological parameters will be profiled (see below). Subjects will then perform a 3-km computer-simulated time trial on the cycle ergometer. This will last approximately 4-7 minutes.

60-minute Sprint Interval – 10 min following completion of the 3-km time trial, subjects will complete 60 min of sprint intervals. **Subjects will only perform the 60-min sprint interval during EX1.** Subjects will pedal at 95% of maximal Watt output (W_{\max}) for 2 min followed by 2 min at 50% W_{\max} . Should subjects fail to maintain a cadence of 50 rpm at 95% W_{\max} , subsequent sprints will be dropped by 10% W_{\max} until cadence is maintained.

Resistance Exercise – 10 minutes following the 60-min sprint interval, subjects will perform 3 sets of 10 at 75% of their peak strength on a leg extension device (Cybex, Medway, MA).

Exercise Trial 2 (n=2, 60 min each)

Subjects will arrive at the human performance lab between 7-9 am, not having consumed alcohol, tobacco, or caffeine 24 hrs prior to testing.

Skeletal Muscle Function – The same muscle soreness ratings described in EX1 will be used. Immediately following, subjects will warm-up for 5 minutes on a treadmill (3.5 mph) after which they will perform peak muscle function testing as described above.

3-km Cycling Time Trial – Immediately following skeletal muscle function testing, subjects will perform a 20-min warm-up on a stationary cycle ergometer (Velotron) (10 min at 60% W_{\max} followed by 10 min at 70% W_{\max}) and perform a 3-km computer-simulated time trial on the cycle ergometer. This will last approximately 4-7 minutes.

Submaximal Physiological Markers:

Heart Rate & Rate of Perceived Exertion (HR & RPE)

During EX trials, HR and RPE will be measured at min 10 and min 20 of the 20-min warm-up preceding the 3-km time trial.

Glucose & Lactate (GLU & LAC)

Plasma GLU and LAC will be obtained via finger-stick samples at min 10 and min 20 of the warm-up preceding the 3-km time trial. Both variables will be assessed using an automated YSI 2300 Stat Plus analyzer (Yellow Springs, OH, USA).

Oxygen Consumption, Ventilation, & Respiratory Exchange Ratio (VO_2 , VE & RER)

VO_2 , VE, and RER will be assessed during EX phases using a Moxus metabolic cart (Pittsburgh, PA, USA). Breath samples will be obtained throughout the 20

min warm-up preceding the 3 km time trials. Minutes 7-10 and 17-20 will be averaged and recorded.

Sleep Protocol:

The first set of EX trials will be separated by either *Sleep+* or *Sleep-*. Subjects will then be assigned the alternate sleep condition 7 days later. Subjects will attempt onset of sleep set at 11:00 pm for both conditions. Onset of sleep will be measured using both an Actigraph accelerometer and a smartphone application that uses motion detection (to be determined – subjects will not be asked to incur possible costs). The accelerometer will be worn on the wrist to detect. The application will be set to wake the subjects 7.5 hrs following sleep onset on *Sleep+*, and 3.5 hrs following sleep onset on *Sleep-*. Sleep will be monitored, but not controlled, during the familiarization phase. Upon arrival for EX2, sleep data will be reviewed to ensure subjects complied with treatment assignment. Should subjects fail to comply, they will be permitted to attempt the phase again after a seven-day washout period. Should subjects fail to comply a second time, the subject will be excluded from the study.

Dietary and Exercise Controls:

Subjects will record all food and beverage intake for 24 hrs preceding EX1. After the initial EX phase, subjects will be provided with copies of their dietary records and instructed to replicate their dietary habits for the second EX phase. Subjects will report to all testing after a >2-hr fast. Subjects will consume Ensure Active High Protein Shake within 1 hour of completing EX1. Additionally, subjects will be instructed to refrain from consuming any other macronutrients during the 2 hrs following EX1. Subjects will be instructed to record all physical activity 72 hrs prior to EX1 in both phases. Subjects will also be instructed to avoid physical activity between EX1 and EX2, and to keep physical activity habits consistent between EX phases.

Risks

Skeletal Muscle Function:

The risks of BioDex muscle function testing and resistance exercise include soreness from exertion 24-48 hours post and potential lightheadedness or loss of consciousness if correct form is not utilized. Participants will be instructed in correct form and breathing techniques prior to testing. Expected soreness will be comparable to that typically experienced following unaccustomed physical activity (i.e. following a pick-up basketball game, long runs, resistance training, etc.)

Sleep Disruption:

The consequences of a single night of sleep restriction comparable to this investigation have not been well documented but include impaired insulin sensitivity, increased sleepiness and fatigue, and reduced alertness and constant attentiveness. The latter have the *potential* to impact short-term academic performance, decision-making and tasks such as driving ability but these have not been documented.

Cardiovascular Testing (3-km Time Trial and VO_{2max} test):

According to the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription, the risk associated with heavy exercise for individuals categorized as "low risk" is very minimal, and physician supervision is not necessary. Any subjects who do not meet the ACSM criteria for "low risk" will not be allowed to participate in the study. In the unlikely event of cardiac or other complications during exercise, an emergency plan is in place. This includes immediate access to a phone to call emergency personnel. In addition, at least one of the listed investigators will be present during the exercise sessions, and all are CPR certified.

Blood Sampling:

The risks of blood sampling using venipuncture include possible mild bruising, and the risk of transfer of blood-borne pathogens, as well as possible risks of infection or skin irritation. These risks are considered to be minimal, and all safety precautions for handling blood samples will be followed according to OSHA protocols, including: investigators will wear latex gloves at all times during blood sampling and testing. A sharps container lined with a biohazard bag will be used for all sharp objects involved in the blood sampling; all other materials (i.e. gloves, gauze pads, etc.) used during the sampling will be put in a separate waste disposal unit lined with a biohazard bag. All investigators who will be involved in blood draws (and handling of blood) have been trained in these phlebotomy techniques, and completed JMU blood-borne pathogen training. A total of ~25 milliliters of blood will be obtained throughout the course of the study, which is roughly 5% of the amount of blood typically obtained during blood donation (1 pint of 473 milliliters)

Benefits

Provided that subjects comply with testing protocols, they will be provided with information regarding their cardiovascular and strength fitness testing. As a whole, the present study may provide useful information for athletes who are particularly susceptible to sleep restriction.

Vulnerable Populations

No data concerning vulnerable populations (minors, prisoners, pregnant woman, neonates, fetuses, cognitively impaired, other protected populations) will be collected during the course of this study.

Research Location

All data will be collected in Godwin Hall, Room 209, at James Madison University.

Deception

No deception will be used.

Time Frame

Data collection will take place immediately following IRB approval until complete data are gathered on 8-15 subjects. Estimated dates: 9/15/15 (pending approval) – 12/1/15.

Data Analysis

Mean power output (Watts) from each 3-km time trial will be used as the performance measure. All data will be log transformed to diminish the effects of non-uniformity. Magnitude-based inferences about the data were derived using methods described by Hopkins and colleagues (Hopkins et al., 2009). For performance, a previously established 'smallest worthwhile change' in performance will be used as the threshold value for a condition effect (EX1 time trial vs. EX2 time trial) (Hopkins, 2004). The smallest worthwhile change in performance will be defined as 0.3 x the within subject variability of select groups of elite cyclists across repeated time trials (CV = 1.5% for time and estimated 4.5% for power)(21). For all other variables, the threshold value for a substantial treatment effect will be defined as 0.2 x within-subject standard deviation, under resting conditions.

Published spreadsheets (Hopkins, 2006a and b) will then be used to determine the likelihood of the true treatment effect (of the population) reaching the substantial change threshold; these percent likelihoods are classified as: <1% almost certainly no chance, 1-5% = very unlikely, 5-25% = unlikely, 25-75% = possible, 75-95% = likely, 95-99% = very likely, and >99% = almost certain. Clinical inference criteria will be used to classify the effects of condition on performance. Specifically, if the percent chance of the effect reaching the substantial change threshold is <25% and the effect is clear, it will be classified as 'trivial'. If the percent chance of the effect reaching the substantial change threshold for benefit exceeds 25% but the chance for harm is >0.5% the effect will be

classified as unclear. An exception to the 0.5% chance of harm criterion will be made if the benefit/harm odds ratio is >66 , in which case the effect will be interpreted as clear and an inference will be assigned.

Separate analyses will be performed on each physiological variable within each condition (*Sleep-* and *Sleep+*) using the spreadsheet mentioned above (Hopkins 2006b). The outcomes from each condition will then be compared using the same spreadsheet (Hopkins 2006b). The classification system detailed above will be applied but mechanistic criteria will be used such that if 90% confidence intervals include values that exceed the substantial change threshold for both a positive and negative effect, effects will be classified as unclear ($>5\%$ chance of reaching the substantial threshold for both a positive and negative effect).

Data Handling

Participation in this research will not be completely anonymous due to the inevitable familiarity of the research team with some of the subjects. However, all subjects will be assigned an individual identification number to ensure that the data remains confidential. All files will be coded with the identification number. Coding sheets with participants' names and corresponding identification numbers along with consent forms will be kept, indefinitely, separately in a locked filing cabinet by Dr. Luden separate from the data files. All hard copies of data (coded with ID number) will be stored, indefinitely, separately in locked file cabinets in the Human Performance Lab. Electronic data and files will be stored, indefinitely, on a password-protected computer, and will only contain de-identified information. Only the identification number will be entered into the computer when creating data spreadsheets and therefore subject's names will not be available to those analyzing and interpreting the data.

Reporting Procedures

Upon request, subjects will receive a summary of their testing, including their VO_{2max} data and performance data within four weeks of the completion of data collection and analysis. Reports will be sent to the subjects by email upon request.

These data will be presented at regional and national conferences, in peer-reviewed exercise science journals, and as pilot data for grant applications. Subjects will not be personally identified in any way in any of these presentations or publications.

Experience of the Researchers

Nicholas D. Luden, Ph.D. is an Associate Professor of Exercise Physiology. He has published >15 peer-reviewed manuscripts in the field of exercise physiology. His primary research interests revolve around skeletal muscle function and how it can be optimized using training and/or nutritional strategies. He has accumulated a substantial amount of

both basic and applied laboratory experience over the past decade, the majority of which has been gained while conducting research on endurance athletes.

Chris Womack, Ph.D. has over twenty years of research experience and has authored or co-authored over forty papers in peer-reviewed exercise science journals. He is a Fellow of the American College of Sports Medicine, the governing body that establishes Guidelines for Exercise Testing and Prescription.

John Chase is a second-year graduate student in Exercise Physiology currently working on his Master's thesis. During his coursework, he has been exposed to numerous research techniques used in exercise testing. This is the first study he has conducted, and he will be under the supervision of his advisor, Nicholas Luden.

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CHAPTER ONE & TWO REFERENCES

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