

The Pennsylvania State University

The Graduate School

Department of Kinesiology

**THE INFLUENCE OF SLEEP AND HEART RATE VARIABILITY ON THE  
OCCURRENCE OF INJURIES, ILLNESSES, AND MISSED PARTICIPATION  
DAYS IN NCAA COLLEGIATE SWIMMERS**

A Thesis in

Kinesiology

by

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**ABSTRACT:**

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Athlete populations are at an increased risk of suffering an injury or illness due to their high level of physical activity. Additionally, athletes may not attain adequate bodily recovery following their training and/or competition sessions. This is particularly true of collegiate athletes who balance the demands of their sport with academic responsibilities. Recent innovations in wearable technology have allowed researchers to track specific recovery metrics such as sleep and heart rate variability. This study utilized the WHOOP Performance Optimization System to measure sleep and heart rate variables in a group of 11 NCAA Division 1 swimmers (Males= 8, Females= 3) for the duration of their season. Data related to covariables including training load and body composition were also recorded. Outcomes of interest were injuries, illnesses, and missed participation days. Descriptive statistics as well as stepwise logistical regression were used to discern the influence of predictor variables on the occurrence of adverse health events. Ten athletes completed data collection and were used for analysis. Average total sleep time was 6.51 ( $\pm$  0.23) hours, and average HRV was 78.08 ( $\pm$  7.54) ms. Regression analysis identified total sleep as a significant predictor of illnesses (OR= 0.70, 95% CI= 0.55-0.89,  $P \leq$  0.01) and missed days (OR= 0.74, 95% CI= 0.61-0.90,  $P \leq$  0.01). Heart rate variability was not identified as a significant predictor of any adverse events. Total yardage (OR= 0.99, 95% CI= 0.99-0.99,  $P \leq$  0.001) and resting heart rate (OR= 1.08, 95% CI= 1.05-1.11,  $P \leq$  0.001) were also identified as significant predictors of missed participation days. These results may be useful in assisting clinicians to make evidence-informed decisions regarding athlete training and recovery in order to reduce the risk of adverse health events. Additional research studies with greater sample sizes are necessary to confirm these preliminary findings.

**TABLE OF CONTENTS**

List of Figures.....	v
List of Tables.....	vi
Acknowledgements.....	vii
Chapter 1: Introduction.....	1
Chapter 2: Methods.....	4
Chapter 3: Results.....	14
Chapter 4: Discussion.....	24
Chapter 5: Conclusion.....	34
Chapter 6: Literature Review.....	35
Appendix A: Recruitment Script.....	70
Appendix B: Participant Informed Consent Form.....	75
Appendix C: Participant HIPAA Release Form.....	80
Appendix D: IRB Approval Letter.....	83
Appendix E: Borg CR10 Perceived Exertion Scale.....	85
Appendix F: Model Variance Inflation Factor Tables.....	86
Appendix G: Individual and Group Moving Average and Acute:Chronic Ratio Tables....	87
Appendix H: 3-day and 7-day Moving Average Figures.....	91
References.....	96

## LIST OF FIGURES

Figure 1: WHOOP Performance Optimization System.....	6
Figure 2: BOD POD.....	10
Figure 3: Total Sleep Data Comparisons Before Injuries.....	91
Figure 4: Total Sleep Data Comparisons Before Illnesses.....	91
Figure 5: Total Sleep Data Comparisons Before First Missed Participation Days.....	92
Figure 6: Total Yardage Data Before Injuries.....	92
Figure 7: Total Yardage Comparisons Before Illnesses.....	93
Figure 8: Total Yardage Data Comparisons before First Missed Participation Days.....	93
Figure 9: RHR Data Comparisons Before Injuries.....	94
Figure 10: RHR Data Comparisons Before Illnesses.....	94
Figure 11: RHR Data Comparisons Before First Missed Participation Days.....	95

**LIST OF TABLES**

Table 3-1: Participant and Group Internal and External Load Data.....	14
Table 3-2: Participant and Group Internal and External Load Data.....	15
Table 3-3: Participant and Group Recovery Data.....	15
Table 3-4: Participant Injury, Illness, and Missed Days Data.....	16
Table 3-5: Participant Illnesses Categorized by Etiology.....	16
Table 3-6: Participant Injuries Categorized by Body Part.....	17
Table 3-7: Participant Injuries Categorized by Mechanism.....	17
Table 3-8: Logistical Regression Model Summary for Injuries.....	19
Table 3-9: Logistical Regression Model Summary for Illnesses.....	19
Table 3-10: Logistical Regression Model Summary for Missed Participation Days.....	20
Table 3-11: Group Descriptive Total Sleep Data.....	21
Table 3-12: Group Descriptive Resting Total Load Data.....	22
Table 3-13: Group Descriptive Resting Heart Rate Data.....	23

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## Chapter 1

### INTRODUCTION

Sports related injuries have a significant effect on the health-related quality of life of student-athletes. The National Collegiate Athletic Association (NCAA) monitors the occurrence of sports related injuries at its member schools through the NCAA Injury Surveillance Program (NCAA-ISP). The most recent report from the NCAA-ISP detailed injury rates during five academic years from 2009-10 through 2013-14. This report documented 478,869 athletes who suffered 1,053,370 injuries during this period, with an average of 210,674 injuries per year. This equated to an estimated 6.0 (95% CI= 5.6-6.0) injuries per 1,000 athlete exposures (i.e. one athlete's participation in one competition or one practice) when analyzing all sports involved in the study. Overall, 21.9% of recorded injuries took greater than 7 days before the athletes could return to full participation.<sup>1</sup> Although less extensive data exists documenting illnesses in athletes, several studies have shown that athlete populations are at high risk of suffering a myriad of pathologies, particularly upper respiratory infections.<sup>2-5</sup> These figures have stimulated increased focus by coaches, clinicians, and researchers on injury/illness prevention strategies, athlete recovery, and objectively-based return-to play progressions. The current study will examine the influence of recovery metrics including sleep and heart rate variability (HRV) on the occurrence of injuries, illnesses, and missed participation days in NCAA Division 1 collegiate swimmers.

Swimming athletes have been used successfully in past studies examining the effect of training load on athlete sleep, injury/illness rates, and psychological profiles.<sup>6-9</sup> . This may be due to the nature of the sport, which emphasizes both high frequency and long duration training sessions. Swimming athletes are also ideal for measuring injury/illness rates as the risk for spontaneous acute injury involved with the activity is low.<sup>1</sup> This is not the case for high impact sports such as football or rugby where an individual could be injured by chance regardless of their state of recovery. The selection of collegiate student athletes as the study cohort is also

important, as these individuals experience life stressors from both academic and athletic responsibilities which could put them at increased risk of injury/illness as well as reduced bodily recovery.<sup>10,11</sup>

The sleep-wake circadian cycle represents a major component of athlete recovery. Although the true functions of sleep are still yet to be fully discovered, it has been reported that sleep is closely related to physical and mental health, cognitive processes, and metabolism.<sup>12</sup> Furthermore, complete rest or sleep is seen as the best method of restoring physical working capacity, as well as mental functioning.<sup>13,14</sup> More specifically, slow wave sleep (SWS) or deep sleep, particularly at the beginning of the night, has been identified as the peak period of human growth hormone (GH) secretion. This hormone has an important effect on muscle growth and repair as well as bone building.<sup>15,16</sup> A notably small number of studies have been conducted exploring the relation between sleep and injury rates in athletes. These have been done in adolescent populations using subjective data and have found that lower amounts of sleep increase the risk of suffering athletically related injuries.<sup>17-19</sup> To our knowledge, no study to date has attempted to discern the effect of sleep variables on the occurrence of adverse health outcomes in a collegiate athlete population using objective data collection methods.

Heart rate variability represents another important parameter related to athlete recovery. Heart rate variability is the variation over time of the period between consecutive heartbeats.<sup>20</sup> This metric is believed to reflect the interplay of the two branches of the autonomic nervous system (ANS): the parasympathetic and sympathetic nervous systems (PNS and SNS, respectively), and their activity as it relates to heart rate (HR) control. Heart rate variability is thought to reflect the health status of the ANS as well as the heart's ability to adapt to changing circumstances.<sup>20</sup> Although specific HRV values are correlated with various disease states and the metric is currently used as a predictor of sudden cardiac death, little research has been conducted to establish HRV norms in athletes or to assess the relation between HRV and the occurrence of injuries/illnesses.

While athlete recovery is a key component in preventing injury/illness it is critical to understand that these negative events do not occur within a vacuum. Other aspects such as training load as well as individual characteristics like body composition also influence an athlete's injury/illness risk.<sup>7,8,21-25</sup> Determining the influence of sleep and HRV on the occurrence of injury/illness in order to identify vulnerable athletes demands a holistic approach that considers multiple inter-related covariables.

Recent advancements in wearable technology have increased the amount and quality of quantitative data related to athletic performance and recovery. To date, no study has explored the relation between sleep and HRV on adverse health outcomes in a collegiate athlete population. The purpose of this study was to utilize an objective method of sleep and HRV detection to discern the influence of these recovery variables on the occurrence of injuries, illnesses, and missed participation days in a group of NCAA Division 1 collegiate swimmers. A wrist-worn activity monitor (WHOOP, Boston, MA) was used to measure sleep and HRV in 11 athlete subjects for the duration of their competitive seasons. Data related to covariables including training load and body composition were also gathered to create descriptive trends as well as a statistical model to quantify the odds of suffering an injury, illness, or missed day. It was hypothesized that sleep restriction and low levels of HRV would increase an athlete's odds of suffering an injury, illness and/or missing participation day when considered along with training load, and body composition.

## Chapter 2

### METHODS

#### Experimental Design

This study utilized a prospective, longitudinal design aimed at discerning the influence of recovery metrics such as sleep and HRV on the occurrence of injuries, illnesses and missed participation days in a group of 11 NCAA Division 1 collegiate swimmers. Athletes were pre-selected by their coaching staff to utilize the WHOOP Performance Optimization System throughout the course of the season to help improve their swimming performance. These 11 individuals were then asked by the research team to allow their data to be analyzed for research purposes. Recruitment materials were approved by Pennsylvania State University's Institutional Review Board (IRB). The approved recruitment materials included a script (Appendix A) recited to the athletes in a private meeting. If a participant met the criteria for inclusion and exclusion, they were asked to read and sign an IRB approved informed consent form (Appendix B).

The research team worked to analyze a range of data collected on subjects by the coaching staff, strength and conditioning team, and sports medicine professionals. These metrics were recorded for 157 consecutive days from September 21, 2017 until February 24, 2018. Recovery metrics HRV, resting heart rate (RHR), total sleep time, sleep efficiency, sleep disturbances, and sleep onset latency, were recorded daily using a wrist-based actigraphy unit. Training load was evaluated via both subjective and objective means. Internal training load was assessed using session ratings of perceived exertion (sRPE) values recorded by the strength and conditioning staff following each team workout (both weight training and swimming) using the Borg CR10 Scale. External load was quantified using both workout duration as well as total swimming distance. Workout duration was recorded daily by the coaches and cross-referenced using notes made by the athletic training staff. Swimming distances were logged by the coaching staff. Height, weight, and body composition data were collected at the beginning of the

competitive season. These testing sessions were conducted by the Sports Nutrition Staff using a BOD POD.

The main outcomes of the study included total injuries, total illnesses, and total missed participation days. Injuries, illnesses, and missed days were calculated using electronic medical record (EMR) data updated by the team's athletic trainers and physicians.

### **Participants**

Study participants included 11 NCAA Division 1 collegiate swimmers (Males= 8, Females= 3) who were independently pre-selected by the coaching staff before the season to wear the WHOOP Performance Optimization System device in an effort to improve swimming performance. The mean age of the subjects was  $20 \pm 1$  years. Subjects were enrolled after providing informed consent to allow their data to be utilized for research purposes. Data was collected on each athlete daily from September 21, 2017 until February 24, 2018. All aspects of this study were approved by the IRB at Pennsylvania State University.

The criteria for inclusion were:

- Male or female athlete on the Penn State Men's and Women's Swimming & Diving Teams between the ages of 18 and 25
- Full-time academic standing as a student taking at least 12 credit hours of coursework
- Medical clearance to participate in sport by the team's head athletic trainers

The criteria for exclusion were:

- Prior history of a diagnosed chronobiological illness or sleep disorder as assessed by the team's physician.

### **The WHOOP Performance Optimization System**

The WHOOP Performance Optimization System (WHOOP, Boston, MA) is a wrist-worn multi-sensor (tri-axial accelerometer, optical sensor, capacitive touch sensor, and ambient temperature sensor) device (Figure1) that samples data at a frequency of 100 Hz.<sup>26</sup> Although

there is currently no peer-reviewed evidence substantiating the validation of this device in assessing objective measures of sleep, actigraphy has been identified by the American Academy of Sleep Medicine as a valid measure to assess sleep-wake periods.<sup>27</sup>



**Figure 1.** WHOOP Performance Optimization System

Source: <http://whoop.com/assets/strap-animations/slide-1/frame.42.png>

WHOOP calculates RHR and HRV via photoplethysmography (PPG). Photoplethysmography involves the use of pulse oximeters which have light emitting diodes (LEDs) that send light waves into the skin. Each cardiac cycle pumps blood to the body's periphery causing distension of vasculature. The change in pulse pressure is detected by illuminating the skin and measuring intensity changes in the light reflected using a photodiode.<sup>28,29</sup> Software algorithms take this signal and combine it with information from the unit's three-dimensional accelerometer to produce meaningful heart rate information.<sup>28</sup> The final HRV metrics produced are equal to the first derivative of the PPG signal.<sup>20</sup> The modulations in

heart rate are measured by WHOOP in the time domain using the root mean square of the successive differences in time between beats (rMSSD)<sup>30</sup> which has been identified in previous studies as a valid means of measuring ambulatory HRV.<sup>31</sup> The system makes this value more intuitive for users by taking the natural log of rMSSD and multiplying it by 20 to create a score ranging from 0-100.<sup>30</sup>

The unique aspect of HRV measurement and calculation by WHOOP is the time when heart rate data is converted to HRV scores. WHOOP has patented its algorithm to compute HRV using a person's heart rate taken during the last 5 minutes of their last stage of slow wave sleep.<sup>30</sup> This is important as many factors affect HRV during waking hours such as hormonal fluctuations, mental health, and physical exertion.<sup>20,32,33</sup> By taking the HRV reading during sleep WHOOP avoids the effects of these variables.<sup>30</sup>

A white paper produced by the WHOOP Analytics Department<sup>26</sup> details the company's efforts to test their product against the gold-standard for sleep measurement, polysomnography (PSG), within a sleep laboratory accredited by the American Academy of Sleep Medicine. WHOOP recruited 30 healthy subjects of a mixed sporting background, 14 of which were Division 1 collegiate athletes. Subjects spent one night in the lab during which their sleep was measured simultaneously using actigraphy and PSG. The WHOOP was 93.1% accurate with a sensitivity of 95.6% a specificity of 80.3% and Cohen's Kappa<sup>2</sup>  $k$  value of 0.75 indicating a high level of statistical agreement compared to PSG. The Band-Altman analysis showed there was almost no bias between WHOOP and the PSG with a mean difference calculation of -0.109 (95% CI= 0.85, -1.07).

In terms of HRV, WHOOP has been tested against a known gold standard, the chest strap Polar model H7 heart rate monitor (Polar, Kempele, Finland). Results of this analysis showed the WHOOP unit to be accurate within 7.9 ms.<sup>34</sup>



### **Sleep and Heart Rate Data**

Sleep variables, RHR and HRV were measured by actigraphy using the WHOOP Performance Optimization System. Sleep and heart rate metrics were defined according to past studies utilizing actigraphy in human subjects research.<sup>35</sup> Total sleep time was defined as the actual time spent asleep in hours calculated from sleep start to sleep end minus any wake time sensed by the WHOOP device. Sleep latency was the difference between sleep onset time calculated by the WHOOP units and the bedtime noted by the participants using the online WHOOP application expressed in minutes. Sleep disturbances were understood as the number of times a person was awakened during a sleep episode, starting from sleep onset to when they became fully awake and did not attempt to go back to sleep. Sleep efficiency was defined as the duration of actual sleep time expressed as a percentage of the total time spent in bed. Heart rate variability was the variation over time of the period between consecutive heartbeats measured in milliseconds.<sup>20</sup> Finally, RHR was expressed as a daily average measured in beats per minute.

Athletes wore the WHOOP device on their non-dominant wrist continuously for the duration of their competitive season. Athletes were instructed to remove the bands only to charge the device or while participating in pool workouts, and competitions. The WHOOP unit's hardware was interfaced with a mobile application via a Bluetooth connection. Data was relayed to a cloud-based analytics platform managed by WHOOP on a daily basis. Representatives from the WHOOP Analytics Department produced reports for each individual athlete compiled together in a spreadsheet format which were provided to researchers weekly throughout the study.

### **Injury, Illness and Missed Participation Day Data**

Student athlete medical records were updated daily by certified athletic trainers as well as physicians upon referral. The electronic medical records of all subjects were reviewed for data pertaining to injuries, illnesses, and missed days following these conditions. Two separate

EMR databases were searched including Blue Ocean (Vivature, Dallas, TX) and PowerChart (Cerner, Kansas City, MO). An injury was defined as any musculoskeletal condition diagnosed by the team's athletic trainers or physicians and noted in the EMR system. An illness was understood to be any adverse general medical condition diagnosed by the athletic trainers or team physicians and noted in the in that athlete's EMR. Finally, a missed day was any day in which an athlete was unable to fully participate in any training session or competition in the opinion of the team's athletic trainers and/or physicians.

Reports spanning from September 21, 2017- February 24, 2018 were generated to include injury and illness incidence using the Vivature-Blue Ocean EMR. An events calendar report was also run using the same start and end dates. This report illustrated the total number of team activities that took place throughout the season, the duration of each of these activities, and which athletes were able to participate. All results within these reports were based on records kept by the team's athletic trainers. The PowerChart EMR was referenced for each subject to acquire information related to injuries specifically referred to a team physician. Demographic information including date of birth, gender, phone numbers, and email addresses were cross referenced from both databases.

Injuries were categorized based on mechanism as well as the affected body region. Mechanism of injury was divided into two groups including acute onset and chronic onset. An acute injury was defined as any injury resulting from a single traumatic event.<sup>36,37</sup> A chronic injury was defined as any injury resulting from multiple repetitive insults.<sup>36,37</sup> In terms of body region, injuries were divided into upper extremity, lower extremity, head and back groupings. Illnesses were categorized based on etiology and categories included upper respiratory, gastrointestinal, skin and other. Injuries, illnesses and missed participation days were each expressed as a total raw value for each individual over the course of their competitive season.

## Body Composition Testing

All subjects participated in body composition testing using the BOD POD (BOD POD, COSMED, Rome Italy) technique to discern their height, weight, and percent body fat. This procedure (Figure 2) involved a 2-compartment body composition model that uses air displacement plethysmography technology to measure both fat and fat-free mass. Past studies have validated this method against a prior gold standard of hydrostatic weighing.<sup>38</sup> The strategy has also been found to possess high test-retest reliability (ICC= 0.991).<sup>39</sup> Prior to using the BOD POD machine, athletes abstained from eating, drinking, and exercise for two hours. The athletes wore form fitting clothing and a swim cap and sat motionless for 8-10 minutes within the BOD POD capsule as the test was conducted. Individual reports with values for height, weight, and percent body fat for all subjects were provided by the team's nutritionist.



**Figure 2.** BOD POD

## **Training Load Data**

External training load was determined based on total swimming distance measured in yards during each team practice by the coaching staff. Additionally, a record of total practice time in minutes was kept for both pool training sessions and lifting/conditioning sessions. All training duration documentation was made on the Vivature-Blue Ocean database via the calendar function by the team athletic trainers. An events calendar report was run, as explained previously within the injury/illness tracking section above, for all team activities between September 21, 2017 and February 24, 2018.

Internal training load was measured using the Borg CR10 Scale.<sup>40</sup> After every training session athletes reported to their sports performance intern and rated their perceived exertion by giving a value from 0-10 as delineated by the Borg CR10 chart seen in Appendix E. Rating of Perceived Exertion are based on the bodily sensations a person experiences during physical activity, including increased heart rate, increased respiration or breathing rate, increased sweating, and muscle fatigue. Session RPE is the overall rating of exertion by subjects during a specified training session or competition. Despite its status as a subjective measure, a person's exertion rating has been shown to provide an accurate estimate of the actual HR during physical activity ( $r= 0.88$ ).<sup>41,42</sup> Total load was expressed in terms of the arbitrary units (AU) method in which load (AU) is the product of the 10-point Borg CR10 rating and the session duration in minutes.<sup>43</sup>

## **Statistical Analysis**

Raw data for 157 consecutive days were analyzed from each subject. Missing data was cleaned using an imputation method based on daily averages. A subject's individual daily average for the given predictor variable was inserted for missing values (e.g. If an athlete was missing their total sleep value on a Monday then this value was replaced with that subject's average total sleep value for all Mondays within the study). A threshold for data cleaning was set at 20% as proposed in past works within this field by Yeats and Martin.<sup>44</sup> Subjects with more

than 20% of their values missing had their data included without cleaning and missing values were accepted as blank inputs.

Descriptive statistics including individual and group means and standard error values were calculated for all variables of interest. Dependent variables were expressed as totals for each outcome across the season to include total injuries, total illnesses, and total missed participation days. Additionally, an incidence rate ratio was created for the injury variable along with a 95% confidence interval (95% CI) as described by Knowles et al.<sup>45</sup> This value was expressed per 1,000 athlete exposures defined as 1 athlete participating in 1 game or practice.<sup>45</sup>

In order to observe the modulation in training load and recovery variables across different time periods leading up to injuries, illnesses, and missed participation days 3-day, 7-day, and 28-day moving averages were calculated. These analyses were done for the following variables including total daily training load (AU), total daily swimming yardage, total daily session duration, total daily sleep, sleep efficiency, sleep disturbances, sleep latency, RHR, HRV, and percent body fat. Acute:chronic ratios were then created by dividing the 7-day moving average for predictor values by the 28-day moving average values as detailed by Gabbet et al.<sup>46</sup>

Multivariate logistical regression modeling with a binomial distribution (1 for the occurrence of an injury/illness/missed day and 0 for non-injury/non-illness/non-missed days) and logit link function was used to quantify the influence of predictor variables on subject odds of suffering an injury, illness, and/or missed participation day. Response variables included total injuries, total illnesses, and total missed days. Predictor variables included RHR, HRV, total sleep time, sleep efficiency, sleep latency, disturbances, total load (AU), total swimming distance per day, total training duration per day, and percentage body fat.

A multivariate model was created for each outcome that included all predictors. An a-priori alpha-level was set at 0.05. A stepwise methodology was used to select the most appropriate predictors from each model according to the Akaike Information Criterion (AIC).<sup>47</sup>

This AIC method has been identified as advantageous in comparison to other variable selection methods within regression analyses<sup>48</sup> The summary statistics used for assessing the adequacy of the models were the null and residual deviances, the chi-square statistic ( $X^2$ ), variance inflation factors (VIF), McFadden's psuedo- $R^2$  and the significance of predictor variables. A chi-square test on the model deviances was executed and  $P$ -values were recorded. A threshold level for variance inflation factors was set at a value of 5<sup>49</sup> and this data can be viewed in Appendix F. The coefficients of the stepwise models were used to produce odds ratios with 95% CI for the occurrence of injuries, illnesses, and missed days. All statistical analyses were conducted using RStudio software and packages (Version 3.4.2, Boston, MA).

## Chapter 3

### RESULTS

Eleven NCAA collegiate swimmers were enrolled in the study and 10 of these subjects completed all aspects of data collection. One individual left the university at the end of the first semester. This subject's data was not included in any statistical analyses as they were not fully exposed to the periodized fluctuations in training and competition load present within a full season and thus their recovery metrics were not considered representative for comparison with those that completed the study.

#### Descriptive Analyses

Demographic information including age, height, weight, and percentage body fat for the subject group can be viewed in Table 3-1. Means and standard errors for recovery variables and training load have been recorded in Table 3-2 and Table 3-3. Table 3-4 shows the total value for all adverse health events collected during the study. Table 3-5 illustrates the categorization of illnesses suffered by the subjects. Tables 3-6 and 3-7 display all injury incidents grouped by body part and mechanism. In terms of the injury rate ratio, 20 total injuries were recorded during 2,194 total athlete exposures during the season. The injury incidence rate ratio was calculated at 9.12 injuries (95% CI= 13.11-5.12) per 1,000 exposure hours.

**Table 3-1.** Participant Demographics

Subject	Age (yrs)	Height (cm)	Weight (kg)	Body Fat (%)
1	21	176.53	85.99	17.8
2	19	182.88	80.46	11.5
3	19	187.96	79.43	13.2
4	21	180.34	73.92	14.7
5	19	170.18	62.07	33.0
6	20	182.88	70.92	9.7
7	20	190.5	83.50	13.6
8	20	185.42	76.39	7.7
9	18	162.56	55.88	17.2
10	18	175.26	70.03	21.8
<b>Group Average</b>	<b>20±1</b>	<b>179.45±2.99</b>	<b>73.86±2.69</b>	<b>16.02±2.29</b>

Mean ± Standard Error, yrs= years, cm= centimeters, kg= kilograms, %= percentage

**Table 3-2. Participant and Group Internal and External Load Data**

Subject	RPE	Total Yardage (yds)	Total Duration (min)	Total Load (AU)
1	3.63±0.20	4369.96±278.29	119.43±6.86	574.57±40.04
2	5.08±0.24	6846.91±395.22	132.71±6.81	812.74±46.31
3	4.64±0.24	4428.50±283.04	116.92±6.92	722.49±49.03
4	4.04±0.25	4131.89±307.14	108.67±7.33	689.19±49.26
5	4.56±0.22	4851.95±431.39	166.66±43.76	746.18±45.98
6	5.45±0.22	7135.07±375.79	135.35±6.47	904.36±46.01
7	5.00±0.24	3310.00±209.30	94.08±6.11	657.49±42.53
8	6.69±0.24	6798.43±370.81	133.71±6.47	1047.88±53.17
9	3.34±0.25	3405.48±299.95	87.48±7.17	529.95±45.11
10	5.28±0.25	6395.31±369.68	128.09±6.81	842.59±49.59
<b>Group Average</b>	<b>4.77±0.31</b>	<b>5167.36±468.66</b>	<b>122.31±7.18</b>	<b>752.74±49.13</b>

Mean ± Standard Error, RPE= Borg Scale Cr-10, yds= yards, min= minutes, AU= arbitrary units

**Table 3-3. Participant and Group Recovery Data**

Subject	Total Sleep (hrs)	Sleep Efficiency (%)	Sleep Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	5.35±0.15	84.11±1.03	6.02±0.31	24.03±8.04	57.55±0.60	74.55±1.91
2	6.13±0.11	88.74±0.46	6.16±0.20	5.45±0.63	41.70±0.26	106.93±1.90
3	6.94±0.14	87.65±0.94	5.80±0.24	10.37±0.92	53.70±0.44	69.63±2.69
4	6.46±0.11	82.54±0.63	5.86±0.20	22.52±1.55	48.50±0.40	82.03±1.70
5	6.67±0.15	90.92±0.95	5.61±0.27	4.21±0.31	57.16±0.66	105.33±2.48
6	7.16±0.10	84.54±0.52	5.76±0.24	19.08±1.36	53.98±0.27	67.81±2.09
7	6.31±0.12	92.26±0.45	5.68±0.27	1.73±0.17	48.18±0.39	77.39±1.39
8	7.09±0.11	90.71±0.35	5.79±0.22	5.47±0.50	61.76±0.28	39.59±0.90
9	7.59±0.08	88.79±0.38	6.12±0.21	12.58±1.11	66.53±0.61	48.29±1.36
10	5.43±0.11	67.71±0.77	4.81±0.17	16.58±1.80	46.74±0.22	109.20±3.47
<b>Group Average</b>	<b>6.51±0.23</b>	<b>85.50±2.25</b>	<b>5.76±0.12</b>	<b>12.20±2.54</b>	<b>53.58±2.37</b>	<b>78.08±7.54</b>

Mean ± standard error, hrs= hours, min= minutes, %= percentage, bpm= beats per minute, ms= milliseconds



**Table 3-4.** Participant Total Injuries, Illnesses, and Missed Days

<b>Subject</b>	<b>Injuries</b>	<b>Illnesses</b>	<b>Missed Days</b>
1	2	7	8
2	3	0	0
3	3	4	5
4	4	5	20
5	0	3	9
6	0	1	0
7	3	4	3
8	1	0	0
9	3	4	46
10	1	3	0
<b>Total</b>	<b>20</b>	<b>31</b>	<b>91</b>

**Table 3-5.** Participant Illnesses Categorized by Etiology

<b>Subject</b>	<b>Respiratory</b>	<b>Skin</b>	<b>GI</b>	<b>Other</b>	<b>Total Illnesses</b>
1	5	1	1	0	7
2	0	0	0	0	0
3	1	0	1	1	3
4	5	0	0	0	5
5	3	0	0	0	3
6	0	0	0	1	1
7	5	0	0	0	5
8	1	0	0	0	1
9	3	0	0	0	3
10	3	0	0	0	3
<b>Total</b>	<b>26</b>	<b>1</b>	<b>2</b>	<b>2</b>	<b>31</b>

**Table 3-6.** Participant Injuries Categorized by Body Part

<b>Subject</b>	<b>Upper</b>	<b>Lower</b>	<b>Head</b>	<b>Back</b>	<b>Total Injuries</b>
1	1	1	0	0	2
2	3	0	0	0	3
3	1	1	0	1	3
4	1	1	2	0	4
5	0	0	0	0	0
6	0	0	0	0	0
7	1	2	0	0	3
8	1	0	0	0	1
9	0	1	0	2	3
10	1	0	0	0	1
<b>Total</b>	<b>9</b>	<b>6</b>	<b>2</b>	<b>3</b>	<b>20</b>

**Table 3-7.** Participant Injuries Categorized by Mechanism

<b>Subject</b>	<b>Acute Injuries</b>	<b>Chronic Injuries</b>	<b>Total Injuries</b>
1	2	0	2
2	0	3	3
3	2	1	3
4	3	1	4
5	0	0	0
6	0	0	0
7	3	0	3
8	0	1	1
9	1	2	3
10	0	1	1
<b>Totals</b>	<b>11</b>	<b>9</b>	<b>20</b>

### **Logistical Regression Statistics**

All predictor variables were included in multivariate analyses during which separate models were created using injuries, illnesses, and missed days as the response variables. A stepwise method was used to select the most significant predictors with AIC representing the criterion measure. Odds ratios with 95% CI and *P*-values were calculated for each predictor. Results from the analyses can be viewed in tables 3-8 through 3-10.

### **Total Injuries**

Multivariate stepwise analysis selected sleep efficiency, RHR, and total load as predictors. This model was found to be statistically significant using a chi-square test for model deviance ( $P \leq 0.05$ ). The multivariate model summary showed that none of these variables were significant predictors of total injuries at the alpha level set at 0.05. Total load ( $P = 0.07$ ) and RHR ( $P = 0.07$ ) approached significance.

### **Total Illnesses**

Multivariate stepwise analysis selected total sleep and total yardage as predictors. This model was identified as statistically significant using a chi-square test for model deviance ( $P \leq 0.01$ ). The multivariate model summary showed that total sleep was a significant predictor of illness with an odds ratio of 0.70 (95% CI= 0.55-0.89,  $P \leq 0.01$ ). This can be interpreted as; those athletes who attained higher amounts of total sleep reduced their odds of suffering an illness by 30%. Total yardage was not found to be a statistically significant predictor of illness (OR= 1.00, 95% CI= 0.99-1.00,  $P = 0.14$ ).

### **Total Missed Participation Days**

Multivariate stepwise analysis selected total sleep, total yardage, RHR, and sleep efficiency as predictors. This model was found to be statistically significant using a chi-square test for model deviance ( $P \leq 0.001$ ). The model summary showed that total sleep (OR= 0.74, 95% CI= 0.61-0.90,  $P \leq 0.01$ ), total yardage (OR= 0.99, 95% CI= 0.99-0.99,  $P \leq 0.001$ ), and RHR (OR= 1.08, 95% CI= 1.05-1.11,  $P \leq 0.001$ ) were significant predictors of missed days. These odds ratios can be interpreted as those athletes who achieved more hours of total sleep per day reduced their odds of missing a participation day by 26%. Athletes who swam more total yards per day reduced their odds of missing a participation day by 1%. Finally, subjects with higher daily RHR increased their odds of missing a participation day by 8%. Sleep efficiency was found to be insignificant when placed in the model (OR= 1.03, 95% CI =0.99-1.07,  $P = 0.16$ ).

**Table 3-8.** Logistical Regression Model Summary for Injuries

<b>Independent Variable</b>	<b>b</b>	<b>se</b>	<b>z-ratio</b>	<b>P-value</b>	<b>OR (95% CI)</b>
(Intercept)	-7.85	3.66	-2.15	0.03*	3.90e-04(1.17e-7-0.05)
Sleep Efficiency	0.07	0.04	1.80	0.07	1.07 (1.00-1.17)
RHR	-0.06	0.03	-1.80	0.07	0.94 (0.87-1.00)
Total Load	6.83e-04	4.82e-04	1.42	0.16	1.00 (0.99-1.00)
<b>Model Diagnostics</b>					
Psuedo-R <sup>2</sup> = 0.05					
$\chi^2= 1074.10$		$P \leq 0.05^*$			
Null Deviance= 163.34 on 1284 df		AIC= 162.75			
Residual Deviance= 154.75 on 1281 df					

OR= odds ratio, se= standard error, df= degrees of freedom

\*Significant at  $P \leq 0.05$

**Table 3-9.** Logistical Regression Model Summary for Illnesses

<b>Independent Variable</b>	<b>b</b>	<b>se</b>	<b>z-ratio</b>	<b>P-value</b>	<b>OR (95% CI)</b>
(Intercept)	-1.24	0.79	-1.58	0.11	0.29(0.05-1.25)
Total Sleep	-0.36	0.12	-3.00	$\leq 0.01^{**}$	<b>0.70 (0.55-0.89)</b>
Total Yardage	-6.73e-05	$\leq 0.001$	-1.46	0.14	1.00 (0.99-1.00)
<b>Model Diagnostics</b>					
Psuedo-R <sup>2</sup> = 3.47e-02					
$\chi^2= 1348.11$		$P \leq 0.01^{**}$			
Null Deviance= 269.66 on 1284 df		AIC= 266.31			
Residual Deviance= 260.31 on 1282 df					

OR= odds ratio, se= standard error, df= degrees of freedom

\*Significant at  $P \leq 0.05$

\*\*Significant at  $P \leq 0.01$

**Table 3-10.** Logistical Regression Model Summary for Missed Participation Days

<b>Independent Variable</b>	<b>b</b>	<b>se</b>	<b>z-ratio</b>	<b>P-value</b>	<b>OR (95% CI)</b>
(Intercept)	-5.87	1.58	-3.71	≤0.001***	0.29 (0.57-1.25)
Total Sleep	-0.29	0.10	-3.07	≤0.01**	<b>0.74 (0.61 – 0.90)</b>
Total Yardage	-1.0e-02	1.99e-04	-5.24	≤0.001***	<b>0.99 (0.99-0.99)</b>
RHR	0.08	0.01	6.00	≤0.001***	<b>1.08 (1.05-1.11)</b>
Sleep Efficiency	0.03	0.02	1.42	0.16	1.03 (0.99-1.07)
<b>Model Diagnostics</b>					
Psuedo-R <sup>2</sup> = 0.43					
χ <sup>2</sup> = 1710.39		P≤ 0.001***			
Null Deviance= 625.95 on 1284 df		AIC= 363.92			
Residual Deviance= 353.92 on 1280 df					

OR= odds ratio, se= standard error, df= degrees of freedom

\* Significant at  $P \leq 0.05$

\*\* Significant at  $P \leq 0.01$

\*\*\* Significant at  $P \leq 0.001$

### **Moving Average and Acute:Chronic Ratio Analyses**

The individual 3-day and 7-day moving averages as well as the acute:chronic ratios prior to the three outcomes of interest for all variables can be seen in Appendix G. Tables 3-11 through 3-13 show group average 3-day and 7-day moving averages as well as acute:chronic ratio data prior to each adverse health outcome for variables identified as significant predictors within the logistic regression analyses. These variables included total sleep, total yardage, and RHR. Group averages for the season are also listed in these tables for comparison. Table 3-11 for total sleep also includes the daily recommendations for total sleep for adults age 18-65 years from the American Academy of Sleep Medicine and Sleep Research Society<sup>50</sup> as well as sleep recommendations for athletes mentioned previously by Bompa et al<sup>14</sup>. Graphical representations of the 3-day and 7-day moving averages have been produced and can be viewed in Appendix H. With regards to the missed days data, if the athletes missed a series of consecutive days each value for the averages was taken using the first missed day in that series as the reference.

Table 3-11 depicts the modulations in sleep across differing time intervals prior to each adverse health event. The season average for total sleep for the group was 6.51 ( $\pm$  0.23). On average subjects got just 6.02 ( $\pm$  0.29) hours of sleep per night during the 3-day period before an injury and 6.16 ( $\pm$  0.36) hours of sleep per night during the 7-day period before an injury. These values were only slightly higher ahead of an illness with the average 3-day moving average calculated at 6.21 ( $\pm$  0.33) hours and the average 7-day moving average calculated at 6.41 ( $\pm$  0.33) hours. Moving averages prior to missed participation days were similar to those prior to injuries with a 3-day moving average of 6.28 ( $\pm$  0.52) hours and a 7-day moving average of 6.20 ( $\pm$  0.48). Average acute:chronic ratios for the group were found to confirm the variations in moving averages with values of 0.96 ( $\pm$  0.01) prior to injury, 0.98 ( $\pm$  0.02) prior to illness, and 0.96 ( $\pm$  0.02) before missed participation days.

**Table 3-11. Group Descriptive Total Sleep Data**

<b>Group Season Average</b>	6.51 $\pm$ 0.23		
<b>Recommendation for Healthy Adults*</b>	7 <sup>3</sup>		
<b>Recommendation for Athletes*</b>	9-10 <sup>4</sup>		
	<b>Condition</b>		
	<b>Before Injury</b>	<b>Before Illness</b>	<b>Before Missed Day</b>
<b>Average 3-day Moving Average*</b>	6.02 $\pm$ 0.29	6.21 $\pm$ 0.33	6.28 $\pm$ 0.52
<b>Average 7-day Moving Average*</b>	6.16 $\pm$ 0.36	6.41 $\pm$ 0.33	6.20 $\pm$ 0.48
<b>Average Acute:Chronic Ratio</b>	0.96 $\pm$ 0.01	0.98 $\pm$ 0.02	0.96 $\pm$ 0.02

Means and standard errors

\*Sleep measured in hours/night

In addition to the athlete recovery data, modulations in total yardage data can be seen within Table 3-12. Total yardage per day during the 3 days leading up to an injury was found to be 5921.33 ( $\pm$  675.38) yards in reference to the season average of 5167.36 ( $\pm$  468.66). The 7-

day moving average value before these events was 5715.64 ( $\pm$  683.41) yards. In contrast, prior to illnesses total yardage was found to be 4727.58 ( $\pm$  434.86) yards per day during the three days leading up to illness and 5173.01 ( $\pm$  445.95) yards per day during the 7 days leading up to these events. Ahead of missed days the group average 3-day average for total yardage was 2691.80 ( $\pm$  530.18) yards and the average 7-day moving average was 3476.15 ( $\pm$  357.46) yards. The acute:chronic ratios were 0.93 ( $\pm$  0.06), 0.93 ( $\pm$  0.06), and 0.95 ( $\pm$  0.11) for days on which athletes suffered an injury, illness, and first missed day respectively.

**Table 3-12.** Group Descriptive Resting Total Yardage Data

<b>Group Season Average*</b>	<b>Condition</b>		
	<b>Before Injury</b>	<b>Before Illness</b>	<b>Before Missed Day</b>
	5167.36 $\pm$ 468.66		
<b>Average 3-day Moving Average*</b>	5921.33 $\pm$ 675.38	4727.58 $\pm$ 434.86	2691.80 $\pm$ 530.18
<b>Average 7-day Moving Average*</b>	5715.64 $\pm$ 683.41	5173.01 $\pm$ 445.95	3476.15 $\pm$ 357.46
<b>Average Acute: Chronic Ratio</b>	1.07 $\pm$ 0.05	0.93 $\pm$ 0.06	0.95 $\pm$ 0.11

Means and standard errors

\*Distance measured in yards

Table 3-13 shows the variation in RHR ahead of each adverse health event. The season average for RHR for the group was 53.58 ( $\pm$  2.37) bpm. A noteworthy variation in RHR can be seen prior to missed day events with an average 3-day moving average of 55.09 ( $\pm$  2.93) bpm and an average 7-day moving average of 55.68 ( $\pm$  2.95) bpm before these events. The acute:chronic ratio was neutral at 1.00 ( $\pm$  0.03). Prior to injuries the group average 3-day moving average was 51.13 ( $\pm$  2.71) bpm, the 7-day average moving average was 51.12 ( $\pm$  2.69) bpm and the average acute:chronic ratio was 0.99 ( $\pm$  0.01). Finally, before illness the 3-day moving RHR average was 53.46 ( $\pm$  1.93) bpm, the 7-day moving average was 54.13 ( $\pm$  2.13) bpm, and the average acute:chronic ratio value was 0.99 ( $\pm$  0.01).

**Table 3-13.** Group Descriptive Resting Heart Rate Data

<b>Group Season Average*</b>	53.58 ± 2.37		
	<b>Condition</b>		
	<b>Before Injury</b>	<b>Before Illness</b>	<b>Before Missed Day</b>
<b>Average 3-day Moving Average*</b>	51.13 ± 2.71	53.46 ± 1.93	55.09 ± 2.93
<b>Average 7-day Moving Average*</b>	51.12 ± 2.69	54.13 ± 2.13	55.68 ± 2.95
<b>Average Acute: Chronic Ratio</b>	0.99 ± 0.01	0.99 ± 0.01	1.00 ± 0.03

Means and standard errors

\*RHR measured in beats per minute (bpm)



## Chapter 4

### DISCUSSION

The purpose of this study was to identify the influence of specific recovery metrics including sleep and HRV on the occurrence of injuries, illnesses and missed participation days in NCAA Division 1 collegiate swimmers. It was hypothesized that fewer hours of sleep and lower values of HRV would be associated with an increased odds of sustaining an injury, illness and/or missing a participation day. A second aim of the study was to produce descriptive statistics for sleep, heart rate, and training load variables. Descriptive data for these metrics have not been well-established in the collegiate athlete population nor have modulations in these variables been observed leading up to adverse health outcomes such as injury, illness, and missed participation days.

#### **Injuries**

The stepwise logistical regression analyses conducted within the current study selected sleep efficiency, RHR and total load as predictor variables of injuries, however, none of these were found to be statistically significant. Both total load and RHR did show a trend toward significance ( $P= 0.07$ ). When considering the descriptive statistics for this group of variables Table-4 shows that the subjects' season average for total daily load was 752.74 ( $\pm 49.13$ ) AU and RHR was 53.58 ( $\pm 2.37$ ) bpm. When considering variations in these independent variables leading up to injury events, 3-day and 7-day moving average tables for injuries in Appendix G show that the average 3-day moving average for total load was 952.43 ( $\pm 63.17$ ) AU and the average 7-day moving average before these incidents was 917.53 ( $\pm 70.66$ ) AU. These moving average values for total load prior to injuries appear to be higher in comparison to the group's season average. The average acute:chronic ratio for load prior to injury seems to support this relation with a value of 1.10 ( $\pm 0.08$ ) suggesting that the 7-day moving average for total load prior to injury was higher in comparison to the 28-day moving average. Results for RHR include an average 3-day moving average prior to injury of 53.46 ( $\pm 1.93$ ) bpm, and an average 7-day

moving average prior to injury of 54.13 ( $\pm$  2.13) bpm. Group average acute:chronic ratio for RHR ahead of injury was  $0.99 \pm 0.01$ .

Of these two variables, training load has been more widely studied in relation to the occurrence of injury. Past research by Sein et al<sup>7</sup> evaluated the pathogenesis of shoulder pain in 80 young elite swimmers and found that supraspinatus tendinopathy could be predicted in 85% of swimmers either from number of hours swum per week alone or in combination with the swimmer's weekly mileage. Additional studies argue that excessive and rapid increases in load identified by high values of acute:chronic ratios increase the likelihood of injury in athletes.<sup>21,22</sup> Greater numbers of subjects in future studies may reveal a statistically significant outcome for either total load or RHR with regard to injury occurrence.

### **Illnesses**

Total sleep was found to be a significant predictor of illness by the logistical regression analyses. Those athletes who achieved more hours of sleep per night during the season had a reduction in their odds of sustaining an illness of 30% (OR= 0.70, 95% CI= 0.55-0.89,  $P \leq 0.01$ ). Descriptive statistics for total sleep in relation to illnesses can be found in Table 3-11 as well as Figure 4 (Appendix H). On average, subjects got just 6.21 ( $\pm$  0.33) hours of sleep per night during the 3 days leading up to an illness and 6.41 ( $\pm$  0.33) hours per night during the 7-day period before these events. These values appear to be lower compared to the group's season average (6.51 ( $\pm$  0.23 hours) as well as sleep recommendations for healthy adults (7 hours)<sup>50</sup> and athletes (9-10 hours).<sup>14</sup> Deficits in total sleep time prior to the occurrence of illness support our findings that athletes who achieve more sleep reduce their odds of suffering these adverse health events.

The protective effect of increased sleep on total illness found in this study is in agreement with multiple researchers that have examined the effect of poor sleep on the immune system. In a review by Bryant et al<sup>51</sup>, the authors concluded that even minor sleep loss, accumulated over time, has a considerable negative impact on the human immune response. A

pair of studies by Irwin et al<sup>52,53</sup> demonstrated how sleep restriction can cause decreases in the number and activity of circulating natural killer (NK) cells of the innate immune system. This same research group also studied the effect of limited sleep on the production of pro-inflammatory cytokines including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-a). Both transcription and monocyte production of these cytokines were found to be increased following sleep loss.<sup>54</sup> Still other researchers have found that sleep restricted individuals have a reduced vaccine immune response<sup>55,56</sup> and an increased likelihood to contract an upper respiratory infection during a pathogen exposure challenge.<sup>57</sup> The findings from Cohen et al<sup>57</sup> are in agreement with the current project as 84% of conditions suffered by the collegiate athlete subjects (Table 3-4) were categorized as upper respiratory infections.

### **Missed Participation Days**

The major findings from the logistical regression analyses were that total sleep, total yardage and RHR were each selected as significant predictors of missed participation days. Those athletes who achieved more hours of sleep per night during the season had a reduction in their odds of missing a day of participation of 26% (OR= 0.74, 95% CI= 0.61-0.90,  $P \leq 0.01$ ). Furthermore, those athletes who swam more yards per day were shown to have a modest decrease in their odds of missing a participation day of 1% (OR= 0.99, 95% CI= 0.99-0.99,  $P \leq 0.001$ ). Finally, athletes with higher daily RHR were found to increase their odds of missing a participation day by approximately 8% (OR= 1.08, 95% CI= 1.05-1.11,  $P \leq 0.001$ ).

Results from the moving average and acute:chronic ratio data seem to support the outcomes of our regression analyses. Table 3-11 as well as Figure 5 (Appendix H) appear to show reduced values of total sleep in reference to the group's season average (6.51 ( $\pm 0.23$  hours) prior to missed days with average 3-day and 7-day moving averages of 6.28 ( $\pm 0.52$ ) hours and 6.20 ( $\pm 0.48$ ) hours respectively. This may suggest that athletes experience reduced sleep during 3-day and 7-day periods before missing participation days. In terms of total yardage before a missed day the average 3-day (2691.80  $\pm$  530.18 yards) and 7-day (3476.15  $\pm$

357.46 yards) moving average values also appear to be lower compared to the group's season average ( $5167.26 \pm 468.66$  yards) as can be observed in Table 3-12 and Figure 8 (Appendix H). Table 3-13 and Figure 11 (Appendix H) show the average 3-day ( $55.09 \pm 2.93$  bpm) and 7-day ( $55.68 \pm 2.95$  bpm) moving averages for RHR prior to missed days compared to the season average ( $53.58 \pm 2.37$  bpm). These descriptive statistics show that RHR across 3-day and 7-day time periods leading up to missed days appear elevated relative to the group's season average.

Missed time has not been well studied in relation to sleep. The protective effect of proper sleep on missed participation days in the current study could be explained by a myriad of different mechanisms. As discussed previously, more sleep appears to reduce the odds of suffering illness in our group of collegiate swimmers which in turn could result in fewer missed participation days. Poor sleep has also been shown to modulate the perception of pain in a variety of different populations. Past studies have shown that sleep restricted individuals were more likely to report musculoskeletal pain<sup>58,59</sup>, had decreased mechanical pain thresholds<sup>59</sup>, had increased pain sensitivity<sup>60</sup>, and had reduced ability to attenuate painful episodes.<sup>60</sup> Increased levels of sleep may play a role in reducing pain perception secondary to injury and/or illness as well as other sources of pain such as delayed onset muscle soreness (DOMS). Reduced pain perception secondary to injury, illness, and/or training could reduce the number of missed participation days in those athletes who attained more sleep. This information may be important for coaches and clinicians as they work to limit the number of days an athlete is unable to participate in their sport.

The negative influence of increased training load on the occurrence of injuries and illnesses in athletes has been well researched,<sup>7,8,21-23</sup> but less work has been done exploring its effect on missed participation days. Considering lack of participation could have resulted from adverse health events, one may expect that higher total yardage would increase the odds of these missed days. Our findings suggest the opposite to be true, however, with higher amounts

of total yardage associated with lower odds of athletes missing a participation day (OR= 0.99, 95% CI= 0.99-0.99,  $P \leq 0.001$ ). One potential reason for this result could be the training intensity covariable. It is common for coaches to decrease the swimming yardage during a training session when the intensity of the training is increased. Therefore, the intensity of a training session and not necessarily the total swimming distance may have had the greatest influence on the odds of missing participation days. Training intensity was measured within the current study using sRPE according to the Borg CR10 scale as well as the total load (AU) metric defined as the product of training duration in minutes (both swimming and weight training) and sRPE. The table for average acute:chronic ratios before first missed participation days in Appendix G shows that the subjects' average acute:chronic ratio for total load was 1.14 ( $\pm 0.12$ ). This value suggests that the 7-day moving average for total load was larger than the 28-day moving average for this metric leading up to these events. The average acute to chronic ratio for sRPE was found to be 1.13 ( $\pm 0.16$ ). Thus, moving average data for total load and sRPE in relation to total yardage data suggests that as yardage decreases before a missed participation day, intensity appears to increase. Further research could attempt to parse this idea by categorizing workouts according to intensity level and relating these groupings back to adverse health events.

The final predictor of missed participation days was RHR. Athletes with higher RHR were found to be at increased odds of missing a participation day. Increases in RHR could be explained by increases in training intensity prior to missed participation days. As the intensity of activity increases, an individual's heart is required to beat faster to supply the body's tissues with oxygen rich blood. An alternative explanation can be derived from the logic used when proposing HRV as a potential significant predictor. Higher heart rates may suggest increased SNS activity,<sup>61</sup> meaning the heart and body are subject to chronically increased levels of stress.<sup>62</sup> High levels of RHR have also been associated with overtraining in athletic populations. A study by Dressendorfer et al<sup>63</sup> found that increased morning RHR was a valid sign of

overtraining in a group of 12 male runners. Medical professionals and coaches commonly allow athletes to miss practice time if they observe signs of overtraining.

In this study injuries and illnesses were recorded regardless of whether they resulted in a missed participation day or not. Missed participation days only occurred secondary to more serious injuries or illnesses. Therefore, while increased RHR was not a significant predictor of the overall number of injuries and illnesses, chronically high levels of RHR may identify individuals at increased risk of suffering the most serious adverse health events. Coaches and clinicians could monitor this metric over time and provide additional rest for individuals with chronically elevated RHRs.

### **Heart Rate Variability**

Heart rate variability was not selected as a significant predictor of any adverse health outcome in the current study. It was proposed that decreased levels of HRV, signaling an increase in the activity of the SNS and/or a decrease in the activity of the PNS<sup>20</sup>, would be associated with the occurrence of injury, illness, and missed participation days. This hypothesis was not supported. Findings within the literature relating to the influence of HRV on injury occurrence is fairly contradictory, possibly due to a lack of agreement with respect to study design.<sup>33</sup> The findings of this project are in support of those from Hedelin et al<sup>64</sup> who found no meaningful difference in HRV indices during pre-post training camp testing in a group of over-trained elite canoeists. Their findings are refuted by a number of other studies which have noted decreases in HRV metrics in injured, and over-trained individuals.<sup>65-70</sup> The effect of HRV on illness is more established with a majority of studies showing decreases in HRV to be related to illness<sup>69,70</sup> and even using HRV to successfully diagnose specific conditions.<sup>71</sup> The reduced sample size of the current study may have affected results for this variable.

### **Sleep and Heart Rate Variability Season Average and General Population Comparisons**

No study to date has attempted to objectively study the sleeping habits of NCAA collegiate athletes. This is surprising considering that past research involving both professional

and adolescent athletes have shown that these populations suffer from reduced sleep duration and sleep quality.<sup>9,35</sup> Additionally, college students have a unique lifestyle compared with the general population and often experience a high level of academic pressure while attempting to maintain a balanced social life which can affect sleeping patterns. Several reports have shown university/college students suffer from chronic sleep problems and disruptions.<sup>72-74</sup> Collegiate athletes may be at an increased risk for such sleep problems as they must balance their academic and social responsibilities while also training for 20 hours per week, attending competitions, and traveling.<sup>75</sup>

The group mean for total sleep in the current study was 6.51 ( $\pm$  0.23) hours. This value is about 29.4 minutes lower than the recent recommendations for sleep in healthy adults aged 18-65 years of 7 hours of sleep per night put forth by The American Academy of Sleep Medicine and Sleep Research Society.<sup>50</sup> Further recommendations made specifically for athletes were established by Bompa and Haff<sup>14</sup> who proposed that athletes require 9 to 10 hours of sleep, with 80-90% of it during the night, in order to properly recover. Our subjects fell short of this standard by approximately 2 and  $\frac{1}{2}$  hours.

Sleep quality was also shown to be reduced compared to the general population according to the group means for the 10 collegiate swimmers in this study. The average sleep efficiency, defined as the time spent asleep divided by the total time spent in bed, of the subjects was 85.8%. This is 4.2% lower than the average for the general population of 90%.<sup>76</sup> The mean for sleep latency, or the time it takes an individual to fall asleep once getting into bed, was just over 12 minutes; a value that falls under the population average of 16-17 minutes.<sup>77</sup> Taken together, the collegiate swimmers in this study appear to have experienced lower sleep quantity and quality during their competitive season compared to the general population.

These low group averages for total sleep and sleep quality are brought into context when one considers the level of energy athletes must exert on a daily basis. A recent study by Trappe et al in a group of college swimmers found that the daily total energy expenditure (TEE)

of these athletes was 5593 ( $\pm$  495) kcal.<sup>78</sup> Li et al<sup>79</sup> studied this same variable in a large group of non-athlete college students and found that TEE was 2373 kcal for females and 2706 kcal for males. Given these values it becomes clear that collegiate athletes are involved in activities that require nearly twice as much TEE compared to their aged matched peers. Although we did not monitor caloric intake during this study, nutritional and sleep deficits may put athletes at increased risk of suffering adverse health events. Future studies should further explore these relations.

In addition to sleep, HRV is a key recovery metric to consider in relation to human well-being particularly due to its association with the health of the human ANS.<sup>20</sup> A systematic review conducted by Nunan et al<sup>80</sup> identified normative HRV data for healthy adults using the rams method for short-term HRV measurement. The absolute value of the mean HRV from healthy adults in their study was 42 ( $\pm$  15 ms). This value is lower than the group average for the collegiate swimmers in the current study of 78.08 ( $\pm$  7.54) ms. This finding is in agreement with conclusions from previous studies suggesting that athletic populations tend to display higher levels of HRV compared with non-athlete controls.<sup>81-84</sup> The current consensus regarding the reason athletes have higher HRV is attributed to the unique adaptations of the athletic heart to increased loads experienced during intensive exercise training.<sup>81-84</sup>

### **Limitations**

A number of limitations were present within this study that may have impacted results. As the Penn State swimming team only had 11 WHOOP devices, the coaching staff aimed to utilize the units on athletes they felt would gain the greatest benefits. Thus, the sample studied was small and non-randomized. The small sample size limits the external validity of the findings while also restricting the statistical analysis for the study's major outcome variables. It is also possible that more data taken from a larger group of subjects would reveal more relations between predictors and outcome variables. For example, RHR and total load exhibited a trend toward significance ( $P= 0.07$ ) as predictors of injury (Table 3-7). Considering collegiate



swimming team are typically divided into separate training groups (sprinters vs distance swimmers) each of which performed different workouts based on their event and distance specializations, a larger sample size could also have allowed researchers to decipher how distance as well as training intensity (sprinting focused vs. endurance focused) effect the odds of injury, illness, and missed days.

Missing data was also a detrimental occurrence that must be considered. One subject dropped out of the university after the first semester and their data could not be used for analyses. Additionally, two subjects misplaced their WHOOP bands and/or chargers during the study and missed portions of data collection. If less than 20% of values were missing from a particular individual, that data was cleaned, however, 6 individuals fell above this threshold with their sleep and hear rate data. In these instances, analyses were conducted on what values were present, but this lack of compliance reduced the total number of raw data points. Overall average compliance using the WHOOP device for the group was 77.13 ( $\pm$  4.0)%.

An important delimitation of the study was the fact that athletes did not wear the WHOOP devices while they were physically in the pool swimming. Although this may not have affected their sleep-related data, not including data from these periods of increased bodily stress could have affected daily averages for variables such as RHR and HRV. The ability to include variables such as real time heart rate during exercise would also have been an interesting objective measurement of training load during training sessions in addition to sRPE, swimming distance, and activity duration.

### **Future Research**

The current project serves as a valuable pilot study upon which many future experiments could be based. Sleep loss was effectively identified as a significant risk factor for increases in total illness and total missed participation days. Future studies should include a larger sample size and ensure better subject compliance so as to increase the amount of data present for the major outcome variables studied here. A larger sample size would provide more valid and

generalizable conclusions. Additionally, researchers should partition subjects based on their training groups (sprinters vs. distance/endurance swimmers) as well as their main stroke (e.g. free-style, breast stroke, butterfly, backstroke) to see if these factors effect outcomes. With a larger sample size, future studies should also attempt to discern the effects of recovery metrics on specific categories of injuries and illnesses. For example, sleep loss may be more strongly related to chronic injuries as opposed to acute injuries, or upper respiratory infections as opposed to gastrointestinal disorders.

Future projects could also consider using a longer study duration to rule out any potential covariables related to time or seasonality. Researchers should identify younger athletes (freshman and sophomores) and follow them over 3 or 4 competitive seasons to discern whether similar results occur. Researchers could also look at the temporal nature of the academic workload placed on the student athlete subjects. For example, questionnaires could be distributed to identify times of high, medium, and low amounts of academic stress/responsibility. These may help explain specific trends in sleep behavior which in turn could lead to variance in health-related outcomes.

Finally, future research should attempt to implement an experimental intervention such as a month of sleep extension by one hour or a month during which early morning practices are eliminated. The methods present in this current project would allow one to observe whether these simple actions have any effect on athlete sleep and the occurrence of adverse health events.

## Chapter 5

### CONCLUSION

The hypotheses presented in this study were partially upheld. Logistical regression analyses showed that as athletes increased the amount of total sleep they attained per night during their competitive season they reduced their odds of suffering an illness by a factor of 30% (OR= 0.70, 95% CI= 0.55-0.89,  $P \leq 0.01$ ) and of missing a day of participation by 26% (OR= 0.74, 95% CI= 0.61- 0.90,  $P \leq 0.01$ ). Total yardage (OR= 0.99, 95% CI= 0.99-0.99,  $P \leq 0.001$ ) as well as RHR (OR= 1.08, 95% CI= 1.05-1.11,  $P \leq 0.001$ ) were also identified as statistically significant predictors of missed days. Those athletes that swam further on a daily basis reduced their odds of suffering a missed day by 1% and those with increased daily RHR increased their odds of missing a participation day by 8%. Sleep was not, however, found to be a significant predictor of athlete injuries. HRV was also not identified as a significant predictor of any adverse health outcome included in the study. The season average for total sleep in these 10 student athletes was 6.51 ( $\pm 0.23$ ) hours which is lower than the recommended duration of 7 hours for the general population as well as the 9-10 hours recommended for athlete populations. Furthermore, mean HRV for the group was calculated at 78.08 ( $\pm 7.54$ ) ms which was higher compared to the 42 ( $\pm 15$  ms) seen in the general population. Larger scale studies similar to this one focused on the relation between recovery variables and adverse health outcomes are warranted. As advancements in technology make these types of individualized, objective recovery data more ubiquitous, clinicians could greatly benefit from the potential ability to identify individuals at increased risk of suffering adverse conditions so as to act early and preventatively to improve patient health.

## Chapter 6

### LITERATURE REVIEW

#### What is Sleep?

Sleep is defined as "...the natural and regular state of inactivity in which consciousness ceases and the bodily functions slow down or cease."<sup>85</sup> From a simple behavioral standpoint sleep is described as a "reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment."<sup>86</sup> What these definitions fail to express, and what many individuals subsequently fail to realize, is that sleep is a brain process and can involve significant cortical activation, greater even than levels occurring during some states of wakefulness.<sup>87</sup> Hobson<sup>88</sup> referred to sleep as "... a dynamic behavior. Not simply the absence of waking, sleep is a special activity of the brain, controlled by elaborate and precise mechanisms. Not simply a state of rest, sleep has its own specific, positive functions." It is critical that athletes are aware of the fact that although sleep is a state of diminished consciousness and slower bodily functions, it plays a key role in the rest-activity cycle with very specific functions occurring during quality sleep.<sup>89</sup>

Within sleep, two separate states have been defined on the basis of a variety of physiologic parameters; these are rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. NREM sleep can be subdivided into stages 1, 2, 3 and 4 based on values gained from electroencephalogram (EEG) readings.<sup>76</sup> In adults, episodes of NREM and REM-sleep recur in a cyclic manner within a period of 90 to 120 minutes.<sup>87</sup> The onset of sleep is through NREM sleep and thus the typical sleep sequence is wake (also called stage 0); NREM stage 1, 2, 3, 4; and finally, REM. The four NREM stages roughly parallel a continuum of sleep depth such that stages 1 and 2 are labeled together as light sleep and stages 3 and 4 are labeled deep sleep or slow-wave sleep (SWS). In REM sleep the brain reactivates into a fast-activity state. Blood flow, heart rate, respiration, body temperature and blood pressure rise and the eyes, underneath closed eyelids, dart back and forth, which may be accompanied by

intermittent muscle twitching.<sup>90,91</sup> This is also the sleep stage where dreaming is thought to occur.

In general, SWS predominates in the first third of the night and is associated with the length of time spent awake while REM sleep predominates in the last third of the night and is linked to the circadian rhythm of body temperature. In general, NREM sleep is usually 75% of sleep with stage 1 taking up 2-5 %, stage 2 taking up 45 -55%, stage 3 constituting 3-8% and stage 4 constituting 10-15%. REM sleep then represents 20-25% of sleep occurring in four to six discrete episodes.<sup>76</sup>

NREM and REM sleep cycles each include unique physiological events that are critical to human bodily function. An article by Siegle<sup>92</sup> explains these unique events in detail. Specifically, NREM sleep is proposed to assist in energy conservation and nervous system recuperation. Comparatively, theories of REM sleep have suggested a role for this state in periodic brain activation, localized recuperative processes, and emotional regulation as well as the establishment of brain connectivity. REM, NREM stage 2, and SWS have also been implicated in memory consolidation and motor learning.

### **Measuring Sleep**

The current gold standard for measuring sleep both for clinical and research purposes is polysomnography (PSG).<sup>93-95</sup> This technique involves the application of numerous surface electrodes, each used to measure specific physiological parameters. These parameters include brain dynamics through the use of electroencephalography, eye movements with electrooculography (EOG), muscle activity with electromyography (EMG), heart physiology with electrocardiography (EKG), and respiratory function.<sup>95</sup> In order to utilize PSG it is usually necessary for patient's to sleep in a sleep laboratory under the continuous supervision of a sleep technician. Time series data are gathered, processed, and either analyzed visually or with the use of a mathematical algorithm.<sup>95</sup>

Despite the advantages of PSG in terms of accuracy, the invasive nature of the technique places restrictions on its use within large population-based research as well as longitudinal studies. Another method for studying sleep parameters is the use of wearable activity monitors, also known as actigraphy. Actigraphy units measure sleep via an accelerometer, operating under the assumption that there will be reduced human motion during sleep compared to waking hours. Many units involve piezoelectric accelerometers in which movements of the wrist cause a mass within the cantilevered system of the device to apply varying pressures to a piezoelectric crystal causing the crystal to emit differing electrical currents.<sup>27</sup> This analog data is band pass filtered (usually 2-3Hz or 0.5-11Hz) to remove gravity and noise artifacts.<sup>96</sup> Next, the filtered signal is digitized and separated into epochs. An epoch is the time over which the movement data is averaged with one minute representing the most common length.<sup>97</sup> Activity counts are then assigned to each epoch.

The digitization process involved in actigraphy can be executed through three modes including the zero-crossing mode (ZCM), time above threshold (TAT), and proportional integration mode (PIM). ZCM counts the amount of time per epoch that the signal crosses an assigned threshold which is set close to zero for sleep analysis.<sup>96,98</sup> TAT mode counts the amount of time the accelerometer signal is above the set threshold.<sup>96,98</sup> Finally, PIM rectifies the accelerometer data and calculates the area beneath this curve.<sup>96,98</sup> Of the three methods PIM has been shown to be most accurate when compared to polysomnography,<sup>99,100</sup> however, technological advancements now allow inputs from all three modes to be considered within algorithms that digitize signal and assign activity counts.<sup>98</sup>

Sleep or wake is typically classified for each epoch based on the activity counts from that time period and applying an algorithm that weights the activity score from surrounding minutes.<sup>96</sup> Specific cut points have been established which are applied to the counts from the epochs and these cut points help distinguish sleep from wake periods. The most commonly applied cut point for adults is <100 counts/minute.<sup>27</sup> Using these thresholds, combined with user

input about their sleep habits, actigraphs report sleep variables including sleep latency, total sleep time, number and frequency of awakenings, and sleep efficiency.

The advantage of actigraphy over traditional PSG is that actigraphy provides a convenient and inexpensive method to record human activity 24-hours a day for weeks at a time.<sup>96</sup> Actigraphy is also a less invasive technique compared to PSG, enabling subjects from a wide range of populations to measure their sleep within their natural sleep environment as opposed to a laboratory setting. Despite these positive attributes, there are additional aspects of actigraphy measurement that lack critical confirmation including the standardization of device settings, reporting of actigraphy analysis parameters in published research, a subjectively determined time of the analyzed rest period, and the establishment of a universal scoring algorithm.<sup>95</sup>

One additional drawback for the use of actigraphy is the accuracy and validity of its sleep data. As compared with PSG, actigraphy is known to overestimate sleep and underestimate wake time.<sup>96</sup> One reason for this shortcoming may be that PSG and actigraphy mark the beginning of sleep periods in different ways. Actigraphs mark the beginning of a sleep period using immobility of the participant whereas PSG analyzes specific changes in brain electrical activity to make this determination.<sup>95</sup> These brain activities often begin long after a period of body immobility, and thus actigraphs will overestimate sleep time.<sup>95</sup> Additionally, actigraphy only analyzes limb movements, and therefore the technique struggles to differentiate between instances of sleep and quiet waking.<sup>95,96</sup>

A number of published studies have attempted to validate the use of actigraphy by comparing its performance to that of PSG. For the purposes of this study, the current literature review will focus on those studies utilizing healthy volunteers. Sitnick et al<sup>101</sup>. compared minute-by-minute sleep-wake scorings based on actigraphy and PSG in young children and found 94% overall agreement, 97% sensitivity, and 24% specificity. De Souza et al<sup>93</sup> found similarly low levels of specificity (34% and 44%) in their comparison of two actigraphy scoring algorithms in

healthy subjects. Likewise, Paquet et al<sup>102</sup> compared two actigraphy scoring systems in a study that included 15 healthy participants and found that increasing wakefulness during sleep periods compromises the minute-by-minute actigraphy PSG correspondence. This resulted from the relatively low specificity of the sleep-wake scoring algorithm.

With the technological advancements that have taken place over the past two decades, the rate of growth in the number of scientific publications that include actigraphy exceeds the rate of growth in publications that include PSG. The American Sleep Disorders Association (ASDA) has identified actigraphy as a reliable and valid sleep assessment method in scientific domains of sleep research and sleep medicine.<sup>32</sup> Additionally, the American Academy of Sleep Medicine has recognized accelerometry as a valid measure to assess sleep-wake periods.<sup>27</sup> Taken together, the scientific literature shows that accelerometry has reasonable validity and reliability in assessing sleep-wake patterns in healthy individuals with average or good sleep quality.<sup>103</sup>

### **Sleep Terminology**

Sleep variables commonly measured using wristwatch actigraphy include time in bed, sleep latency, time asleep, time awake, sleep efficiency, actual sleep percentage, moving minutes, percentage of movement time, and the fragmentation index. A study by Leeder et al<sup>35</sup> provides clear definitions for these variables. Time in bed is the difference between the bedtime and wake time as defined by a participant, typically within a sleep journal or survey. Sleep latency can be understood as the time it takes to fall asleep defined as the difference between sleep onset time calculated by an actigraphy unit and the bedtime noted by a participant. Time asleep is the actual time spent asleep calculated from sleep start to sleep end minus any wake time sensed by an actigraph. Time awake is the time spent awake from sleep start to sleep end. Sleep efficiency is the duration of actual sleep expressed as a percentage of the total time from bedtime noted by the participant to sleep end measured by an actigraph. Actual sleep percentage, is the sleep duration expressed as a percentage of total time from sleep start to



sleep end each detected by the actigraph. Moving minutes are an important measure of the depth of sleep and are defined as the time spent moving during the total time in bed. These moving minutes can also be expressed as a percentage known as percentage moving time defined as the amount of time spent moving as a percentage of time in bed. Finally, the fragmentation index is a measure of restlessness during sleep using the percentage of epochs where measured activity is greater than 0.

Research exploring the effects of sleep on human physiology involves the restriction of normal sleep in experimental subjects. In order to interpret the results of these studies one must become familiar with the varying degrees of sleep restriction. Sleep restriction (SR) occurs when humans fall asleep later or wake earlier than normal; that is, their normal sleep–wake cycle is partially disturbed.<sup>104</sup> This treatment condition can either be considered acutely, such as a single bout of sleep restriction, or involve chronic sleep restriction over time. In contrast, sleep deprivation (SD) refers to extreme cases of sleep loss, whereby humans do not sleep at all for a set period.<sup>104</sup> In general, SR and chronic SR are more prevalent than total SD in human populations and thus the majority of studies included in this review will be those that involve various SR treatment conditions.

### **Sleep in Collegiate Athletes**

The American Academy of Sleep Medicine and Sleep Research Society recently recommended all healthy adults aged 18-65 acquire at least 7 hours of sleep per night to ensure optimal health.<sup>50</sup> Taking this figure into consideration, the 2013 Sleep in America Pole found that individuals 18 years and older slept for an average of 6 hours and 51 min on 'workdays' and 7 and 37 min on 'non-workdays'.<sup>105</sup> College students have a unique lifestyle compared with average Americans and often experience a high level of academic pressure which can affect sleep patterns. In fact, several reports have shown university/college students suffer from chronic sleep problems and disruptions.<sup>72-74</sup> One such report showed that as many as 71% of those 1462 surveyed were dissatisfied with their sleep.<sup>73</sup>

Although both athletes and coaches rate sleep as critical to optimal performance,<sup>106,107</sup> there are few published studies that have investigated normal sleep in collegiate athletes and none that have done so continuously using objective measurement systems over the course of a competitive season. This reality is surprising given the physical, mental, emotional, and social stress athletes are exposed to during training and competitions. In an article written by Bompa and Haff<sup>14</sup> it was proposed that athletes require 9 to 10 hours of sleep, with 80-90% of it during the night, in order to properly recover from these stresses. Recent experiments involving elite athletes suggest that these individuals sleep far less than these specific recommendations as well as those reported for the average adult population by the American Academy of Sleep Medicine and Sleep Research Society expressed previously.

In a study conducted by Sargent et al<sup>9</sup> sleep quality and quantity were measured using wrist activity monitors in 7 elite Australian swimmers during a 14 day high intensity training period. The study found that on average the swimmers slept only 5.4 hours on nights prior to training days. Furthermore, for night-time sleep periods that preceded training days and rest days, the swimmers' sleep efficiency was 71 and 77%, respectively. These values are substantially lower than the typical sleep efficiency of 90% for healthy young adults.<sup>76,108</sup> The swimmers also had relatively longer sleep latency on nights prior to training days (41 min) and rest days (32 min) compared to the average population (16-17 min).<sup>77</sup> In another study by Vantor et al<sup>109</sup> sleep metrics were taken via questionnaire in a group of 890 elite South African athletes. Results showed that the majority (75%) of athletes slept 6 to 8 hours per night, while 11% of respondents reported that they slept less than 6 hours on weekends. Additionally, 41% of athletes stated they had problems falling asleep with interference by noise and light listed as the major causes of these disruptions. A more recent study by Leeder et al<sup>35</sup> attempted to provide normative sleep data for elite athletes using actigraphy and compare these individuals to non-athlete controls. A group of 46 Olympic athletes were studied over 4 days after which the researchers found that the athletes slept for a lower mean total duration (6 hours

and 55 minutes vs. 7 hours and 11 minutes), had poorer sleep efficiency ( $80.6 \pm 6.4\%$  vs.  $88.7 \pm 3.6\%$ ), and a higher fragmentation index ( $36.0 \pm 12.4$  vs.  $29.8 \pm 9.0$ ) compared to an age and sex matched control group.

In addition to projects focusing on normative sleep data in athletes, multiple studies have been done to assess athlete sleep in specific scenarios such as immediately prior to competition. In a study conducted by Erlacher et al<sup>110</sup> a group of 632 German athletes from various sports were asked via questionnaire about their sleep habits during the nights before an important competition. The findings indicated that 62.3% experienced poor sleep in the night(s) before a sports event at least once during the previous 12 months. Athletes involved in individual sports reported more sleep difficulties than team sport athletes. A similar study by Juliff et al<sup>111</sup> was conducted in a group of 283 elite Australian athletes which found that 64% of subjects reported poor sleep prior to an important competition. In both studies, the main sleep problem was an inability to fall asleep and internal factors such as nervousness and thoughts about the competition were rated highest for causing these sleep problems.

Travel schedules have also been shown to have a significant effect on sleep and circadian rhythms in athletes. A greater problem becomes evident when athletes cross multiple time zones. In a study conducted by Richmond et al<sup>112</sup> sleep measurements were made on 19 elite Australian rules footballers using wrist actigraphy to assess the effects of interstate travel across multiple time zones. The study showed no significant differences in any measure of sleep between home and away competitions, however, the athletes self-reported that their sleep at home was significantly better than when away. A similar study conducted on 15 elite male soccer athletes found that 18 hours of predominantly westward travel across 4 time zones caused truncated sleep duration and reduced sleep efficiency.<sup>113</sup>

Despite this informative cohort of studies examining sleep metrics in elite and professional athletes, only two studies could be found that examined normal sleep patterns in college athletes. One such study was conducted by Mah et al<sup>114</sup> as a part of a larger project

examining the effects of increased sleep or sleep extension on basketball performance. Wrist actigraphy readings on 13 NCAA Division 1 basketball players showed the average total nightly sleep time over a 2-4-week baseline period was 6.68 hours. An additional study conducted by Taylor et al<sup>6</sup> examined sleep in college aged female swimmers competitive at a national level using polysomnography readings taken at three different time points across a competitive season including during the onset of training, heavy training and during the pre-competition taper. Total sleep duration for these time points were found to be  $7.5 \pm 0.495$  hours,  $7.968 \pm 1.01$  hours, and  $7.64 \pm 0.257$  hours respectively.

Taken together the results of the current literature seem to suggest that athletes fail to acquire the daily recommended amount of sleep proposed for healthy adults as well as those suggested for optimal recovery in athletic populations. Furthermore, this research highlights the complex lifestyle of athletes and the effects that aspects such as travel, competition, and training volume have on their sleep. Finally, the need for further research focused on the collegiate athlete population is evident as only one study could be found that included NCAA athletes.

### **Sleep's Effects on Cognitive Function**

Sleep studies using cognitive performance as an outcome have focused primarily on tasks that assess the stability of vigilant attention (e.g., psychomotor vigilance test), cognitive processing speed, working memory, decision making and inhibitory control, academic performance, visual-motor behavior, mood states, and reaction time. Of these variables reaction time, visual-motor behavior, vigilant attention, and decision making are particularly pertinent when considering the effect of sleep loss on athletes' risk of suffering an injury/illness.

An example of the sensitivity of cognitive function to acute sleep disruption can be noted with regards to the reaction time variable. Reilly and Deykin<sup>115</sup> conducted a study involving a group of 8 trained men who underwent 3 nights of sleep loss and a single night of recovery sleep. Subjects were measured on subjective mood states, hand steadiness, choice reaction

time, anaerobic power, grip strength, lung function, and endurance capacity. A significant treatment effect was observed for anxiety, hand steadiness, and choice reaction time. Increases in these variables were evident after only one night of reduced sleep. It seems the effect of sleep restriction applies equally to females and males as demonstrated by a study conducted by Reilly and Hales<sup>54</sup>. In this experiment sleep was restricted to 2 hours per night for 3 nights in well-trained females and measurements were made on oral temperature, lung function, grip strength, anaerobic power output, limb steadiness and speed, and subjective sensations at rest and during exercise. Researchers found that gross motor functions were less affected by sleep loss compared to tasks requiring fast reactions. Similar findings were seen in a study conducted by Bonnet<sup>116</sup> on 12 healthy young adults. Subjects had their sleep restricted to 4 hours a night during two separate 4-night periods by systematic disturbances across 10-minute intervals. A unique aspect of this project was that the researchers disturbed SWS only in one 4-night period and compared results on a battery of tests to a 4-night period of general sleep disturbance. Simple reaction time was seen to increase in both groups compared to baseline measures, but no significant differences were seen between SWS restriction and the general restriction groups. One final experiment involving the effects of sleep restriction on reaction time was conducted by Jarraya et al<sup>117</sup> and involved reaction time in 12 handball goalkeepers. The athlete subjects had their sleep restricted to 4-5 hours per night under two randomized sessions, one in which sleep was restricted in the beginning of the sleep period (athletes go to sleep late) and one in which sleep was restricted at the end of the sleep period (athletes wake up early). Reaction time was significantly increased following both treatments but was more sensitive to sleep restriction at the end of the sleep period. Taken together, it appears that reaction time following even minor sleep disruptions is slower compared to baseline levels, an effect that is further enhanced when sleep is shortened due to early rising. These findings are pertinent considering athletes rely heavily on quick reaction times to protect themselves from injury during

sport and often have restrictions placed on their sleep schedule by early morning practice times.

Critical decision making is another important cognitive factor for athletes which could affect their risk of suffering an injury. The susceptibility of this trait to alterations due to sleep is rather equivocal. A review by Harrison et al<sup>118</sup> supports the idea that decision making is impaired following sleep loss. This conclusion contradicts the findings of a study conducted by Khazaie et al<sup>119</sup> which reported no change in abstract reasoning, time reproduction skills, or decision making ability in 26 medical students who underwent a sleep restriction of less than 6 hours per night for five nights. A similar variable to consider is an individual's inhibitory control, defined by Killgore et al<sup>120</sup> as the ability to judge when a behavior or response is appropriate to a specific circumstance, as well as the capability to withhold a response once it is deemed inappropriate or disadvantageous. Inhibitory control has been studied in relation to sleep restriction by Rossa et al<sup>121</sup> in a group of young adults using the balloon analog risk task (PBART). Inhibitory control efficiency was significantly reduced following a single night of sleep restriction resulting in an increase in risk taking behavior. In terms of real-world application, increased risk taking on the PBART has been associated with adolescent self-reported engagement in aggression, alcohol and drug use, and unprotected sexual intercourse.<sup>122</sup> A final study by Yoo et al<sup>123</sup> looked to explore the biological mechanism behind the temporary changes in behavioral inhibition following sleep disruption using functional magnetic resonance imaging. The prefrontal cortex is thought to be most vulnerable to the effects of sleep loss. This brain region has subcortical connections to the amygdala, an area involved in the processing of emotions and goal-oriented actions. The researchers found that sleep deprived individuals show signs of dysfunction in their prefrontal cortical areas resulting in a significantly increased amygdala response to negative stimuli suggesting a loss in inhibitory control.

Visual motor behavior may also be affected by poor sleep. Athletes rely on this sense to avoid injury during their activities and thus it is a critical consideration for this research. In a

review written by Alhola et al<sup>124</sup> it was concluded that sleep deprivation impairs visuomotor performance as measured with tasks of digital symbol substitution, letter cancellation, trail making, and maze tracing. This conclusion is supported by a study by Zils et al<sup>125</sup> in which saccadic eye movements were compared after normal sleep and sleep deprivation in 15 healthy male volunteers. Sleep deprivation had a general impairing effect on the peak velocity and accuracy of saccades demonstrating a deficiency in visual motor function.

Vigilant attention is a final cognitive aspect that is crucial to examine in relation to athletes' risk of adverse health events and may be affected by poor sleep. To avoid injury, athletes must sustain a high level of awareness and attention to their surroundings as they execute the physical tasks necessary for their sport. In a review by Venter<sup>126</sup> it was suggested that the effects of sleep loss among athletes are evident on higher cognitive functions including attention. A review by Fullagar et al<sup>127</sup> also reports that when sleep is reduced to less than 7 hours in healthy adults, cognitive performance is poorer in tests for multiple variables including alertness. The aforementioned study by Jarraya et al<sup>117</sup> involving 12 handball goalkeepers further supports these claims. In addition to reaction time, the study also measured both selective and constant attention. Sleep deprivation that occurred in the beginning and end of a sleep period caused declines in both measures of attention. Finally, in a study by Choudhary et al<sup>128</sup> the effect of sleep restriction on vigilance was explored in a group of 50 night watchmen by measuring visual and auditory reaction time as well as P300 event related potentials; a wavelike signal from the brain shown to reflect information processing and conscious novelty detection. Seven days of sleep restriction resulted in a dose response relationship in which visual and auditory reaction time increased while P300 decreased as sleep restriction was prolonged. Overall, poor sleep appears to have a detrimental effect on multiple measures of human cognitive function.

## **Sleep's Effects on Pain Perception**

Along with illness, bodily pain represents the major factor restricting athlete participation and performance within their sport. Athletes are highly susceptible to pain which can result from many sources including injury as well as muscle soreness secondary to exertion. It is also well accepted that individuals experiencing pain frequently report disturbed sleep.<sup>129,130</sup> Pain has been found to induce an SNS response which can cause night time arousals.<sup>129</sup> The results of more recent studies suggest that sleep deprivation may cause or modulate both acute and chronic pain.<sup>131</sup> Thus, the effect of sleep on pain is a significant consideration for research related to injury rates and time to return to play.

Many early studies involving sleep and pain perception were correlational analyses conducted on patients suffering from fibromyalgia. From these studies, it was established that NREM sleep interruption, in particular, is linked to decreases in pressure pain threshold signaling an increase in myalgia pain. Two studies conducted by Moldofsky et al affirm this assertion. The first of these experiments subjected 6 healthy men to 3 nights of stage 4 sleep restriction and found that the treatment increased pressure pain sensitivity as well as the likelihood that subjects would report musculoskeletal pain.<sup>58</sup> The second study by Moldofsky and Scarisbrick<sup>59</sup> replicated the experimental settings of the first with the addition of a treatment group for REM sleep restriction. Pain thresholds were compared between 7 subjects undergoing 3 nights of REM sleep deprivation and 6 subjects undergoing NREM sleep deprivation. Results showed that NREM sleep again significantly decreased mechanical pain thresholds and increased reporting of musculoskeletal pain, but REM sleep deprivation did not elicit this same effect.

In addition to defining the specific stage of sleep influencing pain perception, recent prospective studies have been conducted to assess whether this influence is reciprocal or possessing of specific directionality. An early review by Lautenbacher et al<sup>132</sup> proposed a reciprocal relationship between sleep and pain, suggesting that sleep deprivation may



enhance or cause pain and pain may induce arousals that in turn interrupt sleep. A more recent review by Finan et al<sup>131</sup>, cites advancements in experimental and data analyses and suggests that the temporal effect of sleep on pain may be stronger than that of pain on sleep. A study by Lewandowsky et al<sup>133</sup> supports this assertion. Researchers took daily actigraphy assessments of sleep in adolescents with heterogeneous chronic pain complaints including headache, back pain, stomach pain, and musculoskeletal pain. Results revealed significant associations of total sleep time and wake after sleep onset on next-day pain reports while pain did not prospectively predict any sleep measurements.

While prospective studies are utilized to assess the temporal relationship between sleep and pain, sleep deprivation studies attempt to provide insights into the mechanisms by which sleep and pain are related. Early studies of this form yielded fairly equivocal results, offering only tentative support for the notion that sleep deprivation increases pain sensitivity. For example, Drewes et al<sup>134</sup> analyzed the effect of one night of total sleep deprivation on 10 healthy individuals and found no change in pain detection and tolerance. In contrast, a study by Kunderman et al<sup>135</sup> involving 20 healthy subjects assigned to a sleep deprivation or control group found that heat pain and cold pain thresholds were significantly decreased in the sleep deprived subjects. The findings from these initial studies are limited by small sample sizes as well as the deprivation techniques utilized which included total sleep and selective sleep stage deprivation.

Most people with sleep impairments achieve some measure of sleep throughout a typical night and therefore partial sleep restriction paradigms are thought to be closer than total sleep deprivation study designs in approximating the effects of common sleep problems on pain. An example of this type of study was carried out by Tiede et al<sup>60</sup> in a group of 10 healthy individuals. Subjects were instructed to restrict themselves to 4 hours of sleep for one night after which they underwent quantitative sensory testing involving laser pulses of radiant heat. Noxious stimuli were rated as significantly more painful following sleep restriction compared to

uninterrupted sleep, while cortical EEG activity in brain regions linked to the descending pain modulatory systems were attenuated. This suggests that not only are sleep deprived individuals more sensitive to pain, but they may also have a reduced capacity to attenuate painful episodes.

Partial sleep deprivation models that disrupt sleep continuity as opposed to maintaining wakefulness for a specified period are suggested to even further mimic sleep disturbance in individuals suffering from chronic pain. A study by Smith et al<sup>129</sup> adopted a forced awakenings model of sleep deprivation in which individuals were awakened pseudorandomly each hour over the course of an 8 hour sleep period. Results from this study showed that disruptions in sleep continuity resulted in a significantly greater amount of next day spontaneous pain reports in an otherwise healthy group of 10 women compared to a sleep deprived group allowed to sleep continuously for the same amount of time as the discontinuous sleep group and a control group allowed to sleep continuously for 8 hours.

Conclusive evidence of a significant relationship has been established between sleep and pain perception. Prospective studies have illustrated that this relationship is directional in nature with poor sleep representing a significant causative factor of increased pain perception. Early experimental studies identified NREM sleep as the major type of sleep associated with pain. More recent experiments have used multiple models to show that poor sleep decreases pain thresholds, increases individual sensitivity to pain, and may inhibit a person's ability to attenuate pain. All of these findings are critical to consider in an athletic population particularly for research involving injury rates and recovery time.

### **Sleep's Effects on Metabolism and Endocrine Function**

Both laboratory and epidemiological studies support the notion that sleep deprivation and sleep restriction can each cause negative effects on metabolism and endocrine function. Observed effects that are pertinent to collegiate athletes include sleep's effect on

glucose metabolism, appetite regulation, growth hormone secretion, the hypothalamic-pituitary-adrenal axis, and the hypothalamic-pituitary-gonadal axis.

Glucose metabolism is a key consideration for athletes considering they require a unique diet of carbohydrate rich foods to fuel their intensive sporting activities. Lack of proper fueling and/or metabolism could lead to increased fatigue and fatigue related injury or illness. According to Knutson et al<sup>136</sup> the exact mechanism by which decreased sleep influences glucose metabolism is thought to be multifactorial and includes decreased brain glucose utilization, alterations in sympathovagal balance, increased evening cortisol, extended night-time growth hormone secretion, and proinflammatory processes. A study by Spiegel et al<sup>137</sup> assessed glucose metabolism in 11 young men under 3 conditions including restricted sleep (4 hours in bed per night), sleep extension (12 hours in bed per night), and baseline sleep (8 hours in bed per night). During the sleep-restriction period, subjects had significantly impaired glucose tolerance, significant reductions in their acute insulin response to glucose, and significant reductions in glucose effectiveness compared with those observed when they were fully rested. Another study conducted by Tasali et al<sup>138</sup> showed that specific suppression of SWS without any reduction in total sleep time resulted in decreased insulin sensitivity, reduced glucose tolerance and increased risk of type 2 diabetes. These findings suggest that a reduction in SWS, independent of the overall duration of sleep, may be particularly important for normal glucose metabolism.

In addition to glucose metabolism, appetite regulation is another important concept for athletes. Poor eating habits can inhibit proper athletic conditioning and poor conditioning is a common risk factor for athletic injury. Leptin is an appetite regulating hormone that exerts a sustained inhibitory effect on food intake while increasing energy expenditure.<sup>139</sup> Ghrelin is an appetite stimulating hormone released by cells in the stomach.<sup>140</sup> Several studies have shown that recurrent partial sleep deprivation and chronic short sleep are associated with a significant decrease in levels of leptin and increase levels of ghrelin. Most notably, a randomized cross-

over study comparing the metabolic effects of 2 nights of short sleep (4 hours per night) with two nights of long sleep (10 hours per night) in a group of 12 healthy men was conducted by Spiegel et al.<sup>141</sup> Results showed a significant decrease in mean blood leptin levels which occurred concomitantly with a significant increase in mean ghrelin levels after sleep restriction compared to sleep extension; these results were seen despite identical caloric intake. In addition, following sleep restriction subjects reported increased hunger and appetite for carbohydrate rich foods.

The hypothalamic-pituitary-adrenal axis, and the hypothalamic-pituitary- gonadal axis each include hormones that are critical to muscle recovery. The functioning of these axes has been shown to be negatively modulated by lack of proper sleep. These effects therefore are important to consider both in terms of athlete injury rates as well as the time it takes athletes to return to play. The process through which damaged cells recover and become replaced by new cells requires proper proliferation, fusion, and differentiation of satellite cells as well as a simultaneous signal of muscle hypertrophy.<sup>142,143</sup> This hypertrophy depends on the rate of protein synthesis being greater than that of degradation. Muscle hypertrophy is thought to be induced by signaling from an insulin-like growth factor (IGF-1) mediated signaling pathway that includes P13K, Akt, and mTOR.<sup>144</sup> The testosterone androgen also plays a key role by increasing transcription,<sup>143</sup> inhibiting protein degradation through the ubiquitin proteasome pathway,<sup>145</sup> and decreasing the functionality of myostatin.<sup>146</sup> Levels of cortisol and other glucocorticoids also play a role as elevations in these hormones stimulate protein degradation also through the ubiquitin proteasome pathway.<sup>145,147,148</sup> It has been hypothesized that poor sleep decreases the production of IGF-1 and testosterone. This causes a decrease in the hypertrophy signal from the IGF-1/p13K/Akt/ and mTOR pathways, and a decrease in the inhibition of myostatin expression causing a subsequent increase in protein degradation. Poor sleep also causes increased cortisol secretion which activates the ubiquitin proteasome system, further increasing protein degradation and muscle atrophy.<sup>143</sup>

Sleep, particularly the first stage of SWS at the beginning of the night, has proven to be the peak period of human growth hormone (GH) secretion with up to 95% of the hormone being produced during NREM-sleep.<sup>15</sup> This hormone has an important effect on muscle growth and repair, bone building and fat burning all of which are critical to consider in relation to injury risk and time to return to play in athletes.<sup>16</sup> Studies have shown that if energy expenditure increases during the day blood levels of growth hormone will rise during sleep. For example, in a study conducted by Taylor et al<sup>6</sup> the amount of SWS in 7 elite swimmers was assessed over the course of a 6 month competitive season including readings taken at the onset of training, during peak training, and during pre-competition taper. Results showed that time spent in SWS was significantly higher during times of higher training volumes, confirming its importance to the recovery process.

Studies analyzing the effect of sleep restriction and deprivation on the release of GH show contradictory results. In a review by Davenne et al<sup>89</sup> it was suggested that when an athlete loses slow wave sleep, levels of GH fall significantly. This situation would logically leave these athletes vulnerable to poor recovery and injury. This assertion is challenged, however, by the results of a controlled study by Brandenberger et al<sup>149</sup> who assessed plasma GH levels in 10 subjects aged 20-26 following 8 hours of sleep and 24 hours of continuous sleep deprivation. This study showed that although the amount of GH secreted during the night was significantly lower during sleep deprivation, the amount of GH secreted during the day in awake participants was significantly increased following deprivation such that the total amount of GH secreted during the 24 h period was similar in both conditions. Studies have also been done on the more common case of chronic sleep restriction and its effects on GH secretion. A study by Spiegel et al<sup>150</sup> assessed GH levels of 11 young men during 6 days of sleep restriction (4 hours per night) and 7 days of extended bedtimes. Both conditions elicited similar total levels of GH secretion, however, the restricted condition led to a partitioning of GH pulses with one large pre-sleep secretion and one large post-sleep secretion.

Evidence supporting the negative effect of poor sleep on human metabolism and endocrine function is both multifaceted and convincing. Poor sleep inhibits glucose metabolism, increases appetite, and alters the hormonal regulation of protein synthesis and degradation thus decreasing the body's ability to appropriately recover. These effects suggest that poor sleep could be a significant risk factor for athlete's both to suffer an injury and/or illness and a to have a prolonged time to return to play following these incidents.

### **Sleep and Injury Rates**

According to the NCAA Injury Surveillance Reporting System, an injury can be defined as any trauma to an individual's body that (1) occurred as a result of participation in an organized intercollegiate practice or competition and (2) required medical attention by a team certified athletic trainer or physician and (3) resulted in restriction of the student-athlete's participation or performance for 1 or more calendar days beyond the day of injury.<sup>151</sup> Few studies have assessed the effect of poor sleep on injury/illness rates in athletes, however, important insights can be gained from reviewing literature on sleep and work-place injuries.

The few observational studies that have included athletes show equivocal results in terms of a direct relationship between sleep and injury rates. One study by Milewski et al<sup>17</sup> was completed at a combined high school/middle school and included 112 student athletes. Subjects self-reported their average number of hours of sleep per night during their respective sports season via a questionnaire. Injuries were defined as any musculoskeletal condition that necessitated a visit to the athletic trainer's room for evaluation and these incidents were quantified based on athletic trainers' records. Data analysis showed that the strongest predictor of injury was attaining less than 8 hours of sleep per night. A total of 65% of athletes who reported sleeping less than 8 hours per night were injured, compared with 31% of athletes who reported sleeping at least 8 hours per night. In addition, athletes who slept less than 8 hours per night on average had a 1.7 times greater risk of being injured than those who slept at least 8 hours per night. These findings were confirmed in a study by Luke et al<sup>18</sup> in a group of 360

athletes of a similar age (6 to 18 years). Subjects visiting a sports-medicine clinic completed a survey to gauge the effects of over-scheduling on injury rates. A physician recorded whether a subject's injury was related to sport and classified the condition based on mechanism. Fatigue-related injuries were significantly related to sleeping less than 6 hours the night before the incident. A more recent study by von Rosen et al<sup>19</sup> measured subjective sleep, nutrition, and injury rates in 340 elite adolescent athletes. Surveys were sent to participants during fall and spring semesters. Of those individuals surveyed, 162 remained uninjured through the fall semester and were followed to the second measurement period. Athletes who reported sleeping at least 8 hours during weekdays reduced their odds of injury by 61% (OR= 0.39, 95% CI= 0.16–0.99). Both of these studies directly contradict findings by Dennis et al<sup>152</sup> who conducted a prospective cohort study on the effect of various sleep patterns on injury occurrence in 22 elite Australian footballers. Subjects had their sleep measured via actigraphy 5 nights a week for the duration of a competitive season. Injury episodes were classified by the senior club physiotherapist and recorded in a database. Sleep in the week of an injury occurrence was compared to the average of the previous 2 weeks and the results showed no significant effect of sleep on injury occurrence. Although this study did have the advantage of an objective measure of sleep, this variable was only recorded for 5 out of 7 nights per week. As a result, there was not enough sleep data to analyze injuries during training sessions, only those incidents that occurred during competition.

In addition to athletic injuries, occupational injuries are also very common with more than 960,000 workers becoming injured daily as a result of accidents at work.<sup>153</sup> Multiple systematic reviews and meta-analyses support the claim that sleep has a significant effect on occupational injury rates. One such meta-analysis conducted by Kucharczyk et al<sup>154</sup> examined 30 studies reporting data on daytime occupational impact in relation to insomnia, insomnia symptoms, or poor sleep quality. Of these studies 11 specifically assessed sleep-related accidents and 8 of the 11 reported excess risk associated with insomnia-like symptoms. The

study concluded that insomnia-type sleep disturbances, are consistently associated with reduced safety and productivity as well as increased levels of illness-related absence from the workplace.<sup>154</sup> Despite these findings, many of the injuries included in this analysis involved motor vehicle accidents and thus are not as applicable to an athletic population. A meta-analysis conducted by Uehli et al<sup>155</sup> examined 27 studies which assessed the effect of general sleep problems on work injuries and excluded studies that recorded injuries secondary to motor vehicle accidents. The review found that workers with sleep problems had a 1.62 times higher risk of being injured at work compared to workers without sleep problems and that approximately 13% of all the work injuries recorded could be attributed to sleep problems.

Aside from these meta-analyses, multiple epidemiological studies support the negative effect of poor sleep on occupational injury rates. A cross sectional study based on data from the US National Health Interview Survey done by Lombardi et al<sup>156</sup> found that shorter daily sleep durations were significantly and independently associated with increased work-related injury risk. In a case control study by Leger et al,<sup>157</sup> individuals meeting DSM-IV criteria for insomnia reported significantly higher levels of work-related accidents within the past year when compared with normal sleepers (8% vs. 1% respectively). In addition to sleep quantity, sleep quality has also been shown to have an effect on occupational injury. In a cross-sectional case-control study of 880 males in the construction industry Chau et al,<sup>158</sup> using logistic regression models, found that workers reporting an occupational injury with subsequent sick leave over the past two years were more likely to report shorter sleep durations (less than 6 hours per day), “not sleeping well”, and the consumption of sleeping tablets compared to controls who had not had an injury. Similar results were found by the same researchers in a case-control study of 2610 male French railway workers which reported that “sleep disorder” symptoms were specifically related to injuries from physical exertion and pain due to movement.<sup>159</sup>

Taken together, results from past studies involving adolescent athletes, elite athletes, and sleep restricted adults in the work force suggest an independent negative relationship



between poor sleep and injury/illness rates. Further research is warranted to assess whether this same effect is present in NCAA division 1 collegiate athletes.

### **Sleep's Effects on the Immune System**

Although the concept of immune function in relation to sleep has not been thoroughly studied in athletes, this topic has been explored in normal, healthy adult subjects. Outcomes within this area of research can take several forms including analyses of immune cell function such as natural killer (NK) T-cells and leukocytes, vaccine immune response, and risk of infection following pathogen exposure. In a review on the role of sleep in the immune system by Bryant et al<sup>51</sup>, the authors concluded: “an increasing body of evidence indicates that even minor sleep loss, accumulated over time, ... has a considerable impact on the immune response.”

Sleep duration has been shown to modulate the function and activity of a number of different immune cells and inflammatory markers. Natural killer T-cells are important effector lymphocytes of the innate immune system that control several types of tumors and microbial infections by limiting their spread and subsequent tissue damage.<sup>160</sup> Leukocytes are cells of the immune system involved in protecting the body against both infectious disease and foreign invaders.<sup>161</sup> These leukocytes accomplish this in part by producing pro-inflammatory cytokines such as interleukins. Interleukin-6 (IL-6) is a key interleukin cytokine that stimulates other cytokines such as tumor necrosis factor- alpha (TNF-a) which plays a role in regulating immune cells during the acute reaction phase secondary to infection.<sup>161</sup>

The production of NK cells in response to sleep restriction has been explored through a pair of studies conducted by Irwin et al. The first study included 23 healthy male subjects who underwent a single bout of late night sleep deprivation between the hours of 03:00 and 07:00. This treatment resulted in a nearly 30% decrease in NK activity compared to baseline norms.<sup>53</sup> In a subsequent study the group analyzed the consequences of early night sleep deprivation from 22:00 to 03:00 in a group of 42 healthy male subjects. This loss of sleep resulted in a

similar suppression of NK cell activity with decreases in both the numbers of circulating NK cells and in their activity.<sup>52</sup>

The production of IL-6 and TNF- $\alpha$  following sleep deprivation was also studied by Irwin et al.<sup>54</sup> Researchers observed proinflammatory cytokine production in 30 healthy adults across 3 baseline periods and after partial sleep deprivation in which subjects slept only 4 hours from 03:00 to 07:00. In the morning after a night of sleep loss, monocyte production of IL-6 and TNF- $\alpha$  were significantly greater compared with morning levels following uninterrupted sleep. Interestingly, sleep loss was also found to induce a more than 3-fold increase in transcription of IL-6 messenger RNA and a 2-fold increase in TNF- $\alpha$  messenger RNA. This finding affirms that sleep restriction induces a functional alteration of the proinflammatory cytokine response of the innate immune system.

Sleep restricted individuals have also been shown to elicit a reduced vaccine immune response. This was illustrated in a study by Prather et al.<sup>55</sup> in which 125 midlife adults were administered a standard 3-dose regimen of recombinant hepatitis B vaccine. Subjects had their sleep analyzed over 6 days using actigraphy and electronic sleep diaries. Immune response was quantified by antibody titers via enzyme-linked immunoassays assessed 6 months after the final immunization. Results showed that shorter sleep duration was associated with lower secondary antibody response and significantly decreased likelihood of clinical protection. A similar study was conducted by Spiegel et al.<sup>56</sup> in which the effects of sleep restriction as well as sleep extension were assessed in relation to immune response to an influenza vaccine in 25 young men. Eleven subjects were assigned to the treatment group and had their sleep restricted to 4 hours for 6 nights and then extended to 12 hours for 7 nights. On the fourth day of sleep restriction the vaccine was given. Mean antibody titers showed that 10 days after immunization, subjects immunized in a sleep restricted state had less than half as many active antibodies in their systems compared to the no-treatment control group.

Risk of infection is also commonly studied using a pathogen exposure challenge model. This strategy was employed by Cohen et al<sup>57</sup> in a study that included 153 healthy male and female volunteers. Subjects reported their sleep duration and efficiency for a period of 14 days after which they were administered nasal drops containing a rhinovirus then quarantined and observed for 5 days for development of a clinical cold. Results of the study showed that those individuals who reported sleeping less than 7 hours daily were 2.94 times more likely to develop a cold than those that reported daily sleep of 8 hours or more. Findings from a prospective study of sleep duration and pneumonia risk in women conducted by Patel et al<sup>162</sup> support this outcome. In this study 56,953 female nurses self-reported their average sleep duration and whether this quantity was adequate for them. Questionnaires ascertaining a new pneumonia diagnosis were then mailed every 2 years. The relative risk of acquiring pneumonia when sleeping 5 hours or less was 1.39. Interestingly, the study also reported an increased risk of 1.38 for those sleeping 9 hours or more.

Taken together, the evidence across several studies support the claim that sleep is a significant modulator of immune function in humans. Decreases in sleep have been shown to cause a negative effect on the production of key disease fighting cells, alter the proliferation and molecular control of pro-inflammatory cytokines, decrease the effectiveness of preventative vaccinations, and increase the likelihood of acquiring specific illnesses such as pneumonia or upper respiratory infection. A review written by Venter<sup>126</sup> summarizes the meaning of these conclusions for athletes, " It is suggested that the ever-increasing pressures to train for longer hours and perform well, combined with other non-training stressors, could affect the quality of the athlete's sleep and compromise immunity."

### **What is Heart Rate Variability?**

Heart Rate Variability is the variation over time of the period between consecutive heartbeats.<sup>20</sup> The most common system used to measure HRV is the electrocardiogram (ECG) which assesses the heart's electrical activity.<sup>28</sup> The electrical activity of the heart falls into a

rhythmic pattern allowing specific phases of the resulting signal to become recognizable. These electrical events have been labeled by researchers such that every heart beat in the ECG signal, has a periodic sequence of observable P, QRS, and T-waves.<sup>163</sup> Of these electrical events, the QRS complex has the highest amplitude and is used to calculate the interval between consecutive heart beats.<sup>163</sup> This is why HRV is commonly referred to as the variation between RR peaks.

The heart is considered a stochastically oscillatory organ<sup>69</sup> and its activity is controlled via inputs from a variety of different bodily systems. One major contributor to cardiac control is the ANS. The ANS is composed of two branches including the SNS and PNS. Sympathetic nervous system stimulation occurs in response to conditions such as stress, exercise and heart disease.<sup>20</sup> This stimulation results in an increase in heart rate by increasing the firing rate of pacemaker cells in the sinoatrial (SA) node.<sup>20</sup> Activity of the PNS primarily results from the function of internal organs, trauma, allergic reactions and the inhalation of irritants.<sup>20</sup> Parasympathetic nervous system input will decrease the firing rate of the SA node and slow heart rate.<sup>20</sup> Contributions from the SNS and PNS modulate heart rate and determine HRV as they control the interval between RR peaks. For this reason, HRV is understood to provide a valid depiction of the overall health of an individual's ANS.<sup>163</sup>

Recent technological advancements have streamlined HRV data acquisition by allowing for the use of wearable devices to collect and analyze this data as opposed to ECG recordings. This is advantageous for researchers as it enables HRV to be collected in subjects continuously through their daily activities, whereas ECG recordings are more restrictive. While an ECG measures HRV via electrical impulses, wrist-based activity monitors typically utilize photoplethysmography (PPG). Photoplethysmography involves the use of pulse oximeters which have light emitting diodes (LEDs) that send light waves into the skin. Each cardiac cycle pumps blood to the body's periphery causing distension of vasculature. The change in pulse pressure is detected by illuminating the skin and measuring intensity changes in the light

reflected using a photodiode.<sup>28,29</sup> Software algorithms then take this signal and often combine it with information from accelerometers within the wearable devices to produce meaningful heart rate information.<sup>34</sup> The final HRV metrics produced are equal to the first derivative of the PPG signal.<sup>20</sup>

### **Heart Rate Variability Indices**

The simplest estimation of HRV is portrayed using time domain measures. Time domain analyses address the question of "how much variability" is present.<sup>32</sup> This category can be further broken down into two classes, those based on inter-beat intervals and those based on comparisons between the lengths of adjacent cycles. Those based on inter-beat intervals include the standard deviation of all normal RR intervals (SDNN), the standard deviation of the means of all 5-minute intervals of a 24-hour period (SDANN), and the mean of the standard deviations of all normal RR intervals for all 5 minutes segments of a 24-hour period (SDNNIDX).<sup>32</sup> These methods based on inter-beat intervals are greatly influenced by external factors such as respiration and circadian rhythms and thus can be subject to inaccuracies.

The second class of time domain variables based on comparisons of adjacent cycles include the proportion of adjacent cycles less than 50 ms apart measured as a percent (pNN50), and the root mean square of successive differences (rMSSD) which is the square root of the averaged sum of squared differences in length between adjacent RR cycles.<sup>32</sup> These variables are independent of long term trends and are suggested to reflect predominantly vagus nerve activity.<sup>32</sup> All time domain indices are typically recorded in units of milliseconds.<sup>32,163</sup>

In addition to time domain analyses, recent developments in microprocessor technology have allowed ECG and PPG signal to be decomposed into its frequency components, thus enabling spectral analyses of HRV.<sup>163</sup> Frequency domain analysis addresses the question of "what are the underlying rhythms" within human heart rate by partitioning the total variance of this signal into the variance accounted for by specific groups of frequencies.<sup>32</sup> Frequency domain analysis requires that the power-spectral density of the RR intervals be calculated. This

procedure is commonly carried out using the Fourier transform (FT) or Fast Fourier transform (FFT).<sup>163</sup> As heart rate is measured in milliseconds, the variance of heart rate, which is referred to as the "power" in a portion of the total spectrum of signal frequencies, is measured in milliseconds squared.<sup>32</sup> Signal amplitude is also reported in some studies and this is the square root of power, and thus is also measured in milliseconds.<sup>32</sup>

The power spectrum in frequency analysis is divided into specific bands. High frequency power (HF) refers to signal in the 0.15 to 0.4 Hz band and is thought to represent primarily respiratory variation. Low frequency (LF) power refers to signal in the 0.04 to 0.15 Hz band. Very low frequency (VLF) power includes signal in the 0.003 to 0.04 Hz band. Finally, ultra-low frequency (ULF) power includes signal <0.003 Hz. The total power (TP) is the total variance within the signal including HF, LF, VLF, and ULF. Low frequency and HF spectra are normalized in some studies by dividing by TP.<sup>113,115</sup>

The SNS and PNS modulate the RR intervals in heart rate recordings at distinct frequencies. High frequency power is mainly associated with PNS activity, specifically through input from the vagus nerve.<sup>32,163</sup> Low frequency power is thought to be controlled by both the SNS and PNS and is strongly affected by the baroreceptor reflex.<sup>32</sup> Ultra low frequency and VLF spectra may represent the influence of the thermoregulators, peripheral vasomotor, or renin angiotensin systems.<sup>164</sup> This difference in frequency ranges allows HRV frequency analysis to separate sympathetic and parasympathetic contributions to the control of heart rate. The ratio of power in the low-to-high frequency bands (LF/HF) is therefore, used as an index of PNS-SNS balance which is important to understanding ANS health.<sup>163</sup>

There are distinct advantages and disadvantages associated with each type of HRV index. Broadly, time domain analyses are simple and easy to calculate, however, these methods do not allow for a clear separation of the influences of the SNS and PNS on heart rate. Frequency domain analyses provide a higher resolution perspective on physiological events such as ANS activity, however, studies such as that conducted by Al Haddad et al<sup>100</sup> have

shown that the reliability of frequency domain indices of HRV are reduced following bouts of exercise. The best assessment of HRV should, therefore, rely on the results of multiple indices including those from the time and frequency domains.

In studies involving wearable devices, spectral analysis may not always be possible for investigators. In these instances, it has been suggested by Plews et al<sup>33</sup> that practitioners and researchers using HRV measurements to evaluate physically active populations choose just one vagally-derived HRV variable for assessment. Further studies have found that rMSSD and Ln rMSSD are the most practical for ambulatory measures<sup>31</sup> as these are not significantly influenced by breathing frequency, and can capture PNS over a short time frame making it convenient for athletes.<sup>165</sup> Additionally, when HRV is used to assess changes in both negative<sup>65</sup> and positive adaptation<sup>166</sup> to exercise, both weekly<sup>65,166</sup> and 7-day rolling averages<sup>65</sup> have been shown to provide better methodological validity compared with measurements taken on a single day. Nunan et al conducted a review of past studies measuring HRV in healthy adult populations using rMSSD and reported a mean of 42 ( $\pm$  15 ms).<sup>80</sup>

### **Heart Rate Variability and Injury Rates**

Several recent studies have suggested intricate involvement of both the PNS and SNS in the initiation and regulation of tissue repair following injury. Danielson et al<sup>167</sup> compared tissues taken from human subjects with painful patellar tendinopathy and compared them with samples from healthy controls. Results showed increases in the location and presence of autonomic neuromediators in the tendinopathy patients. Similar findings were produced by Zeisig et al<sup>168</sup> in samples of tendon from subjects with symptomatic tennis and golfer's elbow. These data suggest that tissue damage as well as active tissue healing necessitates an increased need for ANS stimulation to bring blood supply and neuromediators. This increase can be reflected in HRV measurements which could allow clinicians to gain a more accurate picture of the healing status of tissues and their readiness to progress back to regular exercise.

Despite its distinct variation in relation to stages of tissue healing, there is limited research exploring the specific association between HRV and injury. One association that has received some attention is the relation between HRV and reduced athlete recovery and non-functional overreaching (NFOR) or the overtraining syndrome. These investigations have produced somewhat conflicting results.

An early study by Hedelin et al<sup>64</sup> measured HRV in a group of 9 elite canoeists during a 6-day intensive training camp. Although pre-and post-performance testing showed decreases in run time to fatigue and  $VO_{2max}$  suggestive of overtraining, there was no significant difference observed in HRV indices. These results are contradicted by Uusitalo et al<sup>169</sup> who assessed HRV using spectral analysis in 15 healthy female endurance athletes before and after 6-9 weeks of heavy training. Overall, heavy-training/overtraining induced decreases in maximal aerobic power and this reduction was related to decreased HRV. These results are paralleled by those of Baumert et al<sup>170</sup> who assessed the effects of intensified physical training over 2-weeks on cardiovascular control in 10 healthy athletes. Here, time domain measures of HRV (rMSSD) were found to be reduced following the intensive exercise period suggesting a shift of sympatho-vagal balance toward sympathetic activity.

The relation between HRV and injury has also been explored through the use of case reports. A case study of an elite over-trained cross-country skier conducted by Hedelin et al<sup>171</sup> showed reduced competition performance and lowered profile of mood states, along with substantially increased HF power in HRV spectral analysis as measured over several months of a training season. This suggests dominance of PNS stimulation in NFOR. These findings are refuted by another case study conducted by Plews et al<sup>65</sup> who assessed HRV over a 77-day period in an elite triathlete diagnosed with NFOR compared to their healthy training partner. The NFOR athlete had a decline in HRV leading up to a key competition compared to the healthy control and subsequently performed poorly in the event suggesting modulation in this measure may be strongly predictive of NFOR. In a larger scale longitudinal study, Schmitt et



al<sup>66</sup> aimed to compare HRV in 57 elite Nordic skiers identified via survey to be in either a "fatigue" or "no-fatigue" state. Athletes were studied over 4 years and HRV was found to be lower in athletes in the self-reported "fatigue" state compared with the "no-fatigue" state. Additionally, broader intra-individual variance of HRV parameters was seen in athletes in the "fatigue" state.

One final unique study by Hynen et al<sup>67</sup> evaluated the autonomic balance in 12 athletes who were pre-diagnosed with severe overtraining syndrome compared to a 12 member control group. Time and frequency domain measures of HRV were taken twice during the study, once during subject sleep and once upon subject waking. No differences in any HRV parameters were seen during night sleep, however, SDRRI and LF power were lower in over-trained athletes upon waking. Researchers suggest this alteration was due to decreases in PNS cardiac modulation.

The inconsistency of results taken from these research projects has stalled the use of the HRV metric in clinical practice. One possible reason behind these discrepancies is the differences in experimental design between studies. Researchers typically use two main strategies to explore the relation between HRV and injury. One method involves assessing HRV in a group of subjects as they are exposed to an acute increase in training load over a given time-period; often during a "training camp." This is advantageous as it allows for larger sample sizes and can be done using healthy subjects, however, it relies on the idea that the training regimen will definitively produce the physiological state present in NFOR for all subjects. Additionally, these projects are often not long enough to display a meaningful association. A second strategy is to take a longitudinal approach and follow an athlete or group of athletes through the course of their training season. This allows for more precise HRV averages to be assessed and can provide valid data if an athlete were to develop NFOR, however, these studies are time consuming and involve small sample sizes.

Other common inconsistencies between NFOR studies include the variety of HRV indices that have been used to assess autonomic balance, the lack of a common clinical definition and diagnostic criteria for overtraining and NFOR, and inconsistency in the conditions and devices used to measure heart rate.<sup>172</sup>

### **Heart Rate Variability and Illness Rates**

Perhaps the most notable characteristic of HRV in terms of its clinical relevance is its association with human illness. An early study conducted by Lin et al<sup>173</sup> reported conclusive evidence of diagnostic, prognostic, and predictive value for HRV in pediatric viral infections. Researchers assessed HRV via a 5-minute ECG performed within 24 hours of admission in 66 pediatric patients diagnosed with enterovirus. Results showed that (1) reductions in HRV were associated with enterovirus infection, (2) there was a strong correlation between HRV reduction and disease severity, and (3) HRV accurately identified those patients who subsequently deteriorated to a more advanced stage of the illness. A similar relation is present in adults across a range of disease states. In critically ill adult patients with sepsis studied by Garrard et al<sup>174</sup> and in septic shock patients studied by Piepoli et al<sup>62</sup> and Annane et al,<sup>175</sup> total HRV and LF were reduced during periods of sepsis when compared with periods of recovery and decreased HRV was associated with indices of illness severity.

While these studies in patients already diagnosed with infection are informative, their results are less applicable to clinicians hoping to provide preventative care compared to projects involving healthy subjects. These studies involve the measurement of HRV before and after facilitated injection of an illness vector such as endotoxin. One such study was conducted by Godin et al<sup>69</sup> in a group of 12 subjects experimentally injected with endotoxin. HRV was measured continuously for 8 hours using an ECG and HRV indices included time domain (SD), frequency domain (HF and LF power), and entropy metrics. Infusion of endotoxin caused loss of HRV as measured by SD and power spectra, as well as an increase in heart regularity, as measured by entropy. These findings were upheld in a study conducted by Rassias et al<sup>70</sup> in a

group of 8 healthy volunteers injected with endotoxin. Continuous ECG recordings over the 12-hour testing sessions were used to create time series data of heart rate. Increased regularity and decreased HRV were observed in response to the administration of endotoxin and these changes were noted within the first hour after injection, occurring earlier than increases in body temperature. Interestingly, the increased regularity in HRV also persisted despite a blockade of symptoms by administration of ibuprofen in the subjects and lasted longer than the metabolic abnormalities resulting from the infection. This suggests that HRV may be useful in determining illness prognosis and making decision regarding return to daily activities.

Use of the HRV metric within a clinical context is dependent upon a pathophysiologic explanation of the alterations seen following infection. One prominent theory is that these modulations in HRV are due to dysfunction within the ANS caused by the infection.<sup>173</sup> Typically, this dysfunction involves a marked SNS response and depressed vagal mediation.<sup>173</sup> These responses are consistent with the increased LF/HF HRV commonly seen in clinically ill patients or those injected with endotoxin.<sup>71</sup>

This collection of evidence suggests that HRV may be a valid means of determining both when an individual has been infected with an illness as well as the prognosis of the acquired condition. Wearable technology will make HRV data increasingly available to sports medicine clinicians. A basic understanding of the HRV metric and the physiological insights behind it could significantly improve patient care.

### **Training Load and Injury/illness Rates**

Multiple methods have been used to quantify the concept of practice load in athletes. These strategies can be divided into two main categories: those that measure external load and those that measure internal load. External load involves the actual training or competition stress placed on athletes, such as hours of practice, distance run, games played, or pitches thrown.<sup>176</sup> Internal load quantifies the internal physiological and psychological responses to external load such as heart rate or ratings of perceived exertion (RPE).<sup>176</sup> To date no single marker of an

athlete's response to load has been found to consistently predict maladaptation, injuries, or illnesses.

The majority of studies assessing the relation between load and injury/illness in athletes have used measurements of absolute load, that is, an athlete's external or internal load regardless of the rate of load application or load history.<sup>176</sup> High absolute load has been identified as a risk factor for injury in multiple sports including swimming. One such study by Sein et al<sup>7</sup> evaluated the pathogenesis of shoulder pain in 80 young elite swimmers using a cross-sectional design. Supraspinatus tendinopathy was identified as the major cause of shoulder pain and regression analysis found that the condition could be predicted in 85% of swimmers either from number of hours swum per week alone or in combination with the swimmer's weekly mileage. Additionally, swimmers who trained more than 15 hours per week and swim a weekly distance of more than 35 km were identified as being at increased risk for supraspinatus tendinopathy. A study by Ristolainen et al<sup>177</sup> in 446 endurance athletes including swimmers confirmed these results. Athletes self-reported training load including years of active training, hours trained yearly, competition hours and weekly rest days as well as occurrence of overuse injuries. Athletes with less than 2 rest days per week during training had a 5.2-fold risk (95% CI= 1.89-14.06,  $P= 0.001$ ) for an overuse injury, and athletes who trained more than 700 hours during a year had 2.1-fold risk (95% CI= 1.21-3.61,  $P= 0.008$ ) for an overuse injury. Low number of recovery days and high number of training hours were highlighted as risk factors for overuse injuries.

In addition to these studies detailing the negative effects of high absolute load on injury risk, other investigators reported that high absolute load does not affect injury risk and may even offer protection from injury.<sup>178</sup> These confounding results led to more recent experiments which suggest that excessive and rapid increases in load, and not necessarily the total absolute load, are what lead to increased occurrence of athlete injuries. Gabbett et al<sup>46</sup> introduced the concept of the acute:chronic load ratio to model the relation between training load and injury risk. This

ratio describes the acute training load as being the load within the last week, in relation to the 4-week rolling average of load. Studies by Hulin et al<sup>21</sup> and Blanch et al<sup>179</sup> have shown that when this acute:chronic load ratio exceeds 1.5 the likelihood of injury more than doubles.

Training load has also been studied in relation to athlete illness. In a review written by Drew et al<sup>176</sup> a moderate positive relation was found between the two variables. Piggot et al<sup>180</sup> studied external load expressed as the product of the athlete's self-reported RPE and the number of minutes training above 80% of an individual's maximum heart rate in 16 elite Australian football players. Forty-two percent of recorded illnesses were preceded by a 10% increase in training load above levels seen during the previous week. A study by Foster et al<sup>181</sup> in 25 competitive speed skaters supports these results with 84% of illnesses explained by a preceding spike in training load above an individual's threshold. A more recent study by Brink et al<sup>23</sup> in soccer players showed that greater duration of physical training as well as higher levels of psychosocial stress in the athletes lead to a greater likelihood of sustaining an illness. Hellard et al<sup>8</sup> conducted a study in swimming athletes specifically which also included a more accurate measure of training intensity using blood lactate concentrations and swimming distance. A dose response relationship was discovered between training intensity and illness. Along with this body of evidence, however, studies including work by Fricker et al<sup>182</sup> in runners and Anderson et al<sup>183</sup> in female basketball players show no correlation between training load (mileage, intensity, or RPE x time) and illness.

This evidence suggests that both internal and external load have a significant influence on injury/illness risk in athletes and the collection and discussion of this data by coaches and care providers could be vital to athlete health.

### **Body Composition and Injury/Illness Rates**

Body composition may be another significant predictor of injury and illness in athletes. Common anthropometric measurements include subject height, Body Mass Index (BMI), and percent body fat (%BF). A study by Dane et al<sup>184</sup> explored the association between BMI, body

fat, and power of various muscles to sport injuries in 456 athlete subjects. Body Mass Index was higher in injured athletes than in non-injured athletes, but no correlation was found between body fat and injury. The results involving BMI and injury were upheld by a more recent study by Sayyah et al<sup>185</sup> in a group of 170 female Olympiad athletes. A total of 37 subjects suffered injury during this study and those athletes who sustained an injury were found to have significantly higher BMI values compared to the non-injured athletes. Jespersen et al<sup>24</sup> conducted a longer duration cohort study over 2.5 years involving 632 youth athletes competing in American football. The risk of lower extremity injuries was increased in children found to be overweight as assessed by both BMI and %BF measured by x-ray absorptiometry scans. When comparing the different measures of overweight, overweight by %BF was a higher risk factor for injury compared to BMI. Finally, Gomez et al<sup>25</sup> measured body fatness and injury rates in 215 varsity football lineman. Athletes were divided into high and low fatness groups based on results of skin fold measurements. Athletes in the higher body fatness and BMI groups were found to sustain significantly more injuries.

Along with these studies finding positive correlations between anthropometric data and injury, additional studies found no relation between these variables. Notably, Foss et al<sup>186</sup> found no relation between BMI nor %BF and the incidence of anterior knee pain in 248 middle school basketball athletes.

Overall, these studies appear to suggest that anthropometric measures are important to consider when modeling risk factors of athlete injury. Furthermore, %BF may be a more influential variable compared to BMI when assessing this association.

## Appendix A

### Recruitment Script

Good afternoon everyone, I hope you all had a wonderful summer. My name is Bruin Armwald and I am the athletic trainer for the Penn State men's and women's tennis teams. I want to thank Coach Murphy for the opportunity to speak with you all today about an oftentimes overlooked aspect of life for collegiate athletes, and that is your sleeping habits. In addition to working as an athletic trainer, I am also a graduate student in the Department of Kinesiology and will be conducting a thesis project to study the relation between sleep and injury/illness rates as well as performance in collegiate athletes. So, to begin this short presentation I will present a brief set of statistics detailing what average sleep looks like in the general population as well as what has been established in other athlete populations. I will then draw this in to focus on data that has been gathered about your team from this past competitive season. Finally, I will finish by detailing the specifics of my study and how you can go about being a part of this research.

The American Academy of Sleep Medicine and Sleep Research Society recently recommended all healthy adults aged 18-65 acquire at least 7 hours of sleep per night to ensure optimal health.<sup>1</sup> Due to the high levels of physical stress experienced by athletes, it has been proposed that this population requires as much as 9 to 10 hours of sleep to ensure adequate recovery, with 80-90% occurring during the night.<sup>2</sup> Despite these conclusions, it has been documented that many athletes suffer from sleep restriction and reduced sleep quality.<sup>3,4</sup> In one study involving 7 elite Australian swimmers it was found that subjects slept for an average of 5.4 hours on nights prior to training days. Furthermore, for night-time sleep periods preceding training days and rest days, swimmers' sleep efficiency was 71% and 77% respectively<sup>3</sup>, both of which fall below the average of 90% seen in healthy young adults.<sup>5</sup> In another study conducted by a different group of researchers sleep data was recorded in 46 Olympic athletes. The results showed that athletes slept for a lower mean total duration, had poorer sleep efficiency, and

higher fragmentation indices compared to age and sex matched controls.<sup>4</sup> This data serves to illustrate that in general, athletes appear to require more sleep compared to the average population, however, they are not achieving this level of sleep which could lead to undesirable consequences such as injury, illness, and decreased performance.

Next, I will move on to show some of the data that has already been collected both by WHOOP and the Sports Medicine team here at Penn State from our past competitive season. These data sets have been deidentified to ensure the confidentiality of the individuals who wore the WHOOP system in the past. For those who are not familiar with the system, WHOOP is a wearable activity tracker which collects five major human vital signs including heart rate, heart rate variability, electro-dermal activity, ambient temperature, and three-dimensional acceleration. Using this system along with a complex algorithm WHOOP can also calculate variables such as recovery score and sleep. For this preliminary pilot study, I correlated averages for various sleep variables against illness data taken between September of 2016 and February of 2017 to see what associations could be found.

This first graph is showing the average number of illnesses plotted against the average total sleep duration for the participants and this has been divided up by month. What I would like to call your attention to both on this slide and the slides to come is the spike in illnesses seen in December and January compared to September-October. This does not seem to be completely explained by total sleep as illustrated in this graph. Although December appears to be one of the lowest months in terms of average sleep, there is a recovery in January but the number of illnesses remains constant.

As we searched further, however, there does seem to be a more interesting association between REM sleep, specifically and illness rates. There is a constant decline of REM sleep through the season with December and January representing the months with the lowest averages, while these months also have the highest illness rates. This could suggest some relation between the loss of REM sleep and increased risk of illness.



In this next graph, we see another apparent relation between illness rates and the number of sleep cycles completed per night by the athlete participants. Here again we see as athletes complete fewer sleep cycles there may be an increased risk of suffering illness.

This next graph shows what I view to be the most interesting type of relation between the average duration of illness or how long it took the athletes to return to activity after suffering illness, and average total sleep duration. Here we see that when the average total sleep duration was at a low point in December, the duration of illness was very high, however, when athletes began getting more sleep in January the average duration of the illnesses decreased. This may suggest that the longer sleep helps athletes recover from their illnesses faster.

Another source of data we explored was the team's performance in meets. So, we looked at the team's schedule and tried to find out the average total amount of sleep before meets that the team took first and before meets where the team did not take first. We also looked at sleep before home meets and compared this to the amount of sleep gained before away meets. We began this analysis by looking at the average for an entire week before meets won and meets lost and found only a slight difference of about 19 minutes. This was similar to the results found when we looked at average amount of sleep the week before home meets vs the week before away meets which was just a 5-minute difference.

These differences in average weekly sleep were not very convincing and so we began analyzing sleep habits the day before meet wins versus meet losses and home meets versus away meets. When comparing wins versus losses there is a much greater difference in sleep as can be seen in this graph with participants sleeping an average of 47 minutes longer the night before meets they won versus meets that they did not win. Similarly, the participants were found to sleep for an average of 48 minutes longer the night before a home meet compared to an away meet.

Thus, one can see from this pilot analysis that this recovery data can be extremely informative both to your health, in terms of your risk for injury illness and the duration of recovery associated with these conditions as well as to your performance.

Now I will speak a bit about my research study and what is involved for those individuals who will be participating. The objectives of this project are:

- To collect normative sleep and HRV data in collegiate athletes
- To assess the relation between sleep metrics, HRV, injury/illness rates, and time to return to play in collegiate athletes
- To explore the relation between sleep metrics, HRV, injury/illness rates, and performance in collegiate athletes
- To study the influence of body composition and training load on athlete health and performance

The procedures involved in the study will not require any additional testing or other activities outside of what you are already required to do by your coaching staff and the Athletics Department. Subjects will wear the WHOOP device on their wrist continuously for the duration of the competitive season so that their sleep and heart rate data can be recorded. Specific variables that will be studied by the research team taken from WHOOP include HRV, resting heart rate, time in bed, total sleep time, REM sleep, slow wave sleep, disturbances, sleep onset latency, and sleep cycles completed. Subjects will report all health-related issues to their athletic trainer, Kelly Saxton, who will document these conditions and track how much time is missed due to the injury or illness. Subjects' daily practice time and swimming distance will be recorded for the investigators by athletic trainers and the coaching staff. Additionally, subjects will fill out questionnaires following specific training sessions to give an idea of subjective training load in terms of physical exertion as well as mental stress. Subject's race times will be collected from the coaching staff. Finally, subjects must undergo BOD POD testing to control for the impact of body composition. Variables related to body composition that will be analyzed by researchers

include percent body fat, percent fat-free mass, fat mass, fat-free mass, bod mass, body volume, and body density. Essentially, I am hoping to use the data that is already collected from you during your season and use it for the purpose of this research project.

There are two basic criteria that are necessary to be included in the study. You must be a member of the swimming team and you must also be between the ages of 18-27 years of age. You will be excluded from participation in the study if you do not meet both of these criteria.

There will be no monetary compensation for participating in this study, however, you can receive regular reports on your personal sleep and recovery data upon request. Additionally, your coaching staff will receive an aggregate report for the entire team's recovery metrics, however, individual data will not be provided to your coaches or athletic training staff by any investigator working on this study. As we have already seen this data can be greatly beneficial both to ensure proper health and to improve athletic performance. There are also no risks associated with participation in this study. Your data will be deidentified and no one aside from the study investigators will have access to your individual records. All data will be stored in locked cabinets and/or on a password protected computer both of which are located in the Athletic Training Research Laboratory Located in the Recreation Building. Finally, your participation in this study is completely voluntary and there are no consequences involved with not being part of this research.

Thank you again very much for your time. If you have any questions I can answer them now, or you can feel free to ask me individually. When you feel you have been fully informed regarding all procedures involved with this study and consent to allowing your data to be used please come to me and sign two documents. One is an informed consent form for the study and the other is a HIPAA release document as we will be analyzing medical records.

**Appendix B**  
**Participant Informed Consent Form**

**CONSENT FOR RESEARCH**

The Pennsylvania State University

Title of Project: Sleep and Heart Rate Variability and their Relation to Measures of Sport Performance Injury Rates, and Time to Return to Play in Collegiate Athletes

Principal Investigator: Sayers John Miller III, PhD, PT, ATC

Address: 146 Recreation Building, University Park, PA 16802

Telephone Number: 814- 865-1775

Subject's Printed Name: \_\_\_\_\_

**We are asking you to be in a research study. This form gives you information about the research.**

**Whether or not you take part is up to you. You can choose not to take part. You can agree to take part and later change your mind. Your decision will not be held against you.**

**Please ask questions about anything that is unclear to you and take your time to make your choice.**

**1. Why is this research study being done?**

We are asking you to be in this research because you are currently a varsity student athlete participating on the Penn State Swimming team.

This research is being done to find out whether or not there is a significant relation between recovery metrics (such as sleep and HRV), injury and illness rates, and time to return to play in collegiate athletes.

Approximately 25 people will take part in this research study at this local site.

## **2. What will happen in this research study?**

This study will not involve any direct testing by investigators. The analysis will rely on data that is already set to be collected as a result of your status as a student athlete on the Penn State swimming team. The study team is requesting permission to utilize this data for research purposes. The following is a list of the data the study team would like permission to view:

- BOD POD testing data: height, weight, body fat, percent fat-free mass, fat mass, fat-free mass, body mass, body volume, and body density
- WHOOP Performance Optimization System data: total sleep time, REM sleep, slow wave sleep, disturbances, sleep onset latency, sleep cycles, heart rate and heart rate variability
- Online Medical Records: number of Injuries, number of illnesses, and number of participation days missed due to specific injuries and/or illnesses. Basic demographic information including gender and date of birth will also be recorded
- External load: the distance each participant swims during each team practice session and the amount of time spent training
- Internal load: subjective rating of perceived exertion (RPE) during training, rating of perceived stress due to academic responsibilities, and rating of sleep quality and duration
- Competition Results: race times taken from team coaching staff.

## **3. What are the risks and possible discomforts from being in this research study?**

There is a risk of loss of confidentiality if your information or your identity is obtained by someone other than the investigators, but precautions will be taken to prevent this from happening. The confidentiality of your electronic data created by you or by the researchers will be maintained to the degree permitted by the technology used. Absolute confidentiality cannot be guaranteed. As there will be no direct testing executed by study investigators, the potential loss of confidentiality is the only foreseeable risk associated with this study.

#### **4. What are the possible benefits from being in this research study?**

The results of the study will add to the existing body of literature on sleep metrics and HRV and their relation to athletic performance, injury rates, and time to return to play. The inclusion of body composition and training load will provide a more well-rounded picture of the influence of recovery measures in collegiate athletes. Furthermore, clinicians will be made aware of the benefits of this new recovery data to their clinical practice in terms of identifying individual athletes who are at an increased risk of suffering injury, illness, or prolonged time to return to play. Finally, normative, objective sleep and HRV data will be recorded for a collegiate athlete population for the first time.

#### **5. What other options are available instead of being in this research study?**

You may decide not to participate in this research. If so you will still be required to complete the procedures listed above due to your status as a student athlete participating on the Penn State swimming team.

#### **6. How long will you take part in this research study?**

If you agree to take part, the study will last for the duration of the competitive swimming season. Being in this study does not require any additional time on your part.

#### **7. How will your privacy and confidentiality be protected if you decide to take part in this research study?**

Efforts will be made to limit the use and sharing of your personal research information to people who have a need to review this information.

- A list that matches your name with your code number will be kept in a locked file or password protected file. This master code will be kept within a locked filing cabinet within the Athletic Training Research Laboratory in the Recreation Building. No electronic record of the master code will be kept.
- Your research records will be labeled with your code number, and will be kept within an electronic file saved on a secure hard drive and or computer server inside the Athletic Training Research laboratory in the Recreation Building.

In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

We will do our best to keep your participation in this research study confidential to the extent permitted by law. However, it is possible that other people may find out about your

participation in this research study. For example, the following people/groups may check and copy records about this research.

- The Office for Human Research Protections in the U. S. Department of Health and Human Services
- The Institutional Review Board (a committee that reviews and approves research studies) and
- The Office for Research Protections.

Some of these records could contain information that personally identifies you. Reasonable efforts will be made to keep the personal information in your research record private. However, absolute confidentiality cannot be guaranteed.

#### **8. What are your rights if you take part in this research study?**

Taking part in this research study is voluntary.

- You do not have to be in this research.
- If you choose to be in this research, you have the right to stop at any time.
- If you decide not to be in this research or if you decide to stop at a later date, there will be no penalty or loss of benefits to which you are entitled.

#### **9. If you have questions or concerns about this research study, whom should you call?**

Please call the head of the research study, Dr. Sayers John Miller III at 814-865-6782:

- Have questions, complaints or concerns about the research.
- Believe you may have been harmed by being in the research study.

You may also contact the Office for Research Protections at (814) 865-1775, OR [Protections@psu.edu](mailto:Protections@psu.edu) if you:

- Have questions regarding your rights as a person in a research study.
- Have concerns or general questions about the research.
- You may also call this number if you cannot reach the research team or wish to offer input or to talk to someone else about any concerns related to the research.

Please keep in mind that whether or not you agree to take part in this study, this will have no bearing on our status as a member of the Penn State swimming team.

## **INFORMED CONSENT TO TAKE PART IN RESEARCH**

### **Signature of Person Obtaining Informed Consent**

Your signature below means that you have explained the research to the subject or subject representative and have answered any questions he/she has about the research.

\_\_\_\_\_  
Signature of person who explained this research      Date      Printed Name

(Only approved investigators for this research may explain the research and obtain informed consent.)

### **Signature of Person Giving Informed Consent**

Before making the decision about being in this research you should have:

- Discussed this research study with an investigator,
- Read the information in this form, and
- Had the opportunity to ask any questions you may have.

Your signature below means that you have received this information, have asked the questions you currently have about the research and those questions have been answered. You will receive a copy of the signed and dated form to keep for future reference.

### **Signature of Subject**

By signing this consent form, you indicate that you voluntarily choose to be in this research and agree to allow your information to be used and shared as described above.

\_\_\_\_\_  
Signature of Subject      Date      Printed Name



## Appendix C

## Participant HIPAA Authorization Form

The 330 Building, Suite 205

University Park, PA 16802

814-865-1775

Fax: 814-863-8699

[ORProtections@psu.edu](mailto:ORProtections@psu.edu)

## AUTHORIZATION TO USE & DISCLOSE PROTECTED HEALTH INFORMATION FOR RESEARCH PURPOSES

**IRB#:** STUDY007488

**Project Title:** Sleep and Heart Rate Variability and their Relation to Measures of Sport Performance, Injury and Illness Rates, and Time to Return to Play in Collegiate Athletes

**Principal Investigator:** Sayers John Miller III

The privacy law, Health Insurance Portability & Accountability Act (HIPAA), protects my individually identifiable health information (protected health information<sup>1</sup>). The privacy law requires me to sign an authorization (or agreement) in order for researchers to be able to use or disclose my protected health information (PHI) for research purposes in the above referenced study. I authorize Sayers John Miller III and his/her research staff to use and disclose my protected health information for the purposes described below.

**The following doctors and/or health care providers are authorized to disclose my protected health information for the purposes of this research study:**

- Penn State Swimming Team Physician
- Penn State Swimming Team Orthopedic Surgeon
- Penn State Swimming Team Athletic Trainers

**My protected health information that may be used and disclosed includes:**

- Demographic information:
  - Date of birth
  - Gender
  - Height
  - Weight
- Body composition testing (BOD POD) results:
  - Percentage body fat
  - Percentage fat free mass
  - Fat mass
  - Fat-free mass
  - Body mass
  - Body volume

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<sup>1</sup> Name, Address, Dates Directly Related to an Individual, Telephone/Fax Number, E-mail/Internet Protocol or Web URL Address, Social Security Number, Medical Record or Health Plan Number, Account Number, Certificate of License Number, Photographic Images, Vehicle Identifiers, Device Identifiers, Biometric Identifiers, Any Other Unique Code

- Body density
- Actigraphy (WHOOP) data:
  - Resting heart rate
  - Heart rate variability (HRV)
  - Number of hours in bed
  - Total sleep duration
  - Sleep disturbances
  - Sleep onset latency (SOL)
  - Wake after sleep onset (WASO)
  - Number of sleep cycles completed
  - Time in light sleep
  - Time in deep sleep
  - Time in rapid eye movement (REM) sleep
- Medical History:
  - Injuries
  - Illnesses
  - Missed practices and competitions due to injury and/or illness
- Training Load:
  - External load:
    - Swimming distance
    - Time spent completing team activities (practices, conditioning/weight lifting, competitions)
  - Internal load:
    - Ratings of Perceived Exertion (RPE)
    - Ratings of perceived mental stress
    - Ratings of perceived sleep quality

**My protected health information will be used for:**

- Establishing normative data with regards to sleep and heart rate variability (HRV) in a collegiate athlete population.
- Exploring the relation between recovery metrics (sleep and HRV) and injury/illness rates and time to return to play for the purpose of establishing the predictive power of these recovery measurements.
- Exploring the role of recovery metrics (sleep and HRV) in contributing to athletic performance.

**The Researchers may use and share my health information with:**

- The Pennsylvania State University's Institutional Review Board/Office for Research Protections
- Government representatives, when required by law

Your health information may be used or shared with other specific people or groups in connection with this research study. Research records that identify you will be kept confidential as required by law. You will not be identified by name, social security number, address, phone number or any other direct personal identifier in research records given to someone outside of The Pennsylvania State University (PSU), except when required by law. For records shared outside of PSU, you will be assigned a code number. The list that matches your name with the code number will be kept in a locked file in the principal investigator's office.

The researchers agree to protect my health information by using and disclosing it only as permitted by me in this Authorization and as directed by state and federal law. Should the health information be disclosed by the researcher, to someone outside of this study, it may no longer be covered/protected by the federal regulation HIPAA.

**I do not have to sign this Authorization. If I decide not to sign the Authorization:**

- It will not affect my treatment, payment or enrollment in any health plans or affect my eligibility for benefits.
- I may not be allowed to participate in the research study.
- If applicable, I will not have access to this research-related therapy/treatment.

**After signing the Authorization, I can change my mind and:**

- Not let the researcher disclose or use my protected health information (revoke the Authorization).
- If I revoke the Authorization, I will send a written letter to: Sayers John Miller at 146 Recreation Building, University Park, PA 16802 to inform him of my decision.
- If I revoke this Authorization, researchers may only use and disclose the protected health information **already** collected for this research study.
- If I revoke this Authorization my protected health information may still be used and disclosed should I have an adverse event (a bad effect).
- If I change my mind and withdraw the authorization, I may not be allowed to continue to participate in the study.

This Authorization does not have an expiration date.

**If I have any questions or concerns about my privacy rights, I should contact the Office for Research Protections at (814) 865-1775.**

**I am the participant or am authorized to act on the participant's behalf. I have read this information, and I will receive a copy of this form after it is signed.**

\_\_\_\_\_  
Signature of research participant or \*research participant's legal representative

\_\_\_\_\_  
Date

\_\_\_\_\_  
Printed name of research participant or \*research participant's legal representative

\_\_\_\_\_  
Representative's relationship to research participant

\*Please explain the Representative's relationship to the participant. Include a description of the Representative's authority to act on participant's behalf:

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

## Appendix D

### IRB Approval Letter



IRB Program  
Office for Research Protections

Vice President for Research  
The Pennsylvania State University  
205 The 330 Building  
University Park, PA 16802

Phone : (814) 865-1775  
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Email : [orprotections@psu.edu](mailto:orprotections@psu.edu)  
Web : [www.research.psu.edu/orp](http://www.research.psu.edu/orp)

### APPROVAL OF SUBMISSION

**Date:** August 9, 2017

**From:** Jodi Mathieu, IRB Analyst

**To:** Sayers Miller, III

Type of Submission:	Initial Study
Title of Study:	Sleep and Heart Rate Variability and their Relation to Measures of Sport Performance, Injury and Illness Rates, and Time to Return to Play in Collegiate Athletes
Principal Investigator:	Sayers Miller, III
Study ID:	STUDY00007488
Submission ID:	STUDY00007488
Funding:	Not Applicable
IND,IDE, or HDE:	Not Applicable
Documents Approved:	<ul style="list-style-type: none"> <li>• Master's Thesis hipaa-authorization-form Version 8-4-17.doc (2.01), Category: Other</li> <li>• Masters Thesis HRP-588 - ORP Consent Form Version 7-22-17.pdf (3), Category: Consent Form</li> </ul>

	<ul style="list-style-type: none"> <li>• Masters Thesis HRP-591 - Protocol for Human Subject Research Version 8-8-17.pdf (0.04), Category: IRB Protocol</li> <li>• Masters Thesis Presentation to Swimming Version 8-5-17.pptx (2.01), Category: Recruitment Materials</li> <li>• Master's Thesis Recruitment Presentation Version 7-22-17.docx (2), Category: Recruitment Materials</li> <li>• Master's Thesis Subjective Questionnaire.docx (0.01), Category: Data Collection Instrument</li> </ul>
Review Level:	Expedited
IRB Board Meeting Date:	

On 8/9/2017, the IRB approved the above-referenced Initial Study. This approval is effective through 8/8/2018 inclusive. You must submit a continuing review form with all required explanations for this study at least 45 days before the study's approval end date. You can submit a continuing review by navigating to the active study and clicking 'Create Modification / CR'.

If continuing review approval is not granted before 8/8/2018, approval of this study expires on that date.

To document consent, use the consent documents that were approved and stamped by the IRB. Go to the Documents tab to download them.

In conducting this study, you are required to follow the requirements listed in the Investigator Manual ([HRP-103](#)), which can be found by navigating to the IRB Library within CATS IRB (<http://irb.psu.edu>). These requirements include, but are not limited to:

- Documenting consent
- Requesting modification(s)
- Requesting continuing review
- Closing a study
- Reporting new information about a study
- Registering an applicable clinical trial
- Maintaining research records

This correspondence should be maintained with your records.

**Appendix E****Borg CR10 Perceived Exertion Scale<sup>40</sup>**

<b>0</b>	<b>Nothing at All</b>
<b>0.5</b>	<b>Very, Very Light</b>
<b>1</b>	<b>Very Light</b>
<b>2</b>	<b>Fairly Light</b>
<b>3</b>	<b>Moderate</b>
<b>4</b>	<b>Somewhat Hard</b>
<b>5</b>	<b>Hard</b>
<b>6</b>	
<b>7</b>	<b>Very Hard</b>
<b>8</b>	
<b>9</b>	
<b>10</b>	<b>Very, Very Hard - Maximal</b>

## Appendix F

### Model Variance Inflation Factor Tables

#### Variance Inflation Factors Injury Model

Variable	Variance Inflation Factor
Sleep Efficiency	1.07
RHR	1.09
Total Load	1.02

#### Variance Inflation Factors Illness Model

Variable	Variance Inflation Factor
Total Sleep	1.06
Total Yardage	1.06

#### Variance Inflation Factors Missed Days Model

Variable	Variance Inflation Factor
Total Yardage	1.08
RHR	1.12
Total Sleep	1.21
Sleep Efficiency	1.17

## Appendix G

### Individual and Group Moving Average and Acute:Chronic Ratio Tables

#### Average 3-day Moving Averages Before Injury

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	5668.55	137.50	836.25	4.70	92.19	4.33	5.48	50.33	101.00
2	8681.39	150.00	1058.33	6.17	91.48	6.50	6.73	40.83	109.50
3	7638.51	181.67	1296.67	6.93	91.28	3.94	11.15	55.44	66.89
4	3233.21	103.75	722.50	6.42	87.73	5.36	14.75	46.69	90.75
7	4095.86	120.89	887.32	6.50	94.66	5.33	0.83	49.00	81.67
8	6833.33	155.00	1060.00	5.72	93.48	3.00	2.48	62.00	29.00
9	4319.76	135.00	858.33	7.00	89.95	6.11	8.20	60.78	55.78
10	6900.00	125.00	900.00	5.10	70.16	2.67	9.81	44.00	127.67
<b>Group Average</b>	<b>5921.33 ± 675.38</b>	<b>138.60 ± 8.43</b>	<b>952.43 ± 63.17</b>	<b>6.02 ± 0.29</b>	<b>88.86 ± 2.77</b>	<b>4.66 ± 0.50</b>	<b>7.43 ± 1.62</b>	<b>51.13 ± 2.71</b>	<b>82.78 ± 11.18</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

#### Average 7-day Moving Averages Before Injury

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	4502.27	117.86	657.32	4.24	87.43	5.57	3.84	51.14	94.29
2	7922.41	137.76	945.24	6.43	89.53	6.21	11.69	41.29	109.50
3	6099.17	160.19	1176.90	6.66	89.91	4.56	12.83	54.75	67.44
4	3589.56	110.89	756.96	6.47	87.09	5.91	13.92	46.28	78.78
7	3193.97	92.33	712.42	6.27	91.96	5.83	2.82	47.00	74.83
8	5857.14	139.29	942.86	6.82	92.22	4.61	3.00	61.74	36.76
9	5889.16	155.71	959.64	7.32	90.25	6.43	7.00	61.52	56.67
10	8671.43	161.71	1188.86	5.06	66.87	3.00	12.95	45.25	134.82
<b>Group Average</b>	<b>5715.64 ± 683.41</b>	<b>134.47 ± 8.96</b>	<b>917.53 ± 70.66</b>	<b>6.16 ± 0.36</b>	<b>86.91 ± 2.94</b>	<b>5.27 ± 0.40</b>	<b>8.51 ± 1.71</b>	<b>51.12 ± 2.69</b>	<b>81.64 ± 10.90</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds



### Average Acute:Chronic Ratios Before Injury

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	1.13	1.16	1.24	0.93	1.01	1.15	1.07	0.92	1.18
2	1.04	0.99	0.98	1.00	1.02	0.80	1.09	1.00	0.97
3	1.37	1.41	1.57	0.94	1.02	0.81	1.06	1.01	0.98
4	0.87	0.96	0.95	1.02	1.02	1.05	0.90	0.98	0.95
7	1.07	0.97	1.00	0.97	1.01	0.93	1.39	1.00	0.96
8	0.91	0.96	0.86	0.98	1.02	0.86	0.50	1.00	0.91
9	1.04	1.10	1.10	0.96	1.01	1.07	0.59	1.00	1.01
10	1.17	1.08	1.07	0.92	0.97	0.67	0.77	0.97	1.08
<b>Group Average</b>	<b>1.07 ± 0.05</b>	<b>1.08 ± 0.05</b>	<b>1.10 ± 0.08</b>	<b>0.96 ± 0.01</b>	<b>1.01 ± 0.01</b>	<b>0.92 ± 0.06</b>	<b>0.91 ± 0.10</b>	<b>0.99 ± 0.01</b>	<b>1.00 ± 0.03</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

### Average 3-day Moving Averages Before Illness

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	5154.85	141.75	813.95	4.47	85.65	5.13	2.89	59.73	71.20
2	4405.34	119.13	686.21	7.23	91.04	4.96	6.72	51.83	58.92
3	4831.62	120.00	738.00	6.50	82.65	5.35	17.35	49.17	78.32
4	4597.46	127.06	877.61	6.59	93.59	5.06	2.18	59.78	93.44
7	7000.00	130.00	930.00	5.94	77.34	4.00	12.48	51.00	145.00
8	3506.16	88.48	651.92	6.02	90.70	6.38	1.00	48.33	81.08
9	5383.57	142.50	868.75	7.35	90.21	5.50	15.50	60.08	59.42
10	2941.67	73.83	500.33	5.59	64.17	4.74	31.83	47.72	94.20
<b>Group Average</b>	<b>4727.58 ± 434.86</b>	<b>117.84 ± 8.68</b>	<b>758.35 ± 50.44</b>	<b>6.21 ± 0.33</b>	<b>84.42 ± 3.45</b>	<b>5.14 ± 0.24</b>	<b>11.24 ± 3.68</b>	<b>53.46 ± 1.93</b>	<b>85.20 ± 9.77</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

### Average 7-day Moving Averages Before Illness

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	4704.08	129.67	724.16	4.60	84.78	5.86	3.38	59.29	68.40
3	4490.80	121.23	730.70	7.02	88.76	5.57	7.80	52.99	61.43
4	4891.84	121.71	766.29	6.41	81.40	5.61	22.50	48.69	82.58
5	5898.37	154.45	1048.26	6.56	92.76	5.46	2.95	61.16	92.12
6	7857.14	150.00	1005.00	7.46	78.90	3.60	22.88	54.40	84.80
7	3603.84	102.99	781.03	6.27	90.92	6.24	1.05	47.96	81.63
9	5188.40	137.34	856.52	7.33	88.79	5.68	18.04	61.68	55.64
10	4749.59	93.29	494.93	5.64	60.43	5.25	32.20	46.88	116.16
<b>Group Average</b>	<b>5173.01 ± 445.95</b>	<b>126.34 ± 7.52</b>	<b>800.86 ± 61.52</b>	<b>6.41 ± 0.33</b>	<b>83.34 ± 3.67</b>	<b>5.41 ± 0.28</b>	<b>13.85 ± 4.10</b>	<b>54.13 ± 2.13</b>	<b>80.34 ± 6.76</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

### Average Acute:Chronic Ratios Before Illness

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	1.17	1.22	1.32	0.88	0.97	0.97	0.97	1.04	0.92
2	0.82	0.86	0.72	0.99	1.01	1.01	0.79	0.98	0.92
3	0.89	0.90	0.92	0.97	0.99	0.95	0.96	1.00	0.98
4	0.74	1.13	1.14	0.97	0.99	0.89	1.20	0.97	1.09
7	1.15	1.16	1.31	1.01	0.94	0.66	1.32	1.00	1.30
8	0.84	0.95	1.00	0.94	1.01	0.89	0.99	0.99	1.05
9	1.04	1.07	1.13	0.99	1.00	1.02	1.27	0.97	1.03
10	0.78	0.74	0.64	1.05	0.92	1.07	1.59	0.99	1.10
<b>Group Average</b>	<b>0.93 ± 0.06</b>	<b>1.01 ± 0.06</b>	<b>1.02 ± 0.09</b>	<b>0.98 ± 0.02</b>	<b>0.98 ± 0.01</b>	<b>0.93 ± 0.04</b>	<b>1.14 ± 0.09</b>	<b>0.99 ± 0.01</b>	<b>1.05 ± 0.04</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

### Average 3-day Moving Averages Before First Missed Days

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	2503.15	68.33	401.67	4.58	86.02	5.94	3.48	62.94	71.33
3	4683.31	125.00	900.00	7.49	89.32	5.42	14.36	49.50	46.67
4	3649.91	125.00	805.00	6.02	82.99	4.22	13.54	49.22	77.00
5	2092.81	62.50	430.00	6.76	91.52	5.50	2.82	58.00	101.17
7	2250.02	60.00	420.00	5.13	94.30	4.67	1.32	47.67	90.33
9	971.60	43.44	262.78	7.73	92.49	6.78	6.15	63.22	52.89
<b>Group Average</b>	<b>2691.80 ± 530.18</b>	<b>80.71 ± 14.41</b>	<b>536.57 ± 103.64</b>	<b>6.28 ± 0.52</b>	<b>89.44 ± 1.74</b>	<b>5.42 ± 0.37</b>	<b>6.94 ± 2.31</b>	<b>55.09 ± 2.93</b>	<b>72.23 ± 8.58</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

### Average 7-day Moving Averages Before First Missed Days

Subject	Total Yardage (yds)	Total Duration (min)	Total Load (AU)	Total Sleep (hrs)	Sleep Efficiency (%)	Disturbances	Sleep Latency (min)	RHR (bpm)	HRV (ms)
1	3905.22	120.00	662.86	4.42	84.43	6.36	2.93	63.21	69.10
3	4628.83	131.79	977.14	7.01	89.24	4.71	11.51	53.56	68.04
4	2503.41	80.00	522.14	6.33	83.62	5.12	16.91	48.55	72.83
5	4185.86	120.39	779.93	6.38	91.77	5.31	3.54	57.73	104.64
7	2793.59	98.57	728.57	5.31	93.94	4.20	1.88	47.00	87.80
9	2839.98	85.76	505.48	7.71	91.93	6.38	9.81	64.00	52.57
<b>Group Average</b>	<b>3476.15 ± 357.46</b>	<b>106.09 ± 8.58</b>	<b>696.02 ± 71.83</b>	<b>6.20 ± 0.48</b>	<b>89.16 ± 1.74</b>	<b>5.35 ± 0.36</b>	<b>7.77 ± 2.43</b>	<b>55.68 ± 2.95</b>	<b>75.83 ± 7.54</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

**Average Acute:Chronic Ratios Before First Missed Days**

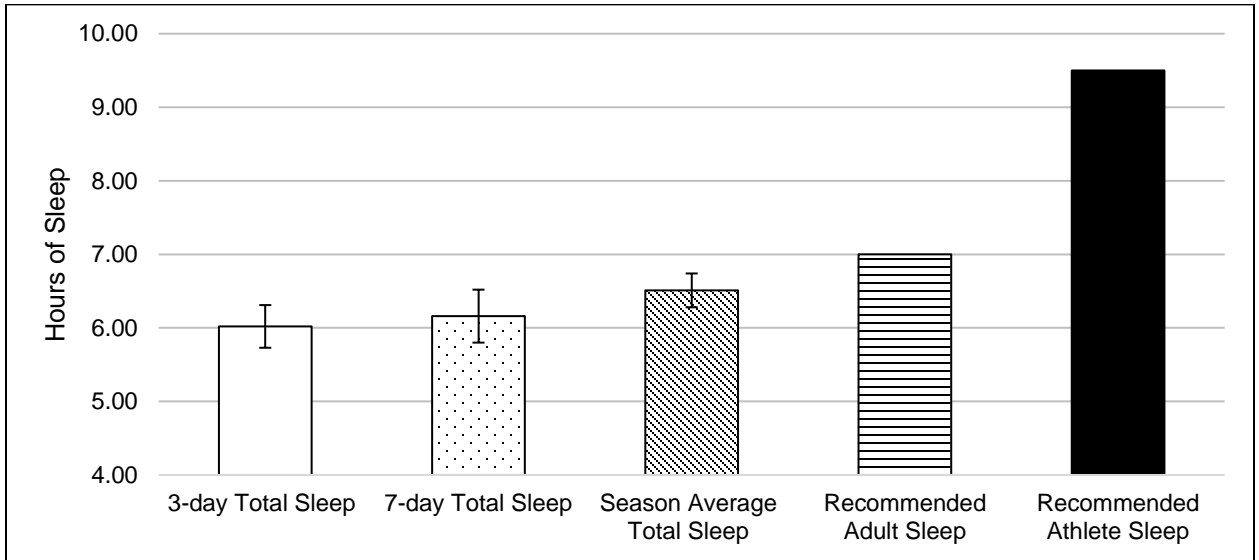
<b>Subject</b>	<b>Total Yardage (yds)</b>	<b>Total Duration (min)</b>	<b>Total Load (AU)</b>	<b>Total Sleep (hrs)</b>	<b>Sleep Efficiency (%)</b>	<b>Disturbances</b>	<b>Sleep Latency (min)</b>	<b>RHR (bpm)</b>	<b>HRV (ms)</b>
1	1.03	1.19	1.21	0.91	0.96	1.15	1.06	1.12	0.85
3	1.01	1.12	1.34	1.00	1.01	0.91	1.09	1.00	1.03
4	0.67	0.74	0.73	0.98	1.00	0.88	0.89	1.01	0.89
5	0.70	0.87	0.89	0.97	0.99	0.97	1.11	0.98	1.05
7	0.89	1.10	1.12	0.88	1.00	0.91	1.50	0.93	1.16
9	1.41	1.44	1.54	1.01	1.03	1.08	0.76	0.98	1.10
<b>Group Average</b>	<b>0.95 ± 0.11</b>	<b>1.08 ± 0.10</b>	<b>1.14 ± 0.12</b>	<b>0.96 ± 0.02</b>	<b>1.00 ± 0.01</b>	<b>0.98 ± 0.05</b>	<b>1.07 ± 0.10</b>	<b>1.00 ± 0.03</b>	<b>1.01 ± 0.05</b>

Mean ± standard error, yds= yards, min= minutes, AU= arbitrary units, hrs= hours, %= percentage, bpm= beats per minute, ms= milliseconds

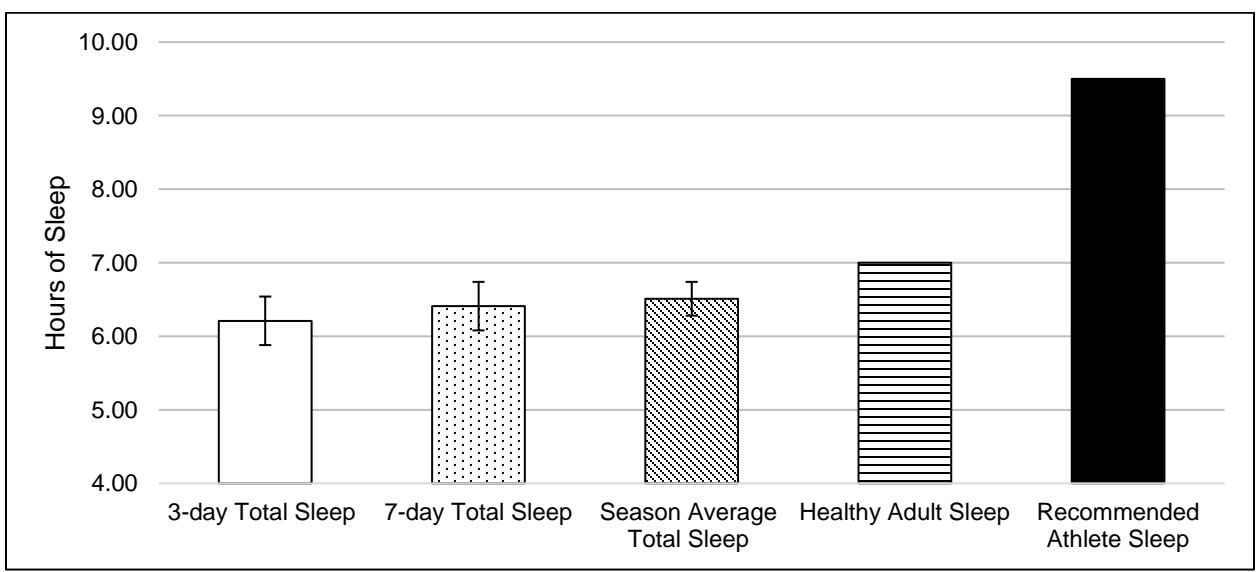
### Appendix H

#### 3-day and 7-day Moving Average Figures

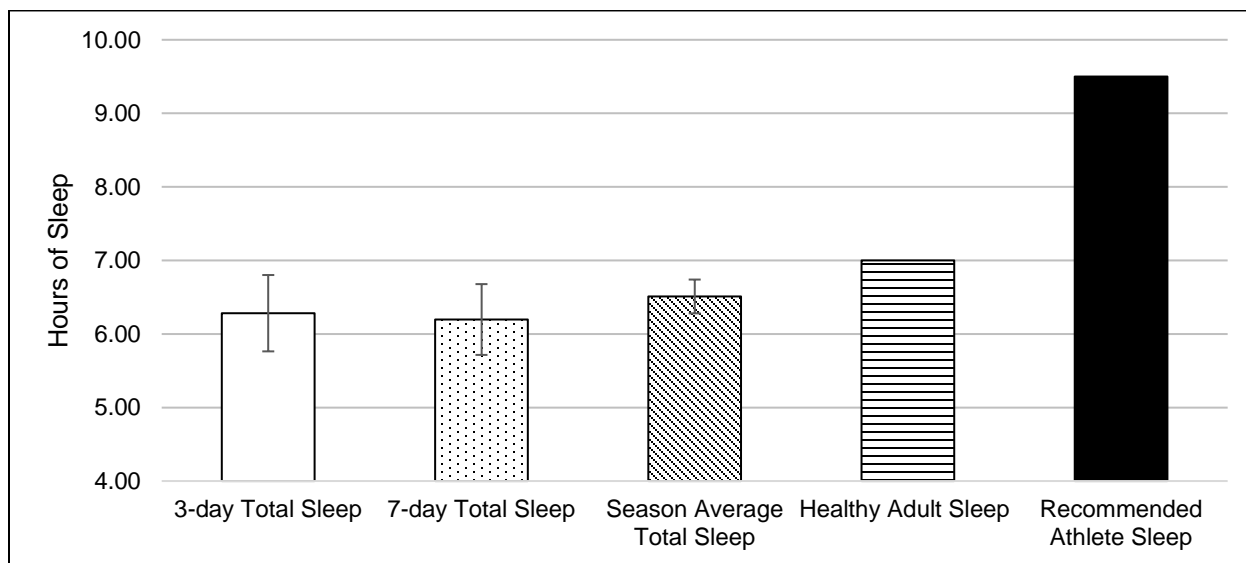
**Figure 3. Total Sleep Data Comparisons Before Injuries**



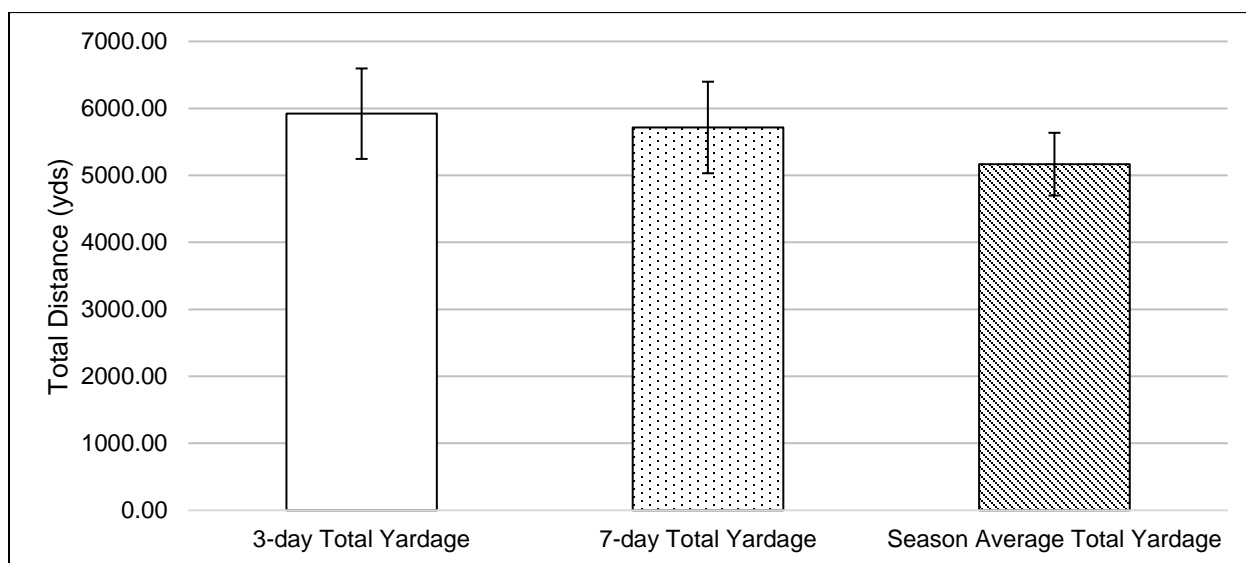
**Figure 4. Total Sleep Data Comparisons Before Illnesses**

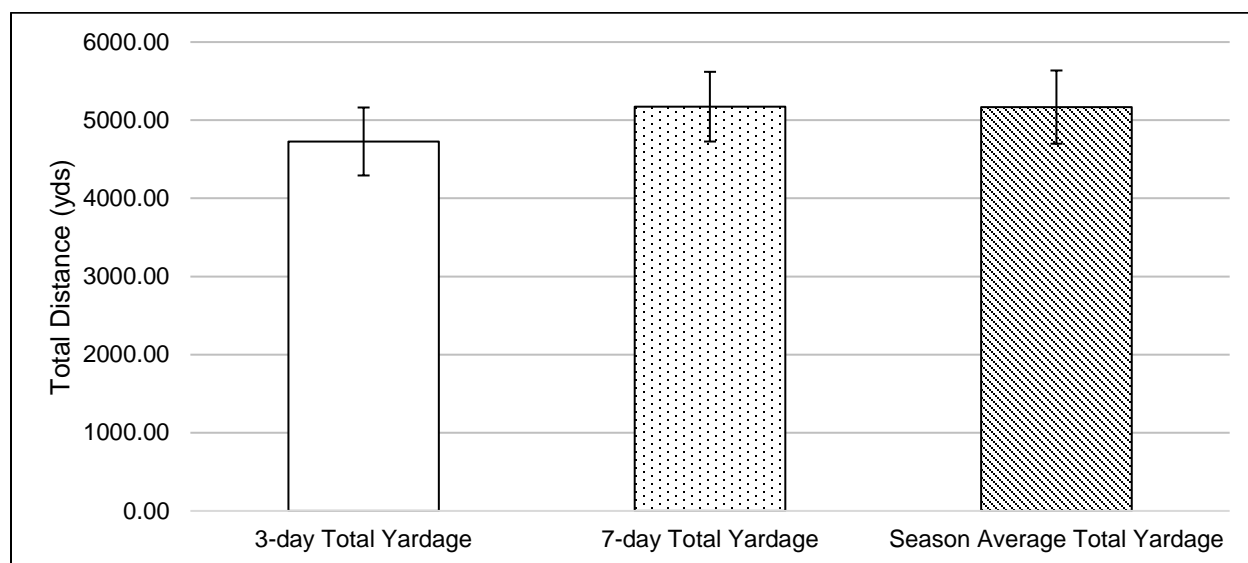
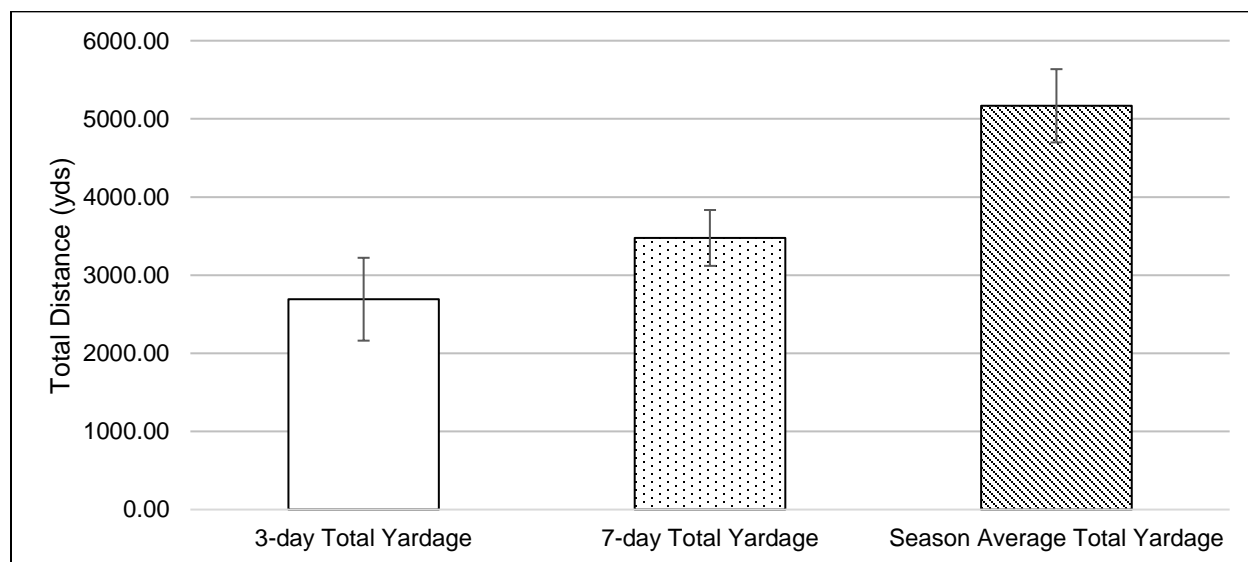


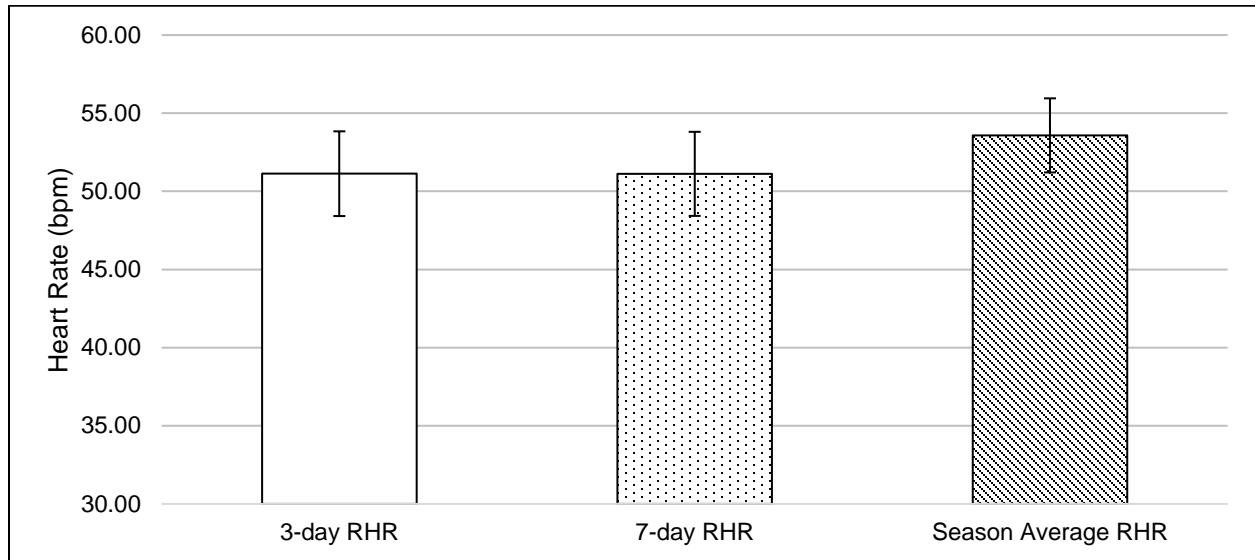
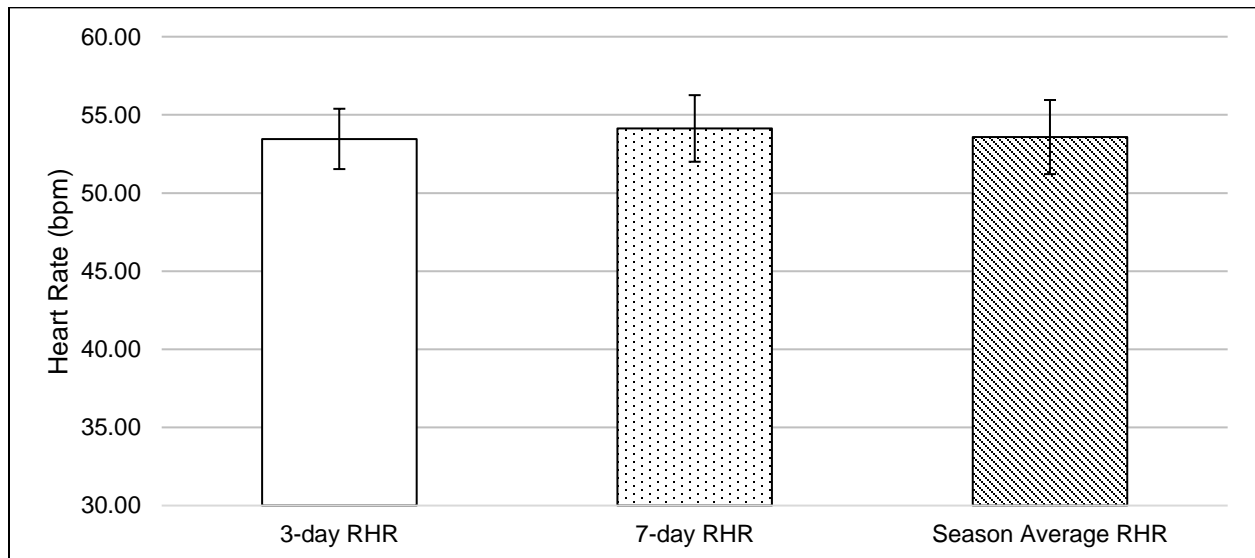
**Figure 5. Total Sleep Data Comparisons Before First Missed Participation Days**

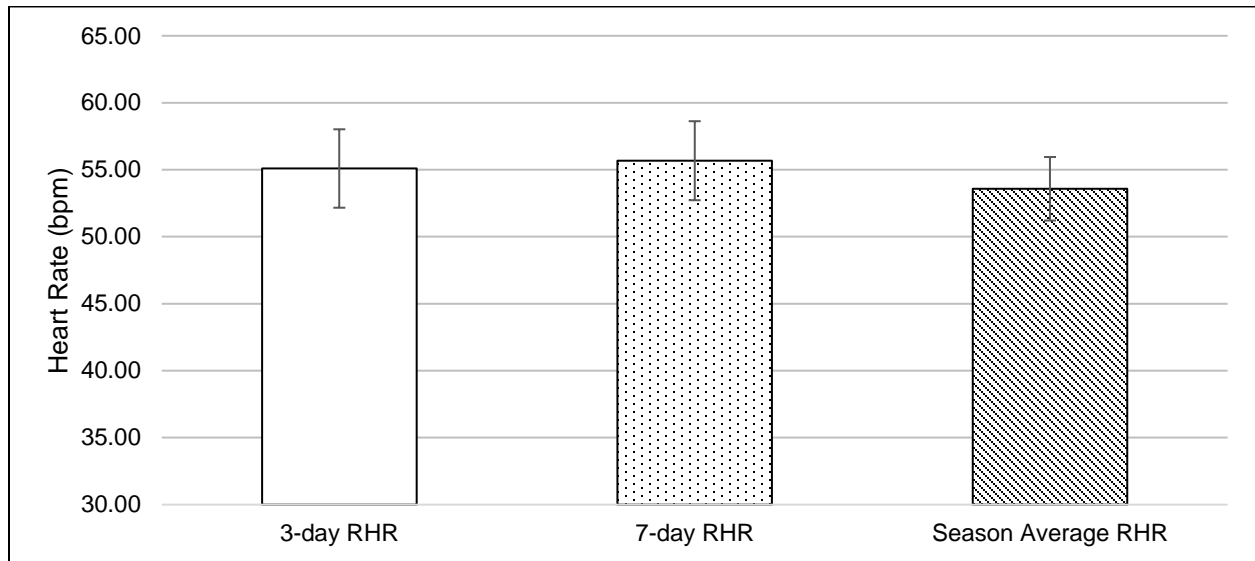


**Figure 6. Total Yardage Data Comparisons Before Injuries**



**Figure 7. Total Yardage Data Comparisons Before Illnesses****Figure 8. Total Yardage Data Comparisons Before First Missed Participation Days**

**Figure 9. RHR Data Comparisons Before Injuries****Figure 10. RHR Data Comparisons Before Illnesses**

**Figure 11.** RHR Data Comparisons Before First Missed Participation Days



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