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# THE EFFECT OF SLEEP QUANTITY AND QUALITY ON DIRECT CURRENT POTENTIAL IN COLLEGIATE AMERICAN FOOTBALL PLAYERS

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# THE EFFECT OF SLEEP QUANTITY AND QUALITY ON DIRECT CURRENT POTENTIAL IN COLLEGIATE AMERICAN FOOTBALL PLAYERS

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DISSERTATION

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A dissertation submitted in partial fulfillment of the
requirement for the degree of Doctor of Philosophy in
the College of Education
at the University of Kentucky

By
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Williamsburg, VA
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ABSTRACT OF DISSERTATION

# THE EFFECT OF SLEEP QUANTITY AND QUALITY ON DIRECT CURRENT POTENTIAL IN COLLEGIATE AMERICAN FOOTBALL PLAYERS

Direct current (DC) potential is an objective measure of the functional state of the human organism. It is a sensitive and accurate indicator of short- and long-term adaptations to stress, adaptive capacities, and it is an important marker of athlete readiness. Sleep is posited to be the most efficacious strategy for improving recovery to enhance sport performance, and adequate sleep is considered vital to normal psycho-physiological function. Thus, optimal sleep may enhance the functional state, in turn enhancing an athlete's adaptability to training stress. However, little is known about the relationship between sleep and DC potential. Therefore, the purpose of this study was to examine the effect of acute (one-night) and extended (two-night) sleep quantity and quality on DC potentials in collegiate American football players. Twenty-four Division 1 American football players (Age: 20.6 ± 1.30 yr; Height: 183.4 ± 6.40 cm; Body mass: 114.40 ± 24.60 kg) wore a wrist-worn actigraphy band seven days per week over the course of 136 days, which spanned the pre-season training camp and competitive season, to measure sleep quantity and quality. DC potential was assessed six days per week using the Omegawave Ltd (Espoo, Finland) athlete monitoring system either 30 minutes upon waking or 75-120 minutes prior to the onset of the football training session. Sleep quantity was stratified into duration categories and sleep quality was stratified within sleep latency, number of awakenings, and sleep efficiency variables. Sleep quantity and quality were evaluated using acute (one night) and extended (rolling average of two consecutive nights) sleep outcomes. Within subject comparisons of DC potential were made across sleep quantity and quality categories using repeated-measures analysis of variance to examine the influence of acute and extended sleep quantity and quality on DC potential outcomes. The level of significance was set at $p \leq 0.025$. Statistically significant main effects were identified for acute sleep ($F_{3,16} = 4.68$, $p < .02$, $\eta_p^2 = 0.47$) and extended sleep durations ($F_{2,17} = 7.71$, $p < 0.005$, $\eta_p^2 = 0.48$). Specifically, for acute sleep durations, there was a 17.1% increase in DC potentials (3.59, $p < 0.01$, Cohen's $d = 0.52$, SE 1.18) for sleep durations ≥7 hours to <9 hours, compared to sleeping <6 hours. For extended sleep, there was a 20% increase in DC potentials (4.53, $p < 0.002$, Cohen's $d = 0.68$, SE = 1.13) when recording a two-day sleep average of ≥7.5 hours and <9 hours, compared to an extended sleep duration of <6 hours. A statistically significant main effect was also identified for extended wake episodes ($F_{2,19} = 4.5$, $p = 0.025$, $\eta_p^2 = 0.32$). For extended sleep periods with >4 wake episodes there was a 12% increase in DC potentials (2.57 ± 2.24mV, $p < 0.25$, Cohen's d = 0.34) compared to extended sleep periods with 2-3 wake episodes. There was not a significant effect of acute ($p \geq 0.20$) sleep quality or extended latency ($p > 0.18$) and efficiency ($p > 0.08$) on DC potentials. These findings suggest that sleep quantity affects DC bio-potentials and thus the functional state of the athlete. Specifically, sleep

durations between 7.00/7.50 to 9 hours correspond with higher measures of DC potentials compared to lesser durations. Given the effect of sleep quantity on biological markers for training adaptability, practitioners should prioritize sleep in the training process and educate athletes on proper sleep hygiene and sleep quantity to enhance their readiness to train.

KEYWORDS: American football, Direct Current potential, Sleep quality, Sleep quantity, Readiness

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Date

# THE EFFECT OF SLEEP QUANTITY AND QUALITY ON DIRECT CURRENT POTENTIAL IN COLLEGIATE AMERICAN FOOTBALL PLAYERS

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# CHAPTER I

## INTRODUCTION

Sleep is an essential biological process (44) that is required for the human organism to grow, adapt (69) and thrive (41, 226). Sleep is also posited to play a critical role in recovery from exercise (182), and it is consistently regarded as the most efficacious strategy for improving recovery to enhance sport performance (80, 107). The literature demonstrates a reciprocal relationship between sleep quantity versus exercise and sport performance (11, 14, 29, 78, 121, 167, 188, 209). However, to date, apart from the National Sleep Foundation's (NSF) general recommendations for sleep quantity and quality, there is a paucity of research regarding optimal sleep parameters among young adults participating in high performance sports.

Sleep plays a critical role in health and normal physiological function (160, 180, 194, 217). The restriction or deprivation of sleep have been shown to negatively affect normal metabolic (33, 194) and endocrine function. Habitually short sleep durations have been implicated in gene down-regulation across several functionally diverse categories including immunity, leukocyte activation and wound healing (227). In addition, sleep loss is considered a risk factor for type 2 diabetes (180), metabolic syndrome (33), and numerous types of cancer (70, 180, 210, 211, 232). Slow wave sleep loss has been implicated in inhibiting protein resynthesis mechanisms which could result in muscle degradation (25).

Consistent and fulfilling sleep is considered to be a critical component to learning and memory consolidation (192). There is a substantial amount of evidence across a broad range of neuroscientific disciplines, complimented by cellular and molecular models of sleep-dependent plasticity, to support sleep-dependent memory processing (22, 50, 224). Sleep deprivation degrades cognitive performance and impairs visual and auditory attention, with visual-motor performance demonstrating increased sensitivity to state instability (109). Tasks requiring divergent problem-solving skills are negatively impacted by sleep deprivation (SD), and these skills are critical to healthy team dynamics and fluid decision making (73, 74, 81).

Research indicates that sleep restriction (SR) and SD can lead to a decrease in endurance, speed, strength and power output as well as sport-specific skill (69, 186). Oliver and colleagues examined the effects of one night of SD on a treadmill endurance test (153). Less distance was covered following one night of SD compared to a separate trial wherein sleep was normalized. Despite the decrease in performance following sleep deprivation, the participants reported a similar perception of effort. This indicates that the altered perception of effort may, in part, account for the decrement in endurance performance following a night without sleep (153). This finding compliments the work of Goel et al. (74), wherein they reported that under conditions of sleep deprivation, “Increased compensatory effort is required to remain behaviorally effective”. Therefore, sleep loss may affect performance despite equivalent applied efforts (186).

Sleep restriction can affect strength (167), endurance (144), sport-specific skill (168) and performance (107). Leger and colleagues (121) examined sleep management and performance during the Tour de France à la Voile yacht race. Analysis revealed that the final rankings of the race were related to sleep management strategies, with the winner achieving a lower total sleep debt (121). Research with dart players (59), handball goalkeepers (105), and tennis players (187) reveal a reduction in sport-specific skill following SR; whereas swim performance, as indicated by lap time, has been shown to be unaffected by SR (69). This is an indication that sports requiring a higher degree of perceptual-cognitive skill may be more affected by sleep restrictions compared to sports relying primarily on gross motor execution (e.g., stroke rate of swimmers). Collegiate American Football requires a unique combination of fine and gross motor skills as well as efficient bioenergetic processes to optimize performance over the duration of three or more hours of competition(42). Therefore, sleep may affect both the cognitive, psycho-motor and physiological processes required in executing the biodynamic and bioenergetic demands of American Football.

The NSF recommends that young adults (18-25 yr) sleep between 7-9 hours per night (86). The NSF has reported that it may be appropriate to sleep as little as 6 hours and as much as 10-11 hours (86). However, less than 6 hours and more than 11 hours is not recommended (86). Unfortunately, the sleep habits of college/university students are declining (83). For instance, Hicks and colleagues (83) noted that 24% of college students indicated dissatisfaction with their sleep in 1978, whereas 71% reported

dissatisfaction in the year 2000. Furthermore, recent literature indicates that 22% of college students report that sleep difficulties impact their academic performance (12).

Ohayon et al. (152) state "Good sleep quality is a well-recognized predictor of physical and mental health, wellness, and overall vitality". Until recently, sleep quality was an ambiguous term that lacked definitional consensus in the scientific community. In 2017, the NSF developed objective sleep quality recommendations for healthy people across the life-span. The NSF dissected sleep into its objective components to define *quality* as "a combination of constituent elements or processes judged as valuable" (152). The NSF determined that four sleep continuity variables (sleep latency, number of awakenings > 5 minutes, wake after sleep onset, and sleep efficiency) were appropriate indicators of good sleep quality. Sleep latency periods ≤ 30 minutes, wake after sleep onset periods ≤ 20 minutes, sleep efficiency ≥ 85%, and having ≤ 1 awakening after sleep onset (>5 mins) were considered appropriate for young adults (18-25 yr) (152).

Despite educational efforts and the accessibility of wearable technology for elite sport communities, the elite athlete population has demonstrated poorer sleep quality markers compared to age and sex matched non-athlete cohorts(120). Juliff, Halson and Peiffer (108) examined the incidence of sleep disturbances prior to important competitions and/or games with a group of 283 elite Australian athletes across a wide variety of sports. The authors found that 64% of the athletes reported sleeping worse than usual in the night(s) prior to a competition or game, with no differences found between sport, gender, or athletes in or out of season (108). Initiating sleep due to

nervousness and thoughts prior to competition was the key complaint amongst the cohort. Potentially even more concerning is that few athletes were aware of sleep strategies that could be utilized to mitigate poor sleep during demanding competitive periods (108).

Poor sleep quality is becoming a prevalent issue in the undergraduate university population. In 2009, Vail-Smith, Felts and Becker (214) reported that amongst 859 university undergraduates, 76.6% of the responders reported occasional sleep problems and 11.8% experience poor sleep quality. Eighteen percent of the students reported a sleep latency period of more than 30 minutes and 28% reported experiencing insomnia in the past 3 months. Finally, the authors reported "Our results indicated that sleep quality (mean SQI scores) were associated with several health risk behaviors including physical aggression, suicide ideation, smoking, alcohol and marijuana use and physical inactivity" (214).

In order to achieve peak performance, athletes engage in an integrated systemic process of athlete preparation termed the *training process* (64). The training process is designed to induce automation of motor skills and develop structural and metabolic functions that lead to an increase in physical performance (190). The training process is an adaptation-dependent process, wherein training loads are applied to an athlete's organism with the intent of inducing an adaptive response that improves athlete preparedness. Preparedness is a "multi-faceted cumulative state" that encompasses the developmental state of the athlete's sports specific-skill, tactical, technical, mental and intellectual factors. There is a physiological cost associated with adaptation which

affects the athlete's state of physiological and psychological readiness to train and adapt to subsequent training loads. The state of readiness is the result of both intrinsic and extrinsic factors, both training and non-training loads (64). By considering the state of readiness, training programs can be designed and modified to target specific regimes of works that can optimize the adaptive process. Morris states "Readiness may be defined as the current functional state of an individual that determines their ability to achieve their performance potential" (141).

The functional state of the human organism can be objectively measured by assessing Direct Current (DC) potentials of the brain. DC potential has been used for over 70 years in the fields of medicine (94, 114), neurophysiology (18), psychophysiology (198) and sports physical preparation (101, 141) for a wide range of scientific applications (96). DC potentials have been demonstrated to be "a physiological marker of integrations of neurohumoral and biochemical interactions, with the central and autonomic systems playing the leading role"(95). DC potentials can be used to evaluate short (102, 145) and long-term (183) adaptations to stress , the functional state (111), and adaptive capacities (96) of the human organism. DC potentials are most frequently collected using the omegametry method which utilizes a discrete and continuous recording of brain biopotentials within the frequency range of 0.00 – 0.50Hz (96). The level of cerebral DC potential (LDCP), as collected using the vertex-thenar method, is a stable potential measured in millivolts which is slow changing and is one of the constant physiological processes of the brain (75). The work of Sychev has demonstrated that the assessment of DC potentials using the vertex-

thenar method permits the "quick diagnosis of the functional state of athletes, optimization of the training process, and enhances performance in sports" (96).

DC potential has been successfully utilized in sport preparation to optimize training processes and outcomes. Research from training centers in the former Soviet Union (e.g., Krasnodar, Bryansk, Pyatigorsk), demonstrate that training adaptations for athletes are most effective when DC potentials are within a range of 20 – 40mV (101). In addition, athlete monitoring parameters were set so that training ceased if DC potentials decreased by 25% or more from baseline during the training process. Conversely, training loads would be continually increased as long as DC potentials did not rise by more than 50% from baseline (101). Recently, Morris demonstrated that objective measures of athlete readiness could be used to guide a fluid periodization model in the physical preparation of collegiate American football players (141). Using objective assessments of athlete readiness, including DC potential, resulted in improvement in athletic performance outcomes concomitant to a reduction in physiological cost to the athletes (141).

Researchers have attempted to summarize the pscyho-physiological significance of DC potential in the assessment of the healthy human's adaptation and compensatory-adaptive abilities to physical loads in sport. For example, DC potentials ranging from 0 – 19 mV are considered to be low and represent decreased arousal, rapid onset of physiological and psychological exhaustion, diminished adaptive-capacity, and low functional reserve. DC potential from 20 – 40 mV are considered to represent an optimal arousal, state of high adaptability to stress, optimal state for learning new

habits, and high functional reserve. Finally, DC potentials ≥41 mV represent a high level of physiological and emotional tension, high psycho-emotional stress and limited adaptability (96, 101, 141). In brief, DC potential can be used to assess the functional state of athletes under the influence of mental and physical loads (101), and as an indicator of acute (102, 145) and long-term adaptive processes (183).

Despite the evidence that sleep affects physiological, psychological, and performance parameters, there is a paucity of research that has explored the relationship between sleep and DC potential. Evaluating this relationship will shed light onto the magnitude of the role sleep plays in determining athlete's readiness to train. Therefore, the purpose of this study was to examine the effect of acute (one-night) and extended (two-night) sleep outcomes on DC potentials. Based on the NSF's sleep quantity and quality recommendations, it was hypothesized that moderate time frames of acute and extended sleep quantity (7-9 hours) would produce greater DC potentials. In addition, it was hypothesized that NSF recommended appropriate ranges for sleep latency, number of wake episodes after sleep onset (>5 mins) and sleep efficiency would produce greater DC potentials.

## Assumptions

Assumptions of this study include the following:

1. It was assumed that all subjects wore their assigned Readiband device as instructed prior to the initiation of the study.

2. It was assumed that the five subjects performing the Omegawave assessment on their own, upon waking, performed that assessment as instructed.

## Delimitations

This study was delimited to the following:

1. Male Division 1 collegiate American football players attending the University of Kentucky between the ages of 18 to 23 years.
2. Male Division 1 collegiate American football players who were cleared for full activity without restriction at the time of the study.
3. Data were only collected during the pre-season training and competitive season.

## Definitions

Sleep – A homeostatically regulated state of reduced movement and sensory responsiveness.

Sleep Restriction – A reduction in sleep quantity that includes deviation from normal sleep onset or wake patterns.

Sleep Deprivation – Long or extreme durations of sleep loss.

Direct Current Potential – Brain biopotential within the frequency range of 0.00 – 0.05 Hz, that can be used to evaluate short and long-term adaptations to stress, the functional state and adaptive capacities of the human organism.

Functional State – A highly sensitive and accurate physiological indicator, which objectively describes individual short- and long-term adaptations to psycho-physiological stressors.

Readiness – The current functional state of an individual that determines their ability to achieve their performance potential.

# CHAPTER II

# REVIEW OF LITERATURE

## Literature Review of Sleep and Its Impact on Athletic Performance

### Introduction

As the English dramatist Thomas Dekker wrote "Sleep is the golden chain that ties health and our bodies together" (87). Sleep is an essential biological process (44) that is required for the human organism to grow, adapt (69), and thrive (41, 226). It is considered a necessity for both cognitive and physiological function (69), and its relationship to post-exercise recovery and performance in athletes has made sleep a topic of great interest in the sports science community (174).

From a behavioral context, sleep can be defined as "a homeostatically regulated state of reduced movement and sensory responsiveness" (7). The sleep state, occurring at habitual intervals during a 24-hour period, is marked by reduced motor activity, fluctuations in body temperature, hormonal activity, eye movement, muscle tone, electroencephalographic (EEG) oscillations (200) and regional brain activity (69). Despite our understanding of the order and synchronicity of sleep, our knowledge of the purpose and regulation of sleep is incomplete, as a single locus of control has yet to be identified (44). Research has yet to pinpoint the exact regulatory mechanism for the sleep-wake cycles, as the body of literature is primarily composed of mechanistic studies conducted on phylogenetically diverse populations. Also, most sleep studies are descriptive in nature, investigating pharmacological interventions and their effects on

neurochemicals, or lesion studies seeking to identify anatomical landmarks that affect sleep (44). None of these studies have identified an anatomical landmark that when lesioned can eliminate sleep. Therefore, considering the ambiguous nature of the biological drivers underpinning sleep, sleep should be viewed as a "broad system-wide phenomenon" without a locus of complete control (44, 148).

Peak sports performance is a compilation of physical, mental, technical, tactical and intellectual components working in harmony to accomplish a motor task. These components are affected by fluctuations in behavioral and physiological processes across a 24-hour period in a rhythmic pattern. These rhythms are generated by circadian clocks, which are considered to be the internal time-keeping machinery of biological systems (148). Circadian clocks are dependent upon cyclic environmental cues called zeitgebers, which is German for time givers. Zeitgebers such as light, temperature, and humidity can alter or reset the timing or phase of circadian rhythmicity (115). Zeitgebers prevent "free running" of endogenous rhythms by entraining them to a 24-hour rhythm (212).

Any process that repeats itself every 24-hours and persists in the absence of external time cues is considered to be a circadian rhythm (164). In mammals, circadian rhythms are controlled by the suprachiasmatic nucleus (SCN) which is found in the anterior hypothalamus (17, 139, 199). The SCN exerts limited control over the sleep-wake cycle, as humans are highly sensitive to changes in their environment.

The circadian process, operating independent of prior sleep and waking, is one of three distinct processes that underlie sleep regulation. The homeostatic process is dependent upon sleep and waking and operates to normalize deviations from average levels of sleep (115). Homeostatic processes increase the drive for sleep in the absence or delay of sleep onset and reduce the drive for sleep in response to excess sleep. In addition to the circadian and homeostatic processes, an ultradian process (shorter than a day but longer than an hour) occurs within sleep and is composed of the alternation of two sleep states: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep (27).

## Non-Rapid Eye Movement and Rapid Eye Movement Sleep States

The normal pattern of sleep in a healthy young adult on a fixed sleep routine consists of approximately 90 to 110-minute cycles of sleep divided into periods of REM and NREM sleep(39). NREM sleep is conventionally subdivided into four stages: stages 1, 2, 3, and 4, and as these stages progress numerically, the depth-of-sleep and arousal thresholds increase. The EEG pattern of NREM sleep is considered to be synchronized, with ubiquitous waveforms such as K-complexes and spindles which are signs progression into stable sleep (34, 46, 157). Stages 3 and 4 are often combined in the literature and are referred to as slow wave sleep (SWS), delta sleep, or deep sleep. SWS is marked by EEG patterns of large amplitude and low frequency waves, and the arousal threshold is the highest of the NREM sleep stages (69, 181, 200). Because NREM sleep is

marked by reduced or fragmented brain activity, the state of NREM sleep can be summed up as a "relatively inactive yet actively regulating brain in a movable body" (157).

NREM sleep is proposed to aid in tissue repair (140), assist in energy conservation (178) and nervous system recuperation (69). Adequate NREM sleep is crucial for athletes as it is during this period that growth hormone (GH) is released from the pituitary gland (76, 140, 176, 206). GH is an anabolic hormone that aids in muscle growth (45), bone building (104), and mobilization of free fatty acids (40). GH is released in a pulsatile manner throughout a 24-hour cycle with the largest pulse occurring in temporal association with the first SWS episode (28, 193). The intensity of SWS, which is quantified through slow-wave activity (SWA) (i.e., the spectral power of the EEG in the 0.50 to 3.50 Hz frequency range), is correlated to the amount of GH secreted during the first SWS episode (76, 193). Research indicates that total sleep deprivation drastically reduces nocturnal GH secretion (28). Thus, for the training athlete, adequate and normalized sleep patterns may play a critical role in restoration from training loads.

In contrast to NREM sleep, REM sleep is marked by low-amplitude high frequency EEG waves with episodic bursts of rapid eye movements and muscle atonia (157).

Davenne states: "During REM sleep, the brain is partly disconnected from the body due to a blocking of cortico-spinal pathways at the brain stem, motor

activity is suppressed, and all muscles are in a state of total relaxation which allows effective myofibril restoration. (46)"

The proposed role for REM sleep is memory consolidation (200), localized recuperative processes and emotional regulation (185). Dreaming is associated with REM sleep based on vivid dream recall following arousal after REM sleep periods (132). Although the function of dreams has yet to be fully elucidated, it has been proposed that dreams "represent the conscious awareness of complex brain systems involved in the reprocessing of emotions and memories during sleep" (201). The reprocessing of emotions and memory are theorized to aid in sleep-dependent memory reprocessing.

Sleep is initiated through NREM stage 1 and progresses through stages 2, 3, and 4 before entering into REM sleep. During the first sleep cycle, REM sleep is brief (under 10 minutes), with SWS accounting for 20 to 40 minutes of the first cycle (157). During the first third of the night, SWS dominates the NREM portion of sleep, but as the evening progresses stage 2 sleep takes the leading role in NREM sleep. Conversely, REM sleep duration increases throughout the night, wherein the longest REM sleep episodes occur in the last third of the night. The REM sleep distribution pattern, which is heavily weighted toward the latter portion of the night, is linked to the circadian nadir of core body temperature. Also, neurophysiological research indicates that clock gene expression within the dorsomedial SCN is associated with REM sleep circadian regulation (119).

## Sleep Regulation

The timing of normative sleep is consistently concentrated to a specific portion of the 24-hour day. Nocturnal sleep duration is largely under volitional control (e.g., early wakening by alarm, or staying up late) by humans; however, circadian modifiers also play a significant role in sleep duration. The two-process model of sleep regulation is a quantitative model which describes the alternation of human sleep and wakefulness as the interaction of homeostatic and circadian processes (17, 26, 212, 216). The model consists of a circadian process (process C) and a homeostatic process (process S) which together determine sleep onset and wakening (215).

Process S represents the drive or need for sleep that accumulates during wakefulness, and can be felt when sleep is extended past habitual bedtimes (215). Process S increases by a saturating exponential function during waking hours and depletes by a declining exponential function upon sleep onset (17). Process C is controlled by the SCN and represents the "daily oscillatory component in the drive for sleep and wakefulness" (215). Process C is proposed to set limits on process S, but the limits are not constant, rather they vary with the time of day (17). As the homeostatic drive for sleep reaches its lower limits during sleep, wakefulness is initiated. Conversely, as Process S reaches its upper limit during wakefulness, sleep is initiated.

Slow-wave activity during NREM sleep behaves as predicted for a measure representing the rise of process S during wakening and decline during sleep (17). In a sleep deprivation study conducted by Finelli et al. (63) theta band power increased

during waking EEG. This increase in theta activity was correlated to an increase in SWA during the first NREM sleep episode of recovery sleep. This relationship of increased theta activity during wakening and SWA activity during sleep supports the two-process model of sleep regulation (115).

The regulation of sleep by homeostatic and circadian processes has a much broader scope of influence than just initiating sleep or wakefulness. Van Dongen and Dinges state "The biological clock also modulates our hour-to-hour waking behavior, as reflected in fatigue, alertness, and performance, generating circadian rhythmicity in almost all neurobehavioral variables" (215). Research indicates that there may be a circadian influence on athletic performance. According to Drust et al. (56), an examination of world record-breaking performances reveals a circadian variation with record-breaking performances occurring in the early evening, which coincides with a peak in body temperature (56). Thun et al. (212) state that: "Performance is better in the afternoon or evening than in the morning for nearly all kinds of sports demanding physical skills. Technical performance may peak some-what earlier in the day than skills demanding more power". Circadian influence on performance has yet to be fully elucidated, but the current literature suggests that it should be considered in preparation for individual and team performance.

Physiological and Epidemiological Implications

The influence of sleep on the function of various physiological systems has yet to be fully elucidated, but there is mounting evidence that sleep plays a critical role in many fundamental biological processes from immune (227) and endocrine function (194) and may play a critical role in health and wellness (160, 180). Recently, Watson and colleagues (227) demonstrated that within healthy monozygotic twin pairs habitual short sleep duration was associated with differential gene expression and pathway enrichment. Habitual short sleep duration was linked to gene down-regulation "…across functionally diverse categories including immunity, wound healing, cell adhesion, chemotaxis, chemokine binding, and leukocyte activation" (227). Also, some of the down-regulated genes were highly enriched in immune-inflammatory pathways. This study indicates that when genetic factors are controlled for, habitual short sleep durations are associated with adverse immune responses that may be related to negative metabolic, cardiovascular and inflammatory outcomes (227).

Sleep loss is considered a risk factor for numerous types of cancer. According to research presented at the American Association for Cancer Research (AACR), sleeping only 3 to 5 hours per night significantly increases the risk of dying of prostate cancer by 55% for men younger than 65 years of age (70). Short sleep durations have also been associated with an increased risk for breast and colorectal cancer (210, 211). In 2007, the International Agency for Research on Cancer, a part of the World Health Organization, categorized "shift work with circadian disruption or chronodisruption as a probable human carcinogen" (61). Shift work with circadian or chronodisruption was

classified as a group 2A carcinogen, which places it in the same risk class as ultraviolet radiation, benzo(a)pyrene, and acrylamide (61). Natural killer (NK) cells are considered to be critical effectors in cancer immunosurveillance, as they spontaneously kill cells deemed dangerous to the host body (77). Wessel et al. (217) found that 5 nights of sleep restriction (SR) (4 hours in bed per sleep episode) led to a 65% reduction in the amount of NK cell concentration (217). Also, moderate sleep restriction of one night has been demonstrated to significantly reduce NK cell immune responses (103).

Sleep restriction, a reduction in sleep quantity that includes deviations from normal sleep onset or wake patterns, can impact normal metabolic and endocrine function. Spiegel et al. (194), investigated carbohydrate metabolism, thryrotropic function, activity of the hypothalamic-pituitary-adrenal (HPA) axis and sympathovagal balance following six nights of SR. Compared to measurements taken at the end of a six-night sleep recovery period (12 hours in bed), SR resulted in significant reductions in glucose tolerance and thyrotropin concentrations as well as an increase in evening cortisol and activation of the sympathetic nervous system. Glucose and insulin responses were consistent with a definite impairment of carbohydrate tolerance during the sleep-debt period. Furthermore, the clearance of glucose after injection was approximately 40% slower in the sleep-debt period compared to the sleep-recovery period (194). This study demonstrates that six-nights of SR has a negative impact on heart rate variability, carbohydrate metabolism, and endocrine function.

Current epidemiological studies indicate that the development of obesity and type 2 diabetes is multifactorial, and that novel risk factors including sleep duration and

disruption of circadian rhythms should be considered alongside traditional risk factors (170). In a recent meta-analysis, Shan et al. (180) investigated the dose-response relationship between sleep quantity and the risk of type 2 diabetes (180). A U-shaped dose-response relationship was observed, with 7-8 hours of sleep being the lowest risk category. In addition, other current meta-analyses indicate a U-shaped relationship between sleep quantity and hypertension, obesity, cardiovascular outcomes (including coronary heart disease, stroke, and total cardiovascular disease) and all-cause mortality (35-37, 225). Finally, in a meta-analysis conducted by Xi and colleagues (232) short sleep duration (≤ 6 hr) was significantly associated with an increased risk for metabolic syndrome.

Sleep duration has been demonstrated to affect concentrations of the anorexigenic hormone leptin (inhibits the feelings of hunger) and the orexigenic hormone ghrelin (stimulated feelings of hunger) which regulate appetite and caloric intake (33). Robertson et al. (170) reported that three weeks of mild SR (habitual bedtime minus 1.50 hours) led to significant reductions in plasma concentrations of leptin, and body weight. In a study conducted by Calvin et al. (33) SR of two-thirds of normal sleep time for eight days/eight nights in a hospital-based clinical research unit resulted in an increase in caloric intake without a concomitant increase in energy expenditure. Finally, in a study by Spiegel and colleagues (195) , two days of SR (4 hours in bed) resulted in a reduction in leptin and elevations in ghrelin along with an increased perception of hunger and appetite.

In 2017, the findings of the National Diet and Nutrition Survey Rolling Programme (NDNS-RP) in the United Kingdom (UK) revealed that longer sleep durations were associated with a lower body mass index (BMI) and favorable metabolic profiles in UK adults (160). The authors used four years of data to investigate if sleep was associated with metabolic syndrome, glucose and lipid metabolism, adiposity, diet and inflammation with 500 adults aged 19 years and over (160). Subjective measures of sleep were obtained through computer-assisted interview questions and participants completed 3 to 4 food diaries on consecutive days. Blood pressure, height, weight, and waist circumference, as well as 35ml of fasted blood, were collected by nurses in the participants' homes. Statistical analysis revealed a positive association between sleep duration and high-density lipo-protein (HDL) cholesterol and a negative association for sleep duration with BMI and waist circumference. These findings support the growing body of research indicating the contribution of short sleep durations to metabolic diseases (160).

It is well documented that sleep exerts significant modulatory control over numerous components of the endocrine system, including sex steroid hormone concentrations (10). Vingren et al. (221) state that "Testosterone is one of the most potent naturally secreted androgenic-anabolic hormones, and its biological effects include promotion of secondary male-sex characteristics…nitrogen retention and muscle growth". Testosterone acts as an anabolic agent in the muscle, stimulating protein synthesis and inhibiting protein degradation. Thus, exercise-induced endogenous testosterone levels act as a critical modulator of muscle hypertrophy (72, 221).

Therefore, it is may be crucial to the training athlete that normative testosterone levels are maintained to prevent maladaptation and to improve physical training outcomes.

Testosterone levels display both circadian and ultradian rhythms. Testosterone increases at the onset of sleep, and in young men peaks during the first REM sleep episode and reaches a nadir in the late afternoon (231). The ultradian rhythm of testosterone is displayed in plasma concentration oscillations that occur every 90 min. These oscillations reflect the underlying pulsatile rhythm of luteinizing hormone secretion (231). In a series of studies conducted by Luboshitzky, it was determined that testosterone levels begin to rise at sleep onset and peak and then plateau at the first REM sleep episode (124, 125). Also, when total fragmentation of sleep architecture occurs, testosterone does not follow its normal circadian pattern, and testosterone levels do not rise (125). This research suggests that there is a relationship between REM sleep and testosterone secretion in young men.

According to Wittert (231): "While studies confirm the effect of total sleep deprivation to lower testosterone, data on the effect of sleep restriction on the hypothalamic-pituitary-gonadal axis remains contradictory". Reynolds et al. (169) determined that five nights of sleep restriction to four hours in bed did not change total testosterone in healthy young men. However, Leproult and Cauter (122) demonstrated that eight nights of SR to five hours in bed resulted in a 10% to 15% reduction in daytime testosterone levels in heathy young men. Research by Schmid et al. (177) indicates that the timing of sleep may be the most important factor in testosterone modulation instead of total sleep duration. When sleep was restricted to 4.5 hours, morning

testosterone levels were reduced, but only when sleep was permitted during the first half of the night (22:30 – 03:30 h) compared to the second half of the night (02:45 – 07:00) (177). The timing of SR may explain the differences in the results of the studies conducted by Reynolds et al. (169) and Leproult and Cauter (122). In Reynolds' et al. study sleep was restricted during the first half of the night (04:00 – 08:00) and in Leproult and Cauter's study sleep was restricted during the morning hours (00:30 – 05:30). Additional investigations into the connection between sleep restriction and testosterone are needed to fully understand the circumstances that surround sleep-dependent androgen suppression.

## Brain Health

Concerns for brain health and the long-term effects of repeated collisions in American football have led to considerable changes in the rules of play for collegiate and professional football (146, 147). Considerable research has been conducted to understand how repetitive head impacts (RHI), cumulative exposure to concussive and subconcussive events(138), are related to both acute (208) and chronic neurological consequences (52, 138, 154, 208). According to Montenigro et al. (138) repetitive subconcussive blows, measured by helmet accelerometer sensors, are associated with "…pre- to post-season cognitive decline, functional brain alterations (e.g., reduced neurophysiological health), and micro structural white matter brain changes in high school football players". Although the research is limited, cumulative RHI are thought to

be key contributors to neurodegenerative disease and chronic traumatic encephalopathy (CTE) (138).

In 2015 it was estimated that 47 million people suffered from dementia and that this number is expected to triple by 2050 (173). Alzheimer's disease (AD), the primary cause of dementia is a growing health crisis. Sleep is an important physiological process for the functional recovery of the brain, as numerous studies indicate that sleep disorders are significant risk factors for neurodegenerative diseases such as Alzheimer's disease (AD) (228, 233). Amyloid-β (Aβ), α-synuclein and tau are proteins present in the interstitial space surrounding the cells of the brain and are linked to neurodegenerative diseases. Amyloid-β plaque deposition in the brain is an early and necessary step in the AD pathogenesis (106, 228). Excessive accumulation of Aβ has been hypothesized to result from an inability to clear toxic Aβ (228).

Until recently, it was thought that unlike peripheral tissues that use lymphatic vessels to usher away interstitial proteins for degradation in the liver, the brain lacked a conventional lymphatic system (233). However, recently Iliff and colleagues (90, 91) discovered a brain-wide paravascular pathway which facilitates the clearance of solutes and waste from the brain by allowing efficient cerebrospinal fluid (CSF) and interstitial fluid (ISF) exchange. This system was termed the "glymphatic" pathway because of its reliance on glial water flux and its similarity to peripheral lymphatic function in interstitial solute clearance (91). Shortly after Iliff et al. (90) used dynamic contrast MRI to confirm key features of the glymphatic pathway, including "para-arterial CSF influx and molecular size-dependent CSF-ISF exchange", Xie et al. (233) revealed that sleep

was associated with a 60% increase in the interstitial space in live mice. This increase in interstitial space allowed for increased exchange in CSF with ISF resulting in increased Aβ clearance (233).

Research has demonstrated that total SD increases Aβ, however, until recently studies have not examined which part of sleep modulates Aβ (106, 155, 228). Ju et al. (106) studied the effects of SWA disruption and the corresponding changes in cerebrospinal concentrations of Aβ, tau, total protein, YKL-40, and hypocretin with healthy young adults (age 35-65). Slow wave activity disruption, not sleep efficiency or sleep duration, was strongly and significantly correlated with increased Aβ. Also, as SWA disruption increased there was a corresponding increase in Aβ. Finally, multiple days of poor sleep efficiency increased tau concentrations. This study indicates that SWA disruption and poor sleep quality modifies neuronally-derived proteins, and these modulations are specific to the type of sleep disruption (106).

## Memory and Learning

It has long been speculated that consistent and fulfilling sleep is a critical component to learning and memory consolidation (192). There now exists a comprehensive body of research from a wide range of neuroscientific disciplines (224), which are complemented by cellular and molecular models of sleep-dependent plasticity (22), to support sleep-dependent memory processing (50). Robert Stickgold describes memory consolidation as a long-term process following initial memory

encoding that includes “a series of cellular, molecular and systems-level alterations” (200). These alterations develop over time and stabilize and enhance the initial memory, outside our conscious awareness and without additional practice. Sleep dependent memory consolidation implies that for most tasks, consolidation is most significant during sleep (off-line) compared to wake (on-line) (192).

“Memory” is often used as a unitary term, implying that the acquisition and storage of memory occur in a linear process (192). Human memory is not a unitary phenomenon. Instead, people can learn, store, and recall different types of information from both conscious acquisition and unconscious environmental awareness (157). Also, acquisition and assimilation occur over variable periods of time with memories considered to belong to multiple specialized memory subdomains that interact in ways that have yet to be elucidated (157).

There is a substantial amount of cellular and molecular evidence to support the role of sleep in promoting brain plasticity (66). In addition, there is a growing body of research regarding the cellular mechanisms of synaptic plasticity that may bring clarity to the neuronal activity of sleep states with changes in synaptic pathways in the brain (66). Sleep has been demonstrated to up-regulate cortical mRNA transcription of calcineurin and camKIV, two genes that are considered important for Long-term Depression (LTD). LTD and Long-term Potentiation (LTP) refer to “use-dependent, persistent alterations in synaptic weights that strengthen (LTP) or weaken (LTD) specific synapses” (66). Research demonstrates that sleep deprivation (long or extreme durations of sleep loss) inhibits the induction or maintenance of LTP, and in some cases,

decreases hippocampal neurogenesis and proteins associated with synaptic remodeling (66).

According to the synaptic homeostasis hypothesis (SHY) “sleep is the price the brain pays for plasticity” (213). Tononi and Cirelli state “SHY proposes that the fundamental function of sleep is the restoration of synaptic homeostasis, which is challenged by synaptic strengthening triggered by learning during wake and synaptogenesis during development” (213). Without homeostatic regulation, synaptic potentiation and depression would lead to oversaturation or obliteration of neural signaling and memory traces. In addition, the SHY proposes that “synaptic normalization by net weakening should occur during sleep” (50). During sleep, the brain goes offline which allows for spontaneous brain activity. This offline state allows neurons to sample the environment comprehensively, free from direct environmental stimulation. In brief, the SHY proposes that new learning occurs from synaptic potentiation, synaptic potentiation occurs primarily in a wakeful state, and synaptic renormalization occurs during sleep (213).

Recently, de Vivo et al. (50) confirmed that synaptic scaling occurs during sleep by measuring 6920 synapses in mouse motor and sensory cortices. Using three-dimensional electron microscopy, it was revealed that after sleep the area of the axon-spine interface (ASI) decreased by 18.9% compared to a state of wakefulness (50). These changes occurred both in the motor and sensory cortices, and the changes in size were proportional to the ASI size, which is indicative of scaling. Finally, the scaling of synaptic size was not uniform, as scaling only occurred in approximately 80% of the

synapses. This is consistent with the SHY in that "selective renormalization during sleep favors memory consolidation, integration, and 'smart' forgetting" (213). This study underscores the critical function that sleep serves in the dynamic process of brain plasticity.

Long-term memories are most commonly divided into declarative and non-declarative memories. Declarative memory is comprised of consciously accessible memories that can easily be described with verbal descriptions (i.e., knowing "what"), with encoding and retrieval being executed explicitly (197). Declarative memories are further subdivided into episodic (memories of one's past with spatial and temporal memory features) and semantic memory (memories of general knowledge without spatial and temporal context) (157, 192).

Nondeclarative memories are those that are acquired unconsciously and are not easily expressed through verbal description (157, 192, 197). Whereas declarative memory provides a means to model the external world into categories of true or false, nondeclarative memory is neither true nor false. Squire expounds upon this concept and states that "It is dispositional and is expressed through performance rather than recollection. Nondeclarative forms of memory occur as modifications within specialized performance systems. The memories are revealed through reactivation of systems within which the learning originally occurred" (196). In contrast to declarative memory, nondeclarative memories are gradually extracted from a series of separate events and are used to build upon current memory models (196). Categories of nondeclarative

memory include the learning of habits and motor skills as well as priming and conditioning (157).

Sleep deprivation studies have revealed that all stages of human sleep may be actively involved in learning and memory consolidation (158). Recent animal and human studies have shed light on how SWS sleep plays a critical role in declarative memory consolidation through covert reactivation of individual memory episodes (68). During SWS, following learning, neuronal reactivation of individual memory episodes is consolidated in the hippocampus and are thus considered to be hippocampus-dependent. According to Fuentemilla et al. (68), "SWS contributes to the facilitating effect of sleep on memory consolidation, presumably by promoting the gradual redistribution of memory traces from the hippocampal to neocortical brain regions for long-term storage".

In a series of experiments, Rasch et al. (162) used an odor (scent of roses) to reactivate memories during human sleep following a visuospatial learning task and a procedural memory task. One group of volunteers performed the learning sessions in the presence of the odor during the evening before sleep with the experimental odor re-presented during the first two periods of subsequent SWS. A second group performed the same learning tasks in the presence of the odor with re-presentation occurring during REM sleep and wake. Follow-up testing revealed an improvement in hippocampus-dependent declarative memories but not of hippocampus-independent procedural memories following odor re-exposure during SWS but not for REM or wake conditions. In addition, functional magnetic resonance imaging revealed "significant

hippocampal activation in response to odor re-exposure during SWS", supporting the specific role of SWS in declarative memory consolidation (162, 192).

Increases in slow oscillation activity and sleep spindle count during post-learning SWS following declarative memory learning tasks (word pair associates) have been shown to increase when the to-be-remembered material is of future importance (157). Wilhelm et al. (230) also demonstrated that retrieval expectancy enhances "sleep associated consolidation of visuospatial (two-dimensional object location task) and procedural memories (finger sequence tapping)". Thus, sleep may be more beneficial in declarative memory consolidation when there is an expectation of memory recall attributed to the information being learned.

Mazza et al. (131) investigated whether sleep-dependent memory consolidation could reduce practice time needed for relearning of a declarative memory task. 40 participants used retrieval-restudy practice to learn the French translation of 16 Swahili words. One group performed the initial learning session in the morning and again in the evening, with sessions separated by 12 hours. The second group performed the learning session in the evening and then again 12 hours later in the morning, with the learning sessions separated by sleep. The first learning session was completed when perfect performance was obtained, and the follow-up relearning session was again repeated until perfect performance was obtained with the number of trials needed to attain this criterion being measured. A midday cued-recall task was performed one week and six months after the relearning session. By interleaving sleep between learning sessions, there was approximately a 50% reduction in the list trials needed to

reach the relearning criterion. In addition, there was a significant difference in forgetting at the one week and 6-month mark with the sleep group dramatically outperforming the wake group. This study indicates that sleeping between learning and relearning is a beneficial strategy for long-term declarative memory retention(131).

REM sleep is considered to play a critical role in episodic memory consolidation (163, 222). REM sleep deprivation has been shown to significantly impair spatial episodic information recall, as well as reduce the vividness of this type of memory (163). REM sleep may also aid in the retention of emotional material. Wagner et al. (222), demonstrated that REM sleep enhanced the memory of emotional versus neutral texts (222). This is indirectly supported by neuroimaging data that indicates conspicuous activation of the amygdala, a vital structure for emotional processing, during REM sleep (129, 222).

As previously mentioned, nondeclarative memories are collectively considered procedures of "how" to perform various tasks in which the learning process is of an unconscious nature (157). Research demonstrates that post-training sleep enhances acquisition levels on perceptual (62, 112), perceptual-motor, non-verbal and procedural learning tasks (51, 62, 112, 157). Fenn et al. (62) demonstrated that a 12-hour sleep period, following initial training, significantly improved memory consolidation and performance accuracy of perceptual learning of spoken language, compared to a 12-hour waking period (62). Stickgold et al. (2000) (202) revealed that visual discrimination task improvement, following overnight sleep, was a direct function of both the amount of SWS in the first quarter of the night and the amount of REM sleep in the last quarter.

The product of early SWS sleep and later REM sleep accounted for 80% of the intersubject variance. This study suggests that improvement in visual discrimination tasks are mediated by a sequence of early SWS, which may prompt memory formation and late REM episodes which perhaps consolidate early memory formations (157, 202).

Dayan et al. (49) describe motor skill learning as a "process by which movements are executed more quickly and accurately with practice". Motor sequence learning, such as learning a piano scale or sequencing actions in throwing a football, is often investigated using the movement sequencing task. The movement sequencing task requires subjects to tap a sequence of keys at varying paces and sequences. Training on the task improves speed and accuracy, which indicates that motor learning has occurred (192). Walker et al. (223) found that a night of sleep following a finger tapping task resulted in a 20% increase in motor speed without a loss in accuracy. Conversely, an equivalent time awake following training provided no extra benefit to performance. Furthermore, the quantity of stage 2 NREM sleep, late in the night, was strongly correlated with improved performance (223). This period of stage 2 NREM sleep also correlates with sleep spindle peak density. According to Spencer et al. (192) "spindles have been shown to increase following a motor task and are associated with cellular plasticity…". In addition, functional MRI data indicates that off-line (sleep) consolidation following motor sequence learning increases neural activity within the corticostriatal system (51). In brief, research has implicated sleep as a foundational component in memory consolidation and as a means of optimizing the learning process.

## Cognitive Performance

It is well established that sleep deprivation degrades cognitive performance, with cognitive performance becoming progressively worse as time on task is extended (74, 81). According to Goel et al. (74) "…this is the classic 'fatigue' effect that is exacerbated by sleep loss". Sleep deprivation has been shown to impair visual and auditory attention with visual-motor-performance demonstrating more sensitivity to state instability (109). It has been proposed that chronic SR leads to 'long-time-constant' changes that serve an adaptive role to stabilize performance (21). However, according to Belenky et al. (21)"…these adaptive changes appear to come at a cost – brain operational capacity is capped in a manner that apparently precludes rapid recovery to baseline levels of alertness and performance when sleep durations are extended to baseline levels".

Cognitive tasks requiring divergent problem-solving skills sometimes referred to as "creative problem solving" have been reported to be negatively affected by SD (73, 74). Divergent cognitive tasks require multitasking, flexible thinking and the creative ability to see new combinations of ideas that may lead to more than one way of solving a problem (6). Divergent cognitive skills that are affected by sleep loss include lateral thinking and innovative abilities, focused attention and assimilation of large amounts of information, developing and updating strategies, assessing risk, mood-appropriate behaviors, effective communication and temporal memory skill (74, 81).

In the largest sleep study to date, which objectively measured real-world sleep and performance, Althoff et al. (8) demonstrated that performance is influenced by prior sleep timing, duration, chronotype (morning/evening preference) and circadian rhythms. Over 18 months, 3 million nights of sleep were tracked by wearable sensors from 31 thousand users with 75 million real-world performance measurements. Cognitive performance was measured by correlating speed of keystroke and click interactions on a web search engine with sleep measures captured by a wearable device over time. The process of entering a query for completion by the search engine and the subsequent response time for choosing a search result captured performance on two different tasks relying on "…different mixes of sensing, reflection, planning and formulating, executing, and monitoring motor plans" (8). Althoff and colleagues found that performance was reduced by up to 31% during habitual sleep times (8). Performance also varied based on chronotype (calculated by created a modified mid-sleep free point on free days), with early sleepers performing slowest at 04:00 hr and medium or late sleepers performing the slowest at 05:00 hr and 06:00 – 07:00 hr, respectively. Keystroke and click time also varied with both acute and chronic sleep duration. Althoff et al. stated: "We demonstrate that very short and very long sleep durations, and irregular timing of sleep are associated with 3%, 4% and 7% lower performance, respectively" (8). Sleeping less than 6 hours and more than 9 hours resulted in significantly impaired performance, with 7.00 – 7.50 hours being the optimal duration of sleep. Finally, they determined that two nights of sleep with fewer than 6 hours of sleep was significantly associated with reductions in performance for six days.

Nir et al. (149) examined the effects of sleep deprivation on individual neuronal firing patterns along with EEG wave activity and how these responses change upon cognitive lapses. Cognitive lapses were deemed as slow trials characterized by delayed behavioral responses. Intracranial electrodes were used to record single-neuron activities and local field potentials (LFPs) in 12 human neurosurgical patients with pharmacologically intractable epilepsy. These subjects performed a face/nonface categorization variant of the psychomotor vigilance task (PVT) in 31 experimental sessions, including a session after a complete night of SD (149). The investigators found that before cognitive lapses, "…the selective spiking responses of individual neurons in the medial temporal lobe (MTL) were attenuated, delayed, and lengthened" (149). The authors also determined that time spent awake (TSA) before the task was the dominant factor influencing performance and that TSA increased the occurrence of cognitive lapses. A time-on-task effect was also observed, whereby cognitive lapses increased as the time spent performing the task also increased. Finally, in responsive LFP channels, cognitive lapses were associated with a "selectively weakened decrease in slow/theta power"(149). The results of this study suggest that during SD there is a dampening effect on brain cell activity, and the selective weakening of slow/theta power implies that select regions of the brain were in a 'sleep-like' state, while the rest of the brain was in a regular operative state.

Sleep restriction leading to a state of excessive sleepiness is considered a significant problem in the general population (53). In 2010, the American Academy of Sleep Medicine reported that 19.5% of US adults reported having moderate to excessive

sleepiness, with 11% reporting severe sleepiness (134). Insufficient sleep is considered one of the leading factors leading to excessive daytime sleepiness. It is estimated that on U.S. roadways there are 328,000 driving crashes associated with drowsy driving each year, with an average of 6,400 fatal drowsy driving crashes (13). These crashes burden the U.S. economy with an estimated societal cost of $109 billion for fatigue-related fatal and injury crashes, which does not include property damage (13).

Sleep restricted impaired performance has been equated to performance impairments caused by alcohol intoxication. Following 18 hours of sustained wakefulness, cognitive, psychomotor performance decreases to that of a person with a Blood Alcohol Content (BAC) of 0.05% (13). After 21 and 24 hours of sustained wakefulness, performance drops to the level equivalent of a BAC of 0.08% (the legal limit in all states) and 0.10% respectively (13, 48). This data reveals how relatively moderate levels of fatigue can be a risk factor for activities of daily living, such as operating a motor vehicle.

## Sleep Quantity and Quality Recommendations

The U.S. Department of Health and Human Services recommends that adults should obtain 7 to 8 hours of sleep to perform optimally, avoid sleep debt and limit daytime sleepiness (1). According to the U.S. Centers for Disease Control (CDC), one-third of the U.S. adult population reports sleeping less than 7 hours a day – which is the minimum threshold for good health and well-being (13, 123). Liu et al. (123) states

"Sleeping <7 hours per night is associated with increased risk for obesity, diabetes, high blood pressure, coronary heart disease, stroke, frequent mental distress, and all-cause mortality". The National Sleep Foundation (NSF) recommends that young adults (ages 18-25 yr) and adults (ages 26-64 yr) should obtain 7 to 9 hours of sleep per night (86). Despite these recommendations, a 2015 nationwide survey of the United States, conducted by the NSF, revealed that adults sleep 6.90 hours on 'work days' and 7.6 hours on 'non-work days' for an average of 7.10 hours per night (65). On work days, the average sleep debt was 26 minutes, as Americans indicated they would prefer to sleep 7.30 hours per night. In addition, when asked about how often they get a good night's sleep, 32% of respondents reported sometimes, and 13% reported rarely or never (65).

Sleep quality also plays an important role in restful and satisfying sleep. According to Ohayon et al. (152): "Good sleep quality is a well-recognized predictor of physical and mental health, wellness, and overall vitality". The NSF has identified nine possible indicators of nocturnal sleep quality with four variables being related to sleep continuity (sleep latency, awakenings >5 minutes, wake after sleep onset, sleep efficiency), and five sleep architecture variables (REM sleep, N1 sleep, N2 sleep, N3 sleep, and arousals). In addition, three nap variables were identified (naps per 24 hours, nap duration, and days per week with at least one nap) (152). The NSF stated that four sleep continuity variables (sleep latency, number of awakenings >5 minutes, wake after sleep onset, and sleep efficiency) are "…appropriate indicators of good sleep quality across the life-span" (152). Table 1 and 2 present the NSF's suggestions for sleep quantity and quality for young adults.

Table 1. National Sleep Foundation's sleep quantity recommendations for young adults.

| Age | Recommended (hours) | May Be Appropriate (hours) | Not Recommended (hours) |
|---|---|---|---|
| Young Adults (18-25 yr) | 7 - 9 | 6, 10 - 11 | <6, >11 |

*Note*. Adapted from Hirshkowitz et al. (86).

Table 2. National Sleep Foundation's sleep quality recommendations for young adults.

| Indicator of Sleep Quality | Appropriate | Uncertain | Inappropriate |
|---|---|---|---|
| Sleep Latency (min) | 0 - 30 | 31 - 45 | ≥ 46 |
| Awakenings (>5 min) | 0 - 1 | 2 - 3 | ≥ 4 |
| Wake After Sleep Onset (min) | ≤ 20 | 21 - 40 | ≥ 41 |
| Sleep Efficiency (%) | 85 - 100 | 65 - 84 | ≤ 64 |

*Note.* Adapted from Ohayon et al. (152).

Sleep in the Athletic Population and University Setting

Although sleep is referred to as the "single best recovery strategy available to elite athletes" there is not a consensus regarding how much sleep is required for elite athletes (82). Bompa and Haff (24) suggest that athletes require 9 to 10 hours of sleep, with 80-90% being obtained during the night, and as stated previously the NSF recommends 7 to 9 hours. Some studies suggest that 8 hours of sleep is required to prevent the neurobehavioral deficits associated with sleep loss (21, 116, 216). Despite

these recommendations, data suggest that athletes sleep far less than what may be required for optimal performance (69). The sleep/wake behavior of 124 elite Australian athletes was examined using self-report diaries and wrist activity monitors (116). It was found that individual sport athletes sleep less than team sport athletes, with individual sport athletes sleeping 6.50 hours per night and team sports athletes obtaining 7 hours per night (116). A recent survey of 130 Division I collegiate student-athletes revealed that 43% of the student-athletes slept six hours or less on weekdays and 15% of those surveyed slept six hours or less on weekends (229). Finally, Carrico et al. (38) found that elite footballers habitual sleep habits on training days and match days were well below the NSF recommendation of 7 to 9 hours.

According to Lastella et al. (116) "Anecdotal evidence indicates that athletes are concerned about the amount of quality sleep they obtain as they believe that good sleep substantially contributes to their capacity to compete at an optimal level"(116). In a recent systematic review by Gupta et al. (78), it was stated that "Insomnia is high among elite athletes, with sleep quality appearing most vulnerable prior to competitive events, during periods of high-intensity training and following long-haul travel to competitions". Shearer et al. (184) report that amongst elite rugby union players, post-competition sleep is deprived and may have detrimental effects on the recovery process (184). In a study of 632 German athletes, it was found that 62.30% of the athletes had experienced poor sleep the night before a competition within the previous 12 months.

Recently Mah et al. (126) investigated the sleep quality and duration of 628 athletes across 29 varsity sports at Stanford University. Sleep duration, onset latency,

subjective sleep symptoms, sleep medication, and disturbances were assessed using the Pittsburgh Sleep Quality Index (PSQI). The Epworth Sleepiness Scale was also used to quantify daytime sleepiness. 42.40% of the athletes, of which 97% lived on-campus, were identified as poor sleepers with PSQI scores >5. Sleep quality was poorer amongst males than females, and 17% of the population surveyed had "fairly bad" or "very bad" sleep quality. Weekday sleep durations of <7 hours were reported by 39% of the athletes, and 59% of the teams (17 teams) averaged < 7 hours during the week. 51% of the athletes indicated high levels of daytime sleepiness (Epworth score ≥ 10), in addition total sleep time was found to be predictive of daytime functioning. In brief, Mah and colleagues found that "collegiate athletes generally experience poor sleep quality, habitually obtain insufficient sleep, and experience substantial daytime sleepiness" (126).

Sleep habits amongst the general college/university student population are declining (83, 84). Hicks and colleagues report that from 1978 to 2000, University students sleep dissatisfaction scores dropped from 24% to 71% (83, 84). According to the Spring 2016 National College Health Assessment, 22.20% of students surveyed reported that sleep difficulties impacted their academic performance, with sleep ranking third behind "stress" and "anxiety" (12). According to Orzech et al. (156), "Young adults rely heavily on digital media devices to complete many daily tasks, and this use is likely to interact with their daily activities, including sleep". Chronic electronic device use, prior to bedtime, has been demonstrated to lead to acute and/or chronic sleep debt (57). Researchers in the fields of sleep science and human computer

interactions (HCI) have found interactions between digital media and daily rhythms (sleep) amongst University students (156). Exposure to digital media two hours before bedtime has been demonstrated to reduce sleep quantity and lead to later bedtimes in first-year University students (156). The relationship between electronic device exposure and physical and cognitive performance in athletes has yet to be fully elucidated as there is limited research on the topic. Recently, Dunican et al. (57) examined the effects of using electronic devices (e.g., smartphones, laptops, computers and televisions) on sleep quality and next-day cognitive and athletic performance in elite judo athletes. Twenty-three elite Australian judo athletes were monitored over six consecutive nights while attending a training camp. Objective measures of sleep quantity and quality were measured using a wrist-worn actigraph (Readiband; Fatigue Science, Vancouver, BC) and qualitative self-reported (dairy) measures were also collected. Physical (single leg triple hop test) and cognitive (Cogstate) performance were also measured at baseline and following removal of electronic devices. Removal of electronic devices during the training camp for 48 hours did not affect sleep quantity or quality or change cognitive or athletic performance. More research needs to be conducted on the effects of electronic device exposure on psycho-physiological outcomes, as the rate of engagement with screen-based media is rapidly rising (71).

Exercise Performance

The relationship between sleep loss, restriction or deprivation, and exercise performance is unclear. Studies investigating this relationship have yielded conflicting results. Fullagar et al. (69) state the following:

> "These varied results are mainly attributed to differences in exercise protocols, participants' fitness, and the experimental environment. For instance, variations in thermoregulatory responses, habituation to sleep loss and the time of day at which activities are performed have a complex interaction with exercise performance, and thus may potentially mask the effects of sleep loss" (69).

Therefore, caution should be applied when evaluating and interpreting the relationship between sleep and exercise performance.

In a landmark study by Thomas and Reilly (209), it was demonstrated that moderate intensity exercise could be maintained continually for 100 hours with matched energy intake. One male competitive cross-country runner, age 31, alternated between three modes of exercise every 20 minutes: walking on a motorized treadmill (0% slope at a speed of 3 mph), bicycle ergometry (40 revolutions/min with a load of 0.50 kp), and a rowing machine (18 strokes/min). No rest pauses were allowed between work-modes or during the 100-hour work period. Heart rate was monitored using an ECG trace using clip-on needle electrodes. Although work rate was controlled, heart rate initially increased then decreased to reach a steady state after 44 hours, which indicated a decrease in sympathetic drive (166). In addition, lung function deteriorated

over the duration of the experiment and visual reaction time slowed with each successive day without sleep (209). This study demonstrates that with considerable intrinsic motivation and adequate energy intake, exercise can be performed for extended periods of time without sleep.

Following one night of SD, Azboy and Kaygisiz (14) demonstrated that time to exhaustion on an incremental cycling test was reduced in volleyball players, but not in runners. Martin (130) found that following 36 hours of total SD, time to exhaustion for prolonged incline treadmill walking (80% of $VO_{2max}$) was reduced by an average of 11%. Racinais et al. (161) reported that 38 hours of total SD did not significantly reduce $VO_{2peak}$, as assessed by the Leger and Gadoury shuttle test, compared to a rested baseline test. Hill et al. (85) reported that 25-30 hours of sleep deprivation did not affect total work nor the aerobic or anaerobic contributions to an exhaustive high-intensity cycling test in both male and female participants. Finally, Oliver et al. (153) examined the effects of 30 hours of SD on endurance performance, 30 min self-paced distance test preceded by a 30 min pre-load treadmill run at 60% $VO_{2max}$. The authors found that 30 hours of sleep deprivation had a detrimental impact on self-paced total distance covered compared to a night of normal sleep (CON). Despite covering less total distance, perception of effort was similar between SD and CON trials. Oliver and colleagues concluded that: “The same perception of effort despite a tendency for lower selected running speed suggests that perception of effort would be greater for the same absolute workload” (153). Therefore, the mechanism driving a reduction in performance following SD may be an increased perception of effort (153).

Despite the number of sports wherein a key contributor to performance is the ability to perform high-intensity intermittent sprints over a sustained period of time, there is little research investigating the relationship of SD to this type of activity. Skein et al. (188) investigated the impact of 30 hours of total SD on consecutive-day intermittent-sprint performance, perceptual, and physiological recovery with ten male team-sport athletes (188). Following SD, mean sprint times were slower, voluntary force and activation during maximal isometric knee extensions were reduced along with muscle glycogen concentrations. In addition, SD led to an elevation of negative mood states and a suppression of positive feelings, as assessed by the modified Profile of Mood States (POMS) questionnaire (188). The authors suggest that SD may affect intermittent-sprint performance through integrated afferent feedback from the periphery. Reduced muscle glycogen and increased perceptual strain may reduce recruitment of active musculature, therefore decreasing intermittent-sprint performance (188).

Sleep deprivation and its effect on expressions of strength, power, and speed are inconclusive in the literature. Takeuchi et al. (207) reported that 64 hours of total SD did not affect 40-meter dash times and isometric handgrip force. However, vertical jump height and isokinetic knee extension force at $60°\cdot s^{-1}$ were significantly reduced following SD. Symons, Vanhelder and Myles (205) reported that 60 hours of SD did not impair muscular strength and endurance, as well as maximal isometric force. Bulbulian et al. (29) investigated how 30 hours of SD and a 40-km intermittent walk carrying 50% of body mass (BM) affected muscular strength and endurance. Two groups of 12 male

U.S. Marine Corps volunteers were tested in two sessions over four separate 2-weeks periods. The first week involved one laboratory visit designed to assess cardiovascular, pulmonary, hematological and psychometric baselines, and the second week a 3-day, 40 km march with 50% of gross BM was conducted. Before and after the 40 km march, muscle power and endurance were tested. All subjects then repeated the field test 2 days later following 30 hours of SD. Isokinetic testing revealed significant reductions in peak knee extension torque, but not peak flexion torque following SD (29). Muscle fatigue, as assessed by 45 consecutive maximal reciprocal contractions at 3.14 $rad \cdot s^{-1}$, was not compromised following SD (29). Blumert et al. (23) studied the effects of 24 hours of total SD on weightlifting performance and mood states with national-caliber male weightlifters. 24 hours of sleep loss did not affect maximal weight lifted in the snatch, clean and jerk and front squat exercises. However, a POMS questionnaire revealed significant changes in vigor, confusion, fatigue and total mood disturbance following SD. The authors concluded that "If an athlete is in an acute period of sleep loss, as noticed by negative mood disturbances, it may be more beneficial to focus on the psychological (motivation) rather than the physiological aspect of the sport" (23).

Similar to total SD, the reported effects of SR on exercise performance are inconsistent, and the number of studies is limited (69). Mougin et al. (144) investigated the effects of 3 hours of SR, in the middle of the night, with seven cyclists on an incremental cycling test to exhaustion. The cyclist performed 20 min of steady state cycling (75% $VO_{2max}$) followed by a progressive increase in workload (10 W every 30 s) until exhaustion. Compared to baseline sleep, SR exercise led to higher heart rates at

submaximal and maximal exercise intensities and decreased peak $VO_{2peak}$. In addition, SR increased maximal minute ventilation ($V_{Emax}$) and enhanced $V_E/VO_2$ which indicated a reduced ventilatory capacity for exercise (144). Similarly, Mamiya et al. (128) found that time to exhaustion during running at 85% of $VO_{2max}$ was significantly impaired following SR of 60% of normal sleep duration in male subjects.

Mejri et al. (136) studied the effects of two types of partial sleep deprivation (PSD) on intermittent aerobic performance, as assessed by the Yo-Yo Intermittent Recovery Test Level 1 (YYIRTL 1), with ten male Taekwondo players. The YYIRTL 1 was performed under three conditions with ≥ 36 hours of recovery between trials: (1) reference night (RN) following a complete night of habitual sleep, (2) partial sleep deprivation of 4 hours at the beginning of the night (PSDB), and (3) partial sleep deprivation of 4 hours at the end of the night (PSDE). In each sleep condition, the subjects performed the YYIRTL 1 between 7 am, and 8 am. PSDB and PSDE did not significantly affect total distance covered or intermittent aerobic performance parameters ($TD_{YYIRTL1}$, plasma lactate concentration, heart rate peak and rate of perceived exertion) in the morning (136). In brief, this study indicated that PSD at the beginning and end of the night did not alter morning performance of intermittent aerobic exercise in Taekwondo players.

In a follow-up experiment, Mejri et al. (137) examined the effect of one night of PSD on intermittent exercise performance with ten male Taekwondo players, the evening following restricted sleep. Again, the subjects completed three experimental sleep conditions in random counterbalanced order with a ≥ 36 hour recovery period in

between conditions. The YYIRTL 1 was completed at 5 pm on the day following PSD. A significant effect of PSD was observed on $TD_{YYIRT}$, with the most pronounced decrement in performance being the PSDEN condition. Mougin et al. (143) observed a similar decrease in afternoon maximal work rate with well-trained endurance athletes following both PSDBN and PSDEN compared to RN(137, 143). According to Mejri et al. (137)"…one night of PSDBN or especially PSDEN before a competition constitute a disturbance agent of high risk on subsequent performance" (137).

Reilly and Deykin (165) reported that three nights of sleep restricted to 2.5 hours did not impact treadmill endurance running, maximal anaerobic power, muscular strength, or lung power. Also, short-term anaerobic power has been reported to be unaffected by one night of SR (142, 219). HajSalem et al. (79) found that PSDE reduced mean and peak power during the Wingate test, yet hand grip strength was unaffected by PSDE. Abedelmalek et al. (2) report a similar decrease in mean and peak power during the Wingate testing in footballers, following PSDE with performance only being affected during afternoon (1800 hours) and not morning (0800 hours). Souissi et al. (191) also reported reductions in short-term maximal performance following PSDE with concomitant decrease in muscle strength and power with afternoon testing. The authors suggested that PSDE might blunt "diurnal variations of short-term maximal exercise" (191).

Finally, Reilly and Piercy (167) investigated the effect of three nights of SR, ration of three hours per night, on submaximal and maximal weight-lifting exercises. The bicep curl, bench press, leg press, and deadlift exercises were performed to assess changes in

strength each evening following restricted sleep. A submaximal load equivalent to 35-45% of maximal load was lifted for 20 repetitions with proper technique, and then a maximal lift was performed for each exercise. Trend analysis indicated a decrease in performance for all submaximal efforts across all four exercises. In addition, there was a decrease in maximal performance for bench press, leg press, and deadlift, but not bicep curl. This study suggests that multiple nights of SR effect both submaximal and maximal weightlifting tasks(167).

Sports Performance

Sleep restriction and deprivation have been demonstrated to impact sport-specific skill and performance negatively. Leger et al. (121) examined sleep management and performance during the Tour de France à la Voile 2002 yacht race. Sleep length, sleep debt and sleepiness before and during competition was compared to race performance. Analysis revealed that the final rankings of the race were related to sleep management strategies, with the winner achieving a lower total sleep debt (121). Recently, Juliff et al. (107) found that during a national multiday netball competition, sleep duration was strongly associated with higher final tournament positions (107).

Sinnerton and Reilly investigated the effects of four nights of SR of 2.5 hours per night on swim times (187). Swim times were unaffected by SR, although a trend was noted for mean performance times improving from the morning to the evening testing sessions. Profile of Mood States questionnaire results revealed significant increases in

negative mood states for tension, depression, fatigue, confusion, vigour, and anger. Sinnerton and Reilly stated that "Results support the brain restitution theory of sleep and indicate that the diurnal variation in swimming performance is greater than any due to partial sleep loss" (187).

Research with dart players reveals that SR negatively impacts hand-eye coordination evidenced by changes in accuracy and variability in dart throwing. Following a single night of SR, less four hours of normative sleep, dart players threw "less accurately and less reliably, and missed the target more frequently" (187). These decrements in performance were interpreted as a general deterioration in psychomotor performance (59). Reyner and Horne (168) reported that SR by one-third of normal sleep reduces serving accuracy in semi-professional tennis players. Conversely, Schwartz and Simon (179) found that extending sleep by 2 hours per night significantly improved serving accuracy in National Collegiate Athletic Association (NCAA) Division III tennis players. It was also reported that with sleep extension the Epworth Sleepiness Scale and Stanford Sleepiness scores declined significantly, indicating an improvement in perceived sleepiness (179).

Over two NCAA seasons, Mah et al. (127) examined the effects of extended nocturnal sleep duration on indices of athletic performance, reaction time, daytime sleepiness and mood with 11 basketball players on the Stanford men's basketball team. Subjects maintained their normal sleep schedules during a 2-4 week baseline period during the competitive season and stayed within the limits of 6-9 hours of subjective sleep per night. Following 5-7 weeks of sleep extension, with a minimum goal of 10

hours in bed, subjects demonstrated significantly faster sprint times, improved free throw percentage and 3-point field goal percentage. In addition, PVT reaction time improved and subjects reported improved mental well-being and physical well-being during practice and games (127).

Juliff et al. (107) state that "Sleep is often regarded as the single best recovery strategy available to an athlete". Sadly, inadequate quantities of sleep are reported to be a significant issue in the collegiate student-athlete populations (107, 229). Total and partial sleep deprivation has been demonstrated to negatively affect physiological and psychological function which can compromise the adaptive process which is the underlying means of improving the training process (66, 73, 74, 109, 192, 194, 221, 227). Therefore, sport coaches and physical preparation coaches should plan for and monitor sleep as part of the global preparation of the athlete.

Literature Review of Direct Current Potential and Its Application to the Training Process

History

In 1935, H. S. Burr and F. S. C. Northrop proposed that a field theory for living systems would allow for the scientific study of "physico-chemical" interactions with bioelectric fields. Burr and Northrop detailed a problem with the current scientific conception of nature, in that it was viewed "as a collection of particles in motion and physico-chemical interaction, there is no meaning to the field as anything more than a mere aggregate and effect of their compounding" (31). Burr and Northrop realized that to progress the study of how bioelectric fields may represent the nature of living systems, a shift needed to occur in how biological fields were viewed. To make sense out of the notion that the "field determines the behavior of any local process or constituent within it" Burr and Northrop put forth *The Electrodynamic Theory of Life (31)*. This new theory opened the door for the legitimate study of how and why measurement tools such as the EEG and EKG represented physiological activity, and that the electrical data derived from such tools represented the by-product of living processes.

In 1939, Pauline A. Davis investigated waking human brain reactions to acoustic stimuli (47). Silver-silver chloride electrodes were placed on multiple parts of the scalp, and EEG reactions to acoustic sounds were measured. Electrical responses "of the same character" were observed in multiple regions but were most clearly observed from the vertex. An "on-effect" was observed when the acoustic stimuli were presented to the

subjects. However, Davis was unable to explain the effect's origin (47). In 1949, Kohler and Held reported that functional interrelations in the brain where coordinated by a continuous field action which consisted of "direct currents which spread through the brain as a continuum"(113). Kohler and Held also reported that this direct current could be measured from the intact human head and that the behavior of these currents agreed with predictions from biophysical theory.

In 1957, H. S. Burr delivered a talk to the Neurological Study Unit at the Yale School of Medicine. Burr, a professor of anatomy at Yale, proposed that the pathway to understanding human behavior was to gain a deeper understanding of the structure and function of the nervous system (30). Burr proposed that: "If field properties could be demonstrated, a common basis for all the electrical phenomena would be at hand, since all the electrical phenomena could be variations in the relatively steady state standing potential of the electrodynamic field" (30). Burr also noted a current problem with the technology available to study electrical field properties. At the time, instruments of observation required a current to be taken from the living system, thus disturbing the organism or physiological system being measured.

As early as 1955, Natalia Bekhtereva began recording and analyzing slower wave forms other than the traditionally studied α-waves in animals and humans (19, 20). Bekhtereva, a neuroscientist and psychologist who is credited for developing a neurophysiological approach to study psychology, first began recording DC potentials by using indwelling electrodes placed in the brain and exposing animals to various environmental factors including sound and light (19). In 1958, Soviet researchers at the

Institute of Biological Physics of the Academy of Sciences of the USSR, N.A. Aladzhalova and A.V. Kol'tsova, found that in different parts of the brain, specifically the hypothalamus and cerebral cortex, electrical activity unlike that usually revealed by the electrocorticogram was able to be recorded (4). Aladzhalova and Kol'tsova reported that it was possible to register "very slow periodic variations in potential, with a frequency of 5-8 per minute and an amplitude of 0.30 – 0.80mv" (4). They named these new frequencies "hyperslow" and "infraslow" (4).

In 1964, O'Leary and Goldring reported DC potentials could undergo temporary or sustained changes wherein "slow aftermaths" would fuse, and named these changes SP shifts (150). Further study revealed that these shifts followed "more enduring depolarization" events arising from anoxia, injury, anesthesia or administration of pharmacological agents (150). SP shifts were also found to occur during and after seizures, sleep, states of activation, and depression. These precedential studies were later supported by numerous neurophysiological studies where DC potentials were confirmed to represent the slow regulatory system of the brain, which only responds to environmental factors that are "exceptional strong or frequent" (141). Whereas the EEG is used to measure the fast regulatory systems of the brain, that respond to stimuli that are weaker or irregular (141). According to Gribanov et al. (75) DC potentials are slow changing and one of the most constant physiological processes of the brain.

In the 1980s, concurrent with the invasive techniques used to study DC potentials wherein gold electrodes were used to penetrate deep structures of the brain, an alternative non-invasive method was being explored. Sychev determined that "the

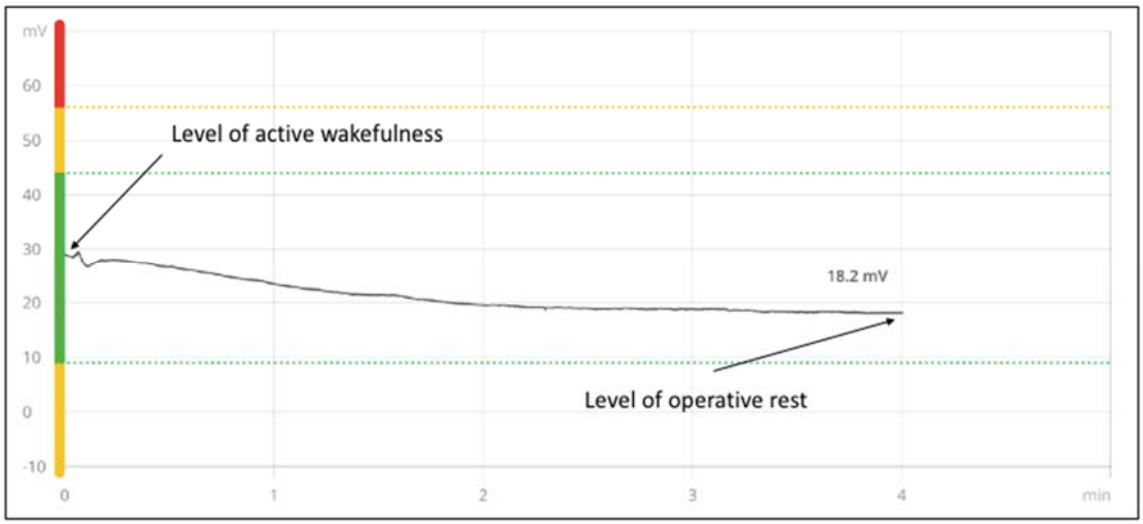
stable potential on the millivolt scale in the vertex-tenar derivation permits quick diagnosis of the functional state of athletes, optimization of the training process, and enhances performance in sport" (96). In 1986, Ilyukhina and Dan'ko revealed the specific hardware required for multiparametric recording of the human functional state (97). This research led to what is now known as the omegametry method for recording DC potentials (96). The omegametry method utilizes a discrete or continuous recording of brain biopotentials within the frequency range of 0.00-0.50 Hz, in both a state of rest and under functional loads (96). A variety of terms have been used to describe DC potential, including Omega-potential (111), ultraslow biological potential oscillations (141), slow rhythmic oscillations (3) , infraslow (92) and the quasi steady difference in potentials (141); however, for the remainder of this review, the term DC potential will be used to provide continuity in terminology.

Although the original works have not been translated into English, later works by Ilyhukina detail how Sychev and colleagues utilized DC potentials in evaluating the stress response of athletes during the training process.

> Ilyukhina states "At the first stage of systemic studies by Sychev et al., the question of differentiating diagnostic markers according to parameters of omegametry in the assessment of adaptation and compensatory – adaptive abilities of the body of healthy humans to mental and physical loads in sports was stated and solved. The data of omegametry were compared to the psychological indices of the efficiency of sports achievements and tolerance of stress effects (prestart state)" (96).

The critical parameters derived from the omegametry method include omega-potential (DC potential), the initial level of active wakefulness (LAW), and the level of operative rest (LOR) (96). The first value recorded during the omegametry assessment is the LAW. Following the plateau of the omega-potential, the sign and value of the LOR are recorded, and this signifies the nonspecific resistance of the body to stress (96).

Figure 1. The omegametry method for recording direct current potentials.

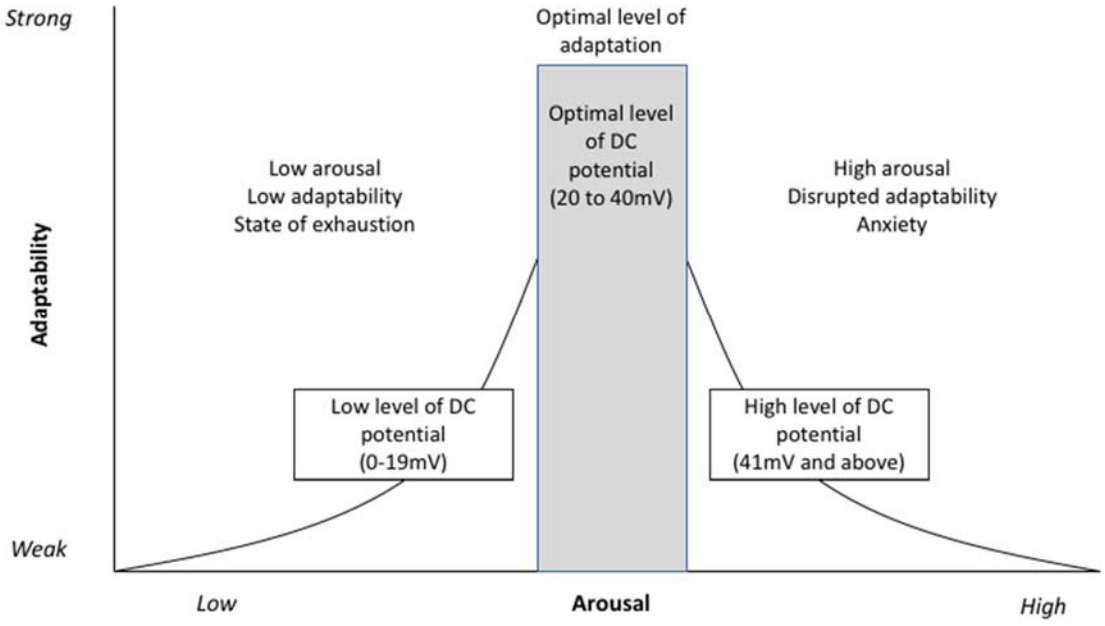



*Note*. Screen capture of DC potential from the Omegawave Ltd (Espoo, Finland) athlete monitoring system.

In 1982, the combined work of Ilyukhina and Sychev was published which outlined quantitative parameters of LOR for the assessment of the healthy human's adaptation and compensatory-adaptive abilities to physical and mental loads in sports (96, 101). According to Ilyukhina, "The data of omegametry were compared to the physiological indices of the efficiency of sports achievements and tolerance of stress

(prestart state). According to the results of the comparative studies, differences in the level of operative rest were differentiated"(96). Figure 2 depicts the relationship between DC potential and adaptability in the human organism.

Figure 2. The relationship between direct current potential and adaptability in the human organism.




*Note.* Adapted from Kara (110).

Low levels of DC potential (0 – 19mV) indicate a decreased level of arousal, a more rapid onset of physiological and psychological exhaustion, diminished adaptive capacity and low functional reserve (96, 101, 141). Moderate levels of DC Potential (20 to 40mV) indicate an optimal level of arousal, a state of high adaptability to endogenous

and exogenous stressors, optimal state for learning new habits and a high functional reserve (96, 101, 141). High levels of DC potential (41mV and above) indicate a high level of physiological and emotional tension, high psycho-emotional stress and limited adaptability to endogenous and exogenous stress (96, 101, 141). Also, high levels of DC potential may indicate a negative adaptive syndrome in athletes (101).

In 1984, Maksimov and Karasj demonstrated how DC potentials could be used as a reliable indicator of long-term adaptation to stress (111). Working as researchers at the Institute of High-Altitude Physiology and Experimental Pathology (former USSR), Maksimov and Karasj investigated how changes in altitude affected the functional state of male engineers (ages 22 to 37 yr) (111). On a daily basis, the engineers would commute from a "living" altitude of 750 m to a "working" altitude of 1750 m above seas level. This short-term transient work conditioning was termed a "pulsatile shift". DC potentials were obtained daily under three conditions: rest (before ascent), following ascent, following descent. The engineers were divided into four groups:

Group 1 – Engineers whose permanent working status was at 1750 m.

Group 2 – Engineers who had worked in a pulsatile shift for over three years.

Group 3 – Engineers working on a pulsatile shift for less than half a year.

Group 4 – Engineers working on a pulsatile shift for half a year to a year.

DC potential recording at rest revealed no significant difference between groups. However, following the ascent and descent, analysis of variance revealed that Group 3

had a 2.50-fold increase in DC potential compared to all other groups. The authors concluded that "…significantly higher omega-potential values reflect the price the body pays for the process of adaptation in the presence of an increased load on the functional systems of the body" (111).

Ilyukhina and Zabolotskikh (102) demonstrated how DC potentials could be used to study acute physiological tolerance to physical loads and typological features of functional states. Eighty-seven subjects, ages 17 to 30 yr attempted two bouts of submaximal exercise (running in place at a self-paced comfortable rate) until self-termination of exercise. Following completion of the exercise protocol, two groups of subjects were singled out. Group I subjects tolerated physical activity well and performed the first bout for 20-45 min and the second bout for 18-25 min. Group II demonstrated "quick fatigability" as they were unable to exceed 10 min of sub-maximal exercise for both bouts. Before the exercise protocol, the participants completed a subjective evaluation of well-being. Group I subjects reported their state as normal and their well-being as good. Almost all Group II subjects reported general fatigue and a low mood state.

DC potentials were recorded at rest and following both the first and second exercise bout. Also, a comprehensive physiological assessment was performed at the same intervals and included evaluations of the autonomic nervous system, internal and external tissue respiration, central hemodynamics, peripheral oxygenation, acid-base, and energy homeostasis.

> Ilyukhina and Zabolotskikh stated that "…significant differences were revealed in the values of central and systemic hemodynamics, physicochemical homeostasis, the level of oxygen consumption by the tissues as well as the values of general non-specific adaptation reactivity of the body at rest in health individuals tolerant of physical stress and age-matched easily fatigued individuals" (102).

Differences in DC potentials at rest and fluctuations following both exercise bouts were evident between Groups I and II. Group I potentials began in an optimal range (32.60 ± 8.70 mV) and following the first exercise bout increased (52.20 ± 0.90 mV) and then decreased following the second exercise bout (22.70 ± 0.80mV). This pattern of change indicated a normative systemic "tension-type shift" of the compensatory-adaptive character of the physiological systems. However, Group II subjects demonstrated a descending pattern of DC potential from rest (12.50 ± 1.70 mV) and following bout one (11.40 ± 0.90 mV) and bout two (7.10 ± 0.80 mV). Following the cessation of the first bout of exercise Group II participants revealed signs of transient circulatory and respiratory hypoxia as well as transient acidosis. The decrease in DC potential following bout one confirms the physiological markers which indicated an imbalance in systemic-compensatory adaptive reactions. The results of this study suggest that DC potential is an accurate indicator of the stress tolerance of the body to external loads, and it confirms the relationship of DC potential to the regulatory systems which maintain homeostasis.

Contributions to Medicine and the Study of Pathological States

DC potential has been used as an objective measure to assess the functional state and adaptive systemic reactions of the body in health and disease (93). As early as 1969, Kambarova and Ivanov (Institute of Experimental Medicine, Academy of Medical Sciences USSR) were using DC potentials to study epilepsy and correlate DC potentials to classical EEG recordings of sleep (18). In 1978, Aladzhalova, Rozhnov, and Kamenetskii (5) began using DC potential to develop a "fresh approach" to the study of the physiology of hypnosis, in the hopes of establishing an objective index of the hypnotic state.

Bechtereva used DC potentials to investigate the neurophysiology of intelligence and emotions in humans (15), psychophysiology (16), and emotional disorders (198). Ilyukhina et al. (93) demonstrated the use of DC potential to enhance medical monitoring when performing exercise tolerance testing (93). Following exercise to exhaustion, hypertensive adult subjects were unable to return to baseline DC potential measures within 15 minutes of completing the exercise protocol. However, normotensive adults returned to baseline measures within 15 minutes. The authors determined that the inability to stabilize body functions indicated a reduced adaptive capacity and suggested the removal of maximal exercise loads for this population (93).

Krupitsky et al. (114) used DC potentials to examine the effects of pharmacological intervention with alcoholic patients with secondary affective disorders (i.e., anxiety and depression). They determined that all pharmacological interventions

significantly decreased DC potentials in the three treatment groups. Krupitsky and colleagues stated that "…the data obtained in this work can be considered as objective electrophysiological confirmation of the positive clinical-electrophysiological dynamics of anxiety observed in the active treatment groups" (114). Ilyukhina and Nikitina (94) also used DC potentials to study alternative pharmacological treatments to mitigate psychological and physiological effects of acute alcohol withdrawal. The authors determined that the use of diphenylhydantoin and standard detoxification therapy (intravenous fluids, cognitive psychotherapy, and occupational therapy) was more effective than standard detoxification therapy alone. Patients who previously presented with sympathetic-adrenal types of autonomic disorders demonstrated significant improvement in mood, aggression and autonomic parameters. Changes in DC potentials demonstrated confirmatory improvement (94).

DC potentials have been used to in the study of a wide variety of pathological states. Ilyukhina, Kozhushoko, and Bokarius (98) used DC potential to examine the functional state and physiological activity of specific structures of the striopallidum and thalamus. Ilyukhina et al. (99) studied factors that dampen compensatory-adaptive reactions of children (6 to 8 yrs) with subclinical forms of pre- and perinatal CNS pathology during transitionary years from preschool to early elementary school. Also, Ilyukhina et al. (100) examined activation levels of the frontal, temporal and parietal cortex of young children (4 to 5 yrs) with normal and delayed speech development. Frolov, Milovanova, and Mekhedova (67) investigated the impacts of music therapy on functional states of subjects with varying levels of anxiety.

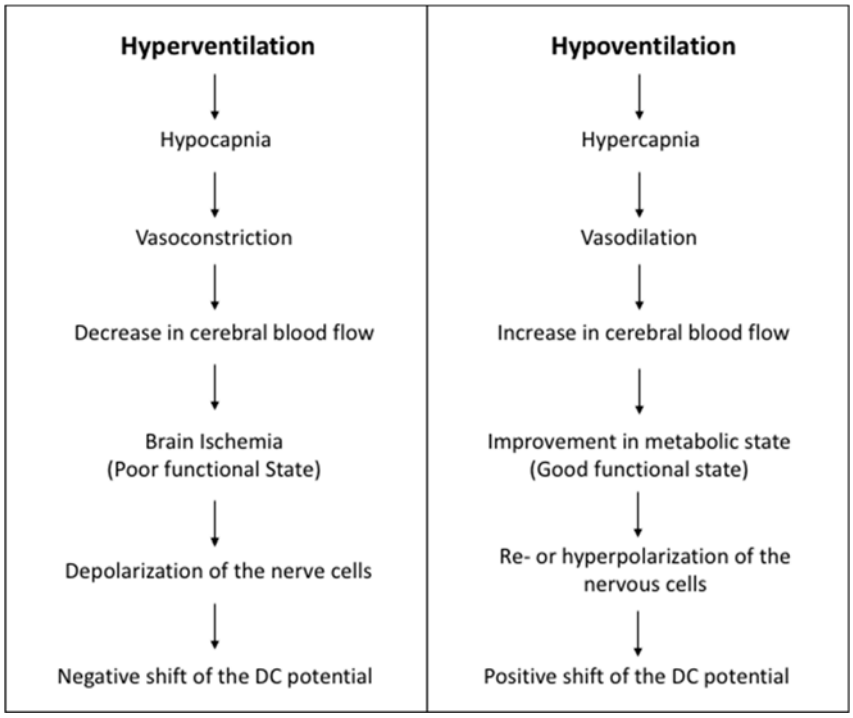
Zhukova used studied changes in DC potentials in 66 women with an endemic goiter (test group) during the third trimester of pregnancy, compared to 66 pregnant women without a goiter (control group) (234). Zhukova recorded DC potentials of both groups in the morning before eating and analyzed the sign and value of the LOR, time to stabilization, and the stability and character of the DC potential curve during a 10-min period. The author found that test group had significantly lower DC potentials, time to stabilization took longer, and the nature of the curves was unstable. Zhukova concluded that the differences in DC potentials between the groups indicated a compensation in the compensatory-adaptive capacity of the regulatory systems, adaptation reserves, non-specific stress response and psycho-emotional lability (234).

Vanhatalo et al. (218) studied hemodynamic changes in the human brain using the direct current EEG (DC-EEG) technique. The goal of the study was to examine how acute manipulations in intracranial hemodynamics would be reflected in changes in DC-EEG. The authors used the following non-invasive manipulations to elicit pressure gradient changes in various intracranial compartments: bilateral jugular vein compression, head-up tilt, head-down tilt, Valsalva, and Mueller maneuvers. Also, changes in cerebral blood volume (CBV) was measured using near-infrared spectroscopy (NIRS). DC shifts were observed with all manipulations, with the most pronounced changes occurring around the vertex. The authors found that for each subject, DC shifts were consistent and reproducible and demonstrated a “clear temporal correlation with changes in CBV” (218). It was proposed that the observed DC shifts could have been caused by changes in intracranial transepithelial potentials, as some transcranial

epithelial layers, specifically those associated with the blood-brain barrier, are known to possess large transcellular potentials.

Murik further examined the use of DC-EEG to estimate the functional and metabolic state of nervous tissue of the brain during hypo- and hyperventilation (145). Six healthy subjects (4 women and 2 men, ages 29 ± 5.40 years) volunteered for this study. The hyperventilation test was composed of 30 deep breathing cycles performed over one minute. A negative shift in DC potential accompanied the weak-ischemic state of hyperventilation. Following a 20-minute rest interval, a hypoventilation test was executed wherein subjects held their breath for as long as possible. A simultaneous positive shift in DC potential was observed during hypoventilation state. Figure 3 details Murik's explanation for the physiological mechanisms that may have produced the polarizing DC potential changes. This study further demonstrates the use of DC-EEG to study functional and metabolic changes in the nervous tissue of the brain (145).

Figure 3. Schematic for the processes occurring in the nervous tissue during hyperventilation and hypoventilation.




*Note.* Adapted from Murik (145).

## The Training Process in Sport Performance

The obtainment of peak performance is achieved through an integrated systemic training process of athlete preparation (64). The training process is designed to induce automation of motor skills and develop structural and metabolic functions that lead to increased physical performance (190). The goal is also to sustain the "highest power output or speed of movement for a given distance or time" while accumulating the least amount of fatigue with the least amount of psycho-physiological cost (190, 220).

The training process is an adaptation-dependent process. To improve the sport-specific skill and the underlying physiological processes of the athlete's organism, a training load is applied by the coach with the intent of improving athlete preparedness. Preparedness is a "multi-faceted cumulative state" which encompasses the developmental state of the athlete's sports specific-skill, technical, tactical, mental and intellectual factors (64). The applied training load, aimed at achieving a specific adaptive response, carries with it a physiological cost of adaptation, which impacts athlete readiness. According to Morris "Readiness may be defined as the current functional state of an individual that determines their ability to achieve their performance potential" (141).

Fomin and Nasedkin (64) define the functional state as: "…a highly sensitive and accurate physiological indicator, which objectively describes individual short- and long-term adaptations of the athlete's body to various stressors, including training load". The concept of the functional state is derived from Anokhin's Functional Systems Theory, wherein functional systems are defined as "dynamic self-regulating organizations whose activity is directed at securing adaptive results, which are useful for the organism" (203). In the context of sport, Verkhoshansky states that "human movement represents an interaction between the human organism and the external environment" (220). The goal of sports movement is to execute the competition exercise. The competition exercise is a system of movements in which the body's segments are coordinated to execute a sporting move (e.g., block, throw, kick). To finalize this task numerous physiological systems (such as the endocrine system, central nervous system,

neuromuscular system) are activated and organized in a functional system focused on executing the competition exercise (220). Therefore, the functional state represents the sum of the individual functional systems and their overall adaptive capacity to allostatic loads (141).

## Direct Current Potential of the Brain: An Objective Measure of Athlete Readiness

To accurately monitor and prescribe training loads for the training process, a non-invasive objective measurement of athlete readiness is required. Ilyukhina describes the brain as a multi-circuit neurodynamic suprasystem that is responsible for information-control processes that vary in rate in the mechanisms controlling the functional state (95). Direct Current potential is considered to be the "universal language" that is most appropriate for studying the central nervous system (CNS) and the functional state (95, 101). Gribanov et al. (75) state that the level of the cerebral DC potential is: "…connected with a complex of biochemical and immunological parameters characterizing the energy expenditure of the brain and the functional state of the adaptive systems of the body" (75). DC potential also characterizes the stability of brain formations and is a quantitative measure of the current functional state, which determines the human organism's physiological activity. DC potential is a slow changing, stable potential measured in millivolts and is one of the most constant physiological processes of the brain (75). Kara states (110) that: "In the realm of athletic

training, DC potential is used as an indicator of the brain's available energetic resources, reflecting the athlete's level of central nervous system (CNS) readiness to perform".

*Applications to the Training Process*

Literature supporting the use of DC potential in the training process is limited because a significant quantity of the research has yet to be translated into English. However, as previously described the literature does support the use of DC potential as an objective measure of short and long-term adaptations to stress (e.g., physiological, psychological, environmental) (102, 111). One of the reasons Sychev developed the vertex-thenar method for the assessment of DC potentials was to assess athletes response to training loads during the training process (101). In the paper, *The Omega-Potential: A Quantitative Parameter of the State of Brain Structures and of the Individual. II. Possibilities and Limitations of the Use of the Omega-Potential for Rapid Assessment of the State of the Individual,* Ilyukina et al. (101) detail the responses of various classes of athletes (i.e., Mater of Sport in the International Class in light athletics) to similar loading stimuli using DC potential. The authors also detail investigations in which G.I. Baryshev correlated DC potentials to a combination of 25 psychophysiological parameters in the members of a "Class A" handball team. Barshev determined that there was a significant correlation between resting DC potential values and the results of the psychophysiological tests. It was found that if resting DC

potentials were between 20 to 40 mV; subsequently, newly introduced sports skills were acquired and executed at a higher rate (101).

Recently, Morris investigated the effects of a fluid periodization model on athletic performance outcomes in collegiate American football players, using an athlete monitoring system (AMS) that assesses the athletes' state of readiness (141). Morris used the Omegawave athlete monitoring system which uses a combination heart rate variability (HRV) and DC potential to assess athlete readiness to train. A longitudinal comparison between two non-randomized groups was used. The treatment group adhered to a fluid periodization model in which volume and intensities of training sessions were modified based upon Omegawave's assessment of athletes' readiness. The control group was not assessed using the Omegwave AMS and performed a similar training regime as the treatment group that was designed by the Strength and Conditioning staff. Following an 8-week training intervention, the treatment group significantly increased in broad jump, vertical jump, lower body vertical power, and aerobic efficiency compared to the unmonitored control group. As a result of using a fluid periodization approach based on DC and HRV output, the treatment group performed 9.5% less arbitrary units of core resistance training volume and 13.2% less volume of accessory exercises. This study indicates that using objective assessments of athlete readiness to guide training, results in improvements in athletic performance outcomes concomitant to a reduction in physiological cost to the athlete (141).

In brief, DC potential has been used in both healthy and diseased populations to assess adaptation and compensatory-adaptive responses to stress (101, 102, 111, 114,

150). The assessment of DC potential, using the vertex-thenar method, allows for the quick and accurate assessment of the functional state of the athlete which can be used in the optimization of the training process (101, 141).

# CHAPTER III

# METHODS

## *Experimental Approach to the Problem*

The purpose of this study was to determine the effect of acute and extended sleep quantity on DC potential outcomes. This study utilized a longitudinal repeated measures design that spanned the 2015 NCAA American football season, including the pre-season training camp and competitive season. Sleep quantity and quality served as the independent variables, whereas DC potential served as the dependent variable.

## *Subjects*

A convenience sample of 24 Division 1 American football players were recruited to participate in this study. The physical characteristics and players' status of the sample are provided Table 3. To qualify, subjects must have been current and active members of the University's football team and cleared to participate in all football related activities. Participation in the study was made available to all players that were projected as starters or reserves that would participate in games. Each subject provided written informed consent and the study was approved by the University of Kentucky's Institutional Review Board.

Table 3. Qualification status and descriptive statistics (mean ± SD) of 24 male Division 1 American football players.

| All Athletes (N = 24) | |
|---|---|
| Age (yr) | 20.6 ± 1.3 |
| Body mass (kg) | 114.6 ± 24.6 |
| Height (cm) | 183.4 ± 6.4 |
| Position | |
| Offensive lineman | 6 |
| Wide receiver | 4 |
| Tight end | 1 |
| Quarterback | 1 |
| Running back | 1 |
| Defensive lineman | 3 |
| Defensive back | 4 |
| Inside linebacker | 1 |
| Outside linebacker | 2 |
| Special teams | 1 |
| Athlete Qualification Status | |
| Freshman | 2 |
| r-Freshman | 1 |
| Sophomore | 4 |
| r-Sophomore | 4 |
| Junior | 4 |
| r-Junior | 3 |
| Senior | 3 |
| r-Senior | 3 |
| *Redshirt (r) | |

*Procedures*

Sleep quantity and quality was assessed using wrist-worn actigraphy (Readiband; Fatigue Science, Vancouver, BC Canada). Actigraphy has been deemed a valid and reliable method for detecting sleep patterns in normal, healthy populations by the American Academy of Sleep Medicine (135). Compared to the "gold-standard" for measuring sleep, polysomnography (PSG), wrist-worn actigraphy correctly scores sleep

with high a degree of sensitivity and accuracy (189), and is considered a convenient method for continuous recordings over a 24-hour period for lengthy periods of time (9). Recently, investigators reported that the Readiband was a valid tool for determining sleep/wake periods, sleep quality and quantity (172). In addition, the Readiband actigraph was found to be 93% accurate in determining sleep scoring compared to data derived from PSG (172). Driller, McQuillan and O'Donnell (54) determined that: "The Readiband™ is a reliable tool for researchers using multiple devices of this brand in sleep studies to assess basic measures of sleep quality and quantity in healthy adult populations". The Readiband has been used to assess daily sleep and mental fatigue within a broad range of personnel and settings including orthopedic surgical residents (133), forestry industry (58), railroad workers (89), elite athletes (including the National Football League) (57, 58), and it has been used to measure alertness and performance by the Federal Aviation Administration (171).

Sleep quantity, efficiency, latency and total wake episodes (>5 min) after sleep onset were all automatically generated using Fatigue Science's proprietary system. Sleep quantity was identified as the longest continuous sleep event in one 24-hour period. Sleep latency was considered the time it took to transition from wakefulness to sleep during the primary sleep event. Sleep efficiency was defined as the percentage of time in bed spent sleeping. Wake episodes were defined as non-sleep periods of >5 minutes. All acute sleep variables were confined to the longest continuous sleep event in one 24-hour period. Extended sleep variables were calculated as the rolling average of two consecutive nights of sleep.

The subjects were instructed to wear the actigraphy device seven days per week, and not to remove the band except during practice, weight lifting sessions, and games. The devices were collected every seven to ten days before practice sessions for data upload and charging. Also, the data were filtered to remove training and game artifacts. The subjects were given a report each week regarding the previous week of sleep, and feedback was provided on how to improve their individual sleep habits.

To measure DC potential, the subjects were assessed six days per week using the Omegawave AMS because they were given one day off from football related activities per week. Optimally, assessments must be conducted within thirty minutes of waking, and subjects self-administered their readiness assessment. Subjects were instructed to perform the assessment by lying supine on the floor for two minutes, before the assessment, in a dimly lit room to eliminate distraction and to assist in relaxation. Before the assessment, the subjects were instructed to remain vertical for at least one minute before placing themselves in a relaxed supine position. DC potential output was sent to the athlete's personal cellular device via a Bluetooth transmission. Upon completion of the assessment, the data were uploaded to Omegawave's cloud server. Five of the twenty-four subjects performed the assessment on their own in this manner. Nineteen of the twenty-four subjects performed the Omegawave assessment 75-120 minutes prior to the onset of football training sessions. These assessments took place in the football training center under the supervision a research assistant. The Omegawave assessment protocol was strictly adhered to. After all assessments were completed the

data were downloaded from Omegawave's cloud server onto the research assistant's laptop computer.

The Omegawave AMS assessed the functional state through the interpretation of DC potential activity. DC potential was measured using the vertex-thenar method, wherein an Ag/AgCl electrode is placed on the forehead and palm of the dominant hand. The difference between the initial level of active wakefulness (LAW) and the level of operative rest (LOR) was calculated following three minutes of rest in the supine position (96). At the three-minute mark, the LOR was automatically recorded by the Omegawave AMS. The resting DC potential, or LOR, represents the functional state of the athlete.

*Statistical Analysis*

Basic statistics (mean, standard deviation, 95% confidence interval) were used to describe the outcome variables. Because measures of DC potential were collected repeatedly across participants throughout the duration of the sampling period, within subject-level means relative to sleep quantity and quality were calculated for sampling independence. The sleep quantity and quality variables were categorized using the NSF's sleep quantity and quality recommendations for both acute and extended timeframes (86, 152). Sleep quantity was stratified into the following categories: <6 hr, 6-7 hr, 7-9 hr, 10-11 hr and ≥ 11 hr. Sleep latency was stratified the three categories: 0-30 min, 31-45 min, ≥46 min. Sleep awakening episodes were stratified into three

categories: 0-1, 2-3, ≥4, and finally sleep efficiency was also stratified into three categories: ≤64%, 65-84%, 85-100%. The normality of the dependent variables' strata distribution were assessed with Shapiro-Wilk's tests of normality. All dependent variable strata were normally distributed ($p > 0.05$).

Repeated-measures analysis of variance (ANOVA) were used to examine the effect of sleep quantity and sleep quality categories on DC potential outcomes. A subject's data were excluded from the repeated-measures ANOVA if data were not present for a level of stratification (Appendix A). Paired samples t-tests with a Bonferroni adjustment were used as the post hoc analysis when significant main effects were identified. Effect sizes for ANOVA outcomes were evaluated using a partial eta squared ($\eta_p^2$) where small, moderate, and large effects were indicated by $\eta_p^2 < 0.06$, $0.06 \leq \eta_p^2 < 0.14$, and $0.14 \leq \eta_p^2$, respectively (88). Effect sizes for post hoc analyses were evaluated using Cohen's d, where a small, moderate, and large effects were indicated by $0.20 \leq d < 0.50$, $0.50 \leq d < 0.80$, and $d \geq 0.80$, respectively (43). The significance level for this study was set at $p \leq 0.025$.

# CHAPTER IV

# RESULTS AND DISCUSSION

## Results

### *Direct Current Potential*

1246 sleep episodes and Omegawave assessments were paired for analysis. There was a 92.20% compliance rate for wearing the Readiband and a 77.90% compliance rate for Omegawave testing. There was a significant effect of player on DC potential ($F_{3,54}$= 9.87, $p$ = 0.03, $\eta_p^2$ = 0.15), where $\eta_p^2$ indicated a large effect size (Table 4). This suggests that the average DC potential varied considerably from day-to-day within each of the subjects in the study.

Table 4. Descriptive characteristics of direct current potential following one night of sleep for all subjects throughout the assessment period.

| | | DC Potential (mV) | | |
|---|---|---|---|---|
| Subject ID | DC Potential | SD | 95% CI | Number of Samples |
| 1 | 18.22 | 11.42 | [6.8, 29.64] | 62 |
| 2 | 24.22 | 12.62 | [11.6, 36.84] | 35 |
| 3 | 15.79 | 9.7 | [6.09, 25.49] | 65 |
| 4 | 20.96 | 11.82 | [9.14, 25.49] | 56 |
| 5 | 27.87 | 9.17 | [18.7, 37.04] | 60 |
| 6 | 19.41 | 10.52 | [8.89, 29.93] | 69 |
| 7 | 15.87 | 8.37 | [7.5, 24.24] | 52 |
| 8 | 16.46 | 15.39 | [1.07, 31.85] | 43 |
| 9 | 22.49 | 10.07 | [12.42, 32.56] | 55 |
| 10 | 9.62 | 12.46 | [-2.84, 22.08] | 18 |
| 11 | 15.47 | 7.48 | [7.99, 22.95] | 24 |
| 12 | 7.7 | 6.78 | [0.92, 14.48] | 21 |
| 13 | 27.53 | 13.33 | [14.2, 40.86] | 55 |
| 14 | 21.59 | 10.59 | [11, 21,18] | 64 |
| 15 | 24.86 | 11.1 | [13.76, 35.96] | 65 |
| 16 | 8.82 | 8.94 | [-0.12, 17.76] | 62 |
| 17 | 19.52 | 8.1 | [11.42, 27.62] | 34 |
| 18 | 34 | 11.07 | [22.93, 45.07] | 62 |
| 19 | 8.99 | 10.01 | [-1.02, 19] | 63 |
| 20 | 20.23 | 8.1 | [12.13, 28.33] | 44 |
| 21 | 21.68 | 9.94 | [11.74, 31.62] | 100 |
| 22 | 30.36 | 10.21 | [20.15, 40.57] | 62 |
| 23 | 23.21 | 7.51 | [15.7, 30.72] | 39 |
| 24 | 10.46 | 8.68 | [1.78, 19.14] | 36 |

*Acute Sleep Quantity Versus DC Potential*

The average acute sleep quantity was 6.87 ± 1.46 hr (SE = 0.04 hr) in all subjects. Table 5 displays the influence of acute sleep quantity strata on DC potential outcomes. Overall, there was a significant effect of acute sleep duration on DC potential ($F_{3,16}$= 4.68, $p$ = 0.02, $\eta_p^2$ = 0.47, power = 0.80), where $\eta_p^2$ indicated a large effect size. Additional pairwise analysis indicated that when compared to sleeping < 6 hours, sleeping ≥ 7 hours and < 9 hours led to a significant increase (mean increase = 17.10%)

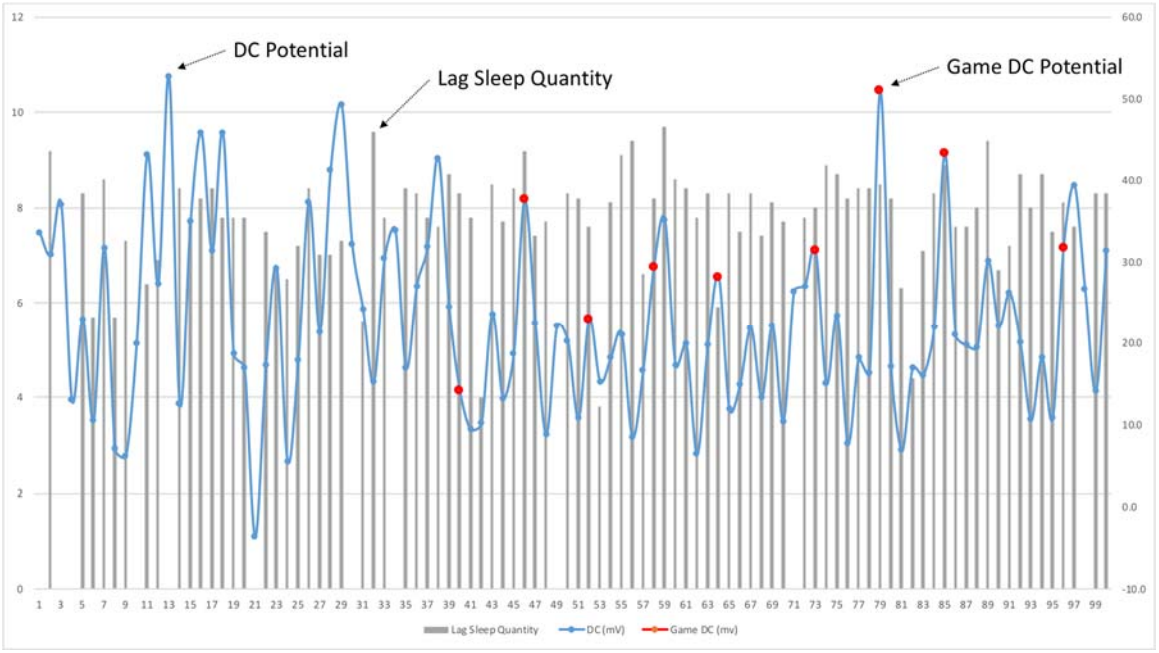
in DC potential of 3.59 mV ($p < 0.01$, Cohen's $d = .52$, SE = 1.18), where Cohen's $d$ suggested a moderate to large effect size. Sleeping ≥ 7 hours and < 9 hours led to an average DC potential of 21.03 mV (N = 19, SD = 7.33 mV) compared to 17.44 mV (N = 19, SD = 6.45 mV) when sleeping < 6 hours.

Table 5. Direct current potential outcomes by acute sleep quantity strata in 24 male collegiate American football players.

| | Hours of Acute Sleep | | | | |
|---|---|---|---|---|---|
| | < 6 | 6-7 | 7-9* | 10-11 | ≥ 11 |
| Mean | 17.44 | 17.81 | 21.03 | 23.96 | 16.66 |
| SD | 6.45 | 7.90 | 7.33 | 9.00 | 11.11 |
| 95% Conf. | [14.33, 20.54 ] | [14, 21.63] | [17.49, 24.56] | [15.57, 32.34 ] | [-83.15, 116.47 ] |

*Significant increase in DC potential compared to sleeping < 6 hr ($p < 0.01$)

Figure 4. A representative subject's time course of acute sleep quantity and direct current potential across the pre-season training camp and competitive season.




*Extended Sleep Quantity Versus DC Potential*

The average extended sleep quantity was 6.79 ± 1.14 hr (SE = 0.03 hr) in all subjects. Table 6 displays the influence of extended sleep duration strata on DC potential outcomes. There was a significant effect of extended sleep duration on DC potential ($F_{2,17}$ = 7.71, $p$ <.005, $\eta_p^2$ = 0.48, power = 0.90), where $\eta_p^2$ indicates a large effect size. Further pairwise analysis indicated that sleep durations of ≥ 7.5 hours and < 9 hours led to a significant increase (mean increase = 20.0%) in DC potential of 4.53 mV ($p$ < .002, Cohen's $d$ = 0.6, SE = 1.13) compared to sleeping < 6 hours, where Cohen's $d$ suggests a moderate to large effect size. Extended sleep durations ≥ 7.50 hours and < 9

hours led to an average DC potential of 22.59 mV (N = 19, SD = 8.43 mV) compared to 18.06 mV (N = 19, SD = 6.80 mV) for extended sleep durations of < 6 hours.

Table 6. Direct current potential outcomes by extended sleep quantity strata in 24 male collegiate American football players.

| | Hours of Extended Sleep | | |
|---|---|---|---|
| | *< 6* | *6-7.5* | *7.5-9** |
| Mean | 18.06 | 19.92 | 22.59 |
| SD | 6.8 | 6.67 | 8.43 |
| 95% Conf. | [14.78, 21.33] | [16.70, 23.13] | [18.53, 26.65] |

*Significant increase in DC potential compared to sleep durations < 6 hr ($p < 0.002$)

Acute Sleep Quality Versus DC Potential

Mean acute sleep quality outcomes are provided in Table 7. Tables 8, 9, and 10 display DC potential outcomes by acute sleep quality strata for wake episodes, sleep latency and efficiency, respectively.

There was not a significant effect of acute sleep wake episodes ($F_{2,19}$ = 1.83, $p$ = 0.19, $\eta_p^2$ = 0.16, power = 0.33; Table 8), sleep latency ($F_{2,21}$ = 1.10, $p$ = 0.35, $\eta_p^2$ = 0.09, power = 0.22; Table 9), or efficiency ($F_{2,15}$ = 1.78, $p$ = 0.20, $\eta_p^2$ = 0.19, power = 0.31; Table 10) on DC potential.

Table 7. Acute and extended sleep quality outcomes in 24 male collegiate American football players.

| | Mean | SD | 95% CI |
|---|---|---|---|
| Acute Latency (min) | 29.13 | 25.75 | [27.68, 30.57] |
| Extended Latency (min) | 28.29 | 18.91 | [27,2, 29.39] |
| Acute Wake Episodes | 4.17 | 2.83 | [4.01, 4.33] |
| Extended Wake Episodes | 4.12 | 2.34 | [3.98, 4.25] |
| Acute Efficiency (%) | 81.73 | 9.18 | [81.22, 82.25] |
| Extended Efficiency (%) | 81.97 | 7.29 | [81.55, 82.39] |

Table 8. Direct current potential outcomes by acute wake episode (>5 min) frequency strata in 24 male collegiate American football players.

| | Wake Episodes (>5 min) | | |
|---|---|---|---|
| | 0 - 1 | 2 - 3 | ≥ 4 |
| Mean | 19.18 | 19.47 | 20.86 |
| SD | 7.27 | 6.5 | 7.35 |
| 95% CI | [15.87, 22.49] | [16.54, 22.41] | [17.52, 24.21] |

Table 9. Direct current potential outcomes by acute sleep latency strata in 24 male collegiate American football players.

| | Latency (min) | | |
|---|---|---|---|
| | 0 - 30 | 31 - 45 | ≥ 46 |
| Mean | 19.57 | 20.25 | 21.63 |
| SD | 6.67 | 8.08 | 7.98 |
| 95% CI | [16.68, 22.45] | [16.76, 23.75] | [18.18,25.08] |

Table 10. Direct current potential by acute sleep efficiency categories in 24 male collegiate American football players.

| | Efficiency (%) | | |
|---|---|---|---|
| | ≤ 64 | 65 - 84 | 85 - 100 |
| Mean | 18.19 | 20.52 | 20.58 |
| SD | 8.38 | 6.68 | 6.76 |
| 95% CI | [13.88, 22.5] | [17.09, 23.96] | [17.11, 24.05] |

Extended Sleep Quality Versus DC Potential

Mean extended sleep quality outcomes are provided in Table 7. Tables 11, 12, and 13 display DC potential outcomes by extended sleep quality strata for wake episodes, sleep latency and efficiency, respectively. There was a significant effect of extended wake episodes on DC potential ($F_{2,19}$ = 4.50, $p$ = 0.025, $\eta_p^2$ = 0.32, power = 0.70), where $\eta_p^2$ indicated a large effect size. Further pairwise analysis revealed that ≥4 wake episodes led to a significant increase (mean increase = 12%) in DC potential of 2.55 mV ($p$ = 0.02, Cohen's $d$ = 0.34, SE = 0.86) compared 2 to 3 wakes episodes, where Cohen's $d$ suggested a small to moderate effect size. Extended wake episodes (≥4) led to an average DC potential of 21.17 mV (N = 21, SD = 6.42 mV) compared to 18.62 mV (N = 21, SD = 8.40 mV) for 2 to 3 extended wake episodes.

There was not a significant effect of sleep latency ($F_{2,19}$ = 1.88, $p$ = 0.18, $\eta_p^2$ = 0.17, power = 0.34; Table 12), or sleep efficiency ($F_{2,6}$ = 3.87, $p$ = 0.08, $\eta_p^2$ = 0.56, power = 0.47; Table 13) on DC potential.

Table 11. Direct current potential by extended wake episode (>5min) strata in 24 male collegiate American football players.

| | Wake Episodes (>5 min) | | |
|---|---|---|---|
| | 0 - 1 | 2 - 3 | ≥4* |
| Mean | 19.31 | 18.62 | 21.17 |
| SD | 8.21 | 8.35 | 6.42 |
| 95% Conf. | [15.58, 23.05] | [14.82, 22.42] | [18.25, 24.09] |

*Significant increase in DC potential compared to 2-3 wake episodes (p = 0.02)

Table 12. Direct current potential by extended sleep latency strata in 24 male collegiate American football players.

| | Latency (min) | | |
|---|---|---|---|
| | 0 - 30 | 31 - 45 | ≥ 46 |
| Mean | 20.28 | 22.22 | 21.1 |
| SD | 6.17 | 6.18 | 9.21 |
| 95% CI | [17.47, 23.09] | [19.4, 25.03] | [17.47, 23.09] |

Table 13. Direct current potential by extended sleep efficiency strata in 24 male collegiate American football players.

| | Efficiency (%) | | |
|---|---|---|---|
| | ≤ 64 | 65 - 84 | 85 - 100 |
| Mean | 27.84 | 20.26 | 22.99 |
| SD | 6.74 | 3.65 | 7.98 |
| 95% CI | [22.2, 33.47] | [17.21, 23.31] | [16.32, 29.66] |

Discussion

The purpose of this study was to examine the influence of acute (one-night) and extended (two-night) sleep outcomes on DC potential. Based on the NSF's quantity recommendations, it was hypothesized that moderate amounts of acute and extended sleep (7-9 hr) would enhance DC potential outcomes. The results of this study suggest that acute sleep durations between 7-9 hours yielded greater DC potential outcomes, compared to non-NSF recommended sleep durations of <6 hours. Thus, the NSF recommendation appears to correspond to sleep requirements necessary to improve the functional state of the athlete. In addition, the results of this study indicate that extended (two-night) sleep durations of ≥7.50 hours and <9 hours yield greater DC potentials compared to two-day average sleep durations of <6 hours. Similar to the acute sleep assessment, extended sleep quantity outcomes matching the NSF recommendations for health benefits also appear to enhance the functional state of the athlete.

DC potential is viewed as a “universal language” that is most appropriate for the study of the "cortico-subcortical organization of the cerebral systems underlying different functional states (sleep and wakefulness), subjective feelings (emotional states), and associative-mnestic and cognitive activities” (95, 101). Therefore, sleep quantities that optimize DC potentials may be related to improved alertness, reaction time, psychomotor vigilance, learning, and cognition. In the largest study to date, which objectively measured real-world sleep and performance, Althoff and colleagues (8) demonstrated that performance is influenced by prior sleep timing and duration, chronotype (morning/evening preference) and circadian rhythms. Over 18 months, 3 million nights of sleep were tracked by wearable sensors from 31 thousand users. Also, "…75 million subsequent real-world performance measurements based on keystrokes and clicks within a web search engine" were obtained. The process of entering a query for completion by the search engine and the subsequent response time for choosing a search result captured performance on two different tasks that rely on, “…different mixes of sensing, reflection, planning and formulating, executing, and monitoring motor plans”(8). Althoff et al. (8) found that keystroke and click time varies with both acute and extended sleep. An inverted U-shaped relationship was established for performance, with 7.00 – 7.50 hours being the optimal duration of sleep. Also, sleeping less than 6 hours and more than 9 hours resulted in significantly impaired performance. Their findings correspond to some of the findings of this study that indicate that acute sleep durations of less than 6 hours result in significantly lower DC potential.

The NSF does not recommend sleeping less than 6 or more than 11 hours for young adults (18-25 yr), as this may lead to serious health problems (86). The NSF's recommendation to avoid durations less than 6 hours corresponded with the data in the present study in which acute sleep durations of less than 6 hours resulted in significantly reduced measures of DC potential. The NSF recommends 7 to 9 hours of sleep for young adults, with 6 to 7 hours and 10 to 11 hours of sleep being potentially appropriate. The findings of this study indicate that sleep between 7 and 9 hours yielded improved measures for DC potential. Additional research with more subjects is needed to identify specific hour-to-hour timeframes of acute and extended sleep for optimal improvement in the functional state.

Althoff et al. (8) also determined that two consecutive nights of insufficient sleep (<6 hours), were associated with significantly decreased performance for six days. The researchers concluded that it took three nights of sufficient sleep (≥6 hours) to compensate for one night of insufficient sleep, and six nights to offset two nights in a row of insufficient sleep (8). The findings of the present study concur with Althoff et al. (8) in that a two-day average of less than 6 hours resulted in significantly lower values for DC potential.

Sleep restriction (SR) has been demonstrated to have deleterious effects on numerous physiological and psychological processes such as immune function, cardiovascular function, motor tasks, cognitive tasks and mood (118, 159). Furthermore, sleep restriction can suppress brain cell proliferation and induce an allostatic overload. The stress that is induced on the brain by SR leads to increased

proinflammatory cytokines, which can cause damage to brain structures and promote systemic inflammation (118). Recently, Lee and colleagues (118) reported that one month of sleep restriction (≤5.50 hours) resulted in functional impairments of the brain through the reduction of rewiring of neuronal fibers in structural brain networks and the reduction of potential alternate neuronal pathways. This may partially explain why DC potential is significantly reduced in sleep durations less than 6 hours for both acute and extended time frames.

Tononi and Cirelli (213) introduced the synaptic homeostasis hypothesis (SHY) which asserts that "...sleep is the price the brain pays for plasticity" (213). As the human organism engages with the environment, new sensory input is synthesized and results in synaptic strengthening. Learning and memory is primarily mediated by the mechanism of synaptic strength perturbations (50). According to Tononi and Cirelli (213) , "SHY proposes that the fundamental function of sleep is the restoration of synaptic homeostasis, which is challenged by synaptic strengthening triggered by learning during wake and by synaptogenesis during development". Learning then should occur primarily through synaptic potentiation during waking hours, therefore, increasing net synaptic strength. Because the animal brain is disconnected from the environment during sleep, synaptic normalization could occur during this period through net weakening (49).

In an exciting new paper by de Vivo and colleagues (2017), synaptic scaling during sleep was confirmed by measuring 6920 synapses in mouse motor and sensory cortices (50). Using three-dimensional electron microscopy, it was determined that axon-spine interface (ASI) size decreased by ~18% after sleep compared to wake.

These proportional changes are indicative of scaling. Also, the scaling occurred in ~80% of the synapses suggesting selective renormalization which would favor memory consolidation, integration, and “smart” forgetting(50).

Because DC potential is considered the “universal language” for the study of the cortico-subcortical organization of cerebral systems underlying different functional states including memory and cognitive activities, DC potential could serve as an indirect measure of the restorative nature of the synaptic renormalization process. Because DC potential represents the level of the frontal brain's system that comprises the integrative center, it could be considered a marker of restorative sleep that mediates learning and memory consolidation, both of which are crucial to the training process. Future studies examining sleep time durations and synaptic scaling differentials may shed light onto why DC potentials are useful in the use of fluid periodization for the enhancement of the training process (141).

In addition to investigating sleep quantity, it was hypothesized that NSF recommended ranges for sleep latency, number of wake episodes after sleep onset (>5 min) and sleep efficiency would yield favorable DC potential outcomes. A significant effect was not found for acute sleep latency, efficiency, or wake episodes, on DC potentials, nor was there a significant effect found for extended sleep latency and efficiency on DC potentials. However, the results of this study indicated that for extended periods of sleep, ≥4 wake episodes yielded greater DC potential outcomes compared to 2 to 3 wake episodes.

Poor sleep quality and sleep disturbances are common in athlete populations (32, 204). Athletes may experience disturbed sleep quality for numerous reasons including, pre-competition anxiety, diet, changes in time zone and sleeping in unfamiliar environments (60). Erlacher et al. (60) reported on findings amongst 632 German athletes and found that 66% slept worse than normal at least once the night before an important competition. Of the 416 athletes in this subset, 80% reported problems falling asleep and 32% reported waking up at night (60). As previously stated, SR has been demonstrated to negatively affect normal physiological processes (160, 180) including immune (227), metabolic and endocrine (194) function which could negatively impact stress adaptation and adaptive capacities.

Therefore, the extended sleep quality results indicating an improvement in the functional state with an increased incidence of wake episodes contradicts the acute/extended sleep quantity findings of this study. In the current study, only wake episodes that where longer than 5 minutes were counted as a wake episode. Thus, subjects were losing sleep quantity during the major sleep episode, as the number of wake episodes increased. All 24 subjects had data that were stratified in the category of ≥4 wake episodes in a sleep period. The average number of wake episodes (>5) minutes, in this stratum, was 5.63 ± 1.75 episodes (95% CI = 5.49, 5.76) per major sleep episode, and the average extended sleep quantity was 6.81 . The relationship of losing sleep quantity and improving DC potential is incongruent with the acute and extended sleep quantity results of this study.

As previously mentioned, there is currently one technical report validating the Readiband, and this report determined that the Readiband had an overall agreement level of 93%, combination of sleep detection sensitivity and identifying "IN-BED Asleep" periods, when compared to PSG (172). Recently, Dunican et al. (58) compared sleep measures from two different wrist-activity monitors, the Actigraph™ and Readiband, to PSG with 50 middle-aged adults in both a laboratory setting and at home. Both devices use accelerometers to define sleep as periods of "no movement" and wake periods as periods of "movement" (58). Paramount to the accurate assessment of sleep measurements (e.g., time in bed, sleep latency, sleep efficiency) using accelerometer-derived measurements of wake and sleep periods, is the accurate measurement of a "lights out" event (58). The ActiGraph™ combines diary-based information, identifying lights out, with accelerometer-based data to objectively measure sleep (58, 151) whereas the Readiband using proprietary scoring algorithms to identify the time of lights out.

Subjects wore both the Actigraph™ and the Readiband on the same wrist (non-dominant) for 8 consecutive nights, with the first night of sleep taking place in a laboratory where the two devices were simultaneously compared to PSG (58). The following seven nights were spent at home with subjects continuing to wear the two wrist-worn devices. Dunican and colleagues (58) determined that compared to the gold-standard for sleep measurement, laboratory based PSG, "…automated scoring algorithm estimation for time at lights out (as used by Readiband) was inaccurate compared to self-reported time at lights out (as used by Actigraph™) and technician

reported time of lights out (as used by PSG)". Sleep measures such as sleep latency and efficiency rely on a precise determination of time at lights for their accurate measurement. The authors did find that sleep measurements not relying on time at lights out for their calculation (e.g., sleep duration, wake time, time at sleep onset) were similar between the Readiband and Actigraph™. Critical to the findings in this study, both the Actigraph™ and Readiband overestimated sleep duration, and a proportional bias was observed for Actigraph™ vs PSG and Actigraph™ vs Readiband (58). Finally the authors concluded that the Readiband may be used in a similar capacity as the Actigraph for assessing sleep duration, however "…measures of sleep latency, sleep efficiency and wake after sleep onset should be interpreted with caution"(58).

Dunican and colleagues' (58) study highlights the challenges of objectively assessing sleep quality by unobtrusive means. At this time, PSG may be the best choice for objectively measuring sleep quality; however, it is an expensive and invasive process that is impractical for real-world data collection across multiple subjects in a training environment (175). Alternative, subjective methods, for sleep quality assessment need to be considered because sleep quality is an issue in the elite (116, 120) and sub-elite athlete (60, 126) populations . Mah et al. (126) recently reported that collegiate student-athletes "…generally experience poor sleep quality, habitually obtain insufficient sleep, and experience substantial levels of daytime sleepiness." Mah and colleagues examined sleep quality, quantity and daytime sleepiness among 628 Stanford University student-athletes using the Pittsburgh Sleep Quality Index (PQSI) and the Epworth Sleepiness Scale (126). It was found that 42% of the student-athletes

experienced poor sleep quality and that 39% regularly slept < 7 hours during the week. Finally, 51% of the student-athletes had Epworth scores ≥ 10, indicating high levels of daytime sleepiness (126).

There is a need for unobtrusive and accurate tools for assessing sleep behavior practices of elite athletes, as elite athletes demonstrate different sleeping patterns and habits compared to non-athlete populations (108, 116, 120). In addition, currently existing sleep screening questionnaires (i.e., Athlete Morningness/Eveningness Scale, Adjusted Neck Circumference, PQSI) have been demonstrated to have poor applicability to the elite athletic population (174). The Athlete Sleep Screening Questionnaire (ASSQ) is a valid tool for screening athletes for sleep disturbances. The ASSQ provides clinical screening with cut-off scores associated with specific clinical interventions to facilitate management of sleep disorders (174). Recently, Driller et. al (55) developed a valid and reliable tool, Athlete Sleep Behavior Questionnaire (ASBQ), that can differentiate between sleep practices of non-athletes and athletes and can identify maladaptive sleep in elite athletes. The ASBQ should receive strong consideration by coaches and practitioners for assessing sleep behaviors in the elite athlete population.

Most often in the literature, specifically cohort and population studies, time in bed and actual sleep time are not distinguished (86). Time in bed is typically greater than actually sleep time, which leads to an inflation in sleep duration estimates. However, intervention studies, using laboratory measured sleep quantities, will generally report shorter sleep durations (86). This is important to take into account when critically examining the acute and extended sleep results from this study.

Research indicates that when compared to objectively measured sleep durations, subjective reporting is overestimated and is only moderately correlated with objective measures (117). The Fatigue Science Readiband filters sleep latency, the length of time it takes to transition from full wakefulness to sleep, and only reports the sleep quantity as the total time asleep for the major sleep period. Therefore, the sleep quantity reported in this study is a more accurate measure of sleep duration and does not represent periods of transition or the subjects' perception of sleep quantity.

An important feature of this study is that it was conducted during the pre-season training camp and competitive season. The data collected includes periods of travel for competition, and changes in daily practice schedules which reflect the organic environment of collegiate athletics. It was the author's intentions to capture real-world biological responses to sleep and its relationship to training and competitive stress (Figure 4). Therefore, these findings should be considered useful as sleep prescription guidelines for collegiate American football players. Genetic factors should be considered, in that Division 1 football players, potentially possess greater adaptive reserves and are more resilient than the standard population. Therefore, caution is recommended in translating these findings to the general population.

There are limitations to the current study that should be noted. There were differences in the total number of Omegawave assessments and the total number of sleep episodes captured for each participant over the duration of the study. These variations are due to individual compliance in wearing the Readiband and performance of Omegawave assessments. The population cohort used in this study consisted of a

moderate size (N = 24) of Division 1 football players who were all members of the same NCAA football team. In addition, physical preparation and technical/tactical training loads were not controlled for, along with dietary intake. These variables could have impacted stress adaptations and future research should attempt to control for these confounding variables. Although the results provide valuable insight into the biological responses to acute and extended sleep durations, as previously stated, caution is recommended when generalizing beyond this specific population. Future research should use similar methodology to investigate biological responses to acute and extended sleep during off-season training periods of Division 1 football players, wherein deliberate means and methods are applied to elicit physiological, psychological and morphological adaptation. It should also be noted that each player displayed different average measurements of DC potential (Table 4). Because of genetic variation, diverse training history, nutrition habits and psychological strategies for managing stress, it is optimal that the coach or practitioner create individual sleep recommendations for each athlete. This recommendation should be derived from the statistical analysis of sleep quantity and biological readiness as assessed by the Omegawave AMS.

## Practical Applications

The findings of this study suggest that sleep quantity is associated with DC bio-potentials and thus the functional state of the athlete. Specifically, sleep durations between 7.00/7.50 to 9 hours correspond with higher measures of DC potentials

compared to lessor sleep durations. According the research by Dunican et al. (58), due to the substantial challenges with accurately and unobtrusively determining time at lights out, wrist-activity monitoring devices may not be optimal for assessing sleep quality. Therefore, subjective assessment tools, such as the ASBQ, may be the best option for sleep quality assessments. Finally, given the relationship between sleep quantity and biological markers for training adaptability, practitioners should prioritize sleep in the training process and educate athletes on proper sleep hygiene and sleep quantity to enhance their readiness to train.

# CHAPTER V

## SUMMARY AND CONCLUSION

In summary, the purpose of this study was to examine the effect of acute (one-night) and extended (two-night) sleep outcomes on DC potentials. Using the NSF's sleep quantity and quality recommendations, it was hypothesized that moderate time frames of acute and extended sleep quantity (7-9 hours) would produce greater DC potentials. In addition, it was hypothesized that NSF recommended ranges for sleep latency, number of wake episodes after sleep onset (>5 min) and sleep efficiency would be produce greater DC potentials. The results from this study confirmed our hypothesis that acute sleep durations between 7-9 hours corresponded with higher measures of DC potentials compared to lessor durations. In addition, extended sleep durations between 7.5-9 hours also corresponded wither higher measures of DC potentials compared to lessor or greater durations. Finally, there was not a significant effect of acute sleep quality on DC potentials, but the results of this study indicated that for extended periods of sleep, ≥4 wake episodes yielded greater DC potential outcomes compared to 2 to 3 wake episodes. There was not a significant effect of extended sleep latency and efficiency on DC potentials.

To the best of our knowledge, this is the first study, that has examined the effect of sleep quantity and quality on DC potentials to evaluate the functional state in collegiate athletes. The findings of this study suggest that sleep quantity influences DC

biopotentials and thus the functional state of the athlete. Although the precise mechanisms have yet to be fully elucidated by which sleep quantity mediates change in the functional state, it can be argued based on the veracity of the measurement of DC potentials that sleep effects the readiness to train in athlete populations.

The insignificant findings regarding the effect of acute and extended sleep quality and DC potentials may or may not accurately represent the reality of said relationship. Wrist-activity monitors are currently incapable of accurately measuring sleep quality variables, therefore unobtrusive and accurate tools for assessing sleep quality are needed for scientists and practitioners to evaluate these behaviors within the organic nature of the sporting environment. Athlete screening questionnaires, such as the ASSQ and the ASBQ, may be the best option until wearable technologies can accurately assess sleep quality.

In conclusion, adequate sleep has been demonstrated to be a critical component for normal psycho-physiological processes and it has been suggested to be one of the most effective recovery methods for athlete populations. The present study sheds light on the relationship between sleep and DC biopotentials and the effect on the functional state. Sleep, therefore, may be one the primary controllable factors in the training process which can enhance the readiness to train and adapt to psycho-physiological training loads.

Appendix A

Descriptive statistics (mean ± SD) of acute sleep latency and subject level DC potentials.

| Player ID | 0 - 30 | | | 31 - 45 | | | ≥ 46 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 16.04 | ± | 11.54 | 20.99 | ± | 12.11 | 20.65 | ± | 11.56 |
| 2 | 21.98 | ± | 10.45 | 11.38 | ± | 8.06 | 32.31 | ± | 13.06 |
| 3 | 17.22 | ± | 10.64 | 14.43 | ± | 5.87 | 15.28 | ± | 8.47 |
| 4 | 20.76 | ± | 12.34 | 21.38 | ± | 13.06 | 18.64 | ± | 10.14 |
| 5 | 29.40 | ± | 9.08 | 24.24 | ± | 6.21 | 25.36 | ± | 8.66 |
| 6 | 17.18 | ± | 9.04 | 20.99 | ± | 12.92 | 20.79 | ± | 11.05 |
| 7 | 18.97 | ± | 10.18 | 13.98 | ± | 11.51 | 18.99 | ± | 10.42 |
| 8 | 15.10 | ± | 14.13 | 9.01 | ± | 19.32 | 19.63 | ± | 13.76 |
| 9 | 20.72 | ± | 8.88 | 19.88 | ± | 15.26 | 26.79 | ± | 8.86 |
| 10 | 4.58 | ± | 10.96 | 17.19 | ± | 14.68 | 29.33 | ± | * |
| 11 | 18.20 | ± | 7.05 | 22.92 | ± | 17.44 | 11.29 | ± | 5.29 |
| 12 | 11.48 | ± | 11.41 | 16.47 | ± | * | 9.07 | ± | 6.15 |
| 13 | 26.85 | ± | 14.00 | 32.28 | ± | 13.19 | 26.38 | ± | 11.88 |
| 14 | 21.14 | ± | 9.76 | 28.28 | ± | 6.52 | 27.04 | ± | 12.29 |
| 15 | 24.58 | ± | 11.70 | 24.00 | ± | 11.97 | 23.00 | ± | 11.02 |
| 16 | 10.43 | ± | 9.16 | 9.47 | ± | 7.05 | 5.64 | ± | 9.29 |
| 17 | 17.96 | ± | 9.10 | 31.82 | ± | 7.11 | 19.29 | ± | 9.39 |
| 18 | 32.88 | ± | 10.72 | 34.22 | ± | 10.45 | 35.32 | ± | 12.74 |
| 19 | 10.21 | ± | 10.28 | 2.50 | ± | 7.95 | 12.17 | ± | 3.43 |
| 20 | 20.94 | ± | 10.12 | 16.15 | ± | 10.67 | 16.60 | ± | 6.92 |
| 21 | 21.39 | ± | 10.04 | 22.98 | ± | 11.72 | 23.37 | ± | 9.70 |
| 22 | 29.08 | ± | 10.60 | 30.29 | ± | 10.07 | 36.99 | ± | 4.31 |
| 23 | 22.97 | ± | 8.16 | 20.96 | ± | 13.41 | 23.61 | ± | 7.95 |
| 24 | 10.76 | ± | 8.60 | 15.12 | ± | 9.45 | ** | ± | ** |

* Only one sample for this duration

** No data available for this duration

Descriptive statistics (mean ± SD) of acute wake episodes (> 5 min) and subject level DC potentials.

| Player ID | 0 - 1 | | | 2 -3 | | | ≥ 4 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 12.30 | ± | 11.41 | 17.96 | ± | 9.92 | 19.52 | ± | 12.29 |
| 2 | 25.38 | ± | 11.39 | 18.67 | ± | 13.70 | 23.98 | ± | 10.56 |
| 3 | 18.28 | ± | 10.80 | 14.31 | ± | 7.92 | 15.50 | ± | 9.01 |
| 4 | 21.59 | ± | 10.20 | 18.80 | ± | 13.71 | 20.58 | ± | 11.46 |
| 5 | 28.58 | ± | 9.53 | 26.51 | ± | 7.40 | 30.16 | ± | 12.18 |
| 6 | 13.60 | ± | 9.92 | 18.50 | ± | 10.12 | 23.12 | ± | 11.02 |
| 7 | 20.04 | ± | 12.91 | 15.13 | ± | 11.34 | 18.18 | ± | 10.58 |
| 8 | 8.17 | ± | 14.20 | 17.01 | ± | 15.09 | 19.61 | ± | 13.69 |
| 9 | 20.40 | ± | 8.69 | 20.70 | ± | 8.62 | 22.53 | ± | 11.48 |
| 10 | 4.70 | ± | 8.47 | 13.19 | ± | 17.22 | 2.37 | ± | 6.03 |
| 11 | 14.61 | ± | 6.64 | 16.31 | ± | 5.75 | 19.95 | ± | 14.85 |
| 12 | * | ± | * | 6.87 | ± | 6.58 | 12.02 | ± | 10.69 |
| 13 | 24.87 | ± | 10.83 | 26.23 | ± | 12.60 | 28.47 | ± | 14.15 |
| 14 | 18.55 | ± | 10.91 | 19.16 | ± | 13.42 | 23.81 | ± | 9.10 |
| 15 | 20.78 | ± | 12.46 | 26.80 | ± | 11.05 | 24.41 | ± | 11.15 |
| 16 | * | ± | * | 10.58 | ± | 10.22 | 9.65 | ± | 8.85 |
| 17 | 18.19 | ± | 10.78 | 21.02 | ± | 9.19 | 17.52 | ± | 8.98 |
| 18 | 32.02 | ± | 10.63 | 33.69 | ± | 13.21 | 34.29 | ± | 9.67 |
| 19 | 8.98 | ± | 16.70 | 5.58 | ± | 8.96 | 10.99 | ± | 9.89 |
| 20 | 15.92 | ± | 6.59 | 16.58 | ± | 14.24 | 20.70 | ± | 9.49 |
| 21 | 23.10 | ± | 7.09 | 23.00 | ± | 9.33 | 21.38 | ± | 10.99 |
| 22 | 31.74 | ± | 6.96 | 28.73 | ± | 11.93 | 31.12 | ± | 9.67 |
| 23 | * | ± | * | 27.18 | ± | 4.88 | 22.17 | ± | 8.60 |
| 24 | 21.03 | ± | 7.60 | 11.01 | ± | 8.87 | 9.87 | ± | 8.04 |

*No data available for this range

Descriptive statistics (mean ± SD) of acute sleep efficiency and subject level DC Potentials.

| Player ID | ≤ 64 | | | 65 - 84 | | | 85 - 100 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 14.84 | ± | 6.96 | 19.11 | ± | 12.67 | 16.15 | ± | 9.75 |
| 2 | ** | ± | ** | 27.22 | ± | 14.55 | 21.00 | ± | 10.67 |
| 3 | ** | ± | ** | 14.13 | ± | 8.49 | 17.12 | ± | 9.90 |
| 4 | 24.62 | ± | 12.98 | 19.10 | ± | 12.34 | 21.61 | ± | 8.42 |
| 5 | ** | ± | ** | 29.60 | ± | 5.61 | 27.57 | ± | 9.33 |
| 6 | 13.47 | ± | * | 20.44 | ± | 10.67 | 17.49 | ± | 11.46 |
| 7 | 23.24 | ± | 12.66 | 16.53 | ± | 10.10 | 18.99 | ± | 7.81 |
| 8 | 9.57 | ± | * | 22.11 | ± | 14.78 | 12.11 | ± | 14.12 |
| 9 | 23.84 | ± | * | 20.71 | ± | 10.51 | 23.55 | ± | 9.29 |
| 10 | ** | ± | ** | 16.68 | ± | 13.74 | 4.69 | ± | 11.35 |
| 11 | 14.71 | ± | 10.11 | 21.02 | ± | 13.97 | 15.69 | ± | 5.10 |
| 12 | 10.54 | ± | * | 10.53 | ± | 10.35 | 13.40 | ± | 11.82 |
| 13 | 23.56 | ± | 11.53 | 28.02 | ± | 13.29 | 27.41 | ± | 14.48 |
| 14 | 12.09 | ± | * | 22.92 | ± | 9.72 | 22.52 | ± | 11.35 |
| 15 | 26.87 | ± | 7.02 | 22.79 | ± | 11.29 | 27.20 | ± | 13.60 |
| 16 | 17.35 | ± | * | 8.26 | ± | 9.00 | 11.68 | ± | 8.65 |
| 17 | ** | ± | ** | 14.97 | ± | 10.19 | 20.36 | ± | 8.87 |
| 18 | 30.95 | ± | * | 34.21 | ± | 11.65 | 33.40 | ± | 10.99 |
| 19 | ** | ± | ** | 9.70 | ± | 10.58 | 10.60 | ± | 9.37 |
| 20 | 17.95 | ± | 6.00 | 19.95 | ± | 10.94 | 18.65 | ± | 8.94 |
| 21 | ** | ± | ** | 21.91 | ± | 11.26 | 21.99 | ± | 9.50 |
| 22 | 26.79 | ± | 6.18 | 29.94 | ± | 8.74 | 31.18 | ± | 11.75 |
| 23 | 21.97 | ± | 8.48 | 22.44 | ± | 8.97 | 25.95 | ± | 3.79 |
| 24 | -3.12 | ± | ** | 10.83 | ± | 8.31 | 12.88 | ± | 8.77 |

* Only one sample for this duration

** No data available for this duration

Descriptive statistics (Mean ± SD) of extended sleep latency and subject level DC potentials.

| Player ID | 0 - 30 | | | 31 - 45 | | | ≥ 46 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 16.00 | ± | 11.54 | 20.56 | ± | 9.58 | 20.20 | ± | 14.15 |
| 2 | 20.17 | ± | 10.64 | 30.82 | ± | 13.15 | 22.26 | ± | 11.18 |
| 3 | 16.58 | ± | 10.55 | 18.52 | ± | 7.68 | 13.64 | ± | 6.95 |
| 4 | 20.28 | ± | 12.88 | 21.58 | ± | 10.94 | 17.59 | ± | 8.14 |
| 5 | 29.49 | ± | 8.81 | 25.24 | ± | 8.04 | 26.92 | ± | 10.49 |
| 6 | 18.26 | ± | 9.88 | 16.60 | ± | 13.24 | 22.38 | ± | 9.13 |
| 7 | 16.37 | ± | 10.72 | 18.13 | ± | 9.80 | 18.84 | ± | 8.81 |
| 8 | 12.38 | ± | 13.44 | 19.15 | ± | 17.65 | 27.61 | ± | 13.35 |
| 9 | 20.44 | ± | 10.61 | 22.84 | ± | 7.06 | 31.41 | ± | 8.30 |
| 10 | 5.95 | ± | 10.61 | ** | ± | ** | 28.45 | ± | 1.25 |
| 11 | 15.33 | ± | 8.57 | 20.62 | ± | 14.48 | 16.80 | ± | 10.65 |
| 12 | 12.47 | ± | 12.36 | 9.29 | ± | 5.29 | 7.84 | ± | 3.81 |
| 13 | 27.63 | ± | 13.30 | 20.77 | ± | 12.40 | 36.15 | ± | 10.30 |
| 14 | 20.96 | ± | 9.62 | 24.22 | ± | 15.32 | 27.24 | ± | 10.33 |
| 15 | 24.63 | ± | 11.45 | 23.34 | ± | 12.09 | 24.53 | ± | 8.59 |
| 16 | 8.44 | ± | 8.44 | 12.68 | ± | 9.30 | -2.60 | ± | * |
| 17 | 18.66 | ± | 8.55 | 24.95 | ± | 3.98 | 20.68 | ± | 1.93 |
| 18 | 34.48 | ± | 10.79 | 31.82 | ± | 9.84 | 34.04 | ± | 15.18 |
| 19 | 9.98 | ± | 10.12 | 7.65 | ± | 5.84 | ** | ± | ** |
| 20 | 21.53 | ± | 10.38 | 17.48 | ± | 6.27 | 16.58 | ± | 5.84 |
| 21 | 22.26 | ± | 11.19 | 22.89 | ± | 7.69 | 17.29 | ± | 9.84 |
| 22 | 27.28 | ± | 10.82 | 33.99 | ± | 8.31 | 34.88 | ± | 7.15 |
| 23 | 22.23 | ± | 7.89 | 31.03 | ± | 8.24 | 17.34 | ± | 6.64 |
| 24 | 10.86 | ± | 8.48 | ** | ± | ** | | ± | ** |

* Only one sample for this duration

** No data available for this duration

Descriptive statistics (mean ± SD) of extended wake episodes (> 5 min) and subject level DC potentials.

| Player ID | 0 - 1 | | | 2 -3 | | | ≥ 4 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 16.24 | ± | 8.00 | 14.59 | ± | 10.50 | 19.63 | ± | 12.47 |
| 2 | 22.92 | ± | 10.02 | 23.24 | ± | 14.14 | 23.06 | ± | 10.19 |
| 3 | 17.22 | ± | 11.50 | 16.18 | ± | 9.10 | 14.94 | ± | 7.63 |
| 4 | 20.45 | ± | 9.96 | 15.69 | ± | 12.60 | 22.87 | ± | 11.12 |
| 5 | 28.37 | ± | 9.46 | 26.68 | ± | 8.39 | 31.81 | ± | 9.08 |
| 6 | 17.20 | ± | 7.94 | 19.84 | ± | 10.71 | 19.23 | ± | 11.78 |
| 7 | 10.91 | ± | * | 13.94 | ± | 21.53 | 17.84 | ± | 9.58 |
| 8 | 6.04 | ± | 15.21 | 15.44 | ± | 14.40 | 18.74 | ± | 14.81 |
| 9 | 33.41 | ± | * | 17.40 | ± | 7.97 | 23.57 | ± | 10.57 |
| 10 | 6.91 | ± | 6.87 | 8.08 | ± | 16.08 | 11.96 | ± | 10.65 |
| 11 | 11.39 | ± | * | 15.31 | ± | 6.71 | 20.36 | ± | 14.45 |
| 12 | ** | ± | ** | 7.02 | ± | 4.99 | 11.94 | ± | 11.13 |
| 13 | 21.22 | ± | * | 27.54 | ± | 10.37 | 27.67 | ± | 14.21 |
| 14 | 31.06 | ± | * | 20.33 | ± | 14.32 | 22.34 | ± | 9.58 |
| 15 | 13.46 | ± | 3.30 | 24.45 | ± | 12.40 | 25.94 | ± | 9.94 |
| 16 | ** | ± | ** | 15.38 | ± | 13.75 | 8.89 | ± | 8.41 |
| 17 | 21.05 | ± | 10.07 | 19.82 | ± | 8.52 | 18.42 | ± | 7.47 |
| 18 | 29.70 | ± | 17.96 | 33.56 | ± | 10.94 | 33.84 | ± | 10.77 |
| 19 | 6.21 | ± | 14.45 | -4.75 | ± | * | 10.21 | ± | 9.73 |
| 20 | 16.75 | ± | 9.80 | 20.71 | ± | 10.98 | 19.95 | ± | 8.71 |
| 21 | 26.02 | ± | 19.01 | 21.30 | ± | 8.63 | 21.85 | ± | 10.84 |
| 22 | 22.76 | ± | * | 31.10 | ± | 10.55 | 29.94 | ± | 10.03 |
| 23 | ** | ± | ** | 31.45 | ± | 0.86 | 21.28 | ± | 8.03 |
| 24 | 26.26 | ± | * | 10.50 | ± | 7.12 | 10.36 | ± | 8.85 |

* Only one sample for this duration

** No data available for this duration

Descriptive statistics (mean ± SD) of extended sleep efficiency and subject level DC potentials.

| Player ID | ≤ 64 | | | 65 - 84 | | | 85 - 100 | | |
|---|---|---|---|---|---|---|---|---|---|
| 1 | ** | ± | ** | 18.38 | ± | 12.25 | 16.35 | ± | 10.12 |
| 2 | ** | ± | ** | 23.80 | ± | 15.15 | 22.73 | ± | 10.12 |
| 3 | ** | ± | ** | 13.17 | ± | 7.26 | 17.26 | ± | 9.96 |
| 4 | 37.57 | ± | 7.49 | 18.27 | ± | 11.38 | 26.28 | ± | 8.29 |
| 5 | ** | ± | ** | 26.30 | ± | 9.66 | 28.25 | ± | 8.85 |
| 6 | ** | ± | ** | 20.36 | ± | 10.39 | 15.88 | ± | 12.67 |
| 7 | 29.00 | ± | 9.09 | 16.90 | ± | 9.02 | 7.47 | ± | 7.64 |
| 8 | ** | ± | ** | 19.24 | ± | 14.70 | 13.11 | ± | 14.83 |
| 9 | ** | ± | ** | 21.65 | ± | 10.76 | 22.08 | ± | 8.01 |
| 10 | ** | ± | ** | 28.45 | ± | 1.25 | 5.95 | ± | 10.61 |
| 11 | 25.09 | ± | * | 17.55 | ± | 11.95 | 14.94 | ± | 5.02 |
| 12 | ** | ± | ** | 12.23 | ± | 10.84 | 4.91 | ± | 7.98 |
| 13 | 33.42 | ± | 15.16 | 27.07 | ± | 13.56 | 28.10 | ± | 12.19 |
| 14 | ** | ± | ** | 22.53 | ± | 9.41 | 21.10 | ± | 13.07 |
| 15 | 26.73 | ± | 1.87 | 24.03 | ± | 11.49 | 25.31 | ± | 10.82 |
| 16 | ** | ± | ** | 9.86 | ± | 9.31 | 8.44 | ± | 8.15 |
| 17 | ** | ± | ** | 19.90 | ± | 3.01 | 19.28 | ± | 9.16 |
| 18 | ** | ± | ** | 32.99 | ± | 10.51 | 34.11 | ± | 11.68 |
| 19 | ** | ± | ** | 9.78 | ± | 10.14 | 9.91 | ± | 9.63 |
| 20 | 24.55 | ± | * | 18.48 | ± | 7.93 | 22.03 | ± | 11.36 |
| 21 | ** | ± | ** | 22.06 | ± | 10.89 | 21.62 | ± | 10.09 |
| 22 | 31.16 | ± | * | 18.07 | ± | * | 30.54 | ± | 10.21 |
| 23 | 15.17 | ± | * | 21.68 | ± | 8.26 | 29.29 | ± | 4.08 |
| 24 | ** | ± | ** | 10.33 | ± | 8.59 | 11.77 | ± | 8.57 |

* Only one sample for this duration

** No data available for this duration

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