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Energy Availability and Sleep Quality on Training Responses and Sport Performance in Collegiate Division I Swimmers

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ABSTRACT

Given their frequent, intense training sessions and competitions, elite collegiate athletes often struggle to adequately fuel for their high energy needs, resulting in a high prevalence of low energy availability (EA) among elite collegiate athletes. In those with low EA, metabolic fuel repartitioning occurs to conserve energy, which, when left unaddressed, can have serious consequences on reproductive and bone health; meanwhile, low EA's impact on training adaptations and athletic performance is less well understood, making this issue one of great importance. Also common among elite collegiate athletes is poor sleep, often attributed to the time demands of balancing academic responsibilities with rigorous training schedules. Inadequate sleep has been found to impact health and appears to impair training responses and athletic performance, although more research is needed to understand the relationship between these variables. Additionally, sleep deprivation is associated with changes in metabolic hormones that are remarkably similar to the changes that occur with metabolic fuel repartitioning in response to low EA, making the relationship between EA, sleep, training responses, and sport performance even more interesting. Therefore, the purpose of this study was to determine the interrelationships between EA, sleep quality, training responses, and sport performance. To do so, this study used WHOOP wearable technology (WHOOP Inc., Boston, MA) to collect data on sleep quality and training (strain, average HR, and maximum HR). Three-day food logs were completed through the MyFitnessPal (Under Armour Inc; Baltimore, MD) app to collect energy intake data. Dual X-ray absorptiometry (DXA) was used to quantify body composition, and a 200-yard time trial swim time (TT_{perf}) was used to quantify athletic performance in 26 male and female NCAA Division 1 Swimmers during heavy training. Collection of EA was matched to sleep and training responses, but not matched to TT_{perf}. Sleep measures were also recorded on the day preceding TT_{perf}. Pearson correlations were utilized to determine relationships between variables when no sex effects persisted, whereas linear regression analyses were utilized to control for sex-differences. In all swimmers, there was a trend toward a correlation between EA and sleep duration_{hrs} (R=0.33; P=0.06). EA positively correlated with REM_{hrs} (R=0.64; p=0.001). When controlling for sex, EA was a predictor of SWS_{hrs} (R²=0.448; p=0.001). Also in all swimmers, sleep duration_{hrs} and sleep debt_{hrs} were related to strain (R=-0.85; p=0.01, R=0.35; p=0.045, respectively), and Ex_{HRavg} (R=-0.65; p=0.001, R=0.51; p=0.01, respectively). SWS_{hrs} was inversely related to Ex_{HRavg} (R=-0.41; p=0.04). When controlling for sex, sleep duration_{hrs} and SWS_% the night preceding the race predicted TT_{perf} (R² = 0.881; p<0.001 and R² = 0.883; p<0.001). Meanwhile, EA was not related to any training responses, and regression analyses revealed that there was no combined effect of EA or sleep quality variables that predicted training responses. We conclude that elite swimmers with lower EA exhibited worse sleep quality. Swim training responses were related to, and performance was predicted by, sleep quantity and quality. Therefore, to avoid negative consequences of poor sleep quality and low EA, athletes should get adequate sleep and consume adequate calories to support energy needs and optimize training and performance.

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Chapter 1

REVIEW OF LITERATURE

Statement of the Problem

The Female and Male Athlete Triad (Triad) is defined as the interrelationship of three conditions—energy availability, reproductive function, and bone mineral density—in which individuals can exist on a spectrum ranging from healthy to unhealthy for each of the three conditions with potential clinical outcomes of eating disorders, hypothalamic amenorrhea or hypogonadotropic hypogonadism, and osteoporosis ^[1, 2]. Due to the role that pressure to achieve a low body weight plays in the Triad's emergence, the Triad is often experienced in the extreme by those involved in aesthetic and endurance sports ^[3]. This makes Triad recognition and prevention education essential for coaches, trainers, and sports administrators.

The energy availability (EA) component of the Triad refers to the amount of dietary energy available for metabolism, thermoregulation, growth, and reproduction once exercise energy expenditure is accounted for, and low EA is the causal factor for development of the Triad condition.^[1] When an individual experiences intense, prolonged low energy availability, metabolic fuel repartitioning occurs to compensate for the under-fueling ^[4], leading to energy reallocation toward vital life-sustaining processes (including locomotion, thermoregulation, and cellular maintenance) and away from non-vital processes (including reproductive function and growth).^[1]

While the hormonal alterations that occur are by themselves not necessarily harmful, the metabolic fuel repartitioning caused by low EA can result in reproductive system suppression, thus, impacting the second component of the Athlete Triad: reproductive function ^[2, 4]. The Triad model was first developed in females, as low EA was demonstrated to impair menstrual function

^[5]. Impaired menstrual function, especially in severe cases such as with secondary amenorrhea, can have serious consequences when it comes to bone health ^[6]. When low EA leads to reproductive system inhibition, estrogen concentrations decline, resulting in an increased risk for alterations in bone health due to the protective nature of estrogen on bone ^[6]. Therefore, when in a low EA state, hypoestrogenemia combined with insufficient energy to rebuild bone breakdown, can result in bone mass loss, making the third component of the Triad range from optimal bone health to osteoporosis ^[6].

In 2021, the Female Triad was expanded to encompass its manifestations within male athletes, and the Male Athlete Triad model was developed based on the scaffolding from the female Triad model, with its three components of low EA, functional hypothalamic hypogonadism, and impaired bone health ^[2]. Further research, including randomized control trials, is currently underway to investigate the exact parameters and gender-specific nuances of the Male Athlete Triad ^[2].

Further studies are needed to accurately assess the extent to which the Triad components manifests in both male and female athletes; however, existing research suggests a concerning prevalence of Triad components, including low EA ^[7]. Because of the apparent prevalence of low EA and its related health consequences ^[6, 7], as outlined by the Triad ^[1, 2], further understanding the broader impacts of low EA on athletes, including their sleep health, is crucial. While existing studies provide some evidence of an association between low EA and poor subjective sleep quality, as assessed through sleep surveys ^[155], subjective measures of sleep have been shown to vary significantly from objective sleep measures ^[18, 19], and more research is needed to assess the relationship between low EA and objective sleep quality. Therefore, the first aim of this study is to examine the relationship between EA and objective sleep quality. Doing so will allow for a better understanding of the impacts of low EA on athlete health, thus, informing evidence-based athletic practices. Specifically, athletes may put greater effort into improving their fueling strategies in light of a relationship between EA and sleep health.

Just as research suggests that low EA is of concerning prevalence among athletes, studies also indicate that sleep deprivation is incredibly common, particularly among elite collegiate athletes who must balance academic responsibilities along with their rigorous sports training schedules ^[10, 11]. For instance, one study researching the prevalence of poor sleep health among collegiate athletes surveyed over a thousand athletes from four different NCAA institutions and found that around 25% of participants have a clinical sleep problem ^[12]. Yet another study investigating the sleep patterns of elite swimmers found the average sleep duration to be 5.4 hours per night during training periods—an average sleep duration so low that it, if prolonged, puts athletes at risk for maladaptive physiological and psychological outcomes ^[13] and poor performance. A third study researching the sleep duration of 18 collegiate athletes found an average sleep duration of only 6 hours during the nights before training days, which is, again, well below a healthy sleep amount ^[14]. Beyond inadequate sleep duration, research also suggests that poor sleep quality is prevalent among athletes ^[15, 16]. Specifically, one study comparing the sleep quality of elite athletes to the sleep quality of non-athletes found that elite athletes had more sleep fragmentation, higher sleep onset latency, and lower sleep efficiency, indicating lower sleep quality for elite athletes compared to the non-athlete group ^[16]. Given the prevalence of poor sleep, in terms of duration and quality, among collegiate athletes and elite swimmers in particular, elucidating the effects of sleep on collegiate swimmers is extremely relevant. Existing research suggests that shorter sleep duration and lower sleep quality correlate with poorer sport performance, which is unsurprising given that sleep is known to play a vital role is physical

recovery and restoration ^[17]. There are, however, a limited number of studies that have assessed the relationship between sleep and sport performance using objective measures of sleep rather than self-reporting methods ^[17, 101], and as mentioned previously, subjective measures of sleep have been shown to vary significantly from objective sleep measures ^[18, 19]. Therefore, the second aim of this study is to investigate the relationship between objective sleep quality and sport outcomes (i.e., training responses and sport performance). Furthermore, understanding how sleep impacts sport outcomes could result in improved evidence-based coaching, training, and athletic practices. For instances, learning of the impacts of sleep on training responses and athletic performance may prompt coaches to discuss sleep hygiene with their athletes or even move training schedules to allow for more sleep opportunities ^[13].

In addition to examining the relationship between EA and sleep quality and the relationship between sleep quality and sport outcomes, we are also interested in accessing the relationship between EA and sport outcomes. In the literature, existing studies provide evidence that impaired reproductive function and impaired bone health, both conditions related to low EA, are associated with poorer athletic performance, yet more research is needed to access the direct relationship between EA and sport outcomes.^[8,9] For this study, because dates associated with EA measures do not coincide precisely with each participant's performance measurement date, analysis of the relationship between EA and sport performance is beyond the scope of this study, so when it comes to investigating the relationship between EA and sport outcomes, will be assessed. Therefore, the third aim of this study is to examine the relationship between EA and training responses. Doing so will allow for a better understanding of the factors that influence training, thus, further informing evidence-based coaching and athletic practices. Specifically, understanding the relationship between EA

and training responses may motivate coaches to put greater effort into helping their athletes improve fueling strategies through emphasizing nutrition and providing athlete education.

Finally, given the high level of similarity between the metabolic alterations associated with low EA and poor sleep health discussed in detail in the next section, as well as the high prevalence of both low EA and poor sleep quality among elite collegiate athletes ^[7, 10, 11, 12, 15, 16], we are also interested in the combined effects of EA and sleep quality on training responses. Therefore, the fourth and final aim of this study is to investigate the combined effects of energy availability and sleep quality on training responses.

Our hope is that by investigating the relationships between EA, sleep quality, and sport outcomes through this study, evidence-based practices may be developed, motivating coaches, trainers, and athletes to adopted fueling and sleep habits that allow them to achieve more optimal performance and health.



Figure 1: Map of Aims

Background

Energy Availability

In examining the effects of EA on training responses in athletes, clear and complete understanding of EA and its current research is essential. In definition, EA refers to the amount of dietary energy remaining after accounting for the energy expended during exercise ^[20]. Therefore, to calculate EA, energy exercise expenditure is subtracted from energy intake, often expressed relative to the individual's fat free mass ^[21].

When EA becomes low, little energy is left to sustain physiology functions, which leads to metabolic fuel repartitioning as the body's way of adapting to survive using the limited energy available ^[22]. Specifically, leptin has been shown to decrease and ghrelin to increase in response to low EA ^[23, 24]. Research has also showed that in low EA states, insulin-like growth factor-1 (IGF-1) decreases while cortisol levels increase ^[25]. Additionally, luteinizing hormone pulse frequency and testosterone levels both have been shown to decrease in response to low EA ^[26, 27].

Such metabolic fuel repartitioning can, when combined, result in reproductive system suppression, which, when prolonged, has serious impacts on one's bone health, as explained above as it pertains to the Triad ^[4]. Decreased bone mass and weaken bones leave athletes more susceptible to bone stress injuries, which can force athletes to take time off for recovery, impede training sessions, and impair competitive performance ^[9, 28]. Not only can low EA indirectly lead to poorer performance through bone stress injuries, but research has shown that low EA is associated with a decrease in neuromuscular performance, as elite endurance athletes experiencing menstrual disturbances, indicative of a low energy state, were determined to have worse neuromuscular performance than their eumenorrheic counterparts ^[8]. Therefore, low EA during heavy training may serve as a mechanism whereby training adaptations are reduced, and

therefore training optimization becomes inadequate, leading to marginalized performance outcomes at critical athletic events during the competitive season.

Measuring Energy Availability

While the central role of EA in athlete health and performance as well as the calculation for EA (energy intake minus exercise energy expenditure, often expressed relative to the individual's fat free mass) have been established, how EA can be accurately measured is another matter that is important to consider when seeking to understand the EA literature.

First, directly calculating EA by measuring energy intake, exercise energy expenditure, and normalizing to fat-free mass brings several challenges. For one, measuring energy intake using food logs, for example, relies on self-reported methods, which are subject to bias as people tend to underreport dietary intake, leading to underestimates of true EA ^[29, 30]. Additionally, measuring exercise energy expenditure requires individuals to carefully track their exercise, which often relies on expensive technological exercise trackers that must be charged and worn appropriately in order to obtain accurate measurements ^[31]. Furthermore, directly calculating EA leads to an EA measurement that is sensitive to short-term changes in EA ^[31]. For example, if during the time of measurement, a person's energy intake increases dramatically compared to their typically energy intake, their calculated EA will reflect their short-term increased energy intake rather than an EA that is consistent for them throughout time.

Another method of assessing energy status is through calculating an individual's resting metabolic rate (RMR) ratio ^[32]. RMR refers to the amount of energy that the body uses while at rest ^[32]. Therefore, during periods of low EA, when body makes metabolic adjustments to survive using the limited available energy, RMR becomes suppressed independent of declines in

overall body weight ^[32]. If an individual's measured RMR is sufficiently below his or her predicted RMR, for example, this would lead to a calculated RMR ratio indicative of energy deficiency ^[32]. However, because RMR suppression is a metabolic response to prolonged low energy availability, RMR ratio represents a more chronic assessment of energy status compared to directly calculating EA based on daily intake and expenditure, and therefore may not reflect day-to-day variations in training, energy expenditure, and eating behaviors ^[34]. Secondly, RMR measurement requires the use of indirect calorimetry in which respiratory gas exchange is measured, in rate and type, to estimate metabolic rate ^[33]. This can be an expensive, timeconsuming, and often inaccessible procedure for coaches and athletes and therefore may not be practical for assessments during a training phase.

Energy Availability Threshold

Given that EA measurements, whether obtained via direct calculation or RMR ratio, are useful tools in assessing energy status ^[37], extensive research has been done to determine the EA threshold in which health consequences occur in order to provide clear, meaningful interpretation to such EA measurements ^[38]. While some researchers assert that 30 kcal/kg LBM/day is the EA threshold under which negative effects are observed, this cut-off is not consistent across individuals ^[38]. Instead, research has shown that some people experience negative effects of low EA with an energy availability higher than 30 kcal/kg LBM/day, meanwhile other individuals do not experience any consequences of low energy availability until their EA reaches a point under 30 kcal/kg LBM/day ^[38]. Given this finding, other researchers suggest that a sliding scale range, where a decrease in EA correlates with an increased risk of reproductive disruption, is a more accurate representation as opposed to a single threshold number ^[39].

Pathways To Low Energy Availability

Beyond the low EA consequences, measurements, and threshold considerations, another piece to consider when seeking to understand the issue of low EA is avenues to low EA. Put simply, for individuals to experience low energy availability, they must have inadequate energy intake to keep up with their energy expenditure. This can occur when individuals unintentionally do not consume enough energy to offset their expenditure, as is often the case for endurance athletes where high training volume leads to high energy expenditure ^[34]. Meanwhile, other individuals experience low energy availability as a result of purposefully restricting their energy intake, engaging in compulsive exercise, or a combination of the two, with several potential etiological factors at play, including social pressure, body dissatisfaction, athletic pressure, and body image ^[21, 34, 153]. Intentional food restriction can involve eating behaviors ranging from disordered eating to clinical eating disorders, both of which are more prevalent among athletes than non-athletes ^[34]. One study researching eating disorders and disordered eating behaviors among elite athletes illustrates this concerning prevalence, finding that around 25% of NCAA Division I collegiate athletes coming from a variety of sports, including swimming, exhibit disorder eating behaviors ^[40]. Compulsive exercise, on the other hand, is characterized by uncontrollable, excessive exercise behavior that often results in negative physical and emotional consequences including injuries and social isolation ^[153, 154]. Often, an individual's pathways to low EA involves a combination of disordered eating and disordered exercise behaviors, making it unsurprising that compulsive exercise has been found to be associated with eating disorder pathology.^[154]

When considering the several pathways to low EA, it is important to consider not only whether total daily energy balance is achieved when considering an entire day's intake and expenditure, but also whether with-in day energy balance is achieved, meaning energy balance is maintained throughout the day, and intake is properly periodized around exercise activity ^[37]. Research has shown that even if an athlete is consuming adequate fuel to meet his or her energy needs, if the athlete is not properly timing their energy intake for athletic activities, such as training sessions or athletic performances, there can be consequences ^[37]. Specifically, one study found that TT_3 , a hormone known to decrease in response to low EA ^[41], was positively correlated to hours in energy balance and negatively correlated with hours of negative hourly energy balance ^[42], reflecting the metabolic repercussions of failing to maintain within-day energy balance. This study also demonstrated that athletes with long-periods of negative energy balance (i.e., backloading eating behavior) resulted in significantly higher ratings of perceived exertion compared to athletes that ate more frequently throughout the day ^[42]. Another study researching the relationship between within-day energy balance and body composition found that participants who were determined to be in a negative hourly energy balance for fewer hours had higher lean body mass percent and lower body fat percent ^[43]. Given the competitive advantage that lean body composition offers athletes across a variety of sports, such a consequence of failing to maintain within-day energy balance is incredibly relevant. This finding highlights the importance of proper fueling amount and timing.

Sleep

Similar to the high prevalence of low EA among elite athletes ^[34], poor sleep health is likewise common among elite athletes and has serious consequences due to the growth, restoration, and immune function benefits of proper sleep that are missed during periods of poor sleep quantity and quality ^[44]. Therefore, between the high prevalence of poor sleep in athletic populations and the known health consequences of poor sleep ^[34, 44], the importance of understanding how sleep impacts athletes motivates this study's aim of investigating of the effect of sleep on training responses and athletic performance. In definition, sleep is an easily reversible state of reduced activity, decreased responsiveness to stimuli, and overall reduced consciousness that plays an important role in physical and mental recovery and restoration and memory consolidation ^[45]. Sleep is assessed through measurements of sleep quantity and quality ^[45].

Sleep Quantity

Adequate sleep quantity, defined as the total amount of time spent asleep per night, is crucial to achieving optimal health, while inadequate sleep quantity (i.e., sleep deprivation), has serious health consequences ^[46, 47]. Demonstrating such health consequences, a metanalysis of 153 studies on sleep duration outcomes found that short sleep duration was significantly correlated with diabetes mellitus, hypertension, cardiovascular disease, coronary heart disease, and earlier mortality ^[46]. While our study is interested in the training responses and sport performance consequences of poor sleep, rather than sleep deprivation's serious, long-term health consequences, extensive research exploring the mechanisms that give rise to such negative health outcomes provides insight into potential mechanisms in which inadequate sleep duration may impair sport performance. Specifically, the literature suggests that inadequate sleep duration is associated with a variety of health consequences due to the hormonal and immune system impairment that arise from inadequate sleep ^[46, 48]; meanwhile, the hormonal and immune system impairment arising from sleep deprivation may also be related to poor performance outcomes in athletes ^[17, 49]. Therefore, it is meaningful to further explore the literature on the hormonal and immune system changes that coincide with inadequate sleep duration.

When it comes to hormones, research has found that when individuals experience inadequate sleep duration, several hormonal aberrations are observed ^[50]. For one, sleep restriction has been shown to decrease leptin levels and increase ghrelin levels ^[51]. As well, inadequate sleep has been related to declines in anabolic hormones testosterone and insulin-like growth factor-1^[52, 53], while the catabolic hormone cortisol has been shown to increase with inadequate sleep ^[27]. Furthermore, consideration of such hormonal aberrations may reveal an avenue in which poor sleep impairs athletic performance ^[49]. Particularly, testosterone is known to play an important role in the muscle growth and bone growth and regulation in males and females ^[54, 55], processes vital to athletes' proper recovery and athletic performance ^[56]. Therefore, research demonstrating an association between short sleep duration and reductions in testosterone suggests a possible avenue in which inadequate sleep quantity may impair athletic performance.

When it comes to the impact of sleep duration on the immune system, researchers have found that short sleep duration is associated with a weaker immune response to vaccines, resulting in a decreased likelihood of vaccine-induced viral protection ^[57, 58]. Further supporting that poor sleep health weakens the immune system, other studies have found sleep deprivation makes individuals more vulnerable to infection, including the common cold, flu, strep throat, and gastroenteritis ^[59, 60, 61]. This is of particular importance to elite athletes whose training sessions and competitive seasons can be greatly affected by illness, as illness impacts athletes' ability to productively train in a variety of ways. For instance, one study showed that on the first day of having sandfly fever, a mild viral infection chosen for its high reproducibility, participants had up to 30% of a decrease in isometric and isotonic muscle strength ^[62]. While this decrease in muscle strength only reflects one day's abilities, other research finds that even after an infection

is over, decline in muscle strength can still be observed, having a correlation with the amount of muscle protein lost during the infection ^[63]. Therefore, depending on the length of time and severity of the infection, illness can set an athlete back considerable in training progress when it comes to muscular strength. When it comes to the impact of infection on aerobic exercise abilities, another researcher found that a febrile infection lasting for one week caused a 25% decrease in aerobic exercise capacity immediately after the infection was over within a previously healthy young adult population ^[64]. This study demonstrates the impact that infection has on athletic strength and endurance, emphasizing the importance of avoiding illness through immune system support within elite athlete populations ^[64]. Altogether, the literature finds that inadequate sleep duration is associated with immune system impairment, demonstrating another avenue in which poor sleep quantity impairs athletic performance.

Having considered several ways in which sleep deprivation impairs performance, it is unsurprising that the literature reveals an association between short sleep duration and impaired sport performance, particularly for aerobic or endurance sports ^[65, 66, 67, 68, 69]. One study investigating the relationship between sleep duration and performance of athletes in a national netball tournament found that there was a strong association between longer time asleep and tournament placement ^[67]. Additionally, a study examining the impact of 42 hours of sleep deprivation on the amount of time until exhaustion is reached while ergometer cycling found that sleep deprivation reduced time until exhaustion, suggesting that performance in endurance activities is impaired following sleep loss ^[68]. Furthermore, a study investigating the effect of extended sleep duration on athletic performance in elite collegiate swimmers found a correlation between extended sleep duration and several measures of performance, including faster sprint time, faster reaction time, faster turn time, and increased kick strokes, indicating that adequate sleep duration is associated with improved athletic performance ^[69]. Considered together, these studies support there being a significant association between short sleep duration and impaired sport performance.

Sleep Quality

Inadequate sleep quantity has several consequences for athletes, including impaired sport performance, elucidating the relationship between sleep and athletic performance requires the consideration of not solely sleep quantity but also sleep quality. Sleep quantity is defined as the total amount of time spent asleep per night, whereas sleep quality measures evaluate the extent to which a person's sleep is restful and restorative ^[70]. Subjective measures of sleep quality assess an individual's perception of their sleep experience, most often through surveys and questionnaires ^[71], and objective measures of sleep quality quantify the extent to which sleep is restful and restorative by evaluating related factors such as number of sleep disturbances, sleep latency, and sleep efficiency ^[72]. Additionally, sleep is a dynamic process involving an array of physiological changes across several stages where stages provide different benefits and contribute differently to restoration, so the amount of time spent in particular stages of sleep and the percent of sleep spend in those stages is also an important consideration when evaluating sleep quality ^[73]. Therefore, to understand the relationship between measures of sleep quality and athletic performance, the literature on sleep stage physiology, sleep stage benefits, particularly those relevant to sport performance, and existing research on the effects of sleep stages on sports performance must be explored.

Sleep Stages

Sleep stages are defined as phases of sleep characterized by distinct changes in brain wave activity, respiratory rate, blood pressure, heart rate, and hormones that occur throughout the night as part of healthy sleep ^[73, 74]. These stages cycle throughout the sleep period with the first cycle lasting 70 to 100 minutes and each subsequent cycle lasting 90 to 120 minutes ^[74]. A typical healthy adult experiences four to six complete cycles each night ^[75]. Several methods, including the usage of electroencephalograms (EEGs) to measure electrical brain activity via electrodes attached to the scalp, have allowed researchers to further understand and categorize distinct stages within the sleep cycle ^[76]. Using such measures, sleep experts have broken sleep down into two main types: non-rapid eye movement sleep and rapid eye movement sleep.

Non-Rapid Eye Movement Sleep (NREM)

Non-rapid eye movement sleep (NREM), true to its name, is the portion of sleep in which the eyes remain still, and this sleep type comes first in the sleep cycle ^[45]. Often described as synchronous, the depth of sleep increases during NREM sleep, which is observed through the increasing dominance of low frequency and high voltage EEG patterns ^[45]. Given the progression of depth that provides variability within the characteristics of NREM sleep, NREM sleep has been further broken down into three separate stages with stage 1 (N1) and stage two (N2) being light sleep and stage 3 (N3), also referred to as slow-wave sleep, being a period of deep sleep ^[75].

N1 and N2 Sleep

N1, which lasts only a few minutes, is entered immediately upon the onset of sleep ^[77]. During this stage, an individual's heartbeat, eye movements, brain waves, and breathing all begin to slow down ^[44, 75]. Next, N2 begins. This stage lasts much longer, as around half of a person's time asleep is generally spent in N2^[74]. During this time, many physiological changes occur as respiratory rate decreases, body temperature drops, muscles further relax, eye movements cease, and heartrate slows ^[44]. Brain waves further slowdown, yet bursts of brain activity called sleep spindles and long delta waves called K-complexes, which are believed to aid in blocking out stimuli and consolidating memories, occur periodically ^[78, 79]. Additionally, there is some evidence that N2 plays a role in learning procedural motor skills as related memories appear to be reprocessed in conjunction with K-complex activity during this stage ^[80]. Although current research is exploring the role of N2 in memory consolidation and motor activity ^[81], the most important role of N1 and N2 is believed to be the slowing down of physiological processes to prepare for the next stage of sleep: slow-wave sleep (SWS)^[73]. Research on the relationship between N1 and N2 sleep and athletic performance is very limited, as SWS and its benefits are believed to be very much relevant to health and athletic performance, making SWS the sleep stage most focused on in the sleep literature ^[44].

Slow-wave Sleep (SWS)

During SWS, also known as deep sleep, muscles begin to further relax, heart rate slows, and respiratory rate decreases ^[44]. In normal subjects, 10-25% of time spent asleep is SWS ^[82], and the length of SWS decreases as an individual moves through subsequent sleep cycles ^[83]. While N2 is distinguished by bursts of brain activity, SWS is named for the characteristic

presence of low frequency, high amplitude slow delta brain waves that occur throughout the stage, as observed through EEG ^[84]. This decreased brain activity taking the form of delta waves during SWS carries many benefits, and ultimately makes SWS the stage central to growth, healing, and immune function ^[85,86,87].

The growth and healing benefits of SWS can be understood in terms of a surge in human growth hormone, a hormone that plays an important role in the building and repairing of bone and muscle tissue ^[88], that is observed during SWS ^[85, 89]. One study investigating the relationship between human growth hormone secretion and delta wave EEG activity reported that growth hormone secretory pulses were concurrent with the delta wave peaks ^[89]. Furthermore, this study found a positive correlation between the amount of secreted human growth hormone and delta wave activity ^[89]. Because human growth hormone is known to play an important role in the building and repairing of bone and muscle tissue ^[88], such research demonstrating the association between SWS and human growth hormone secretion reaffirms the vital role of SWS in growth and healing. Furthermore, studies have found that if there is high energy expenditure throughout the day, as is common among elite endurance athletes, human growth hormone secretion will rise the following night ^[90]; however, when athletes obtain less slow-wave sleep, these levels of human growth hormone drop significantly ^[91]. Such studies demonstrate that the body is equipped to alter hormone secretion in response to exercise to allow for necessary physiological growth and repair, but inadequate sleep interferes, thus, impairing recovery. Moreover, building and repairing muscle tissue are vital processes for all individuals, but especially for elite athletes whose strenuous training sessions and competitions make proper growth and healing all the more important ^[50]. Without experiencing the proper recovery that adequate SWS allows for, athletes are missing opportunities to gain muscle and bone strength

while putting themselves at increased risk of injury ^[92], both serving as potential avenues for inadequate SWS to impair athletic performance.

Another major role of SWS relevant to athletic performance is immune function support ^[93]. Specifically, SWS plays a vital role in coordinating the immune system by promoting the migration of two types of immune cells, professional antigen presenting cells and T cells, to the lymph nodes ^[87]. During SWS, a hormonal network is created between professional antigen presenting cells and T cells to allow for immune system communication ^[87]. In this way, SWS is essential for its role in supporting the immune system.

The immune support offered by SWS is of particular importance to elite athletes whose training sessions and competitive seasons can be greatly affected by a weakened immune system that allows for more frequent illness, as illness impacts athletes' ability to productively train in a variety of ways ^[62, 63, 64]. Additionally, the competitive seasons of elite athletes are both physically and psychologically demanding ^[94, 95], resulting in greater stress experienced ^[96] and further increasing athletes' susceptibility to illness ^[97, 98, 99], making the immune support offered by SWS that much more important. Demonstrating the impact that illness has on athletes, one study showed that one the first day of having sandfly fever, participants had up to 30% of a decrease in isometric and isotonic muscle strength ^[62]. While this decrease in muscle strength only reflects one day's abilities, other research finds that even after an infection is over, decline in muscle strength can still be observed, having a correlation with the amount of muscle protein lost during the infection ^[63]. Therefore, depending on the length of time and severity of the infection, illness can set an athlete back considerable in training progress when it comes to muscular strength. When it comes to the impact of infection on aerobic exercise abilities, another researcher found that a febrile infection lasting for one week caused a 25% decrease in aerobic

exercise capacity immediately after the infection was over within a previously healthy young adult population ^[64]. This study demonstrates the expansive impact that infection has on athletic strength and endurance, emphasizing the importance of avoiding illness through immune system support within elite athlete populations. Coupled with understanding of the role of SWS in supporting the immune system, such research further highlights another potential avenue for inadequate SWS to impair athletic performance.

Furthermore, while there is convincing research demonstrating the role of adequate SWS in human growth hormone secretion and healthy immune function as well as research demonstrating the important role of human growth hormone secretion and healthy immune function on performance, there is limited research specifically investigating the relationship between slow-wave sleep and sport performance ^[100, 101], making this study a novel contribution to the literature.

Rapid Eye Movement Sleep (REM)

Following SWS, the sleep cycle continues, returning to the lighter stages of sleep, first N2 and then N1, before entering rapid-eye movement sleep ^[83]. Rapid eye movement sleep (REM), true to its name, is a type of sleep characterized by the alternation of phasic periods of quick eye movements in a range of directions and tonic periods of decreased movement ^[77]. Opposite to how the length of time spent in slow wave sleep decreases throughout subsequent cycles, the length of time spent in REM sleep increases in subsequent cycles throughout the sleep duration, overall making up around 20-25% of total sleep time for typical adults ^[75, 102]. During REM sleep, dreaming takes place and high levels of brain activity are observed ^[77]. In fact, EEG recordings taken during REM sleep contain beta waves similar to those observed during

wakefulness ^[75]. Furthermore, while the brain activity is high, the body is almost entirely still besides sporadic muscle twitches, which is why REM sleep is commonly referred to as an active brain in a paralyzed body ^[78]. When it comes to the benefits of REM sleep, research suggests that REM sleep's primary role is in memory processing and consolidation, especially for emotional memories ^[103]. Because the memories most relevant to sport performance, procedural motor memories, are processed during N2 sleep K-complex activity, while REM sleep concerns the processing of primarily emotional experiences ^[103], REM is believed to be far less impactful on athletic performance as compared to NREM sleep ^[100, 104]. As a result, the sleep literature lacks research investigating the relationship between REM sleep and athletic performance.

Sleep in Athlete Populations

Considering sleep quantity and sleep quality, including all the stages of sleep, as they relate to athletic performance, exploring the extent to which athletes achieve healthy sleep, as well as the unique barriers elite collegiate athletes face to obtaining adequate sleep, is crucial to understanding the importance of sleep-performance research. Research finds that athletes, particularly elite collegiate athletes whose sleep and performance will be investigated in this study, face unique challenges in maintaining healthy sleep habits ^[11]. A recent student investigating the sleep needs of 175 elite athletes found that, on average, athletes require 8.3 hours of sleep to feel rested ^[105], yet 39.1% of elite collegiate athletes report obtaining fewer than 7 hours of sleep during weekdays ^[11]. Furthermore, research investigating how well self-reported sleep reflects objective sleep measures finds that individuals significantly overestimate their sleep duration when self-reporting, indicating that elite collegiate athletes are likely obtaining even less sleep than reported ^[106].

There are multiple factors that appear to contribute to the poor sleep health among elite collegiate athletes, including their demanding schedules, difficulties of maintaining healthy sleep habits while traveling for competitions, and early morning training sessions that limit sleep opportunities ^[15, 44, 107]. While some factors cannot be modified, such as travel schedules that are predetermined and central to athletic competitions, other factors, such as early morning training sessions, could perhaps be adjusted to allow for more sleep opportunity. In fact, one study investigating the sleeping behavior of elite swimmers found that during night's preceding earlymorning training, swimmers obtained 5.4 hours of sleep, whereas they obtained 7.1 hours of sleep on night's preceding rest days ^[13]. This research indicates that early training sessions significantly reduce the amount of sleep obtained by elite swimmers ^[13]. Another study examining the impact of training sessions on sleep in elite athletes across seven sports found that the amount of sleep obtained prior to early training sessions was significantly lower than on rest days, and shorter sleep times were correlated with pre-training fatigue ^[108]. By continuing the investigation into the relationship between sleep and athletic performance through this study, the hope is to allow for a better understanding of sleep's influence on sport performance, which may create opportunity for improved coaching and athletic practices. Specifically, coaches may discuss sleep hygiene with their athletes or even move training schedules to allow for more sleep opportunities.

Measuring Sleep

While the importance of studying sleep and its connection to athletic performance has been established, how sleep can be accurately measured is another matter. There are two distinct categories of sleep measurement, subjective measures, which assess self-perceived sleep characteristics through surveys or sleep diaries, and objective measures, which utilize technology to assess sleep through quantitative data collection. Interestingly, studies have found that subjective sleep measures are poor predictors of objective sleep measures ^[18]. Specifically, one study reported that subjective sleep measures reflect how individuals view their sleep health, and are related to factors such as perceived overcommitment, self-reported happiness, and perceived level of social support, while is entirely different than objective sleep measures, which reflect actual quantitative sleep data and are unrelated to individuals' perceptions ^[19]. Because this study is interested in the relationship between sleep and athletic performance, rather than perceived sleep and athletic performance, objective measures are of greater interest when reviewing the literature. Nonetheless, in order to fully understand where sleep research has been and where it is going, discussion of subjective and objective measures is valuable.

When it comes to subjective sleep measures, the Pittsburgh Sleep Quality Index (PSQI) stands as the gold standard with a sensitivity of 89.6% and a specificity of 86.5% in distinguishing good and poor sleepers ^[109]. A 19-item questionnaire, the PSQI assesses several variables of sleep, including subjective sleep quality, sleep latency, sleep duration, sleep efficiency, and sleep disturbances, reflecting the respondent's view of their sleep over the span of the previous month ^[109]. Other surveys commonly utilized to assess respondent's view of their sleep include the Stanford Sleepiness Scale, the Epworth Sleepiness Scale, and the Jenkins Sleep Scale; however, the PSQI, as the gold standard for self-perceived sleep quality, remains the predominant subjective sleep measure tool in clinical and non-clinical subjects and is utilized as convergent validity for other questionaries ^[110, 111].

Meanwhile, when it comes to objective measures of sleep, in-laboratory polysomnography holds as the gold standard for its ability to measure sleep quantity and quality

with more accuracy than any other objective assessment tool ^[18]. Polysomnography accomplishes this feat by utilizing pulse oximetry, respiratory airflow, respiratory effort, electroencephalography, electrooculography, electromyography, and electrocardiography to monitoring a variety of bodily functions while a person sleeps overnight in a laboratory setting ^[112]. This data is then analyzed by a sleep technologist to provide accurate and detailed information on sleep onset latency, total sleep time, sleep efficacy, wake after sleep onset, REM sleep latency, number of wake periods, and percent of the sleep period time spent in each sleep stage ^[113]. While an incredibly powerful tool in sleep research, polysomnography is not without its limitations, as its high cost and laboratory setting requirement make it unsuitable for longerterm sleep monitoring and impractical for widespread use by those simply interested in examining their sleep health ^[114].

Actigraphy, another objective sleep measurement tool, is a more cost effective and practical alternative to polysomnography. By utilizing a small accelerometer-containing device, which typically takes the form of a wrist or ankle band, actigraphy measures a person's amount, timing, and type of movement ^[115]. These data are then run through specialized algorithms to calculate sleep parameters, including sleep onset latency, total sleep time, sleep efficacy, wake after sleep onset, and number of awakenings ^[116]. With the advantage of the devices being simple to use and able to be worn at home, actigraphy allow for objective sleep data to be collected in a person's natural environment over a longer period of time, providing data that is reflective of an individual's consistent sleeping behavior. Furthermore, when it comes to estimating sleep parameters, actigraphy has been well validated, and is widely accepted as a useful tool for measuring total sleep time, sleep percentage, and wake after sleep onset, as compared to the gold standard of polysomnography ^[115, 117]. Yet, because actigraphy assess sleep parameter using only

an accelerometer to collect movement data, the technology is limited to assessing sleep-wake, not sleep-stage, data ^[115].

In response to this limitation, sleep wearable technology has emerged, which combines actigraphy's utilization of an accelerometer to measure movement with photoplethysmography heart rate tracking technology—to allow for sleep stage categorization in additional to measuring the various other sleep parameter ^[118]. Specifically, photoplethysmography uses optics, often involving green or infrared LEDS and photodiodes, to detect changes in blood volume, allowing heart rate and heart rate variability data to be collected ^[118]. Using an algorithm, such data can then be related to sleep stages ^[119]. Additionally, while the validity of sleep wearable technology is dependent on the specific device and many devices on the market are still undergoing validation studies ^[120], several sleep wearable technology devices have been validated ^[118, 121]. Specifically, the WHOOP Performance Optimization system (WHOOP Inc., Boston, MA) is one wearable technology device that has been validated for its various sleep measures ^[118, 122, 123]. The ability of everyday consumers, including athlete populations to whom sleep health is especially critical, to obtaining objective sleep measurements through wearable technology devices, such as the WHOOP, offers individuals an opportunity to further understanding their own sleep health—a tool that, before now, has never been accessible to the masses.

WHOOP Wearable Technology

This study in particular utilized wearable technology to obtain sleep data via the WHOOP device (WHOOP Inc., Boston, MA). The WHOOP device incorporates a variety of technology, including a tri-axial accelerometer and temperature, optical, and touch sensors, to measure heart rate, heart rate variability, estimate caloric expenditure, skin temperature, blood oxygen, sleep

data, and derive a strain score, a recovery score, and a stress score with the goal of empowering athletes to optimize their health and performance (WHOOP Inc., Boston, MA). The WHOOP measures numerous variables during sleep, including measurement of total sleep time, sleep disturbances, sleep efficiency, amount and percentage of time in each of the four main stages of sleep (wake light sleep, slow-wave sleep, and REM sleep), and cycle duration. In order to provide such extensive sleep data, the WHOOP utilizes its optical sensors, specifically green/infrared LEDS paired with photodiodes, to gather photoplethysmography data and its triaxial accelerometer to gather movement data (WHOOP Inc., Boston, MA). This information is then run through a sleep staging algorithm to produce the various sleep measures (WHOOP Inc., Boston, MA).

The WHOOP has been validated against the gold-standard, polysomnography, for its measurement of sleep, including its four-stage categorization (wake, light sleep, slow-wave sleep, and REM sleep), when bedtimes were entered manually ^[122]. Additionally, another study, validated the WHOOP's sleep capture ability, including its four-stage categorization of sleep, against polysomnography with manually entered bedtimes as well as auto-detected bedtimes ^[118]. Moreover, a third study validated the WHOOP's sleep measures against polysomnography, finding that the WHOOP accurately measured sleep variables when compared against the gold standard ^[123]. Together, these validation studies support the utilization of the WHOOP as a useful sleep measurement tool, making it an ideal sleep wearable technology device for the purposes of this study.

Low Energy Availability and Poor Sleep Connections

Finally, in reviewing the literature related to the fourth and final aim of this study, examining the combine effects of EA and sleep quality on training responses, the connections between low EA and poor sleep must be evaluated.

As aforementioned, metabolic changes occur in response to poor sleep health ^[124]. Leptin levels have been shown to decrease while ghrelin levels increase during periods of sleep restriction ^[51], which is similar to the changes in leptin and ghrelin observed in response to low EA ^[23, 24]. Furthermore, in response to sleep deprivation, anabolic hormones testosterone and insulin-like growth factor-1 have been reported to decrease ^[52,53], while cortisol, a catabolic hormone, has been shown to increase ^[27], which is also similar to the changes in testosterone, insulin-like growth factor-1, and cortisol observed during periods of low EA ^[25, 27]. Such a remarkable overlap in the metabolic fuel repartitioning that occurs due to energy deficiency and sleep deprivation is depicted in Figure 2 below.


Figure 2. Energy Deficiency and Sleep Deprivation Metabolic Responses

Figure 2. Energy Deficiency and Sleep Deprivation Metabolic Reponses. This figure displays several metabolic changes that occur in response to energy deficiency and sleep deprivation. Responses related only to energy deficiency are recorded in yellow boxes, and responses related only to sleep deprivation are written in blue boxes, while metabolic responses common to both energy deficient and sleep deprived states are recorded in green boxes. Research supporting the occurrence of each metabolic response is referenced near each respective arrow.

Moreover, given that the metabolic fuel repartitioning resulting from low EA can lead to reproductive suppression and even impaired bone health, as outlined by the Triad, the overlap in the metabolic responses of energy deficiency and sleep deprivation is of great interest. Specifically, given the high level of similarity between the metabolic alterations associated with energy deficiency and sleep deprivation, it is possible that for an individual experiencing low energy availability, poor sleep may heighten the degree of metabolic fuel repartitioning ^[124] and possibly even heighten the manifestation of the Triad symptoms. Additionally, if an individual with low EA is experiencing reproductive suppression or poor bone health already, it is possible that inadequate sleep may heighten the degree of metabolic fuel repartitioning and make it more difficult for the individual to return to a healthy state of metabolic functioning that allows them

to recover from the Triad. Further, research is needed to explore these possibilities before any clear determination on the connection between energy availability and sleep can be made; however, the similarity of metabolic responses between low EA and sleep deprivation warrant the mention of such considerations.

Furthermore, these considerations are especially relevant to athletes, particularly endurance athletes, as they are at high risk for experiencing components of the Triad ^[7]. Given the high prevalence of the Triad among athletes and the severity of the Triad's health consequences when it comes to reproductive function and bone health, understanding possible factors that may worsen Triad or impair Triad recovery in athletes is of great importance. Furthermore, athletes, particularly elite collegiate athletes who are the population of interest for this study, are reported to frequently experience poor and insufficient sleep ^[11], making the investigation of sleep's possible effect on Triad components, through its metabolic fuel repartitioning similar to that of low EA, even more interesting.

Altogether, considering the high prevalence of both low energy availability and sleep deprivation among collegiate athletes, the several avenues in which low EA and poor sleep may independently impair performance, and the high degree of similarity in the metabolic effects of low EA and sleep deprivation ^[125, 126], the investigation of the combined effects of EA and sleep on training responses is of great interest, making it the final aim of this study.

Aim and Applications

The overall aim of this study is to assess the interrelationships between EA, sleep quality, and sport outcomes (i.e., training responses and sport performance) in collegiate Division 1 Swimmers. Specifically, the high level of similarity between the metabolic impacts of energy deficiency and sleep deprivation, along with the literature on EA and sleep as they pertain to sport outcomes, provides motivation to investigate the relationships between EA and sleep quality, sleep quality and sport outcomes (i.e. training responses and sport performance), EA and training responses, and the combined effects of EA and sleep quality on training responses.

Furthermore, this study providing further knowledge on low energy's association with impaired training responses may encourage coaches to put greater effort into helping their athletes improve fueling strategies through emphasizing nutrition and providing athlete education. One study investigating the impact of nutritional education on pre-professional ballet dancers found that following the implementation of nutritional education, dancers demonstrated improvements in dietary intake ^[127]. Another study assessing the effect of nutritional education on elite athletes across a variety of sports also found a significant increase in total energy intake following the nutrition education sessions ^[128]. Both of these studies support nutrition education interventions are a valuable tool in improving athlete EA, suggesting meaningful ways this research could be applied to athletics.

Similarly, this study providing further knowledge on the association between poor sleep and impaired training responses and sport performance may encourage coaches to discuss sleep hygiene with their athletes or even move training schedules to allow for more sleep opportunities ^[13, 129, 130]. One study investigating the impact of individualized sleep education on sleep quality in elite athletes found that following sleep hygiene education, there were significant improvements in increased sleep efficiency and decreased sleep latency among the athletes ^[129]. Another study evaluating the impact of sleep hygiene education on sleep quantity in elite athletes found that following a sleep hygiene education session, athletes exhibited a significant increase in total sleep time ^[130]. These studies suggest that sleep education as a valuable tool in improving athlete sleep health ^[129]. Additionally, a study investigating the effect of early training sessions on sleep quantity in elite swimmers by comparing sleep quantity during nights prior to early training sessions to sleep quantity during nights prior to rest days found that sleep quantity was significantly restricted by early training sessions ^[13]. This research suggests that scheduling training sessions to intentionally allow for more sleep opportunity may be another valuable tool in improving athlete sleep health ^[13]. Altogether, the finding that sleep hygiene education and more sleep-centered training session timing are potential strategies to improve athlete sleep health suggests meaningful ways that this study's expansion of the knowledge on the association between poor sleep and impaired training responses and sport performance could be applied to athletics.

In essence, understanding the interrelationships between EA, sleep quality, training responses, and sport performance will allow for more focused athlete education and intervention to improve EA and sleep health, allowing for the optimization of training and athletic performance.

Chapter 2

MATHERIALS AND METHODS

Overview

This study assessing the effect of energy availability (EA) and sleep quality and quantity on training responses and sport performance in elite NCAA Division 1 swimmers, is part of a larger cross-sectional study that measured energy, eating behaviors and attitudes, objective and subjective training load, body composition, metabolism, autonomic nervous system function, subjective stress and recovery, sleep, and swimming performance ^[131]. While all these variables were measured in the larger study, the analysis of energy data, performance data, and extensive sleep data obtained in this study via the WHOOP, including sleep stages, disturbances, and quantity, has yet to be explored.

While this manuscript contains only descriptions of the study procedures performed to collect data relevant to variables included in this analysis, i.e., to determine the effect of EA and sleep quality and quantity on training responses and athletic performance are included below, a comprehensive description of all study procedures of the larger study can be found in Lundstrom's *Effectiveness of Wearable Technology for Predicting Measures of Metabolism and Performance in Collegiate Division 1 Swimmers* [Master's thesis, The Pennsylvania State University].^[131]

Study Design

In order to assess the effect of EA and sleep quality and quantity on athletic performance in elite swimmers, this study employed a cross-sectional design. The participants for this study consisted of 27 NCAA Division I collegiate swimmers (11 males and 16 females), and data collection was carried out over a 6-week period. These 6 weeks of data collection aligned with the intensified training period of the athletes' season in which training load and intensity reached its highest point of the season just before the initiation of "taper" or a period of reduced training intensity to allow for rest in preparation for championship competitions. During the 6-week intensified training period in which all data collection occurred, participants were broken down into two separate waves of participants where each participant underwent 4 weeks of data collection. The first two weeks of data collection had two main objectives: to allow the WHOOP (Figure 3) to fully acclimate to the given participant and to establish participant compliance to wearing the device.

If the participant was compliant during the initial two weeks of data collecting, meaning that he or she wore the WHOOP (Figure 3) for at least 20 hours daily, as instructed, then the participant would continue on to the following 2, and final, weeks of data collection. During these two weeks, physiological data from the WHOOP (Sleep, Recovery, Strain, Heart Rate Variability, Resting Heart Rate), physiological data from laboratory testing (RMR, body composition, and blood sample), and measures of performance (200-yard freestyle time trial, subjective stress and recovery from the Perceived Stress Scale and Recovery Stress Questionnaire for Athletes, both validated questionnaires) were collected. However, for the purpose of this study, discussion of the methodology relevant to our analysis, i.e., energy intake, energy expenditure, and body composition measures (relevant for determination of EA), swim training data, swimming performance, and assessment of sleep quality and quantity will be reported.

Figure 3. WHOOP Wearable Device



Source: https://join.whoop.com/assets/accessories/GEN-4-CartImages-CircleRenders-500w-Onyx.png

Participant Recruitment

In terms of recruitment, all members of the NCAA Division 1 collegiate swim team were provided information about the study via an informational session taking place before the intensified training period began. Following the informational session, interested potential participants approached the study coordinator for more details on the research project, and a verbal recruiting script, approved by the IRB, was utilized by the study coordinator to provide further information. The study's informed consent document and an informational flyer were then provided to interested participants for their review. All recruitment materials were approved by the Pennsylvania State University's Institutional Review Board.

Once an interested participant arrived for their initial laboratory visit, they were first screened to determine eligibility. The several inclusion criteria for this study were that participants must be an active member of Penn State's Division 1 Varsity Swim team, be between 18 and 22 years of age, and be currently wearing the WHOOP or be willing to wear the WHOOP consistently during the data collection period. Exclusion criteria for this study were that

participants could not have any injuries or major training modifications that limit participation in training sessions, could not be taking any medications incompatible with measuring the variables of interest, and could not have any metabolic or heart conditions. Then, provided that the participant met the inclusion criteria and met none of the exclusion criteria, study procedures and the timeline of the study (Figure 4) were explained, and informed consent was obtained.

There were two phases of data collection. Phase 1 included 17 athletes and the second phase consisted of 10 athletes. The first phase of data collection, Phase 1, began after two weeks of the first wave of 17 athletes establishing compliance with wearing the WHOOP and having the WHOOP acclimate to them. Then, the second phase of data collection, Phase 2, began two weeks after the start of the first phase once the second wave of 10 athletes established compliance with wearing the WHOOP and had the WHOOP acclimate to them for a two-week period. During the two-week acclimation period in which participants were required to wear the WHOOP prior to data collection, the WHOOP established a personalized baseline of physiological data for each participant. Both Phase 1 and Phase 2 of data collection occurred during the intensified training period of the athletes' season, concluding before the reduction in training load in preparation for championships. All participants underwent the same study procedures that can be seen in Figure 4.

Figure 4. Adapted Timeline of Study and Procedures



Note: Adapted Timeline of Study and Procedures. Adapted from *Effectiveness of Wearable Technology for Predicting Measures of Metabolism and Performance in Collegiate Division I Swimmers* [Master's thesis, The Pennsylvania State University], by E. Lundstrom, 2020, p. 48. Adapted with permission.

Testing Procedures

Over the entire duration of the study, participants were asked to complete three to four laboratory visits. The first laboratory visit consisted of general screening questions, measurements of height, weight, and BMI, health and medical history surveys, eating behavior and body image surveys, and a WHOOP wear questionnaire. For each participant, data collection spanned two weeks. Throughout this two-week period, WHOOP data, including Sleep, Energy Expenditure, and Cardiovascular Load, or "Strain" data, was collected continuously utilizing the WHOOP dashboard (Figure 5). Additionally, study personal assessed daily training volume, training duration, and individual training session rate of perceived exertion.

During the second and third laboratory visit, indirect calorimetry was used to assess resting metabolic rate, DXA scans were utilized to assess body composition, and blood draw samples were used to measure serum triiodothyronine (TT₃) hormone levels. In addition to these measures, participants were required to attend all trainings, complete WHOOP Log to document their compliance daily, complete the Perceived Stress Scale^[132] and the RESTQ-52 Sport questionnaires^[133], complete a food log for three days (two week days, one weekend day) through the use of the application MyFitnessPal (Under Armour Inc; Baltimore, MD), and participate in a 200-yard freestyle time trial.



Figure 5. WHOOP Dashboard

Source: https://join.whoop.com/assets/landing-content/recovery/en/recovery-

overview.webp

Anthropometrics

During the first laboratory visit, anthropometric measurements were obtained. Participants were first instructed to take off shoes and heavy clothing. Then, total body mass was measured using a traditional physician's scale (Seca Model 770; Seca, Hamburg, Germany) reporting mass in kilograms to the nearest 0.01 kg, while height measurements were recorded in centimeters to the nearest 0.1 cm. Next, using these measurements, weight to height ratio in kg/m² was calculated to obtain the body mass index of each participant.

Body Composition

A Dual-energy X-ray Absorptiometry (DXA) machine (Hologic Horizon-W, Model 201331) was used to assess body composition measures of fat-free mass, lean body mass, fat mass, and total percent body fat in all participants. DXA scans were performed by an International Society of Clinical Densitometry-certified technician on the study team. Prior to their DXA scans, all participants were informed that the scan would expose them to a small amount of radiation. Female participants were additionally required to provide a urine sample yielding a negative urinary pregnancy result before undergoing their scans. Participants were asked to remove any metal-containing clothing or jewelry before lying still on a padded bed for approximately five minutes while a whole body scan was completed to determine lean mass and fat mass. For taller participants who did not fit entirely on the scanning area, the procedure designed and validated by Santos et al., 2012 where two scans are combined for the determination of body composition was followed.^[134]

Training

As stated previously, participants were required to attend all scheduled trainings as part of their participation in the study. The coaches scheduled and provided workout programs for these trainings. These training sessions included both in-water and weightlifting sessions, providing the training plan to each athlete and the study staff at the start of each week. Using these detailed training schedules, study personnel assessed several variables, including training duration, volume, and activity type, in order to determine the overall training load for each week of data collection. The training duration in minutes for each participant as well as the objective training volume as session yardages for each participant were recorded by the study coordinator who attended every training session throughout the data collection period. To quantify objective training load, both weekly and daily measures were calculated; specifically, distance swam per week (yds/wk), sessions attended per week (sessions/wk), hours per week (hrs/wk), minutes per week (mins/wk), distance swam per day (yds/d), hours per day (hrs/d), and minutes per day (mins/d) were calculated. Measures of training intensity were captured via Rating of Perceived Exertion (RPE) on a scale of zero to ten was obtained for each participant after each training session through completion of the Borg CR10 scale ^[135, 136]. To quantify subjective training load, the RPE number (1-10) was multiplied by training duration and reported in arbitrary units per week (AU/week). Both objective and subjective measures of training load calculations were utilized as checks to be sure that high intensity training and heavy training load was consistently maintained throughout the entire data collection period. Training measurements were collected to match 3-day dietary recording and sleep measurement.

WHOOP Data

This study used the WHOOP (WHOOP, Boston, MA), which is a wearable device created to be worn on a person's nondominant wrist that measures several variables with the overall goal of empowering users to optimize their athletic performance. Study participants were required to wear the WHOOP for the entire study duration with the device only being temporarily removed in cases of technical difficulties.

The WHOOP utilizes advanced technology, including a tri-axial accelerometer, optical sensor, capacitive touch sensor, and temperature sensor, capturing data at a frequency of 100 hertz. This data is then used to calculated sleep data, exercise energy expenditure, a strain score, 24-hour energy expenditure, resting heart rate, exercising heart rate, heart rate variability, and a recovery score.

Sleep data are measured by the WHOOP via the WHOOP's tri-axial accelerometer and 3axis gyroscope that gather movement data and its optical sensors, specifically two green lightemitting diodes paired with photodiodes, that measure blood flow. The green light emitting diodes shine light of a specific wavelength onto the skin and the photodiodes measure the light reflected back. Because blood only absorbs specific wavelengths of light, the light reflected back and measured by the photodiodes changes as blood volume fluctuates. In this way, green lightemitting diodes and the photodiodes work in combination to obtain data on changes in blood flow. The blood flow and movement measurements are then used to derive respiratory rate, heart rate, and heart rate variability. These three variable measures are entered into a sleep detection and sleep staging algorithm to produce the various sleep measures (WHOOP Inc., Boston, MA). While the WHOOP's advanced technology and sleep algorithms allow for the auto-detection of sleep, as described above, the WHOOP's application dashboard (Figure 5) also allows users to confirm sleep times or manually adjust sleep times to further improve the accuracy of sleep data. The specific sleep variables measured by the WHOOP are total sleep time, sleep disturbances, sleep efficiency, and amount of time (hours) and percentage of time in each of the four main stages of sleep (wake light sleep, slow-wave sleep, and rapid eye movement sleep), and cycle duration.

Exercise energy expenditure, workout strain, and workout heart rates were measured by the WHOOP, using heart rate and tri-axial accelerometry to analyze heart rate and prompting participants to input their type of exercise and confirm its completion. Together, these data points allow the WHOOP to calculate exercise energy expenditure. Utilizing exercise energy expenditure and exercise heart rate data, the WHOOP also calculates a strain score, a measure of 'cardiovascular load,' on a scale of 0 to 21 with 0 indicating very low strain and 21 indicating very high strain. Workout strain is quantified by a proprietary algorithm utilizing the duration of time spent in heart rate zones based on an athlete's predicted maximum heart rate (0-50%, 50-60%, 60-70%, 70-80%, 80-90%, and 90-100%). Because participants were required to wear the WHOOP at all times during the entire study duration, 24-hour energy expenditure, which included both exercise energy expenditure and non-exercise related activity. was also measured by the WHOOP.

Sleep data, training responses (workout strain, average workout heart rate, and maximum workout heart rate), exercise energy expenditure, and 24-hour energy expenditure were measured for the study using the WHOOP device. For analysis of sleep and training response data against measures of EA, sleep and training response data was collected on matched days with 3-day dietary recording. For analysis of sleep data against swimming performance, sleep data was

retrieved for the night preceding the time trial swim, as well as for the 3 days (averaged) leading up to the time trial performance swim. All WHOOP data assessed for the participants was recorded from the WHOOP application to the WHOOP's cloud analytics platform. Once the data collection period ended, the study team downloaded all of the raw data generated by the WHOOP and converted the csv file into an excel file format that was then de-identified. Once the file had been de-identified, study personnel exported it for final retrieval. Additionally, the WHOOP wearable log was completed by each participant everyday of data collection to document any times in which the WHOOP was not being worn continuously as well as to record comments concerning WHOOP wear for the study personnel to review.

Energy Intake

The application MyFitnessPal premium was utilized as a tool for participants to document their dietary energy intake by keeping a 72-hour diet log, two week days and one weekend day, through the application (Under Armour, Baltimore, MD). During the initial laboratory visit, study personnel provided detailed instructions on how the diet logs should be completed. Specifically, they instructed participants to record all calorie-containing substances that they consume as well as the time of consumption, location of consumption, whether it was a meal or snack, meal type (breakfast, lunch, or dinner), preparation details, and brand of caloriecontaining substances for a 72-hour consecutive period consisting of two weekdays and one weekend day. Upon receiving the 3-day diet logs from each participant, which were saved by each participant as csv file and then sent to study personnel, study personnel examined each log to ensure accuracy. After accuracy was established, dietary energy intake was calculated as the total value of all calorie-containing substances reported throughout the 72-hour food log recording period.

Energy Availability

Energy availability was calculated using the formulas EA=(EI-EEE)/(FFM) (kcal/kg FFM) and EA=(EI-EEE)/(LBM) (kcal/kg LBM). Energy intake (EI) was determined from the information entered by participants into the MyFitnessPal application for 3-days to determine EI in kilocalories. For exercise energy expenditure (EEE), WHOOP data was used to determine EEE, also in kilocalories. Finally, the fat free mass (FFM) and lean body mass (LBM) reported from the DXA scan in kilograms were utilized to normalize the difference in EI and EEE. Sleep data were retrieved for days corresponding to 3-day dietary recording for analysis.

Resting Metabolic Rate

Resting metabolic rate (RMR) was assessed via indirect calorimetry for this study. Specifically, a metabolic cart was utilized to obtain this measurement (Viasys Healthcare, Vmax Encore Metabolic Cart; CareFusion, Yorba Linda, CA). RMR testing occurred in the morning between 0600h and 0900h following either a training rest day in which no training occurred or following a recovery day of training in which training was comparatively light. Participants received several specific instructions concerning the time leading up to their RMR testing appointment including: fast for 12 hours prior to arriving to the lab, consume no alcohol or caffeine in the 24 hours prior to their lab visit, and complete no intense exercise in the 12 hours prior to their appointment.

Once participants arrived at the laboratory for their RMR testing appointment, they were instructed to take off their shoes and heavy clothing before their total body mass was measured

using a physician's scale (Seca Model 770; Seca, Hamburg, Germany) and recorded in kilograms to the nearest 0.01 kg, as described above. Next, participants underwent a rest period in which they laid on a hospital stretcher in a supine position for 20 to 30 minutes. During this time, participants were asked a series of questions concerning the time leading up to the RMR testing to ensure that the specific instructions provided to allow for accurate RMR testing were followed. Once the rest period concluded, RMR testing could begin. At this point, study personnel placed a ventilated hood onto the participant and using indirect calorimetry through the metabolic cart, rates of carbon dioxide production (VCO₂) and oxygen consumption (VCO₂) were measured once every minute for a period of 30 to 45 minutes. During this period, it was required that a 10-minute period of steady state was achieved, referring to a less than ten percent of a fluctuation in oxygen (VO₂; mL·min⁻¹) and less than ten percent of a fluctuation in carbon dioxide (VCO₂; mL·min⁻¹) in order for the RMR to be considered accurate. These data measured via indirect calorimetry through the metabolic cart then were utilized to calculate resting energy expenditure via the Weir equation ^[137].

Additionally, for this study, several RMR prediction equations were used to calculate predicted RMR. These equations were as follows: the Harris-Benedict equation ^[138], the DXA equation ^[139, 140], the Cunningham₁₉₈₀ equation ^[141], and the Cunningham₁₉₉₁ equation ^[142].

Performance Time Trial Swim

One 200-yard freestyle time trial was completed during the study's data collection period as a measure of performance. This time trial, occurring during team practice time, followed a protocol common to elite swimming participation and was overseen but members of the team's coaching staff as well as certified lifeguards. Prior to the time trial date, participants were told to treat the time trial was they would a competition and to prepare accordingly. Then, upon arrival to the time trial designated practice, participants were given 30 minutes to warm up before the time trial began. Participants competed in heats organized to consist of teammates that they normally compete against to simulate a real race environment. Participants dove off the starting block before completing a 200-yard freestyle swim at their maximal exertion level. Both the Colorado Timing system (Colorado Time System; Loveland, CO) and handheld timers were used to time the time trial, which lasted less than two minutes for all participants. Sleep data were retrieved for days corresponding to the night(s) prior to the time trial race (the night prior to the race, and 3-day average of sleep prior to the race) for analysis.

Statistical Analysis

Data were analyzed using SPSS Statistical Software (version 26, Chicago, IL). All variables were tested for normality and outliers prior to conducting statistical analyses. First, normality was tested using the Shapiro-Wilke statistic. Then, outliers were located, removed, and Levene's test was utilized to determine homogeneity of variance. For the analyses, participants were analyzed as a whole group, in sex groupings, and in sex groupings further grouped by the median split of average EA. Independent t-tests were utilized to determine group differences between sexes and between the median split EA groups of each sex. Correlations were calculated using Pearson's correlation analysis to identify any relationships of interest. Linear regression analyses were utilized to determine statistically significant predictors of sleep quality, training responses, and time trial performance. Data were reported as mean +/- SD, and a p-value of <0.05 was considered statistically significant.

Chapter 3

RESULTS

Participant Characteristics

The participants enrolled at the beginning of the study consisted of 27 NCAA Division I collegiate swimmers (11 males and 16 females). There were no dropouts. One male participant was excluded from the analyses due to prematurely entering the taper phase of lower volume training before the data collection period had ended. Therefore, a total of 26 participants were utilized for this study. Of these 26 participants, 24 participants identified themselves as Caucasian, 1 participant identified themselves as African American/Caribbean, and 1 participant identified themselves as Latin American. Further descriptive information is included in Table 1 below. As Table 1 indicates, male swimmers had significantly greater height, weight, BMI, fat free mass, and lean body mass compared to the female swimmers (p<0.05; t-test), as expected.

Descriptive Energy, Sleep, Training and Performance Characteristics in All Swimmers

Average energy intake, average energy expenditure, and average energy availability for the three days of food log recording are included in Table 1. The male swimmers had significantly higher average energy intake, average energy expenditure, and average energy availability compared to the female swimmers (44.4 ± 9.7 vs 35.1 ± 10.5 , p=0.033).

Average nightly hours of sleep, REM sleep hours, slow-wave sleep hours, sleep debt hours, REM sleep percentage, and slow-wave sleep percentage matched to the dietary recording and training days and matched to the night(s) preceding the time trial performance swim are presented in Table 1. In all swimmers, 42% (11/26) had a sleep duration of at least seven hours across the dietary recording and training days, while 38% (6/16) of female swimmers and 50% (5/10) of male swimmers achieved a sleep duration at least seven hours, as recommended by the National Sleep Foundation.¹⁴³ There were no significant differences in sleep duration during the dietary recording days nor during the night(s) prior to the time trial performance swim between male and female swimmers (p<0.05; t-test). Regarding sleep quality, male swimmers had significantly more hours of slow-wave sleep compared to the female swimmers (p=0.004) and significantly higher slow-wave sleep percent compared to female swimmers (p=0.006) when sleep was matched to days of dietary recording and training. Besides slow-wave sleep hours and slow-wave sleep percent matched to dietary recording and training, there were no other significant differences in sleep quality measures between the male and female swimmers during the dietary recording days nor during the night(s) prior to the time trial performance swim.

Time trial performance swim, average training volume, and training response metrics, including workout strain, average workout heart rate, and maximum workout heart rate, are described in Table 1. There were no significant differences in training volume between male and female swimmers during the observation period (p>0.05). Similarly, there were no significant differences in any training metrics between male and female swimmers (p>0.05). Finally, male swimmers had significantly faster time trial times compared to female swimmers (p<0.001), as shown in Table 1.

	All (N=26)	Male (n=10)	Female (n=16)	
	Mean \pm SD	Mean \pm SD	Mean ± SD	p- value
Demographics				
Height (cm)	178.7 ± 7.8	186.4 ± 4.9	174.0 ± 4.8	<0.001
Weight (kg)	74.1 ± 10.3	83.8 ± 8.6	68.0 ± 5.6	<0.001
FFM (kg)	56.5 ± 11.0	68.6 ± 6.7	49.0 ± 4.3	<0.001
LBM (kg)	53.9 ± 10.6	65.6 ± 6.4	46.5 ± 4.0	<0.001
BMI (kg/m2)	23.1 ± 1.9	24.1 ± 1.9	22.5 ± 1.6	0.024
Age (years)	19.6 ± 1.1	20.1 ± 1.0	19.3 ± 1.0	0.094
Energy				
Average EI (kcal/day)	2934.5 ± 990.8	3959.3 ± 715.7	2294.0 ± 437.3	<0.001
Average EE (kcal/day)	2568.6 ± 611.0	3185.0 ± 274.9	601.3 ± 170.1	<0.001
Average EA (kcal/kg FFM)	38.6 ± 11.0	44.4 ± 9.7	35.1 ±10.5	0.033
Sleep				
Matched to Dietary Recording Dates				
Hours of Sleep	6.8 ± 0.9	6.6 ± 1.1	7.0 ± 0.7	0.303
REM Sleep (hours)	1.7 ± 0.7	1.9 ± 0.9	1.5 ± 0.5	0.157
Slow-Wave Sleep (hours)	1.2 ± 0.4	1.5 ± 0.3	1.0 ± 0.3	0.008
Sleep Debt (hours)	1.2 ± 0.5	1.2 ± 0.6	1.2 ± 0.5	0.834
REM Sleep Percentage	22.1 ± 8.0	23.1 ± 9.8	21.4 ± 6.8	0.599
Slow-Wave Sleep Percentage	14.0 ± 4.1	16.4 ± 3.8	12.4 ± 3.6	0.013
Matched to Time Trial Performance				
Night Preceding Time Trial				
Hours of Sleep	6.8±1.2	6.6±1.2	6.9±1.2	0.63
Sleep Debt (hours)	1.0±0.7	1.1±0.6	0.9±0.7	0.56
REM Sleep Percentage	20.8±9.8	21.8±9.2	20.3±10.3	0.713
Slow-Wave Sleep Percentage	14.9±4.7	14.5±4.4	15.1±4.9	0.728
Average of 3 Nights Preceding Time Trial				
Hours of Sleep	6.7±0.9	6.5±0.9	6.9±0.9	0.223
Performance				
Time Trial Time (sec)	112.2 ± 6.2	105.1 ± 3.1	116.6 ± 1.9	<0.001
Average Training Volume (yards/day)	6100±950	5900±1000	6250±850	0.687
Workout Strain (AU: 0-21)	14.0±1.1	14.1±1.5	13.9±0.8	0.549
Average Workout Heart Rate (bpm)	131.6±7.6	131.5±8.5	131.6±7.2	
Maximum Workout Heart Rate (bpm)	175.7±6.6	176.7±5.2	175.0±7.4	0.534

Table 1. Participant characteristics.

FFM: fat-free mass; LBM: lean body mass; BMI: body mass index; EI: energy intake; EE: energy expenditure; EA: energy availability; REM: rapid eye movement; SD: standard deviation.

Relationships Between Energy Availability and Sleep

Correlations between energy availability and sleep are presented in Table 2. There was a trend toward a correlation between energy availability and sleep duration_{hrs} in all swimmers (R=0.33; P=0.06). Average energy availability was positively correlated with hours of REM sleep in all swimmers (r= 0.639, p < 0.001), male swimmers alone (r=0.744, p=0.014), and female swimmers alone (r=0.515, p=0.041), as illustrated in Figure 6. Average energy availability was positively correlated with hours of slow-wave sleep in all swimmers (r= 0.607, p <0.001) and in female swimmers alone (r=0.570, p=0.021); however, average energy availability was not significantly correlated with hours of slow-wave sleep in male swimmers alone (r=0.389, p=0.267). Average energy availability was positively correlated with slow-wave sleep percent in all swimmers (r=0.488, p=0.012); however, average energy availability was not significantly correlated with slow-wave sleep percent in male swimmers alone (r=0.328, p=0.355), nor in female swimmers alone (r=0.378, p=0.149).



Figure 6. The relationship between average EA and REM sleep. (A) The positive correlation between average EA and REM sleep in all swimmers (R=0.639; p<0.001). (B) The positive correlation between average EA and REM sleep in male swimmers (R=0.744, p=0.014). (C) The postive correlation between average EA and REM sleep in female swimmers (R=0.515; p=0.041).

Additionally, because energy availability was significantly different between men and women, t-test analyses examining differences in sleep variables between the lower 50% and upper 50% energy availability groups were performed separately for the sexes. As shown in Table 3, in the male swimmers, there were no significant differences between the lower 50% energy availability group and the upper 50% energy availability group for any of the sleep variables. In the female swimmers, sleep debt was greater in the lower energy availability group than it was in the upper energy availability group $(1.46\pm0.41 \text{ vs } 0.85\pm0.37; p=0.008)$, hours of REM sleep was lower in the lower energy availability group than it was in the upper energy availability group than it was in the upper energy availability group than it was in the upper energy availability group ($1.20\pm0.26 \text{ vs } 1.86\pm0.52; p=0.006$), and hours of slow-wave sleep was lower in the lower energy availability group than it was in the upper energy availability group ($0.85\pm0.26 \text{ vs } 1.19\pm0.26; p=0.002$), as displayed in Table 3. Results illustrating the comparison of hours of sleep debt, REM sleep, and slow-wave sleep between the low 50% energy availability group and the upper 50% energy availability group in both men and women are depicted in Figure 7.

When energy availability was analyzed for its potential influence on measures of sleep quality, linear regression analyses revealed that when controlling for sex, the main effect of energy availability was a significant predictor of SWS_{hrs} ($R^2 = 0.448$; F=9.35, p<0.001). Linear regression analyses also revealed that when controlling for sleep duration_{hrs}, the main effect of energy availability was a significant predictor of REM (hours) ($R^2 = 0.425$; F=8.509, p<0.002). Finally, when controlling for duration of sleep (hours), the main effect of energy availability was a significant predictor of sleep (hours) (R^2 =0.261; F=4.055; p=0.031).



Figure 7: Comparison of hours of sleep debt, REM sleep, and slow-wave sleep between the lower 50% EA group and the upper 50% EA group in both men and women. Statistically significant differences between EA groups are noted with *p<0.05. EA: energy availability; REM: rapid eye movement

Table 2. Correlations between sleep variables and energy availability

	Hours	of Sleep	REM Sle	ep (hours)	Slow-Wave Sleep (hours)		Slow-Wave Sleep Percent	
Average EA	r value	p value	r value	p value	r value	p value	r value	p value
All Swimmers	-0.332	0.098	0.639	<.001	0.607	<0.001	0.488	0.012
Male Swimmers	-0.126	0.728	0.744	0.014	0.389	0.267	0.328	0.355
Female Swimmers	-0.419	0.107	0.515	0.041	0.57	0.021	0.378	0.149

REM: rapid eye movement; EA: energy availability. Correlations are described with R values and p values in table. Bold formatting denotes statistical significance (p<0.05) between variables.

Table 3. t-test examining differences in sleep and performance between lower and higher EA groups by sex

	Male Swimmers			Female Swimmers			
	EA in lower 50%	EA in Upper 50%	<u> </u>	EA in lower 50%	EA in Upper 50%		
Sleep and Performance	$Mean \pm SD$	$Mean \pm SD$	p value	$Mean \pm SD$	$Mean \pm SD$	p value	
Sleep Debt (hours)	1.43 ± 0.62	0.97 ± 0.50	0.231	1.46 ± 0.41	0.85 ± 0.37	0.008	
REM Sleep (hours)	1.51 ± 0.49	2.36 ± 1.09	0.15	1.20 ± 0.26	1.86 ± 0.52	0.006	
Slow-Wave Sleep (hours)	1.38 ± 0.43	1.54 ± 0.21	0.485	0.85 ± 0.26	1.19 ± 0.26	0.002	
Slow-Wave Sleep Percent	16.31 ± 4.87	16.54 ± 2.90	0.929	11.70 ± 2.17	13.15 ± 4.74	0.444	
Time Trial Time (sec)	105.16 ± 2.48	104.95 ± 3.90	0.924	116.60 ± 2.09	116.70 ± 1.91	0.919	

EA: energy availability; REM: rapid-eye movement; SD: standard deviation. Correlations are described with R values and p values in table. Bold formatting denotes statistical significance (p<0.05) between variables.

Relationships Between Sleep and Training Responses

Sleep duration_{hrs} was negatively related to workout strain (r=-0.848, p=0.001), average workout heart rate (r=-0.653, p=0.001), and maximum workout heart rate (r=-0.477, p=0.014) in all swimmers. Similarly, sleep debt_{hrs} was positively related to workout strain (r=0.394, p=0.046), average workout heart rate (r=0.511, p=0.008), and maximum workout heart rate (r=0.566, p=0.003) in all swimmers. Finally, in all swimmers, SWS_{hrs} was negatively related to average workout heart rate (r=-0.405, p=0.04). When examining the sexes separately, sleep duration_{hrs} was negatively related to workout strain (r=-0.927, p=0.001), average workout heart rate (r=-0.822, p=0.004), and maximum workout heart rate (r=-0.726, p=0.017) in male swimmers. However, there were no other sleep variables related to training responses in male swimmers. In female swimmers, sleep duration_{hrs} was negatively related to average workout heart rate (r=-0.517, p=0.04). Similarly, sleep debt_{hrs} was positively related to average workout heart rate (r=-0.511, p=0.043), and maximum workout heart rate (r=0.608, p=0.012) in female swimmers. Finally, REM_{hrs} was negatively related to average workout heart rate (r=-0.601, p=0.014) in female swimmers.

The Relationships Between Sleep Quality and Swimming Performance

When examining the relationships between sleep quality the night(s) prior to the time trial and swimming performance, there were no relationships that achieved statistical significance between sleep duration the night before the race nor when averaging the 3 days leading up to the race and swimming performance in all swimmers, male or female. Additionally, measures of sleep quality (SWS%, REM%, sleep debthrs) assessed the night prior to the race did not significantly relate to actual swimming performance the following day in all swimmers, male or female. However, regression analyses revealed that when controlling for sex, sleep duration_{hrs} the night preceding the race predicted TT_{perf} ($R^2 = 0.881$; p<0.001) whereby swimmers with more hours of sleep swam faster in the time trial. Similarly, when controlling for sex, SWS% the night preceding the race predicted TT_{perf} ($R^2 = 0.883$; p<0.001) whereby swimmers with a greater SWS% swam faster in the time trial. Regression analyses also revealed that sleep debt_{hrs} the night before the race, and the average of the 3 nights leading up to the time trial swim did not significantly predict TT_{perf} in all swimmers.

Energy Availability and Training Responses

In all swimmers, there was no evidence of relationships existing between energy availability and matched training responses (workout strain, average workout heart rate, and maximum workout heart rate). Similarly, when looking at the sexes independently, there were no relationships evident between energy availability and matched training responses (workout strain, average workout heart rate, maximum workout heart rate) in male or female swimmers.

Combined Effects of Energy Availability and Sleep Quality on Training Responses

Regression analyses revealed that when controlling for sex, there was no combined effect of both energy availability and other sleep quality variables tested (SWS_{hrs}, SWS_%, REM_{hrs}, REM_%, and sleep debt_{hrs}) on any training responses (workout strain, average workout heart rate, maximum workout heart rate).

Chapter 4

DISCUSSION

This research contributes meaningfully to the literature as the first study to examine the interrelationships between energy availability on measures of sleep quality and training responses, and sleep quality measures on the training responses and sport performance of elite swimmers. Most notably, we found that (1) swimmers with higher energy availability had significantly better sleep quality for several sleep measures, and (2) higher quality sleep (assessed with several measures) was associated with both more favorable training responses and faster swimming performance. Specifically, energy availability was positively associated with REM sleep hours in all swimmers, male swimmers, and female swimmers and positively associated with slow-wave sleep in all swimmers and female swimmers. Further, energy availability was found to be a significant predictor of hours of REM sleep, slow-wave sleep, and sleep debt when controlling for confounding variables of sex and sleep duration. While energy availability was related to and/or predicted sleep quality, it did not relate to any training responses. Sleep quality, however, was related to both training responses and swimming performance in all swimmers, whereby swimmers with higher sleep quality exhibited lower average workout strain and heart rates during training and swam faster during the Time Trial swim. Considered together, these findings suggest that higher energy availability relates to better sleep quality in populations of elite collegiate endurance athletes, and that higher sleep quality may result in more favorable training and performance outcomes.

Relationships Between Energy Availability and Sleep

EA influences a variety of physiological outcomes. For instance, low EA has been demonstrated to impair reproductive function ^[5], and in severe cases, can have serious consequences when it comes to bone health ^[6], as outlined by the Triad ^[1, 2]. Additionally, low energy availability has been found to impact hormones, with decreases in leptin levels and increases in ghrelin levels being observed during periods of low energy availability ^[23, 24]. Furthering our understanding of EA's influence on physiological outcomes, this study found a relationship between EA and sleep, as swimmers with higher energy availability had significantly better sleep quality for several sleep measures. Our understanding of this finding comes from consideration of the metabolic hormones associated with energy availability and sleep. Specifically, the effects of orexin in increasing arousal during low energy availability ^{[144,} ^{145]} provides a logical interpretation for our study's finding that energy availability was positively correlated with hours of REM sleep in all, male, and female swimmers and positively correlated with hours of slow-wave sleep in all swimmers and female swimmers, as well as the finding that energy availability was found to be a significant predictor of hours of REM sleep and slow-wave sleep when controlling for confounding variables of sex and sleep duration.

The literature supports that leptin inhibits lateral hypothalamic neurons from secreting orexin ^[144], a hormone that increase arousal and wakefulness, decreases REM sleep, and decreases slow-wave sleep ^[145]. Therefore, when leptin levels decrease, such as the case during low energy availability ^[23], lateral hypothalamic neurons may secrete more orexin, resulting in increased arousal and wakefulness, decreased REM sleep, and decreased slow-wave sleep ^[146]. Additionally, ghrelin, a hormone that increases during low energy availability ^[24] has been found to increase orexin levels ^[145], further increasing arousal and wakefulness and also impairing

REM and slow-wave sleep through this mechanism ^[147]. Therefore, given the research demonstrating leptin's role in preserving REM and slow-wave sleep ^[148] along with the finding that leptin decreases in those with low energy availability ^[23], combined with the fact that ghrelin, a hormone that increases during low energy availability ^[149] has been found to increase orexin levels and arousal ^[145], it is unsurprising that lower energy availability was related to less REM sleep and less slow-wave sleep.

The finding that energy availability was a significant predictor sleep debt when controlling for confounding variables of sex and sleep duration in the present study may result from the effects of orexin in increasing arousal during low energy availability ^[145]. Because sleep debt accumulates as individuals fail to obtain the amount of sleep their body demands (WHOOP Inc., Boston, MA), it is likely that increased wakefulness arising from increased orexin in individuals with low energy availability result in less sleep opportunity and increased sleep debt ^[147]. This proposed mechanism for the relationship between low energy availability and sleep quality measures is depicted in Figure 8 below.



Figure 8. Proposed Mechanism of the Relationship Between Low Energy Availability and Several Sleep Quality Measures REM: Rapid-eye movement sleep

Furthermore, our finding that elite swimmers with lower energy availability had significantly worse sleep quality for several sleep measures is not only informative for health maintenance, but it also has valuable, practical applications for athletes. Specifically, finding that athletes with low energy available had significantly worse sleep quality provides coaches with direction in implementing science-driven practices to support the health of their athletes. For instances, coaches may consider putting greater effort into helping their athletes improve fueling strategies through emphasizing nutrition and providing athlete education in light of the apparent interrelationship between energy availability and sleep health. Furthermore, this study's practical application of encouraging nutritional interventions within elite collegiate athlete populations is especially relevant because in endurance sports especially, low energy availability is of high prevalence ^[7].

Relationship Between Sleep, Training Responses, and Swimming Performance

Higher quality sleep was associated with both more favorable training responses and faster swimming performance. Specifically, this study found that greater slow-wave sleep hours related to lower average workout heart rate, and when controlling for sex, slow-wave sleep percent the night preceding the race predicted time trial performance whereby swimmers with a greater slow-wave sleep percent swam faster in the time trial. In understanding these results, we look to the sleep literature. The literature supports that slow-wave sleep carries many physiological benefits, particularly when it comes to growth, healing, and recovery [85, 86, 87]. Such benefits of slow-wave sleep include maintenance of the diurnal rhythm of human growth hormone that supports the building and repairing of bone and muscle tissue ^[88], observed during slow-wave sleep ^[85, 89]. One study investigating the relationship between human growth hormone secretion and slow-wave EEG activity reported that growth hormone secretory pulses were concurrent with the slow-wave peaks ^[89]. Furthermore, Gronfier et al. found a positive correlation between the amount of secreted human growth hormone and slow-wave wave activity ^[89]. As such, research demonstrating the association between slow-wave sleep and human growth hormone secretion reaffirms the vital role of slow-wave sleep in growth and healing. Furthermore, studies have found that if there is high energy expenditure throughout the day, as is common among elite endurance athletes, human growth hormone secretion will rise the following night ^[151]; however, when athletes obtain less slow-wave sleep, these levels of human

growth hormone drop significantly ^[91]. Such studies demonstrate that the body is equipped to alter hormone secretion in response to exercise to allow for necessary physiological growth and repair, but inadequate sleep interferes, impairing recovery, which impacts training responses and sport performance.^[152] Without experiencing the proper recovery that adequate slow-wave sleep promotes, athletes are missing opportunities to gain muscle and bone strength while putting themselves at increased risk of injury ^[92], both serving as additional potential avenues for inadequate slow-wave sleep to impair training response and athletic performance. Altogether, understanding of the role of slow-wave sleep in facilitating proper recovery provides a strong explanation for the findings that more slow-wave sleep was associated with a more optimal training responses and faster time trial swim times.

The present study found that longer sleep duration and less sleep debt, a variable that measures inadequate sleep duration and accumulates as individuals fail to obtain the amount of sleep their body demands (WHOOP Inc., Boston, MA), were associated with more optimal training responses in all swimmers. We also found that when controlling for sex, sleep duration the night preceding the race predicted time trial performance whereby swimmers with more hours of sleep swam faster in the time trial. This could be due to the relationship between sleep duration and slow-wave sleep, whereby shorter sleep durations often result in shorter slow wave sleep durations. As forementioned, when an individual has a short sleep duration, reflected through accumulating a large sleep debt when continued overtime, they spend less total time in each sleep stage, including slow-wave sleep ^[44, 82]. Because slow-wave sleep and its benefits are vital to training responses and athletic performance ^[44, 152], largely due to the interrelationship between slow-wave sleep and human growth hormone as described above ^[90, 91], reductions in slow-wave sleep time resulting from short sleep duration provide a possible explanation for the

finding that longer sleep duration and less sleep debt are related to improved training responses and sport performance .

Energy and Training Responses

While energy availability was related to and predicted several sleep quality measures, it did not relate to any training responses. This finding was unexpected, as we had initially hypothesized that low energy availability would be associated with impaired training responses during matched training sessions, specifically an increased average workout heart rate and increased maximum workout heart rate, given the literature supporting the importance of adequate energy availability in sustaining optimal physiological function ^[4,9,28,8]. Yet, the literature suggests that during the extreme cases of low energy availability, such as the case in individuals with anorexia, cardiac tissue breakdown, often resulting in clinically slow heart rate known as bradycardia ^[150]. Therefore, this extreme outcome of bradycardia that is observed in cases of severe low energy availability complicates the relationship between energy availability and heart rate-related training responses, providing a possible explanation for the finding that energy availability was not related to training responses in this population of elite swimmers.

Combined Effects of Energy Availability and Sleep Quality on Training Responses

Finally, in investigating the combined effect of energy availability and sleep quality on training responses, we found that when controlling for sex, there was no effect of both energy availability and other sleep quality variables tested (SWShrs, SWS%, REMhrs, REM%, and sleep debthrs) on any training responses (workout strain, average workout heart rate, maximum workout heart rate). Given the finding that energy availability did not relate to any training

responses (as understood in terms of slow heart rate observed in cases of severe low energy availability complicating the relationship between energy availability and heart rate-related training responses), it is not surprising that no significant combined effect of energy availably and sleep quality on training responses was found. More research is needed to investigate the casual relationship between energy availability, sleep quality measures, and training responses.

Limitations

While this study was carefully designed, developed, and executed with the highest scientific rigor, several limitations must be disclosed. This study utilized a cross-sectional design. Therefore, it is not possible to draw conclusions about causality; instead, only associations can be analyzed. Additionally, given the elite caliber of athletes enrolled in the study and that data was collected only during the intensified train period of the athletes' season, the applicability of the results in limited in athlete type and season timepoint. Also, while the sample size of 26 athletes used for the analyses is larger than most other elite athlete health-related studies, it is still a relatively small sample size, which limits the statistical power of the results. Furthermore, the significant sex differences between several of the variables of interest (i.e., sport performance variables, energy variables, and slow-wave sleep hours), required certain statistical analyses to be performed separately by sex (specifically, t-tests and correlation analyses), lowering the effective sample size for several of the analyses and, thus, further lowering the statistic power of the result. Aiding the statistical power of the results, however, is the fact that all but one of the 27 participants who enrolled at the start of the study completed all study procedures and demonstrated good adherence to the protocol, which is a notable strength to this study.

Regarding data collection, one limitation of this study was that in-laboratory polysomnography, which is gold standard for measuring sleep due to its ability to collect sleep quantity and quality data with more accuracy than any other assessment tool ^[18], was not used to collected sleep data. Instead, the WHOOP Performance Optimization system (WHOOP Inc., Boston, MA) was utilized. Although not the gold standard, the WHOOP has a notable strength in being a wearable technology device that has been validated against polysomnography for its various sleep measures ^[118,122,123]. However, it is important to note that the WHOOP sleep validation studies were not performed in an athletic population ^[118, 122, 123].

A second limitation of this study to consider when it comes to the data collection is that dates associated with each participant's average energy availability measure (as determined by the dates of the 72-hour diet logs that were utilized to calculated three days of energy availability data for each participant) do not coincide precisely with each participant's time trial swim date. Therefore, analysis of the relationship between energy availability and time trial swim performance is beyond the scope of this study.

Future Directions

While this study contributes novel findings, particularly finding that higher energy availability relates to better sleep quality and that better sleep quality is associated with more favorable training response and swimming performance in our population of elite endurance athletes, more research is needed to clarify the effect of energy availability and sleep quality measures on athletic performance. Particularly, randomized control trials should be conducted to greater explore the casual relationship between these variables. Additionally, future research on the effects of energy availability and sleep quality measure on sport performance may focus on examining these relationships across sports, throughout different points of the training season, and across competitive levels to extend the results of these studies beyond elite collegiate swimmers.

Furthermore, while in-laboratory polysomnography is the gold-standard for measuring sleep due to its ability to collect sleep quantity and quality data with the upmost accuracy ^[18], the accessibility, convenience, and growing validation of wearable technology devices like the WHOOP Performance Optimization system (WHOOP Inc., Boston, MA) against the gold-standard make wearable technology the future of sleep research. As such, WHOOP sleep validation studies conducted specifically in athletic populations would be helpful in increasing the strength of scientific rigor and applicability of future sleep studies to athlete populations.
REFERENCES

- Nattiv, A., Loucks, A. B., Manore, M. M., Sanborn, C. F., Sundgot-Borgen, J., & Warren, M. P. (2007). American College of Sports Medicine position stand. The female athlete triad. *Med Sci Sports Exerc*, *39*(10), 1867-1882. <u>https://doi.org/10.1249/mss.0b013e318149f111</u>
- Nattiv, A., De Souza, M. J., Koltun, K. J., Misra, M., Kussman, A., Williams, N. I., . .
 Fredericson, M. (2021). The Male Athlete Triad-A Consensus Statement From the Female and Male Athlete Triad Coalition Part 1: Definition and Scientific Basis. *Clin J Sport Med*, *31*(4), 345-353. <u>https://doi.org/10.1097/jsm.00000000000946</u>
- Torstveit, M. K., & Sundgot-Borgen, J. (2005). The female athlete triad: are elite athletes at increased risk? *Med Sci Sports Exerc*, *37*(2), 184-193. <u>https://doi.org/10.1249/01.mss.0000152677.60545.3a</u>
- Williams, N. I., Koltun, K. J., Strock, N. C. A., & De Souza, M. J. (2019). Female Athlete Triad and Relative Energy Deficiency in Sport: A Focus on Scientific Rigor. *Exercise and Sport Sciences Reviews*, 47(4), 197-205. https://doi.org/10.1249/jes.00000000000000000000
- Joy, E., De Souza, M. J., Nattiv, A., Misra, M., Williams, N. I., Mallinson, R. J., ... Borgen, J. S. (2014). 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad. *Current Sports Medicine Reports*, 13(4).
- Souza, M. J. D., Nattiv, A., Joy, E., Misra, M., Williams, N. I., Mallinson, R. J., . . .
 Panel, E. (2014). 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International

Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine*, 48(4), 289-289. <u>https://doi.org/10.1136/bjsports-2013-093218</u>

- Gibbs, J. C., Williams, N. I., & De Souza, M. J. (2013). Prevalence of individual and combined components of the female athlete triad. *Med Sci Sports Exerc*, 45(5), 985-996. https://doi.org/10.1249/MSS.0b013e31827e1bdc
- Tornberg Å, B., Melin, A., Koivula, F. M., Johansson, A., Skouby, S., Faber, J., & Sjödin, A. (2017). Reduced Neuromuscular Performance in Amenorrheic Elite Endurance Athletes. *Med Sci Sports Exerc*, 49(12), 2478-2485. <u>https://doi.org/10.1249/mss.00000000001383</u>
- Elliott-Sale, K. J., Tenforde, A. S., Parziale, A. L., Holtzman, B., & Ackerman, K. E. (2018). Endocrine Effects of Relative Energy Deficiency in Sport. *Int J Sport Nutr Exerc Metab*, 28(4), 335-349. <u>https://doi.org/10.1123/ijsnem.2018-0127</u>
- Juliff, L. E., Halson, S. L., & Peiffer, J. J. (2015). Understanding sleep disturbance in athletes prior to important competitions. *Journal of Science and Medicine in Sport*, 18(1), 13-18. <u>https://doi.org/https://doi.org/10.1016/j.jsams.2014.02.007</u>
- Mah, C. D., Kezirian, E. J., Marcello, B. M., & Dement, W. C. (2018). Poor sleep quality and insufficient sleep of a collegiate student-athlete population. *Sleep Health*, 4(3), 251-257. <u>https://doi.org/https://doi.org/10.1016/j.sleh.2018.02.005</u>
- Rabin, J. M., Mehra, R., Chen, E., Ahmadi, R., Jin, Y., & Day, C. (2020). Assessment of sleep health in collegiate athletes using the Athlete Sleep Screening Questionnaire. *Journal of Clinical Sleep Medicine*, *16*(8), 1349-1356.

- Sargent, C., Halson, S., & Roach, G. D. (2014). Sleep or swim? Early-morning training severely restricts the amount of sleep obtained by elite swimmers. *European Journal of Sport Science*, *14*(sup1), S310-S315. <u>https://doi.org/10.1080/17461391.2012.696711</u>
- Merfeld, B., Mancosky, A., Luedke, J., Griesmer, S., Erickson, J. L., Carvalho, V., & Jagim, A. R. (2022). The Impact of Early Morning Training Sessions on Total Sleep Time in Collegiate Athletes. *Int J Exerc Sci*, 15(6), 423-433.
- Gupta, L., Morgan, K., & Gilchrist, S. (2017a). Does Elite Sport Degrade Sleep Quality? A Systematic Review. *Sports Medicine*, 47(7), 1317-1333. <u>https://doi.org/10.1007/s40279-016-0650-6</u>
- Leeder, J., Glaister, M., Pizzoferro, K., Dawson, J., & Pedlar, C. (2012). Sleep duration and quality in elite athletes measured using wristwatch actigraphy. *J Sports Sci*, 30(6), 541-545. <u>https://doi.org/10.1080/02640414.2012.660188</u>
- Watson, A. M. (2017). Sleep and athletic performance. *Current sports medicine* reports, 16(6), 413-418.
- Landry, G. J., Best, J. R., & Liu-Ambrose, T. (2015). Measuring sleep quality in older adults: a comparison using subjective and objective methods. *Frontiers in aging neuroscience*, 7, 166. <u>https://doi.org/10.3389/fnagi.2015.00166</u>
- Jackowska, M., Dockray, S., Hendrickx, H., & Steptoe, A. (2011). Psychosocial Factors and Sleep Efficiency: Discrepancies Between Subjective and Objective Evaluations of Sleep. *Psychosomatic Medicine*, *73*(9), 810-816. https://doi.org/10.1097/PSY.0b013e3182359e77

- 20. Areta, J. L., Taylor, H. L., & Koehler, K. (2021). Low energy availability: history, definition and evidence of its endocrine, metabolic and physiological effects in prospective studies in females and males. *European Journal of Applied Physiology*, 121(1), 1-21. <u>https://doi.org/10.1007/s00421-020-04516-0</u>
- Wasserfurth, P., Palmowski, J., Hahn, A., & Krüger, K. (2020). Reasons for and Consequences of Low Energy Availability in Female and Male Athletes: Social Environment, Adaptations, and Prevention. *Sports Med Open*, 6(1), 44. <u>https://doi.org/10.1186/s40798-020-00275-6</u>
- Wade, G. N., Schneider, J. E., & Li, H. Y. (1996). Control of fertility by metabolic cues. *Am J Physiol*, 270(1 Pt 1), E1<u>https://doi.org/10.1152/ajpendo.1996.270.1.E1</u>
- 23. Koehler, K., Hoerner, N. R., Gibbs, J. C., Zinner, C., Braun, H., De Souza, M. J., & Schaenzer, W. (2016). Low energy availability in exercising men is associated with reduced leptin and insulin but not with changes in other metabolic hormones. *J Sports Sci*, 34(20), 1921-1929. <u>https://doi.org/10.1080/02640414.2016.1142109</u>
- Sovetkina, A., Nadir, R., Fung, J., Nadjarpour, A., & Beddoe, B. (2020). The Physiological Role of Ghrelin in the Regulation of Energy and Glucose Homeostasis. *Cureus*, 12. <u>https://doi.org/10.7759/cureus.7941</u>
- Loucks, A. B., Verdun, M., & Heath, E. M. (1998). Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J Appl Physiol* (1985), 84(1), 37-46. <u>https://doi.org/10.1152/jappl.1998.84.1.37</u>

- Loucks, A. B., & Thuma, J. R. (2003). Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *J Clin Endocrinol Metab*, 88(1), 297-311. <u>https://doi.org/10.1210/jc.2002-020369</u>
- Opstad, P. K. (1992). Androgenic hormones during prolonged physical stress, sleep, and energy deficiency. *The Journal of Clinical Endocrinology & Metabolism*, 74(5), 1176-1183. <u>https://doi.org/10.1210/jcem.74.5.1314847</u>
- Barrow, G. W., & Saha, S. (1988). Menstrual irregularity and stress fractures in collegiate female distance runners. *The American journal of sports medicine*, 16(3), 209–216. <u>https://doi.org/10.1177/036354658801600302</u>
- 29. Schoeller, D. A. (1990). How accurate is self-reported dietary energy intake? *Nutr Rev*, 48(10), 373-379. <u>https://doi.org/10.1111/j.1753-4887.1990.tb02882.x</u>
- Ravelli, M. N., & Schoeller, D. A. (2020). Traditional Self-Reported Dietary Instruments Are Prone to Inaccuracies and New Approaches Are Needed. *Frontiers in nutrition*, 7, 90. <u>https://doi.org/10.3389/fnut.2020.00090</u>
- 31. Tarnowski, C. A., Wardle, S. L., O'Leary, T. J., Gifford, R. M., Greeves, J. P., &
 Wallis, G. A. (2023a). Measurement of Energy Intake Using the Principle of Energy
 Balance Overcomes a Critical Limitation in the Assessment of Energy
 Availability. *Sports Med Open*, 9(1), 16. <u>https://doi.org/10.1186/s40798-023-00558-8</u>
- Sterringer, T., & Larson-Meyer, D. E. (2022). RMR Ratio as a Surrogate Marker for Low Energy Availability. *Current nutrition reports*, *11*(2), 263–272. https://doi.org/10.1007/s13668-021-00385-x

- Mtaweh, H., Tuira, L., Floh, A. A., & Parshuram, C. S. (2018). Indirect Calorimetry: History, Technology, and Application. *Front Pediatr*, 6,
 257. https://doi.org/10.3389/fped.2018.00257
- Fahrenholtz, I. L., Sjödin, A., Benardot, D., Tornberg Å, B., Skouby, S., Faber, J., . . .
 Melin, A. K. (2018). Within-day energy deficiency and reproductive function in female endurance athletes. *Scand J Med Sci Sports*, 28(3), 1139-1146. <u>https://doi.org/10.1111/sms.13030</u>
- 35. Jagim, A. R., Fields, J., Magee, M. K., Kerksick, C. M., & Jones, M. T. (2022). Contributing Factors to Low Energy Availability in Female Athletes: A Narrative Review of Energy Availability, Training Demands, Nutrition Barriers, Body Image, and Disordered Eating. *Nutrients*, 14(5). <u>https://doi.org/10.3390/nu14050986</u>
- Strock, N. C. A., Koltun, K. J., Southmayd, E. A., Williams, N. I., & De Souza, M. J. (2020). Indices of Resting Metabolic Rate Accurately Reflect Energy Deficiency in Exercising Women. *International journal of sport nutrition and exercise metabolism*, 30(1), 14–24. <u>https://doi.org/10.1123/ijsnem.2019-0199</u>
- 37. Logue, D. M., Madigan, S. M., Melin, A., Delahunt, E., Heinen, M., Donnell, S.-J.
 M., & Corish, C. A. (2020). Low Energy Availability in Athletes 2020: An Updated Narrative Review of Prevalence, Risk, Within-Day Energy Balance, Knowledge, and Impact on Sports Performance. *Nutrients*, *12*(3), 835.
- 38. Lieberman, J. L., MJ, D. E. S., Wagstaff, D. A., & Williams, N. I. (2018). Menstrual Disruption with Exercise Is Not Linked to an Energy Availability Threshold. *Med Sci Sports Exerc*, 50(3), 551-561. <u>https://doi.org/10.1249/mss.000000000001451</u>

- 39. De Souza, M. J., Koltun, K. J., Strock, N. C. A., & Williams, N. I. (2019a).
 Rethinking the concept of an energy availability threshold and its role in the Female
 Athlete Triad. *Current Opinion in Physiology*, *10*, 3542. https://doi.org/https://doi.org/10.1016/j.cophys.2019.04.001
- Greenleaf, C., Petrie, T. A., Carter, J., & Reel, J. J. (2009). Female collegiate athletes: prevalence of eating disorders and disordered eating behaviors. *J Am Coll Health*, 57(5), 489-495. <u>https://doi.org/10.3200/jach.57.5.489-496</u>
- 41. Loucks, A. B., & Heath, E. M. (1994a). Induction of low-T3 syndrome in exercising women occurs at a threshold of energy availability. *Am J Physiol*, 266(3 Pt 2), R817-823. <u>https://doi.org/10.1152/ajpregu.1994.266.3.R817</u>
- 42. Lundstrom, E. A., De Souza, M. J., Canil, H. N., & Williams, N. I. (2023). Sex differences and indications of metabolic compensation in within-day energy balance in elite Division 1 swimmers. *Appl Physiol Nutr Metab*, *48*(1), 74-87. https://doi.org/10.1139/apnm-2022-0161
- Bellissimo, M., Licata, A., Nucci, A., Thompson, W., & Benardot, D. (2019).
 Relationships Between Estimated Hourly Energy Balance and Body Composition in Professional Cheerleaders. *Journal of Science in Sport and Exercise*, *1*, 1-9. https://doi.org/10.1007/s42978-019-0004-9
- 44. Venter, R. (2012). Role of sleep in performance and recovery of athletes: A review article. *South African Journal for Research in Sport, Physical Education and Recreation*, *34*, 167-184.
- Doherty, R., Madigan, S., Warrington, G., & Ellis, J. (2019). Sleep and Nutrition Interactions: Implications for Athletes. *Nutrients*, *11*(4), 822.

- 46. Itani, O., Jike, M., Watanabe, N., & Kaneita, Y. (2017). Short sleep duration and health outcomes: a systematic review, meta-analysis, and meta-regression. *Sleep medicine*, 32, 246–256. <u>https://doi.org/10.1016/j.sleep.2016.08.006</u>
- 47. Medic, G., Wille, M., & Hemels, M. E. H. (2017). Short- and long-term health consequences of sleep disruption. *Nature and Science of Sleep*, *9*, 151-161. <u>https://doi.org/10.2147/NSS.S134864</u>
- Liew, S. C., & Aung, T. (2021). Sleep deprivation and its association with diseases- a review. *Sleep Medicine*, 77, 192-204. https://doi.org/https://doi.org/10.1016/j.sleep.2020.07.048
- 49. Blumert, P. A., Crum, A. J., Ernsting, M., Volek, J. S., Hollander, D. B., Haff, E. E., & Haff, G. G. (2007). The acute effects of twenty-four hours of sleep loss on the performance of national caliber male collegiate weightlifters. *The Journal of Strength & Conditioning Research*, 21(4).
- 50. Atkinson, G., & Davenne, D. (2007). Relationships between sleep, physical activity and human health. *Physiology & behavior*, 90(2-3), 229–235. <u>https://doi.org/10.1016/j.physbeh.2006.09.015</u>
- 51. Spiegel, K., Tasali, E., Penev, P., & Van Cauter, E. (2004). Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med*, *141*(11), 846-850. <u>https://doi.org/10.7326/0003-4819-141-11-200412070-00008</u>
- 52. Chennaoui, M., Arnal, P. J., Drogou, C., Sauvet, F., & Gomez-Merino, D. (2016).Sleep extension increases IGF-I concentrations before and during sleep deprivation in

healthy young men. *Applied Physiology, Nutrition, and Metabolism, 41*(9), 963-970. <u>https://doi.org/10.1139/apnm-2016-0110</u> %M 27560704

- 53. Chennaoui, M., Bougard, C., Drogou, C., Langrume, C., Miller, C., Gomez-Merino,
 D., & Vergnoux, F. (2016). Stress Biomarkers, Mood States, and Sleep during a
 Major Competition: "Success" and "Failure" Athlete's Profile of High-Level
 Swimmers. *Front Physiol*, 7, 94. <u>https://doi.org/10.3389/fphys.2016.00094</u>
- Notelovitz, M. (2002). Androgen effects on bone and muscle. *Fertility and* Sterility, 77, 34-41. <u>https://doi.org/https://doi.org/10.1016/S0015-0282(02)02968-0</u>
- 55. Davis, S. R., & Wahlin-Jacobsen, S. (2015). Testosterone in women--the clinical significance. *Lancet Diabetes Endocrinol*, *3*(12), 980992. <u>https://doi.org/10.1016/s2213-8587(15)00284-3</u>
- 56. Cardinale, M., & Stone, M. H. (2006). Is testosterone influencing explosive performance? *The Journal of Strength & Conditioning Research*, 20(1).
- 57. Miller, G. E., Cohen, S., Pressman, S., Barkin, A., Rabin, B. S., & Treanor, J. J. (2004). Psychological stress and antibody response to influenza vaccination: when is the critical period for stress, and how does it get inside the body? *Psychosomatic medicine*, 66(2), 215-223.
- Prather, A. A., Hall, M., Fury, J. M., Ross, D. C., Muldoon, M. F., Cohen, S., & Marsland, A. L. (2012). Sleep and antibody response to hepatitis B vaccination. *Sleep*, *35*(8), 1063–1069. <u>https://doi.org/10.5665/sleep.1990</u>
- 59. Irwin, M. R. (2015). Why Sleep Is Important for Health: A Psychoneuroimmunology Perspective. *Annual Review of Psychology*, 66(1), 143 172. <u>https://doi.org/10.1146/annurev-psych-010213-115205</u>

- Ibarra-Coronado, E. G., Pantaleón-Martínez, A. M., Velazquéz-Moctezuma, J., Prospéro-García, O., Méndez-Díaz, M., Pérez-Tapia, M., Pavón, L., & Morales-Montor, J. (2015). The Bidirectional Relationship between Sleep and Immunity against Infections. *Journal of immunology research*, 2015, 678164. https://doi.org/10.1155/2015/678164
- 61. Orzech, K. M., Acebo, C., Seifer, R., Barker, D., & Carskadon, M. A. (2014). Sleep patterns are associated with common illness in adolescents. *Journal of Sleep Research*, *23*(2), 133-142.
- Friman, G., Wright, J. E., Ilback, N. G., Beisel, W. R., White, J. D., Sharps, D. S., ...
 Vogel, J. A. (1985). Does fever or myalgia indicate reduced physical performance capacity in viral infections? *Acta Medica Scandinavica*, 217(4), 353-361.
- Friman, G. (1977). Effect of acute infectious disease on isometric muscle strength. *Scandinavian Journal of Clinical and Laboratory Investigation*, *37*(4), 303-308.
- 64. Friman, G. (1976). Effects of acute infectious disease on circulatory function. *Acta medica Scandinavica. Supplementum*, *592*, 1-62.
- 65. Kirschen, G. W., Jones, J. J., & Hale, L. (2020). The Impact of Sleep Duration on Performance Among Competitive Athletes: A Systematic Literature Review. *Clinical Journal of Sport Medicine*, 30(5), 503-

512. https://doi.org/10.1097/jsm.000000000000622

66. Thun, E., Bjorvatn, B., Flo, E., Harris, A., & Pallesen, S. (2015). Sleep, circadian rhythms, and athletic performance. *Sleep Med Rev*, 23, 1-

9. <u>https://doi.org/10.1016/j.smrv.2014.11.003</u>

- Juliff, L. E., Halson, S. L., Hebert, J. J., Forsyth, P. L., & Peiffer, J. J. (2018). Longer Sleep Durations Are Positively Associated With Finishing Place During a National Multiday Netball Competition. *J Strength Cond Res*, *32*(1), 189-194. <u>https://doi.org/10.1519/jsc.000000000001793</u>
- Bond, V., Balkissoon, B., Franks, B. D., Brwnlow, R., Caprarola, M., Bartley, D., & Banks, M. (1986). Effects of sleep deprivation on performance during submaximal and maximal exercise. *The Journal of sports medicine and physical fitness*, 26(2), 169-174.
- 69. Mah, C. (2008). Extended sleep and the effects on mood and athletic performance in collegiate swimmers. *Sleep*, *31*.
- 70. Kohyama, J. (2021). Which is more important for health: sleep quantity or sleep quality? *Children*, 8(7), 542.
- Nelson, K. L., Davis, J. E., & Corbett, C. F. (2022). Sleep quality: An evolutionary concept analysis. *Nurs Forum*, 57(1), 144-151. <u>https://doi.org/10.1111/nuf.12659</u>
- 72. Claudino, J. G., Gabbett, T. J., de Sá Souza, H., Simim, M., Fowler, P., de Alcantara Borba, D., . . . D'Almeida, V. (2019). Which parameters to use for sleep quality monitoring in team sport athletes? A systematic review and meta-analysis. *BMJ open sport & exercise medicine*, 5(1), bmjsem-2018-000475.
- 73. Patel, A. K., Reddy, V., & Araujo, J. F. (2022). Physiology, sleep stages.In *StatPearls [Internet]*. StatPearls Publishing.
- 74. Brinkman, J. E., Reddy, V., & Sharma, S. (2023a). Physiology of Sleep.
 In *StatPearls*. StatPearls Publishing. Copyright © 2023, StatPearls Publishing LLC.

- Collop, N. A., Salas, R. E., Delayo, M., & Gamaldo, C. (2008). Normal Sleep and Circadian Processes. *Critical Care Clinics*, 24(3), 449-460. https://doi.org/https://doi.org/10.1016/j.ccc.2008.02.002
- Šušmáková, K. (2004). Human sleep and sleep EEG. *Measurement science review*, 4(2), 59-74.
- Roth, T. (2004). Characteristics and determinants of normal sleep. *J Clin Psychiatry*, 65(Suppl 16), 8-11.
- 78. Carskadon, M. A., & Dement, W. C. (2005). Normal human sleep: an overview. *Principles and practice of sleep medicine*, *4*(1), 13-23.
- Caporro, M., Haneef, Z., Yeh, H. J., Lenartowicz, A., Buttinelli, C., Parvizi, J., & Stern, J. M. (2012). Functional MRI of sleep spindles and K-complexes. *Clinical Neurophysiology*, *123*(2), 303-309. <u>https://doi.org/https://doi.org/10.1016/j.clinph.2011.06.018</u>
- Smith, C., Aubrey, J., & Peters, K. (2004). Different Roles for REM and Stage 2 Sleep In Motor Learning: A Proposed Model. *Psychologica Belgica. Special Issue: Cognition in Slumberland. Mechanisms of Information Processing in the Sleep-Wake Cycle*, 44, 81-104. <u>https://doi.org/10.4324/9780203307991_chapter_5</u>
- 81. Rodríguez-Labrada, R., Galicia-Polo, L., Canales-Ochoa, N., Voss, U., Tuin, I., Peña-Acosta, A., . . . Velázquez-Pérez, L. (2019). Sleep spindles and K-complex activities are decreased in spinocerebellar ataxia type 2: relationship to memory and motor performances. *Sleep Medicine*, 60, 188-

196. https://doi.org/https://doi.org/10.1016/j.sleep.2019.04.005

- Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan. *Sleep*, 27(7), 1255-1273. <u>https://doi.org/10.1093/sleep/27.7.1255</u>
- Biller, M. W., Dunican, I. C., Omond, S. E. T., Boukhris, O., Stevenson, S.,
 Lambing, K., & Bender, A. M. (2023a). Pyjamas, Polysomnography and Professional
 Athletes: The Role of Sleep Tracking Technology in Sport. *Sports*, *11*(1), 14.
- Dijk, D. J. (2009). Regulation and functional correlates of slow wave sleep. *J Clin Sleep Med*, 5(2 Suppl), S6-15.
- Sassin, J. F., Parker, D. C., Mace, J. W., Gotlin, R. W., Johnson, L. C., & Rossman,
 L. G. (1969). Human Growth Hormone Release: Relation to Slow-Wave Sleep and
 Sleep-Waking Cycles. *Science*, *165*(3892), 513-515.
- 86. Léger, D., Debellemaniere, E., Rabat, A., Bayon, V., Benchenane, K., & Chennaoui, M. (2018a). Slow-wave sleep: From the cell to the clinic. *Sleep Medicine Reviews*, *41*, 113-132. <u>https://doi.org/https://doi.org/10.1016/j.smrv.2018.01.008</u>
- Schmitz, N. C. M., van der Werf, Y. D., & Lammers-van der Holst, H. M. (2022).
 The Importance of Sleep and Circadian Rhythms for Vaccination Success and
 Susceptibility to Viral Infections. *Clocks & Sleep*, *4*(1), 66-79.
- 88. Strobl, J. S., & Thomas, M. J. (1994). Human growth hormone. *Pharmacological reviews*, *46*(1), 1-34.
- 89. Gronfier, C., Luthringer, R., Follenius, M., Schaltenbrand, N., Macher, J. P., Muzet,A., & Brandenberger, G. (1996). A quantitative evaluation of the relationships

between growth hormone secretion and delta wave electroencephalographic activity during normal sleep and after enrichment in delta waves. *Sleep*, *19*(10), 817-824.

- 90. Kanaley, J. A., Weltman, J. Y., Veldhuis, J. D., Rogol, A. D., Hartman, M. L., & Weltman, A. (1997). Human growth hormone response to repeated bouts of aerobic exercise. *J Appl Physiol (1985)*, *83*(5), 1756-1761. <u>https://doi.org/10.1152/jappl.1997.83.5.1756</u>
- 91. Kato, Y., Murakami, Y., Sohmiya, M., & Nishiki, M. (2002). Regulation of human growth hormone secretion and its disorders. *Intern Med*, 41(1), 713. https://doi.org/10.2169/internalmedicine.41.7
- Milewski, M. D., Skaggs, D. L., Bishop, G. A., Pace, J. L., Ibrahim, D. A., Wren, T. A., & Barzdukas, A. (2014). Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. *J Pediatr Orthop*, *34*(2), 129-133. <u>https://doi.org/10.1097/bpo.00000000000151</u>
- 93. Besedovsky, L., Lange, T., & Born, J. (2012). Sleep and immune function. *Pflügers Archiv - European Journal of Physiology*, 463(1), 121137. https://doi.org/10.1007/s00424-011-1044-0
- 94. Mehrsafar, A. H., Serrano Rosa, M. A., Moghadam Zadeh, A., & Gazerani, P. (2020).
 Stress, Professional Lifestyle, and Telomere Biology in Elite Athletes: A Growing Trend in Psychophysiology of Sport. *Front Psychol*, *11*, 567214. <u>https://doi.org/10.3389/fpsyg.2020.567214</u>
- 95. Sabato, T. M., Walch, T. J., & Caine, D. J. (2016). The elite young athlete: strategies to ensure physical and emotional health. *Open Access J Sports Med*, *7*, 99-113. <u>https://doi.org/10.2147/oajsm.s96821</u>

- 96. Campbell, E., Irving, R., Bailey, J., Dilworth, L., & Abel, W. (2018). Overview of psychophysiological stress and the implications for junior athletes. *Am. J. Sports Sci. Med*, 6(3), 72-78.
- 97. Drew, M., Vlahovich, N., Hughes, D., Appaneal, R., Burke, L. M., Lundy, B., ... Waddington, G. (2018). Prevalence of illness, poor mental health and sleep quality and low energy availability prior to the 2016 Summer Olympic Games. *British Journal of Sports Medicine*, 52(1), 47-53. <u>https://doi.org/10.1136/bjsports-2017-098208</u>
- 98. Svendsen, I. S., Taylor, I. M., Tønnessen, E., Bahr, R., & Gleeson, M. (2016). Training-related and competition-related risk factors for respiratory tract and gastrointestinal infections in elite cross-country skiers. *British Journal of Sports Medicine*, 50(13), 809-815.
- Gleeson, M., McFarlin, B., & Flynn, M. (2006). Exercise and Toll-like receptors. *Exerc Immunol Rev*, 12(1), 34-53.
- Halson, S. L. (2014). Sleep in Elite Athletes and Nutritional Interventions to Enhance
 Sleep. *Sports Medicine*, 44(1), 13-23. <u>https://doi.org/10.1007/s40279-014-0147-0</u>
- Fullagar, H. H., Vincent, G. E., McCullough, M., Halson, S., & Fowler, P. (2022).
 Sleep and sport performance. *Journal of Clinical Neurophysiology*, 10.1097.
- 102. Payne, J. D., & Nadel, L. (2004). Sleep, dreams, and memory consolidation: the role of the stress hormone cortisol. *Learn Mem*, *11*(6), 671-678. https://doi.org/10.1101/lm.77104
- Miller, K. E., & Gehrman, P. R. (2019). REM sleep: what is it good for? *Current Biology*, 29(16), R806-R807.

- 104. Shapiro, C. M., Bortz, R., Mitchell, D., Bartel, P., & Jooste, P. (1981). Slow-wave sleep: a recovery period after exercise. *Science*, 214(4526), 1253-1254.
- 105. Sargent, C., Lastella, M., Halson, S. L., & Roach, G. D. (2021). How much sleep does an elite athlete need? *International journal of sports physiology and performance*, 16(12), 1746-1757.
- 106. Lauderdale, D. S., Knutson, K. L., Yan, L. L., Liu, K., & Rathouz, P. J. (2008). Self-reported and measured sleep duration: how similar are they? *Epidemiology*, *19*(6), 838-845. <u>https://doi.org/10.1097/EDE.0b013e318187a7b0</u>
- Brauer, A. A., Athey, A. B., Ross, M. J., & Grandner, M. A. (2019). Sleep and Health Among Collegiate Student Athletes. *Chest*, 156(6), 12341245. <u>https://doi.org/https://doi.org/10.1016/j.chest.2019.08.1921</u>
- 108. Sargent, C., Lastella, M., Halson, S. L., & Roach, G. D. (2014). The impact of training schedules on the sleep and fatigue of elite athletes. *Chronobiology International*, 31(10), 1160-1168. <u>https://doi.org/10.3109/07420528.2014.957306</u>
- Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193-213. https://doi.org/https://doi.org/10.1016/0165-1781(89)90047-4
- 110. Fabbri, M., Beracci, A., Martoni, M., Meneo, D., Tonetti, L., & Natale, V. (2021).
 Measuring Subjective Sleep Quality: A Review. *Int J Environ Res Public Health*, 18(3). <u>https://doi.org/10.3390/ijerph18031082</u>
- 111. Mollayeva, T., Thurairajah, P., Burton, K., Mollayeva, S., Shapiro, C. M., &Colantonio, A. (2016). The Pittsburgh sleep quality index as a screening tool for sleep

dysfunction in clinical and non-clinical samples: A systematic review and metaanalysis. *Sleep Medicine Reviews*, 25, 52-

73. https://doi.org/https://doi.org/10.1016/j.smrv.2015.01.009

- 112. Van de Water, A. T., Holmes, A., & Hurley, D. A. (2011). Objective measurements of sleep for non-laboratory settings as alternatives to polysomnography--a systematic review. *J Sleep Res*, 20(1 Pt 2), 183-200. <u>https://doi.org/10.1111/j.1365-2869.2009.00814.x</u>
- Hertenstein, E., Gabryelska, A., Spiegelhalder, K., Nissen, C., Johann, A. F.,
 Umarova, R., . . . Feige, B. (2018). Reference Data for Polysomnography-Measured and Subjective Sleep in Healthy Adults. *J Clin Sleep Med*, *14*(4), 523-532. <u>https://doi.org/10.5664/jcsm.7036</u>
- Walia, H. K., & Mehra, R. (2019). Chapter 24 Practical aspects of actigraphy and approaches in clinical and research domains. In K. H. Levin & P. Chauvel (Eds.), *Handbook of Clinical Neurology* (Vol. 160, pp. 371-379).
 Elsevier. <u>https://doi.org/https://doi.org/10.1016/B978-0-444-64032-1.00024-2</u>
- 115. Martin, J. L., & Hakim, A. D. (2011). Wrist actigraphy. *Chest*, *139*(6), 15141527. <u>https://doi.org/10.1378/chest.10-1872</u>
- Ancoli-Israel, S., Martin, J. L., Blackwell, T., Buenaver, L., Liu, L., Meltzer, L. J., . .
 Taylor, D. J. (2015). The SBSM Guide to Actigraphy Monitoring: Clinical and Research Applications. *Behav Sleep Med*, *13 Suppl 1*, S4s38. https://doi.org/10.1080/15402002.2015.1046356
- Blackwell, T., Redline, S., Ancoli-Israel, S., Schneider, J. L., Surovec, S., Johnson, N.L., . . . Study of Osteoporotic Fractures Research, G. (2008). Comparison of Sleep

Parameters from Actigraphy and Polysomnography in Older Women: The SOF Study. *Sleep*, *31*(2), 283-291. <u>https://doi.org/10.1093/sleep/31.2.283</u>

- Miller, D., Roach, G., Lastella, M., Scanlan, A., Bellenger, C., Halson, S., & Sargent,
 C. (2021a). A Validation Study of a Commercial Wearable Device to Automatically
 Detect and Estimate Sleep. *Biosensors*, *11*,
 185. <u>https://doi.org/10.3390/bios11060185</u>
- 119. Miller, D. J., Sargent, C., & Roach, G. D. (2022). A Validation of Six Wearable Devices for Estimating Sleep, Heart Rate and Heart Rate Variability in Healthy Adults. *Sensors*, 22(16), 6317.
- 120. De Zambotti, M., Cellini, N., Goldstone, A., Colrain, I. M., & Baker, F. C. (2019).
 Wearable sleep technology in clinical and research settings. *Medicine and science in sports and exercise*, *51*(7), 1538.
- Brooke, S. M., An, H.-S., Kang, S.-K., Noble, J. M., Berg, K. E., & Lee, J.-M.
 (2017). Concurrent Validity of Wearable Activity Trackers Under Free-Living Conditions. *The Journal of Strength & Conditioning Research*, *31*(4).
- Miller, D. J., Lastella, M., Scanlan, A. T., Bellenger, C., Halson, S. L., Roach, G. D., & Sargent, C. (2020). A validation study of the WHOOP strap against polysomnography to assess sleep. *J Sports Sci*, *38*(22), 2631-2636. <u>https://doi.org/10.1080/02640414.2020.1797448</u>
- Berryhill, S., Morton, C. J., Dean, A., Berryhill, A., Provencio-Dean, N., Patel, S. I., .
 Parthasarathy, S. (2020). Effect of wearables on sleep in healthy individuals: a randomized crossover trial and validation study. *J Clin Sleep Med*, *16*(5), 775-783. <u>https://doi.org/10.5664/jcsm.8356</u>

Penev, P. D. (2012). Update on Energy Homeostasis and Insufficient Sleep. *The Journal of Clinical Endocrinology & Metabolism*, 97(6), 1792 1801. https://doi.org/10.1210/jc.2012-1067

125. Vanheest, J. L., Rodgers, C. D., Mahoney, C. E., & De Souza, M. J. (2014). Ovarian suppression impairs sport performance in junior elite female swimmers. *Medicine and science in sports and exercise*, 46(1), 156-166. <u>https://doi.org/10.1249/mss.0b013e3182a32b72</u>

- Mah, C. D., Mah, K. E., Kezirian, E. J., & Dement, W. C. (2011). The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep*, *34*(7), 943-950.
- 127. Doyle-Lucas, A. F., & Davy, B. M. (2011). Development and evaluation of an educational intervention program for pre-professional adolescent ballet dancers: nutrition for optimal performance. *Journal of Dance Medicine & Science*, *15*(2), 65-75.
- 128. Elias, S. S. M., Saad, H. A., Taib, M. N. M., & Jamil, Z. (2018). Effects of sports nutrition education intervention on sports nutrition knowledge, attitude and practice, and dietary intake of Malaysian team sports athletes. *Malaysian journal of nutrition*, 24(1).

129. Driller, M. W., Lastella, M., & Sharp, A. P. (2019). Individualized sleep education improves subjective and objective sleep indices in elite cricket athletes: A pilot study. *Journal of Sports Sciences*, *37*(17), 2021-2025. https://doi.org/10.1080/02640414.2019.1616900

- O'Donnell, S., & Driller, M. W. (2017). Sleep-hygiene Education improves Sleep Indices in Elite Female Athletes. *Int J Exerc Sci*, 10(4), 522-530.
- 131. Lundstrom, E. (2020). Effectiveness of Wearable Technology for Predicting Measures of Metabolism and Performance in Collegiate Division 1 Swimmers [Master's thesis]. The Pennsylvania State University.
- Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *J Health Soc Behav*, 24(4), 385-396.
- 133. Kellmann, M., & Kallus, K. W. (2001). *Recovery-stress questionnaire for athletes: User manual*. Human Kinetics.
- Santos, D. A., Gobbo, L. A., Matias, C. N., Petroski, E. L., Gonçalves, E. M., Cyrino, E. S., Minderico, C. S., Sardinha, L. B., & Silva, A. M. (2013). Body composition in taller individuals using DXA: A validation study for athletic and non-athletic populations. *Journal of sports sciences*, *31*(4), 405–413. https://doi.org/10.1080/02640414.2012.734918
- 135. Wallace, L. K., Slattery, K. M., & Coutts, A. J. (2009). The ecological validity and application of the session-RPE method for quantifying training loads in swimming. J Strength Cond Res, 23(1), 33-38. https://doi.org/10.1519/JSC.0b013e3181874512
- Borg, G. A. (1982). Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*, 14(5), 377-381.
- 137. Weir, J. B. (1949). New methods for calculating metabolic rate with special reference to protein metabolism. *J Physiol*, *109*(1-2), 1-
 - 9. https://doi.org/10.1113/jphysiol.1949.sp004363

- Harris, J. A., & Benedict, F. G. (1918). A Biometric Study of Human Basal Metabolism. *Proc Natl Acad Sci U S A*, 4(12), 370-373. https://doi.org/10.1073/pnas.4.12.370
- Hayes, M., Chustek, M., Wang, Z., Gallagher, D., Heshka, S., Spungen, A., . . .
 Heymsfield, S. B. (2002a). DXA: potential for creating a metabolic map of organtissue resting energy expenditure components. *Obes Res*, *10*(10), 969-977. <u>https://doi.org/10.1038/oby.2002.132</u>
- 140. Koehler, K., Williams, N. I., Mallinson, R. J., Southmayd, E. A., Allaway, H. C., & De Souza, M. J. (2016a). Low resting metabolic rate in exercise-associated amenorrhea is not due to a reduced proportion of highly active metabolic tissue compartments. *Am J Physiol Endocrinol Metab*, *311*(2), E480-487. <u>https://doi.org/10.1152/ajpendo.00110.2016</u>
- 141. Cunningham, J. J. (1980). A reanalysis of the factors influencing basal metabolic rate in normal adults. *Am J Clin Nutr*, *33*(11), 2372-2374. https://doi.org/10.1093/ajcn/33.11.2372
- 142. Cunningham, J. J. (1991). Body composition as a determinant of energy expenditure:
 a synthetic review and a proposed general prediction equation. *Am J Clin Nutr*, 54(6), 963-969. <u>https://doi.org/10.1093/ajcn/54.6.963</u>
- 143. Ohayon, M., Wickwire, E. M., Hirshkowitz, M., Albert, S. M., Avidan, A., Daly, F. J., Dauvilliers, Y., Ferri, R., Fung, C., Gozal, D., Hazen, N., Krystal, A., Lichstein, K., Mallampalli, M., Plazzi, G., Rawding, R., Scheer, F. A., Somers, V., & Vitiello, M. V. (2017). National Sleep Foundation's sleep quality recommendations: first report. *Sleep health*, *3*(1), 6–19. <u>https://doi.org/10.1016/j.sleh.2016.11.006</u>

- Yamanaka, A., Beuckmann, C. T., Willie, J. T., Hara, J., Tsujino, N., Mieda, M., Tominaga, M., Yagami, K.i, Sugiyama, F., Goto, K., Yanagisawa, M., & Sakurai, T. (2003). Hypothalamic orexin neurons regulate arousal according to energy balance in mice. *Neuron*, 38(5), 701–713. <u>https://doi.org/10.1016/s0896-6273(03)00331-3</u>
- 145. Inutsuka, A., & Yamanaka, A. (2013). The physiological role of orexin/hypocretin neurons in the regulation of sleep/wakefulness and neuroendocrine functions. *Frontiers in endocrinology*, 4, 18.
- 146. Lauer, C. J., & Krieg, J.-C. (2004). Sleep in eating disorders. *Sleep Medicine Reviews*, 8(2), 109-118. <u>https://doi.org/https://doi.org/10.1016/S1087-0792(02)00122-3</u>
- 147. Lee, M. G., Hassani, O. K., & Jones, B. E. (2005). Discharge of identified orexin/hypocretin neurons across the sleep-waking cycle. *Journal of Neuroscience*, 25(28), 6716-6720.
- 148. Pan, W., & Kastin, A. J. (2014). Leptin: a biomarker for sleep disorders?. Sleep medicine reviews, 18(3), 283-290.
- 149. Sovetkina, A., Nadir, R., Fung, J. N. M., Nadjarpour, A., & Beddoe, B. (2020). The physiological role of ghrelin in the regulation of energy and glucose homeostasis. *Cureus*, 12(5).
- Sall, H., & Timperley, J. (2015). Bradycardia in anorexia nervosa. *Case Reports*, 2015, bcr2015211273.
- 151. Kanaley, J. A., Weltman, J. Y., Veldhuis, J. D., Rogol, A. D., Hartman, M. L., &
 Weltman, A. (1997). Human growth hormone response to repeated bouts of aerobic exercise. *Journal of Applied Physiology*.

- Bishop, P. A., Jones, E., & Woods, A. K. (2008). Recovery from training: a brief review: brief review. *The Journal of Strength & Conditioning Research*, 22(3), 1015-1024.
- 153. Fahrenholtz, I. L., Melin, A. K., Wasserfurth, P., Stenling, A., Logue, D., Garthe, I.,
 ... & Torstveit, M. K. (2022). Risk of low energy availability, disordered eating,
 exercise addiction, and food intolerances in female endurance athletes. *Frontiers in sports and active living*, 160.
- Lichtenstein, M. B., Hinze, C. J., Emborg, B., Thomsen, F., & Hemmingsen, S. D.
 (2017). Compulsive exercise: links, risks and challenges faced. *Psychology research* and behavior management, 10, 85–95. <u>https://doi.org/10.2147/PRBM.S113093</u>
- 155. Groom, J. M. (2019). Associations between low energy availability, stress and anxiety profiles and sleep patterns (Doctoral dissertation, University of Otago).

ACADEMIC VITA

Megan Elizabeth Conklin

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EDUCATION

The Pennsylvania State University: Schreyer Honors College, University Park, PA

Bachelor of Science in Biology Honors in Kinesiology Graduate May 2024

THESIS:

Energy Availability and Sleep Quality on Training Responses and Sport Performance in Collegiate Division I Swimmers

Supervised by Dr. Nancy I. Williams

RELATED EXPERIENCE:

Women's Health and Exercise Lab, Noll Laboratory, University Park, PA, Spring 2022present

Research Assistant

- Review primary literature, develop research questions, and conduct data analysis on thesis project topic
- Communicate findings through scientific writing, culminating in the completion of an honors thesis
- Assist graduate students with participant data collection, including VO2 max testing, cold pressor testing, and RMR testing

University Health Services, Penn State University Park, PA, Spring 2023-Fall 2023 Clinic Intern

- Obtained patients' vital signs and chief complaints upon arrival to the University Health Services Clinic
- Performed basic procedures such as visual acuity
- Utilized an electron medical record system for documentation
- Communicated relevant health information to providers and nursing staff to allow for seamless patient care

Anderson Emergency Department, St. Luke's University Health Network, Easton, PA, Summer 2022-present

Emergency Room Technician

- Record patients' vital signs and chief complaints upon rooming them in the Emergency Department
- Work with nurses, physician assistants, and physicians to respond quickly and effectively to traumas
- Communicate with the health care team about patient care
- Utilize the electronic medical records system MyChart for documentation

Chemistry Department, Penn State University, University Park, PA, Fall 2022-present Honors Introductory Chemistry Grader

- Collaborate with the instructional team to provide valuable feedback to students on homework assignments and quizzes
- Communication with instructors concerning areas that students are struggling throughout the semester

LEADERSHIP AND EXTRACURRICULAR ACTIVITIES

Vice President Spring 2023-present, Secretary Spring 2022, Member 2021 Penn State University Pre-Physician Assistant Club

- Shared passion for the physician assistant profession through collaborating with executive board to design informational presentations
- Assisted in leading meetings and coordinating guest speaker events
- Planned and executed question and answer informational sessions for club members
- Welcomed new and returning members during involvement fair events

Certified Domestic Violence and Sexual Assault Counselor and Advocate Fall 2022present Centre Safe

- Respond to the hospital to provide person-centered support to survivors of sexual assault during their forensic examinations, medical treatments, and police statements
- Encourage and empower victims through Centre Safe's 24-hour hotline
- Initiating shelter intakes, emergency protection from abuse orders, and safety planning
- Listen to victims, creating a safe space for survivors to share their stories and be affirmed that they are not alone

AWARDS

Dean's List: Fall 2021, Spring 2022, Fall 2022, Spring 2023, Fall 2023

Braddock Scholarship Eberly College of Science Fall 2021-present

President Sparks Award Penn State University Spring 2023

President Walker Award Penn State University Spring 2022