Effect of Exercise Training on Sleep:

Implications for the Athlete Population

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Abstract

Sleep is important for physiological and psychological restoration; as such, athletes are encouraged to prioritise sleep following training and competition. Despite this, both team and individual sport athletes appear to achieve inadequate sleep duration and/or quality. Among other potential factors (i.e. schedule, sleep environment and psychological factors), the demands of exercise training are suggested to determine the sleep duration of athletes. However, the effect of exercise training on nocturnal sleep in athletes is not well-understood. Consequently, the aim of this thesis was to elucidate the effect of exercise training on sleep by completing four studies, which tested different training demands encountered by athletes.

Chapter 3 compared the sleeping patterns of athletes and non-athletes during a competition week. Thirty athletes (from team sports and individual sports) and non-athletes were monitored for their sleep over 7d via actigraphy and the Consensus Sleep Diary. Internal training load (duration x RPE) was also attained on each day using a diary. The athlete group had a lower sleep efficiency compared to non-athletes (81.7±4.8 vs. 85.3±4.0 %, p = 0.003, ES: 0.81 [moderate effect]) as a result of a longer sleep onset latency (18 vs. 10 min, p = 0.001, ES: 0.19 [small effect]). Athletes also displayed greater intra-individual variability in sleep onset latency compared with non-athletes (13±9 vs. 7±6 min, p = 0.002, ES: 0.78 [moderate effect]). Analysis of the training load diaries revealed team sport athletes, but not individual sport athletes, had a greater daily training load compared to non-athletes (650 vs. 333 AU, p <0.05), ES: 0.44 [medium effect]). However, the individual sport athletes performed more cardiovascular training sessions in the early evening (between 6 and 7pm) compared with non-athletes (33.3 vs. 19.2 %). These findings suggested sleep quality in the athlete group was lower when compared to non-athletes, which may be explained by the daily training load or constraints related to the training schedule.

To investigate the potential factors contributing to the findings contained in Chapter 3, Chapter 4 looked to examine the effect of exercise training at different intensities in the early evening on nocturnal sleep and cardiac autonomic activity within endurance-trained runners. Eight runners performed either: i) a 1h high intensity interval running session (6x5 min at 90% VO_{2peak} interspersed with 5 min recovery); ii) a 1h low intensity running session (60 min at 45% VO_{2peak}) or no exercise in the early evening (end of exercise 3.5h before bedtime). Subsequent nocturnal sleep was assessed using polysomnography, actigraphy and subjective sleep quality from the Consensus Sleep Diary, whilst cardiac autonomic activity was recorded via a 2-lead electrocardiogram. From the polysomnography analysis, total sleep time increased after high intensity interval exercise (477.4±17.7 min, p = 0.022, ES: 0.79 [moderate effect]) and low intensity exercise (479.6±15.6 min, p = 0.006, ES: 0.96 [moderate effect]) compared with no exercise (462.9±19.0 min). Time awake was lower after high intensity interval exercise (31.8±18.5 min, p = 0.047, ES: 0.77 [moderate effect]) and low intensity exercise (30.4±15.7 min, p = 0.008, ES: 0.90 [moderate effect]) compared with no exercise (46.6±20.0 min). There were no significant differences between conditions for actigraphy variables and subjective sleep quality (p > 0.05). Nocturnal heart rate variability was not different between conditions, but average heart rate increased after high intensity interval exercise (50±5 beats·min^{-1}) compared with low intensity exercise (47±5 beats·min^{-1}, p = 0.02, ES: 1.73 [large effect]) and no exercise (47±5 beats·min^{-1}, p = 0.028, ES: 0.98 [moderate effect]). This suggested endurance athletes may perform high and low intensity exercise interchangeably in the early evening without disruption to nocturnal sleep.

Chapter 5 investigated the effect of a single high intensity interval training session, previously shown to reduce muscle glycogen stores (<200mmol.kg^{-1}DW) on nocturnal

sleep. Maximum voluntary contraction and perceived muscle soreness were also monitored to assess the impact of muscle damage on sleep during the next three nights in the home setting. Seven recreationally trained males completed a 40-min run at 55% VO_{2peak} in the morning. In the afternoon, participants then performed 90-min of high intensity interval training, followed by polysomnography, actigraphy and subjective sleep quality in the sleep laboratory. Pre, post, post 24h, post 48h and post 72h, maximum voluntary contraction and perceived muscle soreness were conducted for markers of exercise-induced muscle damage. Whilst on nights two, three and four, participants sleep was monitored using actigraphy only. From the polysomnography analysis, non-rapid eye movement sleep stage 1 was reduced after the exercise condition compared with no exercise (4.1±2.9 vs. 5.7±4.2 %, p = 0.029, ES: 0.44 [small effect]) on the first night. There were no significant differences between conditions for actigraphy and subjective sleep quality, and there was no change in actigraphic sleep efficiency on nights two, three and four (p > 0.05). There was a reduction in maximum voluntary contraction at post (-6.5±2.6 %, p = 0.001), 24h post (-7.2±6.5%, p = 0.027) and 48h post (-4.4±4.4 %, p = 0.039) compared to pre exercise. Perceived muscle soreness was also higher at post (2±2 AU, p = 0.026), 24h post (4±2 AU, p = 0.027) and 48h post (4±2 AU, p = 0.034) compared to pre exercise (0±1 AU). These findings suggested reduced muscle glycogen stores did not impact sleep the night after exercise, neither did increased muscle damage on the next two nights. Thus, endurance athletes may be able to attain training adaptations using the 'train high, sleep low' paradigm without jeopardising subsequent nocturnal sleep.

Within Chapter 6, an 8-week case study of an International Taekwondo athlete was conducted to explore the effect of low energy availability on sleep leading into competition. A tailored training and nutritional intervention was designed to achieve a 9.5kg reduction in body mass so the athlete could make weight for competition in the Bantamweight category. Each week, energy availability was calculated and actigraphy was used to measure sleep. Additionally, the athletes' hydration status was monitored via urine osmolality, whilst their body composition was assessed using dual x-ray absorptiometry and the sum of skinfolds. There was no negative effect of low energy availability on nocturnal sleep based on the average sleep during -8 weeks to -4 and -3 weeks to the pre-cut stage (-2 before competition). Sleep duration and efficiency were within recommended values during these periods (>7h and 85% respectively [National Sleep Foundation]). However, during the days leading into competition (-5d), there was a noticeable reduction in the athletes' sleep duration, though this was likely a reflection of their internal behaviour (i.e. choosing to make bedtime later). This case study suggested low energy availability does not impact sleep duration or quality.

In conclusion, the four studies resulting from this thesis have provided further understanding of the effects of exercise training on subsequent nocturnal sleep within the athlete population. Based on this research, coaches can be more aware of typical situations that alter sleep. Subsequently, this information can be utilised in the organisation of athletes training regimes.

Dedication

I would like to dedicate this thesis to my late Granddad Ray, Grandma Joan and Uncle Ian. You will never know how much you have influenced me as a person - thank you all so much.

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Declaration

I declare the work contained within this PhD thesis is solely my own.

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List of Abbreviations

α-Lactalbumin = Alpha Lactalbumin
a-MT6 = 6-Sulphatoxymelatonin
AASM = American Academy of Sleep Medicine
ANOVA = Analysis of Variance
BCG = Ballistocardiography
BM = Body Mass
CHO = Carbohydrate
CN = Conditioning
COM = Competition
CON = No Exercise
CV = Cardiovascular
DXA = Dual Energy X-ray Absorptiometry
EA = Energy Availability
ECG = Electrocardiogram
EEG = Electroencephalogram
EMG = Electromyogram
EOG = Electrooculogram
ES = Effect size
EX = Exercise
HIGH = High Intensity Interval Running
HIIT = High Intensity Interval Training
HR = Heart Rate
HRV = Heart Rate Variability
IL-1β = Interleukin 1-Beta
IL-6 = Interleukin-6
IL-8 = Interleukin-8
IIV = Intra-Individual Variability
LEA = Low Energy Availability
LNAA = Large Neutral Amino Acid
LOW = Low Intensity Running
LSD = Least Significant Difference
MEQ = Morningness-Eveningness Questionnaire
MVC = Maximum Voluntary Contraction
NFOR = Non-Functional Overreaching
NREM = Non-Rapid Eye Movement Sleep
PRO = Protein
PSG = Polysomnography
PSQI = Pittsburgh Sleep Quality Index
REM = Rapid Eye Movement Sleep
RMR = Resting Metabolic Rate
RMR_{meas} = Resting Metabolic Rate Measurement
RPE = Rating of Perceived Exertion
SCN = Suprachiasmatic nucleus
SDRR = Standard Deviation of the R-to-R Interval
SPSS = Statistical Package for the Social Sciences
SWS = Slow Wave Sleep
TNF-α = Tumor Necrosis Factor-alpha
TTE = Time to exhaustion
WDEB = Within Daily Energy Balance
VO_2 = Oxygen Uptake
VO_{2peak} = Peak Oxygen Uptake

Chapter 1

General Introduction

1.1. Background

In elite sport, exercise training is necessary to optimise improvements in physical capabilities and enhance performance (Bishop, 2008). The training process involves the careful programming of exercise intensity, volume and frequency over several weeks or months to cause progressive overload (Smith, 2003). This principle is used to stress an athlete above that previously tolerated to provide a stimulus for adaptation and supercompensation (Steinacker and Lehmann, 2002). When this training stress is not balanced with adequate recovery, the acute fatigue from overload may manifest into a state of non-functional overreaching (NFOR) (Barnett, 2006; Kellmann, 2010). This is associated with long term reductions in performance capacity, which may take a number of weeks or months to restore and may be accompanied by physiological and psychological symptoms of maladaptation (Meeusen *et al*, 2012). Given the adaptation process is dependent upon avoiding NFOR, maximising the post exercise recovery of athletes is essential in training management.

The most efficacious recovery strategy for athletes is considered to be sleep (Halson, 2008). Sleep is partially controlled by the amount of sleep pressure accumulated during wakefulness (Borbely *et al*, 2016); as such, it is generally accepted the role of sleep must be to serve a recovery purpose and/or prepare the body for optimal functioning during the subsequent wake period (Halson, 2013). This view is supported by experiments that have used sleep restriction (Souissi *et al*, 2013; Jarraya *et al*, 2013; Reyner and Horne, 2013) and sleep deprivation (Axelsson *et al*, 2008; Oliver *et al*, 2009; Souissi *et* al, 2003) protocols to assess effects of sleep loss on exercise performance. These studies have demonstrated that aerobic, anaerobic, cognitive and sport specific performance is compromised following one or more nights of sleep loss. Such effects may result from the impairment of various physiological functions (i.e. growth hormone release)

associated with less time spent in deep sleep (Non-rapid eye movement [NREM] stage 3) and rapid eye movement (REM) sleep (Fullagar *et al*, 2015; Venter, 2012). To prevent disruption to recovery, it is therefore recommended athletes should increase their sleep duration following strenuous training (Davenne, 2009).

A number of studies have described the sleep patterns of various athlete populations during a training schedule. Compared to non-athlete groups, it has been shown that athletes from both individual and team sports have longer sleep onset latency and lower sleep efficiency (Bender *et al*, 2018; Demirel, 2016; Leeder *et al*, 2012; Whitworth-Turner *et al*, 2017), indicating that sleep quality may be reduced within athletes. Additionally, Leeder and colleagues (2012) reported that Olympic athletes slept markedly below the minimum 8h per night that is necessary to prevent neurobehavioural deficits (Belenky *et al*, 2003; Van Dongen *et al*, 2003). Reduced sleep durations have been documented amongst other athlete groups; for example, Fietze *et al* (2009) showed that sleep duration in ballet dancers was reduced when training for a dance premiere; resulting in a change from 6h 58min in the first week of training to 6h 32min in the final week. Lastella *et al* (2014) also showed that athletes from team (American Rules Football, Basketball, Rugby Union and Football) and individual (Cycling, Mountain Bike, Racewalking, Swimming and Triathlon) sports obtained 6.8h of sleep per night during pre-season. Whilst Caia *et al* (2017) found elite rugby league players slept for 6.9h per night during one pre-season week. Taken together, these findings may suggest that during training periods, sleep patterns within athletes are sub-optimal for recovery and that such factors impacting these sleep patterns warrant further understanding.

One potential factor that may influence sleep within athletes is training-induced stress. According to previous studies, increased training demands (either volume or intensity) are associated with reductions in both sleep duration and quality within athlete

populations (Fullagar *et al*, 2019; Hausswirth *et al*, 2014; Schaal *et al*, 2015; Teng *et al*, 2011; Thornton *et al*, 2017). This may be a result of increased sympathetic activity, muscle soreness, cortisol and/or depleted energy reserves (MacKinnon, 2000; Roberts *et al*, 2018). In contrast, other research has suggested that sleep is unaffected (Caia *et al*, 2017; Knufinke *et al*, 2018; Louis *et al*, 2016) or may even improve with increased training demands (Brand *et al*, 2010a; Taylor *et al*, 1997; Thornton *et al*, 2018; Whitworth-Turner *et al*, 2018). Clearly, at present the sleep response to strenuous exercise training within athletes is unclear. With this in mind, there is a rationale to conduct more investigations that examine the potential effect of exercise training on subsequent nocturnal sleep within athlete groups. This could assist athletes and sports practitioners, such as coaches and performance staff, to distribute and plan training sessions in a way that subsequently benefits nocturnal sleep. Consequently, this may better inform the balance between training stress and recovery, so that adaptation and supercompensation can be facilitated within athlete populations.

1.2. Aims and Objectives

The aim of this thesis is to investigate the effect of exercise training on nocturnal sleep within athletic populations. This will be achieved by completion of the following objectives:

1) Compare sleep of athletes with non-athletes during an in-season week (**Study 1**).
2) Examine the effect of evening exercise training at different intensities on sleep and cardiac autonomic activity within a trained population (**Study 2**).
3) Examine the effect of reduced muscle glycogen stores induced by exhaustive exercise in the evening on sleep within a trained population (**Study 3**).
4) Describe the effect of low energy availability on sleep: a case study of an international Taekwondo athlete making weight for competition (**Study 4**).

Chapter 2

Literature Review

2.1. Sleep

2.1.1. Sleep Structure

Human sleep comprises of NREM sleep and REM sleep (Carskadon and Dement, 2011), and these are defined by the recording of electroencephalography (EEG [brain wave activity]), electrooculography (EOG [eye movement]) and submental electromyography (EMG [chin movement]) (Berry, 2012). NREM is usually observed upon sleep initiation and has three stages differing in depth; stage one has the lowest arousal threshold and stage three has the highest (Carskadon and Dement, 2011). Accordingly, stages one and two are referred to as light sleep and stage three as deep sleep. Within 80-100 min after the onset of NREM sleep, REM commences and is primarily defined by periodic bursts of rapid eye movement (Carskadon and Dement, 2011; Venter, 2012). Other physiological responses include an increase in body temperature, heart rate, blood pressure, blood flow and respiration rate, whilst chin muscle activity is at its lowest during the whole night (Venter, 2012). As such, it is the EOG and EMG recordings that commonly characterise REM sleep, whereas NREM sleep stages are identified through information from the EEG recording (Table 2.1).

Both NREM and REM constitute one sleep cycle, and this lasts approximately ≥90 min (Venter, 2012). In adults, there may be four to six sleep cycles per night (Venter, 2012). NREM is the most dominant type of sleep, accounting for between 75-80% of the cycle with the remaining 20-25% being from REM sleep (Carskadon and Dement, 2011; Venter, 2012). REM does become more prominent as sleep progresses, with the longest episodes occurring in the second half of the night (Carskadon and Dement, 2011; Venter, 2012).

Table 2.1. Characteristics of NREM and REM sleep from EEG, EOG and EMG recordings adapted from Berry (2012).

Type of sleep		Characteristics
Non-rapid eye movement	Stage 1	*Vertex sharp waves* - Sharp contoured waves lasting <0.5 s *Alpha rhythm* – Wave frequency in the range between 8-13 Hz** *Slow eye movements* – Conjugate, regular, sinusoidal eye movements with an initial deflection lasting >500 msec
	Stage 2	*Sleep spindles* – Burst of sharp waves lasting between 0.5-1.5 s* *K complex* – A negative sharp wave (deflection up), followed by a positive sharp wave (deflection down) lasting ≥0.5 s*
	Stage 3	*Slow waves* - Waves with a frequency range of 0.5 to 2 Hz (0.5 – 2 s) and a peak to peak amplitude greater than 75μV
Rapid eye movement		*Saw tooth waves* – Triangular waves of 2 to 6 Hz *Rapid eye movements* – Conjugate, irregular, sharply peaked eye movements with an initial deflection lasting <500 msec *Low EMG tone* – Baseline activity of the chin is no higher than in any other sleep stage and usually at the lowest level of the night recording

* indicates that the characteristic may also be present during stage three of NREM.

** indicates that the characteristic may also be present during REM.

2.1.2. Sleep Regulation

There are two processes that are thought to interact to determine the timing, duration and intensity of sleep (Figure 2.1) (Borbely *et al*, 2016). One is the circadian pacemaker located within the suprachiasmatic nucleus (SCN) of the hypothalamus, which synchronises the internal clock according to information received from environmental cues (Beersma, 1998; Beersma and Gordijn, 2007). Daylight and darkness are the main *zeitgebers* or time givers of the SCN and are relayed via the retinal ganglion cells and retinohypothalamic tract (Schwartz and Roth, 2008; Borbely *et al*, 2016). The circadian rhythm of sleep is delayed in the presence of light but advanced in darkness, which is the usual sleep period for humans (Crowley *et al*, 2007). This is coincided by the secretion of melatonin during night-time (Khalsa *et al*, 2003), which precedes the decrease in body temperature that facilitates sleep propensity (Claustrat *et al*, 2005). Non-photic stimuli such as exercise and meal timing may also act as *zeitgebers* for the SCN and could alter circadian timing for sleep propensity (Mistlberger and Skene, 2005). For example, evening high intensity exercise resulted in a phase advance of melatonin onset, whereas morning, afternoon, nocturnal and no exercise caused phase delays (Buxton *et al*, 2003). Exposure to such *zeitgebers* may explain interindividual differences in sleep between individuals.

Large differences may exist in the circadian timing of sleep between individuals, which may affect performance, health and well-being (Adan *et al*, 2012). Such differences are due to the chronotype preference of the master internal clock controlling the sleep-wake cycle i.e. melatonin and body temperature profiles (Adan *et al*, 2012). Individuals may be a morning chronotype (lark), that is, they tend to wake up and perform activities in the early morning, an evening chronotype (owl), where they wake up later in the morning, or an intermediate chronotype, which is neither a morning or evening chronotype (Adan *et*

al, 2012). Based on constant routine, there can be a one-hour phase advance or delay in temperature than average, for morning and evening types respectively (Kerkhof and Van Dongen, 1996). Waterhouse *et al* (2012) theorised those whose lifestyle predisposes them to live outside of their chronotype preference (i.e. evening types waking earlier) are likely to accrue problems. As such, studies within the current thesis, the participants chronotype will be identified using the morningness-eveningness questionnaires (Horne and Ostberg, 1976; Smith *et al*, 1989).

A second mechanism by which sleep is regulated is suggested to be the sleep homeostat, also referred to as the *hourglass oscillator* (Borbely *et al*, 2016; Dijk and von Schantz, 2005). This infers that during wakefulness there is a rise in sleep propensity due to increased neuronal activity before it is then dissipated during subsequent sleep (Borbely *et al*, 2016). Specifically, it is slow wave sleep (SWS) that is prioritised following prolonged wakefulness (Djik *et al*, 1987) and this is the principle marker of sleep homeostasis (Borbely *et al*, 2016). Several molecules may moderate the rebound in SWS, though adenosine accumulation is currently accepted as the most important molecule of sleep homeostasis (Porrka-Heiskanen, 2013). Experimentally, preventing ATP synthesis, thus causing energy depletion in the basal forebrain resulted in greater extracellular adenosine and NREM sleep in rats (Kalinchuk *et al*, 2003). Blocking the A1 adenosine receptor via caffeine administration also caused an increase in wakefulness, followed by a prolonged increase in SWS, also in rats (Schwierin *et al*, 1996).

Figure 2.1. The two-way process of sleep regulation. Red line = circadian process and the blue line = homeostatic process. Melatonin (sleep regulating hormone) is suppressed from light exposure leading to increased wakefulness and rises in response to darkness causing the body to sleep (Circadian). Sleep pressure is highest during the day and lowest at night when dissipated due to sleep (Homeostasis). Both processes independently regulate sleep, but propensity is at maximum when sleep pressure and melatonin levels are highest (i.e. 23:00h). Figure is from https://www.physoc.org/magazine-articles/what-makes-a-good-nights-sleep/

2.1.3. Sleep Functions

Sleep is thought to serve three important bodily functions relating to physiological repair and growth, neuro-metabolic conservation and cognitive processing (Frank, 2006). These functions are carried out by NREM and REM sleep, and these sleep stages have differing biological actions, both physically and neurologically (Assesfa *et al*, 2015; Davenne, 2009). Although the true sleep function is not fully understood (Halson, 2008; Samuels, 2008), there is evidence that nocturnal sleep is responsible for recovery from wakefulness. It is well-known that metabolic, endocrine, immune and cognitive functioning is negatively affected after sleep restriction and forced desynchrony of the sleep cycle (Samuels, 2008). The amount of time spent asleep is also proportional to the time spent awake, a phenomenon that is tightly regulated by the sleep homeostat or *hourglass oscillator* (Borbely *et al*, 2016). These physiological events would suggest that adequate sleep is important for elite athletes involved in heavy training schedules and competition, as well as critical for optimal performance.

The main argument for physiological repair and growth is the change in hormone secretion that occurs in the transition from wakefulness to sleep (Leproult and Van Cauter, 2010). Shortly after sleep initiation, there is an increase in growth hormone and a reduction in cortisol, which are important for anabolism and catabolism respectively (Leproult and Van Cauter, 2010; Weitzman *et al*, 1974). The increase in growth hormone stimulates protein synthesis for body restitution and has effects on muscle growth and repair that are necessary for elite athletes (Davenne, 2009). Most of the growth hormone is released during SWS of NREM with a much smaller pulse secreted during REM sleep (Takahashi *et al*, 1968; Van Cauter *et al*, 1992). Importantly, in the absence of SWS through sleep restriction, nocturnal growth hormone release can be substantially reduced (Sassin *et al*, 1969; Brandenberger and Weibel, 2004). Furthermore, there is a greater secretion of growth hormone prior to and following sleep initiation in sleep deprived individuals, suggesting a role of sleep (Davidson *et al*, 1991; Spiegel *et al*, 2000). Athletes who have reduced sleep duration may, therefore, not attain the physiological benefits from growth hormone, which may affect their recovery.

Neuro-metabolic conservation is the idea that sleep has a restorative effect on the brain and central nervous system, whereby wake incurs a neural metabolic cost, which nocturnal sleep can recover via specific actions (Frank, 2006). One possibility is sleeping eliminates neural toxins (proteins) from the brain, so the brain then has a 'clear slate to work from' after an individual has slept (Eugene and Masiak, 2015). Xie *et al* (2013) showed sleep improved β-amyloid clearance from the interstitial fluid of mice compared to wake, suggesting sleep can remove waste products formed during wake. Sleep may also help to restore the brain and central nervous system through increasing the storage of brain glycogen, a reserve glucose source for neurons (Benington and Heller, 1995). A strength of this proposal is that brain glycogen is increased during NREM sleep but is then reduced within a few minutes of wakefulness in the rat (Karnovsky *et al*, 1983).

There is also evidence adenosine is a signalling molecule for sleep homeostasis, which would support the need to restore glycogen as adenosine is formed during energy breakdown (Porrka-Heiskanen, 2013). Hence, sleep would seem important for brain restoration in elite athletes.

Another theory, cognitive processing, refers to the potential benefits of sleep on behavioural tasks such as learning and memory. During sleep, certain brain activities are predicted to restore optimal neuronal function from previous wakefulness (Marquet, 2001). Depending on the brain region, NREM and REM both contribute to a reduction in the synaptic activity from wake, and re-normalise synaptic strength (Tononi and Cirelli, 2014). Consequently, sleep is suggested to reduce the burden of plasticity on neurons and other cells, while restoring the ability to learn and consolidate memories (Tononi and Cirelli, 2014). In line with this hypothesis, motor sequence learning is suggested to be poorer after sleep deprivation and even after recovery sleep, normal improvements are prevented (Walker and Stickgold, 2005). Similarly, neurocognitive and academic performance is worsened following sleep restriction, whereas these parameters are improved with normal sleep (Curcio *et al*, 2006). These cognitive benefits from sleep may be pertinent for athletes after training for skill acquisition or tactical situations beyond that of physiological responses alone.

2.2. Assessment of Sleep

2.2.1. Polysomnography

Polysomnography (PSG) is a term used to describe the measurement of EEG, EOG and submental EMG. The combination of these measures provides detailed information of sleep stages and for this reason, PSG is considered the gold standard measure of sleep (Sadeh, 2015). Originally, sleep was staged using standardised criteria developed by Rechtschaffen and Kales (1968). In this manual, NREM consisted of four sleep stages, though sleep stage nomenclature has since changed to combine stages three and four following the publication of the American Academy of Sleep Medicine (AASM) manual for scoring of sleep and associated events (Iber *et al*, 2007). The process of manual scoring is rigorous, consisting of at least two experienced scorers that work together to agree on all aspects of the sleep nomenclature (Penzel and Conradt, 2000). According to the AASM guidelines at least 3 EEG, 2 unipolar EOG and 3 EMG channels are required to accurately stage sleep (Iber, 2007). The three EEG channels are considered the core physiological signals of the set up; these relate to the frontal, central and occipital regions of the head and are referenced against the mastoid process (Berry, 2012; as shown in figure 2.2). The implementation of PSG is somewhat more difficult as it requires a laboratory-controlled environment (Cellini *et al*, 2014) and specialist staff (Shrambroom *et al*, 2012; Kosmadopoulos *et al*, 2014). Furthermore, PSG monitoring can take individuals more than one night to adjust (Agnew *et al*, 1966), therefore, the use of several familiarisation nights may be needed before habitual sleep patterns are captured. These limitations mean PSG is not feasible to assess sleep in large cohorts such as athletes in a longitudinal manner.

The image originally presented here cannot be made freely available via LJMU E-Theses Collection because of copyright. The image was sourced at:
https://trustedacademy.com/courses/eeg/skull-landmarks-10-20-system/

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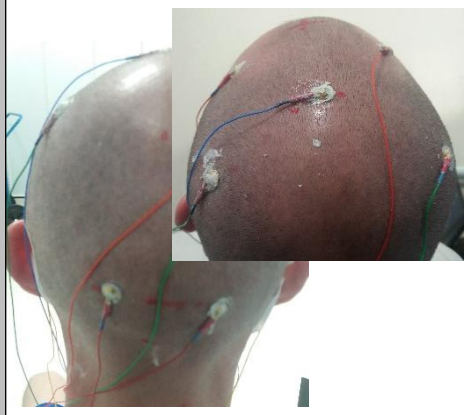

Figure 2.2. EEG channels. F3 = frontal; C3 = central and O1 = occipital a). All channels are referenced against the mastoid process (located behind the ear) as shown by the black arrow. The standard international 10-20 system is used to determine the sites of the EEG channels; the measurement begins from the nasion (front of the head) and the inion (back of the head). Figure is from https://trustedacademy.com/courses/eeg/skull-landmarks-10-20-system/. Example of the EEG electrode placement for the frontal, central and occipital channels b).

2.2.2. Wristwatch Actigraphy

Use of wristwatch actigraphy has become the established gold standard measure of sleep within field settings. Actigraphy offers multiple benefits over PSG in that it is non-intrusive, cost effective and does not require specialist staff or a laboratory-controlled environment (Sadeh, 2011). Actigraphy devices are typically worn on the non-dominant wrist, though the dominant wrist can also be used (Driller *et al*, 2017). They record body movement along three axes via a built-in accelerometer, usually in one-minute epochs, and store this information within a memory chip (Ancoli-Israel *et al*, 2003). The data are downloaded to a computer, which displays periods of activity and inactivity; algorithms then estimate sleep/wake behaviour based on the premise there is less movement during sleep and more movement during wake (Sadeh, 2011). Such devices can be used to measure various sleep parameters such as; total sleep time, sleep onset latency, wake after sleep onset and sleep efficiency (Sadeh, 2011).

In comparison to PSG, actigraphy is deemed to have an acceptable level of agreement and thus, is considered a valid alternative (De Souza *et al*, 2003; Dunican *et al*, 2018; Jean-Louis *et al*, 2001; Pacquet *et al*, 2007; Rupp and Balkin *et al*, 2011). The error that is associated with wristwatch actigraphy is due to low specificity i.e. the ability to identify wake periods (Table 2.2). This is an inherent limitation, as actigraphy is unable to differentiate when an individual is awake and immobile (Tryon, 2004). An important issue is choosing the correct sleep-wake threshold for the population studied as this may influence the accuracy of actigraphy indices. Tonetti *et al* (2008) showed a low threshold (above 20 activity counts scored as wake) produced the best agreement with PSG for total sleep time, sleep efficiency and wake after sleep onset in healthy adults. In contrast, high (above 80 activity counts scored as wake) or medium threshold (above 40 activity counts scored as wake) settings are recommended in athletes (Fuller *et al*, 2017; Sargent *et al*, 2016). This discrepancy may be explained by athletes' tendency to move more during their sleep (Leeder *et al*, 2012), thus requiring a greater activity count.

Perhaps the biggest concern with wristwatch actigraphy is the algorithms used to calculate sleep onset latency. In a commentary, Tryon (2004) argued because sleep onset is indicated by inactivity using actigraphy rather than a change in brain wave frequency, such devices underestimate sleep onset latency and may subsequently overestimate total sleep time and sleep efficiency. The AASM has since recommended a sleep diary be used alongside actigraphy to obtain estimates of lights out and sleep onset, to optimise these algorithms (Morgenthaler *et al*, 2007). A diary designed for research purposes is the Consensus Sleep Diary (Carney *et al*, 2012), which was formed as a result of a collaboration between insomnia experts and potential users. The Consensus Sleep Diary asks a number of questions relating to lights out, sleep onset latency, number of awakenings, time of final awakening and perceived sleep quality, and therefore, may be suitable to implement in studies using wristwatch actigraphy.

Table 2.2. Overview of studies comparing sensitivity and specificity of actigraphy with PSG. Significant difference (P <0.05) compared with PSG is indicated by an asterisk *.

Reference	Actigraphy model	Sleep epochs identified (sensitivity)	Wake epochs identified (specificity)	Epoch by epoch Accuracy	Sleep parameters
De Souza *et al* (2003) N = 21 healthy adults	Mini motionlogger actrigraph 32C	Cole algorithm = 99% Sadeh algorithm = 97%	Cole algorithm = 34% Sadeh algorithm = 44%	Cole algorithm = 91% Sadeh algorithm = 91%	Cole algorithm SOL (min) = +1.4 TST (min) = +18.5 IA (min) = -18.6 SE (%) = +4.2 Sadeh algorithm SOL (min) = +2.5 TST (min) = +8.1 IA (min) = -10 SE (%) = +2.2
Paquet *et al* (2007) N = 15 Healthy adults	Actiwatch-L	95.3%	54.3%	90.7%	SOL (min) = -13.9* TST (min) = +3.6 NA (#) = +7.3* SE (%) = +0.7
Rupp and Balkin (2011) N = 29 Healthy adults	Actiwatch 64	92.2%	57.6%	89.6%	SOL (min) = -6.85* TST (min) = -20.35* NA (#) = +25.9* SE (%) = -4.41*
	Motionlogger watch	96.2%	63.6%	93.6%	SOL (min) = +1.1 TST (min) = -4.7 NA (#) = -3.5 SE (%) = -1.2

Abbreviations: TST = Total sleep time; SE = sleep efficiency; SOL = Sleep onset latency; NA= Number of awakenings; IA = Intermittent awakenings. Underestimation of wristwatch actigraphy (-); Overestimation of wristwatch actigraphy (+).

2.2.3. Partial PSG

Another device designed to monitor sleep in the field is partial-PSG. As the name suggests, partial-PSG is a smaller and simplified version of PSG and uses EEG signals to calculate sleep and wake (Shambroom *et al*, 2012; Kosmadopoulos *et al*, 2014). One example of partial-PSG is the ZEO automated wireless monitoring system (Newton, MA, USA). This system utilises three electrodes embedded within an elastic headband to collect a single channel EEG from the frontalis muscle (forehead) at sites Fp1 and Fp2 (Shambroom *et al*, 2012). It functions by using an ultra-low-power proprietary wireless protocol to communicate brain wave frequency and amplitude (extracted using a signal processing technique [Fast Fourier Transform]) to a base station placed at the bedside, which is able to calculate sleep stages in real time via an artificial neural network algorithm (Shambroom *et al*, 2012).

The wireless monitoring system has previously been validated against PSG during nocturnal sleep (Griessenberger *et al*, 2013; Tonetti *et al*, 2013) and when napping (Cellini *et al*, 2014). Collectively, these studies showed that the wireless monitoring system had high sensitivity, but low specificity compared to PSG. Tonetti and colleagues (2013) attributed the low specificity to there being no electrodes at the back of the headband, where most alpha activity (present when awake) is recorded. Despite this, it has been observed that specificity and the epoch-to-epoch agreement of the wireless monitoring system is higher than that of wristwatch actigraphy (Kosmadopoulos *et al*, 2014; Shambroom *et al*, 2012). An obvious difference is that the headband uses EEG to differentiate between sleep and wake, whereas actigraphy devices use body movement (Shambroom *et al*, 2012). This may suggest that the wireless monitoring system is a potential substitute for actigraphy when assessing sleep in the field.

2.2.4. Ballistocardiography

The technique known as Ballistocardiography (BCG) uses heart rate and breathing rate as well as body movement to calculate sleep and wake. Measures of heart rate and respiration have shown to differentiate between sleep and wake states (Hudgel *et al*, 1984; Panzel *et al*, 2003; Scholz *et al*, 1997; Versace *et al*, 2003). Such physiological measures are detected by a mechanical bed sensor placed either under the pillow, under the mattress or on a bedpost (Brink *et al*, 2006; Cha *et al*, 2008; Paalasmaa *et al*, 2011; Watanabe and Watanabe, 2004). The signal received from the sensor is sent to a Smartphone or web application via a bluetooth connection where an algorithm scores sleep indices (Watanabe and Watanabe, 2004). BCG has been validated against ECG and pulse oximetry for the monitoring of heart rate and breathing rate respectively (Ben Ari *et al*, 2010; Paalasmaa *et al*, 2012).

Among the different types of bed sensors developed, the mattress bed sensor has received the most attention regarding sleep-wake detection. These sensors have shown a high degree of accuracy compared to PSG for the calculation of sleep and wake (81.9-88.5 %) (Guerrero-Mora *et al*, 2012; Tal *et al*, 2017). The mattress bed sensor has also shown promising results against wristwatch actigraphy. Mack *et al* (2009) evaluated the Non-Invasive Analysis of Physiological Signals (NAPS) system and wristwatch actigraphy in a group of healthy sleepers and apneic sleepers. They found the NAPS system had a better agreement with PSG than actigraphy for sleep-wake detection ($k = 0.48$ v 0.34) and was superior in calculating measures of sleep onset time, total sleep time and sleep efficiency. Nonetheless, more validation studies in other populations are warranted before its use in research settings.

2.3. Sleeping Patterns of Athletes

Athletes are encouraged to preserve adequate sleep duration during high training load periods, mainly due to the physiological restoration that occurs when sleeping (Davenne, 2009; Halson, 2013). The National Sleep Foundation recommend adults should obtain at least 7h of sleep per night with a minimum 85% of sleep efficiency (time asleep as a percentage of time in bed) to maintain health and well-being (Hirshkowitz *et al*, 2015; Ohayon *et al*, 2017). Alarmingly, subjective (Bender *et al*, 2018; Demirel, 2016) and objective measures (Leeder *et al*, 2012; Whitworth-Turner *et al*, 2017) of sleep have revealed that athletes sleep efficiency may be lower than that of non-athletes. Leeder and colleagues (2012) studied the sleep of Olympic athletes, via wristwatch actigraphy, during a typical non-competition training phase. Compared to non-athletes, athletes had comparable sleep duration (6:55 vs. 7:11 hh:mm) but a lower sleep efficiency (80.6 vs. 88.7 %), suggesting their sleep quality was lower. Studies have also shown similar sleep efficiency scores in other athlete groups during the competitive training phase (Fietze *et al*, 2009; Shearer *et al*, 2015; Sargent and Roach, 2016; Walsh *et al*, 2019). These findings indicate that athletes may adopt poor sleeping patterns during those periods where the training demand is higher, which may affect their subsequent recovery. That said, there is no study to have objectively compared the sleep patterns of elite athletes, from a range of different sports, to those of non-athletes during a competition period. This information is warranted to understand the sleep differences between these populations during high training load periods; thus, a comparison of athletes and non-athletes sleep will be made in study one of this thesis.

2.4. Effect of Sleep on Sports Performance

2.4.1. Sleep Restriction

From the aforementioned studies, it is likely athletes experience sleep restriction from either falling asleep later or waking earlier, which partially disrupts their sleep cycle (Boonstra *et al*, 2007). Depending on the type of sport, such sleep restriction may result in decrements to performance (Table 2.3) (Fullagar *et al*, 2015). Maximal strength and/or power were impaired after 4h of sleep restriction for one night in judokas (Souissi *et al*, 2013) and footballers (Abedelmalek *et al*, 2013). Specifically, these reductions in performance were observed in the afternoon but not the morning, which may reflect the diurnal variation in short term maximal exercise (Drust *et al*, 2005). Peak power, mean power and peak velocity from the Wingate test were not affected in the morning after a period of similar sleep restriction in trained athletes (Mougin *et al*, 1996), though this study did not assess performance in the afternoon. Submaximal strength was also more affected than maximal strength in healthy individuals following 3h of sleep restriction for three consecutive nights (Reilly and Piercy, 1994). Although maximal strength and power may be more determinant of sporting success (i.e. weightlifting), submaximal strength loss may still affect sports with repeated bouts of activity.

Data from studies assessing specific skills have also shown that sleep restriction may affect darts throwing accuracy (Edwards and Waterhouse, 2009), goalkeeping performance (Jarraya *et al*, 2013), serving accuracy (Reyner and Horne, 2013) and driving ability (Otmani *et* al, 2005: Philip *et al*, 2003). This may be explained by the impairment of psychomotor vigilance i.e. decreased alertness (Edwards and Waterhouse, 2009; Jarraya *et al*, 2012) and increased reaction time (Jarraya *et al*, 2012; Philip *et al*, 2003). In contrast, 6h of sleep restriction over one night had no effect on swim lap times in competitive swimmers compared to normal sleep (Sinnerton and Reilly, 1992), despite a

reduced mood state profile (i.e. increased depression, tension, confusion, fatigue and anger, and decreased vigour). There was also no change in grip and back strength, heart rate or lung function within this study. Collectively, these findings indicate gross motor skills such as stroke rate (Sinnerton and Reilly, 1992) may not be affected by sleep loss in the same way as tasks reliant on fine motor skill execution (Edwards and Waterhouse, 2007; Jarraya *et al*, 2013; Philip *et al*, 2003; Reyner and Horne, 2013).

Sleep restriction may impact sustained exercise performance through delaying recovery from a prior bout of exercise, which is representative of real-world situations (i.e. Tour de France). Morning time trial performance was impaired after 4-5h of sleep restriction for one night, that folllowed an evening time trial effort, in eight recreational level cyclists (Chase *et al*, 2017). Time trial performance was also slower on day 3 of four days of 2-3h sleep restriction compared to normal sleep, in endurance-trained cyclists and triathletes (Roberts *et al*, 2019). This performance loss may be attributed to mood state or perceived exertion (Roberts *et al*, 2019), as cardio-respiratory indices do not tend to change (Chase *et al*, 2017; Mejri *et al*, 2014). However, sustained exercise performance was not affected after one night of sleep restriction without prior exercise (Mejri *et al*, 2014; Mougin *et al*, 1991; Reilly and Deykin, 1983), though these studies were limited by either sample size, type of participant, or the exercise test used. In this respect, endurance-type performance may be compromised following sleep restriction, but it is unclear whether this occurs after one or several nights, which is important considering these types of athletes compete on a day-to-day basis (i.e. Tour de France road race).

2.4.2. Sleep Extension

Given the detrimental effects of sleep loss on sports performance, there has been interest in extending the habitual sleep duration of athletes (Arnal *et al*, 2016; Mah *et al*, 2011; Ritland *et al*, 2019; Roberts *et al*, 2019; Schwartz and Simon Jr, 2015). These studies have indicated that 1-2h of extra sleep per night may improve parameters related to physical and cognitive performance. In the first of these studies, Mah *et al* (2011) investigated the effect of a 5-7 week sleep extension period on athletic performance in university basketball players. Following sleep extension (+110.9±79.7min), athletes had improved sprint times (15.5 vs. 16.2s), free throw performance (+9%) and three-point field goal performance (+9.2%), as well as improved reaction time (310.8 vs. 274.5 ms) compared to baseline. Ritland *et al* (2019) studied the effects of four nights of sleep extension on cognitive and motor performance in tactical athletes enrolled in Reserve Officers Training. In comparison to the control group, sleep extension (+1.36±0.71h) improved immediate reaction time (-16.1 vs. 1.75 ms), trail making test performance (-11.8 vs. -7.1 s) and standing broad jump performance (9.07 vs. -2.31 cm). Moreover, after returning to their habitual sleep for four consecutive nights, the improvement in standing broad jump performance was maintained within the sleep extension group.

Some athletes may have difficulty extending nocturnal sleep duration due to the constraints of their sport; i.e. travel schedules may reduce the amount of time in bed (Mah *et al*, 2011). As a countermeasure, athletes are encouraged to nap during the daytime (Mah *et al*, 2011), preferably post-lunch as it aligns with the dip in circadian alertness (Waterhouse *et al*, 2007). There is currently no clear consensus about the optimal nap duration, but studies have shown that physical and cognitive performance is improved after a ≥20 min nap (Blanchfield *et al*, 2018; Daaloul *et al*, 2019; O'Donnell *et al*, 2018; Waterhouse *et al*, 2007). Interestingly, Blanchfield and colleagues (2018) suggested that

endurance performance was improved to a greater extent after a 20 min nap in individuals that slept less the night before. In this study, eleven trained male runners completed a 30 min morning run (75% VO_{2max}), which was followed by a nap or no nap before a time to exhaustion run (TTE) in the evening. The results showed that despite there being no differences in the TTE between conditions, those individuals who improved their TTE with napping, obtained less sleep the night before. On this basis, athletes should consider using daytime naps to improve performance, especially if they have not achieved an adequate amount of sleep during the nocturnal period.

Table 2.3. Effect of sleep restriction on different exercise performance parameters.

Reference	Participants	Protocol	Performance test	Outcome
Endurance performance				
Chase *et al* (2017)	8 recreational cyclists	4-5h SR for one night	3-km time trial performance	↓ in time trial performance
Mejri *et al* (2014)	10 national Taekwondo athletes	3-4h SR for one night	Yo-Yo intermittent recovery test level 1	No change in distance covered
Mougin *et al* (1991)	7 cyclists	3h SR for one night	20min steady state and incremental test to exhaustion	No change in maximal intensity sustained
Reilly and Deykin (1983)	8 trained participants	2.5h SR for three nights	Incremental treadmill test to exhaustion	No change in time to exhaustion
Roberts *et al* (2019)	9 endurance trained cyclists/triathletes	2-3h SR for 4 nights	Time trial performance	↓ in time trial performance on day three
Strength/power performance				

Abedelmalek *et al* (2013)	12 footballers	4h SR for one night	Wingate anaerobic test	↓ in peak and mean power
Haj-Salem *et al* (2013)	21 judokas	4h SR for one night	Muscle strength and power tests before a judo match	No change in peak power, mean power and handgrip strength
Mougin *et al* (1996)	8 trained participants	4h SR for one night	Wingate anaerobic test	No change in peak power, mean power and peak velocity
Reilly and Piercy (1994)	8 healthy participants	3h SR for three nights	Maximal and submaximal weightlifting	↓ in submaximal strength for biceps curl, leg press and dead lift.
Souissi *et al* (2013)	12 Judokas	3h SR for two nights	Muscle strength and power tests before a judo match	↓ in handgrip strength, MVC of elbow flexors and mean power
Specific skill performance				

Edwards and Waterhouse (2007)	60 darts players	3-4h SR for one night	Dart performance	↓ in mean score and ↑ in number of zeros and variability of score
Jarraya *et al* (2012)	12 handball goalkeepers	4-5h SR for one night	Attention tests	↑ in reaction time and ↓ in attention
Otmani *et al* (2005)	20 healthy individuals	4h SR for one night	Driving performance measures	↓ in alertness
Philip *et al* (2003)	10 healthy individuals	2h SR for one night	Driving performance measures	↑ in reaction time
Reyner and Horne (2013)	16 tennis players	5h SR for one night	Tennis serving drills	↓ in serving accuracy
Sinnerton and Reilly (1992)	8 swimmers	2.5h SR for four nights	Swimming performance tests (50m and 400m lap times)	No change in lap times

2.5. Effect of training load and competition on sleep

Researchers have been interested in using exercise training as a model to improve sleep. Since energy expenditure from the waking period increases SWS and sleep duration, it is suggested exercise should have beneficial effects (Driver and Taylor, 2000; Horne, 1981). Whilst this is likely true for non-athletic populations, data from studies involving athletes are not convincing, suggesting there may be a certain threshold above which the stress from exercise training and competition is non-beneficial (Nedelec *et al*, 2018; Roberts *et al*, 2018).

2.5.1. Training Load

There is evidence that increased training load may disrupt sleep in athletes, although many of these studies have used observational, rather than randomised, crossover designs (Table 2.4). Teng *et al* (2011) monitored the sleep of elite cyclists via actigraphy during a camp consisting of 1-week normal training, 3-weeks high intensity training and 2-weeks recovery training. Across the high training phase, there was a reduction in total sleep time and sleep efficiency, and an increase in mean activity when compared to the normal training phase. Fullagar *et al* (2019) also showed a reduction in total sleep time during both camp (-41 min) and in-season (-56 min) relative to the off-season in American Collegiate Football players, suggesting that intensified training periods may have caused the alteration in sleep duration. Sleep loss may be due to the resultant increase in pain perception, exercise-induced muscle damage, cortisol or sympathetic activity (Chennaoui *et al*, 2015; Roberts *et al*, 2018). However, there is some support that sleep in elite athletes is unaffected (Caia *et al*, 2017; Knufinke *et al*, 2018) or may even be improved (Brand *et al*, 2010a; Taylor *et al*, 1997; Thornton *et al*, 2018; Whitworth-Turner *et al*, 2018) in the face of greater training demands. Taylor *et al* (1997) showed that SWS was significantly increased following both moderate and high intensity aerobic training, but not the taper

period in National female swimmers. Thornton *et al* (2018) also found specific demands (i.e. increased acceleration and deceleration loads) led to greater total sleep time and efficiency in Rugby League players. This may indicate there is a greater requirement for sleep in relation to high training loads.

Other studies have investigated specific scenarios experienced by athletes during periods of increased training load (Dunican *et al*, 2019; Hausswirth *et al*, 2014; Louis *et al*, 2016; Schaal *et al*, 2015). Hausswirth *et al* (2014) examined the sleep patterns of eighteen trained triathletes who were exposed to overload training, where the aim was to cause functional overreaching. During three-weeks of training, nine athletes were diagnosed as overreached based on their declines in performance and VO_{2max}, concomitant with increases in perceptual fatigue. Compared to one-week of normal training, these overreached athletes experienced reductions in sleep duration (6:36 vs. 7:09 hh:mm), sleep efficiency (88.4 vs. 90.0 %) and immobile time (387 vs. 417 min), as well as an increase in upper respiratory tract infections. In athletes not considered overreached, there were no sleep changes between normal and overload training, indicating fatigability rather than training volume alone may cause sleep disruption. Additionally, Louis and colleagues (2016) investigated whether low glycogen availability caused through dietary periodisation may affect sleep, also in a group of trained triathletes. The triathletes were assigned to a control or a sleep low group, where they performed the same training for three weeks but with different nutrition guidelines (i.e. high or low CHO), so that only the sleep low group would initiate sleep with reduced glycogen availability. Despite a small reduction in sleep efficiency (-0.9 %) in the sleep low group, there was no change in total sleep time, sleep onset latency and perceived sleep quality, suggesting that sleeping with lower glycogen availability had minimal effect on sleep. Still, due to factors such as; participants studied, sleep measurement, research design employed, and length

of studies performed, the effect of training load on sleep requires further investigation to further the understanding of this relationship in athlete populations.

Table 2.4. Effect of increased training load on nocturnal sleep in athlete populations.

Reference	Participants	Study design	Training overload	Outcome
Brand *et al* (2010a)	12 adolescent football players	Cross-sectional-observational	14h of vigorous exercise per week	↑ SE and SWS; ↓ SOL and NA
Caia *et al* (2017)	7 Rugby League players	Observational	Increase in intensity for 1WK	No change
Dunican *et al* (2019)	21 Combat sport athletes	Randomised control	Water loading whilst training	No change
Fullagar *et al* (2019)	23 American Collegiate Football players	Observational-longitudinal	Increase in intensity for 4WK during camp and in-season	↓ in TST
Hausswirth *et al* (2014)	21 trained cyclists	Randomised control	Increase in volume for 3WK	↓ in TST, SE and IT
Jurimae *et al* (2004)	21 competitive rowers	Cross-sectional-observational	Increase in volume for 6D	↓ in PSQ
Killer *et al* (2017)	13 highly-trained cyclists	Randomised-counterbalanced	Increase in intensity for 9D	↓ in SE
Knufinke *et al* (2018)	98 from team and individual sports	Mixed methods	Day-to-day variation of perceived training load	No change

Kolling *et al* (2016)	55 National junior rowers	Cross-sectional-observational	Increase in volume and intensity for 2WK	↓ in TST
Louis *et al* (2016)	21 trained triathletes	Randomised control	Increase in intensity for 3WK	↓ in SE
Schaal *et al* (2015)	10 elite synchronised swimmers	Randomised crossover	Increase in intensity for 2WK	↓ in TST and SE; ↑ SOL
Taylor *et al* (1997)	7 National swimmers	Observational-longitudinal	Increase in intensity for 2WK	↑ in SWS; ↓ in S1 and S2
Teng *et al* (2011)	28 elite cyclists	Cross-sectional-observational	Increase in intensity for 3WK	↓ in TST and SE; ↑ in MAS
Thornton *et al* (2018)	14 elite Rugby League players	Observational-longitudinal	Increase in ac/deceleration load	↑ in TST and SE
Walsh *et al* (2019)	12 elite swimmers	Observational-longitudinal	Increase in intensity	↑ in restfulness
Whitworth-Turner *et al* (2018)	10 youth soccer players	Observational-longitudinal	Increase in high speed running	↑ in TST

Abbreviations: TST = Total sleep time; SE = Sleep efficiency; SOL = Sleep onset latency; S1 = NREM stage 1; S2 = NREM stage 2; SWS = Slow wave sleep; IT = Immobile time; NA = Number of awakenings; MAS = Mean activity score; PSQ = Perceived sleep quality.

2.5.2. Competition

To elucidate the effects of competition on sleep, studies have monitored sleep before, on the day of and in the days after competition (Driller and Couples, 2018; Shearer *et al*, 2015). Shearer *et al* (2015) used actigraphy to monitor sleep of professional Rugby Union players and showed sleep duration (6:02 vs. 7:04 hh:mm) was reduced the night after a game compared to two days before the game. Similarly, professional Rugby League players had reduced sleep duration (4:53 vs. 7:11 hh:mm) and efficiency (68 vs. 82 %) the night after a game compared to nights following no competition (Driller and Cupples, 2018). A limitation of these studies is knowing whether the effects on sleep were due to the competition load itself, the influence of kick off times (i.e. afternoon vs. evening kick off) or behaviours taken after matches (i.e. socialising etc.). Lalor and colleagues (2018) recently assessed the impact of match start times and days relative to match day compared to habitual sleep characteristics in elite Australian Football players. They found few effects of match start time on sleep, though evening kick off times caused most sleep disruption such as likely longer sleep onset latency and almost lower sleep efficiency. When assessing sleep on days relative to the match, there was an almost certain reduction in sleep duration (-77 min) the night after a game compared to habitual sleep. This would suggest that whilst match start time does impact sleep, competition load itself is an important factor that affects subsequent sleep in athletes competing at the highest level. The disruption to sleep with competition may also be greater than after training (Lastella *et al*, 2015; O'Donnell *et al*, 2018; Walsh *et al*, 2019), hence sleep changes are likely during the in-season period. Competition load from match play may affect sleep in several ways, including; via the release of pro-inflammatory cytokines (Cunniffe *et al*, 2010), resultant muscle damage (Nedelec *et al*, 2015) and the associated mental stimulation and cognitive fatigue (Fullagar *et al*, 2015). Future research is warranted to determine the

time length in which sleep returns to baseline after competition to inform whether athletes are recovered before competing again.

2.6. Effect of Acute Exercise on Sleep

The majority of studies to current date that have investigated training load and sleep in athletes have utilised observational designs, meaning it is difficult to ascertain causality of effects. Therefore, acute exercise studies may shed further light on the relationship between training and sleep, as they can separate the effects of exercise on sleep from training scheduling.

2.6.1. Exercise Intensity

Studies that have documented the sleep response to exercise intensity have used different exercise protocols, exercise timing and participants. Nonetheless, it seems the time elapsed between performing high intensity exercise and bedtime is the best predictor of the sleep response (Stutz *et al*, 2019). When performed 2-6h before bedtime, experimental studies within non-athletic individuals have shown that high intensity exercise has no effect (Myllymaki *et al*, 2011; Myllymaki *et al*, 2012) or may improve total sleep time, sleep efficiency, wake after sleep onset, sleep onset latency, NREM stage 2, SWS and REM sleep (Dworak *et al*, 2012; Flausino *et al*, 2012; Hayashi *et al*, 2014; Horne and Staff, 1983; Larsen *et al*, 2019; Wong *et al*, 2013). In contrast, only Oda and Shirakawa (2014) have observed sleep disruption i.e. a longer sleep onset latency, after high intensity exercise when performed 1h before bedtime. Moreover, when performed in the morning, there appears to be no effect of intensity on sleep (Jones *et al*, 2008). Therefore, these findings do not support current sleep hygiene guidelines that advise against performing high intensity exercise in the evening, for the reason it may increase arousal and disrupt subsequent night's sleep (American Sleep Association, 2019). In athlete populations, a study by Ramos-Campo *et al* (2019) showed sleep efficiency and the average time of

awakening from actigraphy were worsened after vigorous compared to moderate intensity exercise in the early evening within amateur ultra-endurance runners. This might be an example of where the greater intensity performed by athletes is detrimental to sleep rather than beneficial. However, it should be noted this study was not without limitations, as the authors did not report the bedtime of participants or standardise time in bed, nor did they use a control or use a measure of PSG. Another study in well-trained runners found an increase in NREM sleep and a decrease in REM sleep after simulated late-night trail running, despite an increase in nocturnal HR (Aloulou *et al*, 2019). Taking these findings into account, the effect of exercise intensity on nocturnal sleep is still debatable in athletes, requiring further studies to examine these effects more closely with rigorous study designs.

2.6.2. Exercise Duration

The findings surrounding the effect of exercise duration on sleep in athletes are equivocal, making it difficult to draw firm conclusions. Studies have utilised exhaustive exercise protocols to assess the impact of exercise duration on sleep. Increased wakefulness was observed in fit athletes after an ultra-triathlon (Driver *et al*, 1994) and a marathon (Montgomery *et al*, 1985) but there was an increase in total sleep time the night after an ultra-marathon race, also in fit athletes (Shapiro *et al*, 1981). In terms of sleep stages, REM sleep and SWS were decreased after a marathon (Montgomery *et al*, 1985), though SWS has also shown to be increased (Shapiro *et al*, 1981; Bunnell *et al*, 1983) as well as EEG delta power (Torsvall *et al*, 1984). Importantly, all these studies lack ecological validity because such exhaustive exercise protocols are not commonplace within an athletes training program and are more reflective of competition performances. Another issue is that it cannot be guaranteed intensity was constant throughout the exercise, which would be required to measure the fixed effect of exercise duration. In this respect,

Myllymaki *et al* (2012) provide the best indication of this interaction, who showed no change in actigraphic sleep after 30, 60 and 90-min of running at a HR level corresponding to 60% VO_{2max}, despite increased cardiac autonomic activity in the latter compared to no exercise. Although, this study used active individuals and it is unknown whether these results would be replicated in athletes who may perform at a higher intensity.

2.6.3. Exercise Timing

From the few studies available, it would appear the time of day that exercise is performed can elicit effects on sleep that are independent of both exercise intensity and duration. When performed in the afternoon (14:00h), strenuous intermittent-type exercise resulted in greater total sleep time, sleep efficiency, SWS, REM sleep and reduced wakefulness compared to evening exercise (20:00h), in trained individuals (Baekeland and Lasky, 1966; Souissi *et al*. 2012). The findings surrounding the effects of morning exercise on sleep are somewhat more conflicting. Morning exercise (10:00h) performed at moderate and vigorous intensity led to greater total sleep time and reduced wakefulness compared with evening exercise (20:00h), in ultra-endurance runners (Ramos-Campo *et al*, 2019). There was a greater rating of subjective sleep quality and lower daytime sleepiness after 1h of aerobic exercise in the late evening (20:30h) compared with morning (07:40h) in healthy individuals (Yoshida *et al*, 1998). However, no effect of morning (10:00h) or afternoon (16:00h) cycling prescribed at a low intensity on sleep, in untrained individuals has also been reported (Horne and Porter, 1976). These differences may be explained by the sleep measurement (PSG vs. actigraphy vs. subjective sleep rating) or variations in the populations studied. It is also plausible that rest after morning exercise may serve as a sufficient strategy for recovery instead of sleep in such circumstances (Vyazovskiy,

2015). Based on these findings, the effect of exercise timing in athletes is variable and the responses are subject to further investigation.

2.7. Other Factors Affecting Sleep

2.7.1. Early and late-night schedule

The amount of sleep athletes obtain can vary depending on the time of day they are required to train. Two studies showed the amount of time spent asleep was reduced on training days compared to rest days when sessions were scheduled early in the morning in elite athletes (06:00h) (Sargent *et al*, 2014a; Sargent *et al*, 2014b). Importantly, even when athletes advanced their bedtime to compensate for having to wake up earlier, there was no increase in sleep quantity (Sargent *et al*, 2014a; Sargent *et al*, 2014b). A substantial shift in bedtime may be difficult for athletes to achieve because of many reasons. First, family and social commitments in the evening will limit a person from advancing their bedtime (Folkard and Barton, 1993; Tucker *et al*, 1998). Second, sleep propensity tends to be highest during the night and mid-afternoon but lowest in the early evening; subsequently this time of day is coined the forbidden sleep zone (Lavie, 1986; Folkard and Barton, 1993; Lack and Lushington, 1996). Such a scenario is pertinent for individual sport athletes (i.e. swimmers, rowers, cyclists and runners) who train twice a day to accommodate large training volumes. Factors such as competition timing and travel requirements may also prevent athletes from advancing their bedtime (Sargent and Roach, 2016; Costa *et al*, 2019). However, not all athletes sleep is disrupted by early morning training schedules. Lastella *et al* (2014) collected sleep data on 124 elite athletes from team (Australian Rules Football, Basketball, Rugby Union and Football) and individual (Cycling, Mountain Bike, Racewalking, Swimming and Triathlon) sports who were involved in a typical out of competition training period. They found team sport athletes had greater sleep duration (7.0 vs. 6.5h) than individual sport athletes despite

having a later bedtime (22:27 v 23:24 hh:mm). It was postulated that as well as a shorter sleep latency (22 v 16min) and less wake after sleep onset (18 v 16%), team sport athletes were able to sleep longer due to them adopting a significantly later get up time than the individual sport athletes (06:42 v 07:56h). Team sport athletes often start training later in the morning and in cases, not until the early afternoon, meaning they have greater opportunity during the early morning period to extend their sleep.

Late night competition as opposed to daytime competition can also have an impact on athletes' sleep-wake behaviour. Fullagar *et al* (2016) examined responses of elite male footballers to a subjective sleep questionnaire across day (finished before 18:00h) and night (kicked off after 18:00h) matches as well as training days. The analysis revealed later bedtimes and shorter sleep durations after night matches compared with day matches and training days. Using wristwatch actigraphy, Sargent and Roach (2016) monitored the sleep of Australian Rules footballers after one day match (15:45-17.45h) and one-night match (19:10-21:00h). They showed time in bed was reduced (9.3 v 6.8h), sleep onset was later (23:35 v 01:56 hh:mm) and sleep duration was shorter (7.4 v 5.3h) following night matches. The reduction in sleep duration with late night competition is most likely related to a multitude of factors such as; the scheduling of late kick offs, performing high intensity exercise close to bedtime, using caffeinated products around competition and fulfilling post-match commitments (team meetings, social activities, press conferences and recovery practices) (Fullagar *et al*, 2015; Nedelec *et al*, 2015). Alternatively, one study reported no changes in sleep after a night match (19:00 v 21:00h) compared with a day match and a rest day within elite youth soccer players (Roach *et al*, 2013). The non-competitive nature of friendly games, the lack of expectation on youth soccer players to complete post-match duties and the close proximity of the accommodation to the playing location may explain the discrepancy between results. Whilst research in this area has heavily focused on football, the negative effect of late-night competition on sleep is

probably characteristic of many other sports. Juliff *et al* (2015) surveyed 283 elite Australian athletes from a variety of team (Football, Basketball, Netball, Hockey, Rugby League, Softball, Volleyball and Water polo) and individual (Athletics, Canoeing, Cycling, Gymnastics, Rowing, Sailing, Speed Skating, Swimming, Tennis and Triathlon) sports about their sleep habits around competition. In terms of sleep disturbances, 52% of athletes cited they experienced poor sleep after late night games at least once, although the ratio of team sport to individual sport athletes was not provided. As such, competition timing (night vs. day) would appear to be detrimental to athletes' sleep, but more research is currently warranted to examine whether this effect is sport specific related.

2.7.2. Sleep Environment

Athletes frequently reside in unfamiliar environments (e.g. hotel) the night before playing an away fixture. This is preferred to travelling on the day of a game as it gives athletes more time to adjust to the competition setting (Waters and Lovell, 2002). In these circumstances, both perceived sleep quality and time spent awake are negatively affected compared with staying in the home environment (Erlacher *et al*, 2009; Richmond *et al*, 2004). This is comparable to that of the "first night effect" (Agnew *et al,* 1966), when an individual spends their first night in a sleep laboratory. Many factors may contribute to the disturbance in sleep such as; sleeping in a different bed, sharing a room with teammates and external noise (Erlacher *et al*, 2011; Juliff *et al*, 2015; Savis, 1994). Travelling home later may also contribute to sleep disturbance the night after an away game (Costa *et al,* 2019). Accordingly, it would seem appropriate to monitor athletes sleep when travelling to other locations during the season.

Athletes may also be exposed to scenarios whereby they travel to alternative locations for preparation camps before the competitive season, in an attempt to improve fitness and morale (Buchheit *et al,* 2013). As such, two studies have examined the impact of a

training camp on nocturnal sleep (Thronton *et al*, 2017; Pitchford *et al*, 2017). Pitchford *et al* (2017) studied sleep patterns of Australian Rules footballers across an eight-day home and camp period. They reported lower sleep efficiency (78.7 vs. 84.7 %) and greater wake after sleep onset (96.5 vs. 68.9 min) during camp. Similarly, in Rugby League players, total sleep time (05:51 vs. 07:17 hh:mm), sleep efficiency (79.6 vs. 88.1 %) and time in bed (07:22 vs. 08:16 hh:mm) were reduced in camp compared to home (Thornton *et al*, 2017). However, there are also further external stressors that may affect sleep; including heat and altitude (Okamoto-Mizuno *et al*, 2005; Sargent *et al*, 2013). These findings may highlight the need to optimise athletes sleep during camps, where the desired outcome is to improve competitive performance.

2.7.3. Psychological Factors

Sleep disturbance is often present in individuals with anxiety (Cox and Olatunji, 2016); similarly, athletes do not escape anxiety and nervousness, which may then impact their sleep. Data from retrospective studies indicates athletes have difficulty falling sleep the night prior to competition, which was suggested to relate to nervousness and anxious thoughts (Erlacher *et al*, 2011; Juliff *et al*, 2015; Lastella *et al*, 2014; Savis *et al*, 1997). Erlacher *et al* (2011) suggested individual sport athletes are more at risk of sleep problems the night before competition due to the pressure of being solely responsible for their results. In contrast, Juliff *et al* (2015) found team and individual sport athletes were similar in the reported occurrences of sleep disturbances prior to important competition. These findings are important as sleep loss the night before competition may hinder subsequent next day performance.

2.7.4. Electronic Device Use

Exposure to bright light at short wavelengths prior to bedtime can increase sleep onset latency and decrease sleep propensity (Cajochen *et al*, 1992; Carrier and Dumont, 1995).

Such effects are thought to occur through delaying melatonin onset (Chellappa *et al*, 2011), since light is one of the main time givers for the circadian pacemaker, SCN (Schwartz and Roth, 2008). In the same way, blue light from electronic device use is thought to hinder nocturnal sleep and is not encouraged in the hour(s) prior to bedtime (American Sleep Association, 2019). Seemingly, athletes do not adhere to such recommendations, with use of electronic device at night-time replacing the loss of social interaction when in training or competition (Halson, 2015; Romyn *et al*, 2015). Reducing electronic device use has shown to be useful in increasing sleep duration in Football players (Fullagar *et al*, 2016) and Netball players (O'Donnell and Driller, 2017). Although removing all electronic devices did not change sleep quantity or quality in a group of elite Judo athletes (Dunican *et al*, 2017) and highly-trained netballers (Jones *et al*, 2018). Further information is required around athletes sleep habits in the hours prior to bedtime, so that interventions can be implemented to improve sleep behaviour in this cohort.

2.8. Influence of Nutrition on Sleep

2.8.1. Carbohydrate

High glycemic index (GI) carbohydrates are suggested to promote sleep through increasing the tryptophan to Large Neutral Amino Acid (LNAA) ratio (Afaghi *et al*, 2007). A greater flux of free tryptophan across the blood brain barrier increases brain serotonin levels and thus, melatonin, a substance involved in the circadian timing of sleep (Afaghi *et al*, 2007). The intake of high GI carbohydrate meals increases the tryptophan to LNAA ratio via the direct action of insulin, which promotes the uptake of LNAA's into the muscle (Herrera *et al*, 2011; Lyons and Truswell, 1988; Wurtman *et al*, 2003). Herrera *et al* (2011) reported a higher blood glucose and insulin response 30-45 min after the ingestion of a high GI meal (3212kJ; 90.4% energy from CHO, 1.6% from FAT and 8%

from PRO) compared to a low GI meal. This resulted in a significantly higher peak in the plasma tryptophan to LNAA ratio (23 vs. 8%) 2-4h after ingestion (Herrera *et al*, 2011). Furthermore, adding protein to a carbohydrate meal prevents increases in plasma tryptophan, without affecting insulin secretion, due to contributing more LNAA (Yokogoshi and Wurtman, 1986). The positive effects of high GI carbohydrate meals on nocturnal sleep has been demonstrated by PSG analysis (Afaghi *et al*, 2007; Vlahoyiannis *et al*, 2018). Afaghi and colleagues (2007) showed the same high GI meal reduced sleep onset latency (-8.5 min) compared to a calorie matched low GI meal when consumed 4h before bedtime. No measurement of the insulin response was taken, but it could be assumed there was a higher insulin concentration in the blood after the high GI meal as blood glucose concentrations were significantly elevated. When the timing of the meal was assessed, this improvement in sleep onset latency was maintained when the meal was eaten 4h before bedtime (-5.6 min) compared to 1h before bedtime (Afaghi *et al*, 2007). This effect may be due to the tryptophan to LNAA ratio peak occurring 2-4h after the ingestion of a high GI carbohydrate meal (Lyons and Truswell, 1988; Wurtman *et al*, 2003; Herrera *et al*, 2011). Therefore, the type and timing of carbohydrate may be important when considering sleep.

2.8.2. Alpha-Lactalbumin

Supplementing with the milk protein, α-Lactalbumin, may improve both sleep duration and sleep quality. Ong *et al* (2017) demonstrated 20g of α-Lactalbumin consumed in the form of a milkshake 1h before bedtime increased total sleep time (+54.7 min) and sleep efficiency (+7%) over two nights compared to a placebo (sodium caseinate) in healthy adults. Moreover, this finding was independent of any change in individual sleep variability; indicating differences in sleeping patterns between nights was not responsible for the improvement (Ong *et al*, 2017). Ingestion of α-Lactalbumin acts through

increasing the plasma tryptophan to LNAA ratio, thus influencing brain serotonin (Markus *et al*, 2005). α-Lactalbumin contains the highest amount of tryptophan amongst all other food protein sources (Heine *et al*, 1996) and is recommended as an alternative to pharmacological tryptophan (Hudson *et al*, 2005). Re-feeding young male Wistar rats with a diet containing 30% of α-Lactalbumin was able to fully recover SWS immediately after four days of caloric restriction intended to lower both plasma tryptophan and brain serotonin levels (Minet-Ringuet *et al*, 2004). It was concluded the higher tryptophan to LNAA ratio within the α-Lactalbumin diet favoured the synthesis of brain serotonin and subsequently improved sleep (Minet-Ringuet *et al*, 2004). Markus *et al* (2005) examined the effects of two 20g servings of α-Lactalbumin in the evening (milkshake at 18:30 and at 19:30h) on vigilance and sleepiness in individuals with sleep complaints considered deficient in brain serotonin. The high α-Lactalbumin diet caused a 130% increase in the plasma tryptophan to LNAA ratio, thereby surpassing the 20-40% increase thought to be sufficient to change brain serotonin levels (Fernstrom and Wurtman, 1971; Fernstrom and Fernstrom, 1995). This was also accompanied by reduced morning sleepiness and improved vigilance (assessed via a continuous performance task), suggesting the beneficial effects of α-Lactalbumin were mediated by improved sleep the previous night (Markus *et al*, 2005). This may suggest α-Lactalbumin is beneficial for sleep, particularly in individuals with poor sleep patterns.

2.8.3. Tart Cherry Juice

The consumption of tart cherry juice may alter sleep behaviour as it has multiple sleep promoting properties. Tart cherry juice is made from tart Montmorency cherries, which are abundant in many phytochemicals, including the sleep regulating hormone, melatonin (13.46ng/g) (Burkhardt *et al*, 2001). Supplementing with tart Montmorency cherry juice has been shown to improve objective and subjective sleep parameters (Pigeon *et al*, 2010;

Howatson *et al*, 2012). In the study by Howatson *et al* (2012) two servings (30ml within 30 min of awakening and 30 min before evening meal) of tart Montmorency cherry juice per day over seven days increased both total sleep time (+39min) and total sleep efficiency (+4.9%), as well as urinary 6-sulphatoxymelatonin (aMT6 [major metabolite of melatonin]) compared to a placebo (mixed fruit cordial). It was suggested that the exogenous melatonin from tart cherry juice facilitated the reduction in core temperature and the increase in sleep propensity that is observed with endogenous melatonin (Cajochen *et al*, 2003; Claustrat *et al*, 2005).

In addition, tart cherry juice may influence pro-sommnogenic cytokines involved in sleep regulation (interleukin-1β [IL-1β], interleukin 8 [IL-8] and Tumor Necrosis Factor-α [TNF-α]) (Garcia-Maurino *et al*, 1999; Fjaerly *et al*, 1999). Numerous compounds exist within tart cherries, which may augment pro-sommnogenic cytokines through their anti-inflammatory and antioxidant capacity (McCune *et al*, 2011; Opp, 2004). Garrido and colleagues (2013) provided supporting evidence towards this when assessing the effect of a Jerte-Valley cherry juice product (similar to tart cherry juice) on sleep quality in young, middle aged and older adults. They showed improvements in total sleep time and nocturnal restlessness, as well increases in aMT6 and serum IL-1β, IL-8 and TNF-α across all age groups following five days of the Jerte-Valley cherry juice supplementation. These findings suggest that improvements in sleep with cherry juice ingestion are beyond the effects of exogenous melatonin alone.

2.8.4. Caffeine

Caffeine is beneficial for restoring cognition upon waking, primarily due to its action of blocking adenosine receptors in the brain (Van Dongen *et al*, 2001). However, when consumed in the afternoon or evening, caffeine has the potential to impair subsequent sleep. Drake *et al* (2013) demonstrated that 400mg of caffeine in pill form consumed at

0, 3 and 6h before bedtime decreased total sleep time, sleep efficiency and SWS, compared to a placebo. Caffeine intake has also shown to impair sleep onset in a dose-dependent manner, but critically even doses as low as 75mg can impair nocturnal sleep (Hindmarch *et al*, 2000). This is pertinent for athletes, as many sportspeople consume caffeine in large doses before training and competition to improve their performance (Pickering and Kiely, 2019). Poor sleeping patterns observed within elite athletes may be (in part) attributed to their caffeine consumption.

2.9. Summary

In summary, adequate sleep provides physiological and psychological benefits that are important for the recovery and performance of athletes (Davenne, 2009; Halson, 2013). There is evidence, however, indicating athletes do not meet sleep guidelines and may obtain less sleep quality than non-athletes (Bender *et al*, 2018; Demirel, 2016; Leeder *et al*, 2012; Mah *et al*, 2018). Among the many factors that may impact athletes' sleep (i.e. schedule, sleep environment, psychological, electronic device use and nutrition), training-induced stress has received the most attention, but the findings are generally unclear due to confounding variables. Therefore, it would be appropriate to perform further studies to elucidate the training effect. Specifically, a comparison of athletes from a range of sports, with non-athletes during the competition period would be the first step to understand how these populations may differ due to different training schedule demands. It would also seem important to conduct experimental studies that are well-controlled and that use the gold standard PSG to assess sleep behaviour after different exercise protocols. This may then assist in optimising training prescription (i.e. intensity, duration and timing) so that the balance between training stress and recovery can be managed better in athletes.

Chapter 3

Comparison of Athletes and Non-Athletes sleep patterns

3.1. Introduction

In preparation for competition, the most important goal for coaches is to increase the physical, technical and psychological attributes of their athletes to the highest possible level so that peak performance can be attained (Le Meur *et al*, 2012). To achieve this goal, training and competition need to be carefully balanced with recovery so fatigue does not manifest into a state of NFOR and impair performance (Barnett, 2006; Meeusen *et al*, 2012). Many strategies are used by elite athletes to assist with recovery including; cold water immersion, compression garments and nutritional interventions (Halson, 2013), however, beyond these modalities sleep is considered to be the most efficacious for recovery (Halson, 2008). Sleep is understood to be important for physiological and psychological restoration from previous wakefulness (Samuels, 2008). This is likely explained by a homeostatic response of sleep i.e. the need to sleep is increased with wake, thus reflecting a recovery type process (Porkka-Heiskanen, 2013; Borbely *et al*, 2016). As a result, it is generally accepted elite athletes require more sleep than their sedentary counterparts due to training and competition demands (Davenne, 2009).

Despite the importance of sleep, athlete populations often demonstrate shorter sleep durations and lessened sleep quality (Leeder *et al*, 2012). Anecdotally, athletes may report sleep loss on the night prior to important competition (Erlacher *et al*, 2011; Lastella *et al*, 2014; Juliff *et al*, 2015). Such complaints refer to problems falling asleep and this is attributed to nervousness and thoughts about competition (Erlacher *et al*, 2011; Lastella *et al*, 2014; Juliff *et al*, 2015). In particular, individual sport athletes cite sleep loss more frequently before competition than team sport athletes (Erlacher *et al*, 2011). It is also of the belief these sleep problems are exacerbated during periods of competition in comparison to training (Juliff *et al*, 2015). In some cases, competition sleep has been compared to a baseline period considered representative of athletes' habitual sleep

patterns (Caia *et al*, 2017; Lastella *et al*, 2015; O'Donnell *et al*, 2018; Shearer *et al*, 2015; Walsh *et al*, 2019). Such studies have showed sleep disruption relating to sleep onset and sleep duration during the competition period. Others have compared changes in sleep between competitions scheduled at different times of day and found sleep is reduced following night-time games (Fullagar *et al*, 2016; Sargent and Roach, 2016). Taken together, these results may suggest factors associated with competition such as anxiety, exercise intensity and schedule may cause sleep disruption within athletes.

To date, only a few studies have compared the sleep of athletes to non-athletes (Bender *et al*, 2018; Demirel *et al*, 2016; Leeder *et al*, 2012; Whitworth-Turner *et al*, 2017) and found athletes have similar sleep duration to non-athletes, but reduced sleep quality. However, no research has objectively compared sleep of athletes from multiple sports to non-athletes across consecutive days during the in-season training period. This type of study is warranted to further understand the differences between the general population and that of individuals regularly training within sport. Therefore, the current study aimed to compare the sleep patterns of athletes with non-athletes during an in-season training week using wristwatch actigraphy. Training load during the week leading into competition was also compared to assess additional factors that may affect sleeping patterns. It was hypothesised that the athlete group would have comparable sleep duration to non-athletes, but reduced sleep quality (i.e. sleep efficiency). This information could further the understanding of sleep behaviour and the potential factors influencing sleep within elite athlete populations.

3.2. Materials and Methods

3.2.1. Participants

Thirty athletes (age = 23±5yrs; nine males and twenty-one females) and thirty non-athletes (age = 25±3yrs; seven males and twenty-three females) were recruited. The athlete group consisted of team sport athletes (Soccer, *n* = 14; Rugby Union, *n* = 2; Basketball, *n* = 2; Water Polo, *n* = 1) and individual sport athletes (Endurance [triathlon, duathlon, racewalking, 10,000m], *n* = 5; Track and Field, *n* = 2; Boxing, *n* = 1; Swimming, *n* = 1; Rifle Shooting, *n* =1; Archery, *n* =1) who were competing at national and/or international level. Upon data collection, none of the athletes had completed a long-haul flight or had been diagnosed with a sleep disorder. The non-athlete group were recreationally active, healthy individuals and in either full time education or employment. Prior to commencing the study, a full explanation of the research protocol was given to participants and written informed consent was obtained. In addition, the morningness-eveningness questionnaire (MEQ) (Horne and Ostberg, 1976) was filled in for the assessment of chronotype. Mean chronotype score was 55.4±8 (intermediate) for athletes (55±7.4 [intermediate] for team sport athletes and 56.3±9.4 [intermediate] for individual sport athletes) and 55.6±7.1 (intermediate) for non-athletes. This study was granted ethical approval by the Local University research ethics committee.

3.2.2. Experimental Design

As part of a descriptive, observational study design, wristwatch actigraphy was used to monitor sleep for seven days. A week was chosen to capture the variation in scheduled activities (i.e. different training sessions) within the athlete group, as opposed to monitoring sleep on one off days. The athlete group were monitored during a regular in-season training period. On days one to five, training and rest days were scheduled for all athletes; the order of these activities differed depending on the sport and discretion of

coaches and performance staff. Athletes then competed in their respective sports on day six and used day seven as a recovery day. Both of these days were consistent during the monitoring period to prevent differences in sleep patterns induced by the timing of competition in relation to the amount of days recorded. The non-athlete group were monitored during a seven-day period in which they participated in their habitual daily routines; this involved a mixture of typical working or study days plus days off. Sleep data was only accepted for analysis when participants had worn the actiwatch on at least five nights as this has shown to be the minimum number of nights required to obtain reliable sleep measures using wristwatch actigraphy (Acebo *et al*, 1999). Throughout the monitoring period, participants were encouraged to maintain their habitual sleeping patterns and not change their usual daily activities or schedule. On each day, both athletes and non-athletes were also asked to complete a training diary, so that a subjective assessment of the daily internal training load could be conducted. This was completed to examine the effects of exercise training on sleeping patterns between groups. Table 3.1 provides an overview of the training microcycle for the athlete group and non-athlete group.

Table 3.1. Overview of training microcycle in the athlete and non-athlete groups. Mean±SD training times and number of sessions performed (*n* = absolute number of sessions; % = relative number of sessions; CV = cardiovascular; CN = conditioning; COM = competition).

	Morning	Afternoon	Evening
Non-athletes	*n=42*	*n=36*	*n=47*
Training start (hh:mm)	08:51±01:22	14:19±01:36	18:31±01:02
Training end (hh:mm)	09:50±01:30	15:31±01:52	19:21±01:12
CV (%)	25.6%	15.2%	19.2%
CN (%)	8%	13.6%	18.4%
Team sport athletes	*n=71*	*n=63*	*n=26*
Training start (hh:mm)	10:17±01:01	14:03±01:18	18:47±01:27
Training end (hh:mm)	11:36±01:17	15:08±01:19	20:00±01:48
CV (%)	27.5%	13.1%	10.6%
CN (%)	16.9%	18.1%	1.9%
COM (%)	0%	8.1%	3.8%
Individual sport athletes	*n=24*	*n=16*	*n=23*
Training start (hh:mm)	08:18±01:18	14:16±01:37	18:23±00:51
Training end (hh:mm)	09:13±01:21	15:26±01:54	19:12±00:56
CV (%)	20.6%	12.7%	33.3%
CN (%)	9.5%	4.8%	0%
COM (%)	7.9%	7.9%	3.2%

3.2.3. Experimental Procedures

3.2.3.1. Sleep Monitoring

Participants wore an actiwatch (Actiwatch 4, Cambridge Neurotechnology Ltd, UK) on their non-dominant wrist for sleep monitoring. The actiwatch was set to an epoch length of 1 min and at a medium sensitivity. This level of sensitivity was chosen as it has been shown to have a good agreement (86.5% vs. polysomnography) with PSG for sleep-wake detection and is thus considered a valid alternative to measure sleep (Sargent *et al*, 2016). Participants were instructed to wear the actiwatch at least an hour before they got into bed. When lying down in bed, participants recorded their bedtime (lights off) using the Consensus Sleep Diary (Carney *et al*, 2012) and pressed the marker button on the face of the actiwatch for approximately 2-3s (Figure 3.1). The following morning, upon their final awakening (lights on), participants pressed the marker button again and filled in the remainder of the Consensus Sleep Diary within an hour of being awake. The Consensus Sleep Diary asked questions relating to sleep onset latency, number of awakenings, total duration of awakenings, time of final awakening and get up time. Participants were then free to remove the actiwatch from their non-dominant wrist and begin their day as usual. Both the actiwatch and Consensus Sleep Diary were used to determine bedtime, sleep onset, wake up time and get up time so that sleep behaviour could be automatically calculated using the appropriate actiwatch software (Actiwatch activity and sleep analysis version 5.24, Cambridge Neurotechnology Ltd, UK). From the actiwatch analysis, the following sleep parameters were used:

Bedtime (hh:mm): Time reported when participant went to bed to attempt to sleep.

Final awakening (hh:mm): Time reported when participant woke up and stopped attempting to sleep.

Time in bed (min): The total time spent in bed attempting to sleep between bedtime and get up time.

Sleep duration (min): The amount of sleep obtained between sleep onset and sleep end, minus any wake time.

Sleep onset latency (min): The time between bedtime and sleep onset time.

Sleep efficiency (%): The percentage of time spent asleep whilst in bed.

Wake after sleep onset (min): The amount of time awake between sleep start and sleep end.

Mean activity score (count): The average value of the activity counts per epoch over the assumed sleep period.

Total activity score (count): The total number of activity counts between sleep start and sleep end.

Fragmentation index (%): The percentage of epochs where activity is > 0.

The image originally presented here cannot be made freely available via LJMU E-Theses Collection because of copyright. The image was sourced at: http://susanmorris.com/sun-dial-about/

Figure 3.1. Actiwatch 4 and the marker button used to indicate "lights on" and "lights off".

Figure is from http://susanmorris.com/sun-dial-about/

3.2.3.2. Training Monitoring

A rating of perceived exertion (RPE) score taken from the modified Borg CR10 scale (ranged from 0 [rest] to 10 [maximal exertion]) was used to monitor daily internal training load (Foster *et al*, 2001). Using a handheld diary, the RPE was recorded after the cessation of all exercise sessions, along with the duration of exercise training. The athlete group also recorded these parameters following their competition. All participants were familiarised with the RPE scale and were shown how to fill in the diary. Overall session load was determined by multiplying the exercise duration by the RPE score (Foster *et al*, 2001). The daily training load score was the sum of each overall session load that was calculated.

3.2.4. Statistical Analysis

Statistical Package for the Social Sciences (SPSS v25) was used for data analysis. Using the average of the standard deviations across monitored days, the intra-individual variability (IIV) of each sleep parameter was calculated for both athletes and non-athletes. Normality of data was checked using the Kolmogorov-Smirnov test. Where normality was met for data sets (time in bed, sleep duration, sleep efficiency, wake after sleep onset, mean activity score, total activity score, time in bed IIV, sleep onset latency IIV, fragmentation index IIV, training duration and RPE), an independent samples t-test was conducted to assess significant differences between athletes and non-athletes, and data were presented as mean±SD. In circumstances when normality was violated (bedtime, final awakening, sleep onset latency, fragmentation index, bedtime IIV, final awakening IIV, sleep duration IIV, sleep efficiency IIV, wake after sleep onset IIV, mean activity score IIV, total activity score IIV, training frequency and daily load), the non-parametric Mann-Whitney U test was chosen to test significant differences between athletes and non-athletes', and the median and interquartile range was reported. Effect size (ES) of all

significant differences for normally distributed data was calculated by dividing the difference in group means by the pooled standard deviation (Cohen's d) and was assessed using the following thresholds: <0.20 = trivial effect; 0.20-0.60 = small effect; >0.60-1.20 = moderate effect; >1.20-2.00 = large effect; >2.00-4.00 = very large; >4.00 = extremely large (Hopkins *et al*, 2009). ES of all significant differences for non-normally distributed data was calculated using the eta squared statistic (Fritz *et al*, 2012) and interpreted according to Cohen (1988), where: <0.1 = very small effect; 0.1 = small effect; 0.3 = medium effect; 0.5 = large effect. Statistical significance was set at level $P<0.05$.

3.3. Results

3.3.1. Average Sleep

The sleep parameters in non-athletes and athletes (including a breakdown of team sport and individual sport athletes) are presented in Table 3.2. There was no difference in bedtime (23:09 vs. 23:28 hh:mm, $p = 0.144$), final awakening (07:42 vs. 07:21 hh:mm, $p = 0.309$) or time in bed (517±61 vs. 495±46 min, $p = 0.139$) between athletes and non-athletes. Sleep efficiency was greater in non-athletes (85.3±4.0 %, $p = 0.003$, ES: 0.81 [moderate effect]) compared to athletes (81.7±4.8 %). There was a moderate significant difference in sleep efficiency between team sport athletes and non-athletes (82.3±5.4 vs. 85.3±4.0 %, $p = 0.038$, ES: 0.63 [moderate effect]) and a large significant difference between individual sport athletes and non-athletes (80.4±3.3 vs. 85.3±4.0 %, $p = 0.002$, ES: 1.34 [large effect]). Sleep onset latency was longer in athletes (18 min, $p = 0.001$, ES: 0.19 [small effect]) than in non-athletes (10 min). There was a small significant difference for sleep onset latency between team sport athletes and non-athletes (18 vs. 10 min, $p = 0.004$, ES: 0.19 [small effect]) as well as between individual sport athletes and non-athletes (20.5 vs. 10 min, $p = 0.020$, ES: 0.15 [small effect]). No differences were found between groups for sleep duration (421±56 vs. 421±43 min, $p = 0.976$), wake after sleep onset (51±21 vs. 49±22 min, $p = 0.655$), mean activity score (12±5 vs. 12±6 count, $p = 0.894$), total activity score (5829±2752 vs. 6032±3186 count, $p = 0.799$) and fragmentation index (26.9 vs. 25.7 %, $p = 0.583$).

Table 3.2. Mean±SD and median (interquartile range) where appropriate of objective sleep variables from wristwatch actigraphy in the non-athlete and athlete groups (with a breakdown of team sport and individual sport athletes). * indicates p <0.05 compared to the non-athlete group.

	Non-athletes	Athletes	Team sport athletes	Individual sport athletes
Bedtime (hh:mm)	23:28(00:55)	23:09(01:23)	23:09(01:07)	23:22(01:42)
Final awakening (hh:mm)	07:21(00:31)	07:42(00:55)	07:49(00:58)	07:15(01:14)
Time in bed (min)	495±46	517±61	515±63	518±59
Sleep duration (min)	421±43	421±56	437±51	392±54
Sleep onset latency (min)	10(10)	18(15)*	18(12)*	20.5(20)*
Sleep efficiency (%)	85.3±4.0	81.7±4.8*	82.3±5.4*	80.4±3.3*
Wake after sleep onset (min)	49±22	51±21	51±24	51±15
Mean activity score (count)	12±6	12±5	11±5	14±5
Total activity score (count)	6032±3186	5829±2752	5639±3021	6191±2253
Fragmentation index (%)	25.7(12.5)	26.9(6.8)	27.5(6.7)	26.7(8.2)

3.3.2. Sleep Intra-Individual Variability

Average of the individual standard deviation across the monitoring period for each sleep parameter was calculated for non-athletes and athletes (Table 3.3). There was greater IIV of bedtime in non-athletes (00:45 hh:mm, p = 0.045, ES: 0.07 [very small effect]) compared to athletes (00:36 hh:mm). There was a small significant difference for bedtime between team sport athletes and non-athletes (00:34 vs. 00:45 hh:mm, p = 0.036, ES: 0.10 [small effect]), but no significant difference in bedtime between individual sport athletes and non-athletes (00:36 vs. 00:45 hh:mm, p = 0.338). No difference in IIV was observed between athletes and non-athletes for final awakening (00:52 vs. 01:04 hh:mm, p = 0.207) or time in bed (55±25 vs. 58±26 min, p = 0.607). IIV of sleep onset latency was greater in athletes (13±9 min, p = 0.002, ES: 0.78 [moderate effect]) compared to non-athletes (7±6 min). There was a moderate significant difference for sleep onset latency between team sport athletes and non-athletes (12±8 vs. 7±6 min, p = 0.012, ES: 0.71 [moderate effect]) and between individual sport athletes and non-athletes (16±11 vs. 7±6 min, p = 0.007, ES: 1.02 [moderate effect]). No differences in IIV were found between athletes and non-athletes for sleep duration (39 vs. 53 min, p = 0.533), sleep efficiency (4.6 vs. 4 %, p = 0.798), wake after sleep onset (13 vs. 16 min, p = 0.320), mean activity score (3 vs. 3 count, p = 0.327), total activity score (1400 vs. 1922 count, p = 0.436) and fragmentation index (7.7±3.1 vs. 8.7±3.5 %, p = 0.250).

Table 3.3. Mean±SD and median (interquartile range) where appropriate of IIV of wristwatch actigraphy variables for the non-athlete and athlete groups (with a breakdown of team sport and individual sport athletes). * indicates $p<0.05$ compared to the non-athlete group.

	Non-athletes	Athletes	Team sport athletes	Individual sport athletes
Bedtime (hh:mm)	00:45(00:45)	00:36(00:56)*	00:34(00:16)*	36(01:04)
Final awakening (hh:mm)	01:04(01:01)	00:52(00:46)	00:53(00:51)	00:52(01:00)
Time in bed (min)	58±26	55±25	52±19	60±34
Sleep duration (min)	53(31)	39(24)	47(29)	38(39)
Sleep onset latency (min)	7±6	13±9*	12±8*	16±11*
Sleep efficiency (%)	4(3.2)	4.6(2.4)	3.8(2.5)	5.4(1)
Wake after sleep onset (min)	16(15)	13(7)	12(6)	17(8)
Mean activity score (count)	3(4)	3(3)	3(3)	5(4)
Total activity score (count)	1922(2153)	1400(1253)	1255(1231)	2416(1983)
Fragmentation index (%)	8.7±3.5	7.7±3.1	6.87±2.41	9.28±3.66

3.3.3. Internal Training Load

Training data is shown in Table 3.4. Training frequency was higher in the athlete group (9 number, $p<0.05$, ES: 0.32 [medium effect]) compared to the non-athlete group (5 number). There was a medium significant difference for training frequency between team sport athletes and non-athletes (9 vs. 5 number, $p<0.05$, ES: 0.46 [medium effect]), but no significant difference between individual sport athletes and non-athletes (7 vs. 5 number, $p = 0.064$). There was no difference in training duration between athletes and non-athletes (67±14 vs. 63±22 min, $p = 0.495$). RPE was also similar between athletes and non-athletes (4.9±1.2 vs. 5.3±1.4 AU, $p = 0.296$). Daily load was higher in the athlete group (593 AU, $p = 0.001$, ES: 0.21 [small effect]) compared to the non-athlete group (333 AU). There was a medium significant difference for daily load between team sport athletes and non-athletes (650 vs. 333 AU, $p<0.05$, ES: 0.44 [medium effect]), but no significant difference between individual sport athletes and non-athletes (297 vs. 333 AU, $p = 0.701$).

Table 3.4. Mean±SD and median (interquartile range) where appropriate of training data in the non-athlete and athlete groups (with a breakdown of team sport and individual sport athletes). * indicates $p<0.05$ compared to the non-athlete group. Daily load = sum of session load score (duration x RPE).

	Non-athletes	Athletes	Team sport athletes	Individual sport athletes
Frequency (number)	5(4)	9(3)*	9(4)*	7(5)
Duration (min)	63±22	67±14	71±10	58±16
RPE (AU)	5.3±1.4	4.9±1.2	5.3±1.2	4.3±0.9
Daily load (AU)	333(242)	593(377)*	650(259)*	297(234)

3.4. Discussion

The current study compared the sleep patterns of athletes with non-athletes using wristwatch actigraphy across an in-season training week. A novel aspect was that athletes were recruited from both team and individual sports during the competitive season. In line with the study hypothesis, sleep duration was comparable between groups, but athletes had lower sleep efficiency compared to non-athletes. The significant reduction in sleep efficiency compared to non-athletes seemed to be a consequence of a longer sleep onset latency. The athlete group also displayed greater IIV of sleep onset latency. These findings may suggest that elite athletes have greater disruption to sleeping patterns (i.e. difficulty falling asleep), compared to non-athletes during a regular in-season training week.

Previous research has suggested that athletes have similar sleep duration compared to non-athletes, but reduced sleep quality (Bender *et al*, 2018; Demirel *et al*, 2016; Leeder *et al*, 2012). The current study adds to these findings and is the first to objectively describe the sleeping patterns of athletes and non-athletes across consecutive days within an in-season training week. As in the aforementioned studies, it was found that athletes had comparable sleep duration to non-athletes (421 vs. 421 min), but their sleep efficiency was lower (81.7 vs. 85.3 %), indicating their sleep quality was inferior. It should also be noted that sleep efficiency in the athlete group was below the minimum 85% recommended by the National Sleep Foundation to promote health and optimise daytime functioning (Hirshkowitz *et al*, 2015; Ohayon *et al*, 2017). These findings are also similar to a recent study that measured sleep patterns of youth soccer players and non-athletes during six days of in-season training, using a wireless monitoring system (Whitworth-Turner *et al*, 2017). In comparison to non-athletes, they showed sleep efficiency (93 vs. 96 %) and sleep onset latency (25 vs. 15 min) were worsened in youth soccer players.

The results from previous research and the current study provides insight to suggest that athletes from a wide range of sports may experience greater sleep disruption compared to non-athletes during the in-season training week. Consequently, these findings may have implications for the recovery and performance of elite athletes during training and competition periods.

When the athletes were divided into team and individual sports, both groups had a similar final awakening time to non-athletes. This partly contrasts with Lastella *et al* (2014), who found individual sport athletes obtained less sleep than team sport athletes, due to earlier wake times. It was suggested the earlier wake times were imposed by early morning training (i.e. 06:00h). Individual sport athletes in this study trained at a similar time to non-athletes, though they achieved less sleep efficiency, owing to longer sleep latency rather than wake time. Interestingly, team sport athletes also had lower sleep efficiency, as a result of longer sleep latencies, despite morning training starting 2h after the non-athletes. This coupled with team sport athletes adopting less intra-individual variation in bedtime, would suggest starting training sessions later may not necessarily allow athletes to extend sleep as previously suggested (Lastella *et al*, 2014; Sargent *et al*, 2014a; Sargent *et al*, 2014b). A potential explanation for this might be due to circadian timing of sleep, as the athletes and non-athletes had a similar chronotype based on questionnaire data.

In comparison to non-athletes, individual sport athletes displayed the biggest reduction in sleep efficiency (large size effect). Given their final awakening times were not different compared with non-athletes, this reduction in sleep efficiency is likely due to a longer sleep onset latency (+10.5 min). This suggests the individual sport athletes had more difficulty falling asleep. Such findings are in agreement with retrospective studies, where individual sport athletes cited falling asleep as the main cause of sleep related problems before important competition (Erlacher *et al*, 2011; Juliff *et al*, 2015). These

investigations concluded that pre-competition anxiety or thoughts about competition may be responsible for poor sleep within individual sport athletes. Indeed, this may have contributed to the lower sleep efficiency in this study; however, it is likely that other factors were involved given sleep parameters were averaged across consecutive days in the build up to competition. One possibility is that the sleep behaviour of the individual sport athletes in this study was affected by evening training sessions. The individual sport athletes performed a greater percentage of their cardiovascular sessions in the evening (between 6 and 7pm) compared to the non-athlete group. Exercise training can increase circulating cortisol and sympathetic activity upon bedtime, which may both interfere with sleep initiation (Chennaoui *et al*, 2015; Roberts *et al*, 2018). This could explain the longer sleep onset latency as well as the greater intra-individual variability in sleep onset latency found within the individual sport athletes across the week leading into competition. The manipulation of training schedules (i.e. training earlier) may, therefore, help those individual sport athletes that have difficulty falling asleep (Juliff *et al*, 2015). Nonetheless, more research is required to understand the effects of training in the evening on sleep within athletic populations.

Athletes from the team sports studied also had a reduction in sleep efficiency compared to the non-athlete group. Similar to individual sport athletes, the team sport athletes had a longer sleep onset latency (+8 min), which suggests these athletes also had difficulty with falling asleep. Such sleep latencies are similar to those previously reported within elite Rugby Union players (Shearer *et al*, 2015), Australian Rules football players (Sargent and Roach, 2016), Soccer players (Fullagar *et al*, 2016) and Rugby League players (Caia *et al*, 2017), but are not as long as those within female netballers (O'Donnell *et al*, 2018). A number of factors may have led to the longer sleep latency and reduction in sleep efficiency such as; training-induced stress, social-related activities, media commitments, increased caffeine intake and hyperhydration (Nedelec *et al*, 2015). The

findings from this study may in part support the involvement of training-induced stress. Data collected from the self-report training diaries showed that daily training load was higher within team sport athletes compared to the non-athlete group. This was due to exercising more frequently rather than an increase in exercise intensity or duration (Table 3.4). An increase in training load has previously been associated with a reduction in both sleep efficiency and sleep duration within other athlete populations (Hausswirth *et al*, 2014; Teng *et al*, 2011; Thornton *et al*, 2017). Such sleep disturbances may occur because of increased pain perception and/or the inflammatory response from post-exercise muscle damage (Nedelec *et al,* 2015; Roberts *et al,* 2018). These perturbations may explain the sleep disturbance observed within the team sport athletes. Future investigations should examine the mechanism(s) by which increased training load may contribute to disrupted sleep.

It should be noted this study is not without limitations. Due to using an observational study design, there are confounding factors that were uncontrolled (i.e. electronic device use), which may have contributed to the poorer sleep in the athlete group. Actigraphy measurements are considered a valid alternative to PSG, however, there are issues with its specificity (i.e. ability to identify wake epochs), so there may be error associated with the sleep efficiency and sleep onset latency measures. There may also have been variation in sleep caused by monitoring athletes at different stages of the season.

3.4.1. Conclusions

The current study used wristwatch actigraphy to assess the sleeping patterns of athletes and non-athletes during an in-season training week. In comparison to the non-athlete group, sleep efficiency was lower and sleep onset latency was longer in team and individual sport athletes, suggesting that sleep quality in the athlete group was disrupted. Based on self-report training data, factors relating to the constraints of the training

schedule and daily load may explain the observed differences in sleeping patterns between athlete and non-athlete populations.

Chapter 4

Effect of Exercise Intensity in the Evening on Subsequent Night's Sleep

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Chapter 5

Effect of Reduced Muscle Glycogen Stores on Subsequent Night's Sleep

5.1. Introduction

It is not uncommon for endurance athletes to undertake exercise training in the evening with high muscle glycogen stores, and then initiate nocturnal sleep with reduced glycogen stores before performing a fasted exercise session the next morning (Impey *et al*, 2018). Such a training strategy, also known as 'train high, sleep low', is suggested to promote training adaptation and provide long-term improvements to endurance performance (Impey *et al*, 2018). The acute training response to sleeping with low glycogen (<200mmol.kg^{-1}DW) stores include; the activation of key enzymes involved in mitochondrial biogenesis (i.e. AMPK; Bartlett *et al*, 2013; Chan *et al*, 2004) and favourable changes in substrate availability (i.e. increased fat oxidation) during next morning exercise training (Lane *et al*, 2015). Furthermore, the 'train high, sleep low' paradigm is attractive to the endurance athlete as this may offset the reduction in exercise intensity the next day, as opposed to training twice a day and performing the second session with low muscle glycogen stores (Yeo *et al*, 2008). This is critical given the importance of high intensity exercise sessions for training (Hawley, 2014).

Despite this, empirical evidence only partly supports the maintenance of exercise intensity (Lane *et al*, 2015), in fact, some studies have shown morning exercise capacity (i.e. time trial performance and time to exhaustion) is reduced after sleeping with low muscle glycogen stores (Hearris *et al*, 2019; Paris *et al*, 2019). Given these reductions in exercise capacity, sleeping with low muscle glycogen itself might be counterintuitive towards next day training, if sleep duration and/or quality is affected. This could also explain the lack of sleep disruption in Chapter 4, as muscle glycogen would not have been markedly lowered. To date, only one study has assessed the impact of reduced muscle glycogen stores on nocturnal sleep. In this study, Louis *et al* (2016) assigned twenty-one triathletes to either a control group, where regular CHO intake was maintained, or a sleep

low group, where CHO was restricted between training sessions, hence, they slept with lower glycogen availability. Over three weeks, the triathletes performed a standardised training program, but with these different nutritional guidelines, whilst sleep was measured every night using actigraphy. Exercise training involved six training sessions across four consecutive days, consisting of high intensity interval training (HIIT) in the afternoon and low intensity training the next morning. Though there was a small, significant decrease in sleep efficiency (-0.9%) in the sleep low group, there was no change in total sleep time or sleep onset latency between groups, suggesting sleeping with low muscle glycogen caused minimal sleep disturbance. However, this study did not standardise time in bed, there was no use of PSG and there was a reliance on the triathletes to comply with the dietary protocol. These limitations warrant further investigation of the effects of reduced muscle glycogen stores on subsequent nocturnal sleep.

Therefore, the primary aim of this study was to investigate the effect of reduced muscle glycogen stores (<200mmol.kg^{-1}DW) on nocturnal sleep in comparison to a no exercise control. A secondary aim was to examine whether muscle damage caused from reducing muscle glycogen may impact nocturnal sleep on subsequent nights (nights two to four) within the home environment. Such information could assist with optimising the 'train high, sleep low' training protocol. It was hypothesised that reduced muscle glycogen stores would disrupt nocturnal sleep on the first night compared to no exercise.

5.2. Methods and Materials

5.2.1. Participants

Eleven recreationally trained males were recruited, but four withdrew from the study citing either work or family commitments. Seven participants (age = 28±10.3yrs; height = 1.8±0.1m; body weight = 74.1±6.9kg and peak oxygen uptake = 58.7±7.4ml·kg^{-1}·min^{-1}) completed the experimental protocol. Inclusion criteria consisted of a VO_{2peak} of ≥50 ml·kg^{-1}·min^{-1}, training ≥2 days per week that involved at least one run session and no history of metabolic disease (i.e. Diabetes). None of the participants were using sleep medication, were involved in night shift work or had travelled across different time zones prior to selection but were familiar with training in the early evening. Before commencing the study, a full explanation of the protocol and associated risks were provided, and written informed consent was obtained. In addition, participants completed the PSQI (Buysse *et al*, 1989) and the composite morningness, sleep F/R and L/V (Smith *et al*, 1989) questionnaires for the assessment of sleep quality, chronotype and other personal attributes. Mean PSQI score was 6±2 and mean chronotype score was 39.4±5.7; F/R score, 5.1±7.0; and V/L score, 37.7±6.6. This study was ethically approved from the Local University research ethics committee.

5.2.2. Experimental Design

Following a cardio-respiratory fitness test and a familiarisation night with PSG in the sleep laboratory, participants completed no exercise (CON) followed by exercise (EX), separated by a minimum of seven days. In the EX condition, participants performed a steady state run in the morning to increase insulin sensitivity (Ortega *et al*, 2015) prior to the afternoon HIIT. Pre, post, 24h post, 48h post and 72h post this steady state session, maximal force of the knee extensor muscles was assessed using the isometric Maximum Voluntary Contraction (MVC) test and a rating of muscle soreness was obtained to assess

exercise-induced muscle damage. PSG, actigraphy and subjective sleep quality were used to measure sleep on the first night in the sleep laboratory. Sleep on nights two, three and four was also assessed using actigraphy in participants home. In the 24h prior to the trials, participants were asked to follow the same diet they had consumed before the familiarisation and were not permitted to take part in strenuous exercise. On the day of the trials, participants were asked to abstain from caffeine after 12:00h and a standardised lunch (3g CHO .kg^{-1} BM, 0.19g FAT .kg^{-1}BM and 0.27g PRO .kg^{-1}BM) was provided to eat between the hours of 12 and 14:00 following the morning exercise session. Additionally, a high protein and low carbohydrate meal (0.6g PRO .kg^{-1} BM, 0.40g FAT .kg^{-1}BM and 0.38g CHO .kg^{-1} BM) was consumed after the HIIT session between 19-19:30h. In the CON, participants consumed their habitual diet, which was high in CHO, as confirmed by food diary entries. An overview of the experimental design is shown in Figure 5.1.

5.2.3. Assessment of Cardio-Respiratory Fitness

VO_{2peak} was measured using an incremental exercise test on a motorised treadmill (HP Cosmos, Germany). See the methods section (4.2.3) in Chapter 4 for a full description of the test. Running speeds equating to 55%, 60%, 70%, 80%, 90% and 100% of VO_{2peak} were then calculated for the steady state and HIIT sessions using the regression of VO_2 and velocity.

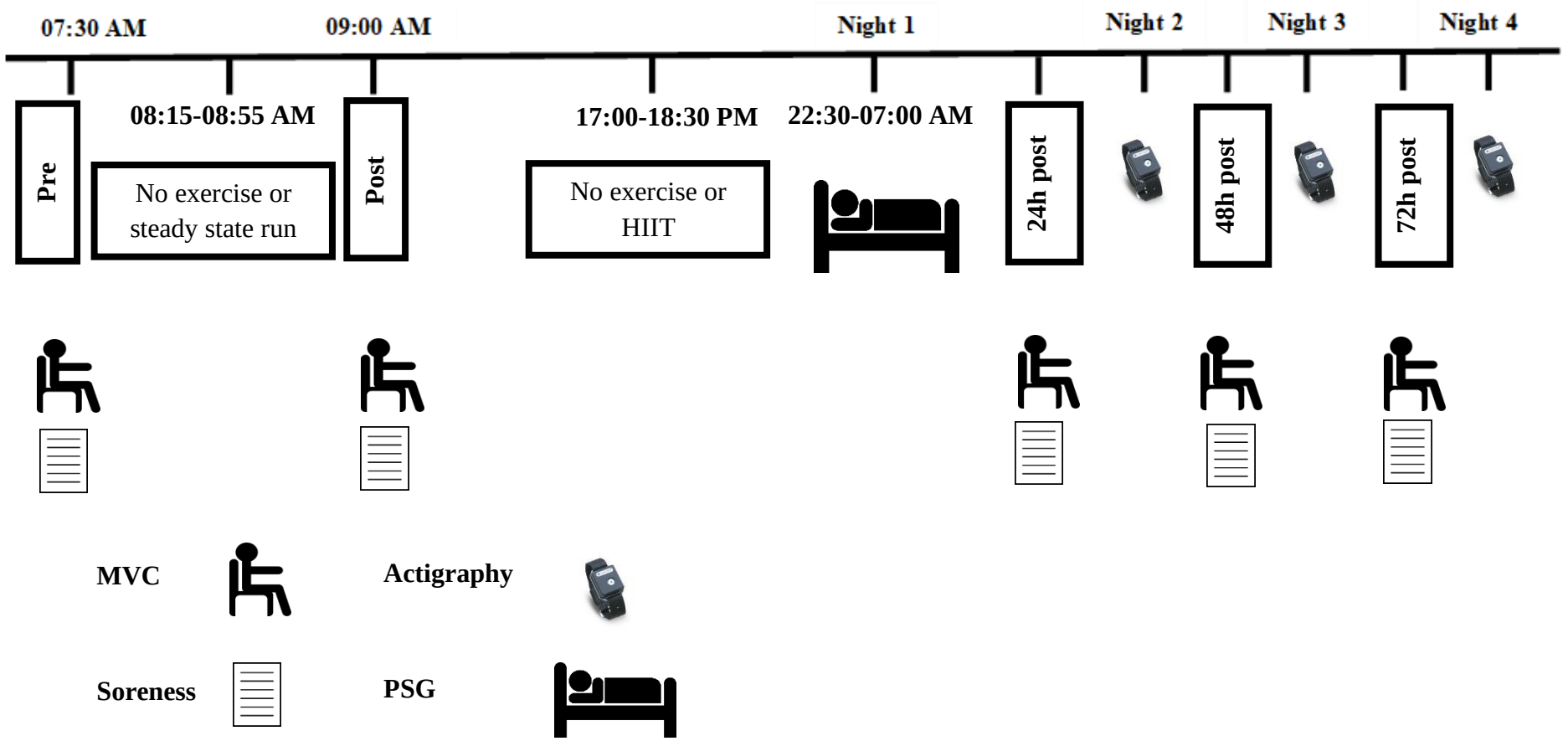

Figure 5.1. Overview of the experimental design.

5.2.4. Experimental Protocol

After consuming a bowl of porridge for breakfast, participants attended the laboratory at 07:30h where they performed an MVC and completed a rating of perceived muscle soreness. Participants subsequently performed a 40-min steady state run at 55% VO_{2peak} on a motorised treadmill. Following the steady state run, the MVC and rating of muscle soreness were repeated immediately, before participants were permitted to leave with a standardised lunch, which was high in CHO to maintain muscle glycogen stores for HIIT in the afternoon. Within the CON trial, participants followed their usual daily activities and did not perform exercise during the day but reported to the sleep laboratory at 19:00h for the overnight sleep.

On exercise days, participants returned to the laboratory at approximately 5pm to perform HIIT. The HIIT was a modified version of the protocol used by Bartlett *et al* (2013), in which they showed muscle glycogen stores were reduced to below 200mmol.kg^{-1}DW in active men. The HIIT session began with 2-min running at a velocity that equated to 100% VO_{2peak} and this was followed by a 2-min recovery performed at 60% VO_{2peak}. Once participants could no longer maintain these intervals, the duration of the running was reduced to 1.5-min and then 1-min whilst at 100% VO_{2peak}. Upon failure of this stage, treadmill velocity was reduced to 90% $VO_{2\ peak}$ and the running duration returned to 2-min, 1.5-min and then 1-min. This pattern continued for 90-min or until participants felt they had reached volitional exhaustion i.e. they were unable to complete 1-min of running at a velocity equating to 70% $VO_{2\ peak}$. Participants received verbal encouragement and were permitted to drink water *ad libitum.*

When the HIIT session was completed, participants showered with the same duration (EX = 04:58±01:32 mm:ss; CON = 05:09±01:54 mm:ss) and water temperature (EX = 35.1±4.9°C; CON = 35.1±4.9°C) used for the control. Duration was documented using a

stopwatch on their mobile phone and water temperature was recorded via a digital temperature probe (accuracy = 1°C) (Electronic Temperature Instruments, Thermamite, UK) placed underneath the showerhead. After showering, participants consumed the high protein meal to increase satiety and maintain protein balance. At 7:45pm, participants were shown to the sleep laboratory to prepare for PSG. Participants were also instructed to wear an actiwatch during the night as in Chapter 4. Bedtime was imposed at 22:30h and get up time was 07:00h the next morning, where participants provided their subjective sleep quality before leaving to start their day.

5.2.5. Sleep Measurements

5.2.5.1. PSG, Wristwatch Actigraphy and Subjective Sleep Quality

See methods section 4.2.5 in Chapter 4 for a description of the procedures used for PSG, wristwatch actigraphy and subjective sleep quality during the first night. From the PSG analysis, the following sleep variables used were: time in bed (min), total sleep time (min), sleep onset latency – stage 1 (min), sleep onset latency – stage 2 (min), time awake (min), NREM stage 1 (%), NREM stage 2 (%), SWS (%), REM sleep (%), NREM stage 1 (min), NREM stage 2 (min), SWS (min), REM sleep (min), deep sleep latency (min) and REM latency (min). Sleep variables used from the actigraphy analysis during the first night were: total sleep time (min), sleep onset latency (min), fragmentation index (%) and the total activity score (count). Subjective sleep quality was based on the Likert scale in the Consensus Sleep Diary ranging from 1-5 (1 = very poor; 2 = poor; 3 = fair; 4 = good; 5 = very good). See methods section 3.2.3.1 in Chapter 3 for the actigraphy procedure used on night 2, 3 and 4. On these nights, only sleep efficiency was used to adjust for time in bed differences.

5.2.6. Muscle Damage Measurements

5.2.6.1. Isometric MVC Test

For the assessment of maximal force from the knee extensors of the dominant leg, testing was performed using a custom-built isometric chair (Figure 5.2). Participants were seated in an upright position with their knee and hip at a 90° angle. Following a warm up protocol, consisting of 4x 50% MVC and 4x 75% MVC, participants completed three isometric MVC of three seconds, which were separated by one min rest. Biofeedback was provided by a graph on a projector screen and standardised verbal encouragement was given for each contraction. MVC was considered the highest of the three attempts and was used for analysis.

5.2.6.2. Perceived Muscle Soreness

A visual analogue scale was used for the measurement of perceived muscle soreness from the knee extensors. The scale ranged from 0 to 10; 0 = not sore and 10 = being very, very sore, and has previously been used by Thompson *et al* (1999). Participants were asked the question "on the scale of 0-10, how sore are your quadriceps muscles?" whilst walking and squatting. The answer given was verbally discussed with the participant and recorded.

Figure 5.2. Custom built chair used to assess maximal force of the knee extensor muscles.

5.2.7. Statistical Analysis

SPSS (v25) was used for data analysis. Data were checked for normality and are presented as mean ± standard deviation. Paired samples T-Tests were performed to compare sleep variables from PSG and wristwatch actigraphy between conditions. The Wilcoxon test was used to assess subjective sleep quality from the Likert scale and perceived muscle soreness in EX. Changes in MVC across time were analysed using a one-way repeated measures ANOVA. Where a significant ANOVA main effect was identified, subsequent post-hoc analyses were conducted using the LSD method. Sleep efficiency from wristwatch actigraphy on nights two, three and four was assessed using a two-way (condition x time) repeated measures ANOVA. ES of all significant differences for sleep data were calculated by dividing the difference in group means by the pooled standard deviation (Cohen's d) and was assessed using the following thresholds: <0.20 = trivial effect; 0.20-0.60 = small effect; >.0.60-1.20 = moderate effect; >1.20-2.00 = large effect; >2.00-4.00 = very large; >4.00 = extremely large (Hopkins *et al*, 2009). Statistical significance was set at level $P<0.05$.

5.3. Results

5.3.1. PSG, Actigraphy and Subjective Sleep Quality

Sleep variables from polysomnography the night immediately after EX and CON are shown in Table 5.1. Time in bed was not different between conditions. The percentage time spent in NREM stage 1 was lower after EX (4.1±2.9 %, $p = 0.029$, ES: 0.44 [small effect]) compared to CON (5.7±4.2 %) as well as the absolute time spent in NREM stage 1 (19.4±13.7 vs. 27.4±20.5 min, $p = 0.032$, ES: 0.46 [small effect]). REM latency was not different between conditions, but there was a trend for this to be longer after EX compared to CON (164.1±46.5 vs. 112.9±36.7 min, $p = 0.060$). There was no difference between conditions for total sleep time (min), sleep onset latency stage 1 (min), sleep onset latency stage 2 (min), deep sleep latency (min), time awake (min), NREM stage 2, SWS and REM sleep ($p > 0.05$). There was also no difference between conditions for sleep variables from wristwatch actigraphy or subjective sleep quality ($p > 0.05$) on the night immediately after EX and CON (Table 5.2). On nights two, three and four, there was no condition ($p = 0.901$), time ($p = 0.545$) or interaction ($p = 0.094$) effect for sleep efficiency between conditions (Figure 5.3).

Table 5.1. Sleep variables from polysomnography after EX and CON. * indicates p <0.05 compared to CON (n=7).

	EX	CON
Time in bed (min)	510±0	510±0
Total sleep time (min)	478.6±21.2	480.0±18.4
Sleep onset latency S1 (min)	14.6±13.4	11.1±8.3
Sleep onset latency S2 (min)	16.6±14.0	14.9±7.5
Deep sleep latency (min)	34.8±21.1	31.9±11.4
REM latency (min)	164.1±46.5	112.9±36.7
Time awake (min)	31.4±21.2	29.8±18
NREM stage 1 (min)	19.4±13.7*	27.4±20.5
NREM stage 2 (min)	290.7±40.6	285.7±37.0
SWS (min)	92.6±33.4	86.5±33.9
REM (min)	76.0±22.1	80.4±15.8
NREM stage 1 (%)	4.1±2.9*	5.7±4.2
NREM stage 2 (%)	60.7±7.6	59.5±7.3
SWS (%)	19.3±6.7	18±6.8
REM (%)	15.9±4.7	16.8±3.5

Table 5.2. Sleep variables from wristwatch actigraphy and subjective sleep quality after EX and CON (n=7).

	EX	CON
Total sleep time (min)	478±28	480±28
Sleep onset latency (min)	16±13	13±10
Fragmentation index (%)	16.3±12.6	17.2±11.2
Total activity score (count)	1398±1685	1627±2013
Subjective sleep quality (1-5)	3.43±0.98	3.43±1.13

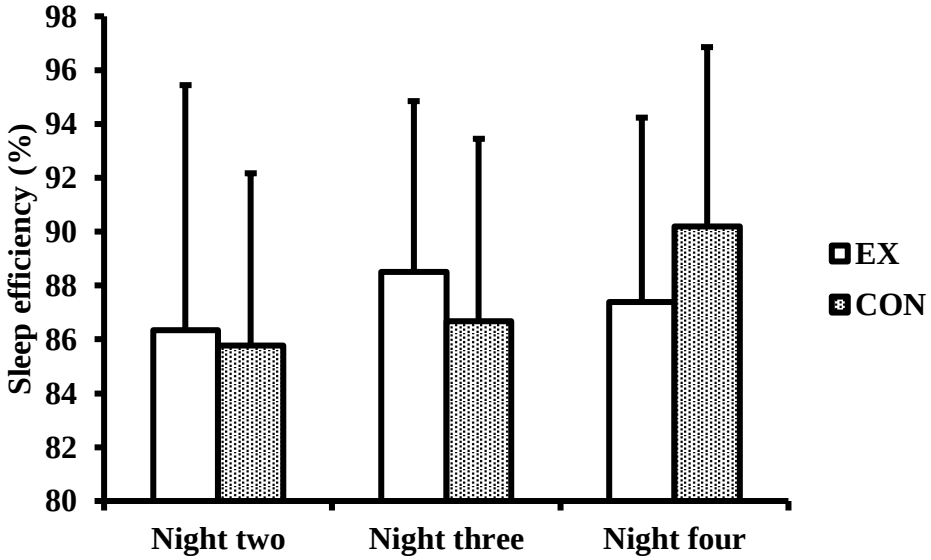

Figure 5.3. Sleep efficiency from wristwatch actigraphy for night two, three and four after EX and CON. White bars represent EX and the white dotted bars indicate the CON (*n*=7).

5.3.2. MVC and Perceived Muscle Soreness

A graphical representation of MVC and perceived muscle soreness is shown in Figure 5.4. There was a significant main effect for the percentage change in MVC (p = 0.001). MVC was reduced at post (-6.5±2.6 %, p = 0.001), 24h post (-7.2±6.5 %, p = 0.027) and 48h post (-4.4±4.4 %, p = 0.039) compared to pre exercise, but recovered at 72h post (2.3±5.9 %, p = 0.409). There was no difference in MVC percentage change at 24h post (-7.2±6.5 %) the morning after the HIIT session and 48h post (-4.4±4.4 %) compared with post after the steady state run (-6.5±2.6 %, $p < 0.05$). Perceived muscle soreness also displayed a significant main effect ($p<0.05$). Muscle soreness was higher at post (2±2 AU, p = 0.026), 24h post (4±2 AU, p = 0.027) and 48h post (4±2 AU, p = 0.034) compared to pre (0±1 AU), but not after 72h post (2±2 AU, p = 0.058). There was no difference in muscle soreness at 24h post (4±2 AU), 48h post (4±2 AU) and 72h post (2±2 AU) compared to post (2±2 AU, $p < 0.05$) after the steady state run.

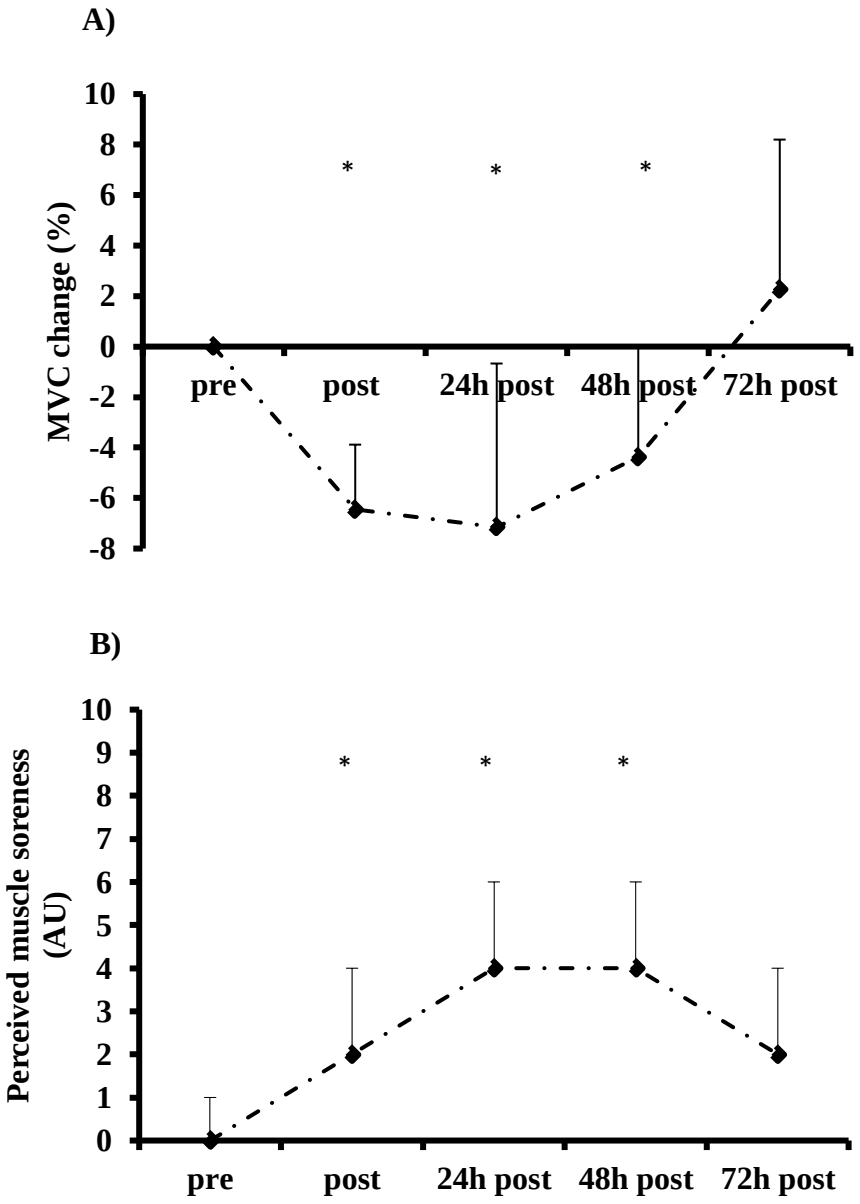

Figure 5.4. Percentage change in MVC a) and perceived muscle soreness b) before and after steady state run (post) and HIIT (24h post). * indicates p <0.05 compared to pre exercise (n=7).

5.4. Discussion

The present study investigated the effect of reduced muscle glycogen stores (<200mmol.kg^{-1}DW) on nocturnal sleep in comparison to a no exercise control. It was hypothesised that reduced glycogen stores would lead to disrupted sleep compared to the CON. The PSG analysis showed there were minimal changes to sleep behaviour; only the amount of NREM stage 1 was lowered with EX on night one. A secondary aim of this study was to examine whether muscle damage from reduced glycogen stores may impact nocturnal sleep on subsequent nights (nights two to four) within the home environment. Indeed, exercise-induced muscle damage was present for 48h post, but this did not subsequently affect actigraphic sleep efficiency on nights two and three.

5.4.1. Effect of Reduced Muscle Glycogen Stores on Nocturnal Sleep

The 'train high, sleep low' paradigm provides training adaptation for the endurance athlete, but its effect on workload during subsequent exercise is debated (Bartlett *et al*, 2015). There is evidence that morning exercise capacity (i.e. time to exhaustion and time trial performance) is impaired after sleeping with low muscle glycogen stores (Hearris *et al*, 2019; Paris *et al*, 2019), though other researchers have shown that exercise training intensity can still be maintained (Lane *et al*, 2015). The current study assessed if sleeping with low glycogen might affect sleep itself. Using PSG, this study observed minimal change in nocturnal sleep after the HIIT session; only stage 1 NREM was lowered the night after EX. This indicates light sleep was shortened following EX compared to the CON condition, however, this did not result in a greater SWS percentage or indeed more time asleep (Table 5.1). Similarly, Louis *et al* (2016) observed a small, significant reduction in sleep efficiency from actigraphy in their sleep low group, suggesting that this strategy had minimal effect on nocturnal sleep. Although the current study did not measure muscle glycogen stores, it is very likely participants initiated sleep with levels

that can be deemed low, as Bartlett and colleagues (2013) showed muscle glycogen levels were below <200mmol.kg^{-1}DW using a similar training protocol. Thus, the 'train high, sleep low' strategy may provide desired training adaptation without causing sleep disruption within endurance athletes. Furthermore, it seems sleeping with low glycogen may not explain the reduction in sleep efficiency within the athletes observed in Chapter 3.

Previously, protein balance has shown to decrease when muscle glycogen stores are low (Howarth *et al,* 2010). As a precaution, participants in this study were provided with a meal high in protein, where the aim was to increase their satiety and maintain protein balance. Unintentionally, this nutritional strategy may have rescued any sleep disturbance the night after exercise, due to the sleep promoting effects of L-tryptophan (Halson, 2008). Participants were provided with tofu, cheese and milk as part of their evening meal, which contain high amounts of L-tryptophan. L-tryptophan is converted into serotonin in the brain, which regulates sleep (Halson, 2013). Dietary intake of L-tryptophan has shown to reduce daytime sleepiness (Markus *et al*, 2005), and improve sleep onset latency, total sleep time and sleep efficiency (Ong *et al,* 2017). CHO before bedtime may also enhance sleep through the tryptophan and serotonin pathway. Afaghi *et al* (2007) reported that sleep onset latency was shortened after a high GI CHO meal. In this way, the benefit from consuming a high CHO diet in the CON, as confirmed by food diaries, may have been negated by the high PRO meal after EX. Whether PRO intake before bedtime offset sleep disturbance cannot be elucidated, as the manipulation of PRO intake was not controlled between the conditions. If protein enhances sleep in this scenario, it could add to other beneficial effects of this strategy relating to AMPK signalling (Taylor *et al,* 2013) and bone resorption (Kerstetter *et al,* 2005). Future research should investigate the effect of protein ingestion on nocturnal sleep following glycogen depleting exercise and its influence on the L-tryptophan and serotonin pathway.

5.4.2. Effect of Exercise-Induced Muscle Damage on Nocturnal Sleep

This study also investigated the effect of exercise-induced muscle damage in relation to sleep. As this type of exercise involved a lowered eccentric element, it could be assumed that the muscle damage from the HIIT would be primarily a result of metabolic perturbations (Tee *et al*, 2007). Reduced muscle glycogen stores are thought to cause metabolic damage through decreased action of ATPase, thus compromising the removal of Ca^{+2}, and leading to metabolic events that cause muscle fibre degeneration (Armstrong *et al*, 1991; Duncan, 1987). The reduction in MVC and increased perceived muscle soreness at 24h post, suggests that metabolic muscle damage was caused from the HIIT session prior to nocturnal sleep. Surprisingly, the HIIT session did not elevate muscle damage following the steady state run in the morning, rather it maintained the level of muscle damage as there was no difference in measures between post and 24h post. This might be explained simply by the timing of the muscle damage measurements; as the measures were taken the morning after HIIT instead of immediately after, it is possible the muscle damage response was dampened when compared to the steady state run. Although the effects of reduced muscle glycogen and increased muscle damage cannot be separated on night one, exercise-induced muscle damage still remained at 48h post. Assuming muscle glycogen stores were recovered on day two, muscle damage alone did not alter sleep efficiency from actigraphy on nights two and three. This concurs with Aloulou *et al* (2019), who found no disruption to nocturnal sleep, despite a reduction in MVC (range = -7.7 to -9.4%), and increased soreness and creatine kinase after simulated trail running. Exercise-induced muscle damage from high intensity training presented in the current study may, therefore, not impact nocturnal sleep in line with previous suggestions (Chennaoui *et al*, 2015; Roberts *et al*, 2018).

Despite the HIIT session appearing to cause muscle damage, the change in MVC was not as much as after performing downhill running exercise (Chen *et al*, 2008; Easthope *et al*, 2010) or repeated sprint exercise (Howatson and Milak, 2009; Leeder *et al*, 2015). For example, Chen and colleagues (2008) reported an approximate 20% decrease in MVC immediately following and 24h post, a 30-min downhill run at 75% VO_{2max}. Additionally, Howatson and Milak (2009) demonstrated a 28% decrease in MVC at 24h post a repeated sprint protocol, consisting of 15 x 30m sprints with a 10m deceleration zone. Such differences in findings are likely due to these activities being predominately eccentric based, as opposed to the HIIT session, which requires a greater emphasis on concentric actions. Previously, muscle strength, creatine kinase and perceived muscle soreness have shown to be substantially more affected following eccentric contractions compared to concentric contractions (Lavender and Nosaka, 2006; Penailillo *et al*, 2013, Willoughby *et al*, 2003). It has been proposed muscle lengthening actions are predisposed to contractile failure because of greater force production (Woledge *et al*, 1985), and decreased motor unit recruitment (Bigland-Richie and Woods, 1976) and cross bridge attachment (McCully and Faulkner, 1986). Due to the lower MVC change after HIIT compared to previous studies (Chen *et al*, 2008; Easthope *et al*, 2010; Howatson and Milak, 2009; Leeder *et al*, 2014), the magnitude of muscle damage could be considered low. For this reason, future research should investigate the impact of other training modalities on nocturnal sleep.

There are some obvious limitations to this study. The sample size was smaller than desired with participants withdrawing due to work and family commitments. Although the HIIT session has previously shown to reduce muscle glycogen stores below 200mmol.kg^{-1}DW, there were no muscle biopsies taken to confirm this had occurred. As there was also no randomisation of the conditions, there may have been a tendency for sleep to be poorer during the CON, despite participants being given a familiarisation night.

5.4.3. Conclusions

In conclusion, reduced muscle glycogen stores (<200mmol.kg^{-1}DW) did not disrupt subsequent night's sleep in recreationally trained males. Exercise-induced muscle damage was also elevated for 48h post, but this did not affect sleep efficiency from wristwatch actigraphy on nights two and three, within the home environment. For those endurance athletes that adopt a 'train high, sleep low' approach to their training, it may be possible to attain adaptation without jeopardising subsequent nocturnal sleep.

Chapter 6

Case Study: Effect of Low Energy Availability on Sleep Duration and Quality in an International Taekwondo Athlete

6.1. Introduction

The findings from Chapter 3 showed that despite similar sleep duration, the sleep efficiency of elite athletes was lower than a non-athlete group, indicating that their sleep quality was inferior. It was also apparent this may be a consequence of either the training load or timing of training. However, Chapter 4 showed that increased cardiac autonomic activity did not affect subsequent nocturnal sleep after high intensity exercise training performed in the early evening. Additionally, Chapter 5 demonstrated that reduced muscle glycogen stores had minimal effect on the next and following night's sleep. Taken together, these results may suggest athletes sleep is disrupted by more chronic exercise exposure, such as that associated with strenuous training and competition.

Whilst it was observed that athletes within Chapter 3 had a higher daily training load than the non-athletes, there was no subsequent information regarding their accompanying energy intake. In combination with low energy intake, athletes with large training demands risk compromising both their health and performance from low energy availability (LEA) (Mountjoy *et al*, 2014). LEA occurs when there is insufficient energy to support normal physiological function, after energy expenditure from exercise training is removed (Logue *et al*, 2018). It is well-established LEA has negative consequences for bone, hormonal balance, metabolism, cardiovascular functioning, immunity and mental health (Mountjoy *et al*, 2018).

In Taekwondo, it is not uncommon for athletes to participate in dieting, extreme weight-control methods and disordered eating to make a certain weight before competition (Sundgot-Bergen and Garthe, 2011). In addition, as there are few weight categories separated by many kilograms and a long wait between the weigh in and competition, it is possible for these athletes to lose a large amount of weight in preparation for competition (Langan-Evans *et al*, 2011; Sundgot-Bergen and Garthe, 2011). Such manipulation of

body mass impacts their energy availability (EA), though this is thought to be masked by acute weight loss strategies that dehydrate and reduce gut content (Burke *et al*, 2018). Due to the need to manipulate their body composition, Taekwondo athletes are at risk of the negative consequences from LEA (Burke *et al*, 2018).

At present, only one study has examined the effect of weight loss on the sleep patterns of combat sport athletes. Dunican *et al* (2019) assigned athletes to either a water loading group (100 $ml.kg^{-1}$) or a control (40 $ml.kg^{-1}$) across 3d, followed by 2d of fluid restriction, in which both groups participated. They showed no disruption to sleep from water loading, suggesting weight loss with or without water loading did not impact sleep, but EA was not reported. Furthermore, due to the intervention length (5d), it cannot be ascertained if effects would differ with gradual weight loss, akin to Taekwondo athletes (da Silva Santos *et al*, 2016).

Therefore, the aim of this case study was to describe the effects of LEA on nocturnal sleep in an International standard Taekwondo athlete making weight for an upcoming competition. Given the negative consequences on other physiological functions, it was hypothesised that LEA from 'making the weight' would disrupt both sleep duration and quality of the athlete.

6.2. Methods

6.2.1. Presentation of the Athlete

The Taekwondo athlete was a Caucasian male (age = 19yrs; height = 1.66m; body weight = 72.5kg) with more than 5 years' experience of competing at international level in the sport. They regularly made weight for the -68kg Featherweight category with total body mass losses of between 4-5kg. However, for their forthcoming competition, they decided to compete in the -63kg Bantamweight category, which required a loss >9.5kg to make the weight-class limit. The competition in question was the 2018 British University Championships, where a semi-final spot would guarantee them National team selection for 2018 European University games. The athlete's aim was to win the competition and achieve a gold medal. In the month prior to the intervention commencing, the athlete was not taking sleep medication, working night-shifts and had not travelled across different time zones. The athlete provided written informed consent to participate in the eight week (WK) intervention and the case study was granted ethical approval by both the Local University research ethics committee and NHS ethics committee.

6.2.2. Study Design

The study intervention manipulated nutrition and training over 8 WK and required minimal participation in typical acute weight loss strategies by the athlete. Sleep patterns were monitored each night via actigraphy and the Consensus Sleep Diary (Carney *et al*, 2012), whilst body composition, hydration status and energy expenditure were taken daily and at set intervals (-8 WK, -4 WK, - 1 WK, PRE-CUT and weigh in [WI]) (Figure 6.1).

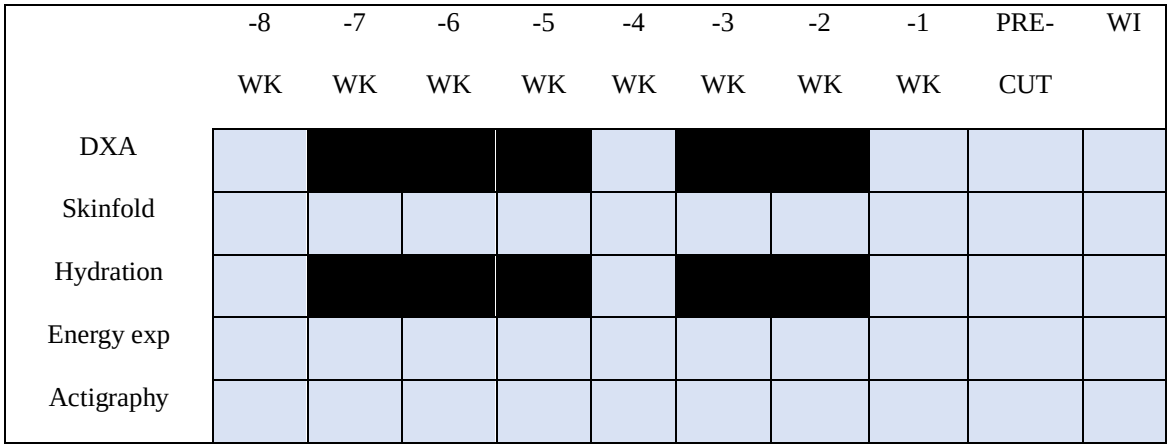

Figure 6.1. Scheduling of physiological measurements taken during the intervention period. Blue denotes measurement was taken and black boxes indicate measurement was not taken. Energy expenditure and actigraphy measurements were taken daily throughout the intervention and DXA, skinfold thickness and hydration were measured once during the week indicated.

6.2.3. Nutrition and Training Intervention

A combination of a calorie restricted diet and exercise training was used to induce weight loss. The athlete ingested an energy intake allowance equivalent to their resting metabolic rate measurement (RMR_{meas}) per day, in line with previous guidelines (Langan-Evans *et al*, 2011). As such, the athletes' average energy intake was 1690 kcal/day^{-1} for the first 7 weeks, which consisted of a low CHO, high PRO and low FAT diet: CHO (3.4g.kgLM^{-1}: 185g/740 kcal/day^{-1}), PRO (2.3g.kgLM^{-1}: 125g/500 kcal/day^{-1}) and FAT (0.9g.kgLM^{-1}: 50g/450 kcal/day^{-1}). Energy intake was exponentially reduced in the final week of the intervention until the WI day with the athlete being restricted to low residue, sodium-based foods to reduce gut content. Similarly, fluid intake during the intervention period was lowered from 2L/day^{-1} to 500 ml in the PRE-CUT stage and no fluid was permitted on the day of the WI to prevent weight gain. There were also no dietary supplements taken by the athlete to aid weight loss and the calorie restricted diet prescribed during the intervention period was fully adhered to by the athlete.

In conjunction, the athlete followed a training programme consisting of five cardio-respiratory sessions (three aerobic and two anaerobic), two strength and conditioning sessions and four Taekwondo specific sessions, which included one sparring session,

totalling 12-15h per week. Steady state sessions were performed fasted in the early morning to cause maximal fat oxidation (Achten and Jeukendrup, 2004) and lasted 45-60min. The two high intensity interval running sessions performed in the morning and evening were designed with the specific competition demands in mind and these differed in work: rest patterns (1:1 min x 10 repetitions at 120% VO_{2peak} and 3:1 min x 6 repetitions at 90% VO_{2peak}). Resistance training performed in weeks 1-4 focused on improving general strength via incremental increases in volume load and intensity. From week 5, specific strength training consisting of varied volume load and a higher average intensity was performed, which was achieved by adding in a combination of maximal strength and speed/reactive strength exercises. During the one-week taper before competition (-1 WK), no resistance training was performed. Figure 6.2. depicts the weekly training schedule during the preparation period.

	MON	**TUE**	**WED**	**THUR**	**FRI**	**SAT**	**SUN**
Early AM	SS run	Interval session			SS run		SS run
Late AM							
Early PM		Weight training		Taekwondo specific	Weight training		
Late PM	Taekwondo specific		Taekwondo specific	Interval session	Taekwondo specific		

Figure 6.2. Training schedule for the athlete during the intervention. SS = Steady state run.

6.2.4. Measurements

6.2.4.1. Anthropometry

Body mass was obtained using a set of digital scales (Seca 702, Seca, Hamberg, Germany) and was recorded to the nearest 0.01kg. Dual x-ray absorptiometry (DXA) (QDR Series Discovery A, Hologic Inc, Massachusetts, USA) was utilised for measures of body fat mass (kg) and LM (kg). The athlete was asked to empty their bladder/bowels and remove jewellery/clothing except for their underwear, before they laid supine in the middle of the bed. The athletes head and legs were then manipulated to ensure straight spinal alignment, hands were placed at the side of their body and feet were turned inwards towards the centre line. Throughout the DXA scan, the athlete was asked to remain in this position. Skinfold ($\sum_{8SKf}$) thickness was also assessed at eight standard anatomical sites (Iliac crest, supra iliac, abdomen, biceps, triceps, subscapular, quadriceps and calf) on the right side of the body using skinfold callipers (Harpenden, Baty Int, West Sussex, UK). The mean of two measurements were taken, unless there was a 5% difference between the first two, in which a third measure was taken before the sum of the skinfolds was recorded. All anthropometric procedures were carried out by a certified sports physiologist.

6.2.4.2. Assessment of Cardio-Respiratory Fitness

To determine $VO_{2peak,}$ an online gas analysis system (CPX, Ultima Series, Medgraphics, Saint Paul, MN, USA) and the same treadmill as in section 4.2.3 within Chapter 4 was used. The only difference was from speeds 6-11km.h^{-1}, stages were completed in 3 min and were followed by 2 min at 12-16km.h^{-1}. In addition, upon 16km.h^{-1}, the incline was increased by 1% each 1 min, until volitional exhaustion despite verbal encouragement.

6.2.4.3. Hydration Status

Urine osmolality was measured using a handheld portable unit (Osmocheck, Vitech Scientific, West Sussex, UK) for the assessment of hydration status. The handheld unit was calibrated by placing a small volume of distilled water onto the unit sample plate and pressing the zero-calibration button. Once the athlete had collected a urine sample using a 5 ml sterilised container (Fisher Scientific, Loughborough, UK), urine was placed onto the sample plate using a disposable pipette and the start button was subsequently pressed to begin the analysis. This process repeated another two times with the mean value recorded as the urine osmolality.

6.2.4.4. Training Monitoring

The internal daily training load of the athlete was monitored using the modified Borg CR10 scale (Foster *et al*, 2001). Within an hour after the cessation of every training session, the athlete was asked to rate their RPE via WhatsApp messenger (WhatsApp, California, USA). The overall session load was then determined by multiplying the training duration by RPE.

6.2.4.5. Resting Metabolic Rate

An indirect calorimetry open hood system (GEM Open Circuit Indirect Calorimeter, GEMNurition Ltd, Warrington, UK) was used to assess RMR. The testing was performed after an overnight fast. Following calibration using O_2, CO_2 and a zero-calibration span gas, the athlete was asked to lay supine on a medical bed. A ventilated hood was then placed over their head and shoulders and they were asked to remain still and breathe normally for a total duration of 30 minutes within a dark, quiet laboratory. The computer connected to the system recorded breath by breath VO_2. The first 10 minutes was discarded to reduce artefact associated with familiarisation and only the last 20 min average was used for analysis (Compher *et al*, 2006). Average breath by breath VO_2 was multiplied by 60 and 24 to the average kcal, calculated from the table in Lusk (1924).

6.2.4.6. Energy Intake and Energy Expenditure

Energy intake and macronutrient intake for each meal was calculated using periodic DXA fat free mass data and Atwater factors (Atwater and Benedict, 1902) for CHO, FAT and PRO. This information was then used by an external food preparation company (Fuel Station Ltd, Liverpool, UK) to provide pre-packaged, ready to eat meals for the athlete each day. Throughout the intervention, fluid intake was monitored using a 1 litre sports bottle with a scale measured to 10 ml, in which the athlete reported the amount ingested via WhatsApp messenger. Exercise energy expenditure from each training session was assessed using an Acitheart 4 unit (Actiheart 4, Cambridge Neurotechnology Ltd, UK) positioned at their chest level. Non-exercise activity thermogenesis was calculated from the Polar V800 HR watch (Polar Electro UK Ltd, Warwick, UK) that the athlete wore during each day of the intervention. The total energy expenditure was calculated from the sum of RMR_{meas}, dietary induced thermogenesis (10% of meal in 6h postprandial period [see Reed and Hill, 1996], non-exercise thermogenesis and exercise energy expenditure.

6.2.4.7. Daily Energy Balance and Energy Availability

Within daily energy balance (WDEB) examines the change in EB over the daily 24h period and this was established using the methodology from Torstveit and colleagues (2018). Additionally, retrospective analysis of EA was calculated hour-by-hour and as a daily total via subtracting exercise energy expenditure from energy intake divided by LM.

6.2.4.8. Sleep Monitoring

Actigraphy and the Consensus Sleep Diary (Carney *et al*, 2012) were used to monitor sleep patterns in the athletes' home. The athlete was provided with an actiwatch (Actiwatch 4, Cambridge Neurotechnology Ltd, UK), which was set to an epoch length of 1-min and at a medium sensitivity. See section 3.2.3.1 in Chapter 3 for procedure.

6.3. Results

6.3.1. Body Mass and Composition Changes During Intervention

At the weigh-in, the athlete recorded an official body mass of 62.7kg and therefore, successfully made the weight for the sanctioned -63kg Bantamweight category competition. Total body mass was reduced from 72.5kg at -8 weeks to 67.4kg at -4 weeks of the intervention, equating to a 5.1kg loss and this was accompanied by a 17.3mm reduction in $\sum_{8SKf}$ thickness. Between -8 weeks and -4 weeks, there was also a reduction of 2.6kg in fat mass though the athletes' LM was maintained (54.5 to 55.0kg), as recorded by DXA measurement. Upon -7d, total body mass had reduced to 66kg, a 1.4kg loss from 67.4kg at -4 weeks, concomitant with a 6.5mm reduction in $\sum_{8SKf}$ thickness and a 1.8kg reduction in fat mass. During the final week before the pre-cut, the athlete had reduced total body mass to 64.5kg, which was accompanied by a 4.1mm reduction in $\sum_{8SKf}$ thickness. At PRE-CUT, LM was reduced by 1.6kg and then by a further 2.1kg at the WI (water loss), whereas fat mass was relatively stable. Figure 6.3 displays the overall change in total body mass, $\sum_{8SKf}$ thickness, DXA fat mass and DXA LM across the intervention.

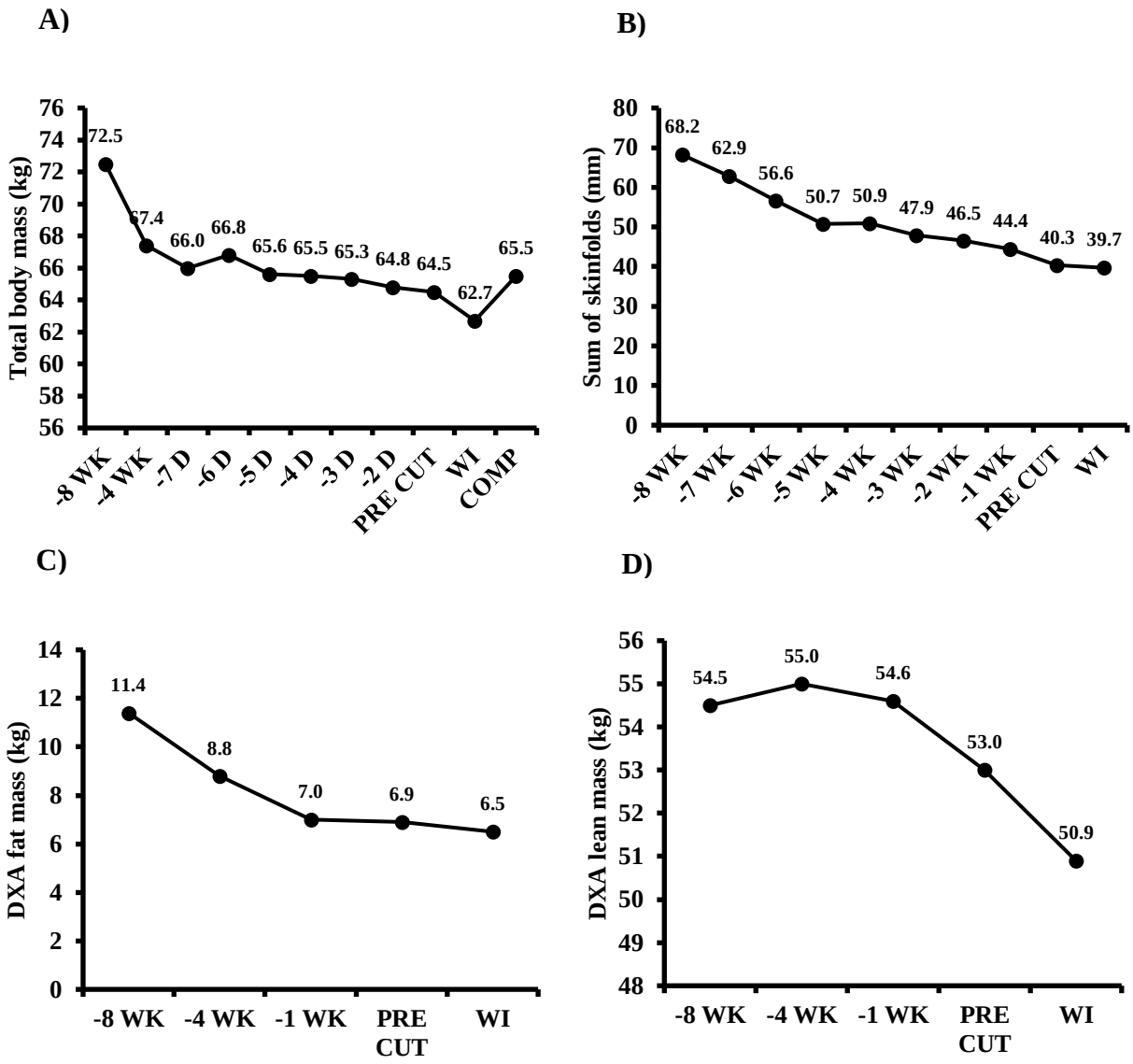

Figure 6.3. Change in body mass (kg) a), $\sum_{8SKf}$ thickness (mm) b), DXA fat mass (kg) c) and DXA LM (kg) d) over the intervention. Black markers and labels represent values at each stage.

6.3.2. Hydration Status from Urine Osmolality During Intervention

Based on the measurement of urine osmolality, the athlete was hypohydrated throughout the intervention, as their values were consistently over 800 mOsmols.kg^{-1} (Figure 6.4). Unsurprisingly, urine osmolality was highest prior to the weigh in, however, the athlete appeared to be around euhydrated values (100-300 mOsmols.kg^{-1}) on competition day.

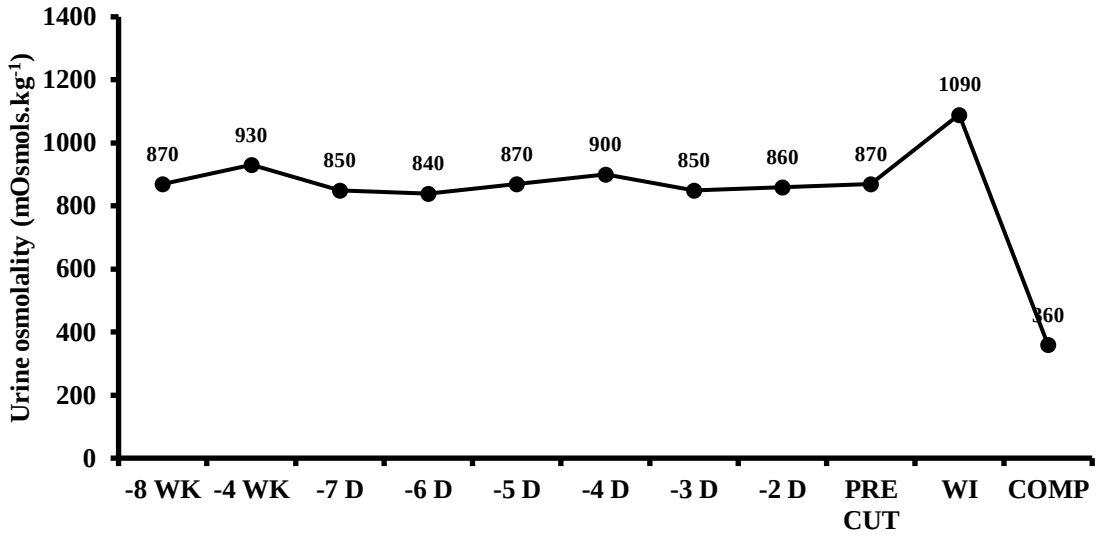

Figure 6.4. Urine osmolality (mOsmols.kg^{-1}) during the intervention period and on the day of competition. Black markers and labels represent urine osmolality values at each stage.

6.3.3. Energy Balance, Energy Intake, Energy Expenditure and Training Load

Figure 6.5 shows a graphical representation of energy intake, energy expenditure, WDEB and training load. During the intervention period, the average daily energy expenditure was higher than daily energy intake, meaning the athlete was consistently within negative energy balance. Due to a weekly average energy intake of 11,900 Kcal and weekly average energy expenditure of 22,000 Kcal, WDEB averaged -13,089 Kcal per week, cumulating in a 104,859 Kcal per week deficit at the end of the intervention. All cardiovascular, strength and sport specific training sessions were completed by the athlete; the training load was slightly increased from -3 weeks to 1 week due to the higher intensity. This was reflected by the greater average exercise energy expenditure during -3 weeks to 1 week compared to -8 weeks to 4 weeks. There was also a reduction in exercise energy expenditure prior to competition due to the taper.

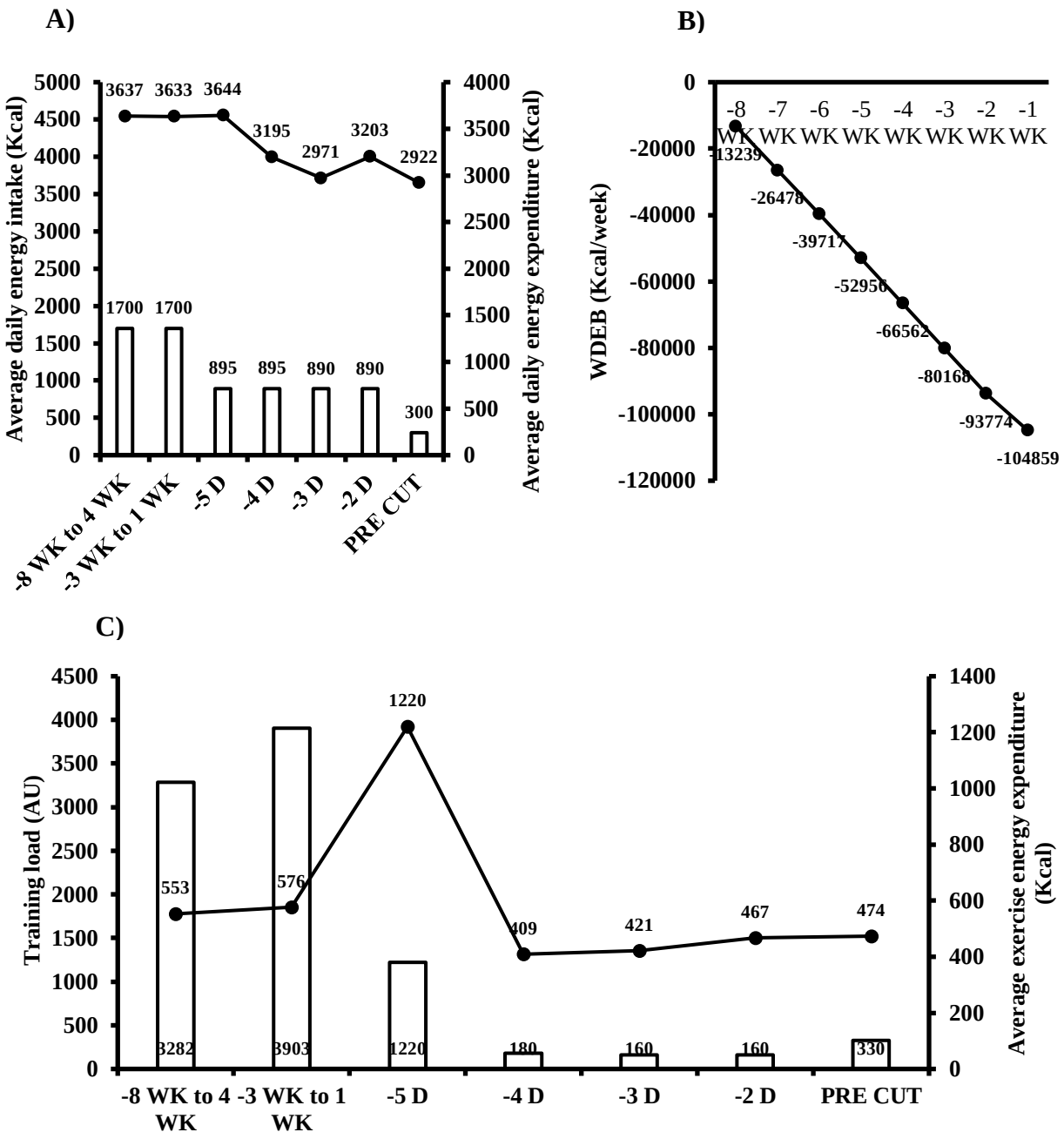

Figure 6.5. Average energy intake and energy expenditure (Kcal) a) WDEB (Kcal) b) and training load (AU) and average exercise energy expenditure (Kcal) c) during the intervention period.

6.3.4. Effect of Energy Availability Status on Sleep Measures

Figure 6.6 displays the sleep variables in relation to changes in EA during the intervention. Across the intervention, the athlete was within values of LEA (<30 Kcal·kg·LM·day^{-1}), with the weekly average EA highest during -8 to 4 weeks and lowest during -3 weeks to PRE-CUT. Day-to-day EA throughout the entire intervention ranged between -7 and 30 kcal·kg·LM·day^{-1}. On average, the athletes' bedtime was 22:02hh:mm and their final time of awakening was 07:35hh:mm during -8 to 4 WK. Similarly, the athletes' average bedtime was 22:09hh:mm and their final time of awakening was 07:22hh:mm during -3 WK through to the PRE-CUT day. Based on the intra-individual variability of athletes in Chapter 3, there was no meaningful change in total sleep time, sleep onset latency or fragmentation index across the intervention. Sleep efficiency and wake after sleep onset increased (+4%) and decreased (-18min) respectively during -3 weeks to PRE-CUT from -8 weeks to 4 weeks. In the days prior to the competition, the athletes' average bedtime was 00:00hh:mm and their average time of final awakening was 06:42hh:mm. No obvious impact of LEA on total sleep time, sleep efficiency, sleep onset latency, wake after sleep onset and fragmentation index was observed, though the total sleep time was altered on days -3 and -5 (Figure 6.7). A weekly average of total sleep time and sleep efficiency across the intervention are presented in Figure 6.8.

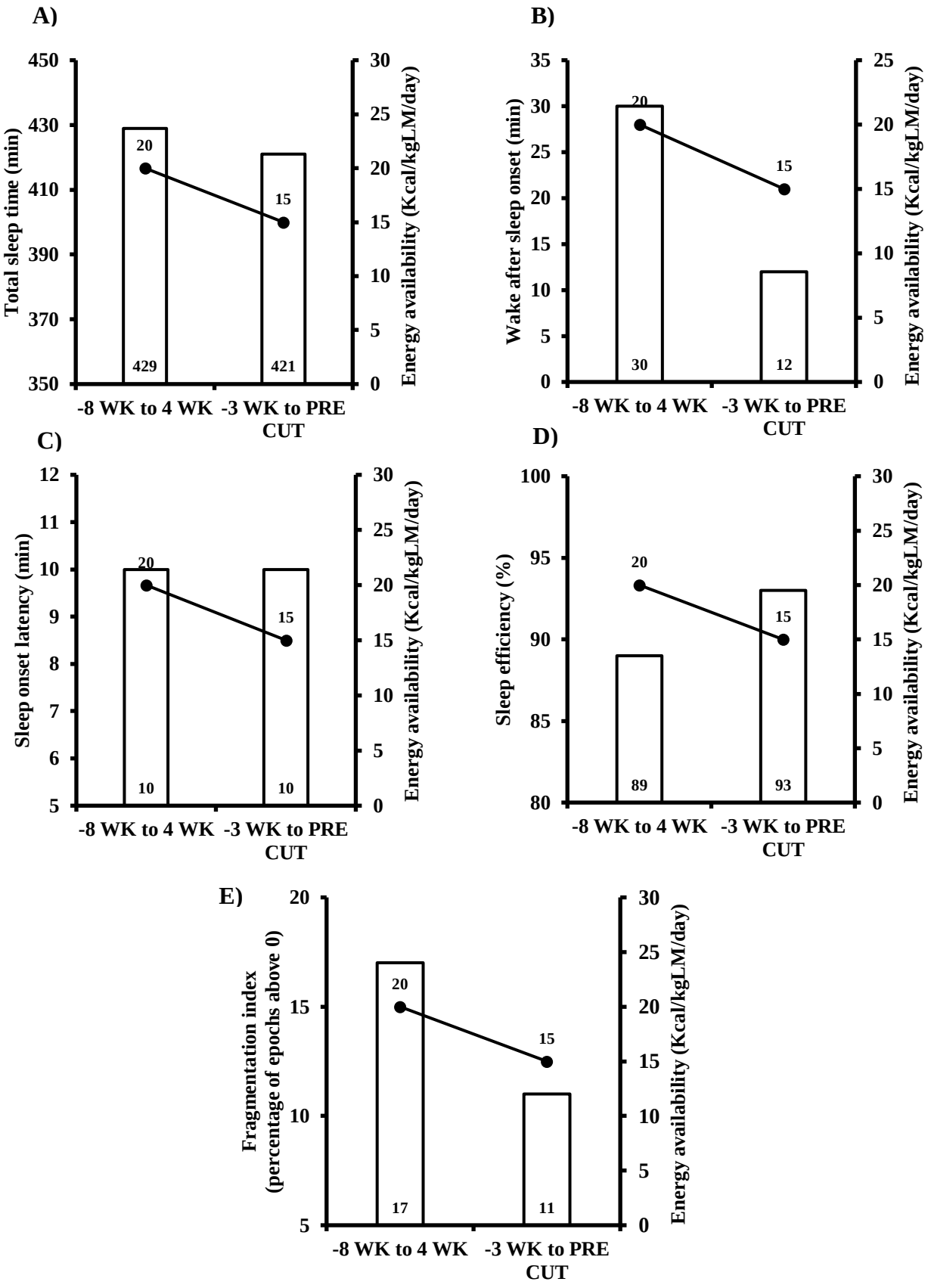

Figure 6.6. Total sleep time (min) a), wake after sleep onset (min) b), sleep onset latency (min) c), sleep efficiency (%) d) and fragmentation index (% epochs > 0) e) relative to EA (Kcal/kgLM/day^{-1}) in the intervention. White bars donate sleep values and black markers indicate EA.

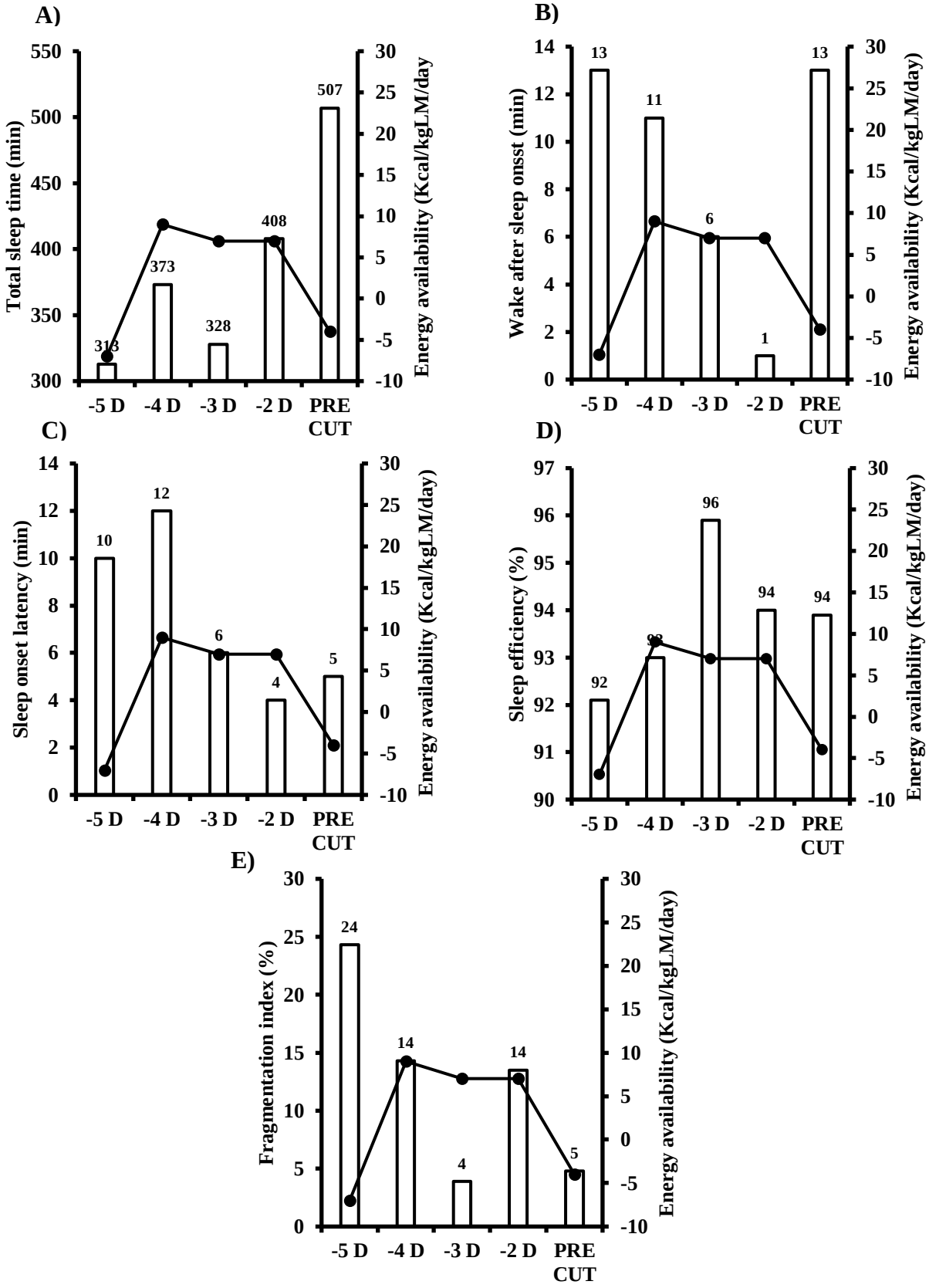

Figure 6.7. Total sleep time (min) a), wake after sleep onset (min) b), sleep onset latency (min) c), sleep efficiency (%) d) and fragmentation index (% epochs > 0) e) relative to EA (Kcal/kgLM/day^{-1}) before competition. White bars donate sleep values and black markers indicate EA.

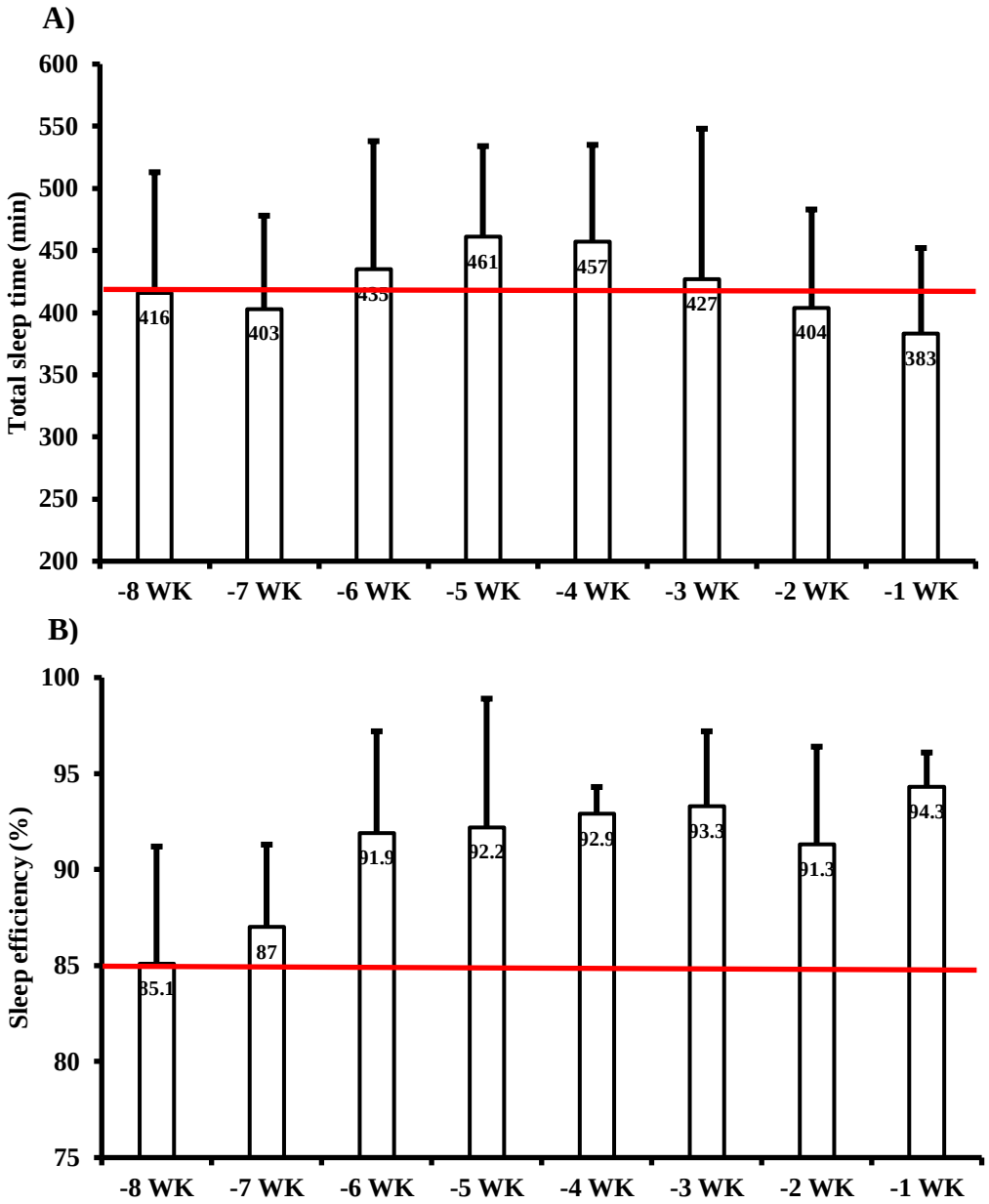

Figure 6.8. Weekly average±SD of total sleep time a) and sleep efficiency b) during the intervention. Red solid line indicates minimum recommended values for each sleep variable.

6.4. Discussion

For the first time, this case study has described the effect of LEA on nocturnal sleep in an international Taekwondo athlete making weight for competition. Both the calorie restricted diet and training programme resulted in the athlete being within values of LEA (<30 Kcal·kg·LM·day^{-1}) throughout the entire intervention. Despite this, there appeared to be no negative effect of LEA on total sleep time, wake after sleep onset, sleep efficiency, sleep onset latency or fragmentation index, as assessed via actigraphy.

As expected, the athlete was within LEA during the entire intervention due to both the greater training demand and calorie restricted diet required to make weight for the -63kg limit. These data are consistent with findings from professional boxers (Morton *et al*, 2010), jockeys (Wilson *et al*, 2013), endurance athletes (Melin *et al*, 2015) and bodybuilders (Fagerberg, 2018), who also restrict energy intake and expend a high degree of energy in training. Previously, it has been shown that LEA has negative health and performance consequences (Mountjoy *et al*, 2014); however, in this case study, there was no critical impact on the sleep of the athlete. This finding concurs with Dunican *et al* (2019), who found no sleep disruption with or without water loading, plus fluid restriction in combat sport athletes. The National Sleep Foundation recommends adults should obtain 7-9h of sleep with at least 85% sleep efficiency to maintain health (Hirshkowitz *et al*, 2015; Ohayon *et al*, 2017). Both recommendations were adhered to by the athlete, albeit sleep duration was more towards the minimum value of 7h when the average of the four-week blocks were considered. Surprisingly, the athletes sleep efficiency was increased during -3 weeks to the PRE-CUT when EA was lowest, which was likely due to the lower wake after sleep onset. This may be explained by increased sleep pressure from molecules such as adenosine, due an energy deficit (Borbely *et al*, 2016). Given the athletes sleep duration and efficiency were within recommended values across the

intervention, LEA from training and calorie restriction may not explain the sleep disturbance of the athletes within Chapter 3.

Interestingly, sleep duration was low in the days prior to competition, with the athlete sometimes sleeping below 6h (-5 d and -3 d), though it is unlikely LEA caused this change. Prior to competition, EA was indeed at its lowest point throughout the intervention due to using acute weight loss strategies (i.e. dehydration and reducing both sodium intake and gut content). Nonetheless, their sleep duration was around recommended values (Hirshkowitz *et al*, 2015) on -2 d and PRE-CUT, suggesting behaviours such as their personal schedule (e.g. choosing to delay bedtime) was responsible for the reduction in sleep duration on the other days. In the days leading into competition, the athlete went to bed on average nearly ~2h later and woke up ~40 min earlier than other days. Roberts *et al* (2019) highlighted that travel and training start times might interfere with athletes' habitual bedtimes and final awakening times, however, this was not the case with this athlete. Their tapered training began later in the day and they did not travel until the day of the weigh in, which may indicate the athlete deliberately chose to delay their bedtime and wake up earlier. The finding of a later bedtime and earlier final awakening in the absence of early/late training is novel, particularly as the athlete was known to sleep within recommended values on other days. To encourage the athlete to maintain or even extend their sleep duration from previous weeks, their coach could deliver sleep education sessions to help maximise recovery and performance.

The athletes' sleep efficiency remained high in the days leading into the competition, suggesting there was no adverse effects of LEA from the acute weight loss on sleep quality. Sleep efficiency during this time was very high relative to elite Rugby Union players (Shearer *et al*, 2015), Rugby League players (Caia *et al*, 2017), endurance cyclists (Lastella *et al*, 2015) and the athletes within Chapter 3. In comparison to these athletes, the Taekwondo athlete initiated sleep much quicker and spent less time awake during the

night, which contributed to the greater sleep efficiency. In fact, the sleep onset latency of the athlete was within normative values (10-20 min) for healthy adults (Boulos *et al*, 2019). These findings may be unique to combat athletes, as they have few distractions during the taper, whereas the sleep of team sport athletes and endurance cyclists may be affected by prior training/competition load or thoughts about upcoming/previous competition (Erlacher *et al*, 2011; Juliff *et al*, 2015). Endurance athletes also compete away for long periods, meaning they often reside in unfamiliar environments, which has been reported to affect sleep (Erlacher *et al*, 2009; Pitchford *et al*, 2017; Thornton *et al*, 2017). The athlete in this case study was sleeping within their home environment up until the night before the competition, which may have facilitated the shorter sleep onset latency. Another possibility is that the shorter sleep onset latency of the Taekwondo athlete is reflective of them using strategies to facilitate sleep. Juliff *et al* (2015) reported that 59% of team sport athletes had no strategy to assist with sleep on the night prior to competition compared with only 33% of individual sport athletes. Nonetheless, although the athletes' sleep efficiency in this case study was not affected by the acute weight loss, further large-scale experimental studies are warranted to confirm these findings.

There are a couple of limitations that are characteristic of a case study. A sample size of one means the findings cannot be generalised. It would have also been novel to measure the athletes sleep using PSG at different stages of the intervention (i.e. start, middle and end). This would have provided vital information about sleep staging, which is lost when utilising field-based measurements such as wristwatch actigraphy, as in this study.

6.4.1. Conclusions

In conclusion, LEA induced via calorie restriction and training did not impact sleep in a Taekwondo athlete, manipulating body mass over 8 weeks to make weight for a

competition. For combat sport athletes, being within LEA to make the limit for a weight category may not be detrimental to either the duration or quality of nocturnal sleep.

Chapter 7

Synthesis of Findings

7.1. Introduction

The aim of this thesis was to investigate the effect of exercise training on nocturnal sleep within athlete populations by completing four objectives, as were stated in section 1.2 of Chapter 1. Accordingly, the main findings from this thesis will be revisited in chronological order to determine 1) whether the aim was sufficiently achieved and 2) the significance of the research. This will then be followed by a general discussion of the findings, limitations section, practical implications for coaches and athletes, before ending with directions for future research.

7.2. Summary of Main Findings

As demonstrated in Figure 7.1 the work from this thesis has added to the current literature and provides additional knowledge for coaches, which they could utilise to plan and organise their athletes training programmes. The main findings were:

1) In Chapter 3, it was demonstrated that athletes from team and individual sports had increased sleep onset latency and reduced sleep efficiency compared to non-athletes. Additionally, athletes adopted a greater training load and trained more in the evening.
2) Chapter 4 showed that high intensity interval exercise in the early evening increased total sleep time compared to no exercise, despite increased cardiac autonomic activity. Low intensity exercise performed at the same time of day also increased total sleep time and this was comparable to the increase following high intensity interval exercise.
3) In Chapter 5, reduced muscle glycogen stores from a HIIT session did not affect sleep on the subsequent night and muscle damage did not alter sleep on following nights.

4) In Chapter 6, there was no obvious effect of LEA on sleep during an 8-week intervention in a Taekwondo athlete who was making weight for competition.

7.3. General Discussion of Main Findings

7.3.1. The Effect of Exercise Intensity on Sleep

According to previous research, sleep duration and/or quality is reduced in response to increased training intensity or volume (Fullagar *et al*, 2019; Hausswirth *et al*, 2014; Schaal *et al*, 2015; Teng *et al*, 2011; Thornton *et al*, 2017). In contrast, others have shown sleep is unaffected (Caia *et al*, 2017; Knufinke *et al*, 2018; Louis *et al*. 2016) or improved with greater training demands (Brand *et al*, 2010a; Taylor *et al*, 1997; Thornton *et al*, 2018; Whitworth-Turner *et al*, 2018). In the current thesis, the findings from both Chapters 4 and 5 suggest that high intensity exercise increases sleep need (i.e. total sleep time) rather than causes disruption. An important consideration between the studies within Chapters 4 and 5, and previous studies that have shown disturbance to sleep is the acute vs. chronic effect of increased training load. The majority of studies demonstrating a disturbance to sleep in response to high training loads have monitored athletes within their habitual environment across a number of weeks or months. For example, Fullagar *et al* (2019) recently found that total sleep time was almost certainly reduced during the in-season compared to the off season in American Collegiate Footballers. Given the studies in Chapters 4 and 5 were acute, this may explain the difference in findings. The findings from the acute studies (Chapters 4 and 5) in this thesis and those of Chapter 6, however, concur with those chronic studies, where a greater training load was shown to be beneficial for sleep (Taylor *et al*, 1997; Thornton *et al*, 2018; Whitworth-Turner *et al*, 2018). Interestingly, a study by Brand and colleagues (2010a) also showed improved sleep efficiency, sleep onset latency and SWS in adolescent footballers (14h vigorous exercise per week) compared to aged matched controls (1.5h of vigorous exercise per

week) over one night, suggesting vigorous exercise leads to beneficial EEG changes irrespective of acute exercise. Nonetheless, it would seem important for future research to conduct chronic exercise training studies that can elucidate the impact of a prolonged training stimulus on nocturnal sleep.

7.3.2. Athlete Population

A moderating variable of the effect of exercise training on sleep may be the population studied (Leeder *et al*, 2012; Whitworth-Turner *et al*, 2017). Leeder *et al* (2012) showed Olympic sport athletes had reduced sleep efficiency compared to non-athletes and suggested this could be explained by the greater training-induced stress imposed on the athletes. Similarly, in Chapter 3 of this thesis, athletes had a lower sleep efficiency than non-athletes, which may have been attributed to differences in training times or a higher daily training load. However, when these training factors were examined within Chapters 4-6, there was no impact of increased cardiac autonomic activity, reduced muscle glycogen or LEA on nocturnal sleep. Collectively, these findings and those within non-athletic individuals (Myllymaki *et al*, 2011; Myllymaki *et al*, 2012; Dworak *et al*, 2012; Flausino *et al*, 2012; Larsen *et al*, 2019), may suggest exercise training has a beneficial effect on sleep regardless of the population studied. The discrepancy between the findings within Chapter 3 and Chapters 4-5 might be due to differences in the study design, which ultimately affects the control of confounder variables. By utilising an observational design in Chapter 3, environmental (i.e. location) and social (i.e. electronic device use) behaviours may have altered sleep (Fullagar *et al*, 2016; Pitchford *et al*, 2017), whereas this was not the case within Chapters 4 and 5, due to the cross over design used. This idea was referred to in Chapter 6, where seemingly the athlete made a choice to delay their bedtime and wake up earlier, thus lowering their sleep duration in the week before competition. Therefore, instead of training load, it is plausible that athletes might have

worse sleep habits than non-athletes before bedtime, which may account for differences in sleeping patterns. Future research should further attempt to separate the effects of exercise training and these behaviours on sleep within athlete populations. This would assist practitioners with developing and implementing strategies to improve sleeping patterns within their athletes.

7.3.3. Measurement of Sleep

The gold standard measurement of sleep is PSG, due to its ability to identify sleep stages based on measures of EEG, EOG and submental EMG (Sadeh, 2015). In the current thesis, this technique was utilised in both Chapters 4 and 5, alongside the use of wristwatch actigraphy. Perhaps not surprisingly, in line with previous research (Rupp and Balkin, 2011), the actiwatch 4 underestimated total sleep time (-20 min) and as a result overestimated total wake time in Chapter 4. However, this was not the case in Chapter 5, where total sleep time was similar to PSG, thus raising a potential issue regarding the outcome data from studies using wristwatch actigraphy. The outcome data from wristwatch actigraphy is primarily dependent upon the level of sensitivity chosen, which refers to the activity count needed to score an individual as awake. Within individuals whom are restless during their sleep (i.e. athletes), a medium to high sensitivity is recommended due the higher activity count (Fuller *et al*, 2017; Sargent *et al*, 2016), whereas a low sensitivity is suggested in individuals who move less during their sleep (i.e. healthy adults) (Tonetti *et al*, 2008). Throughout all the studies (Chapters 3-6) in this thesis, a medium sensitivity (>40 counts scored as wake) was used as participants were either athletes or highly active. Given the actiwatch 4 underestimated total sleep time in Chapter 4, but not within Chapter 5, choosing the sensitivity setting based on the type of participant may not be fully appropriate, potentially due to the interindividual variability in sleep. Participants within Chapters 4 and 5 were of similar fitness and athletic ability,

but those within Chapter 5 may have been less restless during their sleep than those in Chapter 4, hence, a greater sensitivity setting may have been warranted within Chapter 4 (i.e. a high sensitivity). Despite this, in the current thesis, because PSG was used alongside wristwatch actigraphy and subjective sleep quality, this made the study designs within Chapters 4 and 5 more robust, which overall adds to the value of the findings from the studies within these Chapters.

7.4. Limitations

The major strength of the present thesis is the use of PSG and more controlled studies, which builds upon the research examining training load and sleep within observational designs. However, by conducting such studies, there were a number of limitations regarding the sample size, type of participant and ecological validity. The main limitations will now be addressed:

7.4.1. Comparison of Athletes Sleep with Non-Athletes During an In-Season Week

The use of actigraphy has been deemed a valid alternative to PSG, though there are issues surrounding its specificity (i.e. the ability to identify wake epochs), so it cannot be ruled out there may be error associated with the measures of sleep efficiency and sleep onset latency. There were also many confounding factors that were unable to be controlled; such as electronic device use, nutrition and sleep environment, meaning these may have contributed to the poorer sleep patterns within the athletes, rather than training load or schedule alone. Additionally, as this study utilised a longitudinal design, data was collected at different stages of the competitive season, which may inherently cause unwanted variation in sleep patterns.

7.4.2. Effect of Exercise Intensity in the Evening on Subsequent Night's Sleep

As with many sleep laboratory studies, Chapter 4 had a lower sample size than desired, probably due to the intrusive nature of PSG and the difficulty of the HIGH exercise protocol. This ultimately justifies caution surrounding the generalisation of the findings from this study. Given it was a laboratory-controlled study, the ecological validity was somewhat compromised. Certainly, participants sleep may have differed if they had slept within their own bed, and it is acknowledged that some endurance runners may perform exercise closer to their bedtime. Participants in this study were also good sleepers according to data from the PSQI, and it might be that those athletes who are affected by training load are poor sleepers initially.

7.4.3. Effect of Reduced Muscle Glycogen Stores on Subsequent Night's Sleep

Similar to Chapter 4, the sample size within Chapter 5 was lower than desired, as indicated participants who withdrew felt it difficult to fit around their work and family commitments. An added effect of this was that endurance-trained runners were not able to be recruited, so whether the exercise stimulus would have had a different outcome on sleep is unknown. Despite the HIIT session within Chapter 5 has shown to reduce muscle glycogen below 200mmol.kg^{-1}DW, no muscle biopsies were collected to be able to confirm glycogen stores. As there was also no randomisation of conditions within this study, it cannot be ruled out that there was a bias towards sleeping poorer in the CON, despite a familiarisation night.

7.4.4. Case Study: Effect of Low Energy Availability on Sleep Duration and Quality in an International Taekwondo Athlete

An obvious issue with a case study is the sample size and as such, the findings cannot be generalised. It would have been an advantage if the athletes' sleep was measured by PSG at different stages of the intervention (i.e. start, middle and end). This type of analysis

would have provided a comparison with wristwatch actigraphy and information on their sleep staging. Energy intake was controlled, but as a consequence this also lowers the ecological validity.

7.5. Practical Implications

Based on the findings from the four studies undertaken as part of this thesis (Figure 7.1), there are many practical implications that athletes and coaches may consider during training periods.

1) Given the sleep analysis of athletes in Chapter 3, there is a need to implement strategies that improve athletes sleep onset latency and sleep quality. Strategies should target relaxation and reducing core temperature to initiate sleep sooner. This may then improve the recovery of athletes and lead to better performance.
2) Despite the suggestion morning training should be pushed backwards to extend sleep, Chapter 3 also showed this may not necessarily work depending on the athletes' chronotype. Thus, knowledge of the athletes' chronotype (i.e. morning, intermediate or evening) is important when considering moving training sessions.
3) Through the use of PSG in Chapters 4 and 5, it is possible for coaches to schedule training sessions in the afternoon or evening without disruption to athletes' sleep, despite increases in cardiac autonomic activity and reduced glycogen stores.
4) From the findings of Chapter 4, coaches should consider sleep when planning a microcycle for their athletes. For example, where possible, afternoon or evening training sessions could lead to better nocturnal sleep and thus recovery.
5) Due to the differences between PSG and the actiwatch 4 in their outcome data in Chapters 4 and 5, athletes should be cautious when interpreting results from actigraphy. Use of wristwatch actigraphy may lead to under or overestimation of some sleep variables and therefore, cause misinterpretation of the data.

6) As stated in Chapter 6, the variation of sleep duration in athletes prior to competition may be due to their personal schedule (i.e. choice to delay bedtime). Coaches should consider using sleep education and attending to their athletes evening routine, whereby they focus on more appropriate bedtimes.

7.6. Directions for Future Research

The present thesis provides further knowledge of the effect of exercise training on sleep in athlete populations through the four investigations undertaken in Chapters 3-6. Based on the thesis limitations and study findings, a direction for future research is warranted to gain a greater understanding of this research area, beyond that provided in this thesis.

1) Practical strategies to improve sleep in athletes should be considered. Chapter 3 suggested athletes have a lower sleep efficiency than non-athletes, due to increased sleep onset latency. This issue was reflected by the greater IIV of sleep onset latency. Future research should explore ways in which athletes could initiate sleep sooner, as this would help to increase their sleep efficiency to within recommended values. Carbohydrate intake has shown promise for improving sleep onset latency, and its efficacy within real world settings should be investigated by assessing if such effects are maintained within individuals who have not fasted before their evening meal.
2) The timing of training effect on sleep in endurance runners should be revisited. From the findings in Chapter 4, high intensity exercise in the early evening may not disrupt nocturnal sleep in endurance runners, but some runners may train closer to bedtime. Future research should investigate whether exercise training 2h before bedtime may disrupt sleep in endurance runners, and if this is moderated by their chronotype. A between subjects' design could be used to compare sleep after high intensity exercise in individuals that have different chronotypes (i.e. morning, intermediate and evening).
3) Additionally, the mechanisms influencing nocturnal sleep are unclear. Based on the findings from Chapters 4-6, increased cardiac autonomic activity, reduced muscle glycogen, exercise-induced muscle damage and LEA do not impact

subsequent nocturnal sleep. Future research should investigate these perturbations further and understand how exercise may improve sleep, as this could influence the design of training programmes. Identifying markers at the peripheral level from glycogen breakdown after exercise could be studied, and if they link with changes in sleep.

4) The acute effect of exercise on sleep in other athlete groups is unclear. This thesis has focused on the exercise training schedules of individual sport athletes, namely those of endurance runners and Taekwondo athletes, of which differ to other athlete groups. Future research should investigate if team sport athletes sleep is impacted by their training schedule, specifically, whether exercise type and frequency affect sleep. Exercise frequency could be assessed by comparing two sessions a day vs. one session a day on subsequent sleep, which is a scenario faced by many team sport athletes.
5) Chronic exercise training studies would be desirable in the future. The two laboratory studies in this thesis were acute and the case study, though performed over eight weeks, only monitored one athlete, therefore this is not representative of other athletes. Future research could investigate whether exercise training over a longer period is maladaptive toward subsequent night's sleep and other measures of recovery. Whilst these studies would be difficult to perform, exercise training could be supervised within the laboratory and PSG could be conducted at different stages of the training programme.

7.7. Conclusions

Taken together, the findings from the present thesis and the available literature provide evidence that acute exercise training may be beneficial for nocturnal sleep in athletes. Specifically, exercise training, independent of timing and intensity, can increase sleep need (i.e. total sleep time), which may subsequently lead to greater recovery (Davenne, 2009). Although this thesis gives an insight into the effect of chronic exercise training, there are studies with larger sample sizes that indicate long term-training may have a negative effect on sleep. Figure 7.1 provides a theoretical model of the effect of exercise training on sleep in athletes, and this is intended to be used for designing future investigations in this research area.

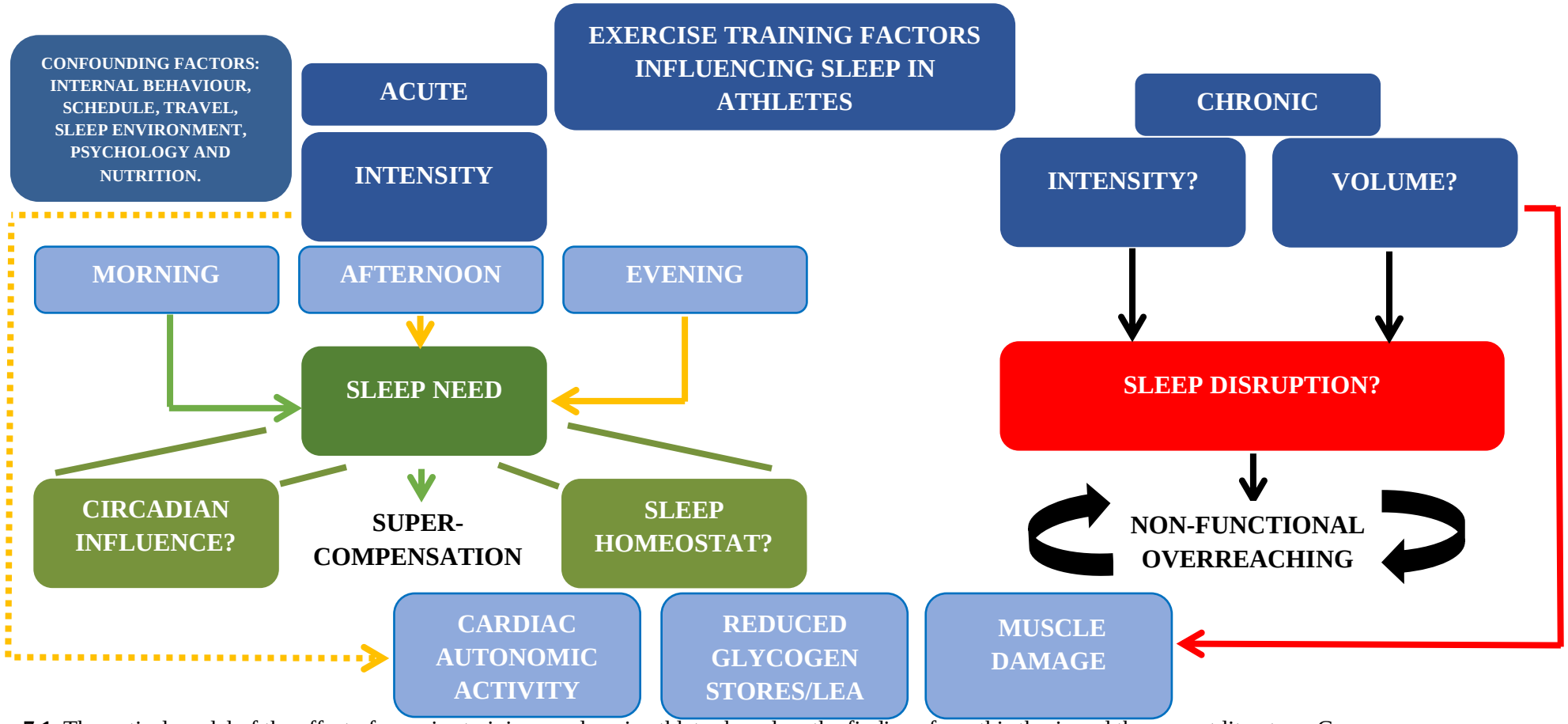

Figure 7.1. Theoretical model of the effect of exercise training on sleep in athletes based on the findings from this thesis and the current literature. Green arrows = positive effect on the outcome variable. Orange arrows = shows findings from this thesis has added to the literature. Dashed lines and arrows = outcome variable is overridden. Solid red line = outcome variable is not overridden and may have negative effect on sleep.

Chapter 8

Appendix

Appendix 1 – The Morningness-Eveningness Questionnaire (Horne and Ostberg, 1976)

MORNINGNESS-EVENINGNESS QUESTIONNAIRE
Self-Assessment Version (MEQ-SA)1

Name: _____ Date: _____

For each question, please select the answer that best describes you by circling the point value that best indicates how you have felt in recent weeks.

1. *Approximately* what time would you get up if you were entirely free to plan your day?
[5] 5:00 AM–6:30 AM *(05:00–06:30 h)*
[4] 6:30 AM–7:45 AM *(06:30–07:45 h)*
[3] 7:45 AM–9:45 AM *(07:45–09:45 h)*
[2] 9:45 AM–11:00 AM *(09:45–11:00 h)*
[1] 11:00 AM–12 noon *(11:00–12:00 h)*

2. *Approximately* what time would you go to bed if you were entirely free to plan your evening?
[5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
[4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
[3] 10:15 PM–12:30 AM *(22:15–00:30 h)*
[2] 12:30 AM–1:45 AM *(00:30–01:45 h)*
[1] 1:45 AM–3:00 AM *(01:45–03:00 h)*

3. If you usually have to get up at a specific time in the morning, how much do you depend on an alarm clock?
[4] Not at all
[3] Slightly
[2] Somewhat
[1] Very much

4. How easy do you find it to get up in the morning (when you are not awakened unexpectedly)?
[1] Very difficult
[2] Somewhat difficult
[3] Fairly easy
[4] Very easy

5. How alert do you feel during the first half hour after you wake up in the morning?
[1] Not at all alert
[2] Slightly alert
[3] Fairly alert
[4] Very alert

6. How hungry do you feel during the first half hour after you wake up?
[1] Not at all hungry
[2] Slightly hungry
[3] Fairly hungry
[4] Very hungry

7. During the first half hour after you wake up in the morning, how do you feel?
[1] Very tired
[2] Fairly tired
[3] Fairly refreshed
[4] Very refreshed

8. If you had no commitments the next day, what time would you go to bed compared to your usual bedtime?
[4] Seldom or never later
[3] Less than 1 hour later
[2] 1-2 hours later
[1] More than 2 hours later

9. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week, and the best time for him is between 7-8 AM *(07-08 h).* Bearing in mind nothing but your own internal "clock," how do you think you would perform?
[4] Would be in good form
[3] Would be in reasonable form
[2] Would find it difficult
[1] Would find it very difficult

10. At *approximately* what time in the evening do you feel tired, and, as a result, in need of
sleep?
[5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
[4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
[3] 10:15 PM–12:45 AM *(22:15–00:45 h)*
[2] 12:45 AM–2:00 AM *(00:45–02:00 h)*
[1] 2:00 AM–3:00 AM *(02:00–03:00 h)*

11. You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last two hours. You are entirely free to plan your day. Considering only your "internal clock," which one of the four testing times would you choose?
[6] 8 AM–10 AM *(08–10 h)*
[4] 11 AM–1 PM *(11–13 h)*
[2] 3 PM–5 PM *(15–17 h)*
[0] 7 PM–9 PM *(19–21 h)*

12. If you got into bed at 11 PM *(23 h),* how tired would you be?
[0] Not at all tired
[2] A little tired
[3] Fairly tired
[5] Very tired

13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which one of the following are you most likely to do?
[4] Will wake up at usual time, but will not fall back asleep
[3] Will wake up at usual time and will doze thereafter
[2] Will wake up at usual time, but will fall asleep again

[1] Will not wake up until later than usual

14. One night you have to remain awake between 4-6 AM *(04-06 h)* in order to carry out a
night watch. You have no time commitments the next day. Which one of the alternatives would suit you best?
[1] Would not go to bed until the watch is over
[2] Would take a nap before and sleep after
[3] Would take a good sleep before and nap after
[4] Would sleep only before the watch

15. You have two hours of hard physical work. You are entirely free to plan your day. Considering only your internal "clock," which of the following times would you choose?
[4] 8 AM–10 AM *(08–10 h)*
[3] 11 AM–1 PM *(11–13 h)*
[2] 3 PM–5 PM *(15–17 h)*
[1] 7 PM–9 PM *(19–21 h)*

16. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week. The best time for her is between 10-11 PM *(22-23 h).* Bearing in mind only your internal "clock," how well do you think you would perform?
[1] Would be in good form
[2] Would be in reasonable form
[3] Would find it difficult
[4] Would find it very difficult

17. Suppose you can choose your own work hours. Assume that you work a five-hour day
(including breaks), your job is interesting, and you are paid based on your performance. At *approximately* what time would you choose to begin?
[5] 5 hours starting between 4–8 AM *(05–08 h)*
[4] 5 hours starting between 8–9 AM *(08–09 h)*
[3] 5 hours starting between 9 AM–2 PM *(09–14 h)*
[2] 5 hours starting between 2–5 PM *(14–17 h)*
[1] 5 hours starting between 5 PM–4 AM *(17–04 h)*

18. At *approximately* what time of day do you usually feel your best?
[5] 5–8 AM *(05–08 h)*
[4] 8–10 AM *(08–10 h)*
[3] 10 AM–5 PM *(10–17 h)*
[2] 5–10 PM *(17–22 h)*
[1] 10 PM–5 AM *(22–05 h)*

19. One hears about "morning types" and "evening types." Which one of these types do you consider yourself to be?
[6] Definitely a morning type
[4] Rather more a morning type than an evening type
[2] Rather more an evening type than a morning type
[1] Definitely an evening type

_____ Total points for all 19 questions

Appendix 2 – The Composite Morningness Questionnaire (Smith *et al*, 1989)

SECTION 1 - The Composite "Morningness questionnaire". (Barton *et al.*, 1990)

For each of the questions circle the response to the response that best describes you.

		Scores
1. Considering only your own "feeling best" rhythm at what time would you get up if you were entirely free to plan your day?	5.00-6.30 am	5
	6.30-7.45 am	4
	7.45-9.45 am	3
	9.45-11.00 am	2
	11.00-12.00 (noon)	1
2. Considering only your own "feeling best" rhythm, at what time would you go to bed if you were entirely free to plan your evening?	8.00-9.00 pm	5
	9.00-10.15 pm	4
	10.15-12.30 am	3
	12.30-1.45 am	2
	1.45-3.00 am	1
3. Assuming normal circumstance, how easy do you find getting up in the morning?	Not at all easy	1
	Slightly easy	2
	Fairly easy	3
	Very easy	4
4. How alert do you feel during the first half hour after having awakened in the morning?	Not at all alert	1
	Slightly alert	2
	Fairly alert	3
	Very alert	4
5. During the first half hour after having awakened in the morning, how tired do you feel?	Very tired	1
	Fairly tired	2
	Fairly refreshed	3
	Very refreshed	4
6. You have to engage in physical exercise. A friend suggests that you do this one hour twice a week and the best time for him is 7.00-8.00 a.m. Bearing in mind nothing else but your own "feeling best" rhythm, how do you think that you will perform?	Would be in good form	4
	Would be in reasonable form	3
	Would find it difficult	2
	Would find it very difficult	1
7. At what time in the evening do you feel tired and, as a result in need of sleep?	8.00-9.00 pm	5
	9.00-10.15 pm	4
	10.15-12.30 am	3
	12.30-1.45 am	2
	1.45-3.00 am	1
8. You wish to be at your peak performance for a test which you Know will be mentally exhausting and lasting for two hours. You are entirely free to plan your day and considering only your own "feeling best" rhythm. Which one of the four testing times would you choose?	8.00-10.00 am	4
	11.00am-1.00 pm	3
	3.00-5.00 pm	2
	7.00-9.00 pm	1
9. One hears about "morning" and "evening" types of people. Which one of these types do you consider yourself to be? yourself to be?	Definitely a morning type	4
	More a morning type than an evening type	3
	More an evening type than a morning type	2
	Definitely an evening type	1

10. When would you prefer to rise (provided you Have a full day's work – 8 hours) if you were totally free to arrange your time?	Before 6.30 am 6.30-7.30 am 7.30-8.30 am 8.30 am or later	4 3 2 1
11. If you always had to rise at 6.00 am what do you think it would be like?	Very difficult and unpleasant Rather difficult and unpleasant A little unpleasant but no - great problem Easy and not unpleasant	1 2 3 4
12. How long does it usually take before you "recover your senses" in the morning after rising from a night's sleep?	0-10 minutes 11-20 minutes 21-40 minutes More than 40 minutes	4 3 2 1
13. Please indicate to what extent you are a morning or evening active individual?	Pronounced morning active (morning Alert and evening tried) To some extent, morning active To some extent, evening active Pronounced evening active (morning Tired and evening alert)	4 3 2 1

Scoring

Evening type: 22 and less
Intermediate type: 22-43
Morning type: 44 and above

SECTION 2 – Languidness/vigorous and Flexibility/rigidity

Please work through the questions as quickly as possible. It is your immediate reaction to the questions that we are interested in, rather than a carefully deliberate answer. There are no "right" or "wrong" answers to any of the questions. For each question we simply want you to indicate which of the five alternatives best describes you, or your preferences, by circling the appropriate number.

	Almost Never	Seldom	Some-times	Usually	Almost Always	
1 Do you tend to need more sleep than other people?	**1**	**2**	**3**	**4**	**5**	**L/V**
2. If you are feeling drowsy can you easily overcome it if you have something to do?	**1**	**2**	**3**	**4**	**5**	**F/R**
3. Do you find it fairly easy to get to sleep whenever you want to?	**1**	**2**	**3**	**4**	**5**	**F/R**
4. Can you miss out a night's sleep without too much difficulty?	**1**	**2**	**3**	**4**	**5**	**F/R**
5. Do you find it difficult to "wake-up" properly if you are awoken at an unusual time?	**1**	**2**	**3**	**4**	**5**	**L/V**
6. If you had to do a certain job in the middle of the night do you think that you could do it almost as easily as at a more normal time of day?	**1**	**2**	**3**	**4**	**5**	**F/R**
7. Do you find it easy to "sleep in" in the morning if you got to bed very late do you think you could do it almost as easily as at a more normal time of day?	**1**	**2**	**3**	**4**	**5**	**F/R**

	Almost Never	Seldom	Some-times	Usually	Almost Always	
8. If you go to bed very late do you need to sleep in the following morning?	**1**	**2**	**3**	**4**	**5**	L/V
9. Can you easily keep alert in boring situations?	**1**	**2**	**3**	**4**	**5**	F/R
10. Are you fairly unaware as to what time it is?	**1**	**2**	**3**	**4**	**5**	L/V
11. If you are tired do you have difficulty keeping awake even though you need to?	**1**	**2**	**3**	**4**	**5**	L/V
12. Do you enjoy working at unusual times of day or night?	**1**	**2**	**3**	**4**	**5**	F/R
13. Do you feel sleepy for a while after waking in the morning?	**1**	**2**	**3**	**4**	**5**	L/V
14. Do you get up later than normal when you are on holiday?	**1**	**2**	**3**	**4**	**5**	L/V
15. If you have a lot to do can you stay up late to finish it off without feeling too tired?	**1**	**2**	**3**	**4**	**5**	F/R
16. Does the time of day have a large effect on your mood and abilities?	**1**	**2**	**3**	**4**	**5**	L/V
17. Do you find it as easy to work late at night as earlier in the day?	**1**	**2**	**3**	**4**	**5**	F/R
18. If you have to get up very early one morning do you tend to feel tired all day?	**1**	**2**	**3**	**4**	**5**	L/V
19. Do you “nod-off” if you are listening to, or watching a boring programme?	**1**	**2**	**3**	**4**	**5**	L/V
20. Can you easily go to sleep earlier than normal to “catch-up” on lost sleep e.g. after several late nights?	**1**	**2**	**3**	**4**	**5**	F/R
21. Do you have no strong preference as to when you sleep?	**1**	**2**	**3**	**4**	**5**	F/R
22. Can you manage with only a few hours sleep each night for several days in a row without too much difficulty?	**1**	**2**	**3**	**4**	**5**	F/R
23. Do you find it fairly difficult to overcome tiredness even in a challenging situation?	**1**	**2**	**3**	**4**	**5**	F/R

	Almost Never	Seldom	Some-times	Usually	Almost Always	
24. Would you be just as happy to do something in the middle of the night as during the day?	**1**	**2**	**3**	**4**	**5**	F/R
25. Do you rely on an alarm clock, or someone else, to wake you up in the morning?	**1**	**2**	**3**	**4**	**5**	L/V
26. Do you get to sleep fairly quickly when you have gone to bed earlier than normal?	**1**	**2**	**3**	**4**	**5**	F/R
27. Do you go to parties, or have evenings out with friends if you have to get up early the following morning?	**1**	**2**	**3**	**4**	**5**	F/R
28. Do you need a cup of coffee or tea to wake you up properly after you have been asleep?	**1**	**2**	**3**	**4**	**5**	L/V
29. Are there particular times of day when you Would avoid doing certain jobs if you could?	**1**	**2**	**3**	**4**	**5**	L/V
30. If you could do so, would you rather wait for half-an-hour or so after waking in the morning before eating a large breakfast?	**1**	**2**	**3**	**4**	**5**	L/V

Scoring

Please add up the scores for the questions with F/R at the end, and then the same for those with L/V.

Flexibility/Rigidity:

Scores over 37 and up to 75 indicate more 'Flexibility'.
Scores lower than 37 indicate more 'rigidity'.

Languidness/Vigorous:

Scores over 37 and up to 75 indicate more 'Languidity'.
Scores lower than 37 indicate more 'vigorous'.

Appendix 3 – Pittsburgh Sleep Quality Index (Buysse *et al,* 1989)

The Pittsburgh Sleep Quality Index

Instructions:
The following questions relate to your usual sleep habits during the past month *only*. Your answers should indicate the most accurate reply for the *majority* of days and nights in the past month. Please answer all the questions.

1. During the past month, when have you usually gone to bed at night?

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

3. During the past month, when have you usually got up in the morning?

4. During the past month, how many hours of *actual* sleep did you get at night? (This may be different than the number of hours you spend in bed).

For each of the remaining questions, check the one best response. Please answer *all* questions.

5. During the past month, how often have you had trouble sleeping because you……

(a) Cannot get to sleep within 30 minutes

Not during the past month____	Less than once a week_____	Once or twice a week_____	three or more times a week_____

(b) Wake up in the middle of the night or early morning

Not during the past month____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

(c) Have to get up to use the bathroom

Not during the past month____	Less than once a week_____	Once or twice a week_____	three or more times a week_____

(d) Cannot breathe comfortably

Not during the past month____	Less than once a week_____	Once or twice a week_____	three or more times a week_____

(e) Cough or snore loudly

Not during the past month____	Less than once a week_____	Once or twice a week_____	three or more times a week_____

(f) Feel too cold

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

(g) Feel too hot

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

(h) Had bad dreams

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

(i) Have pain

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

(j) Other reason(s), please describe_____
_____
_____

How often during the past month have you had trouble sleeping because of this?

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

6. During the past month, how would you rate your sleep quality overall?

Very good_____
Fairly good_____
Fairly bad_____
Very bad_____

7. During the past month, how often have you taken medicine (prescribed or " over the counter") to help you sleep?

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month____ Less than once a week_____ Once or twice a week_____ three or more times a week_____

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all_____
Only a very slight problem___
Somewhat of a problem_____
A very big problem_____

Appendix 4 – Training Load Diary

Date							
How many training sessions have you completed? What time did you train?							
What type of training was involved in your session(s)?							
What was the duration of your training session(s)?							
How difficult did you find your training session(s)?							

Appendix 5 – Analogue Scale for Muscle Soreness (Thompson *et al*, 1999)

0 – Not sore

1

2

3

4

5

6

7

8

9

10 – Very, very sore

Chapter 9

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