Sleep, Nutrition and Recovery in Athletes.

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Research undertaken in the Faculty of Health and Life Sciences and in collaboration with the Sport Ireland Institute.

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Abstract

Elite athletes are vulnerable to sleep difficulties due to high training and competition loads, while inadequate sleep has a detrimental impact on both cognitive performance (i.e. alertness, reaction time, memory and decision making) and physical performance as well as injury risk being increased. Adequate sleep can counteract the negative performance, cognitive, immunity, oxidative stress (OS), non-functional overreaching (NFO) and increased pain outcomes that are consequences of sleep debt. Reduced sleep is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis, blunting training adaptations and recovery which may lead to compromised performance. The aim of Chapters 1-3 was to scope the literature to determine a) was there a problem with sleep in athletes, b) is it related to recovery and c) what non-pharmacological options were there to manage both. The research demonstrated a problem with sleep in athletes and a relationship with recovery, it also became clear that Chrononutrition is an area that shows promise, therefore it was necessary to characterise sleep in athletes (Chapter 4), and under specific circumstances (Competition travel – Chapter 5). This allowed the indetification of the specific sleep amd recovery areas of concern in this population with a view to implementing a chrononutritional intervention (Kiwifruit – Chapter 6) to ascertain whether it improved sleep and recovery in general, and specifically in those domains that were most affected in the first two studies.

An investigation of the sleep and recovery practices of athletes, outlined in Chapter 4, demonstrated that 64% of athletes were classified as 'poor sleepers', while 21% reported excessive daytime sleepiness. Total sleep time (TST) was lower in the elite athlete group on both training/competition days and rest days, adding to the evidence that elite athletes are particularly vulnerable to sleep difficulties. Significantly, higher levels of sport-specific recovery were observed in the elite athlete group. Pain was reported by 50% of athletes while anxiety/depression was reported by 34% of athletes. In terms of nutrition, the most consumed supplements were whey protein, caffeine, multivitamins, creatine, fish oil, probiotics and vitamin D, while sub-elite athletes reported drinking more alcohol that the elite athletes.

The results of Chapter 5 indicated the following impact of long-haul eastward travel across 7 time zones on sleep: actigraphy derived time in bed, total sleep time and sleep efficiency, and sleep diary derived time in bed, total sleep time, fatigue going to bed and sleep quality. Each were significantly negatively impacted by long haul travel particulalry on the travel day and the following day i.e. the 48 hours post travel.

Chapter 6 highlighted the positive impact of Kiwifruit consumption (2 kiwifruit 1 hour before bed) on key aspects of sleep and recovery in elite athletes. From baseline to post-intervention there were clinically significant improvements in sleep quality (i.e. improved PSQI global scores and sleep quality component scores) and improvements in recovery stress balance (reduced general stress and sports stress scales). Moreover, the intervention improved sleep as evidenced by significant increases in TST and sleep efficiency % and significant reductions in number of awakenings and wake after sleep onset. The findings broadly suggested that Kiwifruit does impact positively on sleep and recovery in general, and on some but not all of the sleep and recovery doamins highlighted throughout this thesis.

Declaration

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of others.

Any ethical clearance for the research presented in this thesis has been approved. Approval has been sought and granted by the Faculty of Health and Life Sciences Ethics Committee on 21/5/2017.

I declare that the Word Count of this Thesis is 49,332 words

Name: Rónán Doherty

Signature:

Date: 10th October 2021

Abbreviations

ANOVA – analysis of variance

ANS – autonomic nervous system

ATP – adenosine triphosphate

AU – arbitrary units

BMAL1 – brain and muscle ARNT like protein 1

CAR – cortisol awakening response

CAT – catalase

CBTmin – daily minimum core body temperature

CBTmax – daily maximum core body temperature

CDC – Centre for Diseases Control

CF – central fatigue

CLOCK – circadian locomotor output cycles kaput

CNS – central nervous system

cps – cycles per second

CRSWDs – circadian rhythm sleep wake disorders

CVD – cardiovascular disease

DLMO – dim light melatonin onset

DNA – deoxyribonucleic acid

EEG – electroencephalogram

FO – functional overreaching

GAS – general adaptive syndrome

GH – growth hormone

GHRH – growth hormone releasing hormone

GLM – general linear model

GPx – glutathione peroxide

GSH – glutathione

HPA – Hypothalamic-pituitary-adrenal axis

HR – heart rate

HRV – heart rate variability

Hz – hertz

IGF-1 – insulin like growth factor 1

ISCD-3 – International classification of sleep disorders (3rd edition)

LDL – low density lipoprotein

MPS – muscle protein synthesis

MSLT – multiple sleep latency test

NCAA – National Collegiate Athletic Association

NFO – non-functional overreaching

NSAIDs – non steroidal anti-inflammatory drugs

OS – oxidative stress

OSA – obstructive sleep apnea

OTS – overtraining syndrome

Pi – inorganic phosphate

PCV – percent coefficient of variation

PF – peripheral fatigue

PLMD – periodic limb movement disorder

PSG – polysomnography

PVT – psychomotor vigilance test

REM – rapid eye movement

RLS – restless legs syndrome

RPE – rate of perceived exertion

RR – relative risk

SCN – suprachiasmatic nuclei

SOD – superoxide dismutase

SpO_2 – oxygen saturation

TLR-4 – toll-like receptor-4

TNFα – tumour necrosis factor-α

URTI – upper respiratory tract infection

URTS – upper respiratory tract symptoms

UUPS – unexplained underperformance syndrome

VPLO – ventrolateral preoptic neclei

WHO – World Health Organisation

Glossary of terms

Term	Definition
Acrophase	Time of peak hormone concentration.
Antioxidant	Any substance that significantly delays or prevents oxidative damage of a target molecule.
Arousals	An abrupt change of NREM sleep stage or REM towards waking.
Awakenings (#)	Number of awakenings from sleep onset until final waking.
Central disorders of hypersomnolence	Excessive daytime sleepiness that cannot be attributed to another sleep disorder.
Central fatigue	Fatigue originating in the central nervous system i.e. brain and spinal cord.
Chrononutrition	Describes the interaction between food and the circadian clock system and how human's internal clock can be altered by changing the timing of food intake.
Chronotype	Individual circadian rhythmicity that is categorised as follows: morning types, intermediate types, and evening types.
Circadian sleep wake disorders	Chronic sleep-wake disruption caused by an alteration to the endogenous circadian or desychronisation of the circadian rhythm and the sleep-wake schedule, causing sleep-wake disturbance and distress or impairment.
Clock genes	Any of a number of genes that interact with eachother to make up an autoregulatory feedback loop, in whick its activation and repression takes approximately 1 day.
Fatigue	A symptom whereby physical and cognitive function are limited by interactions between perceived fatigability and performamce fatigability. Or
Fatigue (Sports Performance)	An acute decrease in force production, or an inability to regenerate the original force in the presence of an increased perception of effort. Or
Fatigue (Exercise Performance)	An exercise induced reduction in the maximal voluntary force production during muscular contraction and can be attributed to contractile failure, suboptimal motor cortical output (supraspinal fatigue) and/or altered afferent inputs (spinal fatigue).
Febrile response	Elevation in core body temperature which usually occurs as a response to infection or inflammation.
Free radicals	Molecules or any chemical species that have one or more unpaired electron, formed in cells by either losing or gaining one electron.
Health	A state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.
Hormesis	An adaptive response of cells and organisms to a moderate (usually intermittent) stress e.g. exercise.
Insomnia	Difficulty falling asleep, staying asleep, waking too early with daytime symptoms of fatigue, resistance to going to bed and/or difficulty sleeping without intervention.
Intermittent sports	Sports characterised by repeated bouts of high intensity activity interspersed with short periods of active recovery or passive rest.
N1 sleep (%)	Ratio of time spent in N1 to total sleep time.
N2 sleep (%)	Ratio of time spent in N2 to total sleep time.

N3 sleep (%)	Ratio of time spent in N3 to total sleep time.
Obstructive sleep apnea (OSA)	A frequent condition characterised by repeated episodes of partial or complete reduction in breathing activity during sleep.
Other sleep disorders	All sleep disorders that do not meet the criteria for another sleep disorder.
Oxidative stress	An imbalance between oxidants and antioxidants due to increased oxidants, causing a disruption of redox (oxidation-reduction) signalling, control and/or molecullar damage.
Parasomnias	Underisable movements or behaviours that occur during sleep e.g. sleep-walking, sleep-talking, night terrors and REM sleep behaviour disorder.
Percieved fatigability	Sensations that regulate homeostasis (balanced internal state) and the psychological state of the individual.
Performance fatigability	A result of the contractile capabilities of the muscles involved and the capacity of the nervous system to provide activation signals for a given task.
Periodisation	A training plan where peak performance is elicited through potentiation of biomotors and the management of fatigue and accommodation.
Peripheral fatigue	Fatigue at or distal to the neuromusculsr junction.
Phagocytosis	The ingestion of bacteria or other material by phagocytes.
Rate of perceived exertion	The numeric estimate of someone's exercise intensity (e.g. 1-10. 1=Easy-10=Extremely difficult).
REM sleep (%)	Ratio of time spent in REM to total sleep time.
REM sleep density	The cumulated duration of each REM burst divided by the duration of each REM sleep period.
REM sleep latency	The interval between the first epoch of N2 and the first epoch of REM sleep
Sleep continuity	The amount of sleep versus wakefulness within a given sleep period including sleep initiation and sleep maintenance.
Sleep debt	The difference between an individual's sleep requirement and actual sleep duration.
Sleep deprivation	Not getting enough sleep.
Sleep disturbance	Difficulty initiating or maintaining sleep.
Sleep efficency (%)	Ratio of sleep time to time in bed.
Sleep health	A multidimensional pattern of sleep-wakefulness adapted to individual, social and environmental demands, which promotes physical and mental wellbeing.
Sleep inertia	Severe subjective feeling of sleepiness upon waking.
Sleep maintenance	Staying asleep after sleep onset.
Sleep need	The optimum amount of sleep required to remain alert and function throughout the day.
Sleep onset	Transition from wakefulness to sleep.
Sleep onset latency	Length of time (mins) it takes to transition from wake to sleep.
Sleep related movement disorders	An unpleasant crawling, deep-aching sensation in the legs or arms that is relieved through movement.
Sleep window	Time of day most suited to sleep.
Time in bed (TIB)	The time spent in bed between going to bed and getting up.
Total sleep time	The amount of time spent asleep.
Waking after sleep onset (WASO)	Time (mins) spent awake after sleep onset and before final awakening.

Zeitgebers	(time cues) the cues in our environment that sychronise our internal body clock to the external environment.

Publications and research dissemination

Journal Articles

- Doherty, R., Madigan, S.M., Nevill, A., Warrington, G. and Ellis, J.G. (2021) The sleep and recovery practices of athletes. *Nutrients*, 13(4): 1330. Link
- Doherty, R., Madigan, S., Warrington, G. and Ellis, J. (2019) Sleep and nutrition interactions: Implications for athletes. *Nutrients*, 11(4): 822. Link

Guest-editor

- Nutrients Special Issue: Sleep, Fatigue and Recovery: Current Update on Nutrition and Chrononutrition. Link

Presentations

- 12/8/21 Invited presentation: The sleep and recovery practices of athletes. Sleep4Performance Seminar Series, Perth, Australia (remote).
- 5/5/21 Invited presentation: Tokyo Ready: Sleep and Nutrition. Olympic Federation of Ireland. Irish Olympic team athlete and staff preparation camp, Dublin, Ireland.
- 22/11/19 Invited presentation: Sleep, Nutrition and Athlete Recovery. British Sleep Society, Annual Scientific Conference 2019, Birmingham, England.
- 13/09/19 Invited presentation: Sleep and Recovery: Implications for Athletes. RCSI Faculty of Sports and Exercise Medicine, Annual Scientific Conference 2019, Dublin, Ireland.
- 25/10/18 Invited presentation: HPx High Performance Sport Knowledge Exchange. Sleep, Nutrition and Recovery. How they interact. Sport Ireland Conference Centre, Dublin, Ireland.
- 7/9/18 Conference Presentation: European College of Sports Science Congress 2018. The Sleep and Recovery Practices of Athletes. National Convention Centre, Dublin, Ireland.

Media

- 2/8/21 Invited contribution: Dairy and Sleep from folklore to science. The National Dairy Council, Dairy Nutrition Forum. Link
- 1/7/21 Podcast: Sleep4Performance Season 6 Episoide 11: Sleep and nutrition interactions for athletes. Link
- 12/6/20 Article: RTÉ Brainstorm: How food affects the sleep of athletes and sports people. Link

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Go raibh maith agaibh go léir!

Chapter 1: Introduction

A concise version of Chapters 1-3 has been published:

Doherty, R., Madigan, S., Warrington, G. and Ellis, J. (2019) Sleep and nutrition interactions: Implications for athletes. *Nutrients,* 11(4): 822. Link

1.1 Introduction

The potential role of nutrition and sleep in athlete recovery has recently become a key area of research focus. The concept that nutritional interventions may improve athlete sleep and recovery times via mechanisms such as improving hormonal status, muscle protein synthesis and/or muscle glycogen stores has stimulated increased research in this area. However, the research is in it's infancy and further research is necessary to develop nutrition guidelines, products, protocols and tailored interventions for athletes designed to enhance athlete sleep, recovery and performance. The current thesis sought to add to the existing body of knowledge and understanding of the interaction of sleep, nutrition and athlete recovery. The aims of this thesis were to:

- Outline the current research in relation sleep and nutrition and the implications for athletes (Chapters 1-3).
- Characterise the sleep and recovery practices of elite and sub-elite athletes (Chapter 4).
- Assess the impact of long haul travel on the sleep of elite athletes (Chapter 5).
- Assess the impact of kiwifruit consumption on the sleep and recovery of elite athletes (Chapter 6).

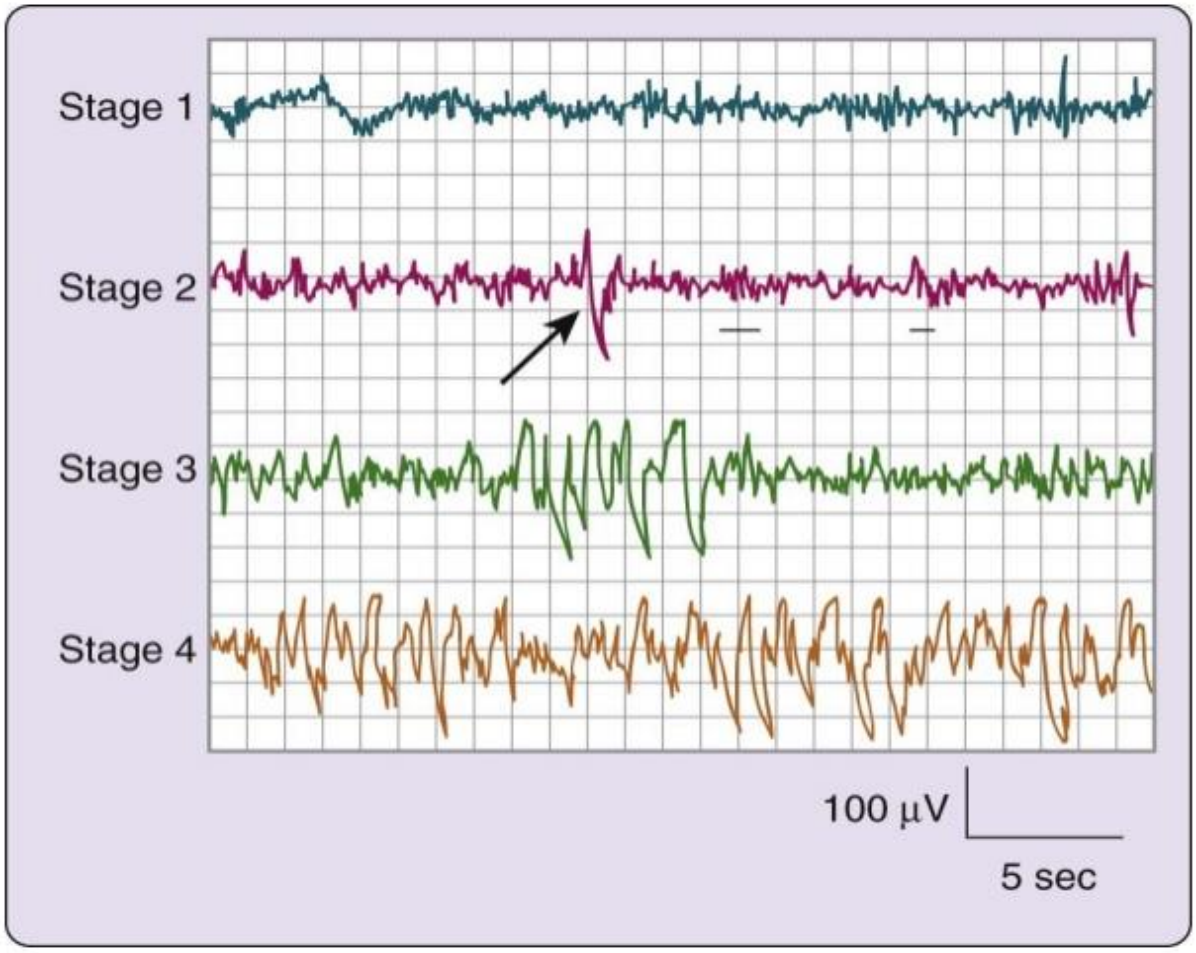
Sleep is defined as a reversible behavioural state whereby a person is perceptually disengaged from, and unresponsive to, their environment (Halson, 2013). Sleep architechture has two basic states based on physiological measurement parameters, non-rapid eye movement sleep (NREM) and rapid eye movement (REM) sleep (Halson, 2013). NREM and REM sleep are as distinct from one another as both are from wakefulness (Carskadon and Dement, 2005). Several methods can be used to assess sleep architecture, the 'gold-standard' is Polysomnography (PSG) where brain activity, eye movements, muscle tone and cardiac activity are measured, providing information on sleep staging (Carskadon and Dement, 2005) [See section 1.4.1 for a discussion of common sleep assessment methods]. Arousal states fall along a continuum from fully awake to deep sleep (i.e. highly synchronous electroencephalogram (EEG) and high arousal thresholds) (Irwin and Opp, 2017). The arousal threshold is the propensity to wake up from sleep, with higher arousal thresholds being less likely to result in a spontaneous awakening (Edwards et al., 2014). In

terms of brain activity, the EEG pattern of NREM sleep is commonly described as synchronous, with characteristic waveforms (sleep spindles, K-complexes and high voltage waves) (Carskadon and Dement, 2005). NREM is usually associated with minimal or fragmented mental activity.

Sleep is distinct from wake (W), when a person is awake EEG shows predominantly alpha (7.5 – 12.5Hz) and beta (12.5 – 30Hz) activity, with beta activity more pronounced during periods of concentration. As a person becomes drowsy, the eyes close and alpha activity increases (Miyazaki et al., 2017). Traditionally, NREM sleep has been subdivided into 4 stages associated with arousal thresholds generally lowest in Stage 1 and highest in Stage 4 (Carskadon and Dement, 2005). Sleep can be characterised based on the wave patterns within the EEG, with stage 1 consisting of high frequency low amplitude waves while stages 3-4 (delta or deep sleep) consisting of low frequency high amplitude waves (Sriraam, 2016). The characteristics of each NREM sleep stage (see Figure 1.1) are as follows:

1. Stage 1: sleep is easily discontinued by internal and external stimuli (e.g. noise, a light touch, etc.) plays a key role in the initial wake to sleep transition and also acts as a transitional stage throughout the night.
2. Stage 2: a more intense stimulus is required to produce an arousal. Indicated by K-complexes (V-shaped wave with and initial negative wave followed by a positive wave indicating a transition into deeper sleep [see Figure 1.1] and/or sleep spindles (separated discharges of oscillatory neural activity) in the EEG. As Stage 2 sleep progresses, high voltage slow wave EEG activity will become more frequent.
3. Stage 3: Characterised by high voltage (75μV) slow wave (2 cycles per second [cps]) activity that is ≥ 20% but < 50% of EEG activity (30 second epochs).
4. Stage 4: identified when high voltage slow wave activity is ≥ 50% of EEG activity (30 second epochs). Stages 3 and 4 combined are often referred to as slow wave sleep, deep sleep or delta sleep.

(Kryger et al., 2010)



*The arrow indicates a K-complex and the underlined areas highlight two sleep spindles.

Figure 1.1: Stages of NREM Sleep (Adapted from: Kryger et al., 2010)

It should be noted that the American Academy of Sleep Medicine (AASM) recommend alternative terminology for sleep staging. Wake is referred to as W, NREM sleep is referred to as N and is divided into three stages: N1 – Stage 1, N2 – Stage 2 and N3 – Slow Wave Sleep or Deep Sleep, i.e., Stage 3 and 4 combined; while REM is referred to as R (Berry et al., 2017; see Table 1.1).

Table 1.1: Comparison of sleep stageing Rechtschaffen and Kales (1968) and the American Academy of Sleep Medicine.

Rechtschaffen and Kales	American Academy of Sleep Medicine
W - Wake	W - Wake
NREM stage 1	N1
NREM stage 2	N2
NREM stage 3 NREM stage 4	N3
REM stage 5	REM

In contrast to NREM, REM sleep is defined by (EEG) activation, muscle atonia and episodic bursts of rapid eye movement (Carskadon and Dement, 2005). The mental activity of REM sleep is associated with dreaming and emotional regulation, while brain mechanisms inhibit spinal motor-neurons limiting movement. Hence, REM sleep has been defined as a highly-activated brain in a paralysed body (Carskadon and Dement, 2005).

1.2 Sleep Regulation

The brain is essentially an electrical system with circuits that switch on and off to promote either wakefulness or sleep. Since the arousal and sleep-promoting systems are mutually inhibitory, a sleep switch or 'flip-flop' model has been proposed (Saper et al., 2005). A flip-flop switch contains mutually inhibitory elements where activity in one of the competing sides shuts down inhibitory inputs from the other side producing two discrete states with sharp transitions (Saper et al., 2001). Activation of arousal systems inhibits sleep active neurons facilitating sleep while activation of sleep-promoting neurons inhibits arousal-related neurons reinforcing consolidated sleep episodes providing a mechanism for stabilisation of sleep and waking states (McGinty and Szymusiak, 2005). During wake, orexin (hypocretin) neurons located in the lateral hypothalamus project to and excite wake promoting neurons in the hind and mid-brain including monoaminergic neurons which release histamine, dopamine, noradrenaline and serotonin; cholinergic neurons that release acetylcholine; and an important group of widely distributed neurons that release glutamate (Foster, 2020). These neurotransmitters promote wakefulness and consciousness within the cortex. Acute activation of the hypothalamic-pituitary-adrenal (HPA) axis (stress axis) also contributes to sleep regulation by promoting wake and inhibiting sleep (Foster, 2020). The stress hormone cortisol is the end product of the HPA axis and the cortisol awakening respone (CAR) refers to the rapid rise in cortisol levels observed immediately following wakening (Elder et al., 2014). During the CAR cortisol levels increase by 38-75% peaking 30-45mins after waking (Elder et al., 2014). Cortisol has a marked circadian rhythm reaching a nadir during deep sleep at night and then increasing in the early hours of the morning before sunrise and awakening, likely reflecting the hormonal preparatory mechanism for waking (Weitzman et al., 1986). During wake, the monoaminergic neurons project to and inhibit the ventrolateral preoptic nuclei (VPLO) (Foster, 2020). While during sleep, circadian and homeostatic sleep drivers activate the VPLO triggering the release of the neurotramsmitters gamma-aminobutyric (GABA) and galanin which inhibit orexin neurons in the lateral hypothalmus and the monoaminergic, cholinergic and glutamatergic

neurons (see Figure 1.2) (Foster, 2020). A subpopulation of interneurons in the cortex project to the cerebral cortex and release GABA, the activation of these interneurons is proportional to the homeostatic sleep drive (Foster, 2020).

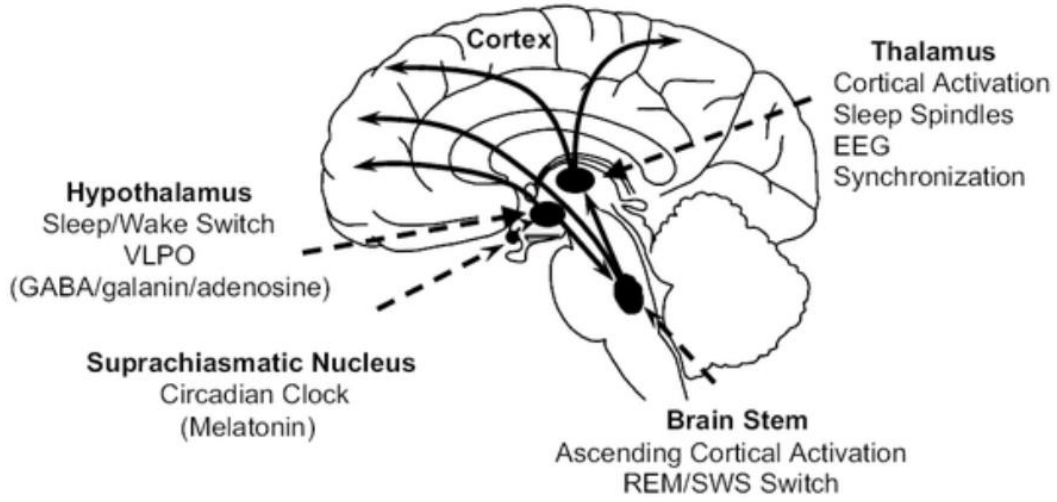


Figure 1.2: Brain regions and sleep/wake regulation (Owens et al, 2013).

1.2.1 Circadian Rhythmns

Circadian rhythms are the oscillation of physiological processes over a 24 hour period, the sleep/wake cycle is the most obvious manifestation of these oscillations (Hill et al, 2020). Circadian oscillators are networks of biochemical feedback loops within the body that generate 24 hour rhythms (Saini et al., 2019). Circadian rhythms must be synchronised to the external environment using signals that provide time of day information (zeitgebers) and the light/dark cycle, thereby acting as a cue in the regulation of the internal body clock. (Foster, 2020). Circadian rhythms are generated endogenously and synchronised to the external 24 hour environment (Foster and Kreitzman, 2004). The circadian rhythm in humans has been estimated in young males (24.18 ± 0.04 hours; percent coefficient of variation (PCV 0.54 %) and older adults (24.18 ± 0.04 hours; PCV 0.58 %), low percentage coefficients of variation and no significant difference between the groups indicated a small range of variability in circadian rhythms (Czeisler et al., 1999).

Circadian rhythms are generated by the suprachiasmatic nuclei (SCN) located in the hypothalamus (Thun et al., 2014). The SCN, a pair of nuclei located above the optic chiasm at the base of the third ventricle (Borbély et al., 2016), are the 'master clock' of the mammalian circadian system (Buhr and Takahashi, 2013). The SCN projects directly to approximately 35 brain regions, particularly those regions of the hypothalamus that control hormone release (Foster, 2020). The SCN regulates the autonomic nervous system (ANS),

which acts as a time stamp for physiological aspects such as hormone sensitivity of target tissues (Kalsbeek et al., 2006). The SCN has melatonin receptor cells, as darkness falls and light stimulus to photosensitive ganglion cells in the retina via the retinohypothalamic tract are suppressed, melatonin is secreted by the pineal gland making the individual sleepy (Venter, 2012). The pineal gland is derived from, and surrounded by, brain tissue and secretes melatonin into the cerebrospinal fluid and blood (Wurtman, 2020). Melatonin activates receptors on brain neurons providing a time signal for various circadian rhythms to promote sleep onset (transition from wakefulness to sleep) and sleep maintenance (staying asleep after sleep onset) during the night (Wurtman, 2020).

The circadian system regulates feelings of sleepiness and wakefulness throughout the day (Samuels et al., 2016), which can both impact on general health and performance. Animal studies have demonstrated that exogenous melatonin and ramelteon (an MT1/MT2 melatonin receptor agonist) function as non-photic entrainers, which phase advance the SCN (Rawashdeh et al., 2011). Inappropriate timing of lifestyle behaviours can cause disruption to the circadian rhythm, resulting in an altered physiological response (e.g. poor sleep). Lifestyle factors (e.g. caffeine consumption, work-rest patterns, alcohol consumption and timing of sleep) can cause alterations in environmental cues which may negatively impact circadian rhythms and in turn result in negative physiological consequences (Golem et al., 2014; Gupta, 2019). For example, increased use of technology and resultant ‘blue’ light exposure adversely impacts circadian rhythmns and is implicated in the development of conditions such as depression, cancer, cardiovascular disease (CVD), and diabetes (Gupta, 2019).

The circadian system is regulated by clock genes via feedback from oscillators, endocrine and neural signals (Khan et al., 2018). Clock genes play a crucial role in sleep regulation and normal physiological function such as hormone secretion, body temperature, appetite and metabolism (Takahashi et al., 2008). The main clock genes are Period (Per 1-3), brain and muscle ARNT like protein 1 (BMAL1), circadian locomotor output cycles kaput (CLOCK), cryptochrome (Cry1 and Cry2) and neuronal PAS domain protein 2 (Npas2) (Albrecht, 2002). At a cellular level, BMAL1 and CLOCK form two different loops for the complete organisation and activation of the circadian clock (Khan et al., 2018). Altered light-dark cycles and lifestyle factors can results in the deactivation or overexpression of core CLOCK genes (e.g. BMAL1 and CLOCK mutations can affect glucose metabolism, behavioural sensitisation to psychostimulants and lipogenesis, and altered sleep patterns) (Khan et al., 2018).

1.2.2 The Two Process Model of Sleep Regulation

Sleep is a dynamic process largely regulated by two factors; the circadian rhythm and the sleep homeostat. Humans' tendency to sleep is governed by the homeostatic process (S) which represents sleep debt and regulates the amount and quality of sleep, in conjunction with the circadian process (C) which regulates the timing of sleep (Borbély, 1980; Daan et al., 1984; Toporikova et al., 2017; Hill et al., 2020). The Two Process Model for Sleep Regulation (see Figure 1.3) was developed to illustrate the interaction of S and C in the timing and duration of sleep (Borbély, 1980; Borbély, 1982; Daan et al., 1984). Sleep pressure accumulates during waking and triggers sleep when it reaches its upper limit (Borbély, 1982; Borbély et al., 2016). S, is an exponential function that decreases during sleep and increases during wakefulness (Toporikova et al., 2017). The underlying mechanisms of S remain unclear, however, it has been demonstrated that adenosine in the basal forebrain is a significant modulator of homeostatic control (Borbély et al., 2016). Adenosine is produced by the breakdown of adeonisine triposphate (ATP). When humans require energy (i.e. when awake, working, training, competing) ATP is broken down and adenosine accumulates in the forebrain (Bjorness and Greene, 2009). High levels of adenosine elicit a sleep promoting effect by inhibiting wake promoting regions or exciting sleep promoting cell groups (Bjorness and Greene, 2009). Caffeine exerts a stimulant effect promoting alertness by blocking adenosine receptors (Foster, 2020). C is an endogenous mechanism, relying on exogenous cues to regulate it to approximately 24 hours. The three main external cues that regulate the circadian system are light and dark, food intake and exercise (Wang, 2017; Johnston, 2014).

While the physiology of S has not been fully determined, a biological marker has been identified as the power of low frequency, delta range (0.5-4Hz) oscillations in EEG during NREM (Toporikova et al., 2017; Daan et al., 1984; Borbély, 1980). Similarly, prolonged waking has been correlated with increasing theta activity (4-7Hz) which is considered a reliable marker of sleep pressure (Vyazovskiy and Tobler, 2005). Core body temperature and melatonin rhythms are markers of C (Rosenthal et al., 2001). The ‘sleep window’ (time of day most suited to sleep) occurs as a result of the combined effects of S and C. Sleep pressure is highest during the first part of the night but increasingly reduced as S dissipates (Foster, 2020). The circadian rhythm ebbs and flows throughout the day but sleepiness is highly likely between 2:00-4:00am and between 1:00-3:00pm (Foster, 2020).

Borbély (1982) originally suggested that S and C exhibited independent influences on sleep, it is now accepted that there is some crosstalk between the processes (Deboer, 2018)

Interaction between S and C may allow individuals to stay alert, even towards the end of the day when sleep pressure has accumulated but not yet reached its peak (Meerlo et al., 2015). A Three Process Model of Sleep Regulation has also been proposed whereby sleepiness and alertness are stimulated by the combined action of a homeostatic process, a circadian process and sleep inertia process, the model has been extended to include sleep onset latency (the length of time of the transition from wakefulness to sleep), sleep length and performance (Åkerstedt and Folkard, 1997).

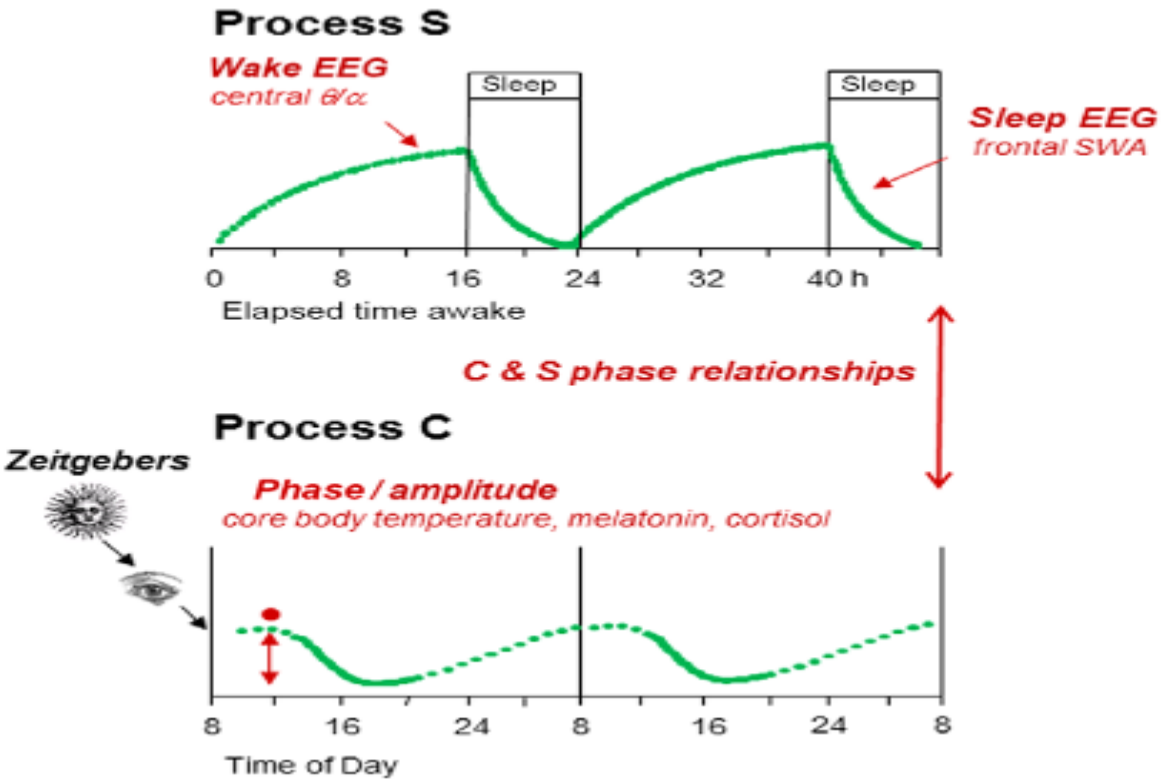


Figure 1.3: The Two Process Model for Sleep Regulation (Adapted from: Borbély et al., 2016).

1.2.3 The Functions of Sleep

The classic view of sleep is as a recovery process (Adam and Oswald, 1977; Hartman, 1973). Sleep is an active anabolic state (i.e. promoting growth, cellular repair and regeneration, energy restoration and stimulating the immune system) (Chokroverty, 2017). Sleep has been hypothesised to have numerous functions including synaptic downscaling, energy restoration and metabolite clearance (Hill et al., 2020). Aspects of cardiovasuclar health i.e. myocardial oxygen consumption, contractility, heart rate recovery, endothelial integrity, angiogenic augmentation and anti-inflammatory and antioxidative functions have all been linked to sleep (Yuksel et al., 2014; Jelic and Le Jemtel, 2008). In contrast, irregular

sleep patterns have been identified as an independent risk factor for CVD in older adults (Huang et al., 2020). Sleep has a vital role in restorative processes and immunity (Ong, 2017). Deficiencies in sleep can adversely affect health and contribute to increased risk of morbidity and all-cause mortality (Ong et al., 2017). Sleep disturbances (difficulty initiating or maintaining sleep) and sleep deprivation (not getting enough sleep) are risk factors for inflammation [Samuels et al., 2016; Hirshkowitz et al., 2015].

Fatigue whether central (originating in the central nervous system (CNS) [brain and spinal cord] or peripheral (at or distal to the neuromuscular junction) is disspiated by the recovery of the energy systems (anabolism), with the majority of this recovery occuring during sleep (Chandrasekaran et al, 2020). It has been hypothesised that sleep, especially slow wave sleep (i.e. N3), is vital for physiological recovery, due to the relationship between growth hormone (GH) release and slow wave sleep (Halson, 2013; Palaniappan and Thenappan, 2015). GH is secreted from the anterior pituitary gland of the brain in the acidophilic, somatotrophic cells and its production is regulated by complex feedback mechanisms that respond to sleep, stress, exercise, nutrition, and GH itself (Bergan-Roller and Sheridan, 2018). GH plays a role in maintaining the homogeneity of tissue and organs during daily living and after injury (Chennaoui et al., 2020). GH synthesis and secretion are controlled by hypothalamic peptides, with stimulation by GH releasing hormone (GHRH) and inhibition by somatostatin (Chennaoui et al., 2020). It must be noted that there are gender differences in hormonal regulation with more pronounced GH production in males coupled with deep sleep, this does not appear to be the case in young women, also the peak levels of plasma GH for females are lower in males but can happen more often through the night (Kimura et al., 2005). Other hormonal regulators of GH release include insulin like growth factor 1 (IGF-1), ghrelin, glucocorticoids, gonadal sex hormones (e.g. testosterone and oestrogen) and thyroid hormone (Chennaoui et al., 2020). GH directly results in increased amino acid uptake, increased ribonucleic acid (RNA) synthesis, increased protein synthesis, increased cartilage synthesis and hypertrophy (Bergan-Roller and Sheridan, 2018). Circulating GH stimulates the synthesis and secretion of IGF-1 from the liver, cell growth and differentiation are stimulated by IGF-1 in a variety of target tissues via IGF-1 receptors (Laviola et al., 2007).

Sleep is essential for the regulation of cerebral glycolsis and reducing lactic acid and subsequent hydrogen accumulation induced central fatigue (Chandrasekaran et al., , 2020). Sleep deprivation impairs insulin sensitivity and glucose homeostasis (Depner et al., 2014). Sleep has a key role in the replenishment of energy stores facilitating improved immune

responses, indeed adaptive immunity peaks during sleep (Schmidt, 2014). Sleep deprivation induced increases in inflammatory markers such as interleukins, cytokines, C-reactive protein (CRP), leukotrine's, GH and tumour-necrosis factor, all of which decrease immune function increasing the risk of illness (Zielinski and Krueger, 2011). Sleep deprivation also decreases T cell count and increases inflammation (Zielinski and Krueger). Sleep also has a role to play in endocrine function as sleep deprivation can contribute to hormonal imbalances (e.g. GH, thyroid, adrenaline and serum cortisol; Meerlo et al., 2008), anabolic hormones (e.g. testosterone, GH and IGF-1) are reduced with inadequate sleep, which reduces muscle protein synthesis (MPS) impacting recovery (Dattilo et al., 2011).

Cognitive recovery also takes place dring sleep and it has been suggested that sleep is essential for skill development, learning, memory, synaptic plasticity and psychological recovery (Ong, 2017; Venter, 2008; Frank, 2006). Sleep deprivation adversely affects mood state (Fullagar et al., 2015) and mood state can be enhanced through sleep extension (> 10hours per night) (Mah et al., 2011). Whilst there is continuing debate in the literature regarding the exact function of sleep, the importance of sleep is clear as it has a role in myriad physiological processes.

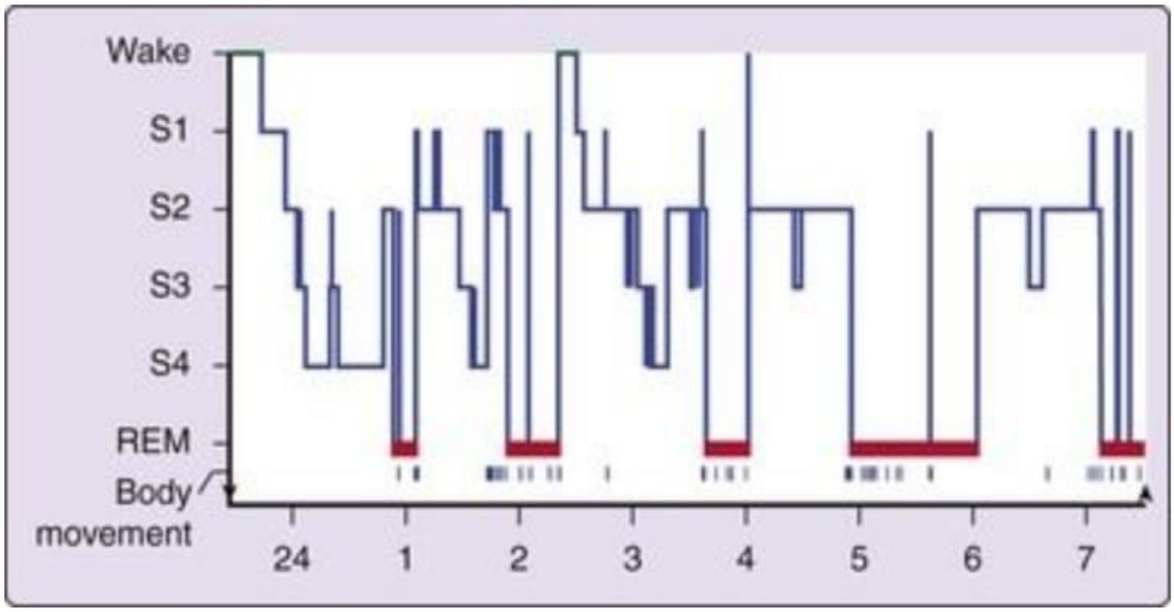


Figure 1.4: The progression of sleep stages across a single night in a healthy young adult (Adapted from: Kryger et al., 2011; p.18).

1.3 Normal Sleep

The most commonly used group to demonstrate normal sleep are healthy young adults (Kryger et al., 2010), however sleep requirements differ across the lifespan (Hirshkowitz et

al., 2015; see Table 1.1). As illustrated in Figure 1.4, in terms of sleep staging, the transition from wakefulness to sleep is via entry into NREM sleep and a later transition to REM sleep (Irwin, 2015; Kryger et al., 2011). REM sleep does not occur until > 90 minutes after the initiation of sleep (Kryger et al., 2011). Following a period of REM sleep a brief arousal/awakening may occur prior to entry into NREM sleep again, over the course of the night four to six cycles of sleep occur, with each cycle lasting approximately 90 - 110 minutes (Irwin, 2015). NREM usually constitutes 75%-80% of sleep while REM makes up the remaining 20%-25% of sleep (Kryger et al., 2011).

1.4 Sleep Duration and Quality

Sleep requirement or sleep need is defined as the optimum amount of sleep required to remain alert and function throughout the day (Chokroverty, 2017). A recent study in elite athletes (n = 175) included a self-report assessment of sleep need, athletes reported an average sleep need of 8.3 ± 0.9 hours (Sargent et al., 2021). The length of sleep depends on a number of factors, not least volitional control (e.g. staying up late, waking by alarm, socialising, etc.), which can make it difficult to characterise a 'normal' sleep pattern due to high individual variation (Kryger et al., 2011). Sleep length is also dictated by genetic determinants (Lassi and Tucci, 2019). Each cell in the body has a molecular clock that dictates the expression of clock controlled-genes which vary depending on cellular subtype, to a periodicity (the time required for one oscillation) or phase (time of awakening relative to the start of the solar day) (Kurien et al., 2019). Sleep length is also determined by processes associsted with circadian rhythms, thus when an individual sleeps has a role in determining how long the individual sleeps (Kryger et al., 2011). Sleep debt is defined as the difference between an individual's sleep requirement and actual sleep duration obtained (Chokroverty, 2017). For sleep to have a restorative effect on the body, it must be of adequate duration and quality, which is dependent on age (Hirshkowitz et al., 2015). Older adults tend to have more fragmented sleep due to factors such as pain and the need to urinate (Hirshkowitz et al., 2015). After waking in the morning, the average individual remains alert until 2pm, when alertness is reduced and lasts approximately 30-60 minutes, then alertness rises again peaking between 6-8pm, when sleepiness begins to increase, facilitating sleep at bedtime (Lastella, 2016). The National Sleep Foundation has produced guidelines regarding sleep duration recommendations (Hirshkowitz et al., 2015). Sleep needs change over the lifespan from adolescents (recommended 8 – 10h), to adults (recommended 7 – 9h), and older adults (7 – 8 h) (see Table 1.1) [Hirshkowitz et al., 2015].

Table 1.2: Sleep duration recommendations.

Population	Recommended (h)	May be appropriate (h)	Not recommended (h)
Adolescents	8 - 10	7 - 11	< 7; > 11
Adults (18 – 64 years)	7 - 9	6 - 11	< 6; > 11
Older adults (≥ 65 years)	7 - 8	5 - 9	< 5; > 9

(Adapted from: Hirshkowitz et al., 2015)

1.4.1 Assessment of Sleep

A common global approach to the assessment of sleep quality is the use of self-report ratings reflecting an individual's satisfaction with their sleep (Ohayon et al., 2017, Buysse, 2014). In an extension of this approach the data gathered can be correlated to enviromental factors, the timing of sleep, physiological data, polysomnography, behaviours, medication use, and sleep disorders to assess accuracy and identify factors that impact sleep (Ohayon et al., 2017). For a more comprehensive approach to assessment, sleep quality can be deconstruced into components i.e. sleep continuity, sleep architechture and naps. Sleep continuity is the amount of sleep versus wakefulness within a given sleep period including sleep initiation and sleep maintenance (Mezick, 2013). The National Sleep Foundation have proposed 12 indicators of sleep quality (see Table 1.2 for definitions) including: 4 sleep continuity variables (sleep latency, awakenings > 5 mins, wake after sleep onset and sleep efficiency), 5 sleep architecture variables (REM sleep, N1 sleep, N2 sleep, N3 sleep and arousals) and 3 nap-related variables (naps per 24 hours, nap duration and days per week with at least one nap) (See Table 1.2) (Ohayon et al., 2017).

Table 1.3: Definition of sleep quality indicators.

Indicator	Definition
Sleep latency	Length of time (mins) it takes to transition from wake to sleep
Awakenings (> 5 mins)	Number per night
Waking after sleep onset	Time (mins) spent awake after sleep onset and before final awakening
Sleep efficency (%)	Ratio of sleep time to time in bed
REM sleep (%)	Ratio of time spent in REM to total sleep time
N1 sleep (%)	Ratio of time spent in N1 to total sleep time
N2 sleep (%)	Ratio of time spent in N2 to total sleep time
N3 sleep (%)	Ratio of time spent in N3 to total sleep time
Arousals	An abrupt change of NREM sleep stage or REM towards waking
Naps per 24 hours	Number of naps per 24 hour period
Nap duration	Mean length of each nap (mins)
Days per week with at least one nap	Number of days in the last week that a nap occurred

(Adapted from: Ohayon et al., 2017)

Sleep can be considered adequate when there is no daytime sleepiness or dysfunction. Sleep is controlled by the circadian clock system, sleep-wake homeostasis and volitional behaviour (Peukhuri et al., 2012). Such analysis of sleepiness can be used to differentiate between arousal states along a continuum (fully awake to deep sleep). PSG records sleep continuity, sleep architecture and REM sleep. Sleep continuity is commonly assessed using sleep diaries and measures include time the subject went to bed, time the subject tried to initiate sleep, the length of time from turning off the lights until sleep onset (sleep onset latency), number and duration of awakenings, the degree of sleep maintenance during the night (sleep efficiency or the ratio of wake time to time in bed; awake time after sleep onset) sleep duration (total sleep time), time the subject woke up, time the subject got out of bed and sleep quality (subjective rating of sleep) (Carney et al., 2012; Irwin, 2015).

Actigraphy is also used to assess sleep, regularly in combination with sleep diaries. Actigraphy involves wearing a small monitor (usually on the non-dominant wrist) which records body movement. High levels of activity are used as a measure of wakefulness and low levels of activity are classified as sleep (Irwin and Opp, 2017). Activity monitors record movement as a function of time (Quante et al., 2015), typically a tri-axial accelerometer is used to determine sleep/wake based on a proprietary algorithm (Kolla et al., 2016). A limitation of actigraphy is that all activity is recorded as wake unless sleep diaries show an attempt to sleep (i.e. lying down trying to sleep) and the activity counts are low enough to indicate the subject is stationary (Halson et al., 2013). Actigraphy has, however, been shown to be reliable and valid in relation to PSG for general measures of sleep (Sedah, 2011; Ancoli-Isreal et al., 2003). Actigraphy (e.g. Actiwatch 2) has demonstrated reliability assessing true sleep (93% sensitivity), ability to assess true wake (63% specificity) and ability to assess both sleep and wake (87% accuracy) (Toon et al., 2016). A similar study compared three different activity monitors to PSG. All monitors displayed good validity for measures of total sleep time (Actiwatch $r = 0.836$; Sleepwatch $r = 0.822$; Actical $r = 0.722$; $P < 0.001$) while two monitors displayed validity for measures of sleep efficiency (Actiwatch $r = 0.651$; Sleepwatch $r = 0.619$; $P<0.001$) (Weiss et al., 2010). In another study the Actiwatch (87.7%) and MyCadian (91.3%) showed excellent agreement with PSG indicating reliability in terms of sleep-wake scoring (Taylor et al., 2017). However, it must be noted both devices tended to underestimate wake epochs and overestimate sleep epochs, compared to PSG. Actigraphy is a valuable clinical and research tool as units are relatively inexpensive,

non-intrusive and does not require a sleep technician. In contrast to PSG, actigraphy offers a cost effective and non-intrusive assessment of sleep which can be implemented in the field.

1.5 Sleep Health

Good sleep health is characterised by satisfaction, appropriate timing, adequate duration, high efficiency, and sustained alertness during waking hours (Buysse, 2014). Sleep health is a concept which involves a holistic view of sleep as opposed to individual symptoms and disorders (Hale et al., 2020). It has been recognised that sleep duration may not be the only sleep characteristic that contributes to health and wellbeing (Matricciani et al., 2018). Sleep health has been defined in an attempt to move from focussing on individual sleep characteristics and health outcomes towards a multidimensional construct, that is a modifiable component of the 24-hour day (Matricciani et al., 2018). Moreover, sleep health is a multidimensional pattern of sleep-wakefulness adapted to individual, social and environmental demands, which promotes physical and mental wellbeing (Buysse, 2014). Sleep has been identified as one of the components of a healthy lifestyle along with diet and exercise (Matricciani et al., 2018).

Sleep can be measured through self-report, behavioural, physiological, circuit, cellular and genetic levels of assessments and each type of analysis can be further charactierised along multiple dimensions i.e. quantity, continuity and timing (Buysse, 2014). Five dimensions of sleep appear to be the most relevant to the definition and measurement of sleep health:

1. Sleep duration: total amount of sleep per 24 hours
2. Sleep continuity or efficiency: ease of falling asleep and returning to sleep
3. Timing: placement of sleep within the 24-hour day
4. Alertness/sleepiness: ability to maintain waking
5. Satisfaction/quality: subjective assessment of ‘good, or ‘poor’ sleep (Buysse, 2014).

These dimensions of sleep health are not unique to any specific sleep disorder and can be assessed by self-report, to provide a comprehensive assessment (Ensrud et al., 2020). Sleep health not only identifies what ‘normal’ sleep is but also quantifies it within a normal range (Buysse, 2014). Each dimension is associated with health outcomes (e.g. mortality, CVD risk, glucose metabolism, obesity and hypertension) and can be expressed as positive or negative (Buysse, 2014). Sleep duration and sleep timing are ‘good’ if they fall within certain ranges but ‘poor’ if they deviate in either direction from these ranges (Buysse, 2014). As with nutrition and physical activity, sleep is a modifiable factor for promoting health

(Benítez et al., 2020). The concept of sleep health is consistent with the World Health Organisation's (WHO) definition of general health i.e. health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 2006).

1.6 Sleep Disorders and Disturbance

Sleep disorders are identified by a wide range of symptoms that impact health and quality of life (Medic et al., 2017), cognitive performance (Yegneswaran and Shapiro, 2007) and physical performance (Erlacher et al., 2011; Fullagar et al., 2015). Over 80 sleep disorders are listed in the third edition of the International Classification of Sleep Disorders (ISCD-3) (Sateia, 2014). The ISCD-3 has reviewed and revised the criteria for each diagnosis for sensitivity, however, there are still considerable uncertainties regarding the classification of sleep disorders particularly regarding the metrics used and the severity of disturbance required to achieve clinical significance (Sateia, 2014). The ICSD-3 includes 7 major categories of sleep disorders: insomnia, sleep-related breathing disorders, central disorders of hypersomnolence, circadian rhythm sleep wake disorders (CRSWDs), sleep related movement disorders, parasomnias and other sleep disorders (Sateia, 2014).

In the general population, the most common sleep disorders are obstructive sleep apnea (OSA), insomnia and restless legs syndrome (RLS) (Adams et al., 2016). Sleep related breathing disorders are characterised by breathing issues during sleep (Thorpy, 2012). OSA is a frequent condition characterised by repeated episodes of partial or complete reduction in breathing activity during sleep (Marra et al., 2019). Insomnia is characterised by difficulty falling asleep, staying asleep, waking too early with daytime symptoms of fatigue, resistance to going to bed and/or difficulty sleeping without intervention occurring at least 3 times per week over a period of three months (Dunican et al., 2019; Riemann et al., 2017). Central disorders of hypersomnolence are typified by excessive daytime sleepiness that cannot be attributed to another sleep disorder (Sateia, 2014). CRSWDs are chronic ($\geq$ 3 months) patterns of sleep-wake disruption caused by an alteration to the endogenous circadian rhythmn or desychronisation of the circadian rhythm and the sleep-wake schedule, causing sleep-wake disturbance and distress or impairment (Sateia, 2014). Sleep related movement disorders may be observed as an unpleasant crawling, deep-aching sensation in the legs or arms that is relieved through movement (Vaughn and O'Neill, 2011). Parasomnias are underisable movements or behaviours that occur during sleep e.g. sleep-walking, sleep-talking, night terrors and REM sleep behaviour disorder (Vaughn and

O'Neill, 2011). The category of 'other sleep disorders' includes all sleep disorders that do not meet the criteria for another sleep disorder classifications (Thorpy, 2012).

Sleep disturbances encompasss disorders of initiating sleep, disorders of excessive somnolence, disorders of the sleep-wake schedule and dysfunctions with sleep, sleep stages or partial arousals (Cormier, 1990), which are not sufficient to meet the diagnostic criteria for a sleep disorder. Sleep disturbances and long or short sleep durations are risk factors for inflammation, linked to all cause mortality and morbididty (Irwin et al., 2016; Vgontzas et al., 2013; Cappuccio et al., 2011; Dew et al., 2003, Kripke et al., 2002; Mallon et al., 2002). Sleep disturbance is also linked to impaired emotional function (Mauss et al., 2013). Short sleep durations adversely affect glucose metabolism and neuroendocrine function that can affect carbohydrate metabolism, appetite, energy intake and protein synthesis (Halson, 2014). Sleep deprivation (< 7 hours) has been identified as a risk factor for stroke (OR 1.45; 95% CI 1.23 – 1.70), heart failure (OR 1.65; 95% CI 1.40 – 1.95) diabetes mellitus (OR 1.35; 95% CI 1.23 – 1.49) and hyperlididemia (OR 1.12; 95% CI 1.04 – 1.22) (Krittanawong et al., 2020). While long sleep duration (> 9 hours) is associated with increased risk of stroke (OR 1.81; 95% CI 1.37 – 2.34) and heart failure (OR 1.47; 95% CI 1.08 – 1.97) (Krittanawong).

1.7 Immunity

Innate immunity acts as the body's first line defence against tissue damage and infection (Medzhitov, 2008). Monocytes (type of white blood cell) which can differentiate into macrophages (recognise, engulf and destroy target cells) and dendritic cells (antigen presenting cells) are the cells involved in the innate immune system. These cells circulate around the body utilising pattern recognition recpetors to detect a variety of pathogens or damaged cells (Irwin and Opp, 2017). Activation of these cells occurs within minutes to hours of recognition of a foreign challenge, initiating a cascade of inflammatory processes in an attempt to curtail infection and promote healing and recovery (Medzhitov, 2008). Microbes or pathogen associated molecular patterns are detected by 'hard-wired' or highly conserved receptors of innate immunity cells, therefore innate immunity relies on a relatively small number of receptors to detect and initiate a response to a wide variety of microbes (Irwin and Opp, 2017). When pattern recognition receptors are activated, an increase in inflammatory activity occurs both locally (i.e. at the site of tissue injury or infection) and systemically (Medzhitov, 2008).

1.7.1 Sleep and Immunity

Sleep has a restorative effect on the immune system and the endocrine system, facilitates the recovery of the nervous and metabolic cost of the waking state and has an integral role in learning, memory and synaptic plasticity (Frank, 2006). In a study by Spiegel et al., (2002) antibody levels (Antibody titers) were found to be decreased (>50%) 10 days after receiving the influenza vaccination in participants who were vaccinated immediately following 6 days of sleep restriction compared to participants who were vaccinated after 6 nights of habitual sleep. However, 3-4 weeks following vaccination there was no difference in antibody levels between the groups. In a similar study, attenuation of the febrile response (elevated body temperature usually a response to infection or inflammation) to an endotoxin challenge (*Escherichia Coli*) was observed in sleep restricted participants, following 10 nights (4 hours), compared to non-restricted controls (8 hours per night) (Balachandran et al., 2002). Those who were exposed to total sleep deprivation demonstrated reductions in circulating levels of GH (Radomski et al., 1992; Seifritz et al., 1995), leptin (Mullington et al., 2003), thyroid axis hormones (Allan and Cziesler, 1994; Gary et al., 1996) and increases in cortisol (Weitzman et al., 1983; Akerstedt et al., 1979), with no effect on melatonin (Morris et al., 1990).

Research has demonstrated that sleep disorders (e.g. insomnia) and abnormal sleep durations (i.e. below 6 or above 10 hours) are risk factors for inflammatory disease and contribute to all-cause mortality (Irwin et al., 2016; Vgontzas et al., 2013; Dew et al., 2003, Kripke et al., 2002; Mallon et al., 2002). Observational studies have reported a moderate to strong correlation between sleep quality and inflammation (CRP) ($r = 0.37$-0.56) (Zeydi et al., 2014; Liu et al., 2014). Research in sleep health has focused on the biological mechanisms underpinning these effects, with particular focus on the effect of sleep disturbance on measures of innate immunity (Irwin, 2015). Increased levels of circulating inflammatory markers (i.e. CRP and interleukin-6 [IL-6]) have been shown to predict cardiovascular events (Ridker et al., 2003); body mass gain in older adults (Barzilay et al., 2006); hypertension (Sesso et al., 2003) and type 2 diabetes (Irwin and Cole, 2011). Recently, Irwin et al., (2016) conducted a meta-analysis of evidence highlighting the link between sleep disturbance, sleep duration and inflammation. Sleep disturbance (i.e. poor sleep quality, insomnia complaints) was associated with increased levels of IL-6 (ES: 0.20 [0.08-0.31]) and CRP (ES: 0.12 [0.05-0.19]) (Irwin et al., 2016). Short sleep duration (< 7h per night) was associated with increased IL-6 (ES: 0.29 [0.05-0.52]), while long sleep duration (> 8h per night) was associated with increased IL-6 (ES: 0.11 [0.02-0.20]) and CRP

(ES: 0.17 [0.01-0.34]). Similarly, a meta-analysis of sleep duration and all-cause mortality demonstrated a U-shaped association, whereby long sleep (> 8h per night) has a 30% (RR: 1.30 [1.22-1.38]) greater risk while short sleep (< 7h per night) has a 12% (RR: 1.12 [1.06-1.18]) greater risk, compared to normal sleep reference (7-8h per night) (Cappuccio et al., 2011).

During sleep, both circadian and sleep-dependent processes (e.g. cytokine production) contribute to the regulation of inflammatory processes and changes in immune function during sleep are influenced by both sleep and circadian oscillators (Irwin and Opp, 2017). For example, serum concentration levels of IL-6 have been shown to peak twice (≈19:00pm and again at 05:00am) and these peaks appear to be aligned to circadian processes (Vgontzas et al., 1999). However, total sleep deprivation has been found to reduce IL-6 concentration peaks by ≈50%, but a transient peak was still observed at ≈1:00am indicating continued circadian influences on IL-6 levels (Redwine et al., 2000). Acute sleep deprivation and sleep disturbance (short sleep duration or reduced sleep efficiency) impair adaptive immunity which is associated with reduced response to vaccinations and increased vulnerability to infectious diseases, attributed to reduced GH release during deep sleep and increased sympathetic output (Irwin, 2015). Tumour necrosis factor-α (TNFα) along with other cytokines are considered key to the regulation of sleep in normal physiological conditions (Dimitrov et al., 2015). TNFα is an inflammatory cytokine which has an important role in resistance to infection which is produced by macrophages/monocytes during acute inflammation and causes a variety of signalling events within cells, leading to necrosis and apoptosis (Idriss and Naismith, 2000). While increases in TNFα are primarily controlled by circadian factors (Parameswaran and Patial, 2010). During sleep, serum TNF-α levels decrease however, there is evidence of a profound night-time increase in the ability of monocytes to respond to challenge (i.e. toll-like receptor-4 (TLR-4) activation with lipopolysaccharide (LPS) (Dimitrov et al., 2015). Sleep is associated with increases in adaptive and inate immunity (i.e. inflammation) (Irwin, 2015). While, sleep deprivation and sleep disturbance (i.e. short duration or reduced sleep efficiency) impair adative immunity reducing response to vaccines and increasing suceptibiity to infection (Irwin, 2015). This evidence points to sleep's role in maintain the immune response to a challenge, while inadequate sleep can adversely affect the immune system and health.

1.8 Oxidative Stress (OS)

OS is an imbalance between oxidants and antioxidants due to increased oxidants, causing a disruption of redox (oxidation-reduction) signalling, control and/or molecullar damage (Seis and Jones, 2007; Sies, 2015). OS occurs due to an imbalance between reactive species (i.e. reactive oxygen species, reactive nitrogen species and reactive chloride species) and antioxidants (Strobel et al., 2011). Reactive species are normally produced during numerous physiological processes and play an important regulatory role as mediators of redox signalling processes (Strobel et al., 2011). Reactive oxygen species (ROS) are produced from mitochondrial metabolism (Bardaweel et al., 2018). ROS is a general term that refers to oxygen-centred radicals but also derivatives of oxygen that are non-radical but reactive (e.g. hydrogen peroxide [H_2O_2], superoxide [O_2^-], singlet oxygen [$^1O_2$], and the hydroxyl radical [$^{\bullet}OH$] (Halliwell and Gutteridge, 2015). ROS are continuously produced in skeletal muscle at rest but the rate of production increases during exercise (Reid, 2008), in relation to the intensity of the exercise and the antioxidant capacity of the individual (see Figure 1.5) (Pingitore et al., 2015). The cellular enzymes nicotinamide adenine dinecleotide phosphate (NADPH) oxidases also produce ROS (Lasségue et al., 2012). Other cellular sources of ROS include neutrophils, monocytes, cardiomyocytes, endothelial cells, xanthine oxidases, cytochrome P450, lipoxygenases and nitric oxide synthase (Izyumov et al., 2010; Cubero and Nieto, 2012). ROS 'steal' electrons form other nearby molecules, via an oxidative reaction which damages the structure of the molecules (Dunnill et al., 2017). Overproduction of ROS results in OS, which is considered a deleterious process, causing damage to cell structures that is implicated in undesireable health outcomes (Bardaweel et al., 2018). Increases in reactive species cause damage to lipoproteins, lipids, deoxyribonucleic acid (DNA) and proteins and OS induced modifications of these molecules has been linked to disease pathways e.g. CVD (Halliwell and Gutteridge, 2015; Strobel et al., 2011).

ROS have a key role in the preservation of cellular homeostasis, abnormaly low levels induce cell cycle arrest (cytostatic - i.e. inhitbits cell growth and multiplication), basal levels maintain cell function and homeostasis, increased ROS induce a number of transcription factors that drive a cell-mediated defence response, excessive ROS induction activates pro-apoptotic proteins that result in cell death (triggered by body processes with no inflammation) and in extreme cases cellular necrosis (triggered by trauma, infection or toxins) (Dunhill et al., 2017; Shen et al., 2009; Trachootham et al., 2008). ROS also regulates vascular constriction (vasoconstriction) and vascular relaxation (vasodilation) (Dunhill et al., 2017). ROS also plays a role in ageing, in a cycle where toxicity of ROS causes damage

to the mitochondria which generates additional ROS (Balaban, 2005). Further, ROS plays a role in the activation and assembly of inflammasomes, regulateing the inflammatory response (Salminen et al., 2012).

1.8.1 Sleep and OS

Increases in inflammatory, oxidative, substances and increased lactate following exercise may adversely impact sleep, decreasing REM and increasing sleep fragmentation (Jelic et al., 2008). One of the functions of sleep is the promotion of antioxidant mechanisms, while sleep deprivation induces OS (Atrooz and Salim, 2020). It has been suggested that sleep facilitates the removal of free radicals accumulated during waking (Reimund, 1994). Sleep deprivation has been associated with increased catabolic and reduced anabolic hormones which result in impaired MPS (Fullagar and Bartlett, 2016). It has been proposed that sleep deprivation may alter MPS through OS (Atrooz and Salim, 2020). Exercise can have a positive or negative effect on OS depending on the type (acute or chronic), specificity, load, volume and the basal level of fitness (Vassalle et al., 2015). Generally, beneficial changes to physiological and laboratory parameters have been observed as a result of regular moderate exercise, however acute and strenuous exercise may paradoxically induce OS and adverse health effects (Vassalle et al., 2015).

Few studies have explored the relationship between sleep quality, inflammation, oxidative stress and antioxidant levels (Dowd et al., 2007; Pingitore et al., 2015). Although regular moderate training appears beneficial for OS and health, acute and strenuous bouts of aerobic and anaerobic exercise can induce ROS overproduction (Pingitore et al., 2015). However, although exercise induces OS, the same stimulus is necessary to allow up-regulation in endogenous antioxidant defences i.e. superoxide dismutase [SOD], catalase [CAT], glutathione peroxidase [GPx], glutathione reductase, glutathione-S-transferase, protein and low molecular weight scavengers such as uric acid, coenzyme Q and lipoic acid (Poljsak et al., 2013). The human antioxidant defense system is complex and must balance ROS levels to miminise cellular damage while maintaining beneficial functions of ROS producton i.e. cell signalling and redox regulation (Halliwell, 2011). Under normal physiological conditions, the body's antioxidant defences can cope with the production of free radicals. Free radicals are molecules or any chemical species that have one or more unpaired electron, formed in cells by either losing or gaining one electron (Halliwell and Gutteridge, 2015). Unpaired electrons lead to molecular instability making radical highly reactive molecules that can promote oxidative damage to carbohydrates, proteins, lipids and

DNA (Galano et al., 2016; Stear et al., 2012). ROS can become toxic when generated in excess or in the presence of a deficiency in the bodys antioxidant defences (Vassalle et al., 2015). The imbalance between free radical generation and antioxidant defences leads to an OS state which plays a role in aging and many pathological conditions (e.g. cardiovascular disease, neurodegenerative diseases and cancer) (Vassalle et al., 2015). Exercise increases the production of reactive species (Lewis et al., 2015b), and if levels exceed an individual's antioxidant capacity, muscle and immune function can be impaired (Verhagen et al., 2006).

1.8.2 Hormesis

Hormesis theory refers to a biphasic dose response to an environmental agent, characterised by a low dose stimulation or beneficial effect and a high dose inhibition or toxic effect (Mattson, 2008). In terms of biology, Hormesis is defined as an adaptive response of cells and organisms to a moderate (usually intermittent) stress e.g. exercise (Mattson, 2008). Hormesis theory applied to ROS is based on the hypothesis that the body's reaction to repeated increases in ROS production (i.e. after each training session), via exercise, involves adaptive mechanisms (see Figure 1.5) (Pingitore et al., 2015). ROS are not always considered harmful metabolic byproducts, fulfilling the role of intracellular signalling molecules when strictly regulated (Ray et al., 2012; Scandalios, 2002). At a cellular level ROS, contribute to essential processes such as blood pressure regulation (Dhalla et al., 2000), cognitive function (Qin et al., 2006), and immune response (Krause and Bedard, 2008). Hormesis causes antioxidant up-regulation, a shift towards a more reducing environment (i.e. oxidation is prevented) and increased OS resistance (Greilberger et al., 2015; Radák et al., 2008). These adaptive responses and the detoxifying function of engogenous antioxidant enzymes (SOD, CAT, GPx, glutathione reductase, glutathione-S-transferase) and non-enzymatic antioxidants (e.g. Vitamins A, C and E; glutathione [GSH] and uric acid) are involved in the prevention of excessive OS related to performance enhancement, aging and pathological risk in athletes (Greilberger et al., 2015). Sleep clearly has a role to play in the recovery and adaptation from exercise bouts that is crucial for athletes.

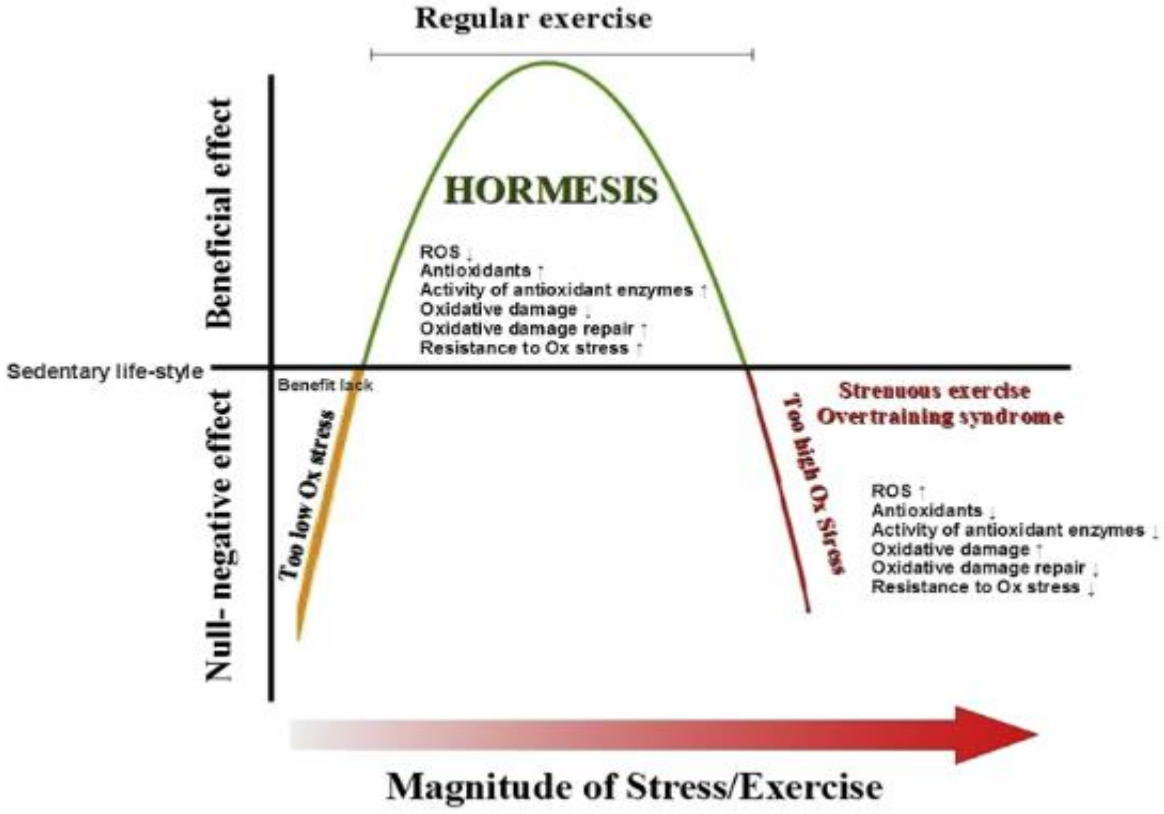


Figure 1.5: Hormesis and Exercise (Adapted from: Pingitore et al., 2015).

1.9 Research Aim

The aim of this research was to investigate the association between sleep, nutrition and athlete recovery. Although it is accepted that sleep is important for athletes and nutrition has a key role to play in both sleep (i.e. chrononutrition) and recovery the research is emerging and evolving (see Chapter 2 & 3). The current thesis sought to add to the understanding of the interaction between sleep, nutrition and athlete recovery.

1.9.1 Research Objectives

The objectives of this research were to:

- Scope the current literature in relation sleep and nutrition and the implications for athletes to determine a) is there a sleep problem in this population, b) is it related to recovery and c) what non-pharmacological options are there to manage both (Chapters 1-3).
- Characterise the sleep of athletes and recovery practices of athletes in general (Chapter 4) and under specific circumstances i.e. long-haul eastward travel (Chapter 5), to identify

the specific sleep and recovery areas of concern in this population with a view to implementing a chrononutrition intervention.

- Assess the impact of kiwifruit consumption on the sleep and recovery of elite athletes (Chapter 6).

1.10 Conclusion

Sleep is vital to maintain physical and mental health, recovery and performance. Sleep also has a restorative effect on the immune system and the endocrine system. Sleep requirements differ across the lifespan but for sleep to have a restorative effect on the body, it must be of adequate duration, timing, and quality. Good sleep is characterised by satisfaction, appropriate timing, adequate duration, high efficency and alertness during waking hours. Sleep disturbance and sleep deprivation can have negative health consequences, and both are implicated in inflammatory disease and all-cause mortality. Sleep deprivation adversely impacts carbohydrate metabolism, appetite, energy intake and protein synthesis affecting an individual's ability to recover from the energy demands of daily living and regulate fatigue. Sleep's role in overall health and wellbeing has been established. However, the relationship between sleep and recovery in specific populations (e.g. athletes) warrants further investigation.

Chapter 2: Sleep and Athletes

2.1 Introduction

Chapter 2 focuses on the current research investigating sleep and athletes. It is clear from the current literature that sleep inadequacy has been reported to be high among elite athlete populations due to both sport-specific factors (i.e. training, injury travel and competition) and lifestyle/social factors (i.e. habitual caffeine use, technology and social media use in an 'always connected' society) (Walsh et al., 2021). Moreover, research has highlighted athlete specific factors for sleep inadequacy such as high training loads short and long-haul travel, evening competition after 6:00pm, and early morning start times before 8:00am. Inadequate sleep may have a negative impact on athlete sleep and performance, particularly elite athletes who may have greater physical and mental recovery needs than the general population. When sleep is reduced to < 7 hours cognitive performance and physical performance and injury risk are adversely affected (Charest and Grandner, 2020; Laux et al., 2015; LeMeur et al., 2013). Adequate sleep including afternoon naps can counteract the negative performance, cognitive, immunity, OS, and pain outcomes that are consequences of sleep debt.

The repetitive demanding nature of an annual training and competition cycle can test athletes' physiological and psychological capacity. Training, competition, work, education, nutrition and other lifestyle factors and exposure to technology (i.e. blue light exposure), can have a detrimental impact on athletes' ability to match their circadian phase with the opportunity for sleep. If the circadian phase and sleep schedule are not matched, the duration and quality of sleep can be negatively affected (Lastella et al., 2016), which can negatively impact training adaptations, increase the risk of maladaption and reduce subsequent performance. Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery (Venter, 2014). In terms of general health, optimising sleep prevents and/or reduces the risk of illness and benefits energy levels, mood state and cognition, improves immunity and the recovery from illness (Irwin, 2015). Additional benefits for athletes include a reduced risk of overtraining/under recovery and reduced injury risk (Samuels et al., 2016; von Rosen et al., 2016; Milewski et al., 2014; Venter, 2014; Halson, 2013). Similar to nutrition and physical activity, sleep disturbances and long or short sleep durations are behavioural risk factors for inflammation (Irwin et al., 2016). For sleep to be truly restorative it must be of adequate duration, of sufficient quality and be well timed (Samuels et al., 2016). This is especially true for elite

athletes who due to typically high training loads and competition demands may have greater physical and mental recovery needs than the general population.

2.2 Napping

While the majority of the sleep period should occur at night in order to align with the normal circadian rhythm (Simpson et al., 2017), when spending the right amount of time in bed at night is not possible or when sleep is compromised (e.g. training camps, travelling or competing late at night) recouperative daytime naps may be used to increase total sleep time within a 24hour period. Afternoon naps should be restricted to 30 minutes (Romyn et al., 2018, Lastella et al., 2016; Mah et al., 2011) in an attempt to avoid sleep inertia (severe subjective feeling of sleepiness upon waking [Tassi and Muzet, 2000]) and prevent any adverse impact on the following night's sleep duration and quality. Three types of nap have been proposed which differ in their relationship with daytime sleepiness: prophylactic sleeping (anticipation of sleep loss), replacement/compensatory napping (response to sleep loss) and appetitive napping (for convenience or enjoyment) (Broughton and Dinges, 1989). Athletes must strive to maintain a regular and consistent sleep/nap routine, a comfortable sleeping environment and monitor their sleep to maximise sleep quality (Samuels et al., 2016). When schedule changes are not possible to prioritise sleep, napping can supplement insufficient night-time sleep (Walsh et al., 2021).

Napping has been suggested as a behavioural measure to dissipate sleep debt, with the afternoon being the most frequent time that napping occurs (Petit et al., 2014). It has been reported that athletes nap frequency was 11% over a 7-night period with a mean duration 0:59 ± 1:02 hr:min (Lastella et al., 2015). In study of elite Karate combat athletes (n=13) a 30-minute nap (1:00pm) following both a normal night's sleep and partial sleep deprivation (sleep 11:00pm-3:00am) enhanced alertness, cognitive funtion and performance (Daaloul et al., 2019). An afternoon nap was suggested as an effectice strategy for athletes to attenuate the cognitive and physical deterioration in performane resulting from either sleep loss or fatigue induced by training/competition (Daaloul et al., 2019). Equally, naps have been shown to enhance mood, alertness and cognitive performance in those who typically get the amount of sleep they need on a nightly basis (Milner and Cote, 2009), therefore napping may be an effective strategy even for athletes who get adequate sleep. In terms of duration, it has been suggested that when athletes have a nap opportunity, < 30mins is preferrable to avoid sleep inertia (Walsh et al., 2020). A 15-20min 'coffee-nap' in mid-afternoon has also been proposed whereby athletes consume caffeine (150-200mg) immediately before napping to

counterbalance sleepiness (Horne and Reyner, 1996; Hayashi et al., 2003). Alternatively, 90mins is also considered an optimal nap period as this facilitates a complete sleep cycle (NREM and REM) to occur, reducing the effects of sleep inertia (Davies et al., 2010).

2.3 Chronotype

Humans typically display individual differences in the timing of their behaviour (e.g. social activities, daytime activities and sleep) (Roenneberg et al., 2003). Chronotype is the preferential expression of individual circadian rhythmicity and has been categorised as follows: morning types, intermediate types, and evening types (Vitale and Weydahl, 2017; Roeneberg et al., 2007). Research in the general population has demonstrated that most people are intermediate types (70%) with the remainder being either morning types (14%) or evening types (16%) (Vitale and Weydahl, 2017). Chronotype is in part genetic but cultural and environmental factors also affect an individual's sleep pattern. Morning types prefer to wake up and perform tasks in the early morning and display difficulty remaining awake beyond their usual bedtime (Adnan et al., 2012). Evening types prefer to go to bed later and have difficulty waking up early (Adnan et al., 2012). Intermediate types lie somewhere on a scale between morning types and evening types. Athletes' sleep/wake behaviours are often based on their training plan (Lastella et al., 2015), and the timing of training/competition has the potential to impact on performance. There is evidence that elite athletes tend to peruse sports that involve training and competition schedules matching their chronotype (Lastella et al., 2016). In a study of swimmers (n=26, 18 males and 8 females), morning type swimmers (p=0.036) and those who habitually trained in the morning ($p=0.011$) were significantly faster during a morning (06:30am) 200m time trial compared to an evening time trial (18:30pm) accompanied with lower rate of perceived exertion (RPE) scores post warm-up, higher vigour and lower fatigue scores (Rae et al., 2015). A recent study highlighted a time of day effect in the VO_2max of athletes (n=17, 10 males and 7 females) the mean diurnal variation difference was 5.0 ± 1.9 ml·kg·min^{-1} (95% CI: 4.1-6.0) across 6 assessments completed every 3 hours between 7:00am and 21:00pm (Knaier et al., 2019).

A recent meta-analysis identifided body temperature as the most important marker of diurnal variance in athletic performance (Kusumoto et al., 2020). Morning types display earlier peaks of several psychophysiological variables (RPE and fatigue) during the day compared to evening types (Vitale and Weydahl, 2017). Research has demonstrated differences between morning and evening types in terms of circadian rhythmicity in relation

to physiological variables, synchronisation to jetlag, personality, mood and cognitive performance (Adnan et al., 2012; Vitale et al., 215). Significantly ($p<0.05$) delayed peaks in oral temperature (2 hours) and serum cortisol (55minutes) have been observed in evening types compared to morning types (Baehr et al., 2000; Bailey and Heitkemper, 2001). Morning types displayed an early acrophase (time of peak hormone concentration) of serum and salivary melatonin concentrations (≈3 hours) compared to evening types (Adnan et al., 2012). Melatonin is a hormone associated with circadian rhythms (Ramis et al., 2015), that has displayed sedative effects (Bonnefont-Rousselot et al., 2010). The secretion of melatonin is regulated by the SCN, it is produced by the pineal gland, influenced by light/dark cycles and is integral in the sleep/wake cycle (Ramis et al., 2015). Since endogenous melatonin influences core temperature, facilitating sleep, increased exogenous melatonin could positively impact on core temperature and subsequent sleep quality (Howatson et al., 2011).

2.4 Fatigue

A variety of metabolic and/or neural factors of central (brain) or peripheral (muscle) origin contribute to fatigue. Peripheral alterations in skeletal muscle, cardiovascular function and metabolic strain are linked to acute fatigue (Bangsbo et al., 2006) and investigation of the CNS contribution to acute fatigue is an emerging field of research (Roelands et al., 2013). Athletic performance induces physiological disturbance but also causes psychological stress due to the need for sustained periods of concentration, perception skills and decision making. During field based team sports, the athletes' environment is in a constant state of change and players must synthesise information regarding the ball, teammates and opponents before choosing an appropriate action based upon set objectives (e.g. strategy, tactics) and action constraints (e.g. technique, physical capacity) (Williams, 2000). Such cognitively demanding tasks often lead to mental fatigue, adversely impacting performance.

2.4.1 Fatigue and Athletes

Fatigue has many potential drivers including dehydration, thermoregulation, glycogen depletion, muscle damage and mental fatigue. Recovery of muscle function is predominantly a matter of reversing the main causes of fatigue. In theory, the rate of post-exercise recovery is relative to the physiological and neuromuscular demands of the exercise bout (Nédélec et al., 2012). It is assumed that physiological bases of recovery are dependent on restoration and reversal of the stresses placed on the exercising muscles (Minett and Duffield, 2014). In contrast, the regulation of performance during exercise has increasingly been interpreted as a cohesive, multifaceted process involving both the CNS and PNS (Knicker et al., 2011;

Noakes, 2012). While there is debate whether the regulation of exercise performance is derived from the CNS or PNS (St Clair Gibson and Noakes, 2004) and whether the regulation is conscious (Marcora, 2008) or anticipatory (Marino, 2004); it is clear that changing CNS drive and motor unit recruitment are associated with fatigue. Therefore, if the brain is linked to all the core physiological systems (e.g. endocrine, muscloskeletal, nervous and immune) critical to the processes that lead to fatigue (performance detriments) then it has a role in the post-exercise recovery process (De Pauw et al., 2013), which could be facilitated through sleep.

Training is the process of preparing an individual physically, technically, tactically, and psychologically to perform (Bompa and Buzzichelli, 2015). At the elite level, athletes and their support teams continually strive for marginal gains over time to improve performance (Soligard et al., 2016). Training and competition load elicit a number of homeostatic responses and adaptations, with the main aim of training being to exploit these in order to elicit an improvement in performance. The training process involves exploitation, manipulation and coordination of numerous variables (e.g. physiology, biomechanics and psychology) to improve performance. Athletes continually strive to improve their performance, as such variations in training load are necessary e.g. increased frequency, duration and/or intensity, in order to optimise the training response (Halson, 2014). As part of a periodized training plan, depending on the phase of the season (e.g. pre-season, general preparation, competition, etc.), loads must be manipulated and managed to increase or decrease fatigue and optimise recovery, to enhance training adaptations or performance (Halson, 2014).

2.4.2 General Adaptive Syndrome and the Fitness-Fatigue Model

Hans Seyle's General Adaptive Syndrome (GAS) principle is based on a series of rodent studies which investigated the stress response to non-lethal doses of different drugs (e.g. morphine) and stimuli (e.g. exercise). Similar responses were observed (gastrointestinal ulceration, thymico-lymphatic atrophy and adrenocortical ulceration) and classified into three stages: alarm, resistance and exhaustion (Bucker et al., 2017). This has led to the application of the GAS model in relation to exercise programming. Although the GAS concept was developed in the 1930's it has been widely accepted in this field following inclusion in a theoretical model of strength training in the 1980's (Stone et al., 1982) and played a role in the development of Periodisation. Periodisation has been defined as a training plan where peak performance is elicited through potentiation of biomotors and the

management of fatigue and accommodation (Turner, 2011). In terms of the GAS applied to resistance training, it is suggested that there are three phases of adaptation: alarm, resistance and overtraining (Buckner et al., 2017). Based on the GAS, training load should be divided into unequal portions (cycles), with the ultimate aim being to avoid exhaustion i.e. periodised. Periodisation is usually based on intentional peaking for competitions or events of perceived greatest priority or difficulty over the course of a competitive season (Robertson and Joyce, 2015). In real terms this typically involves manipulation of training intensity and volume over specificed time periods in order to maximise athlete preparedness for upcoming competitions/events.

Similarly, the fitness-fatigue model argues that different training stressors result in different physiological responses (Bannister, 1991). The model is based on the premise that training results in two after-effects which either positively, or negatively, influence performance: fitness (+) and fatigue (-) (Chui and Barnes, 2003). For athletes, factors that affect fitness include muscle cross-sectional area, muscle contractile protein composition, muscle metabolic enzyme concentrations, maximal oxygen consumption, mitochondrial density and muscle capillarisation (Chui and Barneas, 2003). The fitness-fatigue model is not necessarily an alternative to GAS but an enhanced representation of stimulus and response and the distinction of fitness and fatigue after-effects. This is important for the development of training paradigms e.g. periodisation (Chui and Barnes, 2003).

2.4.3 Functional and Non-functional Over-reaching

At certain times of the training cycle, overload is programmed to put the athlete into an overreached state. Successful training outcomes must involve overload but avoid excessive overload coupled with inadequate recovery (Meeusen et al., 2013). Overreaching or short-term overtraining is an accumulation of training and/or non-training stress characterised by acute performance reductions with or without related physiological and psychological signs and symptoms of maladaptation, which are reversible (within 1-2 weeks) and can result in super-compensation (enhanced adaptations and/or improved performance) (Meeusen et al., 2013). When appropriate periods of recovery are provided a ‘Supercompensation’ effect is observed whereby the athlete’s performance improves beyond baseline levels. This response is defined as functional overreaching (FO) (Meeusen et al., 2013). While short term overreaching is typically an important component of the training plan (e.g. training camps), prolonged overreaching pushes an athlete into a non-functional overreaching (NFO) state or over-trained state, which can negatively affect performance and health (Sands, 2016). When

this intensive training schedule is prolonged the athlete can evolve into a state of NFO i.e. fatigue lasting weeks to months (see Figure 2.1), that will lead to a reduction in performance for several weeks or months (Meeusen et al., 2013).

Under-recovery and NFO can be attenuated through the systematic application of recovery strategies and rest alongside lifestyle related factors such as sleep, nutrition and social interaction (Kellmann et al., 2018). Continued underrecovery and NFO are a precursor for overtraining syndrome (OTS) (Kellmann et al., 2018). OTS is characterised by physical symptoms such as prolonged muscle soreness, pain sensations, clinical and/or endochrinological disturbance (Kellmann et al., 2018). Recovery from OTS requires a prolonged period of rest and recovery lasting weeks to months with subsequent reduced performance (Kellmann et al, 2018; Meeusen et al., 2013). Prolonged imbalance between fatigue and recovery results in negative consequences such as underecovery, NFO, or OTS. Hence, monitoring training load is important to determine if an athlete is adapting to training and to minimise the risk of NFO, injury and/or illness (Soligard et al., 2016; Schwellnus et al., 2016).

Process	Training (Overload)		
Outcome	Acute fatigue	Functional Overreaching (Short-term)	Non-functional Overreaching (extreme overreaching)
Recovery	Day(s)	Days-weeks	Weeks-months
Performance	Increase	Temporary Performance Decrement (e.g. Camp)	Stagnation or decrease

Figure 2.1: Functional and non-functional overreaching (Adapted from: Meeusen et al., 2013).

The relationship between training load and health can be considered on a continuum of well-being (Soligard et al., 2016; Schwellnus et al., 2016), with training load and recovery as antagonists. Stress (training and non-training) is imposed on athletes, altering their physical and psychological well-being along a continuum: homeostasis, acute fatigue, subclinical tissue damage, FO, NFO, clinical symptoms, overtraining syndrome, time-loss injury or illness and with continued loading in extreme cases, death (Soligard et al., 2016; Schwellnus et al., 2016). A recent meta-analysis has linked psychosocial stress (r = 0.27, 80% CI 0.20-0.37) and history of stressors (r = 0.13, 80% CI 0.11-0.15) to injury rates in athletes (Ivarsson et al., 2017). Athletes' injury risks were affected by their responses to

multiple stressors that result in not only physical, psychological and attentional changes (e.g. increased reaction time, narrowing of peripheral vision, increased distracability) but also behavioural changes (e.g. poor sleep quality, poor diet and impaired self-care) (Ivarsson et al., 2017). As such, athletes should have a detailed, multi-faceted and structured recovery plan involving nutrition, hydration, sleep and psychological recovery strategies (Schwellnus et al., 2016). Given the high training and competition load that athletes undertake; it is clear that they must adopt strategies that promote sleep quality and duration. Fatigue can be managed, and recovery can be enhanced through adequate passive rest and sufficient sleep (Meeusen et al., 2013), it is generally recommended that athletes have one 'rest' day per week. Rest days can serve to alleviate boredom and stress perception while the absence of a 'rest day' during periods of intense training has been related to the onset of overreaching and inadequate recovery (Meeusen et al., 2013).

2.4.4 Fatigue and Performance

Despite the plethora of literature exploring the influence of fatigue on performance, limited attempt has been made to bridge the gap between theory and practice in terms of translational research (Enoka and Duchateau, 2016). This is due to the compartmentalisation of knowledge relating to fatigue across disciplines such as medicine, physiology and psychology. In an attempt to standardise the approach to evaluating fatigue, a framework has been proposed by Enoka and Duchateau, (2016) that can be used to assess the functional significance of fatigue. Through the framework it is proposed that fatigue can be defined as a symptom whereby physical and cognitive function are limited by interactions between perceived fatigability and performamce fatigability (Kluger et al., 2013; see Figure 2.2). Percieved fatigability is a result of the sensations that regulate homeostasis (balanced internal state) and the psychological state of the individual (Enoka and Duchateau, 2016). Performace fatigability is a result of the contractile capabilities of the muscles involved and the capacity of the nervous system to provide activation signals for a given task (Enoka and Duchateau, 2016). The level of fatigue experienced can be modulated by disturbances to psychological state, reduction is muscle contractile function and the capacity to deliver activation signals to the muscles involved (Enok and Duchateau, 2016). Athletes experience fatigue not only due to competition but also due to the training stimulus required to maintain and develop the many capacities necessary for optimal performance i.e. speed, power, strength and endurance; in addition to technical and tactical skills (Jones et al., 2017).

In terms of sports performance, fatigue is defined as an acute decrease in force production, or an inability to regenerate the original force in the presence of an increased perception of effort (St Clair Gibson and Noakes, 2004). The central governor theory proposes the brain regulates exercise within a neurally contolled safe exertion level by continuously modifying the number of motor units that are recruited in the exercising limbs (Noakes et al., 2005). The brain uses uncomfortable sensations of fatigue to ensure that exercise intensity and duration are always within an individual's capacity (Noakes, 2012). In terms of exercise performance, fatigue is classically defined as an exercise induced reduction in the maximal voluntary force production during muscular contraction and can be attributed to contractile failure, suboptimal motor cortical output (supraspinal fatigue) and/or altered afferent inputs (spinal fatigue) (Gandevia, 2001). Due to the extremely complex nature of fatigue (Chiu and Barnes, 2003; Noakes, 2012), and individualised responses (Maan et al., 2014; McLean et al., 2012), it has become common practice to monitor global athlete fatigue (i.e. comprising physical, mental and neural aspects) to assess the response to training loads in an attempt to minimise injury risk and illness (Jones et al., 2017). Physical fatigue (e.g. muscular, cellular, metabolic, thermoregulatory) results from exertion (i.e. training/competition) and induces an acute reduction in the force production capacity of the muscle which is traditionally referred to as muscle fatigue (Pageaux and Lepers, 2016; Gandevia, 2001). The reduction in force production capacity is caused by an inability of the CNS to maximally recruit the muscles (i.e. central fatigue [CF]) and/or through changes at or distal to the neuromuscular junction adversely affecting the contractile properties of the muscle (i.e. peripheral fatigue [PF]) (Allen at al., 2008). Sustained physical activity requires co-ordination of various physiological systems to facilitate gas exchange, energy supply and repeated muscular contraction.

Muscle fatigue is linked to skeletal muscle acidosis during high intensity activity (e.g. sprinting) whereby lactate accumulation causes an increase in hydrogen ions leading to a reduction in cellular pH and negatively impacting muscle contraction, or glycogen depletion during more prolonged activity (e.g. marathon) (Juel et al., 2004; Robergs et al., 2004). Mental fatigue is a psychobiological state caused by prolonged bouts of cognitively demanding activity (Desmond and Hancock, 2001; Job and Dalziel, 2001). Mental fatigue has been demonstrated to negatively impact endurance, technical skill and decision making (Smith et al., 2018; Van Cutsem et al., 2017). Mental fatigue manifests both behaviourally and/or physiologically through a decline in accuracy and reaction time, feelings of tiredness, lack of energy, reduced motivation and alertness (Van Cutsem at al., 2017).

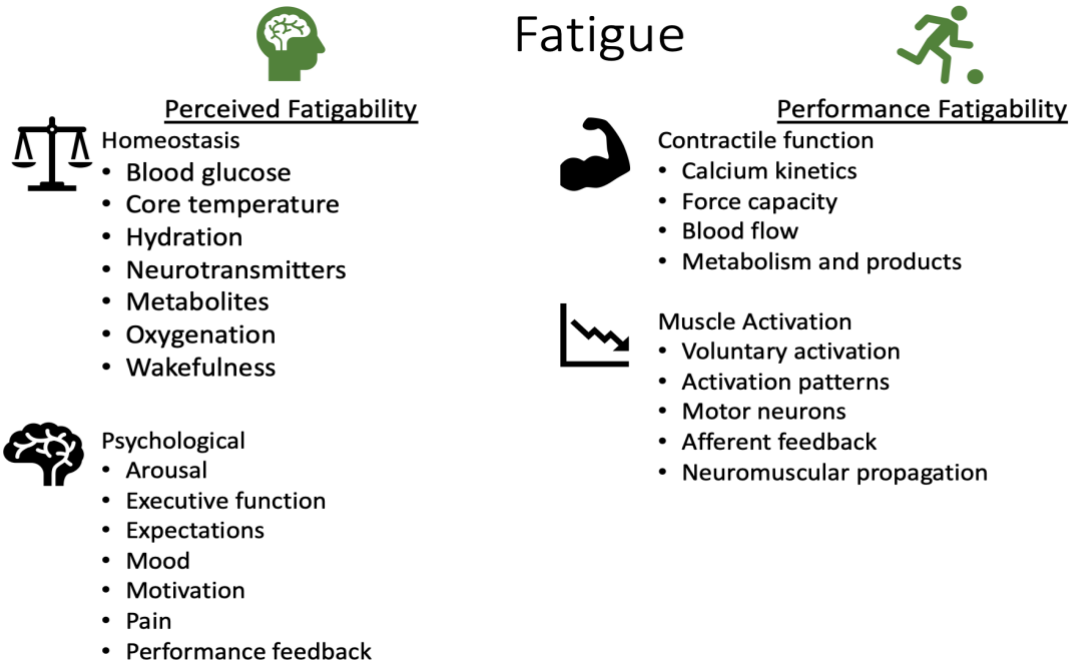


Figure 2.2: Taxonomy of fatigue (Adapted from: Kluger et al., 2013).

In terms of intermittent sports (repeated bouts of high intensity activity interspersed with short periods of active recovery or passive rest) fatigue can be defined as a decline in peak sprint speed or power output between multiple efforts (Bishop, 2012). Intermittent team sports (e.g. Gaelic football, soccer, Rugby and Australian rules football, etc.) involve repetitive intense activities such as sprinting, quick accelerations and decelerations to stop or change direction, kicking, tackling, jumping and collisions. Such activities involve repeated eccentric (lengthening) muscle contraction causing muscle damage (Duffield et al., 2009; Coutts et al., 2010; Akenhead et al., 2013; Bradley and Noakes, 2013; Howatson and Milak, 2009; Ispirlidis et al., 2010). Muscle damage during exercise is related to mechanical disruption of the muscle fibre such as membrane damage, myofibrillar disruptions (myofilament disorganisation and loss of z-disc integrity) (Raastad et al., 2010) and subsequent damage is caused by inflammatory process and altered excitation-coupling within the muscle (Clarkson et al., 1992). Laboratory based simulations of intermittent team sports tend not to include collisions, tackles and kicking which all induce muscle damage. This is a limitation of such research, as individualised recovery strategies should be devised based on the muscle damaging activities performed during competition (Young et al., 2012).

Research has highlighted that fatigued participants were still able to complete over-learned, automatic skills; however, the performance of tasks that demand voluntary allocation of attention significantly deteriorated (Boksem et al., 2005; Sanders, 1998; Lorist et al., 2005). Greig et al. (2007) demonstrated that the cumulative effect of completing a

continuous grid based vigilance task (n=10, semi-professional soccer players) during a laboratory simulation of a 90 minute soccer match (intermittent treadmill protocol), errors significantly ($p < 0.05$) increased during the final 30 minutes of the protocol, with mean response time during the final 15 minutes (0.71 ± 0.04 seconds) significantly ($p < 0.05$) slower than during the first 15 minutes (0.65 ± 0.05 seconds). Tolerance to high intensity cycling (n=16; time to exhaustion at 80% peak power output) significantly deteriorated due to mental fatigue (90 mins demanding cognitive performance test) (Marcora et al., 2009). Following completion of a 90 minute cognitively demanding task, a mood questionnaire revealed a state of mental fatigue ($p=0.005$) that was related to significantly reduced time to exhaustion (i.e. reduced performance capacity [640 ± 316s]) compared with the control group who watched a 90 minute emotionally neutral documentary (754 ± 339s) ($p = 0.003$) (Marcora et al., 2009).

Athletes experience both acute and residual fatigue during periods of heavy training and competition (Borresen and Lambert, 2009). Field based team sports are characterised by repeated bouts of intermittent activity (sprinting) with short rest periods representing high physiological stress (Spencer et al., 2005), neuromuscular stress (Rampini et al., 2011; Duffield et al., 2012) and RPE (Impellizzeri et al. 2004). Relative stress is accumulated when successive bouts compromise the athlete's rate of recovery and can limit subsequent performance (Bishop et al., 2008). Hence, decreasing the natural timeframe of the bodies' regenerative processes via recovery strategies is vital for performance (Barnett, 2006). Such recovery strategies can be divided into physiological strategies (e.g. sleep, cold water immersion, cryotherapy, massage and compression), pharmacological (e.g. non-steroidal anti-inflammatory drugs [NSAIDs]) and nutritional (e.g. nutrient timing and supplementation) (Minett and Duffield, 2014). The aim of each strategy is to limit post exercise physiological disturbances and inflammation within the exercised muscle cells. An understanding of the physiological mechanisms driving recovery is vital to assess the beneficial effects of each strategy or combination of strategies. A certain degree of fatigue is necessary to elicit performace enhancement (i.e. functional overreaching) which can be counterbalanced through adequate recovery (Kellmann et al., 2018), which can be facilitated through sleep.

2.5 Oxidative Stress (OS) and Athletic Performance

Performance is influenced by the complex inter-play between various distinct factors: nutrition, genetics, training and conditioning, psycho-social and lifestyle. Therefore,

performance is related to training and physical adaptation and correct nutrition in individuals with specific genetic characteristics can facilitate such adaptations (Banfi et al., 2012). However, the OS related effects of sustained and strenuous training performed by elite athletes are still debated (Perrone et al., 2020; Magherini et al., 2019; Park and Kwak, 2016). While some studies report that strenuous and sustained exercise induces OS, inflammatory response and structural damage to both skeletal and cardiac muscle cells, there is conflicting evidence that training can increase resistance to OS and reduce inflammation (Neubauer et al., 2008, Pinto et al., 2012; Nun et al., 2010.) While strenuous exercise increases OS, it also appears to upregulate endogenous antioxidant production which has beneficial effects such as increased antioxidant enzyme activity and repair of OS (Heaton et al., 2017). The contradiction may be explained by the adaptive response of the body to repeated OS induced by training, resulting in the ability to better cope with OS of different origins (Teramoto and Bungum, 2010).

ROS are involved in immunity by acting against antigens during phagocytosis (the ingestion of bacteria or other material by phagocytes) (Fehrenbach and Northoff, 2000), this role increases during inflammation. Inflammation is caused by training, particularly intense eccentric (i.e. muscle lengthening) exercise. ROS have an important role in cellular signals serving as cell messengers or modifying oxidation reduction (redox) status (Finaud et al., 2006). ROS are also involved in enzyme activation, in drug detoxification and facilitation of glycogen repletion (Jenkins, 1988). ROS are potentially harmful as they can alter the size and shape of compounds they interact with (Jenkins, 1988) and every type of cell can be damaged. ROS can cause apoptosis in healthy cells, inflammation and alter cellular functions (Tavazzi et al., 2000). ROS initiate lipoprotein oxidation, particularly low density lipoprotein (LDL). This is dependent on blood antioxidant capacity and can increase due to OS caused by exercise (Pincemail et al., 2000). However, it must be noted that these effects are partially or totally compensated in athletes as exercise reduces CVD risk (Pincemail et al., 2000). ROS can also oxidise blood and structural proteins and inhibit the proteolytic system (Szweda et al., 2002). During oxidation proteins, can be fragmented or lose amino acids, leading to alterations in structural proteins or of enzyme functions (Szweda et al., 2002).

Increased ROS production is an important factor in the aging process and a major determinant of lifespan (Vina et al., 2013). Epidemiological studies have reported that elite athletes, particularly endurance athletes, live longer than the general population and have hypothesised an inverse dose response relationship between the volume of physical training and all-cause mortality (Corbi et al., 2012; Lee et al., 2001). A paradox exists in terms of the

oxidative and inflammatory effects of exercise and the beneficial effects of physical activity. Repeated exercise bouts result in increased ROS production causing antioxidant up-regulation, a shift towards a more reducing environment (oxidation is prevented) and increased OS resistance (Greilberger et al., 2015; Radák et al., 2008). These adaptations may protect against increased ROS production, resulting in improved exercise performance and health (Vassalle et al., 2015). Similarly, in animal studies, regular exercise has been shown to up-regulate glutathione levels and reduce ROS production and inflammation in rats (Rádak et al., 2008; Rádak et al., 1999).

Humans are bound by the oxygen paradox, namely they cannot live without oxygen, but it can represent a hazard to health status, the risk is presented through the inherent formation of oxidants during cellular respiration (Galano et al., 2016; Park and Kwak, 2016). Free radicals are highly reactive molecules and can damage carbohydrates, proteins, lipids and DNA (Halliwell and Gutteridge, 2015; Galano et al., 2016; Stear et al., 2012). A growing body of evidence indicates that increased ROS production in skeletal muscle is related to fatigue during prolonged sub-maximal exercise bouts (>30mins), due to oxidative damage of proteins and lipids reducing muscle force production (Galano et al., 2016; Halliwell and Gutteridge, 2015; Stear et al., 2012). A minimal amount of ROS is necessary for muscular contraction; however, OS causes increased intramuscular ROS concentrations and is associated with fatigue during contraction and has been related to post-exercise muscle damage (Reid, 2008). The alteration of mitochondrial function by ROS, is considered a major factor in muscle fatigue (Reid, 2008). Mitochondria are particularly susceptible to ROS-induced oxidative damage of lipids, proteins and DNA. Mitochondria DNA damage can alter aerobic pathway efficiency due to changes to the respiratory complexes causing decreased electron transfer and adenosine triphosphate (ATP) formation (Finaud et al., 2006), resulting in increased anaerobic pathway utilisation. Anaerobic pathways cause increased inorganic phosphate (Pi) levels and acidosis contributing to muscular fatigue (Reid, 2008), this reusltant fatigue could adversely impact training adaptations or reduce performance during competition.

2.6 Recovery

Post-exercise recovery is vital for all athletes. If the balance between training stress and physical recovery is inadequate, the adaptation from and performance in subsequent training sessions or competition may be adversely affected (Venter, 2014). Elite athletes have intense training and competition schedules, with most sports containing cycles of competition

combined with a periodised training plan over the course of a competitive season. Athletes train at high intensity to maintain or improve sport specific physical qualities such as speed, strength, power and technique. Training followed by optimum recovery strategies can induce favourable physiological; and psychological adaptations resulting in improved performance (Hartwig et al., 2009). Cognition, tissue repair and metabolism are critical psychological and physiological factors that contribute to training capacity, recovery and ultimately performance (Samuels et al., 2016).

In addition to training stress, the disruption associated with the stress of travel due to competition schedules contributes to both physical and mental fatigue. The process of travel, jet lag, travel fatigue or arriving at a destination in the middle of the night can cause circadian rhythm disruption. Further, associated stress, restricted motion during the flight(s) and unfamiliar surroundings can lead to sleep disruption (Bishop, 2004) and poor quality sleep (Richmond et al., 2007).

2.7 Sleep and Recovery

The relationship between sleep and recovery can be viewed in terms of 3 key factors that affect the recuperative outcome:

1. Sleep length (total sleep requirement; hours/night, plus naps)
2. Sleep quality (sleep disorders, environmental disturbance or sleep fragmentation)
3. Sleep phase (circadian timing of sleep)

(Samuels et al., 2016)

The aetiology of sleep disturbances during periods of intense training is unclear. It remains to be determined as to whether poor sleep is a symptom of overtraining, or intense training negatively affects sleep and recovery (Hausswirth et al., 2014). Muscle fatigue or soreness may adversely affect sleep, with inflammatory cytokines linked to disruption of normal sleep (Hausswirth et al., 2014; Imeri and Opp, 2009), while poor sleep increases muscle soreness (Hagenauer et al., 2017). Inadequate recovery can reduce autonomic nervous system (ANS) resources, with an associated reduction in heart rate variability (HRV) and increased heart rate (Hynynen et al., 2006). HRV responds to changes in training load and is negatively affected by total sleep deprivation (12h -7.3; 24h -9.09; 36h -9.6; $P<0.05$) (Zhong et al., 2005), following adequate recovery, HRV values increase due to a slower heart rate and reduced ANS excitability.

Athletes may experience significant problems sleeping due to lack of an appropriate sleep routine relating to changing training schedules, timetables and other sleep-

incompatible behaviours e.g. late night blue light exposure (Tuomiletho et al., 2016). For athletes, post competition routines and heightened arousal (i.e. medical care, recovery strategies, meals, media commitments and travel) can lead to later bedtimes, which can adversely affect sleep quality and quantity. Reduced sleep is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis (Fullagar and Bartlett, 2016), blunting training adaptations and recovery. Extensive sleep loss (≥ 30h sleep deprivation) has been associated with a reduction in muscle glycogen content (Skein et al., 2011).

Sleep was reported as the most important recovery modality utilised by South African athletes (n = 890; International n = 183, National n = 474, Club n = 233) (Venter et al., 2010). While Erlacher et al., (2011) found that 66% (n = 416) of elite German athletes (n = 632) reported pre-competition sleep problems including difficulty falling asleep, waking during the night and early final waking times. Similarly, modest sleep loss has been associated with reduced psychomotor performance in adults as demonstrated by a significant increase (2.5 ± 0.5; p = 0.01) in psychomotor vigilance task (PVT) lapse totals (Vgontzas et al., 2004).

Sleep patterns have been shown to influence athletic performance while athletic performance has also been shown to impact sleep patterns. Following a single night of sleep restriction (5 hours), mean Tennis serve accuracy declined significantly from baseline 53% to 37% (P < 0.001) (Reyner and Horne, 2013). Tuomiletho et al., (2016), investigated the sleep patterns of professional male Ice Hockey players (n = 23) using PSG and found than mean total sleep time (415mins; 95% CI 378-450) was inadequate. Sleep duration (< 8hours) has been identified as the strongest predictor of injury in adolescent athletes (RR = 2.1; 95% CI: 1.2 - 3.9; P = 0.01) (Milewski et al., 2014). The Karalinska Athlete Screening Injury Prevention (KASIP) study investigated injury occurrence in Swedish adolescent elite athletes (n = 340; 178 males and 162 females) and demonstrated that athletes reaching the National Sleep Foundation (Hirshkowitz, 2015) sleep guidelines (> 8 hours) reduced injury risk by 61% (OR: 0.39; 95% CI 0.17 - 0.96) while athletes who consumed the recommended nutrition guidelines reduced injury risk by 64% (OR: 0.36; 95% CI 0.14 - 0.91) (von Rosen et al., 2016). These findings illustrate the interactions between sleep and athlete recovery. Sleep extension (> 10hours per night for 2 weeks) demonstrated significantly (P < 0.001) improved sprint time (16.2 vs 15.5 seconds), free throw shooting accuracy (7.9 vs 8.8), 3-point shooting accuracy (10.2 vs 11.6), mean reaction time (PVT 310.84 ± 77.13ms vs 274.51 ± 42.01) in collegiate Basketball players (n=11) (Mah et al., 2011). Subjective ratings of physical (7.8 vs 8.8; P < 0.0001) and mental (6.9 vs 8.8; P < 0.0001) wellbeing in training

and games also improved following the period of sleep extension (Mah et al., 2011), however, the absence of a control group must be noted. More research is necessary to investigate the sleep of athletes and potential interventions to improve overall sleep quality and quantity.

2.8 Sleep and Pain

Sleep is vital for pain regulation which can impact both the general population and athletes. An investigation of 'mildly sleepy' (indicative of inadequate sleep duration) but otherwise healthy males (n=24) showed sleep extension (time in bed 10 hours) increased pain tolerance by 20% (Roehrs et al., 1989). While chronic sleep restriction (50% of habitual time for 12 days) was related to increased levels of muscle soreness. Sleep deprivation has also been linked to increased pain sensitivity (Hagenauer et al., 2017). Total sleep deprivation (TSD) (88 hours of continual wakefulness) resulted in small 5%-14% but significant ($P<0.05$) increases in spontaneous generalised pain (i.e. headache, muscle pain, stomach pain, generalised body pain and physical discomfort) (Haack et al., 2009). However, it must be noted this study involved a very small sample size (n=8) and pain was self-reported. Onen et al., (2001) showed an 8% (range 8.1%-8.8%) decrease in pain threshold after a single night of sleep deprivation. As such, it is clear that sleep has a key role to play in athletes' pain regulation. Increased pain tolerance could allow athletes to train at higher intensities, be more competitive and fully engage in rehabilitation following injury (Simpson et al., 2017).

2.9 Conclusion

The importance of sleep for not only athletes' recovery but also their performance is clear. When sleep is reduced to < 7 hours cognitive performance (i.e. alertness, reaction time, memory and decision making) and physical performance and injury risk are adversely affected (Charest and Grandner, 2020; Laux et al., 2015; LeMeur et al., 2013). Adequate sleep including afternoon naps can counteract the negative performance, cognitive, immunity, OS, and pain outcomes that are consequences of sleep debt. Due to the demanding nature of athletes' schedules which can impact sleep, reducing recovery, sleep has previously been recognised as important for optimal training and performance in athletic populations (Chennaoui et al., 2015; Juliff et al., 2015; Lastella et al., 2015). Indeed, athletes have acknowledged sleep as their most important recovery modality (Venter et al., 2010). Sleep is essential to recover from the fatigue accumulated by athletes during both training and

competition. Unless an athlete recovers quickly their subsequent training, workload and ultimately performance will suffer (Boompa and Haff, 2009).

Chapter 3: Sleep and Nutrition Interactions

3.1 Chrononutrition

Chapter 3 investigates the between sleep and nutrition, while the number of studies investigating the effect of nutritional interventions on sleep are increasing (Samuels et al.,

2016; Irwin et al., 2016; Lastella et al., 2016, Peukhuri et al., 2012, Halson, 2013), more research is necessary in elite athletic populations. Unless an athlete recovers quickly their subsequent training, workload and ultimately performance will suffer (Boompa and Haff, 2009). If the athlete does not recovery properly fatigue accumulates resulting in maladaption and reduced performace, if this is not addressed the athlete can develop NFO or unexplained underperformance syndrome (UPPS) in the short term and ultimately over-training syndrome (OTS) in the longer term (Lewis et al., 2015a; Meeusen et al., 2013). Optimal nutrition, hydration and sleep are the most effective strategies for recovery in athletes. The adaptive response to training is dictated by a number of variables: duration, intensity, frequency and type of exercise in combination with timing, quality and quantity of nutrition both pre and post-exercise (Jeukendrup, 2017). Training adaptations can be maximised by optimal nutrition practices or reduced by suboptimal nutrition practices. Nutrition support must be periodised in relation to the demands of the athlete's daily training load and overall nutrition goals (Close et al., 2016). Researchers and practitioners now consider 'Competition Nutrition' which is performance focused and 'Training Nutrition' which is adaptation focused, as two separate entities (Close et al., 2016). Elite athletes are particularly vulnerable to sleep difficulties due to high training and competition demands (Walsh et al., 2021), as such investigations the sleep and recovery practices of athletes and potential nutritional interventions to improve sleep duration and quality are warranted (Ordóñez et al., 2017).

Chrononutrition refers to the relationship between food intake and the circadian clock system (Tahara and Shibata, 2014). Chrononutrition is essentially an extension of chronopharmacology. Chronopharmacology involves determining the timing of drug administration in relation to in targeted kinase activity, the protein quantities necessary to enhance the potency of a medication, and/or the absorption and excretion of a medication (Tahara and Shibata, 2014). Chronopharmacotherapy can be utilised to maintain or improve human health through the timing of medication in relation to circadian changes. The circadian system responds to external and internal signals because the oscillation period is not precisely 24 hours (Tahara and Shibata, 2014). The SCN receives environmental cues such as the light-dark cycle and additional information from other areas of the brain (e.g. when we eat or exercise). Nutrients such as glucose, amino acids, sodium, ethanol and caffeine, as well as the timing of meals can affect circadian rhythms (Froy, 2007). Recently the term Chrononutrition has been used to describe the interaction between food and the circadian clock system and how human's internal clock can be altered by changing the timing of food intake. Chrononutrition has been defined as including two aspects:

1. Timing of food intake or contributions of food components to the maintenance of health.
2. Timing of food intake or contributions of food components to rapid changes in or resetting of human's system of internal clocks.

(Tahara and Shibota, 2014).

Chrononutrition is becoming an area of increased interest in relation to nutrition and circadian system interactions. Neurotransmitters in the brain such as serotonin, gamma-aminobutyric acid (GABA), orexin, melanin-concentrating hormone, cholinergic, galanin, noreadrenaline and histamine are involved in the sleep-wake cycle (Saper et al., 2005), nutritional interventions that act upon these neurotransmitters may therefore influence sleep, and vice versa. Dietary precursors can influence the rate of synthesis and function of neurotransmitters. Serotonin synthesis is dependent on the availability of its precursor Tryptophan in the brain (Halson, 2013). Tryptophan is transported across the blood brain barrier by a system that shares transporters with a number of Large Neutral Amino Acids (LNAA). The ratio of Tryptophan:LNAA in the blood is vital to the transport of Tryptophan into the brain and can be increased by consumption of either Tryptophan or Tryptophan rich protein (Silber and Schmitt, 2010). In terms of general health further research involving functional foods and supplements is necessary to clarify the interactions between nutrition and the circadian system as there is potential to reduce the prevalence and burden of chronic diseases, through the promotion of sleep health. In terms of athletes, further research is necessary to clarify if functional foods and supplements can be used to benefit health, performance and/or recovery.

Light information obtained from the retina is the typical external entrainable factor in mammals, other external factors include food, temperature, exercise, drugs and psycho-social stress. Among these factors food has been shown to be the best circadian system synchronizer (comparable to light) (Garaulet and Madrid, 2010; Mistlberger, 2011; Shibata et al., 2010; Delezie and Challet, 2011). An understanding of circadian changes in the digestive system enables optimal timing of food. Humans can alter the timing of their 'internal clock' by altering the timing of food intake (Tahara and Shibata, 2014). Sleep deprivation can also impact appetite and glucose metabolism due to the influence of leptin (appetite supressant) and ghrelin (stimulates appetite) that may be produced by the pituitary-hypothalamus axis (Chandrasekaran et al., 2020).

The adaptive response to training is dictated by a number of variables: duration, intensity, frequency and type of exercise in combination with timing, quality and quantity of nutrition both pre and post-exercise (Jeukendrup, 2017). Training adaptations can be maximised by

optimal nutrition practices or reduced by suboptimal nutrition practices. Contemporary research has demonstrated the pivotal role of both macronutrient and micronutrient availability in regulating skeletal muscle adaptations to exercise (Heaton et al., 2017, Jeukendrup, 2017, Close et al., 2016). Nutrition support must be periodised in relation to the demands of the athlete's daily training load and overall nutrition goals (Close et al., 2016). Researchers and practitioners now consider 'Competition Nutrition', which is performance focused, and 'Training Nutrition', which is adaptation focused, as two separate entities (Close et al., 2016). There are numerous other effects of nutrition and interactions between nutrition and exercise that ultimately determine exercise performance, these effects and interactions and their impact on performance warrant further investigation.

3.2 Antioxidants

Antioxidants are any substance that significantly delay or prevent oxidative damage of a target molecule (Halliwell and Gutteridge, 2015). Cells contain an endogenous antioxidant system composed of enzymatic and non-enzymatic antioxidants. A range of antioxidants are active within the body including enzymatic (endogenous) and non-enzymatic (mainly dietary) antioxidants (Powers and Lennon, 1999), all of which can function at an extracellular and intracellular level. Antioxidant enzymes including Superoxide Dismutase (SOD), Catalase (CAT) and Glutathione Peroxidase (GPx) scavenge ROS (Bouayed et al., 2010; Finaud et al., 2006). Non-enzymatic antioxidants include a range of free-radical scavengers such as Vitamin A, Vitamin E, Flavonoids, Thiols and Iron, Copper, Zinc, Selenium and Manganese which act as enzymatic cofactors (Finaud et al., 2006). Synergy between endogenous and exogenous antioxidants protects the body from damage caused by OS and maintains redox balance. The efficiency of the antioxidant system depends on dietary intake of antioxidants and endogenous antioxidant enzyme production which can be affected by factors such as exercise, training, nutrition and aging.

Both the general population and athletes can benefit from nutritional and supplementation support to boost immunity and reduce acute and chronic inflammation during periods of increased training load and competition. Appropriate nutrient intake is necessary for the various cells of the immune system to function optimally and respond to challeneges e.g. viruses, bacteria and injury (Nieman and Mitmesser, 2017). Immune cells require substrates (glucose, amino acids and fatty acids) and micronutrients to divide and produce protective chemicals; move, engulf, and/or destroy pathogens and produce proteins (e.g. immunoglobulins and cytokines) and lipid mediators (prostaglandins, leukotrienes and

specialised pro-resolving mediators) (Nieman and Mitmesser, 2017). It is well established that exercise results in increased free-radical production in skeletal muscle (Stear et al., 2012). Research indicates that exercise-induced free-radical production results in oxidative damage to cells and plays a role in muscular fatigue during prolonged exercise (Powers, 2008). The fact that exercising muscles produces free radicals has motivated many athletes to consume antioxidant supplements in an attempt to reduce exercise induced free-radical damage and/or muscle fatigue. The antioxidant capacity of several dietary micronutrients is an emerging area of interest to support the endogenous antioxidant defence system of athletes and attenuate the negative effects of oxidative damage due to free radicals. Supplemental antioxidants such as Vitamin E, Vitamin C, Carotenoids, Flavenooids (e.g. Quercetin), Coenzyme Q10 and Anthocyanidins are commonly consumed by athletes in an attempt to reduce OS, following training. Optimal nutrition, hydration and rest are the most effective strategies for recovery in athletes (Robson-Ansley et al., 2009).

Dietary antioxidants (e.g. Vitamin C and Vitamin E) augment endogenous antioxidant content within skeletal muscle (Stear et al. 2009). Various studies have investigated the effects of antioxidant supplementation (Vitamin C, Vitamin E, Beta-Carotene, N-acetylcysteine, Zinc, Vitamin B_{12} and Folic Acid (Vitamin B_9) in relation to exercise induced OS (Nieman and Mitmesser, 2017, Calder, 2013, Petersen et al., 2012; Ferreira et al., 2011). Vitamin E is a fat-soluble vitamin made up of several isoforms known as tocopherols, with α-tocopherol being the most active and abundant (Fuchs et al., 2003). Vitamin E is an important antioxidant due to its abundance within cells, mitochondrial membranes and its ability to act directly on ROS (Evans, 2000). Vitamin E reacts with other antioxidants such as Vitamin C, Beta-Carotene and Lipoic Acid, which have the capacity to regenerate Vitamin E from it oxidised form (Coombes et al., 2001). Trained participants have demonstrated increased Vitamin E status (Gleeson, 2007; Dawson et al., 2002; Goldfarb, 1999). However, supplementation with high doses (800IU/day) of Vitamin E did not counteract OS in triathletes, the intervention group demonstrated higher levels of post-race inflammation and OS than the control group (Nieman et al., 2004). N-Acetylcysteine (NAC) blunts acute exercise induced adaptations in skeletal muscle, specifically phosphorylation of Jun N-terminal kinase (JNK) and mRNA expression of Manganese Superoxide Dismutase (MnSoD) (Petersen et al., 2012), but enhances acute exercise performance, specifically repeated sprint performance by 50% (Yo-Yo Intermittent Recovery Test) ($p<0.05$) and significantly reduced rate of perceived exertion (RPE) ($p=0.038$) (Cobley et al., 2011). Similarly, NAC supplementation (150 mg/kg dissolved in 100ml saline) resulted in a 32%

(p<0.05) increase in time to fatigue during a submaximal handgrip exercise (Matuszczak et al., 2005). However, it has been noted that NAC supplementation can induce acute gastrointestinal discomfort, with doses ≤70mg/kg not eliciting any adverse effects (Ferreira et al., 2011).

Vitamin A is a fat-soluble vitamin present in many lipid substances, Beta-Carotene can be converted into Vitamin A when necessary within the body (Finaud et al., 2006). Beta-Carotene deactivates ROS and reduces lipid peroxidation, supplementation has demonstrated beneficial effects in terms of exercise induced OS (Schröder et al., 2001). Vitamin C is a water-soluble vitamin and is extremely effective in extracellular fluids but is also effective in the cytosol (Finaud et al., 2006). Vitamin C is abundant in tissues where ROS production is important, this has been defined as an adaptation to OS (Palmer et al., 2003). Vitamin C deficiency impairs athletic performance and supplementation, particularly in conjunction with other antioxidants (e.g. Vitamin E) aids maintenance of adequate Vitamin C status (Laursen, 2001). Modest improvements in recovery (reduced plasma Il-6 levels 2 hours post exercise and muscle soreness) from unaccustomed exercise (90mins intermittent shuttle running) were demonstrated following 2 weeks of Vitamin C supplementation (400mg/day) (Thompson et al., 2001).

It must be noted that antioxidants are heterogeneous, they function in a distinct manner and do not solely regulate ROS (Murphy et al., 2011). Consumption of an antioxidant does not guarantee that the compound will act as an antioxidant within the body, therefore positive findings from one antioxidant or combination of antioxidants cannot be generalised (Nieman and Mitmesser, 2017). It has been suggested that a high intake of antioxidants could potentially reduce training adaptations (Gomez-Cabrera et al., 2015). It is accepted that repeated exercise bouts (i.e. training) induce disruption in skeletal muscle homeostasis that regulate training adaptations (Cobley et al., 2015a; Cobley et al., 2015b). It has been reported that high doses of antioxidants could reduce training adaptations of muscle mitochondrial biogenesis and VO_{2max} (Gomez-Carbrera et al., 2008). However, not all antioxidant studies have demonstrated negative effects and it has been suggested that the specific antioxidant used, the dose and timing of ingestion all affect outcomes (Mankowski et al., 2015).

Further research is necessary to develop practical guidelines for antioxidant supplementation to enhance training adaptation and/or post exercise recovery. It must be noted that the majority of research investigating the effects of antioxidant supplementation to date, has employed high doses. Given the adoption of a ‘food first’ approach by many athletes (Close et al., 2016), there is scope for investigation of food based interventions

designed to promote athlete recovery and/or enhance sleep health. At present, there has been no data published that suggests that consumption of high antioxidant fruit and/or vegetables reduces training adaptations.

3.3 Tryptophan

Tryptophan is an essential amino acid that is a precursor to Serotonin and Melatonin and has the ability to cross the blood-brain barrier by competing for transport with other large neutral amino acids (LNAA) (Halson, 2013). Conversion to Serotonin is dependent on sufficient precursor availability in the brain. An increase in brain Tryptophan occurs when the ratio of free tryptophan to branched chain amino acids increases (Halson, 2013), following Tryptophan conversion to Serotonin, Melatonin is produced. Early research concluded there was a significant positive effect of Tryptophan (doses $\geq$ 1g) on sleep, particularly sleep latency (time to sleep) (Hartman, 1983). Recently, doses $\leq$ 1g have produced comparable results but it has been noted that doses $\leq$ 5g do not appear to affect sleep stages (Silber and Schmitt, 2010), and Tryptophan may have minimal effects on sleep in healthy adults who fall asleep easily.

Dietary sources of Tryptophan include milk, turkey, chicken, fish, eggs, pumpkin seeds, beans, peanuts, cheese, and leafy green vegetables. Dietary Tryptophan has been shown to improve sleep, in a comparison of the effect on sleep of tryptophan enriched muesli bars (de-oiled gourd seed: 22mg/1g protein) plus glucose with bars containing 250mg pharmaceutical Tryptophan plus glucose and glucose alone (Hudson et al., 2005). The muesli bars and the pharmaceutical dose produced similar results (5.5% and 6.5% respectively) for reduction of time awake during the night (Hudson et al., 2005), indicating that relatively small doses (250mg) of dietary Tryptophan can positively impact sleep. However, pharmaceutical Tryptophan has been associated with eosinophilia myalgia syndrome (EMS), a serious medical condition which can be fatal (Hudson et al., 2005).

The milk protein, α-lactalbumin has been reported as having the highest natural levels of tryptophan among all protein food sources (Heine et al., 1996). Ingestion of α-lactalbumin enriched Whey protein, significantly ($p<0.05$) increased Tryptophan:LNAA by 48% compared to a casein enriched diet (Markus et al., 2000). In a similar study, healthy adults (n=14) with sleep complaints consumed milkshakes containing either α-lactalbumin (20g) or a casein placebo, evening ingestion of α-lactalbumin resulted in a 130% increase in Tryptophan:LNAA prior to bed and modest but significant reduction in sleepiness and improved alertness the following morning (Markus et al., 2005). Tryptophan depletion

studies have demonstrated decreased Tryptophan plasma concentrations affected sleep fragmentation (Arousal Index [events/h]), REM sleep latency (the interval between first epoch of N2 and the first epoch of REM sleep), and REM density (the cumulated duration of each REM burst divided by the duration of each REM sleep period) compared to baseline and placebo (Arnulf et al., 2002; Bhatti et al., 1998).

3.4 Melatonin

Melatonin is a hormone secreted naturally by the Pineal Gland, particularly at night. Studies have indicated that pharmacological doses of Melatonin are effective for inducing and maintaining sleep in both children and adults with normal sleeping patterns (Hashimoto et al., 1996). However, the effect is relative to the person's endogenous Melatonin levels. In many Western countries, Cow's milk has traditionally been considering a sleep promoting beverage. Melatonin is a naturally occurring compound in cow's milk, but its concentration increases significantly if cows are milked in darkness at night (Peukhuri et al., 2012). Among the molecules that offer chemical protection against OS, Melatonin and related compounds are particularly effective (Ramis et al., 2015; Galano et al., 2011; Hardeland, 2005). Melatonin has extremely low toxicity even at relatively high doses (Jahnke et al., 1999) and can easily cross physiological barriers due to its optimal size, partial water solubility and high lipid solubility (Ceraulo et al., 1999; Bonnefont-Rousselot et al., 2010). However, it must be noted there appears to be no added benefit of doses > 3mg (Bonnefont-Rousselot et al., 2010).

Metabolism does not reduce Melatonin antioxidant protection against OS; it is maintained or even increased due to the antioxidant capacity of its metabolites (Gurer-Ohran et al., 2015). In humans Melatonin is the final product in the metabolism of the amino acid Tryptophan (Trp), and is secreted by the pineal gland at the onset of darkness, triggering sleep due it its hypothermic effect (Halson, 2008). Ingestion of Melatonin affects sleep propensity and has hypnotic effects enhancing sleep quality and duration (Brzezinski, 1997), pharmacological Melatonin is commonly used in athletes to manipulate sleep patterns. A positive effect of dosages of either 0.3mg or 1mg of exogenous Melatonin on sleep latency have been observed, when administered between 6:00pm and 8:00pm (Pires et al., 2001). However, the impact was time dependent as the 0.3mg dose increased sleep latency and there was no effect when the 1mg dose was administered at 9:00pm (Pires et al., 2001). A dose response relationship was not evident as the 0.3mg dose, which is similar to endogenous Melatonin concentrations, was as effective as the 1mg dose when administered between

6:00pm and 8:00pm. Tart Cherries contain high concentrations of melatonin. Significantly reduced insomnia severity index scores (13.2 ± 2.8 versus control 14.9 ± 3.6; $p<0.05$) and wake after sleep onset time (62.1 ± 37.4min versus control 79.1 ± 38.6; $p<0.01$), was obserbved in older adults following consumption of a Tart Cherry Juice blend, compared to a placebo (Pigeon et al., 2010). Research was conducted to investigate if Melatonin is the mechanism of Tart Cherry Juice (2 x servings of 30mls concentrate) sleep enhancement and improved sleep time and quality (Howatson et al., 2012). Total Melatonin content was significantly elevated and significant increases in time in bed (+24 minutes), total sleep time (+34 minutes) and sleep efficiency total (82.3%) and a significant reduction in daytime napping (-22%) ($P<0.05$) were associated with cherry juice supplementation (Howatson et al., 2012). Although no difference was observed in timing of the Circadian Rhythm, there was a trend to a higher mesor and amplitude. The range of phenolic compounds in Cherries which have anti-inflammatory and antioxidant properties may enhance post exercise recovery as well as sleep (McHugh, 2011).

It has been proposed that Melatonin may be synthesised in mitochondria (Slominski et al., 2014), making Melatonin and its metabolites available to protect the muscle against OS. Melatonin also increases the protective effects of Glutathione, Vitamin C and Trolox, through regeneration by electron transfer processes (Galano et al., 2016). A placebo controlled double blind study on older adults, demonstrated a food based supplement containing 5mg Melatonin, 225mg Magnesium and 11.25mg Zinc, significantly ($P<0.05$) improved subjective measures of sleep and total sleep time assessed via actigraphy (Rondanelli et al., 2011). The effects were attributed to the crucial roles both magnesium and Zinc have in endogenous melatonin production (Rondanelli et al., 2011). Indeed, there is evidence that Tart Cherry Juice supplementation post-exercise reduces muscle soreness (Howatson et al., 2010), however, it is unclear if this as a result of a direct scavenging effect (Cobley et al., 2015b). Recent research has demonstrated Tart Cherry Juice supplementation (30mls, twice per day for 7 days) reduced the post-exercise decline in functional performance following intermittent sprint activity (maximal voluntary isometric contractions, 20m sprint, counter movement jump and 505 agility test), delayed onset muscle soreness (DOMS) and reduced inflammatory response (Il-6) (Bell et al., 2016). With regards the reduction in post-exercise inflammatory response, in practice the researchers suggested that this might be beneficial during periods of high volume training e.g. (Pre-season) or where athletes are required to produce multiple performances in a short space of time (e.g. double training sessions), when recovery periods are short (Bell et al., 2016).

3.5 B Vitamins and Magnesium

Vegetables and wholegrains are a feature of self-help advice to improve sleep due to their B Vitamin and Magnesium content (Halson, 2008). Vitamin B_{12} contributes to Melatonin secretion, Vitamin B_6 is involved in the synthesis of Serotonin from Tryptophan and Niacin (Vitamin B_3) may elicit a Tryptophan sparing effect (Peuhkuri et al., 2012). Mixed effects have been observed, with different doses of Vitamin B_{12} on sleep-wake rhythm and delayed sleep phase syndrome, while no effect was observed for sleep duration (Peukhuri et al., 2012). Niacin (Vitamin B_3) has been shown to increase REM sleep in healthy adults, and improved sleep efficiency in participants with moderate to severe insomnia (Peukhuri et al., 2012). Niacin is synthesised from dietary Tryptophan via the Kynurenine Pathway. It has been suggested that administration of Niacin causes an increase in Nicotinamide Adenine Dinucleotide, which may reduce Tryptophan conversion to Niacin, increasing Tryptophan availability for the synthesis of Serotonin and Melatonin (Peukhuri et al., 2012). Magnesium is also believed to enhance Melatonin secretion and act as a GABA agonist, the main inhibitory neurotransmitter that acts on the CNS (Peukhuri et al., 2012). It has been noted that deficiencies in B Vitamins and Magnesium may also disrupt sleep (Peukhuri et al., 2012).

3.6 Phenolic Compounds

Phenolic compounds have been widely investigated in relation to antioxidant capacity and their ability to protect against OS (Galano et al., 2016). Phenolic compounds scavenge free radicals by Hydrogen Atom Transfer (Cao et al., 2014), Proton Coupled Electron Transfer (Nakayama and Uno, 2015) and Sequential Proton Loss Electron Transfer (Marković et al., 2015). Phonolic compounds are frequently consumed in the human diet as they are abundant in a wide variety of foods and beverages e.g. fruit, vegetables, wine, coffee and tea (Perron and Brumaghim, 2009). However, it has been reported phenolic compounds have poor bioavailability, in an investigation involving an average intake of 10-100mg of phenolic compounds, plasma concentrations rarely exceeded 1mM (Scalbert and Williamson, 2000). However, it has been suggested that total phenol concentration in plasma is higher due to the presence of metabolites formed in tissue and by the colonic microflora, which were not accounted for (Scalbert and Williamson, 2000).

3.7 Kiwifruit

The health benefits of consuming fruit are well documented (Boeing, 2012). Kiwifruit appeal due to their nutrient density, health benefits and palatability (Ferguson and Ferguson,

2003). Kiwifruit are the edible berry of the *Actinidia* vine, the most common are *Actinidia Deliciosa* (Green Kiwifruit) and *Actinidia Chinensis* (Gold Kiwifruit) (Ferguson and Ferguson, 2003). The chemical composition of Kiwifruit is of considerable interest in terms of nutritional value and health benefits. The composition of Kiwifruit varies depending on multiple factors such as horticulture, region, soil type, storage, ripening condition and maturity of the fruit (Drummond, 2013). Interest in the antioxidant capacity, enzyme, polyphenolic and phytochemical content of Kiwifruit has increased steadily over the last decade. It has been suggested that the various bioactive components in Kiwifruit may act synergistically affecting various physiological and metabolic processes (Singletary, 2012). Contemporary research has focused on the health benefits of Kiwifruit particularly in relation to antioxidant capacity, digestion, iron nutrition, metabolic health and immune function (Singletary, 2012). Kiwifruit contain significant amounts of Vitamin C but also contain a range of other health promoting nutrients such as Vitamin E, Vitamin K, Folate, Beta-Carotene, Lutein, Potassium, Copper and Fibre (Stonehouse et al., 2012). Compared to controls, Kiwifruit fed mice exhibited significantly lower levels of urinary OS markers, while Cytokine production was increased indicting a potential positive affect on immune function (Iwasawa et al., 2010).

Kiwifruit are nutritionally dense containing a range of nutrients that can benefit sleep and recovery including Serotonin, Vitamin C, Vitamin E, Folate, Anthocyanidins and Carotenoids (Lin et al., 2011). Kiwifruit also contain Lutein, Zeaxanthin and a range of phytochemicals which have antioxidant properties (Fiorentino et al., 2009). The Vitamin C content of Kiwifruit has been demonstrated to be high across a range of species *Actinidia Latifolia* 671-2140mg/100g FW (Huang et al., 2004; Du et al., 2009) and *Actinidia Kolomikta* 1008mg/100g FW (Latocha et al., 2010). Kiwifruit contain relatively high levels of Vitamin E and it was originally assumed that Vitamin E was contained mainly in Kiwfruit seeds, reducing bioavailability (Ferguson and Ferguson, 2002). However, recent analysis has highlighted that the main α-tocopherol form of Vitamin E in Kiwifruit, is contained in the flesh in cell membranes, suggesting otherwise (Fiorentino et al., 2009). A new form of Vitamin E, α-tocomonoenol was also discovered in Kiwifruit which was shown to have a similar antioxidant capacity as α-tocopherol (Fiorentino et al., 2009). The bioactivity of Kiwifruit is attributed to its Vitamin C, Vitamin E, Folate, Naringenin, Quercetin and Epicatechin content (Sun-Waterhouse et al., 2009; Collins et al., 2001).

Regular consumption of Kiwifruit has been found to significantly ($p \leq 0.05$) increase plasma Vitamin C (Prior et al., 2007; Beck et al., 2011; Hunter et al., 2012) Vitamin E

(Chang and Liu, 2009; Hunter et al., 2012) and Lutein/Zeaxanthin (Bøhn et al., 2010; Beck et al., 2011; Hunter et al., 2012) concentrations. Kiwifruit consumption in conjunction with a meal was associated with increased plasma antioxidant capacity (Prior et al., 2007), and may therefore play a role in combatting OS. A small, randomised crossover study comprising of 6 males and 8 females, demonstrated consumption of varying doses of Kiwifruit (1-3/d x 3 weeks, with 2-week washout between doses) resulted in a significant increase in plasma Vitamin C levels (Collins et al., 2003). Compared to baseline, consumption of 2 Kiwifruit daily significantly raised plasma Vitamin C levels by 20% (73μM±4; $p<0.01$), while consumption of 3 Kiwifruit daily raised plasma Vitamin C levels by 26% (77μM±3; $p<0.001$) (Collins et al., 2003). Additionally, improved antioxidant status was evident, lymphocytes isolated from blood collected from participants demonstrated decreased sensitivity to oxidative attack by (H_2O_2), in vitro and endogenous oxidation of lymphocyte DNA was also decreased (Collins et al., 2003). However, it must be noted that the results of this study may not be generalizable due to the very small sample size.

A study involving healthy adult volunteers (n=25) who self-reported sleep disturbance demonstrated consumption of 2 Kiwifruit, 1 hour before bedtime for 4 weeks significantly improved actigraphy measured total sleep time (16.9%) and sleep efficiency (2.4%) ($p<0.001$) (Lin et al., 2011). Self-report measures also improved significantly, wake time after sleep onset reduced (-28.9%), sleep latency reduced (-35.4%) while sleep efficiency increased (5.4%) ($p<0.002$) (Lin et al., 2011). It is clear that sleep quality was significantly improved following the 4-week Kiwifruit intervention. These promising findings warrant further investigation within athletic populations. The Serotonin content in Kiwifruit may contribute to improved sleep while the rich antioxidant content may supress free radical expression and inflammatory cytokines. Folate deficiency has been linked to insomnia and restless leg syndrome, the folate in Kiwifruit may improve folate status and consequently improve sleep (Lin et al., 2011). Although Folates are widely consumed in the diet, they are destroyed by cooking or processing. Kiwifruit are consumed in their raw form and contain 0.23±0.04μg/g of total folate, 80% higher than carrot juice and 15% higher than orange juice (Wyatt et al., 1970), and it has been estimated that a Kiwifruit contains 10% of the average daily requirement (Ferguson and Ferguson, 2003).

Numerous processes including physiological or emotional stress and inadequate sleep increase the concentration of ROS. The abundance of antioxidants and other health promoting compounds in Kiwifruit might be utilised to promote athlete recovery and improve sleep quality. Further research is warranted to investigate the potential role of

Kiwifruit in reducing OS, improving recovery and promoting sleep. Kiwifruit consumption potentially increases resistance to OS through up regulation of genes involved in DNA repair (Bøhn et al., 2010). Further research is necessary to investigate the potential benefits and practical application of Kiwifruit supplementation to promote post-exercise recovery and promote or improve sleep in athletes.

3.8 Conclusion

In general, sleep can be promoted either by inhibiting wake-promoting mechanisms or by increasing sleep promoting factors either through nutritional interventions or other means (Peukhuri et al., 2012). While the number of studies investigating the effect of nutritional interventions on sleep are increasing (Samuels et al., 2016; Irwin et al., 2016; Lastella et al., 2016, Peukhuri et al., 2012, Halson, 2013), future research needs to focus on specific populations, such as athletes (elite and sub-elite). Antioxidants are commonly consumed by athletes in an attempt to reduce oxidative stress, following training. Further research is necessary to develop practical guidelines for antioxidant supplementation to enhance training adaptation and/or post-exercise recovery. It must be noted that the majority of research investigating the effects of antioxidant supplementation to date, has employed high doses. Given the adoption of a 'food first' approach by many athletes, there is scope for investigation of 'functional food' (e.g. Kiwifruit) based interventions designed to promote athlete recovery and/or enhance sleep quality and quantity. Research is necessary to investigate the potential role of Kiwifruit in reducing oxidative stress, improving recovery and promoting sleep. Further research is also necessary to investigate if the quality, quantity and timing of sleep can be manipulated to promote the uptake of supplementations designed to reduce OS, following training. Research investigating the sleep and physiological outcomes from ingestion of 'functional foods' would add to the current literature. Ideally this research will lead to the development of nutritional interventions for optimising sleep quality, sleep quantity and enhancing post-exercise recovery.

3.9 Summary

The purpose of this research was to investigate the association between sleep, nutrition and athlete recovery. It is clear from the research reviewed in Chapters 1-3 that sleep is important for athletes and nutrition has a key role to play in both sleep (i.e. chrononutrition) and recovery, and the research is emerging and evolving (see Chapter 2 & 3). The aim of Chapters 1-3 was to scope the literature to determine a) was there a problem with sleep in athletes, b) is it related to recovery and c) what non-pharmacological options were there to

manage both. Sleep can be promoted either by inhibiting wake-promoting mechanisms or by increasing sleep promoting factors (Peukhuri et al., 2012). Athletes may experience significant problems sleeping due to the lack of an appropriate sleep routine due to ever changing training and competition schedules, general lifestyle factors and other sleep-incompatible behaviours e.g. late night blue light exposure (Tuomiletho et al., 2016). Irregular sleep-wake patterns influence the homeostatic and circadian regulation of sleep, which reduces both sleep quality and quantity (Fischer et al., 2008). For athletes, post-competition routines and heightened arousal (i.e. medical care, recovery strategies, meals, media commitments and travel) can lead to later bedtimes, which can adversely affect sleep quality, sleep quantity and recovery. Reduced sleep is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis (Fullagar and Bartlett, 2016), blunting training adaptations and recovery which may lead to compromised performance

The importance of sleep for not only athletes' recovery but also their performance is clear. When sleep is reduced to < 7 hours there is a detrimental impact on both cognitive performance (i.e. alertness, reaction time, memory and decision making) and physical performance while injury risk is increased (Charest and Grandner, 2020; Laux et al., 2015; LeMeur et al., 2013). Adequate sleep including afternoon naps can counteract the negative performance, cognitive, immunity, oxidative stress (OS), non-functional overreaching (NFO) and increased pain outcomes that are consequences of sleep debt (Walsh et al., 2021). Sleep is essential to recover from the fatigue accumulated by athletes during both training and competition, and athletes have reported sleep as their most important recovery modality (Venter et al., 2012). Unless an athlete recovers quickly their subsequent training, workload and ultimately performance will suffer (Boompa and Haff, 2009). If the athlete does not recovery properly fatigue accumulates resulting in maladaption and reduced performace, if this is not addressed the athlete can develop NFO or unexplained underperformance syndrome (UPPS) in the short term and ultimately over-training syndrome (OTS) in the longer term (Lewis et al., 2015a; Meeusen et al., 2013). Elite athletes are particularly vulnerable to sleep difficulties due to high training and competition demands (Walsh et al., 2021), as such investigations the sleep and recovery practices of athletes and potential nutritional interventions to improve sleep duration and quality are warranted (Ordóñez et al., 2017).

The research demonstrated a problem with sleep in athletes and a relationship with recovery, it is also clear that Chrononutrition is an area that shows promise, therefore it is

necessary to characterise sleep in athletes (Chapter 4), and under specific circumstances (Competition travel – Chapter 5). This will allow the indetification of the specific sleep and recovery areas of concern in this population with a view to implementing a chrononutritional intervention (Kiwifruit – Chapter 6) with a view to seeing whether it improves sleep and recovery.

Chapter 4: The sleep and recovery practices of athletes.

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4.1 Introduction

Post-exercise recovery is vital for all athletes and the balance between training stress and physical recovery must be managed to maximise the adaptation from, and performance in subsequent training sessions or competitions (Venter, 2010; Hartwig et al., 2009). The repetitive demanding nature of a competitive season can test athletes' physiological and

psychological capacity. Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery and performance in subsequent training/competition (Venter, 2010). The regulation of performance during exercise has increasingly been interpreted as a cohesive, multifaceted process involving both the central nervous system (CNS) and peripheral nervous system (PNS) (Knicker et al., 2011; Noakes, 2012). While there is debate whether the regulation of exercise performance is derived primarily from the CNS or PNS (Gibson and Noakes, 2004) and whether the regulation is conscious (Marcora, 2008) or anticipatory (Marino, 2004); changing CNS drive and motor unit recruitment is widely considered to be associated with fatigue (i.e. reduced physical and mental capacity) (Knicker et al., 2011). In contrast, physical fatigue has many potential drivers (dehydration, glycogen depletion, muscle damage and mental fatigue), recovery of muscle function is predominantly a matter of reversing the main causes of fatigue. Sleep deprivation (< 7 hours) increases circulating stress hormones (e.g. cortisol) (Meerlo et al., 2008); decreases the regeneration of carbohydrate stores (i.e. glycogen) (Morselli et al., 2010); deregulates appetite and impacts on energy expenditure (Knutson et al., 2007); increases catabolism and reduces anabolism, impacting the rate of muscle repair (MPS) (Atrooz and Salim, 2020; Fullagar and Bartlett, 2016). Therefore, sleep plays a key role in facilitation of post exercise recovery or the reduction of fatigue and the reversal of the processes that lead to fatigue (De Pauw et al., 2013).

Athletes experience stress for various reasons (e.g. training, competition, travel and lifestyle) including periods of both acute and residual fatigue due to heavy training and competition schedules (Borresen and Lambert, 2009). For example, field based team sports are characterised by repeated bouts of intermittent activity (sprinting) with short rest periods representing high physiological stress (Spencer et al., 2005), neuromuscular stress (Rampini et al., 2011; Duffield et al., 2012) and high rates of perceived exertion (i.e. how hard exercise seems) (Impellizzeri et al. 2004). While individual endurance athletes experience fatigue due prolonged activity resulting in glycogen depletion, thermal stress and/or dehydration (Costa et al., 2019). Relative stress is accumulated when successive bouts of training are combined with suboptimal recovery (under-recovery) impacting subsequent performance in training and competition (Bishop et al., 2008). It has been suggested that decreasing the natural timeframe of the bodies' regenerative processes via recovery strategies is vital for performance (Barnett, 2006). Such recovery strategies can be divided into physiological strategies (e.g. sleep, cold water immersion, cryotherapy, contrast therapy, massage and compression), pharmacological (e.g. non-steroidal anti-inflammatory drugs [NSAIDs]) and

nutritional (e.g. nutrient timing, composition and supplementation) (Minett and Duffield, 2014). However, it must be noted that some research has suggested that interfering with the body's natural recovery processes particularly inflammatory responses and OS could reduce training adaptations (Harty et al., 2019). A recent review by Owens et al., (2019) addressed these concerns in relation to the application of nutritional strategies to reduce muscle damage, suggesting that a periodised approach is necessary to achieve the greatest benefits for the athlete.

4.2 Sleep

As discussed in Chapter 1, sleep has previously been self-reported as the most important recovery modality utilised by both elite and sub-elite athletes (Erlacher et al., 2011; Venter et al., 2010; Tuomiletho et al., 2016). Furthermore, Leeder et al., (2012) suggested that sleep was a new frontier in performance enhancement for athletes. Sleep has a restorative effect on the immune system and the endocrine system (Chennaoui et al., 2015; Juliff et al., 2015; Lastella et al., 2015), facilitates the recovery of the nervous and metabolic cost of the waking state and has an integral role in cognitive function (Frank, 2006). The relationship between sleep, nutrition and recovery is an emerging area of interest (Halson, 2019; Biggins et al., 2019; Lastella et al., 2018; Bonnar et al., 2018; Tuomilehto et al., 2016; Watson, 2017; Gupta et al., 2017; Samuels et al., 2016; Irwin et al., 2016; Lastella et al., 2016, von Rosen et al., 2016; Milewski et al., 2014; Venter, 2014; Halson, 2013; Peukhuri et al., 2012). It has been hypothesised that sleep, especially slow wave sleep (N3) is vital for physical recovery, due to the relationship with growth hormone release (Palaniappan and Thenappan, 2015; Halson, 2013). Tuomiletho et al. (2016), investigated the sleep patterns of professional male Ice Hockey players (n = 23) using PSG and found mean total sleep time was 415 mins; 95% CI 378-450. Leeder et al. (2012) investigated the sleep of elite athletes (n = 47) using actigraphy and compared to non athletic controls (n=20), elite athletes' mean sleep time was relatively low (6:55 ± 0.43 hr:min), they also had a lower sleep efficiency (80.6 ± 6.4 %), higher sleep fragmentation index (36.0 ± 12.4) and higher sleep onset latencies (18.2 ± 16.5 mins). In healthy adults when sleep is reduced to < 7 hours cognitive performance (i.e. alertness, reaction time, memory and decision making) is adversely affected (LeMeur et al., 2013). An association between sleep duration (< 8hours) and injury risk has been identified in adolescent athletes (RR = 2.1; 95% CI: 1.2 - 3.9; P = 0.01) (Milewski et al., 2014). A similar study of Swedish adolescent elite athletes (n = 340; 178 males and 162 females) demonstrated that achieving the National Sleep Foundation (Hirshkowitz, 2015) sleep

guidelines (> 8 hours) reduced injury risk by 61% (OR: 0.39; 95% CI 0.17 - 0.96) (von Rosen et al., 2016). In a recent study of endurance athletes (n = 95) new injury risk significantly increased with 14 day lag < 7h/day sleep quantity (HR = 1.51, 95% CI = 2.02 – 1.13, p<0.01) (Johnston et al., 2020). Optimal sleep duration is subject to individual variance, according to the National Sleep Foundation (Hirshkowitz et al., 2015), however, 7 – 9 hours sleep is recommended for adults. However, it has been argued that elite athletes may require more quality sleep than non-athletes (Walsh et al., 2021; Sargent et al., 2021; Bird, 2013).

Sleep inadequacy is common in athletes and can be attributed to lack of an appropriate sleep routine due to changing training schedules, timetables and other sleep-incompatible behaviours e.g. late night blue light exposure (Tuomilehto et al., 2016; Bird, 2013). Previous research in athletes has reported sleep durations < 7 hours (Roberts et al., 2019), long sleep onset latency (Tuomilehto et al., 2016, Schaal et al., 2011), daytime sleepiness (Swinbourne et al., 2016), and daytime fatigue (Sargent et al., 2014). Studies investigating sleep quality in elite athletes have demonstrated that 50% - 80% experience sleep disturbance and 22% - 26% experience highly disturbed sleep (Gupta et al., 2017; Swinbourne et al., 2016; Samuels, 2008). Irregular sleep-wake patterns influence the homeostatic and circadian regulation of sleep, which reduces both sleep quality and quantity (Fischer et al., 2008). For athletes, post-completion routines and heightened arousal (i.e. medical care, recovery strategies, meals, media commitments and travel) can lead to later bedtimes, which can adversely affect sleep quality and quantity. Reduced sleep is associated with increased catabolic and reduced anabolic hormones which results in impaired MPS (Fullagar et al., 2016), potentially blunting training adaptations and recovery.

The inconvenience and stress of travel due to competition schedules contributes to both mental and physical fatigue. The process of travel, jet-lag or arriving at a destination in the middle of the night can cause circadian rhythm disruption along with associated stress, restricted motion and unfamiliar surroundings creating further sleep disruption (Bishop, 2004) and poorer quality of sleep (Richmond et al., 2007). The repetitive demanding nature of a competitive season can also test athletes' physiological and psychological capacity, reinforcing the athletes' need for quality sleep. Actigraphy based sleep assessments reveal suboptimal sleep in athletes i.e. low TST and high WASO causing low sleep efficiency and resultant low sleep duration (Lastella et al., 2015; Leeder et al., 2012) which improves following a rest day (Fowler et al., 2014). However, the athletes' experience of suboptimal sleep remains unclear as sleep need varies between individuals; some may report poor sleep while objective measures indicate sufficient sleep (Halson, 2019). Therefore, subjective

measures of sleep quality, quantity and timing are a valuable addition to objective sleep assessments. Subjective markers of sleep (e.g. total sleep time, time in bed, sleep efficiency, sleep quality and sleep onset latency), combined, can highlight the sleep need and recovery status of athletes and identify areas to be addressed in terms of sleep optimisation. Moreover, the use of subjective measures within an athletic population allows the assessment of large cohorts of athletes that are difficult to access i.e. elite athletes.

Animal models have demonstrated that nutrients such as glucose, amino acids, sodium, ethanol and caffeine, as well as the timing of meals can affect circadian rhythms (Froy, 2007). Neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), orexin, dopamine, melanin-concentrating hormone, galanin, noradrenaline and histamine that are involved in the sleep-wake cycle (Saper et al., 2005), are affected by nutrition. In terms of recovery, the adaptive response to training is dictated by a number of variables: duration, intensity, frequency and type of exercise in combination with timing, quality and quantity of nutrition both pre and post-exercise (Jeukendrup, 2017). Recovery can be maximised by optimal nutrition practices or reduced by suboptimal nutrition practices. Contemporary, research has demonstrated the pivotal role of both macronutrient and micronutrient availability in regulating skeletal muscle adaptations to exercise (Heaton et al., 2017, Jeukendrup, 2017, Close et al., 2016). It is important to characterise the sleep quality, quantity and recovery practices of sub-elite and elite athletes. This study aimed to investigate: (i) the quality, quantity and timing of sleep among sub-elite and elite athletes and (ii) the recovery/stress balance of sub-elite and elite athletes and (iii) supplement use and alcohol intake of sub-elite and elite athletes. The study also aimed to investigate the difference between elite and sub-elite athletes in terms of their subjective sleep, recovery and nutritional practices. It was hypothesised that the sleep, recovery and nutrition practices of elite athletes would be superior to those of sub-elite athletes.

4.3 Methodology

4.3.1 Participants

A sample (n = 338) comprising of elite (n = 115; male n = 74 and female n = 41) and sub-elite (n = 223; male 129 and female 94) athletes was recruited from both Ireland and the United Kingdom (see Table 2.1). The elite athletes were recruited directly through Sport Ireland and the national governing bodies (NGBs) of each sport within Ireland and the United Kingdom. The sub-elite athletes were recruited via social media and the researcher's network within high performance sport. The recruitment e-mail and social media posts

included the inclusion and exclusion criteria and a link to the online questionnaire. In line with Swann et al., (2015) elite athletes were defined as: (a.) currently receiving support/funding through the international carding scheme and/or (b.) members of a national/professional team or a recruitment/academy squad and/or (c.) nationally ranked in their sport. Sub-elite athletes were defined as those competing at a regional, university and/or national level of organised sport that trained and/or competed for a combined minimum of 400 minutes per week. Athletes, at either level, were excluded if they were (i.) aged ≤ 18 years (ii.) training and competing for < 400 minutes per week or (iii.) had a diagnosed sleep disorder.

4.3.2 Procedure

All eligible athletes were invited to take part in an online survey. All procedures were approved by the research ethics committee of the Faculty of Health and Life Sciences, Northumbria University. After reading the participant information sheet (See Appendix 9.3, participants were invited to provide informed consent then completed an online survey on Qualtrics[xm] which consisted of a battery of previously validated and reliable widely used questionnaires assessing sleep, recovery and nutritional practices. The online survey was available between 1/7/17 and 31/12/17. Following completion of the survey participants received a debrief sheet with details of how they could contact the researcher if they wished to receive feedback from the survey.

4.3.3 Measures

In the initial section of the survey the participants completed demographic data. Participants recorded their gender, age, body mass (kg), height (cm), sport, athlete type (elite or sub-elite), phase of season (pre-season, competition or off-season), normal training time (before 8am, 8am to 5pm and after 5pm) and training/competition duration per week (mins).

4.3.4 EuroQoL (EQ-5D-5L)

The EQ-5D-5L is a self-report measure of health status as defined across five dimensions: mobility, self-care, activity, pain and depression/anxiety with one question per dimension. Each dimension is scored on a 5-point Likert Scale (0=No problem to 5=Severe problem) (Herdman et al., 2011). The EQ-5D-5L also includes a visual analogue scale on which respondents are instructed to rate their perceived current health state (0-100). The EQ-5D-5L has capacity to discriminate between slight, moderate and severe issues within each domain compared to previous versions (Davies et al., 2017).

4.3.5 Pittsburgh Sleep Quality Index (PSQI)

The PSQI is a self-report measure of sleep quality (Buysse et al, 1989). The PSQI consists of 19 items grouped into seven component scores (0-3) which are equally weighted. Although overall global scores (GPSQI) are calculated by summing the seven components (range 0 – 21: with higher scores indicating poorer sleep quality) the component scores provide subscale ratings of: (i.) subjective sleep quality, (ii.) sleep latency, (iii.) sleep duration, (iv.) sleep efficiency, (v.) sleep disturbances, (vi.). use of sleep medication and (vii.) daytime dysfunction (Hinz et al., 2017). Global scores ≥ 5 are generally used to indicate poor sleep quality (Hinz et al., 2017). The PSQI has demonstrated good reliability (Cronbach's alpha = 0.83, test retest reliability r = 0.85) The PSQI has also demonstrated a diagnostic sensitivity (89.6%) and specificity (86.5%) in distinguishing between 'good' and poor' sleepers (Buysse et al., 1989) however, more conservative scores of ≥ 8 have been used in athletes to indicate poor sleep, potentially due to the increased sleep needs in this population (Samuels, 2009). Although the empirical discussion around the PSQI cut-offs for athletes is ongoing (Samuels et al, 2016; Samuels, 2008), given that athletes often strive for marginal gains in their performance which can be facilitated through optimised sleep, the identification of both 'poor' and 'moderate' sleep quality is warranted (Venter, 2014), hence the standard cut-off (≥ 5) was employed.

4.3.6 Epworth Sleepiness Scale (ESS)

The ESS is an eight item self-report measure of general daytime sleepiness (Johns, 1993). Respondents report their daytime sleepiness in particular situations on a Likert scale (0 = Would never doze to 4 = High chance of dozing). Scores are summed to yield a global ESS score (0 - 24). The EES global score is indicative of daytime sleepiness (Kryger et al., 2011). Higher scores indicate greater sleepiness, scores > 10 suggest excessive daytime sleepiness (Johns, 1993). In general, ESS scores are interpreted in terms of daytime sleepiness as follows: 0 – 5 low normal, 6 – 10 higher normal, 11 – 12 mild excessive, 13 – 15 moderate excessive and 16 – 24 severe (Johns, 1993). In the present sample a Cronbach's alpha of 0.827 was observed.

4.3.7 The Recovery Stress Questionnaire for Athletes (RESTQ Sport)

The RESTQ-Sport is a 52-item self-report measure of general stress and recovery levels of athletes (Kellmann and Kallus, 2001). The RESTQ-Sport consists of seven general stress components with two items per scale (general stress, emotional stress, social stress, conflicts/pressure, fatigue, lack of energy, and physical complaints), five general recovery

components with two items per scale (success, social recovery, physical recovery, general well-being, and sleep quality), three sport-specific stress components with four items per scale (disturbed breaks, burnout/emotional exhaustion, and fitness/injury) and four sport-specific recovery components with four items per scale (fitness/being in shape, burnout/personal accomplishments, self-efficacy, and self-regulation) (Kellmann and Kallus, 2001). Sub-scale item mean scores can be combined to give a total score for each of the four major sub-scales (i.e. general stress, general recovery, sport-specific stress and sport-specific recovery). Each item is scored on a Likert scale (0 = Never to 6 = Always) based on how often the respondent engaged in a specified activity over the previous three days/nights. With a response of 0 indicating never having experienced the feeling and 6 indicating always experiencing the associated feeling. High scores on stress scales indicate a high level of stress, while high scores on the recovery scales indicate a high level of recovery (Kallus and Kellmann, 2001). A Cronbach's alpha of 0.784 was observed in the current sample.

4.3.8 Athlete Morningness/Eveningness Questionnaire (AMES)

The AMES which is based on the Horne-Östberg morningness-eveningness questionnaire (Horne and Östberg, 1975), is a four-item questionnaire used to classify an athlete's chronotype in terms of self-identification as being a morning or evening type, preferred sleep/wake phase and preferred competition and training time (Bender et al., 2019). The AMES provides a global score which is used to categorise chronotype: extreme evening type (10-12), moderate evening type (13-17), mid-range type (18-23), moderate morning type (24-28) and extreme morning type (29-31) (Samuels, 2008). A Cronbach's alpha of 0.698 was observed in the current sample.

4.3.9 Consensus Sleep Dairy – Core (CSD-C)

Participants were instructed to complete the CSD-C for two nights (1 'training/competition' day and 1 'rest' day). The CSD is a standardised sleep diary developed for use in both research and clinical settings (Carney et al., 2012). The CSD-C included 8 items e.g. bed time, time it took to fall asleep, number of awakenings, duration of awakenings, time of final awakening, time the respondent got out of bed, and a Likert scale self-report rating of sleep quality (Maich et al., 2016). There was also a comments section where participants could record specific comments about each night's sleep (i.e. 1 training/competition day and 1 rest day). The data collected was then used to compute indices of sleep continuity such as total time in bed (TIB), total sleep time (TST), sleep onset

latency (SOL; time from light out to N1), wakefulness after sleep onset (WASO; amount of time awake after sleep onset), number of awakenings (NoA) and sleep efficiency (SE; ratio of TST:TIB) (Maich et al., 2016).

4.3.10 Supplementation

All participants were instructed to complete questions relating to supplement use (name, dose, frequency and reason for use) on both training/competition days and rest days. Athletes also reported their alcohol consumption (number of drinking sessions and unit consumption per session) in the last month prior to completion of the questionnaire.

4.3.11 Data Analysis

All data was analysed using the Statistical Package for the Social Sciences (SPSS Version 25, IBM Corporation) and Jamovi Version 1.6.18 (The Jamovi Project). Frequency distribution and descriptive statistics were used to present findings (Thomas et al., 2015). All data were presented in mean ± standard deviation, and/or frequency. The differences between the groups for athlete type were explored using independent samples t-tests, Chi square tests, Mann-Whitney U and one-way ANOVA (Thomas et al., 2015).

4.4 Results

4.4.1 Participant Characteristics

A total of 338 (elite n = 115 and sub-elite n = 223) athletes were recruited from a variety of team and individual sports (see Table 4.1 and 4.2.). The sample consisted of both male (n = 203; ~ 60%) and female (n = 135; ~ 40%) athletes.

Table 4.1: Participant characteristics (mean ± SD).

	All (n = 338)	Elite (n = 115)	Sub-elite (n = 223)	t/x^2 value
Gender	Male n = 203; Female n = 135	Male n = 74; Female n = 41	Male n = 129; Female n = 94	X^2 = 1.72
Age*	24.94 ± 5.93	23.44 ± 4.91	25.71 ± 6.27	t = 3.384
Body mass (kg)	72.95 ± 13.26	73.95 ± 12.55	72.44 ± 13.61	t = -0.995
Height (cm)	175.60 ± 9.70	176.6 ± 8.78	175.08 ± 10.12	t = -1.361
Training (mins·wk)*	675.12 ± 306.59	801.35 ± 338.81	610.02 ± 266.90	t = -5.682

*Statistically significant difference

A Chi square analysis demonstrated no significant differences between the goups for gender (X^2[1, n =338] = 1.72, p = 0.189). While there were statistically significant differences between the groups for age (elite 23.44 ± 4.91 years and sub-elite 25.71 ± 6.27 years; t(336) = 3.38; p = 0.001) and minutes trained per week (elite 801.35 ± 338.81 and sub-elite 610.02 ± 266.90; t(336) = -5.68; p ≤ 0.001). An independent samples t-test indicated no significant differences between the groups in terms of height and body mass (see Table 4.1).

Table 4.2: Participant breakdown.

Sport	All	Elite n = 115	Sub-elite n = 223
Athletics	64	10	54
Boxing	12	11	1
Gaelic games	89	26	63
Hockey	10	9	1
Rowing	29	8	21
Rugby	20	8	12
Sailing	4	3	1
Soccer	31	10	21
Swimming	8	4	4
Other	71	26	45

Chi square analyses demonstrated a statistically significant difference between the groups for sport ($X^2[9, n =338] = 1.72, p \leq 0.001$). There was statistically significant difference between the groups for normal training time; before 8 am (elite n = 8 and sub-elite n = 25), between 8am and 5pm (elite n = 50; sub-elite n = 58), and after 5pm (elite n = 57; sub-elite n = 140) ($X^2[2, n =338] = 10.9, p \leq 0.004$). There were no statistically significant differences between the groups for phase of season; pre-season (elite n = 31; sub-elite n = 57), competition (elite n = 65; sub-elite n = 115), off-season (elite n = 19; sub-elite n = 51) ($X^2[2, n =338] = 1.88, p = 0.39$).

4.5 EuroQoL

A Cronbah's alpha of 0.70-0.95 is considered "acceptable" for a scale used in human research (Vaske et al., 2017; Tavakol and Dennick, 2011). Cronbach's alpha of 0.609 was observed in the current sample most likely due to the low number of items (5), as the size of alpha depends on the number of items in a scale (Streiner, 2003). There was no statistically significant difference between the groups for their perceived general health rating (0-100) with the elite athlete group reporting slightly higher levels of general health than the sub-elite athlete group (83.05 ± 13.65 vs 81.05 ± 12.57; t = -1.37; p = 0.172). There were no statistically significant differences between the groups in terms of each of the domains of the quality of life measure (See Table 4.3). Slight to severe problems with mobility were reported by 19% (n = 65) of participants (elite n = 19 [17%]; sub-elite n = 46 [21%]). Some issues regarding the completion of usual activities (e.g. work, study, training, housework, family or leisure activities) were reported by 19% (n = 64) of participants (elite n = 23 [20%]; sub-elite n = 41 [18%]). Issues with self-care were not evident within the athletes as slight to moderate issues were reported by 3% of participants (elite n = 2 [2%]; sub-elite n = 9 [4%]). Pain was reported by 50% (n = 169) of participants (elite n = 53 [46%]; sub-elite n = 116 [52%]). Anxiety/depression was reported by 35% (n = 116) of participants (elite n = 43 [37%]; sub-elite n = 73 [33%]).

Table 4.3: Athlete responses to the EuroQOL

		None	Slight	Moderate	Severe	Extreme
Mobility	Elite	96	14	5		
	Sub-elite	177	42	2	1	1
Self-care	Elite	113	1	1		
	Sub-elite	214	7	2		
Usual activities	Elite	92	18	3	1	1
	Sub-elite	182	33	8		
Pain	Elite	62	47	6		
	Sub-elite	107	102	14		
Anxiety/Depression	Elite	72	33	8	2	
	Sub-elite	150	58	13	2	

4.6 Pittsburgh Sleep Quality Index

An independent samples t-test was used to compare PSQI data for the elite and sub-elite athlete groups. A statistically significant difference was observed between the groups for PSQI habitual sleep efficiency % (elite 88.62 ± 8.84 vs sub-elite 86.55 ± 9.09; t = - 2.01; p = 0.046). While no other statistically significant differences were observed, the majority of athletes (64%; n = 220) were classified as poor sleepers (i.e. global PSQI score ≥ 5 - elite 64% [n = 74]; sub-elite 65% [n = 146]). Overall self-reported sleep quality did not reflect this as the athletes rated their sleep quality as very good (elite n = 19 [17%]; sub-elite n = 45 [20%]), fairly good (elite n = 68 [59%]; sub-elite n = 123 [55%]), fairly bad (elite n = 26 [23%]; sub-elite n = 50 [22%]) and poor (elite n = 2 [1%]; sub-elite n = 5 [2%]). Mean total sleep time (hours) varied between the elite athlete (7.58 ± 1.06; range 5 – 10 hours) and the sub-elite athlete groups (7.35 ± 1.05; range 4 - 10 hours) but this was not statistically significant. The athletes reported total sleep time ≤ 6 hours (elite n = 16 [14%]; sub-elite n = 43 [19%]), 7 hours (elite n = 38 [33%]; sub-elite n = 80 [36%]), 8 hours (elite n = 39 [34%]; sub-elite n = 70 [32%]) and ≥ 9 hours (elite n = 22 [19%]; sub-elite n = 30 [13%]). The athletes' responses to the PSQI are summarised in Table 4.3. The athletes reported total time in bed ≤ 8 hours (elite n = 53 [46%]; sub-elite n = 109 [49%]), 9 - 10 hours (elite n = 50 [44%]; sub-elite n = 110 [49%]) and 11 - 12 hours (elite n = 12 [10%]; sub-elite n = 4 [2%]).

The reasons reported for poor sleep quality were not getting to sleep within 30 minutes, waking during the night or early morning, waking to use the bathroom and feeling too hot in bed. A feeling of lack of enthusiasm for general tasks at least once per week was reported by 44% (n = 51) of the elite group and 41% (n = 92) of the sub-elite group. The use of sleep medication was low in both groups, with 5% (n = 6) of the elite group and 7% (n = 16) of the sub-elite group using medication on a weekly basis (see Table 4.4).

Table 4.4: Athlete responses to the PSQI.

		Not during the last month	Less than once per week	Once or twice per week	Three or more times per week
Cannot get to sleep within 30 minutes	Elite	40	24	27	34
	Sub-elite	99	56	82	48
Wake up in the middle of the night or early morning	Elite	30	24	27	34
	Sub-elite	37	56	82	48
Have to get up to use the bathroom	Elite	38	24	29	24
	Sub-elite	63	76	44	40
Cannot breathe comfortably	Elite	101	11	1	2
	Sub-elite	192	16	10	5
Cough or snore loudly	Elite	88	14	5	8
	Sub-elite	167	33	16	7
Feel too cold	Elite	79	27	7	2
	Sub-elite	160	41	19	3
Feel too hot	Elite	54	32	26	3
	Sub-elite	82	77	54	10
Have bad dreams	Elite	63	34	16	2
	Sub-elite	114	75	27	7
Have pain	Elite	81	22	11	1
	Sub-elite	152	48	19	4
Other reasons	Elite	101	9	3	2
	Sub-elite	180	27	9	7
Problems staying awake	Elite	66	29	13	7
	Sub-elite	125	66	27	5
Lack of enthusiasm	Elite	35	29	37	14
	Sub-elite	50	81	69	23
Use of sleep medication	Elite	104	5	3	3
	Sub-elite	189	18	7	9

4.7 Epworth Sleepiness Scale

An independent samples t-test demonstrated no significant differences between the elite and sub-elite athlete groups on ESS scores ($p > 0.05$). A Chi square test highlighted no significant difference between the groups' ESS classification ($X^2[20, n = 338] = 21.1$, $p = 0.391$). Approximately 21% (n = 70) of athletes (elite n = 25; 22% and sub-elite n = 45; 20%) reported clinically significant excessive daytime sleepiness (ESS total score ≥ 10) (see Table 4.5).

Table 4.5: ESS classification

Classification (ESS score)	Elite (n = 115)	Sub-elite (n = 223)
Low Normal (0 - 5)	53	114
Higher Normal (6 -10)	45	70
Mild Excessive (11 - 12)	6	20
Moderate Excessive (13 - 15)	8	14
Severe (16 – 24)	3	5

4.8 Recovery Stress Questionnaire

An independent samples t-test highlighted significant differences between the elite and sub-elite athlete groups for recovery i.e. the sport specific recovery scale (3.22 ± 0.90 vs

2.91 ± 0.90; t=-2.984; p <0.001). While no statistically signicant differences were observed for the general stress, general recovery and sport specific stress subscales. Recovery stress scale scores were similar in both the elite and sub-elite groups with similar scores observed for the general stress scale (1.96 ± 0.91 vs 2.01 ± 0.86), general recovery scale (2.97 ± 0.79 vs 2.97 ± 0.77) and sport specific stress scale (1.97 ± 0.87 vs 1.99 ± 0.85).

Table 4.6 RESTQ scales (Mean ± SD).

	All (n=338)	Elite (n=115)	Sub-elite (n=223)	T=	p=
General Stress	1.7 ± 1.31	1.77 ± 1.39	1.67 ± 1.26	-0.6602	0.51
Emotional Stress	1.95 ± 0.983	1.9 ± 0.98	1.97 ± 0.99	0.6858	0.493
Social Stress	1.85 ± 1.03	1.83 ± 1.04	1.86 ± 1.02	0.2199	0.826
Conflicts/Presure	2.35 ± 1.24	2.24 ± 1.26	2.41± 1.24	1.1382	0.256
Fatigue	2.52 ± 1.32	2.46 ± 1.32	2.55 ± 1.32	0.6125	0.541
Lack of Energy	2 ± 1.06	1.95 ± 1.19	2.02 ± 1	0.5755	0.565
Physical Complaints	1.61 ± 1.22	1.59 ± 1.34	1.61± 1.16	0.1638	0.87
Success	2.85 ± 1	2.92 ± 1.01	2.81 ± 1	-0.9189	0.359
Social Relaxation	3.3 ± 1.28	3.19 ± 1.26	3.36 ± 1.29	1.1573	0.248
Physical Relaxation	2.53 ± 1.06	2.59 ± 1.09	2.49 ± 1.04	-0.8265	0.409
General Wellbeing	3.35 ± 1.16	3.37 ± 1.22	3.35 ± 1.13	-0.1497	0.881
Sleep Quality	2.81 ± 0.83	2.77 ± 0.78	2.83 ± 0.85	0.6552	0.513
Disturbed Breaks	1.68 ± 0.92	1.71 ± 0.91	1.67 ± 0.94	-0.4119	0.681
Burnout/Emotional Exhaustion	1.83 ±1.13	1.87 ± 1.22	1.81 ± 1.09	-0.4695	0.639
Fitness/Injury	2.43 ± 1.12	2.32 ± 1.17	2.48 ± 1.09	1.2827	0.2
Fitness/Being in Shape**	3.01 ± 1.06	3.22 ± 1.08	2.9 ± 1.04	-2.6563	0.008
Burnout/PersonalAccomplishment *	2.82 ± 1.01	2.97 ± 1.04	2.74 ± 0.98	-1.9984	0.048
Self-Efficacy**	2.94 ± 1.07	3.15 ± 1.12	2.83 ± 1.04	-2.5747	0.01
Self-Regulation**	3.31 ± 1.2	3.55 ± 1.19	3.18 ± 1.18	-2.7121	0.007

Data presented as mean ± SD * p<0.05, ** p<0.01

An independent samples t-test displayed no statistically significant differences between the groups for the majority of the subscales with both groups recording similar scores (see Table 4.6). However, significant differences between the groups were observed for the following sport specific recovery subscales: being in shape (3.22 ± 1.08 vs. 2.90 ± 1.04; t = -2.66; p = 0.008), personal accomplishment (2.97 ± 1.04 vs. 2.74 ± 0.98; t = -1.98; p = 0.048), self-efficacy (3.15 ± 1.12 vs. 2.83 ± 1.04; t = -2.58; p = 0.010) and self-regulation (3.55 ± 1.19 vs. 3.18 ± 1.18; t = -2.71; p = 0.007), with higher levels being observed across each domain in the elite athlete group (See Figure 4.1).

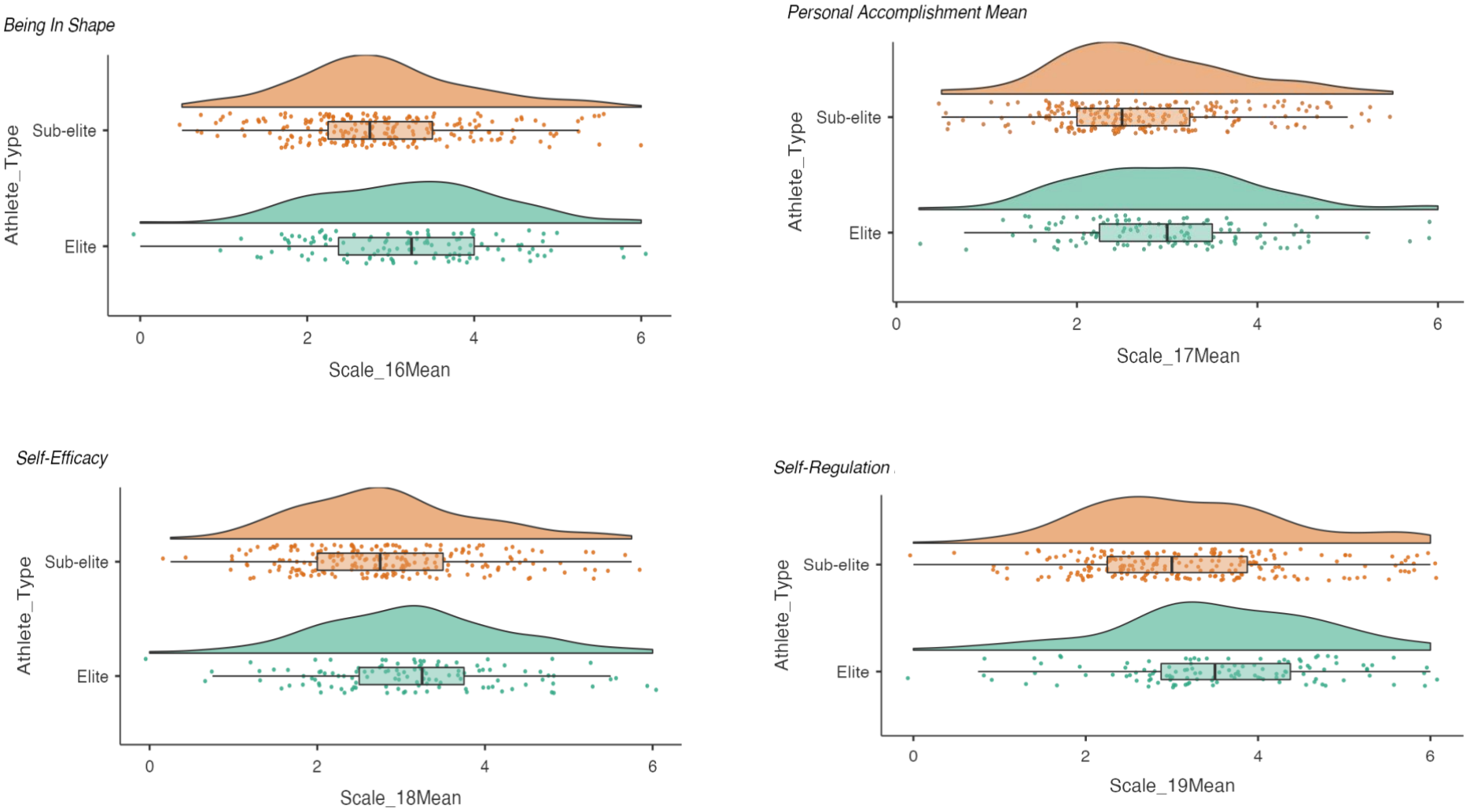

Figure 4.1: Comparison of the sport specific recovery subscales.

4.9 AMES

An independent samples t-test demonstrated a statistically significant difference between the groups for preferred competition time, ($t(336) = -2.45$; $p = 0.015$) with a higher percentage of the elite athlete group (77% [n = 89]) preferring afternoon competition times compared to the sub-elite group (60% [n = 113]) (see Table 4.7). There was no significant differences between the groups for chronotype, time they usually get tired and preferred training time.

Table 4.7: Athlete response to the AMES.

Chronotype	Morning type	More morning type	More evening type	Evening type	
Elite (n =)	24	40	36	15	
Sub-elite (n =)	45	84	65	29	
Preferred training time	6am-9am	9am-Noon	Noon-3pm	3pm-6pm	6pm-9pm
Elite (n =)	12	31	29	18	25
Sub-elite (n =)	26	75	47	41	34
Preferred competition time*	6am-9am	9am-Noon	Noon-3pm	3pm-6pm	6pm-9pm
Elite (n =)	5	21	47	21	24
Sub-elite (n =)	12	78	62	47	21
Time you usually get tired	8pm-9:30pm	9:31pm-10:45pm	10:46pm-12:30am	12:30am-1:45am	1:46am-3:00am
Elite (n =)	27	51	26	3	8
Sub-elite (n =)	50	94	66	9	4

*Statistically significant difference ($p<0.05$).

4.10 Consensus Sleep Dairy–Core

All athletes also completed a sleep diary for a training/competition day and a rest day. A one-way ANOVA was conducted to assess the difference between the groups for TIB, TST, SL, NoA and WASO on both the training/competition day and rest day. While there were no statistically significant differences for TIB, SL and WASO, there were statistically significant differences between the groups (elite vs. sub-elite) for TST on the training/competition day (8.01 ± 1.3 vs 8.2 ± 1.38; $F(1, 238) = 3.91$; $p = 0.049$) and NoA on the rest day (1.03 ± 1.17 vs 1.52 ± 2.44; $F(1, 334) = 6.34$; $p = 0.012$), with the sub-elite athlete group reporting higher TST but more awakenings (see Table 4.8). The majority of athletes in both groups (elite n = 155 [70%]; sub-elite n = 77 [67%]) reported wakening 1-5 times each night. Athletes in both groups reported that it took ≥ 30 minutes to fall asleep on the training/competition day (elite n = 33 [29%]; sub-elite n = 72 [32%]) and the rest day (elite n = 35 [30%]; sub-elite n = 70 [31%]). While there was no statistically significant difference between the groups, poor habitual sleep efficiency (< 85%) was reported by 20% (n = 23) of the elite athlete group and 25% (n = 55) of the sub-elite athlete group. In the comments section of the sleep diary a subset of athletes (n = 73 [22%]) reported the reasons

for waking at night, the most common reasons included injury (n = 15 [4%]), children (n = 11 [3%]), anxiety (n = 19 [6%]), energy restriction (i.e making weight) (n = 7 [2%]) and waking to use the bathroom (n = 21 [6%]).

Table 4.8: Sleep Dairy responses (Mean ± SD).

Sleep Measure		Training/Competition Day	Rest Day
Time In Bed (h)	Elite	9.1 ± 1.18	9.53 ± 1.49
	Sub-elite	9.2 ± 1.42	9.6 ± 1.5
Total Sleep Time (h)	Elite	8.01 ± 1.30*	8.58 ± 1.4
	Sub-elite	8.2 ± 1.38*	8.59 ± 1.44
Sleep Latency (Min)	Elite	22.85 ± 20.74	21.62 ± 18.7
	Sub-elite	22.65 ± 17.70	23.72 ± 22.37
Number of Awakenings (#)	Elite	1.38 ± 1.43	1.03 ± 1.17*
	Sub-elite	1.51 ± 1.73	1.52 ± 2.44*
Wake After Sleep Onset (Min)	Elite	11.06 ± 17.06	7.31 ± 9.99
	Sub-elite	10.14 ± 16.51	9.56 ± 12.60
Sleep Efficiency (%)	Elite	88.2 ± 10.18	90.21 ± 6.6
	Sub-elite	89.77 ± 7.14	89.1 ± 7.05

* Statistically significant differemnce ($p<0.05$)

4.11 Nutrition

The athletes also reported their supplement and alcohol consumption in the month prior to completion of the questionnaire. A Mann-Whitney U test indicated no significant differences between the elite and sub-elite athlete groups for supplemenation and alcohol consumption ($p \geq 0.05$). The most commonly used supplements were whey protein, caffeine, creatine, multivitamins, fish oil, probiotics and vitamin D (see Table 4.9).

Table 4.9: Athlete supplement use, frequency, average dose and reason for use.

Supplement	Frequency	Dose	Reason	Elite (n = 115)	Sub-elite (n = 223)
Caffeine	Daily	100mg	Performance	23	37
Creatine	Daily	Varied	Performance	13	20
Fish Oil	Daily	1 capsule	Health	18	12
Iron	Daily	Varied	Anaemia/Performance	4	10
Multivitamin	Daily	1 capsule	Health	24	32
Nitrate	Daily	1 shot	Performance	11	1
Probiotics	Daily	1 capsule	Health	13	25
Vitamin D	Daily	1,000-4,000IU	Health/Performance	21	5
Whey	Daily	25-40g	Recovery	22	48
Other (e.g. BCAA, beta – alanine, HMB, antioxidants)	Daily/weekly	Varied	Health/Performance	30	19

(BCAA = Branched chain amino acids; HMB = Hydroxymethylbutrate)

Spearman's rank order correlation was used to assess the relationship between supplement use and various sleep and recovery variables. There were small significant

correlations between supplement use and the RESTQ scales: sleep quality, disturbed breaks, emotional exhaustion, being in shape and self-efficacy (see Table 4.10).

Table 4.10: Relationship between supplement use and recovery.

	Sleep Quality	Disturbed Breaks	Emotional Exhaustion	Being in Shape	Self-Efficacy
Supplement Use	-0.167**	0.119*	0.137*	-0.114*	-0.108*
	p=0.002	p=0.029	p=0.012	p=0.036	p=0.048

Statistically significant differemnce (* $p < 0.05$; ** $p < 0.01$)

The athletes reported the number of times they consumed alcohol in the last month 1-4 times (elite n = 10 [9%]; sub-elite n = 10 [5%]), 5 – 9 times (elite n = 11 [10%]; sub-elite n = 5 [2%]), and > 10 times (elite n = 3 [3%]; sub-elite n =11 [5%]). The athletes also reported the number of units they usually consumed during each drinking session < 4 units (elite n = 11 [10%]; sub-elite n = 6 [3%]), 5 – 10 (elite n = 9 [8%]; sub-elite n = 8 [4%]) and > 10 (elite n = 4 [3%]; sub-elite n = 12 [5%]).

4.12 Discussion

This study recruited a large cohort of elite (n = 115) and sub-elite (n = 223) athletes from a wide variety of sports. Elite athletes were either international athletes, members of a national/professional team, a recruitment/academy squad and/or nationally ranked in their sport (Swann et al., 2015). Sub-elite athletes were defined as those competing at a regional, university and/or national level of organised sport that trained and/or competed for a combined minimum of 400 minutes per week (Swann et al., 2015). To the authors' knowledge this is one of the largest cohorts of athletes to have been investigated from a sleep and recovery perspective. This study aimed to investigate: the quality, quantity and timing of sleep among sub-elite and elite athletes and characterise their recovery and nutrition practices. It was hypothesised that the sleep, recovery and nutrition practices of elite athletes would be superior to those of sub-elite athletes. Interestingly similar levels of poor sleep were reported by both the elite and sub-elite athlete groups while there was a significant difference in sport specific recovery practices.

4.13 Sleep

Poor sleep quality was reported in the PSQI, the REST-Q and it was notable in the sleep diaries that athletes reported improved TIB, TST and WASO on rest days. Excessive daytime sleepiness was also observed in both groups. Similarly, previous research has suggested that the quality and quantity of elite atletes sleep was inferior to sub-elite athletes and potentially

inadequate in relation to optimal recovery and performance (Halson, 2019; Knufinke et al., 2018: Gupta et al., 2017; Lastella et al., 2015; Leeder et al., 2012). It is important to note that athletes who had a diagnosed sleep problem were excluded from the current study, hence the prevalence of sleep problems are indicative of levels within elite and sub-elite athlete populations.

4.13.1 Pitsburg Sleep Quality Index

The PSQI has demonstrated good reliability (Buysse et al., 1989), acceptable internal consistency and has been shown to be reliable (Backhaus et al., 2002; Spira et al., 2011) and valid (Backhaus et al., 2002; Buysse et al., 1989; Hinz et al., 2017; Spira et al., 2011) measure of sleep quality. Cronbach's a 0.744 was observed in the current sample. The majority of athletes (~ 65%; n = 220) were classified as poor sleepers (Global PSQI score ≥ 5). This is consistent with previous research in elite athletes (Knufinke et al., 2018, Swinbourne et al., 2016; Sargent et al., 2014; Samuels, 2008), and sub-elite athletes (Mah et al., 2018; Elbayoumy and Elbayoumy, 2015). A relatively high proportion of athletes (~30%) self-reported their sleep quality as either poor or very poor on the training/competition day compared to rest day (elite 10% [n = 12] and sub-elite 16% [n = 36]). The PSQI data highlighted reasons for poor sleep on both training/competition days and rest days such as feeling too hot in bed and a lack of enthusiasm for general tasks. Poor sleep quality is of particular concern for elite athletes as it can result in a reduction in recovery and/or subsequent athletic performance (Juliff et al., 2015; Jarraya et al., 2013; Reyner and Horne 2013; Edwards and Waterhouse, 2009).

Interestingly the PSQI mean TST (<8h) was lower than that reported in the CSD-C (>8h), it has been suggested that athletes tend to overestimate their sleep (Caia et al., 2018). A recent review suggested that sleep in athletes is limited to 7.2 hours per night with all studies reporting < 8 hours per night and mean SE was 86.3% ± 6.8% (Vlahoyiannis et al., 2020), which is in line with the PSQI and CSD-C data from the current study. The PSQI mean TST for both groups in the current study is adequate according to current sleep recommendations (7-9h) (Hirshkowitz et al., 2015). However, optimal sleep duration is subject to individual variance and it has been argued that elite athletes may require more sleep than non-athletes (Walsh et al., 2021; Sargent et al., 2021; Bird, 2013). It has previously been reported that athletes tend to sleep less (6.5-6.7h) and that their sleep quality is poor (Hagenauer et al., 2017; Sargent et al., 2014; Hausswirth et al., 2014; Leeder et al., 2012; Mah et al., 2011).

Optimising sleep gives athletes an advantage when it comes to maximising adaptations from training and performance enhancement (Simpson et al., 2017).

4.13.2 Consensus Sleep Diary-Core

There were significant differences between the groups for TST on the training/competition day and NoA on the rest day. TST was lower in the elite athlete group on both days, however it did improve on the rest day which was most likely a reflection of their behaviour e.g. choosing to go to bed earlier. Although not statistically significant, there was a trend towards increased TIB, TST and WASO in both groups on the rest day while the elite athlete group also demonstrated a trend towards reduced SL, NoA and increased SE on the rest day. Similarly, a small study of Australian athletes (n = 6) using objective measures of sleep demonstrated that sleep improved (longer duration) on a rest day (71.6% reported no sleep disturbance following one rest day) (Fowler et al., 2014). A study involving elite swimmers (n=7) showed that the athletes went to bed later but slept longer on rest days (Sargent et al., 2014), where the opportunity for extended sleep provided the athletes with an opportunity to partially recover the sleep debt accumulated during the training week (Merdad et al., 2014). In the current study poor sleep was attributed by the athletes in both groups to a number of factors i.e. injury, children, anxiety, making weight (boxing) and bathroom use. Previous research has highlighted isssues that impair an athlete's sleep such as stress (Halson, 2019; Pires et al., 2016), pain/injury (Biggins et al., 2019; Halson, 2019; Watson, 2017) and anxiety (Juliff et al., 2015; Erlacher et al., 2011). The relationship between poor sleep and impaired mood has been reported in non-athletic populations (Dinges et. al., 1997), however the study involved sleep restricton to 4.98 hours per night. Monitoring athletes' mood (e.g. through wellness monitoring) could identify athletes who require sleep related intervention.

In the current study, poor habitual SE% previously quantified as < 85% (Ohayon et al., 2017) was reported by 20% (n = 23) of the elite athlete group and 25% (n = 55) of the sub-elite athlete group. Previous research has demonstrated that habitual sleep efficiency of elite athletes was 88.47% ± 5.45% (Knufinke et al., 2018) 80.6% ± 6.4% (Leeder et al., 2012), 86.3% ± 6.1% (Lastella et al., 2015) and 79% ± 9.2% (Shearer et al., 2015). A recent systematic review reported the pooled average sleep efficiency for athletes (86% ± 5%; range 79% - 96%) (Gupta et. al., 2017) which straddled and for many athletes was less than the threshold of 85%, below which insomnia symptoms are indicated (Sánchez-Ortuño et al., 2010). While the range of sleep efficiency observed can in part be explained by

methodological inconsisitencies, the pooled averge nonetheless indicated sleep problems and poor sleep quality. There is a need for clear athlete-friendly interventions that could promote improved sleep and recovery.

4.13.3 Daytimne Sleepiness

The ESS score is comparable to objective sleepiness measures such as the multiple sleep latency test (MSLT) and is considered a valid and reliable measure of objective sleepiness (Johns, 1993). The ESS has been widely used in athletic populations such as Australian rules football (Antic et al., 2013), collegiate basketball players (Mah et al., 2011) and American football players (George et al., 2003). Approximately 21% of athletes in the current study reported excessive daytime sleepiness. Similar levels of excessive daytime sleepiness have been reported in Rugby players and cricketers (Swinbourne et al., 2016), American footballers (George et al., 2003), Australian rules footballers (Antic et al., 2013) and college athletes (Mah et al., 2018). Similarly, previous research reported that 44% (n = 12) Brazilian Paralympians experienced excessive daytime sleepiness (Silva et al., 2012). However, it must be noted that these athletes may have had physical impairments (e.g. spinal cord injury) that could impact sleep quantity and quality.

The levels of excessive daytime sleepiness observed in the current study may be due to sleep disorders such as obstructive sleep apnea (OSA) and periodic limb movement disorder (PLMD). In the general population the most common sleep disorders are (OSA), insomnia and restless legs syndrome (RLS)/(PLMD) (Adams et al., 2016). OSA is a frequent condition characterised by repeated episodes of partial or complete reduction in breathing activity during sleep (Marra et al., 2019). PLMD is a condition characterised by repetitive limb movements during sleep that cause sleep disruption (Thorpy, 2017). A recent systematic review highlighted the prevalence of insomnia symptoms (longer SOL, increased sleep fragmentation and excessive daytime sleepiness) in elite athletes (Gupta et al., 2017). Other sleep problems, such as OSA, are less prevalent but appear to be higher in strength and power athletes (e.g. Rugby players) most likely due to increased body mass and neck circumference (> 42cm) which are anatomical features related to OSA (Swinbourne et al., 2016). A recent study using a combination of PSG and subjective measures demonstrated high prevalence of sleep disorders in Rugby union players (n = 25), all players displayed insomnia symptoms and 24% (n= 6) had OSA and 12% (n= 3) had RLS/PLMD (Dunican et al., 2019). In a similar study using home-based PSG in Rugby league players (n=22), 45% (n = 10) had OSA (Caia et al., 2020). A previous study of NFL players (n = 137) demonstrated the 19%

(n = 26) had OSA (Rice et al., 2010). Previous research in elite ice hockey players (n =107) has demonstrated sleep problems, 11% (n =14) had insomnia, 10% (n = 13) had OSA and 3% (n = 4) had RLS/PLMD (Tuomilehto et al., 2016). Athletes with poor sleep habits and/or a sleep disorder must be identified, diagnosed and individual interventions (e.g. sleep hygiene, nutrition) must be implemented in order to facilitate athlete recovery and performance.

4.13.4 Athlete Morningness Eveningness

Although there was no significant difference between the groups for chronotype, time they usually got tired or preferred training time, a statistically significant difference was evident for preferred competition time, ($p = 0.015$), with the elite athlete group preferring afternoon competition times, while the sub-elite athlete group preferred morning competition times. The vast majority of the athletes from both groups 58% (n = 197) indicated that their normal training time was after 5pm. Training time and chronotype may have an influence on sleep (Lastella et al., 2016). A study investigating the sleep quality of morning and evening types after a morning (8:00 am) and evening (20:00 pm) high intensity interval training session reported poorer sleep quality (reduced total sleep time, increased sleep disturbance and reduced sleep efficiency) in morning types after the evening session while sleep quality after the morning session was similar for both groups (Vitale et al., 2017). The late training times reported by the athletes in the current study may have adversely impacted their sleep and recovery. Sleep following training is recognised a being important for recovery (Skein et al., 2013), reduced sleep quality following evening training sessions (particularly vigorous training) may negatively impact subsequent recovery and performance, the effect may be more pronounced in morning type athletes.

4.14 Recovery

Recovery is a process in time, dependent on the duration of stress and requires a reduction of stress, a change in stress or a break from stress (Frank et al., 2018; Kellmann, 2010). Relatively high levels of fatigue, stress and pain were reported in both groups. A range of supplements were used regularly by athletes in both groups; indeed, whey was the most commonly used recovery supplement in both groups. The results suggest future research is warranted to further the development of individualised inventions focused on sleep, nutrition and athlete recovery.

4.14.1 EuroQoL

The EQ-5D-5L has demonstrated reliability (mean intraclass correlation coefficients 0.69; range 0.43 – 0.84) and convergent validity (mean Spearman rank coefficients 0.99; range 0.97 – 0.99) (Janssen et al., 2008). The mean general health rating scores for the elite athlete group (83.1 ± 12.6) and the sub-elite athlete group (81 ± 13.7) were relatively high, which was consistent with current research in athletes (Timpka et al., 2019). In the current study the elite athlete group reported higher mean health rating scores. Elite athletes tend to have their training and recovery sessions scheduled for them (Sargent et al., 2014), hence, they are likely to complete regular if not daily mobility type sessions. Whereas, the sub-elite athletes may have had less free time due to work, social and family commitments. A high prevalence of pain was reported by 50% (n = 169) of participants (elite n = 53 sub-elite n = 116). An investigation of 'mildly sleepy' (indicative of inadequate sleep duration) but otherwise healthy males (n = 24) showed sleep extension (time in bed 10 hours) increased pain tolerance by 20% (Roehrs et al., 1989). While chronic sleep restriction (50% of habitual time for 12 days) is related to increased levels of muscle soreness and increased pain sensitivity (Hagenauer et al., 2017). While mobility issues were noted in both groups, there were higher levels mobility issues reported by the sub-elite athlete group coupled with issues completing usual activities. However, it has recently been suggested that elite and high level athletes have increased pain tolerance (cold pressor test) and that the training time per week has a positive impact on the tolerance (Pettersen et al., 2020). Anxiety/depression was reported by 35% of athletes (elite n = 43 and sub-elite n = 73) in the current study, which is consistent with previous research that has highlighted that anxiety is related to and has a negative effect on the quality of athletes' sleep (Juliff et al., 2015; Erlacher et al., 2011). The relationship between poor sleep and impaired mood has also been reported in non-athletic populations (Dinges et. al., 1997), however the study involved sleep restricton to 4.98 hours per night. Monitoring athletes' mood and levels of anxiety/depression (e.g. through wellness monitoring/POMS) could identify athletes who require specific interventions.

4.14.2 REST-Q sport

The RESTQ-Sport has been shown to be valid in athletic populations (Laux et al., 2015; Tibbert et al., 2009). The scales have displayed good internal consistency (0.67 - 0.89) and high test-retest reliability (> 0.79) (Kellmann, 2010; Kellmann and Kallus, 2001). Relatively high levels of stress and fatigue were evident from the REST-Q. Stress and fatigue are factors

for illness, which must be managed by elite athletes (Keaney et al., 2019; Schwellnus et al., 2016), during their competitive seasons to avoid missed training/competitions. Significant differences between the elite and sub-elite athletes were observed for 4 of the REST-Q subscales relating to athletic performance, with higher mean score for each subscale: being in shape, personal accomplishment, self-efficacy and self-regulation reported by the elite athlete group. The injury (2.31 ± 1.17 vs 2.48 ± 1.09), fatigue (2.45 ± 1.32 vs 2.54 ± 1.32) subscale scores were relatively high in both the elite athletes and sub-elite athletes, while the sleep quality scores were low (2.76 ± 0.78 vs 2.83 ± 0.85). The current findings are consistent with previous research which reported that injury risk was significantly positively related to injury subscale scores for disturbed breaks, fatigue, and lower values on the sleep quality subscale score (Laux et al., 2015). The relationship between training load and health can be considered on a well-being continuum (Kellmann et al., 2018; Soligard et al., 2016; Schwellnus et al., 2016), with training load and recovery as antagonists. Stress is imposed on athletes, altering their physical and psychological well-being along a continuum: homeostasis, acute fatigue, subclinical tissue damage, functional overreaching, non-functional overreaching, clinical symptoms, overtraining syndrome, time-loss injury or illness and with continued loading in extreme cases death (Soligard et al., 2016; Schwellnus et al., 2016). A recent meta-analysis has linked psychological stress (r = 0.27, 80% CI 0.20 - 0.37) and history of stressors (r = 0.13, 80% CI 0.11 - 0.15) to injury rates in athletes (Ivarsson et al., 2017). Athletes' injury risks are affected by their responses to multiple stressors that result in not only physical, psychological and attentional changes (e.g. increased reaction time, narrowing of peripheral vision, increased distracability) but also behavioural changes (e.g. poor sleep quality and impaired self-care) (Ivarsson et al., 2017).

In the current study significantly higher levels of sports specific recovery (3.22 ± 0.91 vs 2.91 ± 0.90) were reported by the elite athlete group compared to the sub-elite athlete group. This result potentially highlights the fact that elite athletes tend to be under the supervision of a multidisciplinary team e.g. medical, strength and conditioning, nutrition, physiology and psychology, who are involved in all aspects of the athletes training and recovery. The sub-elite athletes would typically not receive the same access to multidisciplinary support services. It is imperative that athletes have a detailed recovery plan compromising of nutrition, hydration, sleep and psychological recovery (Schwellnus et al., 2016). Given the high training and competition load that athletes undertake; they must adopt strategies that promote sleep across the domains of quality, quantity and timing. Fatigue can be managed, and recovery enhanced through adequate passive rest and sufficient sleep

(Meeusen et al., 2013), it is generally recommended that athletes have at least one 'rest' day per week. Rest days can serve to alleviate boredom and stress perception while the absence of a 'rest day' during periods of intense training has been related to the onset of over-reaching and inadequate recovery (Meeusen et al., 2013). It is suggested from the current results that sleep tends to improve on rest days i.e. increased perceived sleep quality, TIB, TST and reduced WASO in both goups, while SL, NOA and SE also improved in the elite athlete group.

4.14.3 Nutrition

In the current sample the elite athletes tended to consume more supplements, at higher doses with increased frequency compared to the sub-elite athletes. Those athletes who used supplements reported high usage of caffeine, whey protein, creatine, multivitamins, fish oil, probiotics and vitamin D while the use of iron and nitrate was reported to a lesser extent. This is similar to previous research in elite Dutch athletes (n = 778) where the most commonly consumed supplements were multivitamins, caffeine, vitamin D, sports drinks, protein, beta-alanine and sodium bicarbonate (Wardenaar et al., 2016). It has also been demonstrated previously that elite athletes tend to take more supplements than sub-elite athletes (Knapik et al., 2016). Despite the relatively low number of athletes reporting supplement use the correlations between supplement use and RESTQ scales indicated a relationship between supplement use, stress and recovery. Future studies should include a detailed analysis of the supplements used by athletes and their impact on sleep and recovery. Whey protein was one of the most prevalent supplements used while casein use was also reported. While research is emerging supporting pre-sleep protein ingestion for muscle recovery (Snijders et al., 2019; Falkenberg et al., 2021), the impact of pre-sleep ingestion of 40g doses of Whey and/or Casein warrants further investigation with regards both muscle recovery and sleep improvement.

Daily caffeine use was reported by approximately 20% of the athletes which could negatively impact sleep. The low level of caffeine use reported is most likely due to the fact that athletes were asked to report their supplement use and may have neglected to include habitual caffeine consumption. Caffeine exerts a stimulant effect promoting alertness by blocking adenosine receptors (Foster, 2020). The levels of caffeine consumption reported are lower than previous research which has suggested that 75-90% of athletes consume caffeine before or during competition (Del Coso et al., 2011; Van Thuyne et al., 2005; Desbrow and Leveritt, 2006). While, it has been suggested that chronic low dose caffeine

ingestion may blunt any potential ergogenic effects (Beaumont et al., 2017), moderate doses (~ 3mg/kg/d) appear to pose no problems for most athletes (Pickering and Kiely, 2019). However, in terms of sleep, moderate caffeine doses have been shown to increase SOL and decrease TST, REM sleep and SE (Miller et al., 2014). Hence, athletes training/competing in the late afternoon (> 5pm) need to consider its potentially detrimental effect on sleep. It has recently been suggested that athletes should adopt a strategic individualised approach to caffeine consumption during competition (Dunican et al., 2018).

In the current study higher alcohol consumption was observed in the sub-elite athletes and they tended to consume more units of alcohol during a drinking bout. In line with previous research the actual amount of alcohol consumed by athletes 'in training' is low (O'Brien and Lyons, 2000). Elite athletes tend to have less opportunity to socialise and their schedules (e.g. early morning training) do not lend themselves to regularly consuming alcohol. Alcohol consumption by athletes often occurs post-competition where it can be seen as a reward for 'hard work' (Barnes, 2014). Alcohol consumption has been associated with poorer sleep quality and quantity, reduced REM sleep and increased sleep disturbance in the second half of the sleep bout (Roehrs et al., 2001).

4.15 Limitations

Due to logistical reasons, the sleep diary was only completed for one training/competition day and one rest day, this may have been insufficient in terms of data collection. It has been recommended that sleep diaries should be completed for a duration of 1 week (Carney et al., 2012; Anderson et al., 2018). The aim of the 2 day diary was to limit participant burden and recall bias (Reynolds, 2011). However, sleep diaries may be more accurate than sleep questionnaires (Halson, 2019). The intrinsic limitations of self-report measures (i.e. questionnaires and diaries) are measurement error and recall bias (Thomas et al., 2015). Indeed, it has been demonstrated that athletes tend to overestimate their sleep duration (Caia et al., 2018). However, self-report measures have their place within athletic settings, as they are a relatively simple and inexpensive approach to athlete monitoring, affording a more representative overview of the target population (Halson, 2014). Within elite athlete populations the use of subjective measures of sleep are often employed, particularly during the competitive season due to the more invasive nature of both PSG and actigraphy (Fullagar et al., 2016). A growing body of research has suggested that self-report measures may be more sensitive and reliable than physiological, biochemical and performance measures (Halson, 2014; Buccheit et al., 2013; Meeusen et al., 2013; Coutts et

al., 2007). When choosing a particular measure, ultimately the aim is to maintain a balance between the need to obtain meaningful data from an athlete whilst minimising the burden involved in completion of any self-report measure (Saw et al., 2015). Athletes in the current study were not instructed to record naps, however it was possible to record additional information and notes in the Consensus Sleep Diary-Core, and no particants reported napping. In the current study it was not feasible or practical due to the large sample size to include an objective assessment of sleep. However, future research should incorporate both objective (e.g. PSG, actigraphy) and subjective measures (e.g. sleep diaries) of sleep to provide a more accurate estimates of sleep including napping and because some individuals may self-report poor sleep quality despite objective measures indicating adequate sleep (Krystal and Edinger, 2008). There was little difference between the elite and sub-elite athlete groups in terms of sleep. The inclusion of a healthy control group would have allowed for comparison and exploration of the differnces between the sleep of athletic population and healthy adults.

A specific section in relation to anxiety/depression could have been included in the battery of questionnaires given their potential to impact on sleep and vice versa. The Profile of Mood States (POMS) (McNair et al., 1981) is widely used in wellness assessments of athletic populations and has subscales that specifically relate to anxiety and depression. However, as the EuroQoL has a dimension for anxiety/depression, the POMS was omitted to reduce participant burden and survey fatigue which could have negatively impacted the reliability of the data collected.

The demographic difference between the groups was a limitation in that there was a statistically significant difference between the groups with the sub-elite group being significantly older which could have affected the results. This issue was directly related to the sampling method employed where participants were recruited based on their accessibility (Bornstein et al., 2013). However, care was taken to recruit a large cohort (n = 338) and strict inclusion and exclusion criteria were applied (Swann et al., 2015).

4.16 Future research

Future research should replicate this investigation of the sleep and recovery practices of large cohorts of athletes. Such studies should include a combination of subjective and objective measures of sleep and recovery, for a minimum of 1 week (Carney et al., 2012; Anderson et al., 2018). The validity and reliability of combinations of subjective and objective measures in athletic populations warrants further investigation. While this may not

be practical during the competitive season there may be a window of opportunity at the end of the season or in preseason.

As the majority of athletes in the current cohort have reported sleep problems future research is warranted to identify the specific sleep problems that affect athletic populations. It is also necessary in future research to identify if athletes are affected by acute disturbances e.g. competition anxiety or chronic disorders e.g OSA, insomnia and PLMD (Tuomilehto et al., 2016). Further, future research should investigate the effects of specific nutritional recovery strategies (e.g. antioxidants, protein, carbohydrate) on sleep in athletic populations. Such practices may already be an established part of an athlete's daily routine but the potential additional benefit of improved sleep must be explored.

4.17 Practical Applications

A strength of this novel study is that it presents 'real-life' data from a training/competition day and a rest day relating to the sleep and recovery practices of athletes. Poor sleep and inadequate recovery practices were evident in both the elite and sub-elite athlete groups. In a recent study 95% of swimmers (n=82) identifed their coaches (n=10) as the primary source of recovery information while the coaches highlighted conferences and workshops as their primary source of recovery information (Shell et al., 2020). In order to promote sleep and adequate recovery practices in athletes a comprehensive coach and athlete education curriculum may need to be developed and implemented.

The athletes generally reported improved sleep quality and quantity on rest days which has implications for athlete health, wellbeing and performance. Optimising the sleep and recovery practices of athletes would impact performance. Monitoring of sleep behaviour, nutrition and recovery-stress responses of athletes aids the identification of irregularities (e.g. due to travel or illness) and allows for early interventions with individual athletes as and when necessary (Heidari et al., 2018). The ongoing collection of data from athletes such as the data collected in the current study, could be used by coaches, medical and support staff to implement individual sleep, recovery and nutrition interventions and plans.

4.18 Conclusion

Due to the symbiosis between sleep and recovery it is clear from the current findings that athletes should have a detailed individualised and multi-faceted recovery plan in place involving sleep, nutrition, hydration, as well as other physiological and psychological aspects. At the elite level, athletes and their support teams continually strive for marginal gains over time to improve performance (Soligard et al., 2016). Training and competition

load elicit a number of homeostatic responses and adaptations, the main aim of training is to exploit these in order to elicit an improvement in performance. The training process involves exploitation, manipulation and coordination of numerous variables (e.g. physiology, biomechanics and psychology) to improve performance. Athletes continually strive to improve their performance, as such variations in training load are necessary e.g. increased frequency, duration and/or intensity, in order to optimise the training response (Halson, 2014). Depending on the phase of the season (e.g. pre-season, general preparation, competition, etc.) loads must be managed to increase or decrease fatigue, to enhance training adaptations or performance (Halson, 2014). Rest days should also be incorporated into the recovery plan, which could serve to improve sleep quality, alleviate boredom and stress perception.

The majority of athletes were classified as poor sleepers and reported excessive daytime sleepiness even though their TST met current adequate sleep guidelines. The importance of a rest day was highlighted by the fact that sleep improved in both groups. While relatively low levels of physical recovery were observed in both groups coupled with relatively high levels of stress. The elite athlete group reported significantly higher levels of sport specific recovery. A higher prevalence of supplement use was reported by the elite athlete group while higher levels of alcohol consumption were reported by the sub-elite asthlete group. Given the high training and competition load that athletes undertake, particularly elite athletes; it is clear that they must adopt strategies that promote sleep and recovery. There is a need for athletes to receive individualised support and education regarding their sleep and recovery practices.

4.19 Study 1 - Link to Next Chapter

The aim of this study was to characterise the sleep and general recovery practices of athletes. Poor sleep was high with 64% of athletes classified as ‘poor sleepers’, while 21% reported excessive daytime sleepiness. Total sleep time (TST) was lower in the elite athlete group on both training/competition days and rest days, consolidating the existing evidence that elite athletes are particularly vulnerable to sleep difficulties. Significantly, higher levels of sport-specific recovery were observed in the elite athlete group. Pain was reported by 50% of athletes while anxiety/depression was reported by 35% of athletes. In terms of nutrition, the most commonly consumed supplements were whey protein, caffeine, multivitamins, creatine, fish oil, probiotics and vitamin D, while sub-elite athletes reported drinking more alcohol than the elite athletes. Having characterised the sleep and recovery of athletes in

general, it is now necessary to assess athlete sleep under specific circumstances (i.e. long-haul eastward travel).

Chapter 5: The impact of long haul travel on the sleep of elite athletes.

5.1 Introduction

The Olympic Games were to be held in Tokyo, Japan in July-August 2020, unfortunately due to the Covid-19 pandemic, the games were postponed until 2021. For athletes and

support staff, travel from Europe to Japan involves a considerable eastward journey crossing 7 timezones and up to 24 hours travel time, depending on stopovers (Roach and Sargent, 2019). It is vital that the impact of such journeys on sleep is investigated in order to develop strategies to cope with and minimise the effects of travel fatigue and jet lag. Some teams will travel directly to Japan or other countries in the region weeks before their competition to train and acclimatise, while others will arrive days before their event. Irrespective, athletes will want to adjust to the new time zone as quickly as possible to train and compete (Roach and Sargent, 2019). Acclimitisation is complex and will be made more difficult by the Tokyo summertime conditions which could lead to the hottest Olympics ever, it has been predicted that the temperatures in Tokyo in August will average 31°C with relative humidity above 60% (Vanos, 2020).

The main determinant of the human 'body clock' is situated in the hypothalmus and this master clock becomes synchronised with the external environment, predominately through light/dark cycles (≈ 24 hours 'circadian rhythmn') (Hastings et al., 2018). The circadian system responds to external and internal signals via the SCN (within the hypothalmus) which receives environmental cues such as the light-dark cycle and additional information from other areas of the brain in alignment with certain behaviours (e.g. the timing of nutritional intake and/or exercise) (Tahara and Shibata. 2014). Zeitgebers (time cues) are the cues in our environment that sychronise our internal body clock to the external environment (Choy and Salibu, 2011), through cyclical changes in the external environment and rhythmic signals from melatonin (originating in the pineal gland, an internal zeitgeber) (Hastings et al., 2018; Reppert and Weaver, 2002). Sunlight is the most powerful zeitgeber but eating, physical activity, social contact and other lifestyle factors also influence our circadian rhythmns (Mistlberger and Skene, 2004).

Humans' sleep schedules are predominantly regulated by exposure to light and the secretion of melatonin which have opposing effects on circadian rhythmns (Coste and Lagarde, 2009; Sack, 2009). Melatonin is secreted by the pineal gland for 10-12 hours in the evening and night eliciting a sleep inducing effect (Sack, 2009; Beaumont et al., 2004; Atkinson et al., 2003). While light inhibits the secretion of melatonin and stiumulates arousal (Sack, 2009, Atkinson et al., 2003). The production of endogenous melatonin begins ~ 2 hours before habitual bedtime (Burgess and Eastman, 2005), the daily minimum core body temperature (CBTmin) occurs ~ 7 hours after melatonin onset and corresponds to the low point of the circadian cycle (Eastman et al., 2000), while the daily peak core body

temperature (CBTmax) corresponds to the daily peak of the circadian cycle and occurs ~ 12 hours after CBTmin (Dijk et al., 1992). The propensity for sleep and poorer mental/physical performance occurs 2-3 hours either side of CBTmin, while greatest alertness and optimal mental/physical performance occur in the 2-3 hours either side of CBTmax (Dijk et al., 1992). For example, a person who normally goes to sleep from 11:00pm-7:00am will have a melatonin onset time at ~ 9:00pm, CBTmin at ~ 4:00am and CBTmax at ~ 4:00pm (Waterhouse et al., 2007).

The body clock is connected to a series of peripheral clocks, that have a role in increasing alertness, mental and physical activity during the daytime, and preparing the body for sleep in the evening (Walsh et al., 2020). All the major organs such as the heart, lungs, liver and pancreas have their own circadian oscillators which are controlled by the SCN under normal conditions (Schibler et al., 2015). The circadian rhythm facilitates consolidated sleep at night and prepares the body for waking via rhythmic changes in the autonomic nervous system, hormones, core body temperature and the sleep-wake cycle (Walsh et al., 2020). Falling asleep, the ability to maintain sleep, and the likelihood of awakenings are also associated with core body temperature. Getting to sleep and maintaining sleep are easiest at and after the onset of melatonin secretion (body temperature is reducing or low) and most difficult when melatonin secretion has ceased (body temperature is increasing or high) (Edwards and Waterhouse, 2013).

The normal synchronicity between the body clock and the environment is disrupted by rapid transmeridian travel with associated negative symptoms that are referred to as ‘jet lag” (Waterhouse et al., 2007). The conditions experienced during long haul travel, such as uncomfortable and confined seating positions, noise levels and stopovers can cause fatigue and disrupt sleep (Fowler et al., 2021). Numerous symptoms commonly manifest as a result of jet lag that impact athletic performance such as fatigue, disturbed sleep, insomnia, excessive daytime sleepiness, decreased alertness, headaches, mood disturbance, decreased motivation, appetite change and gastrointestinal distress (Diekman and Bose, 2018; Forbes-Robertson et al., 2012; Choy and Salibu, 2011; Kolla and Auger, 2011). The consequences of long haul travel on athletes’ sleep and well-being could compromise both training adaptations and/or performance. Keeping athletes healthy in the period prior to important competitions is paramount for optimal performance (Keaney et al., 2019). It has been demonstrated that elite athletes who have fewer injuries and illness and complete ≥ 80% of planned trainings in the 6 months prior to major events have a greater chance of achieving their predefined goals (Raysmith and Drew 2016).

5.1.1 Travel Fatigue and Jet Lag

Elite athletes are frequently required to travel both short (e.g. National championships/leagues, squad camps, etc.) and long haul (e.g. Olympic Games, World Championships, Continental events, World Student Games, etc.) for training or competitions which causes travel fatigue and jet lag (Biggins et al., 2020; Van Rensburg et al., 2019; Samuels, 2012; Arendt, 2009). The circadian system has to either advance (eastward travel) or delay (westward travel) however, eastward travel is more difficult as endogenous circadian rhythmns have an ~ 24.25h period making it more difficult to delay the circadian system (Van Rensburg et al., 2019). During any long journey travel fatigue develops over time, irrespective of the mode of travel (Samuels, 2012). Travel fatigue is associated with frequent travel while jet lag is associated with time zone displacement (Walsh et al., 2020). Travel fatigue is caused by the demands of travel (e.g. getting to the airport, poor prior sleep, delays and/or flight changes) (Waterhouse et al., 2007). Travel fatigue refers to a variety of symptoms that occur during or immediately following travel including fatigue, disorientation and headache (Waterhouse et al., 2004). These symptoms are caused by sleep loss, dehydration, hypoxia, discomfort, low air pressure and low himudity (Roach et al., 2018; Brown et al., 2001). Interuptions to sleep, training, and eating patterns while in transit may also induce travel fatigue in athletes (Halson et al., 2019). The degree of fatigue can be managed through careful slection of mode, timing and itinary of travel and management of behaviour during travel (e.g. eating, napping) (Halson et al., 2019). Travel fatigue usually abates after 1-2 days following a good night's sleep (Sack, 2010; Waterhouse et al., 2007).

Jet lag is recognised as a sleep disorder (Thorpy, 2017; Sateia, 2014) that individuals experience after transmeridian travel (across multiple timezones). Travellers with rigid sleep habits appear to be more susceptible to jet lag (Flower et al., 2003). Jet lag involves the sychronisation of the body clock to the new environment (destination time) and it has been suggested that recovery takes place at a rate of approximately 0.5 (west) to 1 day (east) per time zone crossed (Van Rensburg et al., 2020; Sack et al., 2010; Waterehouse et al., 2007). Jet lag is caused by travel across multiple timezones (> 3) with resultant desychronisation of the circadian system to the light/dark cyle of the destination (Van Rensburg et al., 2019). Following long-haul east-west travel involving crossing multiple timezones, the circadian system cannot immediately entrain to the new time zone (Wever, 1983) and initially upon arrival the circadian system is sychronised with the time zone of departure (Winglet et al., 1984). It has been suggested that north-south long haul travel can also cause jet lag due to changes in day length even when no timezones are crossed (Diekman and Bose, 2018).

While the human circadian system takes time to adjust to the destination time, jet lag symptoms such as sleep disturbance, daytime sleepiness, difficulty sleeping at night, irritability, gastrointestinal disruption and reduced cognitive and physical performance persist (Samuels, 2012; Waterhouse et al., 2004). As the body clock adjusts to the new time zone, nocturnal sleep improves and the symptoms of daytime fatigue, reduced motivation, poor mental performance begin to dissipate (Edwards and Waterhouse, 2013). In order to manage and implement strategies to alleviate the symptoms of jet lag it is essential to assess the impact of jet lag in athletes.

5.2 Jet Lag and Athletes

While the research regarding jet lag and athletes is relatively limited some risk factors for jet lag in athletes have been identified:

5.2.1 Chronotype

Chronotype typically influences individual's reaction to jet lag, morning types typically have less difficultly travelling eastward while evening types usually have less difficulty travelling westward (Baehr et al., 2000; Kerkhof and van Dongen, 1996). It has been suggested that the difference is because morning types cope better with the earlier bedtime and waking time required for eastward travel, while evening types are better able to cope with the delayed bedtime and waking time required for westward travel (Reilly et al., 2009; Baehr et al., 2000; Kerkhof and van Dongen, 1996). Research has reported a skew towards morningness in elite athletes (Bender et al., 2019; Lastella et al., 2016; Silva et al., 2012), indicating they may have less difficulty travelling eastward.

5.2.2 Gender

While jet lag tends to affect males and females in the same way, there a number of gender differences that could influence levels of jet lag experienced. Males go to sleep earlier and experience less fatigue after crossing multiple time zones during eastward travel (Waterhouse et al., 2002). It has also been demonstrated the the onset of melatonin secretion is earlier for females hence sleep may occur at a later biological time during travel which could impact the effects of jetlag (Cain et al., 2010; Sack et al., 2007).

5.2.3 Travel Direction

While it has been suggested that jetlag affects people in a similar way regardless of the direction of travel (Sack et al., 2010), it is generally accepted that jetlag is more pronounced on eastbound trips (lengthen the day) than on westbound trips (shorten the day) and it is easier to stay awake than to go to 'bed' early (Eichner, 2020). Fowler et al., (2017)

demonstrated signigicantly ($p<0.05$) higher jet lag symtoms (jet lag, fatigue and reduced motivation) and a greater effect on sleep (later sleep onset, final waking and reduced sleep duration) and performance (YoYo intermittent recovery and 20m sprint) due to westward versus eastward travel in student athletes (n=10). Flying west is associated with less jetlag due to ease of sleep onset following a delay in bedtime. This is likely due to increased fatigue and time awake, the fact that circadian rhythmns are ~ 24.25h, hence delaying is easier than advancing, and the effect of light exposure at the destination promoting adjustments of the circadian rhythm (Reilly et al., 2009). Research in athletes has previously demonstrated that following westward travel (crossing 6 timezones), jetlag peaked after 3 days, while following eastward travel (crossing 8 timezones) the effects are more pronounced and last for as long as 7 days (Lemmer et al., 2002). Roach and Sargent (2019) suggested that following westward travel an athlete will be sleepy in the late morning and early afternoon, experience difficulty falling asleep at their habitual bedtime, and struggle to train and/or compete in the late morning or early afternoon.

A study of masters (i.e. aged > 40 years) triathletes (n=12; age 48 ± 14 years) investigated the impact of northeasterly travel (Australia to Hawaii) on sleep during travel compared to baseline (-4.8 ± 1.2 hours), sleep upon arrival (+0.7 ± 1.0 hours), napping on arrival (36 ± 65mins), and self-reported fatigue (1.1 ± 1.6 Arbitrary Units [AU]) (Stevens et al., 2018). Upon arrival all measures returned to baseline levels within 48 hours and it was also noted that 25% (n=3) of the athletes reported symptoms of illness (2 x upper respiratory tract infection [URTI] and 1 x chest infection) 3-5 days after arrival (Stevens et al., 2018). Recent mathematical modelling of jetlag has highlighted the assymetry in the severity of jetlag between eastward and westward travel (Diekman and Bose, 2018). While north-south travel may cause jet lag even when no timezones are crossed due to the change in day length (Diekamn and Bose, 2018).

A study of elite German athletes (n=13) investigated the impact of both eastward and westward travel on HR and blood pressure and jet lag symptoms (Lemmer et al., 2002). Interestingly, an investigation of collegiate swimmers (n = 40; 18 females and 22 males) who crossed 4 time zones, did not experience negative physiological (sleep, blood pressure, heart rate and cortisol), perceptual (muscle soreness and RPE) or affective (POMS) changes during heavy training (4 x 182.9m swin @ 90% of the swimmer's maximal velocity) (O'Connor et al., 1991). While skeleton athletes (n = 5) travelling from Australia to Canada displayed no significant change in performance despite a significant desrease in pre and post travel salivary cortisol concentration (Bullock et al., 2007). Jet lag has been demonstrated to

have a significant negative effect on performance. The longer the duration of the athletes' events the more likely performance will be influenced by sleep loss (Waterhouse et al., 2004).

5.2.4 Arrival Time and Number of Times Zones Crossed

Arrival time may impact the severity of jet lag symptoms. An investigation of athletes, coaches and academics (n=85) who crossed 10 times zones (UK to Australia), demonstrated those who arrived at midday had less jet lag symptoms than morning arrivals (Waterhouse et al., 2002). In a similar study, investigating the impact of travel from the UK to Australia (10 time zones) in elite athletes (n=39), less jet lag symptoms were reported by those who had made the journey previously (Waterhouse et al., 2000). It has recently been shown that late arrival times cause subsequent reductions in both time in bed and sleep duration (Fowler et al., 2017), as late arrival at their destination and/or transfers reduce the sleep opportunity available to athletes. Travel schedules that minimise the intervals between the last full night's sleep before departure and the first full night's sleep upon arrival reduce jet lag (Fowler et al., 2017; Waterhouse et al., 2000).

5.2.5 Fitness

Physical fitness has been shown to promote sleep in female athletes (Silva and Paiva, 2018), and has been associated with mental toughness which may improve athletes' ability to cope with travel fatigue and jetlag (Reilly et al., 2007). It has been suggested that the diurnal changes in mental and physical performance can influence other circadian rhythmns and could aid the resychronisation to new timezones (Malhotra, 2017), therefore shifting training schedules prior to departure may reduce the impact of jetlag on performance.

5.2.6 Athletic Performance

It has been proposed that there are 3 areas of evidence regarding how jetlag impacts athletic performance (1) the circadian rhythm influences athletic performance (Reilly and Waterhouse, 2009), (2) the negative effects of jet lag (i.e. nocturnal sleep loss, daytime fatigue: reduced motivation and cognitive performance) which can affect athletic performance (Walsh et al., 2020): and (3) the results of games/matches (i.e. win-loss records). American football, basketball and hockey teams, who routinely travel long haul for games, showed teams had a better chance of winning if the team played at a time coinciding with daytime in the time zone they departed from and/or travelling eastward (Huyghe et al., 2021, Roy and Forest, 2018; Jehue et al., 1993).

While there are methodological issues regarding the assessment of the impact of jetlag on athletic performance (Reilly et al., 1997) such as using proxy measures of performance (jump height, grip strength) or simulated competition (e.g. treadmill protocols) and the difficulty measuring athletic performance in team sports (e.g. win vs. loss), there is evidence of circadian rhythmnicity in athletic performance, with time of day effects for jump height, anaerobic and aerobic performance being observed (Leatherwood and Dragoo, 2013). Desychronisation of the circadian rhythmn, as a result of transmeridian travel, has the potential to impact performance (Halson et al., 2019). Until the rhythmns synchronise with the new time zone: sleep, fatigue, motivation and performance may be adversely affected (Zubac et al., 2020). Disruption of the morning nadir and late afternoon peak in performance may occur, with both physical and cognitive performance reduced outside of this circadian peak 'window' (Leatherwood et al., 2013; Reilly et al., 2001).

5.2.7 Cognitive Performance

Jet lag, the resultant sleep loss and/or disruptions to the circadian rhythmn can alter cognitive function (Lee and Galvez, 2012). Mood and complex cognitive tasks deteriorate more quickly than simpler tasks (Reilly et al., 2008). Mental performance is adversely impacted by sleep loss which is directly related to jet lag and crossing multiple timezones (Waterhouse et al., 2004). Previous research has demonstrated that athletes competing in international competitions immediately following long haul travel suffered a decline in performing complex cognitive tasks coupled with feeling of lethargy and loss of motivation (Reilly et al., 1997).

During the preparatory phase for major competitions, athletes undertake international travel to camps and the competitions themselves. Research investigating the sleep of athletes during and after long haul travel is limited and warrants further investigation (Janse et al., 2020; Reilly and Edwards, 2007; Lee and Galves, 2012; Waterhouse et al., 2002). Further research is necessary to assess the impact of jetlag on athletes' sleep as it has has the potential to negatively affect both health and performance. The aim of this study was to assess the impact of long-haul travel and jet lag on the sleep of elite athletes. In order to assess the impact of long-haul travel and jet lag on their sleep the athletes were monitored before, during and after travel (see Figure 5.1). Therefore, the objectives of the study were to: 1.) assess baseline sleep, 2.) the impact of travel on their subjective and objective sleep measures and 3.) assess sleep upon arrival at the destination.

5.3 Methodology

5.3.1 Participants

An entire elite national track cycling squad (n = 7; n = 3 males and n = 4 females; see table 5.1) were recruited through the National Sports Institute. After reading the participant information sheet (see Appendix 9.4.1) written informed consent was provided prior to data collection. The athletes were regarded as elite in line with published definitions i.e. members of a national/professional team (Swann et al., 2015).

5.3.2 Procedure

All procedures were approved by the research ethics committee of the Faculty of Health and Life Sciences, Northumbria University. After providing written informed consent the participants were given an ActiWatch 2 (Philips Respironics ActiWatch 2, Phillips, Amsterdam, Netherlands), instructions and a link to the online sleep diary (see Appendix 9.4). Participants were instructed the wear the ActiWatch 2 daily, except when washing, and complete the CSD–C daily. Following completion of the study all particpants received feedback and a summary of their data (see Appendix 9.4.4).

5.3.3 Measures

Participants wore an activity monitor for 5 days prior to travel, during the long haul travel and 5 days upon arrival at their destination and completed a daily online sleep diary (see Figure 5.1). The athletes did not recive any sleep education and were not emplying any sleep/jet lag countermeasures. All participants travelled in economy class for all flights which involved travel across 7 time zones:

Flight 1: Majorca to Madrid (1h 25mins)

Transit: 2 hours

Flight 2: Madrid to Dubai (7h 15 mins)

Transit: 3 hours

Flight 3: Dubai to Hong Kong (7h 50mins)Travel time: 21.5 hours

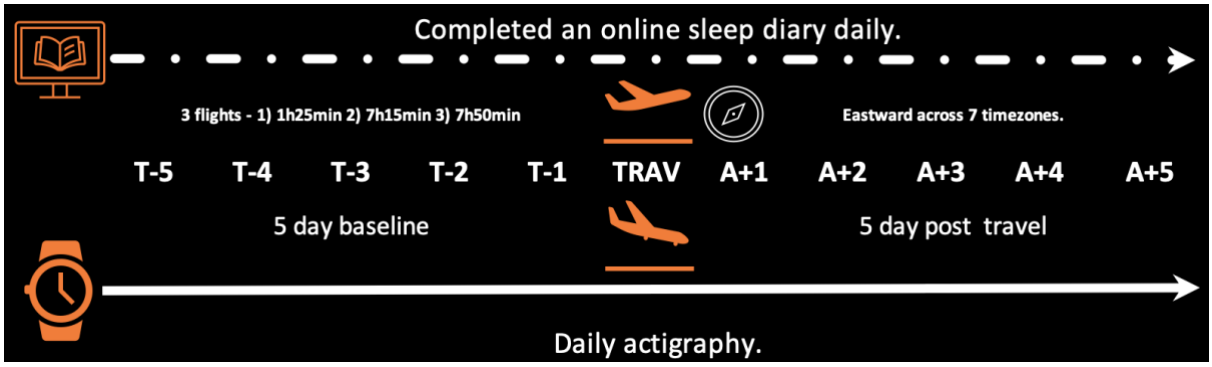

Figure 5.1: Schematic of study design.

5.3.4 Actigraphy

As discussed in Chapter 1, actigraphy is a valid and reliable alternative to PSG for sleep assessment which is widely used in athletic populations (Halson, 2019). The algorithmns for sleep and wake have previously been shown to be accurate in healthy sleepers. However, it must be noted both devices tended to underestimate wake epochs and overestimate sleep epochs, compared to PSG (Toon et al., 2016). As such, activity monitors may have reduced accuracy in individuals with fragmented sleep who have increased waking after sleep onset (Quante et al., 2015). Actigraphy is a valuable clinical and research tool as units are relatively inexpensive, non-intrusive and does not require a sleep technician. In contrast to PSG, actigraphy offers a cost effective and field-based assessment of sleep which can be implemented in 'real-world' settings with athletes (Taylor et al., 2017). The following sleep indices were derived from the actigraphy data (see Table 5.2):

- Time in bed (min): the time spent in bed between going to bed and getting up
- Total sleep time (min): the amount of time spent asleep
- Sleep onset latency (min): the amount of time it took to get to sleep
- Sleep efficiency (%): sleep duration expressed as a percentage of time in bed
- Wake after sleep onset (min): time spent awake between start and end of sleep
- Awakenings (#): number of awakenings

5.3.5 Consensus Sleep Dairy – Core

Sleep diaries are commonly used in conjunction with actigraphy and represent a simple and cost-effective means of subjectively assessing sleep. It has been recommended that varibles to such as bed and wake time, lights out time and sleep quality are assessed for a minimum duration of 1 week (Anderson, 2018). Nevertheless, some limitations exist with sleep diaries due to recall bias, social desireability and expectation, however, these limiations are minimised when sleep diaries are used in conjunction with actigraphy (Quante et al., 2015). Participants were instructed to complete the CSD-C a standardised research sleep diary for each nights sleep for the duration of the study (Carney et al., 2012). The CSD-C included 8 items e.g. bed time, time it took to fall asleep, number of awakenings, duration of awakenings, time of final awakening, time the respondent got out of bed, and a Likert scale self-report rating of sleep quality (Maich et al., 2016). Particpants were also instructed to record levels of fatigue both going to bed and in the morning on a likert scale (1 = Completely exhausted to 7 = Fully Alert) (See Table 5.3).

5.3.6 Data Analysis

All data was analysed using the Statistical Package for the Social Sciences (SPSS Version 26, IBM Corporation) and Jamovi Version 1.6.18 (The Jamovi Project). Q-Q plots and Shapiro-Wilks tests were used to assess the distribution of data. Frequency distribution and descriptive statistics were used to present findings with all data being presented as mean ± standard deviation, mode and/or frequency (Thomas et al., 2015). One way ANOVA, Kruskal-Wallis tests, post hoc analysis and Linear Regression were used to assess the impact of jet lag (day) on the athletes' sleep. To examine the difference in objective sleep measures due to long haul travel, linear mixed models were fitted, considering each sleep variable and day, their interaction as fixed effects, with participant as a random effect.

5.4 Results

At baseline participants' age and body mass were recorded (see Table 5.1), they were given an Actiwatch and began daily sleep diary completion (see Table 5.2 and 5.3). A Chi square analysis demonstrated no significant differences between the participants for gender ($X^2[1, n = 77] = 1.57$, $p = 0.21$).

Table 5.1: Subject characterisitcs

Participant ID	Gender	Age (Y)	Body Mass (kg)
1	Female	25	69
2	Male	28	68
3	Male	25	76
4	Female	33	59.1
5	Female	36	54
6	Female	30	66
7	Male	24	77

Participants' sleep was assessed before, during and after long haul travel using the ActiWatch 2 wrist worn activity monitor (see Table 5.2).

Table 5.2: Summary of actigraphy assessments (mean ± SD)

Day	TIB (min)	TST (min)	SOL (min)	SE (%)	WASO (min)	Awakenings (#)
T-5	650 ± 138	567 ± 126	21.2 ± 38.8	87.4 ± 9.26	37.5 ± 36.2	33.9 ± 25.1
T-4	523 ± 146	426 ± 138	44.4 ± 48.3	80.3 ± 11.8	34.9 ± 13.6	36.9 ± 14.1
T-3	564 ± 74.9	442 ± 114	51.1 ± 65.9	77.3 ± 10.8	48 ± 34.4	44.3 ± 18.7
T-2	577 ± 116	489 ± 125	35.4 ± 40.6	82.7 ± 5.29	39.5 ± 20.3	38.6 ± 17.6
T-1	570 ± 90	472 ± 43.7	28.2 ± 47.8	83.9 ± 7.83	34.3 ± 16.2	40.4 ± 13.5
Trav	363 ± 124	211 ± 77	64.4 ± 42.8	59.8 ± 15.9	27 ± 12.6	20.6 ± 11.1
A+1	384 ± 129	237 ± 83.7	62.7 ± 63.9	65.5 ± 19.9	46.9 ± 41.6	28 ± 23.3
A+2	574 ± 191	463 ± 154	3.43 ± 7.28	84.1 ± 8.44	32.9 ± 19.9	32.7 ± 8.94
A+3	487 ± 85.2	410 ± 91.5	23.4 ± 13.4	85.3 ± 5.91	30.9 ± 18.3	30.4 ± 14
A+4	567 ± 33.8	500 ± 33.7	20.2 ± 13.2	88.7 ± 3.96	35.1 ± 13.4	40 ± 12.9
A+5	595 ± 42	434 ± 176	51.2 ± 54.3	84.4 ± 8.42	37.4 ± 18.5	43.3 ± 20.7

Key
T-5: 5 days before travel
T-4: 4 days before travel
T-3: 3 days before travel
T-2: 2 days before travel
T-1: day before travel
Trav: travel day
A+1: day after arrival
A+2: 2 days after arrival
A+3: 3 days after arrival
A+4: 4 days after arrival
A+5: 5 days after arrival

5.5 Actigraphy

5.5.1 Time in bed (min)

There were statistically significant differences for TIB based on day. A mixed model was used to assess the impact of day on TIB, a significant regression was found ($F(10, 77) = 4.78$; $p < 0.001$; $R^2 = 0.386$). TIB was significantly reduced by 213.1 minutes ($p < 0.001$, SE 44.2, 95% CI [-299.7 to -126.5]) on TRAV and 157.01 minutes ($p < 0.001$, SE 43.6, 95% CI [-242.4 to -71.6]) on A+1 compared to all other days (see Firgure 5.2)

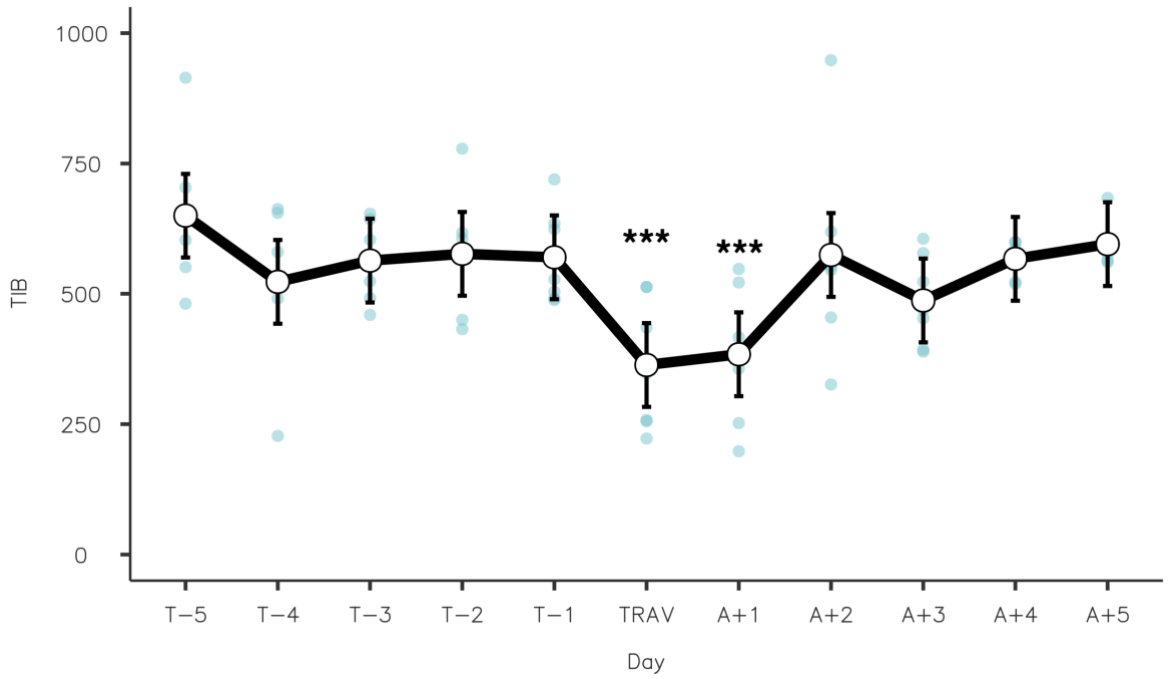

Figure 5.2: Changes in TIB by day.

5.5.2 Total sleep time (min)

TST significantly varied based on day. A mixed model was used to assess the impact of day on TST, a significant regression was found ($F(10, 70) = 7.36$; $p < 0.001$; $R^2 = 0.495$). TST was significantly reduced by 140.43 minutes ($p = 0.015$, SE 56, 95% CI [-250.23 to -30.6]), on T-4, 268.63 minutes ($p < 0.001$, SE 43.6, 95% CI [-353.68.4 to -183.6]) on TRAV, 197.93 minutes ($p < 0.001$, SE 42.8, 95% CI [-281.79 to -114.1]) on A+1 and increased by 87.38 minutes ($p = 0.04$, SE 41.8, 95% CI 5.4 to -169.2]) on A+4 compared to all other days (see Firgure 5.3 And Table 5.3)

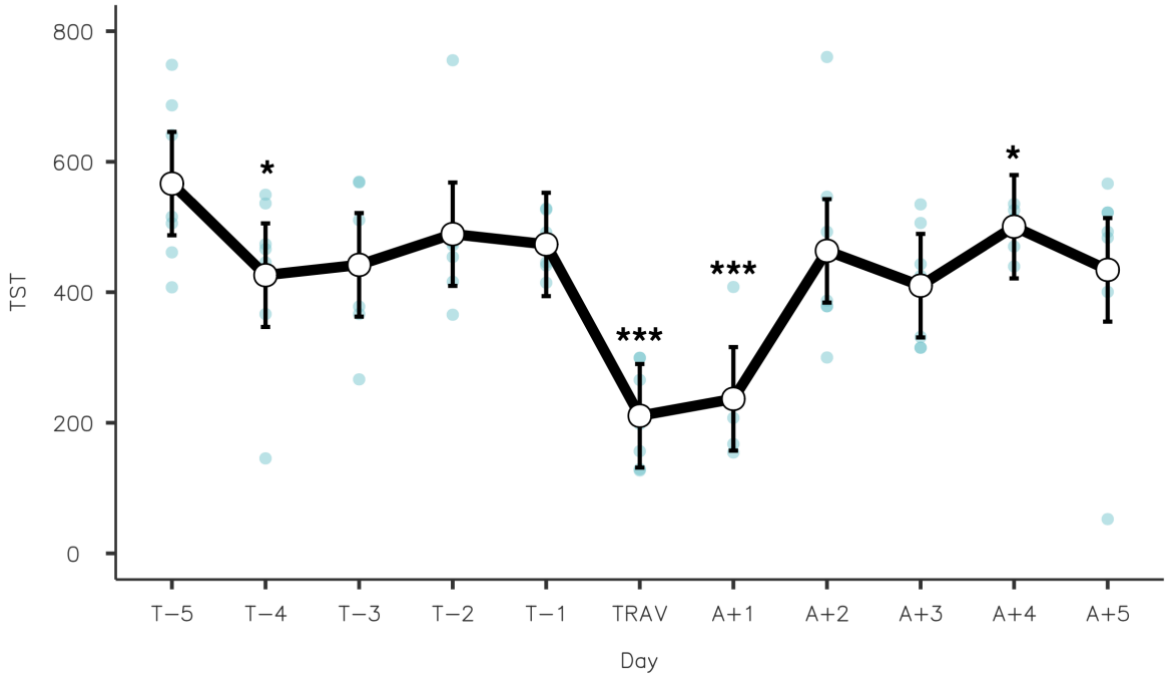

Figure 5.3: Changes in TST by day.

5.5.3 Sleep latency

While SOL increased on TRAV and A+1, a one way ANOVA (Kruskal-Wallis) demonstrated there were no statistically significant differences between SOL scores (X^2[1, n =10] = 19, p>0.05).

5.5.4 Sleep efficiency

SE significantly varied based on day, a mixed model was used to assess the impact of day on SE, a significant regression was found (F(10, 70) = 6.11; p < 0.001; R^2 = 0.452). SE was significantly reduced by 22.63% (p < 0.001, SE 4.08, 95% CI [-30.5 to -14.56]) on TRAV, 13.12% (p = 0.002, SE 4.02, 95% CI [-20.99 to -5.24]) on A+1 and increased by 10.27% (p = 0.011, SE 3.92, 95% CI 2.58 to -17.96]) on A+4 compared to all other days (see Firgure 5.4 and Table 5.2)

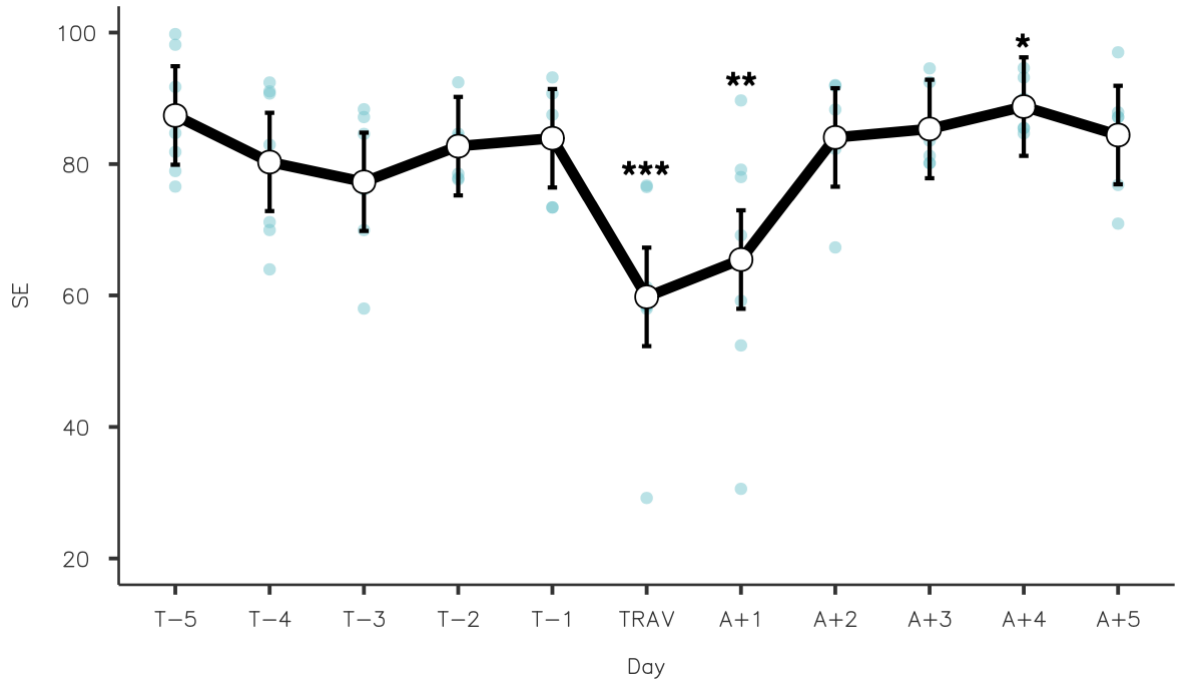

Figure 5.4: Changes in SE by day (*p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.005)

5.5.5 Awakenings

A one way ANOVA was used to assess the effect of day on WASO. There was no statistically significant difference between WASO scores by day ($F(10, 66) = 0.468$, $p = 0.905$). A one way ANOVA was used to assess the effect of day on number of awakenings. There was no statistically significant difference between WASO scores by day within participants ($F(10, 66) = 1.219$, $p = 0.296$).

5.6 Sleep Diary

Participants' self-reported their sleep and fatigue levels both before bed and in the morning after waking before, during and after long haul travel using online sleep diaries (see Table 5.3).

Table 5.3: Summary of sleep dairy responses (mean ± SD; mode)

Day	TIB (min)	TST (min)	SOL (min)	SE (%)	WASO (min)	Awakenings (#)	Fatigue (Bedtime)	Fatigue (Morning)	SQ

T-5	666 ± 139	624 ± 156	12.1 ± 12.9	93 ± 5.67	30.1 ± 26.6	5.14 ± 3.48	3 (Moderate)	3 (Moderate)	4 (Good)
T-4	536 ± 140	474 ± 132	26.4 ± 27.3	88.2 ± 6.03	35.1 ± 14.2	4.43 ± 1.81	3 (Moderate)	3 (Moderate)	4 (Good)
T-3	560 ± 80.8	500 ± 100	29.3 ± 41.8	88.6 ± 9.12	30.6 ± 23.8	4.29 ± 1.25	3 (Moderate)	3 (Moderate)	4 (Good)
T-2	587 ± 126	530 ± 135	21.7 ± 23.6	89.8 ± 5.39	35.4 ± 23.7	4.29 ± 1.7	3 (Moderate)	5 (Somewhat fresh)	4 (Good)
T-1	558 ± 70.3	504 ± 54.1	24.6 ± 43.4	90.7 ± 6.39	29.7 ± 13.5	5 ± 2.89	3 (Moderate)	3 (Moderate)	4 (Good)
Trav	426 ± 119	357 ± 116	44.3 ± 33	82.9 ± 9.31	24.1 ± 12.5	4.29 ± 2.63	1 (Exhausted)	1 (Exhausted)	1 (Very Poor)
A+1	405 ± 140	327 ± 123	42.1 ± 28.8	81 ± 11.2	36.6 ± 38.3	3.29 ± 2.69	2 (Very tired)	2 (Very tired)	1 (Very poor)
A+2	591 ± 183	558 ± 163	5.71 ± 7.32	94.7 ± 2.03	28.1 ± 21.1	5.43 ± 3.26	2 (Very tired)	2 (Very tired)	3 (Fair)
A+3	508 ± 75.8	460 ± 82.6	19.3 ± 12.4	90.4 ± 5.16	28.4 ± 16.3	4.57 ± 2.15	3 (Moderate)	3 (Moderate)	4 (Good)
A+4	578 ± 28.8	528 ± 38	20.7 ± 15.9	91.4 ± 3.09	28.9 ± 21.7	4.14 ± 3.13	3 (Moderate)	4 (A little tired)	4 (Good)
A+5	615 ± 48.4	542 ± 50.8	47.1 ± 48.3	88.3 ± 7.87	26.3 ± 14	3.57 ± 2.07	4 (A little tired)	3 (Moderate)	4 (Good)

5.6.1 Sleep dairy

As the data were not normally distributed one way ANOVAs (Kruskal-Wallis) were used to assess the effect of day on the various sleep parameters from participants' sleep diaries. There were no statistically significant differeences for SOL ($X^2[1, n =10] = 17.47$, $p = 0.065$; $\varepsilon^2 = 0.23$), WASO ($X^2[1, n =10] = 3.05$, $p = 0.98$; $\varepsilon^2 = 0.04$) and awakenings ($X^2[1, n =10] = 3.57$, $p = 0.97$; $\varepsilon^2 = 0.05$).

Sleep diary TIB varied significantly by day ($X^2[1, n =10] = 25.21$, $p \leq 0.005$; $\varepsilon^2 = 0.33$) (see figure 5.5). Dwass-Steel-Critchlow-Fligner post hoc analysis indicated no pairwise comparisons. A linear regression model was used to assess the impact of day on sleep diary TIB, there was a significant effect found for TIB x day ($F(10, 66) = 3.24$; $p \leq 0.002$; $R^2 = 0.228$).

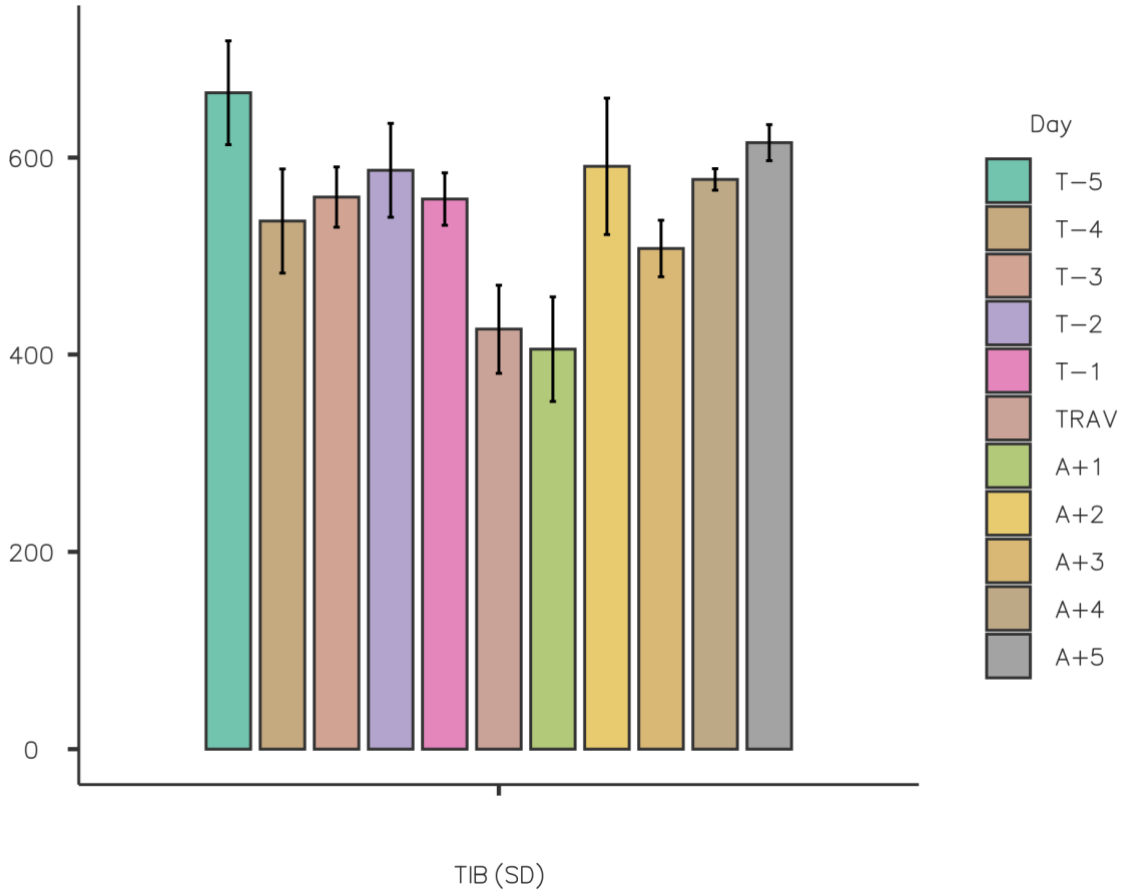

Figure 5.5: Sleep diary total TIB by day (mins).

Sleep diary TST significantly varied based on day ($X^2[1, n = 10] = 28.08$, $p \leq 0.002$; $\varepsilon^2 = 0.37$) (see figure 5.6). Dwass-Steel-Critchlow-Fligner post hoc analysis indicated no pairwise comparisons. A linear regression model was used to assess the impact of day on sleep diary TST, there was a significant effect found for TST x day ($F(10, 66) = 4.1$; $p \leq 0.01$; $R^2 = 0.38$).

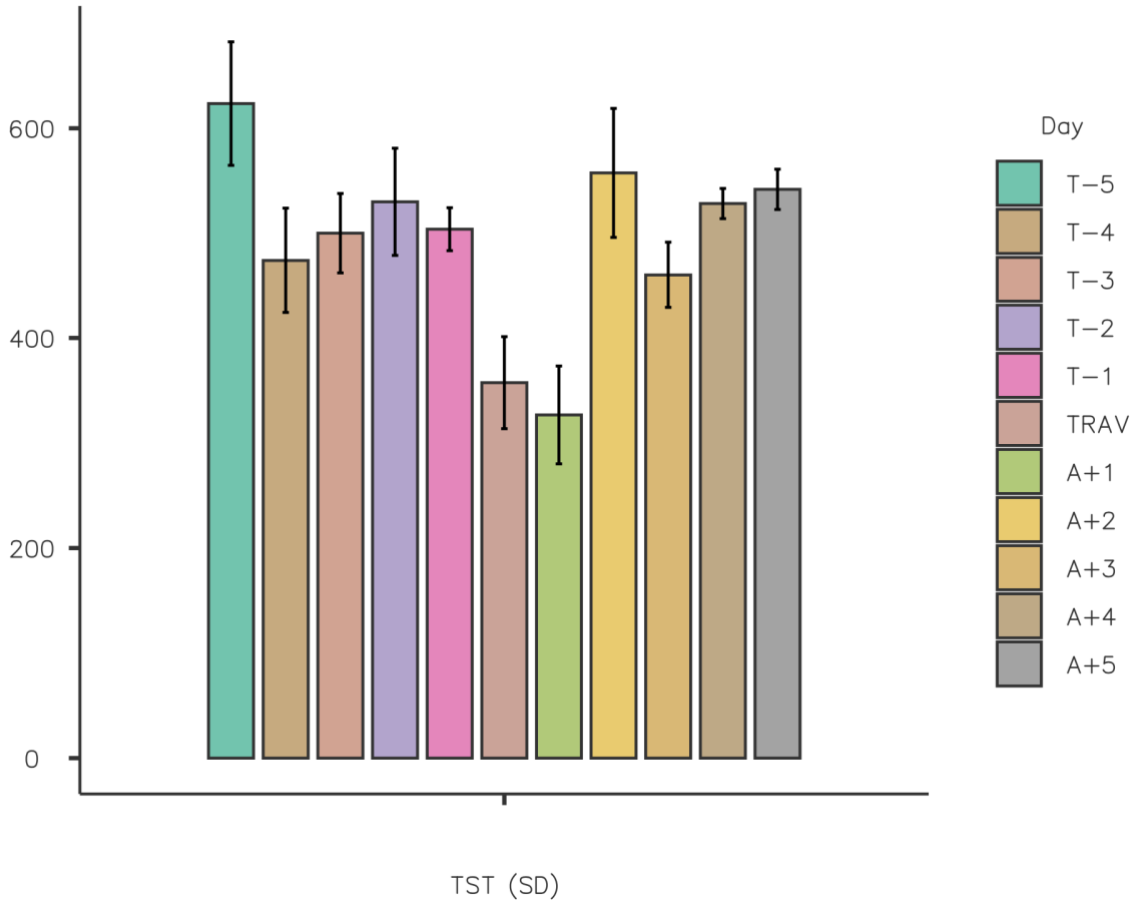

Figure 5.6: Sleep diary total TST by day (mins).

Sleep dairy SE % significantly varied based on day (see figure 5.7), a one way ANOVA (Kruskal-Wallis) ($X^2[1, n = 10] = 19.1$, $p \leq 0.04$; $\varepsilon^2 = 0.25$). Dwass-Steel-Critchlow-Fligner post hoc analysis indicated no pairwise comparisons. A linear regression model was used to assess the impact of day on fatigue going to bed. There was a significant effect found for SE % x day ($F(10, 66) = 2.32$; $p = 0.021$; $R^2 = 0.26$).

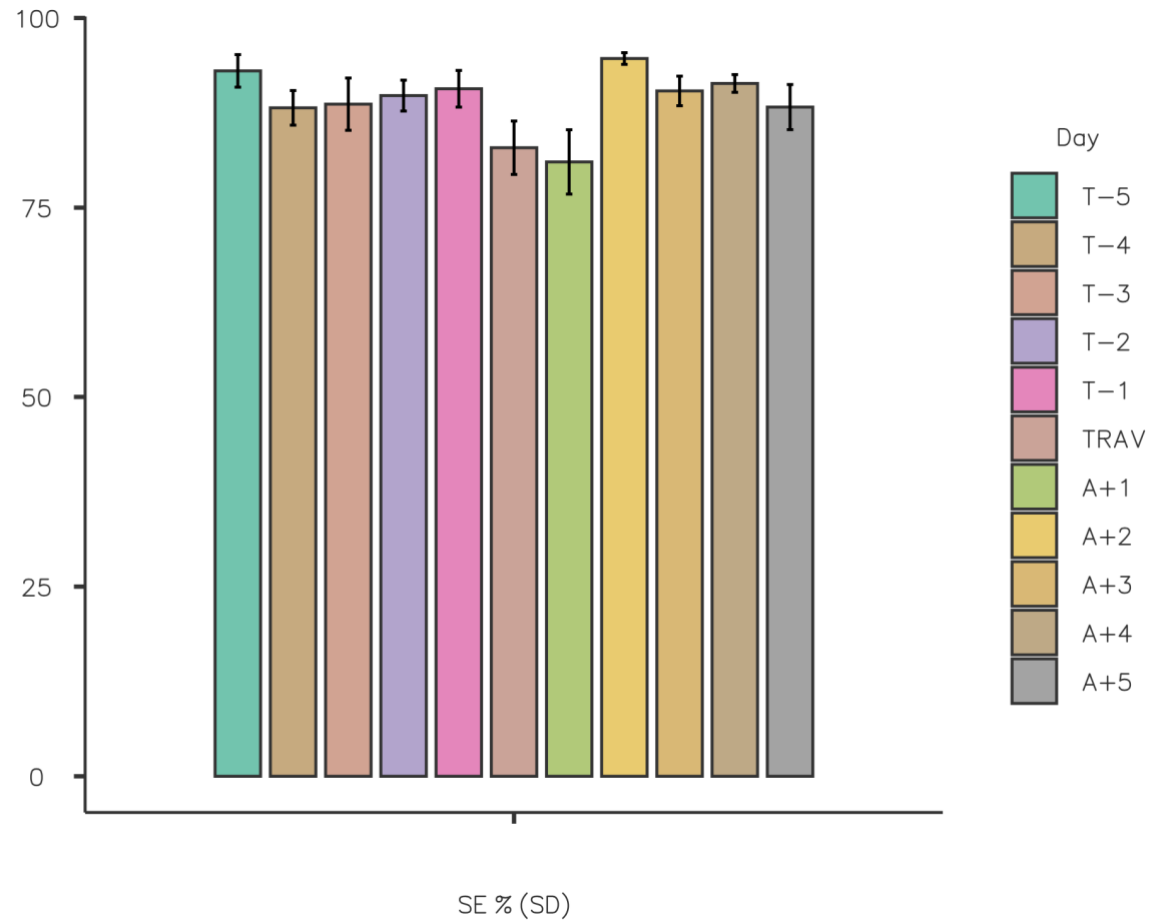

Figure 5.7: Sleep diary sleep efficiency (%).

5.6.2 Fatigue

Fatigue significantly varied based upon day (see figure 5.8), one way ANOVAs (Kruskal-Wallis) were used to assess the effect of day on fatigue as the data were not normally distributed. There was a statistically significant difference between fatigue going to bed scores by day ($X^2[1, n = 10] = 40.1$, $p \leq 0.001$; $\varepsilon^2 = 0.528$). Dwass-Steel-Critchlow-Fligner post hoc analysis indicated significant differences between TRAV and T-5, T-4, T-3, T-2, T-1, A+4 and A+5. A linear regression model was used to assess the impact of day on fatigue going to bed. There was a significant effect found for fatigue going to bed x day ($F(10, 66) = 8.22$; $p \leq 0.001$; $R^2 = 0.555$). There was no statistically significant difference between fatigue in the morning scores by day ($X^2[1, n = 10] = 10.8$, $p = 376$; $\varepsilon^2 = 0.142$).

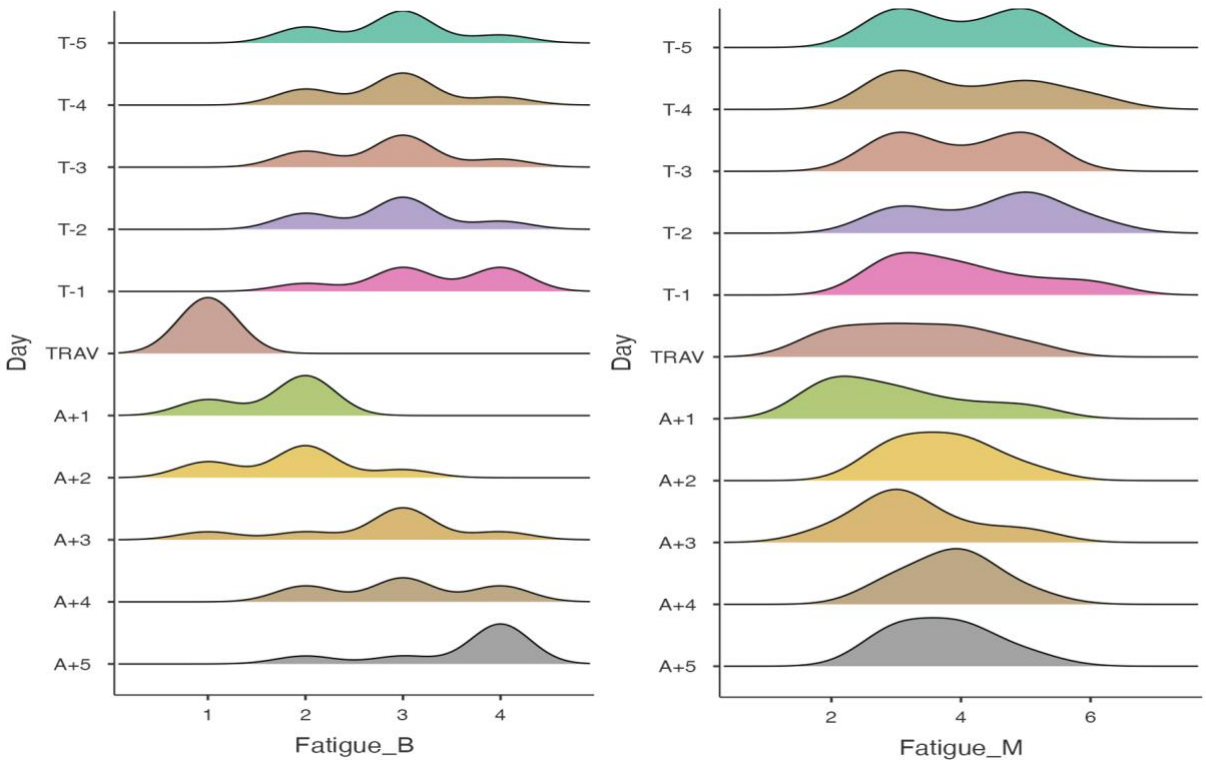

Figure 5.8: Comparision of self-reported fatigue going to bed and in the morning.

5.6.3 Sleep quality

SQ significantly varied based on day (see figure 5.9), a one way ANOVA (Kruskal-Wallis) was used to assess the effect of day on SQ as the data were not normally distributed. There was a statistically significant difference between SQ by day ($X^2[1, n = 10] = 3.15$, p ; $\varepsilon^2 = 0.588$). Dwass-Steel-Critchlow-Fligner post hoc analysis indicated significant differences between TRAV and T-5, T-4 and T-3. A linear regression model was used to assess the impact of day on sleep quality, there was a significant effect ($F(10, 66) = 9.54$; $p \leq 0.001$; $R^2 = 0.529$).

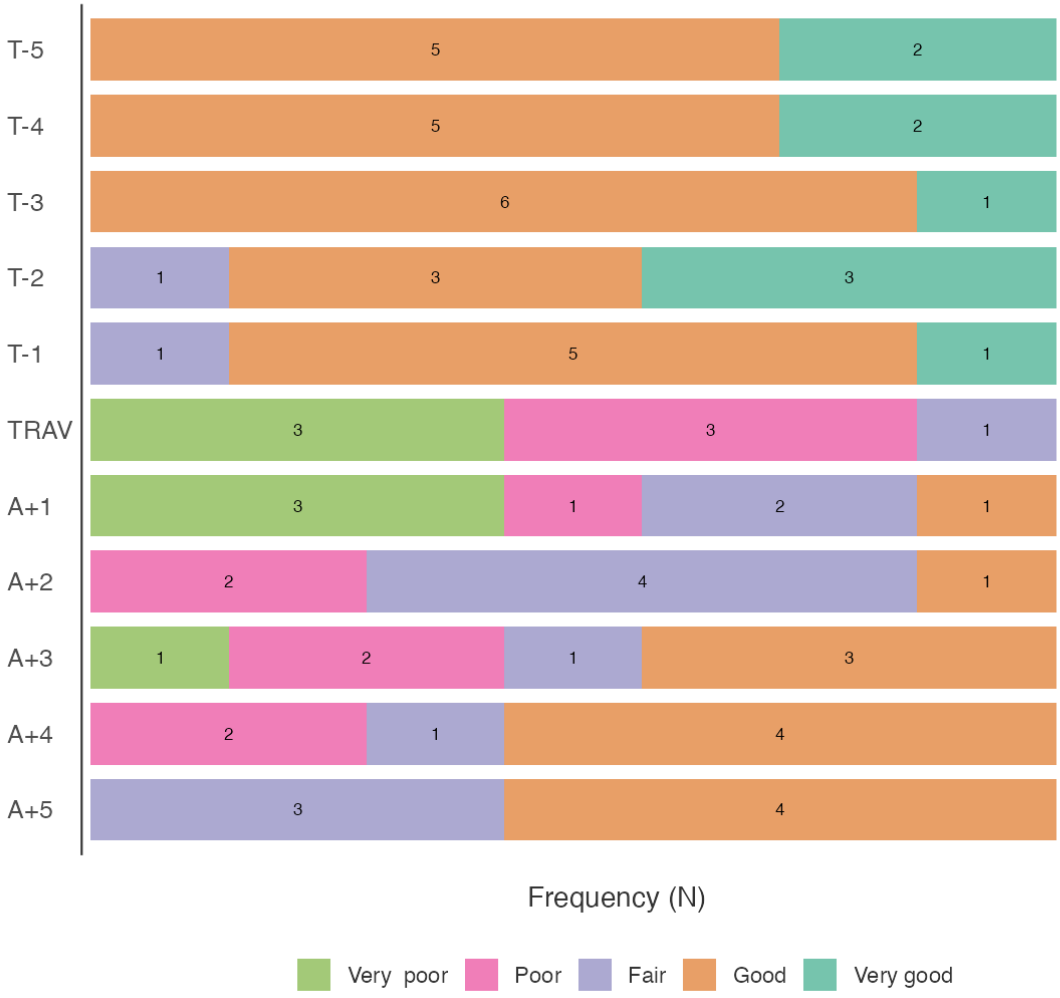

Figure 5.9: Self report sleep quality by day.

5.7 Discussion

The aim of the current study was to assess the sleep of elite athletes before long haul travel and the subsequent impact on sleep whilst in transit and upon arrival at their destination, in order to provide a) an indicator of what sleep parameters are affected by competition travel which will inform what sleep parameters an intervention is needed to address and b) an overview of when the intervention may be most beneficial over the course of competition travel. It has previously been proposed that jetlag affects athetes in 3 key ways (1) the circadian rhythm influences athletic performance (Reilly and Waterhouse, 2009); (2) the negative effects of jet lag (Walsh et al., 2020), and (3) the impact of travel on competition outcomes (Huyghe et al. 2021; Roy and Forest, 2018; Jehue et al., 1993). Unless the negative effects of both long haul travel and the resultant jet lag upon arrival can be managed or minimised then athlete health, well-being and performance are likely to be

affected (Fowler et al., 2020). A recent study investigating the impact of jet lag on the sleep of an elite Rugby union team (n = 37) following long haul westward travel, showed TST reduced during and after travel but returned to normal after 2 days of adjustment upon arrival (Smithies et al., 2021). A strength of this study is the fact that the athletes had not received any sleep education and did not employ any sleep strategies or jet lag counter measures during travel, and the findings could be used to inform future practice. The results of the current study indicated that the following sleep parameters: actigraphy derived TIB, TST and SE and sleep diary derived TIB, TST, fatigue going to bed and SQ were significantly negatively impacted by long haul travel. No significant effects were observed for long haul travel on WASO, number of awakenings and fatigue in the morning. In the current study, the majority of sleep disruption was observed during the initial 48h post travel period, this is consistent with previous research that highlighted that the greatest disruption occurred in the first 48 hour period following travel (Smithies et al., 2021; Stevens et al., 2018). It is clear from the current study and previous research that teams/athletes should complete long-haul travel 5-6 days prior to competition to allow sufficient time for athletes to recover from travel fatigue and jetlag.

Actigraphy measured TIB was significantly reduced as a result of travel (363 ± 124mins) and on the first night (384 ± 129) after arrival, these findings were also demonstred in the sleep dairy TIB for TRAV and A+1. Indeed, mean TIB for TRAV and A+1 were less than current sleep recommendations (7-9h) for adults (Hirshkowitz et al., 2015), hence there was not enough TIB to gain adequate total sleep times. The negative impact of long haul travel on TIB on TRAV and A+1 was observed for all athletes. It has been suggested that sleep can be difficult on long haul flights due to an uncomfortable upright seating/sleeping position, light, noise and the environment and routine on the flight (Fowler et al., 2020; Roach et al., 2018; Stevens et al., 2018). It is common practice to advise athletes to schedule their sleep on a long haul flight to coincide with nighttime at their destination to prevent their sleep/wake cycle from ‘anchoring’ to the departure timezone (Roach and Sargent, 2019; Reilly et al., 2007). However, it has recently been recommended that in order to maximise sleep and minimise the impact of jet lag athletes should sleep during a ‘sleep window’, that corresponds to night-time at the place of departure (i.e. during biological night time when the circadian system is promoting sleep) (Fowler et al., 2020). Fowler et al., (2020) demonstrated that this approach reduced sleep disruption during travel but also had no negative effect on sleep or jetlag symptoms post travel.

A range of 7-9 hours sleep has been suggested as appropriate for healthy adults (Hirshkowitz et al., 2015; Watson et al., 2015), however, athletes may require more sleep due to the physical and psychological demands of their sport associated with training and competition (Bird, 2013). Actigraphy TST was significantly reduced on TRAV (211 ± 77mins) and A+1 (237 ± 83.7mins) compared to the other days. Sleep diary TST was also reduced on TRAV and A+1. While TST during baseline was adequate, TST was less than current sleep recommendations (i.e. 7-9h) (Hirshkowitz et al., 2015; Watson et al., 2015), on TRAV, A+1 and A+3, therefore it was not possible for the athletes to get adequate sleep. Previous research has suggested that sleep duration was decreased during travel but increased on the first night after arrival (Stevens et al., 2018). This contrasts with the current study whereby TST was significantly reduced on TRAV and A+1. Indeed, for all athletes the range of sleep time on both TRAV (127 - 300 mins) and A+1 (155 - 408 mins) was below the recommended range for athletes (7-9h) (Hirshkowitz et al., 2015), potentially chronotype had a role to play i.e. being uable to delay wake time. Lastella et al., (2014) noted reduced TST (6.6 ± 1.3h per night compared to baseline 7.5 ± 1.3h) immediately following long haul eastward travel crossing 8 timezones (Sydney to Denver, Colorado) however, it was noted that the altitude of the destination may have been a confounding factor.

Sleep preservation is an important strategy in the management of jetlag (Arendt, 2018). Sleep hygiene has been shown to help healthy adults overcome travel fatigue following 24-hours of simulated international travel and while good sleep hygiene may not phase shift the circadian system, some sleep habits (e.g. use of electronic devices) can induce phase shifts (Fowler et al., 2015). Long haul and overnight travel are most likely to cause disrupted sleep patterms and adversely affect both sleep quality and quantity (Fowler et al., 2016). Similar to the current study, an investigation of the impact of long haul international travel (18h, -4h timezone shift) in elite male football players (n=15) on sleep highlighted significantly ($p>0.05$) reduced sleep duration and sleep efficiency during and immediately following travel (Fullagar et al., 2016).

In athletic populations when sleep is reduced to < 7 hours cognitive performance (i.e. alertness, reaction time, memory and decision making) and physical performance and injury risk are adversely affected (Charest and Grandner, 2020; Laux et al., 2015; LeMeur et al., 2013). Sleep loss is central to the negative impact of jet lag on performance, daytime fatigue and gastrointestinal comfort (Van Rensburg et al., 2020; Fowler et al., 2017). A recent study in Rugby 7's players (n=17; aged 25.4 ± 5.1 year) during the competitive season indicated that long haul travel had a neglible impact on actigraphy assessed sleep quantity and quality

in the immediate period after travel, however it was suggested that the team had implemented efficient and robust travel strategies (Leduc et al., 2021), it must also be noted that given their age this group may have more plasticy in their sleep system (Ohayon et al., 2004). Previous research in Rugby 7's has indicated that a simple dose response relationaship does not exist between travel duration, competition demands, and number of time zones crossed and that highly individual responses are observed reinforcing the need for individualised support (Fowler et al., 2019). The role of sleep is increasingly recognised in terms of both general health and athletic performance (Jukic et al., 2021) and it is clear that in the current study jet lag had a significant negative impact for 48h post travel. Athlete sleep need could be assessed subjectively, indeed a recent study in elite athletes (n = 175) included a self-report assessment of sleep need, athletes reported an average sleep need of 8.3 ± 0.9 hours (Sargent et al., 2021). An individualised approach incorporating an assessment of the athlete's perceived sleep need should be employed. As such, a one size fits all approach to athlete sleep recommendations, sleep hygiene strategies and interventions to reduce the symptoms and impact of jetlag may be inadequate (Walsh et al., 2020).

A common symptom of jet lag following long haul eastward travel is sleep disruption due to delayed SOL (Fowler et al., 2017; Reilly et al., 2009). In the current study SOL significantly increased on TRAV (64.4 ± 42.8min) and A+1 (62.7 ± 63.9min), however, SOL exceeded 20 minutes on all days except A+2 (3.43 ± 7.48 mins), which may account for the lack of significant pairwise comparisons in SOL by day. Long SOL has previously been reported as an issue in elite athletes related to sleep inadequacy in general, not just during travel, which can be attributed to insomnia (Tuomilehto et al., 2017; Schaal et al., 2011).

Reduced TIB and TST due to travel and jetlag resulted in poorer SE% <85% (i.e. poor sleep), it was observed on T-4 (80.3%), T-3 (77.3%), T-2 (82.7%), T-1 (83.9%), TRAV (59.8%), A+1 (65.5%), A+2 (84.1%) and A+5 (84.4%). However, SE was reduced in the 48h period post travel then began to return to baseline levels, this was consistent with previous research that demonstrated significant reductions in SE both during travel and the subsequent 48hours (Fullagar et al., 2016). It has recently been shown that SE is reduced following long haul travel for approximately 72 hours (Fowler et al., 2020).

In the current study there was a significant difference between fatigue going to bed between TRAV and T-5, T-4, T-3, T-2, T-1, A+4 and A+5. There was no significant impact observed for jetlag on awakenings, number of awakenings or fatigue in the morning. It has previously been suggested that athletes report more fatigue during long haul travel than following training and competition (Calleja-Gonzalez et al., 2020). In order to support

athlete health, travel experience, and performance the implementation of sleep hygiene practices, nutritional programming, load monitoring, fatigue reporting and recovery practices are paramount particualry during travel (Calleja-Gonzalez et al., 2020).

There was a significant negative effect on SQ as a result of long haul travel with TRAV being significantly different than T-5, T-4 and T-3. Travel has previously been indicated as a sport related risk factor for inadequate and/or non-restorative sleep (Nédélec et al., 2018; Gupta et al., 2017). Recovery from jetlag and subsequent improvement in sleep quality requires resynchronisation of the circadian rhythm to the destination light-dark cycle (Roach and Sargent, 2019). Following eastward travel, the circadian rhythm requires time to advance which is generally more difficult, it has previously been suggested that resychronisation following eastward travel requires 1 day per timezone crossed (Van Rensburg et al., 2020). However, in the current study the negative effects of jetlag began to dissipate after 48 hours.

5.7.1 Strengths and Limitations

A strength of the current study is the use of actigraphy and the inclusion of a baseline assessment period. The American Academy of Sleep Medicine (AASM) recommend that actigraphy be conducted for a minimum of 72 hours to 14 days (Smith et al., 2018). It has been suggested that actigraphy is useful for sleep monitoring in athletes and can be incorporated during training cycles for a 1-2 week period (Halson, 2019). The Actiwatch 2 has demonstrated reliability and accuracy for assessing true sleep, true wake, and the ability to assess both sleep and wake (Toon et al., 2016). The Actiwatch 2 has also been validated in athletic populations (Fuller et al., 2017; Sargent et al., 2016). Actigraphy is useful in athletic populations as it allows for longitudinal monitoring in 'real-world' settings which allows assessment of sleep/wake across multiple days, with low subject burden and the removal of recall bias (Quante et al., 2015). However, due to the Actiwatch's sampling rate (32Hz), memory (1Mbit) and a maximum battery life of ~ 22 days at 15 sec epochs (McDevitt et al., 2021), the data collection period was limited to 11 days which could have impacted the reliability of the data. However, as all sleep variables started to return to baseline levels 48h post travel hence, the data collection period appears to have been long enough to assess long haul travel related changes in sleep variables.

The fact that chronotype was not assessed in the current study is a limitation as jet lag following eastward travel may be less pronpounced in morning types (Reilly et al., 2009; Baehr et al., 2000; Kerkhof and van Dongen, 1996). As discussed, previous research has suggested a skew towards morningness in elite athletes (Bender et al., 2019; Lastella et al.,

2016; Silva et al., 2012) which might partially explain the findings of the current study. However, the aim of the study was not to assess mediators or moderators of jet-lag but to identify the sleep parameters that are most affected by competition travel.

A further limitation of the current study is the small sample size (n=7) however, it must be noted that an entire national squad completed the study. The rareness of elite athletes makes recruiting such participants in large numbers difficult (Sands et al., 2019).

5.7.2 Practical applications

These findings support the suggestion that when athletes are required to travel across multiple timezones, particularly for major competitions it is essential to allow enough time between arrival at the destination and the start of the event for athletes to recover and adjust to the new time zone (Lastella et al., 2019). In the current study TST, TIB, SE (%), fatigue going to bed and SQ were significantly adversly affected both during and after (+48 hours) long haul travel, all measures began to return to baseline values by A+3. These findings are similar to a recent study of Rugby players which suggested that long haul travel should completed at least 6 days prior to competition (Smithies et al., 2021). The development and employment of individualised jetlag interventions, including sleep hygiene practices, may help reduce jetlag symptoms and severity (Fowler et al., 2014).

5.8 Conclusion

Eastward long haul travel had significant negative impacts on athletes' sleep (TIB, TST, SOL, SE, fatigue going to bed and SQ) particuarly in the initial 48h period post travel. Actigraphy highlighted significant reductions in TIB, TST and SE (%) due to long haul eastward travel, particaurly in the 48 hoursd after travel, after this period all measures began to return towards baseline. Sleep diary data exhibited significant reductions in TIB, TST, SE, SQ and a significant increase in fatigue going to bed as a result of long haul eastward travel. The level of sleep disruption observed in the 48h post eastward travel in the current study could negatively impact both althete health and performance. In order to facilitate the development of interventions to reduce the symptoms and severity of jetlag objective and subjective asessements of sleep should be coupled with assessments of chronotype and perceived sleep need.

5.9 Study 2 - Link to Next Chapter

The aim of this study was the asses the impact of long-haul eastward travel (across 7 timezones) on the sleep of elite athletes. The results indicated that the following sleep parameters: actigraphy derived TIB, TST and SE and sleep diary derived TIB, TST, fatigue

going to bed and SQ were significantly negatively impacted by long haul travel particualry on TRAV and A+1 i.e. the 48 hours post travel. Characterising the sleep and recovery of athletes in general (Study 1), and under specific circumstances (i.e. long-haul eastward travel: Study 2) allowed the identification of the specific sleep and recovery areas of concern in this population. Together, the results of these studies are of particular interest in terms of tragets for sleep interventions in athletes. The next step in the research was the implementation and assessment of the impact of a simple nutritional intervention (kiwifruit) on athlete sleep and recovery (Study 3), with the aim to determine whether a) it improves sleep generally and b) whether it improves those sleep dimensions most poorly affected in athletes.

Chapter 6: Kiwifruit consumption and sleep.

The previous studies (Study 1 and Study 2) suggest that sleep (TST, TIB, SE and SQ) and recovery (sport specific recovery and fatigue) parameters are affected in athletes, due to competing factors including competition travel. The aim of the present study is to determine whether a simple nutritional intervention can alieviate these issues.

6.1 Introduction

Elite athletes are predisposed to challenges to their sleep, such as habitual short sleep duration (< 7 hours per night) and poor sleep quality (e.g. fragmented sleep) (Walsh et al., 2021). The multifaceted demands placed on elite athletes including the frequency, volume, intensity and timing of training and competition (Walsh et al., 2021; Halson, 2019; Sargent et al., 2014), performance anxiety (Juliff et al., 2015; Erlacher et al., 2011) and travel requirements (Biggins et al., 2020; Fowler et al., 2015) can all negatively impact sleep health. Good sleep health is characterised by satisfaction, appropriate timing, adequate duration, high efficiency, and sustained alertness during waking hours, and can be assessed in athletes using the Regulatory, Satisfaction, Alertness, Timing, Efficiency and Duration questionnaire (RU SATED) (Buysse et al., 2014). Sleep health is a concept which involves a holistic view of sleep as opposed to individual symptoms and disorders (Hale et al., 2020). Other lifestyle factors (e.g. nutrition, caffeine use) and exposure to technology (i.e. blue light exposure at night), can also have a detrimental impact on athletes' sleep (Walsh et al., 2021). Poor sleep and resultant under-recovery can negatively impact training adaptations, increase the risk of maladaption and reduce subsequent performance (Gupta et al., 2017), which may lead to non-functional overreaching (NFO) in the shorter term and over-training syndrome (OTS)/unexplained underperformance syndrome (UUPS) in the longer term (Lewis et al., 2015a; Meeusen et al., 2013).

The health benefits of consuming fruit are well documented (Boeing, 2012), it has previously been suggested that athletes' sleep could be improved by analysing and improving their eating habits by for example, increasing their intake of fruit, vegetables and fish while reducing their intake of processed foods (Ordóñez et al., 2017). Due to the 'food first' approach adopted by many athletes, there is scope for investigation of 'functional food' based interventions (i.e. Kiwifruit) designed to promote athlete recovery and/or enhance sleep quality and quantity. As discussed in Chapter 3, Kiwifruit are platabale nutritionally dense containing a range of nutrients that can benefit sleep and recovery in athletes. Contemporary research has focused on the health benefits of Kiwifruit, particularly in

relation to antioxidant capacity, digestion, iron nutrition, metabolic health and immune function (Singletary, 2012).

Given the adoption of a 'food first' approach by many athletes (Close et al., 2016), there is scope for investigation of food based interventions designed to promote athlete recovery and/or enhance sleep health. Antioxidants are commonly consumed by athletes in an attempt to reduce oxidative stress, following training. Further research is necessary to develop practical guidelines for antioxidant supplementation to enhance training adaptation and/or post-exercise recovery. It has been suggested that a high intake of antioxidants could potentially reduce training adaptations (Gomez-Cabrera et al., 2015). It is accepted that repeated exercise bouts (i.e. training) induce disruption in skeletal muscle homeostasis that regulate training adaptations (Cobley et al., 2015a; Cobley et al., 2015b). It has been reported that high doses of antioxidants could reduce training adaptations of muscle mitochondrial biogenesis and VO_{2max} (Gomez-Carbrera et al., 2008). However, not all antioxidant studies have demonstrated negative effects and it has been suggested that the specific antioxidant used, the dose and timing of ingestion all affect outcomes (Mankowski et al., 2015). It must be noted that the majority of research investigating the effects of antioxidant supplementation to date, has employed high doses/supplementation as opposed to wholefood interventions. At present, there has been no data published that suggests that consumption of high antioxidant fruit and/or vegetables reduces training adaptations.

Kiwifruit have also been shown to contain melatonin (Morin and Benca, 2012), which plays an important role in circadian rhythm regulation i.e. getting to sleep and maintaining sleep are easiest at and after the onset of melatonin secretion. The Serotonin content in Kiwifruit may contribute to improved sleep while the rich antioxidant content may supress free radical expression and inflammatory cytokines. Folate deficiency has been linked to insomnia and restless leg syndrome, the folate in Kiwifruit may improve folate status and consequently improve sleep (Lin et al., 2011). Although folates are widely consumed in the diet, they are destroyed by cooking or processing. Kiwifruit are consumed in their raw form and contain 0.23 ± 0.04μg/g of total folate, 80% higher than carrot juice and 15% higher than orange juice (Wyatt et al., 1970), and it has been estimated that a Kiwifruit contains 10% of the average daily requirement (Ferguson and Ferguson, 2002). A small, randomised crossover study comprising of 6 males and 8 females, demonstrated consumption of varying doses of Kiwifruit (1-3/d x 3 weeks, with 2-week washout between doses) resulted in a significant increase in plasma Vitamin C levels (Collins et al., 2003). Compared to baseline, consumption of 2 Kiwifruit daily significantly raised plasma Vitamin C levels by 20%

(73µM ± 4; $p<0.01$) (Collins et al., 2003). Additionally, improved antioxidant status was evident, lymphocytes isolated from blood collected from participants demonstrated decreased sensitivity to oxidative attack by (H_2O_2), in vitro and endogenous oxidation of lymphocyte DNA was also decreased (Collins et al., 2003). However, it must be noted that the results of this study may not be generalizable due to the very small sample size.

In the last decade Kiwifruit has received attention in terms of potential sleep promoting properties. To date, the research has focused on populations either self-reporting or having a diagnosed sleep problem. A study involving volunteers (n=25) who self-reported sleep disturbance demonstrated consumption of 2 Kiwifruit 1 hour before bedtime for 4 weeks significantly improved actigraphy measured total sleep time (16.9%) and sleep efficiency (2.4%) ($p<0.001$) (Lin et al., 2011). Self-report measures of sleep also improved significantly, WASO reduced (-28.9%), sleep onset latency reduced (-35.4%) while sleep efficiency increased (5.4%) ($p\leq0.002$) (Lin et al., 2011). Sleep quality and duration were significantly improved following the 4-week Kiwifruit intervention, however it must be noted that sleep was not monitored during the intervention period In a similar study, students (n =74) with diagnosed insomnia (using the Bergen Insomnia scale) consumed either 130g of Kiwifruit or a placebo (130g pear), 1 hour before bed for 4 weeks and sleep was assessed by both actigraphy and sleep diaries. While there were no statistically significant differences in objective measures of sleep, there were statistically significant group x time effects for subjective sleep quality ($F1,51 = 5.88$, $p<0.05$) and daytime function ($F1,51 = 4.79$, $p<0.05$) (Nødtvedt et al., 2017). These promising findings warrant further investigation within athletic populations in relation to Kiwifruit consumption and their interaction with sleep and recovery. Further research is necessary to investigate the potential benefits and practical application of Kiwifruit consumption to promote post-exercise recovery and/or improve sleep quality and quantity in athletes.

The current study is the first step in the development of a specific 'whole-food' nutritional intervention for optimising sleep quality, sleep quantity, sleep health and/or enhancing post-exercise recovery in elite athletes. This is the first study to investigate the impact of Kiwifruit consumption on the sleep and recovery of elite athletes. The aims of this study were to:

1. Characterise the baseline sleep and recovery levels of elite athletes.
2. Assess the impact of Kiwifruit supplementation on the sleep and sleep health of elite athletes.
3. Reassess the sleep and recovery levels of elite athletes after the 4 week intervention

6.2 Methodology

6.2.1 Design

This study was an open label trial of the impact of Kiwifruit supplementation on sleep and recovery in athletes, and as such the purpose of the trail was not withheld from particpiants (Sedgwick, 2014). The participants were told that the purpose of the research was to investigate the impact of kiwi ingestion on athlete recovery.

6.2.2 Participants

A group of elite athletes (n=15) from a national sailing squad (n = 9; 7 males and 2 females) and a national athletics squad (n = 6; 2 males and 4 females; see table 6.1) were recruited through the National Sports Institute. Athletes were regarded as elite in line with published definitions i.e. members of a national team (Swann et al., 2015). No participants reported using sleep medication at baseline or post-intervention.

6.2.3 Procedure

All procedures were approved by the research ethics committee of the Faculty of Health and Life Sciences, Northumbria University. After reading the participant information sheet (see Appendix 9.5), all participants provided written informed consent prior to data collection. The participants were the provided with a link to the baseline and post-intervention questionnaire battery and the daily sleep diary. Participants were instructed to complete the baseline questionnaire battery, commence completion of the daily sleep dairy for the duration of the study (5 weeks; 1 control week and 4 intervention weeks) and complete the post-intervention questionnaire battery upon completion of the study i.e. after week 5 (see Figure 6.1).

6.2.4 Measures

All participants completed demographic data before completing the baseline and post-intervention questionnaires. Participants recorded their sex, age, body mass (kg), height (cm), sport, athlete type (elite or sub-elite), phase of season (pre-season, competition or off-season), normal training time (before 8 am, 8 am to 5 pm and after 5 pm) and training/competition duration per week (mins) (see Table 6.1).

6.2.5 The Recovery Stress Questionnaire for Athletes (RESTQ Sport)

The RESTQ-Sport is explained in detail in Chapter 4. High scores on stress scales indicate a high level of stress, while high scores on the recovery scales indicate a high level of recovery (Kallus and Kellmann, 2001). Each item is scored on a Likert scale (0 = Never to 6 = Always) based on how often the respondent engaged in a specified activity over the

previous three days/nights. With a response of 0 indicating never having experienced the feeling and 6 indicating always experiencing the associated feeling. High scores on stress scales indicate a high level of stress, while high scores on the recovery scales indicate a high level of recovery (Kallus and Kellmann, 2001).

6.2.6 Pittsburgh Sleep Quality Index (PSQI)

As discussed in detail in Chapter 4, the PSQI is a self-report measure of sleep quality, consisting of 19 items grouped into seven component scores which are equally weighted (Buysse et al, 1989). Overall global scores (GPSQI) were calculated by summing the seven components (range 0 – 21: with higher scores indicating poorer sleep quality) and the component scores were also calculated to provide subscale ratings of: (i.) subjective sleep quality, (ii.) sleep latency, (iii.) sleep duration, (iv.) sleep efficiency, (v.) sleep disturbances, (vi.). use of sleep medication and (vii.) daytime dysfunction (Hinz et al., 2017). As athletes often strive for marginal gains in their performance which can be facilitated through optimised sleep, the identification of both 'poor' and 'moderate' sleep quality is warranted (Venter, 2014), hence the standard cut-off (≥ 5) was employed for GPSQI.

6.2.7 Consensus Sleep Dairy – Core (CSD-C)

The CSD-C a standardised sleep diary is explained in detail in Chapter 4. The data collected was used to compute indices of sleep continuity such as sleep onset latency (SOL), number of awakenings (NoA), wake after sleep onset (WASO), time in bed (TIB), total sleep time (TST), and sleep efficiency (SE) (Maich et al., 2016) (See Table 6.4). Additional Likert scales were used to report fatigue both before going to bed and on getting up in the morning (1 = Completely Exhausted to 8= Fully Alert) and sleep quality (1 = Very Poor to 5= Verry Good). There was also a question relating to adherence where the participants recorded if they consumed Kiwifruit or not each day.

6.2.8 The Regulatory, Satisfaction, Alertness, Timing, Efficiency and Duration (RU SATED) Questionnaire

The RU SATED was developed to assess Sleep Health (Buysse et al., 2014). Sleep health is identified through regulation, satisfaction, appropriate timing, adequate duration, high efficiency, and sustained alertness during waking hours (Buysse, 2014). Sleep health is a concept which involves a holistic view of sleep as opposed to individual symptoms and disorders (Hale et al., 2020). The RU-SATED assesses six dimensions of sleep health:

1. Regulation: consistent sleep wake schedule (within 1 hour)
2. Satisfaction/quality: subjective assessment of 'good, or 'poor' sleep

3. Alertness/sleepiness: ability to maintain waking
4. Timing: placement of sleep within the 24-hour
5. Efficiency: ease of falling asleep and returning to sleep
6. Duration: total amount of sleep per 24 hours (Buysse, 2014).

Each dimension is scored on a 3-point likert scale from 0 (rarely/never) to 2 (usually/always), the scores from each dimension can be converted to a total score (0-12) with higher scores indicative of good sleep health (Ravyts et al., 2021).

6.2.9 Kiwifruit Intervention

Following the baseline assessment (Week 1) all subjects began the intervention (Weeks 2-5). During the 4-week intervention participants were asked to consume 2 medium-sized Green Kiwifruit (*Actinidia Deliciosa*) an hour before bed (See Figure 6.1). The dose was based on doses employed in previous studies (2 x Kiwi [Lin et al., 2011]) and (130g [Nødtvedt et al., 2017]) and the timing was proposed to coincide with the melatonin secretion. The participants reported adherence to the intervention when completing the daily questionnaire. As the research was conducted under 'lockdown' conditions during the Covid-19 pandemic participants were instructed to purchase the Kiwfruit themselves and were reimbursed upon completion of the study.

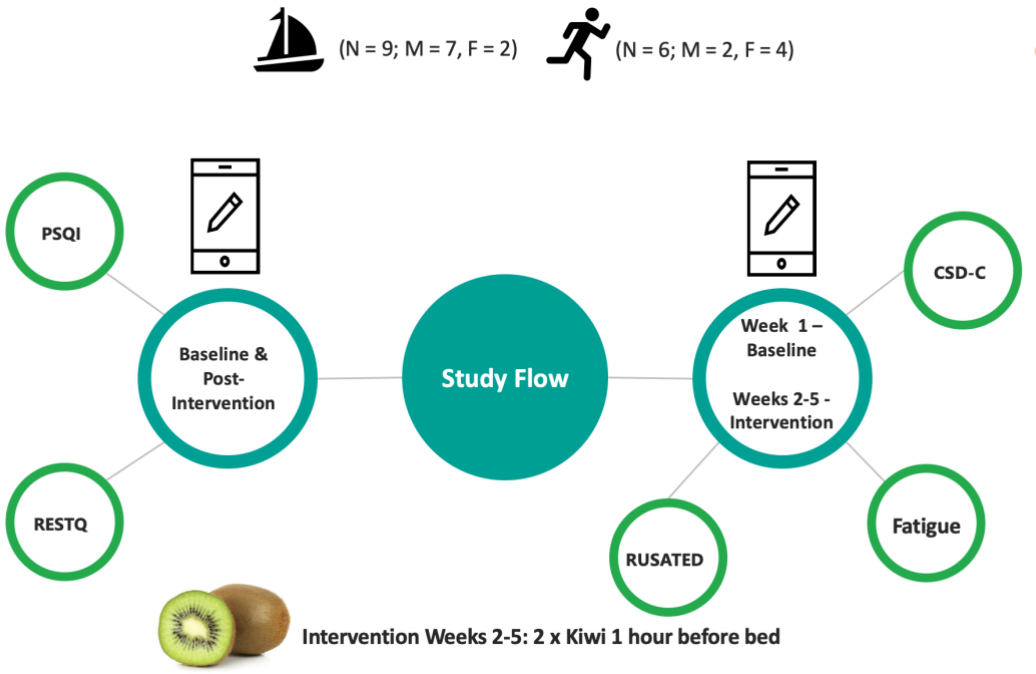

Figure 6.1: Study flowchart.

6.3 Data Analysis

All data was analysed using the Statistical Package for the Social Sciences (SPSS Version 26, IBM Corporation) and Jamovi (Version 1.6, The Jamovi Project). Frequency distribution and descriptive statistics were used to present findings (Thomas et al., 2015). All data were presented in mean ± standard deviation, and/or frequency. Shapiro-Wilks tests were used to assess the distribution of data. Paired samples t-test and Wilcoxon signed rank tests were used to examine the changes in scores from baseline to post-intervention. Repated measures ANOVA and Friedmans test were used to assess the difference in scores from baseline and week by week during the intervention. For variables that demonstrateed significant differences, pairwise comparisons were performed to identify each timepoint where significant differences occurred compared to baseline.

6.4 Results

In total 15 elite athletes took part from a national sailing squad (n = 9; male n = 7 and female n= 2) and a national athletics squad (n = 6; male n = 2 and female n= 4). An independent samples t-test highlighted significant differences between the groups at baseline for body mass ($t=-4.931$; $p < 0.001$), height ($t=-2.338$; $p < 0.05$) and training/competition duration per week ($t=-3.066$; $p < 0.01$), which is indicative of the different characteristics of Sailing and Athletics. However, a Wilcoxon signed rank test revealed no statistically significant differences from baseline to post-intervention for body mass, normal training time, training duration and phase of season ($p > 0.05$) (see Table 6.1).

Table 6.1: Characteristics of participants.

	All	Sailing	Athletics
Gender	15 (M = 9 / F = 6)	9 (M = 7 / F = 2)	6 (M = 2 / F = 4)
Age (Y)	23.2 ± 3.9	24.56 ± 4	21.17 ± 2.93
Body mass (kg)	70.39 ± 13.34	78.89 ± 7.42***	57.65 ± 9.25
Height (cm)	175.37 ± 8.99	179.22 ± 7.31*	169.58 ± 8.58
Phase of season	Pre-season n = 10	Pre-season n = 4	Pre-season n = 6
	Competition n = 2	Competition n = 1	Competition n = 1
	Off-season n = 3	Off-season n = 1	Off-season n = 2
Normal training time	8am -5pm n = 14	8am to 5pm n = 9	8am to 5pm n = 5
	After 5pm n = 1		After 5pm n = 1
Training/competition duration per week (mins)	912 ± 359.19	636.67 ± 237.8**	1095.56 ± 309.32

Statistically significant ***p ≤ 0.001; **p ≤ 0.01; *p<0.05.

6.5 Baseline vs. Post-Intervention (PSQI and RESTQ)

In order to investigate changes in sleep quality and recovery/stress balance over the duration of the study, participants completed the PSQI and RESTQ at baseline and post-intervention. There were no significant gender or sport effects for any measures in the current study.

6.5.1 Sleep Quality

A Wilcoxon signed rank test was used to compare the PSQI component scores from baeline to post-intrvention (see Table 6.2). Sleep quality improved significantly from baseline (1.53 ± 0.84) to post-intervention (0.27 ± 0.46; z=78, p = 0.002) (see Table 6.2 and Figure 6.2). PSQI global scores reduced significantly from baseline (6.47 ± 2.17) to post-intervention (4.13 ± 1.19; z=91, p = 0.002) (see Table 6.2 and Figure 6.3), this was also clinically relevant indicating a significant reduction in sleep problems among the athletes. While there were improvements from baseline to post-intervention in sleep onset latency, sleep duration and sleep efficiency, no significant differences were observed (p > 0.05). Similarly, daytime dysfuntion component scores did not change from baseline and post-intervention.

Table 6.2: Comparison of global PSQI score and component scores (mean ± SD) baseline vs. post-intervention.

	Baseline	Post-Intervention	Mean Difference	95% CI	Effect size	p-value
Sleep Quality	1.53 ± 0.84	0.27 ± 0.46	1.27	1-2	1	0.002**
Sleep Latency	1.67 ± 0.49	1.33 ± 0.62	1	-1.79-1	0.46	0.18
Sleep duration	0.34 ± 0.49	0.14 ± 0.35	1	-0.02-0.43	1	0.15
Sleep Efficiency	0.6 ± 0.91	0.2 ± 0.42	1.5	1-2	1	0.09
Sleep Disturbance	1.2 ± 0.56	1.2 ± 0.56	0	0	0	1
Medication	0	0	0	0	0	0
Daytime Dysfunction	1.13 ± 0.74	1 ± 0.54	4.31	-1.98-1	0.33	0.48
PSQI Global Score	6.47 ± 2.17	4.13 ± 1.19	2.5	1.5-3.5	1	0.002**

Statistically significant **p ≤ 0.01

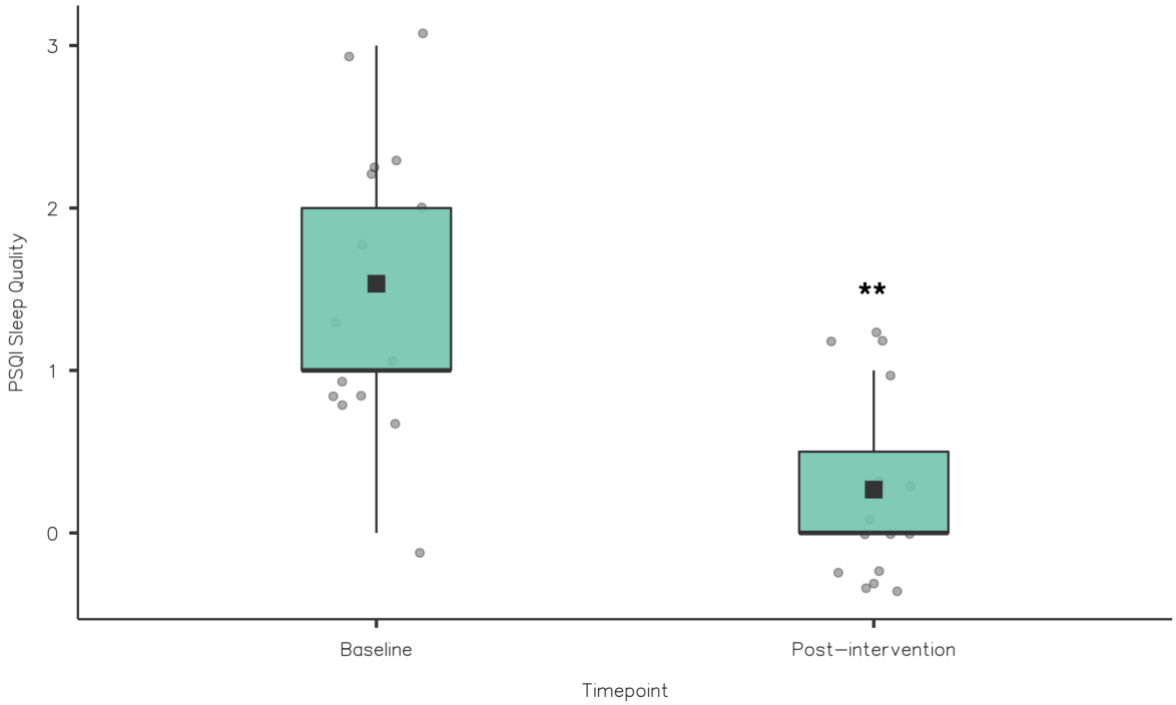

Figure 6.2: PSQI Sleep Quality Baseline Vs. Post-Intervention (p≤0.01).**

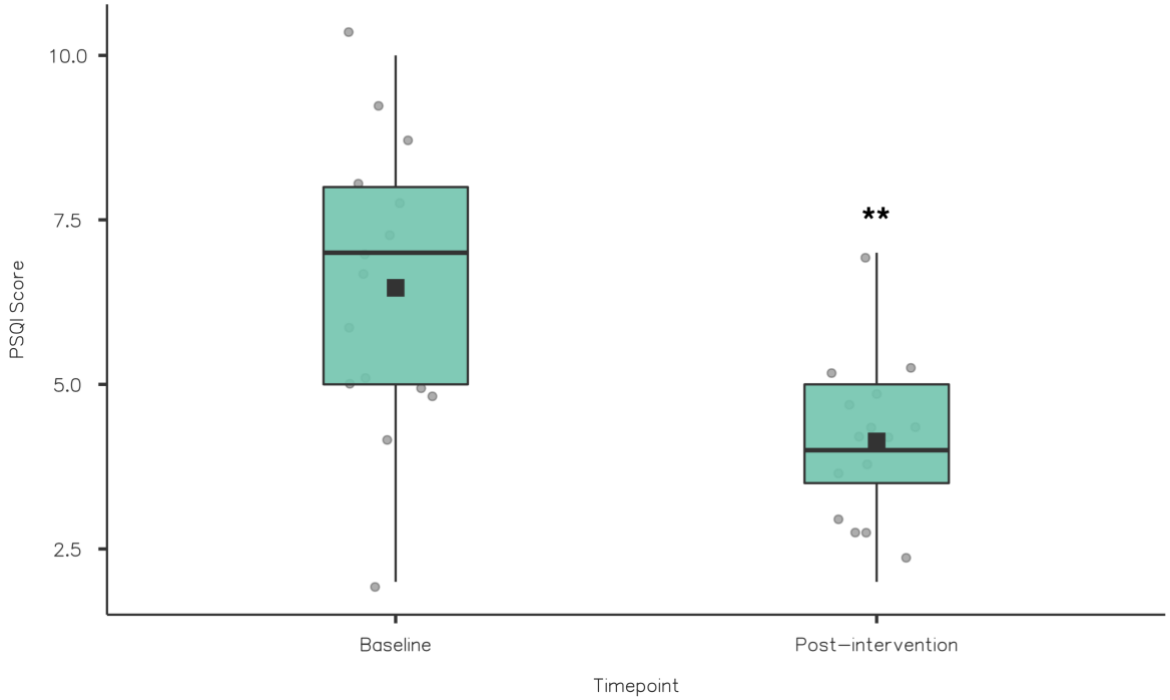

Figure 6.3: PSQI Global scores baseline vs. post-intervention (p≤0.01).**

6.5.2 Recovery

Wilcoxon signed rank tests were used to compare the baseline and post-intervention RESTQ scores. There were statistically significant improvements from baseline to post-intervention for the RESTQ scales general stress (3 ± 0.86 vs 2.58 ± 0.58; z=2.77,p=0.015)

and sport stress (2.72 ± 0.65 vs. 2.39 ± 0.63; z =2.85, p=0.019), conversely while both increased there were no statistically significant differences between general recovery (3.83 ± 0.79 vs. 4.11 ± 0.84; z=-2.09 , p=0.71), and sport recovery (4.03 ± 1.06 vs. 4.09 ± 0.92; z=-0.4 ,p=0.65).

Table 6.3: Recovery Stress (Mean ± SD) Subscales Baseline vs Post-Intervention.

	Baseline	Post-Intervention	Mean difference	95% CI	Effect size	p-value
General Stress	2.83 ± 1.54	2.37 ± 1.01	0.5	-0.13-1.06	0.48	0.16
Emotional Stress	2.9 ± 0.95	2.47 ± 0.61	0.5	-2.71-1.25	0.44	0.17
Social Stress	3.03 ± 1.03	2.57 ± 0.84	0.75	-0.5-1.75	0.64	0.95
Conflicts/Pressure	3.43 ± 1.31	3.17 ± 0.92	0.75	-0.5-1.75	0.5	0.3
Fatigue	3.4 ± 1.42	2.8 ± 1.21	1	0.5-1.5	0.84	0.02*****
Lack of Energy	3.07 ± 1.18	2.87 ± 1.09	0.5	-4.26-1	0.56	0.15
Physical Complaints	2.37 ± 0.92	1.8 ± 0.6	0.75	0.5-1.25	1	0.005******
Success	3.47 ± 0.9	3.73 ± 0.93	-0.5	-1.5-0.5	-0.43	0.4
Social Recovery	4.13 ± 1.1	4.43 ± 1.1	-0.68	-1.25-0.5	-0.61	0.14
Physical Recovery	3.53 ± 1.19	3.83 ± 1.18	-0.5	-1-0.25	-0.5	0.17
General Wellbeing	4.47 ± 1.27	4.5 ± 0.95	-2.7	-1.5-1.25	-0.02	1
Sleep Quality	3.57 ± 1.22	4.07 ± 1.35	-1	-1.75-0.5	-0.64	0.1
Disturbed Breaks	2.38 ± 0.8	2.03 ± 0.75	0.5	5.9-0.88	0.73	0.04*****
Emotional Exhaustion	2.7 ± 1.14	2.33 ± 0.98	0.5	-2.3-1	0.67	0.051
Injury	3.08 ± 0.68	2.8 ± 0.94	0.38	-0.13-0.75	0.42	0.2
Being in Shape	4.23 ± 1.25	4.4 ± 1.1	-0.25	-1-0.5	-0.37	0.33
Personal Accomplishment	3.58 ± 1.08	3.7 ± 1.13	-0.16	-0.5-0.38	-0.35	0.3
Self-Efficacy	3.95 ± 1.27	3.93 ± 1.09	6.01	-0.75-0.75	0.03	0.96
Self-Regulation	4.33 ± 1.15	4.32 ± 0.84	0.13	-0.75-0.88	0.13	0.77

Statistically significant **p ≤ 0.01; *p<0.05

A Wilcoxon signed rank test was also used to compare the 19 RESTQ sub scale items from baseline to post-intervention (see Table 6.3). There were statistically significant reductions in fatigue (3.4 ± 1.42 vs. 2.8 ± 1.21; z=50.5, p=0.02), physical complaints (2.37 ± 0.92 vs. 1.8 ± 0.6; z=55, p=0.005) and disturbed breaks (2.38 ± 0.8 vs. 2.03 ± 0.75; z=57, p=0.036) (see Figures 6.4 6.5 And 6.6).

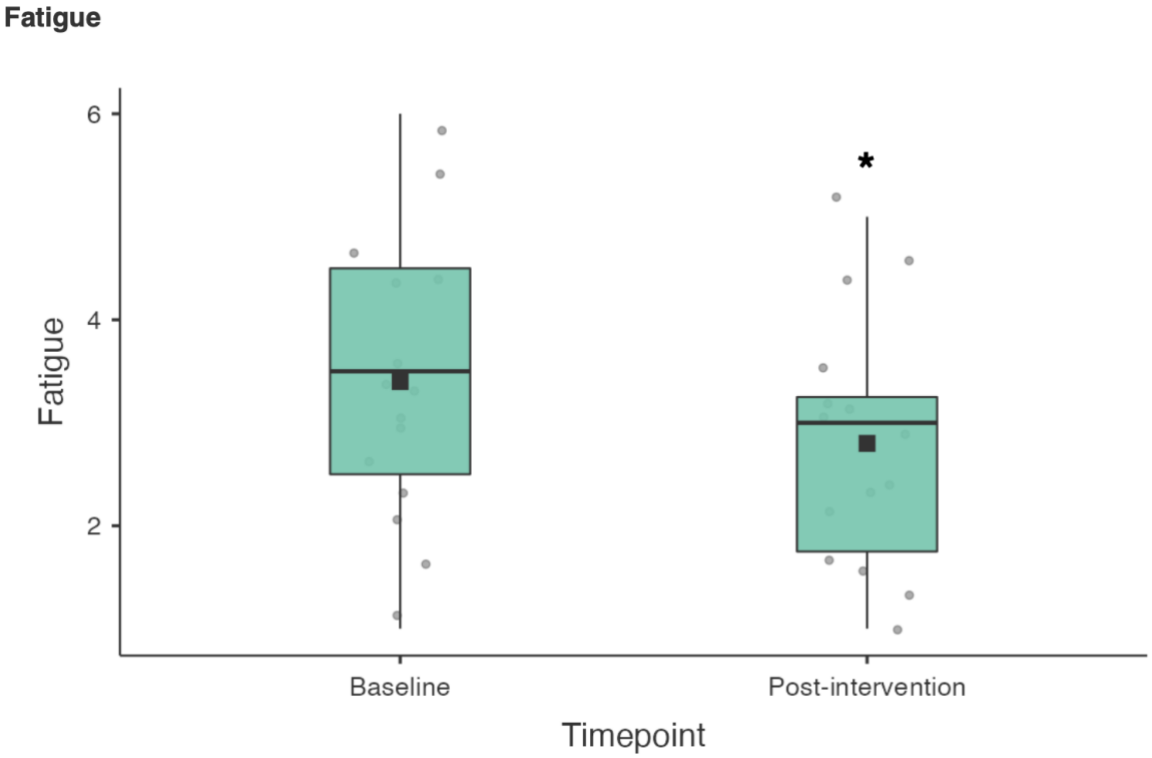

Figure 6.4: RESTQ Fatigue scores baseline vs. post-intervention (*p<0.05).

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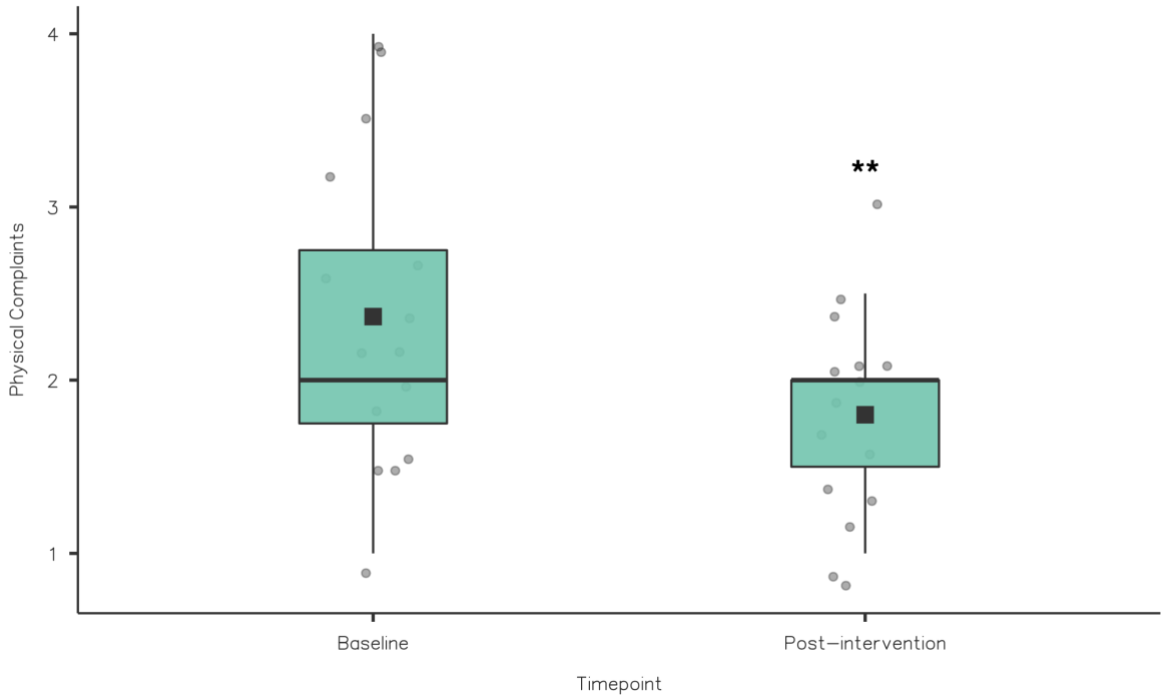

Figure 6.5: RESTQ Physical Complaints scores baseline vs. post-intervention (p≤0.01).**

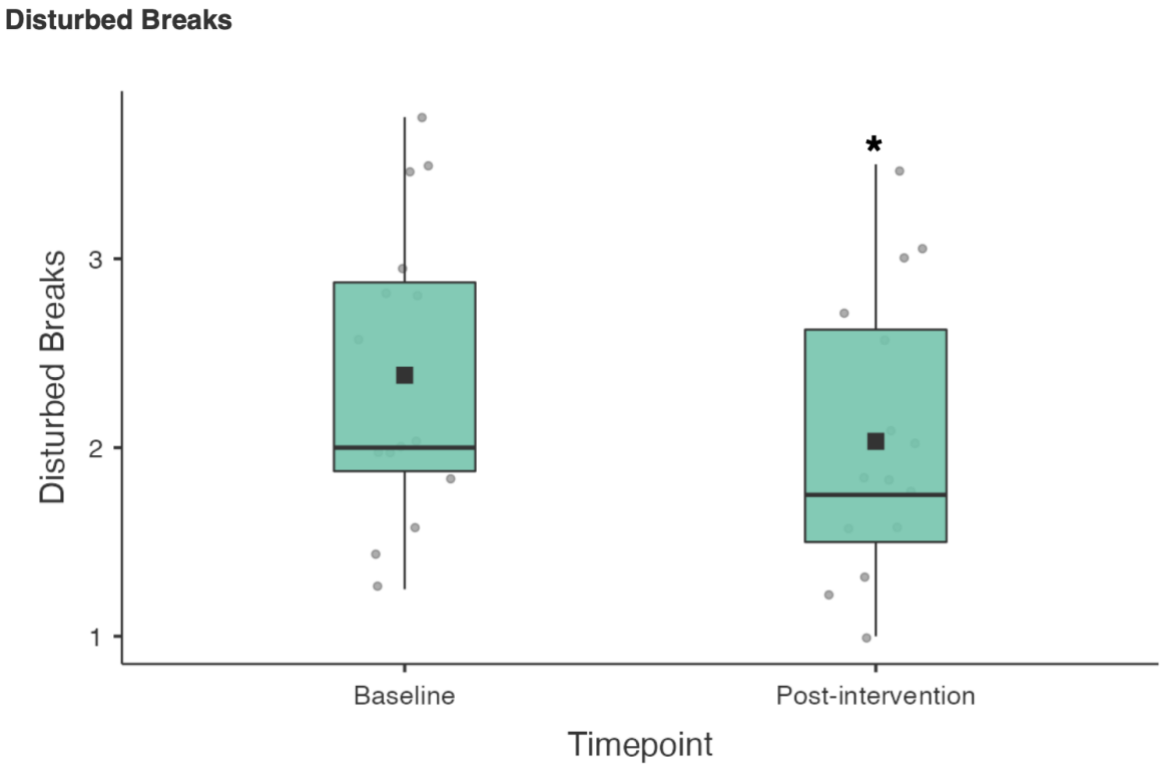

Figure 6.6: RESTQ Disturbed Breaks scores baseline vs. post-intervention (*p<0.05).

6.6 Intervention (Weeks 2-5)

Motivating, enabling and supporting athletes is key to behaviour change, increasing athlete self-awareness and promoting long-term nutritional changes (Logue et al., 2021).

Adherence (90 ± 6.64%; range 82.14 - 100%) to the Kiwifruit intervention was high for all participants. It has previously been suggested that athlete adherence to nutritional guidance is seasonal (i.e. higher adherence in-season vs. off-season) (Bentley et al., 2021), the majority of athletes in this study (n=10) were in pre-season which may have improved adherence. In order to investigate changes in sleep quality and sleep health particpants completed the CSD-C and RU-SATED daily for the duration of the study.

6.6.1 Sleep Diary

The daily sleep diary data was averaged and analysed on a week by week basis (see Table 6.4). A repeated measures ANOVA and Friedmans test were used to assess the difference between the baseline and intervention weeks for the sleep diary data. The normally distributed variables (SOL, TIB and TST) were analysed using a repeated measures ANOVA, while Friedmans test was used to analyse the non-normally distributed variables (Awakenings, WASO, SE, Fatigue and SQ). Where there were significant differences, pairwise comparisons were performed to assess if there were significant differences between variables at each timepoint during the intervention compared to baseline.

A repeated measures ANOVA demonstrated that although SOL reduced during the intervention compared to baseline there were no statistically significant differences ($F(1.81, 19.92) = 2.689$, $p > 0.05$). Conversely, TIB increased from baseline to intervention but the differences were not statistically significant ($F(1, 11) = 0.393$, $p > 0.05$). TST improved week on week from baseline to intervention ($F(4, 44) = 6.653$, $p = 0.001$ partial $\eta^2 = 0.38$). TST increased from baseline 7.6 ± 0.75 h to 8.55 ± 0.44 h at week 4, a statistically significant increase of 0.83 ± 0.23 [mean ± standard error], $p < 0.05$) (see Figure 6.7).

Table 6.4: Sleep diary comparison week by week (mean ± SD).

	Baseline	Week 2	Week 3	Week 4	Week 5
SOL (mins)	24.6 ± 15.9	18.8 ± 11	14.9 ± 9.51	16.1 ± 10.3	13.9 ± 8.4
Awakenings	1.22 ± 0.87	1.2 ± 0.98	0.89 ± 0.94**	0.95 ± 0.96*	0.98 ± 0.96
WASO (mins)	10.8 ± 10.2	7.8 ± 6.45	5.15 ± 6.12**	5.56 ± 4.43**	5.68 ± 5.19*
TIB (h)	8.84 ± 0.75	9.18 ± 0.5	9.02 ± 0.41	9.2 ± 0.54	9.24 ± 0.47
TST (h)	7.6 ± 0.75	8.4 ± 0.62	8.42 ± 0.34	8.55 ± 0.44*	8.63 ± 0.47
SE (%)	86.2 ± 5.31	91.5 ± 3.8*	93.4 ± 2.7***	93 ± 2.54***	93.3 ± 2.43***
Fatigue (Bed)	3.23 ± 0.78	3.1 ± 0.58	3.28 ± 0.57	3.24 ± 0.49	3.16 ± 0.69
Fatigue (Morning)	3.85 ± 1.15	3.67 ± 1.08	4.03 ± 1.31	4.1 ± 1.3	3.7 ± 1.24*
Sleep Quality	3.45 ± 0.74	3.48 ± 0.78	3.76 ± 0.7	3.76 ± 0.68	3.58 ± 0.83

Statistically significant differemnce (* p < 0.05; **p < 0.01)

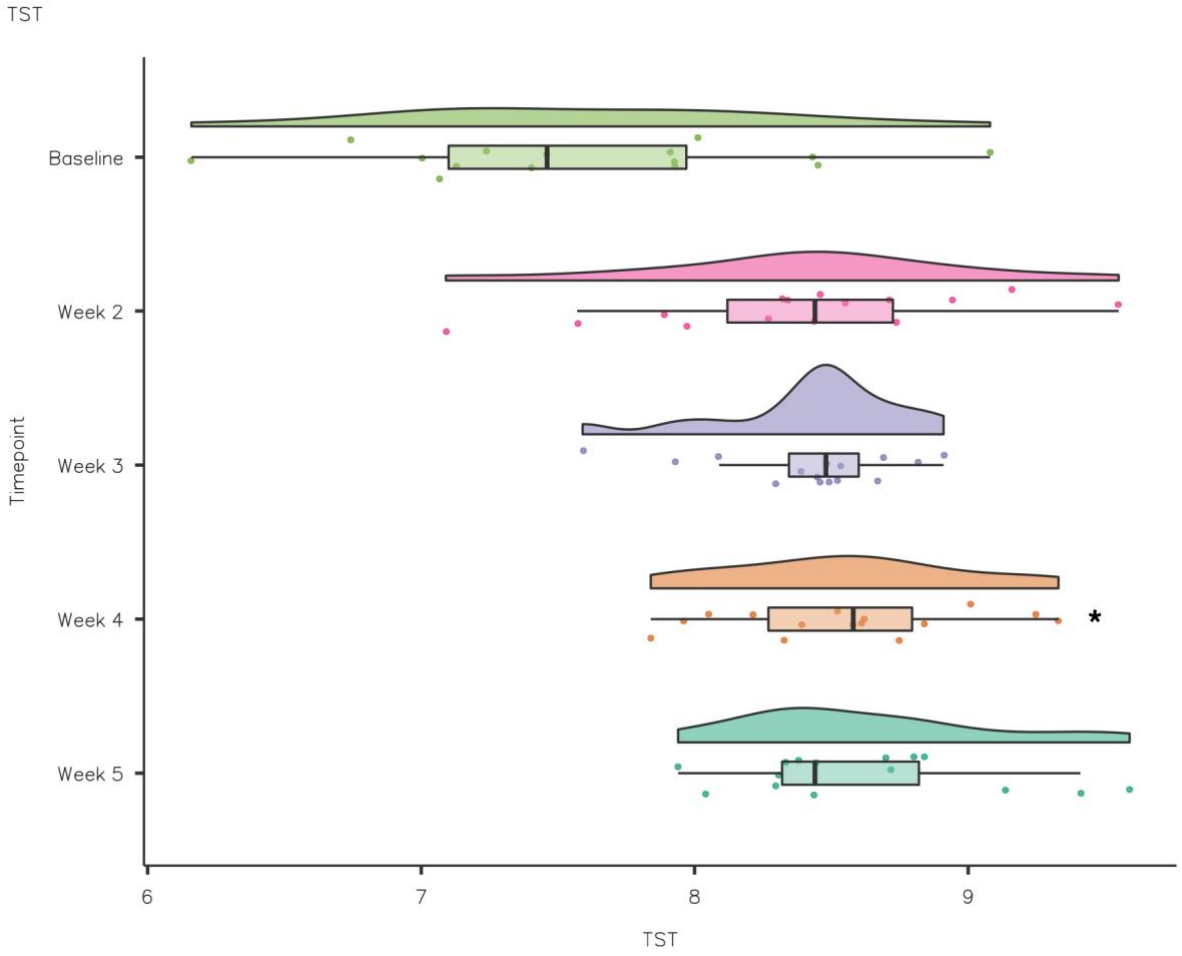

Figure 6.7: CSD-C Total Sleep Time data comparison week by week (*p<0.05).

A Friedmans test highlighted that Number of Awakenings reduced significantly from baseline to intervention $\chi^2(4) = 12.6$, $p<0.05$. Pairwise comparisons (Durbin-Conover) were performed to assess if there were significant differences between timepoints during the intervention compared to baseline. Pairwise comparisions deomonstrated that there was a statistically significant reduction in NoA compared to baseline in weeks 3 (p=0.003) and 4 (p=0.012) (see Figure 6.8).

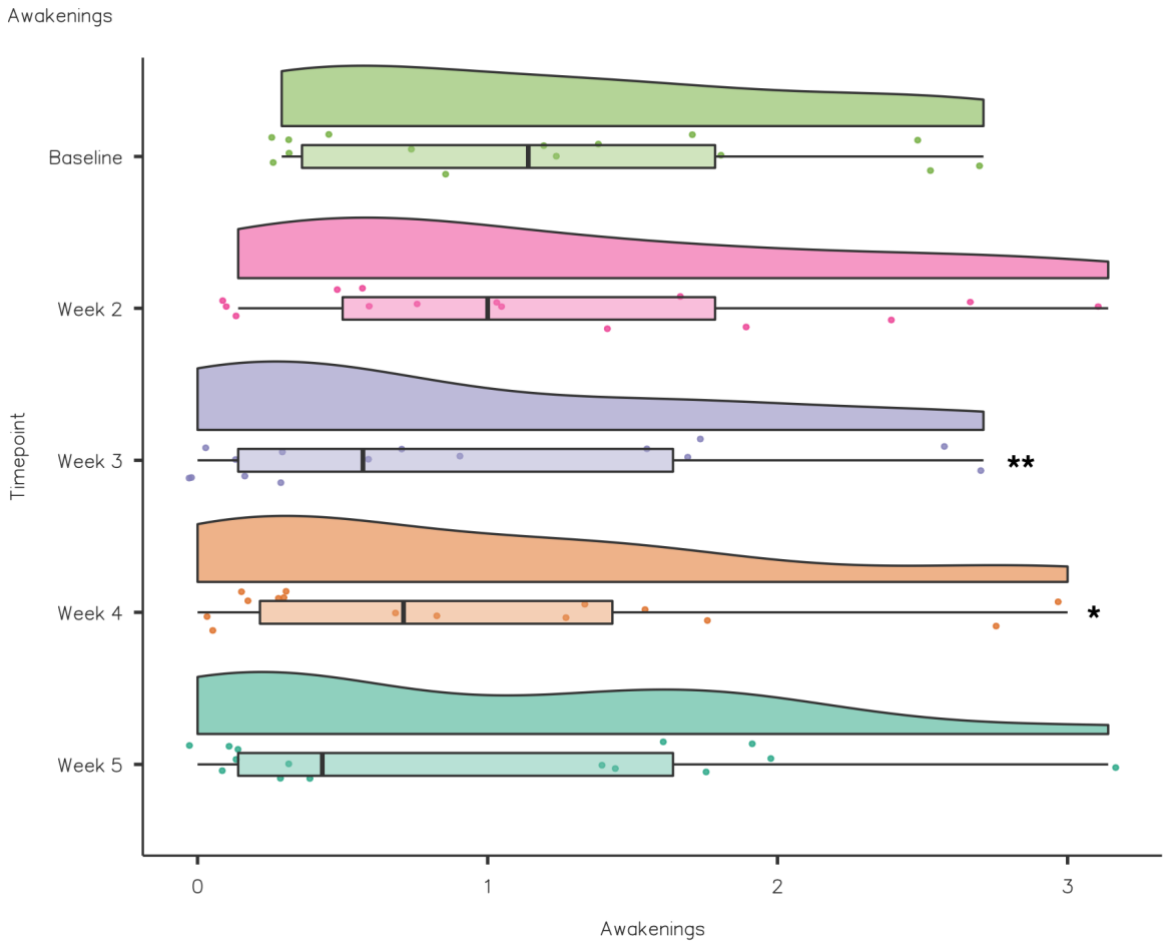

Figure 6.8: CSD-C Awakenings data comparison week by week (p≤0.01; *p<0.05).**

A Friedmans test highlighted that WASO reduced significantly from baseline to intervention $\chi^2(4) = 12.5$, $p<0.05$. Pairwise comparisions (Durbin-Conover) deomonstrated that there was a statistically significant reduction in WASO compared to baseline in week 3 (p=0.002), week 4 (p=0.003) and week 5 (p=0.014) (see Figure 6.9).

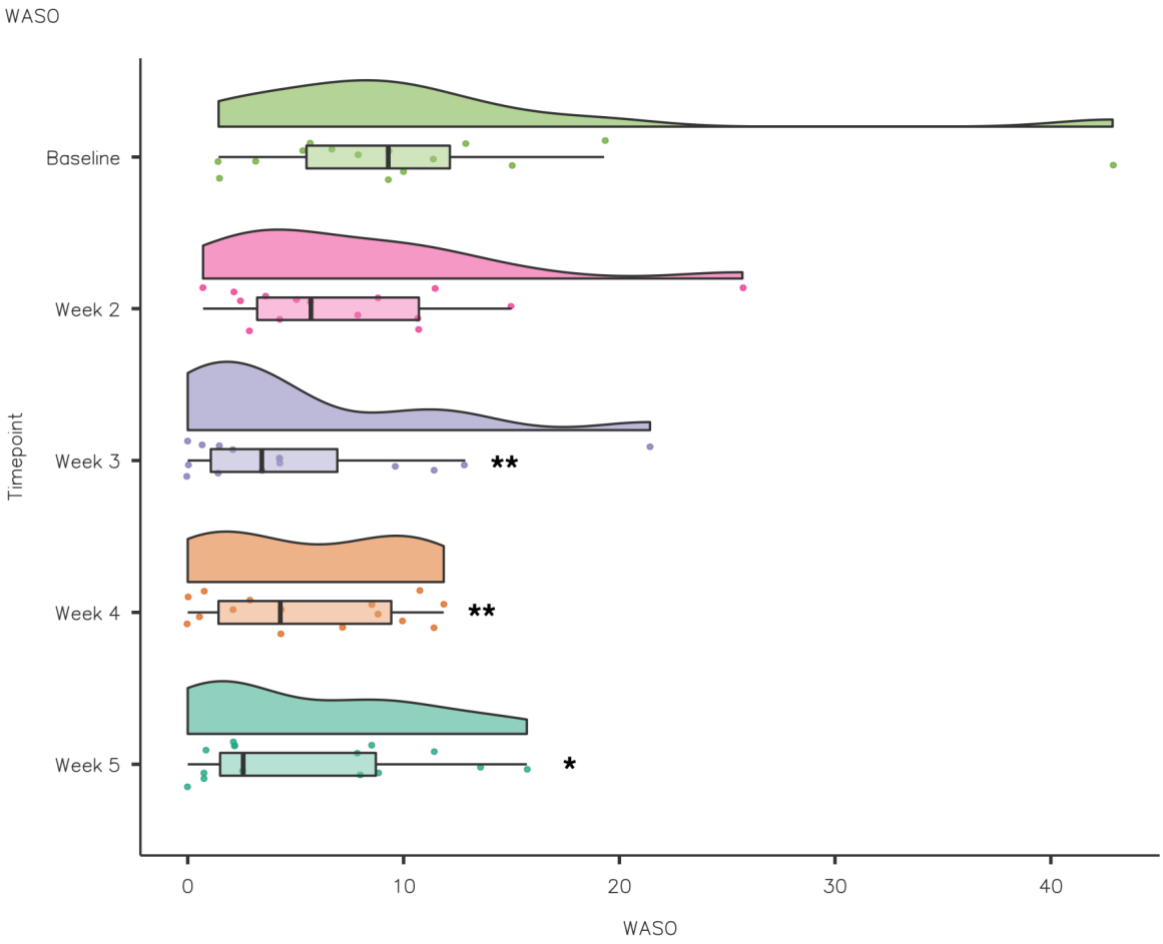

Figure 6.9: CSD-C WASO data comparison week by week (p≤0.01; *p<0.05).**

A Friedmans test showed that SE increased significantly from baseline to intervention $\chi^2(4) = 21.2$, $p \leq 0.001$. Pairwise comparisions (Durbin-Conover) deomonstrated that there was a statistically significant increase in SE compared to baseline in week 2 (p=0.018), week 3 (p < 0.001), week 4 (p < 0.001) and week 5 (p < 0.001) (see Figure 6.10). Self-report Fatigue Going to Bed did not differ significantly from baseline to intervention $\chi^2(4) = 3.05$, p = 0.55. While there was a significant difference in Fatigue in the Morning from baseline to intervention $\chi^2(4) = 15.6$, p=0.004. Pairwise comparisions (Durbin-Conover) deomonstrated that there was a statistically significant reduction in Fatigue in the Morning compared to baseline in week 5 (p=0.041) (see Figure 6.11).

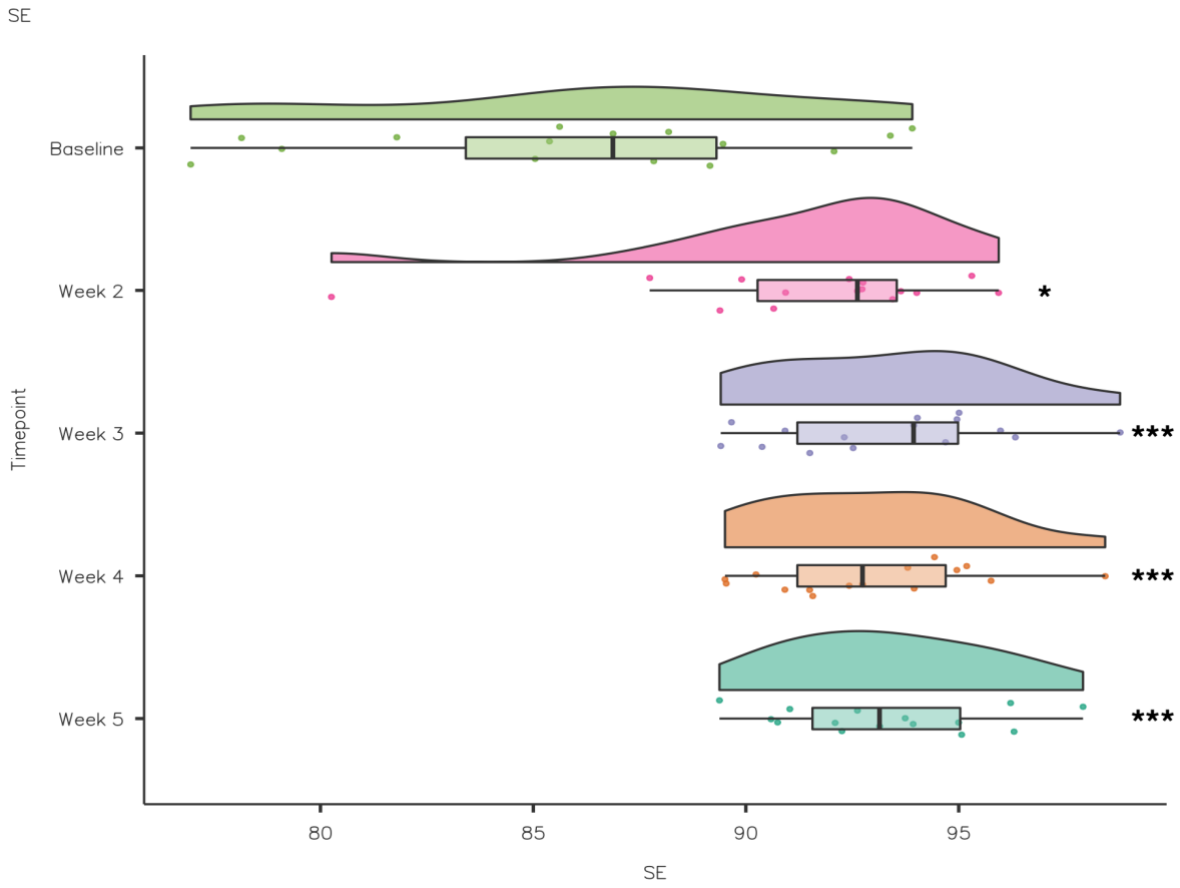

Figure 6.10: CSD-C Sleep Efficiency data comparison week by week ***p ≤ 0.001).

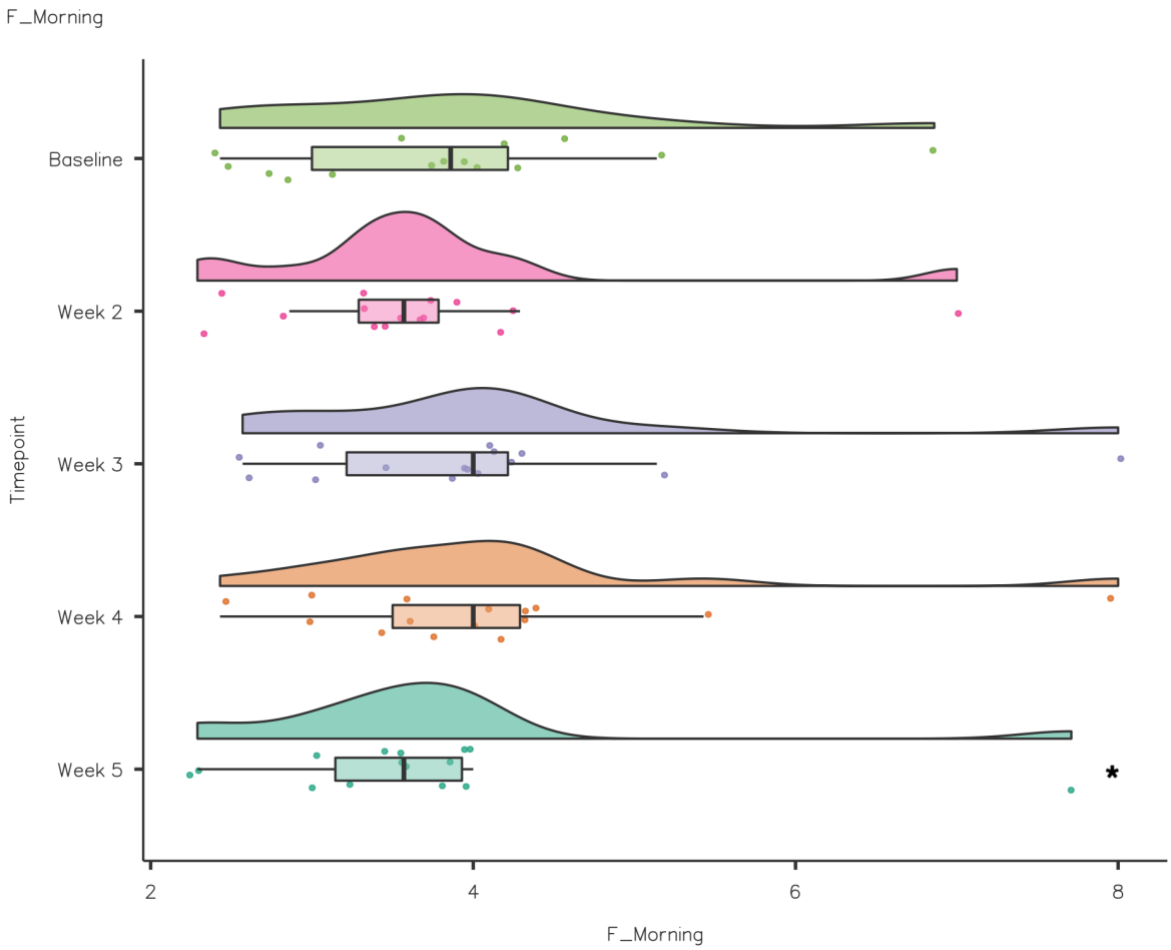

Figure 6.11: CSD-C Fatigue in the Morning data comparison week by week (*p<0.05).

While all Sleep Quality scores were higher during the intervention compared to baseline, the differences were not significant $\chi^2(4) = 8.62$, p=0.071.

6.6.2 Sleep Health

A repeated measures ANOVA demonstrated that although the Sleep Health scores increased during the intervention (weeks 3-5) compared to baseline there were no statistically significant differences ($F(4, 44) = 1.178$, $p > 0.05$). A Friedmans test were used to assess the difference between the basline and intervention weeks for the RU-SATED data (see Table 6.5).

Table 6.5: Sleep health comparison week by week (mean ± SD).

	Baseline	Week 2	Week 3	Week 4	Week 5
Regulation	2.6 ± 0.46	2.56 ± 0.47	2.58 ± 0.48	2.62 ± 0.46	2.59 ± 0.52
Satisfaction/Quality	2.33 ± 0.51	2.27 ± 0.61	2.25 ± 0.68	2.3 ± 0.62	2.31 ± 0.62
Alertness/Sleepiness	2.78 ± 0.36	2.83 ± 0.31	2.88 ± 0.28	2.9 ± 0.26	2.9 ± 0.27
Timing	2.98 ± 0.08	2.97 ± 0.08	2.99 ± 0.04	2.98 ± 0.07	2.99 ± 0.04
Efficiency	2.37 ± 0.66	2.43 ± 0.63	2.47 ± 0.65***	2.46 ± 0.65*	2.46 ± 0.62**
Duration	2.81 ± 0.36	2.75 ± 0.43	2.74 ± 0.43	2.74 ± 0.45	2.74 ± 0.44
Sleep Health Score	9.88 ± 1.63	9.81 ± 1.75	9.9 ± 1.65	9.97 ± 1.68	9.98 ± 1.7

Statistically significant ***$p \leq 0.001$; ** $p \leq 0.01$; *$p < 0.05$

A Friedmans test showed that Efficiency increased significantly from baseline to intervention $\chi^2(4) = 10.2$, $p \leq 0.036$. Pairwise comparisions (Durbin-Conover) deomonstrated that there was a statistically significant improvement in Efficiency compared to baseline in week 3 (p=0.005), week 4 (p=0.021) and week 5 (p=0.009) (see Figure 6.12). There were no significant differences between Regulation $\chi^2(4) = 8.62$, p=0.417 and Satisfaction/Quality $\chi^2(4) = 2.55$, p=0.637. While Alertness/Sleepiness scores improved week on week the changes were not significant $\chi^2(4) = 8.48$, p=0.075. There were no significant differences between Timing $\chi^2(4) = 1.00$, p=0.91, Duration $\chi^2(4) = 4.11$, p=0.392.

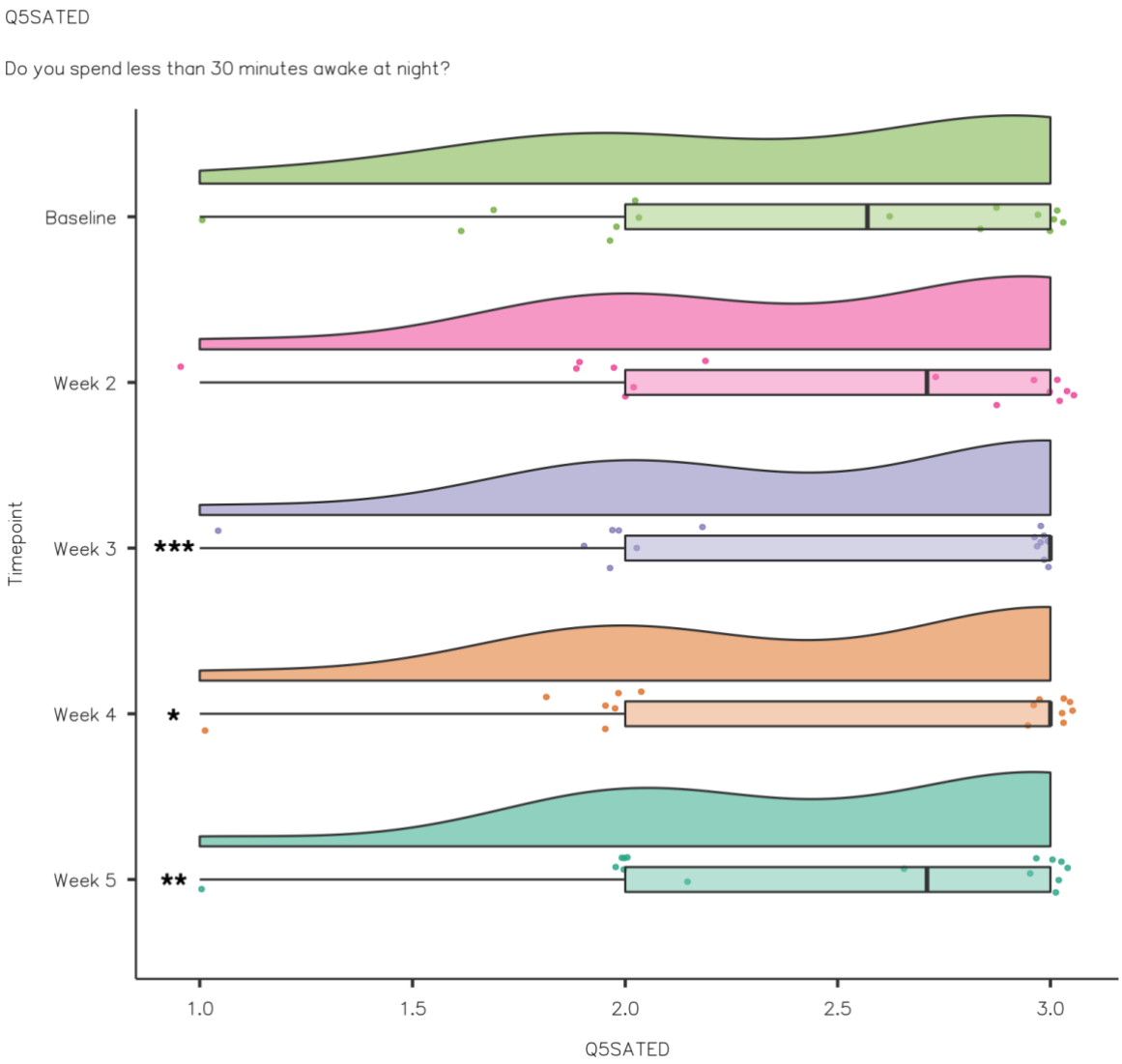

Figure 6.12: RU-SATED Efficiency data comparison week by week (*p ≤ 0.001; ** p≤0.01; *p<0.05).**

6.7 Discussion

The aims of the current study were to (i) assess the baseline sleep and recovery levels of elite athletes, (ii) assess the impact of Kiwifruit supplementation on the sleep of elite athletes and (iii) reassess the sleep and recovery levels of elite athletes after the intervention. This is the first study to assess the impact of Kiwifruit consumption on the sleep and recovery of elite athletes. To date very limited research exists investigating the potential sleep promoting properties of Kiwifruit and this study is the first to investigate the impact of Kiwifruit consumption on the sleep and recovery of elite athletes. As such, further research is necessary to develop practical guidelines for supplementation to enhance sleep and/or post exercise recovery.

6.7.1 Baseline vs Post-Intervention Measures

In the current study participants completed the PSQI and RESTQ at baseline and post-intervention to assess changes in sleep quality and recovery/stress balance. At baseline 87% (n=13) of athletes were classified as poor sleepers (global PSQI score ≥ 5), which was consistent with previous research in elite athletes (Knufinke et al., 2018, Swinbourne et al., 2016; Sargent et al., 2014; Samuels, 2008). A growing body of research has highlighted the prevalence of sleep problems in athletes including insomnia symptoms (Dunican et al., 2019; Gupta et al., 2017) and obstructive sleep apnea (OSA) (Swinbourne et al., 2016). In the current study there was a significant reduction in mean global PSQI scores (6.47 ± 2.17 to 4.13 ± 1.19) from baseline to post-intervention. Global PSQI scores improved significantly at post-intervention with fewer athletes (33%; n=5) being classified as poor sleepers, this change was clinically significant in that there were less sleep problems observed post-intervention.

Specifically, PSQI sleep quality improved significantly from baseline to post-intervention (1.53 ± 0.84 to 0.27 ± 0.46). Previous research using both subjective (Bender et al., 2019) and objective measures (Leeder et al., 2012) has suggested that elite athletes have inferior sleep quality compared to non-athletes. Poor sleep quality is of particular concern for elite athletes as it can result in a reduction in recovery and/or subsequent athletic performance (Juliff et al., 2015; Jarraya et al., 2013; Reyner and Horne 2013; Edwards and Waterhouse, 2009). While there were improvements from baseline to post-intervention in PSQI dimensions of sleep onset latency (1.67 ± 0.49 to 1.35 ± 0.62), sleep duration (0.34 ± 0.49 to 0.14 ± 0.35) and sleep efficiency (0.6 ± 0.91 to 0.2 ± 0.42) no significant differences were observed. Similarly, daytime dysfuntion component scores did not change from baseline and post-intervention indicating no impact on levels of daytime sleepiness from baseline to post-intervention.

No participants reported using sleep medication at baseline or post-intervention however, the small sample size of the current study (n=15) must be noted. This is in stark contrast to a recent investigation in Finland (n = 228) which demonstrated that 33.9% (n=76) used sleep medication (Penttilä et al., 2021). A report from the National Collegiate Athletic Association (NCAA) indicated sleep medication use accounted for 10.3% of miscellaneous substance use across all sports in American student athletes (Rexroat, 2014). The lack of sleep medication usage in the current study despite 87% (n=13) of the athletes reporting poor sleep at baseline, highlights the potential need for evidence based nutritional interventions and protocols (e.g. Kiwifruit) to promote sleep health in elite athletes.

In the current study, there were statistically significant improvements from baseline to post-intervention for the RESTQ scales general stress (3 ± 0.86 vs 2.58 ± 0.58) and sport stress (2.72 ± 0.65 vs. 2.39 ± 0.63). Compared to previous research in Rugby players (n =41), general stress (forwards 1.38 ± 0.62 and backs 1.57 ± 0.68) and sport stress (forwards 1.26 ± 0.51 and backs 1.67 ± 0.73) scores in the current study were higher (i.e. indicating more stress) at baseline and post-intervention, however it must be noted that this sample were student athletes and not necessarily competing at the same level as the participants in the current study (Grobbelaar et al., 2010).

In terms of the 19 RESTQ sub scale items from baseline to post-intervention, there were statistically significant reductions in fatigue (3.4 ± 1.42 vs. 2.8 ± 1.21), physical complaints (2.37 ± 0.92 vs. 1.8 ± 0.6) and disturbed breaks (2.38 ± 0.8 vs. 2.03 ± 0.75). Significant associations between the RESTQ subscales fatigue (OR 1.7) and disturbed breaks (OR 1.84) have been demonstrated in German professional soccer players (n=22) suggesting that injury risk increased due to insufficient rest periods and/or if players feel exhausted or overtrained (Laux et al., 2015). The REST-Q scores at baseline suggested that the athletes in the current study would benefit from an intervention aimed at promoting sleep and/or recovery. While the changes in the REST-Q scores from baseline to post-interventuon suggest a small but potentially meaningful change in athletes' recovery stress balance.

6.7.2 Over the course of the Intervention – Sleep Diary

To assess the impact of Kiwifruit consumption on sleep quality, sleep duration, fatigue and sleep health, for the duration of the intervention (1 baseline week and 4 intervention weeks) participants completed the CSD-C including additional questions relating to fatigue. It has recently been suggested that elite athletes are prone to sleep inadequacies charaterised by habituial short sleep duration (< 7 hours/night), unrefreshing sleep, long SOL, daytime sleepiness, daytime fatigue and poor sleep quality (Walsh et al., 2021). In the current study mean TST (hours) improved from baseline (7.6 ± 0.75) to week 2 (8.4 ± 0.62), week 3 (8.42 ± 0.34), week 4 (8.55 ± 0.44) and week 5 (8.63 ± 0.47), mean values week on week met current sleep guidelines (i.e. 7-9h) for adults. Overall, mean TST moved from inadequate during the baseline week to within the recommended 8-10hour range, for athletes, during the intervention. However, it must be noted that the athletes self-reported their sleep behaviours using a sleep diary, which can be affected by recall bias e.g. overestimation of sleep duration and efficiency (Walsh et al., 2021). Shorter sleep durations can directly impact athletic performance through negative effects on heart rate, breathing

rate and lactate concentrations (Mougin et al., 1996) or indirectly through an impact on mood, motivation or rate of perceived exertion (RPE) (Roberts et al., 2019; Martin, 1981). The amount of sleep an individual habitually obtains has implications for their ability to function effectively (Sargent et al., 2021) hence improvements in sleep duration as seen in the current study could positively impact health and performance.

A recent study which assessed sleep, using actigraphy, in a large elite athlete sample (n = 175) demonstrated that habitual sleep duration was 6.7 ± 0.8 hours while self-identifed sleep need was 8.3 ± 0.9 hours and suggested that individual athletes get less sleep that team sport athletes (Sargent et al., 2021). It is possible that some athletes require < 7-9 hours sleep while others require more (Sargent et al., 2021). More research is necessary to gain an understanding of the sleep needs of athletes, how often athletes actually achieve these sleep needs and possible interventions (e.g. nutrition) than can positively impact athlete sleep.

Previous research has suggested that there are no differences between the sexes for habitual sleep duration (Sargent et al., 2021; Leeder et al., 2012), however it must be noted that female athletes tend to be under-represented in research investigation the sleep and recovery of athletes; and these comparisons were not sport specific. While differences in male and female physiology and biochemistry have been established e.g. males typically have greater muscle mass and less adipose tissue which contributes to greater strength, aerobic and anaerobic power compared to females (McGuigan et al., 2021; Sandbakk et al., 2018). The impact of alterations in female sex hormone concentrations during the menstrual cycle on sleep and recovery of elite athletes warrants further investigation. In the current study > 50% (n=8) of the sample were female athletes. In the current study there were no significant gender or sport effects on measures of sleep duration or quality. Further research is, however, warranted that focuses on gender differences within sports as research in athletes tends to focus on comparisons among athlete groups rather than comparison within specific sports.

It has recently been suggested sleep fragmentation is a contributing factor to poor sleep quality in athletes (Walsh et al., 2021). It is estimated that athletes need 8.3 ± 0.9 hours sleep (Sargent et al., 2021), the increase in TIB during the intervention would increase the likelihood of an athlete achieving their sleep need. Awakenings reduced from baseline (1.22 ± 0.87) to intervention with significant reduction in week 3 (0.89 ± 0.94) and week 4 (0.95 ± 0.96). WASO also reduced from baseline and significantly in week 3 (10.8 ± 10.2), week 4 (5.56 ± 4.43) and week 5 (5.68 ± 5.19).

Good sleep quality is recognised as a predictor of physical health, mental health and wellness, while poor sleep quality can lead to fatigue, drowsiness and changes in mood (Chennaoui et al., 2015). Although SE is a good starting point in terms of sleep improvement, athletes also need to focus on sleep quality (de Moura Simim et al., 2020). Sleep quality can be difficult to assess, especially in athletes (de Moura Simim et al., 2020), however, it has been recommended that sleep efficiency should be used to monitor sleep quality using actigraphy in athletes (Claudino et al., 2019). Previous research has highlighted that athletes' sleep quality as measured by SE (%) was lower (3%-4%) the night before competition (Roberts et al., 2019). Differences have also been observed in the sleep characteristics of team sport and individual athletes whereby individual athletes had poorer sleep efficiency than team sport athletes (Lastella et al., 2015). In the current study SE (%) increased significantly from baseline (86.2 ± 5.31) to week 2 (91.5 ± 3.8), week 3 (93.4 ± 2.7), week 4 (93 ± 2.54) and week 5 (93.3 ± 2.43) which is reflective of the increased TST and/or reductions in WASO and SOL. SE < 85% is considered poor (Ohayon et al., 2017), in the current study baseline SE (%) scores straddled and for a minority of athletes (n=4) were below the threshold of 85%, indicating insomnia symptoms (Sánchez-Ortuño et al., 2010). The improvement in SE (%) observed from throughout the intervention resulted in less insomnia symptomology among the sample but could also impact performance. Insufficent sleep has been negatively associated to physical performance (speed and anaerobic power), neurocognitive function (attention and memory) and physical health (illness and injury risk) (Simpson et al., 2017; Chennaoui et al., 2015; Fullagar et al., 2015). The scores from the current study are similar to previous research which has demonstrated that habitual sleep efficiency of elite athletes was 88.47% ± 5.45% (Knufinke et al., 2018), 80.6% ± 6.4% (Leeder et al., 2012), 86.3% ± 6.1% (Lastella et al., 2015) and 79% ± 9.2% (Shearer et al., 2015). A recent systematic review reported the pooled average sleep efficiency for athletes (86% ± 5%; range 79% - 96%) (Gupta et. al., 2017).

Self-report Fatigue Going to Bed did not differ significantly from baseline to intervention however, there was a significant reduction in Fatigue in the Morning from baseline to week 5, which coincided with the improvements reported in the sleep diaries. The improvement in Fatigue in the Morning is beneficial as sleep problems in athletes have been noted previously, a recent systematic review demonstrated the prevalence of insomnia symptomology (i.e. increased SOL, greater sleep fragmentation, non-restorative sleep and excessice daytime fatigue) (Gupta et al., 2017). While no significant difference was

observed, Sleep Quality scores improved during the intervention compared to baseline. These improvements in sleep duration, and quality, highlight Kiwifruit as a potential athlete-friendly intervention that could promote improve sleep and recovery.

6.7.3 Over the course of the Intervention - Sleep Health

The RU-SATED has demonstrated adequate internal consistency (Cronbach's $\alpha = 0.64$) (Ravyts et al., 2021), most likely due to the low number of items (6), as the size of alpha depends on the number of items in a scale (Streiner, 2003). However, mean inter-item correlations ($r = 0.29\text{-}0.5$) were moderate (Ravyts et al., 2021) and it has previously been suggested that inter-item correlations should fall between 0.15-0.5 (Clark and Watson, 1995). The RU-SATED is a valid instrument for the assessment of sleep health in adults that is related to but distinct from other sleep constructs (Ravyts et al., 2021).

To assess the impact of Kiwifruit consumption on sleep quality, sleep duration, fatigue and sleep health, for the duration of the intervention (1 baseline week and 4 intervention weeks) participants completed the RU-SATED. Poor sleep health can impair physical health, recently it was demonstrated that students with poor sleep health were more likely to have poor physical health (Benham, 2019). Sleep health in athletes is characterised by good sleep quality, minimal daytime dysfunction, strategic napping, if necessary, and good sleep hygiene (Biggins et al., 2020). In the current study the particpiants reported relatively good sleep health scores at baseline, as a result there were no significant differences from baseline to intervention for Regulation, Satisfaction/Quality, Timing and Duration. SE increased significantly from baseline (2.37 ± 0.66) to intervention and there was a significant improvement in week 3 (2.47 ± 0.65), week 4 (2.46 ± 0.65) and week 5 (2.46 ± 0.62) similar to the PSQI sleep efficiency scores. Acute sleep deprivation and sleep disturbance (short sleep duration or reduced sleep efficiency) can impact immunity, which has been attributed to reduced growth hormone release during deep sleep and increased sympathetic output (Irwin, 2015), reduced growth hormone release could also negatively impact athlete recovery following training or competition.

The results of this study suggest that consuming two Kiwifruit one hour before bed is a wholefood based intervention that has potential to promote sleep and recovery in athletes. Further research is warranted in athletic populations to investigate the impact of Kiwifruit consumption on sleep and recovery.

6.8 Limitations

This study is a novel investigation of the impact of a wholefood nutrition intervention (2 x Kiwifruit 1 hour before bed) on the sleep and recovery of elite athletes. Two elite athlete squads were recruited for this research however, the sample size is small (n=15; n = 9 sailing and n = 6 athletics) but it must be noted that the sample represented all the members of both squads, it is recognised that recruitment and retention of elite athletes for research can be difficult, but it is essential as this research informs evidence based practice (Haugen, 2020). The RU-SATED sleep health scores increased from baseline to weeks 2, 3, 4 and 5 indicating an improvement in sleep health during the intervention, however, the differences were not significant, possibly due to the small sample size or a ceiling effect.

Another limitation of the current study is the reliance on self-report measures of sleep and recovery. As noted previously self-report measures (i.e. questionnaires and diaries) are prone to measurement error and recall bias (Thomas et al., 2015), and athletes may overestimate their sleep duration (Caia et al., 2018; Dunican et al., 2017). However, self-report measures are accepted within athletic settings, as they are a relatively simple and inexpensive approach to athlete monitoring, affording a more representative overview of the target population (Halson, 2014), and subjective sleep tends to relate to complaints and help seeking behaviour. Future research into the potential role of Kiwifruit supplementation in the facilitation of athlete sleep and recovery should incorporate both subjective and objective measures of sleep.

The absence of objectives measurs of sleep (e.g. PSG, actigraphy) must be acknowledged as a limitation but unfortunately such measures were not feasible as the research was conducted during lockdown as a result of the global Covid-19 pandemic. The entire study was conducted during lockdown, the pandemic severly curtailed research as countries were forced to go into lockdown as the virus spread (Weiner et al., 2020). Similar to the current study, the majority of research during the pandemic had to be modified to facilitate data collection and maintain particpaint risk of Covid-19 infection (Weiner et al., 2020). When it is possible to do so, this study should be replicated using a larger cohort of elite athletes incorporating a combination of subjective and objectives measures of sleep and recovery in a randomised control trial.

6.9 Practical applications

The potential roles for specific foods and/or nutrients in promoting sleep quantitiy or quality and athlete recovery is an emerging area of interest within sport nutrition research.

Potential nutritional interventions that could positively impact athletes' sleep, causing resultant improvements in recovery warrant investigation. The potential of nutrition to influence sleep is related to various neurotransmitters associated with the sleep wake cycle (e.g. melatonin and serotonin in Kiwifruit). Although the rsearch in this field is in its infancy the current study adds to the limited body of evidence that Kiwifruit consumption can postiviely impact sleep. The manipulation of the timing and dose of kiwifruit may have applications in terms of sleep and recovery (e.g. antioxidant consumption in relation to training) in athletes that warrants further investigation. The results presented suggest a potential role for Kiwifruit consumption in sleep promotion and recovery protocols for elite athletes. Consuming 2 Kiwifruit 1 hour before bed is a practical wholefood based intervention that can easily be implemented in real-world settings. Kiwifruit is available in wholefood form, but it is also consumed in various processed forms e.g. drinks, sweets, lyophilised products (i.e. freeze dried), dehydrated products and juices (Zehra et al., 2020). Further research is warranted to develop protocols and/or products designed specifically to promote sleep and/or recovery in elite athletes.

6.10 Conclusion

The consumption of two Kiwifruit, one hour before bed, for four weeks has potential to positively impact the sleep and recovery of athletes. The results of the current study demonstrated a positive impact of Kiwifruit consumption on key aspects of sleep and recovery in elite athletes. In summary, from baseline to post-intervention there were clinically significant inprovements in sleep quality (i.e. improved PSQI global scores and sleep quality component scores) and improvemets in recovery stress balance (i.e. reduced general stress and sports stress scales). During the intervention consumption of two Kiwifruit one hour before bed improved sleep as evidenced by significant increases in TST and SE % and significant reductions in number of awakenings and WASO.

Chapter 7: Summary, Practical applications and Conclusion.

7.1 Summary

Sleep inadequacy has been reported to be high among elite athlete populations due to both sport-specific factors (i.e. training, injury travel and competition) and lifestyle/social factors (i.e. habitual caffeine use, technology and social media use in an 'always connected' society) (Walsh et al., 2021). Moreover, research has highlighted athlete specific factors for sleep inadequacy such as high training loads (Dumortier et al., 2018; Kölling et al., 2016; Hausswirth et al., 2014), short and long-haul travel (Fowler et al., 2020; Fowler et al., 2015; Samuels et al., 2012), competition anxiety (Juliff et al., 2015; Erlacher et al., 2011), evening competition after 6:00pm (Fullagar et al., 2016; Nédélec et al., 2019), and early morning start times before 8:00am (Sargent et al., 2014). The potential role of nutrition in sleep quality, quantity and athlete recovery has recently become a key area of research focus. The concept that nutritional interventions may improve athlete sleep and recovery times via mechanisms such as improving hormonal status, muscle protein synthesis and/or muscle glycogen stores has stimulated increased research in this area. However, the research is in it's infancy and further research is necessary to develop nutrition guidelines, products, protocols and tailored interventions for athletes designed to enhance athlete sleep, recovery and performance. The current thesis sought to add to the existing body of knowledge and understanding of the interaction of sleep, nutrition and athlete recovery. The aims of this thesis were to:

- Outline the current research in relation sleep and nutrition and the implications for athletes (Chapters 1-3).
- Characterise the sleep and recovery practices of elite and sub-elite athletes (Chapter 4).
- Assess the impact of long haul travel on the sleep of elite athletes (Chapter 5).
- Assess the impact of kiwifruit consumption on the sleep and recovery of elite athletes (Chapter 6).

7.1.1 Chapters 1-3: Sleep and nutrition interactions – implications for athletes.

As outlined in Chapters 1-3, sleep can be promoted either by inhibiting wake-promoting mechanisms or by increasing sleep promoting factors (Peukhuri et al., 2012). Athletes may experience significant problems sleeping due to the lack of an appropriate sleep routine due to ever changing training and competition schedules, general lifestyle factors and other sleep-incompatible behaviours e.g. late night blue light exposure (Tuomiletho et al., 2016). Irregular sleep-wake patterns influence the homeostatic and circadian regulation of sleep,

which reduces both sleep quality and quantity (Fischer et al., 2008). For athletes, post-competition routines and heightened arousal (i.e. medical care, recovery strategies, meals, media commitments and travel) can lead to later bedtimes, which can adversely affect sleep quality, sleep quantity and recovery. Reduced sleep is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis (Fullagar and Bartlett, 2016), blunting training adaptations and recovery which may lead to compromised performance

Based on the research reviewed in Chapters 1-3, the importance of sleep for not only athletes' recovery but also their performance is clear. When sleep is reduced to < 7 hours there is a detrimental impact on both cognitive performance (i.e. alertness, reaction time, memory and decision making) and physical performance while injury risk is increased (Charest and Grandner, 2020; Laux et al., 2015; LeMeur et al., 2013). Adequate sleep including afternoon naps can counteract the negative performance, cognitive, immunity, oxidative stress (OS), non-functional overreaching (NFO) and increased pain outcomes that are consequences of sleep debt (Walsh et al., 2021). Sleep is essential to recover from the fatigue accumulated by athletes during both training and competition, and athletes have reported sleep as their most important recovery modality (Venter et al., 2012). Unless an athlete recovers quickly their subsequent training, workload and ultimately performance will suffer (Boompa and Haff, 2009). If the athlete does not recover properly fatigue accumulates resulting in maladaption and reduced performace, if this is not addressed the athlete can develop NFO or unexplained underperformance syndrome (UPPS) in the short term and ultimately over-training syndrome (OTS) in the longer term (Lewis et al., 2015a; Meeusen et al., 2013).

Elite athletes are particularly vulnerable to sleep difficulties due to high training and competition demands (Walsh et al., 2021), as such investigations the sleep and recovery practices of athletes and potential nutritional interventions to improve sleep duration and quality are warranted (Ordóñez et al., 2017). The research demonstrated a problem with sleep in athletes and a relationship with recovery, it is also clear that Chrononutrition is an area that shows promise, therefore it was necessary to characterise sleep in athletes both in general (Chapter 4), and under specific circumstances (Competition travel – Chapter 5). This allowed the identification of the specific sleep and recovery areas of concern in this population with a view to implementing a chrononutritional intervention (Kiwifruit – Chapter 6) to investigate whether it improves sleep and recovery.

7.1.2 Chapter 4: The sleep and recovery practices of athletes.

While there has been an upsurge in research in this particular field, and the quality of the research is improving, more research is necessary to elucidate the relationship between the sleep and recovery of athletes. The available evidence suggests that athletes are predisposed to sleep inadequacies that are characterised by habitual short sleep durations (< 7 hours per night) and poor sleep quality (e.g. fragmented sleep) (Walsh et al., 2021). The aim of the research outlined in Chapter 4 was to investigate the sleep and recovery practices of a large cohort of elite (n = 115) and sub-elite (n = 223) athletes on both a training/competition day and a rest day, in order to characterise their sleep and recovery behaviours, including an examination of between status (elite vs sub-elite) factors.

The key findings were that the prevalence of poor sleep and potential sleep problems were high, 64% (elite n = 74 and sub-elite n = 146) of athletes were classified as 'poor sleepers' (PSQI global score ≥ 5), while 21% (elite n = 25 and sub-elite n = 45) reported excessive daytime sleepiness (ESS total score ≥ 10), which is of clinical significance. TST was lower in the elite athlete group on both the training/competition day and the rest day, adding to the evidence that elite athletes are particularly vulnerable to sleep difficulties. TST improved in both groups on the rest day, highlighting a potential positive impact on athlete sleep which could benefit recovery. Significantly higher levels of sport-specific recovery were observed in the elite athlete group. Pain was reported by 50% of athletes (elite n = 53 and sub-elite n = 116) while anxiety/depression was reported by 34% of athletes (elite n = 43 and sub-elite n = 73). In relation to nutrition, the most consumed nutrition supplements were whey protein, caffeine, multivitamins, creatine, fish oil, probiotics and vitamin D, while sub-elite athletes reported drinking more alcohol that the elite athletes. This suggests that increasing TST is an important dimension for sleep health in this population and the strategies currently employed do not appear to be efficacious in terms of sleep (as noted by the high overall PSQI scores).

7.1.3 Chapter 5: The impact of long haul travel on the sleep of elite athletes.

Unless the negative effects of both long haul travel and the resultant jet lag can be managed or minimised athlete health, well-being and performance are likely to suffer (Fowler et al., 2020). In order to support athlete health, travel experience, and performance the implementation of sleep hygiene practices, nutritional programming, load monitoring, fatigue reporting and recovery practices are paramount particuarly during long haul travel (Calleja-Gonzalez et al., 2020). The aim of the study described in Chapter 5 was to asses

the impact of long-haul eastward travel (across 7 timezones) on the sleep of elite athletes. The objectives of the research were to: 1.) assess baseline sleep, 2.) the impact of travel on their subjective and objective sleep measures and 3.) assess sleep upon arrival at the destination. The results of the current study indicated that the following sleep parameters: actigraphy derived TIB, TST and SE and sleep diary derived TIB, TST, fatigue going to bed and SQ were significantly negatively impacted by long haul travel particualry on TRAV and A+1 i.e. the 48 hours post travel. Together, the results from these studies suggest that TIB, TST, SE, SQ, sport-specific recovery and fatigue are of particular interest in terms of targets for sleep interventions in this population. The final study aimed to introduce an easily accessible nutrition intervention to determine whether a) it improved sleep generally and b) whether it improved those sleep dimensions most poorly affected in this population.

7.1.4 Chapter 6: Kiwifruit consumption and sleep.

This was the first study to recruit a group of elite athletes to investigate the impact of Kiwifruit on sleep and recovery. The results in Chapter 6, demonstrated a positive impact of Kiwifruit consumption on key aspects of sleep and recovery in elite athletes. In summary, from baseline to post-intervention there were clinically significant improvements in sleep quality (i.e. improved PSQI global scores and sleep quality component scores) and improvements in recovery stress balance (reduced general stress and sports stress scales). Moreover, the intervention (i.e. consumption of two Kiwifruit one hour before bed) improved sleep as evidenced by significant increases in TST and SE % and significant reductions in number of awakenings and WASO.

7.2 Practical applications and recommendations

It is clear from the evidence presented in Chapters 1-3, that more research is necessary to explore and characterise the sleep and recovery practices of athletes, however, a number of practical applications may be proposed:

- The sleep and recovery practices of athletes has been investigated by a growing body of peer-reviewed publications (particularly in the last decade). The quality of the research is subject to variance due to inconsistent, and in some instances unreliable and invalid research methodologies (Walsh et al., 2021). That said, a solid foundation has been established but more research is necessary to investigate and characterise the sleep and recovery of athletes. Future research of various aspects of athletes' sleep such as the

impact of sleep education strategies, screening athletes for sleep problems, investigating napping behaviour/napping interventions and sleep extension are also warranted.

- There is scope for additional research which investigates sport specific and individualised interventions designed to promote the sleep and recovery of elite athletes. While the number of studies investigating the effect of nutritional interventions on sleep are increasing (Samuels et al., 2016; Irwin et al., 2016; Lastella et al., 2016, Peukhuri et al., 2012, Halson, 2013), future research needs to focus on elite athletic populations. It is also recommended that future research includes clear definitions of the athlete populations used such as the framework developed by Swann et al., (2015) employed in the current thesis.
- Optimal nutrition, hydration and sleep are the most effective strategies for recovery in athletes. Further research is necessary to develop practical guidelines/protocols that can be implemented by multi-disciplinary support staff that work with elite athletes/teams. This may require the development of a coach/support staff specific athlete recovery education programme tailored to the specific demands of the sport and individual needs of each athlete.
- Given the adoption of a 'food first' approach by many athletes, there is scope for investigation of 'functional foods' based interventions designed to promote athlete recovery and/or enhance sleep quality, sleep quantity and/or recovery. The adaptive response to training is dictated by a number of variables: duration, intensity, frequency and type of exercise in combination with timing, quality and quantity of nutrition both pre and post-exercise (Jeukendrup, 2017). Training adaptations can be maximised by optimal nutrition practices or reduced by suboptimal nutrition practices. Nutrition support must be periodised in relation to the demands of the athlete's daily training load and overall nutrition goals (Close et al., 2016). Researchers and practitioners now consider 'Competition Nutrition' which is performance focused and 'Training Nutrition' which is adaptation focused, as two separate entities (Close et al., 2016). Further research is recommended to begin the process of developing 'functional food' based periodised nutrition protocols that can be implemented at different phases of the season to promote both athlete sleep and recovery.
- Similarly, further research is also necessary to investigate if the quality, quantity and timing of sleep can be manipulated to promote the uptake of supplements designed to promote recovery following training/competition, where possible elite athlete

populations should be recruited Ideally this research will lead to the development of nutritional interventions for optimising sleep quality, sleep quantity and enhancing post-exercise recovery.

Based on the findings of Chapter 4 a number of recommendations and practical applications were formulated:

- This study collected 'real-life' data from a training/competition day and a rest day relating to the sleep and recovery practices of athletes. Poor sleep and inadequate recovery practices were evident in both the elite and sub-elite athlete groups. In order to promote sleep health and adequate recovery practices in athletes a comprehensive coach and athlete, evidence based education curriculum may need to be developed and implemented.
- Optimising the sleep and recovery practices of athletes would positively impact performance. Monitoring of sleep behaviour, nutrition and recovery-stress responses of athletes aids the identification of irregularities (e.g. due to travel or illness) and allows for early intervention with individual athletes as and when necessary (Heidari et al., 2018). The data collection methods employed in the current study to assess sleep (PSQI, ESS, AMES and CSD_C) and recovery (RESTQ, EuroQoL and nutrition information), could be used by coaches, medical and support staff to monitor athletes and aid the implementation of individual sleep, recovery and nutrition interventions and plans.
- The athletes generally reported improved sleep quality and quantity on rest days which has implications for athlete health, well-being and performance. Fatigue can be managed, and recovery can be enhanced through adequate passive rest and sufficient sleep (Meeusen et al., 2013). Rest days can serve to alleviate boredom and stress perception while the absence of a 'rest day' during periods of intense training has been related to inadequate recovery and the onset of NFO/OTS (Meeusen et al., 2013). A certain degree of overload leading to fatigue is necessary to elicit training adaptation and performace enhancement (i.e. functional over-reaching [FO]) which can be counterbalanced through adequate recovery (Kellmann et al., 2018), facilitated through sleep. Based on the findings presented for the rest day (i.e. reduced WASO and increased TIB, TST and subjective sleep quality), it is recommend that coaches and multi-disciplinary support staff are mindful of the need for adequate rest. Rest and

resultant recovery can be facilitated through the inclusion of sufficient 'rest days' when programming training/competition plans.

- Future research should replicate this investigation of the sleep and recovery practices of athletes, incorporating a combination of subjective and objective measures of sleep and recovery, for a minimum of 1 week (Carney et al., 2012; Anderson et al., 2018). The validity and reliability of combinations of subjective and objective measures in athletic populations warrants further investigation. While this may not be practical during the competitive season there may be a window of opportunity at the end of the season or in preseason.
- The majority of athletes in the current cohort have reported sleep problems, future research is warranted to identify the specific sleep problems that affect athletic populations. It is also necessary to identify if athletes are affected by acute disturbances e.g. competition anxiety or chronic disorders e.g OSA, insomnia and PLMDs (Tuomilehto et al., 2016).
- Future research should investigate the effects of specific nutritional recovery strategies (e.g. Kiwifruit) on sleep in athletic populations. Such practices may already be an established part of an athlete's daily routine, but the potential additional benefit of improved sleep must be explored.
- Whey protein was one of the most prevalent supplements used while casein use was also reported. While research is emerging supporting pre-sleep protein ingestion for muscle recovery (Snijders et al., 2019; Falkenberg et al., 2021), the impact of pre-sleep ingestion of 40g doses of Whey and/or Casein warrants further investigation with regards to both muscle recovery and sleep improvement in athletic populations.

Based on the findings of the research outlined in Chapter 5, it is clear that:

- During long-haul travel disruption of the morning nadir and late afternoon peak in performance may occur, with both physical and cognitive performance reduced outside of this circadian peak 'window' (Leatherwood et al., 2013; Reilly et al., 2001).
- An individualised approach incorporating an assessment of the athlete's perceived sleep need should be employed. As such, a one size fits all approach to athlete sleep recommendations, sleep hygiene strategies and interventions to reduce the symptoms and impact of jetlag may be inadequate (Walsh et al., 2020). A simple subjective assessment of perceived sleep need could be included in future research into the

impact of long haul travel on the sleep of athletic populations. Such an approach has recently been incorporated in a study of elite athletes (n = 175), which included a self-report assessment of sleep need, athletes reported an average sleep need of 8.3 ± 0.9 hours (Sargent et al., 2021).

- A common symptom of jet lag following long-haul travel is a delayed sleep onset (Fowler et al., 2018), which is influenced by direction of travel, number of time zones crossed and time of arrival relative to departure time at country of origin. The results of the current study indicated that the following sleep parameters: actigraphy derived TIB, TST and SE and sleep diary derived TIB, TST, fatigue going to bed and SQ were significantly negatively impacted by long haul travel. In the current study the majority of poor sleep was observed during the initial 48h post travel period, this is consistent with previous research (Smithies et al., 2021; Stevens et al., 2018). These findings support the suggestion that when athletes are required to travel across multiple time zones, particularly for major competitions it is essential to allow enough time between arrival at the destination and the start of the event for athletes to recover and adjust to the new time zone (Lastella et al., 2019). It is clear from the current study and previous research that teams/athletes should complete long-haul travel a minimum 5-6 days prior to competition to allow sufficient time for athletes to recover from travel fatigue and jetlag. However, other factors such as number of time zones crossed, temperature and humidity at the destination may also need to be considered when deciding upon travel protocols. The development and employment of individualised jet lag interventions, including sleep hygiene practices, may help reduce jetlag symptoms and severity (See Appendices 9.2.3 and 9.2.4).

Based on the findings of Chapter 6 several recommendations and practical applications can be made:

- The results presented suggest a potential role for Kiwifruit consumption in sleep promotion and recovery protocols for elite athletes. The potential of nutrition to influence sleep is related to various neurotransmitters associated with the sleep wake cycle (e.g. serotonin in Kiwifruit). Although the research in this field is in its infancy the current study adds to the limited body of evidence that Kiwifruit consumption can positively impact on sleep parameters. Consuming 2 Kiwifruit 1 hour before bed is a practical wholefood based intervention that can easily be implemented in real-world settings. Future research is necessary to develop and investigate the impact of

tailored interventions based on sport specific and individual sleep and recovery needs.

- Kiwifruit are available in wholefood form but are also consumed in various processed forms e.g. drinks, sweets, lyophilised products (i.e. freeze dried), dehydrated products and juices (Zehra et al., 2020). Further research is warranted to develop kiwifruit products and/or protocols designed specifically to promote sleep and/or recovery in elite athletes.
- The current study should be replicated with a larger sample size, including a control group and the use of both subjective and objective measures of sleep.

7.3 Conclusion

While this thesis adds to the body of evidence supporting the notion that sleep plays a vital role in athlete recovery and that nutrition can promote sleep and recovery, further investigation of potential nutritional interventions to improve sleep duration, sleep quality and/or recovery in athletes is warranted. Poor sleep and inadequate recovery practices were evident in both the elite and sub-elite athlete groups assessed in this thesis; future research is needed to identify the specific sleep problems that affect athletic populations.

This thesis highlighted that jet lag following long haul eastward travel significantly negavtively impacted the actigraphy derived parameters of TIB, TST and SE and sleep diary derived parameters of TIB, TST, fatigue going to bed and SQ. The majority of sleep disruption was observed during the initial 48h post travel period. It is clear from the current study and previous research that teams/athletes should complete long-haul travel at least 5-6 days prior to competition to allow sufficient time for athletes to recover from travel fatigue and jetlag.

This thesis has also highlighted the potential role for nutrition interventions in the promotion of sleep and recovery of athletes. Given the adoption of a ‘food first’ approach by many athletes, ‘functional foods’ (i.e. kiwifruit) based interventions designed to promote sleep quality, sleep quantity and/or recovery are essential. Although the research in this field is in its infancy the current thesis adds to the limited body of evidence that Kiwifruit consumption can postiviely impact sleep. Kiwifruit consumption resulted in significant increases in TST and SE % and significant reductions in number of awakenings and WASO. Consuming 2 Kiwifruit 1 hour before bed for four weeks is a practical wholefood based intervention that can easily be implemented in real-world settings by athletes.

Although it is accepted that sleep is important for athlete recovery, the research is emerging and evolving. The current thesis sought to add to the understanding of the sleep, nutrition and athlete recovery. It is clear from the research conducted within this thesis that nutrition and sleep interact and impact athlete recovery, the sleep and recovery practices of elite and sub-elite athletes are inadequate, long-haul eastward travel negatively impacts the sleep of elite athletes (particularly in the 48 hours post-travel) and kiwifruit consumption (2 x kiwis 1 hour before bed) has a positive impact on the sleep and recovery of elite athletes.

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Appendices

9.1 Sleep and Nutrition Interactions: Implications for Athletes.

nutrients

MDPI

Review

Sleep and Nutrition Interactions: Implications for Athletes

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Abstract: This narrative review explores the relationship between sleep and nutrition. Various nutritional interventions have been shown to improve sleep including high carbohydrate, high glycaemic index evening meals, melatonin, tryptophan rich protein, tart cherry juice, Kiwifruit and micronutrients. Sleep disturbances and short sleep duration are behavioural risk factors for inflammation, associated with increased risk of illness and disease, which can be modified to promote sleep health. For sleep to have a restorative effect on the body, it must be of adequate duration and quality; particularly for athletes whose physical and mental recovery needs may be greater due to the high physiological and psychological demands placed on them during training and competition. Sleep has been shown to have a restorative effect on the immune system, the endocrine system, facilitate the recovery of the nervous system and metabolic cost of the waking state and has an integral

role in learning, memory and synaptic plasticity, all of which can impact both athletic recovery and performance. Functional food-based interventions designed to enhance sleep quality and quantity or promote general health, sleep health, training adaptations and/or recovery warrant further investigation.

Keywords: sleep; athletes; chrononutrition.

1. What is Sleep?

Sleep, in humans, is defined as a complex reversible behavioural state where an individual is perceptually disengaged from and unresponsive to their environment [1]. Sleep architecture has two basic states based on physiological parameters: non-rapid eye movement sleep (NREM) and rapid eye movement (REM) sleep [2]. Sleep stages fall along a continuum from fully awake to deep sleep [3]. NREM has been defined as "a relatively inactive yet actively regulating brain in a moveable body" [2], (p.17). In terms of brain activity, the electroencephalogram (EEG) pattern of NREM sleep is commonly described as synchronous (increasing depth of sleep is indicated by progressive dominance of high voltage, low frequency EEG patterns), with characteristic waveforms (sleep spindles, K-complexes and high voltage waves) [2]. NREM is usually associated with minimal or fragmented mental activity. Table 1 shows the traditional four stages of NREM which are associated with differing levels of depth of sleep, with arousal thresholds generally lowest in Stage 1 and highest in Stage 4 sleep [2].

Table 1. Characteristics of NREM Sleep.

Stage	Characteristics
1	Sleep is easily discontinued (e.g., noise, a light touch, etc.) Sleep is easily interrupted Key role in the initial wake to sleep transition Transitional stage throughout the sleep cycle
2	More intense stimuli required to produce arousal (e.g., bright light or loud noise) Indicated by K-complexes or sleep spindles in the EEG High voltage slow wave EEG activity will become apparent
3	High voltage (75 μ V) slow wave (two cycles per second [cps]) activity that is ≥ 20% but < 50% of EEG activity
4	High voltage slow wave activity is ≥ 50% of EEG activity.

(Adapted from: [4]).

In contrast, REM sleep is defined by EEG activation, muscle atonia (paralysis) and episodic bursts of rapid eye movement [2]. REM sleep is associated with cognitive activity, while brain stem mechanisms inhibit spinal motor neurons limiting movement. Hence, REM sleep has been defined as "an activated brain in a paralysed body" [2], (p.16). It should be noted that the American Academy of Sleep Medicine (AASM) have recommended alternative terminology for Sleep staging. Wake is referred to as W, NREM sleep is referred to as N and is divided into three stages: N1 – Stage 1, N2 – Stage 2 and N3 – Slow Wave Sleep or Deep Sleep, i.e., Stage 3 and 4 combined; while REM is referred to as R [5].

Sleep health is a multidimensional pattern of sleep-wakefulness adapted to individual, social and environmental demands, which promotes physical and mental wellbeing [6]. Good sleep health is characterised by satisfaction, appropriate timing, adequate duration, high efficiency and sustained alertness during waking hours [6]. Sleep deprivation adversely affects glucose metabolism and neuroendocrine function which can affect carbohydrate metabolism, appetite, energy intake and protein synthesis [1]. These factors may negatively impact an athlete's nutritional, metabolic and endocrine status impacting athletic performance and recovery [1], (e.g., impaired glucose metabolism could reduce glycogen repletion while impaired protein synthesis could reduce recovery and adaptation from training). This narrative review examines and evaluates the interaction between nutrition and sleep.

1.1. How and Why Sleep Occurs

The brain is essentially an electrical system with circuits that switch on and off to promote either wakefulness or sleep. Since the arousal and sleep-promoting systems are mutually inhibitory, a sleep switch or 'flip-flop' model has been proposed [7]. A flip-flop switch contains mutually inhibitory elements where activity in one of the competing sides shuts down inhibitory inputs from the other side producing two discrete states with sharp transitions [8]. Activation of arousal systems inhibits sleep active neurons facilitating sleep while activation of sleep-promoting neurons inhibits arousal-related neurons reinforcing consolidated sleep episodes providing a mechanism for stabilisation of sleep and waking states [9].

The circadian rhythm in humans has been estimated in young males (24.18 ± 0.04 h; PCV 0.54%) and older adults (24.18 ± 0.04 h; PCV 0.58%), low percentage coefficients of variation and no significant difference between the groups indicated a small range variability in circadian rhythms [10]. HuHPAmans however, typically display individual differences in their behaviour (e.g., social activities, daytime activities and sleep). Chronotype is the expression of individual circadian rhythmicity and has been categorised as follows: morning types, intermediate types and evening types [11]. Chronotype is, in part, genetic but cultural and environmental factors also affect an individual's sleep pattern. Research in the general population has demonstrated that most people are intermediate types (70%) with the remainder being either morning types (14%) or evening types (16%) [12].

Sleep is a dynamic process largely regulated by two factors; the circadian systems and the sleep homeostat. The Two Process Model for Sleep Regulation was developed to illustrate the interaction of the homeostatic sleep drive (sleep pressure or urge to sleep that accumulates during wakefulness) and the circadian system (endogenous timing system) in the timing and duration of sleep [13,14]. The homeostatic process (S) is a function of sleep and waking, while the circadian process (C) is controlled by a circadian oscillator [10]. S increases during waking and declines during sleep and it interacts with C, which is independent of sleep and waking and receives cues (e.g., light) from the environment [13,14]. The suprachiasmatic nucleus (SCN) in the brain is central to this process but secondary clock systems have been identified throughout the body [14].

Process S is an endogenous mechanism, relying on exogenous cues to regulate it to approximately 24 h. Process S represents sleep debt which increases during waking and reduces during sleep within a range that oscillates within a period that is normally entrained to day and night by process C [14]. When S reaches the lower boundary of the range, awakening is triggered and when S reaches the upper boundary sleep is triggered [14]. In terms of process C, the Two-Process Model focuses on time-of-day effects on sleep propensity, specifically that sleep propensity is minimal near midday and is strongly promoted in the early hours of the morning [13]. This circadian rhythmicity in sleep propensity is combined with S by C dictating the threshold values at which S transitions from sleep to wake, and vice versa [13,14]. Core body temperature and melatonin rhythms are markers of C [11]. The SCN has melatonin receptor cells, as darkness falls, melatonin is secreted by the pineal gland making the individual sleepy [15]. Animal studies have demonstrated that exogenous melatonin and ramelteon (an MT1/MT2 melatonin receptor agonist) function as non-photic entrainers, which phase advance the SCN [16]. A Three-Process Model of Sleep Regulation has also been proposed whereby sleepiness and alertness are stimulated by the combined action of a homeostatic process, a circadian process and sleep inertia process, the model has been extended to include sleep onset latency (the length of time of the transition from wakefulness to sleep), sleep length and performance [17].

Sleep has a restorative effect on the immune system and the endocrine system, facilitates the recovery of the nervous and metabolic cost of the waking state and has an integral role in learning, memory and synaptic plasticity (ability of synapses to strengthen or weaken over time) [18,19]. Sleep, particularly slow wave sleep (or N3) early in the night promotes prolactin release, while the anti-inflammatory actions of cortisol and catecholamines are reduced [18]. Acute sleep deprivation and sleep disturbance (short sleep duration or reduced sleep efficiency) impair adaptive immunity which is associated with reduced response to vaccinations and increased vulnerability to infectious diseases, attributed to reduced growth hormone release during deep sleep and increased sympathetic output [20]. Tumour necrosis factor-α (TNFα) along with other cytokines are considered key to the regulation of sleep in normal physiological conditions [21]. Research has demonstrated that sleep disturbance (i.e. insomnia) and extremes of sleep durations affect risk factors of inflammatory disease and contribute to all-cause mortality [18,22]. Increased levels of circulating inflammatory markers (i.e., C-reactive protein [CRP] and Interleukin-6 [IL-6]) predict body mass gain in older adults [23] and type 2 diabetes [24]. Sleep disturbance is believed to have proximal effects on IL-6, which induces CRP [18], therefore, increases in CRP may be attributed to persistent or severe sleep disturbance. In a recent meta-analysis sleep

disturbance (i.e., poor sleep quality, insomnia) was associated with increased levels of IL-6 (ES: 0.20 (0.08–0.31)) and CRP (ES: 0.12 (0.05–0.19)) [18]. Short sleep duration (< 7 h per night) was associated with increased IL-6 (ES: 0.29 (0.05–0.52)), while long sleep duration (> 8 h per night) was also associated with increased IL-6 (ES: 0.11 (0.02–0.20)) but also increased CRP (ES: 0.17 (0.01–0.34)) [18]. Similarly, a meta-analysis of sleep duration and all-cause mortality demonstrated a U-shaped association, whereby long sleep (> 8 h per night) has a 30% (RR: 1.30 (1.22–1.38)) greater risk while short sleep (<7 h per night) has a 12% (RR: 1.12 (1.06–1.18)) greater risk compared to normal sleep reference (7–8 h per night) [25].

Inappropriate timing of lifestyle behaviours can cause disruption to the circadian rhythm, resulting in an altered physiological response (e.g., poor sleep). Lifestyle factors (e.g., caffeine consumption, alcohol consumption and timing of sleep) can cause alterations in environmental cues which may negatively impact circadian rhythms and in turn result in negative physiological consequences [26]. The SCN receives environmental cues such as the light-dark cycle and additional information from other areas of the brain (e.g., when we eat or exercise). Give that Process C can be modified by exogenous cues [27], there is scope for investigation of nutrition interventions to enhance sleep quality and quantity. Similarly, the effect of nutrition interventions that promote athlete recovery on sleep quality and quantity should be investigated.

2. Sleep and Athletes

The classic view of sleep is that it is a recovery process, with the circadian system regulating feelings of sleepiness and wakefulness throughout the day [28]. Cognition, tissue repair and metabolism are critical psychological and physiological factors that contribute to training capacity, recovery and ultimately performance [28]. The relationship between sleep, performance and recovery can be viewed in terms of 3 key factors that affect the recuperative outcome:

4. Sleep length (total sleep duration; hours/night, plus naps)
5. Sleep quality (i.e., the experience and perceived adequacy of sleep)
6. Sleep phase (circadian timing of sleep) [28].

Post-exercise recovery is vital for all athletes. If the balance between training stress and physical recovery is inadequate, performance in subsequent training sessions or competition may be adversely affected [15]. Muscle fatigue or soreness may adversely affect sleep, with inflammatory cytokines linked to disruption of normal sleep [29]. Inadequate recovery can reduce autonomic nervous system (ANS) resources, with an associated reduction in heart rate variability (HRV) and increased resting heart rate [30]. Sleep deprivation is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis [31], blunting training adaptations and recovery.

Sleep disturbances and inadequate sleep duration have been reported in athletic populations. Assessment of the sleep patterns of professional male ice hockey players (n = 23) using polysomnography (PSG), demonstrated mean total sleep duration was 6.92 h; 95% CI 6.3–7.5 h [32]. Similarly, sleep was self-reported as the most important recovery modality utilised by South African athletes (n = 890; international n = 183, national n = 474, club n = 233) [15]. While a similar study found that 66% (n = 416) of elite German athletes (n = 632) reported pre-competition insomnia symptomology including difficulty falling asleep, waking during the night and early final waking times [33]. Sleep duration (< 8 h) has been identified as the strongest predictor of injury in adolescent athletes (RR = 2.1; 95% CI: 1.2–3.9) [34]. The Karolinska Athlete Screening Injury Prevention (KASIP) study investigated injury occurrence in Swedish adolescent elite athletes (n = 340; 178 males and 162 females) and demonstrated that athletes sleeping >8 h were less likely to suffer an injury (OR: 0.39; 95% CI 0.17–0.96) [35]. The aetiology of sleep disturbances is unclear during periods of intense training, it is unclear whether poor sleep is a symptom of overtraining, or intense training negatively affects sleep and recovery [30]. Sleep also has a pivotal role to play in performance, training adaptations and recovery [1,18]. Given the importance of sleep for athlete recovery, further research is warranted to investigate potential nutritional interventions to promote improved sleep quality and/or duration and recovery in athletes.

2.1. Sleep, Nutrition and Athletes

Nutrition support needs to be periodised in relation to the demands of the athlete's daily training and overall nutritional goals [36]. The focus of 'training' nutrition is to promote adaptations while the focus of 'competition' nutrition is optimal performance [36]. Athletes also have added responsibility to adhere to the World Anti-Doping Agency (WADA) code and are subject to testing for prohibited substances. If an athlete chooses to take any supplement they must do so in a safe and effective manner. Athletes should check that any

supplement they take has been tested for banned substances, and independent testing programmes (e.g., Informed Sport and Informed Choice) offer additional protection. Athletes are advised to seek the professional advice of a qualified sports dietician/nutritionist regarding any nutritional supplement. Training adaptations and recovery can be maximised by optimal nutrition practices or impaired by suboptimal nutrition practices [36–38]. Nutrients such as carbohydrate (high glycaemic index evening meal reduced sleep onset latency), protein (consumption of dairy sources may increase sleep duration), ethanol (reduced REM sleep) [37] and caffeine (increased sleep onset latency, reduced total sleep duration and reduced sleep quality) [39], as well as the timing and quantity of meals (large portions and/or meals later in the evening can negatively impact sleep potentially due to the thermogenic effect of digestion) can affect circadian rhythms [40]. Caffeine consumption can lead to poor sleep which, in turn, can lead to increased caffeine consumption. Caffeine increases the state of alertness, antagonising adenosine receptors, which also leads to a reduction in the inclination to sleep [39]. Alcohol consumption has been associated with poorer sleep quality and quantity, reduced REM sleep and increased sleep disturbance in the second half of the sleep bout [41]. Similar to nutrition, sleep disturbances (difficulty initiating or maintain sleep) and sleep deprivation (not getting enough sleep) are risk factors for inflammation [18,42], which can be treated or managed to promote recovery and/or performance. For sleep to have a restorative effect on the body, it must be of adequate duration which is dependent on age [28,42]. Sleep recommendations particularly the amount of sleep required, change over the lifespan from adolescents (8–10 h), adults (7–9 h), and older adults (7–8 h) [42].

2.1. Chrononutrition

Recently the term Chrononutrition has been used to describe the interaction between food and the circadian system [39]. It has been suggested that the internal clock can be altered by changing the timing and nature of food intake [39]. Chrononutrition has been characterised as including two aspects:

3. Timing of food intake or contributions of food components to the maintenance of health; and
4. Timing of food intake or contributions of food components to rapid changes in or resetting of a human's system of internal clocks [39].

Several neurotransmitters are involved with the sleep-wake cycle including 5–hydroxytryptophan (5-HT), GABA, orexin, melanin concentrating hormone, cholinergic, galanin, noradrenaline and histamine [7]. Therefore, nutrition interventions that act on these neurotransmitters could positively impact sleep. Dietary precursors can influence the rate of synthesis and function of neurotransmitters (e.g., serotonin synthesis is dependent on the availability of its precursor tryptophan in the brain) [1]. Tryptophan is transported across the blood brain barrier by a system that shares transporters with several large neutral amino acids (LNAA) [1]. The ratio of tryptophan:LNAA in the blood is vital to the transport of tryptophan into the brain and can be increased through consumption of tryptophan, a high carbohydrate/low protein diet or α-lactalbumin (whey derived protein) [43].

2.1. Carbohydrate

Carbohydrate consumption has been shown to increase plasma tryptophan concentrations [44]. Carbohydrates affect plasma tryptophan:LNAA ratio and may compliment the sleep enhancing effect of consuming tryptophan rich protein [40]. Insulin influences the transport of tryptophan across the blood brain barrier after a carbohydrate rich meal, as it is an anabolic agent it also facilitates the uptake of LNAA by muscle [26]. Consumption of high glycaemic index (GI) carbohydrate increases the ratio of circulating tryptophann:LNAA via direct action of insulin which promotes muscle uptake of LNAA [45]. This increases tryptophan availability for synthesis of serotonin and ultimately melatonin. GI has been shown to affect sleep onset latency (length of time of the transition from wake to sleep) [44]. A high GI meal consumed four hours before bed, significantly (p = 0.009) reduced sleep onset latency (9.0 ± 6.2 min) compared to a low GI meal (17.5 ± 6.2 min) and the same meal consumed 1 hour before bed (14.6 ± 9.9 min) [44]. Among a large sample (n = 4452) from the National Health and Nutrition Examination survey lower carbohydrate intake (24–h recall and structured interview) has been significantly associated (OR 0.71; 0.55–0.92, p = 0.01) with insomnia symptoms (difficulty maintaining sleep) [46]. Consumption of a high-carbohydrate meal (130 g) when compared to a low-carbohydrate meal (47 g), or a meal containing no carbohydrate, 45 min before bedtime increased REM and decreased light sleep and wakefulness [47]. The timing of carbohydrate evening meals and the carbohydrate content of evening meal on sleep and athlete recovery requires further investigation within athletic populations.

2.1. Melatonin

Melatonin is a hormone secreted by the pineal gland, that has displayed sedative effects [48,49]. Since endogenous melatonin influences core temperature facilitating sleep, increased exogenous melatonin could affect changes in core temperature improving sleep quality [50]. However, the effect is relative to the person's endogenous melatonin levels. In many Western countries, Cow's milk has traditionally been considered a sleep promoting beverage. Melatonin is a naturally-occurring compound in cow's milk, but its concentration increases significantly if cows are milked in darkness at night referred to as 'night time milk' [40]. Increased tryptophan and melatonin concentrations appear to be the property responsible for the sleep promoting effect of night time milk. Consumption of night time milk (melatonin concentration of 39.43 pg/mL) compared to daytime milk (4.03 pg/mL) significantly increased circulating melatonin (26.5%) concentration in rats [51], indicating that high melatonin concentrations are necessary for milk to affect blood melatonin concentrations. When the night time milk was supplemented with tryptophan (2.5 g/L) circulating melatonin concentrations significantly increased further (35.5%) [51].

Melatonin has extremely low toxicity even at relatively high doses and can easily cross physiological barriers due to its optimal size, partial water solubility and high lipid solubility however, it must be noted that there appears to be no added benefit to doses >3 mg [49]. Ingestion of melatonin affects sleep propensity and has hypnotic effects enhancing sleep quality and duration, pharmacological melatonin can be used to manipulate circadian timing [49]. A positive effect of low doses (0.3 mg or 1 mg) of exogenous melatonin (gelatin capsules) on sleep onset latency has been observed in a small group of healthy males (n = 6), when administered at either 6:00 pm (0.3 mg 16.5 ± 19.9 min; 1 mg 12.3 ± 13.6 min; Placebo 23.1 ± 22.7 min) and 8:00 pm (0.3 mg 19.6 ± 14.1 min; 1 mg 20.7 ± 17.7 min; Placebo 53.4 ± 51.9 min) [52]. However, the impact was time dependent as a 0.3 mg dose increased sleep onset latency when consumed at 9:00 pm (0.3 mg 25.1 ± 10.5 min; 1 mg 12.1 ± 7.4 min; Placebo 8.8 ± 4 min) and there was no evidence of an effect when the 1 mg dose was administered at 9:00 pm [52]. The results indicate a low dose of melatonin similar to nocturnal physiological concentrations can elicit a sleep-inducing effect. A dose response relationship was not evident as the 0.3 mg dose, which is similar to endogenous melatonin concentrations, was as effective as the 1 mg dose when administered at 6:00 pm or 8:00 pm.

2.1. Tryptophan Rich Protein

Tryptophan is an essential amino acid that is a precursor to serotonin and melatonin, which can cross the blood-brain barrier by competing for transport with other LNAA [1]. Conversion to serotonin is dependent on sufficient precursor availability in the brain, an increase in brain tryptophan occurs when the ratio of free tryptophan to branched chain amino acids increases, following tryptophan conversion to serotonin, melatonin is produced [1]. Dietary sources of tryptophan include milk, turkey, chicken, fish, eggs, pumpkin seeds, beans, peanuts, cheese, and leafy green vegetables. Dietary tryptophan has been shown to improve sleep, in a comparison of food bars (Food 1: 25 g deoiled butternut squash seed meal and 25 g dextrose, Food 2: 250 mg of pharmaceutical tryptophan and Food 3: 50 g rolled oats [control]) [53]. Food 1 and Food 2 produced significant results ($p \leq 0.05$) for reduction of time awake during the night (19.2% and 22.1%), increased sleep efficiency (% of time spent in bed; asleep) (5.19% and 7.36%) and increased subjective sleep quality (12.2% and 11.8%) [53], indicating that relatively small doses (250 mg) of dietary tryptophan can positively impact sleep. The milk protein, α-lactalbumin has been reported as having the highest natural levels of tryptophan among all protein food sources [54]. Ingestion of α-lactalbumin enriched whey protein, significantly ($p < 0.05$) increased tryptophan:LNAA by 48% compared to a casein enriched diet [54]. In a similar study, healthy adults (n = 14) with sleep complaints consumed milkshakes containing either α-lactalbumin (20 g) or a casein placebo. Evening ingestion of α-lactalbumin resulted in a 130% increase in tryptophan:LNAA prior to bed and modest but significant reduction in morning sleepiness and improved alertness the following morning [55]. Tryptophan depletion studies have demonstrated decreased tryptophan plasma concentrations affected sleep fragmentation (arousal index (events/h)), REM sleep latency (the interval between first epoch of stage 2 and the first epoch of REM sleep), and REM density (the cumulated duration of each REM burst divided by the duration of each REM sleep period) compared to baseline and placebo [56,57]. Consumption of tryptophan rich protein (e.g., milk) could affect changes in core temperature improving sleep quality [51]. The effects of tryptophan rich protein (e.g., α-lactalbumin enriched whey and casein) interventions on sleep and recovery, warrant further investigation.

2.1. Antioxidants

Both the general population and athletes can benefit from nutritional and supplementation support to boost immunity and reduce acute and chronic inflammation during periods of increased training load and competition. Antioxidants are any substance that significantly delay or prevent oxidative damage of a target molecule [58]. The fact that exercising muscles produce free radicals has motivated many athletes to consume antioxidant supplements in an attempt to reduce exercise induced free-radical damage and/or muscle fatigue. The antioxidant capacity of several dietary micronutrients is an emerging area of interest to support the endogenous antioxidant defence system of athletes and attenuate the negative effects of oxidative damage due to free radicals. Antioxidant consumption may influence recovery from exercise but may also influence sleep since sleep regulation is influenced by pro-inflammatory cytokines [59]. Dietary antioxidants (e.g., vitamin C and vitamin E) augment endogenous antioxidant content within skeletal muscle [59]. Vitamin E is a fat-soluble vitamin made up of several isoforms known as tocopherols, with α-tocopherol being the most active and abundant [60]. Vitamin E is an important antioxidant due to its abundance within cells, mitochondrial membranes and its ability to act directly on reactive oxygen species [60]. Vitamin E reacts with other antioxidants such as vitamin C, beta-carotene and lipoic acid, which have the capacity to regenerate vitamin E from its oxidised form. However, supplementation with high doses (800 IU/day) of vitamin E did not counteract OS in triathletes (n = 38), the intervention group demonstrated significantly ($p \leq 0.05$) higher levels of post-race inflammation and OS (Plasma F_2-isoprostanes increased 181% versus 97% and IL-6 166 ± 28 $pg \cdot mL^{-1}$ versus 88 ± 13 $pg \cdot mL^{-1}$) than the control group [60]. Interestingly, despite increased markers of OS and inflammation in the intervention group, there was no significant difference between the groups in terms of race performance.

Vitamin A is a fat-soluble vitamin present in many lipid substances, beta-carotene can be converted into vitamin A, when necessary, from within the body [61]. Vitamin C is a water-soluble vitamin and is extremely effective in extracellular fluids, but is also effective in the cytosol [61]. It must be noted that antioxidants are heterogeneous, they function in a distinct manner and do not solely regulate ROS [61]. Consumption of an antioxidant does not guarantee that the compound will act as an antioxidant within the body, therefore positive findings from one antioxidant or combination of antioxidants cannot be generalised [59]. It has been suggested that a high intake of antioxidants could potentially reduce training adaptations. It is accepted that repeated exercise bouts (i.e. training) induce disruption in skeletal muscle homeostasis that regulate training adaptations [62,63]. While it has been reported that high doses of antioxidants could reduce training adaptations of muscle mitochondrial biogenesis and VO_{2max}, not all antioxidant studies have demonstrated negative effects and it has been suggested that the specific antioxidant used, the dose and timing of ingestion all affect outcomes [64]. It must be noted that the majority of studies have been conducted on healthy adults and there is inconsistency in terms of supplementation protocols, duration and also a wide variety of exercise protocols have been utilised. Antioxidants reduce OS, play a key role in immunity and may improve recovery following exercise [58,59]. Further research is necessary to investigate the recovery promoting doses of antioxidants within athletic populations [38,65]. The potential sleep promoting benefits of antioxidant consumption/supplementation should be investigated also.

2.7.1. Tart Cherries

Tart cherries contain high concentrations of melatonin and a range of phenolic compounds that have both antioxidant and anti-inflammatory properties [66,67]. A recent study was conducted to investigate the effect of tart cherry juice (2 × servings of 30 mL concentrate) on sleep enhancement, sleep duration and sleep quality [50]. This was the first investigation to demonstrate that tart cherry juice supplementation increased circulating melatonin levels and improved sleep time and quality in healthy adults. In the intervention group tart cherry juice supplementation resulted in significantly elevated total melatonin content, increased time in bed (+24 min), increased total sleep duration (+34 min) improved sleep efficiency total (82.3%) and a significant reduction in daytime napping (–22%) ($p < 0.05$) [50]. It must be noted that elevated melatonin concentrations may not be only mechanism at work as sleep regulation is also influenced by proinflammatory cytokines [3]. Tart cherries also contain numerous compounds that have antioxidant and anti-inflammatory properties. A similar study demonstrated that tart cherry juice consumption resulted in significantly reduced insomnia severity index scores (13.2 ± 2.8 versus control 14.9 ± 3.6; $p < 0.05$) and wake after sleep onset time (62.1 ± 37.4 min versus control, 79.1 ± 38.6 mins; $p < 0.01$), in older females with insomnia ($n = 7$) compared to a placebo [68].

Indeed, there is evidence that tart cherry juice supplementation post exercise may aid recovery from running a marathon [67]. The intervention group demonstrated a more rapid return of baseline isometric knee extension strength (pre-race 432 ± 114 vs. 48h 435 ± 109), 48 h post-marathon which was not demonstrated in the control group (pre-race 384 ± 112 vs. 48 h 349 ± 96) [67], indicating that consumption of tart cherry juice may blunt the secondary muscle damage response (localised inflammation). Post-race levels of inflammation were significantly reduced in the intervention group (IL-6 41.8 pg/mL) compared to the control group (IL-6 82.1 pg/mL) [67]. Similarly, post-race elevations in CRP and uric acid were significantly reduced in the intervention group ($p < 0.001$) [67]. Total antioxidant capacity was increased in both groups post-race (intervention 124% of baseline and control 112% of baseline, $p < 0.01$) and remained elevated at 24 h in the intervention group (114% of baseline) but not the control group [67]. During recovery athletes can suffer from delayed onset muscle soreness (DOMS) [66], which can reduce sleep quantity and quality. A recent study has demonstrated tart cherry juice supplementation (30 mL, twice per day for seven days) reduced the post-exercise decline in functional performance following intermittent sprint activity (maximal voluntary isometric contractions, 20 m sprint, counter movement jump and 505 agility test), DOMS and inflammatory response (Il-6) [66]. With regards the reduction in both DOMS and the post-exercise inflammatory response, in practice, the researchers suggested that this might be beneficial during periods of high-volume training (e.g., pre-season) or where athletes are required to produce multiple performances in a short space of time (e.g., double training sessions), when recovery periods are short [66]. The range of phenolic compounds in cherries which have anti-inflammatory and antioxidant properties may enhance post exercise recovery as well as sleep [50]. It has been proposed that melatonin may be synthesised in mitochondria, making melatonin and its metabolites available to protect the muscle against oxidative stress. Melatonin also increases the protective effects of glutathione, vitamin C and trolox, through regeneration by electron transfer processes [69].

2.7.2. Kiwifruit

Kiwifruit are nutritionally dense containing a range of nutrients that can benefit sleep, health and recovery including serotonin, vitamin C, vitamin E, vitamin K, folate, anthocyanidins, carotenoids, beta-carotene, lutein, potassium, copper and fibre [70]. Interest in the antioxidant capacity, enzyme, polyphenolic and phytochemical content of Kiwifruit has increased steadily over the last decade [70–72]. It has been suggested that the various bioactive components in Kiwifruit may act synergistically affecting various physiological and metabolic processes (e.g., inhibition of oxidative and inflammatory responses, improved gastrointestinal tract health and bowel function) [73]. Contemporary research has focused on the health benefits of Kiwifruit particularly in relation to antioxidant capacity, digestion, iron nutrition, metabolic health and immune function [73].

Regular consumption of Kiwifruit has been found to significantly ($p \leq 0.05$) increase plasma vitamin C [74] vitamin E [71] and lutein/zeaxanthin concentrations [71,74]. A study involving volunteers ($n = 25$) with a self-reported sleep disturbance demonstrated consumption of two Kiwifruit one hour before bedtime for four weeks significantly improved actigraphy-measured total sleep duration (+16.9%, baseline 354.5 ± 17.1 min; post-intervention 395.3 ± 17.4 min) and sleep efficiency (+2.4%, baseline 93.9 ± 1.03 min; post-intervention 95.9 ± 0.67 min) ($p \leq 0.005$) [70]. Self-report measures also improved significantly, wake after sleep onset reduced (time awake during sleep period) (–28.9%), sleep onset latency reduced (–35.4%) while sleep efficiency increased (5.4%) ($p \leq 0.002$) [70]. Sleep quality was significantly improved following the four-week Kiwifruit intervention however, the lack of a control group must be noted and there was a high level of subject drop out ($n = 5$). The findings may be prone to bias as participants were recruited based on interest in participation in a dietary intervention study relating to sleep.

Serotonin is the end product of L-tryptophan metabolism and is related to REM [70]. The serotonin content in Kiwifruit may contribute to improved sleep while the rich antioxidant content may supress free radical expression and inflammatory cytokines. Folate deficiency has been linked to insomnia (difficulty initiating or maintaining sleep, extended periods of wakefulness and/or insufficient sleep) and restless leg syndrome (repeated movement of or undesirable sensations in legs leading to sleep disruption) [70], the high folate content in Kiwifruit may improve folate status and consequently improve sleep [70]. Although folates are widely consumed in the diet, they are destroyed by cooking or processing, however, Kiwifruit are typically consumed in their raw form. Further research is warranted within athletic populations to investigate the potential sleep promoting properties of Kiwifruit and the effect of Kiwifruit consumption on both sleep quality, sleep quantity and recovery.

2.7. B Vitamins and Magnesium

Vitamin B_{12} contributes to melatonin secretion, pyridoxine (vitamin B_6) is involved in the synthesis of serotonin from tryptophan and niacin (vitamin B_3) may elicit a tryptophan sparing effect [40]. Niacin can be synthesised endogenously from tryptophan via the Kynurenine Pathway, therefore, consuming a sufficient amount of niacin is necessary to inhibit 2,3-dioxygenase activity eliciting a tryptophan sparing effect, increasing its availability for synthesis serotonin and melatonin [40]. Folate (vitamin B_9) and pyridoxine are involved in the conversion of tryptophan into serotonin [45]. The reduced form of folate (5-methyltetratrahydrofolate) increases tetrahydrobiopterin is a co-enzyme of tryptophan-5-hydroxylase which converts tryptophan into 5-hydroxytryptamine (5-HT) [45]. Pyridoxine's role in the conversion of tryptophan into serotonin is related to the amino acid decarboxylase which speeds up the conversion rate of 5-HT to serotonin [45]. Mixed effects have been observed, with different doses of cobalamin (vitamin B_{12}) on sleep-wake rhythms and delayed sleep phase syndrome (a significant delay in circadian rhythm), while no effect was observed for sleep duration [41].

Magnesium is also believed to enhance melatonin secretion promoting sleep onset and act as a GABA agonist, the main inhibitory neurotransmitter that acts on the CNS [40]. Magnesium is important for the production of the enzyme N-acetyltransferase which converts 5-HT into N-acetyl-5-hydroxytryptamine, which can then be converted to melatonin [44]. A placebo controlled double blind study on older adults (n = 43), demonstrated a food based supplement containing 5 mg melatonin, 225 mg magnesium and 11.25 mg zinc, significantly ($p < 0.001$) improved subjective sleep quality scores in the intervention group but not the controls (difference between the groups 6.8; 95% CI 5.4–8.3) and total sleep duration (182.18 min; 95% CI 160.02–204.34) assessed via actigraphy [75]. The effects were attributed to the synergy between magnesium, zinc and melatonin [75]. It must be noted that supplementing these nutrients will most likely only have an effect in cases of deficiency or insufficiency.

3. Conclusion

Nutrients such as antioxidants, tryptophan rich protein, carbohydrate, melatonin, micronutrients and fruit can affect sleep [37,39,40]. Sleep can be promoted either by inhibiting wake-promoting mechanisms or by increasing sleep promoting factors through nutritional interventions [40]. Based on this review of the existing scientific literature, there appears to be considerable scope for further investigation of nutrition interventions designed to enhance sleep quality and quantity or promote general health, sleep health, training adaptations and/or recovery in both general and athletic populations.

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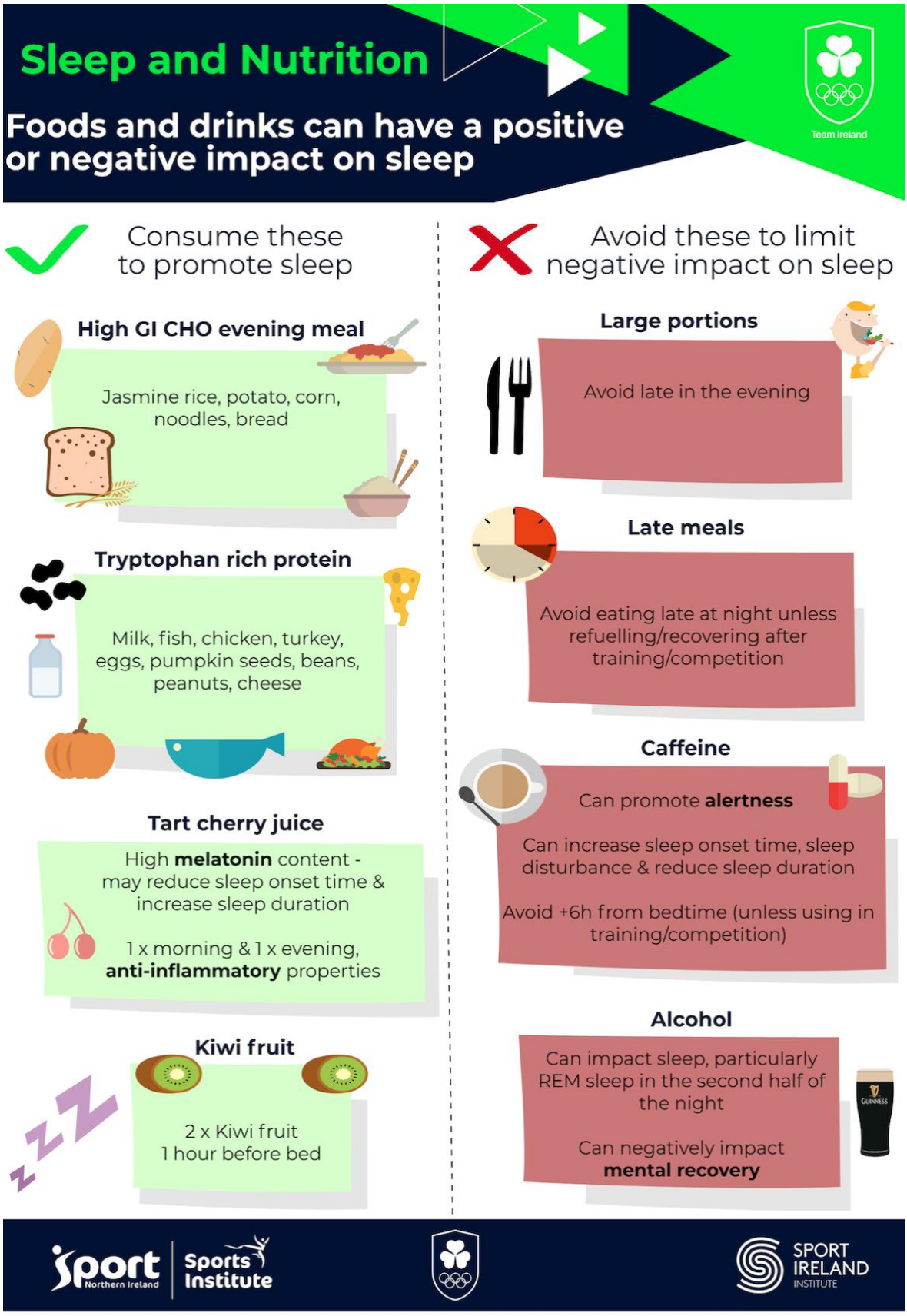
9.2 Infographics

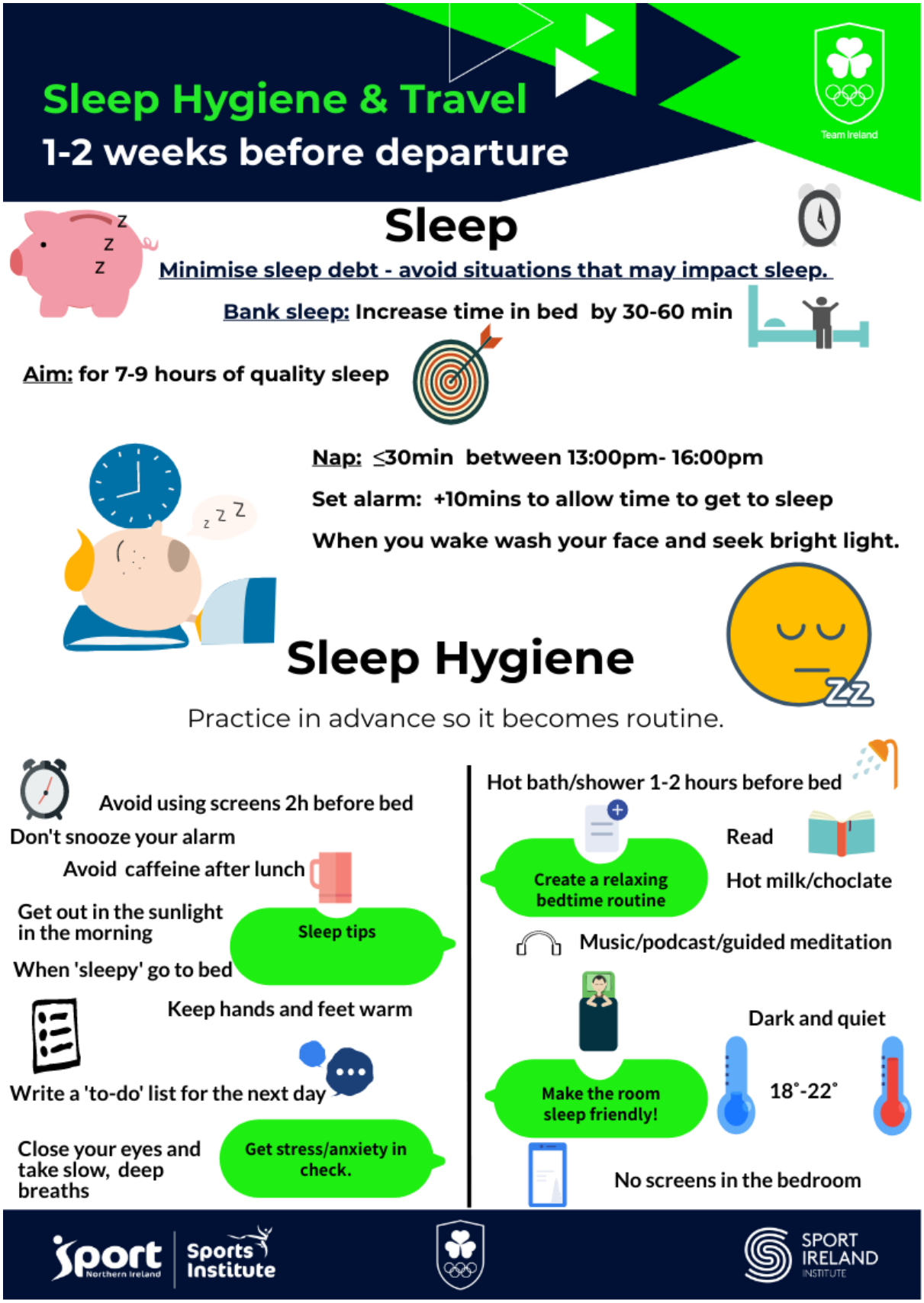
9.2.1 Sport Ireland Infographic – Sleep and Nutrition Interactions: Implications for athletes.

9.2.2 YLM Infographic – Sleep and Nutrition Interactions: Implications for athletes.

9.2.3 Olympic Federation of Ireland, Tokyo Olympic Games 2021 – Sleep and Nutrition.



9.2.4 *Olympic Federation of Ireland, Tokyo Olympic Games 2021 – Pre-travel and Sleep.*



9.3 Study 1 – Appendices

9.3.1 Study 1 – Participant Information Sheet

PARTICIPANT INFORMATION SHEET

The purpose of this Information Sheet is to provide you with sufficient information so that you can give your informed consent. It is very important that you read this document carefully, and raise any issues/questions you may have with the investigator.

Name of Researcher: Rónán Doherty

Name of Supervisor: Professor Jason Ellis

Project Title: Sleep, nutrition and athlete recovery.

1. **What is the purpose of this project?**

   Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery. The purpose of this study to investigate the sleep quality and quantity of athletes and their current recovery practices. This study aims to investigate: (i) the quality and quantity of sleep among elite athletes, (ii) the timing of sleep in elite athletes and (iii) recovery practices of elite athletes.

2. **Why am I eligible to take part?**

   You have been identified because you are an athlete with a team/sport and/or organisation which was targeted for this research and/or with whom I the researcher work. You are being invited to participate in this research because you are a member of an athletic group of interest with respect to sleep.

3. **What will I have to do?**

   This study requires you to complete an online questionnaire investigating your sleep quality and quantity of athletes and recovery practices.

4. **Will my participation involve any physical discomfort, psychological discomfort or embarrassment?**

   No.

5. **How will confidentiality be assured?**

   The research team have put in place a number of procedures to protect the confidentiality of participants. You will be assigned a participant identification number that will be used

to anonymise your data. Your name will not be associated with your data and only the research team will have access to any identifiable information.

6. **Who will have access to the information I provide?**

   Any information and data collected during this study will only be available to the research team identified in this Information Sheet. Should the research be presented or published in any form, the information will be generalised and anonymised. Your personal information or data will not be identifiable.

7. **How will my information be stored/used in the future?**

   Any paper records including questionnaires and consent forms will be kept in a locked filing cabinet. All electronic data will be securely stored on a password protected PC in a locked office. Data will be backed-up to a password protected portable hard-drive. All information and data collected during this study will be stored in accordance with the Data Protection Act.

   The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected.

8. **Has the project received appropriate ethical clearance?**

   Yes, this study has received full ethical approval from the Faculty of Life and Health Sciences Ethics Committee, Northumbria University.

9. **How can I withdraw from the project?**

   If you decide that you do not wish to take any further part in the study, you are able to withdraw at any time, without prejudice and without having to give any reason. Please inform any member of the research team (see contact details in section 11), as soon as possible, and they will facilitate your withdrawal and explain how your data will be treated. Having completed the study you are still free to withdraw your data. However, please contact a member of the research team within one month of your participation in the study. After this point it will not be possible to withdraw your individual data. All data are anonymised, your individual data will not be identifiable in any way.

10. **If I require additional information who should I contact?**

    If you have further questions or wish to withdraw from the study, please contact the researcher, Rónán Doherty (ronan.doherty@northumbria.ac.uk) or his supervisor, Professor Jason Ellis (jason.ellis@northumbria.ac.uk).

Thank you!

9.3.2 Study 1 – Informed Consent Form

INFORMED CONSENT FORM

Name of Researcher: Rónán Doherty

Name of Supervisor: Professor Jason Ellis

Project Title: Examining sleep as a moderator in the link between nutritional supplementation and oxidative stress in elite athletes.

I, the undersigned, confirm that (please tick box as appropriate):

1.	I have read and understood the information about the project, as provided in the Participant Information Sheet.	☐
2.	I have been given the opportunity to ask questions about the project and my participation.	☐
3.	I voluntarily agree to participate in the project.	☐
4.	I understand I can withdraw at any time without giving reasons and that I will not be penalised for withdrawing nor will I be questioned on why I have withdrawn.	☐
5.	The procedures regarding confidentiality have been clearly explained (e.g. use of names, pseudonyms, anonymisation of data, etc.) to me.	☐
7.	The use of the data in research, publications, sharing and archiving has been explained to me.	☐

I agree to the University of Northumbria at Newcastle recording and processing this information about me. I understand that this information will be used only for the purpose(s) set out in the information sheet supplied to me, and my consent is conditional upon the University complying with its duties and obligations under the Data Protection Act 1998.

Participant:

_____ _____ _____

Name of Participant Signature Date

Researcher:

_____ _____ _____

Name of Researcher Signature Date

9.3.3 Study 1 – Debrief Sheet

DEBRIEF FORM

Name of Researcher: Rónán Doherty

Name of Supervisor: Professor Jason Ellis

Project Title: Examining sleep as a moderator in the link between nutritional supplementation and oxidative stress in elite athletes.

11. Aims and expected findings of this project?

Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery. The purpose of this study was to investigate the sleep quality and quantity of athletes and their current recovery practices. The expected findings of this study will characterise normative sleep and recovery practices of athletes.

12. How will the data be used?

The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected.

13. Withdrawal from the project?

All data are anonymised, your individual data will not be identifiable in any way. However, if you decide that you do not wish for your data to be used in the study, you are able to withdraw at any time, without prejudice and without having to give any reason.

14. If you require any additional information or wish to register a complaint?

If you have further questions or wish to withdraw from the study, please contact the researcher, Rónán Doherty (ronan.doherty@northumbria.ac.uk) or the Faculty of Health and Life Sciences, Research Ethics Director (nick.neave@northumbria.ac.uk).

Thank you for participating in this study!

9.4 Study 2 Appendices

9.4.1 Study 2 – Participant Information Sheet

Northumbria University
NEWCASTLE

PARTICIPANT INFORMATION SHEET

The purpose of this Information Sheet is to provide you with sufficient information so that you can give your informed consent. It is very important that you read this document carefully, and raise any issues/questions you may have with the investigator.

Name of Researcher: Rónán Doherty **Name of Supervisor:** Professor Jason Ellis **Project Title:** Examining the sleep of elite athletes and coaches.

1. What is the purpose of this project?

During the build-up to the Olympic Games in Tokyo 2020, numerous test and warm-up events will take place in Asia. In the run up to the games athletes and coaches are keen to combat jet lag and minimise its impact on performance. The purpose of this study is to investigate the sleep quality and quantity of athletes and coaches. The expected findings of this study will characterise normative sleep of coaches and athletes and highlight the impact of eastward travel on their sleep.

2. Why am I eligible to take part?

You have been identified because you are an athlete or coach with a team/sport and/or organisation which was targeted for this research and/or with whom I the researcher work. You are being invited to participate in this research because you are a member of a group of interest with respect to sleep.

3. What will I have to do?

This study requires you to complete a daily online questionnaire and wear an activity monitor to investigate your sleep quality and quantity of athletes and recovery practices.

4. Will my participation involve any physical discomfort, psychological discomfort or embarrassment?

No.

5. How will confidentiality be assured?

The research team have put in place a number of procedures to protect the confidentiality of participants. You will be assigned a participant identification number that will be used to

anonymise your data. Your name will not be associated with your data and only the research team will have access to any identifiable information.

6. Who will have access to the information I provide?

Any information and data collected during this study will only be available to the research team identified in this information sheet. Should the research be presented or published in any form, the information will be generalised and anonymised. Your personal information or data will not be identifiable.

7. How will my information be stored/used in the future?

Any paper records including questionnaires and consent forms will be kept in a locked filing cabinet. All electronic data will be securely stored on a password protected PC in a locked office. Data will be backed-up to a password protected portable hard-drive. All information and data collected during this study will be stored in accordance with the Data Protection Act. The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected.

8. Has the project received appropriate ethical clearance?

Yes, this study has received full ethical approval from the Faculty of Life and Health Sciences Ethics Committee, Northumbria University.

9. How can I withdraw from the project?

If you decide that you do not wish to take any further part in the study, you are able to withdraw at any time, without prejudice and without having to give any reason. Please inform any member of the research team (see contact details in section 10), as soon as possible, and they will facilitate your withdrawal and explain how your data will be treated. Having completed the study you are still free to withdraw your data. However, please contact a member of the research team within one month of your participation in the study. All data are anonymised, your individual data will not be identifiable in any way.

10. If I require additional information who should I contact?

If you have further questions or wish to withdraw from the study, please contact the researcher, Rónán Doherty (ronan.doherty@northumbria.ac.uk) or his supervisor, Professor Jason Ellis (jason.ellis@northumbria.ac.uk).

Thank you!

9.4.2 Study 2 – Informed Consent Form

Northumbria University
NEWCASTLE

CONSENT FORM

Project Title: Examining the sleep of elite athletes and coaches.

Principal Investigator: Rónán Doherty

	please tick or initial where applicable
I have carefully read and understood the Participant Information Sheet.	☐
I have had an opportunity to ask questions and discuss this study and I have received satisfactory answers.	☐
I understand I am free to withdraw from the study at any time, without having to give a reason for withdrawing, and without prejudice.	☐
I agree to take part in this study.	☐
I also consent to the retention of this data under the condition that any subsequent use also be restricted to research projects that have gained ethical approval from Northumbria University.	☐

Signature of participant..... Date.....……..   (NAME IN BLOCK LETTERS).....……..
Signature of Parent / Guardian in the case of a minor   .....
Signature of researcher..... Date.....……..   (NAME IN BLOCK LETTERS).....……..

9.4.3 Study 2 – Debrief Sheet

Northumbria University NEWCASTLE

Participant code:

PARTICIPANT DEBRIEF SHEET

Name of Researcher: Rónán Doherty

Name of Supervisor (if relevant): Professor Jason Ellis

Project Title: Examining the sleep of elite athletes and coaches.

1. What was the purpose of the project?

During the build-up to the Olympic Games in Tokyo 2020, numerous test and warm-up events will take place in Asia. Athletes and coaches are keen to combat jet lag and minimise its impact on performance. The purpose of this study was to investigate the sleep quality and quantity of athletes and coaches. The expected findings of this study will characterise normative sleep of coaches and athletes and highlight the impact of eastward travel on their sleep.

2. How will I find out about the results?

The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected. Approximately 4 weeks after taking part, the researcher will e-mail you a general summary of your results.

3. If I change my mind and wish to withdraw the information I have provided, how do I do this?

The data obtained If you wish to withdraw your data then email (ronan.doherty@northumbria.ac.uk) within 1 month of taking part and given them the code number that was allocated to you (this can be found on your debrief sheet). After this time, it might not be possible to withdraw your data as it could already have been analysed.

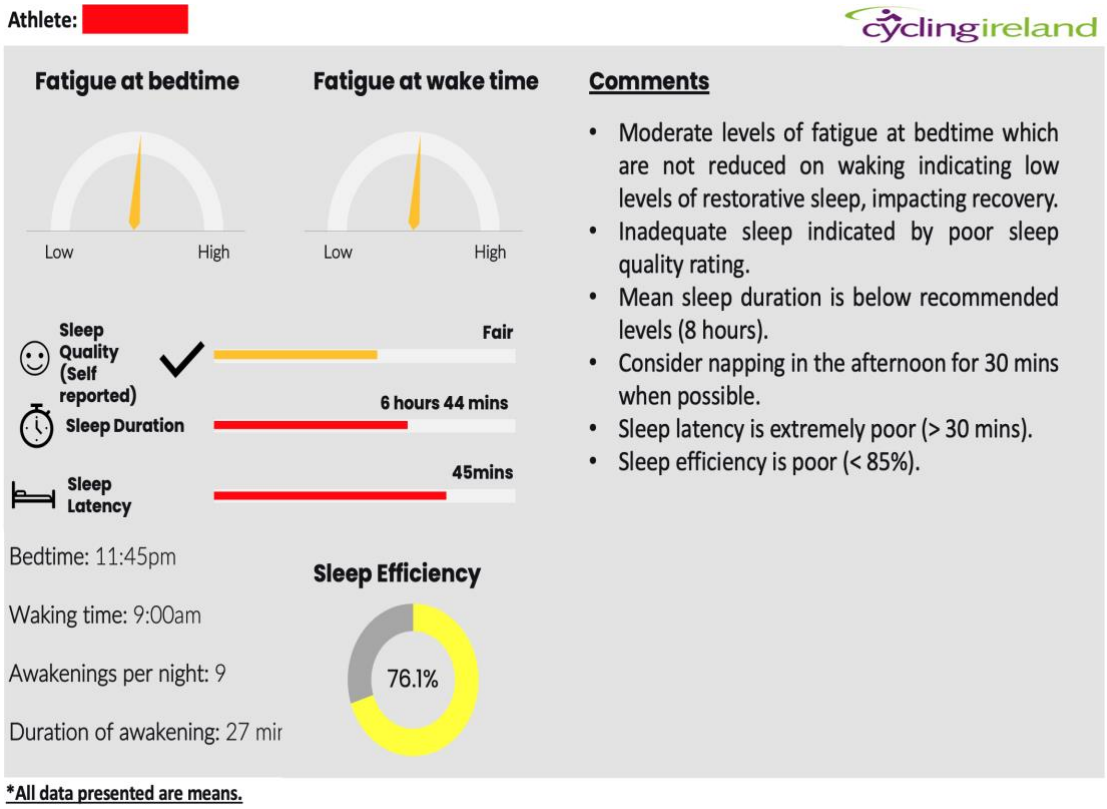
The data collected in this study may also be published in scientific journals or presented at conferences. Information and data gathered during this research study will only be available to the research team identified in the information sheet. Should the research be presented or published in any form, all data will be anonymous (i.e. your personal information or data will not be identifiable).

All information and data gathered during this research will be stored in line with the Data Protection Act and GDPR. The data will be destroyed in line with the guidelines following the conclusion of the study. If the research is published in a scientific journal it may be kept for longer before being destroyed. During that time the data may be used by members of the research team only for purposes appropriate to the research question, but at no point will your personal information or data be revealed. Insurance companies and employers will not be given any individual's personal information, nor any data provided by them, and nor will we allow access to the police, security services, social services, relatives or lawyers, unless forced to do so by the courts.

If you wish to receive feedback about the findings of this research study, then please contact the researcher at ronan.doherty@northumbria.ac.uk

This study and its protocol have received full ethical approval from Faculty of Health and Life Sciences Research Ethics Committee. If you require confirmation of this, or if you have any concerns or worries concerning this research, or if you wish to register a complaint, please contact the Chair of this Committee Nick Weave (nick.weave@northumbria.ac.uk) stating the title of the research project and the name of the researcher.

9.4.4 Study 2 Participant Report Example



9.5 Study 3 Appendices

9.5.1 Study 3 – Participant Information Sheet

Northumbria University
NEWCASTLE

PARTICIPANT INFORMATION SHEET

The purpose of this Information Sheet is to provide you with sufficient information so that you can give your informed consent. It is very important that you read this document carefully, and raise any issues/questions you may have with the investigator.

Name of Researcher: Rónán Doherty

Name of Supervisor: Professor Jason Ellis

Project Title: Examining the impact of kiwifruit supplementation on the sleep of elite athletes.

1. **What is the purpose of this project?**

   Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery. The purpose of this study to investigate the sleep quality and quantity of athletes and the impact of kiwifruit supplementation on their sleep.

2. **Why am I eligible to take part?**

   You have been identified because you are an athlete with a team/sport and/or organisation which was targeted for this research and/or with whom I the researcher work. You are being invited to participate in this research because you are a member of an athletic group of interest with respect to sleep.

3. **What will I have to do?**

   This study requires you to complete an online questionnaire investigating your sleep quality and quantity of athletes and recovery practices for 5 weeks. You will also be required to consume two kiwis per day, one hour before bed for the final 4 weeks of the study. You will have to purchase the kiwifruit and you will be reimbursed.

4. **Will my participation involve any physical discomfort, psychological discomfort or embarrassment?**

No.

5. **How will confidentiality be assured?**

   The research team have put in place a number of procedures to protect the confidentiality of participants. You will be assigned a participant identification number that will be used to anonymise your data. Your name will not be associated with your data and only the research team will have access to any identifiable information.

6. **Who will have access to the information I provide?**

   Any information and data collected during this study will only be available to the research team identified in this Information Sheet. Should the research be presented or published in any form, the information will be generalised and anonymised. Your personal information or data will not be identifiable.

7. **How will my information be stored/used in the future?**

   Any paper records including questionnaires and consent forms will be kept in a locked filing cabinet. All electronic data will be securely stored on a password protected PC in a locked office. Data will be backed-up to a password protected portable hard-drive. All information and data collected during this study will be stored in accordance with the Data Protection Act.

   The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected.

8. **Has the project received appropriate ethical clearance?**

   Yes, this study has received full ethical approval from the Faculty of Life and Health Sciences Ethics Committee, Northumbria University.

9. **How can I withdraw from the project?**

   If you decide that you do not wish to take any further part in the study, you are able to withdraw at any time, without prejudice and without having to give any reason. Please inform any member of the research team (see contact details in section 11), as soon as possible, and they will facilitate your withdrawal and explain how your data will be treated. Having completed the study you are still free to withdraw your data. However, please contact a member of the research team within one month of your participation

in the study. After this point it will not be possible to withdraw your individual data. All data are anonymised, your individual data will not be identifiable in any way.

10. If I require additional information who should I contact?

If you have further questions or wish to withdraw from the study, please contact the researcher, Rónán Doherty (ronan.doherty@northumbria.ac.uk) or his supervisor, Professor Jason Ellis (jason.ellis@northumbria.ac.uk).

Thank you!

9.5.2 Study 3 – Informed Consent Form

Northumbria University
NEWCASTLE

CONSENT FORM

Project Title: Examining the impact of kiwifruit supplementation on the sleep of elite athletes.

Principal Investigator: Rónán Doherty

	please tick or initial where applicable
I have carefully read and understood the Participant Information Sheet.	☐
I have had an opportunity to ask questions and discuss this study and I have received satisfactory answers.	☐
I understand I am free to withdraw from the study at any time, without having to give a reason for withdrawing, and without prejudice.	☐
I agree to take part in this study.	☐
I also consent to the retention of this data under the condition that any subsequent use also be restricted to research projects that have gained ethical approval from Northumbria University.	☐

Signature of participant..... Date.....……. (NAME IN BLOCK LETTERS).....…….
Signature of Parent / Guardian in the case of a minor .....
Signature of researcher..... Date.....……. (NAME IN BLOCK LETTERS).....…….

9.5.3 Study 3 – Debrief Sheet

Northumbria University
NEWCASTLE

Participant code:

PARTICIPANT DEBRIEF

Name of Researcher: Rónán Doherty

Name of Supervisor (if relevant): Professor Jason Ellis

Project Title: Examining the impact kiwifruit supplementation on the sleep of elite athletes.

1. What was the purpose of the project?

During the build-up to the Olympic Games in Tokyo 2020, numerous test and warm-up events will take place in Asia. Athletes and coaches are keen to combat jet lag and minimise its impact on performance. The purpose of this study was to investigate the sleep quality and quantity of athletes and coaches. The expected findings of this study will characterise normative sleep of and highlight the impact of kiwifruit supplementation on their sleep.

2. How will I find out about the results?

The data obtained will be analysed and may be used by the research team for purposes appropriate to the research questions or for scientific publications, but at no point will any personal information or data be unprotected. Approximately 4 weeks after taking part, the researcher will e-mail you a general summary of your results.

3. If I change my mind and wish to withdraw the information I have provided, how do I do this?

The data obtained If you wish to withdraw your data then email (ronan.doherty@northumbria.ac.uk) within 1 month of taking part and given them the code number that was allocated to you (this can be found on your debrief sheet). After this time, it might not be possible to withdraw your data as it could already have been analysed.

The data collected in this study may also be published in scientific journals or presented at conferences. Information and data gathered during this research study will only be available to the research team identified in the information sheet. Should the research be presented or published in any form, all data will be anonymous (i.e. your personal information or data will not be identifiable).

All information and data gathered during this research will be stored in line with the Data Protection Act and GDPR. The data will be destroyed in line with the guidelines following the conclusion of the study. If the research is published in a scientific journal it may be kept for longer before being destroyed. During that time the data may be used by members of the research team only for purposes appropriate to the research question, but at no point will your personal information or data be revealed. Insurance companies and employers will not be given any individual's personal information, nor any data provided by them, and nor will we allow access to the police, security services, social services, relatives or lawyers, unless forced to do so by the courts.

If you wish to receive feedback about the findings of this research study, then please contact the researcher at ronan.doherty@northumbria.ac.uk

This study and its protocol have received full ethical approval from Faculty of Health and Life Sciences Research Ethics Committee. If you require confirmation of this, or if you have any concerns or worries concerning this research, or if you wish to register a complaint, please contact the Chair of this Committee Nick Weave (nick.neave@northumbria.ac.uk) stating the title of the research project and the name of the researcher.

9.6 The sleep and recovery practices of athletes.

nutrients

MDPI

Article

The Sleep and Recovery Practices of Athletes

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Abstract: Background: Athletes maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery and performance. Optimal TST is subject to individual variance. However, 7–9 h sleep is recommended for adults, while elite athletes may require more quality sleep than non-athletes. Methods: A total of 338 (elite n = 115, 74 males and 41 females, aged 23.44 ± 4.91 years; and sub-elite n = 223, 129 males and 94 females aged 25.71 ± 6.27) athletes were recruited from a variety of team and individual sports to complete a battery of previously validated and reliable widely used questionnaires assessing sleep, recovery and nutritional practices. Results: Poor sleep was reported by both the elite and sub-elite athlete groups (i.e., global PSQI score ≥5—elite 64% [n = 74]; sub-elite 65% [n = 146]) and there was a significant difference in sport-specific recovery practices (3.22 ± 0.90 vs. 2.91 ± 0.90; $p < 0.001$). Relatively high levels of fatigue (2.52 ± 1.32), stress (1.7 ± 1.31) and pain (50%, n = 169) were reported in both groups. A range of supplements were used regularly by athletes in both groups; indeed, whey (elite n = 22 and sub-elite n = 48) was the most commonly used recovery supplement in both groups. Higher alcohol consumption was observed in the sub-elite athletes (12%, n = 26) and they tended to consume more units of alcohol per drinking bout. Conclusion: There is a need for athletes to receive individualised support and education regarding their sleep and recovery practices.

Keywords: sleep; recovery; nutrition; alcohol; athletes

1. Introduction

Post-exercise recovery is vital for all athletes and the balance between training stress and physical recovery must be managed to maximise the adaptation from, and performance in,

subsequent training sessions or competitions [1,2]. The repetitive demanding nature of a competitive season can test athletes' physiological and psychological capacity. Athletes must maintain a balance between stress and recovery and adopt recovery modalities that manage fatigue and enhance recovery and performance in subsequent training/competition [1]. The regulation of performance during exercise has increasingly been interpreted as a cohesive, multifaceted process involving both the central nervous system (CNS) and the peripheral nervous system (PNS) [3,4]. While there is debate on whether the regulation of exercise performance is derived primarily from the CNS or PNS [5] and whether the regulation is conscious [6] or anticipatory [7], changing CNS drive and motor unit recruitment is widely considered to be associated with fatigue (i.e., reduced physical and mental capacity) [3]. In contrast, physical fatigue has many potential drivers (dehydration, glycogen depletion, muscle damage and mental fatigue), and recovery of muscle function is predominantly a matter of reversing the main causes of fatigue. Sleep deprivation (<7 h) increases circulating stress hormones (e.g., cortisol) [8]; decreases the regeneration of carbohydrate stores (i.e., glycogen) [9]; deregulates appetite and impacts on energy expenditure [10]; increases catabolism and reduces anabolism, impacting the rate of muscle repair (MPS) [11,12]. Therefore, sleep plays a key role in facilitation of post-exercise recovery or the reduction in fatigue and the reversal of the processes that lead to fatigue [13].

Athletes experience stress for various reasons (e.g., training, competition, travel and lifestyle) including periods of both acute and residual fatigue due to heavy training and competition schedules [14]. For example, field-based team sports are characterised by repeated bouts of intermittent activity (sprinting) with short rest periods, representing high physiological stress [15], neuromuscular stress [16,17] and high rates of perceived exertion (i.e., how hard exercise seems) [18]. Further, individual endurance athletes experience fatigue due to prolonged activity, resulting in glycogen depletion, thermal stress and/or dehydration [19]. Relative stress is accumulated when successive bouts of training are combined with suboptimal recovery (under-recovery) impacting subsequent performance in training and competition [20]. It has been suggested that decreasing the natural timeframe of the bodies' regenerative processes via recovery strategies is vital for performance [21]. Such recovery strategies can be divided into physiological strategies (e.g., sleep, cold water immersion, cryotherapy, contrast therapy, massage and compression), pharmacological (e.g., non-steroidal anti-inflammatory drugs [NSAIDs]) and nutritional (e.g., nutrient timing, composition and supplementation) [22]. However, it must be noted that some research has suggested that interfering with the body's natural recovery processes, particularly inflammatory responses and OS, could reduce training adaptations [23]. A recent review addressed these concerns in relation to the application of nutritional strategies to reduce muscle damage [24].

Sleep has previously been self-reported as the most important recovery modality utilised by both elite and sub-elite athletes [1,25,26]. Furthermore, it has been suggested that sleep was a new frontier in performance enhancement for athletes [27]. Sleep has a restorative effect on the immune system and the endocrine system [28–30], facilitates the recovery of the nervous and metabolic cost of the waking state and has an integral role in cognitive function [31]. The relationship between sleep, nutrition and recovery is an emerging area of interest [26,32–45]. Sleep has two basic states—non-rapid eye movement sleep (NREM) and rapid eye movement (REM) sleep. NREM is subdivided into three stages based on a continuum from light sleep (Stage N1 and N2) to deep sleep (Stage N3). It has been hypothesised that sleep, especially slow-wave sleep (Stage N3), is vital for physical recovery, due to the relationship with growth hormone release [44,46]. The National Sleep Foundation has proposed 12 indicators of sleep quality including 4 sleep continuity variables (sleep latency, awakenings >5 min, wake after sleep onset and sleep efficiency), 5 sleep architecture variables (REM sleep, N1 sleep, N2 sleep, N3 sleep and arousals) and 3 nap-related variables (naps per 24 h, nap duration and days per week with at least one nap) [47]. Sleep can be considered adequate when there is no daytime sleepiness or dysfunction.

For sleep to have a restorative effect on the body, it must be of adequate duration, quality, and appropriately timed [38,48]. The National Sleep Foundation has produced guidelines regarding

sleep duration for adolescents (recommended 8–10 h), adults (recommended 7–9 h), and older adults (7–8 h) [48]. It has been argued that elite athletes may require more quality sleep than non-athletes [49]. It has recently been suggested that a one-size-fits-all sleep recommendation (7–9 h) may be inappropriate for athlete performance and health and an individual approach should be adapted including an assessment of perceived sleep needs [50].

Sleep inadequacy is common in athletes and can be attributed to the lack of an appropriate sleep routine due to changing training schedules, timetables and other sleep-incompatible behaviours, e.g., late night blue-light exposure [26,50]. Previous research has reported sleep durations <7 h [51], long sleep onset latency [26,52], daytime sleepiness [53], and daytime fatigue [54]. Studies investigating sleep quality in elite athletes have demonstrated that 50–80% experience sleep disturbance and 22–26% experience highly disturbed sleep [37,53,55]. Irregular sleep–wake patterns influence the homeostatic and circadian regulation of sleep, which reduces both sleep quality and quantity [56]. For athletes, post-completion routines and heightened arousal (i.e., medical care, recovery strategies, meals, media commitments and travel) can lead to later bedtimes, which can adversely affect sleep quality and quantity. Reduced sleep is associated with increased catabolic and reduced anabolic hormones, which results in impaired muscle protein synthesis [12], potentially blunting training adaptations and recovery.

Sleep disorders are identified by a wide range of symptoms that impact health and quality of life [57], cognitive performance [58] and physical performance [25,59]. Over 80 sleep disorders are listed in the third edition of the International Classification of Sleep Disorders (ISCD-3) [60]. The ICSD-3 includes seven major categories of sleep disorders: insomnia, sleep-related breathing disorders, central disorders of hypersomnolence, circadian rhythm sleep wake disorders (CRSWDs), sleep-related movement disorders, parasomnias and other sleep disorders [60]. In the general population, the most common sleep disorders are obstructive sleep apnoea (OSA), insomnia and restless legs syndrome (RLS) [61]. Sleep-related breathing disorders are characterised by breathing issues during sleep [62]. OSA is a frequent condition characterised by repeated episodes of partial or complete reduction in breathing activity during sleep [63]. Insomnia is characterised by difficulty falling asleep, staying asleep, waking too early with daytime symptoms of fatigue, resistance to going to bed and/or difficulty sleeping without intervention occurring at least 3 times per week over a period of one month ([64,65]. Central disorders of hypersomnolence are typified by excessive daytime sleepiness that cannot be attributed to another sleep disorder [60]. CRSWDs are chronic (≥3 months) patterns of sleep–wake disruption caused by an alteration to the endogenous circadian or desychronisation of the circadian rhythm and the sleep–wake schedule, causing sleep–wake disturbance and distress or impairment [60]. Sleep-related movement disorders may result from an unpleasant crawling, deep-aching sensation in the legs or arms that is relieved through movement [66]. Parasomnias are undesirable movements or behaviours that occur during sleep, e.g., sleep walking, sleep talking, night terrors and REM sleep behaviour disorder [66]. Other sleep disorders include all sleep disorders that do not meet the criteria for another sleep disorder classifications [62].

Polysomnography (PSG) is the 'gold-standard' method of sleep assessment and records sleep continuity, sleep architecture and REM sleep. A common global approach to the assessment of sleep quality is the use of self-report ratings reflecting an individual's satisfaction with their sleep [47,67]. Sleep continuity is commonly assessed using sleep diaries and measures include time the subject went to bed, time the subject tried to initiate sleep, the length of time from turning off the lights until sleep onset (sleep onset latency), number and duration of awakenings, the degree of sleep maintenance during the night (sleep efficiency or the ratio of wake time to time in bed; awake time after sleep onset) sleep duration (total sleep time), time the subject woke up, time the subject got out of bed and sleep quality (subjective rating of sleep) [68,69].

Actigraphy is also used to assess sleep, regularly in combination with sleep diaries. Actigraphy involves wearing a small monitor (usually on the non-dominant wrist) which records body movement, high levels of activity are used as a measure of wakefulness and low levels of activity are classified as sleep [69]. Activity monitors record movement as a function of time [70], typically a tri-

axial accelerometer is used to determine sleep/wake based on a proprietary algorithm [71]. A limitation of actigraphy is that all activity is recorded as waking unless the sleep diaries show an attempt to sleep (i.e., lying down trying to sleep) and the activity counts are low enough to indicate the subject is stationary [32]. However, actigraphy has been shown to be reliable and valid in relation to PSG for general measures of sleep [72,73].

Athletes' schedules can negatively impact their sleep and recovery [51,52], and the repetitive demanding nature of a competitive season can also test athletes' physiological and psychological capacity, reinforcing the athletes' need for quality sleep [74-77]. Actigraphy based sleep assessments reveal suboptimal sleep in athletes, i.e., low TST and high WASO, causing resultant low sleep efficiency [27, 30], which improves following a rest day [78]. However, the athletes' experience of suboptimal sleep remains unclear as sleep need varies between individuals; some may report poor sleep while objective measures indicate sufficient sleep [32]. Therefore, subjective measures of sleep quality, quantity and timing are a valuable addition to objective sleep assessments. Combined subjective markers of sleep (e.g., TST, time in bed, sleep efficiency, sleep quality and sleep onset latency) can highlight the sleep need and recovery status of athletes and identify areas to be addressed in terms of sleep optimisation. Moreover, the use of subjective measures within an athletic population allows the assessment of large cohorts of athletes that are difficult to access, i.e., elite athletes.

Animal models have demonstrated that nutrients such as glucose, amino acids, sodium, ethanol and caffeine, as well as the timing of meals can affect circadian rhythms [79]. Neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), orexin, dopamine, melanin-concentrating hormone, galanin, noradrenaline and histamine that are involved in the sleep–wake cycle [80] are affected by nutrition. In terms of recovery, the adaptive response to training is dictated by a number of variables: duration, intensity, frequency and type of exercise, in combination with timing, quality and quantity of nutrition both pre- and post-exercise [81]. Recovery can be maximised by optimal nutrition practices or reduced by suboptimal nutrition practices. Contemporary research has demonstrated the pivotal role of both macronutrient and micronutrient availability in regulating skeletal muscle adaptations to exercise [81–83]. It is important to characterise the sleep quality and quantity of sub-elite and elite athletes and recovery practices. This study aimed to investigate: (i) the quality, quantity and timing of sleep among sub-elite and elite athletes; (ii) the recovery/stress balance of sub-elite and elite athletes; and (iii) the supplement use and alcohol intake of sub-elite and elite athletes. This study also aimed to investigate the difference between elite and sub-elite athletes in terms of their subjective sleep, recovery and nutritional practices. It was hypothesised that the sleep, recovery and nutrition practices of elite athletes would be superior to those of sub-elite athletes.

2. Materials and Methods

2.1. Participants

A sample (n = 338) comprising elite (n = 115; male n = 74 and female n = 41) and sub-elite (n = 223; male 129 and female 94) athletes were recruited from both Ireland and the United Kingdom (see Table 1). The elite athletes were recruited directly through Sport Ireland and the national governing bodies (NGBs) of each sport within Ireland and the United Kingdom. The sub-elite athletes were recruited via social media and the researcher's network within high-performance sport. In line with Swann et al. [84], elite athletes were defined as: (a) currently receiving support/funding through the international carding scheme and/or (b) members of a national/professional team or a recruitment/academy squad and/or (c) nationally ranked in their sport. Sub-elite athletes were defined as those competing at a regional, university and/or national level of organised sport that trained and/or competed for a combined minimum of 400 min per week. Athletes, at either level, were excluded if they were (i) aged <18 years, (ii) training and competing for <400 min per week or (iii) reported a sleep disorder.

2.2. Procedure

All eligible athletes were invited to take part in an online survey. All procedures were approved by the research ethics committee of the Faculty of Health and Life Sciences, Northumbria University (date of approval 2 July 2019; Submission ID: 17406). After reading the participant information sheet, participants were invited to provide informed consent and then completed an online survey on Qualtrics[xm] which consisted of a battery of previously validated and reliable widely used questionnaires assessing sleep, recovery and nutritional practices. Following completion of the survey, participants received a debrief sheet with details of how they could contact the researcher if they wished to receive feedback from the survey.

2.3. Measures

In the initial section of the survey, the participants completed demographic data. Participants recorded their gender, age, body mass (kg), height (cm), sport, athlete type (elite or sub-elite), phase of season (pre-season, competition or off-season), normal training time (before 8 am, 8 am to 5 pm and after 5 pm) and training/competition duration per week (mins).

2.3.1. EuroQoL (EQ-5D-5L)

The EQ-5D-5L is a self-report measure of health status as defined across five dimensions—mobility, self-care, activity, pain and depression/anxiety—with one question per dimension. Each dimension is scored on a 5-point Likert scale (0 = No problem to 5 = Severe problem) [85]. The EQ-5D-5L also includes a visual analogue scale on which respondents are instructed to rate their perceived current health state (0–100). The EQ-5D-5L has capacity to discriminate between slight, moderate and severe issues within each domain compared to previous versions [86].

2.3.2. Pittsburgh Sleep Quality Index (PSQI)

The PSQI is a self-report measure of sleep quality [62]. The PSQI consists of 19 items grouped into seven component scores (0–3) which are equally weighted. Although overall global scores (GPSQI) are calculated by summing the seven components (range 0–21, with higher scores indicating poorer sleep quality) the component scores provide subscale ratings of: (i) subjective sleep quality, (ii) sleep latency, (iii) TST, (iv) sleep efficiency, (v) sleep disturbances, (vi) use of sleep medication and (vii) daytime dysfunction [63]. Global scores > 5 are generally used to indicate poor sleep quality (63). The PSQI has demonstrated a diagnostic sensitivity (89.6%) and specificity (86.5%) in distinguishing between 'good' and poor' sleepers [87]. However, more conservative scores of ≥8 have been used in athletes to indicate poor sleep, potentially due to the increased sleep needs in this population [55]. Although the empirical discussion around the PSQI cut-offs for athletes is ongoing [38,55], given that athletes often strive for marginal gains in their performance, which can be facilitated through optimised sleep, the identification of both 'poor' and 'moderate' sleep quality is warranted [43], hence the standard cut-off (≥5) was employed.

2.3.3. Epworth Sleepiness Scale (ESS)

The ESS is an eight-item self-report measure of general daytime sleepiness [89]. Respondents report their daytime sleepiness in particular situations on a Likert scale (0 = Would never doze to 4 = High chance of dozing). Scores are summed to yield a global ESS score (0–24). The EES global score is indicative of daytime sleepiness [90]. Higher scores indicate greater sleepiness, scores > 10 suggest excessive daytime sleepiness [89]. In general ESS scores are interpreted in terms of daytime sleepiness as follows: 0–5 low normal, 6–10 higher normal, 11–12 mild excessive, 13–15 moderate excessive and 16–24 severe [65].

2.3.4. The Recovery Stress Questionnaire for Athletes (RESTQ Sport)

The RESTQ-Sport is a 52-item self-report measure of general stress and recovery levels of athletes [91]. The RESTQ-Sport consists of seven general stress components with two items per scale (general stress, emotional stress, social stress, conflicts/pressure, fatigue, a lack of energy, and physical complaints), five general recovery components with two items per scale (success, social recovery, physical recovery, general well-being, and sleep quality), three sport-specific stress components with four items per scale (disturbed breaks, burnout/emotional exhaustion, and fitness/injury) and four sport-specific recovery components with four items per scale (fitness/being in shape, burnout/personal accomplishments, self-efficacy, and self-regulation) [91]. Sub-scale item mean scores can be combined to give a total score for each of the four major sub-scales (i.e., general stress, general recovery, sport-specific stress and sport-specific recovery). Each item is scored on a Likert scale (0 = Never to 6 = Always) based on how often the respondent engaged in a specified activity over the previous three days/nights, with a response of 0 indicating never having experienced the feeling and 6 indicating always experiencing the associated feeling. High scores on stress scales indicate a high level of stress, while high scores on the recovery scales indicate a high level of recovery [91].

2.3.5. Athlete Morningness/Eveningness Questionnaire (AMES)

The AMES, which is based on the Horne–Östberg morningness/eveningness questionnaire [92], is a four-item questionnaire used to classify an athlete's chronotype in terms of self-identification as being a morning or evening type, preferred sleep/wake phase and preferred competition and training time [93]. The AMES provides a global score which is used to categorise chronotype: extreme evening type (10–12), moderate evening type (13–17), mid-range type (18–23), moderate morning type (24–28) and extreme morning type (29–31) [55].

2.3.6. Consensus Sleep Diary—Core (CSD-C)

Participants were instructed to complete the CSD-C for two nights (1 'training/competition' day and 1 'rest' day). The CSD is a standardised sleep diary developed for use in both research and clinical settings [68]. The CSD-C included 8 items, e.g., bed time, time it took to fall asleep, number of awakenings, duration of awakenings, time of final awakening, time the respondent got out of bed, and a Likert scale self-report rating of sleep quality [94]. There was also a comments section where participants could record specific comments about each night's sleep (i.e., 1 training/competition day and 1 rest day). The data collected were then used to compute indices of sleep continuity such as total time in bed (TIB), total sleep time (TST, sleep onset latency (SOL; time from lights out to N1), wakefulness after sleep onset (WASO; amount of time awake after sleep onset), number of awakenings (NoA) and sleep efficiency (SE; ratio of TST:TIB) [94].

2.4. Supplementation

All participants were instructed to complete questions relating to supplement use (name, dose, frequency and reason for use) on both training/competition days and rest days. Athletes also reported their alcohol consumption (number of drinking sessions and unit consumption per session) in the last month prior to completion of the questionnaire.

2.5. Data Analysis

All data were analysed using the Statistical Package for the Social Sciences (SPSS Version 25, IBM Corporation) and Jamovi (Version 1.8.16). Frequency distribution and descriptive statistics were used to present findings [95]. All data were presented as the mean ± standard deviation, and/or frequency. The differences between the groups for athlete type were explored using independent-samples t-tests, chi square tests, Mann–Whitney U and one-way ANOVA [95].

3. Results

3.1. Participant Characteristics

A total of 338 (elite *n* = 115 and sub-elite *n* = 223) athletes were recruited from a variety of team and individual sports (see Tables 1 and 2.). The sample consisted of both male (*n* = 203; ~60%) and female (*n* = 135; ~40%) athletes.

Table 1. Participant characteristics (mean ± SD).

	All (*n* = 338)	**Elite (*n* = 115)**	**Sub-elite (*n* = 223)**	**t/x² Value**
Gender	Male *n* = 203; Female *n* = 135	Male *n* = 74; Female *n* = 41	Male *n* = 129; Female *n* = 94	$X^2 = 1.72$
Age *	24.94 ± 5.93	23.44 ± 4.91	25.71 ± 6.27	$t = 3.384$
Body mass (kg)	72.95 ± 13.26	73.95 ± 12.55	72.44 ± 13.61	$t = -0.995$
Height (cm)	175.60 ± 9.70	176.6 ± 8.78	175.08 ± 10.12	$t = -1.361$
Training (mins·wk) *	675.12 ± 306.59	801.35 ± 338.81	610.02 ± 266.90	$t = -5.682$

* Statistically significant difference.

A chi square analysis demonstrated no significant differences between the groups for gender ($X^2[1, n = 338] = 1.72$, $p = 0.189$). While there were statistically significant differences between the groups for age (elite 23.44 ± 4.91 years and sub-elite 25.71 ± 6.27 years; $t(336) = 3.38$; $p = 0.001$) and minutes trained per week (elite 801.35 ± 338.81 and sub-elite 610.02 ± 266.90; $t(336) = -5.68$; $p \leq 0.001$). An independent-samples t-test indicated no significant differences between the groups in terms of height, body mass and normal training time (time of day when training occurred) (see Table 1).

Table 2. Participant breakdown.

Sport	**All**	**Elite *n* = 115**	**Sub-elite *n* = 223**
Athletics	64	10	54
Boxing	12	11	1
Gaelic games	89	26	63
Hockey	10	9	1
Rowing	29	8	21
Rugby	20	8	12
Sailing	4	3	1
Soccer	31	10	21
Swimming	8	4	4
Other	71	26	45

Chi square analyses demonstrated a statistically significant difference between the groups for sport ($X^2[9, n = 338] = 1.72$, $p \leq 0.001$). There were no statistically significant differences between the groups for phase of season: pre-season (elite *n* = 31; sub-elite *n* = 57), competition (elite *n* = 65; sub-elite *n* = 115), off-season (elite *n* = 19; sub-elite *n* = 51) ($X^2[2, n = 338] = 1.88$, $p = 0.39$). There were statistically significant differences between the groups for normal training time: before 8 am (elite *n* = 8 and sub-elite *n* = 25), between 8 am and 5 pm (elite *n* = 50; sub-elite *n* = 58), and after 5 pm (elite *n* = 57; sub-elite *n* = 140) ($X^2[2, n = 338] = 10.9$, $p \leq 0.001$).

3.1.1. EuroQoL

There was no statistically significant difference between the groups for their perceived general health rating (0–100) with the elite athlete group reporting slightly higher levels of general health than the sub-elite athlete group (83.05 ± 13.65 vs. 81.05 ± 12.57; t = −1.37; $p = 0.172$). There were no statistically significant differences between the groups in terms of each of the domains of the quality of life measure (see Table 3). Slight to severe problems with mobility were reported by 19% (*n* = 65) of participants (elite *n* = 19 [17%]; sub-elite *n* = 46 [21%]). Some issues regarding the completion of usual activities (e.g., work, study, training, housework, family or leisure activities) were reported by

19% (*n* = 64) of participants (elite *n* = 23 [20%]; sub-elite *n* = 41 [18%]). Issues with self-care were not evident within the athletes as slight to moderate issues were reported by 3% of participants (elite *n* = 2 [2%]; sub-elite *n* = 9 [4%]). Pain was reported by 50% (*n* = 169) of participants (elite *n* = 53 [46%]; sub-elite *n* = 116 [52%]). Anxiety/depression was reported by 34% (*n* = 116) of participants (elite *n* = 43 [37%]; sub-elite *n* = 73 [33%]).

Table 3. Athlete responses to the EuroQOL.

		None	Slight	Moderate	Severe	Extreme
Mobility	Elite	96	14	5		
	Sub-elite	177	42	2	1	1
Self-care	Elite	113	1	1		
	Sub-elite	214	7	2		
Usual activities	Elite	92	18	3	1	1
	Sub-elite	182	33	8		
Pain	Elite	62	47	6		
	Sub-elite	107	102	14		
Anxiety/Depression	Elite	72	33	8	2	
	Sub-elite	150	58	13	2	

3.1.2. Pittsburgh Sleep Quality Index

An independent-samples t-test was used to compare PSQI data for the elite and sub-elite athlete groups. A statistically significant difference was observed between the groups for PSQI habitual sleep efficiency % (elite 88.62 ± 8.84 vs. sub-elite 86.55 ± 9.09; $t = -2.01$; $p = 0.046$). While no other statistically significant differences were observed, the majority of athletes (64%; *n* = 220) were classified as poor sleepers (i.e., global PSQI score ≥5—elite 64% [*n* = 74]; sub-elite 65% [*n* = 146]). Overall self-reported sleep quality did not reflect this as the athletes rated their sleep quality as very good (elite *n* = 19 [17%]; sub-elite *n* = 45 [20%]), fairly good (elite *n* = 68 [59%]; sub-elite *n* = 123 [55%]), fairly bad (elite *n* = 26 [23%]; sub-elite *n* = 50 [22%]) and poor (elite *n* = 2 [1%]; sub-elite *n* = 5 [2%]). Mean total sleep time (hours) varied between the elite athlete (7.58 ± 1.06; range 5–10 h) and the sub-elite athlete groups (7.35 ± 1.05; range 4–10 h) but this was not statistically significant. The athletes reported total sleep time ≤6 h (elite *n* = 16 [14%]; sub-elite *n* = 43 [19%]), 7 h (elite *n* = 38 [33%]; sub-elite *n* = 80 [36%]), 8 h (elite *n* = 39 [34%]; sub-elite *n* = 70 [32%]) and 9 h (elite *n* = 22 [19%]; sub-elite *n* = 30 [13%]). The athletes' responses to the PSQI are summarised in Table 3. The athletes reported total time in bed 8 h (elite *n* = 53 [46%]; sub-elite *n* = 109 [49%]), 9–10 h (elite *n* = 50 [44%]; sub-elite *n* = 110 [49%]) and 11–12 h (elite *n* = 12 [10%]; sub-elite *n* = 4 [2%]).

The reasons reported for poor sleep quality were not getting to sleep within 30 min, waking during the night or early morning, waking to use the bathroom and feeling too hot in bed (see Table 3). The feeling of a lack of enthusiasm for general tasks at least once per week was reported by 44% (*n* = 51) of the elite group and 41% (*n* = 92) of the sub-elite group. The use of sleep medication was low in both groups, with 5% (*n* = 6) of the elite group and 7% (*n* = 16) of the sub-elite group using medication on a weekly basis (see Table 4).

Table 4. Athlete responses to the PSQI.

		Not during the Last Month	Less than Once per Week	Once or Twice per Week	Three or More Times per Week
Cannot get to sleep within 30 min	Elite	40	24	27	34
	Sub-elite	99	56	82	48
Wake up in the middle of the night or early morning	Elite	30	24	27	34
	Sub-elite	37	56	82	48
Have to get up to use the bathroom	Elite	38	24	29	24
	Sub-elite	63	76	44	40
Cannot breathe comfortably	Elite	101	11	1	2
	Sub-elite	192	16	10	5
Cough or snore loudly	Elite	88	14	5	8
	Sub-elite	167	33	16	7
Feel too cold	Elite	79	27	7	2
	Sub-elite	160	41	19	3
Feel too hot	Elite	54	32	26	3
	Sub-elite	82	77	54	10
Have bad dreams	Elite	63	34	16	2
	Sub-elite	114	75	27	7
Have pain	Elite	81	22	11	1
	Sub-elite	152	48	19	4
Other reasons	Elite	101	9	3	2
	Sub-elite	180	27	9	7
Problems staying awake	Elite	66	29	13	7
	Sub-elite	125	66	27	5
Lack of enthusiasm	Elite	35	29	37	14
	Sub-elite	50	81	69	23
Use of sleep medication	Elite	104	5	3	3
	Sub-elite	189	18	7	9

3.2. Epworth Sleepiness Scale

An independent-samples t-test demonstrated no significant differences between the elite and sub-elite athlete groups for ESS scores ($p > 0.05$). A chi square test highlighted no significant difference between the groups' ESS classification (X2[20, $n = 338$] = 21.1, $p = 0.391$). Approximately 21% ($n = 70$) of athletes (elite $n = 25$; 22% and sub-elite $n = 45$; 20%) reported clinically significant excessive daytime sleepiness (ESS total score ≥10) (see Table 5).

Table 5. ESS classification.

Classification (ESS Score)	Elite ($n = 115$)	Sub-elite ($n = 223$)
Low Normal (0–5)	53	114
Higher Normal (6–10)	45	70
Mild Excessive (11–12)	6	20

Moderate Excessive (13–15)	8	14
Severe (16–24)	3	5

3.3. Recovery Stress Questionnaire

An independent-samples t-test highlighted significant differences between the elite and sub-elite athlete groups for recovery, i.e., the sport-specific recovery scale (3.22 ± 0.90 vs. 2.91 ± 0.90; t (−2.984); $p < 0.001$). While no statistically significant differences were observed for the general stress, general recovery and sport-specific stress subscales. Recovery stress scale scores were similar in both the elite and sub-elite groups with similar scores observed for the general stress scale (1.96 ± 0.91 vs. 2.01 ± 0.86), general recovery scale (2.97 ± 0.79 vs. 2.97 ± 0.77) and sport-specific stress scale (1.97 ± 0.87 vs. 1.99 ± 0.85).

An independent-samples t-test displayed no statistically significant differences between the groups for the majority of the subscales with both groups recording similar scores (see Table 6). However, significant differences between the groups were observed for the following sport-specific recovery subscales: being in shape (3.22 ± 1.08 vs. 2.90 ± 1.04; $t = -2.66$; $p = 0.008$), personal accomplishment (2.97 ± 1.04 vs. 2.74 ± 0.98; $t = -1.98$; $p = 0.048$), self-efficacy (3.15 ± 1.12 vs. 2.83 ± 1.04; $t = -2.58$; $p = 0.010$) and self-regulation (3.55 ± 1.19 vs. 3.18 ± 1.18; $t = -2.71$; $p = 0.007$), with higher levels being observed across each domain in the elite athlete group (see Figure 1). While not statistically significant poor sleep quality was observed (2.77 ± 0.78 vs. 2.83 ± 0.85), concerns related to injury (2.48 ± 1.09 vs. 2.32 ± 1.17) and relatively high levels of fatigue (2.46 ± 1.33 vs. 2.54 ± 1.31).

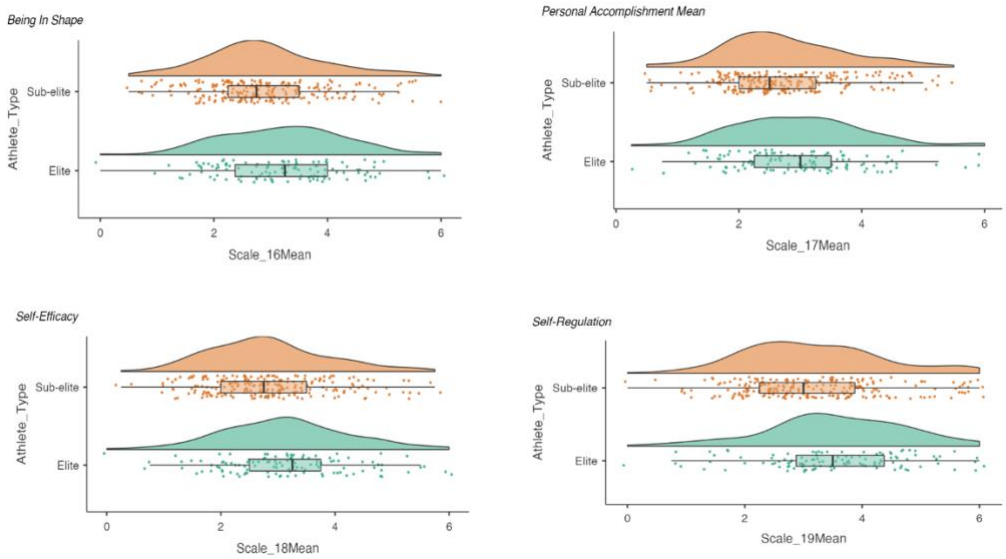

Figure 1. Comparison of the sport-specific recovery subscales.

Table 6. RESTQ scales (Mean ± SD).

	All (*n* = 338)	**Elite (*n* = 115)**	**Sub-Elite (*n* = 223)**	**T=**	***p*=**
General Stress	1.7 ± 1.31	1.77 ± 1.39	1.67 ± 1.26	−0.6602	0.51
Emotional Stress	1.95 ± 0.983	1.9 ± 0.98	1.97 ± 0.99	0.6858	0.493
Social Stress	1.85 ± 1.03	1.83 ± 1.04	1.86 ± 1.02	0.2199	0.826
Conflicts/Pressure	2.35 ± 1.24	2.24 ± 1.26	2.41 ± 1.24	1.1382	0.256
Fatigue	2.52 ± 1.32	2.46 ± 1.32	2.55 ± 1.32	0.6125	0.541
Lack of Energy	2 ± 1.06	1.95 ± 1.19	2.02 ± 1	0.5755	0.565
Physical Complaints	1.61 ± 1.22	1.59 ± 1.34	1.61 ± 1.16	0.1638	0.87
Success	2.85 ± 1	2.92 ± 1.01	2.81 ± 1	−0.9189	0.359
Social Relaxation	3.3 ± 1.28	3.19 ± 1.26	3.36 ± 1.29	1.1573	0.248
Physical Relaxation	2.53 ± 1.06	2.59 ± 1.09	2.49 ± 1.04	−0.8265	0.409
General Well-Being	3.35 ± 1.16	3.37 ± 1.22	3.35 ± 1.13	−0.1497	0.881
Sleep Quality	2.81 ± 0.83	2.77 ± 0.78	2.83 ± 0.85	0.6552	0.513
Disturbed Breaks	1.68 ± 0.92	1.71 ± 0.91	1.67 ± 0.94	−0.4119	0.681
Burnout/Emotional Exhaustion	1.83 ± 1.13	1.87 ± 1.22	1.81 ± 1.09	−0.4695	0.639
Fitness/Injury	2.43 ± 1.12	2.32 ± 1.17	2.48 ± 1.09	1.2827	0.2
Fitness/Being in Shape **	3.01 ± 1.06	3.22 ± 1.08	2.9 ± 1.04	−2.6563	0.008
Burnout/Personal Accomplishment *	2.82 ± 1.01	2.97 ± 1.04	2.74 ± 0.98	−1.9984	0.048
Self-Efficacy **	2.94 ± 1.07	3.15 ± 1.12	2.83 ± 1.04	−2.5747	0.01
Self-Regulation **	3.31 ± 1.2	3.55 ± 1.19	3.18 ± 1.18	−2.7121	0.007

Data presented as the mean ± SD * $p < 0.05$, ** $p < 0.01$.

3.3.1. AMES

An independent-samples t-test demonstrated a statistically significant difference between the groups for preferred competition time (t (336) = −2.45; p = 0.015), with a higher percentage of the elite athlete group (77% [n = 89]) preferring afternoon competition times compared to the sub-elite group (60% [n = 113]) (see Table 7). There was no significant difference between the groups for chronotype, time they usually become tired and preferred training time.

Table 7. Athlete response to the AMES.

Chronotype	Morning type	More morning type	More evening type		Evening type
Elite (*n* =)	24	40	36		15
Sub-elite (*n* =)	45	84	65		29
Preferred training time	6 am–9 am	9 am–Noon	Noon–3 pm	3 pm–6 pm	6 pm–9 pm
Elite (*n* =)	12	31	29	18	25
Sub-elite (*n* =)	26	75	47	41	34
Preferred competition time *	6 am–9 am	9 am–Noon	Noon–3 pm	3 pm–6 pm	6 pm–9 pm
Elite (*n* =)	5	21	47	21	21
Sub-elite (*n* =)	12	78	62	47	24
Time you usually get tired	8 pm–9:30 pm	9:31 pm–10:45 pm	10:46 pm–12:30 am	12:30 am–1:45 am	1:46 am–3:00 am
Elite (*n* =)	27	51	26	3	8
Sub-elite (*n* =)	50	94	66	9	4

* Statistically significant difference ($p < 0.05$).

3.3.2. Consensus Sleep Diary—Core

All athletes also completed a sleep diary for a training/competition day and a rest day. A one-way ANOVA was conducted to assess the difference between the groups for TIB, TST, SL, NoA and WASO on both the training/competition day and rest day. While there were no statistically significant differences for TIB, SL and WASO, there were statistically significant differences between

the groups (elite vs. sub-elite) for TST on the training/competition day (8.01 ± 1.3 vs. 8.2 ± 1.38; F(1, 238) = 3.91 ; p = 0.049) and NoA on the rest day (1.03 ± 1.17 vs. 1.52 ± 2.44; F(1, 334) = 6.34; p = 0.012), with the sub-elite athlete group reporting higher levels of both measures (see Table 8). The majority of athletes in both groups (elite n = 155 [70%]; sub-elite n = 77 [67%]) reported wakening 1–5 times each night. Athletes in both groups reported that it took ≥30 min to fall asleep on the training/competition day (elite n = 33 [29%]; sub-elite n = 72 [32%]) and the rest day (elite n = 35 [30%]; sub-elite n = 70 [31%]). While there was no statistically significant difference between the groups, poor habitual sleep efficiency (<85%) was reported by 20% (n = 23) of the elite athlete group and 25% (n = 55) of the sub-elite athlete group. In the comments section of the sleep diary a subset of athletes (n = 73 [22%]) reported the reasons for waking at night, the most common reasons included injury (n = 15 [4%]), children (n = 11 [3%]), anxiety (n = 19 [6%]), energy restriction (i.e., making weight) (n = 7 [2%]) and waking to use the bathroom (n = 21 [6%]).

Table 8. Sleep diary responses (mean ± SD).

Sleep Measure		Training/Competition Day	Rest Day
TIB (h)	Elite	9.1 ± 1.18	9.53 ± 1.49
	Sub-elite	9.2 ± 1.42	9.6 ± 1.5
TST (h)	Elite	8.01 ± 1.30 *	8.58 ± 1.4
	Sub-elite	8.2 ± 1.38 *	8.59 ± 1.44
SL (Min)	Elite	22.85 ± 20.74	21.62 ± 18.7
	Sub-elite	22.65 ± 17.70	23.72 ± 22.37
NoA (#)	Elite	1.38 ± 1.43	1.03 ± 1.17 *
	Sub-elite	1.51 ± 1.73	1.52 ± 2.44 *
WASO (Min)	Elite	11.06 ± 17.06	7.31 ± 9.99
	Sub-elite	10.14 ± 16.51	9.56 ± 12.60
SE (%)	Elite	88.2 ± 10.18	90.21 ± 6.6
	Sub-elite	89.77 ± 7.14	89.1 ± 7.05

* Statistically significant difference (p < 0.05).

3.3.3. Nutrition

The athletes also reported their supplement and alcohol consumption in the month prior to completion of the questionnaire. A Mann–Whitney U test indicated no significant differences between the elite and sub-elite athlete groups for supplementation and alcohol consumption (p ≥ 0.05). The most commonly used supplements were whey protein, caffeine, creatine, multivitamins, fish oil, probiotics and vitamin D (see Table 9).

Table 9. Athlete supplement use, frequency, average dose and reason for use.

Supplement	Frequency	Dose	Reason	Elite (n = 115)	Sub-elite (n = 223)
Caffeine	Daily	100 mg	Performance	23	37
Creatine	Daily	Varied	Performance	13	20
Fish Oil	Daily	1 capsule	Health	18	12

Iron	Daily	Varied	Anaemia/Performance	4	10
Multivitamin	Daily	1 capsule	Health	24	32
Nitrate	Daily	1 shot	Performance	11	1
Probiotics	Daily	1 capsule	Health	13	25
Vitamin D	Daily	1000–4000 IU	Health/Performance	21	5
Whey	Daily	25–40 g	Recovery	22	48
Other (e.g., BCAA, beta—alanine, HMB, casein, antioxidants)	Daily/weekly	Varied	Health/Performance	30	19

Spearman's rank order correlation was used to assess the relationship between supplement use and various sleep and recovery variables. There were small significant correlations between supplement use and the RESTQ scales: sleep quality, disturbed breaks, emotional exhaustion, being in shape and self-efficacy (see Table 10).

Table 10. Relationship between supplement use and recovery.

	Sleep Quality	Disturbed Breaks	Emotional Exhaustion	Being in Shape	Self-Efficacy
Supplement Use	−0.167 **	0.119 *	0.137 *	−0.114 *	−0.108 *
	$p = 0.002$	$p = 0.029$	$p = 0.012$	$p = 0.036$	$p = 0.048$

Statistically significant * $p \leq 0.05$; ** $p \leq 0.01$.

The athletes reported the number of times that they consumed alcohol in the last month 1–4 times (elite $n = 10$ [9%]; sub-elite $n = 10$ [5%]), 5–9 times (elite $n = 11$ [10%]; sub-elite $n = 5$ [2%]), and >10 times (elite $n = 3$ [3%]; sub-elite $n = 11$ [5%]). The athletes also reported the number of units they usually consumed during each drinking session < 4 units (elite $n = 11$ [10%]; sub-elite $n = 6$ [3%]), 5–10 (elite $n = 9$ [8%]; sub-elite $n = 8$ [4%]) and >10 (elite $n = 4$ [3%]; sub-elite $n = 12$ [5%]).

4. Discussion

This study recruited a large cohort of elite ($n = 115$) and sub-elite ($n = 223$) athletes from a wide variety of sports. Elite athletes were either international athletes, members of a national/professional team, a recruitment/academy squad and/or nationally ranked in their sport [84]. Sub-elite athletes were defined as those competing at a regional, university and/or national level of organised sport that trained and/or competed for a combined minimum of 400 min per week [84]. To the authors' knowledge, this is one of the largest cohorts of athletes to have been investigated from a sleep and recovery perspective. This study aimed to investigate: the quality, quantity and timing of sleep among sub-elite and elite athletes and characterise their recovery and nutrition practices. It was hypothesised that the sleep, recovery and nutrition practices of elite athletes would be superior to those of sub-elite athletes. Interestingly, similar levels of poor sleep were reported by both the elite and sub-elite athlete groups, whereas there was a significant difference in sport-specific recovery practices.

4.1. Sleep

Poor sleep quality was reported in the PSQI, the REST-Q and it was notable in the sleep diaries that athletes reported improved TIB, TST and WASO on rest days. Excessive daytime sleepiness was also observed in both groups. Similarly, previous research has suggested that the quality and quantity of elite athlete's sleep was inferior to sub-elite athletes and potentially inadequate in relation to optimal recovery and performance [27, 30, 32, 37, 96].

4.2. Pittsburg Sleep Quality Index

The PSQI has demonstrated good reliability (Cronbach's alpha = 0.83, test–retest reliability $r = 0.85$) [87]. The PSQI having demonstrated acceptable internal consistency and has been shown to be reliable [97,98] and valid [87,88,97,98] measure of sleep quality. Cronbach's alpha 0.744 was observed in the current sample. The majority of athletes (~65%; $n = 220$) were classified as poor sleepers (Global

PSQI score ≥5). This is consistent with previous research in elite athletes [53–55,96], and sub-elite athletes [99,100]. A relatively high proportion of athletes (~30%) self-reported their sleep quality as either poor or very poor on the training/competition day compared to rest day (elite 10% [*n* = 12] and sub-elite 16% [*n* = 36]). The PSQI data highlighted reasons for poor sleep on both training/competition days and rest days such as feeling too hot in bed and a lack of enthusiasm for general tasks. Poor sleep quality is of particular concern for elite athletes as it can result in a reduction in recovery and/or subsequent athletic performance [29,101–103].

Interestingly the PSQI mean TST (<8 h) was lower than that reported in the CSD-C (>8 h), it has been suggested that athletes tend to overestimate their sleep [104–105]. A recent review suggested that sleep in athletes is limited to 7.2 h per night, with all studies reporting <8 h per night and mean SE was 86.3 ± 6.8% [106], which is in line with the PSQI and CSD-C data from the current study. The PSQI mean TST for both groups in the current study is adequate according to current sleep recommendations (7–9 h) [48]. However, optimal TST is subject to individual variance and it has been argued that elite athletes may require more quality sleep than non-athletes [49]. It has previously been reported that athletes tend to sleep less (6.5–6.7 h) and that their sleep quality is poor [27,54,107–109]. Optimising sleep gives athletes an advantage when it comes to maximising adaptations from training and performance enhancement [110].

4.3. Consensus Sleep Diary-Core

There were significant differences between the groups for TST on the training/competition day and NoA on the rest day. TST was lower in the elite athlete group on both days. However, it did improve on the rest day which was most likely a reflection of their behaviour, e.g., choosing to go to bed earlier. Although not statistically significant there was a trend towards reduced TIB, TST and WASO in both groups on the rest day while the elite athlete group also demonstrated a trend towards reduced SL, NoA and increased SE on the rest day. Similarly, a small study of Australian athletes (*n* = 6) using objective measures of sleep demonstrated that sleep improved (longer duration) on a rest day (71.6% reported no sleep disturbance following one rest day) [78]. A study involving elite swimmers (*n* = 7) showed that the athletes went to bed later but slept longer on rest days [54], where the opportunity for extended sleep provided the athletes with an opportunity to partially recover the sleep debt accumulated during the training week [111]. In the current study, poor sleep was attributed by the athletes in both groups to a number of factors, i.e., injury, children, anxiety, making weight (boxing) and bathroom use. Previous research has highlighted issues that impair an athlete's sleep such as stress [32,112], pain/injury [26,32,33] and anxiety [25,29]. The relationship between poor sleep and impaired mood has been reported in non-athletic populations [113]. However, the study involved sleep restriction to 4.98 h per night. Monitoring athletes' mood (e.g., through wellness monitoring) could identify athletes who require sleep-related intervention.

In the current study, poor habitual SE% previously quantified as <85% [47] was reported by 20% (*n* = 23) of the elite athlete group and 25% (*n* = 55) of the sub-elite athlete group. Previous research has demonstrated that habitual sleep efficiency of elite athletes was 88.47 ± 5.45% [96] 80.6 ± 6.4% [27], 86.3 ± 6.1% [30] and 79 ± 9.2% [114]. A recent systematic review reported the pooled average sleep efficiency for athletes (86 ± 5%; range 79–96%) [37] which straddled and for many athletes overlapped the threshold of 85%, below which insomnia symptoms are indicated [115]. While the range of sleep efficiency observed can in part be explained by methodological inconsistencies, the pooled mean nonetheless indicated sleep problems and poor sleep quality. There is a need for clear athlete-friendly interventions that could promote improved sleep and recovery.

4.3.1. Daytime Sleepiness

The ESS score is comparable to objective sleepiness measures such as the multiple sleep latency test (MSLT) and is considered a valid and reliable measure of objective sleepiness [89]. The ESS has been widely used in athletic populations such as Australian rules football [116], collegiate basketball players [109] and American football players [117]. In the present sample, Cronbach's alpha was 0.827.

Approximately 21% of athletes in the current study reported excessive daytime sleepiness. Similar levels of excessive daytime sleepiness have been reported in Rugby players and cricketers [53], American footballers [117], Australian rules footballers [116] and college athletes [99]. Similarly, previous research reported that 44% (n = 12) Brazilian Paralympians experienced excessive daytime sleepiness [118]. However, it must be noted that these athletes may have had physical impairments (e.g., spinal cord injury) that could impact sleep quantity and quality.

The levels of excessive daytime sleepiness observed in the current study may be due to sleep disorders such as obstructive sleep apnoea (OSA) and periodic limb movement disorder (PLMD). In the general population, the most common sleep disorders are (OSA), insomnia and restless legs syndrome (RLS)/(PLMD) [61]. OSA is a frequent condition characterised by repeated episodes of partial or complete reduction in breathing activity during sleep [61]. PLMD is a condition characterised by repetitive limb movements during sleep that cause sleep disruption [62]. A recent systematic review highlighted the prevalence of insomnia symptoms (longer SOL, increased sleep fragmentation and excessive daytime sleepiness) in elite athletes [37]. Other sleep problems such as OSA are less prevalent but appear to higher in strength and power athletes (e.g., Rugby players) most likely due to increased body mass and neck circumference (>42 cm) which are anatomical features related to OSA [53]. A recent study using a combination of PSG and subjective measures demonstrated a high prevalence of sleep disorders in Rugby union players (n = 25), all players displayed insomnia symptoms and 24% (n = 6) had OSA and 12% (n = 3) [64]. In similar study using home-based PSG in Rugby league players (n = 22), 45% (n = 10) had OSA [119]. A previous study of NFL players (n = 137) demonstrated that 19% (n = 26) had OSA [120]. Previous research in elite ice hockey players (n = 107) has demonstrated sleep problem, 11% (n = 14) had insomnia, 10% (n = 13) had OSA and 3% (n = 4) had RLS/PLMD [26]. Athletes with poor sleep habits and/or a sleep disorder must be identified and diagnosed and individual interventions (e.g., sleep hygiene, nutrition) must be implemented in order to athlete recovery and performance.

4.3.2. Athlete Morningness/Eveningness

A Cronbach's alpha of 0.698 was observed in the current sample. Although there was no significant difference between the groups for chronotype, time they usually become tired or preferred training time, a statistically significant difference was evident for preferred competition time, (p = 0.015), with the elite athlete group preferring afternoon competition times, while the sub-elite athlete group preferred morning competition times. The vast majority of the athletes from both groups 58% (n = 197) indicated that their normal training time was after 5 pm. Training time and chronotype may have an influence on sleep [40]. A study investigating the sleep quality of morning and evening types after a morning (8:00 am) and evening (20:00 pm) high intensity interval training session types reported poorer sleep quality (reduced total sleep time, increased sleep disturbance and reduced sleep efficiency) in morning types after the evening session while sleep quality after the morning session was similar for both groups [121]. The late training times reported by the athletes in the current study may have adversely impacted their sleep and recovery. Sleep following training is recognised a being important for recovery [122], reduced sleep quality following evening training sessions (particularly vigorous training) may negatively impact subsequent recovery and performance, the effect may be more pronounced in morning type athletes.

4.4. Recovery

Recovery is a process in time, dependent on the duration of stress and requires a reduction in stress, a change in stress or a break from stress [123,124]. Relatively high levels of fatigue, stress and pain were reported in both groups. A range of supplements were used regularly by athletes in both groups; indeed, whey was the most commonly used recovery supplement in both groups. The results suggest that future research is warranted to further the development of individualised inventions focused on sleep, nutrition and athlete recovery.

4.4.1. EuroQoL

The EQ-5D-5L has demonstrated reliability (mean intraclass correlation coefficients 0.69; range 0.43–0.84) and convergent validity (mean Spearman rank coefficients 0.99; range 0.97–0.99) [85]. Cronbach's alpha of 0.70–0.95 are considered "acceptable" for a scale used in human research [125,126]. Cronbach's alpha 0.609 was observed in the current sample most likely due to the low number of items (5), as the size if alpha depends on the number of items in a scale [127]. The mean general health rating scores for the elite athlete group (83.1 ± 12.6) and the sub-elite athlete group (81 ± 13.7) were relatively high, which was consistent with current research in athletes [128]. In the current study, the elite athlete group reported higher mean health rating scores. Elite athletes tend to have their training and recovery sessions scheduled for them [54], hence, they are likely to complete regular if not daily mobility type sessions. Whereas the sub-elite athletes may have had less free time due to work, social and family commitments. A high prevalence of pain was reported by 50% (n = 169) of participants (elite n = 53 sub-elite n = 116). An investigation of 'mildly sleepy' (indicative of inadequate TST) but otherwise healthy males (n = 24) showed sleep extension (time in bed 10 h) increased pain tolerance by 20% [129]. While chronic sleep restriction (50% of habitual time for 12 days) is related to increased levels of muscle soreness and increased pain sensitivity [107]. While mobility issues were noted in both groups, there were higher levels mobility issues reported by the sub-elite athlete group coupled with issues completing usual activities. However, it has recently been suggested that elite and high-level athletes have increased pain tolerance (cold pressor test) and that the training time per week has a positive impact on the tolerance [130].

4.4.2. REST-Q sport

The RESTQ-Sport has been shown to be valid in athletic populations [131,132]. The scales have displayed good internal consistency (0.67–0.89) and high test–retest reliability (>0.79) [91,124]. A Cronbach's alpha of 0.784 was observed in the current sample.

Relatively high levels of stress and fatigue were evident from the REST-Q. Stress and fatigue are factors for illness, which must be managed by elite athletes [133,134], during their competitive seasons to avoid missed training/competitions. Significant differences between the elite and sub-elite athletes were observed for four of the REST-Q subscales relating to athletic performance, with higher mean score for each subscale: being in shape, personal accomplishment, self-efficacy and self-regulation reported by the elite athlete group. The injury (2.31 ± 1.17 vs. 2.48 ± 1.09), fatigue (2.45 ± 1.32 vs. 2.54 ± 1.32) subscale scores were relatively high in both the elite athletes and sub-elite athletes, while the sleep quality scores were low (2.76 ± 0.78 vs. 2.83 ± 0.85). The current findings are consistent with previous research which reported that injury risk was significantly positively related to injury subscale scores for disturbed breaks, fatigue, and lower values on the sleep quality subscale score [131]. The relationship between training load and health can be considered on a well-being continuum [123,134,135], with training load and recovery as antagonists. Stress is imposed on athletes, altering their physical and psychological well-being along a continuum: homeostasis, acute fatigue, subclinical tissue damage, functional overreaching, non-functional overreaching, clinical symptoms, overtraining syndrome, time-loss injury or illness and, with continued loading in extreme cases, death [134,135]. A recent meta-analysis has linked psychological stress (r = 0.27, 80% CI 0.20–0.37) and history of stressors (r = 0.13, 80% CI 0.11–0.15) to injury rates [136]. Athletes' injury risks are affected by their responses to multiple stressors that result in not only physical, psychological and attentional changes (e.g., increased reaction time, narrowing of peripheral vision, increased distraction) but also behavioural changes (e.g., poor sleep quality and impaired self-care) [136].

In the current study, significantly higher levels of sport-specific recovery (3.22 ± 0.91 vs. 2.91 ± 0.90) were reported by the elite athlete group compared to the sub-elite athlete group. This result potentially highlights the fact that elite athletes tend to be under the supervision of a multidisciplinary team, e.g., medical, strength and conditioning, nutrition, physiology and

psychology, who are involved in all aspects of the athletes training and recovery. The sub-elite athletes would typically not receive the same access to multidisciplinary support services. It is imperative that athletes have a detailed recovery plan compromising of nutrition, hydration, sleep and psychological recovery [134]. Given the high training and competition load that athletes undertake, it is clear that they must adopt strategies that promote sleep across the domains of quality, quantity and timing. Fatigue can be managed, and recovery enhanced through adequate passive rest and sufficient sleep [137], it is generally recommended that athletes have at least one 'rest' day per week. Rest days can serve to alleviate boredom and stress perception while the absence of a 'rest day' during periods of intense training has been related to the onset overreaching and inadequate recovery [137]. It is suggested from the current results that sleep tends to improve on rest days, i.e., increased perceived sleep quality, TIB, TST and reduced WASO in both groups, while SL, NOA and SE also improved in the elite athlete group.

4.5. Nutrition

In the current sample, the elite athletes tended to consume more supplements, at higher doses with increased frequency, compared to the sub-elite athletes. Those athletes who used supplements reported high usage of caffeine, whey protein, creatine, multivitamins, fish oil, probiotics and vitamin D while the use of iron and nitrate was reported to a lesser extent. This is similar to previous research in elite Dutch athletes (n = 778) where the most commonly consumed supplements were multivitamins, caffeine, vitamin D, sports drinks, protein, beta-alanine and sodium bicarbonate [138]. It has also been demonstrated previously that elite athletes tend to take more supplements than sub-elite athletes [139]. Despite the relatively low number of athletes reporting supplement use, the correlations between supplement use and RESTQ scales warrant further investigation. Whey protein was one of the most prevalent supplements used while casein use was also reported. While research is emerging supporting pre-sleep protein ingestion for muscle recovery [140,141], the impact of pre-sleep ingestion of 40 g doses of whey and/or casein warrants further investigation with regards both muscle recovery and sleep improvement.

Daily caffeine use was reported by approximately 20% of the athletes which could negatively impact sleep. The low level of caffeine use reported in the current study was most likely due to the fact that athletes were asked to report their supplement use and may have neglected to include habitual caffeine consumption. Caffeine exerts a stimulant effect promoting alertness by blocking adenosine receptors [142]. The levels of caffeine consumption reported were lower than previous research which has suggested that 75–90% of athletes consume caffeine before or during competition [143–145]. While, it has been suggested that chronic low dose caffeine ingestion may blunt any potential ergogenic effects [146], moderate doses (~3 mg/kg/d) appear to pose no problems for most athletes [147]. However, in terms of sleep, moderate caffeine doses have been shown to increase SOL and decrease TST, REM sleep and SE [148]. Hence, athletes training/competing in the late afternoon (>5 pm) need to consider its potentially detrimental effect on sleep. It has recently been suggested that athletes should adopt a strategic individualised approach to caffeine consumption during competition [149]. In the current study, higher alcohol consumption was observed in the sub-elite athletes and they tended to consume more units of alcohol per drinking bout. In line with previous research, the actual amount of alcohol consumed by athletes "in training' is low [150]. Elite athletes tend to have less opportunity to socialise and their schedules (e.g., early morning training) do not lend themselves to regularly consuming alcohol. Alcohol consumption by athletes often occurs post-competition, where it can be seen as a reward for 'hard work' [151]. Alcohol consumption has been associated with poorer sleep quality and quantity, reduced REM sleep and increased sleep disturbance in the second half of the sleep bout [152].

4.6. Limitations

Due to logistical reasons, the sleep diary was only completed for one training/competition day and one rest day, and this may have been insufficient in terms of data collection. It has been recommended that sleep diaries should be completed for a duration of 1 week [68,153]. The aim of the 2 day diary was to limit participant burden and recall bias [154]. However, sleep diaries may be more accurate than sleep questionnaires [32]. The intrinsic limitations of self-report measures (i.e., questionnaires and diaries) are measurement error and recall bias [95]. Indeed, it has been demonstrated that athletes can overestimate their TST [104,105]. However, self-report measures have their place within athletic settings, as they are a relatively simple and inexpensive approach to athlete monitoring affording a more representative overview of the target population [44]. Within elite athlete populations, the use of subjective measures of sleep are often employed, particularly during the competitive season due to the more invasive nature of both PSG and actigraphy [12]. A growing body of research has suggested that self-report measures may be more sensitive and reliable than physiological, biochemical and performance measures [44,137,153–156]. When choosing a particular measure, ultimately the aim is to maintain a balance between the need to obtain meaningful data from an athlete whilst minimising the burden involved in completion of any self-report measure [154–156]. In the current study, it was not feasible or practical due to the large sample size to include a subjective assessment of sleep. However, future research should incorporate both objective (e.g., PSG, actigraphy) and subjective measures (e.g., sleep diaries) of sleep to provide a more accurate estimates of sleep and because some individuals may self-report poor sleep quality despite objective measures indicating adequate sleep [155,156,158]. There was little difference between the elite and sub-elite athlete groups in terms of sleep. The inclusion of a healthy control group would have allowed for comparison and exploration of the differences between the sleep of athletic population and healthy adults.

A specific section in relation to anxiety/depression could have been included in the battery of questionnaires given the potential to impact on sleep and vice versa. The Profile of Mood States (POMS) [157] is widely used in wellness assessments of athletic populations and has subscales that specifically relate to anxiety and depression. However, as the EuroQoL has a dimension for anxiety/depression, the POMS was omitted to reduce participant burden and survey fatigue which could have negatively impacted the reliability of the data collected.

The demographic difference between the groups was a limitation in that there was a statistically significant difference between the groups with the sub-elite group being significantly older which could have affected the results. This issue was directly related to the sampling method employed where participants are recruited based on their accessibility. However, care was taken to recruit a large cohort (n = 338) and strict inclusion and exclusion criteria were applied [84].

4.7. Future Research

Future research should replicate this investigation of the sleep and recovery practices of large cohorts of athletes. Such studies should include a combination of subjective and objective measures of sleep and recovery, for a minimum of 1 week [54,153]. The validity and reliability of combinations of subjective and objective measures in athletic populations warrants further investigation. While this may not be practical during the competitive season there may be a window of opportunity at the end of the season or in preseason.

As the majority of athletes in the current cohort have reported sleep problems future research is warranted to identify the specific sleep problems that affect athletic populations. It is also necessary in future research to identify if athletes are affected by acute disturbances, e.g., competition anxiety or chronic disorders, e.g., OSA, insomnia and PLMD [26].

Future research should investigate the effects of specific nutritional recovery strategies (e.g., antioxidants, protein, carbohydrate) on sleep in athletic populations. Such practices may already be an established part of an athlete's daily routine, but the potential additional benefit of improved sleep must be explored.

4.8. Practical Applications

A strength of this novel study is that it presents 'real-life' data from training/competition days and a rest day relating to the sleep and recovery practices of athletes. Poor sleep and inadequate recovery practices were evident in both the elite and sub-elite athlete groups. In a recent study, 95% of swimmers (n = 82) identified their coaches (n = 10) as the primary source of recovery information while the coaches highlighted conferences and workshops as their primary source of recovery information [159]. In order to promote sleep hygiene and adequate recovery practices in athletes, a comprehensive coach and athlete education curriculum may need to be developed and implemented.

The athletes generally reported improved sleep quality and quantity on rest days which has implications for athlete health, well-being and performance. Optimising the sleep and recovery practices of athletes would impact performance. Monitoring of sleep behaviours, nutrition and recovery-stress responses of athletes aids the identification of irregularities (e.g., due to travel or illness) and allows for early interventions with individual athletes as and when necessary [158]. The ongoing collection of data from athletes such as the data collected in the current study could be used by coaches and medical and support staff to implement individual sleep, recovery and nutrition interventions and plans.

5. Conclusions

Due to the symbiosis between sleep and recovery, it is clear from the current findings that athletes should have a detailed individualised and multifaceted recovery plan in place involving sleep, nutrition, hydration, and other physiological and psychological aspects. At the elite level, athletes and their support teams continually strive for marginal gains over time to improve performance (135). Training and competition load elicit a number of homeostatic responses and adaptations, and the main aim of training is to exploit these in order to elicit an improvement in performance. The training process involves exploitation, manipulation and coordination of numerous variables (e.g., physiology, biomechanics and psychology) to improve performance. Athletes continually strive to improve their performance, and, as such, variations in training load are necessary, e.g., increased frequency, duration and/or intensity in order to optimise the training response [44]. Depending on the phase of the season (e.g., pre-season, general preparation, and competition), loads must be managed to increase or decrease fatigue, to enhance training adaptations or performance [44]. Rest days should also be incorporated into the recovery plan, which could serve to improve sleep quality, alleviate boredom and stress perception.

The majority of athletes were classified as poor sleepers and reported excessive daytime sleepiness even though their TST met current adequate sleep guidelines. The importance of a rest day was highlighted by the fact that sleep improved in both groups. Relatively low levels of physical recovery were observed in both groups coupled with relatively high levels of stress. The elite athlete group reported significantly higher levels of sport-specific recovery. A higher prevalence of supplement use was reported by the elite athlete group, while higher levels of alcohol consumption were reported by the sub-elite athlete group. Given the high training and competition load that athletes undertake, particularly elite athletes, it is clear that they must adopt strategies that promote sleep and recovery. There is a need for athletes to receive individualised support and education regarding their sleep ad recovery practices.

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