Effects of sleep on endurance cycling performance and heart rate indices of athlete readiness

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Effects of Sleep on Endurance Cycling Performance and Heart Rate Indices of Athlete Readiness

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Bachelor of Exercise and Sport Science (Honours)

Submitted in fulfilment of the requirements for the degree of

Doctor of Philosophy

Deakin University

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Effects of total sleep deprivation on endurance performance cycling performance and heart rate indices used for monitoring athlete readiness		Journal of Sports Sciences
Name of executive author	School/Institute/Division if based at Deakin; Organisation and address if non-Deakin	Email or phone
Mr Spencer Roberts	Institute for Physical Activity and Nutrition (IPAN)	rspen@deakin.edu.au

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SR was involved in the conception of the study, and was responsible for participant recruitment, data collection, data analysis, drafting the manuscript, and approving the final manuscript.		
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Doctor Stuart Warmington, IPAN, Deakin University	SW was involved in conception of the study, analysis and interpretation of results, revising of manuscript, and approved the final manuscript

Doctor Wei-Peng Teo, PESS, Nanyang Technological University	WPT was involved in conception of the study, and revising of manuscript

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Mr Spencer Roberts	Institute for Physical Activity and Nutrition (IPAN)	rspen@deakin.edu.au

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SR was involved in the conception of the study, and was responsible for participant recruitment, data collection, data analysis, drafting the manuscript, and approving the final manuscript.		
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Doctor Stuart Warmington, IPAN, Deakin University	SW was involved in conception of the study, analysis and interpretation of results, revising of manuscript, and approved the final manuscript

Doctor Wei-Peng Teo, PESS, Nanyang Technological University	WPT was involved in conception of the study, and revising of manuscript

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Publications and Presentations

Peer-reviewed journal publications arising from this thesis

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. Effects of total sleep deprivation on endurance cycling performance and heart rate indices used for monitoring athlete readiness. *J Sports Sci* 2019;37(23):2691-701.

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. Extended sleep maintains endurance performance better than normal or restricted sleep. *Med Sci Sports Exerc* 2019;51(12): 2516-523.

Other related peer-reviewed journal publications

Roberts SSH, Teo W-P, Warmington SA. Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis. *Br J Sports Med* 2019;53(8):513-22.

Trounson KM**, Roberts S**, Balloch A, Warmington SA. Light exercise heart rate on-kinetics: a comparison of data fitted with sigmoidal and exponential functions and the impact of fitness and exercise intensity. *Physiol Rep.* 2017; 5(12):e13312

Conference presentations

Roberts SSH, Teo W-P, Warmington SA. Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis (Oral presentation). British Association of Sport and Exercise Sciences Conference. Leicester, United Kingdom, 19-20 November 2019.

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. Effects of sleep extension and restriction on the performance and cardiac autonomic function of endurance cyclists (Oral presentation and poster). The Physiological Society: Sleep and Circadian Rhythms from Mechanisms to Function. London, United Kingdom, 5-6 December 2018.

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. The effects of sleep extension and sleep restriction on the performance of endurance cyclists (Oral presentation and poster). Australasian Sleep Association: Sleep DownUnder. Brisbane, Australia, 17-20 October 2018.

Roberts SSH, Teo W-P, Warmington SA. Effects of sleep deprivation on cardiac autonomic modulation and endurance performance in trained cyclists (Oral presentation). European College of Sport Science: 22nd Annual Congress. Essen, Germany, 5-8 July 2017.

Industry presentations

Roberts SSH. Sleeping to win: the effects of sleep on athletic performance. Presented to coaching and high-performance staff at Geelong Football Club. GMHBA Stadium, Geelong. January 2019.

Roberts SSH. Sleeping to win: examining the influence of sleep on athletic performance. Presented to Athletics Victoria Talent Pathway Program. Victorian Institute of Sport, Lakeside Stadium, Albert Park. July 2018.

Roberts SSH. Sleep and athletic performance. Presented to delegation of Chinese high-performance managers. Deakin University, Geelong. August 2017.

Contents

List of Abbreviations

a	Baseline heart rate (exponential function parameter)
A	Heart rate amplitude (exponential function parameter)
AT	Anaerobic threshold
ATP	Adenosine triphosphate
AU	Arbitrary units
CNS	Central nervous system
CV	Coefficient of variation
D1-D4	Laboratory testing days 1-4
$g \cdot dL^{-1}$	Grams per decilitre
HR	Heart rate
kJ	Kilojoules
Ln rMSSD	natural logarithm of the square root of the mean sum of the squared differences between R-R intervals
PBM	Psychobiological model of exercise tolerance
$pg \cdot mL^{-1}$	Picogram per millilitre
POMS	Profile of mood state questionnaire
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
PVT	Psychomotor vigilance task
REM	Rapid-eye-movement
$rev \cdot min^{-1}$	Revolutions per minute
rHRI	Maximal rate of heart rate increase
RPE	Rating of perceived exertion
SE	Sleep extension
SR	Sleep restriction
SD	Total sleep deprivation / Standard deviation
STAI-Y	State-Trait Anxiety Inventory
SWS	Slow wave sleep
τ	Tau (exponential function parameter)
t	Time in seconds (exponential function parameter)

TD	Time delay (exponential function parameter)
TT	Time-trial
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_{2\,max}$	Maximal oxygen consumption
W	Watts
W_{AT}	Watts at anaerobic threshold

List of Definitions

Key terms relating to sleep and sleep monitoring:

Bedtime - the self-reported time of day one starts attempting to sleep.

Sleep onset - the time of day following bedtime at which sleep commences

Get-up time - the self-reported time of day one wakes from a sleep period for the final time.

Time in bed - the total time spent in bed between bedtime and get-up time.

Sleep period – the time between initial sleep onset and final wake-up time.

Total sleep time - total time spent asleep during a sleep period.

Sleep efficiency - total sleep time as a percentage of time in bed.

Sleep latency – time between bedtime and initial sleep onset

Sleep phase - sleep period from the time of day at sleep onset to the time of day on waking. It is dependent on the circadian process which can be delayed or advanced by environmental or psychosocial factors.

Total sleep deprivation – complete lack or absence of sleep for a period to time.

Sleep restriction – a decrease in one's habitual total sleep time.

Sleep extension - an increase in one's habitual total sleep time.

Sleep opportunity – a period during which sleep can be obtained.

Sleep hygiene – the practice of behaviours that facilitate sleep and avoidance of behaviours that impede sleep.

Key terms relating to heart rate and its use for athlete monitoring:

Readiness to perform (i.e., readiness) – an athlete's relative capacity to perform that reflects the interaction between their overall fitness level and any accumulated

fatigue, and which depends on balancing the psychophysiological stress of training and competition with adequate recovery.

Resting heart rate - mean heart rate (beats·min^{-1}) recorded during a period of rest.

Heart rate variability - a measure of the variation in time between adjacent heart beats (i.e., R-R intervals).

Maximal rate of heart rate increase - maximum rate of heart rate increase (beats·min^{-1}·s^{-1}) at the onset of constant-load submaximal exercise derived from the first derivative maxima of an exponential function fitted to the heart rate response.

Steady-state heart rate - mean heart rate recorded (beats·min^{-1}) during a period of constant-load submaximal exercise and once heart rate has reached steady-state.

Heart rate recovery - magnitude of change in heart rate (beats·min^{-1}) that occurs within one minute of ceasing exercise.

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Thesis Abstract

Introduction: Sleep is considered an important recovery behaviour for athletic success. However, relatively few studies have examined the effects of sleep on the sports-specific performances of athletes, particularly endurance athletes. Studies that *have* examined the effects of sleep loss on the performance of endurance athletes have not assessed performance using prolonged self-paced efforts (e.g., time-trials) that mimic the demands of many endurance sports (e.g., road cycling, triathlon). Further, no prior research has examined the *cumulative* effects of sleep restriction (i.e., reduced habitual sleep time) or sleep extension (i.e., increased habitual sleep time), over consecutive nights, on the performance of endurance athletes. In addition, although heart rate (HR) indices are popular for monitoring athletes' fatigue status or 'readiness to perform', no prior research has examined whether these HR indices are sensitive to any effects of sleep on the readiness of endurance athletes. Understanding the effects of sleep on HR indices may help practitioners interpret such indices, and thus inform better athlete management decisions (e.g., sleep recommendations). Therefore, across two studies, the current research investigated the effects of a single night of total sleep deprivation (*Study 1*), and three consecutive nights of both sleep restriction and sleep extension (*Study 2*), on the performance of endurance athletes. Studies also investigated whether HR indices were sensitive to any effects of sleep on endurance athletes' readiness to perform. **Methods:** *Study 1* - Thirteen endurance athletes completed a crossover experiment comprising a normal sleep (NS) and total sleep deprivation (SD) condition. Each condition required completion of an endurance time-trial (TT) on

two consecutive days (D1, D2) separated by either normal sleep or total sleep deprivation. For each TT, finishing time and perceived exertion (RPE) were recorded. Prior to each TT, participants had their mood and psychomotor vigilance (PVT) assessed. Resting and exercising heart rate (HR) indices were examined, including the subjective to objective (RPE:HR) and internal to external (W:HR) intensity ratio for each TT. *Study 2* - Nine endurance athletes completed a crossover experiment with three conditions: sleep restriction (SR), normal sleep (NS), and sleep extension (SE). Each condition required completion of an endurance TT on four consecutive days (D1 - D4). Participants slept habitually prior to the first day (D1); however, time in bed was either reduced by 30% (SR), remained normal (NS), or was extended by 30% (SE) on the three subsequent nights. Sleep was monitored using actigraphy to confirm sleep restriction and extension were achieved. For each TT, finishing time and RPE were recorded. Prior to each TT, participants had their mood and PVT assessed. Resting and exercising heart rate (HR) indices were examined, including the subjective to objective (RPE:HR), and internal to external (W:HR), intensity ratio for each TT. For *both studies*, data were analysed using Generalised Estimating Equations, and Pearson's correlations examined relationships between changes in respective HR indices and changes TT finishing time within the SD condition (*Study 1*), and within the SR and SE conditions (*Study 2*). For *Study 2*, publishing constraints (e.g., word and referencing limits) prevented findings from being published in a single paper, thus findings concerning effects on endurance performance (*Study 2a*) and effects on HR indices (*Study 2b*) are presented separately. **Results:** *Study 1* - TT finishing time on D2 of SD was 10% slower than on D2 of NS (64 ± 7 vs 59 ± 4 min, $P < 0.01$), and 11% slower than D1 of SD (58 ± 5 min, $P < 0.01$). The subjective to objective intensity

ratio (RPE:HR) for the TT was higher on D2 of SD compared with D2 of NS and D1 of SD ($P < 0.01$). There were no statistical differences between or within conditions for any other HR indices, and there were no significant correlations between changes in respective HR indices and changes TT finishing time within the SD condition. Mood disturbance and PVT mean response time increased on D2 of SD compared with D2 of NS and D1 of SD. *Study 2a* - On nights D1, D2, and D3, total sleep time was longer ($P < 0.001$) in the SE condition (8.6 ± 1.0; 8.3 ± 0.6; 8.2 ± 0.6 h, respectively), and shorter ($P < 0.001$) in the SR condition (4.7 ± 0.8; 4.8 ± 0.8; 4.9 ± 0.4 h) compared with NS (7.1 ± 0.8; 6.5 ± 1.0; 6.9 ± 0.7 h). Compared with the NS condition, TT performance was slower ($P < 0.02$) on D3 of SR (58.8 ± 2.5 vs 60.4 ± 3.7 min) and faster ($P < 0.02$) on D4 of SE (58.7 ± 3.4 vs 56.8 ± 3.1 min). RPE was not different between or within conditions. Compared with the NS condition, mood disturbance was higher and PVT mean response time slower, following sleep restriction. Compared with the NS condition, PVT mean response time was faster following sleep extension. *Study 2b* - The subjective to objective intensity ratio (RPE:HR) for the TT was lower on D4 of the SE condition compared with both NS and SR conditions ($P < 0.02$). Within the SR condition RPE:HR was higher on D3 and D4 compared with D1 ($P < 0.02$). Strong correlations were found between percentage changes in external to internal intensity ratio (W:HR) for the TT from D1 to D4 and changes in TT finishing time for both SR ($r = -0.67$, $P < 0.05$) and SE ($r = -0.69$, $P < 0.05$) conditions. There were no statistical differences between or within conditions for any other HR indices, and there were no other significant correlations. **Conclusion:** Sleep is an important recovery behaviour that optimises endurance performance. Compared with normal sleep, a single night of total sleep deprivation, and two consecutive

nights of sleep restriction, impaired endurance performance. In contrast, compared with normal sleep, three consecutive nights of sleep extension improved endurance performance. Findings suggest endurance athletes should aim to sleep > 8 hours per night to optimise performance. Findings may suggest that total sleep time accumulated over 1-3 nights affects endurance performance by altering the perceived exertion of a given exercise intensity. Intensity ratios recorded during high-intensity exercise (e.g., endurance races) that incorporate mean HR (i.e., RPE:HR, W:HR) may, to some extent, be sensitive to the effects of sleep on endurance athletes' readiness to perform. No other HR index examined showed any sensitivity to the effect of sleep on endurance performance. Further research is required to examine relationships between sleep, endurance performance, and HR indices over longer timeframes and in other applied sport settings. Finally, an individualised sleep extension strategy that prescribed relative time-in-bed increases (e.g., +30% of habitual time-in-bed) and tailored bed / get-up times to individual chorotype effectively extended sleep, and may be a useful approach for practitioners recommending sleep extension to athletes.

Chapter 1: General Introduction

1.1 Sleep: a *not-so* new frontier in sport science

The question as to the importance of sleep has long been a focus of human inquiry.[1] Hippocrates (c. 460-375 BCE), the central figure in ancient Greek medicine, believed sleep was necessary for the maintenance of human health.[1] Later, the Scottish surgeon and philosopher Robert MacNish prophetically described healthy sleep as necessary for the "renovation of bodily organs and mental faculties" in his 1834 book 'The Philosophy of Sleep'(Page 11).[2] Despite these long-held intuitions, the absence of supportive empirical evidence led some researchers in the 20th century to suggest that sleep may serve no important function at all.[3] This prompted the pioneering sleep researcher Alan Rechtschaffen to declare that "if sleep doesn't serve an absolutely vital function, it's the biggest mistake evolution ever made."[3] In recent decades, research has indeed discovered sleep serves *many* vital functions such as up-regulating immune defence, removing metabolic waste from the brain, restoring brain energy stores, and consolidating the brain's neural connections.[4] As such, it is now accepted that sleep is essential for human health and survival.[4 5]

In athletic populations too, sleep has long been considered important for recovery and performance.[6] In 1923, The Boston Medical and Surgical Journal published an article in which sleep was identified as one of the "main considerations" that affect the training of athletes, recommending that athletes obtain "abundant sleep at regular hours".[6] The article also identified sleep disturbance as a symptom of an athlete "going stale".[6] Over the last 40 years, research has shown sleep affects many of the physiological (e.g., maximal oxygen uptake) and cognitive (e.g., reaction time) determinants of athletic performance, as well as exercise performance itself.[7] Thus, not surprisingly, regular healthy sleep is now considered essential for the recovery and performance of athletes.[7-9] Indeed, a recent survey of team-sport

athletes found sleep was considered the most important recovery behaviour for athletic success.[10] This growing appreciation of sleep in recent decades coincided with the development of new methods for monitoring sleep.[11] While sleep monitoring traditionally required expensive laboratory equipment, actigraphy emerged as a valid objective means by which to study sleep without infringing on habitual routines.[11 12] Consequently, over the last ~ 15 years, there has been a proliferation of research aimed at understanding athletes' sleep,[13] and it is now understood that athletes often sleep poorly, and are susceptible to sleep disturbances due to factors associated with training and competition (e.g., nervousness, late night competition).[8 13 14] More recently, this improved understanding of athletes' sleep has prompted the development of athlete-specific tools that screen for sleep disorders (e.g., Athlete Sleep Screening Questionnaire) and evaluate sleep behaviours (e.g., Athlete Sleep Behaviour Questionnaire).[15 16] The use of such tools, along with strategies to extend time-in-bed, and/or provide sleep hygiene education, has proven effective for improving sleep outcomes in athletes.[17-20]

1.2 Thesis rationale and objectives

Despite the apparent consensus that sleep is important for athletic recovery and performance,[7-10] relatively few studies have actually examined the effects of sleep on the sport-specific performance of trained athletes.[7 20-26] Indeed, a recent review stated more research was needed to examine the effects of sleep loss on the sport-specific performance of athletes in order to clarify whether the importance of sleep differs depending on the nature of the exercise or sport.[7] Studies that *have* investigated sports-specific performances have often examined the effects of sleep on sports-specific skill execution.[20 21 23] While those examining endurance athletes have reported equivocal findings, and have not assessed performance using

prolonged self-paced efforts (e.g., time trials lasting ≥ 60 minutes) akin to those required by many endurance sports, such as road cycling and triathlon.[24-26] Therefore, there is a need to examine the effects of sleep on the prolonged self-paced endurance performance of endurance-trained athletes.

Another issue of interest to practitioners is how to monitor athletes' sleep and its effect on performance and health.[27] In elite sporting contexts, the utility of wearable devices is limited, largely due to concerns over cost, accuracy, and/or violation of privacy.[27 28] Therefore, sleep is typically monitored using simple subjective tools (e.g., visual analogue scales), despite evidence that subjective data does not always correlate with objective data.[29 30] Given the barriers to monitoring sleep itself, it is important practitioners understand whether indices routinely used to monitor athletes' fatigue status or 'readiness to perform' are sensitive to sleep. This will help practitioners better interpret readiness indices, and thus inform better athlete management decisions (e.g., sleep recommendations). Heart rate (HR) indices are popular for monitoring athlete readiness, and although there is evidence that HR regulation is influenced by prior sleep duration,[31 32] it remains unclear whether HR indices are sensitive to any effects of sleep on athletes' readiness to perform.

Given the issues highlighted in this introduction, the current thesis aimed to:

1. Examine the effects of total sleep time, ranging from total sleep deprivation to sleep extension, on the recovery of endurance performance over acute timeframes (i.e., 1-3 nights).
2. Examine the effects of total sleep time, ranging from total sleep deprivation to sleep extension, on HR indices used to monitor athlete

readiness, and determine whether any changes in HR indices correspond to changes in endurance performance.

1.3 Thesis structure

This thesis comprises six chapters (Figure 1.1). Following the current introduction, Chapter 2 reviews literature on sleep and its importance for athletic recovery and performance. It highlights specific gaps in our understanding of the importance of sleep for the recovery of endurance performance. Chapter 2 includes excerpts from a systematic review and meta-analysis published by the author in the *British Journal of Sports Medicine.* Chapter 3 contains original research examining the effects of total sleep deprivation on endurance cycling performance and HR indices of athlete readiness (Study 1). This study has been published in the *Journal of Sports Sciences*. Chapter 4 contains original research examining the cumulative effects, over three consecutive nights, of both sleep restriction and sleep extension, on endurance cycling performance (Study 2a). This study has been published in *Medicine and Science in Sports and Exercise*. Chapter 5 contains original research examining the effects of both sleep restriction and sleep extension on HR indices of athlete readiness (Study 2b). This chapter is being prepared for publication. Chapters 4 and 5 report findings from a single study; however, due to publishing constraints (i.e., word and referencing limits), findings concerning endurance performance and HR responses were prepared for separate publications, and have thus been presented in separate chapters in this thesis. Finally, Chapter 6 provides a general discussion of the overarching findings of the research, their implications for athletes, and recommendations for future research.

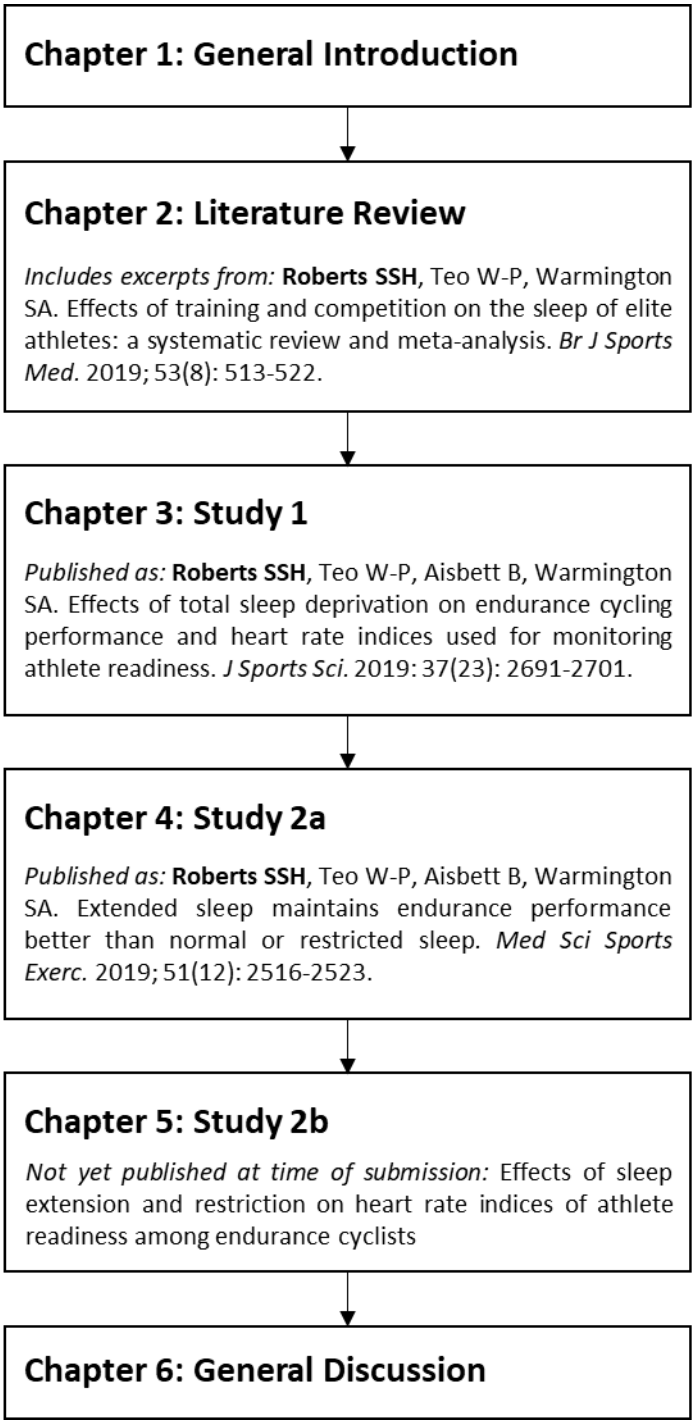

Figure 1.1. Thesis structure. Overall structure showing individual chapters, and associated publications arising from the thesis.

Chapter 2: Literature Review

Sections 2.3.3.1 and 2.3.3.2 of this chapter include excerpts published in:

Roberts SSH, Teo W-P, Warmington SA. Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis. *Br J Sports Med* 2019; 53(8): 513-522 (Appendix A).

2.1 Overview

This literature review comprises two main sections. The first examines the *endurance athlete*, including the key determinants of endurance performance, the demands of training and competition, and the importance of recovery. The second section identifies *sleep* as an important recovery behaviour, examining the psychophysiological benefits of sleep for recovering athletes, the sleep challenges faced during training and competition, and the available strategies for improving sleep. This section includes a critical evaluation of previous literature examining the effects of sleep on endurance performance, particularly among endurance-trained athletes, and highlights a need for research that systematically manipulates the sleep of endurance athletes to measure effects on performance. This is followed by an examination of tools used for monitoring sleep, and the barriers to their use in athletic populations. This section also reviews the sensitivity of HR to changes in sleep, thus rationalising an examination of the effects of sleep on HR indices commonly used to monitor athletes' readiness to perform. Although the sleep and performance of *endurance* athletes is of primary concern for this thesis, the paucity of data on such athletes occasionally necessitates a wider examination of the literature concerning athletes or athletic performance in general. In such cases, the limitations of drawing inferences for endurance athletes are acknowledged.

2.2 The endurance athlete

Aerobic endurance describes the ability to sustain dynamic exercise, such as running and cycling, using large muscle groups.[33] The energetic demands of aerobic endurance tasks are met predominantly by the combustion of carbohydrate and fat in the presence of oxygen (i.e., aerobic respiration).[34] Thus, any continuous effort lasting around 75 seconds or more, whereby > 50% of energy is derived from

aerobic respiration, can be considered an aerobic endurance task.[33] Compared with anaerobic pathways, the relative contribution of aerobic respiration to overall energy supply increases with the duration of the task, such that longer endurance tasks rely almost exclusively on aerobic respiration.[34] For instance, for a 60-minute cycling time-trial, more than 95% of energy is aerobically supplied.[35] This thesis examines prolonged aerobic endurance performance. For brevity, henceforth the term 'endurance' will be used when describing 'aerobic endurance'.

As a necessary foundation with which to explain the findings of this thesis, the following sections review the potential mechanisms of fatigue that limit muscular force production during endurance exercise (section 2.2.1), and how these fatiguing mechanisms are thought to be centrally integrated with cognitive factors to dictate 'race-pace' (section 2.2.2). Exercise economy is not examined in this thesis; however, for completeness, a very short section summarises its influence on endurance performance (section 2.2.3). This is followed by a brief review of the training and competition demands of endurance sports (section 2.2.4), which highlights the importance of recovery, and leads to a brief review of recovery strategies available to endurance athletes, whereby *sleep* is identified as a recovery behaviour requiring further examination (section 2.2.5).

2.2.1 Fatigue: the limits of muscular force production

The ability to prevent or delay 'fatigue' is critical for endurance performance.[36] Fatigue can be defined as an inability to maintain muscular force production,[37 38] typically in the presence of an increased perception of effort.[39] The biological basis of fatigue has been the focus of study for over a century,[40] with both peripheral (i.e., skeletal muscle) and central (i.e., central nervous system) origins identified.[41]

2.2.1.1 Peripheral fatigue

Peripheral fatigue occurs when skeletal muscle is unable to generate force despite unchanged or increasing neural drive.[42] The most influential model of peripheral fatigue, first postulated by Archibald Hill and colleagues in the 1920s, is the muscle anaerobiosis model.[43-45] This model suggests that when the oxygen demands of the *muscle* exceed oxygen supply from the *heart*, muscle anaerobiosis will proliferate lactate accumulation and inhibit muscle contraction and/or relaxation.[38] Subsequent research downplayed the role of lactate *per se*, and instead implicated other 'toxic' metabolites such as hydrogen ions (H^+) and inorganic phosphate (P_i).[46 47] However, this has also been contested based on studies examining *in situ* human muscle.[48] Nonetheless, the notion that muscle anaerobiosis creates a metabolic milieu that inhibits contraction and/or relaxation is central to the peripheral fatigue hypothesis. As such, any mechanism that inhibits the *supply* or *use* of oxygen may indirectly cause peripheral fatigue. For example, plasma volume expansion following total sleep deprivation has been speculated to impair endurance performance by decreasing haemoglobin concentration (i.e., haemodilution) and thus reducing oxygen delivery.[49] Likewise, the potential depletion of catecholamine stores following sleep loss,[50] may impair endurance performance by attenuating the sympathetic activation of cardiac muscle required for oxygen delivery.[51]

To avoid muscle anaerobiosis, endurance athletes require a high aerobic capacity[52 53]. Maximal oxygen uptake ($\dot{V}O_{2\,max}$) is the upper limit of aerobic respiration and reflects the heart's capacity to eject blood (i.e., cardiac output) and the muscle's ability to extract oxygen (arterial-venous oxygen difference). $\dot{V}O_{2\,max}$ typically ranges between 70-90 $mL \cdot kg^{-1} \cdot min^{-1}$ in elite male endurance athletes,[35 54 55] and is slightly lower in female athletes due in-part to differences in blood volume, body

fat percentage, and haemoglobin concentration.[36] Although endurance athletes often have high $\dot{V}O_{2\,max}$ values, little time during a prolonged (e.g., ≥ 60 minutes) endurance race is spent at an intensity that elicits $\dot{V}O_{2\,max}$, which would induce muscle anaerobiosis.[36] Rather, the highest sustainable pace during races typically corresponds to the maximal intensity at which energy supply remains almost exclusively oxidative. This intensity has been described as the second lactate, or anaerobic threshold (AT), and in this context, the AT is akin to 'critical power', or 'maximal lactate steady state'.[56] Typically, the relative exercise intensity (i.e., percentage of $\dot{V}O_{2\,max}$) at which AT occurs is dictated by the oxidative capacity of mitochondria and the metabolite removal capacity of capillaries.[57 58] In elite cyclists and rowers, respective AT values of 90% and 83% of $\dot{V}O_{2\,max}$ have been recorded,[59 60] and elite cyclists' AT strongly correlates (r = -0.9) with their time-trial performance (> 60 minute duration) at the Tour de France.[61]

2.2.1.2 Central fatigue

Central fatigue occurs when the central nervous system (CNS) is unable to recruit sufficient motor units to sustain muscular force.[62] Monitoring CNS activity during exercise is difficult, thus central fatigue has not been widely studied in athletes. Nonetheless, prolonged endurance exercise (e.g., 100-km cycling time-trial),[63] and/or hyperthermia[64 65] are thought to be two causes of central fatigue. In fact, a core temperature threshold (e.g., 39°C) may exist above which motor activity is inhibited.[64 66] Previous research found only ~50% of the potential activation of knee extensor muscles was achieved during maximal voluntary contraction after cycling to exhaustion in 40°C conditions, compared with ~ 80% of potential activation achieved after cycling in 18°C conditions.[65]

Serotonin is thought to be the primary neurotransmitter mediating central fatigue.[67-69] Prolonged exercise increases plasma levels of unbound tryptophan, a precursor of serotonin, which enters the CNS via the blood-brain barrier and increases brain serotonin levels.[64] Alpha-motoneurons responsible for activating skeletal muscle receive serotonergic inputs,[70] and increased serotonin may inhibit their activation.[67 69] Serotonin may also explain central fatigue induced by hyperthermia, as serotonergic neurons project to the hypothalamus, the thermoregulatory centre of the brain.[64] Another mechanism implicated in central fatigue is the depletion of brain glycogen.[71] Brain glycogen is an energy source for active neurons (e.g., motor neurons).[71] Brain glycogen reduces during prolonged exercise and may down-regulate motor unit recruitment to prevent energy depletion and neuronal death.[71] Whatever the precise mechanism, impaired endurance performances previously reported following sleep loss are unlikely the result of central fatigue,[24 26] as impaired performances did not coincide with changes in the activation of knee extensor muscles during maximal contraction following either total sleep deprivation,[26] or sleep restriction (i.e., a reduction in habitual sleep time).[24]

2.2.1.3 Mental fatigue

Models describing fatigue that is 'central' in origin have not traditionally distinguished between an *inability* of the CNS to recruit motor units, and an athlete's *unwillingness,* or *perceived inability,* to maintain muscular force.[66] This distinction has recently been made, and mental fatigue and central fatigue can thus be considered distinct phenomena.[72 73] Mental fatigue is recognised as a change in psychobiological state,[74] that leads to impaired cognitive performance and increased feelings of mental tiredness.[75] Magnetic resonance imaging has shown *central fatigue* is predominantly associated with reduced activation of the

supplementary motor area of the brain, whereas mentally fatiguing tasks affect the *pre*-supplementary motor area and *anterior cingulate cortex.*[73] This distinction is significant, as the supplementary motor area projects directly to motor neurons, whereas the *pre*-supplementary motor area projects to the pre-frontal cortex; a region synonymous with planning, decision making, problem solving, and self-control (i.e., executive functions).[76] Further, the anterior cingulate cortex plays an important role in emotion processing and self-regulation.[77]

Adenosine accumulation in the brain is thought to be the primary mediator of mental fatigue.[78] Adenosine is released as a by-product of ATP hydrolysis, and increased neural activity increases adenosine concentrations in the brain.[79] [80] Adenosine accumulates with prolonged wakefulness (e.g., sleep deprivation) and dissipates during sleep.[81] Its accumulation is also accelerated by intense physical activity.[82] As an inhibitory neurotransmitter, adenosine lowers brain activity and inhibits the release other neurotransmitters, such as dopamine.[78] [81] Figure 2.1, proposes that mental fatigue induced by adenosine accumulation impairs endurance by increasing the perceived effort of exercise.[78] Perceived effort is thought to reflect the neurocognitive processing of corollary signals sent from motor to sensory areas of the brain that signal the level of neural drive required to activate muscle.[83] Thus, the inhibitory action of adenosine may create a milieu whereby greater neural activation is required to produce a given motor output.[78] As an adenosine antagonist, caffeine is thought to improve athletic performance by blocking the inhibitory action of adenosine in the brain.[84] Figure 2.1 also shows that mental fatigue is typically associated with decreased motivation, likely caused by the decreased release (i.e., via the inhibitory action of adenosine) of dopamine in the brain.[78] The anterior cingulate cortex contains dopamine receptors that regulate

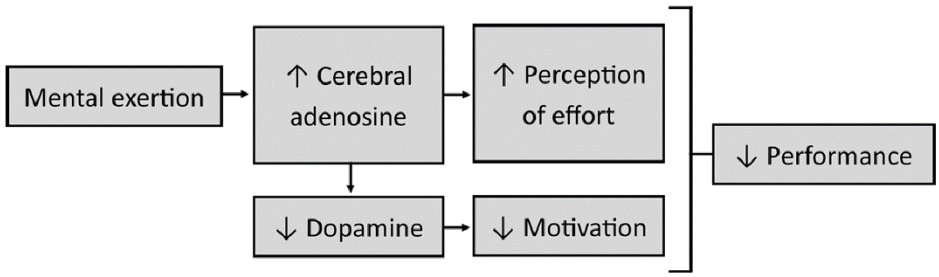

Figure 2.1. Schematic representation of the proposed mechanism of mental fatigue. Inhibitory action of adenosine in the brain directly increases perception of effort during exercise, and indirectly reduces dopamine release, which in-turn reduces motivation levels. Source: Martin et al.[78]

effort based decision making.[85] For example, infusion of a dopamine receptor antagonist into the anterior cingulate cortex of rodents reduced their preference for high-effort high-reward options in favour of low-effort low-reward options.[85] In humans, elevated dopamine increases the propensity to choose high-cost high-reward options (i.e., behaviour orientation), and increases the actual effort invested into a task (i.e., behaviour intensity).[86]

2.2.2 Integrated models for understanding endurance performance

In the 'real-world' of endurance sport, the race pace adopted by an athlete is rarely constant.[87] For example, the final sprint (i.e., "end spurt") observed in most endurance races is faster, and requires greater muscle activation, than the pace adopted for most of the race. Therefore, one-dimensional, 'linear' models implying concentrations of a given chemical directly, and absolutely, affect muscle contractility or efferent brain activity are too simplistic.[39] As such, models attempting to explain endurance performance suggest sensations of fatigue are centrally integrated with additional cognitive factors to dictate race pace.[39 88]

The Central Governor Model can be credited for triggering the paradigm shift away from one-dimensional models of fatigue, towards a focus on central integration.[89] The model postulates that in order to preserve whole-body homeostasis and prevent organ failure, an upstream 'governor' continuously regulates the number of motor units recruited during exercise.[39] It suggests that the subconscious brain selects the tolerable level of motor unit recruitment based on inputs from both feedback (e.g. H^+ levels, muscle glycogen) and feedforward (e.g., neurotransmitter levels) mechanisms.[38] When the integrated input suggests the current cost of exercise is a threat to homeostasis, an afferent signal is sent to the conscious brain triggering

sensations of fatigue, and increases in perceived effort, that discourage further muscle activation.[39] Thus, fatigue and perceived effort gradually increase as exercise intensity approaches the upper limit set by the central governor, ensuring task disengagement occurs before catastrophic organ failure.[38] According to the Central Governor Model, cognitive processing of situational factors (e.g., race importance, race distance, prior experiences), and the use of psychological skills (e.g., self-talk, imagery, goal-setting),[33 90] allow athletes to resist sensations of fatigue and increase muscle activation so long as there is no threat to homeostasis.[38]

More recently, the Psychobiological Model (PBM) has been proposed to explain endurance performance.[91] The PBM agrees that both central and peripheral factors influence muscle activation; however, it downplays the notion that a life-threatening disruption to homeostasis determines the upper limit of muscle activation.[92] Rather, the PBM, which is based on motivational intensity theory, suggests the effort invested in a task reflects the importance of success,[88] and that central and peripheral factors influence performance in-so-far as they alter either the *perceived effort* of the task, or *motivation* levels.[91 93] As such, the tolerable limit of muscle activation occurs when either (1) the perceived effort required to maintain muscular force is equal to the maximal effort the athlete is willing to exert to succeed, or (2) the perceived effort required to maintain muscular force is equal to the maximum effort the athlete perceives they are capable of producing.[94] Proponents of the PBM suggest the perceived effort of exercise reflects the level of neural drive required to activate muscle[95]. Therefore, peripheral fatigue (e.g., H^+ accumulation in muscle) may impair performance by increasing the neural drive, and thus the perceived effort, required to sustain muscular force[95], and mental fatigue may impair performance by both increasing the perceived effort of exercise,

and decreasing motivation (Figure 2.1).[78] The PBM suggests sensations of fatigue or pain do not directly down-regulate muscle activation, but rather indirectly affect muscle activation by reducing motivation (i.e., willingness to maintain force production).[95] According to the PBM, cognitive processing of situational factors and the use of psychological skills may alter either perceived effort or motivation to ultimately shift the exercise intensity that is tolerable during a race.[33 96] While integrated models explaining endurance performance continue to be revised,[89] the PBM is widely used and accepted for explaining endurance performance, and thus it will be used in this thesis to interpret findings.

2.2.3 Exercise economy: converting force into velocity

While the level of muscular force production sustained during an endurance task is important, exercise economy also influences race pace. Exercise *economy* refers to the metabolic (i.e., oxygen) cost of producing a given velocity,[36] and is similar to exercise *efficiency*, which refers to the metabolic cost of producing a given amount of mechanical work (e.g., Watts).[97] A more *economic* athlete uses less oxygen for a given velocity, and is thus able to conserve energy, or increase velocity, compared with a less economic competitor.[53] Differences in *economy* account for much of the variation in performance between endurance athletes.[98] For example, the oxygen cost, relative to $\dot{V}O_{2\,max}$, of running 15 km hour^{-1} is a significant predictor of elite triathlon performance.[99] Intrinsic factors including anthropometry (e.g., height, body composition), biomechanics (e.g., elastic energy, flexibility, technique) and physiology (e.g., muscle fibre composition) influence exercise economy.[58 100 101] While extrinsic factors associated with equipment (e.g., aerodynamics, shoe-surface interface) and the environment (e.g., altitude, wind) also influence exercise economy.

2.2.4 The demands of endurance training and competition

Endurance athletes undertake high training loads to improve fatigue resistance to physical work, and exercise economy.[102] Internal training and/or competition load is often quantified as the product of session duration and intensity, which can be measured subjectively (e.g., RPE) or objectively (e.g., HR).[103] Training loads of endurance athletes have increased significantly over the years; however, there has been a shift towards longer, less intense sessions.[104] Among elite endurance athletes, > 70% of training is typically undertaken at relatively low intensities (i.e., lactate < 2 $mmol \cdot L^{-1}$), with intensity significantly increasing as competition approaches.[105] Athletes also dedicate many hours to training; champion rowers train 12-15 hours per week,[106] while a professional cyclist, and an Olympic champion cross country skier, were both found to train up to 18 hours per week.[107] [108] An 80% increase in training load was observed over a five year period during which a road cyclist transitioned from an 'amateur' to a 'professional' racer[107]. In fact, road cycling is one of the most arduous endurance sports, with professionals pedalling > 30,000 km per year during training and competition, including 90-100 competition days.[52] [59] [109] During multi-day stage races, cyclists compete for 4-6 hours per day on consecutive days.[52] [59] [110] Given the high training and competition demands of endurance sport, it is no surprise that endurance athletes are susceptible to overuse injuries, including tendinitis, stress fractures, and back pain,[111-113] as well as upper respiratory tract infections caused by suppressed immune function.[114] Moreover, the psychological stress of high training loads has been associated with elevated levels of anxiety among endurance athletes.[115] Thus, in order to tolerate the demands of training and competition, and optimise performance, it is important that endurance athletes prioritise recovery.

2.2.5 The recovery of endurance performance

Recovery can be defined as a multi-faceted (e.g., psychological, physiological, social) time-sensitive process required to re-establish athletic performance.[116] Sufficient recovery restores or helps improve performance,[117] whereas insufficient recovery leads to performance stagnation, accumulated fatigue, and in severe cases, injury and illness.[118] [119] The time-course of recovery is athlete-specific, and depends on the severity of psychophysiological stress incurred during training and competition.[116] Approaches to recovery can be passive, active, or proactive.[120] [121] Passive recovery requires little or no active involvement from the athlete, and includes sleeping, resting, and interventions *applied* to an inactive athlete, such as massage, compression garments, water immersion, and cryotherapy.[120] Active recovery typically involves light physical activity immediately after training or competition (i.e., "cool down") that is primarily intended to facilitate removal of the metabolic by-products of exercise.[120] [122] While active recovery involves action-oriented behaviours that are typically prescribed by practitioners, *proactive* recovery refers to *self-determined* action-oriented behaviours that an athlete deems important for recovery; and can include light physical activity, diet choices, social activities, relaxation techniques, and sleep hygiene.[120]

Although many strategies / interventions are purported to promote recovery,[116] [123] [124] research does not always support their use among endurance athletes.[125] [126] For example, both compression garments,[125] and massage,[126] have demonstrated little positive effect on the recovery of endurance performance. In contrast, it is well-established that adequate dietary protein and carbohydrate is essential for the recovery of endurance performance.[127] [128] For example, endurance exercise depletes muscle glycogen, the predominant energy source during high-intensity

endurance tasks, thus post-exercise carbohydrate ingestion restores muscle glycogen and performance capacity.[129] Another recovery behaviour that is generally associated with physical and mental well-being is *sleep.*[130] While sleep itself is a *passive* recovery behaviour, *proactive* behaviours, such as the adoption of healthy sleep hygiene, or the deliberate extension of time-in-bed, can improve the quality and / or quantity of sleep.[18 20 131] However, few studies have examined the effects of sleep on the recovery of endurance performance, especially among endurance athletes.[24-26]

2.3 Sleep: an important recovery behaviour

This section begins by outlining the characteristics and regulatory mechanisms of normal human sleep (section 2.3.1). This is followed by a review of the potential psychophysiological benefits of sleep for recovering athletes (section 2.3.2). An examination of *sleep loss* in athletic populations follows, which includes a discussion of some of the causes of sleep loss among athletes, as well as a critical evaluation of previous research examining the effects of sleep loss on endurance performance (section 2.3.3). An examination of *sleep extension* in athletic populations discusses strategies for extending athletes' sleep, and provides a critical evaluation of previous research examining the impact of sleep extension on athletic performance (section 2.3.4). The final section reviews current approaches and barriers to monitoring sleep and its effects on athletes. This section highlights that despite the popularity of HR indices for monitoring athletes' readiness, an investigation of their sensitivity to any effects of sleep on athletes' readiness has not been undertaken (section 2.3.5). Figure 2.2 provides a schematic representation of the topics discussed in the following sections, and highlights the specific line of inquiry (i.e., in bold) that is the focus of this thesis.

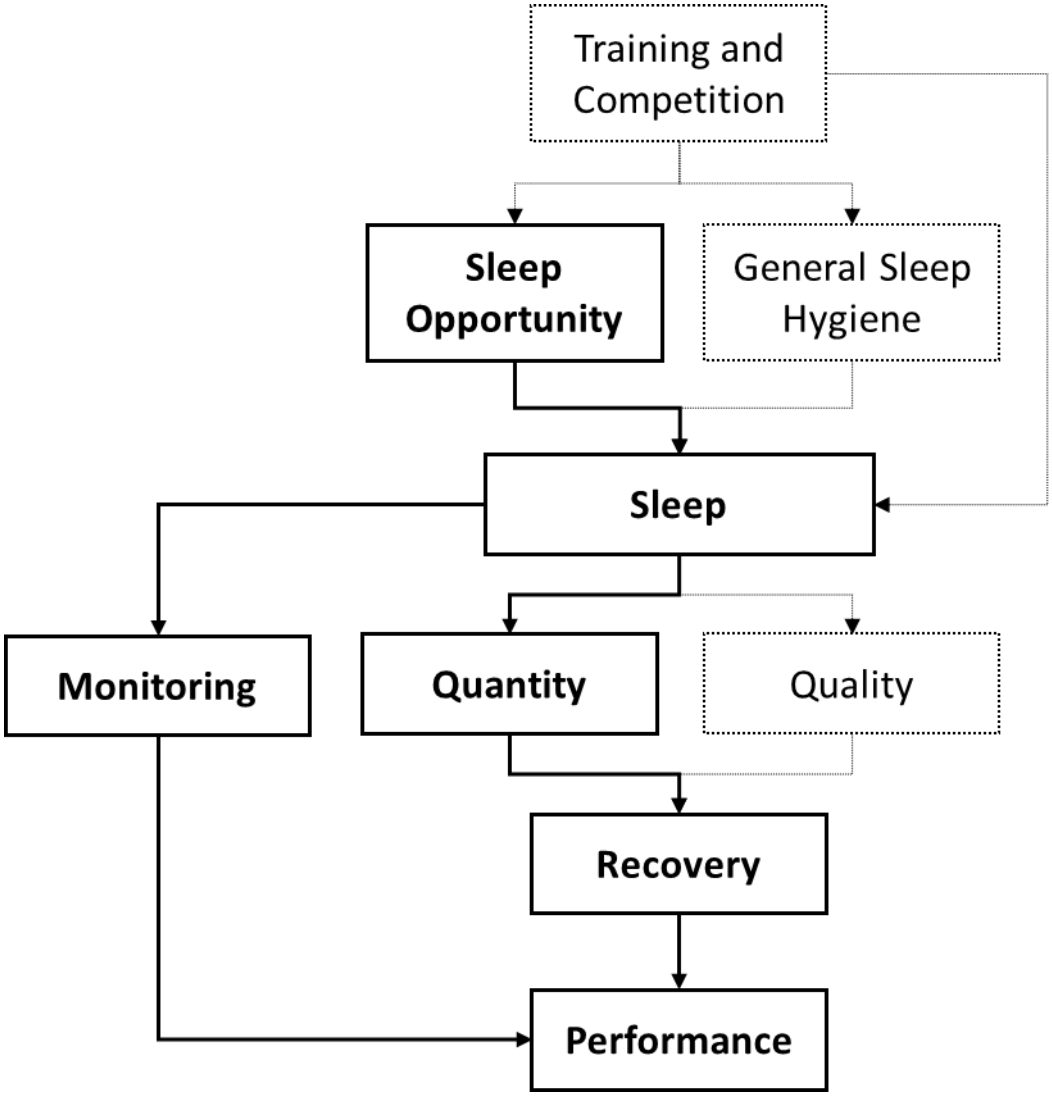

Figure 2.2. Schematic representation of factors affecting athletes' sleep and their relationship with recovery and performance. Factors related to training and competition may negatively affect sleep either directly (e.g., hyperarousal due to nervousness) or indirectly by reducing sleep opportunity (e.g., early morning training) or interfering with healthy sleep hygiene (e.g., athletes consuming caffeine before evening competition). In contrast, efforts to increase sleep opportunity (e.g., increase time-in-bed, napping) and educate athletes on sleep hygiene can positively affect sleep. Sleep can be assessed by measuring either sleep quantity (i.e., total sleep time) or quality (e.g., frequency of arousals across the night). Both sleep quantity and quality may influence recovery (e.g., via effects on metabolism, cognition, immunity, and brain plasticity) and thus affect subsequent performance. Given the likely impact of sleep on athletes' recovery and performance, there is a desire to monitor sleep, and its effects on recovery and performance. Pathway in bold identifies the line of inquiry that is the focus of this thesis.

2.3.1 Normal human sleep

Sleep is a reversible behavioural state characterised by immobility and reduced perceptual awareness of the environment.[132] There are two separate sleep states; rapid eye movement (REM) and non-REM sleep.[133] Non-REM sleep is comprised of four stages, whereby the depth of sleep and arousal threshold increases with each stage, such that stages 1 and 2 constitute shallow sleep, and stages 3 and 4 constitute deep or slow wave sleep (SWS).[133] REM sleep is characterised by high levels of brain activity and complete loss of muscle tone.[134] A normal sleep cycle involves a progression through the stages of non-REM sleep followed by a brief period of REM sleep.[135] This process takes approximately 70-120 minutes and is repeated throughout the night.[132] Within cycles, the time spent in each sleep stage changes across the sleep period, with SWS concentrated in earlier cycles and REM sleep concentrated in later cycles.[135]

The regulation of sleep is attributed to the interaction of two processes; the homeostatic process (i.e., sleep need or pressure) and the circadian process (i.e., biological clock).[136 137] This two-process model, shown in Figure 2.3, suggests the homeostatic need for sleep, which accumulates during wakefulness and dissipates during sleep, interacts with daily oscillations in physiologic, behavioural and cognitive functions (i.e. circadian variations) to determine sleep and wake thresholds.[138 139] Adenosine is the primary neurotransmitter moderating the homeostatic process by inhibiting the action of wake promoting neurons (e.g., cholinergic neurons).[81] It accumulates in the extracellular space of the brain during wakefulness as a by-product of the ATP breakdown required for brain activity.[81] A cluster of hypothalamic neurons called the superchiasmatic nucleus (i.e., circadian pacemaker') controls the circadian process.

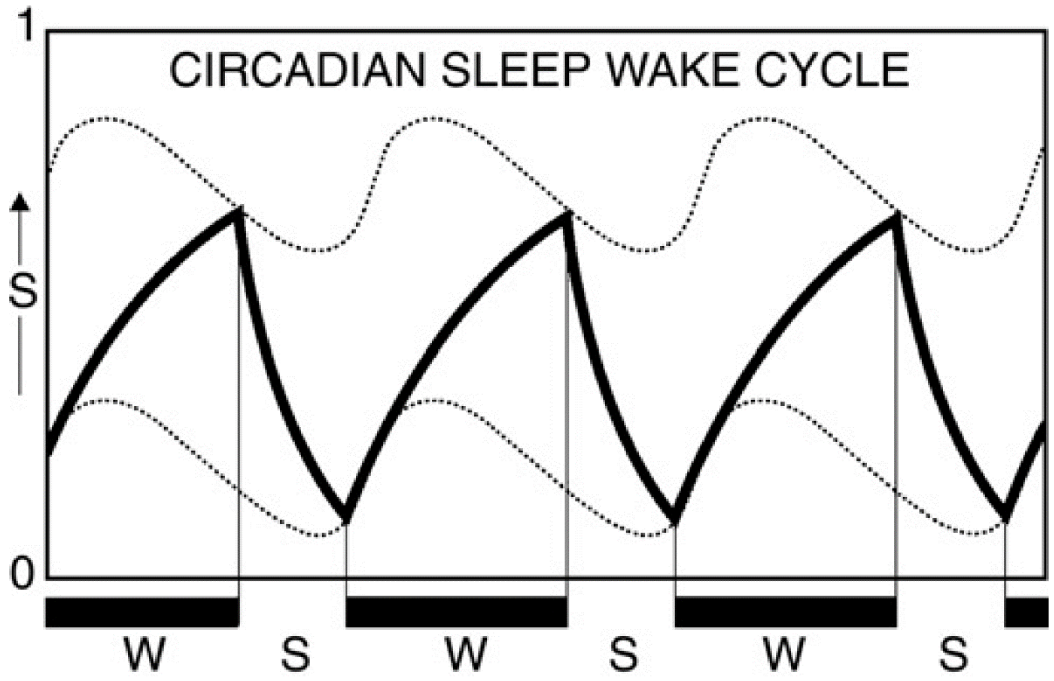

Figure 2.3. The two-process model of sleep regulation.

The two-process model of sleep regulation. Thick black line represents sleep need (S), which increases during wakefulness (W-abscissa) and decreases during sleep (S-abscissa). Its variation is restricted to a range (dotted lines) which represent fluctuations in the arousing influence of the circadian process. Sleep need increases over time until the current upper limit of circadian arousal is reached (i.e., arousal level not high enough to prevent sleep onset). Sleep need decreases during sleep until the lower limit of circadian arousal is reached (i.e., arousal level not low enough to prevent waking). Source: Beersma & Gordijn[139]

The molecular oscillations of the superchiasmatic nucleus are principally entrained by the light-dark cycle, such that exposure to light in the morning synchronises circadian rhythms with the external day / night environment.[140]

Although the two-process model identifies the main processes involved in sleep-wake regulation,[141] several additional factors affect sleep tendency.[136 142 143] For example, sleep inertia has been included as a 'third process' in some models for its effects on sleep tendency immediately after waking.[142] While frequent (i.e., hourly) so-called ultradian rhythms, such as the cycling between Non-REM and REM sleep states, are also known to influence sleep-wake thresholds.[143] Finally, the effects of decisions, stress, and even pain can transiently modulate the underlying circadian process and alter sleep-wake thresholds.[136]

2.3.2 Recovery benefits of sleep for athletes

Athletes identify sleep as an important recovery behaviour for athletic success,[10] and indeed there is *some* empirical evidence to support this (sections 2.3.3.3, 2.3.4.2, and 2.3.4.3). There is also evidence that sleep may moderate injury risk.[144-147] A survey of national age-group athletes found sleeping > 8 hours per night reduced injury risk by 61%.[144] And among college athletes, daytime sleepiness greatly increased the likelihood of sustaining a sports-related concussion.[147] There are many physiological processes occurring during sleep that may augment recovery.[8] These processes broadly affect (1) metabolic functions (2), cognitive functions, (3) immune functions, or (4) brain plasticity.

2.3.2.1 Sleep and metabolism

The metabolic functions of sleep, particularly SWS, may be crucial for athletes needing to conserve and replenish energy,[148 149 150] as well as promote tissue growth

and repair.[151 152] All physiological processes require energy, and sleep may have evolved as a strategy to conserve energy when food was scarce.[134 148] The reduction in energy expenditure during sleep corresponds to the energy expended during prior weakefulenss.[149 150] Indeed, SWS has the lowest metabolic rate of all sleep stages,[153] and increases as a proportion of total sleep time when daily energy expenditure increases.[149] Sleep also removes metabolic waste from the brain,[154] and replenishes brain glycogen stores, a critical energy substrate required for brain activity.[148] In fact, adenosine accumulates in the brain as a by-product of ATP hydrolysis,[124 129] and this accumulation of adenosine drives sleep propensity and increases the expression of SWS during subsequent sleep that is necessary for brain glycogen resynthesis.[148 155 156] Sleep also up-regulates anabolic pathways.[151 152] Growth hormone and testosterone promote tissue growth / repair by stimulating protein synthesis and 24-hour plasma levels of these hormones peak shortly after sleep onset, during the first episode of SWS.[157-159] In contrast, sleep disruptions supresses nocturnal growth hormone secretion.[160 161] SWS is also negatively correlated with the release of the catabolic hormone cortisol,[162 163] a key hormone that signals skeletal muscle atrophy.[164] To illustrate the importance of SWS for athletic recovery, Figure 2.4 shows that when the sleep of six experienced marathon runners was monitored for four nights following a 92-kilometre ultra-marathon, SWS as a percentage of total sleep time increased.[165] Similarly, when the sleep of elite female swimmers was monitored across an entire competitive season, SWS formed a high percentage of total sleep time during peak training periods (i.e., 31%) but was significantly reduced following pre-competition taper (i.e., 16%).[166] More recently, adolescent soccer players training 14 hours per week obtained 6% more SWS and 5% less REM sleep than controls training just 1.5 hours per week.[167]

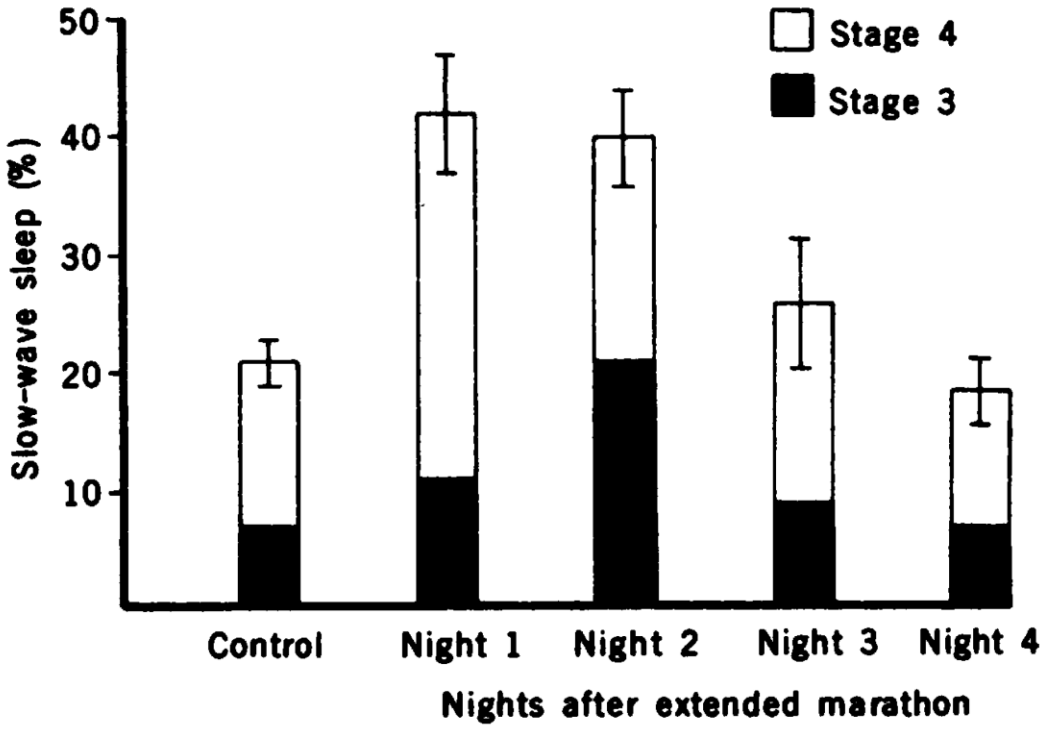

Figure 2.4. Slow wave sleep as a percentage of total sleep before and after completion of a 92-km ultramarathon. Control reflects mean of two nights sleep, one recorded two weeks prior to-, and another two weeks post, the ultramarathon. Source: Shapiro[168]

2.3.2.2 Sleep and cognition

Sleep loss impairs daytime cognitive function, perhaps due to an accumulation of metabolic by-products, such as adenosine, that inhibit brain activity.[78 81] Indeed, sleep loss decreases metabolic activity in the brain, particularly in the thalamus, the pre-frontal, and the anterior cingulate cortex.[169 170] These regions govern many cognitive processes including executive functions, emotions, and attention.[170] Consequently, sleep loss impairs decision making, particularly in situations that require innovative solutions, or flexibility in changing environments.[171 172] Athletes are routinely confronted by such situations, particularly in sports involving an adversarial opponent (e.g., team sports).[173] Sleep loss also impairs reaction time and / or psychomotor vigilance.[174] Sleep restriction for a single night slowed the reaction times and decreased the attentional capacities of handball goalkeepers.[175] Such effects not only have implications for goalkeepers, but also for other athletes reliant on reaction time and attentional capacity (e.g., cricketers, motor racing drivers).[7] Seep loss also increases feelings of depression, confusion, and mood disturbance.[174 176 177] Indeed, inadequate sleep is believed to be a key contributing factor to poor mental health among athletes.[28 178] Sleep disturbances have been associated with feelings of fatigue in overreached triathletes,[179] and decreased total sleep time during training negatively affected subjective wellbeing in female youth athletes.[180] Sleep loss also impairs the ability to communicate effectively and to demonstrate empathy,[171] which may impair the ability to develop strong, productive relationships with coaches and teammates.

2.3.2.3 Sleep and immunity

The relationship between sleep and immune function is well-established.[130] Healthy sleep improves resistance to infection,[181] and the time-course of wound healing.[182] Plasma levels of pro-inflammatory cytokines peak during SWS.[130 183 184] In fact, several pro-inflammatory cytokines have been shown to promote SWS.[185] Therefore, in addition to the metabolic factors discussed, increases in pro-inflammatory cytokines, which have been observed following endurance training among rowers,[186] may explain higher amounts of SWS in athletes.[165 166] The pro-inflammatory milieu of SWS may help repair the cellular damage induced by training or competition, and defend against infection.[130] Indeed, sleep disturbances have been associated with a higher risk of infection among triathletes.[179] Given endurance training can supress immune function and increase the risk of upper respiratory tract infection,[187] healthy sleep is likely crucial for the immunity of endurance athletes.[181] While a transitory pro-inflammatory state *during sleep* may promote acute recovery, a persistent and systemic increase in inflammation is associated with inadequate recovery and overtraining.[188] Sleep restriction has been shown to increase 24-hour levels of pro-inflammatory cytokines,[188 189] and may thus contribute to the chronic inflammation that is believed to explain many of the symptoms of overtraining.[176] For example, chronic inflammation increases plasma levels of catecholamine and cortisol,[188] hormones which can negatively affect mood and up-regulate catabolic metabolism.[164 188 190]

2.3.2.4 Sleep and brain plasticity

Sleep helps to process and commit to long-term memory information acquired throughout the day.[191] Both declarative (e.g., recall of events),[192 193] and procedural memories (e.g., motor skills)[194 195] are consolidated during sleep. For example,

improvements in motor skill execution following sleep are greater than those observed over a similar timeframe without sleep.[196] Even daytime naps as short as six minutes have been shown to improve free recall.[193] Individual sleep stages may influence learning and memory in different ways; however, most sleep stages seem to play a role in brain plasticity.[196 197] For example, the extent of overnight motor skill improvement recorded after exposure to a motor task has been strongly correlated with the percentage of subsequent Stage 2 Non-REM sleep ($r = 0.66$, $P < 0.05$).[196] The effects of sleep on brain plasticity are likely important for the technical (e.g., skill acquisition) and tactical learning of athletes. Accordingly, sleep extension (i.e., increased habitual sleep time) has been shown to improve the shooting accuracy of college basketballers,[198] and the serving accuracy of college tennis players[23].

2.3.3 Sleep loss in athletic populations

Although sleep is considered an important recovery behaviour,[8 10 199] athletes are vulnerable to sleep disturbances,[14 200] and often fail to achieve the minimum 7 hours per night at 85% sleep efficiency (i.e., percentage of time-in-bed spent asleep) recommended for optimal health.[13 201 202] The author recently published a systematic review and meta-analysis in the *British Journal of Sports Medicine* characterising athletes' sleep, and factors associated with training and competition that negatively affect sleep (Appendix A).[13] The following sections (sections 2.3.3.1 and 2.3.3.2) include excerpts from the 'results' and 'discussion' of that publication, and highlight that sleep loss is common and sometimes unavoidable in athletes. For brevity, only sections discussing commonly experienced sleep challenges that may affect endurance athletes are included. For a more comprehensive discussion of the sleep challenges faced by athletes, including the

effects of hypoxia, and air-travel, the reader is referred to the published paper,[13] and other reviews by Gupta and colleagues,[14] and Nédélec and colleagues.[8] For consistency within the thesis, the author has made minor changes to the structure and syntax of published excerpts. Following sections discussing sleep challenges faced by athletes, there is a critical review of research that has examined the effects of sleep loss on endurance performance (section 2.3.3.3).

2.3.3.1 Effects of competition on athletes' sleep

Qualitative assessments suggest pre-competition sleep disturbances are prevalent among athletes.[200 203 204] However, the author's systematic review and meta-analyses did not find consistent objective evidence of this.[13] Compared with training, total sleep time was shorter the night before a race among cyclists,[205] but longer the night before a match among Australian rules footballers.[206 207] Individual-sport athletes were reported to have shorter sleep times, and lower sleep efficiency than team-sport athletes during out of competition training.[208] However, when sleep was examined prior to competition, comparisons of individual- and team-sport athletes have delivered equivocal findings.[203 200] Nonetheless, it is suggested that individual-sport athletes,[203] particularly those competing in aesthetic-sports (e.g., gymnastics), may be most susceptible to sleep disturbances.[209] In addition, while it has been found that sleep efficiency during training was lower in males compared with females,[210] there is some evidence that prior to competition, sleep disturbances are more prevalent among females.[203 209] The principal cause of pre-competition sleep disturbance is nervousness or thoughts about competition.[200 203] Individual-sport athletes (e.g., most endurance athletes) may experience particularly high levels of anxiety because they cannot share the psychological burden of competition with team members,[203 208 209] which may be

further exacerbated in aesthetic-sport athletes whose success depends on the judgement of others.[209]

Athletes rarely achieve total sleep time or sleep efficiency recommendations on the night of competition.[13] Total sleep time is typically 60 minutes shorter the night of competition compared with pre-competition nights (i.e., the 1-2 nights immediately preceding competition day).[13] Moreover, on the night of *evening* competition (i.e., start time ≥ 6 pm), sleep time is typically 80 minutes shorter and sleep efficiency 3-4% lower, than pre-competition nights.[13] Reductions in sleep time the night of competition are often attributed to a delay in bedtime that reduces overall time-in-bed.[211-216] Several factors may explain delayed bedtimes, and lower sleep efficiency, on the night of competition. For example, increased circulating cortisol,[217] sympathetic hyperactivity,[218] elevated core body temperature,[219] and muscle pain,[220] may persist post-competition and increase arousal.[221 222] Cortisol levels following a race have been strongly ($r = -0.90$, $P < 0.05$) correlated with sleep efficiency in swimmers.[223] While elevated cortisol post-competition was associated with a reduction in total sleep time the night of competition in netballers.[217] Additionally, the ergogenic use of caffeine[224], and exposure to bright light (e.g., stadium lighting), may interfere with neurological processes that promote sleep.[84 225] In support, increases in salivary caffeine on match day have been moderately correlated with increases in sleep latency ($r = 0.53$, $P < 0.05$), and decreases in sleep efficiency ($r = 0.52$, $P < 0.05$) in rugby union players.[224] Finally, delayed bedtimes may be attributed to post-competition recovery / medical interventions, meetings, travel, and media commitments.[226 212]

2.3.3.2 Effects of training on athletes' sleep

Total sleep time is shorter prior to training days than prior to rest days,[212 227-229] particularly when training starts before 0700, as in rugby league players during pre-season,[212] and rowers / swimmers preparing for competition.[227 229] A study that examined the effect of different training start times found athletes who started training between 0500 and 0600 obtained less than 5 hours of sleep, and those who started between 1000 and 1100 obtained more than 7 hours.[228] It is speculated that shorter sleep times reported for individual-sport athletes compared with team-sport athletes may reflect a tendency for individual sports to commence training early in the morning.[208] While athletes try to offset the effects of early training by going to bed earlier, it is often difficult to prevent reductions in total sleep time.[228] For example, on a training camp, rowers woke approximately 2.5 hours earlier on training days compared with rest days, but went to bed just 25 minutes earlier the night before.[227] The inability to go to bed early has been attributed to the fact that most social engagements are scheduled in the evening, and the so-called 'forbidden zone' - a period of heightened arousal mediated by the body's circadian rhythms - typically reduces sleep propensity between 2000 and 2200.[228 230]

Total sleep time and sleep efficiency have been reported to reduce following large increases (>25%) in training load.[179 231-233] In contrast, most studies that suggest sleep is unaffected, or even improves with higher training loads have not examined large increases in load, but rather subtle within-subject changes in load.[166 212 234 235] Therefore, reductions in total sleep time and sleep efficiency may reflect a negative adaptation to training in response to large increases in training load. Heavy training can lead to increases in circulating cortisol,[236] and sympathetic activity,[237] both of which may prevent the normal down-regulation of the human stress systems (i.e.,

hypothalamic-pituitary-adrenal, sympathy-adrenal-medullary) required for healthy sleep.[238 239] In support, changes in resting alpha-amylase, a marker of sympathetic activity, have been moderately correlated ($r = -0.54$, $p < 0.05$) with changes in sleep efficiency among synchronised swimmers.[232] It is also possible that increases in training load may disturb sleep via pain associated with muscle soreness, and frequent micturition in the night associated with rehydration efforts.

2.3.3.3 Sleep loss and endurance performance

This thesis examines effects of both total sleep deprivation and sleep restriction (i.e., reduced habitual sleep time) on the performance of endurance athletes. Therefore, this section critically reviews previous literature examining effects of total sleep deprivation and sleep restriction on endurance performance. Although effects on the performance of endurance athletes is of primary interest, this section also includes studies that examined untrained populations.

Two studies have investigated effects of *total sleep deprivation* on the performance of *endurance* athletes, and these studies have contradictory findings.[25 26] Compared with normal sleep, one night without sleep reduced time-to-exhaustion in endurance athletes by ~1.5 minutes across a ~20-minute incremental cycling test that followed a 40-minute preload exercise period at 50-65% maximal aerobic power.[26] Whereas 25-30 hours of sleep deprivation did not affect time-to-exhaustion in distance runners during a short ~13-minute incremental cycling test.[25] A limitation of these studies is that performance was assessed using time-to-exhaustion protocols whereby exercise intensity was externally controlled.[25 26] Such protocols have less ecological validity for endurance athletes who typically compete in self-paced time-trials or races.[240] Another study that did not strictly

examine endurance athletes found distance covered by *team-sport* athletes during a 50-minute intermittent-sprint protocol was lower after 30 hours of total sleep deprivation than after normal sleep.[241] However, again, the intermittent nature of this testing protocol has limited ecological validity for endurance athletes who undertake sustained efforts (e.g., road cyclists, triathletes).[240]

Most studies examining effects of *total sleep deprivation* in *untrained* populations have reported impaired endurance performances.[25 242-245] Sleep deprivation has been found to reduce running, walking, and cycling time-to-exhaustion.[25 242-244] A single night of sleep deprivation also led to a 3% reduction in distance covered by active males during a 30-minute treadmill run that followed a 30-minute preload run at 60% $\dot{V}O_{2\,max}$.[245] Despite these findings, some studies have found total sleep deprivation did not impair endurance performance.[246 247] Sixty hours without sleep did not affect time-to-exhaustion in female students undertaking a ~ 20-minute incremental cycling test.[246]

Three studies have investigated the effects of *sleep restriction* on the performance of *endurance* athletes, and these report contradictory findings.[24 248 249] Compared with 7.1 hours sleep, a single night of 2.4 hours sleep prior to a 3-km cycling time-trial slowed the performance of trained cyclists by 4%.[24] While this study used a time-trial to assess performance, it's duration was relatively short compared with the prolonged efforts (e.g., ≥ 60 minutes) often undertaken by endurance athletes (e.g., cyclists). In addition, the severe sleep restriction protocol (i.e., > 4-hour reduction in sleep time) used has limited ecological validity as athletes typically experience more subtle sleep restriction (i.e., < 3-hour reduction in sleep time) during training and competition.[13] Other studies examining effects of sleep

restriction on endurance athletes also used severe sleep restriction protocols. For example, the maximal workload achieved by endurance athletes during an incremental test that followed a period of constant-load submaximal exercise decreased by 5% (i.e., from 309 to 293 W) when prior sleep opportunity was reduced by four hours (total sleep time not reported),[248] but was unaffected when prior sleep opportunity was reduced by three hours.[249]

Four studies have investigated the effects of *sleep restriction* on the performance of *untrained* participants.[250-252] Two studies assessed performance by recording distance covered during an intermittent running test.[251 252] Compared with normal sleep, reducing sleep opportunity to 3-4 hours for one night (total sleep time not reported) did not affect distance covered in the morning,[251] but reduced distance covered in the evening.[252] Two studies have investigated the impact of consecutive nights of sleep restriction on endurance performance,[250 253] but none of these has examined endurance athletes despite the fact that endurance athletes routinely train, and compete (e.g., cycling tour races), on consecutive days. Nonetheless, compared with normal sleep, three consecutive nights of 2.5 hours sleep did not affect time-to-exhaustion of healthy males during a short incremental treadmill test.[250] Similarly, 400-m swim performances over four consecutive days were unaffected when sleep was restricted to 2.5 hours per night on intervening nights compared with when participants slept normally on intervening nights.[253] Importantly, while participants were identified as ‘swimmers’, no information on training history was provided, and finishing times > 7 minutes suggest they were not well-trained.[253]

Several limitations of previous research justify further investigation of the effects of sleep loss on endurance performance. First, few studies have examined effects

of sleep loss on the performance of *endurance athletes*, and these studies report contradictory findings.[24-26 248 249] Second, sleep restriction protocols have tended to reduce nightly sleep time by ≥ 3 hours,[24 248 251 252 250 253] which has limited ecological validity in athletes, who typically experience more subtle sleep restriction.[13] Third, while two studies have examined the effects of sleep restriction over consecutive nights on the performance of untrained participants,[250 253] no such study has been undertaken on endurance athletes. Fourth, most research to date has not monitored sleep prior to experimental interventions,[24-26 248 249] and thus findings may be confounded by discrepancies in cumulative sleep time (e.g., an accumulated sleep debt) between experimental conditions that occurred *prior* to testing.

Finally, a primary criticism of research to date is that most studies examining the effects of sleep loss on endurance performance have used time-to-exhaustion or workload-at-exhaustion tests.[25 26 242-244 248 249] These tests have limited ecological validity for endurance athletes.[240] Cyclists routinely compete in time-trials but rarely in events that require cycling at a constant-load until exhaustion. Compared with protocols in which intensity is externally controlled, time-trials require self-regulation of pacing, and thus have different psychological demands. In cyclists, plasma glucose oxidation was greater during variable-intensity exercise (i.e., time-trials) than during constant-load exercise of the same duration and mean power output.[254] Previous research found no significant correlation between time-to-exhaustion at ventilatory threshold and cycling performance during a triathlon,[255] but a strong correlation ($r = 0.8$, $P < 0.05$) between time-trials undertaken in laboratory and 'real world' competition settings.[256]. Importantly, time-trials also have better internal reliability than time-to-exhaustion tests.[240] When the reliability of protocols of similar duration (i.e., one hour) and intensity were compared in

endurance athletes, the mean coefficient of variation for finishing time was 27% for a time-to-exhaustion test but 3% for a time-trial.[257]

In summary, the literature seems to suggest sleep loss is more likely to impair endurance performances of longer duration (e.g., > 30 min); following more severe sleep loss (e.g., > 3 hours); or following extended periods of wake after sleep loss. However, several limitations of previous research justify further examination of the effects of sleep loss on endurance performance:

1. Few studies have investigated the effects of total sleep deprivation or sleep restriction on the performance of endurance athletes, and these report contradictory findings.[24-26 248 249]
2. Studies examining effects of sleep restriction on endurance performance have adopted severe sleep restriction protocols (e.g., ≥ 3 hour reduction in sleep time / sleep opportunity)[24 248 251 252 250 253] that have limited ecological validity in athletes who typically experience more subtle sleep restriction (e.g., < 3 hour reduction in sleep time).[13]
3. Although endurance athletes train and occasionally compete (e.g., road cyclists) on consecutive days, no prior research has investigated the cumulative effects of sleep restriction over consecutive nights on the performance of endurance athletes.
4. Studies have typically not monitored sleep prior to experimental interventions,[24-26 248 249] and so it is possible that differences in sleep prior to interventions have confounded findings.
5. Studies that have examined effects of sleep loss on the performance of endurance athletes have used short (< 15-minute) performance tests,[24 25]

and/or time-to-exhaustion / workload-at-exhaustion tests,[25 26 248 249] that have limited ecological validity for many endurance athletes.

2.3.4 Sleep extension in athletic populations

Sleep extension refers to an increase in habitual sleep time that is typically prescribed under the assumption that a residual sleep debt exists.[20 258] Athletes may incur a sleep debt due to inadequate habitual sleep (i.e., chronic sleep debt), or an acute disruption to habitual sleep, such as on the night of competition (i.e., acute sleep debt). In either case, sleep extension will help resolve, or at least reduce, the accrued sleep debt.[258] Practitioners and researchers have explored strategies to extend athletes' sleep, which typically focus on increasing sleep opportunity (i.e., via extending nightly time-in-bed, or allowing daytime nap opportunities) and / or sleep hygiene education. However, despite growing interest in extending athletes' sleep, relatively few studies have examined the effects of sleep extension on the sports-specific performance of trained athletes.[20 23] The following sections examine strategies for extending athletes' sleep (section 2.3.4.1), and review literature examining the effects of both nightly sleep extension (section 2.3.4.2) and daytime napping (section 2.3.4.3) on athletic performance.

2.3.4.1 Strategies for extending athletes' sleep

Providing increased opportunities for sleep, via extending nightly time-in-bed, or undertaking daytime naps, is essential for facilitating sleep extension.[20 23 259] In college basketballers, prescribing a minimum 10 hours in bed per night increased nightly sleep time from 6.7 to 8.5 hours per night.[20] While in college tennis players, prescribing 9 hours in bed per night increased self-reported sleep time from 7.1 to 8.9 hours per night.[23] Moreover, when rugby players were encouraged to sleep 10

hours per night during pre-season, total sleep time increased by 6.3%.[259] In addition, among soccer players, sleep obtained during a two-hour daytime nap opportunity was able to compensate for sleep lost by reducing nightly time-in-bed by two hours.[260] Interestingly, to date, studies aiming to extend sleep have prescribed absolute time-in-bed targets (e.g., 10 hours per night) for all athletes. While this has proven effective, such an approach does not account for individual differences in sleep needs or lifestyle preferences. For example, absolute time-in-bed targets for some athletes (e.g., anxious athletes, genetically short sleepers)[261] may be counterproductive, as excessive time-in-bed is a key behavioural mechanism known to perpetuate insomnia.[262] Therefore, it is possible that individualised approaches to sleep extension, which may include relative increases in nightly time-in-bed (e.g., 30% increase in habitual sleep time), and / or the tailoring of bedtimes and get-up times to individual chronotype, may be more effective in applied sport settings. Accordingly, the research in this thesis adopts an individualised approach to prescribing sleep extension.

Sleep extension is also facilitated by adopting behaviours that promote healthy sleep (i.e., sleep hygiene). Such behaviours include maintaining a consistent sleep / wake schedule, exposure to natural light during the day, maintaining a cool (e.g., 16-20° C), dark, and quiet bedroom, avoiding screens and reducing light in the evening, and avoiding caffeine in the late afternoon.[16 263-265] A 60-minute sleep hygiene education session, that included advice on relaxation strategies (e.g., progressive muscle relaxation), increased total sleep time among elite netballers by an average of 22 minutes per night during pre-season.[131] Similarly, sleep hygiene advice increased total sleep time following strenuous training among professional tennis players.[266] While a crossover study examining semi-professional soccer

players found that a sleep hygiene intervention increased total sleep time from four to six hours on the night of a match.[19] Sleep hygiene education can also improve other aspects of athletes' sleep.[18 16 17] For example, sleep hygiene education improved sleep efficiency among professional Australian rules footballers,[18] and combining sleep hygiene education with individualised advice informed by responses to the Athlete Sleep Behaviour Questionnaire – a tool developed to identify maladaptive sleep habits in athletes - improved the sleep efficiency, sleep onset latency, and sleep onset variance, of international cricketers.[16 17]

In addition to examining the effects of general sleep hygiene, researchers have also examined the impact of isolated behaviours or interventions on athletes' sleep, such as electronic device use,[267-269] red-light treatment,[270] diet,[271] brainwave entrainment,[272] mattress quality,[273] muscle relaxation,[274] cryostimulation,[232 275] and hot showers.[276] For example, although avoidance of light emitting devices before bed is recommended, the use of such devices may not affect *athletes'* sleep.[267 277] In contrast, progressive muscle relaxation reduced sleep onset latency in dancers with high anxiety,[274] and cryostimulation extended nightly sleep time among elite synchronised swimmers during a heavy training period.[232]

2.3.4.2 Nightly sleep extension and athletic performance

There is mounting evidence that nightly sleep extension improves human function and performance. In non-athletes, sleep extension has been shown to improve alertness, psychomotor vigilance, mood,[278] and pain tolerance.[279] Among tennis players, extended sleep following strenuous training reduced next-day perceived soreness.[266] Among rugby players, extended sleep during pre-season reduced salivary cortisol and improved reaction times during a psychomotor vigilance

task.[259] In female track athletes, extending sleep time by an average of 20 minutes per night for one week, improved mood and peak power during a 30-second Wingate test.[280] And in college athletes there is evidence that sleep extension may improve reactive strength measured during a 'drop-jump' test.[281] Moreover, in military tactical athletes, four nights of sleep extension improved reaction time, executive function, standing jump distance, and motivation levels.[282]

Given evidence demonstrating the benefits of sleep extension for human function, it is surprising that only two peer-reviewed studies have examined the effects of nightly sleep extension on the sports-specific performance of athletes.[20 23] When nine collegiate basketballers spent at least 10 hours in bed per night for 5-7 weeks, mean nightly sleep time measured via actigraphy increased by almost 2 hours per night during the sleep extension period.[20] Sleep extension led to a 9% improvement in both 3-point field goal and free throw shooting accuracy, as well as improvements in sprint times, mood states, and psychomotor vigilance.[20] However, this study did not have a control arm, so improvements may have been attributable to normal training adaptation across the intervention period rather than sleep extension *per se*. In another study, college tennis players had their serving accuracy assessed (i.e., percentage of 'second serves' that hit a circular target deep in the service box) before and after one-week of sleep extension that required players obtain at least 9 hours of sleep per night. Compared with their normal sleep, players self-reported sleeping 1.7 hours per night more during the sleep extension period, and serving accuracy improved from 36% to 42%[23]. However, as previously highlighted, self-reported sleep data often correlates poorly with objective data,[29 30], and thus it is not certain that athletes actually slept more than normal during the sleep extension period. In another, non-peer-reviewed study, the time-in-bed of five

college swimmers was extended to 10 hours per night for a period of 6-7 weeks, and was reported to improve 15-metre sprint times, reaction times, tumble-turn times, and overall mood.[198] However, again, this study did not include a control arm, thus findings may reflect normal training adaptations rather than any effects of sleep extension. Finally, while not strictly investigating sleep extension, a novel study examining the sleep of Australian Under-21 netballers during a one-week national championship, found that members of successful teams (i.e., placed first or second) averaged ~1 hour more sleep per night more during the tournament than members of unsuccessful teams (Figure 2.5).[283]

Based on the current review, three key limitations of previous research justify the need to examine the effects of nightly sleep extension on the performance of endurance athletes:

1. No study has examined the effects of nightly sleep extension on endurance performance. Thus, more specifically, no study has examined the effects of sleep extension on the sports-specific performance of endurance athletes.
2. While two peer-reviewed studies have examined the effects of sleep extension on athletic performance (i.e., basketball, tennis), one did not have a control arm,[20] and the other used self-reported sleep to confirm sleep extension.[23] Thus, no previous study has *both* objectively confirmed sleep extension *and* incorporated a control condition, in order to examine whether sleep extension affects athletic performance.
3. No prior study has examined the efficacy of an individualised sleep extension strategy for extending the nightly sleep time of athletes.

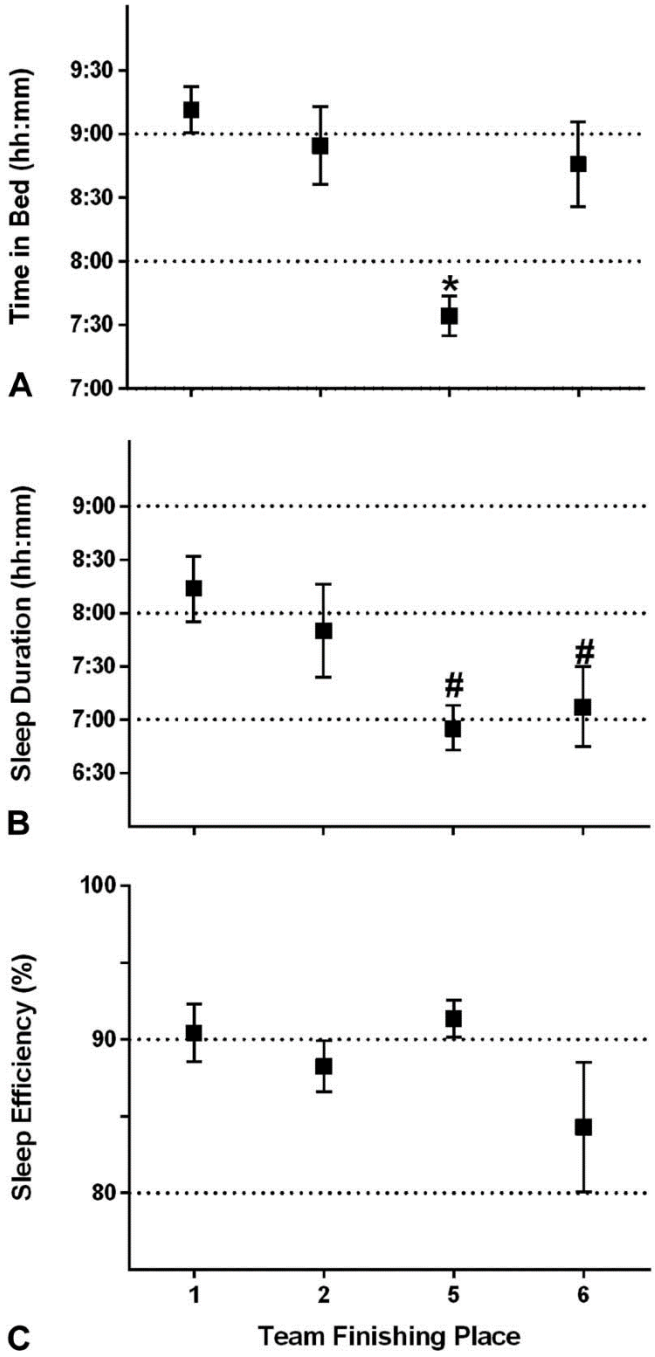

Figure 2.5. Sleep characteristics of netball teams during national championship tournament. Results presented (means and 95% confidence intervals) based on final finishing position for four of seven state representative Under-21 teams competing in the tournament for (A) Time in Bed, (B) Sleep Duration, and (C) Sleep Efficiency. # Different ($P \leq 0.05$) to both team 1 and 2. * Different ($P \leq 0.05$) to team 1, 2, and 4. Source: Juliff[283]

2.3.4.3 Napping and athletic performance

Daytime napping is effective for extending 24-hour sleep time, particularly when nightly sleep is disturbed.[260] Nap frequency (i.e., percentage of days on which a nap is taken) is typically < 20% among elite athletes,[208 284 285] with team sport athletes less likely to nap than individual-sport athletes.[286] However, a recent survey of professional soccer players found 95% of players napped for at least 30-minutes prior to evening matches.[287] There is evidence that napping improves athletic performance.[288-294] For example, compared with a control (i.e., no nap) condition, a 20-minute nap improved the time-to-exhaustion of endurance runners habitually sleeping ~6.4 hours per night, but did not improve the time-to-exhaustion of runners sleeping ~7.5 hours per night.[288] This led authors to suggest that napping may only benefit athletic performance among athletes not habitually sleeping > 7 hours per night,[288] as is recommended for optimal health.[202] In agreement, A 20-minute nap did not affect 30-second Wingate performance of amateur athletes that habitually slept > 8 hours per night.[295] Similarly, while a 30-minute nap among national-level karate athletes did not affect squat jump performance, napping *did* overcome squat jump performance decrements associated with prior sleep restriction.[290] In professional netballers, napping for less than 20 minutes on match days improved overall player performance as judged by coaching staff.[289] In healthy adults, daytime naps of 25, 35, and 45 minutes improved the distance covered during a 5-minute shuttle run.[291] While a 30-minute lunch-time nap following partial sleep deprivation improved 20-metre sprint times in healthy adults.[292] Interestingly, napping for ≤ 30 minutes led to *impaired* 20-metre sprint performance among adolescent athletes, where it was speculated that sleep inertia may have impaired performance.[296]

2.3.5 Monitoring sleep in athletic populations

Given the potential importance of sleep for athletes' health and performance, there is a strong desire to monitor athletes' sleep and address sleep problems.[27] Athlete-specific tools that screen for sleep problems (e.g., Athlete Sleep Screening Questionnaire) and evaluate sleep hygiene (e.g., Athlete Sleep Behaviour Questionnaire),[15 16] are intended for relatively infrequent use (i.e., not daily) and are thus not overly burdensome for athletes. In contrast, frequent (e.g., daily) monitoring of sleep and / or sleepiness is difficult, as data must be accurate and rapidly available, whilst also collected with minimal impost on the athlete. A recent survey found 44% of coaches and support staff working within high performance team-sport do not monitor their athletes' sleep.[297] The main barriers to monitoring sleep identified were a lack of resources, time, and knowledge.[297] This section will review available tools for monitoring sleep and limitations to their use with athletes.

Polysomnography (PSG) provides a composite measure of electrical activity occurring in the brain, heart, and muscles of the face and lower extremities, and is the 'gold standard' for sleep monitoring.[132 133 298] While polysomnography has been useful in athletic populations for investigating sleep pathologies,[299] and sleep disruptions caused by concussion,[300] it is expensive and typically requires specialised laboratory equipment, making it impractical for frequent use in the field.[301] Although, more recently, portable polysomnography systems have demonstrated good agreement with traditional polysomnography,[302] such systems still require electroencephalogram electrodes to be worn, which may be considered invasive and an unnecessary burden by athletes.

Athletes' sleep is typically monitored using subjective tools (e.g., visual analogue scales, diaries).[285 297 303 304] A recent survey of practitioners working in elite sport found that almost 80% of practitioners that monitor sleep use subjective methods.[297] Subjective tools are inexpensive and thus useful for monitoring large cohorts of athletes. While reductions in subjective sleep quality are used to indicate elevations in athletes' stress,[119 305] the accuracy of such assessments is questionable as subjective sleep data often correlates poorly with objective data.[29 30] In addition, subjective sleep assessments are likely susceptible to bias due to recall error,[27] as well as 'respondent fatigue' arising from repeated assessments over extended periods that can result in athletes responding in a random manner.[119]

Research-grade activity monitors provide objective sleep data without affecting the habitual sleep routine of athletes.[210 286 301] Activity monitors, usually worn on the wrist, have embedded accelerometers that detect movement, and device-specific algorithms assess recorded movements across successive epochs (e.g., one-minute) to determine sleep / wake states.[11 298 306 307] Actigraphy provides summary measures such as 'total sleep time' and 'sleep efficiency' (i.e., the percentage of time-in-bed spent asleep).[11 298] Comparison with polysomnography has shown activity monitors to be valid and reliable devices for monitoring sleep in athletes.[11 298 301 308-312] For example, in elite cyclists, an activity monitor demonstrated 87% and 90% agreement (i.e., percentage of epochs correctly identified) with PSG using medium (i.e., > 40 counts·min^{-1} scored 'wake') and high (i.e., > 80 counts·min^{-1} scored 'wake') thresholds respectively.[301] Actigraphy tends to overestimate total sleep time due to quiescent wakefulness being misidentified as sleep.[30 310 313] However, this bias is minimised when used in combination with sleep diaries.[298] Although among athletes, actigraphy may actually *underestimate* total sleep time, as frequent

movements during sleep are common among athletes and may be misidentified as 'wake'.[301] Although useful for research purposes, activity monitors have limited utility in the field because they are relatively expensive (e.g., $1000 USD per monitor), and data typically requires retrospective analysis, as well as expertise to consolidate and interpret.[27] Thus, research grade activity monitors have limited utility for informing 'real-time' athlete management decisions. In a recent interview, Tom Allen, Sport Scientist at Arsenal Football Club stated:[305]

"We look for the most bang for our buck. If there is one (wearable technology) that provides us with reasonable information that can add to the story and it's quick, then that will win over another which gives us really good information but takes a day to get it. We need to be as efficient as possible."

Consumer-grade wearable devices claiming to monitor sleep are also becoming increasingly available. Such devices (e.g., Fitbit, Whoop, Oura Ring) are relatively inexpensive and provide data in 'real-time'. However, most consumer-grade devices have no published data demonstrating their agreement with PSG,[27] or have shown poor agreement with PSG.[314] Nonetheless, recent validation studies have indicated *some* devices (e.g., Oura Ring, Fitbit Charge 2) may demonstrate acceptable agreement with PSG.[315 316] For example, a validation study of the Oura Ring found estimated total sleep time was within the a-priori clinically acceptable range (i.e., ≤ 30 minute difference) for 87.8% of participants examined.[316]. However, further research is required, as validation studies to date have examined the sleep of non-athletes for just a single night.[315 316] Thus, it is unclear whether devices are accurate over long periods and/or in athletic populations. In any case, the use of consumer-grade wearable devices also raises ethical issues over privacy,

including who should have access to data.[28] Moreover, athletes may view such devices as an unnecessary invasion of privacy. As such, it is recommended that any use of sleep monitoring devices should be voluntary and conducted under the guidance of sleep experts intent on educating athletes about their sleep.[28 297]

In summary, the literature reviewed in this section highlights several barriers to monitoring athletes' sleep:

1. Polysomnography, the 'gold standard' for sleep monitoring, while useful for screening purposes or investigating sleep pathologies, is impractical for frequent monitoring of athletes in the field.
2. Subjective tools (e.g., visual analogue scales) are practical and popular for monitoring athletes' sleep; however, they are susceptible to bias, and data does not necessarily correlate with objective sleep outcomes.
3. Research-grade activity monitors are valid and useful for research purposes; however, they are typically expensive, require expertise to use, an often cannot provide actionable 'real-time' data.
4. Some consumer-grade activity monitors demonstrate acceptable agreement with PSG; however, it is unclear whether they are accurate over extended periods in athletic populations.
5. Athletes may view the use of any wearable sleep-monitoring device as an excessive burden and/or a privacy concern.
6. Practitioners often believe they lack the resources, time, and knowledge to monitor athletes' sleep.

2.3.6 Monitoring the effects of sleep on athletes' readiness

Whilst it is important to monitor sleep itself, there is also value in understanding the influence of sleep on popular athlete monitoring indices, such as those used for monitoring athletes' wellbeing and / or 'readiness to perform'. This is particularly true given the barriers to monitoring sleep itself, including concerns over privacy, and the need for expertise in analysing sleep data. Understanding the *effects of sleep* on indices already used to assess athlete readiness may help practitioners better interpret such indices by knowing whether sleep is a moderating factor and thus help inform better athlete management decisions (e.g., facilitation of sleep extension). It would also allow sleep to be considered in existing athlete monitoring protocols without the additional athlete burden, or need for specific sleep-monitoring expertise, that is associated with monitoring sleep itself.

There is evidence that subjective wellbeing assessments are sensitive to sleep.[20 227] Subjective tools include the Profile of Mood States (POMS),[317] the Recovery Stress Questionnaire for Sport (REST-Q),[318] and the Acute / Short Recovery and Stress Scales (ARSS / SRSS).[319] Such tools assess multiple aspects of wellbeing in a time-efficient and inexpensive manner,[320] and are recommended for identifying fatigued or overtrained athletes.[119] Sleep extension for 5-7 weeks improved POMS subscale scores for 'vigour' and 'fatigue' among college basketballers.[20] While one night of sleep restriction decreased SRSS scores for 'overall recovery' and increased scores for 'negative emotional state' among elite junior rowers.[227] However, it's important to note that subjective wellbeing assessments are susceptible to bias when athletes want to be seen in a positive light (e.g., to ensure team selection), or when they want to appear fatigued so that training demands are reduced.[119] Also, repeated wellbeing assessments can lead to respondent fatigue.[119]

Heart rate (HR) is another popular measure used to assess athletes' readiness. It is popular because it is not susceptible to subjective bias, it's inexpensive, and data can be rapidly available and interpreted.[321] However, it is unclear whether HR indices often used to monitor athlete readiness are sensitive to sleep.

2.3.6.1 Using heart rate to monitor athletes' readiness

The rationale for using HR indices to monitor athlete readiness is based on the assumption that changes in cardiac autonomic activity reflect changes in autonomic nervous system balance (i.e., parasympathetic vs sympathetic tone), cardiac function, and / or haemodynamics that can provide an insight into an athletes' ability to perform.[322 323] For example, progressive overload training over several weeks in middle distance runners led to a gradual reduction in HR variability due to an increase in sympathetic tone that is indicative of heightened autonomic stress.[324] Prolonged exercise has also been shown to reduce the fractional shortening of cardiac muscle in fatigued athletes, leading to a reduction in stroke volume that requires a compensatory increase in HR during exercise to sustain cardiac output.[325] Likewise, metabolic by-products released during exercise signal afferent nerves and chemoreceptors to increase sympathetic activity and exercising HR, and may also slow HR recovery post-exercise.[326 327]

The efficacy of HR indices to monitor changes in athletes' readiness has been previously reviewed by Buchheit,[321] and Bellenger and colleagues,[306] and the author is referred to these reviews for a thorough discussion of the topic. Collectively, the evidence indicates several HR indices are effective for monitoring the acute fatigue induced by isolated endurance-based exercise sessions, as well as the accumulated fatigue and fitness improvements induced by extended endurance-

based training blocks.[321] However, while previous research has typically examined the effects of training and / or competition on HR indices,[321 323] the current thesis will systematically examine the effects of *total sleep time* on HR indices. Definitions, monitoring techniques, and typical responses of HR indices examined in this thesis are outlined below:

Resting heart rate: Mean resting HR is typically derived from short (5-10 minute) recordings taken in the supine or seated position upon waking.[321] These conditions are favoured because of the stable conditions they provide (e.g., same time of day, same posture, quiet environment etc.).[321] Acute bouts of fatiguing endurance exercise have been shown to increase resting HR,[325 328] whereas fitness improvements lower resting HR.[329]

Resting heart rate variability: Heart rate variability refers to the variation in time between successive heart beats (i.e., R-R intervals).[330] It is useful for its ability to assess the balance between sympathetic and parasympathetic activity.[331-334] The natural logarithm of the square root of the mean sum of squared differences between adjacent R-R intervals (i.e., Ln rMSSD), which reflects levels of parasympathetic activity, is recommended for athlete monitoring because it can be derived from short (e.g., 5-minute) HR recordings and its sensitivity to respiratory rate is low.[321] Reduced HR variability is typically associated with accumulated fatigue.[324 335-341] However, *elite* endurance athletes occasionally have such high parasympathetic predominance that acetylcholine receptors become saturated, and thus the normal HR variability associated with respiration is eliminated.[342]

Maximal rate of heart rate increase: The maximal rate of HR increase (rHRI) recorded during constant-load submaximal exercise indicates autonomic nervous system responsiveness, particularly parasympathetic withdrawal, during the transition from rest to exercise.[343-349] The rHRI is derived from the first derivative maxima of exponential or sigmoidal functions fitted to the HR kinetic response to exercise; however, for endurance athletes, using the exponential response to exercise at 60% $\dot{V}O_{2\,max}$ is recommended.[348] The rHRI was faster in endurance athletes following two-weeks of light training compared with two-weeks of overload training.[349] While the acute fatigue induced by a 2-hour treadmill run led to a reduction in rHRI that correlated ($r = 0.6$, $P < 0.05$) with the reduction in work completed during a cycling time-trial.[347]

Steady-state heart rate: Mean steady-state HR recorded during constant-load submaximal exercise is recommended for its ability to inform on changes in relative exercise intensity (i.e., the physiological cost of exercising at a given workload).[321] Steady-state HR is usually averaged over the final minute of a relatively short (e.g., 5 minutes) constant-load exercise test, and tends to increase with accumulated fatigue,[350 351] and decrease with improved fitness.[333 352 353]

Heart rate recovery: Typically measured as the magnitude of change in HR (i.e., in beats·min^{-1}) within 60 seconds of stopping exercise,[354] HR recovery reflects the interaction between sympathetic withdrawal and parasympathetic reactivation that occurs following exercise.[355 356] Cross-sectional studies show HR recovery is greater in trained versus untrained

individuals,[357-359] and fitness improvements often lead to faster HR recovery.[353 360-362] Reduced HR recovery tends to indicate an accumulation of fatigue.[363-365] However, in elite endurance athletes, greater HR recovery has been associated with short-term performance decrements,[366-369] and may reflect reduced sympathetic responsiveness during exercise, or reduced sensitivity of adrenergic receptors.[366 367] Therefore, interpretation of HR recovery should be made in the context of the current training phase / load and the perceived exertion of exercise.[368]

Exercise intensity ratios: Recent studies have shown that incorporating HR indices into intensity ratios (e.g., subjective to objective, external to internal) can also inform practitioners about athletes' fatigue status.[109 370] Exercising HR collected during training or competition is typically used to assess either the internal physiological cost of completing an external workload (i.e., internal to external intensity ratio), or the perceived exertion required for a given internal load (i.e., subjective to objective intensity ratio).[371-373] The relationship between HR and running velocity ($m \cdot min^{-1}$) decreased as fatigue accumulated across a match in professional footballers.[370] While Figure 2.6 shows that ratios for mean power to mean HR (i.e., W:HR), and perceived exertion to mean HR (i.e., RPE:HR), increased with fatigue across a three-week tour race among cyclists.[109]

2.3.6.2 Using heart rate to monitor the effects of sleep on readiness

There is a close coupling of central and autonomic nervous system activity during sleep.[374] Indeed, parasympathetic activity increases and sympathetic activity decreases during sleep.[238 375]

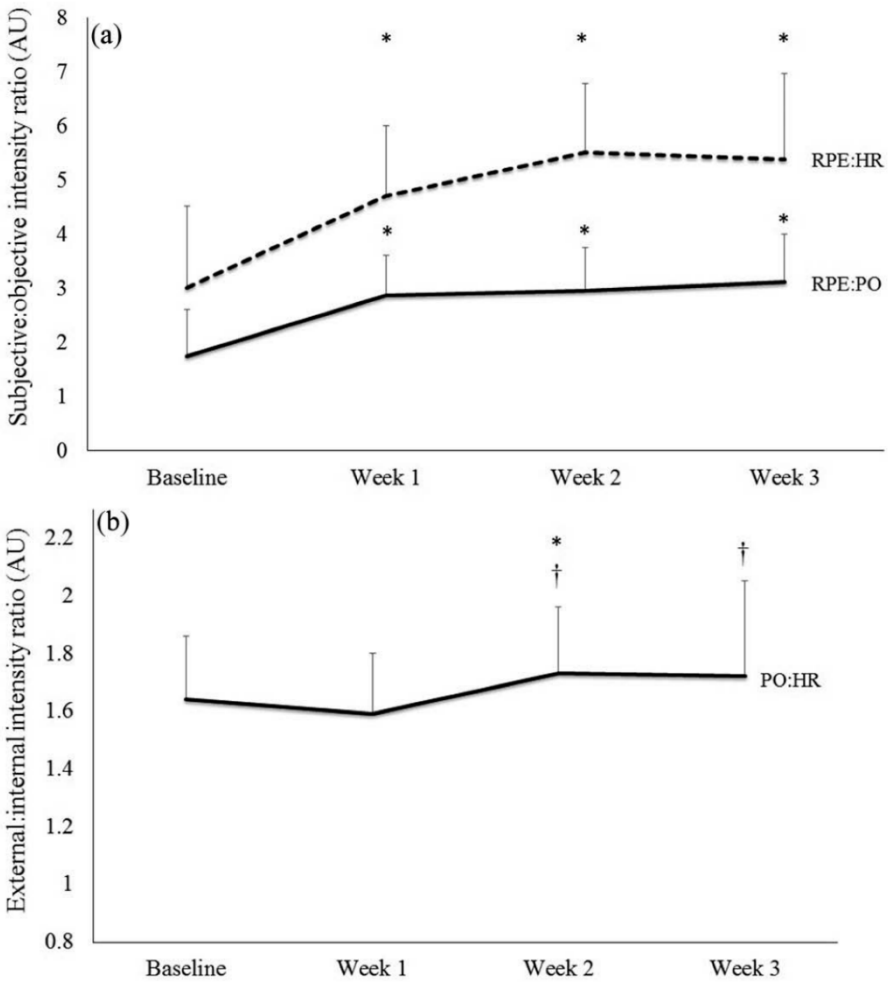

Figure 2.6. Intensity ratios among professional cyclists during baseline training and three weeks of a Grand Tour. Perceived exertion, RPE; mean HR, HR; and mean power, W; obtained during baseline training and Grand Tour race stages and computed to determine subjective to objective intensity ratio (RPE:HR) and external to internal intensity ratio (PO:HR). AU, arbitrary units. * Different to baseline training ($P<0.05$). † Different to week 1 ($P<0.05$). Source: Sanders et al.[109]

This is evidenced by research showing that, compared with wakefulness, sleep is associated with increases in HR variability,[238 376] reductions in HR,[374 377] and reductions in circulating catecholamines – a marker of sympathetic activity.[378-380] When comparing sleep stages, parasympathetic activity is higher during stages 2-4 of non-REM compared with REM sleep,[377 381] while sympathetic activity increases during REM sleep.[382] The coupling of central and autonomic nervous system activity is further demonstrated by research showing nightly HR variability can identify and distinguish sleep stages.[383-385] Moreover, sleep disorders such as insomnia and sleep apnoea are associated with night time sympathetic hyperactivity.[221 386] Similarly, insomnia symptoms in athletes are thought to reflect sympathetic hyperactivity before and during sleep.[14] Among soccer players, HR variability (i.e., Ln rMSSD) during sleep was reduced ($P < 0.001$) following training and matches compared with nights following rest days.[387]

Research has also investigated the effects of sleep, specifically sleep loss, on daytime HR regulation. While there is some evidence that *total sleep deprivation* does not affect HR,[245 388-390] there is also evidence that total sleep deprivation reduces both resting,[244 390-392] and exercising (e.g., steady-state) HR for a given workload.[51 393-395] Total sleep deprivation has been shown to either not affect,[389 396] or reduce daytime plasma catecholamine levels.[244] While analysis of HR variability suggests there may be a shift towards parasympathetic predominance following total sleep deprivation,[391 392] which may serve to protect against cardiac overload.[391 393 397] It has also been speculated that lower HR, and increased HR variability, following sleep deprivation may reflect an exhaustion of catecholamine supply, that dampens the HR response to sympathetic activity,[393 397] Otherwise, increases in plasma volume that normally occur over prolonged periods of wakefulness may

trigger baroreflex-mediated reductions in HR.[49 246] In contrast, most studies show *sleep restriction* either does not affect HR,[251 398] or increases both resting,[399-403] and exercising HR for a given workload.[249 404] Moreover, sleep restriction has been shown to reduce resting HR variability due to elevated sympathetic modulation of the heart.[398 405] Sleep restriction has also been associated with increases in urinary and plasma catecholamines.[405-407] In summary, total sleep deprivation tends to decrease HR and increase HR variability,[51 244 390-395] whereas sleep restriction tends to increase HR and reduce HR variability.[249 398-405] These findings encourage speculation that sleep restriction may be a stress that shifts autonomic balance towards sympathetic predominance, whereas total sleep deprivation may be such a *severe* stress that it exhausts catecholamine supply, or triggers protective mechanisms that result in parasympathetic predominance. Nonetheless, it is also noted that several studies show daytime HR is unaffected by sleep loss.[245 251 388-390 398] Discrepant findings between studies *may* reflect differences in HR recording conditions (e.g., posture, time-of-day, prior activity), the severity of sleep restirction, and / or participant characteristics (e.g., fitness, age, sex).

Although sleep loss may alter daytime HR regulation, and HR indices are widely used for athlete monitoring, no prior research has examined the sensitivity of HR indices to any effects of sleep on athletes' readiness. However, in non-athletes, previous research *has* shown that HR variability can track decrements in psychomotor vigilance caused by sleep loss.[408 409] One study examined healthy men across a 40-hour total sleep deprivation period that required a 10-minute psychomotor vigilance task to be completed every two hours.[408] This study found that participants' HR variability measured using either power spectral density in the 0.02-0.08 Hz range, or the mean of the standard deviation of all R-R intervals,

strongly correlated ($r = 0.7$, $P < 0.05$) with lapses in psychomotor vigilance across the sleep deprivation period.[408] Another study examined healthy males over five consecutive nights of sleep restriction (i.e., 4 hours sleep per night) that required a 10-minute psychomotor vigilance task to be completed four times each day.[409] This study also found that power spectral density in the 0.01-0.08 Hz range correlated ($r = 0.60$, $P < 0.05$) with lapses in psychomotor vigilance.[409] Authors of both studies recommended HR variability be used in lieu of more invasive or expensive tools to monitor fatigue induced by sleep loss in occupational settings.[408 409] Importantly, the HR variability indices used in these studies are not recommended for athletes, as they typically require long HR recordings and are sensitive to breathing rate.[321] In contrast, tests used for monitoring athletes are preferably short (e.g., ≤ 5 minutes) and relatively simple to administer. Nonetheless, these findings pose the question; do changes in HR variability, or any other HR index, caused by sleep loss or sleep extension, similarly track changes in *athletic* performance?

2.4 Research objectives

Sleep may be an important recovery behaviour for athletic success.[10 410] However, to date, few studies have investigated the impact of sleep on the performance of endurance athletes.[24-26 248 249] Studies that *have* investigated effects of sleep loss (i.e., total sleep deprivation or sleep restriction) on the performance of endurance athletes report contradictory findings, and have not assessed performance using prolonged self-paced time-trials that mimic the efforts required by many endurance sports (e.g., road cycling, triathlon).[24-26 248 249] Further, although endurance athletes train, and occasionally compete, on consecutive days, no prior research has examined the *cumulative* effects of sleep restriction (i.e., reduced habitual sleep time) or sleep extension (i.e., increased habitual sleep time) over consecutive nights

on the performance of endurance athletes. Thus, it remains unclear the extent to which cumulative sleep time affects the performance of endurance athletes.

Monitoring athletes' sleep may help practitioners understand sleep challenges faced by athletes and explore the effects of sleep on athletic performance. However, current monitoring tools have limitations for use with athletes; mostly due to concerns over their cost and practicality (e.g., PSG, research-grade devices), their accuracy (e.g., subjective tools, consumer-grade devices), and their intrusion on athletes' privacy.[27] Therefore, not surprisingly, many practitioners do not monitor their athletes' sleep due to a perceived lack of time, resources, and knowledge.[297] An alternate approach is to better understand the effects of sleep on existing indices used for monitoring athlete readiness. This could improve practitioners' ability to interpret readiness indices and thus inform better athlete management decisions (e.g., facilitation of sleep extension). Heart rate indices are widely used to monitor athletes' readiness,[321 323] and sleep loss has been shown to alter HR regulation.[51 244 249 390-395 398-405] However, it remains unclear whether prior sleep time moderates the relationship between HR indices and athletes' readiness to perform.

The primary aim of this thesis was to investigate whether total sleep time, accumulated over 1-3 nights, affects the recovery of prolonged self-paced endurance performance among endurance athletes. The secondary aim of this thesis was to investigate whether HR indices used for monitoring athletes' readiness were sensitive to any effects of total sleep time on the recovery of endurance performance. Data collected in addressing these aims also allowed for an exploration of potential mechanisms through which sleep may affect endurance performance. The specific aims of the two studies included in this thesis were:

Study 1: The aims of Study 1 were to (1) examine the effect of a single night of total sleep deprivation on the recovery of endurance cycling performance, and (2) to examine whether HR indices were sensitive to any effects of total sleep deprivation on athletes' readiness to perform.

Study 2: The aims of Study 2 were to (1) examine the effects of three consecutive nights of sleep restriction and sleep extension on the recovery of endurance cycling performance (Study 2a), and (2) to examine whether HR indices were sensitive to any effects of sleep restriction or sleep extension on athletes' readiness to perform (Study 2b).

Chapter 3: Study 1

This chapter is presented as per the manuscript accepted for publication in the Journal of Sports Sciences. However, online supplementary material referenced in the accepted manuscript has been included in this chapter, thus table numbers differ to the accepted manuscript:

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. Effects of total sleep deprivation on endurance cycling performance and heart rate indices used for monitoring athlete readiness. *J Sports Sci.* 2019;37(23):2691-701.

3.1 Abstract

Introduction: This study investigated effects of total sleep deprivation on self-paced endurance performance, and heart rate (HR) indices of athletes' "readiness to perform". **Methods:** Endurance athletes (n = 13) completed a crossover experiment comprising a normal sleep (NS) and sleep deprivation (SD) condition. Each required completion of an endurance time-trial (TT) on consecutive days (D1, D2) separated by normal sleep or total sleep deprivation. Finishing time, perceived exertion (RPE), mood, psychomotor vigilance (PVT), and HR responses were assessed. **Results:** Time on D2 of SD was 10% slower than on D2 of NS (64 ± 7 vs 59 ± 4 min, $P < 0.01$), and 11% slower than D1 of SD (58 ± 5 min, $P < 0.01$). Subjective to objective (RPE: mean HR) intensity ratio was higher on D2 of SD compared with D2 of NS and D1 of SD ($P < 0.01$). Mood disturbance and PVT mean response time increased on D2 of SD compared with D2 of NS and D1 of SD. Anaerobic threshold and change in TT time were correlated ($R = -0.73$, $P < 0.01$). **Conclusion:** Sleep helps to optimise endurance performance. Subjective to objective intensity ratios appear sensitive to effects of sleep on athletes' readiness to perform. Research examining more subtle sleep manipulation is required.

3.2 Introduction

Sleep is considered an important recovery behaviour for athletic success; however, athletes often sleep poorly during training and competition periods.[13] Sleep disturbances have been attributed to pre-competition thoughts or nervousness,[200] post-competition hyperarousal,[287] transmeridian travel,[411] and early-morning training sessions.[228] Several studies have examined the effects of sleep loss on the performance of endurance-trained athletes.[24-26 248 249] However, these studies report contradictory findings, and have often used short (i.e., < 15 minutes) performance

tests,[24 25] or time-to-exhaustion[25 26] / workload-at-exhaustion[248 249] tests, that do not reflect the prolonged (e.g., ≥ 60 minutes) self-paced nature of many endurance sports (e.g. road cycling). For example, compared with a normal night of sleep (7.1 hours), reducing sleep to 2.4 hours the night before a 3 km time-trial slowed performance by 4%.[24] While the maximal workload achieved by endurance athletes during an incremental test that followed a 20-minute submaximal preload decreased when prior sleep opportunity was reduced by four hours,[248] but was unaffected when sleep opportunity was reduced by three hours.[249] Furthermore, studies investigating *total* sleep deprivation have shown that, compared with normal sleep, one night without sleep reduced the time-to-exhaustion of endurance athletes during a ~20-minute incremental test that followed a 40-minute preload at 50-65% of $\dot{V}O_{2\ max}$.[26] Whereas 25-30 hours of sleep deprivation did not affect the time-to-exhaustion of distance runners but reduced the time-to-exhaustion of volleyball players.[25] Given the equivocal findings to date, and the paucity of data examining self-paced performance, further investigation of the effects of sleep deprivation on endurance performance is required.

The mechanisms underpinning impaired endurance performances following sleep loss remain unclear. Previous research found endurance performance decrements following total sleep deprivation did not coincide with impaired neuromuscular function (i.e., central fatigue).[26] However, there is a close coupling of central and autonomic nervous system activity during sleep,[374] and sleep loss has been shown to alter cardiac autonomic function.[31 390] For example, sleep restriction has been shown to decrease daytime HR variability compared with sleep extension.[31] Altered cardiac autonomic function *may* explain, to some extent, endurance performance decrements previously reported. If so, HR indices typically used for athlete

monitoring may be sensitive to the effects of prior sleep on athletes' fatigue status (i.e., "readiness to perform"). HR indices can inform the training (e.g., load) and competition (e.g., game-time) adjustments required to optimise performance, and are popular because data can be easily collected and quickly interpreted.[109 321] HR indices can be recorded at rest (e.g., HR variability) or during exercise (e.g., intensity ratios).[321] For example, reduced resting HR variability has been reported in fatigued Nordic-skiers,[340] whereas increased HR variability occurred with fitness improvements in runners.[412] Moreover, subjective to objective, and external to internal intensity ratios incorporating mean HR have been shown to increase in professional cyclists during a three-week grand tour.[109] However, no study has investigated whether such routinely measured athletic indices are sensitive to the acute effects of prior sleep.

This study aimed to (1) quantify the effect of a single night of sleep deprivation on prolonged endurance cycling performance, and (2) examine whether HR indices used for athlete monitoring are sensitive to any effects of sleep deprivation on athletes' readiness to perform.

3.3 Methods

Participants

Thirteen males (mean ± SD, Age = 33 ± 6 years; $\dot{V}O_{2\,max}$ = 64 ± 7 mL·kg^{-1}·min^{-1}) were recruited from cycling (n=8) and triathlon (n=5) clubs. Participants were considered 'trained' according to adapted criteria for classifying cyclists (≥ 1 year of competitive racing, ≥ 3 × training sessions per week, $\dot{V}O_{2\,max}$ ≥ 55 mL·kg^{-1}·min^{-1})[35]. To screen for pre-existing sleep problems and high anxiety, inclusion criteria required a score of ≤ 5 in the Pittsburgh Sleep Quality Index (PSQI) and ≤ 40 in the

State-Trait Anxiety Inventory (STAI-Y).[413] [414] Participants were not permitted to be undertaking shift work or taking medication that may affect sleep. The Morningness-Eveningness Questionnaire categorised participants as 'intermediate' (n = 8) and 'moderate morning' types (n = 5).[415] Participants were not high caffeine consumers (mean ± SD, 2 ± 1 caffeine products per day). The study was approved the Deakin University Human Research Ethics Committee and informed written consent to participate was obtained.

Study design

Participants completed a randomised counterbalanced crossover study comprising two conditions; normal sleep (NS) and sleep deprivation (SD). Prior to testing, three familiarisation sessions required completion of an incremental test, and two practice time-trials (TT). Each condition comprised six consecutive days and nights (-2, -1, D1, D2, +1, +2) during which sleep, morning HR, training load, and diet were monitored (Figure 3.1). Dietary monitoring served to control for potential differences in energy availability between conditions. Diet was recorded (e.g., 8am: 2 slices of toast with jam) for the duration of the first condition and replicated for the second condition. Caffeine, alcohol, and exercise (excluding that required for the study) was prohibited on days -1 to D2. However, to accommodate preferred preparation and recovery strategies, participants were permitted to undertake light exercise on days -2, +1 and +2. Training load was quantified according to methods previously described.[416] There were no differences between conditions on days prior to-, or post, laboratory testing (Table 3.1). On D1 and D2, testing was conducted at the Deakin University Human Research and Performance Laboratory, whereby endurance performance, subjective mood, psychometric vigilance, plasma volume, plasma catecholamines, and HR responses during exercise were assessed.

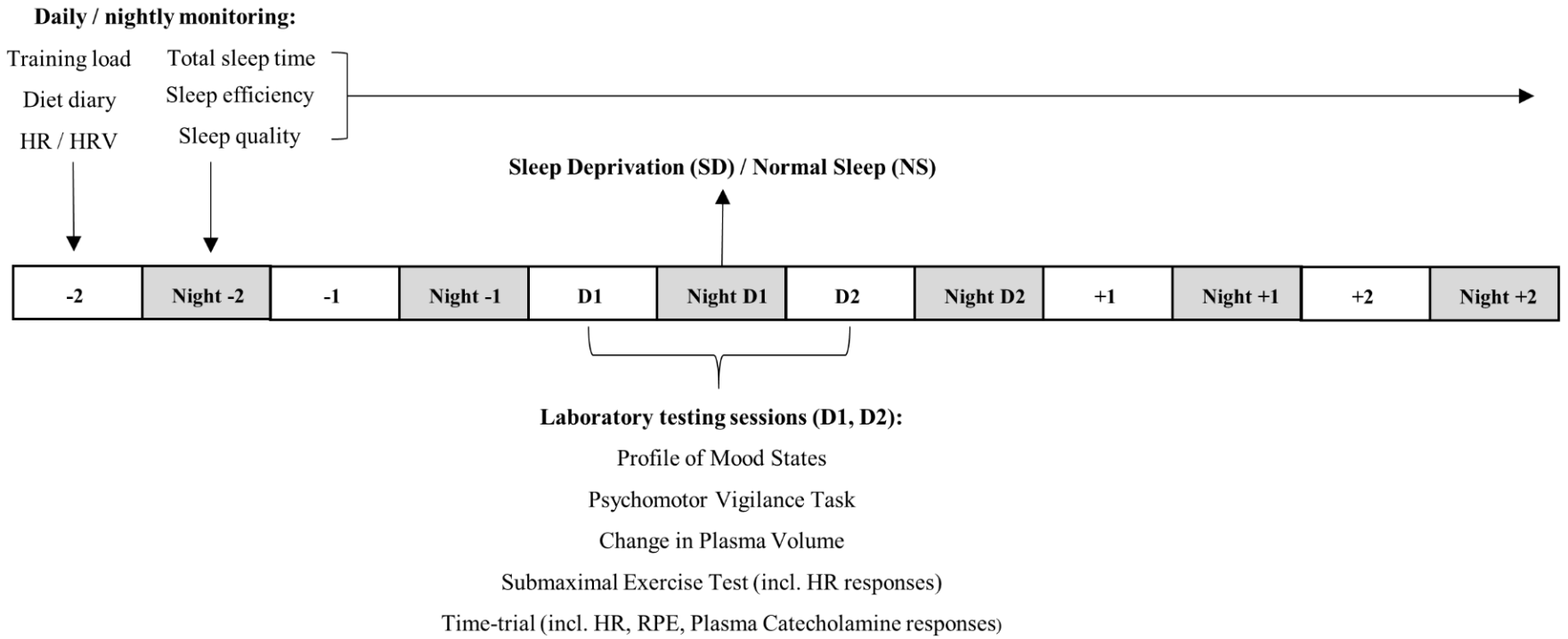

Figure 3.1. Data collection timeline. Training load and diet were recorded daily. Resting heart rate and heart rate variability (HR / HRV), total sleep time, sleep efficiency, and subjective sleep quality were monitored every day / night (-2 to +2). On laboratory testing days (D1, D2) participants initially completed mood, psychomotor vigilance, and plasma volume assessments. Participants then completed a five-minute constant load submaximal exercise test, which served as a warm-up, before an endurance time-trial. Heart rate responses to the submaximal exercise test and the time-trial were assessed. Perceived exertion (RPE) upon completion of the time-trial, and plasma catecholamine pre- and post the time-trial, were also assessed. The study intervention (i.e., normal sleep or total sleep deprivation) occurred on night D1.

Table 3.1. Self-reported training load.

	Training Load					
Days	**-2**	**-1**	**D1**	**D2**	**+1**	**+2**
Sleep Deprivation	427±417	43±83	507±66	564±48*	277±210	529±223
Normal Sleep	388±259	43±84	520±47	517±42	357±353	569±488

Data shows arbitrary units for the product of session exercise time (min) and perceived exertion (CR-10 scale).[412] Participants recorded all exercise undertaken during each condition (-2 to +2) including laboratory TTs on D1 and D2. Note: Although training was prohibited on day -1, two participants were required to undertake very light exercise to commute to employment. This exercise was replicated for both conditions. There were no differences between conditions on days -2, -1, D1, +1, or +2. * Different ($P < 0.01$) compared with D2 of normal sleep. Data shows mean ± SD.

Upon completion of testing on D1 of SD, participants undertook their usual daily activities before returning to the laboratory at 9:00 pm to undertake overnight sleep deprivation under supervision of the research team. Testing on D2 commenced at the same time of day as on D1, therefore, when considering the time required for participants to wake and travel to the laboratory on D1 of SD, participants were sleep deprived for ~25 h prior to commencement of testing on D2. During sleep deprivation, participants undertook sedentary activities (e.g., watched TV) and consumed water *ad libitum* in low light (<20 lux) conditions. No other food or drink was permitted, other than the participant's usual breakfast the morning of D2. Participants slept habitually at home on all other nights of the study. All laboratory testing commenced between 06:00–09:00 am to control for circadian variation. There was a six-day washout period between conditions.

Familiarisation sessions

Incremental test

On a cycle ergometer (Excalibur Sport, Lode, Groningen, Netherlands) controlled using compatible software (Lode Ergometry Manager 9, Lode, Groningen, Netherlands), the incremental test commenced at 75W and increased by 50W every three minutes. After nine minutes, workload increased by 25W every minute until volitional exhaustion. Gas was analysed using an Innocor metabolic system (DK-5260, Innovision, Odense, Denmark) to determine maximal oxygen uptake ($\dot{V}O_{2\,max}$) and anaerobic threshold (AT).[417]

Time-trial protocol

Target workload for the TT was equivalent to the estimated energy expended when cycling at AT for one hour:

$$Work\ (kJ) = \frac{(W_{AT}\ \times 3600)}{1000}$$

whereby the linear relationship between oxygen uptake ($\dot{V}O_2$) and power (W) was calculated for the first three workloads of the incremental test, and the resulting regression used to calculate power at AT (W_{AT}). Pedalling resistance was determined according to the formula.

$$W_{AT} = Linear\ Factor \times Preferred\ pedal\ rate^{\ 2}$$

whereby the linear factor ensured W_{AT} was achieved at the participant's preferred pedal rate per minute (rev·min^{-1}). There is a strong correlation between power at AT and one-hour TT performance ($r = 0.8$, $P < 0.05$).[418] During the TT, work completed (kJ) was displayed on a computer screen, but no other feedback was provided. Participants completed two practice TTs so that pacing strategies could be refined.

Experimental conditions

Sleep

Participants wore an activity monitor (Actical MiniMitter/Philips Respironics, Bend, OR) on their non-dominant wrist for all sleep periods. The Actical device is an objective, validated tool for indirectly monitoring sleep.[298] The monitor recorded activity counts in one-minute epochs. Raw scores were downloaded using a device specific interface unit (ActiReader, Philips Respironics, Bend, OR) and processed with a manufacturer algorithm (Actical v3.10) set to a medium threshold (<40 counts·min^{-1} scored as sleep) to distinguish between sleep-wake states. This threshold has demonstrated 87% agreement with polysomnography in cyclists.[301] Participants recorded bed- and wake-times in a diary in order to crosscheck

sleep/wake states identified with actigraphy. The total sleep obtained (i.e., total sleep time – TST), and the percentage of time in bed spent asleep (i.e., sleep efficiency) were calculated for all sleep periods. While careful monitoring of participants during sleep deprivation ensured no napping occurred, actigraphy data was nonetheless analysed for the sleep deprivation periods for confirmation. To evaluate 24-hour patterns, aggregate TST for all sleep periods from the end of a night's main sleep to the end of the next night's main sleep was calculated. Mean SE was calculated for all sleep episodes during the same period. Upon waking, participants rated sleep quality according to a 5-point Likert scale (i.e., 1 = Very Good, 2 = Good, 3 = Average, 4 = Poor, 5 = Very Poor).

Time-trial outcomes

Finishing time (minutes), peak HR, mean HR, and mean power (W) were recorded for each TT. Upon completion of the TT, participants recorded the perceived exertion (RPE) required to complete the TT on a CR-10 scale.[419] The CR-10 scale was chosen as previous research used this scale to examine intensity ratios in professional cyclists.[109] The external to internal intensity ratio (mean power to mean HR; W:HR), and subjective to objective intensity ratio (perceived exertion to mean HR; RPE:HR) were analysed.[109]

A subset of participants (n = 7) had plasma catecholamines monitored as a measure of sympathetic nervous system activity. Blood was collected immediately prior to the warm-up (i.e., submaximal exercise test, see Preliminary testing) and within five minutes of completing the TT. On each occasion, 8 mL of whole blood was collected (K_2EDTA, BD Vacutainer, Franklin Lakes, NJ), and then spun for 10 minutes at 3000 rev·min^{-1} (Heraeus Megafuge 8R, Thermo Fisher Scientific,

Waltham, MA). Plasma was extracted and stored at -80°C for later analysis. Catecholamines were assayed in duplicate using a single ELISA kit (Abnova, KA1877, Taipei City, Taiwan) for both adrenaline and noradrenaline.

Preliminary testing

Participants undertook a series of tests prior to the TT (Figure 1). Subjective mood was assessed using the Profile of Mood States (POMS), which assessed the feelings of participants "right now" across 65 mood descriptors using a 5-point Likert scale, providing scores for total mood disturbance, tension, depression, anger, vigour, fatigue, and confusion.[420] Participants then completed a validated touchscreen version of the psychomotor vigilance task (PVT) on a tablet device using the application sleep-2-Peak (v2.2.1, Proactive Life LLC, New York, NY).[421] The PVT measured reaction times to visual stimuli occurring at varying time intervals over 10 minutes. Mean reaction time, and the number of lapses > 500 milliseconds were recorded. Both the POMS and PVT have shown sensitivity to total sleep deprivation and served as positive controls to confirm expected mood disturbances and psychomotor impairments caused by sleep loss.[422] [423]

Potential changes in plasma volume, which have been reported following total sleep deprivation and may influence HR regulation,[394] were indirectly assessed using capillary blood samples[424]. Hemoglobin ($g \cdot dL^{-1}$) was determined using a Hemo Control Hemoglobin Analyser (EFK Diagnostics, Cardiff, UK), and hematocrit (%) was manually read from a centrifuged sample (Sigma 1-15, Sigma Laborzentrifugen GmbH, Germany).

A submaximal exercise test, which served as a warm-up for the TT, required participants to cycle for five minutes at a workload corresponding to 60%$\dot{V}O_{2\,max}$.

Participants rested on the ergometer for at least two minutes prior to the test, and were not pre-warned when the test was to begin in order to avoid an anticipatory increase in HR. Upon completion, participants rested on the ergometer for one minute. Data were transferred to Table Curve 2D software (SYSTAT Software Inc., San Jose, CA) and fitted to a mono-exponential function:

$$HR\ (beats \cdot min^{-1}) = a + A\ (1 - e^{\frac{-(t-TD)}{\tau}})$$

Where; a is baseline HR (beats·min^{-1}); A is the amplitude of HR response (beats·min^{-1}); t is time (s); TD is the time delay before HR increases (s); and τ is the curvature parameter (s). Curve fitting constraints applied to ensure an optimal fit have been previously described.[348] Steady-state HR (i.e., mean HR for final minute of exercise), external to internal intensity ratio (i.e., power to steady-state HR; W:HR), and HR recovery (beats·min^{-1} recovered) were recorded. Additionally, cardiac autonomic responsiveness at the onset of exercise was assessed using the maximal rate of HR increase (rHRI, beats·min^{-1}·s^{-1}) derived from the first derivative maxima of the function according to:

$$HR\ (beats \cdot min^{-1} \cdot s^{-1}) = \frac{A\ (e^{-1(\frac{t-TD}{\tau})})}{\tau}$$

Daily heart rate and heart rate variability

Participants recorded R-R series data for seven minutes, in the supine position, every morning upon waking (V800 HR Monitor, Polar, Kempele, Finland). Recording HR upon waking is optimal because testing conditions are highly standardised (i.e., same bed, same time of day, low light)[321]. The data were uploaded to the Polar FlowSync (v2.6.2) interface, and then exported to Kubios

HRV software (v3.0.1 University of Eastern Finland, Finland). Artefacts and ectopic beats were visually identified and interpolated with the cubic spline method using the lowest detection threshold necessary. The last five minutes of each R-R series was analysed, as the first two minutes served as a stabilisation period. Mean HR and the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) were calculated.

Statistical analysis

Mean and SD were calculated for all variables. Generalised Estimating Equations with exchangeable correlation structures and robust standard errors analysed mean changes in outcome variables. Initial models tested for period and carryover effects; however, no such effects were found ($P > 0.05$). Models then analysed two- or three-way interactions for the factors; 'condition', 'day', and 'pre-post' time point (i.e., catecholamines only). Where interactions were significant ($P < 0.05$), pairwise models were run for each 'day' or 'pre-post' time point. A p-value < 0.025 was used to account for multiple analyses. Additional models analysed main effects of 'day' within a condition, and main effects of 'day' for each 'pre-post' time point within a condition. A p-value <0.05 was used. Data from days / nights -2 and -1 served as baseline values in separate models for data collected over six days / nights. Cohen's *d* effect sizes were calculated for significant differences between conditions.

Anaerobic threshold (AT) is indicative of cardiorespiratory fitness and increases with endurance training.[425] Therefore, to examine the relationship between cardiorespiratory fitness or training history and athletes' response to total sleep deprivation, a Pearson correlation and linear regression examined the relationship

between AT and the change in TT finishing time from D1 to D2 of the SD condition. Correlations and linear regressions also examined relationships between changes in HR indices and changes in TT finishing time in the SD condition. Percentage changes were examined to account for individual differences in HR regulation and TT finishing times. Analyses were performed in IBM SPSS statistics for Windows (v24.0, New York, NY).

3.4 Results

Sleep

Table 3.2 shows TST was shorter in the SD condition on night D1 ($P < 0.001$, $d = 11.8$) and longer on night D2 ($P < 0.001$, $d = 2.5$) compared with NS. Within the SD condition, TST was shorter on night D1 and longer on night D2, compared with baseline nights -2 and -1 ($P < 0.001$). Within the NS condition, TST was longer on night D2 compared with baseline nights -2 ($P < 0.01$) and -1 ($P < 0.02$). Neither sleep efficiency nor sleep quality were recorded on night D1 of SD, as participants did not sleep. However, there were no differences in sleep efficiency or sleep quality between conditions on any other night. Within the SD condition, sleep efficiency was higher on night D2 compared with -2 and -1 ($P < 0.05$). Within the SD condition, sleep quality was better on night D2 compared with baseline nights -2 and -1 ($P < 0.01$)

Table 3.2. Morning heart rate, and nightly sleep outcomes recorded across the six days / nights of each condition.

	Days	-2	-1	D1	D2	+1	+2
Sleep Deprivation	**HR (beats·min^{-1})**	53±6	52±6	53±7	50±6	51±9*	54±7^{+}
	Ln rMSSD (ms)	4.26±0.53	4.26±0.44	4.26±0.52	4.39±0.52	4.19±0.41	4.19±0.43
	Total sleep time (h)	5.9±1.4	5.9±1.0	0.0±0.0*$^{+\wedge}$	9.7±1.7*$^{+\wedge}$	6.1±0.8	5.8±0.7
	Sleep efficiency (%)	82±10	82±10	NA	84±7$^{+\wedge}$	81±10	80±9
	Sleep quality (au)	3±1	3±1	NA	2±1$^{+\wedge}$	3±1	3±1
Normal Sleep	**HR (beats·min^{-1})**	54±8	53±9	53±7	53±8	55±9	55±9
	Ln rMSSD (ms)	4.22±0.47	4.18±0.40	4.26±0.42	4.19±0.46	4.16±0.52	4.21±0.37
	Total sleep time (h)	5.9±1.0	5.9±1.2	5.9±1.0	6.5±0.9$^{+\wedge}$	5.8±0.9	5.8±0.8
	Sleep efficiency (%)	82±11	83±7	81±10	82±8	82±7	81±8
	Sleep quality (au)	3±1	3±1	3±1	3±1	3±1	3±1

Heart rate data recorded the *morning* of the day indicated (-2 to +2). Sleep data recorded the *night* of the day indicated (-2 to +2). *D1-D2*, laboratory-testing days; *TST*, total sleep time; *h*, hours; *SE*, sleep efficiency; *SQ*, subjective sleep quality; *au*, arbitrary units; *HR*, mean heart rate; *beats·min^{-1}*, beats per minute; *Ln rMSSD*, natural logarithm of the square root of the mean sum of the squared differences between adjacent R-R intervals; *ms*, milliseconds. * Different ($P < 0.025$) to normal sleep condition. $^{+}$ Different ($P < 0.05$) to -1 of condition. $^{\wedge}$ Different ($P < 0.05$) to -2 of condition. Data shows mean ± SD.

Time-trial

Finishing time (Figure 3.2A) was slower on D2 of SD compared with D2 of NS ($P < 0.01$, d =1.0), and D1 of SD ($P < 0.01$). Mean power, mean HR, and peak HR were lower ($P < 0.01$) on D2 of SD compared with D2 of NS (d = 0.3, 1.0, 0.7, respectively), and D1 of SD (Table 3.3). There were no differences in RPE between or within conditions. Subjective to objective (RPE:HR) intensity ratio was higher ($P < 0.01$) on D2 of SD compared with D2 of NS (d =0.8), and D1 of SD (Figure 3.2B). There were no differences in external to internal (W:HR) intensity ratio between or within conditions (Figure 3.2C).

No differences in adrenaline concentration were found for either the 'pre' (SD: D1, 60 ± 19, D2 60 ± 40; NS: D1, 53 ± 27, D2, 45 ± 18 pg·mL^{-1}) or 'post' (SD: D1, 200 ± 132, D2, 112 ± 54; NS: D1, 229 ± 130, D2, 147 ± 117 pg·mL^{-1}) time points between conditions, or between days within condition ($P > 0.05$). Noradrenaline was higher post TT on D2 of SD (2213 ± 495 pg·mL^{-1}) compared with D1 of SD (1916 ± 722 pg·mL^{-1}, $P < 0.05$). No other differences in noradrenaline were found for 'pre' (SD: D1, 770 ± 333, D2, 636 ± 279; NS: D1, 688 ± 386, D2, 836 ± 231 pg·mL^{-1}) or 'post' (NS: D1, 2238 ± 699, D2, 1956 ± 444 pg·mL^{-1}) time points.

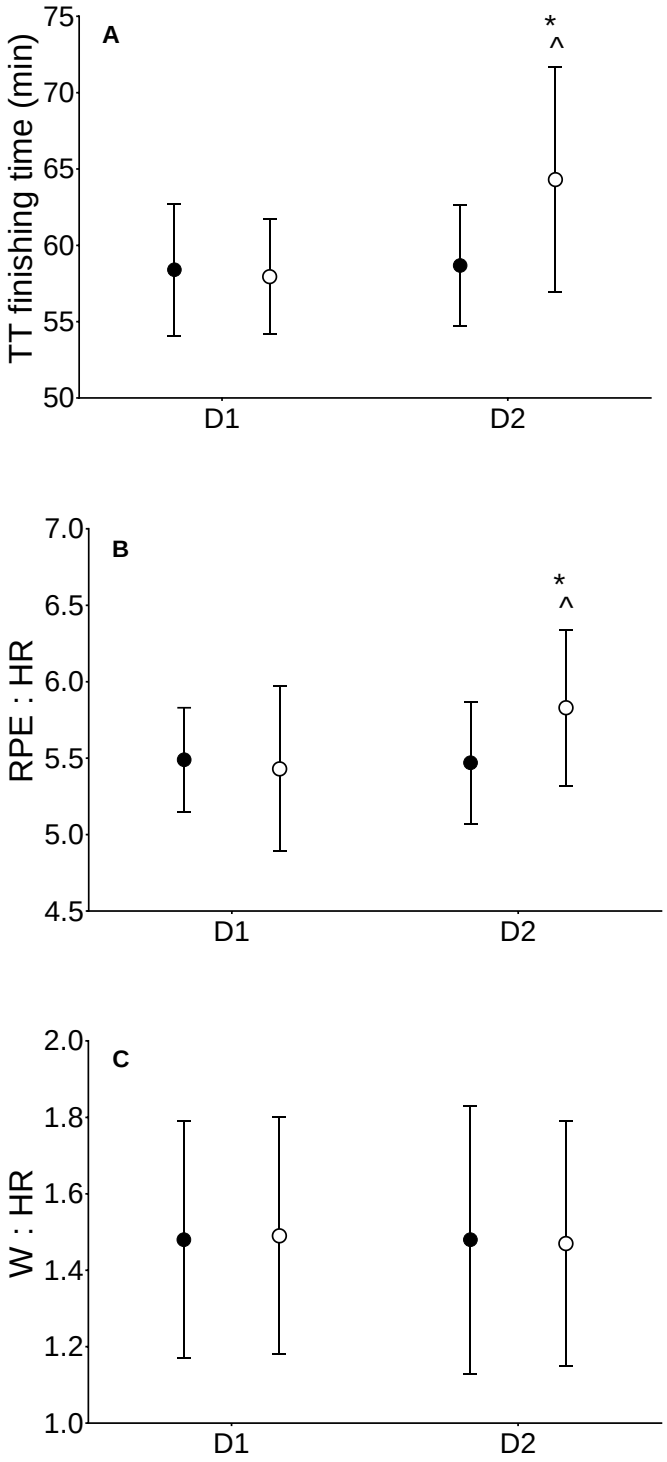

Figure 3.2. Time-trial (TT) finishing time (A), subjective to objective intensity ratio, RPE:HR (B), and external to internal intensity ratio, W:HR (C) for normal sleep (black circles) and sleep deprivation (white circles) conditions. *Different ($P<0.05$) compared with testing day one (D1) of same condition. ^Different ($P<0.025$) compared with testing day two (D2) of Normal Sleep condition. Data presented as mean ± SD.

Table 3.3. Time-trial outcomes.

	Normal Sleep		Sleep Deprivation	
Days	**D1**	**D2**	**D1**	**D2**
Finishing Time (min)	58.4±4.3	58.7±4.0	57.9±3.8	64.3±7.4*^
Peak HR (beats·min^{-1})	174±11	174±11	175±11	165±14*^
Mean HR (beats·min^{-1})	162±11	161±8	162±10	151±13*^
Mean Power (W)	239±59	238±58	241±58	222±66*^
RPE (CR-10 scale)	8.9±0.8	8.8±0.9	8.8±1.1	8.8±1.0
W:HR	1.48±0.31	1.48±0.35	1.49±0.31	1.47±0.32
RPE:HR (×100)	5.49±0.34	5.47±0.40	5.43±0.54	5.83±0.51*^

*Different ($P < 0.05$) compared with D1 of sleep deprivation condition. ^Different ($P < 0.025$) compared with D2 of normal sleep condition. Data shows mean ± SD.

Preliminary testing

Table 3.4 shows total mood disturbance, confusion, and fatigue were higher ($P < 0.01$) on D2 of SD compared with D2 of NS ($d = 1.0, 1.1, 1.1$, respectively) and D1 of SD. Vigour was lower ($P < 0.01$) on D2 of SD compared with D2 of NS ($d = 1.3$) and D1 of SD. PVT mean response time was longer ($P < 0.01$) on D2 of SD compared with D2 of NS ($d = 1.3$) and D1 of SD. PVT lapses were higher ($P < 0.01$) on D2 of SD compared with D2 of NS ($d = 1.4$) and D1 of SD. Neither the HR response to the submaximal exercise test, nor the estimated change in plasma volume from D1 to D2, was different between or within conditions (Table 3.4).

Daily heart rate and heart rate variability

Ln rMSSD was not different for any day between or within conditions. Compared with NS, mean HR was lower ($P < 0.01$) in the SD condition on +1. In the SD condition, mean HR was higher ($P < 0.05$) on +2 compared with baseline -1 (Table 3.2). There were no differences in mean HR between conditions.

Correlations

A strong correlation was found between participants' AT and the percentage change in TT finishing time from D1 to D2 of the SD condition ($R = -0.73$, $R^2 = 0.53$, $P < 0.01$, Figure 3.3A). Although one participant was identified as a potential outlier (i.e., change in TT performance > 3 × SD of the sample mean), removal of this participant from the analysis only slightly reduced the strength of this correlation ($R = -0.65$, $R^2 = 0.42$, $P < 0.03$, Figure 3.3B). There were no significant correlations between changes in respective HR indices and changes in TT finishing time either with (Figure 3.4), or without the outlier participant ($P > 0.05$).

Table 3.4. Preliminary testing outcomes recorded prior to time-trials.

	Normal Sleep		Sleep Deprivation	
Days	**D1**	**D2**	**D1**	**D2**
Profile of Mood States				
Total Mood Disturbance	9±21	6±11	7±17	22±21*^
Anger	4±3	4±3	4±3	3±2
Confusion	6±4	5±2	5±3	9±5*^
Depression	5±5	4±4	4±4	4±3
Fatigue	5±5	5±3	5±6	10±6*^
Tension	7±5	5±4	8±7	7±5
Vigour	18±5	17±4	19±5	11±5*^
Psychomotor Vigilance Task				
Mean Response (ms)	365±36	352±35	355±31	403±43*^
Lapses (>500ms)	3±2	2±2	2±2	7±5*^
Submaximal Exercise Test				
Baseline HR (beats·min^{-1})	66±10	66±13	65±9	65±10
HR Amplitude (beats·min^{-1})	63±10	64±10	63±10	63±11
Time Delay (s)	0.2±0.8	0.7±1.3	0.9±1.5	1.1±1.6
Tau (s)	22.1±6.8	22.6±6.7	20.9±3.8	22.7±5.4
Power (W)	185±44	185±44	185±44	185±44
Steady-state HR	130±8	132±7	130±8	129±7
W:HR	1.43±0.33	1.41±0.35	1.42±0.32	1.44±0.35
rHRI (beats·min^{-1}·sec^{-1})	3.2±1.1	3.3±1.3	3.2±0.5	3.1±1.0
HR recovery (beats·min^{-1})	46±9	44±10	42±9	44±7
Plasma volume assessment				
Haematocrit (%)	42±3	41±3	42±3	42±2
Haemoglobin (g·dL^{-1})	14.5±1.2	14.6±1.3	14.5±1.2	14.5±0.8
Change in plasma volume (%)	-	-0.2±6.7	-	-0.7±5.8

*Different ($P < 0.05$) compared with D1 of sleep deprivation condition. ^Different ($P < 0.025$) compared with D2 of normal sleep condition. Data shows mean ± SD.

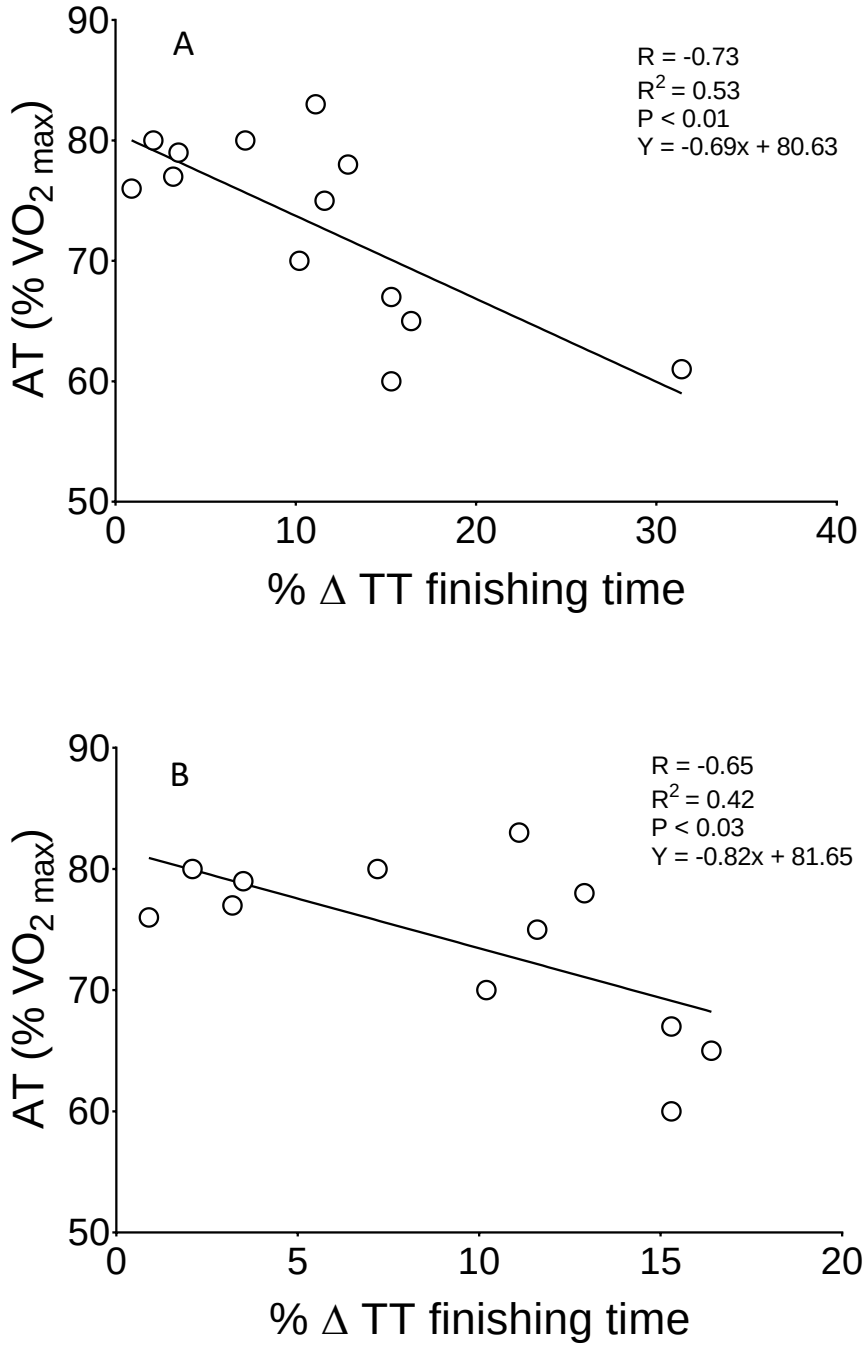

Figure 3.3. Correlations between individual anaerobic threshold (AT) and percentage change in time-trial finishing time from D1 to D2 of the sleep deprivation condition. Panels show correlation for all participants (A) and following the removal of a single outlier participant that recorded a change in TT finishing time of 31.4% (a change >3×SD of the sample) (B).

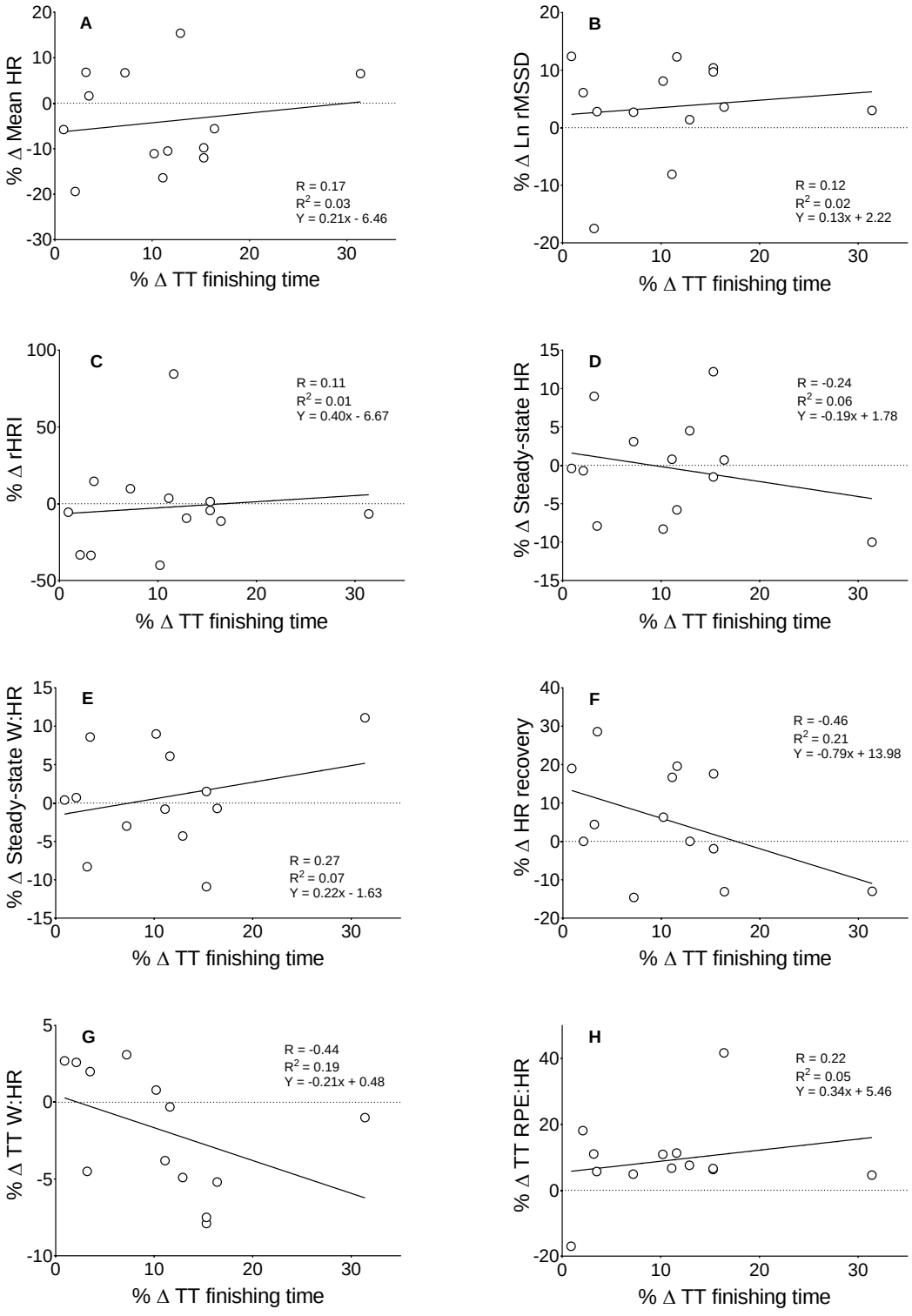

Figure 3.4. Correlations between percentage changes in TT finishing time from D1 to D2 of the SD condition and changes in resting mean HR (A); resting Ln rMSSD (B); maximal rate of heart rate increase at onset of the submaximal exercise test, rHRI (C); steady-state HR during the submaximal exercise test (D); external to internal intensity ratio at steady-state of the submaximal exercise test, steady-state W:HR (E); HR recovery post submaximal exercise test (F); external to internal intensity ratio for the TT, TT W:HR (G); and subjective to objective intensity ratio for the TT, TT RPE:HR (H). There were no statistically significant correlations ($P > 0.05$).

3.5 Discussion

This study examined effects of a single night of total sleep deprivation on prolonged (~60-minute) self-paced endurance cycling performance. Compared with normal sleep, total sleep deprivation impaired performance by 10% (5 min) and increased subjective to objective intensity ratio (RPE:HR) during exercise. However, sleep deprivation did not affect other HR indices often used for monitoring athletes' readiness or fatigue status.

Sleep deprivation and endurance performance

Few studies have examined the effects of sleep loss on *self-paced* endurance performance, and this study is the first to do so using a prolonged ~60-minute TT protocol intended to mimic the psychophysiological demands of many endurance sports (e.g., road cycling, triathlon).[61 426] While total sleep deprivation is uncommon, athletes often experience severe sleep loss, particularly on the night of competition.[13] Therefore, our findings are pertinent for endurance athletes who routinely compete or train on consecutive days (e.g., road cyclists). Previous studies examining the effects of sleep loss on self-paced endurance performance have reported similar findings.[24 241 245] In team sport athletes, *total* sleep deprivation slowed intermittent sprint times across a 50-minute exercise protocol.[241] While in active males, *total* sleep deprivation reduced distance covered during a 30-minute treadmill run that followed a 30-minute preload at 60% $\dot{V}O_{2\,max}$.[245] Similarly, one night of severe sleep *restriction* (i.e., reduced habitual total sleep time), whereby cyclists slept just 2.4 hours prior to undertaking a 3 km time-trial, led to a 4% slower finishing time compared with performance following a normal night of sleep (7.1 hours).[24]

Other studies investigating the effects of total sleep deprivation on endurance performance have assessed *time-to-exhaustion* at prescribed workloads.[26 242-244] Most studies have found that total sleep deprivation reduces walking,[242 243] and cycling time-to-exhaustion.[26 244] However, in endurance athletes, time-to-exhaustion has been shown to be both unaffected,[25] and reduced following total sleep deprivation.[26] Such discrepant findings may reflect differences in the duration of the endurance task used to assess performance. For example, total sleep deprivation reduced the time-to-exhaustion of endurance athletes during a ~20-minute incremental test that followed a 40-minute submaximal exercise preload,[26] but did not reduce the time-to-exhaustion of endurance runners during a ~12-minute stand-alone test.[25] We speculate that longer time-to-exhaustion efforts are more likely to be impaired by sleep deprivation, as these tasks require more sustained periods of self-regulation.

The strong negative correlation between AT and percentage change in TT time in the SD condition (Figure 3.3) suggests cardiorespiratory fitness or training history may also moderate the effect of total sleep deprivation on endurance performance. Anaerobic threshold has been shown to increase with endurance training,[425] and the high thresholds recorded in elite endurance athletes have been attributed to their history of strenuous training.[60] Thus, Figure 3.3 suggests fitter or better-trained athletes may be less susceptible to performance impairment following total sleep deprivation. This is consistent with a previous study that found total sleep deprivation reduced time-to-exhaustion in volleyball players, but not endurance-trained runners.[25] Previously, it was demonstrated that professional cyclists were more resistant to mental fatigue than recreational cyclists, and it was speculated that endurance training develops critical mental capacities that help athletes better

tolerate mental fatigue,[427] which might explain the strong correlation between AT and change in TT time in the SD condition. Future research should examine the impact of sleep deprivation on the endurance performance of athletes with differing fitness levels to further explore this issue. In addition, research should examine whether psychological interventions[33] can mitigate the effects of sleep loss on performance.

Sleep deprivation and perception of effort

Sleep deprivation led to a reduction in mean HR and power output during the TT, but did not reduce perceived exertion (Table 3.3). In addition, POMS scores for fatigue, confusion, and total mood disturbance increased. While sleep deprivation also increased PVT mean response time and number of lapses (Table 3.4). These findings are consistent with previous research showing total sleep deprivation slows reaction times, reduces time-to-exhaustion, and increases RPE at a given exercise intensity.[26 242] Previous research has found no impact of sleep deprivation on neuromuscular function, suggesting central fatigue does not impair performance.[26] However, mental fatigue can be considered distinct from central fatigue in that it increases the perceived exertion required for muscle activation, rather than inhibits muscle activation itself.[72] While mental fatigue was not directly measured in the present study, effects on performance and perceived exertion observed following sleep deprivation parallel the effects previously reported following mentally fatiguing tasks.[74] Furthermore, perturbations in mood and psychomotor vigilance, as observed in the present study, are recommended as surrogate indications of mental fatigue when working with athletes in the field.[72] Therefore, current findings suggest sleep deprivation may have impaired performance by exacerbating mental fatigue during exercise.

Perceived exertion is a measure of the conscious effort required to overcome fatigue,[38 94] and reflects the level of central motor drive required to recruit muscle.[83] According to the psychobiological model, athletes disengage from an endurance task when the perceived effort required exceeds the maximum effort they are willing to exert, or believe they are capable of.[94] Perceived exertion was consistently near maximal (e.g., 9 ± 1) between conditions, suggesting the *perceived* effort athletes were willing to exert was unaffected by sleep deprivation. However, sleep deprivation increased the perceived exertion of a given power output, thus reducing mean power output during the TT.

It is difficult to elucidate the mechanisms that increase mental fatigue, and/or perceived exertion for a given workload, during endurance exercise. However, an increase in extracellular cerebral adenosine is thought to be the primary mediator.[78] Adenosine accumulates in the brain during wakefulness, and as an inhibitory neurotransmitter,[81] may increase perceived exertion via increases in the neural activity required for muscle activation,[83] or for important higher-order cognitive processes, such as self-regulation of pacing or pain.[96 428] Another factor that may, to some extent, explain the current findings is a reduction in muscle glycogen, as total sleep deprivation previously led to modest (e.g., ~65 $mmol.kg^{-1}dw$) reductions in muscle glycogen, which may signal fatigue in order to down-regulate muscle activation and prevent glycogen depletion.[241]

Sleep deprivation and heart rate indices

Sleep deprivation led to reductions in mean and peak HR, and an increase in RPE:HR, during the TT. While mean HR monitored in isolation during self-paced efforts has limited utility, when combined with subjective metrics to compare

perceived intensity with actual (i.e., physiological) intensity, it can provide insightful information about the perceived 'cost' of exercise.[371] Increases in RPE:HR were recently reported in professional cyclists when exercise load increased during a three-week grand tour, and it was suggested RPE:HR may be a useful tool for monitoring fatigue during competition.[109] While the present findings provide evidence that this ratio is moderated, to some extent, by prior sleep time, further research is required to determine whether more subtle sleep manipulations influence RPE:HR. Interestingly, there was no significant correlation between the change in RPE:HR and the change in TT time in the sleep deprivation condition (Figure 4.4). Therefore, while an increase in RPE:HR following sleep loss suggests endurance performance may be impaired, the magnitude of RPE:HR increase is not necessarily indicative of the magnitude of performance impairment. Sleep deprivation did not perturb other HR indices recorded at rest, or during the submaximal exercise test. Thus, collectively, findings highlight the importance of including psychometrics, such as RPE, in athlete monitoring protocols.

Increased subjective to objective intensity ratio (RPE:HR) was indicative of lower mean HR, rather than increased RPE, following sleep deprivation. Lower mean HR will occur with a drop in workload, which we speculate is caused by down regulation of pacing due to mental fatigue. However, additional peripheral factors speculated to lower exercising HR following sleep deprivation include increases in plasma volume,[394 395] reduced supply of catecholamines,[51 243] or decreased sensitivity of catecholamine receptors.[109] In the present study, estimated changes in plasma volume from D1 to D2 were consistent between conditions (Table 3.4), and sleep deprivation did not affect adrenaline concentrations. However, sleep deprivation did lead to *higher* noradrenaline concentration post TT on D2 compared

with D1 of SD. Such elevated noradrenaline levels despite lower peak HR on D2 compared with D1 of SD, suggest a decrease in catecholamine receptor sensitivity *may* have occurred. Indeed, perhaps decreased receptor sensitivity is a contributing mechanism explaining the increase in perceived exertion for a given workload following sleep deprivation (i.e., due to increased neural activation of cardiac muscle). Nonetheless, given previous research has found time-to-exhaustion reduced following sleep deprivation without any change in plasma catecholamines or mean HR,[243] and that we found no statistical difference in plasma catecholamines between conditions on D2 despite a difference in TT performance, any potential decrease in catecholamine receptor sensitivity is unlikely to fully explain performance decrements following sleep deprivation.

Limitations

The present study recruited well-trained male athletes, thus generalising findings for elite or female athletes may require caution. The study analysed HR indices typically used for monitoring athletes in the field. Therefore, findings do not represent an exhaustive examination of cardiac autonomic function. Caffeine withdrawal symptoms peak 20-50 hours post-abstinence,[429] thus TT performances on D1 may have been impaired by withdrawal symptoms. However, given the crossover nature of the study, this is unlikely to influence conclusions. Finally, participants' normal sleep duration (~6 hours, Table 3.2) was less than the 7 hours per night recommended for good health[202], thus findings may not reflect the effects of sleep deprivation on athletes who habitually sleep longer. Nonetheless, athletes often sleep ~6 hours per night during training and competition periods,[13] therefore, conclusions remain relevant for athletic populations.

Conclusion

Sleep is important for optimising endurance performance, as total sleep deprivation impaired prolonged (~ 60-minute) self-paced endurance performance by 10%. Subjective to objective intensity ratios (e.g., RPE:HR) may be sensitive to the effects of sleep on the fatigue status, or readiness, of endurance athletes. Results highlight the importance of psychometric indices in athlete monitoring protocols. Future research should examine more subtle manipulations of sleep in athletes.

Chapter 4: Study 2a

This chapter is presented as per the manuscript accepted for publication in Medicine and Science in Sports and Exercise. However, online supplementary material referenced in the accepted manuscript has been included in this chapter, thus table numbers differ to the accepted manuscript:

Roberts SSH, Teo W-P, Aisbett B, Warmington SA. Extended sleep maintains endurance performance better than normal or restricted sleep. *Med Sci Sports Exerc* 2019;51(12):2516-523.

4.1 Abstract

Purpose: The cumulative influence of sleep time on endurance performance remains unclear. This study examined effects of three consecutive nights of both sleep extension and restriction on endurance cycling performance. **Methods:** Endurance cyclists/triathletes (n = 9) completed a counterbalanced crossover experiment with three conditions; sleep restriction (SR), normal sleep (NS), and sleep extension (SE). Each condition comprised seven days/nights of data collection (-2, -1, D1, D2, D3, D4, +1). Sleep was monitored using actigraphy throughout. Participants completed testing sessions on days D1-D4 that included an endurance time-trial (TT), mood, and psychomotor vigilance assessment. Perceived exertion (RPE) was monitored throughout each TT. Participants slept habitually prior to D1; however, time in bed was reduced by 30% (SR), remained normal (NS), or extended by 30% (SE) on nights D1, D2, and D3. Data were analysed using Generalised Estimating Equations. **Results:** On nights D1, D2, and D3, total sleep time was longer ($P < 0.001$) in the SE condition (8.6 ± 1.0; 8.3 ± 0.6; 8.2 ± 0.6 h, respectively), and shorter ($P < 0.001$) in the SR condition (4.7 ± 0.8; 4.8 ± 0.8; 4.9 ± 0.4 h) compared with NS (7.1 ± 0.8; 6.5 ± 1.0; 6.9 ± 0.7 h). Compared with NS, TT performance was slower ($P < 0.02$) on D3 of SR (58.8 ± 2.5 vs 60.4 ± 3.7min) and faster ($P < 0.02$) on D4 of SE (58.7 ± 3.4 vs 56.8 ± 3.1min). RPE was not different between or within conditions. Compared with NS, mood disturbance was higher, and psychomotor vigilance impaired, following sleep restriction. Compared with NS, psychomotor vigilance improved following sleep extension. **Conclusion:** Sleep extension for three nights led to better maintenance of endurance performance compared with normal and restricted sleep. Sleep restriction impaired performance. Cumulative sleep time affects performance

by altering the perceived exertion of a given exercise intensity. Endurance athletes should sleep > 8 hours per night to optimise performance.

4.2 Introduction

Endurance athletes experience high levels of physical and psychological stress during training and competition.[52] For example, elite road cyclists pedal more than 30,000 km·yr^{-1}, and during stage races, will compete for 4-6 h·day^{-1} on consecutive days.[52] Sleep is considered an important recovery behaviour that may help athletes tolerate such demands;[7] however, the influence of sleep on endurance performance remains unclear.

No study, to our knowledge, has investigated the effects of sleep *extension* (i.e., increased habitual total sleep time) on endurance performance. In non-endurance athletes, sleep extension has been reported to improve the serving accuracy of tennis players,[23] and the shooting accuracy and sprint times of basketballers.[20] However, in the latter study, the absence of a control arm suggests improvements may have been attributable to training adaptations rather than sleep extension.[20] Studies investigating effects of sleep *restriction* (i.e., decreased habitual total sleep time) on endurance performance have reported equivocal findings.[24 248-250 252 430] Moreover, these studies have often recruited untrained participants,[250 252 430] assessed performance using relatively brief (<30 minutes) intermittent[252 430] or graded exercise tests,[250] or examined effects of a single night of sleep restriction.[24 249 252 430]

Given many endurance athletes (e.g., road cyclists) train or compete for prolonged periods (≥ 60 minutes), and on consecutive days, and in light of evidence that athletes' sleep is often disturbed during training and competition,[13] further

investigation of the cumulative effects of sleep time on endurance performance is required. The present study examined the effects of both sleep *extension* and *restriction* across three consecutive nights on endurance cycling performance.

4.3 Methods

Participants

Nine males (mean ± SD, Age: 30 ± 6 years, $\dot{V}O_{2\,max}$: 63 ± 6 mL·kg^{-1}·min^{-1}) were recruited from cycling (n = 7) and triathlon (n = 2) clubs. Athletes were considered 'trained' according to adapted criteria for classifying cyclists (≥ 1 year competitive racing, ≥ 3 training sessions per week, $\dot{V}O_{2\,max}$ ≥ 55 mL·kg^{-1}·min^{-1}).[35] To screen for sleep problems and high anxiety, inclusion criteria required a score ≤ 5 in the Pittsburgh Sleep Quality Index (PSQI),[414] and ≤ 40 in the State-Trait Anxiety Inventory (STAI-Y).[413] Participants did not habitually consume high levels of caffeine (mean ± SD, caffeine products per day: 2±1). The Morningness-Eveningness Questionnaire determined participants were mostly 'moderate morning' types (n = 5), with the remainder being 'definite morning' (n = 2) or 'intermediate' (n = 2) types.[415] The study was approved the Deakin University Human Research Ethics Committee, and informed consent was obtained before participation.

Overview

Participants completed a counterbalanced crossover experiment with three conditions; sleep restriction (SR), normal sleep (NS), and sleep extension (SE). Beforehand, participants had their habitual sleep monitored for a minimum four nights, and undertook two familiarisation sessions comprising an incremental exercise test and a practice time-trial (TT), respectively. Each condition comprised seven consecutive days / nights (-2, -1, D1, D2, D3, D4, +1) of data collection

(Figure 4.1). Participants undertook four testing sessions (D1-D4) at the Deakin University Human Research and Performance Laboratory. During these sessions, participants completed an endurance TT, subjective mood evaluation, and a psychomotor vigilance task. For all conditions, participants slept habitually prior to D1. However, for the three subsequent 'intervention' nights (D1, D2, D3), habitual 'time-in-bed' was either reduced by 30% (SR), extended by 30% (SE), or remained normal (NS). Required time in bed for the intervention nights was calculated according to participants' habitual sleep recorded prior to the experiment. Participants were prescribed bedtimes and get-up times on nights D1, D2, and D3 to ensure the required time in bed was achieved. Bedtimes and get-up times were tailored to individual chorotype to maximise the likelihood of modifying 'total sleep time' rather than simply 'time-in-bed'. For example, sleep extension for a 'morning type' was prescribed by predominantly advancing bedtime rather than delaying get-up time. To minimise the effect of circadian variations on performance, all testing commenced between 6:00-9:00 am. Testing start times were consistent for each participant on D1 of each condition, and on D2, D3, and D4 of the NS condition (mean ± SD, start-time, 7:08 am ± 31 min). Testing start times on D2, D3, and D4 of the SE condition were slightly later to allow for prescribed time in bed increases (mean ± SD, start time, 7:48 am ± 37 min). Testing start times on D2, D3, and D4 of the SR condition were slightly earlier to reduce idle time after waking and thus minimise the risk falling back asleep (start-time, 6:32 am ± 30 min). No circadian variation in prolonged (e.g., 60-minute) endurance performance has been established for time of day differences such as those that occurred in the present study (e.g., 6:30am vs 7:50am).[431]

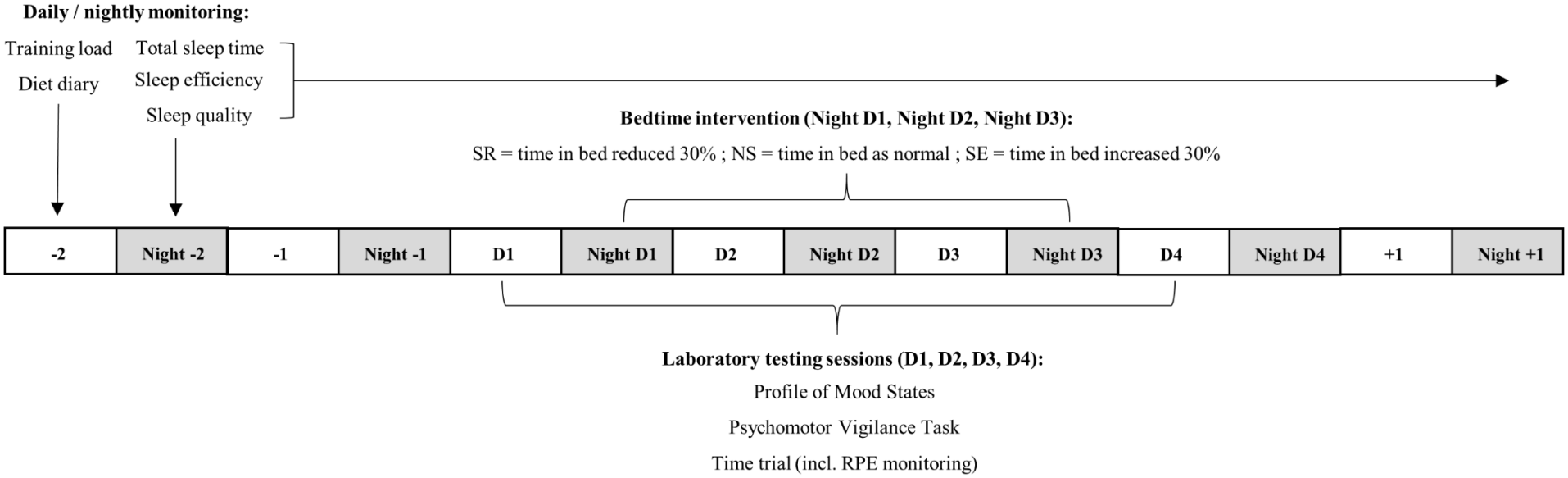

Figure 4.1. Overview of data collection across the seven days / nights of each condition. Training load and diet were self-reported every day. Total sleep time, sleep efficiency, and subjective sleep quality were monitored throughout (-2 to +1). Bedtimes were prescribed on nights D1 to D3 according to the experimental condition being undertaken. Laboratory testing was undertaken on days D1 to D4.

All participants had either morning or intermediate chronotypes, and all routinely trained in the morning. Thus, all testing was undertaken at a time when participants would normally be awake,[415] and when they would often be training. Consumption of caffeine and alcohol was prohibited on days -1 to D4. Athletes were experienced racers, so dietary requirements were self-determined. However, to prevent discrepancies in energy availability, athletes recorded (e.g., 7 am; 1 cup oats with milk) and replicated their dietary intake for each condition. Exercise was prohibited on days -1 to D4 (other than that required for the experiment). However, to accommodate preferred preparation and recovery strategies, participants were permitted to exercise lightly on days -2, +1, and +2, and were required to replicate this exercise between conditions. Participants recorded all exercise so load could be quantified.[416] No differences between conditions were noted on days prior to, or post laboratory testing (Table 4.1). A minimum seven-day washout period was required between D4 of a condition and D1 of the next condition.

Incremental test

On a cycle ergometer (Excalibur Sport, Lode, Groningen, Netherlands) controlled using compatible software (Lode Ergometry Manager 9, Lode, Groningen, Netherlands) participants cycled for three minutes at 75, 125, and 175 W respectively, before workload increased by 25 W every minute until volitional exhaustion. An Innocor metabolic system (DK-5260, Innovision, Odense, Denmark) determined maximal oxygen uptake ($\dot{V}O_{2\,max}$) and anaerobic threshold (AT).

Table 4.1. Self-reported training load.

Day	-2	-1	D1	D2	D3	D4	+1	+2
Sleep Restriction	405±139	0±0	538±45	543±56	564±55	572±63^{+}	258±153	342±101
Normal Sleep	392±134	0±0	545±29	541±51	555±32	555±56^{+}	252±150	387±117
Sleep Extension	403±111	0±0	543±46	532±56	543±47	531±47	267±113	395±106

Data shows arbitrary units for the product of exercise time (min) and session perceived exertion (CR-10 scale).[412] Participants recorded all exercise sessions undertaken for the duration of each condition (-2 to +2), including laboratory TTs on days D1-D4. Participants were required to avoid exercise on day -1 prior to the commencement of laboratory testing the next day. $^{+}$ Different ($P < 0.025$) compared with sleep extension. Data shows mean ± SD.

Time-trial protocol

Target work for the TT was the estimated work expended when cycling at AT for one hour:

$$Work\ (kJ) = \frac{(W_{AT}\ \times 3600)}{1000}$$

Power at AT (W_{AT}) was determined from a regression of the relationship between oxygen uptake ($\dot{V}O_2$) and power (W) for the first three workloads of the incremental test. The ergometer was set to linear mode and pedalling resistance was calculated according to the following formula:

$$W_{AT} = Linear\ Factor \times Preferred\ Pedal\ Rate^2$$

where the linear factor ensured W_{AT} ocurred at the participant's preferred pedal rate per minute (rev·min^{-1}). A strong correlation has been demonstrated between W_{AT} and one-hour TT performance (r = 0.8, P<0.05).[418] Participants completed one practice TT to refine their pacing strategy. During the TT, work completed (kJ) was displayed on a computer screen. No other feedback or encouragement was provided.

Measures

Sleep

Participants wore activity monitors (Actical MiniMitter / Philips Respironics, Bend, OR) on their non-dominant wrist from day -2 to +2 to monitor sleep.[301 310] Activity counts were recorded in one-minute epochs and downloaded using a device specific interface unit (ActiReader, Philips Respironics, Bend, OR). Raw data was processed with a validated manufacturer proprietary algorithm (Actical v3.10) set to a medium sleep-wake threshold (<40 counts.min^{-1} scored sleep).[301 310]

This threshold has shown 87% agreement with polysomnography when identifying sleep and wake states in elite cyclists.[301] In order to verify, or identify misclassified sleep / wake states, participants completed a sleep diary that required them to record the time of day (i.e., to the nearest minute) they *'began attempting to sleep'*, and the time of day they '*woke up for the last time'* for all sleep episodes.[432] No daytime naps were permitted from day -1 until completion of testing on D4. For all sleep episodes, the total amount of sleep obtained (i.e., total sleep time – TST), and the percentage of time in bed spent asleep (i.e., sleep efficiency) were determined. For analysis, TST was aggregated from the end of one night's main sleep to the end of the next night's main sleep. Mean sleep efficiency was calculated for all sleep episodes during the same period. Subjective sleep quality (SQ) was recorded in the sleep diary upon waking each morning on a 5-point Likert scale (i.e., 1=Very Good, 2=Good, 3=Average, 4=Poor, 5=Very Poor).

Time-trial

Overall finishing time (minutes) was recorded. Target work was divided into four equal splits and perceived exertion (6-20 scale) recorded during the final minute of splits 1-3, and immediately upon completion of split four.[419]

Preliminary testing

Prior to the TT, upon arriving at the laboratory, participants completed psychometric testing. The Profile of Mood States (POMS) assessed the feelings of participants "right now" across 65 mood descriptors, providing scores for total mood disturbance, tension, depression, anger, vigour, fatigue, and confusion.[420] Participants completed a touchscreen version of the psychomotor vigilance task (PVT) on a tablet device using the application sleep-2-Peak (v2.2.1, Proactive Life LLC, New York, NY). This version of the PVT has been validated against

traditional PVT methods.[421] The PVT measured reaction times to visual stimuli occurring at varying intervals over 10 minutes. Mean response time and the number of lapses >500 milliseconds were recorded.

Statistical analysis

Mean and SD were calculated for all variables. Generalised Estimating Equations with exchangeable correlation structures and robust standard errors analysed mean changes in outcome variables. Initial models tested for period and carryover effects, however no such effects were found ($P > 0.05$). Models analysed two-, or three-way interactions for the factors 'condition', 'day', and 'split' (RPE only). Where interactions were significant ($P < 0.05$), pairwise models were run for each 'day'. A p-value < 0.025 was used to account for multiple comparisons. Additional models analysed main effects of 'day' for each condition. A p-value < 0.05 was used. For sleep variables, nights -2 and -1 served as baseline values in separate models. For all other variables, D1 served as a baseline value. Analyses were performed in IBM SPSS statistics for Windows (v24.0,Armonk,NY).

4.4 Results

Sleep

Total sleep time (Figure 4.2A) on nights D1, D2, and D3 was longer ($P < 0.001$) in the SE condition (8.6 ± 1.0; 8.3 ± 0.6; 8.2 ± 0.6 h, respectively), and shorter ($P < 0.001$) in the SR condition (4.7 ± 0.8; 4.8 ± 0.8; 4.9 ± 0.4 h), compared with NS (7.1 ± 0.8; 6.5 ± 1.0; 6.9 ± 0.7 h). On night -2 (i.e., two nights prior to commencement of laboratory testing) TST was longer ($P < 0.01$) in the SR condition (7.4 ± 1.0 h) compared with SE (6.9 ± 1.0 h). On night D4 (i.e., following the final laboratory testing session) TST was longer in the SR condition (7.5 ± 0.8 h) compared with SE (6.6 ± 0.9 h, $P < 0.001$) and NS (7.1 ± 0.7 h, $P < 0.02$), while

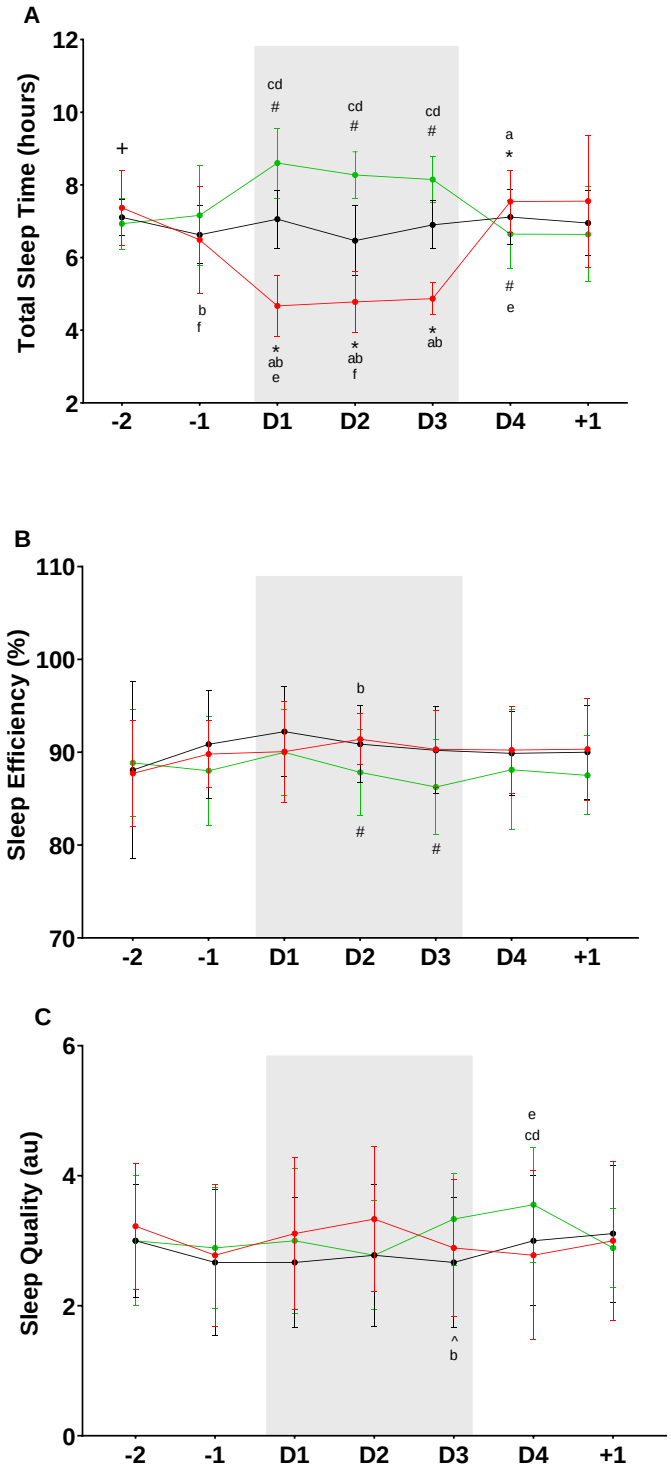

Figure 4.2. Total sleep time (A), sleep efficiency (B) and subjective sleep quality (C) for sleep restriction (red line), normal sleep (black line) and sleep extension (green line) conditions. Data shows mean ± SD. Shaded area represents the nights where bedtime interventions were implemented. # Different ($P < 0.025$) to both normal sleep and sleep restriction. * Different ($P < 0.025$) to both normal sleep and sleep extension. + Difference ($P < 0.025$) between sleep restriction and sleep extension only. ^ Difference ($P < 0.025$) between normal sleep and sleep extension only. a,b Differences ($P < 0.05$) within sleep restriction condition compared with -1 (a) and -2 (b). c,d Differences ($P < 0.05$) within sleep extension condition compared with -1 (c) and -2 (d). e,f Differences ($P < 0.05$) within normal sleep condition compared with -1 (e) and -2 (f)

TST was also longer ($P < 0.02$) in the NS condition compared with SE. On night +1, TST tended ($P = 0.025$) to be longer in the SR (7.6 ± 1.8 h) condition compared with SE (6.6 ± 1.3 h).

Within the SR condition, TST was shorter ($P < 0.01$) on nights D1, D2, and D3 compared with nights -2 and -1, longer ($P < 0.02$) on night +1 compared with night -1, and shorter ($P < 0.01$) on night -1 compared with night -2. Within the NS condition, TST was shorter ($P < 0.05$) on nights -1 and D2 compared with night -2, and longer ($P < 0.05$) on nights D1 and D4 compared with night -1. Within the SE condition, TST was longer ($P < 0.01$) on nights D1, D2, and D3 compared with nights -2 and -1.

On night D2, sleep efficiency (Figure 4.2B) was lower ($P < 0.01$) in the SE condition (88 ± 5%) compared with SR (91 ± 3%) and NS (91 ± 4%). On night D3, sleep efficiency was lower ($P < 0.025$) in the SE condition (86 ± 5%) compared with SR (90 ± 4%) and NS (90 ± 5%). Within the SR condition, sleep efficiency was higher ($P < 0.01$) on night D2 compared with baseline night -2.

On night D3, sleep quality (Figure 4.2C) was better ($P < 0.01$) in the NS condition (2.7 ± 1.0) compared with SE (3.3 ± 0.7). On night D4, sleep quality tended to be better ($P = 0.039$) in the SR condition (2.8 ± 1.3) compared with SE (3.6 ± 0.9). Within the SR condition, sleep quality was better ($P < 0.05$) on night D3 compared with baseline night -2. Within the NS condition, sleep quality was worse ($P < 0.05$) on night D4 compared with baseline night -1 Within the SE condition, sleep quality was worse ($P < 0.05$) on night D4 compared with baseline nights -2 and -1.

See Table 4.2 for a numerical summary of sleep outcomes. This table was submitted as supplementary material for the accepted manuscript.

Table 4.2. Sleep and time-trial outcomes for each of the experimental conditions.

		-2	-1	D1	D2	D3	D4	+1
Sleep Restriction	**Bedtime (hh:mm)**	22:08±00:40	22:30±01:06	23:52±00:33	23:50±00:30	23:48±00:20	21:54±28	22:22±01:12
	Get-up time (hh:mm)	06:32±00:26	05:42±00:36	05:02±00:21	05:08±00:24	05:12±00:16	06:12±26	06:46±00:48
	Time-in-bed (h)	8.4±1.1	7.2±1.7	5.2±0.9	5.3±0.9	5.4±0.6	8.3±0.9	8.4±2.0
	Total sleep time (h)	7.4±1.0	6.5±1.5	4.7±0.8	4.8±0.8	4.9±0.4	7.5±0.8	7.6±1.8
	Sleep efficiency (%)	88±6	90±4	90±5	91±3	90±4	90±5	90±6
	Sleep quality (au)	3.2±1.0	2.8±1.1	3.1±1.2	3.3±1.1	2.9±1.0	2.8±1.3	3.0±1.2
	TT Time (min)	-	-	57.6±1.7	59.5±2.0	60.4±3.7	62.0±5.2	-
	TT Power (W)	-	-	273±42	266±44$^{\wedge}$	263±54	257±62$^{+\wedge}$	-
Normal Sleep	**Bedtime (hh:mm)**	22:10±00:22	22:22±00:34	22:05±00:33	22:26±00:44	22:08±00:27	22:08±00:26	22:32±00:35
	Get-up time (hh:mm)	06:16±00:20	05:40±00:20	05:47±00:21	05:38±00:22	05:49±00:21	06:02±00:22	06:20±00:25
	Time-in-bed (h)	8.1±0.7	7.3±0.9	7.7±0.9	7.2±1.1	7.7±0.8	7.9±0.8	7.8±1.0
	Total sleep time (h)	7.1±0.5	6.6±0.8	7.1±0.8	6.5±1.0	6.9±0.7	7.1±0.7	7.0±0.9
	Sleep efficiency (%)	88±10	91±6	92±5	91±4	90±5	90±5	90±5
	Sleep quality (au)	3.0±0.9	2.7±1.1	2.7±1.0	2.8±1.1	2.7±1.0	3.0±1.0	3.1±1.1
	TT Time (min)	-	-	58.4±2.1	58.0±2.8	58.8±2.5	58.7±3.4	-
	TT Power (W)	-	-	270±44	272±48	269±46*	269±52^{+}	-
Sleep Extension	**Bedtime (hh:mm)**	22:18±00:39	22:03±00:46	20:46±00:38	20:50±00:25	20:54±00:23	22:56±00:31	22:37±00:51
	Get-up time (hh:mm)	06:06±00:27	06:09±00:44	06:22±00:28	06:14±00:17	06:24±00:19	06:26±00:29	06:07±00:39
	Time-in-bed (h)	7.8±1.1	8.1±1.5	9.6±1.1	9.4±0.7	9.5±0.7	7.5±1.0	7.5±1.5
	Total sleep time (h)	6.9±1.0	7.2±1.4	8.6±1.0	8.3±0.6	8.2±0.6	6.6±0.9	6.6±1.3
	Sleep efficiency (%)	89±6	88±6	90±5	88±5	86±5	88±6	88±4
	Sleep quality (au)	3.0±1.0	2.9±0.9	3.0±1.1	2.8±0.8	3.3±0.7	3.6±0.9	2.9±0.6
	TT Time (min)	-	-	57.4±3.4	57.0±3.9	58.2±4.4	56.8±3.1	-
	TT Power (W)	-	-	274±52	276±55	271±58	277±50	-

Sleep data collected the *night of* the day listed (i.e., -2 to +2). Time-trial (TT) data recorded the *morning of* the day listed (i.e., -2 to +2). *D1-D4*, testing days; *hh:mm*, hours: minutes; *au*, arbitrary units; *h*, hours; *min*, minutes; *W*, watts. Statistical analysis of sleep and TT finishing time data is provided in the manuscript. For TT power data, $^{+}$ different (P<0.025) compared with sleep extension, * different (P<0.025) compared with normal sleep, $^{\wedge}$ different (P<0.05) compared with D1 of condition. NB: Additional analysis found no difference (P>0.05) between conditions when comparing the sum of TST over the 48 hours (i.e., sum -2 and -1) prior to laboratory testing (mean ± SD, sleep restriction, 13.9±2.4; normal sleep, 13.7±1.1; sleep extension, 14.1±2.0 h). Data shows mean ± SD.

Time-Trial Performance

Shown in Figure 4.3, time was slower ($P<0.02$) on D3 of SR (60.4±3.7min) compared with NS (58.8 ± 2.5 min). Time was slower ($P < 0.02$) on D4 of SR (62.0 ± 5.2 min) and NS (58.7 ± 3.4 min) compared with SE (56.8 ± 3.1 min). Within the SR condition, time was slower ($P < 0.05$) on D2 and D4 compared with D1, and tended to be slower ($P = 0.053$) on D3 compared with D1. See also Table 4.2 for a numerical summary of TT outcomes.

Time-Trial Perceived Exertion

There was no statistical difference in perceived exertion for any split between conditions, or any split between days within conditions (Table 4.3)

Psychomotor Vigilance Task

Mean response time (Table 4.4) was faster ($P < 0.025$) on D3 and D4 of SE compared with SR and NS, and faster ($P < 0.025$) on D4 of NS compared with SR. Within the SR condition, mean response time was slower ($P < 0.05$) on D2, D3, and D4 compared with D1. Within the NS condition, mean response time was slower ($P < 0.05$) on D2 and D4 compared with D1. Within the SE condition, mean response time was faster ($P < 0.05$) on D4 compared with D1. Lapses were fewer ($P < 0.025$) on D3 and D4 of SE compared with SR and NS. Lapses were fewer on D4 of NS compared with SR. Within the SR condition, lapses were greater ($P < 0.05$) on D3 and D4 compared with D1.

Profile of Mood States

Total mood disturbance (Table 4.4) was higher ($P < 0.025$) on D3 and D4 of SR compared with NS and SE. Within the SR condition, total mood disturbance was higher ($P < 0.05$) on D2, D3, and D4 compared with D1. Confusion was higher

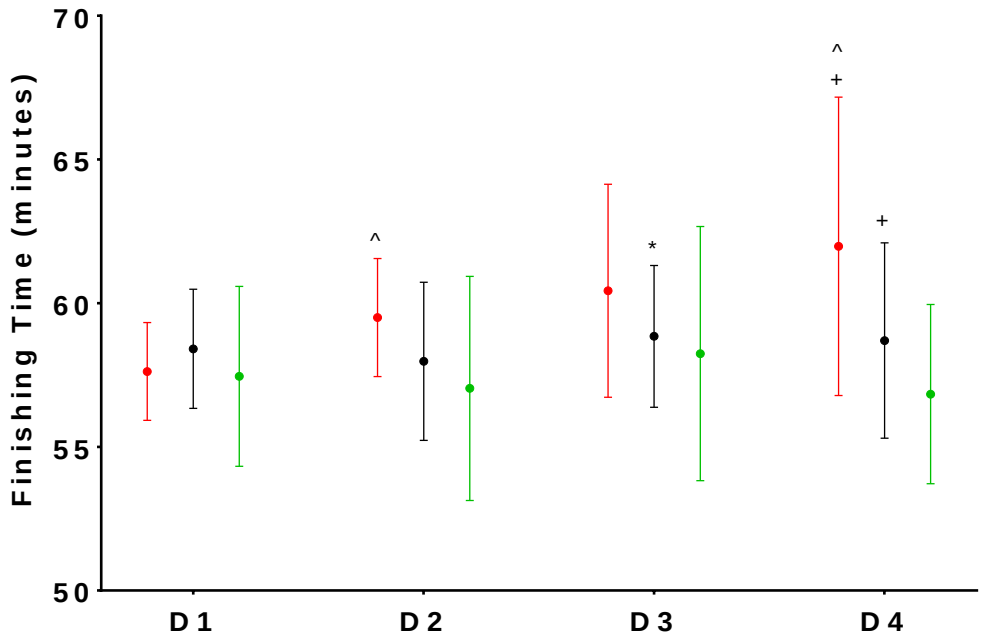

Figure 4.3. Finishing time for each time-trial across the four days (D1-D4) of testing. Sleep restriction (red line), normal sleep (black line), and sleep extension (green line). * Different ($P < 0.025$) to sleep restriction. + Different ($P < 0.025$) to sleep extension. ^ Different ($P < 0.05$) to D1 of the same condition. Data presented as mean ± SD.

Table 4.3. Ratings of perceived exertion recorded for each split during the time-trials.

Day	D1				D2				D3				D4			
Time-Trial Split	**1**	**2**	**3**	**4**	**1**	**2**	**3**	**4**	**1**	**2**	**3**	**4**	**1**	**2**	**3**	**4**
Sleep Restriction	14±1	16±1	17±1	19±1	14±2	16±2	17±2	19±1	14±2	16±1	17±2	19±1	14±1	16±1	17±2	19±1
Normal Sleep	14±1	16±1	17±1	19±1	15±1	16±1	17±1	19±1	15±1	16±1	17±2	19±1	15±1	16±2	17±1	19±1
Sleep Extension	14±1	16±1	17±1	19±1	15±1	16±1	17±1	19±1	15±2	16±1	17±2	19±1	15±2	16±2	17±2	19±1

D1-D4, testing days one to four. No significant differences for any split between conditions ($P > 0.025$), or any split between days within conditions ($P > 0.05$). Data shows mean ± SD.

Table 4.4. Preliminary testing outcomes recorded prior to time-trials.

	Sleep Restriction				Normal Sleep				Sleep Extension			
	D1	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**
Profile of Mood States												
Total mood disturbance	2±11	10±10$^{\wedge}$	25±14$^{+\wedge}$	28±12$^{+\wedge}$	3±13	3±14	9±14*	13±18*	2±10	6±23	7±13	4±10
Anger	2±2	2±1	3±2	2±2	3±2	2±1	2±2	2±2	3±2	4±6	4±5	2±3
Confusion	4±2	6±3	8±2$^{+\wedge}$	9±3$^{+\wedge}$	5±3	4±3	5±4*	5±4*	4±2	4±5	4±3	4±2
Depression	2±2	2±2	4±4	3±3	2±2	2±3	3±2	1±1	3±2	4±6	4±5	2±3
Fatigue	4±4	7±4$^{+\wedge}$	11±4$^{+\wedge}$	14±5$^{+\wedge}$	4±3	6±4	7±5*	9±6$^{*\wedge}$	4±2	4±3	7±5$^{\wedge}$	6±4$^{\wedge}$
Tension	8±6	5±3	6±4	7±4	6±4	4±3	6±3	6±3	6±4	7±5	6±2	5±2
Vigour	17±4	13±3$^{+\wedge}$	7±5$^{+\wedge}$	7±3$^{+\wedge}$	18±5	14±6	13±5*	10±8^{+}	17±6	17±6	17±7	16±5
Psychomotor Vigilance Task												
Mean response time (ms)	347±26	365±30$^{\wedge}$	374±31$^{+\wedge}$	392±40$^{+\wedge}$	348±34	363±30$^{\wedge}$	360±28^{+}	363±28$^{+*\wedge}$	349±32	353±22	346±27	332±29$^{\wedge}$
Lapses (>500ms)	2±1	3±2	4±2$^{+\wedge}$	5±5$^{+\wedge}$	2±1	3±2	3±1^{+}	3±2^{+*}	2±1	2±1	1±1	1±1

D1-D4, laboratory testing days.* Different ($P < 0.025$) compared with Sleep Restriction. $^{+}$ Different ($P < 0.025$) compared with Sleep Extension. $^{\wedge}$ Different ($P < 0.05$) compared with D1 of same condition. Data shows mean ± SD.

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(P < 0.025) on D3 and D4 of SR compared with NS and SE. Within the SR condition, confusion was higher (P < 0.05) on D3 and D4 compared with D1. Fatigue was higher (P < 0.025) on D2, D3, and D4 of SR compared with SE, and higher (P < 0.025) on D3 and D4 of SR compared with NS. Within the SR condition, fatigue was higher (P < 0.05) on D2, D3, and D4 compared with D1. Within the NS condition, fatigue was higher (P < 0.05) on D4 compared with D1. Within the SE condition, fatigue was higher (P<0.05) on D3 and D4 compared with D1. Vigour was lower (P < 0.025) on D2, D3, and D4 of SR compared with SE, and lower on D3 of SR compared with NS. Vigour was higher (P < 0.025) on D4 of SE compared with NS. Within the SR condition, vigour was lower (P < 0.05) on D2, D3, and D4 compared with D1. Within the NS condition, vigour was lower (P < 0.05) on D3 and D4 compared with D1.

4.5 Discussion

Three nights of sleep extension better maintained endurance performance compared with both normal and restricted sleep. Compared with normal sleep, extending sleep time for three consecutive nights by an average of 90, 108, and 78 min, respectively, improved performance by 3%, or ~2 minutes across a ~60-minute TT. In contrast, reducing sleep for two consecutive nights by an average of 144 and 102 minutes respectively, slowed TT performance by 3%, or ~1.5 minutes. Within the sleep restriction condition, performance was slower on day two and four compared with day one. However, performance was consistent over time in the normal and extended sleep conditions.

Sleep extension and endurance performance

Few studies have examined the effects of sleep extension on athletic performance. While extending sleep has been reported to improve sport-specific skill execution

and sprint times,[20 23] this is the first study to examine the performance of endurance athletes. Moreover, previous studies examining sleep extension in athletes have used self-reported sleep times,[23] or have not included a control arm.[20] In contrast, the present study objectively monitored sleep and adopted a three-armed crossover design. In the present study, athletes habitually slept ~6.5-7.0 hours per night, similar to sleep durations reported in elite athletes. While a minimum seven hours of sleep per night is recommended for good health,[202] our findings suggest this may not be sufficient to optimise endurance performance. In fact, on sleep extension nights, athletes slept, on average, 8.4 hours per night (Figure 4.2A), similar to previous studies reporting improved athletic performance when sleep time was extended to 8.4,[20] and 8.9[23] hours per night. Therefore, we recommend athletes sleep >8 hours per night to optimise performance. Sleep efficiency was consistently above 85% (Figure 4.2B), the minimum efficiency recommended for good health.[201] However, sleep extension led to lower sleep efficiency compared with normal and restricted sleep, and poorer subjective sleep quality over time, perhaps indicative of reduced homeostatic sleep pressure (i.e., sleep 'need').[433] Therefore, sleep extension led to better maintenance of performance despite a reduction in sleep efficiency. Although future research should examine the precise effect of sleep efficiency and subjective sleep quality on endurance performance, we recommend practitioners, with the help of valid monitoring / assessment tools,[15 301] work with athletes to optimise both sleep quantity *and* quality.

Sleep restriction and endurance performance

The extent of accumulated sleep pressure may moderate the effect of sleep restriction on endurance performance. Compared with normal sleep, we found performance was unaffected by one night-, but impaired following two nights, of

sleep restriction (i.e., < 5 hours TST per night). Previously, a severe sleep restriction protocol whereby cyclists slept 2.4 hours for one night, led to slower 3 km TT performance compared with 7.1 hours of sleep.[24] In endurance athletes, the maximal workload achieved during a graded exercise test was unaffected when the prior night's sleep opportunity was reduced by three hours,[249] but was lower when sleep opportunity was reduced by four hours.[248] In taekwondo athletes, reducing sleep by 3-4 hours for one night did not affect distance covered during an intermittent test in the morning,[430] but reduced distance covered in the evening.[252] Collectively, these findings suggest performance is likely impaired as sleep pressure/debt accumulates. Apparently contrary to this hypothesis, one study found time to exhaustion during a graded exercise test was unaffected following three consecutive nights of 2.5 hours sleep.[250] Moreover, in the present study, we found performance was not statistically slower (P = 0.09) on day four of sleep restriction, compared with normal sleep. This may reflect, on the part of at least some of the athletes tested, a subconscious increase in motivation for the final TT of the sequence as the fear of premature fatigue diminishes, akin to the 'end-spurt' effect demonstrated within endurance tasks.[434] Nonetheless, within the sleep restriction condition, performance *was* slower on day two and four compared with day one. Therefore, collectively, the present findings suggest athletes should avoid short or restricted sleep, particularly on consecutive nights, for optimal endurance performance.

Cumulative sleep time and perceived exertion

Cumulative sleep time did not affect RPE scores, which were consistently near maximal upon TT completion, despite differences in TT finishing times between conditions (Table 4.3). According to the linear nature of the TT protocol, finishing

times corresponded to mean power output (Table 4.2), thus compared with normal sleep, athletes' perceived exertion for a given power output was higher following sleep restriction (e.g., D3), and lower following sleep extension (e.g., D4). Perceived exertion reflects the effort required to overcome fatigue, and according to the psychobiological model of exercise tolerance, athletes disengage from an endurance task when perceived effort is greater than the maximum effort they are willing to exert, or believe they are capable of exerting.[94] Our findings suggest total sleep obtained over 2-3 nights appears to alter the intensity (i.e., power output) at which these 'effort thresholds' occur. Increased perceived exertion during exercise has been associated with mental fatigue.[435] While we did not measure mental fatigue *per se*, we speculate that prior cumulative sleep time affects the level of mental fatigue experienced, or tolerated, during an endurance task. In fact, sleep extension has been shown to increase pain tolerance (i.e., ability to withstand pain) in healthy adults,[436] which may explain higher power outputs for a given RPE after three nights of sleep extension. Evidence that sleep restriction *impaired* mood and psychomotor vigilance, while sleep extension *improved* vigour and psychomotor vigilance (Table 4.4), further supports speculation that mental/psychological determinants of endurance performance (e.g., attentional focus on pacing, response inhibition etc.)[96] were likely affected by sleep extension and restriction.

Limitations

Participants were well-trained male endurance athletes; therefore, inferences for elite and/or female athletes may require caution. Caffeine withdrawal symptoms peak 20-51 hours post-abstinence,[429] therefore, symptoms may have impaired performances on D1. However, given the crossover nature of the experiment this is unlikely to affect findings. Participants slept ~30 minutes more on night -2 of SR

compared with SE, potentially confounding results. However, total sleep time for the 48 hours prior to D1 was no different (~ 14 hours, see Table 4.2) between conditions, therefore, differences on night -2 are unlikely to affect findings. On D2, D3, and D4, mean start times of testing sessions differed slightly between conditions (see methods 'overview' section), potentially confounding results due to circadian variation in endurance capacity. However, performance differences between conditions did not manifest until after consecutive days of either sleep restriction (e.g., D3) or extension (e.g., D4). Thus, circadian effects cannon explain findings as any effects on performance should have occurred as soon as start times differed (e.g., D2). In addition, findings from studies examining time of day effects on *prolonged endurance performances* (e.g.,~60 minutes) have been equivocal,[431] and any effects of small time of day changes, such as those occurring in the current study (e.g.,~40 minute difference between start times of the NS condition and the SR / SE conditions), have not been established.

Conclusions

Sleep extension for three consecutive nights better maintained prolonged self-paced endurance performance compared with both normal and restricted sleep. Sleep restriction impaired endurance performance. Sleep time accumulated over 2-3 nights appears to influence performance by altering the perceived exertion of a given exercise intensity. Athletes should aim to sleep >8 hours per night to optimise endurance performance.

Chapter 5: Study 2b

This chapter reports findings from Study 2 concerning effects of sleep restriction and extension on HR indices of athlete readiness. Performance data is not presented in this chapter; however, given this chapter examines whether HR indices track changes in athletes' readiness to perform, performance data is used for correlation analysis and is referred to in the 'Discussion' section.

5.1 Abstract

Purpose: It is unclear whether HR indices are sensitive to the cumulative effects of sleep on endurance athletes' readiness to perform. This study examined whether HR indices are sensitive to the effects of three consecutive nights of sleep extension and sleep restriction on athlete readiness. **Methods:** Endurance cyclists / triathletes (n = 9) completed a counterbalanced crossover experiment with three conditions; sleep restriction (SR), normal sleep (NS), and sleep extension (SE). Each condition comprised seven days / nights of data collection (-2, -1, D1, D2, D3, D4, +1). Participants slept habitually prior to D1; however, time in bed was reduced by 30% (SR), remained normal (NS), or extended by 30% (SE) on nights D1, D2, and D3. On D1-D4, HR responses to constant-load submaximal exercise and a cycling TT were examined. Mean HR for the TT was incorporated into intensity ratios (e.g., RPE:HR, W:HR). Resting HR and HR variability (Ln rMSSD) were recorded from day -2 to +2. Data were analysed using Generalised Estimating Equations and Pearson's Correlations examined relationships between HR indices and TT performance. **Results:** On D4, RPE:HR was lower ($P < 0.02$) in the SE condition compared with both NS and SR conditions. Within the SR condition, RPE:HR was higher ($P < 0.02$) on D3 and D4 compared with D1. Strong correlations were found between percentage changes in W:HR from D1 to D4 and changes in TT finishing time for both SR ($r = -0.67$, $P < 0.05$) and SE ($r = -0.69$, $P < 0.05$) conditions. **Conclusions:** Intensity ratios during high-intensity exercise (e.g., races) that incorporate mean HR may be sensitive to the effects of cumulative sleep time, over 1-3 nights, on endurance athletes' readiness to perform. Cumulative sleep time should be considered a potential mediating factor when using intensity ratios to analyse endurance performances.

5.2 Introduction

Heart rate indices recorded at rest (e.g., HR variability), during short submaximal exercise tests (e.g., rHRI),[321] or computed with external or subjective intensity indices (e.g., RPE:HR),[109] are commonly used to monitor athletes' fatigue status or readiness to perform. For example, HR variability was lower in 'fatigued' Nordic skiers compared with 'non-fatigued' skiers,[340] and RPE:HR increased over the course of a three-week Grand Tour among professional cyclists.[109]

There is a close coupling of central and autonomic nervous system activity during sleep[374], and sleep restriction has been shown to alter daytime HR regulation[249 398-405]. Compared with sleep extension (i.e., 12 hours in bed), sleep restriction (i.e., 4 hours in bed) decreased 24-hour HR variability among healthy males.[31] Despite this, it remains unclear whether HR indices are sensitive to any effects of sleep extension or restriction on endurance athletes' readiness to perform. Therefore, the present study examined the effects of both sleep *extension* and *restriction* across three consecutive nights on HR indices typically used to examine athlete readiness.

5.3 Methods

For a detailed description of the participants, the overall study design, and the incremental test and TT protocols, the reader is referred to the 'methods' section of Chapter 4: Study 2a. The following sections detail the additional tests undertaken and measures obtained during Study 2b that were used to examine whether HR indices are sensitive to any effects of sleep extension and/or restriction on athletes' readiness. All HR data were recorded using a Polar monitor (V800 HR monitor, Polar, Kempele, Finland) and uploaded to the Polar FlowSync (v2.6.2) interface prior to analysis.

Measures

Time-trial

For each TT undertaken on days D1 to D4, peak HR, mean HR, and mean power were recorded. Mean power and mean HR were used to determine the external to internal intensity ratio (i.e., W:HR) for the TT. Participants also provided an overall RPE score for the TT ten minutes after completion using the CR-10 scale.[7] This was used in conjunction with mean HR to determine the subjective to objective intensity ratio (i.e., RPE:HR) for the TT.[109] Importantly, the RPE rating provided ten minutes after completing the TT (i.e., using the CR-10 scale) was *in addition to* RPE scores reported (i.e., using the 6-20 scale) for each split during the TT (Chapter 4: Study 2a, Page 98). The additional use of the CR-10 scale to provide an overall RPE score for the TT was necessary to determine intensity ratios, as the ratio scaling characteristics of the CR-10 are recommended for comparing perceived exertion with physiological responses such as HR.[419 437] The CR-10 scale was previously used when calculating intensity ratios among professional cyclists.[109]

A subset of participants (n = 7) had plasma catecholamine levels assessed on D1 and D4 from blood samples collected prior to (i.e., 'Pre') the submaximal exercise test (see Preliminary testing) and within five minutes of completing the TT (i.e., 'Post'). This provided an assessment of sympathetic nervous system activity in order to clarify the underlying autonomic mechanisms associated with any changes in HR observed. A description of blood collection methods and analysis techniques is provided in Chapter 3: Study 1 (Page 68).

Preliminary testing

On testing days D1 to D4, the HR response to submaximal exercise was examined. A description of the submaximal exercise test protocol, and the mono-exponential

curve fitting techniques used to analyse HR data is provided in Chapter 3: Study 1 (Page 70). Parameters derived from curve fitting included baseline HR (beats·min^{-1}), HR amplitude (beats·min^{-1}), time delay before HR increase (s), and curvature constant (i.e. tau; s).[348] These parameters were then used to determine the maximal rate of HR increase (i.e., rHRI; beats·min^{-1}·s^{-1}) from the first derivative maxima of the function.[348] Mean steady-state HR (beats·min^{-1}) for the final minute of exercise, external to internal intensity ratio (i.e., W:HR), and HR recovery (i.e., beats·min^{-1} recovered in one minute) upon test cessation were also examined.

Daily heart rate and heart rate variability

Participants recorded resting HR upon waking each morning across the seven days of data collection (i.e., -2 to +1). The recording protocol and HR data analysis techniques were described in Chapter 3: Study 1 (Page 70). Mean HR, and the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) were examined.

Statistical analyses

Mean and SD were calculated for all variables. Generalised Estimating Equations with exchangeable correlation structures and robust standard errors analysed mean changes in outcome variables. Initial models tested for period and carryover effects, however no such effects were found ($P > 0.05$). Models analysed two- or three-way interactions for the factors 'condition', 'day', and 'pre-post' time point (i.e., pre and post the TT on D1 and D4 for catecholamines only). Where interactions were significant ($P < 0.05$), pairwise models were run for each 'day'. A p-value < 0.025 was used to account for multiple analyses. Additional models analysed main effects of 'day' for each condition, and main effects of 'day' for each 'pre' or 'post' time

point within condition, with a p-value < 0.05 considered significant. For variables recorded on laboratory testing days, D1 served as a baseline value. For variables recorded across seven days, days -2 and -1 served as baseline values in separate models. Pearson's correlations and linear regressions examined relationships between changes in HR indices and TT finishing times from D1 to D4 in both SR and SE conditions. Percentage changes were examined to account for individual differences. For correlations, a p-value < 0.05 was considered significant. Analyses were performed in IBM SPSS statistics for Windows (v24.0, Armonk, NY).

5.4 Results

Time-trial

Table 5.1 shows peak HR, mean HR, mean power, and RPE results, and Figure 5.1 shows resulting intensity ratios. Please note, although mean power results were presented in Chapter 4: Study 2a (Table 4.2, Page 102), it is presented again in the current chapter to provide context for the W:HR intensity ratio. There were no statistical differences in peak HR between conditions; however, on D4, peak HR tended to be lower in the SR condition compared with NS (P = 0.037) and SE (P = 0.046). Within the SR condition, peak HR was lower on D4 compared with D1 (P < 0.05). On D1, mean HR was lower in the NS condition compared with SR and SE (P < 0.025). On D2, mean HR was higher in the SE condition compared with NS and SR (P < 0.025). On D4, mean HR was higher in the SE condition compared with SR (P < 0.025), and tended to be higher than NS (P = 0.033)

Within the SR condition, mean HR was lower on D2, D3, and D4 compared with D1. On D3, mean power was lower in the SR condition compared with NS (P < 0.025). On D4, mean power was higher in the SE condition compared with both SR

and NS ($P < 0.025$). Within the SR condition, mean power was lower on D2 and D3 compared with D1 ($P < 0.05$). There were no statistical differences between or within conditions for RPE. On D4, RPE:HR was lower ($P < 0.02$) in the SE condition compared with both NS and SR conditions. Within the SR condition, RPE:HR was higher ($P < 0.02$) on D3 and D4 compared with D1. There were no statistical differences in W:HR between or within conditions. Table 5.2 shows catecholamine results. Adrenaline was lower pre-TT on D4 of the SR condition compared with the NS and SE conditions. Within the SR condition, adrenaline was lower pre-TT on D4 compared with D1. Within the SE condition, adrenaline was higher pre-TT on D4 compared with D1.

Submaximal exercise test

There were no statistical differences in curvature parameters, steady-state HR, rHRI, HR recovery, or W:HR between or within conditions (Table 5.3)

Daily heart rate and heart rate variability

There were no statistical differences in resting HR or Ln rMSSD between conditions (Table 5.4). In the SR condition, mean HR was higher, and Ln rMSSD lower, on +1 compared with -1. In the NS condition, mean HR was higher, and Ln rMSSD lower, on +1 compared with -2. In the SE condition, mean HR was higher on D2 and +1 compared with -2.

Table 5.1 Time-trial outcomes across the four laboratory testing days.

	Sleep Restriction				Normal Sleep				Sleep Extension			
	D1	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**
Peak HR (beats·min^{-1})	177±4	177±9	171±13	168±12$^{\wedge}$	176±8	175±5	173±9	175±6	177±6	177±5	174±4	175±3
Mean HR (beats·min^{-1})	161±5	154±9$^{+\wedge}$	154±11$^{\wedge}$	151±13$^{+\wedge}$	155±8*$^{+}$	157±5^{+}	155±7	156±9	161±6	162±6	158±6	161±3
Mean Power (W)	273±42	266±44$^{\wedge}$	263±54	257±62$^{+\wedge}$	270±44	272±48	269±46*	269±52^{+}	274±52	276±55	271±58	277±50
RPE (0-10 scale)	9.3±0.7	9.1±0.8	9.3±0.7	9.2±0.7	9.1±0.3	9.2±0.7	9.3±0.5	9.4±0.5	9.4±0.5	9.4±0.5	9.3±0.5	9.3±0.5
W:HR	1.70±0.32	1.72±0.34	1.71±0.34	1.70±0.33	1.74±0.34	1.73±0.30	1.74±0.32	1.72±0.30	1.71±0.28	1.70±0.28	1.71±0.29	1.72±0.29
RPE:HR(×100)	5.78±0.40	5.91±0.72	6.04±0.57$^{\wedge}$	6.09±0.45$^{+\wedge}$	5.87±0.30	5.86±0.34	6.00±0.37	6.03±0.47^{+}	5.85±0.24	5.82±0.28	5.90±0.39	5.78±0.34

D1-D4, laboratory testing days; W, mean power in Watts; RPE, perceived exertion; HR, heart rate. Note: Intensity ratios use mean HR.* Different ($P < 0.025$) compared with Sleep Restriction. $^{+}$ Different ($P < 0.025$) compared with Sleep Extension. $^{\wedge}$ Different ($P < 0.05$) compared with D1 of same condition. Data shows mean ± SD.

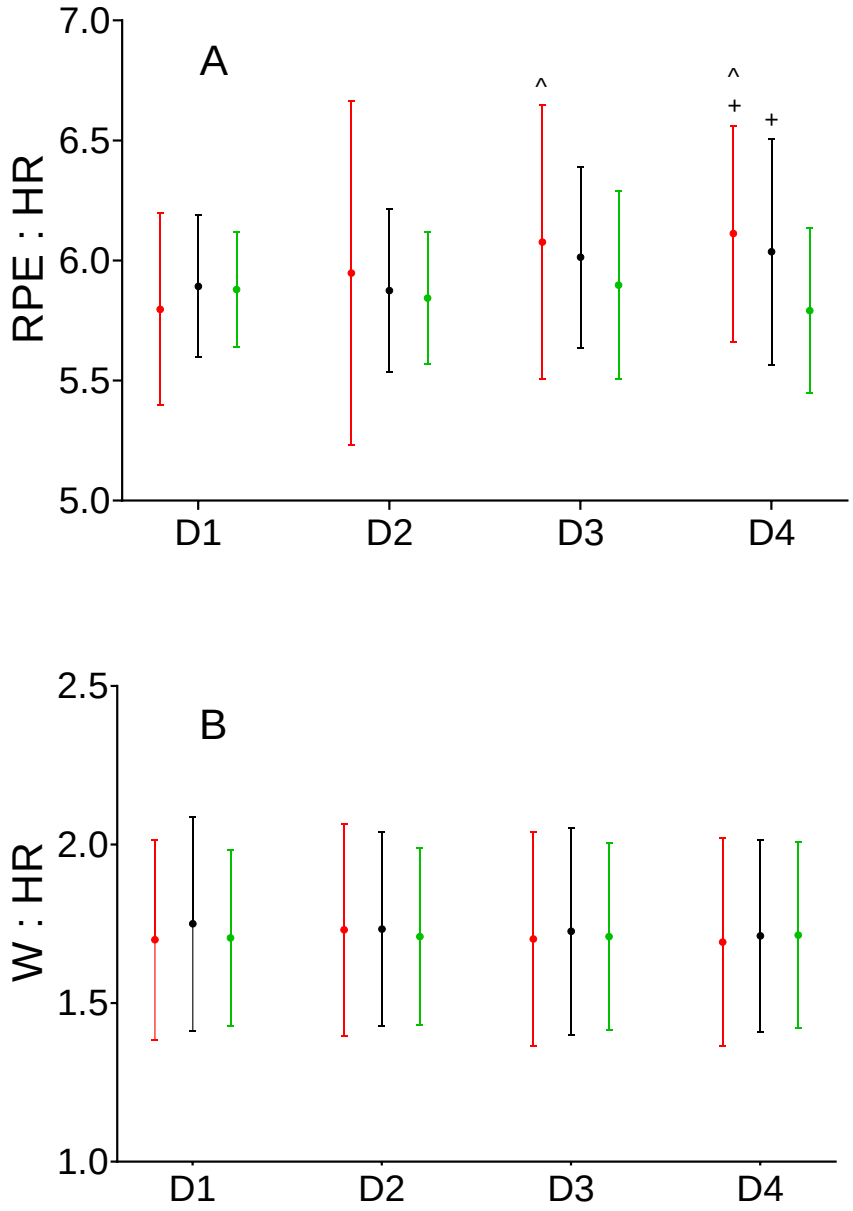

Figure 5.1 Subjective to objective intensity ratio, RPE:HR (A), and external to internal intensity ratio, W:HR (B) for each time-trial across the four days (D1-D4) of testing. Sleep restriction (red), normal Sleep (black), and sleep Extension (green). $^{+}$ Different ($P < 0.025$) to Sleep Extension. $^{\wedge}$ Different ($P < 0.05$) to D1 of same condition. Data presented as mean ± SD.

Table 5.2 Plasma catecholamines measured prior to (pre), and immediately post each time-trial.

		Sleep Restriction		Normal Sleep		Sleep Extension	
		D1	**D4**	**D1**	**D4**	**D1**	**D4**
Adrenaline (pg·mL^{-1})	**Pre**	51±34	25±22$^{+\wedge}$	49±26	42±18*	45±30	67±40$^{\wedge}$
	Post	165±68	231±224	171±103	159±62	178±56	259±202
Noradrenaline (pg·mL^{-1})	**Pre**	609±328	693±229	515±275	594±227	582±314	607±166
	Post	2873±1652	3226±986	2640±1701	3342±3024	2881±1007	3439±1439

D1, testing day one; D4, testing day four. * Different ($P < 0.025$) compared with same day and time point of Sleep Restriction. $^{+}$ Different ($P < 0.025$) compared with same day and time point of Sleep Extension. $^{\wedge}$ Different ($P < 0.05$) compared with the same time point (i.e., 'pre' or 'post') on D1 of same condition. Data shows mean ± SD.

Table 5.3 Curve parameters and heart rate indices derived from the submaximal exercise test conducted prior to time-trials.

	Sleep Restriction				Normal Sleep				Sleep Extension			
	D1	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**	**D1**	**D2**	**D3**	**D4**
Baseline HR (beats·min^{-1})	66±11	69±7	73±8	71±12	68±10	67±6	70±9	70±7	69±13	71±7	70±10	70±9
HR amplitude (beats·min^{-1})	63±12	62±6	59±14	59±14	61±8	59±7	60±10	59±9	60±8	61±12	57±10	59±12
Time delay (s)	0.7±0.9	0.1±0.2	0.3±0.3	0.6±0.8	0.4±0.8	0.2±0.2	0.1±0.3	0.8±1.3	0.4±0.6	0.1±0.3	0.5±1.1	0.4±0.7
Tau (s)	21±4	21±4	22±6	23±6	21±6	20±6	21±4	22±6	21±7	23±9	20±6	23±8
rHRI (beats·min^{-1}·sec^{-1})	3.1±0.9	3.0±0.6	3.0±1.1	2.8±1.2	3.2±1.1	3.2±1.0	3.1±0.9	3.0±0.8	3.2±1.1	3.1±1.5	3.1±0.6	3.0±1.8
Steady-state HR (beats·min^{-1})	131±6	132±9	133±9	132±8	131±9	128±9	131±8	130±11	130±11	134±10	129±10	129±9
HR recovery (beats·min^{-1})	48±11	48±6	49±12	44±10	50±10	47±10	49±8	49±8	51±13	47±8	47±10	45±5
W:HR	1.60±0.36	1.60±0.35	1.59±0.38	1.60±0.39	1.60±0.36	1.64±0.40	1.61±0.37	1.61±0.35	1.61±0.37	1.56±0.35	1.65±0.42	1.63±0.37

D1-D4, laboratory testing days one to four; HR, heart rate, rHRI , maximal rate of HR increase, W:HR, mean power to steady-state HR ratio. There were no statistical differences for any variable between or within conditions. Data shows mean ± SD.

Table 5.4 Resting heart rate and heart rate variability recorded every morning upon waking.

		-2	-1	D1	D2	D3	D4	+1	+2
Sleep Restriction	**Resting HR**	54±6	53±5	54±5	53±5	54±3	54±6	56±5^	54±5
	Ln rMSSD	4.17±0.58	4.26±0.57	4.17±0.64	4.13±0.48	4.05±0.40	4.03±0.52	3.93±0.47^	4.09±0.61
Normal Sleep	**Mean HR**	53±5	54±6	54±7	54±5	53±7	55±8	56±5#	55±3
	Ln rMSSD	4.28±0.51	4.23±0.67	4.21±0.61	4.12±0.39	4.03±0.48	4.02±0.36	3.96±0.45#	4.10±0.49
Sleep Extension	**Mean HR**	53±5	54±7	54±7	56±5#	56±5	56±4	57±7#	54±5
	Ln rMSSD	4.25±0.52	4.20±0.54	4.18±0.64	4.10±0.49	4.04±0.47	4.08±0.41	4.01±0.58	4.21±0.59

HR, mean heart rate, Ln rMSSD, natural logarithm of the square root of the mean sum of the squared differences between R-R intervals. ^ Different ($P<0.05$) compared with baseline day -1 of condition. # Different compared with baseline day -2 of condition. There were no statistical differences for either mean HR or Ln rMSSD for any day between conditions. Data shows mean ± SD.

Correlations

A strong correlation was found between the percentage change in W:HR from D1 to D4 and the change in TT finishing time for both the SR ($r = -0.67$, $P < 0.05$, Figure 5.2G) and SE ($r = -0.69$, $P < 0.05$, Figure 5.3G) conditions. However, one participant in the SR condition was identified as a potential outlier (i.e., change in TT finishing time from D1 to D4 > 2 × SD of the sample mean[438]), and after removal from the analysis this correlation (i.e., Figure 5.2G) was no longer significant. There were no other significant correlations between changes in respective HR indices and changes in TT finishing time for either the SR or SE conditions.

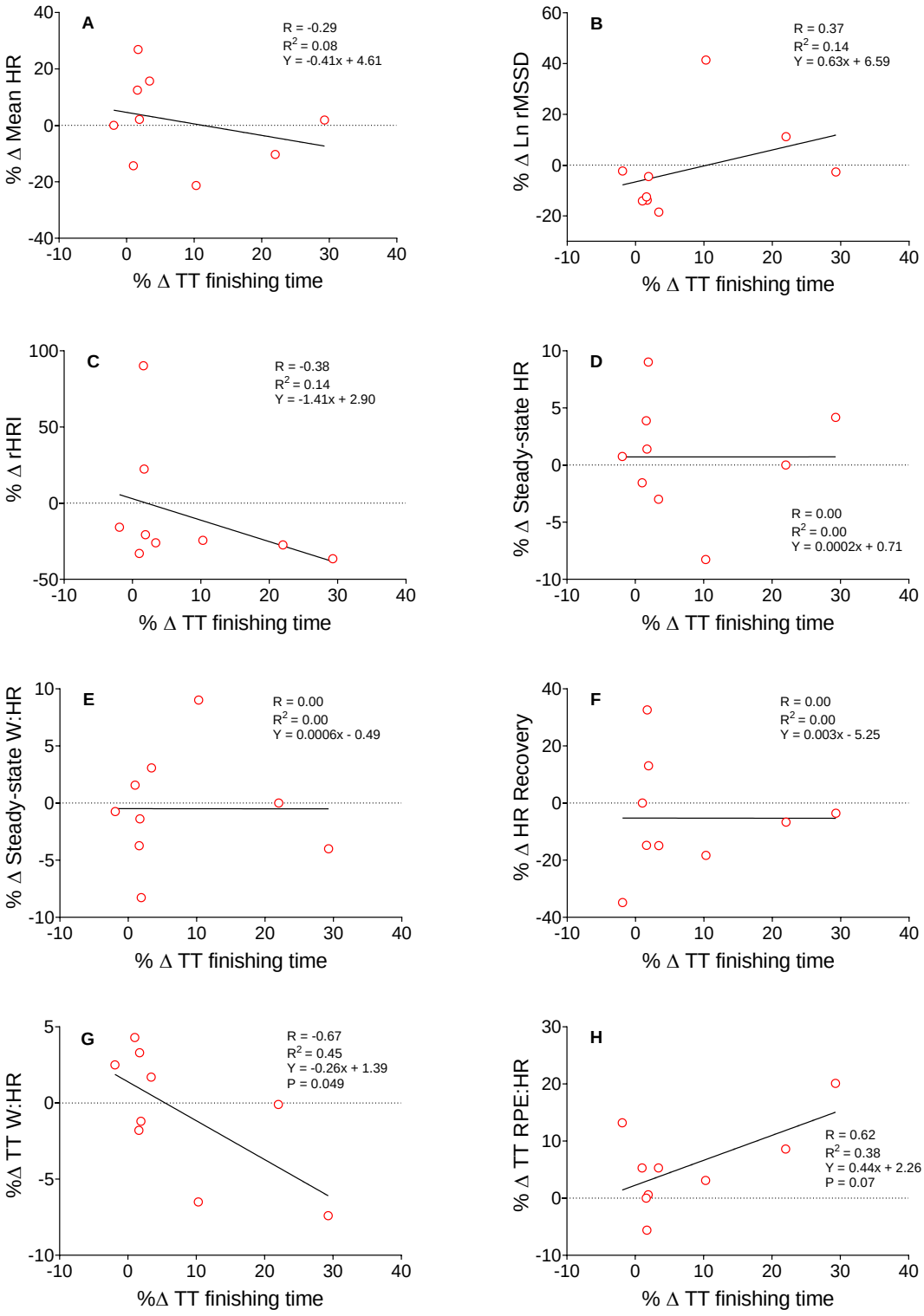

Figure 5.2 Correlations between percentage changes in time-trial finishing time from D1 to D4 of the Sleep Restriction condition, and changes in resting mean HR (A); Ln rMSSD (B); maximal rate of HR increase at onset of submaximal exercise test, rHRI (C); steady-state HR during submaximal exercise test (D); external to internal intensity ratio during submaximal exercise test, steady-state W:HR (E); HR recovery following the submaximal exercise test (F); external to internal intensity ratio for the TT, TT W:HR (G); and subjective to objective intensity ratio for the TT, TT RPE:HR (H). Significant correlation ($P < 0.05$) for panel G only. However, after removal of outlier (change in finishing time > 2 × SD of the sample mean), this correlation was no longer significant.

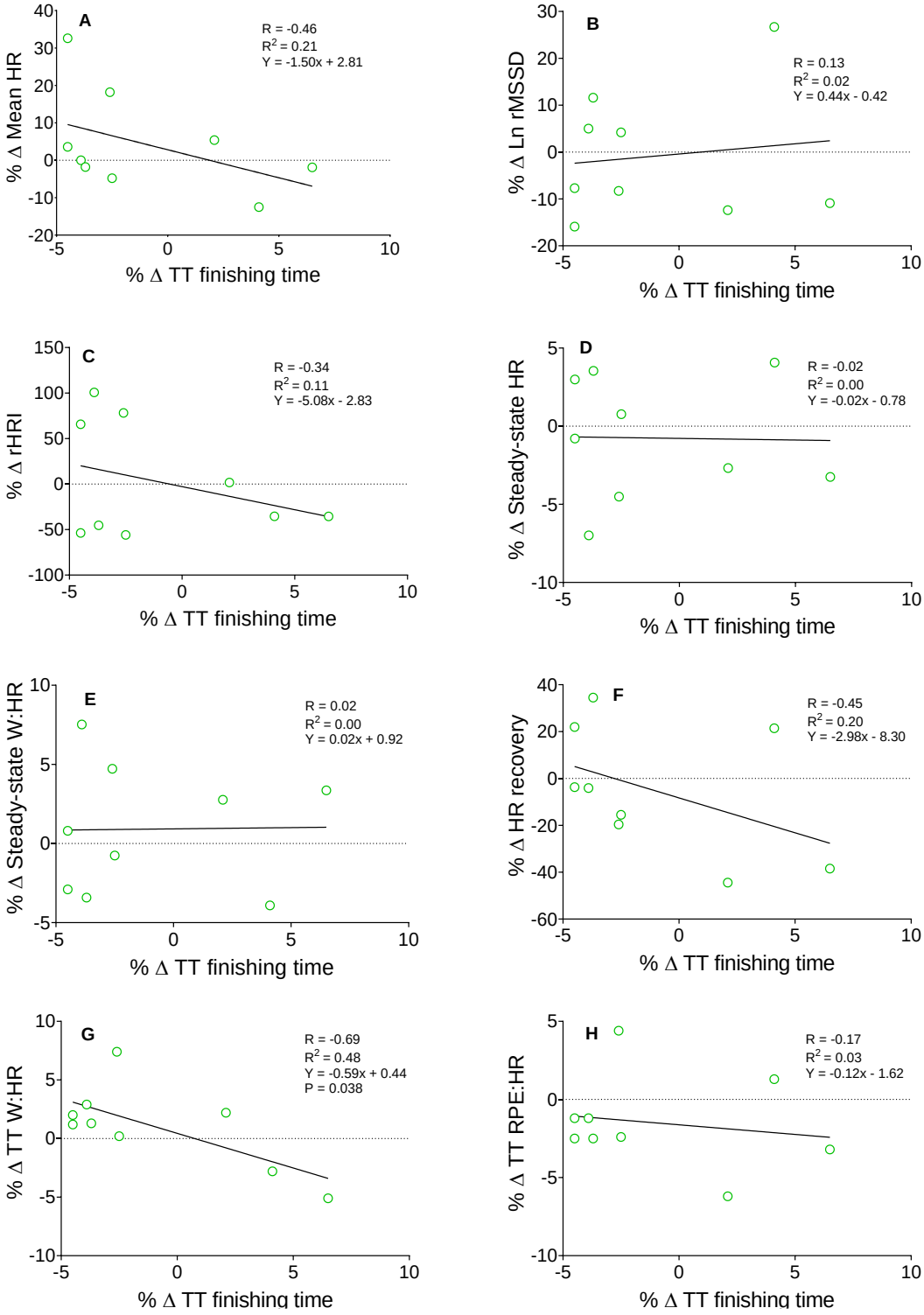

Figure 5.3 Correlations between percentage changes in time-trial finishing time from D1 to D4 of the Sleep Extension condition, and changes in resting mean HR (A); Ln rMSSD (B); maximal rate of HR increase at the onset of submaximal exercise test, rHRI (C); steady-state HR during submaximal exercise test (D); external to internal intensity ratio during submaximal exercise test, steady-state W:HR (E); HR recovery following submaximal exercise test (F); external to internal intensity ratio for the TT, TT W:HR (G); and subjective to objective intensity ratio for the TT, TT RPE:HR (H). Significant correlation ($P < 0.05$) for panel G only.

5.5 Discussion

The aim of this study was to determine whether HR indices commonly used to assess athlete readiness are sensitive to the effects of sleep restriction and / or sleep extension over 1-3 nights on athletes' readiness to perform a ~60-minute TT. A key finding was that RPE:HR for the TT was lower after three nights of sleep extension compared with normal and restricted sleep (Figure 5.1A). This mirrors results from Chapter 4: Study 2a whereby three nights of sleep extension led to better maintenance of endurance performance compared with normal and restricted sleep. Another key finding was that changes in W:HR during the TT from D1 to D4 significantly correlated with changes in TT finishing time in both SR ($r = -0.67$, $P < 0.05$, Figure 5.2G) and SE ($r = -0.69$, $P < 0.05$, Figure 5.3G) conditions.

Intensity ratios

The finding that RPE:HR during the TT was lower after three nights of sleep extension compared with normal and restricted sleep, together with the RPE results presented in Chapter 4: Study 2a, supports the notion that cumulative sleep time affects the perceived exertion of a given exercise intensity. For example, on D4, while overall RPE for the TT was consistently near maximal between conditions (i.e., 9 ± 1), mean HR was higher in the SE condition compared with the SR condition ($P < 0.025$), and *tended* to be higher compared with the NS condition ($P = 0.033$). Thus, it appears sleep extension allowed athletes to work at a higher internal exercise intensity for a given RPE. Previous research has shown RPE:HR increases among professional cyclists across a three-week Grand Tour, and it was recommended that this ratio be used to monitor fatigue during endurance races.[109] Findings indicate cumulative sleep time moderates RPE:HR during exercise and should be considered when using this ratio to analyse performances. For example,

in cyclists, an increase in RPE:HR during a multi-day race may indicate, in-part, sleep loss, and thus prompt support staff to enquire with the athlete about their prior sleep and, if required, intervene to facilitate sleep extension or improve sleeping arrangements (e.g., change from a twin-share to a single occupancy room).

Most athletes recorded a decrease in RPE:HR after three nights of sleep extension (n = 7 / 9 participants) and an increase in RPE:HR after three nights of sleep restriction (n = 7 / 9 participants). Despite this, changes in RPE:HR after three nights of either sleep restriction (Figure 5.2H) or sleep extension (Figure 5.3H) did not significantly correlate with changes in TT finishing time ($P > 0.05$). Although for the SR condition this correlation was approaching statistical significance ($r = 0.62$, $P = 0.07$). This is consistent with findings from Chapter 3: Study 1, whereby total sleep deprivation increased RPE:HR for the TT; however, changes in RPE:HR did not correlate with changes in TT finishing time. Therefore, while an increase in RPE:HR following total sleep deprivation may signal impaired endurance performance, and a decrease in RPE:HR following sleep extension may signal improved performance, the magnitude of changes in RPE:HR cannot be used to accurately predict the *magnitude* of performance changes.

Although TT W:HR was not statistically different for any comparison between or within conditions, individual changes in TT W:HR from D1 to D4 strongly correlated ($r = - 0.7$, $P < 0.05$) with changes in TT finishing time in both SR and SE conditions. However, after the removal of a potential outlier, this correlation was no longer significant in the SR condition. Nonetheless, in the SE condition at least, this correlation suggests performance tended to improve from D1 to D4 in athletes whose TT W:HR increased but tended to decline in athletes whose TT

W:HR decreased. In the SE condition, most participants (n = 6 / 7) who recorded an increase in TT W:HR improved performance from D1 to D4. Given the W:HR relationship is linear below the anaerobic threshold, and then becomes curvilinear at higher intensities,[439] such increases in W:HR may reflect an ability to produce more power (i.e., "push harder") as HR begins to plateau at high exercise intensities. This is consistent with the notion that sleep extension may elevate the effort threshold, and/or pain tolerance, of the endurance athlete.[436] Importantly, changes in W:HR for the *submaximal exercise test* (i.e., steady-state W:HR) did not significantly correlate with changes in TT finishing time (Figure 5.2E and 5.3E). Thus, W:HR was associated with performance only at high exercise intensities. This supports speculation that an ability to sustain a higher power output when HR is plateauing may be a mechanism through which sleep extension improves performance. On the other hand, a decline in performance from D1 to D4 that is associated with a decrease in TT W:HR, suggests an increase in the internal physiological cost of a given external load. For example, in both the SR and SE conditions, the participant that recorded the greatest decrease in TT W:HR, also recorded the greatest increase in TT finishing time. An increase in HR for a given external load occurs with training fatigue.[109 350 351] Thus, the increases in TT W:HR observed in some participants may reflect an accumulation of physiological fatigue across the four days of testing. Such an accumulation of physiological fatigue could potentially mask any proposed psychological benefits of sleep extension. In the SE condition, two of the three participants who recorded a decline in performance from D1 to D4 also recorded a decrease in TT W:HR.

In summary, intensity ratios recorded during high-intensity exercise may be sensitive to the effects of cumulative sleep time on endurance athletes' readiness to

perform. These findings may help practitioners interpret intensity ratios when analysing endurance performances. For example, in cyclists, increases in RPE:HR during tour races may be indicative of fatigue that, to some extent, may be mediated by insufficient sleep. In such cases, practitioners could recommend a period of sleep extension. Likewise, the potential benefit of sleep extension could potentially be monitored by measuring increases in W:HR during competition. Although further research is required in applied sport settings to further clarify the relationship between sleep, intensity ratios, and endurance performance.

Daily heart rate and heart rate variability

There were no statistical differences in resting HR or Ln rMSSD between conditions, and changes in these indices did not correlate with changes in TT finishing time in either the SR or SE conditions. Resting HR indices were also similar between conditions in Chapter 3: Study 1. While some studies have shown resting HR indices are unaffected by sleep restirction,[251 398] others have hound sleep restriction increases resting HR,[399-403] and decreases resting HR variability.[398 405] It is difficult to explain discrepant findings between studies; however, compared with most studies showing perturbations in resting HR indices following sleep restriction,[251 398] the current study adopted a relatively subtle sleep restriction protocol whereby sleep time was reduced by just ~2 hours per night, and examined well-trained endurance athletes. Indeed, parasympathetic predominance is an adaptation to endurance training,[329] which may prevent elevated sympathetic activity following sleep loss.

Compared with baseline days (i.e., days -2 and -1), resting HR was elevated on day +1 in all conditions, and Ln rMSSD was reduced on day +1 in the SR and NS

conditions (Table 5.4). These findings *may* suggest that there was an accumulation of physiological fatigue (i.e., autonomic nervous system imbalance) over the four testing days in both the SR and NS conditions. Previous research found increases in resting HR and reductions HR variability upon waking in overreached athletes.[337 338 341] In the current study, TT performance was consistent over time in both the NS and SE conditions, suggesting an accumulation of fatigue did not affect TT performance. However, given statistical differences in resting HR and Ln rMSSD did not emerge until day +1 (i.e., the morning after the final testing day), perhaps performance may have deteriorated in the NS and SE conditions had testing continued for a fifth consecutive day. Interestingly, the only condition not to record a reduction in Ln rMSSD on day +1 was the SE condition. Thus, while Ln rMSSD was not different between conditions, it is possible that over a longer testing period, such as a three-week cycling tour, extended sleep *may* help prevent the autonomic imbalances associated with accumulated fatigue.[337 338 341] However, further research is required to clarify the long-term relationships between cumulative sleep time, HR variability, and endurance performance.

Submaximal exercise test

Cumulative sleep time did not affect HR indices derived from the submaximal exercise test (Table 5.3). Moreover, changes in these indices did not correlate with changes TT finishing time from D1 to D4 in either the SR or SE conditions. These findings suggest the autonomic mechanisms that increase HR at the onset of exercise, such as the rapid withdrawal of parasympathetic activity and the gradual increase in sympathetic activity,[440] were unaffected by sleep restriction / extension. In support, W:HR during the submaximal exercise test was unaffected by sleep restriction / extension, indicating the linear relationship between power output and

HR at intensities below anaerobic threshold was preserved between conditions.[439] Findings suggest HR indices recorded during (e.g., rHRI, W:HR) or following (e.g., HR recovery) submaximal exercise at 60% $\dot{V}O_{2\,max}$ are insensitive to effects of cumulative sleep time over 1-3 nights on endurance athletes' readiness to perform.

Plasma catecholamines

Pre-TT adrenaline was lower after three nights of sleep restriction compared with normal and extended sleep (Table 5.2). Noradrenaline was unaffected by sleep interventions. Previous research found sleep restriction for a single night *increased* urinary catecholamines[406] while total sleep deprivation has been found to both reduce,[244] and not affect resting plasma catecholamines.[389 396] In Chapter 3: Study 1, catecholamines were not different between conditions following total sleep deprivation. A possible explanation for the current findings is that during REM sleep central nervous system stores of catecholamines are replenished,[50] and thus sleep restriction, which could reduce cumulative REM sleep time,[441] may deplete catecholamine stores.[243] Such depletion of catecholamines could attenuate the sympathetic response to exercise and impair endurance performance.[243] However, given sleep stages were not assessed in the current study, it is impossible to substantiate this explanation. Furthermore, given post-TT catecholamines on D4 were similar between conditions, it appears that the sympathetic response to exercise was unaffected by sleep interventions. An alternative explanation for this finding is a potential resetting of the baroreflex set-point following sleep restriction. Such a re-setting to a higher mean arterial pressure has been demonstrated after total sleep deprivation, and is speculated to occur predominantly via an increase in peripheral vasoconstriction that leads to a reduction in sympathetic activity that may occur without a change in resting HR.[388] In summary, it is unclear why pre-

TT adrenaline was lower after three nights of sleep restriction compared with normal sleep; and thus this finding provides little insight into the mechanisms behind changes in intensity ratios or endurance performance reported in the current study.

Limitations

Limitations concerning the performance outcomes of this study are provided in Chapter 4: Study 2a. However, there are some additional limitations concerning the analysis of HR. First, the study examined popular HR indices used for monitoring athletes in the field; therefore, it is not an exhaustive examination of the cardiac autonomic response to sleep restriction and extension in athletes. Second, the study examined HR responses across a short monitoring period; therefore, although differences were not statistically different in the current study, HR indices may be sensitive to the moderating effects of sleep on athletes' readiness to perform over longer periods (e.g., three-week tour races). Future research should, accordingly, investigate the responsiveness of HR indices over longer periods.

Conclusions

Intensity ratios recorded during high-intensity exercise may be useful for monitoring effects of cumulative sleep time on athletes' readiness to perform endurance exercise. The current findings may help practitioners interpret intensity ratios during high-intensity endurance exercise (e.g., races), and thus help inform athlete management decisions, such as sleep extension recommendations. In contrast, cumulative sleep time over 1-3 nights does not appear to influence HR indices recorded at rest (e.g., resting HR, Ln rMSSD) or during moderate intensity submaximal exercise (e.g., rHRI, steady-state HR, HR recovery, W:HR) Whether the observed relationships persist over longer timeframes requires further research.

Chapter 6: General Discussion

6.1 Overview

This chapter summarises the major findings of this thesis and their contribution to the current state of knowledge concerning the effects of sleep on endurance performance, the sensitivity of HR indices to effects of sleep on athlete readiness to perform, and the potential mechanisms through which sleep affects endurance performance. This chapter also focuses on four emergent themes from the thesis that address (1) the sleep needs of athletes, (2) practical considerations for sleep extension strategies, (3) recommendations for mitigating the negative effects of sleep loss on performance, and (4) methods for monitoring the effects of sleep on athlete readiness and wellbeing. Each of these sections includes recommendations for future research.

6.2 Thesis summary and advances in the state of knowledge

Sleep is considered an important recovery behaviour for athletic success.[7-9] However, to date, few studies have investigated the impact of sleep on the sports-specific performance of athletes.[20 23 24 249] The aim of this thesis was to examine the effects of total sleep time on the performance of endurance athletes. Across two studies, this thesis examined the effects of a single night of total sleep deprivation, and three consecutive nights of both sleep restriction and sleep extension, on endurance cycling performance. A novel aspect of this research was the examination of prolonged (i.e.,~60-minute) self-paced endurance efforts similar to those required by many endurance sports (e.g., road cycling, triathlon). Given the coupling of central and autonomic nervous system activity that is required to initiate and regulate sleep,[374] and evidence that changes in total sleep time can alter daytime HR regulation,[31] a secondary aim of this thesis was to examine whether sleep time affected HR indices often used to monitor athlete readiness to perform. No prior

research had examined the sensitivity of HR indices to any effects of sleep on athlete readiness. Finally, data collected in addressing the key aims of this thesis provided an opportunity to discuss the potential mechanisms through which sleep may influence endurance performance. The major findings of this thesis were:

1. Compared with normal sleep, sleep loss (i.e., one night of total sleep deprivation *and* two consecutive nights of sleep restriction) impaired endurance performance.
2. Compared with normal sleep, three consecutive nights of sleep extension improved endurance performance.
3. Sleep time accumulated over an acute (i.e., 1-3 days) timeframe appears to influence endurance performance by altering the perceived exertion of a given exercise intensity.
4. Intensity ratios (e.g., RPE:HR, W:HR) during *high-intensity* exercise (e.g., time-trials) demonstrated some sensitivity to the effects of sleep time on athlete readiness to perform. There was no evidence that any other HR indices were sensitive to the effects of sleep on athlete readiness.

6.2.1 Sleep loss and endurance performance

The current research was the first to examine the effects of sleep loss on the performance of endurance athletes using a prolonged time-trial, and thus provides novel evidence that both total sleep deprivation and sleep restriction impair the prolonged self-paced performance of endurance athletes. These findings are pertinent for athletes competing in sports requiring such efforts, including road cyclists and triathletes. To date, studies examining the effects of sleep loss on the performance of endurance athletes have delivered equivocal findings, with sleep

loss found to both impair,[24 26 248] and not affect endurance performance.[25 249] Studies that found performance was impaired by sleep loss assessed performance using long protocols (i.e., > 40 minutes) that typically included a preload exercise period followed by an incremental test,[26 248] or using a self-paced time-trial.[24] Therefore, when the current findings are considered alongside previous research, is appears that longer and/or self-paced endurance efforts may be more susceptible to performance impairment following sleep loss.

Study 2a is the first to show a cumulative impact of sleep restriction on the performance of endurance athletes. Compared with normal sleep, finishing time was slower after two nights, but not after one or three night/s, of sleep restriction. Although there was a tendency for performance to be slower after three nights of sleep restriction ($P = 0.09$). Two previous studies examined the impact of three consecutive nights of sleep restriction on endurance performance.[250 253] While these studies found performance was unaffected, participants were not trained endurance athletes, and performances were assessed using relatively short testing protocols (e.g., 400 m swim, uphill treadmill run).[250 253] Only three previous studies have examined the impact of sleep restriction on endurance athletes' performances, and these all examined the effects of a single night of sleep restriction.[24 248 249] Interestingly, impaired performances have only been reported following relatively severe sleep restriction (i.e., ≥ 4 hour reduction in sleep time or time-in-bed).[24 248] In contrast, the sleep restriction protocol used in Study 2a reduced total sleep time by an average of ~2 hours per night, thus illustrating for the first time, that relatively subtle reductions in sleep time, when accumulated over consecutive nights, also impair endurance performance. This supports the hypothesis that the extent of accumulated sleep debt may moderate effects on performance. These findings also

have ecological validity, as athletes more often experience sleep restriction of ≤ 2 hours per night during training and competition periods.[13]

6.2.2 Sleep extension and endurance performance

No prior study has investigated the effects of sleep extension on endurance performance, thus evidence that compared with normal sleep, three consecutive nights of sleep extension improved performance is an important and novel contribution to the field. While two previous studies suggested sleep extension improves sports-specific skill execution and anaerobic performance,[20 23] these studies were confounded by either the lack of a control condition,[20] or the use of self-reported sleep data.[23] In contrast, Study 2a adopted a three-armed crossover design that included a 'control' (i.e., normal sleep) condition, and objectively monitored sleep using validated methods.[301 310] Therefore, it is the most rigorous investigation undertaken to date to determine the effects of sleep extension on the sports-specific performance of trained athletes.

Given Study 2a examined performance across four consecutive days, findings are pertinent for the majority of endurance athletes who routinely train, and for the few who often compete (e.g., road cyclists), on consecutive days. Indeed, given the effects of sleep extension on performance did not manifest until the fourth consecutive day of testing, findings highlight the importance of cumulative sleep time during multi-day endurance events. Sleep extension may also be beneficial for non-endurance athletes who either compete over consecutive days (e.g., cricket players) or have congested competition schedules (e.g., tennis players, soccer players). Moreover, the normal sleep time of athletes in Study 2 was similar to sleep times reported among elite endurance athletes (i.e., ~7 hours per night).[13 205 208 229]

Therefore, recommendations to extend sleep to > 8 hours per night in order to optimise performance are ecologically valid. Finally, the smallest worthwhile change recommended to affect the prospect of cyclists finishing on the podium in a time-trial is 0.6 × coefficient of variation in performance.[442] Given the coefficient of variation in performance between participants' familiarisation time-trial and their first experimental time-trial of Study 2a was 2.8%, the ~ 2-minute improvement in performance after three nights of sleep extension was almost double the smallest worthwhile change recommended for a ~60-minute time-trial. Thus, findings are not only statistically significant but also likely to be competitively relevant for endurance athletes.

6.2.3 Explaining effects of sleep on endurance performance

According to the psychobiological of endurance performance, the perceived effort of exercise is a key determinant of endurance performance.[91] In both the current studies, RPE scores were consistent across all time-trials despite differences in finishing times, and thus mean power output. Therefore, it is proposed that total sleep time accumulated over 1-3 days affects performance by altering the perceived effort of a given exercise intensity (i.e., power output). To the author's knowledge, Study 2 is the first to demonstrate that sleep extension *reduces* RPE for a given exercise intensity. In contrast, several studies support findings from Studies 1 and 2 showing total sleep deprivation and sleep restriction *increase* RPE for a given exercise intensity. Indeed, total sleep deprivation has consistently been shown to elevate RPE for a given exercise intensity without affecting mean HR.[26 242 245 394] Similarly, sleep restriction reduced distance covered during an intermittent running test but did not alter RPE recorded upon test completion.[252]

Increased RPE for a given exercise intensity is associated with mental fatigue,[74 443] and mentally fatigued athletes exercise at lower intensities during self-paced time-trials.[443 444] Therefore, findings suggest total sleep time accumulated over 1-3 nights may influence the level of mental fatigue experienced or tolerated during exercise. Direct assessment of mental fatigue requires the monitoring of brain activity; however, surrogate measures such as subjective changes in mood, or objective changes in cognitive performance, are effective and recommended for monitoring athletes.[72] In contrast, physiological metrics such as HRV have proven ineffective for monitoring mental fatigue.[445] Therefore, evidence that sleep loss *impaired* mood and psychomotor vigilance whereas sleep extension *improved* mood and psychomotor vigilance (Study 1 and 2a), but that neither sleep loss nor sleep extension affected isolated HR indices (Study 1 and 2b), supports the hypothesis that cumulative sleep time affects the level of mental fatigue experienced or tolerated during exercise. An accumulation of adenosine in the brain, particularly in the anterior cingulate cortex, is thought to be the primary cause of mental fatigue.[72] The anterior cingulate cortex is associated with perception of effort, and is highly active during tasks that require response inhibition (e.g., suppression of negative emotions),[72 444 446] which is a key cognitive determinant of endurance performance.[447] Not surprisingly, as a sleep promoting neurotransmitter, adenosine accumulates in the brain during prolonged wakefulness.[81 448 449] Interestingly, sleep extension has actually been speculated to improve brain function by down-regulating adenosine receptors in the brain.[450]

The current findings also support this mental fatigue hypothesis by downplaying the likelihood of other peripheral sources of fatigue. For example, it is proposed that total sleep deprivation exacerbates the gradual increase in plasma volume that

occurs during prolonged wakefulness,[49 394] thus reducing the oxygen carrying capacity of blood.[394] However, findings from Study 1 suggested total sleep deprivation did not affect athletes' plasma volume despite impairing performance. Furthermore, catecholamines play a key role in increasing ventilation and cardiac output during exercise, and catecholamine release is thought to be attenuated following sleep loss.[50] However, in Study 1, catecholamine levels were similar for all time points between conditions. In addition, in Study 2, while adrenaline was lower prior to the TT on D4 of the sleep restriction condition compared with other conditions, catecholamine levels post time-trial on D4 were similar between conditions. Therefore, it is unlikely that differences in catecholamine availability explain the observed differences in performance. Finally, previous studies have also found no evidence for central fatigue following sleep loss, as performance decrements do not coincide with impaired neuromuscular function.[24 26]

6.2.4 Monitoring effects of sleep on athlete readiness using heart rate

No prior study has investigated whether HR indices are sensitive to the effects of sleep on athletes' readiness to perform. Compared with normal sleep, RPE:HR for the TT was higher after total sleep deprivation (Study 1), and lower after three nights of sleep extension (Study 2b). These findings are the first to demonstrate that RPE:HR may be sensitive to effects of sleep on endurance performance. Given other HR indices (i.e., excluding intensity ratios) were unaffected by sleep interventions, it is unlikely that changes in RPE:HR reflect changes in autonomic function, but rather reflect changes in the perceived effort of a given exercise intensity. These findings highlight the importance of including psychometrics such as RPE in athlete monitoring protocols, and suggest cumulative sleep time should

be considered a moderating factor when interpreting RPE:HR, an index previously shown to increase with fatigue in professional cyclists during a three-week tour.[109]

While neither study found differences in W:HR between conditions, a novel finding from Study 2b was that individual changes in TT W:HR from D1 to D4 strongly correlated (r = - 0.7) with changes in TT finishing time in both conditions. Albeit, after removal of an outlier participant, this correlation was no longer significant in the SR condition. These correlations may indicate that cumulative sleep time affects athletes' capacity to tolerate high intensity exercise. For example, it is proposed that increases in W:HR following sleep extension may reflect an ability to produce higher power outputs when HR is plateauing. These findings provide novel evidence that sleep could be considered a potential moderating factor when interpreting W:HR recorded during high-intensity exercise (e.g., endurance races).

Resting HR and Ln rMSSD were unaffected by changes in total sleep time, despite research in non-athletes showing daytime HRV was lower during a period of sleep restriction compared with sleep extension.[31] Changes in total sleep time also had no effect on HR responses during the submaximal recovery test (i.e., rHRI, HR recovery, W:HR, HR kinetic curve parameters), despite previous studies showing acute fatigue slows rHRI,[347] and functional overreaching leads to more rapid HR recovery.[366 368] Collectively, these findings provide important evidence that HR indices, when examined in isolation (i.e., not used in intensity ratios), are insensitive to the effects of sleep time on athletes' readiness to perform endurance exercise - at least over an acute timeframe (i.e., 1-3 nights).

6.2.5 Limitations of the research

Specific limitations pertinent to each study are detailed in their respective chapters. However, some additional overarching limitations of the research warrant mention. First, although the use of actigraphy for sleep monitoring is valid, it is less accurate than PSG - the 'gold-standard' for sleep monitoring. The method used to monitor sleep in the current studies has demonstrated 87% agreement (i.e., percentage of sleep and wake epochs correctly detected) with PSG in cyclists. However, sleep time is typically underestimated by ~ 50 minutes per night using this method.[301] Nonetheless, total sleep time, the independent variable of the research, clearly differed between conditions on intervention nights as evidenced by the clear statistical differences observed. Second, while performance testing was conducted in a laboratory to control potential confounders, when extrapolating 'real-world' implications from the findings it is important to consider that several factors (e.g., environmental conditions, race circumstances, caffeine use etc.) could moderate the impact of sleep on performance. Third, it is widely understood that sleep is good for human function and performance.[4] Therefore, given the inherent difficulty of blinding participants to changes in their sleep, expectations that sleep extension may improve (i.e., placebo effect), and sleep loss impair (i.e., nocebo effect), performance, may have influenced athletes' efforts. To reduce this possibility, participants were reminded to complete each time-trial as fast as possible and were encouraged not to anticipate effects of interventions. Participants were also blinded to any information about their performance for the duration of the studies. In future, research could potentially blind athletes to subtle changes in sleep time by removing access to clocks and by controlling bed / wake times in laboratory settings. Fourth, although participants completed a diet diary and were required to

replicate dietary intake between conditions, it cannot be guaranteed that this was done, and that performances were not influenced by changes in energy availability. However, diaries for each participant were manually checked for any discrepancies at the conclusion of testing, and no such discrepancies were found. In addition, participants were frequently reminded of the dietary requirements of the research, and in personal communications participants indicated they were adhering to these requirements. Fifth, the current research examined the effects of sleep on endurance performances of athletes in the morning (i.e., between 0600-0900), and thus it is impossible to know whether observed effects on performance would persist if testing was conducted at a different time of day. Finally, Study 2 had a relatively small sample size of nine participants, which reflected both the stringent inclusion criteria and the extremely demanding nature of the study. Although a small sample size limits the statistical power of analyses, similar sample sizes have been used in previous research examining effects of sleep on endurance performance.[241 242 245 252] Moreover, the treatment effect for sustained sleep restriction and extension was large enough to be detected as statistically significant in nine participants.

6.3 Implications and future directions

6.3.1 How much sleep do athletes need to optimise performance?

Study 2a found endurance performance was better maintained when athletes slept > 8 hours per night. Previous studies also found increasing total sleep time to > 8 hours per night improved sports-specific performances[20 23]. In addition, an examination of national age-group netballers during a tournament found players from the winning team typically slept > 8 hours per night, more than the ~7 hours per night recorded by lower ranked teams[283]. Players also indicated that 8 hours of sleep per night was needed to feel well rested[283]. Therefore, current findings

contribute to an emerging consensus that athletes need > 8 hours of sleep per night to optimise performance. Given sleep efficiency generally ranges between 80-90%[13], athletes should aim to spend 9-10 hours in bed per night to achieve > 8 hours sleep.

Although general sleep recommendations are important, sleep needs will vary between athletes, and ideally recommendations should consider athlete-specific factors.[451] For example, in Study 2a, the benefits of sleep extension, compared with normal sleep, did not manifest until the fourth TT, and so sleep extension seems to protect against an accumulation of fatigue over consecutive days. Therefore, maximising sleep time may be particularly important for athletes competing across consecutive days (e.g., cyclists, cricketers, racing drivers), or undertaking congested competition schedules (e.g., tennis players, soccer players, Olympic athletes). Such athletes may easily accumulate fatigue and/or stress due to limited recovery between competition days. In addition, findings from both studies suggest sleep may affect the mental determinants of endurance performance. Thus, athletes competing in sports with particularly high or sustained mental demands may be especially reliant on sleep for optimal performance. For example, sleep extension, which improved the mean reaction times of athletes in Study 2a, may be particularly beneficial for athletes reliant on reaction time (e.g., goalkeepers, baseball batters, racing drivers etc.). Conversely, sleep restriction has been shown to slow the reaction times and reduce the attentional capacity of handball goalkeepers.[175] Additional factors that may influence an athlete's sleep need include their genotype,[261 452] age,[202] and sex.[453] For example, sleep need decreases across the lifespan, such that teenagers typically need more sleep than adults.[202] While women typically sleep more than men, in-part due to different sex hormone profiles.[453]

Given the myriad of athlete-specific factors that likely influence sleep need, identifying an athlete's individual sleep need, or 'optimal sleep time', should be a priority for practitioners. Optimal sleep time is described as the total sleep per night required to eliminate homeostatic sleep debt.[258] Knowledge of an athlete's optimal sleep time could inform an individualised approach to managing sleep, similar to approaches used for training prescription. An athlete's optimal sleep time may change across a season depending on training / competition demands, making it difficult to know precisely how much sleep an athlete needs at any given time. For example, in youth soccer players, there is evidence that increases in training load necessitate increases in total sleep time during recovery.[454] Nonetheless, tools that assess 'daytime sleepiness' may be useful for determining whether a residual sleep debt exists.[455-457] Subjective tools (Stanford Sleepiness Scale, Karolinska Sleepiness Scale) for monitoring sleepiness are practical in the field, as they are inexpensive and easily administered.[456 457] Wearable devices could also be used to supplement subjective sleepiness assessments.[315 316] For example, increases in total sleep time that occur concomitantly with increases in sleep latency and decreases in sleep efficiency generally signal the recovery of sleep debt.[458]

The current research focused on the acute effects of sleep on athletic performance; therefore, future research should examine longer-term effects of sleep on athletes. It is possible that even very mild sleep restriction, if experienced for prolonged periods of time, could affect athletes' ability to train and compete. Conversely, minor increases in habitual sleep time may lead to long-term benefits for athletes (e.g., improved overall health, greater training adaptations). Research could explore these questions by examining relationships between total sleep time and athlete outcomes (e.g., performance, health) over the course of competitive seasons or even

athletes' careers. Research should focus on athletes that compete over consecutive days, or in sports with high or sustained mental demands, as these athletes may rely more heavily on sleep for optimal performance. Potential differences in the sleep needs of athletes that may be moderated by sex, age, and even genotype should also be investigated. Finally, more research examining sleep using PSG, or portable electroencephalography,[459] is required to gain a more accurate and detailed understanding of athletes' sleep and its influence on performance.

6.3.2 Extending sleep to optimise athletic performance

The sleep extension achieved in Study 2 indicates athletes were carrying a sleep debt due to inadequate habitual sleep, or at least inadequate sleep immediately prior to the sleep extension intervention.[13] Sleep extension is defined as an increase in habitual sleep time, that is prescribed under the assumption that a residual sleep debt exists.[20 436] If habitual sleep time matches optimal sleep time, a sleep debt will not exist and sleep extension cannot occur. While athletes should aim to ensure habitual sleep time matches optimal sleep time, this is not always possible.[13] A recent study found professional soccer players slept < 6.5 hours per night after training days, and < 5 hours per night after evening matches.[287] As such, players likely had a chronic sleep debt that was periodically exacerbated by evening competition.[287] Given athletes often experience sleep debt, it is important that practitioners can effectively facilitate sleep extension.

Understanding the dynamics of recovery sleep in response to a sleep debt will help inform sleep extension strategies. In Study 2, extending time-in-bed to ~9.5 hours per night increased total sleep time from ~7 to ~8.4 hours per night. However, when healthy adults extended time-in-bed to *12 hours per night* for nine nights, total sleep

time increased from ~7 to ≥ 9.5 hours per night for three consecutive nights, before it reduced to ~ 8.4 hours by the fourth night and remained steady thereafter, illustrating participants' optimal sleep time was ~ 8.4 hours per night.[258] This suggests the sleep extension protocol used in Study 2 was probably not sufficient to maximise total sleep time in athletes habitually sleeping ~ 7 hours per night. It also demonstrates that when recovering a sleep debt, the initial 'rebound' in sleep time, which reflects the severity of the residual sleep debt, is greater than the long-term optimal sleep time.[258] Practitioners should understand these dynamics when prescribing sleep extension, and facilitate large increases in sleep opportunity for at least 2-3 days after a sleep debt is incurred or identified (e.g., after evening competition). This will ensure sleep debt is recovered and optimal sleep time established / re-established as quickly as possible. Sleep opportunity can be increased by either extending nightly time-in-bed and/or undertaking daytime naps.

Study 2 adopted an individualised approach to extending nightly sleep such that each athlete had their habitual time-in-bed extended by 30%, and bed / get-up times were tailored to individual chronotype to ensure athletes were in bed when sleep propensity was high. In contrast, previous research achieved sleep extension by prescribing absolute time-in-bed targets for all athletes (e.g., ≥ 10 hours in bed per night) for periods ranging from one week up to 5-7 weeks.[20 23] This approach does not account for individual differences in sleep need, and may lead to excessive or unrealistic time-in-bed targets (e.g., for genetically short sleepers). Excessive time-in-bed may paradoxically increase sleep disruption by instilling an unhealthy preoccupation with sleep, and is a key behavioural mechanism known to perpetuate insomnia.[262] Such targets could also interfere with other aspects of athlete wellbeing, such as social and family life. To individualise sleep recommendations

among athletes, practitioners should assess individual chronotype, either by using valid questionnaires, or by measuring 'dim light melatonin onset'.[460] Understanding an athlete's chronotype will not only help inform bed / get-up time recommendations, it could also help practitioners ensure training start times do not encroach on an athlete's habitual sleep phase. Indeed, early morning training (e.g., start time < 7 am) has been shown to reduce prior total sleep time in athletes.[228 456] Despite this, a recent study of professional soccer players found that although players went to bed after 2 am following evening matches, they typically woke before 9 am in order to attend 10 am recovery sessions.[287] Not surprisingly, players obtained < 5 hours sleep following evening matches.[287] In summary, while large increases in sleep opportunity may be required to recover a sleep debt and establish optimal sleep time, practitioners should try to individualise sleep extension strategies by recommending time-in-bed changes relative to an athlete's habitual sleep routine, and by using individual chronotype to inform both sleep recommendations and training schedules.

It is important to note that sleep extension is also facilitated by undertaking daytime naps and adopting healthy sleep hygiene. Study 2 prohibited daytime napping in order to control sleep via the manipulation of nightly time-in-bed. However, in Chapter 2, napping was highlighted as an effective strategy for supplementing nightly sleep (Section 2.3.4.1) that can benefit subsequent athletic performance (section 2.3.4.3). Despite this, nap frequency (i.e., percentage of days on which a nap is taken) is typically ≤ 20% among elite athletes.[208 284 285] Napping is particularly useful when nightly sleep is disturbed; however, naps should generally be < 2 hours in duration, and should be avoided in the late afternoon to avoid disrupting nightly sleep.[16] In Study 2, participants were not provided with sleep

hygiene recommendations. However, Chapter 2 highlighted the efficacy of sleep hygiene education in facilitating sleep extension in athletes (section 2.3.4.1). There is little data on the sleep hygiene of athletes; however, a survey of national and international level youth athletes found sleep hygiene could be improved primarily by reducing the irregularity of bedtimes and the use of evening artificial light.[285] Interestingly, an *individualised* approach to sleep hygiene recommendations was effective for improving sleep outcomes among international cricketers.[17]

Sleep extension is necessary to recover a residual sleep debt; however, more research is required to determine optimal sleep extension protocols for athletes. Future research should examine how best to use increases in sleep opportunity and effective sleep hygiene to rapidly resolve sleep debts incurred during training and competition. Indeed, research could focus on developing optimal sleep extension protocols for scenarios commonly experienced by athletes (e.g., sleeping < 5 hours following evening competition). Future research should also focus on examining individualised sleep extension protocols, including the use of athlete-specific interventions that may promote sleep, such as psychotherapies for nervous or hyperaroused athletes,[461] or cryotherapy for athletes prone to muscle soreness.[275]

6.3.3 Mitigating effects of sleep loss on athletic performance

Although it is preferable to address an athlete's sleep debt by facilitating sleep extension, in some situations this may not be possible before training or competition resumes (e.g., competing over consecutive days). In such situations, strategies that mitigate the effects of sleep loss on performance may be useful.

Compared with normal sleep, TT finishing time in Study 2 was slower after two nights, but not after one night, of sleep restriction. This indicates that athletes can

tolerate a level of sleep loss before *endurance performance* is affected. Indeed, Study 1 suggested athletes with higher anaerobic thresholds may be more resistant to the negative effects of sleep loss, as there was a strong negative correlation between anaerobic threshold and change in TT finishing time. Previous research found minor sleep loss (i.e., < 1 hour) did not affect the performance of Dutch athletes, and in agreement with the current findings, authors suggested extreme sleep loss or accumulated sleep debt is likely more detrimental to performance.[462] Therefore, it is important athletes do not catastrophize after one 'bad night' of sleep, as doing so may be more harmful to performance than any sleep loss itself (i.e., nocebo effect). Practitioners should emphasise the importance of cumulative sleep time, perhaps by examining sleep trends over weekly or monthly timeframes, rather than on a nightly basis.

The current findings show impaired endurance performances following both total sleep deprivation and sleep restriction were associated with an increase in perceived effort for a given exercise intensity, impaired psychomotor vigilance, and perturbed mood states. These findings are consistent with the hypothesis that sleep loss may increase mental fatigue during exercise. Therefore, increasing athletes' tolerance of mental fatigue may help mitigate the negative effects of sleep loss on athletic performance. In fact, evidence from Study 1 suggesting athletes with higher anaerobic thresholds may be more resistant to the effects of sleep loss may reflect an adaptation to years of strenuous training whereby cerebral fuel becomes better conserved during exercise, thus minimising the accumulation of extracellular adenosine.[78 427] Compared with recreational cyclists, professional cyclists have been found to be more resistant to increases in perceived effort, and to demonstrate superior inhibitory control, when mentally fatigued.[427] Greater tolerance of mental

fatigue could also be fostered by educating athletes on the use of psychological strategies, such as goal-setting, imagery, and self-talk.[33] For example, motivational self-talk (e.g., "I'm feeling good", "push through this") has been shown to reduce perceived effort for a given exercise intensity and improve endurance performance.[90] Furthermore, 'brain endurance training', a method of training that combines mentally fatiguing cognitive tasks with standard endurance training, increases time-to-exhaustion and reduces RPE when compared with standard endurance training alone.[463] Given these findings, future research could explore whether specific psychological skills or cognitive training can mitigate the negative effects of sleep loss on performance.

Finally, as an adenosine antagonist, caffeine ingestion mitigates athletic performance decrements caused by both sleep loss,[464] and mental fatigue.[465] Caffeine ingestion has been found to reduce RPE for a given exercise intensity and improve endurance performance in mentally fatigued males.[465] However, it should be noted that chronic use of caffeine develops a tolerance that attenuates performance benefits,[466] and when taken prior to afternoon or evening competition, caffeine can disturb nightly sleep.[224] [467] Therefore, when not essential for performance, caffeine should be used sparingly by athletes.

6.3.4 Monitoring the effects of sleep on athlete readiness

Evidence that RPE:HR and W:HR recorded during high-intensity exercise were, to some extent, sensitive to the effects of total sleep time on athletes' readiness to performance suggests prior sleep time should be considered when interpreting these indices. While intensity ratios during high-intensity exercise are not necessarily useful for informing decisions immediately prior to training or competition, as

high-intensity exercise at such times will exacerbate existing fatigue, they *are* useful for analysing (1) trends in athlete readiness over time, or (2) for informing athlete management decisions for athletes competing over consecutive days. For example, when analysing a trending increase in RPE:HR that occurs over time (e.g., across a pre-season training period), practitioners should consider inadequate sleep as a potential cause, and thus provide sleep hygiene education and/or facilitate sleep extension in order to hopefully improve readiness for subsequent training and/or competition. Such interventions may help optimise both training adaptation and competition performance. Similarly, practitioners supporting cyclists in multi-day/stage tour races could analyse RPE:HR and W:HR during each stage and promote sleep among cyclists whose ratios show signs of fatigue. For example, practitioners may offer a single occupancy hotel room as opposed to a normal twin-share for cyclists showing increases in RPE:HR. However, it should be noted, more research is needed to examine whether sleep outcomes are associated with intensity ratios in 'real-world' training and competition settings.

The current findings also show sleep loss increased POMS scores for 'total mood disturbance', 'confusion' and 'fatigue', and reduced scores for 'vigour'. Whereas sleep extension increased scores for 'vigour'. In agreement, sleep extension for 5-7 weeks improved POMS subscale scores for 'vigour' and 'fatigue' among college basketballers.[20] Similarly, one night of sleep restriction decreased SRSS scores for 'overall recovery' and increased scores for 'negative emotional state' among elite junior rowers.[227] Therefore, practitioners should also consider the impact of sleep when interpreting subjective mood and wellness tools, such as the POMS. Previous research found subjective self-reported measures, including the POMS, were more effective than objective tools such as HR for monitoring athlete wellbeing.[468]

Indeed, the greater effectiveness of subjective measures may reflect, in-part, their sensitivity to sleep. Therefore, while subjective wellbeing assessments are susceptible to bias when athletes want to be seen in a positive light by practitioners, or when they want to appear fatigued so that training demands are reduced,[119] they remain a critical component of athlete monitoring protocols.

6.4 Conclusions

This thesis examined the effects of sleep on endurance cycling performance and found that total sleep time over accumulated over 1-3 nights affects endurance performance, with sleep loss impairing performance and sleep extension improving performance, compared with normal habitual sleep. This thesis also found that intensity ratios recorded during high-intensity exercise (i.e., time-trials) were somewhat sensitive to the effects of sleep on endurance athletes' readiness to perform. Therefore, practitioners should consider the impact of prior sleep when interpreting intensity ratios. Sleep appears to influence performance by altering the perceived effort of a given exercise intensity. The current findings suggest athletes need to sleep > 8 hours per night to optimise performance. However, sleep needs will vary between individual athletes, and practitioners should aim to understand the individual sleep needs of athletes, and provide individualised recommendations, when prescribing sleep extension.

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Appendices

Appendix A

Review

Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis

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ABSTRACT

Objectives To characterise the sleep of elite athletes and to identify factors associated with training and competition that negatively affect sleep.

Design Prognosis systematic review.

Data sources Three databases (PubMed, SCOPUS and SPORTDiscus) were searched from inception to 26 February 2018.

Eligibility criteria for selecting studies Included studies objectively reported total sleep time (TST) and/or sleep efficiency (SE) in elite athletes. Studies were required to be observational or to include an observational trial.

Results Fifty-four studies were included. During training, many studies reported athletes were unable to achieve TST (n=23/41) and/or SE (n=16/37) recommendations. On the night of competition, most studies reported athletes were unable to achieve TST (n=14/18) and/or SE (n=10/16) recommendations. TST was shorter (60 min) the night of competition compared with previous nights. SE was lower (1%) the night of competition compared with the previous night. TST was shorter the night of night competition (start ≥18:00; 80 min) and day competition (20 min) compared with the previous night. SE was lower (3%–4%) the night of night competition but unchanged the night of day competition compared with previous nights. Early morning training (start <07:00), increases in training load (>25%), late night/early morning travel departure times, eastward air travel and altitude ascent impaired sleep.

Conclusion Athletes were often unable to achieve sleep recommendations during training or competition periods. Sleep was impaired the night of competition compared with previous nights. Early morning training, increases in training load, travel departure times, jet lag and altitude can impair athletes' sleep.

PROSPERO registration number CRD42017074367.

INTRODUCTION

Sleep is associated with many physiological processes that may facilitate recovery from, and adaptation to, athletic training and competition.[1] During sleep, anabolic metabolism is upregulated,[2] procedural memories are consolidated[3] and immune responses are augmented.[4] Not surprisingly, many athletes identify sleep as the most important recovery behaviour for athletic success.[5] Extending sleep may be beneficial for sports-specific skill execution.[6] Nightly sleep duration has been positively correlated with team netball performance[7] and associated with injury risk in adolescent athletes.[8]

It is important to understand how the demands of training and competition affect sleep, and this has become a priority for coaches and scientists.[9] It is recommended that adults sleep at least 7 hours per night and achieve a sleep efficiency (ie, time asleep as a percentage of time in bed) of at least 85% on a regular basis to promote health.[10 11] However, there is a high prevalence of insomnia symptoms among athletes.[9] Much of the research examining athletes' sleep has been derived from subjective tools (eg, diaries).[12] This is a problem because subjective tools, while useful for monitoring large cohorts, typically correlate poorly with objective data.[13]

The 'gold standard' for sleep monitoring is polysomnography (PSG), which uses surface electrodes to monitor physiological parameters such as brain, muscle, heart and respiratory activity.[14] PSG is particularly useful for investigating sleep pathologies, including sleep-disordered breathing[15] and sleep disturbances caused by concussion.[16] However, PSG is expensive and requires specialised laboratory equipment, making it impractical for monitoring athletes in the field.[17] Actigraphy, on the other hand, uses accelerometers embedded in portable devices to record movements that can be analysed using algorithms that estimate sleep quality and quantity.[18] Actigraphy is inexpensive and can be administered with minimal impact on habitual sleep or training routines. As such, actigraphy is the preferred method for objectively monitoring the sleep of athletes.[17]

In recent years, growing interest in understanding the sleep of athletes has seen an increase in published studies, such that objective data are now readily available. Therefore, in this systematic review we aimed to characterise the sleep of athletes in the context of current sleep recommendations, and to examine whether there were training and competition factors that negatively affected sleep.

METHODS

This review conformed to the Meta-Analysis of Observational Studies in Epidemiology (MOOSE) guidelines for Meta-Analyses and Systematic Reviews of Observational Studies,[19] and has adopted the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA),[20] with PROSPERO trial registration number CRD42017074367.

Search strategy

Three electronic databases (PubMed, SCOPUS and SPORTDiscus) were searched from inception to 26 February 2018 using combinations of the following keywords: sleep, elite, competitive, athlete/s, sport and player/s (online supplementary file 1). Reference lists of relevant studies were also cross-checked. Figure 1 illustrates the search process.

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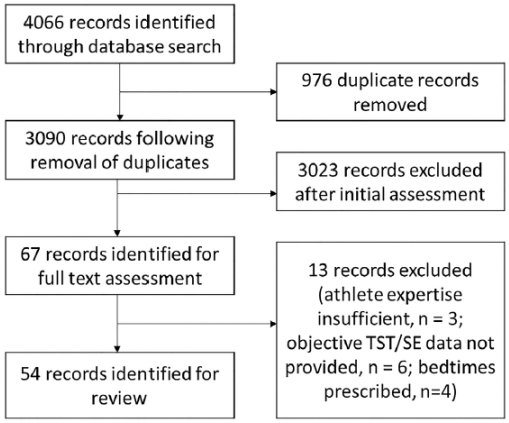

Figure 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow chart for the selection of included studies. SE, sleep efficiency; TST, total sleep time.

Table 1 Sleep-related definitions

Term	Definition
Sleep phase	The sleep period from the time of day at sleep onset to the time of day on waking. It is dependent on the circadian process, and can be delayed or advanced by environmental or psychosocial factors.
Sleep pattern	The consistency or lack of consistency in the sleep phase of an individual over consecutive days or weeks.
Bedtime	The time of day at which an individual goes to bed and attempts to sleep.
Sleep onset	The time of day following bedtime at which sleep commences.
Wake time	The time of day at which the final awakening from a sleep period occurs.
Time in bed (TIB)	Total time spent in bed from bedtime to wake time.
Sleep period (SP)	Total time from sleep onset to wake time.
Wake after sleep onset (WASO)	Total time spent awake after the initial sleep onset and before final awakening (ie, WASO=SP–TST).
Total sleep time (TST)	Total time spent sleeping (ie, TST=SP–WASO).
Sleep efficiency	TST expressed as a percentage of TIB (ie, TST/TIB × 100). A measure of sleep quality.
Sleep onset latency	Time elapsed between bedtime and sleep onset.

Eligibility criteria

Included studies met the following criteria: (1) participants could be identified as elite athletes (see 'Athlete expertise appraisal' section); (2) the study reported objective (eg, PSG, actigraphy) sleep outcomes; (3) the mean age of participants was ≥15 years; (4) the study was observational or had an observational (ie, control) trial whereby sleep/wake behaviour was not manipulated; (5) the study investigated able-bodied athletes; and (6) the study was published in English as a full-text article in a peer-reviewed journal.

Study selection and data extraction

After removal of duplicates, the title and abstract of each retrieved record were screened by one reviewer (SSHR). The full text of each screened record was then examined independently by two reviewers (SSHR, SAW) to determine whether the inclusion criteria had been satisfied. Discrepancies were discussed until a consensus was reached. Data from eligible studies were extracted by one reviewer (SSHR) into a spreadsheet designed to record information on the study design and sample population. Data were categorised according to the explanatory variable evident in the study design. The mean and SD for sleep outcomes of interest (see 'Sleep definitions and outcomes of interest' section) were recorded. Where required, SD was calculated from reported CIs. Where data were reported graphically, Plot Digitizer (V.2.6.8) was used to extract numerical values.

Athlete expertise appraisal

Two reviewers (SSHR, SAW) independently assessed athlete expertise using a modified version of the Model for Classifying the Validity of Expert Samples in Sport Psychology (online supplementary file 2).[21] The model evaluated three subscale items: the level or performance reached by the athlete, the competitiveness of the sport in the country and the competitiveness of the sport in the world. The modified version was necessary due to limited reporting of athlete experience or success in the literature. Scores were awarded according to the criteria for each item, and a modified algorithm was used to calculate an overall score.[9] Included studies were required to recruit athletes identifiable as either 'competitive-elite' (ie, competing at the highest level in their sport) or 'semi-elite' (ie, competing below the highest level in their sport).[9 21]

Evidence quality appraisal

Two reviewers (SSHR, SAW) independently assessed the methodological quality of each study using an adapted version of the Newcastle-Ottawa Scale (NOS) (online supplementary file 3).[22] The NOS was adapted for use with cross-sectional studies as previously demonstrated.[22] The NOS assessed participant selection, control of confounders and outcome reporting using eight subscale items. The criteria for each item were established by two reviewers (SSHR, SAW), so that the evidence quality of each study could be assessed in the context of the aims of the review. Each subscale item was awarded a 'score' according to the extent to which the criteria had been satisfied (eg, 0=not satisfied, 1=satisfied, 2=satisfied using a validated tool or established model (two items only)). Discrepancies were discussed until a consensus was reached. The sum of subscale item scores was used to provide an overall assessment of evidence quality for each study (ie, >7=high quality, 5–7=moderate quality, <5=low quality). No risk of bias was undertaken at the outcome level because all included studies objectively measured sleep outcomes.

Sleep definitions and outcomes of interest

Outcomes examined in this review were total sleep time (TST) and sleep efficiency (SE). These were selected because they provide quantitative measures of sleep quantity (TST) and quality (SE), and are frequently reported in the literature. Table 1 shows the definitions of key sleep-related terms used in this review.

Meta-analysis

For studies that reported sleep outcomes on multiple nights surrounding competition, random-effects meta-analyses were performed to compute the standardised mean difference (SMD) between studies. Studies deemed of moderate quality or higher following evidence quality appraisal were eligible for the meta-analysis. Analyses were performed for several time-point

comparisons: A (−2 vs −1), B (−2 vs 0), C (−2 vs +1), D (−2 vs +2) and E (−1 vs 0), whereby −2=two nights before, −1=the night before, 0=the night of, +1= the night after and +2= two nights after competition day/night. Two nights before competition was selected to represent a 'control' night for analyses because it is most removed from potential sleep influencing factors such as precompetition anxiety and post competition fatigue, whilst still being reported frequently in the literature. The additional comparison E was included due to the plethora of data in the literature for nights -1 and 0. Prespecified subgroup analyses and meta-regression were undertaken to assess if 'sleeping environment' (home vs away) and 'competition start time' (day (start time <18:00) vs night (start time ≥18:00)) affected sleep. The size of significant effects was estimated by back transforming pooled SMDs using representative studies,[23–25] as recommended by previously established guidelines.[26] The I^2 statistic was used to assess statistical heterogeneity, whereby I^2<25% was considered low, I^2=25%–50% was considered moderate and I^2>50% was considered high heterogeneity.[27] We used funnel plots and Egger's regression test (non-significant asymmetry indicated no bias) to assess for publication bias.[28] Statistical analyses were performed using Comprehensive Meta-Analysis (V.3.0, Biostat, Englewood, USA). An alpha level of p<0.05 was used to determine statistical significance.

RESULTS

The database search yielded 4066 records. After removal of duplicates and initial screening, the full text of 67 records was examined. Thirteen were excluded for not meeting the inclusion criteria, leaving 54 studies for review (figure 1).

Study characteristics and athlete expertise

The included studies were published between 1997 and 2018,[7 13 23–25 29–77] with 81% (n=44 studies) published since 2014.[7 13 23–25 29–34 36–39 41–50 52 53 55–57 60–63 65–70 73 74 76 77] Women were recruited by 37% of studies (n=20 studies),[7 13 32 35 40–44 50 51 55 56 61–63 65 67 71 74] but only 19% (n=10 studies) recruited women exclusively.[7 40 41 55 56 61 65 67 71 74] The mean age of athletes ranged from 16 to 28 years; however, one case study recruited a 31-year-old athlete.[53] Athletes were recruited from 21 sports including soccer (n=14),[25 36–38 42 46 47 50 52 53 60 64 72 73] Australian rules football (n=11),[23 33 39 45 50 52 57–59 63 70] cycling (n=6),[42 48–50 54 63] rugby league (n=6),[29–31 39 68 69] swimming (n=5),[32 50 62 63 71] rugby union (n=5),[24 34 50 52 66] netball (n=6),[7 41 55 56 61 74] basketball (n=3),[50 63 67] rowing (n=3),[43 44 51] mountain biking (n=3),[42 50 63] triathlon (n=3),[42 50 63] American football,[75 76] race walking (n=2),[50 63] ice hockey,[77] diving (n=1),[51] running (n=1),[40] volleyball (n=1),[42] judo (n=1),[13] handball (n=1),[42] canoeing (n=1),[51] speed skating (n=1),[51] synchronised swimming (n=1)[65] and ballet (n=1).[35] 'Competitive-elite' athletes were recruited by 65% (n=35) of studies,[23–25 29–34 36 38 39 42 45 48–53 55–59 63 65–70 74 75 77] and 37% (n=20) recruited 'semi-elite' athletes (online supplementary file 2).[7 13 29 35 37 40 41 43 44 46 47 54 60–62 64 71–73 76]

Evidence quality and data collection methods

The average evidence quality of included studies was moderate (NOS mean±SD=7±1), with 28% (n=15) considered high quality,[7 31 33 34 39 42 50–52 57 63 70] 70% (n=38) considered moderate quality,[13 23–25 29 30 32 35–38 40 41 43–49 53–56 58–62 64–67 69 71–74] and 2% (n=1) considered low quality (the results for each subscale item are provided in online supplementary file 3).[68] Studies used actigraphy (n=46),[7 13 23–25 29–38 40–53 55–63 65–70 72 74] PSG (n=6),[39 54 64 71 75 77] headband electroencephalography (n=1)[73] or photoplethysmography (n=1)[76] to monitor sleep. Of those that used actigraphy, 9% (n=4) used a high sleep–wake threshold (>80 activity counts),[29 30 42 72] 33% (n=15) used a medium threshold (>40 activity counts),[7 23 36 41 45 47–50 52 60 62 63 70 78] 18% (n=9) used the 'Readiband' device-specific algorithm,[13 25 31 33 34 38 55 56 74] 7% (n=3) used the 'SenseWear' device-specific algorithm,[24 43 44] 2% (n=1) used the Cole-Kripke algorithm,[67] 2% (n=1) used the Sadeh algorithm[37] and 2% (n=1) used a modified version of Cole's algorithm.[40] The sleep–wake threshold/algorithm was not reported by 29% (n=13) of studies.[31 32 35 46 51 57–59 61 65 66 68 69]

Quantitative synthesis

Thirteen studies reported sleep outcomes for multiple nights surrounding competition and were included in the meta-analysis.[23–25 30 34 36 38 45 55 58 66 67 79] All included studies recruited 'competitive-elite' athletes competing at the highest level in their sport. Pooled effects for each time-point comparison (A–E) are shown in figure 2 (TST) and figure 3 (SE). Funnel plots examining publication bias are provided in online supplementary file 4.

TST was shorter (approximately 60 min) the night of compared with two nights before (comparison B, p<0.01) and the night before competition (comparison E, p<0.01) (figure 2). For comparisons B and E, TST was shorter the night of competition in both home (p<0.01) and away (p<0.01) environments. For comparison E, TST was shorter (approximately 80 min) the night of night competition (p<0.01) and shorter (approximately 20 min) the night of day competition (p=0.032) compared with the night before competition (figure 4). No differences (p>0.05) for TST were found for any other time-point (A, C, D) or subgroup comparisons. TST heterogeneity was moderate for comparisons A (I^2=39%), C (I^2=48%) and D (I^2=36%), and high for comparisons B (I^2=66%) and E (I^2=76%).

SE was lower (approximately 1%) the night of competition compared with the night before competition (comparison E, p=0.02) (figure 3). This effect was driven by night competition, as SE was lower (approximately 3%) the night of night competition (p<0.01) but unchanged the night of day competition (p=0.378) (figure 4). SE tended to be lower compared with two nights before competition (comparison B, p=0.058). However, when just night competition was examined, SE was lower (approximately 4%) the night of competition (p=0.012) compared with two nights before (note: meta-analysis of 'day' subgroup for comparison B was not possible due to limited data) figure 5. No differences (p>0.05) for SE were found for any other time-point (A, C, D) or subgroup comparison. Heterogeneity was low for all comparisons (A–D=0%, E=6%).

Qualitative synthesis

General sleep characteristics

Seven studies examined sleep across a competitive week, month or season.[7 30 33 45 49 61 67] Of these, few found athletes, on average, did not achieve current recommendations for TST (n=2/7 studies)[30 33] and SE (n=1/7 studies; online supplementary file 5).[33] Twenty-one studies examined sleep on one or more individual nights (−2 to +2) surrounding the competition.[7 23–25 30 32 34 36 38 41 45 49 53–55 58 59 66 67 72 74] Some studies reported cases where athletes were unable to achieve sleep recommendations on nights −2 (TST: n=2/7, SE: n=5/6 studies),[24 25 30 34 38 49 66] −1 (TST: n=2/15, SE: n=6/13 studies),[23 25 30 34 38 49 55 66] +1 (TST: n=4/11, SE: n=5/9

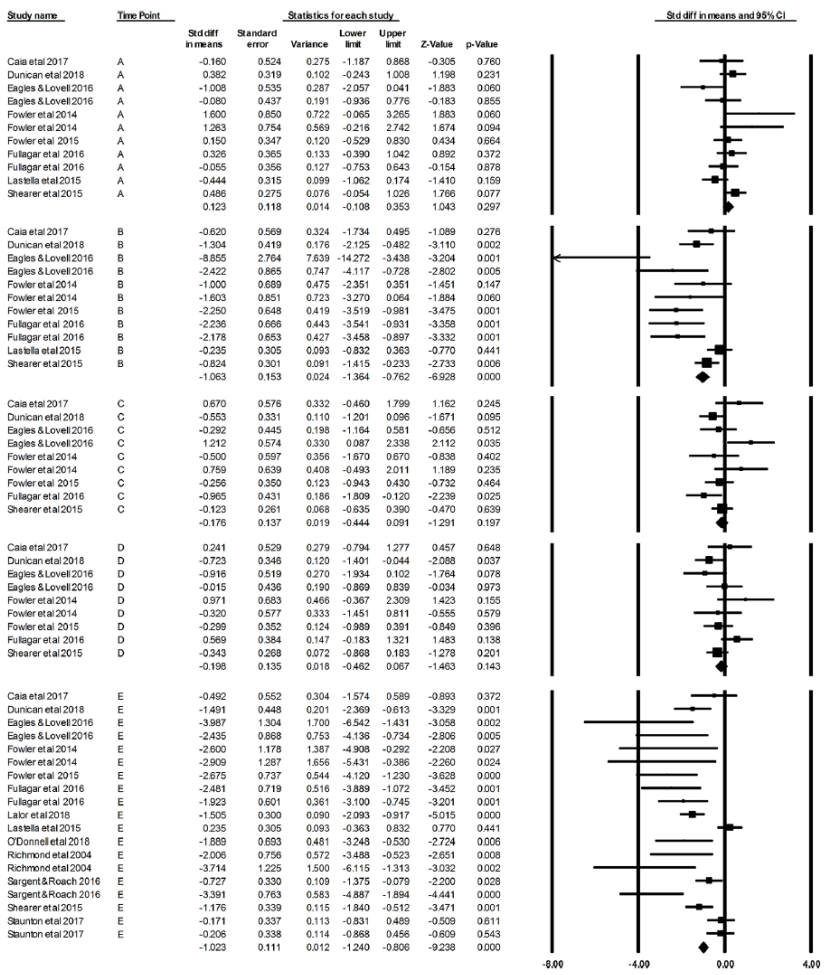

Figure 2 Forest plots showing total sleep time effects for time-point comparisons A (−2 vs −1), B (−2 vs 0), C (−2 vs +1), D (−2 vs +2) and E (−1 vs 0). Effects to the left of 0.00 indicate higher values for the first night of the comparison, whereas effects to the right of 0.00 indicate higher values for the second night of the comparison.

studies)[25 34 36 38 55 66] and +2 (TST: n=3/8, SE: n=2/6 studies).[25 34 36 66] On the night of competition (0), most studies found athletes were unable to achieve recommendations for TST (n=14/18 studies)[23–25 30 34 36 38 49 53 55 58 66 72 74] and SE (n=10/16 studies; online supplementary file 6).[23 25 34 38 53 55 58 66 72 74] Forty-two studies examined sleep during a training or non-competition period.[7 13 29–31 35 37 39 40 42–54 56–58 60–65 67–77] Of these, many reported cases whereby athletes were unable to achieve recommendations for TST (n=23/41 studies)[29–31 35 40 43 44 46 47 50–53 57 62 63 65 68 69 72 75–77] and SE (n=16/36 studies; online supplementary file 7).[35 37 43 45 47 48 51 56 57 62 64 65 69 70 72 74]

The night of competition

Eighteen studies examined sleep the night of competition.[7 23–25 30 34 36–38 41 45 53 55 58 66 67 74 79] Shorter TST the night of competition compared with surrounding nights (−2 to +2) was frequently reported.[24 25 30 34 36 38 45 55 58 66] Only three studies reported no difference in TST the night of competition compared with surrounding nights.[37 49 67] However, one of these found that TST was shorter the night of competition when 'double-header' fixtures (ie, two matches in 3 days) were played,[67] and another found TST was shorter the night of competition compared with prior training.[49] Similarly, the TST of netballers was shorter the night of a game compared with the night of a rest day[41 74] and the night of a training day,[74] while some evidence suggested TST was shorter the night of night-time compared with day competition,[7 23] and the night of away compared with home competition.[58]

Most studies reported no change in SE the night of competition compared with surrounding nights or training

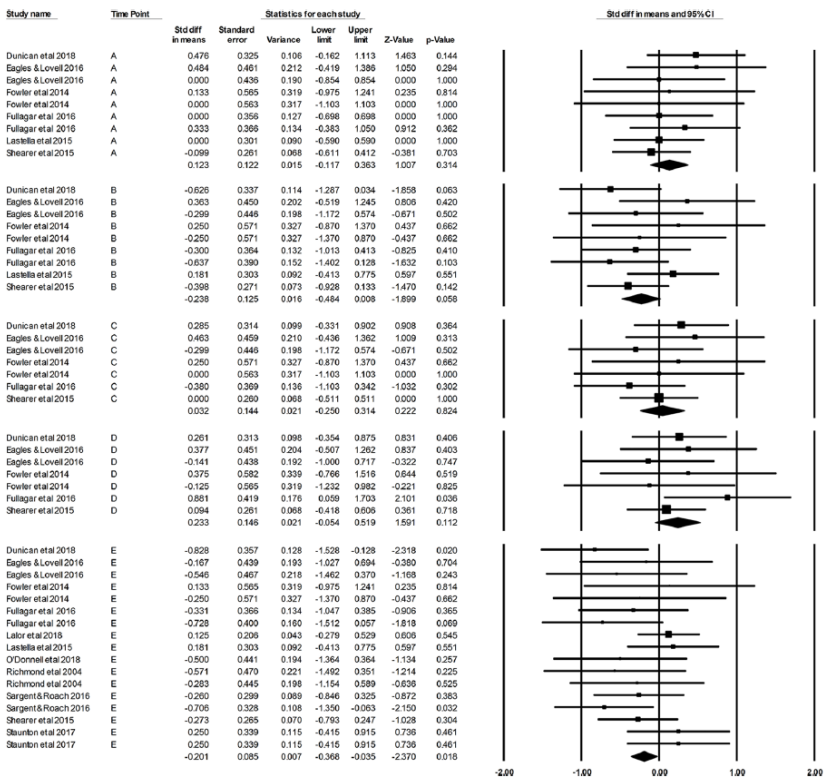

Figure 3 Forest plots showing sleep efficiency effects for time-point comparisons A (−2 vs −1), B (−2 vs 0), C (−2 vs +1), D (−2 vs +2) and E (−1 vs 0). Effects to the left of 0.00 indicate higher values for the first night of the comparison, whereas effects to the right of 0.00 indicate higher values for the second night of the comparison.

periods.[24 25 38 45 4955 58 66 67] However, SE was lower the night of a match compared with surrounding nights in rugby union players,[34] and compared with the night of a rest day among netballers.[41] Similarly, SE was very low (70%±10%) the night of competition and outside preseason intraindividual variability (90%±3%) in a soccer player.[53] However, conversely, compared with sleep monitored during preseason, SE was *almost certainly* higher the night of competition among Australian rules footballers.[45]

The night before competition

Fifteen studies examined sleep the night before competition.[23–25 30 32 36 3845 49 53 58 59 66 67] Most reported no difference in TST or SE the night before competition compared with surrounding non-competition nights (−2, +1, +2).[23–25 30 36 38 45 49 66 67] However, TST the night before competition was longer compared with the night after (+1) in netballers,[55] and was *likely* longer than the night after (+1) and two nights after (+2) competition in Australian rules footballers.[45] Additionally, both TST and SE the night before a match were *almost certainly* greater than during preseason among Australian rules footballers.[45] Several other studies found TST to be longer the night before competition compared with prior training.[58 67 80] However, one study found TST was shorter the night before competition compared with prior training among cyclists.[49] Interestingly, TST was longer before a final than before a semifinal in swimmers who were unable to achieve a top-four finish at national championships.[32] Finally, SE was lower on nights prior to a match where an injury occurred (66%–79%) than the intraindividual variability (90%±3%) recorded during preseason.[53]

Training schedule

Six studies examined the effects of training schedules on sleep.[13 30 43 62 63 68] TST was shorter prior to training days than prior to rest days,[30 43 62 63] particularly when training started before 07:00, as in rugby league players during preseason[30] and rowers/swimmers preparing for competition.[43 62] No difference in TST prior to training versus rest days was observed in rugby league players when training commenced at 07:30[68] or 09:00.[30] Shifting training start from 06:30 to 09:30 increased prior TST in judo athletes,[13] while a study that examined the effect of different training start times found athletes who started between 05:00 and 06:00 obtained less than 5 hours of TST, and those who started between 10:00 and 11:00 obtained more than 7 hours.[63] It was predicted that TST will reduce by just 6 min when start is advanced from 08:00 to 07:00, but by 48 min when advanced from 08:00 to 06:00.[63]

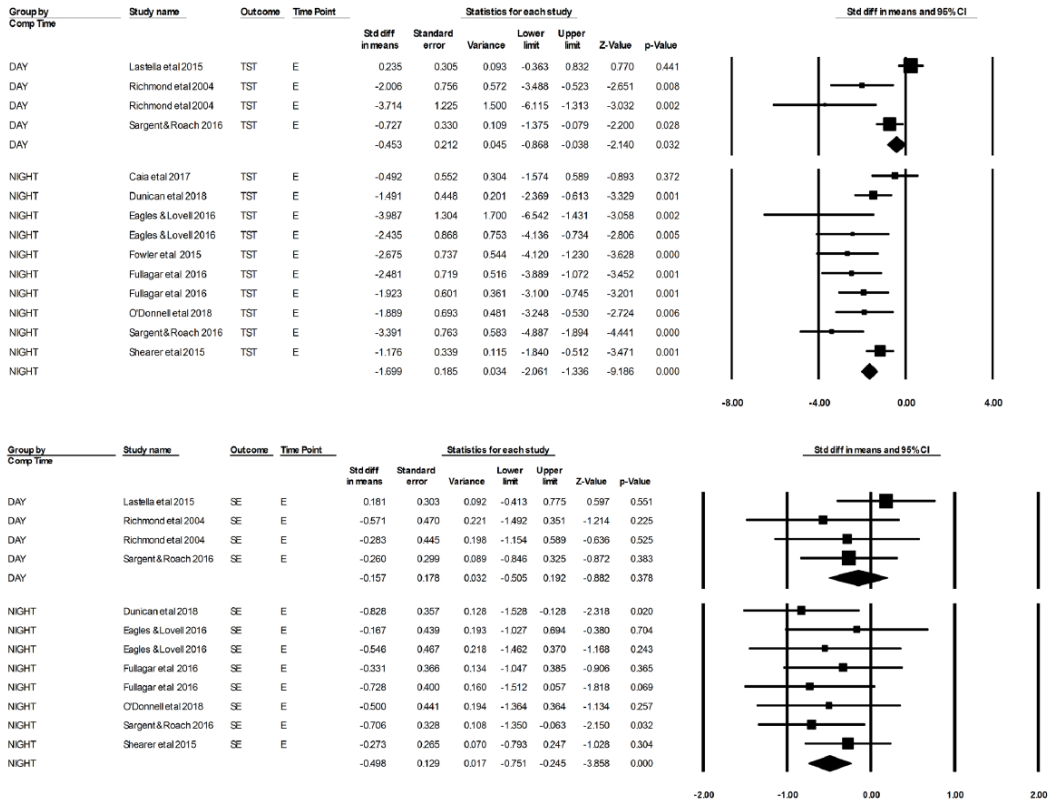

Figure 4 Forest plots showing total sleep time (TST) (top) and sleep efficiency (SE) (bottom) effects for time-point comparison E (–1 vs 0) when subgroup analysis 'competition start time' was applied. Includes data from studies that reported competition started either before (ie, 'day') or after (ie, 'night')18:00. Effects to the left of 0.00 indicate higher values for the first night of the comparison, wheras effects to the right of 0.00 indicate higher values for the second night of the comparison

Only when training commenced after 09:00 did athletes consistently achieve 7 or more hours of TST.[63] No effects of training schedule on SE were reported.

Training load

Eleven studies examined the effects of training load on sleep.[30 42–44 48 57 60 65 68 69 71] Rugby league players, synchronised swimmers and cyclists had reduced TST and SE following large increases (>25%) in training load.[48 65 69] Rowers also had shorter TST, but no change in SE, during periods of higher training load,[43] while distance covered during training negatively correlated with subsequent TST ($r=-0.31$) among rugby league players.[69] Shorter TST was also observed among rowers in the final days of a training camp compared with subsequent nights at a competition when sleep opportunities increased.[44] In contrast, among soccer players, no difference in TST or SE was found on nights following light training versus heavy training,[60] and the TST of swimmers was the same during peak and tapered training periods.[71]

Four studies examined sleep in response to day-to-day variations in individual training load. In a study of athletes from several sports, neither TST nor SE was associated with day-to-day variation in load.[42] However, in rugby league players, a small correlation between within-player load and both TST and SE ($r=0.11$, respectively) was reported.[30] Interestingly, the TST of rugby league players was reduced the more within-player training load differed (whether it increased or decreased) from the individual mean training load.[57] It was also suggested, using magnitude-based inferences, that increased acceleration/deceleration during training may increase SE and TST,[68] while increased distance covered and distance covered at high speed may lead to lower TST and lower SE, respectively.[68]

Hypoxia

Four studies examined the effects of hypoxia on sleep.[40 46 64 72] Three examined soccer players who ascended altitude (ie, hypobaric).[46 64 72] Compared with sleeping near sea level (430 m), TST at high altitude (3600 m) was reduced the first night after ascending and on 5 of 12 subsequent nights.[72] Similarly, SE was reduced on the first night after ascending and on 3 of 12 subsequent nights.[72] In a subset of these players, PSG found SE and TST were *possibly* lower on the first few nights of exposure,[64] but that after almost 2 weeks at altitude SE was *possibly* higher than was recorded at sea level.[64] Similarly,

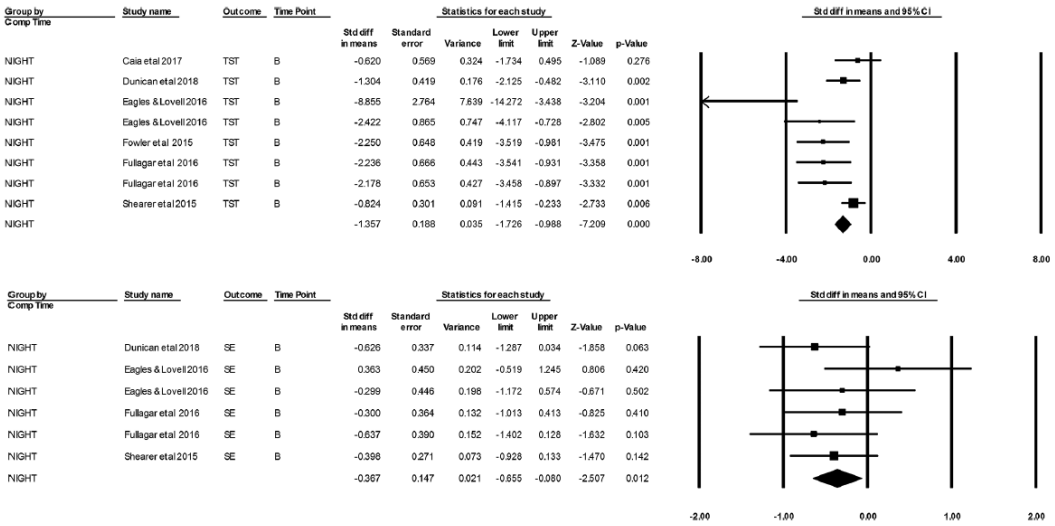

Figure 5 Forest plots showing total sleep time (TST) (top) and sleep efficiency (SE) (bottom) effects for time-point comparison B (−2 vs 0) when subgroup analysis 'competition start time' was applied. Includes only data from studies reporting a 'night' start time, as only one study reported a 'day' start time for comparison B. Effects to the left of 0.00 indicate higher values for the first night of the comparison, whereas effects to the right of 0.00 indicate higher values for the second night of the comparison.

TST was shorter in the days after travelling from sea level to 1600 m, without being further affected following an additional ascent to 2150 m.[46] No changes in TST or SE were observed in runners undertaking six nights of normobaric altitude (2000 m) exposure.[40]

Air travel

Eight studies examined the effects of air travel on sleep.[25 36 38 44 46 58 59 72] Early morning and late night departures reduced TST and SE prior to departure compared with non-travel nights.[36 46 81] Soccer players had shorter TST the first night after long-haul eastward travel compared with baseline sleep.[46 72] However, rowers had longer TST on arrival after long-haul westward travel compared with nights prior to travel.[44] No change in TST or SE has been observed in response to short-haul travel.[25 58 59] However, in Australian rules footballers, TST was shorter on nights of away matches when a postgame flight was undertaken compared with home matches where no flight was required.[58]

Electronic devices

Two studies examined the effects of electronic device use on sleep. In judo competitors, the use of electronic devices during a training camp had no effect on TST or SE,[13] while in netballers no significant association between duration of electronic device use and subsequent sleep outcomes was found during either training or competition.[61]

Additional factors

One study reported lower SE in male compared with female athletes,[51] while another found SE was lower when sleeping away compared with sleeping at home despite no air travel or difference in training load between environments.[57] One study found individual sport athletes have lower TST and SE than team sport athletes,[50] and one study found professional rugby league players obtained more sleep than semielite players.[29]

Results summary

The major findings of this review are that (1) athletes are often unable to achieve ≥7 hours of TST and ≥85% SE during training periods or on the night of competition; (2) both TST and SE are reduced the night of competition compared with previous nights, particularly after night (start ≥18:00) competition; (3) athletes typically achieve ≥7 hours of TST and ≥85% SE the night before competition, and neither TST nor SE is reduced the night before competition compared with surrounding nights; (5) scheduling training before 07:00 reduces TST the night before training and starting after 09:00 may be necessary to consistently achieve ≥7 hours of TST; (6) large increases (ie, ≥25%) in training load often lead to reductions in both TST and SE; (7) TST and SE may be reduced in the initial days after ascending altitude (≥1600 m); (8) early morning and late night travel departure times reduce TST and SE, while long-haul eastward travel reduces TST immediately on arrival; and (10) no effect of electronic device use on TST or SE has been reported.

DISCUSSION

This review aimed to (1) characterise the sleep of athletes in the context of current sleep recommendations; and (2) identify factors associated with training and competition that negatively affect sleep.

Sleep characteristics

Athletes are often unable to achieve ≥7 hours of TST and ≥85% SE during training and on the night of competition. These results are especially concerning as studies with mean data indicating athletes achieve these recommendations likely

include individuals and/or nights that do not. Previously, athletes recorded a SE below 85% on 22% of nights examined despite a mean SE of 88%.[42]

The night of competition

Athletes rarely achieved TST or SE recommendations on the night of competition. Meta-analyses found TST and SE were reduced the night of competition compared with precompetition nights, particularly following a night competition. Reductions in TST the night of competition are often attributed to a delay in bedtime, which reduces overall time in bed.[23 24 30 66 81 82] Several factors may explain delayed bedtimes and lower SE the night of competition. For example, increased circulating cortisol,[74] sympathetic hyperactivity,[83] elevated core body temperature[84] and muscle pain[85] may persist postcompetition and increase arousal.[86 87] Cortisol levels following a race have been negatively ($r=-0.90$) correlated with SE in swimmers,[32] while elevated cortisol postcompetition was associated with a reduction in TST the night of competition in netballers.[74] Additionally, the ergogenic use of caffeine[34] and exposure to bright light (eg, stadium lighting) may interfere with the neurological processes that promote sleep.[88 89] In support, increases in salivary caffeine on match day have been correlated with increases in sleep latency ($r=0.53$) and decreases in SE ($r=0.52$) in rugby union players.[34] Finally, delayed bedtimes may be attributed to postcompetition recovery/medical interventions, meetings, travel and media commitments.[30 38] This review featured predominantly male field sport athletes; thus, poor sleep the night of competition may reflect unique sports-specific challenges faced by these athletes. For example, Australian rules football, rugby union and rugby league are sports characterised by frequent high-speed collisions,[90] perhaps suggesting postcompetition muscle soreness may contribute to sleep disturbances.[87] Likewise, within-sport factors, such as travel, may explain sleep disturbances. For example, in Australian rules footballers, TST the night of away games, which required players to fly home immediately postgame, was lower than TST the night of home games that required no travel.[58] These findings may have implications for the time-course of recovery in the days postcompetition and highlight the need to prioritise sleep during this period.

The night before competition

Qualitative assessments suggest precompetition sleep disturbances are prevalent among athletes.[91–93] However, neither the systematic review nor meta-analyses found consistent evidence of this. Compared with training, TST was shorter the night before a race among cyclists,[49] but longer the night before a match among Australian rules footballers.[58 59] This review reported that individual sport athletes have lower TST and SE than team sport athletes during out-of-competition training.[50] However, when sleep was examined prior to competition, comparisons of individual and team sport athletes have delivered equivocal findings.[91 92] Nonetheless, it is suggested that individual athletes,[91] particularly those competing in aesthetic sports, may be most susceptible to sleep disturbances.[94] In addition, while this review found SE during training was lower in men compared with women,[51] there is some evidence that prior to competition sleep disturbances are more prevalent among women.[91 94]

The principal cause of precompetition sleep disturbance is anxiety or thoughts about the competition.[91 92] Individual athletes may experience particularly high levels of anxiety because they cannot share the psychological burden of competition with team members,[50 91 94] which may be exacerbated in aesthetic sport athletes whose success depends on the judgement of others.[94] Moreover, anxiety disorders have been shown to be more prevalent among female versus male athletes.[94] Therefore, the limited representation of individual sport, aesthetic sport or female athletes in the present review may explain why consistent evidence for precompetition sleep disturbances were not found.

Early morning training

Reductions in TST on nights prior to training were noted primarily when training commenced at or before 07:00. It is speculated that shorter TST among individual sport athletes compared with team sport athletes may reflect a tendency for individual sports to commence training early in the morning.[50] While athletes try to offset the effects of early training by going to bed earlier, it is often difficult to prevent reductions in TST.[63] For example, on a training camp, rowers woke approximately 2.5 hours earlier on training days compared with rest days, but went to bed just 25 min earlier the night before.[43] The inability to go to bed early has been attributed to the fact that most social engagements are scheduled in the evening, and the so-called 'forbidden zone'—a period of heightened arousal mediated by the body's circadian rhythms—typically reduces sleep propensity between 20:00 and 22:00.[63]

Increased training load

TST and SE were reduced following large increases (>25%) in training load.[48 65 69] This is consistent with reductions in TST and SE among subelite triathletes following a 30% increase in training load.[95] In contrast, most studies that suggested sleep was unaffected or even improved with higher training loads did not involve large increases in training load, rather investigated sleep responses to subtle within-subject changes in load.[30 57 68 71] Therefore, reductions in TST and SE may reflect a negative adaptation to training in response to large increases in training load. Heavy training can lead to increases in circulating cortisol[96] and sympathetic activity,[97] both of which may prevent the normal downregulation of the human stress systems (ie, hypothalamic-pituitary-adrenal, sympathetic-adrenal-medullary) required for healthy sleep.[98 99] In support, changes in resting alpha-amylase, a marker of sympathetic activity, have been negatively correlated with changes in SE among synchronised swimmers.[65] Increases in training load may also disturb sleep via pain associated with muscle soreness and frequent micturition at night associated with rehydration efforts.

Hypoxia

Exposure to high altitude was associated with reductions in TST and SE. This is consistent with previous research suggesting hypoxia causes a shift towards lighter, more fragmented sleep.[100] Both actual (ie, hypobaric) and simulated (ie, normobaric) altitude exposure have been shown to reduce TST and SE.[100 101] Sleep disturbances occur predominantly at altitudes above 2000 m in the initial days after exposure,[102] and are attributed to arousals caused by the hyperventilatory response to arterial desaturation[101] and sympathetic hyperactivity.[103] These findings suggest that when ascending altitude, sufficient time should be allowed for sleep to normalise before training or competition commences. A staged, gradual ascent may expedite this process, as demonstrated in soccer players whose sleep was consistent when ascending from 1600 m to 2150 m, despite initial reductions in TST after ascending to 1600 m.[46]

Air travel

Sleep disturbances associated with air travel in this review can be attributed to either jet lag (ie, circadian misalignment) or the encroachment of travel times on habitual sleep phase. Both late night and early morning departure times were shown to reduce TST and SE prior to departure,[36 38 46] which may have detrimental effects on athlete preparation. For example, high ratings of fatigue among soccer players following northward travel have been attributed to an early morning flight that reduced TST the night before departure, as jet lag was unlikely given the flight only crossed one time zone.[36] This review reported lower TST upon arrival after eastward travel,[46 72] which has also been reported in athletes whose sleep was monitored using self-reported diaries'.[104] In contrast, following westward travel, this review found evidence for an increase in TST on arrival.[44] However, this finding may be confounded by the fact that pretravel sleep was monitored during a training camp, and that there was an increase in sleep opportunities on arrival.[44] Nonetheless, there is evidence in subelite athletes that eastward travel is more disruptive to sleep than westward travel.[105] These findings suggest consideration for the timing of flight departures and the direction of travel is necessary to mitigate the negative effects of air travel.

Electronic devices

Evening exposure to light, particularly blue light, can cause sleep disturbances.[106] Delayed bedtimes and reductions in TST have been associated with electronic device use among school-aged children.[107 108] Sleep disturbances are usually attributed to light emitted from such devices that inhibits melatonin release.[109] However, this review found no evidence that electronic device use in the evening affects the sleep of athletes.[13 61] In support, an examination of the sleep of netballers found that despite melatonin levels rising more in players who read rather than used a tablet device before bed, no difference in subsequent sleep quality or quantity was observed.[110] Moreover, a study examining high school athletes found that restricting electronic media use after 22:00 did not improve sleep habits.[111] It may be speculated that the homeostatic pressure for sleep among athletes is such that any negative effects of electronic device use are masked. Blue light has been shown to preferentially reduce slow-wave sleep,[112] which is the type of sleep believed to promote recovery in athletes and which increases with prior energy expenditure.[113]

Sleep disorders

This review examined TST and SE in athletes during training and competition. As such, the prevalence of diagnosed sleep disorders was not reported in the results. It is important to acknowledge that the TST and SE outcomes reported in this review may reflect, to some extent, sleep disorders among the athletes investigated. Previous studies have examined the prevalence of sleep disorders among athletes.[75–77 114 115] Mild sleep-disordered breathing has been found to occur in 8% of college American footballers[76] and 19% of professional American footballers.[114] One in four professional ice hockey players has significant problems sleeping,[77] while a study examining rugby and cricket players found 38% defined themselves as snorers.[115] Athletes with high body mass index, large neck circumference or high levels of adiposity may be at a heightened risk of developing a sleep disorder.[76 114] This review included relatively few studies that examined such athletes (eg, rugby players); therefore, it is not expected that undiagnosed sleep disorders would affect the findings of the review.

Limitations

The findings of this review may not reflect the sleep of all athletes, as most reviewed studies recruited male field sport athletes. Only 28% of the studies included in this review were deemed of high quality, with most concerns arising from small sample sizes and the limited number of sports represented, which make it difficult to apply findings generally to all elite athletes. It is likely that the quality and quantity of sleep vary significantly between athletes and sports. As such, the high heterogeneity of TST reported in the meta-analysis may reflect numerous characteristics that vary between athletes (eg, age, sex, body composition) and the nature of their sport (eg, individual, team, aesthetic, contact). Another factor that may explain high heterogeneity is variation in data collection protocols. Studies that monitored sleep with actigraphy in this review used several different sleep–wake thresholds. This is a concern given the interpretation of data will vary depending on the threshold used.[17] In addition, while field data are necessary to determine how 'real-world' training and competition pressures affect sleep, an inherent limitation of such data is that potentially confounding factors, such as caffeine and alcohol intake, which may influence sleep, cannot be eliminated or controlled for. Methodological limitations include that initial screening of titles and abstracts was conducted by one reviewer, and that only articles published in English and in peer-reviewed journals were included.

Future research

Studies using actigraphy to monitor sleep should report the sleep–wake threshold used. There is contradictory evidence for which threshold (ie, medium vs high) is most appropriate for athletes[17 39]; therefore, research should explore how athlete type (ie, endurance vs power) and training phase (heavy vs light) affect bias of different thresholds.[39] Moreover, while it is important to develop strategies that can mitigate sleep disturbances (eg, sleep hygiene) with the aim of improving performance,[6] more objective data are required to better understand which athletes will benefit and when they will benefit from such strategies. For instance, despite reports that individual sport, aesthetic sport and female athletes are susceptible to sleep disturbances before competition,[94] there are limited supportive, objective data. Finally, given strong evidence in this review that sleep is disturbed the night of competition, future research should examine whether this affects the time-course of recovery, which may have implications for athlete management postcompetition.

CONCLUSIONS

This review found that athletes often sleep poorly during training and on the night of competition. Despite previous reports that athletes' sleep is disturbed prior to competition, this is not supported by objectively recorded data. Early morning training, large increases in training load, exposure to hypoxia, travel departure times and jet lag may all negatively affect athletes' sleep. Therefore, sleep during these times should be monitored and prioritised by athletes and support staff. Due to a paucity of literature objectively examining the sleep of individual sport and female athletes, the findings of this review may not reflect the sleep of all athletes.

SUMMARY

- Athletes are often unable to achieve total sleep time (TST) and sleep efficiency (SE) recommendations during training and on the night of competition (National Sleep Foundation).
- TST and SE are reduced the night of competition compared with previous nights

- Early morning training, increases in training load, exposure to hypoxic conditions, air travel departure times and jet lag can all negatively affect TST and SE.
- Future research should investigate individual sport and female athletes.

Correction notice This article has been corrected since it was published Online First. Reference 41 has been updated to reflect the actual details of the publication, and a sentence in the 'Air travel' section (page 9) has been edited for clarity.

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Appendix B

Human Research Ethics

Deakin Research Integrity
Burwood Campus
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research-ethics@deakin.edu.au

Memorandum

To: Dr Stuart Warmington
School of Exercise and Nutrition Sciences
B

cc: Mr Spencer Roberts

From: Deakin University Human Research Ethics Committee (DUHREC)

Date: 02 November, 2015

Subject: 2015-200
Using heart rate indices to examine the effect of sleep on endurance performance

Please quote this project number in all future communications

The application for this project was considered at the DU-HREC meeting held on 24/8/2015.

Approval has been given for Mr Spencer Roberts, under the supervision of Dr Stuart Warmington, School of Exercise and Nutrition Sciences, to undertake this project from 2/11/2015 to 2/11/2019.

The approval given by the Deakin University Human Research Ethics Committee is given only for the project and for the period as stated in the approval. It is your responsibility to contact the Human Research Ethics Unit immediately should any of the following occur:

- Serious or unexpected adverse effects on the participants
- Any proposed changes in the protocol, including extensions of time.
- Any events which might affect the continuing ethical acceptability of the project.
- The project is discontinued before the expected date of completion.
- Modifications are requested by other HRECs.

In addition you will be required to report on the progress of your project at least once every year and at the conclusion of the project. Failure to report as required will result in suspension of your approval to proceed with the project.

DUHREC may need to audit this project as part of the requirements for monitoring set out in the National Statement on Ethical Conduct in Human Research (2007).

Human Research Ethics Unit
research-ethics@deakin.edu.au
Telephone: 03 9251 7123

Appendix C

PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO:

Consent Form

Date:

Full Project Title: Using heart rate to examine the effect of sleep on endurance performance

Reference Number: 2015-200

I have read and understand the attached *Plain Language Statement.*

I freely agree to participate in **Study 1 / Study 2 / both studies** according to the conditions in the *Plain Language Statement.*

I have been given a copy of the Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal my identity and personal details, including where information about this project is published, or presented in any public form.

In addition, I would **(not) like** to receive copies of any publications arising from this research

Note: please cross out words in bold according to your preference

Participant's Name (printed) .....

Signature ..... Date .....

Contact: Spencer Roberts
Email: rspen@deakin.edu.au
Phone: 0422 352 936

Appendix D

The Pittsburgh Sleep Quality Index (PSQI)

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

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times week (3)
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
	Very good (0)	Fairly good (1)	Fairly bad (2)	Very bad (3)
9. During the past month, how would you rate your sleep quality overall?				

Component 1	#9 Score	C1_____
Component 2	#2 Score (≤15min=0; 16-30 min=1; 31-60 min=2, >60 min=3) +#5a Score (if sum is equal 0=0; 1-2=1; 3-4=2; 5-6=3)	C2_____
Component 3	#4 Score (>7=0; 6-7=1; 5-6=2; <5=3)	C3_____
Component 4	(total # of hours asleep)/(total # of hours in bed) x 100 >85%=0, 75%-84%=1, 65%-74%=2, <65%=3	C4_____
Component 5	Sum of Scores #5b to #5j (0=0; 1-9=1; 10-18=2; 19-27=3)	C5_____
Component 6	#6 Score	C6_____
Component 7	#7 Score + #8 Score (0=0; 1-2=1; 3-4=2; 5-6=3)	C7_____

Add the seven component scores together_____**Global PSQI Score**_____

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R., & Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Journal of Psychiatric Research, 28*(2), 193-213.

Appendix E

State-Trait Anxiety Inventory for Adults

Self-Evaluation Questionnaire

STAI Form Y-1 and Form Y-2

Developed by Charles D. Spielberger
in collaboration with R.L. Gorsuch, R. Lushene, P.R. Vagg, and G.A. Jacobs

Published by Mind Garden
1690 Woodside Road Suite 202, Redwood City, CA 94061 USA 650-261-3500
www.mindgarden.com

SELF-EVALUATION QUESTIONNAIRE STAI Form Y-1

Please provide the following information:

Name_____Date_____S_____

Age_____ Gender (*Circle*) **M F** T_____

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

STAIP-AD Test Form Y
www.mindgarden.com

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name_____ Date_____

DIRECTIONS

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
21. I feel pleasant	1	2	3	4
22. I feel nervous and restless	1	2	3	4
23. I feel satisfied with myself	1	2	3	4
24. I wish I could be as happy as others seem to be	1	2	3	4
25. I feel like a failure	1	2	3	4
26. I feel rested	1	2	3	4
27. I am "calm, cool, and collected"	1	2	3	4
28. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
29. I worry too much over something that really doesn't matter	1	2	3	4
30. I am happy	1	2	3	4
31. I have disturbing thoughts	1	2	3	4
32. I lack self-confidence	1	2	3	4
33. I feel secure	1	2	3	4
34. I make decisions easily	1	2	3	4
35. I feel inadequate	1	2	3	4
36. I am content	1	2	3	4
37. Some unimportant thought runs through my mind and bothers me	1	2	3	4
38. I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
39. I am a steady person	1	2	3	4
40. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

STAIP-AD Test Form Y
www.mindgarden.com

State-Trait Anxiety Inventory for Adults Scoring Key (Form Y-1, Y-2)

Developed by Charles D. Spielberger in collaboration with R.L. Gorsuch, R. Lushene, P.R. Vagg, and G.A. Jacobs

To use this stencil, fold this sheet in half and line up with the appropriate test side, either Form Y-1 or Form Y-2. Simply total the scoring **weights** shown on the stencil for each response category. For example, for question # 1, if the respondent marked 3, then the **weight** would be **2**. Refer to the manual for appropriate normative data.

Form Y-1	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1.	4	3	2	1
2.	4	3	2	1
3.	1	2	3	4
4.	1	2	3	4
5.	4	3	2	1
6.	1	2	3	4
7.	1	2	3	4
8.	4	3	2	1
9.	1	2	3	4
10.	4	3	2	1
11.	4	3	2	1
12.	1	2	3	4
13.	1	2	3	4
14.	1	2	3	4
15.	4	3	2	1
16.	4	3	2	1
17.	1	2	3	4
18.	1	2	3	4
19.	4	3	2	1
20.	4	3	2	1

Form Y-2	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
21.	4	3	2	1
22.	1	2	3	4
23.	4	3	2	1
24.	1	2	3	4
25.	1	2	3	4
26.	4	3	2	1
27.	4	3	2	1
28.	1	2	3	4
29.	1	2	3	4
30.	4	3	2	1
31.	1	2	3	4
32.	1	2	3	4
33.	4	3	2	1
34.	4	3	2	1
35.	1	2	3	4
36.	4	3	2	1
37.	1	2	3	4
38.	1	2	3	4
39.	4	3	2	1
40.	1	2	3	4

Appendix F

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

Name: _____ Date: _____

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

1. *Approximately* what time would you get up if you were entirely free to plan your day?

   [5] 5:00 AM–6:30 AM *(05:00–06:30 h)*
   [4] 6:30 AM–7:45 AM *(06:30–07:45 h)*
   [3] 7:45 AM–9:45 AM *(07:45–09:45 h)*
   [2] 9:45 AM–11:00 AM *(09:45–11:00 h)*
   [1] 11:00 AM–12 noon *(11:00–12:00 h)*

2. *Approximately* what time would you go to bed if you were entirely free to plan your evening?

   [5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
   [4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
   [3] 10:15 PM–12:30 AM *(22:15–00:30 h)*
   [2] 12:30 AM–1:45 AM *(00:30–01:45 h)*
   [1] 1:45 AM–3:00 AM *(01:45–03:00 h)*

3. If you usually have to get up at a specific time in the morning, how much do you depend on an alarm clock?

   [4] Not at all
   [3] Slightly
   [2] Somewhat
   [1] Very much

---

[1]Some stem questions and item choices have been rephrased from the original instrument (Horne and Östberg, 1976) to conform with spoken American English. Discrete item choices have been substituted for continuous graphic scales. Prepared by Terman M, Rifkin JB, Jacobs J, White TM (2001), New York State Psychiatric Institute, 1051 Riverside Drive, Unit 50, New York, NY, 10032. January 2008 version. Supported by NIH Grant MH42931. *See also:* automated version (AutoMEQ) at www.cet.org.

Horne JA and Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. International Journal of Chronobiology, 1976: 4, 97-100.

4. How easy do you find it to get up in the morning (when you are not awakened unexpectedly)?

   [1] Very difficult
   [2] Somewhat difficult
   [3] Fairly easy
   [4] Very easy

5. How alert do you feel during the first half hour after you wake up in the morning?

   [1] Not at all alert
   [2] Slightly alert
   [3] Fairly alert
   [4] Very alert

6. How hungry do you feel during the first half hour after you wake up?

   [1] Not at all hungry
   [2] Slightly hungry
   [3] Fairly hungry
   [4] Very hungry

7. During the first half hour after you wake up in the morning, how do you feel?

   [1] Very tired
   [2] Fairly tired
   [3] Fairly refreshed
   [4] Very refreshed

8. If you had no commitments the next day, what time would you go to bed compared to your usual bedtime?

   [4] Seldom or never later
   [3] Less that 1 hour later
   [2] 1-2 hours later
   [1] More than 2 hours later

9. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week, and the best time for him is between 7-8 AM *(07-08 h)*. Bearing in mind nothing but your own internal "clock," how do you think you would perform?

   [4] Would be in good form
   [3] Would be in reasonable form
   [2] Would find it difficult
   [1] Would find it very difficult

10. At *approximately* what time in the evening do you feel tired, and, as a result, in need of sleep?

   [5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
   [4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
   [3] 10:15 PM–12:45 AM *(22:15–00:45 h)*
   [2] 12:45 AM–2:00 AM *(00:45–02:00 h)*
   [1] 2:00 AM–3:00 AM *(02:00–03:00 h)*

11. You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last two hours. You are entirely free to plan your day. Considering only your "internal clock," which one of the four testing times would you choose?

   [6] 8 AM–10 AM *(08–10 h)*
   [4] 11 AM–1 PM *(11–13 h)*
   [2] 3 PM–5 PM *(15–17 h)*
   [0] 7 PM–9 PM *(19–21 h)*

12. If you got into bed at 11 PM *(23 h)*, how tired would you be?

   [0] Not at all tired
   [2] A little tired
   [3] Fairly tired
   [5] Very tired

13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which one of the following are you most likely to do?

[4] Will wake up at usual time, but will not fall back asleep
[3] Will wake up at usual time and will doze thereafter
[2] Will wake up at usual time, but will fall asleep again
[1] Will not wake up until later than usual

14. One night you have to remain awake between 4-6 AM *(04-06 h)* in order to carry out a night watch. You have no time commitments the next day. Which one of the alternatives would suit you best?

[1] Would not go to bed until the watch is over
[2] Would take a nap before and sleep after
[3] Would take a good sleep before and nap after
[4] Would sleep only before the watch

15. You have two hours of hard physical work. You are entirely free to plan your day. Considering only your internal "clock," which of the following times would you choose?

[4] 8 AM–10 AM *(08–10 h)*
[3] 11 AM–1 PM *(11–13 h)*
[2] 3 PM–5 PM *(15–17 h)*
[1] 7 PM–9 PM *(19–21 h)*

16. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week. The best time for her is between 10-11 PM *(22-23 h)*. Bearing in mind only your internal "clock," how well do you think you would perform?

[1] Would be in good form
[2] Would be in reasonable form
[3] Would find it difficult
[4] Would find it very difficult

17. Suppose you can choose your own work hours. Assume that you work a five-hour day (including breaks), your job is interesting, and you are paid based on your performance. At *approximately* what time would you choose to begin?

[5] 5 hours starting between 4–8 AM *(05–08 h)*
[4] 5 hours starting between 8–9 AM *(08–09 h)*
[3] 5 hours starting between 9 AM–2 PM *(09–14 h)*
[2] 5 hours starting between 2–5 PM *(14–17 h)*
[1] 5 hours starting between 5 PM–4 AM *(17–04 h)*

18. At *approximately* what time of day do you usually feel your best?

[5] 5–8 AM *(05–08 h)*
[4] 8–10 AM *(08–10 h)*
[3] 10 AM–5 PM *(10–17 h)*
[2] 5–10 PM *(17–22 h)*
[1] 10 PM–5 AM *(22–05 h)*

19. One hears about "morning types" and "evening types." Which one of these types do you consider yourself to be?

[6] Definitely a morning type
[4] Rather more a morning type than an evening type
[2] Rather more an evening type than a morning type
[1] Definitely an evening type

____ Total points for all 19 questions

INTERPRETING AND USING YOUR MORNINGNESS-EVENINGNESS SCORE

This questionnaire has 19 questions, each with a number of points. First, add up the points you circled and enter your total morningness-eveningness score here:

Scores can range from 16-86. Scores of 41 and below indicate "evening types." Scores of 59 and above indicate "morning types." Scores between 42-58 indicate "intermediate types."

16-30	31-41	42-58	59-69	70-86
definite evening	moderate evening	intermediate	moderate morning	definite morning

Occasionally a person has trouble with the questionnaire. For example, some of the questions are difficult to answer if you have been on a shift work schedule, if you don't work, or if your bedtime is unusually late. Your answers may be influenced by an illness or medications you may be taking. *If you are not confident about your answers, you should also not be confident about the advice that follows.*

One way to check this is to ask whether your morningness-eveningness score approximately matches the sleep onset and wake-up times listed below:

Score	16-30	31-41	42-58	59-69	70-86
Sleep onset	2:00-3:00 AM *(02:00-03:00 h)*	12:45-2:00 AM *(00:45-02:00 h)*	10:45 PM-12:45 AM *(22:45-00:45 h)*	9:30-10:45 PM *(21:30-22:45 h)*	9:00-9:30 PM *(21:00-21:30 h)*
Wake-up	10:00-11:30 AM *(10:00-11:30 h)*	8:30-10:00 AM *(08:30-10:00 h)*	6:30-8:30 AM *(06:30-08:30 h)*	5:00-6:30 AM *(05:00-06:30 h)*	4:00-5:00 AM *(04:00-05:00 h)*

If your usual sleep onset is earlier than 9:00 PM *(21:00 h)* or later than 3:00 AM *(03:00 h),* or your wake-up is earlier than 4:00 AM *(04:00 h)* or later than 11:30 AM *(11:30 h),* you should seek the advice of a light therapy clinician in order to proceed effectively with treatment.

We use the morningness-eveningness score to improve the antidepressant effect of light therapy. Although most people experience good antidepressant response to light therapy when they take a regular morning session using a 10,000 lux white light device *(see www.cet.org for recommendations)* for 30 minutes, often this will not give the best possible response. If your internal clock is shifted relative to external time (as indirectly measured by your morningness-eveningness score), the timing of light therapy needs to be adjusted.

The table at the top of the next page shows the recommended start time for light therapy for a wide range of morningness-eveningness scores. If your score falls beyond this range (either very low or very high), you should seek the advice of a light therapy clinician in order to proceed effectively with treatment.

Morningness-Eveningness Score	Start time for light therapy
23-26	8:15 AM
27-30	8:00 AM
31-34	7:45 AM
35-38	7:30 AM
39-41	7:15 AM
42-45	7:00 AM
46-49	6:45 AM
50-53	6:30 AM
54-57	6:15 AM
58-61	6:00 AM
62-65	5:45 AM
66-68	5:30 AM
69-72	5:15 AM
73-76	5:00 AM

If you usually sleep longer than 7 hours per night, you will need to wake up somewhat earlier than normal to achieve the effect – but you should feel better for doing that. Some people compensate by going to bed earlier, while others feel fine with shorter sleep. If you usually sleep less than 7 hours per night you will be able to maintain your current wake-up time. If you find yourself automatically waking up more than 30 minutes before your session start time, you should try moving the session later. Avoid taking sessions earlier than recommended, but if you happen to oversleep your alarm clock, it is better to take the session late than to skip it.

Our recommended light schedule for evening types – say, 8:00 AM (08:00 h) for a morningness-eveningness score of 30 – may make it difficult to get to work on time, yet taking the light earlier may not be helpful. Once you have noted improvement at the recommended hour, however, you can begin inching the light therapy session earlier by 15 minutes per day, enabling your internal clock to synchronize with your desired sleep-wake cycle and work schedule.

The personalized advice we give you here is based on a large clinical trial of patients with seasonal affective disorder (SAD) at Columbia University Medical Center in New York. Patients who took the light too late in the morning experienced only half the improvement of those who took it approximately at the times indicated. These guidelines are not only for SAD, but are also helpful in treatment of nonseasonal depression, for reducing insomnia at bedtime, and for reducing the urge to oversleep in the morning.

Our advice serves only as a *general guideline* for new users of light therapy. There are many individual factors that might call for a different schedule or dose (intensity, duration) of light. *Any person with clinical depression should proceed with light therapy only under clinical guidance.*

Reference: Terman M, Terman JS. Light therapy for seasonal and nonseasonal depression: efficacy, protocol, safety, and side effects. CNS Spectrums, 2005;10:647-663. (Downloadable at www.cet.org)

Appendix G

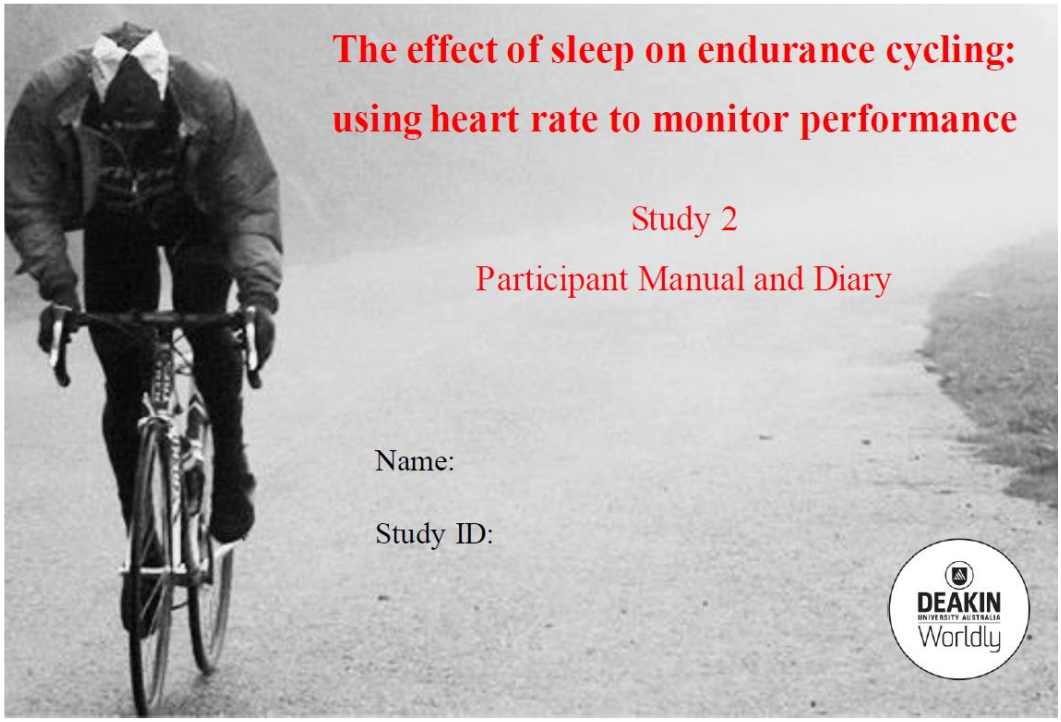
PROFILE OF MOOD STATES

Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in ONE circle that best describes HOW YOU HAVE BEEN FEELING DURING THE PAST WEEK INCLUDING TODAY?

	Not at all	A little	Moderately	Quite a bit	Extremely
1. FRIENDLY	0	0	0	0	0
2. TENSE	0	0	0	0	0
3. ANGRY	0	0	0	0	0
4. WORN OUT	0	0	0	0	0
5. UNHAPPY	0	0	0	0	0
6. CLEAR HEADED	0	0	0	0	0
7. LIVELY	0	0	0	0	0
8. CONFUSED	0	0	0	0	0
9. SORRY FOR THINGS DONE	0	0	0	0	0
10. SHAKY	0	0	0	0	0
11. LISTLESS	0	0	0	0	0
12. PEEVED	0	0	0	0	0
13. CONSIDERATE	0	0	0	0	0
14. SAD	0	0	0	0	0
15. ACTIVE	0	0	0	0	0
16. ON EDGE	0	0	0	0	0
17. GROUCHY	0	0	0	0	0
18. BLUE	0	0	0	0	0
19. ENERGETIC	0	0	0	0	0
20. PANICKY	0	0	0	0	0
21. HOPELESS	0	0	0	0	0
22. RELAXED	0	0	0	0	0
23. UNWORTHY	0	0	0	0	0
24. SPITEFUL	0	0	0	0	0
25. SYMPATHETIC	0	0	0	0	0
26. UNEASY	0	0	0	0	0
27. RESTLESS	0	0	0	0	0
28. UNABLE TO CONCENTRATE	0	0	0	0	0
29. FATIGUED	0	0	0	0	0
30. HELPFUL	0	0	0	0	0
31. ANNOYED	0	0	0	0	0

	Not at all	A little	Moderately	Quite a bit	Extremel
32. DISCOURAGED	0	0	0	0	0
33. RESENTFUL	0	0	0	0	0
34. NERVOUS	0	0	0	0	0
35. LONELY	0	0	0	0	0
36. MISERABLE	0	0	0	0	0
37. MUDDLED	0	0	0	0	0
38. CHEERFUL	0	0	0	0	0
39. BITTER	0	0	0	0	0
40. EXHAUSTED	0	0	0	0	0
41. ANXIOUS	0	0	0	0	0
42. READY TO FIGHT	0	0	0	0	0
43. GOOD NATURED	0	0	0	0	0
44. GLOOMY	0	0	0	0	0
45. DESPERATE	0	0	0	0	0
46. SLUGGISH	0	0	0	0	0
47. REBELLIOUS	0	0	0	0	0
48. HELPLESS	0	0	0	0	0
49. WEARY	0	0	0	0	0
50. BEWILDERED	0	0	0	0	0
51. ALERT	0	0	0	0	0
52. DECEIVED	0	0	0	0	0
53. FURIOUS	0	0	0	0	0
54. EFFICIENT	0	0	0	0	0
55. TRUSTING	0	0	0	0	0
56. FULL OF PEP	0	0	0	0	0
57. BAD TEMPERED	0	0	0	0	0
58. WORTHLESS	0	0	0	0	0
59. FORGETFUL	0	0	0	0	0
60. CAREFREE	0	0	0	0	0
61. TERRIFIED	0	0	0	0	0
62. GUILTY	0	0	0	0	0
63. VIGOROUS	0	0	0	0	0
64. UNCERTAIN ABOUT THINGS	0	0	0	0	0
65. BUSHED	0	0	0	0	0

Appendix H



Important contacts

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Dr. Stuart Warmington

Email: stuart.warmington@deakin.edu.au

Dr. Wei-Peng Teo

Email: weipeng.teo@deakin.edu.au

1

About you

Full Name: _____

Date of Birth: _____ Age: _____

Cycling Profile

Are you a cyclist or a triathlete? _____

How long have you been competing (i.e., years)? _____

On average, how often do you cycle (i.e., weekly)? _____

What is the typical duration of a cycling session (i.e., hours)? _____

Do you undertake any training other than cycling? _____

How many hours do you spend training (i.e., weekly)? _____

2

Psychometric Profile

Pittsburgh Sleep Quality Index: _____

State-Trait Anxiety Inventory: _____

Morningness-Eveningness Questionnaire: _____

Epworth Sleepiness Scale: _____

Profile of Mood States: _____

Consultation with general practitioner recommended? Yes □ No □

3

Physical / Physiological Profile

Height (m): _____

Weight (kg): _____

Resting Heart Rate (beats.min^{-1}): _____

Haemoglobin (g.dl^{-1}): _____

Haematocrit (g.dl^{-1}): _____

$VO_{2\ max}$ (l.min^{-1}): _____

$VO_{2\ max}$ (ml.kg^{-1}.min^{-1}): _____

Maximum Aerobic Power (W): _____

ESSA Screen (risk factors): _____

Consultation with general practitioner recommended? Yes □ *No* □

4

Lifestyle Profile

Do you consume caffeinated products? Yes □ No □

If YES, how often (i.e., products per day)? _____

Are you currently employed? Yes □ No □

If YES, what is your current profession? _____

If YES, Which best describes you employment status?

Full time □ Part-time □ Casual □ Seasonal □ Self-employed □

Do you currently undertake shift work? Yes □ No □

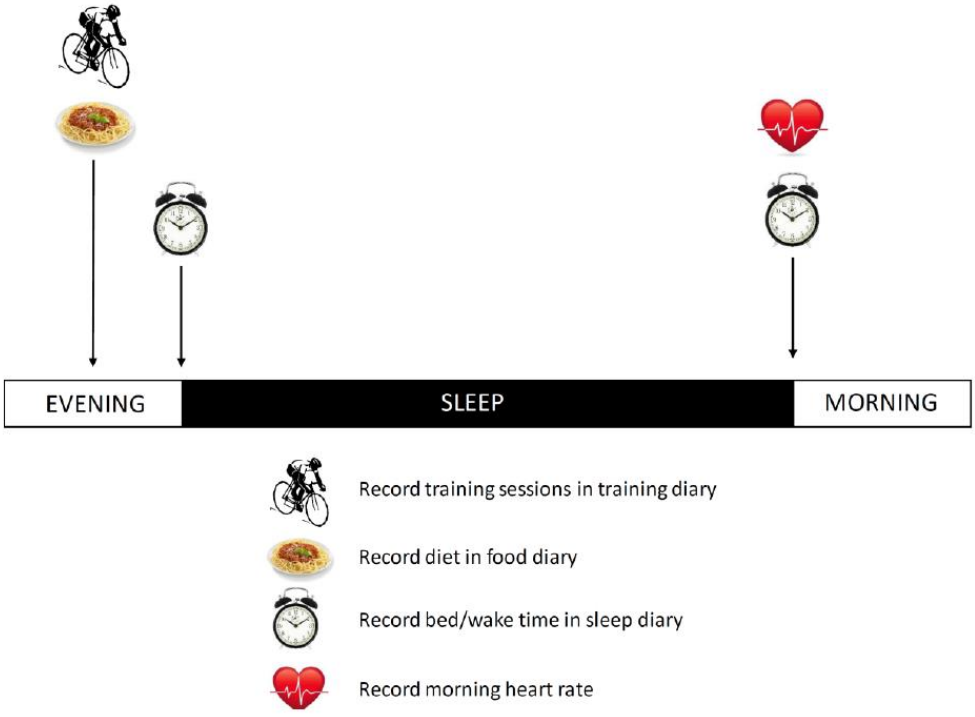
5

Data Collection

In addition to the data collected at Deakin University on testing days, participation in this study will require information to be recorded about your **(1) diet, (2) training**, and **(3) sleep** behaviours. It will also require frequent monitoring of resting **(4) morning heart rate**.

This information will be collected by YOU every day for the duration of the study, using equipment provided by the researchers and the diary included in this booklet. You will be educated on how to use the equipment before you commence the study. A diagram of your daily data collection is provided on the next page.

6



7

1. Diet

In order to control the effect of diet on the results, you are asked to replicate your diet in the 24 hours prior to, and during, the two days of each laboratory trial. To facilitate this you will record your daily food and drink consumption, including the quantity consumed. For example, if you eat five Weet-Bix for breakfast this should be recorded as; Weet-Bix (5). See below for an example of a full day's consumption.

	TIME	FOOD / DRINK (INCL. QUANTITY)
DIET	0700	Weet-Bix (5) w/ Milk (1 cup) & Orange Juice (1 cup)
	1000	Coffee (1 cup) & Muesli Bar (1)
	1300	Sandwiches w/ tomato lettuce, cheese, ham, wholemeal bread (2)
	1600	Banana (1) & Gatorade Sports Drink (600ml)
	1900	Stir-fry w/ beef, vegetables, rice (1 bowl or 2 cups cooked rice)

8

2. Training

As changes in training load may influence the results, you are asked to keep your weekly training loads consistent for the duration of the study. In addition, you are asked to refrain from exercise in the 24 hours prior to, and during, the four days of each laboratory trial (this excludes exercise undertaken as part of the study). To ensure these requirements are met you will record all training sessions throughout the study. You will record the time of day, exercise mode (e.g., cycling, swimming, running, resistance training), and duration (i.e., in minutes) of each session undertaken. In addition, you will rate the perceived exertion (RPE) required to complete each session. Please use the table on the next page as a guide when determining the RPE for each session.

9

RATING OF PERCIEVED EXERTION (RPE)	
0	**Rest** (e.g., sleeping)
1	**Really Easy** (e.g., watching TV, reading)
2	**Easy** (e.g., walking)
3	**Moderate** (i.e., comfortable but not easy exercise)
4	**Somewhat Hard** (i.e., heavy breathing but comfortable)
5	**Hard** (i.e., no longer comfortable)
6	**Very Hard** (i.e., uncomfortable, hard to breath)
7	**Really Hard** (i.e., very uncomfortable, short of breath)
8	**Really, Really, Hard** (i.e., extremely uncomfortable)
9	**Extremely Hard** (i.e., very difficult to maintain intensity)
10	**Maximal** (i.e., nothing left to give)

10

Below is an example of how you would record your training on a day that included a hard 90 minute bike ride in the morning (i.e., leaving at 6am), a somewhat hard 45 minute resistance training session at lunchtime (i.e., commencing at 1pm), and a light or moderate intensity run for 60 minutes in the evening (i.e., leaving at 7pm). It is not expected that you would undertake more than three training sessions in a single day, however, if you do, please speak to a researcher about how to record this.

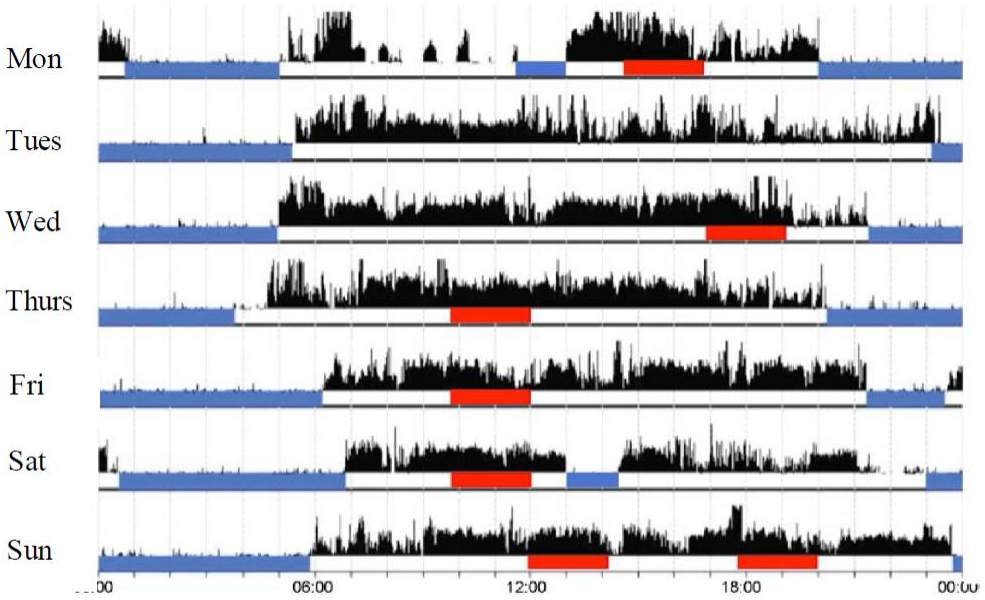
	TIME	MODE	DURATION	PERCIEVED EXERTION (RPE)
TRAINING	0600	Cycling	90 min	1 2 3 4 (5) 6 7 8 9 10
	1300	Resistance	45 min	1 2 3 (4) 5 6 7 8 9 10
	1900	Run	60 min	1 2 (3) 4 5 6 7 8 9 10

11

3. Sleep

To enable us to monitor your sleep, you will need to: (i) wear an activity monitor, and (ii) keep a sleep diary. The information obtained from the activity monitor and sleep diary can be combined to inform us about the quantity and quality of your sleep. The next page shows an example of seven days of data collected using an activity monitor and sleep diary. Each line represents one day of data, from midnight to midnight. The blue horizontal bars represent bedtimes as recorded in a sleep diary. The black vertical bars represent the level of activity as recorded by an activity monitor. This data is input into software that can provide quantitative information about your sleep (e.g., total sleep time, sleep efficiency etc.).

12



13

(i) Activity Monitor

An activity monitor is a device worn like a wristwatch that continuously records body movement. The monitor provided must be worn at ALL times. It is water resistant but not waterproof, so it must be taken off when swimming or showering. It is important that you always wear the activity monitor on the same wrist (non-dominant). If you take the monitor off don't forget to put it back on! Please take care of the monitor, replacement value = $3,000.

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(ii) Sleep Diary

The data collected by the activity monitor cannot be analysed unless we know the times you have been attempting to sleep. Therefore, you are required to complete a sleep diary. You will need to record the time you go to bed (i.e., attempting to sleep - not reading/watching TV) each night, as well as the time you wake up. It may be best to put your sleep diary and a pen next to your bed to ensure completion. Please note: you are encouraged not to nap during the day for the duration of the study. However, if you fall asleep accidently, you must record the time you were asleep in the diary. You are also asked to rate the quality of all sleep episodes according to the following 5-point scale:

15

RATING OF SLEEP QUALITY	
1	**Very Good** (i.e., no disruptions, feel energised)
2	**Good** (i.e., minimal disruptions, feel good)
3	**Average** (i.e., a few disruptions, still a little tired)
4	**Poor** (i.e., many disruptions, feel tired)
5	**Very Poor** (i.e., lots of disruptions, feel exhausted)

Below is an example entry for the sleep diary on a day where you accidently fell asleep – with a few disruptions - from 3pm to 4pm, before going bed for a good night's sleep from 10pm to 6am.

	BED-TIME	WAKE-TIME	WAS THIS A NAP?	SLEEP QUALITY
SLEEP	1500	1600	Yes ✓ No □	1 2 (3) 4 5
	2200	0600	Yes □ No ✓	1 (2) 3 4 5

16

4. Morning Heart Rate

You will be recording heart rate every morning for the duration of the study (21 mornings). The morning heart rate test (MHRT) is a 7-minute test that will be undertaken upon waking. The test requires you to lay still, and on your back for 7-minutes whilst recording your heart rate. It is important you do this immediately upon waking and do not undertake any activities prior to the test. This is to ensure that your heart rate reflects your true recovery status rather than any activity you have previously been undertaking (e.g., showering or eating etc.). You may want to have your HR monitor beside you bed so that when you wake up it is ready for you to put on. You will be provided with a Polar V800 HR monitor for the duration of the study.

17

Please follow the steps below to complete the MHRT:

1. Moisten the electrode area of the strap with water. You can do this by simply dipping you finger in some water and rubbing it on the electrodes (the harder plastic part of the strap). It may be helpful to sleep with a cup or drink bottle beside your bed.

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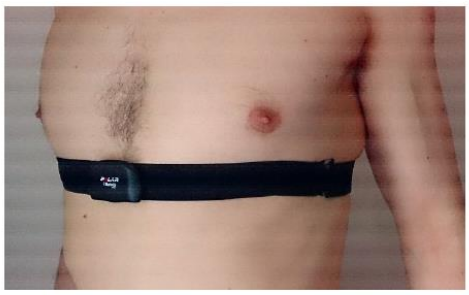
2. The sensor should remained attached to the strap at all times. However, if has been removed please make sure it is attached before you proceed.

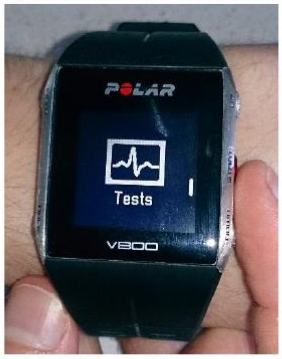
19

3. Fit the strap around your chest by sliding the hook through the loop. The strap should be positioned just below the chest muscles (i.e., 2-3cm below nipples), with the sensor directly above the sternum.



20

4. From the home screen of the watch press the 'down' button (bottom right) five times until the 'Tests' option appears. Select this using the red 'start' button. Press the down button three more times until the 'RR recording' test appears and select using the red 'start' button.



21

5. Press the red start button again to 'Start recording' your heart rate. The watch will momentarily search for your heart rate before the 'Heart rate found' screen appears. The test will automatically begin.

22

Note: If heart rate cannot be detected the screen will display 'Couldn't find heart rate'. If this occurs you may need to check that your heart rate strap is correctly positioned (see step 3) or you may need to apply more water to the electrode.

23

6. The time elapsed on the test will be displayed on the watch below 'RR recording'. You must remain completely still (except when checking the watch) and lying on your back for seven minutes. At the end of the test (i.e., when elapsed time reads seven minutes) press and hold the 'back' button (bottom left) for three seconds to end the test.

Elapsed time shown here

24

7. The screen will display 'recording ended' and a summary of the test will appear. Please note, the test duration should read at least 7-minutes. The test will be automatically saved so that it can be synced with Polar software when you visit Deakin University for your next laboratory session. Press 'back' to return to the home screen.

25

8. The diary that follows this instruction manual includes a question asking whether you recorded your heart rate upon waking. This serves as a reminder so that you don't forget to do the test. Please tick 'yes' when the test is completed.

HEART RATE	DID YOU RECORD HEART RATE FOR 7-MINUTES UPON WAKING?
	Yes ✓ No □

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Participant Diary

27

DATES:	to			DAY/NIGHT: 01
DIET	TIME	FOOD / DRINK (INCL. QUANTITY)		
TRAINING	TIME	MODE	DURATION	PERCIEVED EXERTION (RPE)
				1 2 3 4 5 6 7 8 9 10
				1 2 3 4 5 6 7 8 9 10
				1 2 3 4 5 6 7 8 9 10
SLEEP	BED-TIME	WAKE-TIME	WAS THIS A NAP?	SLEEP QUALITY
			Yes □ No □	1 2 3 4 5
			Yes □ No □	1 2 3 4 5
			Yes □ No □	1 2 3 4 5
HEART RATE	DID YOU RECORD HEART RATE FOR 7-MINUTES UPON WAKING?			
	Yes □ No □			