

Effects of Acute Sleep Restriction on Performance with Napping Interventions and Sleep Measurement Considerations

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Declaration

I declare that the work in this thesis was conducted in accordance with the regulations of Liverpool John Moores University. Apart from the help and advice acknowledged, the work presented is my own original work and it has not been written or assisted by artificial intelligence (AI) tools.

The thesis has not been presented to any other University for examination in the United Kingdom or overseas. No portion of the work referred to in this thesis has been submitted in support of an application for another degree or qualification of this or any other university.

Some chapters of this thesis have been published in peer-reviewed journals and, where this is the case, acknowledgements have been provided.

Abstract

Acute sleep restriction is associated with a host of negative effects on an individual's health and wellbeing. However, the extent in which these outcomes influence exercise performance, cognitive function and perceptual responses are still not fully understood. This thesis explores acute sleep restriction and evaluates the effectiveness of nap interventions in mitigating the consequences of sleep loss on performance. Chapter 1 introduces sleep restriction and outlines different interventions, such as daytime napping, that have been proposed to alleviate the impact of sleep loss on performance. Chapter 2, presents a mixed methods narrative review examining 33 studies that were extracted from the literature on acute sleep restriction and the effects on exercise types, cognitive domains and perceptual responses. The review highlights methodological inconsistencies such as sleep measurement, that limit comparability of findings between studies. Chapter 3 outlines the standardised testing protocols used throughout the experimental chapters, with further information on the equipment, protocols and techniques used were outlined. Chapter 4 explores the validity of consumer sleep devices and research grade actigraphy compared to polysomnography in the laboratory versus the home setting. The findings suggest that consumer devices have significant discrepancies when compared to polysomnography and are not yet suitable for research or clinical use. Collectively, Chapters 5, 6 and 7 evaluated the effectiveness of naps, with consideration for timing and duration of the nap session. Despite employing rigorous research designs such as dietary control and objective sleep assessment with polysomnography, post lunch naps did not improve afternoon submaximal or maximal performance when compared to a no nap condition. However, nap interventions were effective in improving cognitive function and perceptual responses, likely due to reducing homeostatic pressure, excessive fatigue and restoring alertness. Chapter 8 synthesises the findings from the 4 experimental chapters and collectively concludes that the effectiveness of nap interventions following sleep restriction is likely task dependent. While longer nap durations allow for more restorative benefits however the translation to performance enhancement depends on the performance type. Tasks requiring greater cognitive demand and attentional focus over longer bouts appear to gain greater benefits from naps following sleep restriction. The synthesis further highlights the importance of rigorous research designs to account for individual variability in sleep need and performance outcomes.

Publications and research dissemination

Journal articles:

Pullinger SA, Adhikari R, Bawari B, Pérez Armendáriz ML, Manathanath A, Bommasamudram T, **Gallagher C**, Bampouras TM, Edwards BJ. (2025). Sleep quality, behaviour, disorders prevalence and chronotype in elite junior and senior athletes. *Biological Rhythm Research*, 1–17. <https://doi.org/10.1080/09291016.2025.245383>.

Gallagher C, Austin V, Dunlop KA, Dally J, Taylor K, Pullinger SA and Edwards BJ. (2024). Effects of supplementing zinc magnesium aspartate on sleep quality and submaximal weightlifting performance, following two consecutive nights of partial sleep deprivation. *Nutrients*, 16(2), p.251. <https://doi.org/10.3390/nu16020251>.

Edwards BJ, Adam RL, Drummond D, **Gallagher C**, Pullinger SA, Hulton AT, Richardson LD, Donavan TF. (2024). Effects of an Acute Dose of Zinc Monomethionine Aspartate and Magnesium Aspartate (ZMA) on Subsequent Sleep and Next-Day Morning Performance (Counter Movement Jumps, Repeated Sprints and Stroop Test). *Nutrients*, 16(15), 2466; <https://doi.org/10.3390/nu16152466>.

Edwards BJ, Adam RL, **Gallagher C**, Germaine M, Hulton AT, Pullinger SA and Chester N. (2024). In individuals with adequate dietary needs who present no sleep disturbances, is an acute intake of Zinc Magnesium Aspartate following either two consecutive nights of 8 or 4-h sleep deprivation, beneficial for sleep and morning cognitive performance. *Behavioural Sciences*. 14(7), 622; <https://doi.org/10.3390/bs14070622>.

Munnilar M, Bommasamudram T, Easow J, Tod D, Varamenti E, Edwards BJ, Ravindrakumar A, **Gallagher C** and Pullinger SA. (2024). Diurnal variation in variables related to cognitive performance: a systematic review. *Sleep and Breathing*, 28(1), pp.495-510. <https://doi.org/10.1007/s11325-023-02895-0>.

Gallagher C, Green CE, Kenny ML, Evans JR, McCullagh GD, Pullinger SA and Edwards BJ. (2023). Is implementing a post-lunch nap beneficial on evening performance, following two nights partial sleep restriction?. *Chronobiology International*, 40(9), pp.1169-1186. <https://doi.org/10.1080/07420528.2023.2253908>.

Edwards BJ, **Gallagher C**, Pullinger SA, de Mello TM & Walsh NP. (2023). Athletic performance; effects of sleep loss. *The Encyclopedia of Sleep and Circadian Rhythms*, 2nd edition Edited by Clete A. Kushida, Elsevier. Pp.434-443, ISBN 9780323910941, <https://doi.org/10.1016/B978-0-12-822963-7.00249-8>.

Langan-Evans C, Hearn MA, **Gallagher C**, Long S, Thomas C, Moss AD, Cheung W, Howatson G and Morton JP. (2022). Nutritional Modulation of Sleep Latency, Duration, and Efficiency: A Randomised, Repeated-Measures, Double-Blind Deception Study. *Medicine and Science in Sports and Exercise*. <https://doi.org/10.1249/MSS.0000000000003040>.

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Chapter 1:

General Introduction

This chapter provides an overview on the importance of sleep and gives insight to the prevalence and effects of sleep loss on general and athletic populations. Napping interventions are introduced along with methods of sleep measurement that are currently employed in the field.

Introduction

Sleep is a fundamental behavioural state required by all human beings to maintain a healthy lifestyle and ultimately survive. It is defined as a reversible yet recurring process of reduced responsiveness to external stimuli, with marked reductions in muscle activity. Ultimately, sleep allows the brain and body to rest and undergo physiological adaptations (Ravindra and Kutty, 2012; Tester and Foss, 2018). The latest statistics for the United Kingdom, report 9 in 10 people are currently experiencing sleep issues (The Sleep Charity, 2024). Unfortunately, these figures span across the globe with data from the United States reporting that 1 in 3 Americans (approximately 84 million people) do not achieve recommended sleep guidelines (Gallup Report, 2022). A round-the-clock activity within society (a drift from the 09-17:00 hours [h] work schedules and changes in lifestyle habits) have contributed to an ever-growing proportion of the population who are neglecting sleep, resulting in greater prevalence of sleep complaints. This presents a major public health concern as there is strong evidence to support that inadequate sleep leads to a chronic inflammatory state and increases the risk of cardiometabolic, autoimmune and neurodegenerative diseases. Hence why sleep deprivation has been linked to 5 of the top 15 leading causes of death (Wells and Vaughn, 2012; Garbarino et al. 2021). To clarify, sleep deprivation/restriction can either be total sleep deprivation, when no sleep is achieved, or partial deprivation/restriction when total sleep time is less than required for functioning (i.e. $< 7 - 9$ h per night; Reynolds and Banks, 2010).

Latest statistics report that 74% of UK adults have experienced a decline in sleep quality over the past 12 months. With young adults (aged 35-44 years old) reporting the least sleep, where only 33% of this population achieve 7-8 h per night (Healthier Nations Index, 2022). It has been suggested that athletic populations require greater sleep quality and duration than recommended by 'The National Sleep Foundation' (NSF) guidelines. Where an individualised approach to sleep needs and demand may be more appropriate due to the individual and environmental factors that influence sleep (refer to Figure 1.1; Doherty et al. 2021). Despite this recommendation it is difficult to quantify this as you cannot simply prescribe greater sleep quality to an athlete as this term alone is made up of a combination of elements: Sleep efficiency (ratio of time spent asleep relative to time in bed); sleep latency (time taken to fall asleep after lights out); sleep duration (quantity of time a person sleeps) and wake after sleep onset (time spent awake after initially falling asleep). There are also subjective aspects of sleep quality including how an individual feels upon waking and their perception of the prior sleep period (Nelson et al. 2021). With the aim of further clarifying the term 'sleep quality' the NSF assembled a panel of experts to address "What

is good sleep quality?”. According to the NSF sleep continuity contributes to indicators of sleep quality with shorter latencies, fewer awakenings and reduced wake after sleep onset indicates good overall sleep quality. In regard to nap durations the literature is quite vague, however for adults those who nap for > 100 does not indicate good sleep quality (Ohayon et al. 2017). It is important to identify the specific sleep parameters that are affecting an individual’s overall sleep to ensure relevant recommendations are provided. For example, if wake after sleep onset is a concern, interventions need to be targeted at reducing nighttime awakenings to subsequently increase sleep duration and sleep quality. Similar to general populations of the same age and biological sex, 50-80% of elite athletes experience sleep disturbances, which may be attributed to training demands, scheduling of competitions and associated travel (Walsh et al. 2021). Certain populations such as athletes, appear to be more susceptible to sleep loss and the accumulation of reduced sleep which may lead to greater daytime fatigue, mood degradation, impaired memory and cognitive function. All factors that contribute to a reduction in training adaptations and recovery (Craven et al. 2022; Fullagar and Bartlett, 2016).

To understand the importance of sleep, one approach is to explore changes that happen due to lack of quality and quantity of sleep, at a physiological and behavioural level. The consequences of sleep loss are likely to have numerous aetiologies (Kujawa et al. 2020). Regarding physiological functions, sleep loss can negatively affect muscle strength and/or endurance. However, the results are conflicting dependent on the timing and duration of sleep restriction (late retiring or early awakening sleep restriction and chronic or acute) and type of exercise performed i.e. submaximal or maximal exercise (Reilly and Piercy, 1994). Studies that adopted ‘normal retiring and early rising’ sleep restriction protocols (retiring at 23:00 and waking at 04:00 h), report more detrimental effects on gross muscular strength and power compared to ‘late retiring and normal rising times’ (retiring at 03:00 and waking at 07:00 h (Souissi et al. 2013). In addition, tasks that require more ‘time on task’ or are of a repetitive nature, report greater detriments to performance (Brotherton et al. 2019). Previous studies investigating effects of sleep loss on cognitive components report hindered cognitive processing abilities and alterations in mood state, which may increase perceived effort and reduce motivation to complete the task (Axelsson et al. 2020). It has been previously hypothesised that as sleep loss progresses the homeostatic drive for sleep increases and there is greater variability in cognitive performance as more effort is required to perform. Although effects of sleep loss on cognitive performance are conflicting, executive function tasks have shown to be highly sensitive after only

one night of sleep deprivation (such as the Stroop test [Stroop, 1935] which evaluate word fluency, response time and decision making; McCarthy and Waters, 1997).

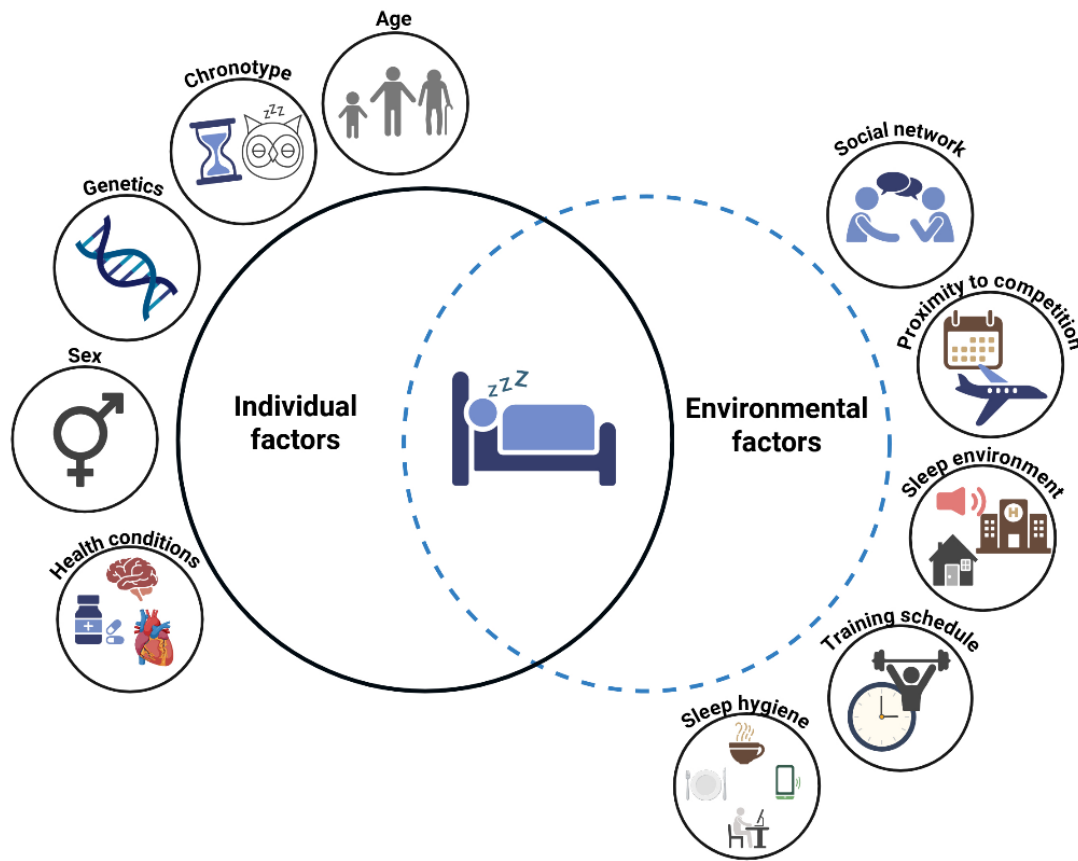


Figure 1.1. Venn diagram outlining the individual and environmental factors that influence sleep.

Partial sleep restriction is sometimes unavoidable, i.e. due to training schedule or overseas travel for competition (Davenne, 2009). Therefore, it is important that interventions to alleviate effects of sleep loss are explored, to assist these individuals and reduce the impact on performance. An intervention that is commonly implemented in athletic and general populations are ‘naps’ as they are non-invasive, have no associated cost and can be time effective (Saletin et al. 2017). Naps are typically distinguishable from nocturnal sleep because of the composition of the physiological sleep stages that occur. Yet this is dependent on the nap duration and age of the individual. In addition, naps that proceed a period of sleep restriction or deprivation differ architecturally to naps following normal nocturnal sleep (Mantua and Spencer, 2017). Previous studies have shown that a nap of 30 minutes (min) can reduce the homeostatic drive for sleep and improve alertness, memory and emotional regulation for 2 hours

following termination of the nap (Stickgold, 2005). Although naps may restore alertness and functioning following sleep restriction, the long-term effects of these benefits are unknown. The effectiveness of a nap can also be influenced by sleep inertia, which can impair performance, and is typically more present following naps of longer durations (> 30 minutes) as it is exacerbated when waking from deeper stages of sleep (Hilditch et al. 2017a). Sleep inertia is a state of impaired cognition that can make an individual feel groggy and disorientated upon waking (Trotti, 2017). It can be influenced by prior wakefulness therefore sleep restriction may exacerbate sleep inertia immediately after waking. Previous studies suggest that it can take up to 60 min for effects of sleep inertia to dissipate therefore longer naps should only be employed if the individual has sufficient time to recover post nap (Hilditch and McHill, 2019).

To assess for sleep disturbances and potential disorders, an objective measurement of sleep is necessary. In recent years there has been significant advances in technology for sleep assessment with the aim of producing devices that are more consumer friendly, unobtrusive and can be used in the home setting (Scott et al. 2020). Wearables and nearables are available on the market in a variety of forms from mattress devices, rings such as Oura or Go2Sleep ring and watches such as FitBit, WHOOP or Garmin. Despite the attractiveness of these products, they often under or overestimate sleep metrics due to lack of sensitivity, when compared to research grade devices and gold standard polysomnography (PSG; Crivello et al. 2019). Performing polysomnography provides measurement of brain electrical activity (electroencephalography; EEG), eye movement (electrooculography; EOG), muscle activity (electromyography; EMG) and cardiac activity (electrocardiography; ECG), which collectively are essential for detection of sleep stages and multiple physiological variables. Hence the importance of performing PSG particularly for diagnosis of sleep disorders (Kline, 2020). As mentioned previously, the growing popularity of consumer devices is predominately due to the ease of use and accessibility. To conduct PSG a qualified sleep technician is required. It is typically performed in a laboratory or clinical setting therefore it may be difficult to access this service and is often expensive (Mitchell and Werkhaven, 2020). Therefore, it is important to bridge the gap between consumer and research validated devices to ensure a thorough assessment can be conducted. Other factors should be considered such as comfort of the participant and the possibility of developing devices that can be used in the home environment, where individuals are likely to sleep better due to the familiarity (Kelly et al. 2012).

Aims and Objectives

The primary aim of this thesis is to provide greater insight into acute sleep restriction and the effects on physical performance, cognitive function and perceptual responses through a mixed model review of the topic and original investigations. These findings will help to provide practical recommendations and interventions that can be implemented, when experiencing acute sleep loss. The experimental studies will assess different durations of sleep restriction and explore the potential dose response between 3 versus 4 h of sleep restriction on subsequent performance. Optimal timing and duration of nap interventions will be evaluated as well as the factors that contribute to the effectiveness of a nap. In addition, a further aim was to explore methods of sleep measurement, from gold standard polysomnography compared to research grade devices and consumer products (wearables and nearables), with consideration for sleep environment i.e. home environment versus the laboratory.

The aims of Chapter 2 were to review the existing literature on acute sleep restriction and the effects on exercise performance, considering the secondary impacts on cognitive function and perceptual responses. Based on the findings from the systematic search the aim was to discuss important methodological considerations that are often overlooked when conducting research in sleep and chronobiology and the relevance for future research.

The aims of Chapter 3 were to provide in-depth information on the participants, procedures, methods and equipment used throughout the experimental chapters. This section avoids unnecessary repetition in each chapter as a lot of the methods were replicated across the studies.

The aims for Chapter 4 were to investigate the validity of polysomnography compared to surrogate sleep devices during an 8-h sleep opportunity. A further aim was to assess the potential difference of sleep when assessed in the laboratory versus the participants home setting.

The aim of Chapter 5 was to: (1) determine the physiological effects on muscle strength measures following partial sleep restriction (4 h sleep per night, over two consecutive nights) and the psychological effects related to changes in mood state, cognitive function, intra-aural temperature and subjective responses. (2) to investigate the effectiveness of a 30 versus 60-min nap at 13:00 h and establish whether a nap would improve evening submaximal weightlifting performance compared to no nap.

The aims of Chapter 6 were to investigate the dose response between 3 versus 4 h of sleep restriction for two consecutive nights and effects on exercise performance, cognitive function and perceptual responses.

The aims of Chapter 7 were to determine the mechanisms of action of a nap regarding temperature regulation and sleep architecture, when prior sleep is restricted to 3 h per night for two consecutive nights. A further aim was to investigate the effectiveness of a nap opportunity of 30 or 60 min opposed to no nap opportunity (yet remaining awake in the same environment) at 13:00 h on cognitive function pre nap, post nap and 45 minutes post nap. We explored the diurnal responses of hunger ratings, mood state and perceptual responses as well as submaximal weightlifting performance at 17:00 h.

The aims of Chapter 8 were to address the original research questions and integrate the findings from all previous chapters, providing a summary of the overall outcomes. A further aim was to reflect on the research as a whole, discuss potential limitations and outline future directions for this field based on the results.

Chapter 2:

Mixed methods literature review exploring the effects of partial sleep restriction on exercise performance

This chapter provides a systematic review of the literature in relation to acute sleep restriction and the effects on exercise performance, in addition to time-of-day effects on cognitive function and perceptual responses. Practical recommendations and future considerations for the research field are discussed.

2.1 Background

It is often emphasised that as humans we spend approximately one third of our lives asleep, yet the exact mechanisms that regulate the sleep-wake cycle are still not fully understood. Sleep is an essential process comprised of complex physiological functions that contribute to memory, metabolic functions, emotional regulation and cellular maintenance (Vyazovvskiy, 2015). Despite variations in sleep patterns throughout history, it has long been known that the timing and duration of sleep can have significant impacts on an individual. In the past two decades we have witnessed large shifts in society and technological advancements that have contributed to a decrease in average sleep durations (Keyes et al. 2015; George et al. 2024). There has been a growth of research on the effects of insufficient sleep on health yet the number of individuals reporting poor sleep is still on the rise. A recent survey on the UK population found that 1 in 20 are unaware of the link between poor sleep and health problems (The Sleep Charity, 2024) despite public health costs estimated at £40 billion a year as a result of sleep loss. Even acute bouts of sleep restriction, often referred to as a short-term reduction in sleep duration < 7 hours (h) lasting one to several consecutive nights, can result in negative consequences. These may include neurobehavioral deficits, changes in hormone regulation and impaired immune response (Besedovsky et al. 2012; Fullager et al. 2015; Walsh et al. 2021).

Acute sleep restriction is often unavoidable, whether it be due to travel, work schedules, health or social commitments (Reynolds and Banks, 2010). Athletic populations are often more prone to sleep loss because of early morning and late-night training schedules, as well as regular travel for training camps and/or competition. High training volumes and the associated stressors that accompany being an athlete have shown to significantly increase the likelihood of injuries, overtraining and reduced performance output; hence the importance of prioritising optimal sleep (Bonnar et al. 2018; Watson et al. 2020). Our understanding on the direct impact of sleep loss on exercise performance has expanded in recent years. However, findings often vary even when assessing the same exercise modality in similar populations (Craven et al. 2022). There is also a lack of studies that incorporate participants from recreational through to elite level athletes, which poses a significant gap in the research. Individual differences in sleep exist due to age, genetics and environmental factors which highlights the need for studies to encompass a broad range of individuals and exercise types (Van Dongen et al. 2005). A common challenge amongst researchers is to reduce bias, the main biases in individual studies are “bias in measurement of the outcome” and “bias in selection of the reported result”, with a lack of large and well powered studies. It is recommended that future studies conduct an A-priori test prior to recruitment to achieve the estimated

sample size but also account for participant dropouts (Edwards et al. 2025). The absence of a sample size estimation may lead to underpowered data, making it difficult to gather meaningful conclusions from the result. Overall, these factors pose challenges for practitioners and coaches to draw clear conclusions on the effects of sleep loss on sport specific performance.

The purpose of this review is to evaluate the research to date and provide an overview on the effects of acute sleep loss on physical performance, with insight on specific exercise types. The initial plan for this review was to conduct a systematic review and meta-analyses of the current literature. However, due to multiple reviews being published in recent years in this topic area (i.e. Fullager et al. 2015; Craven et al. 2022; Hatia et al. 2024; Kong et al. 2025) another review would not contribute anything further to the literature. The mixed methods approach was therefore adopted to conduct an extensive systematic search of the literature and extract the relevant data. The reviews will discuss other factors that influence performance such as psychological factors, cognitive function and time-of-day effects. In order to critically discuss the current knowledge on acute sleep restriction and physical performance an extensive computerised literature search was conducted on 4 electronic databases: PubMed, Cochrane Trials, Web of Science and Scopus. The databases were searched from their inception until December 2024, with no language restrictions using the Boolean expression: (Sleep* OR intervention* OR partial sleep deprivation OR sleep restriction OR total sleep deprivation OR sleep loss OR insufficient sleep) AND (Exercise OR performance test OR physical activity OR aerobic OR anaerobic OR competition OR strength) AND (PSG OR polysomnography OR actigraphy OR wearable OR nearable OR actimetry) AND (Health OR recreational OR active OR young OR male OR female OR athlete). The star symbol (*) was used to capture derivatives of the search terms. Two investigators (CG and BE) independently screened the potential texts. All study characteristics are outlined in Table 2.1. All search outputs were exported into Endnote Citation Manager (Version 20, Philadelphia, USA). Additionally, we manually sourced articles from reference lists of original manuscripts and previous reviews.

Table 2.1: Summary of study characteristics.

Author and year	Participants characteristics			Methodological considerations			Performance type
	Sex	Age (years)	Fitness classification/Sport type	Standardised dict/sample size estimation	Familiarisation prior to testing	Sleep conditions	
Abdelmalek et al. 2013	12M	21.2 ± 1.2	Insufficient information*/Football	No/No	No	Control versus PSD (4.5 h)	Anaerobic
Ajjimaporn et al. 2020	11M	20.0 ± 1.0	Tier 2: Trained/Football	Yes/Yes	No	Control versus PSD (3.5 h)	Anaerobic
Baati et al. 2020	10M	22.8 ± 1.3	Tier 2: Trained/Soccer	No/No	Yes	Control, PSDBN (3 h), PSDEN (4.5 h)	Anaerobic
Brotherton et al. 2019	16M	22.0 ± 1.6	Tier 1: Recreationally active/Not specified	No/No?	Yes	Control, versus PSR (3 h)	Strength
Chase et al. 2017	6M, 2F	24.0 ± 7.0	Tier 1: Recreationally active/Cyclists	Yes/No	Yes	Control versus PSR (3 h)	Anaerobic & Strength
Cook et al. 2012	16M	20.9 ± 0.9	Tier 3: Highly trained, national level/Rugby	Yes/No	Yes	Control versus PSD (< 6 h)	Strength
Cullen et al. 2019	10M	27.0 ± 6.0	Tier 1: Recreationally active/Not specified	Yes/No	Yes	Control, versus PSD (4 h)	Anaerobic & Strength
Daaloul et al. 2019	13M	23.0 ± 2.0	Tier 3: Highly trained, national level/Karate	Yes/No	Yes	Control versus PSR (4 h)	Anaerobic, endurance
Dean et al. 2023	10M	29.9 ± 10.7	Tier 2: Trained/Cycling	Yes/No	Yes	Control versus PSR (3 h)	Anaerobic & Strength
HajSalem et al. 2013	21M	19.1 ± 1.2	Not specified*/JudoKas	No/No	Yes	Control versus PSDEN (4.5 h)	Anaerobic & Strength
Khemila et al. 2021	12M	20.3 ± 2.0	Tier 1: Recreationally active/ Not specified	Yes/Yes	Yes	Control versus PSD (4.5 h)	Anaerobic
Mejri et al. 2014	10M	17.6 ± 0.5	Tier 3: Highly trained, national level/Taekwondo	Yes/No	Yes	Control, PSDEN, PSDBN (3 h)	Anaerobic

Table 2.1 continued.

Author and year	Participants characteristics			Methodological considerations			Performance type
	Sex	Age (years)	Fitness classification/Sport type	Standardised diet/sample size estimation	Familiarisation prior to testing	Sleep conditions	
Mejri et al. 2016	10M	17.6 ± 0.5	Tier 3: Highly trained, national level/Taekwondo	Yes/No	Yes	Control, PSDBN (4 h), PSDEN (3 h)	Anaerobic
Mougin et al. 1996	8M	19.2 ± 1.8	Highly trained*	No/No	No	Control versus PSD (4 h)	Anaerobic
Mougin et al. 2001	8M	24.0 ± 0.8	Highly trained endurance athletes*	No/No	No	Control versus PSR (4 h)	Anaerobic
Omiya et al. 2009	16M	21.5 ± 2.6	Tier 1: Recreationally active/Not specified	No/No	No	Control versus PSR (< 3 h)	Anaerobic
Paryab et al. 2021	33M	20.0 ± 2.0	Insufficient information	Yes/No	No	Control versus PSD (4 h)	Anaerobic & Balance
Reilly and Deykin, 1983	8M	18-26 years	Insufficient information*/Various sports	No/No	Yes	Control versus PSD (2.5 h)	Aerobic & Strength
Roberts et al. 2019	9M	30.0 ± 6.0	Tier 2: Trained/Cycling	Yes/No	Yes	Control versus PSR (30% reduction)	Aerobic
Romdhani et al. 2019	9M	18.5 ± 0.9	Tier 2: Trained/Judokas	Yes/No	Yes	Control, PSDEN, PSDBN (4 h)	Anaerobic
Romdhani et al. 2020	9M	18.8 ± 1.1	Tier 3: Highly trained, national level/Judokas	Yes/No	Yes	Control versus PSD (4.5 h)	Anaerobic
Romdhani et al. 2021	9M	18.8 ± 1.1	Tier 2: Trained/Judokas	Yes/No	Yes	Control versus PSD (4 h)	Anaerobic
Sinnerton and Reilly, 1992	5M, 3F	19-28 years	Insufficient information*/Swimming	No/No	No	Control versus PSD (2.5 h)	Anaerobic & Strength
Souissi et al. 2008	11M	21.7 ± 1.3	Tier 1: Recreationally active/Not specified	Yes/No	Yes	Control, PSDBN, PSDEN (4 h)	Anaerobic & Strength

Table 2.1 continued.								
Author and year		Participants characteristics		Methodological considerations			Performance type	
	Sex	Age (years)	Fitness classification/Sport type	Standardised diet/sample size estimation	Familiarisation prior to testing	Sleep conditions	Method of sleep measurement	
Souissi et al. 2013	12M	18.6 ± 2.4	Tier 2: Trained/Judokas	Yes/No	Yes	Control (7.5 h), PSDBN, PSDEN (3 h)	Monitored by researchers	Anaerobic & Strength
Souissi et al. 2015	14M	23.6 ± 2.0	Insufficient information*/Football	Yes/No	No	Control (8.5 h) versus PSD (4.5 h)	Actigraphy	Anaerobic & Power
Souissi et al. 2018	12M	20.7 ± 1.2	Tier 1: Recreationally active/Not specified	Yes/No	No	Control (8.5 h) versus PSD (4.5 h)	Actigraphy	Anaerobic
Souissi et al. 2013	12M	18.6 ± 2.4	Tier 2: Trained/Judokas	Yes/No	Yes	Control, PSDBN, PSDEN (3 h)	Monitored by researchers	Anaerobic & Strength
Souissi et al. 2015	14M	23.6 ± 2.0	Insufficient information*/Football	Yes/No	No	Control versus PSD (4.5 h)	Actigraphy	Anaerobic & Power
Souissi et al. 2018	12M	20.7 ± 1.2	Tier 1: Recreationally active/Not specified	Yes/No	No	Control versus PSD (4.5 h)	Actigraphy	Anaerobic
Souissi et al. 2020	19M	20.5 ± 1.5	Tier 1: Recreationally active/Not specified	Yes/No	Yes	Control versus PSD (5 h)	Actigraphy, sleep diaries and monitored by researchers	Anaerobic
Sweeney et al. 2020	19M	25.0 ± 0.8	Tier 1: Recreationally active/Not specified	Yes/Yes	Yes	Control versus PSD (4 h)	Actigraphy and time stamped	Anaerobic
Vardar et al. 2007	13M	22.0 ± 1.1	Tier 1: Recreationally active/Not specified	Yes/No	Yes	Control versus PSD (4 h)	Monitored by researchers	Anaerobic
PSD: Partial sleep deprivation; PSR: Partial sleep restriction; Control: > 7 h sleep per night; PSDBN: partial sleep deprivation at the beginning of the night; PSDEN: partial sleep deprivation at the end of the night; *study did not provide sufficient information to classify using the participant classification framework.								

2.2 Fundamentals of sleep

Many theories have been proposed to explain *why* we sleep including the inactivity, energy conservation and restoration theories. Prior findings support each of these theories and it is believed that no one single process is responsible for the role of sleep. To develop a greater understanding of the biological mechanisms of sleep, the two-process model was first introduced by Borbely (1982) to provide a deeper understanding of sleep/wake regulation. This model discusses the interaction between two oscillatory processes whereby the homeostatic component (Process S) interacts with a circadian component (Process C). Process S increases during wakefulness, resulting in a rise in sleep propensity and only dissipates once an individual retires to sleep (Landolt, 2008; Rempel et al. 2010). Whereas Process C refers to the body's internal 24-h biological clock which regulates both physiological and behavioural processes. Melatonin is a key biological marker of circadian phase. The transition from wake to sleep occurs when Process S approaches the upper threshold of Process C to induce sleep, with sleep to wake occurring when Process S reaches the lower threshold to trigger waking (Bailey et al. 2018). Therefore, when Process C is misaligned with Process S, due to scenarios such as sleep restriction or jet lag, difficulties initiating and maintaining sleep can arise.

Importantly, circadian timing not only influences sleep propensity but also mood and performance cycles. Chronobiology research highlights that daily rhythms of mood state and cognitive functioning are typically higher during the day and more vulnerable to impairments at night (i.e. past midnight). The period between 02:00-04:00 h is typically referred to as the 'devil hours' where awakening during this window can result in feelings of disorientation, partly because this time coincides with REM sleep where heart rate and body temperature reach their lowest values.

In healthy individuals relatively predictable patterns are observed, with cyclical transitions between non-rapid eye movement (NREM) and rapid eye movement (REM) sleep stages. Across the night an individual will typically have 4-5 sleep cycles each lasting approximately 90 mins (Carskadon and Dement, 2005). The interplay between sleep stages is crucial to obtain optimal sleep as each stage serves an important role, as outlined below. Therefore, irregular cycling or absence of particular sleep stages are often a consequence of disordered sleep.

When an individual retires to sleep, they will generally enter non rapid eye movement (NREM) stage 1 sleep and progress to NREM 2 and 3 before transitioning to rapid eye movement (REM) sleep. It is important to note that these sleep stage transitions are not linear and can be rather variable and dynamic throughout the night. It is relatively common for the brain to shift between lighter and deeper stages of sleep or briefly awaken before re-

entering sleep throughout the night. For example, after REM sleep, an individual may return to NREM2 opposed to NREM3 or restarting from NREM1 (Patel et al. 2024). As the brain transitions from NREM stages 1-3, heart rate and core body temperature begin to decrease. This transition facilitates unique restorative processes such as cellular repair, tissue regeneration and immune function (Le Bon, 2020). In the second half of the night, REM sleep is most prominent and despite only constituting around 20-25% of total sleep, it is fundamental for memory consolidation and emotional regulation.

Disruption to sleep architecture due to factors such as the amount of prior sleep (i.e. deprived state), restriction of caffeine consumption or noise and light exposure can negatively impact an individual's sleep quality and daytime functioning (Patel et al. 2024). In regard to caffeine, the effects on sleep are time and dose dependent, with consumption later in the day increasing the chance of delayed sleep onset and nocturnal sleep disturbance.

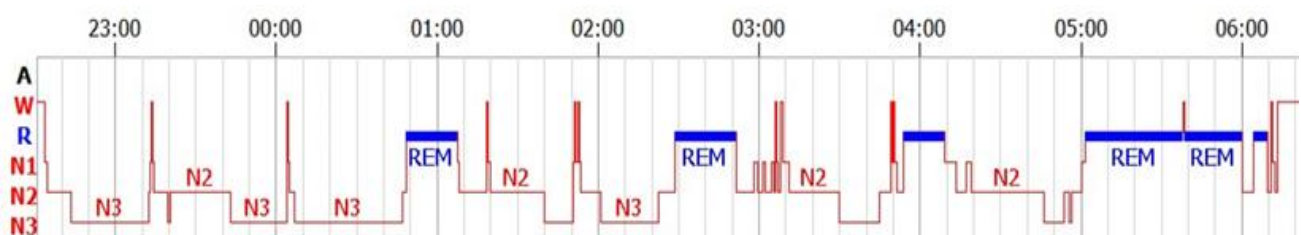


Figure 2.1: Example of a hypnogram from a representative participant, showing sleep stage transitions across an 8-hour overnight polysomnography recording.

2.3 Sleep guidelines for general and athletic populations

It is broadly understood that a ‘good night’s sleep’ is critical to sustain overall health and wellbeing, however the amount of sleep required differs between individuals (Mukherjee et al. 2015). The National Sleep Foundation recommends 7 to 9 h of sleep per night to achieve optimal sleep. Although these guidelines are useful as a benchmark, they do not take into consideration the influence of individual needs (such as sleep restriction due to training late into the night) or emotional state (the night before a big competition). Younger adults and athletic individuals are cohorts that are encouraged to achieve towards the upper limit of this recommendation as they

typically possess poor sleep patterns. In younger populations it may be greater screen use that contributes to poor sleep hygiene (Owens et al. 2014). For athletes it is proposed there is a greater sleep need due to higher physical demands and a greater requirement for physiological and psychological recovery (Fullager et al. 2015). Despite these recommendations there has been a concerning decline in sleep duration amongst the general population, especially in the last 20 years likely in line with societal changes. Insufficient sleep appears to be a global issue and is now considered a ‘public health epidemic’ with more than one third of adults in the United States and 43% of the UK population sleeping less than seven hours per night (The Sleep Charity, 2024; Chattu et al. 2018). These concerning statistics are also prominent amongst athletic populations, with poor subjective sleep quality and greater sleep disruption being commonly reported across a range of sports (Gupta et al. 2017). Athletes that engage in individual sports are reported to obtain less sleep than team sport athletes which may be attributed to greater performance pressure and pre-competition anxiety. Individual sports such as swimming and triathlon typically have demanding training schedules with early rising times that may restrict their sleep opportunity compared to team sport athletes (Sargent et al. 2014; Lastella et al. 2015; Sargent et al. 2021).

2.4 Measurement of sleep

Sleep can be assessed by behavioural observation, physiological monitoring or subjective self-reporting via questionnaires and sleep diaries. Both objective and subjective methods of assessment have been extensively validated, however when they are employed simultaneously in research studies there are often disparities (Mendonça et al. 2019). Objective methods provide more robust values of key sleep variables such as sleep duration, sleep efficiency and wake bouts. However, this does differ dependent on the objective method as some sleep measurement devices still have poor accuracy when trying to obtain sleep variables (i.e. consumer device compared to research grade actigraphy). Whereas subjective measures are unable to capture this information and are largely influenced by emotional state. There are advantages and disadvantages to objective and subjective measures therefore it is crucial both approaches are employed to reduce the gap in understanding and provide a comprehensive overview of an individual’s sleep. The gold standard method of measurement is polysomnography (PSG) and incorporates multiple skin electrodes to record brain activity (electroencephalogram), eye movements (electrooculogram), muscle tone (electromyogram), electrocardiogram and leg movements (Rundo and Downey, 2019). This array of measures provides a thorough overview of an individual’s sleep and is therefore typically used for diagnostic purposes. Following a PSG recording trained individuals interpret the data and produce a

hypnogram (refer to Figure 2.1) which is a graph that represents the sleep stages that occur across the sleep duration. For ease of use and field studies, wearables devices such as actigraphy are often adopted as they have been extensively validated in the research field and provide values comparable to PSG (Berryhill et al, 2020; De Zambotti et al. 2024).

2.5 Sleep restriction

Sleep restriction, also referred to as sleep debt, is characterised by a reduction in sleep duration below the normal amount required to feel rested and maintain functioning (Reynolds and Banks, 2010). This is unlike total sleep deprivation, where no sleep is taken over a period of 24 h or more. Sleep restriction typically alters an individual's sleep architecture, however the changes to each sleep stage differ dependent on the timing and duration of SR (acute or chronic). Interestingly, some sleep stages are conserved or completely diminished, while others intensify or occur sooner (Elmenhorst et al. 2008).

Given the restorative processes of sleep, it is critical for athletic populations to optimise sleep opportunity and have an aligned inner body clock as this will determine optimal performance. Recent findings report that athletes are more prone to achieving less sleep due to shorter time in bed and long sleep latencies than general populations. A recent study assessed subjective sleep responses of 202 elite Indian athletes and reported that 59% of athletes state they have poor sleep quality (Pullinger et al. 2025). Similarly, Juliff and colleagues (2015) studied a cohort of 283 elite Australian athletes and found that 64% experience poor sleep quality. The reasons associated with poor sleep were training schedules (up early and late to bed), travel requirements (for training and competition), psychological stress and high training volumes. In addition to these factors', individual differences such as chronotype can impact an athlete's ability to adapt to the varying training and competition schedules.

The variety of exercise modalities, physiological differences and impact of competing in team based versus individual sport warrants more research in elite athletes to further understand the prevalence and associated impacts on sleep. It is also important to highlight that recruiting elite athletes is very difficult hence why most of the research is conducted in recreationally active/trained individuals, which may not replicate professional athletes. The following sections will review the effects of acute sleep restriction on exercise performance with consideration for cognitive and psychological responses.

2.5.1 Sleep restriction and exercise performance

Among the first studies to investigate the interaction between partial sleep restriction and exercise performance they consistently report no effect when compared to a ‘control’ condition (normal sleep) for measures of strength, anaerobic threshold and aerobic capacity (Reilly and Deykin, 1983; Sinnerton and Reilly, 1992; Mougin et al. 1996). Each of these studies recruited trained individuals from a variety of sports, however they employed different sleep restriction protocols. Mougin and colleagues (1996) employed 1 night of 4 h sleep restriction (03:00-07:00 h) whereas the other two studies employed 3 consecutive nights of 2.5 h sleep (03:00-05:30 h; Reilly and Deykin, 1983; Sinnerton and Reilly, 1992). The study concluded that short duration and higher intensity exercise bouts (i.e Wingate or repeated sprints) were maintained as the exercise stimulus provided sufficient arousal to counteract the influence of sleep loss. Task duration is thought to be a significant factor following SR with less attentional lapses and greater performance when task duration is shorter as less cognitive effort is required (Wüst et al. 2024). Despite the lack of effect for experimental conditions, when performance was conducted at different time points there was a significant time-of-day effect with greater performance in the afternoon (17:00 h) compared to morning (06:30 h; Sinnerton and Reilly, 1992). Differences were attributed to the diurnal peak in temperature in the evening. A few years later Mougin and colleagues (2001) conducted another study, with a 4-h delayed SR protocol (03:00-07:00 h) for 1 night as well as an early awakening sleep restriction protocol (22:30-03:00 h; PSDBN). They employed a time to exhaustion test on a cycle ergometer and reported reductions in maximal work rate for delayed bedtime and the early awakening condition (Table 2). Researchers proposed that reduced cortisol concentrations 30 minutes after maximal exercise reflects the heightened fatigue individuals experience under sleep restriction conditions (Mougin et al. 2001). Post exercise values were significantly lower compared to normal sleep conditions for early awakening and delayed bedtime protocols (MD: 6.4 and 5.0 ng.100 ml⁻¹, respectively; $P < 0.05$). Although exercise may induce general stress responses such as high concentrations of cortisol and growth hormone, the direct interaction between this response and sleep loss studies remains inconclusive. Hormone secretion is dependent on an array of factors (intensity of stimulus, time of day hormone is measured) and have high circadian components therefore outcomes may differ dependent on the protocol.

The contrast in findings amongst the literature remains, with more recent studies reporting impaired Wingate performance with reduced peak power (PP) and mean power (MP) output by 2-11% (HajSalem et al. 2013; Soussi et al. 2013; Souissi et al. 2018; Khemila et al. 2021). Performance decline was more pronounced following early

awakening restriction opposed to delayed bedtime. A prolonged waking period increases neuronal activity that drives sleep homeostasis and increases sleep propensity (Landolt, 2008). These studies suggest that perceptual changes such as reduced positive mood state and motivation, result from sleep restriction. Both mood state and motivation are critical when performing tests such as an all-out effort Wingate as these factors can impact an athlete's ability to perform optimally during both training and competition (Axelsson et al. 2020). Interestingly strategies such as listening to motivational music when performing a task have shown to be effective at increasing an athlete's perception of confidence and arousal; resulting in enhanced performance at different times of day even under conditions of SR (Khemila et al. 2021). This highlights the importance of mood state monitoring in conjunction with physiological tests, particularly following sleep restriction when an individual may be more irritable (Bolin, 2019). To add, small sample sizes and individual differences such as participant fitness level were noted as potential contributing factors. The remaining studies from our literature search report no change in PP or MP during the Wingate test when compared to a control condition (Vardar et al. 2007; Souissi et al. 2008; Abdelmalek et al. 2013; Souissi et al. 2015; Paryab et al. 2021). However, of these five studies, three did not consider time-of-day effects and solely conducted morning testing which is consistently shown to be unaffected following sleep restriction (Souissi et al, 2008; Souissi et al. 2015; Paryab et al, 2021). In terms of the re-test reliability of Wingate tests they have been validated as a reliable tool for measures of PP and MP in healthy individuals (Hachana et al. 2012). Differences between study protocols such as prior familiarisation, timing and duration of sleep restriction, difference in training status and underpowered samples make it difficult to confirm the effect on anaerobic type performance.

Our literature search identified nine studies investigating muscle strength, with females only representing 3% of the participant populations. This alarming underrepresentation highlights a significant sex imbalance in the existing literature which raises concerns regarding the generalizability of the findings. It is crucial to address this disparity to ensure future studies are inclusive and reflect the broader population. Among these nine studies, strength was measured through resistance exercise (i.e. bench press, back squat), handgrip or maximal voluntary contractions (MVC). Overall, these studies indicate that acute SR has a negative influence on strength outcomes (see Table 2). The findings are less apparent for maximal handgrip with three studies reporting no difference between experimental conditions or only reporting significance for left handgrip (non-dominant hand) values (Reilly and Deykin, 1983; Sinnerton and Reilly, 1992; HajSalem et al. 2013; Cullen et al. 2019). It appears that

short term gross-muscular efforts remain intact under sleep loss. The results also highlight that submaximal strength measures were predominately affected when performance was isolated to the afternoon/evening (Reilly and Deykin, 1983; Sinnerton and Reilly, 1992; Souissi et al. 2013; Chase et al. 2017; Brotherton et al. 2019; Ajjimaporn et al. 2020). The two studies that measured strength in the morning reported no change in peak torque, handgrip or MVC values between 09:00-11:00 h (Chase et al. 2017; Souissi et al. 2013). The time-of-day effect observed in submaximal strength is linked to an increase in homeostatic pressure, which adversely affects the circadian rhythm and muscular performance in the evening (Souissi et al. 2013; Brotherton et al. 2019). Diurnal changes are also more prominent when the cognitive component of the task is greater. For example, submaximal exercise involves longer time on task and a high motivational component as the individual must perform repeated bouts opposed to singular maximal efforts (Fullager et al. 2015). These physiological responses may also be attributed to greater ratings of perceived effort (RPE), yet these theories remain unclear. Fifteen of the studies included in our search did measure RPE, however nine reported no differences between conditions. Despite the widespread use of Borg's RPE scale to assess exertion and effort, particularly during exercise interventions, the single item response is often said to oversimplify the rating. It is on a 6-20 scale with 6 being no exertion and 20 classified as maximal exertion. Although used interchangeably definitions of exertion and effort slightly differ with previous literature reporting different responses when participants were asked to report them separately (Hutchinson and Tenenbaum, 2019). Individual perception of effort and exertion can be largely variable, and caution should be taken when comparing RPE responses across studies. Hence, further research is needed to assess the interaction between performance outcomes and subjective ratings for standardised tests.

Table 2.2: Performance outcomes extracted from the thirty studies included in our literature search.

Reference	Exercise intervention	Timing of testing (h)	Performance outcome (compared to control)	Cognitive function and/or subjective variables
Abdelmalek et al. 2013	30 s Wingate test on cycle ergometer	08:00 and 18:00 h (h values	No significant effect for PP or MP	No
Ajiimaporn et al. 2020	Anaerobic sprint test (35-minute intervals x6, 10s rest between intervals) and isometric leg strength test	16:00 h	<p>↓AP by 16.8%</p> <p>↓Leg muscle strength by 15.9%</p> <p>↓Minimum power by 17.1%</p> <p>↓Max power by 21.2%</p>	Yes
Baati et al. 2020	Repeated cycling (10 x 6 s maximal cycling with 24 s between intervals)	17:00 h	No significant effect for PP	Yes
Brotherton et al. 2019	HG and 40, 60 and 80% of 1RM for bench press and leg press		<p>↓Grip strength by 2.7%</p> <p>↓AP by 11.2% for Bench press</p> <p>↓AP by 5.7% for Leg press</p>	Yes
Chase et al. 2017	3km TT on cycle ergometer and Peak isokinetic leg extensions	15:00-16:00 h	<p>↓TT finish time was slower by 2.3% for EX2 and 1.1% for EX1.</p> <p>No significant effect for VO₂ and VE</p> <p>No change in peak muscle torque</p>	No
Cook et al. 2012	Bench press, back squat and bent rows at 85% of 1RM for four sets	11:00 h	<p>↓BP by 28.8%</p> <p>↓BS by 20%</p> <p>↓BR by 11.7%</p> <p>↓Total workload by 19.8%</p>	No
Cullen et al. 2019	HG, CMJ and 15-minute self-paced TT	07:00-09:00 h	<p>↓Aerobic performance by 4.1%</p> <p>↓CMJ by 5.2%</p>	Yes
Daaloul et al. 2019	Karate specific test, CMJ and SJ	14:00 h	<p>No significant difference for SJ.</p> <p>↓CMJ by 2.8%</p> <p>↓Time to exhaustion for karate specific task by 12.2%</p>	Yes
Dean et al. 2023	90 min and 30-minute fixed paced cycling including two 6s PP, 4 and 20-minute TT	Not specified	<p>↓6-s PO by 7.3%</p> <p>↓20min TT PO by 6.7%</p>	Yes
HajSalem et al. 2013	Wingate Test and HG	Not specified	<p>↓PP by 1.6% before the judo match, 2.5% after the judo match</p> <p>↓MP by 3.5% before the judo match, 3.6% after the judo match</p> <p>No significant effect for HG.</p>	Yes

Table 2.2 continued.

Reference	Exercise intervention	Timing of testing (h)	Performance outcome (compared to control)	Cognitive function and/or subjective variables
Khemila et al. 2021	30 s Wingate Test	07:00 and 17:00 h	↓ PP by 10.5% at 07:00 h and 9.6% at 17:00 h ↓ MP by 10.4% at 17:00 h	Yes
Mejri et al. 2014	YoYo Intermittent Test	07:00-08:00 h	No significant effect for Total distance or HR.	Yes
Mejri et al. 2016	YoYo Intermittent Test	17:00 h	↓ TD by 9.0% for PSDBN and 28.1% for PSDEN	Yes
Mougin et al. 1996	30 s Wingate Test	09:00-12:00 h	No significant effect for maximal ventilation, MP, PP or PV.	No
Mougin et al. 2001	TTE on cycle ergometer	14:00 h	↓ Maximal work rate by 4.9% after PSDBN and 5.5% after PSDEN	No
Omiya et al. 2009	Cardiopulmonary test on cycle ergometer	17:00-21:00 h	↓ AT by 15.0% ↓ Time to reach AT by 18.6%	No
Paryab et al. 2021	Wingate Test and Dynamic Balance	08:00 h	No significant effect on balance, relative anaerobic power and relative MP.	
Reilly and Deykin 1983	HG, standing broad jump, stair run test, treadmill TTE	17:00 h	No significant effect for HG, SBJ, PP or endurance performance. ↓ Anaerobic power on testing day 1	Yes
Roberts et al. 2019	TT	06:30-07:50 h	↓ TTT finished time on day 3 of testing only.	Yes
Romdhani et al. 2019	Running sprint test	15:00 h	↓ MP for PSDBN and PSDEN ↓ Maximum power for PSDEN ↓ Minimum power for PSDBN and PSDEN	Yes

Table 2.2 continued.

Reference	Exercise intervention	Timing of testing (h)	Performance outcome (compared to control)	Cognitive function and/or subjective variables
Romdhani et al. 2020	Running sprint test	15:00 h	<p>↓ P_{max} by 13.6%</p> <p>↓ P_{mean} by 14.0%</p> <p>↓ P_{min} by 18.8%</p>	Yes
Romdhani et al. 2021	Running sprint test	15:00 h	<p>↓ P_{max} by 15.7%</p> <p>↓ P_{mean} by 16.8%</p> <p>↓ P_{min} by 24.8%</p>	Yes
Sinnerton and Reilly, 1992	HG, back strength, 4 x 50m swim sprints, 400m swim	06:30 and 17:30 h	<p>↓ Left HG</p> <p>No significant effect on right HG,</p>	Yes
Souissi et al. 2008	10 s Wingate and Force velocity	07:00 and 18:00 h	No significant effect for P _{max} , PP, MP or braking force (P > 0.05).	No
Souissi et al. 2013	Wingate, MVC, Handgrip	09:00-10:00 & 16:00-17:00 h	<p>↓ HG and MVC for SDE afternoon testing.</p> <p>↓ PP for SDE afternoon testing only.</p>	Yes
Souissi et al. 2015	30 s Wingate Test and SJ	08:00 h	No significant effect for MP or	Yes
Souissi et al. 2018	10 s Wingate Test and 5m shuttle test	18:00 h	<p>↓ PP by 11.1%</p> <p>↓ Peak distance by 12.4 %</p> <p>↓ TD by 10%</p>	Yes
Souissi et al. 2020	5-minute Shuttle Test	18:00 h	<p>↓ TD by 1.4%</p> <p>↓ Peak distance by 2.1%</p>	Yes
Sweeney et al. 2020	30 s sprint intervals	08:00 h	No significant effect for PP or Total work done.	No
Vardar et al. 2007	Wingate Test	14:00-16:00 h	No significant effect for PP, MP or AF (P > 0.05).	Yes

AF: Anaerobic Fatigue; AT: Anaerobic threshold; BR: Bent over rows; CMI: Countermovement jump; HG: Handgrip strength; HR: Heart rate; MP: Mean Power; MVC: Maximal voluntary contraction; PO: Power output; PP: Peak Power; SDN: Sleep restriction delayed bedtime; SDE: Sleep restriction early awakening; SJ: Squat jump; Standing broad jump; TD: Total distance; TOD: Time of day; TT: Time trial; TTE: Time to exhaustion.

2.5.2 Effects of sleep restriction on cognitive performance and perceptual responses

Twenty-two of the studies identified in our literature search assessed cognitive function and/or perceptual measures in addition to the physical performance tests. Consistent with the pattern observed in studies employing exercise tasks, only one of these cognitive studies included female participants. This highlights a persistent sex bias in the literature, which significantly reduces the applicability and translation of the findings.

It has been consistently documented that following SR, cognitive function and perceptual responses are influenced by a range of biological and hormonal factors that can differ between sexes. Failing to include females in these studies ignores these sex-based differences but also risks knowledge gaps in understanding how sleep restriction affects cognitive function in half of the population. Psychological factors are particularly impaired following sleep loss which may influence physical performance. Subjective responses for fatigue and sleepiness tend to dramatically increase, paired with reductions in vigour and alertness. The absence of alertness makes the ability to engage in cognitive tasks extremely difficult for an individual, particularly when the time on task is greater. Souissi and colleagues (2018, 2020) reported greater subjective fatigue as well as slower reaction times and a reduction in vigilance, when compared to habitual sleep. These outcomes are thought to be heavily influenced by a circadian component as vigilant attention is often impaired when there is an increase in homeostatic pressure. Further studies within the search report impaired cognitive function with increases in reaction time assessed using simple and choice reaction time tasks (Souissi et al. 2015; Souissi et al. 2018; Romdhani et al. 2019; Daaloul et al. 2019; Souissi et al. 2020; Romdhani et al. 2020; Khemila et al. 2021). However, two of these studies report increases in choice reaction time but not simple reaction time (Daaloul et al. 2019; Romdhani et al. 2020). The lack of significance was possibly due to minimal cognitive load and limited time required to complete the task. Overall, when the findings are related to athletes, reaction time and information processing are highly meaningful in terms of achieving optimal athletic performance. Yet the relevance of specific cognitive domains differs dependent on the specific sport. Given this it is important that future research considers sex specific responses to sleep loss to better inform strategies in athletic populations.

Affective functioning is another domain that was measured in ten of the studies within this review. It is often assessed in the form of mood; defined as a positive or negative state of mind that is longer lasting when compared to emotional state. The majority of studies investigate mood using the POMS questionnaire (Profile of Mood States) and report increases in states of anger, depression, confusion, anxiety and fatigue after sleep loss compared to a control sleep (Sinnerton and Reilly, 1992; Romdhani et al. 2019; Souissi et al. 2018; Roberts et al. 2019;

Romdhani et al. 2020; Souissi et al. 2020; Khemila et al. 2021). When sleep restriction is extended over consecutive days, the disturbance of mood state intensifies, and positive mood states decline (Roberts et al. 2019). Although the proposed mechanisms are still unknown, prior research has suggested REM sleep may be a potential mechanism as alterations in REM have been associated with changes in daytime affective state. Following SR there may be noticeable reductions in REM sleep, particularly if the restriction protocol involves early awakening as REM dominates the latter half of the night. A reduction in REM may therefore increase negative mood outcomes. Another proposed mechanism is the disruption of prefrontal cortex functioning, as SR has shown to cause reductions in connectivity between neural networks (Killgore, 2010). Despite these findings it is important to recognise that some individuals cope better with loss of sleep and can compensate for reductions in alertness while others appear much less resistant. Assessing chronotype and personal state traits such as flexibility and rigidity as well as languidity and vigour can give insight on how adaptable an individual is and why certain individuals adjust better to situations like sleep loss (Brotherton et al. 2019). Therefore, studies that have also assessed affective functioning using POMS and state-trait anxiety scores have reported no differences following SR (Vardar et al. 2007; Dean et al. 2023). Discrepancies between findings may be due to different interpretation and understanding of mood states, therefore it is difficult to draw conclusions on overall affective functioning when addressing single item mood states (i.e. anger and confusion; Groeger et al. 2022).

Table 2.3: Considerations for future research.

Additional considerations	Problem	Solution
Method of sleep measurement	Sleep restriction studies typically ensure compliance to protocols by monitoring the participant; objective measurement (i.e. actigraphy/polysomnography) or subjective measurement (i.e. sleep diary). These methods often disagree with one another as they each capture different elements of sleep. Inconsistency between studies and the absence of objective measures may reduce compliance with restriction protocols.	Recommendation: Incorporate an objective and subjective method of measurement. Why: This provides insight on key sleep variables (e.g. duration, efficiency, wake bouts). It also allows researchers to assess subjective sleep perception as this can influence performance outcomes regardless of the objective findings.
Participant sex	Amongst the studies conducted to date there are very few that include female participants. There are known sex differences in human sleep, which are mainly due to hormonal changes. The absence of	Recommendation: Aim to conduct more female research and understand the difficulties of recruiting female participants. Ensure you refer to methodological guidelines such as those proposed

	<p>research on females limits the ability to apply evidence informed interventions and therefore as researchers we are failing to maximise the potential of these athletes.</p>	<p>by Elliott-Sale et al. 2021, to achieve good practice standards.</p> <p>Why: There is high demand for female research due to a rise in the professionalism of female sports. By incorporating females into sleep research, we can expand our knowledge and develop sex specific, evidenced based guidelines.</p>
Fitness level	<p>Most sleep studies recruit individuals who are recreationally active opposed to elite level athletes. Studies involving restriction protocol can be quite burdensome for those participating making them impractical for athletes to participate. The impact on performance may vary between these populations due to differences in training demands and recovery needs of elite athletes.</p>	<p>Recommendation: Research should aim to recruit diverse populations and incorporate more elite athletes. To standardise across studies, participants should be categorised using a framework such as ‘Participant Classification Framework’ (McKay et al. 2021).</p> <p>Why: This will develop our understanding of sleep restriction and performance in different populations.</p>
Chronotype	<p>Assessing chronotype during the pre-screening process is still not routine. Research indicates that chronotype can influence physical and cognitive tasks, with individuals performing better when tasks are aligned with their preference. By not assessing chronotype it may significantly impact study outcomes, especially if the time of testing misaligns with the participants chronotype.</p> <p>To assess chronotype there are multiple questionnaires that have varying criteria which results in lack of standardisation and variability.</p>	<p>Recommendation: Participant chronotype should always be assessed prior to testing for any studies investigating sleep.</p> <p>For assessment of chronotype future studies should adopt a standardised and validated tool that clearly defines chronotype.</p> <p>Why: Allows researchers to account for individual differences, understand the impact on study outcomes and reduce bias.</p> <p>It will allow comparable assessment between studies in regard to chronotypes and expand our knowledge on more extreme chronotypes.</p>
Caffeine	<p>Studies often ask participants to abstain from caffeinated products due to the stimulant effects. Sensitivity and the half-life of caffeine vary amongst individuals. If a study requires participants to abstain but does not screen for habitual intake, there may be withdrawal effects such as excessive sleepiness that mask performance outcomes.</p> <p>Consumption of caffeine is very common. Requiring participants to abstain is unrealistic and does not reflect real-world conditions.</p>	<p>Recommendation: Researchers should obtain information on habitual caffeine consumption (quantity, type and regularity) to assess if participants are low, middle or high consumers.</p> <p>Why: Screen habitual intake to help control potential withdrawal effects for studies that employ abstinence.</p> <p>Rather than abstaining tailored protocols such as limiting consumption to time windows could be developed (i.e. no caffeine after 13:00 h).</p>

Familiarisation sessions	If a study does not employ familiarisation sessions prior to testing it may result in a ‘learning effect’ where performance improves with more exposure, not due to the intervention. Participants may not comply or make mistakes as they do not understand the protocol. This can lead to unreliable data.	Recommendation: Before data collection, ensure all participants complete at least one familiarisation session where they are introduced to study procedures and tasks. Why: Reduces any learning effect and ensures the participant is aware of the protocol and able to adhere.
Sample size	Due to the nature of sleep restriction protocols, it is often a challenge to achieve large sample sizes, which leads to underpowered data.	Recommendation: Sample size and power estimations should be calculated to determine recruitment numbers. Why: This will ensure the study does not lack sufficient power and the sample is large enough to detect differences and account for dropouts.

2.6 Additional considerations

The review sought to explore the current literature on the effects of acute sleep loss on exercise performance. There is still a lack of evidence to provide a clear consensus on this topic, particularly when trying to provide athlete specific recommendations. There are several additional considerations that are proposed and should be incorporated into future studies to begin addressing some significant gaps in the literature (see Table 2.3).

2.7 Conclusion

The review of the literature confirms that there is a significant impact of sleep restriction on aerobic, anaerobic, power and strength-based exercise. However, these findings are not consistent across all studies. Differences between studies are dependent on exercise timing (morning versus afternoon), fitness level (recreational versus trained/elite), prior habitual sleep and the sleep restriction protocol (delayed bedtime versus early awakening). To ensure participants are achieving sufficient sleep prior to testing, studies should monitor habitual sleep using sleep diaries and actigraphy. When exercise is scheduled in the afternoon and following an early awakening restriction protocol, the effect on performance is generally impaired to a greater extent than morning exercise testing following delayed bedtime restriction. In addition, cognitive function and perceptual changes are apparent after exposure to sleep restriction, that likely contribute to a reduction in exercise performance. Currently there is insufficient evidence to draw conclusions on the effects of sleep loss on specific sports/performance tests. More in depth research is needed into the physiological effects of sleep restriction. To add, standardised and rigorous

research designs should be employed in future studies as there are inconsistencies between protocols. Of the 33 studies included in our review, seven did not provide sufficient evidence to classify the population using the framework by McKay and colleagues (2021) making it difficult to compare between participant populations. Another important factor to consider is dietary intake, however this was relatively consistent with a total of twenty-one studies either monitoring food consumption through a dietary log or providing standardised dietary intake. On the other hand, factors such as sample size estimation were only reported by three of the studies which raises concerns regarding statistical power; essential for detecting a true effect (Table 2.1 and 2.2). By adopting consistent methodologies, it will allow for easier comparison between studies and improve the reliability of findings to further support practitioners/coaches in providing evidence informed guidelines and recommendations (Table 2.3).

Chapter 3:

General Methods

The purpose of this section is to provide an overview of the methods that were conducted across Chapters 4, 5, 6, and 7 and to justify the experimental design.

3.0 General methods

3.1 Participants

To pre-screen participants, the following questionnaires were completed prior to all experimental conditions: Physical Activity Readiness Questionnaire (PAR-Q; Chisholm et al. 1975); Composite Morningness/Eveningness Questionnaire (CMEQ) to assess chronotype (Smith et al. 1989); Caffeine Expectancy Questionnaire to assess caffeine consumption (Huntley and Juliano, 2012); Pittsburg Sleep Index Questionnaire (PSQI; Buysse et al. 1989) and Epworth Sleepiness Scale (Johns, 1991) in Chapters 4, 5, 6 and 7. To clarify the PSQI was employed as it is a well validated instrument used in a range of populations that assess multiple domains of sleep. The PSQI was used to gain greater insight of the participants habitual sleep in the one month prior to participating, it was not used to strictly exclude individuals based off their index score. For assessment of chronotype, there are numerous questionnaires available however we opted for the CMEQ as it is easy to score and assesses subjective preference opposed to factors that can be influenced by external factors (i.e. actual sleep and wake times). In Chapter 4 we also included the Berlin Sleep Apnoea Questionnaire (Netzer et al. 1999) as it was easy to employ, well validated and provided an indication of individuals who may be at risk of sleep apnoea. The Arousal Disorder Questionnaire (Loddo et al. 2021) was also employed in Chapter 4 to assess for symptoms related to sleep disorders (i.e. sleep walking) to ensure participants were safe to sleep overnight in the university laboratory. Exclusively in Chapters 5, 6 and 7 participants were classified as recreationally active, using the 'Participant Classification Framework' (McKay et al. 2021), as suggested in the future considerations within the review (see Table 2.3). All participants had > 2 years of strength and weight-based training experience. To meet the *inclusion criteria* participants had to identify as male (for Chapters 5, 6 and 7), as identified by sex and gender, have no diagnosed sleep disorders, no musculoskeletal injuries, caffeine intake is not in excess of > 200mg (classed as moderate to high intake), classified as an intermediate chronotype using CMEQ or travel outside of the local time-zone in the past month and/or completed shiftwork (refer to Table 3.1, Edwards et al. 2024). The original aim was to include female participants within the inclusion criteria due to the lack of research on female participants. Unfortunately, this was not possible due to monetary restrictions that accompany self-funded studies. To achieve gold standard method of measurement and conduct menstrual tracking it would have required hormone detection via serum or saliva samples that would have incurred significant costs hence our inclusion of only male participants in Chapters 5, 6 and 7. All participants provided written consent, and all experimental procedures were explained. The research investigations were approved and conducted in accordance with the Human Ethics

Committee at Liverpool John Moores University and complied with the Declaration of Helsinki. Individual ethics codes will be included in the relevant chapters, in the method sections.

Table 3.1 Participant characteristics across the four experimental studies (Mean \pm SD).

	Chapter 4 (n = 10)	Chapter 5 (n = 15)	Chapter 6 (n = 18)	Chapter 7 (n = 11)
Age (years)	24.5 \pm 2.1	22.0 \pm 1.6	22.0 \pm 1.9	22.4 \pm 2.1
Height (cm)	179.3 \pm 3.7	177.0 \pm 5.7	178.4 \pm 5.9	178.6 \pm 5.7
Body mass (kg)	82.8 \pm 14.0	79.4 \pm 10.4	75.0 \pm 1.2	83.5 \pm 11.4
Habitual sleep duration (hh:mm)	07:17 \pm 00:32	07:53 \pm 00:39	07:59 \pm 00:42	08:16 \pm 00:44
Chronotype (13-52)	37 \pm 4	33 \pm 4	35 \pm 6	36 \pm 5
Flexibility/rigidity (14-70)	45 \pm 7	46 \pm 4	48 \pm 7	49 \pm 8
Languidity/vigour (16-80)	38 \pm 6	44 \pm 5	40 \pm 7	38 \pm 8
Participant classification (Tier: 0-5)	Recreational	Recreational	Recreational	Recreational
Time of year testing was conducted	September-June	October - May	October-April	November-April

3.2 Research Design

3.2.1 Pre-screening process

Each participant was required to complete 7-days of habitual sleep recording via actimetry and a sleep diary to capture data over weekdays and weekends. During this 7-day recording period, habitual food/fluid intake (including caffeine consumption) was also recorded for the first 5-days to obtain basic dietary information and ensure participants were consuming a balanced diet. The habitual data was collected two weeks prior to the first condition to ensure participants were maintaining healthy sleeping habits prior to testing. Participants were not eligible to participate if habitual caffeine consumption was towards moderate to high quantities (> 200 mg per day; Temple et al. 2017). Prior to testing participants were required to complete the questionnaires outlined in section 3.1, 'Participants'. The initial laboratory visit (for Chapters 5, 6 and 7) involved completion of one repetition max (1RM) of bench press and back squat at the university gym.

3.2.2 Familiarisation sessions

The initial two visits were familiarisation sessions and consisted of: Recording height and mass (Seca, Hamburg, Germany); completion of questionnaires; Stroop test (see section 3.4 for explanation of this cognitive test) and exercise protocol. Familiarisation sessions were conducted to ensure that participants were aware of the protocol and to reduce the risk of failed lifts during testing sessions. Between each experimental condition participants were under free living conditions with no lifestyle restrictions (diet, exercise etc). However, in the 24 h prior to experimental protocol participants were asked to refrain from vigorous exercise and alcohol consumption. In addition, participants were asked to refrain from food consumption in the 1-2 h before all exercise sessions. Between all experimental conditions participants were given a minimum of 7 days to ensure adequate recovery. For Chapters 5, 6 and 7 experimental conditions involved two nights of partial sleep restriction to be taken at the participants' home; participants were required to wear an actigraphy (Motionwatch 8, CamnTech) watch to measure sleep and ensure compliance to the protocol. Researchers also checked in with the participants via direct message prior to bed and upon waking. Conditions were completed in a counterbalanced order of administration to reduce any potential learning effect in Chapters 4, 5, 6 and 7 (Monk and Leng, 1982). A stacking method was used to group participants into 3 groups based on their physical abilities from the 1RM session for bench press and back squat values. This method was used to control for confounding variables that could influence the outcomes of the study such as differences in strength.

3.2.3 Sleep restriction protocol

Sleep restriction conditions (followed in Chapters 5, 6 and 7) required participants to either retire to sleep at 02:30 and rise at 06:30 h (Chapters 5 and 6) or retire at 03:30 and rise at 06:30 h (Chapters 6 and 7). A delayed bedtime restriction protocol was employed to replicate real world scenarios of sleep loss. For many athletes' their competition time is scheduled in the evening which often results in retiring to sleep in the early hours of the morning once they have left the competition and returned to their accommodation. The following morning athletes may have to vacate the property early which restricts their sleep window to similar sleep times that were adopted in the current studies. This approach allows the research outcomes and recommendations to be directly relatable to those in the applied setting. In the hour prior to sleep participants were asked to refrain from screen use (mobile phone, laptop etc). During the two nights of sleep restriction and on the third day of testing participants were

instructed to refrain from driving and advised to commute to and from the university by walking or public transport for their health and safety.

Following partial sleep restriction (PSR), on the third day participants were allocated to no nap, 30-min nap or a 60-min nap; all commencing at 13:00 h. Although participants were not able to be blinded to the sleep restriction protocol, participants were not informed of their napping condition until entering the room at 13:00 h. This reduced any anticipatory effects on the outcomes. For the two nap conditions (30 min and 60 min nap) participants were given the opportunity to sleep on a single orthopaedic hospital bed provided in the university sleep laboratory and were required to stay in bed until the end of the session when the researcher re-entered the room. Upon re-entering the room, the researcher subjectively asked participants if they napped with participants responding that they “managed to sleep” and others “rested their eyes”. These responses were not officially recorded which may be deemed as a limitation; a questionnaire that asked the participant how long they managed to sleep may have been more appropriate. Throughout the testing day (for Chapters 5, 6 and 7) researchers regularly checked in with participants to ensure compliance and participants were asked to remain on campus until testing finished.

3.3 Measurements

3.3.1 Polysomnography

Sleep was recorded using polysomnography with an ambulatory monitor (SOMNOMedics, GmbH, Germany) in Chapters 4 (overnight sleep monitoring) and 7 (during nap periods). The PSG montage consisted of electroencephalography (EEG) at 8 electrode brain sites to create 6 standard EEG channels referenced to contralateral mastoid processes for F3-M2, F4-M1, C3-M2, C4-M1, O1-M2 and O2-M1. Left and right electrooculograms (EOG) were fitted, in addition to two lead electrocardiogram (ECG) and one chin electromyogram (EMG). A 6-channel set up was implemented opposed to a typical 10-20 system as the equipment was purchased by the department at the beginning of the PhD and there was no on-site technical support available. Due to limited experience this set up was more appropriate and provided sufficient information required for the two studies. The potential drawbacks of not employing a larger montage were reduced precision when identifying key markers such as K complexes and sleep spindles, as well as greater risk of misinterpreting artifact.

To start each recording, all impedances were $<10\text{ k}\Omega$. Electrode site placements were measured and applied in accordance with standard criteria for American Society of Sleep Medicine (ASSM). The PSG unit was fitted for

an 8-hour sleep opportunity in Chapter 4 and a 30 or 60-min duration in Chapter 7. To determine sleep onset latency (SOL; time in minutes from lights out to sleep onset), we defined sleep onset as the first epoch of stage N1 sleep, as N1 is associated with the transition from wakefulness to sleep. Despite using this cutoff, this method may underestimate SOL as individuals can transition from N1 to wakefulness. Therefore, defining sleep onset based on the presence of N1 followed by a sustained N2 may have been more appropriate.

Prior to testing, participants were not familiarised with the PSG equipment. This is a limitation of the study design due to the nature of the equipment, and the discomfort participants may experience when wearing the PSG. However, the primary reason for not conducting a familiarisation was due to time constraints and available resources as the study designs were already rather burdensome for those who participated.

All recordings were downloaded onto DOMINO Software (SOMNOMedics, Germany) and scored manually in 30s epochs in accordance with the American Academy of Sleep Medicine (AASM) guidelines for the standard adult. Two trained sleep technologists independently scored each recording. This protocol was followed for Chapters 4 and 7.

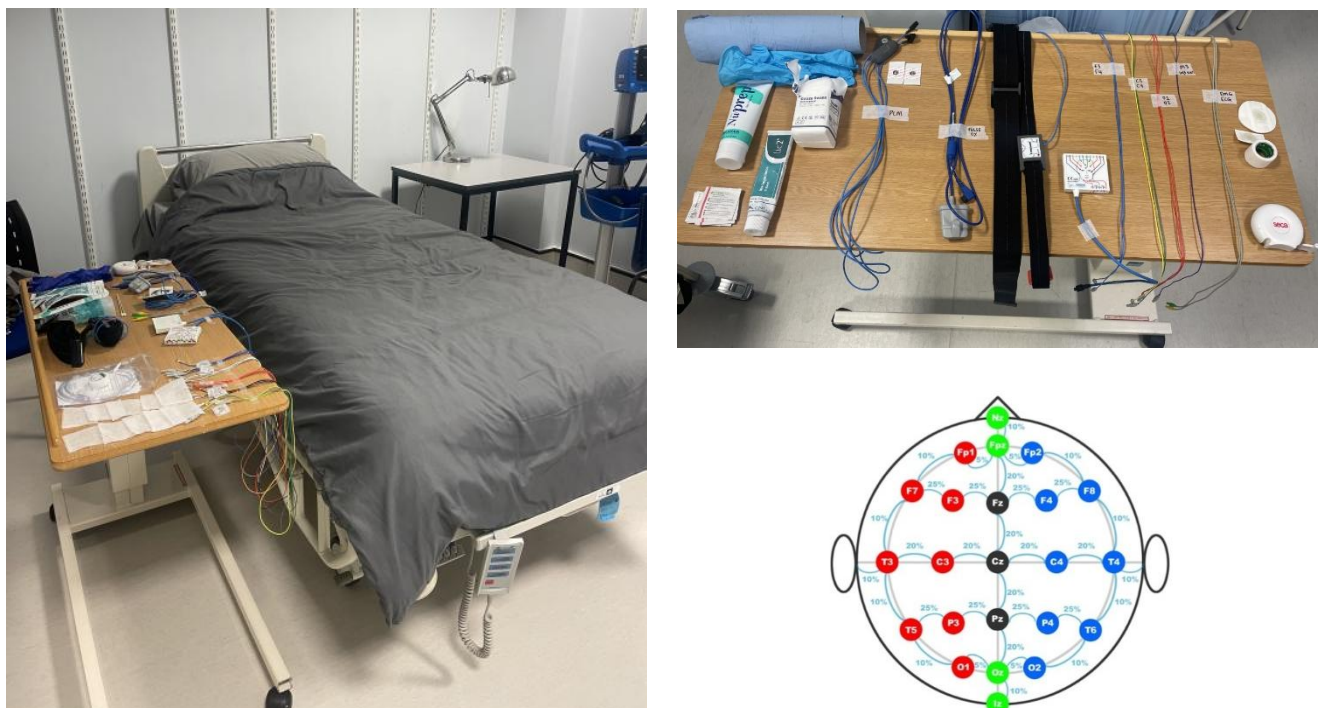


Figure 3.1 Sleep laboratory preparation for nap sessions and overnight sleep assessment.



Figure 3.2 Example of preparation for polysomnography for an afternoon nap (13:00 h) following sleep restriction.

3.3.2 Exercise performance

Before experimental conditions participants would complete a one repetition max (1RM) session for both bench press and back squat lifts. The values obtained in this session were used to determine 40, 60 and 80% of their 1RM values and conducted to ensure each participant could safely perform the lifts. These values were selected as they replicate training weights that resistance trained individuals would typically lift. During each experimental protocol participants would conduct an exercise session. Prior to submaximal weightlifting, an active warm-up was conducted that consisted of 5 minutes at 150 W on a cycle ergometer (Lode Corival, Furth, Germany), followed by a series of dynamic stretching involving: Squats (x10), single leg lunges (x5 each leg), single leg Romanian deadlifts (x5 each leg) and press ups (x10), repeated twice. Following the warm-up, participants had three attempts at left- and right-hand grip strength using a dynamometer (Takei Kiki Kogyo, Tokyo, Japan). To record performance variables for bench press and back squat, a force velocity linear encoder (MuscleLab, Ergotest, version 4010, Norway) were attached to a 20 kg Olympic bar to record values for displacement (D), average power (AP), peak velocity (PV), average velocity (AV) and time to peak velocity (tPV). Participants completed 3 attempts at each incremental load (40, 60 and 80%), as detailed above, for bench press and back squat, with a 2-minute rest between each set. After each set participants were asked for a rating of perceived

exertion, breathing and muscle fatigue using the RPE 6-20 scale (Borg, 1982) and rating of effort presented as a visual analogue scale (VAS: 0-10 scale where 0 is no effort and 10 is maximal; Birk and Birk, 1987). To analyse the performance data, the highest of the three AP outputs (and associated D, PV, AV and tPV values) were used for analysis of each mass on the bar for bench press and back squat, respectively.

3.3.3 Cognitive tests

The Stroop test was divided into 3 trials; participants had 45 seconds (s) to perform each trial and had to read their responses aloud. For the first trial participants were presented with an A4 sheet that had names of colours printed in black ink. The second trial participants were presented with congruent colour-words, meaning they were displayed in the same colour they were named (i.e. the word 'Red' is displayed in red font) and the third trial contained incongruent words, where the word was different to the colour displayed (i.e. the word 'Red' is in blue font). For the second trial participant first read aloud the meaning of the word (word-not-colour) and for the third trial read aloud the colour of the ink ignoring the meaning of the word (colour-not-word). Each trial was filmed by a researcher to capture the number of errors and total score.

3.3.4 Questionnaires

Throughout the experimental conditions participants were asked to complete the following questionnaires on the day of testing to assess an array of psychological variables:

- Profile of Mood States (POMS Version 32; Terry et al. 2003): The questionnaire presents 32 different types of feelings that people may experience. The participant was required to select the relevant response rated on a likert scale: 'Not at all' = 0, 'A little' = 1, 'Moderately' = 2, 'Quite a bit' = 3, 'Extremely' = 4'. The 32 feelings were organised into six mood states for analysis: Tension, depression, anger, vigour, fatigue and confusion (questionnaire was used in Chapters 4, 5, 6 and 7).
- Stanford Sleepiness Scale is a tool to assess an individual's level of sleepiness at a given moment. It is presented on the following 7-point scale: 1 = Feeling alert, 2 = Functioning at high levels, 3 = Awake but relaxed, 4 = Somewhat foggy, 5 = Foggy, 6 = Extremely sleepy and 7 = No longer fighting sleep (questionnaire was used in Chapters 4, 5, 6 and 7).

- Waterhouse questionnaire contains sleep questions from the Liverpool Jet-lag questionnaire (Waterhouse et al. 2007). It contains 5 questions regarding the prior night's sleep with questions in relation to ease falling asleep, rising and retiring time, sleep quality and alertness 30 min upon waking (questionnaire was used in Chapters 4, 5, 6 and 7).
- Caffeine withdrawal questionnaire is a 23-item tool used to assess the magnitude of withdrawal following caffeine abstinence (used in Chapter 7 only). Each symptom is rated on a scale of 0-4, with 0 being 'not at all' and 4 'severe'. The items are categorised into 7 factors for analysis: Drowsiness/fatigue, decreased alertness, mood disturbances, decreased sociability, nausea/upset stomach, flu-like feelings and headache.

Table 3.2: Description of all questionnaires and scales employed throughout the thesis.

Measure	Dimensions	No. of items	Scoring	Score range	Direction
Arousal Disorder Questionnaire	Screening for disorders (i.e. sleepwalking)	15	Yes/No responses	Sum of total score	‘Yes’ responses indicate high risk for arousal disorders.
Berlin Sleep Apnoea Questionnaire	Risk of obstructive sleep apnoea	10 questions across 3 categories	Yes/No responses and Likert scales	Classified as high risk if > 2 categories are positive	High risk = more likely to have OSA
Caffeine Expectancy Questionnaire	Individuals’ expectations of how caffeine affects them.	47	6-point Likert scale	1 (very unlikely) to 6 (very likely) Total score range 47-282	Higher score = higher expectancy
Caffeine Withdrawal Questionnaire	Assesses withdrawal symptoms	23	0-4 scale	Sum of scores for each symptom (7 categories)	Higher score = greater withdrawal
Composite Morningness/Eveningness Questionnaire	Section 1: Assesses chronotype. Section 2: Assesses languidness/vigorous (L/V) and flexibility/rigidity (F/R)	Section 1: 13 questions Section 2: 14 L/V questions and 16 F/R questions	Multiple choice scored 1-5	Section 1: 13-55 Section 2: 14-70 (L/V), 16-80 (F/R)	Section 1: ≤ 22 evening type, 22-43 intermediate, ≥ 44 morning type. Section 2: scores < 37 more vigorous/rigidity, scores > 37 more languid/flexible
Epworth Sleepiness Scale	Daytime sleepiness	8	0-3 scale per item	0-24	Higher score = greater daytime fatigue
Hunger and Satiety Scale	Subjective responses regarding hunger and fullness	8	Participant marks on the 10cm line, anchored by ‘Not at all’ and ‘Extremely’	0-100mm	Higher score = more intensity (more hunger etc)
Physical Activity Readiness Questionnaire (PAR-Q)	Determine if an individual is safe to exercise	7	Yes/No responses	Any ‘Yes’ responses require referral	All ‘No’ responses, individual is cleared to exercise
Pittsburg Sleep Quality Index (PSQI)	Sleep quality	19	Each component scored 0-30; Global score is the sum of components	0-20	Higher score = poorer sleep quality
Profile of Mood States – Short form (POMS)	Six mood states	32	Five-point Likert scale	Mood states split into subgroups with score range depending on items per subscale.	Higher score on negative subscale = worse mood Higher score for vigour = better mood
Rating of perceived exertion (RPE)	Perceived exertion	1	6-20 scale	6-20	Higher score = greater perceived exertion
Stanford Sleepiness Scale	Subjective sleepiness	7	Scale from 1 to 7	1-7	Higher score = greater sleepiness
Visual Analog Scale	Subjective response to exercise task	1	Participant marks on the 10cm line, anchored by ‘Not at all’ and ‘Extremely’	0-100mm	Higher score = greater intensity
Waterhouse Questionnaire	Assesses prior nights sleep quality in 5 questions	5	-5 to 5 scale	-5 to 5	-5 indicates poorer sleep than normal 0 indicates no difference in sleep +5 indicates better than normal sleep

3.4 Statistical Analysis

The Statistical Package for the Social Sciences (SPSS IBM) version 29, for Windows was for analysis of Chapters 5 and 6. In Chapters 4 and 7, JAMOVI version 2.6.25 statistical package was used. Differences within conditions were evaluated using a general linear model with repeated measures. For Chapters 5, 6 and 7 within subject factors for nap condition (three levels), time-of-day (two levels for exercise performance and 4 levels for psychological variables), 'load on the bar' (three levels; 40, 60 and 80%) and interactions between all conditions were conducted. For Chapter 6, a between subject factor for group was also ran (sleep restriction protocol with two levels, SR₃ and SR₄). All data was checked for normality by conducting visual checks of the residual plots and Q plots. To correct for violations of sphericity, the degrees of freedom were corrected using Huynh-Feldt ($\epsilon > 0.75$) or Greenhouse-Geisser ($\epsilon < 0.75$) values for ϵ , as appropriate. Graphical comparisons between means and Bonferroni pairwise comparisons were made where main effects were present. For non-normally distributed data and missing data points a generalised mixed model (GMM) was conducted in Chapters 4 and 7. For GMM, results are reported as chi squared values (χ^2), degrees of freedom (df) and associated P-values (P). The level of statistical significance was set at $P < 0.05$ for all outcomes. Effect sizes are referred to as partial eta squared values (η^2_p) with values of 0.01, 0.06 and 0.14 corresponding to a small, medium and large effect respectively (Cohen, 1988). All results in text, figures and tables are presented as mean \pm standard deviation (SD) unless otherwise stated. Ninety-five percent confidence intervals are presented where appropriate as well as the mean difference between pairwise comparisons. To determine sample size, a power calculation software (G*Power, version 3.1.9.6; Faul et al. 2007) was used and the relevant effect size and power value for each research study (refer to Table 3.2). Figures presented in Chapters 4, 6 and 7 were designed in Prism (Version 8, Graphpad, USA).

In Chapter 7 Bland and Altman plots were created to demonstrate the mean differences between each sleep device compared to PSG against the average of each sleep variable to determine the level of bias. The bias is represented as the mean difference between PSG and each device with a negative mean representing an underestimation and a positive mean difference representing an overestimation. Upper and lower limits of agreement were based on 95% confidence intervals and were plotted to determine the significance of mean differences.

To analyse polysomnography data in Chapters 4 and 7, two trained sleep researchers (CG, SP) independently scored all PSG recordings, in 30s epochs according to the criteria outlined by AASM. Sleep onset latency was

determined by comparing self-reported sleep time with sleep onset determined by PSG. All data from actigraphy watches was downloaded and analysed with the relevant software (Motionware) and were scored in 15s epochs.

Table 3.3 Values used to determine sample size estimations for each study.

	P value	Power	Effect size	Estimated sample	Reference
Chapter 4	0.05	0.80	0.75	23	Chinoy et al. 2021 for total sleep time (n = 16).
Chapter 5	0.05	0.80	0.80	12	Brotherton et al. 2019 for average power values.
Chapter 6	0.05	0.80	0.80	12	Brotherton et al. 2019 for average power values.
Chapter 7	0.05	0.80	1.13	7	Petit et al. 2014, mean and standard deviation values for REM sleep.
Using our familiarisation data					
Chapter 4	0.05	0.80	4.20	3	Calculated using total sleep time (n = 8).
Chapter 7	0.05	0.80	0.78	12	Calculated using average power from familiarisation data set.

Chapter 4:

A preliminary study investigating polysomnography and surrogate devices for sleep assessment in home and laboratory settings.

This chapter provides insight into the issues of consumer sleep devices compared to validated methods and highlights the importance of a familiarisation night when assessing sleep in the laboratory.

4.1 Introduction

The consumer demand to measure sleep has grown significantly as has the global sleep technology devices market, that is forecast to exceed \$60 billion by 2025 (Goldstein, 2020). Sleep tracking via the use of consumer devices (such as wearables, nearables and airables) is rapidly growing, as is the availability of these products in the market. Although these devices may allow consumers to self-monitor their sleep, most of these products have not been validated (Ko et al. 2015). It is essential to scientifically test all devices against the gold standard measure of polysomnography (PSG) to ensure inaccurate information isn't provided and the results do not have unintended effects on sleep behaviours (Bianchi, 2018). Previous studies that have assessed sleep trackers report difficulties detecting wake bouts, sleep onset latency and proportion of sleep stages (De Zambotti et al. 2024). The issue attaining accurate results is due to a high sensitivity to identify sleep but low specificity to detect wake bouts, with device algorithms creating assumptions based on physical movements (Lee et al. 2018). Although there are fundamental issues, consumer devices often have an easy user interface and are more affordable. The devices also provide an array of information regarding sleep and other behaviours, that can be measured over extensive periods without the need for specialist support (De Zambotti et al. 2019). In addition to wearables/nearables, research grade devices such as actigraphy watches are often used, particularly in non-laboratory settings due to extensive validation (Montgomery-Downs et al. 2012). Recent studies have reported that when comparing consumer sleep devices to actigraphy they performed equivalent to, if not better for detecting wake bouts. The mixed findings across studies, highlights the need for further validation (Chinoy et al. 2021).

Polysomnography recordings are typically conducted in the laboratory which does not mimic the natural sleeping environment. When assessing sleep in the laboratory, key variables such as total sleep time and sleep efficiency were significantly reduced compared to at home (Portier et al. 2000; Ameen et al. 2019). This phenomenon is referred to as the 'first night effect' which has been frequently observed in healthy individuals. Greater sleep fragmentation is likely due to unfamiliarity of the equipment, discomfort of the electrodes and restricted movement due to the equipment (Newell et al. 2012). With the growing demand to measure sleep and limited sleep laboratories, overnight assessments are being restricted to one night, although the reference standard is two nights due to the 'first night effect' (Samson, 2021). Ambulatory polysomnography in the home is therefore becoming popular as it reduces the 'first night effect' and the strain on public health services (Iber et al. 2004). Despite the concerns of conducting polysomnography in the home, due to issues with signal loss and the inability

to adjust sensors, studies that compared the home versus hospital showed no difference in study quality or accuracy (Mykityn et al. 1999).

Therefore, the aims of this study were i) to compare surrogate sleep devices (wearable, nearable, research grade) to polysomnography and ii) investigate the differences between sleeping in the home versus laboratory environment. As the overarching aim of the thesis was to support future recommendations, sleep measurement is a high priority for individuals, yet the current literature is very conflicting. Therefore, this chapter explores validation of consumer grade products compared to research grade devices that are employed in the following experimental chapters (actigraphy and polysomnography) to support guidelines and further insight.

4.2 Methods

4.2.1 Participants

Ten males, as identified by sex and gender (refer to Table 3.1 for characteristics), participated in the study. Sample size estimation is presented in Table 3.2 of the General Methods, and the inclusion criteria are detailed in Chapter 3: General Methods, section 3.2.1. Female participants were able to participate; however, no females applied to the study. Prior to participation, all volunteers were presented with an information sheet and provided with a verbal explanation of the experimental procedures, explaining the possible risks associated. Pre-screening process consisted of 7 questionnaires to assess eligibility as outlined in Chapter 3, section 3.1. Experimental procedures were approved by the Human Ethics Committee at Liverpool John Moores University (Ethics number: 23/SPS/048) and complied with the Declaration of Helsinki.

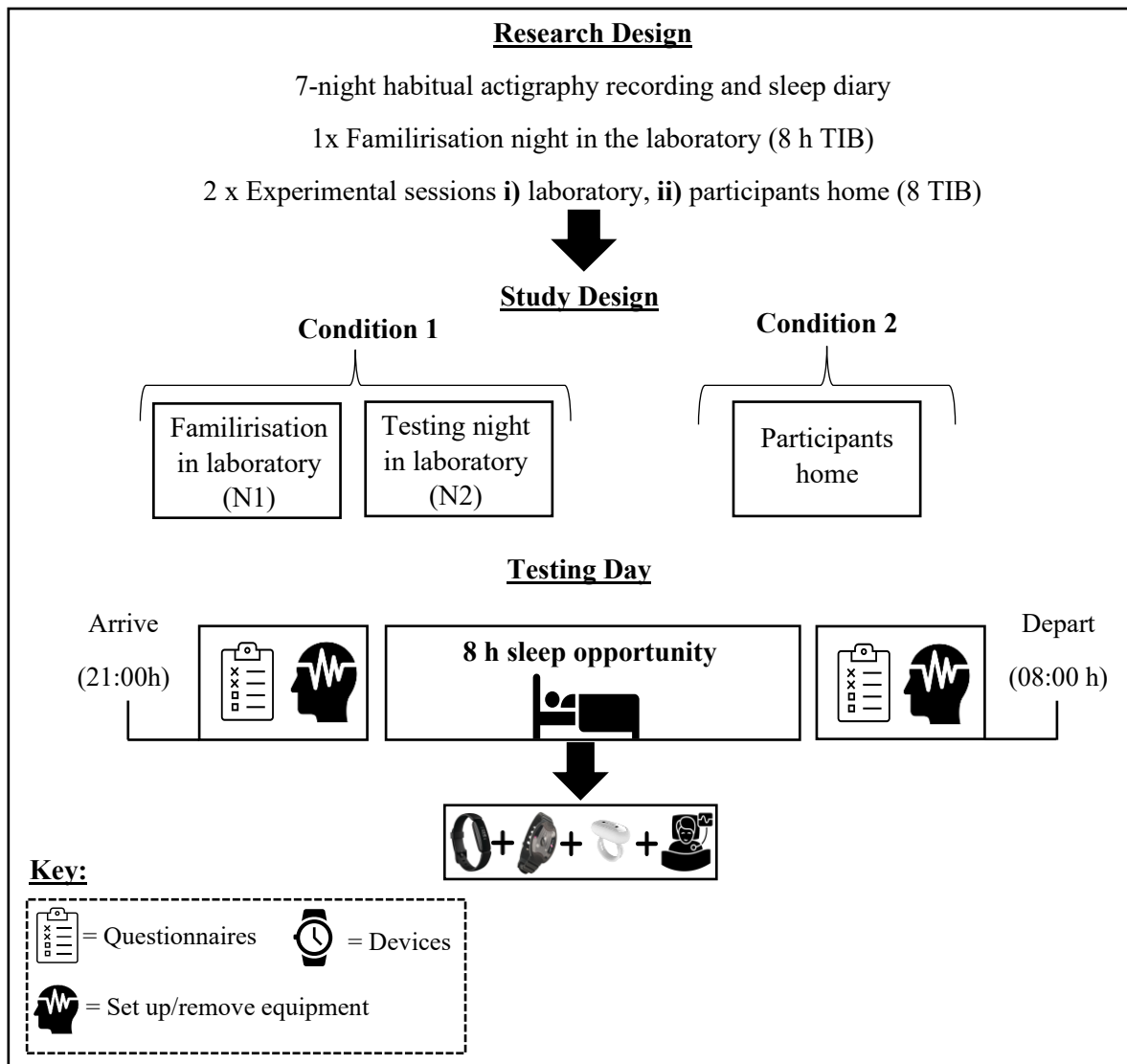






Figure 4.1. Study schematic, outlining the research design, study design and testing day protocol.

4.2.2 Research Design

All participants were required to visit the University sleep laboratory on two occasions, in addition to one visit at the participants' home. Two weeks prior to the first experimental condition, participants wore an actigraphy watch (Motionwatch 8, CamnTech) and used a sleep diary to record habitual sleep for a 7-day duration to ensure participants were maintaining healthy sleeping habits. Thereafter participants completed 1 familiarisation night in the laboratory (N1) and 2 experimental conditions i) overnight stay in the University sleep laboratory (N2) ii) overnight home condition (Home), conducted in randomised and counterbalanced order. The familiarisation night took place within the 5 days prior to the N2 condition with the purpose of habituating the participant to the

environment. Each overnight stay involved an 8 h sleep opportunity, with retiring and rising times relative to the participants habitual sleep schedule. Participants reported to the laboratory 2 h prior to their habitual bedtime. At the beginning of the testing session participants underwent 10 channel polysomnography electrode application by a trained researcher, followed by applying the remaining devices: FitBit Inspire 2 (FBI2; FitBit Inc., San Francisco, California, USA), actigraphy, Go2Sleep ring (GS2) and Withings Sleep Analyzer (nearable sensor pad placed under the mattress; Withings, Paris, France) to ensure they were fitted correctly and connected to the associated applications. Unfortunately, technical difficulties were experienced with the Withings pad on multiple occasions resulting in the nearable device being removed from the study. In the 30 minutes prior to their scheduled bedtime participants were asked to refrain from using any light emitting devices. This was to reduce the exposure of blue light in close proximity to the scheduled bedtime as blue light can delay sleep onset due to melatonin suppression as well as impair sleep quality (Silvani et al. 2022). Participants slept in the university sleep laboratory which was sound attenuated and had no windows or clocks. Prior to bed once all equipment was fitted, participants were asked to complete 3 questionnaires: Perceived stress scale, Profile of Mood States (POMS) and Stanford Sleepiness Questionnaire. At the scheduled bedtime the researcher would exit the sleep laboratory and turn off all the lights. Researchers monitored the participant throughout the night from the laboratory next door, using a tablet (Samsung Electronics, Seoul, South Korea) that was linked to the polysomnography system. At the participants rising time researchers returned to the sleep laboratory, turned on the lights and woke the participant. Following this all the equipment was removed, participants were asked to complete the Waterhouse Questionnaire which asked questions on the prior night's sleep, before exiting the laboratory. For the Home condition, two researchers arrived at the participants home approximately 2 hours before their scheduled bedtime to allow sufficient time to apply all the equipment. Instructions were provided verbally and in writing on how to properly initiate the devices and complete the questionnaires at bedtime and upon waking. The researchers would return to the participants home in the morning to remove the equipment. Refer to Figure 1 for the study schematic.

Each condition was separated by a minimum of 7 days to ensure the participants were well rested. During the first assigned condition participants were allowed to continue normal daily living activities, however they were asked to replicate the same activities and dietary intake for the remaining conditions. Participants were asked to refrain from vigorous exercise and alcohol consumption in the 24 hours before testing and abstain from caffeine consumption after midday on the day of testing (12:00 h).

Table 4.1. Outlines the components of each device used in this study for assessment of sleep.				
Device	Motionware 8	Go2Sleep Ring	FitBit Inspire 2	Withings Sleep Pad
				
Accelerometer type	Tri-axial (samples at 50 Hz)	Triaxial (sampling frequency not documented)	Triaxial (sampling frequency not documented)	No accelerometer in the device
Additional sensors		<ul style="list-style-type: none"> • Photoplethysmogram (optical heart sensor) • Infrared light (blood oxygenation) 	<ul style="list-style-type: none"> • Photoplethysmogram (optical heart sensor) 	<ul style="list-style-type: none"> • Ballistocardiograph (heart rate) • Ultrasonic sensor (monitor body movement) • Pressure sensors (breathing patterns)
Detection of sleep	Movement counts and light intensity (lux) in one second epochs	Heart rate, movement and proprietary algorithm	Heart rate, movement and proprietary algorithm	Heart rate, movement and proprietary algorithm
Data extraction	Motionware software	Bluetooth	Bluetooth	Bluetooth or Wifi

4.2.3 Polysomnography recording

To obtain overnight PSG recordings a wireless PSG recorder was used with the associated DOMINO software (SOMNOMedics, GmbHTM, Germany). Details of the PSG montage are explained in Chapter 3, section 3.3.1.

4.2.4 Statistical Analysis

To examine the effects of experimental conditions and sleep devices, a generalized mixed model was conducted with gamma distribution. Fixed effects included experimental condition (Home, N1 and N2) and sleep device (PSG, GS2, FBI2, ActiWatch) and random effects were included to account for individual differences. To extract data from the wearable devices (GS2 and FBI2) the relevant mobile applications were used (SLEEPON Version V2.4 and FitBit Inc., San Francisco, California, USA). Refer to Chapter 3, section 3.4 of General Methods for further detail on the statistical analysis.

4.3 Results

4.3.1 Sleep variables

4.3.2 Sleep efficiency

The model explains 77.5% of the variance due to fixed effects (marginal $R^2 = 0.775$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition ($P = 0.029$, Table 7.2), with the greatest sleep efficiency reported for N2 compared to both N1 and Home conditions ($88.2 \pm 6.0\%$, $87.6 \pm 6.6\%$ and $85.9 \pm 7.5\%$, respectively). There was also a significant effect for sleep devices ($P < 0.001$), with actigraphy, GS2 and FBI2 underestimating SE compared to PSG (mean difference compared to PSG: 3.96, 3.44 and 11.96 %, respectively). There was no interaction between condition and device ($P = 0.680$).

4.3.3 Total sleep time

The model explains 24.4% of the variance due to fixed effects (marginal $R^2 = 0.244$) and 33.3% when including random effects (conditional $R^2 = 0.333$). There was no significant difference for condition, with similar total sleep durations reported across Home, N1 and N2 experimental conditions ($P = 0.411$, Table 7.2). However, there was a significant effect for sleep device ($P < 0.001$), with actigraphy, GS2 and FBI2 underestimating values when compared to PSG (MD: 00:36:50, 00:07:46 and 00:13:16 hh:mm:ss, respectively). There was no interaction between condition and device ($P = 0.668$).

4.3.4 Time in bed

The model explains 23.7% of the variance due to fixed effects (marginal $R^2 = 0.237$) and 31.0% when including random effects (conditional $R^2 = 0.310$). There was no significant effect for condition for time in bed ($P = 0.713$, Table 7.2), with similar values across the three experimental conditions. There was a significant effect for device ($P < 0.001$), where actigraphy and FBI2 devices overestimated compared to PSG (MD: 22:24 and 20:54 mm:ss, respectively). No significant interaction was reported between condition and device ($P = 0.845$).

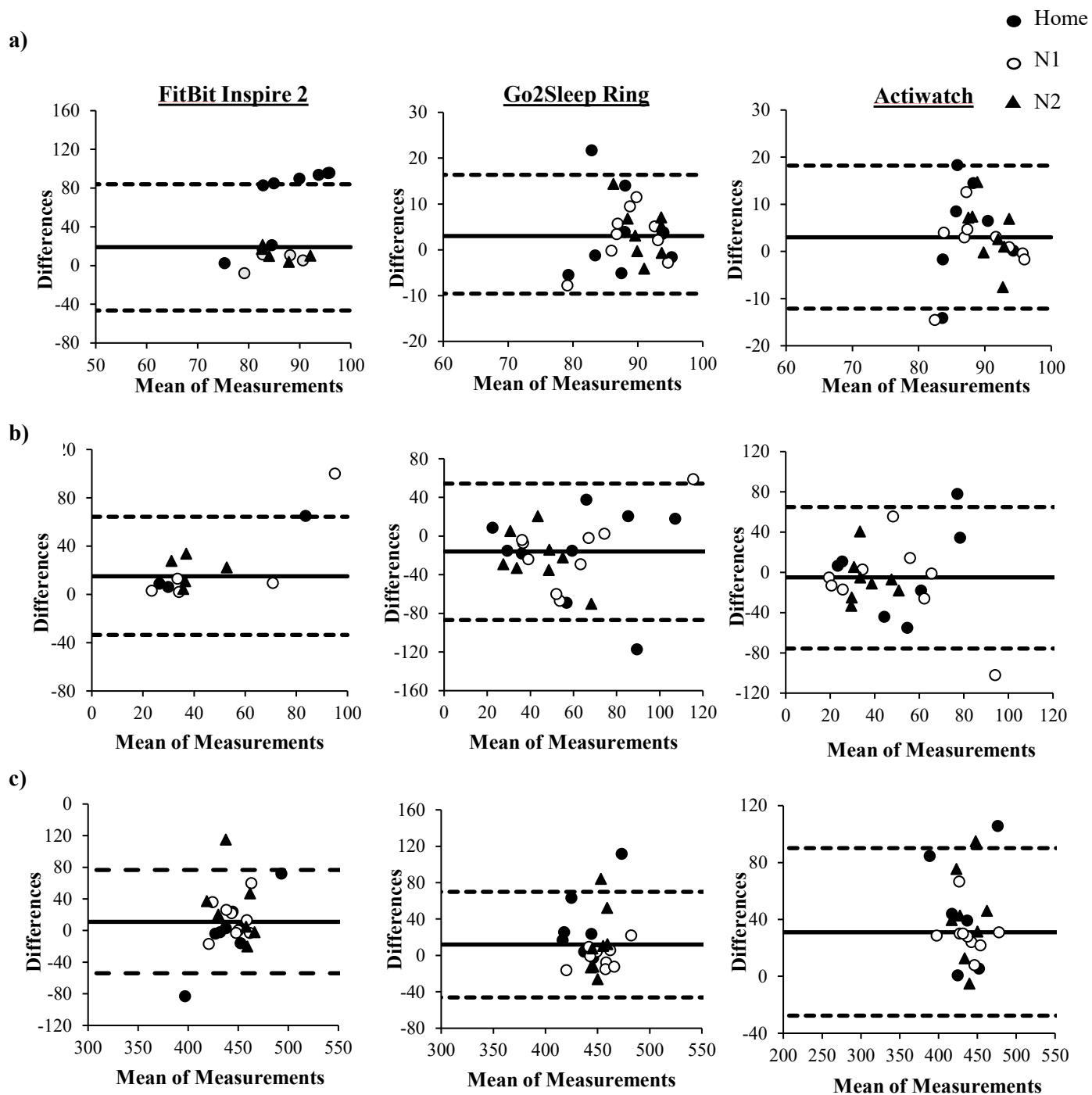


Figure 4.2. Bland-Altman plots depict the mean bias (solid black line) and upper and lower limits of agreement (dashed lines) for mean differences between polysomnography and each device (y-axis; FitBit, Go2Sleep ring, actigraphy) for the following variables **a)** sleep efficiency (%), **b)** wake bouts (min), **c)** total sleep time (min). The plots representing FitBit data have reduced data points due to missing data.

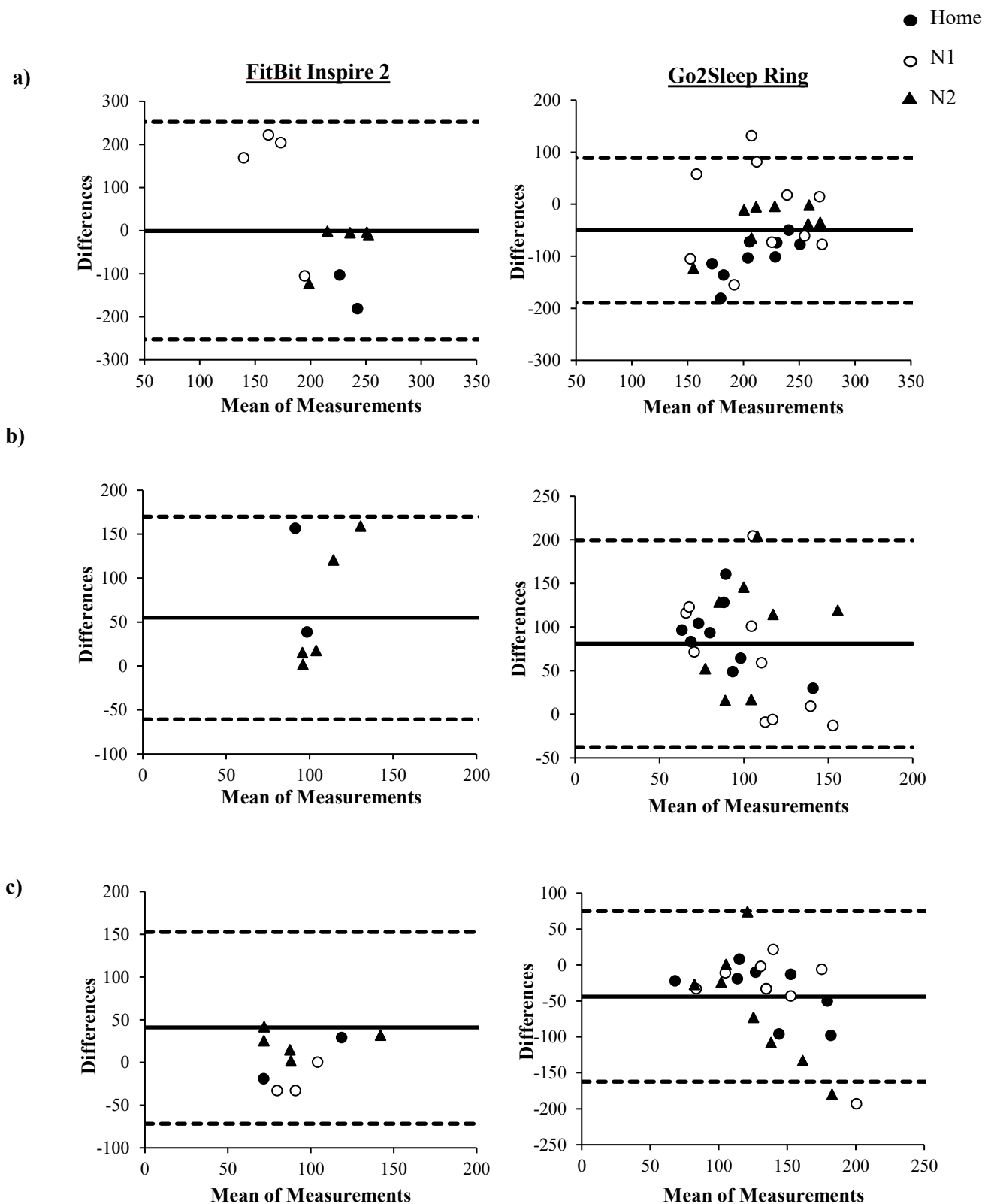


Figure 4.3. Bland-Altman plots depict the mean bias (solid black line) and upper and lower limits of agreement (dashed lines) for mean differences between polysomnography and each device (y-axis; FitBit and Go2Sleep ring) for the following variables **a)** light sleep (min), **b)** deep sleep (min), **c)** REM sleep (min). The plots representing FitBit data have reduced data points due to missing data.

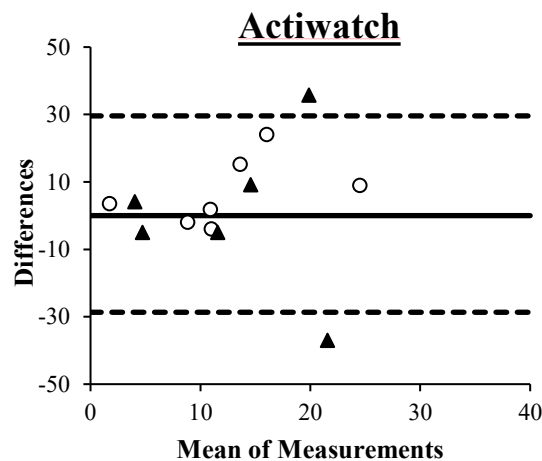


Figure 4.4. Bland-Altman plots depict the mean bias (solid black line) and upper and lower limits of agreement (dashed lines) for mean differences between polysomnography and actigraphy (y-axis) for measurements of sleep latency (min).

4.3.5 Wake after sleep onset (WASO)

The model explains 76.4% of the variance due to fixed effects (marginal $R^2 = 0.764$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant effect for condition for wake time ($P < 0.001$, see Table 7.2), with the greatest time spent awake in the home condition ($54:56 \pm 32:20$ mm:ss) compared to N1

(51:00 ± 27:54 mm:ss) and N2 (42:15 ± 18:52 mm:ss). A significant effect for device ($P < 0.001$) was found, where the GS2 significantly overestimated wake time compared to PSG (MD: 20:31 mm:ss, 95% CI: 17:36 - 22:50 mm:ss) and FBI2 underestimating compared to PSG (MD: 05:33, 95% CI: 2:41 – 08:24 mm:ss). There was a significant interaction between condition and device ($P < 0.001$).

4.3.6 Sleep latency

The model explains 15.2% of the variance due to fixed effects (marginal $R^2 = 0.152$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition ($P = 0.039$, see Table 2), where sleep latency was significantly shorter in N2 condition compared to home (MD: 03:44 mm:ss, 95% CI: 00:51 – 06:36 mm:ss, $P = 0.033$). No significant main effect between PSG and actigraphy devices ($P = 0.934$) was reported and no interaction between condition and device ($P = 0.076$).

4.3.7 Light sleep

The model explains 66.3% of the variance due to fixed effects (marginal $R^2 = 0.663$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition ($P < 0.001$, Table 7.2), with light sleep significantly shorter in the home condition compared to N1 (MD: 07:12 mm:ss, 95% CI: 02:32-11:53, $P = 0.008$) and N2 condition (MD: 17:25, 95% CI: 12:51-22:00 mm:ss, $P < 0.001$). There was a significant effect for device ($P < 0.001$), where the FBI2 overestimated the quantity of light sleep compared to PSG (MD: 01:18:51, 95% CI: 01:12:35 – 01:25:07 hh:mm:ss; see Table 7.2). A significant interaction was found between condition and device ($P < 0.001$). Across all three conditions the FBI2 values were highly variable. Whereas PSG was comparable between home and N2 yet significantly different for N1. In contrast the GS2 achieved similar values between home and N1 ($P = 1.000$) but differed significantly on N2.

4.3.8 Deep sleep

The model explains 85.5% of the variance due to fixed effects (marginal $R^2 = 0.858$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant effect for condition ($P < 0.001$) with less deep sleep achieved in the home condition when compared to N1 (MD: 12:52, 95% CI: 08:55-16:49 mm:ss, $P < 0.001$) and N2 (MD: 18:33, 95% CI: 14:56-22:10 mm:ss, $P < 0.001$). There was a significant main effect for device ($P <$

0.001) with the GS2 and FBI2 significantly underestimating duration of deep sleep compared to PSG (MD: 01:46:17, 95% CI: 01:40:43 - 01:51:51 hh:mm:ss, $P < 0.001$ and MD:01:16:22, 95% CI: 01:12:29-01:20:16 hh:mm:ss, $P < 0.001$; respectively). We report a significant interaction between condition and device ($P < 0.001$).

4.3.9 REM Sleep

The model explains 80.9% of the variance due to fixed effects (marginal $R^2 = 0.809$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect of condition ($P < 0.001$, see Table 7.2), with greater REM sleep in the home condition compared to N1 (MD: 11:10 mm:ss, 95% CI: 05:13-17:02 mm:ss, $P < 0.001$). There was also significantly greater REM sleep in N1 compared to N2 conditions (MD: 11:53 mm:ss, 95% CI: 05:23-18:24 mm:ss, $P = 0.001$). Between devices there was a significant effect ($P < 0.001$) with the GS2 overestimating REM sleep compared to PSG (MD: 01:04, 95% CI: 00:59-01:08 hh:mm, $P < 0.001$) and the FBI2 underestimating REM compared to PSG (MD: 08:08, 95% CI: 04:52-11:24 mm:ss, $P < 0.001$). There was a significant interaction between condition and device ($P < 0.001$).

Table 4.2: Summary of chi-squared (χ^2), degrees of freedom (df) and p values (P) from the generalized mixed model analysis. **Bold** values indicate significance (P < 0.05)

	Condition			Device			Interaction (Condition*Device)		
	χ^2	df	P	χ^2	df	P	χ^2	df	P
Sleep Efficiency	7.10	2.00	0.029	38.81	3.00	< 0.001	3.97	6.00	0.680
Sleep Latency	6.47	2.00	0.039	0.01	1.00	0.934	5.15	2.00	0.076
Total sleep time	1.78	2.00	0.411	35.99	3.00	< 0.001	4.06	6.00	0.668
Time in bed	0.68	2.00	0.713	35.91	3.00	< 0.001	2.70	6.00	0.845
Wake bouts	82.10	2.00	< 0.001	244.20	3.00	< 0.001	282.00	6.00	< 0.001
REM	15.10	2.00	< 0.001	769.10	2.00	< 0.001	569.60	4.00	< 0.001
Light Sleep	56.60	2.00	< 0.001	725.10	2.00	< 0.001	1417.70	4.00	< 0.001
Deep Sleep	129.00	2.00	< 0.001	2539.00	2.00	< 0.001	330.00	4.00	< 0.001

Table 4.3 Summary of results for sleep variables measured by each device. Values are presented for the three experimental conditions (Home, N1, N2). Mean values are presented for each device. Bias represents the mean difference between PSG and the device, the positive bias indicating overestimation and negative bias indicating underestimation compared to PSG. Lower and upper limits of agreement are presented

Device (Mean)				Bias		Lower limit of agreement			Upper limit of agreement			
Home	N1	N2	Home	N1	N2	Home	N1	N2	Home	N1	N2	
Total sleep time (hh:mm:ss)												
FBI2	07:22:00	07:17:40	07:12:45	20.8	18.1	25.0	-32.9	-14.0	-34.0	78.1	53.7	91.2
GS2	07:03:00	06:48:24	07:24:07	25.0	-1.1	11.5	-41.0	-18.5	-49.8	91.0	16.4	72.8
Actiwatch	06:49:00	07:02:40	06:56:15	28.0	26.9	33.8	-45.4	-5.6	-28.1	101.4	59.4	95.8
Sleep efficiency (%)												
FBI2	74.0	82.7	79.6	70.8	4.9	12.4	2.8	-10.2	0.3	138.7	20.0	24.5
GS2	86.1	87.6	88.8	3.7	2.9	3.9	-12.9	-8.1	-6.7	20.3	14.0	14.5
Actiwatch	85.1	86.8	88.7	3.2	1.3	4.0	-13.8	-12.0	-8.2	20.3	14.6	16.2
Wake bouts (hh:mm)												
FBI2	00:35:20	00:42:12	00:41:48	8.0	25.5	19.9	-17.1	-34.9	-6.6	45.8	99.0	40.9
GS2	01:02:36	01:00:18	01:00:18	9.0	-22.6	-22.0	-84.5	-83.6	-72.5	103.5	54.3	54.3
ActiWatch	00:51:00	00:42:13	00:40:00	1.9	-10.1	-6.6	-81.7	-87.3	-48.0	85.4	67.1	34.8

Table 4.3 continued.

	Device (Mean ± SD)				Bias		Lower limit of agreement			Upper limit of agreement		
	Home	N1	N2	Home	N1	N2	Home	N1	N2	Home	N1	N2
Light sleep (hh:mm:ss)												
FBI2	05:05:30	01:46:00	04:05:00	-142.0	123.0	-29.0	-218.4	-137.3	-121.3	467.1	383.3	63.3
GS2	03:35:47	03:41:54	03:57:00	32.6	68.9	72.0	-24.0	-18.4	14.3	89.2	156.2	129.7
Deep sleep (hh:mm:ss)												
FBI2	00:46:00	01:04:30	01:16:36	97.6	90.6	62.9	-17.8	31.4	-62.9	213.0	149.8	188.6
GS2	00:43:20	01:18:48	00:54:45	89.9	99.6	79.7	16.2	-35.7	-21.6	163.6	199.1	220.8
REM Sleep												
FBI2	01:32:30	01:42:20	01:27:53	23.9	21.8	23.2	13.8	-8.1	-4.0	33.9	51.8	50.3
GS2	02:47:40	03:00:30	02:38:37	75.7	85.8	81.3	-13.5	-34.1	-25.2	164.9	205.7	187.8
Sleep latency												
Actiwatch	16:30	09:00	13:23	9.1	8.6	15.9	-8.0	-6.6	-12.7	26.6	23.7	44.4

4.3.10 Psychological measures (pre sleep and upon waking)

There was no significant main effect for condition for responses to the Perceived Stress Scale ($P = 0.361$), Profile of Mood States ($P > 0.05$), Waterhouse questionnaire ($P > 0.05$) or Stanford Sleepiness ($P = 0.952$; see Table 4.4).

Table 4.4 Mean \pm SD, F and P values for all psychological variables measured in the study: Stanford Sleepiness, Waterhouse Questionnaire, Perceived Stress Scale and Profile of Mood States (POMS).

Variables	Home	N1	N2	Significance Condition
Stanford Sleepiness	3.4 \pm 1.1	3.4 \pm 0.7	3.3 \pm 1.0	$F_{2,0, 16,0} = 0.05$ ($P = 0.952$)
Waterhouse				
Q1: How easily did you get to sleep?	-0.2 \pm 1.7	0.0 \pm 2.1	-0.3 \pm 2.2	$F_{1,8, 16,9} = 0.08$ ($P = 0.918$)
Q2: What time did you get to sleep?	-0.0 \pm 1.6	-0.2 \pm 1.8	-0.1 \pm 1.5	$F_{1,3, 11,6} = 0.07$ ($P = 0.851$)
Q3: How well did you sleep?	-0.6 \pm 1.8	0.1 \pm 2.7	-0.5 \pm 2.1	$F_{1,3, 11,9} = 0.45$ ($P = 0.568$)
Q4: What was your waking time?	-0.2 \pm 1.3	-0.2 \pm 1.5	0.0 \pm 0.9	$F_{1,4, 13,0} = 0.18$ ($P = 0.763$)
Q5: How alert did you feel after 30 minutes of waking?	-0.4 \pm 1.3	-0.5 \pm 1.6	-0.5 \pm 1.4	$F_{2,0, 18,0} = 0.03$ ($P = 0.967$)
Perceived stress scale	17.0 \pm 2.6	17.5 \pm 1.9	18.4 \pm 2.4	$F_{2,0, 18,0} = 1.08$ ($P = 0.361$)
Profile of Mood States				
Vigour	6.0 \pm 2.0	6.0 \pm 2.0	5.0 \pm 3.0	$F_{1,5, 13,9} = 1.55$ ($P = 0.244$)
Anger	0.0 \pm 0.0	0.0 \pm 0.0	1.0 \pm 1.0	$F_{1,4, 12,6} = 4.17$ ($P = 0.052$)
Tension	1.0 \pm 1.0	1.0 \pm 2.0	1.0 \pm 2.0	$F_{2,0, 18,0} = 0.15$ ($P = 0.862$)
Calm	11.0 \pm 3.0	10.0 \pm 2.0	11.0 \pm 3.0	$F_{1,9, 17,0} = 1.43$ ($P = 0.265$)
Happiness	10.0 \pm 3.0	8.0 \pm 3.0	9.0 \pm 3.0	$F_{10,5, 18} = 1.66$ ($P = 0.219$)
Confusion	1.0 \pm 1.0	1.0 \pm 1.0	1.0 \pm 2.0	$F_{1,5, 13,9} = 0.23$ ($P = 0.777$)
Depression	0.0 \pm 0.0	0.0 \pm 1.0	0.0 \pm 0.0	$F_{1,5, 13,5} = 0.86$ ($P = 0.414$)
Fatigue	5.0 \pm 2.0	6.0 \pm 3.0	6.0 \pm 2.0	$F_{2,0, 18,0} = 1.07$ ($P = 0.363$)

4.4 Discussion

The aim was to assess the accuracy of consumer and research grade sleep devices against PSG when given an 8-hour opportunity to sleep. Consumer devices accurately detected total sleep time (TST) but their ability to specify sleep staging was relatively poor. Mixed results were found, with consumer devices (FBI2 and GS2) performing as well as, or better than the research validated device for some measures. Another aim was to compare participants sleep in their home environment versus the laboratory. While objective measures differed, no differences were found for subjective measures of emotional state and sleep quality.

Due to the growth and advancement of consumer devices for measurement of sleep, there have been numerous studies conducted in previous years. However, the accuracy of each device differs dependent on the sleep metrics you wish to obtain, therefore it may be too premature to recommend these devices for research and clinical purposes (Evenson et al. 2015). Amongst the most fundamental metrics of sleep, actigraphy significantly underestimated TST by an average of 37 minutes compared to PSG. While actigraphy watches are widely used in research and clinical settings for their high reliability estimating TST, other studies have also reported an overestimation due to difficulty detecting brief awakenings (Montgomery-Downs et al, 2012). Interestingly, the GS2 and FBI2 had good agreement with PSG for TST ($P = 1.00$ and $P = 0.289$, respectively), outperforming actigraphy. Mantua and Colleagues (2016) found that FitBit devices reported no difference to PSG for measures of TST in healthy populations but experienced the greatest device failures. Including issues regarding device initiation and data retrieval due to software errors. The issues with FitBit devices have been previously documented and despite contacting FitBit for support they are often unresponsive or unable to rectify the issue (Baroni et al. 2016). This precluded the use of this device for research purposes.

For measures of sleep efficiency, FBI2 significantly underestimated values compared to PSG by an average of 11%, as did actigraphy but by smaller margins of 4%. Surprisingly, the GS2 did not differ to PSG for measures of SE, TIB or TST (see Table 7.1 and 7.2). Hence, the GS2 may be a promising consumer alternative for basic measures of sleep duration and quality, although few studies to date have assessed the GS2 compared to PSG. A recent study that compared GS2 to the Oura and Circul ring, found the GS2 and Oura to perform similar for sleep/wake classification in healthy individuals (Herberger et al. 2025). However large variation between individuals were very apparent, therefore the authors stated that between ring estimates with PSG values were too large for clinical application. Despite these other studies that have compared the Oura to PSG in healthy

populations, have reported values in agreeable ranges (de Zambotti et al. 2019b; Svensson et al. 2024). Therefore, further validation of sleep ring devices is warranted.

Sleep latency is another key parameter for assessment of sleep despite difficulty detecting it (de Souza et al. 2003). Sleep diaries are typically used to determine ‘lights off’ in addition to an objective measure, however actigraphy has shown to systematically under and overestimate SL compared to PSG (Chae et al. 2009; Sadeh, 2011). In the current study no differences were found between PSG and actigraphy for SL which suggests a promising alternative for determining this measure of sleep. To determine WASO, the accelerometer component in actigraphy and consumer devices often leads to a high sensitivity yet low specificity to detect wake bouts during sleep as the analysis is determined by movement (Sadeh, 2011; see Table 7.1). Differences between these devices and PSG for measures of WASO tend to be more pronounced when participants experience greater sleep fragmentation, as illustrated in the Bland-Altman plots (Figure 7.2). This pattern of dispersion has been consistently reported in previous studies (de Zambotti et al. 2015, Mantua et al. 2016; Chinoy et al. 2021).

To assess sleep stages, the binary nature of actigraphy limits the device to wake and sleep classification opposed to individual stages (Marino et al. 2013). Consumer devices typically report a sleep stage summary but often have difficulty distinguishing between REM and deep sleep, as both stages are characterised by reduced movement (de Zambotti et al. 2024). In agreement, the GS2 significantly overestimated REM sleep compared to PSG by 65 minutes, whereas FBI2 underestimated by 7 min (see Table 7.2). This was also apparent for deep sleep where both GS2 and FBI2 underestimated values compared to PSG by an average of 01:22 and 01:15 hh:mm, respectively. For light sleep, only the FBI2 differed to PSG values by 21%, which has been demonstrated previously (Lim et al. 2023; de Zambotti et al. 2024). Most consumer devices estimate sleep stages using limited physiological signals such as heart rate, heart rate variability or movement and sleep onset (see Table 7.1). This poses a great challenge to confidently classify each stage (Roomkham et al. 2018). The lack of EEG, EOG and EMG signals, used for PSG to give insight on characteristics such as spindle activity, eye movements and muscle tone, are vital for sleep stage classification. The majority of consumer sleep devices lack essential components such as brain activity and electrical signal measurement which are necessary to achieve high comparison with PSG (Rentz et al. 2021). Devices such as sleep headbands and ear worn devices, use EEG electrodes and show promising results (Arnal et al. 2020). However, there are currently no established standards or guidelines that

outline or define the sleep metrics required to deem a consumer device valid, contributing to variation across sleep devices. If clear standards were created it may encourage alternative approaches in product development (Evenson et al. 2015).

Recent advances in ambulatory PSG aim to measure sleep out of the laboratory to offer a cost-effective approach and assess sleep in real-life settings. Traditionally PSG is conducted in clinical environments which may cause a ‘first night effect’ where sleep is disrupted until the participant habituates to the unfamiliar setting (Samson, 2021). Of the limited studies that have compared PSG in the home versus laboratory, outcomes often favour the home with greater sleep efficiency, longer sleep durations and shorter sleep latency (Johns et al. 1976; Bruyneel et al. 2011). In the current study all sleep variables, except TST and TIB, significantly differed between conditions despite a counterbalanced design. Contrary to the original hypothesis, greater sleep efficiency, less awakenings and shorter sleep latency were reported in the laboratory condition (N2) compared to home. Greater awakenings also occurred at home, followed by N1 and the least during N2. This finding supports the hypothesis of a ‘first night effect’ in the laboratory and reiterates the importance of employing a familiarisation condition when assessing sleep in a clinical setting (Tamaki et al. 2005; Rentz et al 2021). Durations of light sleep and deep sleep were greater when participants slept in the laboratory (N1 and N2), with similar durations of REM sleep achieved for home and N2. Overall, the findings suggest that participants slept best on the second night in the laboratory. This is potentially due to the participant population residing in student or shared accommodation at the time of the study. The windowless, soundproof laboratory may reduce these environment disruptions to sleep (Altun et al. 2012). Despite this, the subjective data collected upon waking regarding prior night’s sleep, did not identify any differences between conditions (Table 7.3). The discrepancy between objective and subjective findings may be due to individual differences in perceiving ‘sleep quality’ as there is no standard definition. Therefore, global ratings of sleep quality may refer to different aspects of sleep for different people (Krystal and Edinger, 2008).

4.4.1 Conclusion

Collectively the findings suggest that wearable sleep devices are a promising alternative to actigraphy and PSG, though further development is warranted due to substantial variability. Device performance varies dependent on the sleep variable of interest, as certain metrics such as TST, TIB and SE did not differ between PSG and consumer

devices (GS2 and FBI2). However, challenges remain including the ability to detect sleep stages, battery life and device misuse (Chaudry et al. 2020). The lack of clarity surrounding manufacturer algorithms and the ability of these devices to approximate gold standard measures remain a concern. Further development to improve the validity of sleep tracking devices are encouraged as they typically offer lower costs, accessible user interface and do not require specialist input (Montgomery-Downs et al. 2012). Regarding the sleep environment the findings support the implementation of a familiarisation night when measuring sleep in a clinical setting to allow the individual to adjust to the unfamiliar environment (Ameen et al. 2019). The findings also suggest that if a prior familiarisation night is given, realistic insights on an individual's sleep can be obtained in the laboratory. However, these outcomes may vary across different age groups (children and older adults) and populations (clinical).

4.4.2 Strengths and limitations

Sleep was also assessed in the laboratory and the participants home, with additional subjective measures. This is quite uncommon as most studies typically conduct overnight sleep research in a laboratory. It is acknowledged that there are limitations of the current study. The sample size was $n=11$ participants, initially it was estimated that $n=16$ was required based on the ES for total sleep time (Chinoy et al. 2021; see Table 3.1). Interestingly, when sample size estimation was undertaken using the current studies familiarisation data (rather than others work, where random variation and systematic bias may be different), $n=3$ was calculated. Regardless, due to the challenging nature of the study design such large sample sizes are difficult to recruit. Missing data due to software errors may have also compromised the results and increased bias. The impact of missing data on the outcomes was further amplified by the small sample. Particularly regarding the FitBit device where the majority of data was absent due to issues with the mobile application despite trying to seek technical support from FitBit on multiple occasions. Furthermore, the findings are limited to a small population, as all the participants were male and of a similar age. Future studies should encourage more diverse populations to include different age groups and both sexes.

Chapter 5:

Is implementing a post-lunch nap beneficial on evening performance, following two nights partial sleep restriction?

The work has been published in Chronobiology International (Gallagher et al. 2023). This chapter explores the effectiveness of different nap durations on maximal and submaximal performance in healthy resistance trained individuals, following acute sleep restriction.

5.1 Introduction

Partial sleep restriction (restricted but not complete elimination of habitual sleep within a 24 h period), is a common occurrence in society, with 45% of the western population failing to obtain the recommended 7–9 h per night (Bambaeichi et al. 2005; Craven et al. 2022). The susceptibility and prevalence of athletes experiencing poor sleep is discussed in Chapters 1 and 2 (Sargent et al. 2014; Simpson et al. 2017; Walsh et al. 2021). Refer to Chapter 2, section 2.2 on the fundamental importance of the sleep-wake cycle (Reilly and Edwards, 2007; Souissi et al. 2008).

Previous research suggests that when partial sleep loss is employed over multiple nights, impairments on weightlifting performance are more pronounced on the second and third day of sleep loss. This suggests that tasks requiring greater activation and of larger muscle groups, are more susceptible to sleep loss (Bambaeichi et al. 2005; Reilly and Piercy, 1994; Thun et al. 2015). These differences along with an increase in homeostatic pressure and accumulation of sleep propensity over the course of a day, signifies the importance of scheduling exercise sessions, to ensure optimal performance outcomes (Jarraya et al. 2013). A possible intervention strategy to combat adverse effects of sleep loss is the implementation of a nap, a safe and non-invasive intervention that can help increase total sleep time over the 24 h period. It has been reported that 43% of athletes already use some form of napping, however timing of the nap can be very difficult due to training and competition schedules (Lastella et al. 2015; Romyne et al. 2018). Literature suggests that an afternoon nap between 13:00-15:00 h, lasting between 20-60 min in duration should be encouraged, as this is when there is a transient fall in alertness and core temperature values decrease (Brotherton et al. 2019; Waterhouse et al. 2007).

Therefore, the purpose of the present study was to: (1) determine the physiological and psychological effects on muscle strength measures when partially sleep restricted (4 h per night PSR, over two consecutive nights). As well as changes in mood state, cognitive abilities, intra-aural temperature, tiredness, sleepiness and alertness subjective values. (2) To investigate the effectiveness of a 30 versus 60-min post lunch nap (PSR₀ versus PSR₃₀ versus PSR₆₀), and whether it will improve evening physiological and subjective psychological measures.

5.2 Method

5.2.1 Participants

Fifteen males, as identified by sex and gender, participated in the study. Please refer to ‘General Methods’, Chapter 3.1 for participant characteristics and inclusion criteria. All participants gave their written informed consent (Ethics code: M19_SPS_140).

5.2.2 Research Design

Each participant attended the laboratory on seven occasions (dry temperature of 19°C, 35–45% humidity and a barometric pressure of 750–760 mmHg, respectively). Experimental conditions consisted of retiring to sleep at 02:30 and rising at 06:30 h, and either followed (PSR₀) no nap, (PSR₃₀) 30-min nap or (PSR₆₀) 60-min nap all commencing at 13:00 (Figure 4.1). When completing the PSR₃₀ and PSR₆₀ experimental conditions, participants were required to sleep/rest on a bed provided in a dark, quiet room in the university sleep laboratory and were not permitted to get up from the bed until the end of the session. At 13:00 h those in the PSR₀ condition were allowed to undertake free living conditions and were instructed not to nap or exercise. All participants were blinded to their assigned nap condition until arrival at the laboratory at 13:00 h to minimise anticipatory effects across experimental conditions. Researchers checked in with participants via direct messages to ensure compliance and participants remained on university campus throughout this time. To ensure recovery between trials there was at least a week between testing conditions for all participants.

5.2.3 Measurements

Following two consecutive nights of sleep restriction (02:30–06:30 h), participants arrived at the laboratory at 07:30, 11:00, 14:00 and 17:00 h for recordings of intra-aural (ear) temperature using a thermometer (Genius 1000, Mark 2, Sherwood, Nottingham, UK); rating of mood (Profile of Mood State questionnaire; Terry et al. 2003) and quality of sleep and sleepiness (Stanford Sleepiness Scale; Hoddes et al. 1973). Across the two-day sleep restriction protocol participants were allowed to follow free living conditions during the daytime, however they were asked to refrain from vigorous exercise and abstain from alcohol and caffeine consumption. Please refer to General Methods, Chapter 3.3 for the further detail on the measurements.

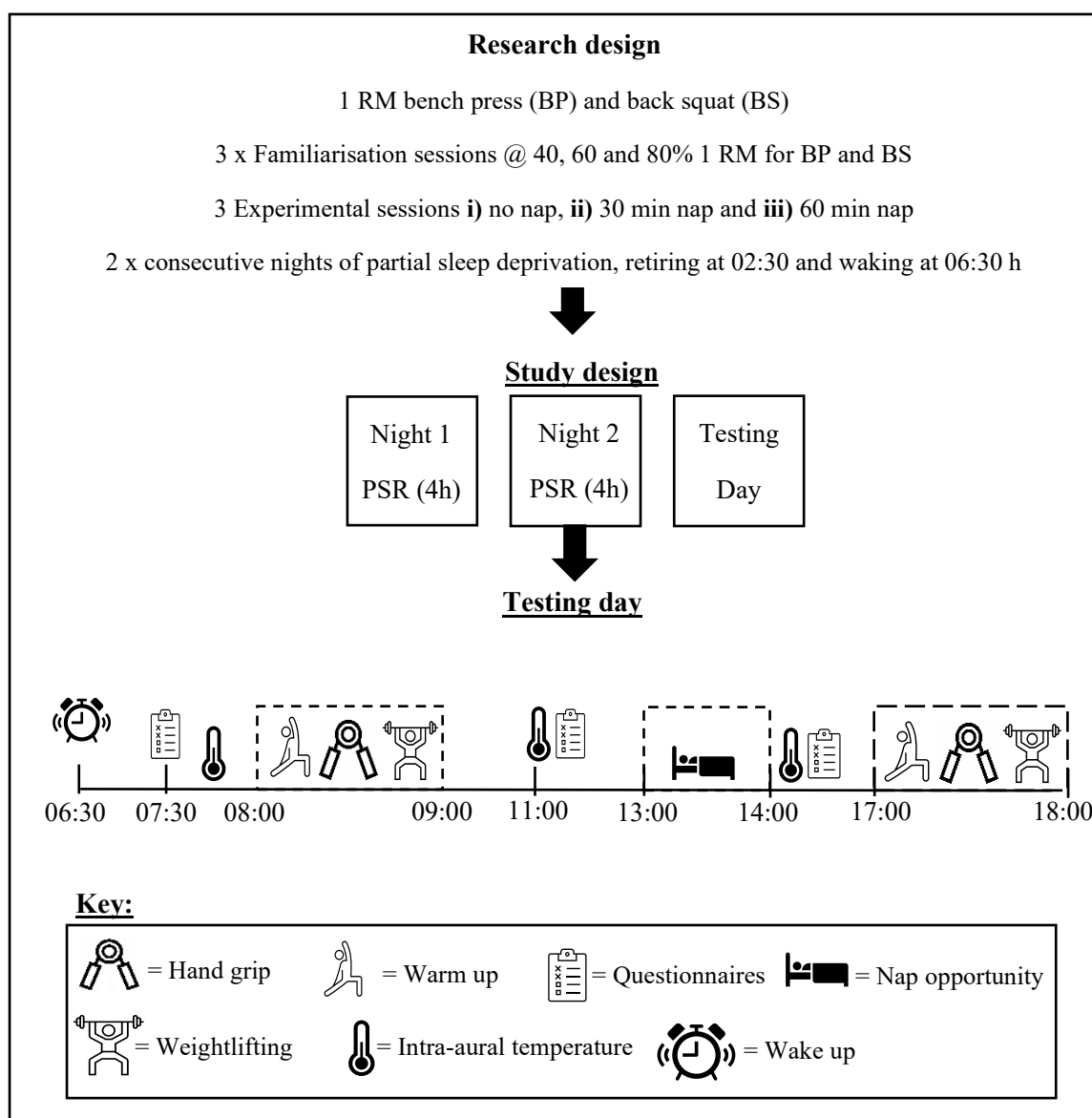


Figure 5.1: Schematic of experimental protocol outlining research design, study design and testing day protocol.

5.2.4 Statistical Analysis

Refer to Chapter 3, section 3.4 of General Methods for detail of the statistical analysis. Values to determine sample size are displayed in Chapter 3, Table 3.2 of the General Methods. Fifteen participants were recruited to account for dropouts.

5.3 Results

5.3.1 Performance measures (measured at 07:30 and 17:00 h)

Mean \pm SD values and the results from the ANOVA statistical analysis are displayed in Table 5.1. Statistical significance of the results can be seen in Figures 5.2 and 5.3.

5.3.2 Grip Strength

There was no significant effect of nap on left or right-hand grip strength values ($P = 0.211$, $P = 0.176$; respectively, Table 5.1). However, there was a significant main effect for time of day for left ($P = 0.018$, $\eta^2_p = 0.33$) and right grip strength ($P = 0.05$, $\eta^2_p = 0.25$), with pairwise comparisons showing greater values at 17:00 compared to 07:00 h for left and right hand (mean difference [MD]: 2.00 Nm^{-1} , 95% CI: $0.39 - 3.61 \text{ Nm}^{-1}$; MD: 1.58 Nm^{-1} , 95% CI: $0.01 - 3.15 \text{ Nm}^{-1}$, respectively).

5.3.3 Bench Press

There was no significant main effect for nap condition for all bench press performance variables (Table 5.1). There was a significant main effect for time of day for PV ($P < 0.05$, $\eta^2_p = 0.54$), where pairwise comparisons showed that participants produced significantly higher PV values at 17:00 h compared to 07:30 h (1.0 ms^{-1} , $P = 0.001$; Figure 5.1). No other bench press variables were significant for time of day. There was a significant main effect of load for all bench press variables measured (Table 5.1). For AP, AV, D and PV, values were highest at 40 % 1RM ($320.1 \pm 22.9 \text{ W}$; $0.81 \pm 0.02 \text{ ms}^{-1}$; $42.9 \pm 1.2 \text{ cm}$; $1.33 \pm 0.05 \text{ ms}^{-1}$, respectively) and lowest at 80 % 1RM ($265.9 \pm 14.9 \text{ W}$; $0.43 \pm 0.02 \text{ ms}^{-1}$; $41.8 \pm 1.9 \text{ cm}$; $0.67 \pm 0.04 \text{ ms}^{-1}$). Whereas tPV was significantly lower at 40 % 1RM ($0.33 \pm 0.01 \text{ ms}^{-1}$) and highest at 80 % 1RM ($0.71 \pm 0.05 \text{ ms}^{-1}$). As expected, there was a corresponding significant main effect for load on subjective effort and RPE values ($P < 0.05$), with 40 % of 1RM eliciting lower subjective values (Effort: 3 ± 0 ; RPE: 9 ± 0 ; RPE Breathing: 8 ± 0 ; RPE Muscle Fatigue: 9 ± 0) and 80 % producing the highest (Effort: 8 ± 0 ; RPE: 15 ± 0 ; RPE Breathing: 12 ± 1 ; RPE Muscle Fatigue: 15 ± 0). There was no significant interaction between condition, time of day and load for any variable, such that the values across all conditions at both time points for the three loads, rose or fell in the same manner (Figure 5.1 and 5.2).

5.3.4 Back Squat

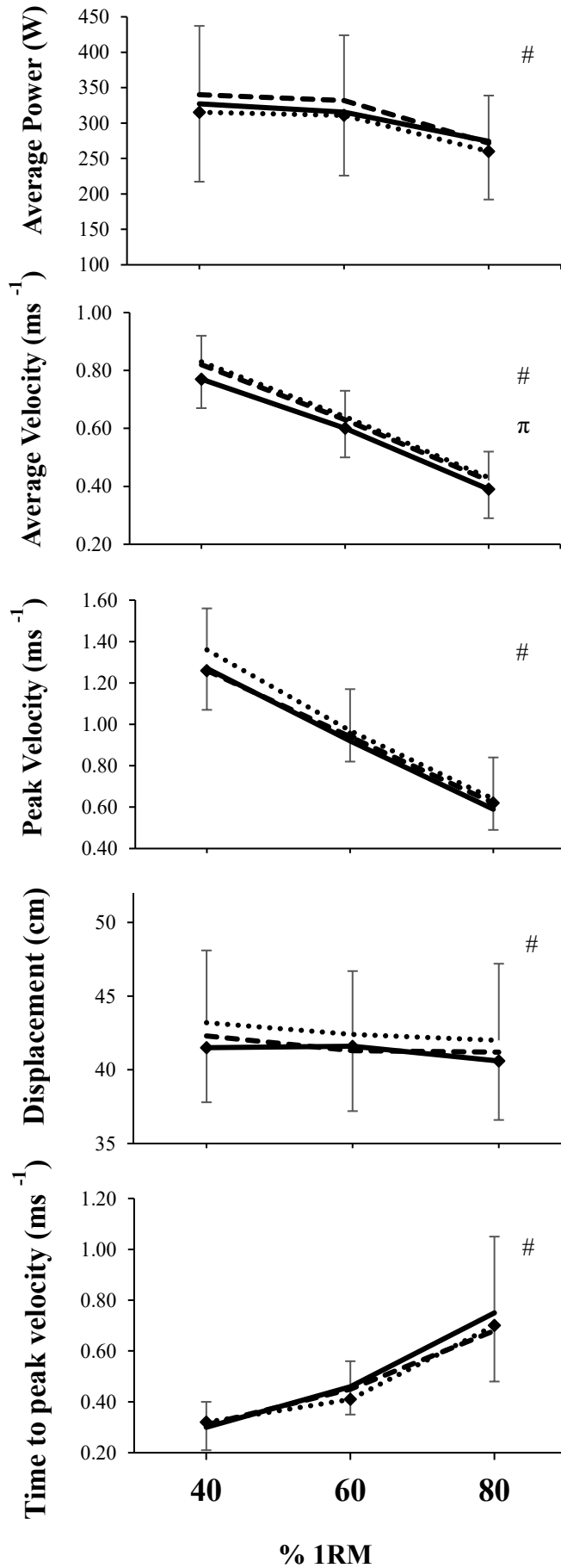
There was no significant main effect for nap condition for all back squat performance variables (Table 5.1). However, there was a significant main effect for time of day for AP ($P = 0.052$, $\eta^2_p = 0.24$), AV ($P = 0.034$, $\eta^2_p = 0.28$), PV ($P = 0.012$, $\eta^2_p = 0.38$) and RPE ($P = 0.02$, $\eta^2_p = 0.32$), yet no significance for time-of-day for D, tPV or perceived effort. Pairwise comparisons showed that participants had significantly lower AP values at 07:30 h (942.0 ± 44.9 W) than at 17:00 h (983.7 ± 48.7 W; MD: 41.7 W). There was a significant main effect for load on all back squat variables, as anticipated tPV, perceived effort, RPE, breathing and muscle fatigue were significantly lower at 40 % and higher at 80 % 1 RM ($P < 0.05$; Table 5.1). Conversely, AP, AV, PV and D were significantly highest at 40 % and lowest at 80 % 1RM ($P < 0.05$). A significant interaction was present between condition and time of day for AV ($P = 0.03$), where values were greater at 17:00 h in the PSR₆₀ than PSR₃₀ and PSR₀ conditions. There were also significant interactions for time of day and load for AV, PV and tPV ($P = 0.03$, $P = 0.02$, $P < 0.05$; respectively), with greater mean values for load at 17:00 compared to 07:00 h.

5.3.5 Physiological and psychological variables (measured at 07:30, 11:00, 14:00 and 17:00 h)

5.3.6 Intra-aural temperature

There was a significant main effect for sleep condition ($P < 0.05$, $\eta^2_p = 0.44$; Table 5.2) on intra-aural temperature, with PSR₀ producing the lowest average values (35.5 ± 0.1), compared to PSR₃₀ (36.0 ± 0.1) and PSR₆₀ (36.2 ± 0.1). There was a significant main effect for time of day ($P = 0.01$, $\eta^2_p = 0.30$) on intra-aural temperature with a drop in temperature between 07:30 h (36.1 ± 0.2) and 11:00 h (35.5 ± 0.2), followed by a progressive rise at 14:00 h (35.9 ± 0.1) and 17:00 h (36.1 ± 0.1).

Bench Press (07:30 h)



Bench Press (17:00 h)

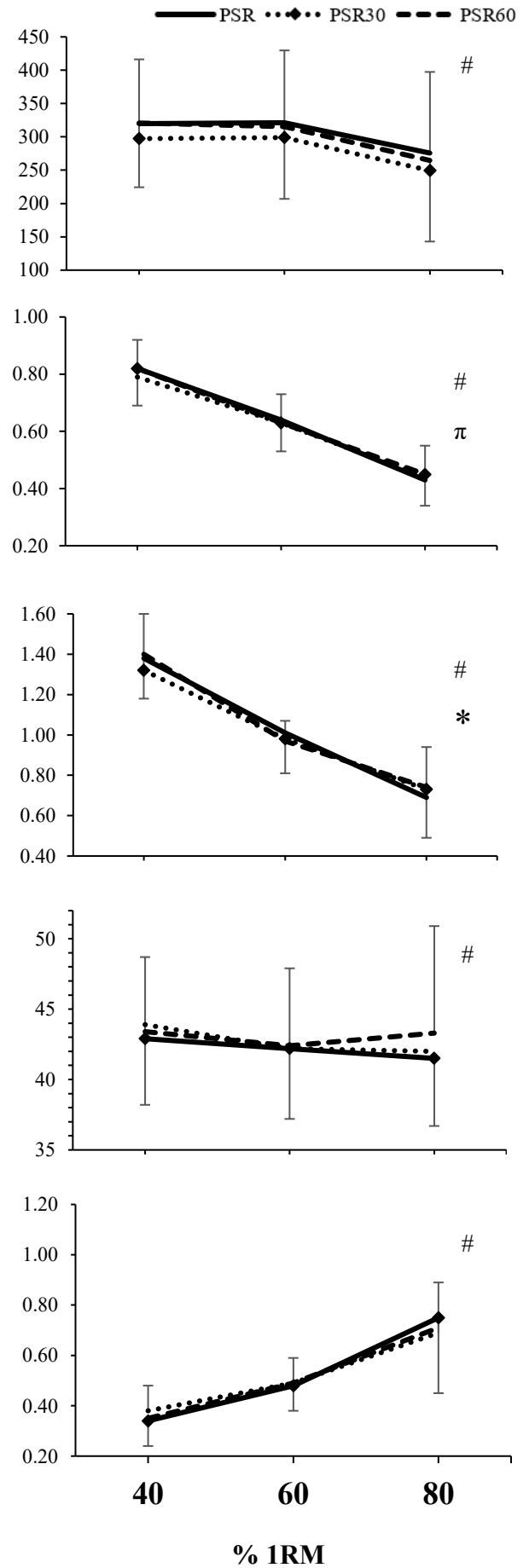
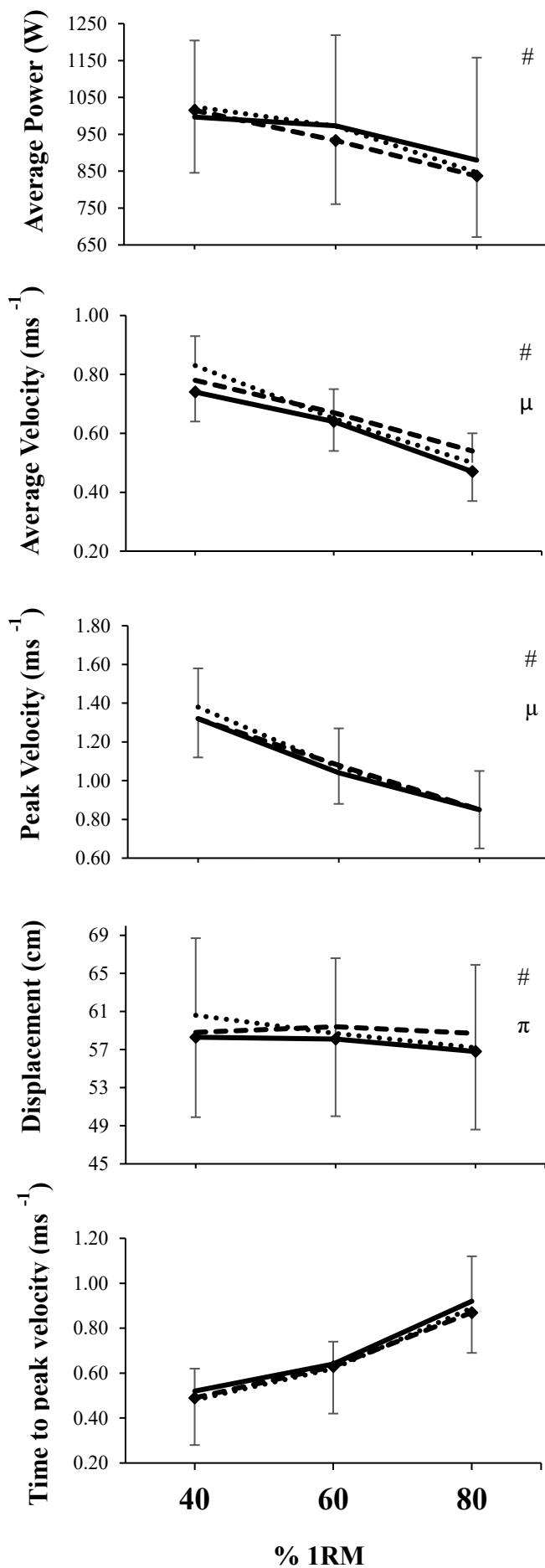


Figure 5.2: Mean \pm SD values of performance variable at 07:30 h and 17:00 h for bench press at 40, 60 and 80% 1RM loads for three experimental conditions. # denotes main effect for load ($P < 0.05$), * denotes main effect for “time-of-day” ($P < 0.05$) and π denotes condition and “time-of-day” interaction.

Back Squat (07:30)



Back Squat (17:00 h)

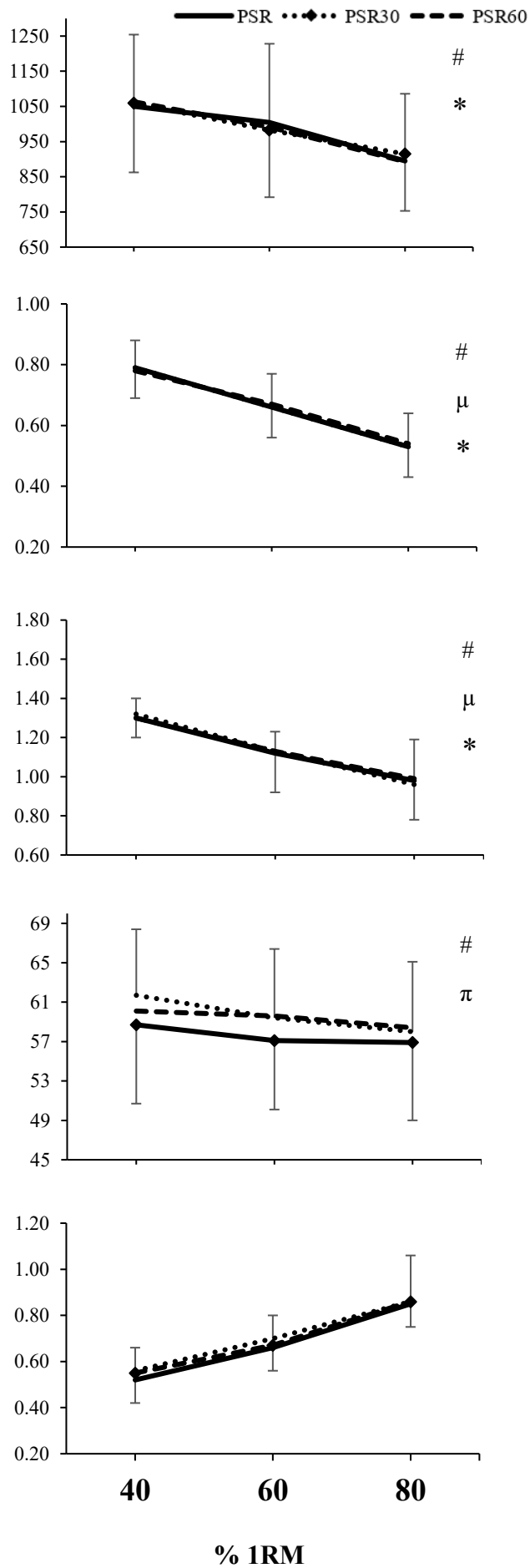


Figure 5.3. Mean \pm SD values of each performance variable for 07:30 h and 17:00 h back squat at 40, 60 and 80% 1RM loads for three experimental conditions. # denotes main effect for load ($P < 0.05$), * main effect for “time-of-day” ($P < 0.05$), π denotes condition and “time-of-day” interaction, μ denotes “time-of-day” and load interaction.

Table 5.1: F and P values for all performance variables measured across experimental conditions and time of day (TOD). RPE (rating of perceived exertion) and visual analog scale (VAS) are presented for grip strength, bench press and back squat. **Bold** values indicate significance ($P < 0.05$).

Variable	Significance Condition	Significance TOD	Significance of Load	Interactions (COND * TOD * LOAD)	COND*TOD	COND*LOAD
Left grip strength (N)	$F_{2,0, 25.8} = 1.66$ ($P = 0.211$)	$F_{1,0, 13,0} = 6.42$ ($P = 0.025$)				
VAS (0-10)	$F_{2,0, 28,0} = 0.31$ ($P = 0.740$)	$F_{1,0, 14,0} = 0.01$ ($P = 0.919$)			$F_{1,9, 26,3} = 0.90$ ($P = 0.415$)	
Right grip strength (N)	$F_{1,4, 20,0} = 1.95$ ($P = 0.176$)	$F_{1,0, 14,0} = 4.61$ ($P = 0.050$)				
VAS (0-10)	$F_{2,0, 28,0} = 0.65$ ($P = 0.531$)	$F_{1,0, 14,0} = 0.58$ ($P = 0.458$)			$F_{1,4, 19,4} = 0.46$ ($P = 0.570$)	
Bench Press Average Power (W)	$F_{2,0, 28,0} = 1.62$ ($P = 0.215$)	$F_{1,0, 14,0} = 1.34$ ($P = 0.266$)	$F_{1,3, 17,8} = 14.94$ ($P < 0.0005$)	$F_{2,0, 27,5} = 0.21$ ($P = 0.806$)	$F_{1,5, 21,4} = 0.41$ ($P = 0.612$)	$F_{2,3, 32,8} = 0.61$ ($P = 0.575$)
Displacement (cm)	$F_{1,5, 20,9} = 0.70$ ($P = 0.467$)	$F_{1,0, 14,0} = 4.32$ ($P = 0.056$)	$F_{1,6, 22,3} = 6.90$ ($P = 0.007$)	$F_{2,1, 29,4} = 0.38$ ($P = 0.696$)	$F_{1,9, 27,1} = 0.60$ ($P = 0.553$)	$F_{2,7, 38,3} = 1.48$ ($P = 0.238$)
Average Velocity (ms^{-1})	$F_{1,5, 21,3} = 1.91$ ($P = 0.179$)	$F_{1,0, 14,0} = 3.69$ ($P = 0.075$)	$F_{1,3, 17,7} = 296.73$ ($P < 0.0005$)	$F_{2,3, 32,0} = 0.52$ ($P = 0.626$)	$F_{2,0, 28,0} = 3.36$ ($P = 0.049$)	$F_{2,1, 29,6} = 0.59$ ($P = 0.568$)
Peak Velocity (m s^{-1})	$F_{2,0, 28,0} = 2.12$ ($P = 0.141$)	$F_{1,0, 14,0} = 16.54$ ($P < 0.0005$)	$F_{1,4, 20,2} = 617.66$ ($P < 0.0005$)	$F_{2,6, 36,1} = 1.20$ ($P = 0.320$)	$F_{2,0, 28,0} = 2.12$ ($P = 0.141$)	$F_{2,4, 34,0} = 0.27$ ($P = 0.809$)
Time to Peak Velocity (s)	$F_{2,0, 28,0} = 0.21$ ($P = 0.811$)	$F_{1,0, 14,0} = 2.59$ ($P = 0.130$)	$F_{1,1, 15,6} = 59.42$ ($P < 0.0005$)	$F_{2,8, 39,2} = 0.47$ ($P = 0.695$)	$F_{2,0, 28,0} = 0.21$ ($P = 0.811$)	$F_{2,1, 29,0} = 1.40$ ($P = 0.262$)
RPE (6-20)	$F_{2,0, 28,0} = 1.27$ ($P = 0.297$)	$F_{1,0, 14,0} = 1.35$ ($P = 0.265$)	$F_{1,4, 19,5} = 132.93$ ($P < 0.0005$)	$F_{2,9, 40,5} = 0.69$ ($P = 0.561$)	$F_{1,8, 25,4} = 0.46$ ($P = 0.618$)	$F_{2,3, 31,9} = 0.56$ ($P = 0.597$)
RPE Breathing (6-20)	$F_{2,0, 28,0} = 0.09$ ($P = 0.911$)	$F_{1,0, 14,0} = 1.06$ ($P = 0.321$)	$F_{1,0, 14,0} = 1.06$ ($P = 0.321$)	$F_{2,1, 31,1} = 0.35$ ($P = 0.727$)	$F_{2,0, 28,0} = 0.18$ ($P = 0.834$)	$F_{2,7, 37,2} = 0.37$ ($P = 0.749$)
RPE Muscle Fatigue (6-20)	$F_{2,0, 28,0} = 1.55$ ($P = 0.230$)	$F_{1,0, 14,0} = 1.23$ ($P = 0.286$)	$F_{1,0, 14,0} = 1.23$ ($P = 0.286$)	$F_{2,9, 40,2} = 2.39$ ($P = 0.086$)	$F_{1,6, 22,1} = 2.26$ ($P = 0.137$)	$F_{2,3, 32,3} = 0.22$ ($P = 0.835$)
VAS (0-10)	$F_{1,6, 21,9} = 0.92$ ($P = 0.392$)	$F_{1,0, 14,0} = 0.25$ ($P = 0.627$)	$F_{1,3, 18,7} = 148.42$ ($P < 0.0005$)	$F_{2,3, 32,2} = 0.82$ ($P = 0.465$)	$F_{2,0, 28,0} = 0.56$ ($P = 0.580$)	$F_{2,8, 39,5} = 1.22$ ($P = 0.315$)

Table 5.1 continued.

Variable	Significance Condition	Significance TOD	Significance of Load	Interactions (COND * TOD * LOAD)	COND*TOD	COND*LOAD
Back Squat						
Average Power (W)	$F_{1,9, 25,9} = 0.12$ ($P = 0.873$)	$F_{1,0, 14,0} = 4.44$ ($P = 0.054$)	$F_{1,2, 17,4} = 54.88$ ($P < 0.0005$)	$F_{3,1, 44,0} = 1.43$ ($P = 0.247$)	$F_{1,2, 16,6} = 0.24$ ($P = 0.685$)	$F_{2,3, 32,5} = 1.04$ ($P = 0.372$)
Displacement (cm)	$F_{2,0, 28,0} = 1.65$ ($P = 0.210$)	$F_{1,0, 14,0} = 0.52$ ($P = 0.483$)	$F_{1,2, 16,8} = 5.30$ (P = 0.029)	$F_{3,3, 45,8} = 0.33$ ($P = 0.819$)	$F_{1,5, 20,3} = 0.24$ ($P = 0.717$)	$F_{2,7, 37,8} = 2.50$ ($P = 0.080$)
Average Velocity (ms⁻¹)	$F_{2,0, 28,0} = 1.03$ ($P = 0.372$)	$F_{1,0, 14,0} = 5.54$ ($P = 0.034$)	$F_{1,1, 16,0} = 182.95$ ($P < 0.0005$)	$F_{2,6, 37,1} = 1.80$ ($P = 0.170$)	$F_{1,8, 24,8} = 4.18$ ($P = 0.031$)	$F_{1,9, 26,9} = 1.05$ ($P = 0.361$)
Peak Velocity (m s⁻¹)	$F_{2,0, 28,0} = 0.21$ ($P = 0.810$)	$F_{1,0, 14,0} = 8.41$ ($P = 0.012$)	$F_{1,2, 16,7} = 110.37$ ($P < 0.0005$)	$F_{2,0, 28,6} = 0.15$ ($P = 0.865$)	$F_{1,5, 20,4} = 0.18$ ($P = 0.765$)	$F_{2,3, 31,8} = 0.84$ ($P = 0.453$)
Time to Peak Velocity (s)	$F_{1,9, 26,4} = 0.06$ ($P = 0.930$)	$F_{1,0, 14,0} = 0.85$ ($P = 0.372$)	$F_{1,1, 15,5} = 112.87$ ($P < 0.0005$)	$F_{2,6, 37,0} = 0.76$ ($P = 0.507$)	$F_{1,9, 26,6} = 1.62$ ($P = 0.217$)	$F_{2,5, 34,3} = 0.46$ ($P = 0.674$)
RPE (6-20)	$F_{2,0, 28,0} = 0.19$ ($P = 0.830$)	$F_{1,0, 14,0} = 6.58$ ($P = 0.022$)	$F_{1,5, 20,5} = 218.55$ ($P < 0.0005$)	$F_{2,9, 40,5} = 0.75$ ($P = 0.523$)	$F_{2,0, 27,4} = 2.23$ ($P = 0.127$)	$F_{3,0, 42,4} = 0.83$ ($P = 0.488$)
RPE Breathing (6-20)	$F_{2,0, 27,8} = 0.23$ ($P = 0.797$)	$F_{1,0, 14,0} = 0.06$ ($P = 0.812$)	$F_{1,4, 18,9} = 81.46$ ($P < 0.0005$)	$F_{3,2, 45,2} = 0.90$ ($P = 0.454$)	$F_{1,9, 26,1} = 0.00$ ($P = 1.000$)	$F_{2,6, 35,8} = 0.57$ ($P = 0.610$)
RPE Muscle Fatigue (6-20)	$F_{2,0, 28,0} = 1.32$ ($P = 0.284$)	$F_{1,0, 14,0} = 1.12$ ($P = 0.309$)	$F_{1,5, 21,5} = 232.77$ ($P < 0.0005$)	$F_{2,5, 34,5} = 1.06$ ($P = 0.369$)	$F_{1,6, 22,0} = 1.51$ ($P = 0.242$)	$F_{2,9, 40,1} = 1.80$ ($P = 0.166$)
VAS (0-10)	$F_{1,8, 25,8} = 0.20$ ($P = 0.805$)	$F_{1,0, 14,0} = 0.37$ ($P = 0.552$)	$F_{1,2, 16,7} = 249.45$ ($P < 0.0005$)	$F_{2,8, 38,8} = 0.48$ ($P = 0.687$)	$F_{1,4, 19,0} = 1.65$ ($P = 0.218$)	$F_{2,6, 35,8} = 0.87$ ($P = 0.451$)

5.3.7 Tiredness and Alertness

There was no significant main effect of nap length on subjective tiredness and alertness ($P > 0.05$, see Table 5.2), indicating the powernap did not have a significant effect on average feelings of alertness and sleepiness; however, there was a significant main effect on time-of-day for both variables ($P < 0.0005$). With tiredness being the reciprocal of alertness, as anticipated subjective tiredness values decreased whereas alertness levels increased from 07:30 h (Alertness: 4 ± 1 , Tiredness: 8 ± 1) to 17:00 h (Alertness: 6 ± 1 , Tiredness: 5 ± 1). No interaction between condition and time-of-day were identified for tiredness ($P = 0.345$) or alertness ($P = 0.685$) values.

5.3.8 Profile of Mood State

Regarding mood, there was no significant effect of condition on all mood profiles, however there was a significant effect of time of day for vigour, happiness, confusion, and fatigue ($P < 0.05$; see Table 5.2). Vigour and happiness were significantly lower in the morning compared to the evening, whereas tiredness and confusion were significantly higher in the morning than the evening.

5.3.9 Stanford Sleepiness and Waterhouse questions

There was a no significant main effect of condition on subjective sleepiness rating, yet there was a significant time of day effect ($P < 0.05$, $\eta^2_p = 0.53$; see Table 5.2) where highest values of sleepiness were reported in the morning at 07:30 h (4 ± 0) and lowest levels at 17:00 h (3 ± 0). There was a significant main effect for condition for Waterhouse question 5 ($P = 0.048$; How alert did you feel after 30 minutes of waking?) with greatest alertness in PSR₆₀ condition compared to PSR₃₀ and PSR₀, with no main effect for the remaining questions.

5.3.10 Stroop task

5.3.11 Incongruent total

There was a significant main effect of condition ($P = 0.02$, $\eta^2_p = 0.29$; see Table 4.2), with PSR₀ achieving the lowest total (60.2 ± 2.3 ; 95% CI = 55.4 – 65.0) with a stepwise increase in PSR₃₀ (62.8 ± 2.0 , 95% CI = 58.5 –

67.1) and PSR₆₀ (66.8 ± 2.7, 95% CI = 61.1 – 72.5; respectively). However, there was no main effect of time-of-day or significant interaction (Table 5.2).

Table 5.2: Mean ± SD, F values and *P* values for all physiological and psychological variables measured in the study across condition and time of day (TOD). **Bold** values indicate significant figures (*P* < 0.05).

Variables	PSR ₀	PSR ₃₀	PSR ₆₀	Significance Condition	Significance TOD	Interaction (Condition*TOD)
Intra-aural Temperature (°C)	35.5 ± 0.1	36.0 ± 0.1	36.2 ± 0.1	F_{2.0, 28.0} = 11.15 (P < 0.0005)	F_{1.9, 27.0} = 6.01 (P = 0.007)	F _{3.0, 41.4} = 0.99 (P = 0.405)
Tiredness (0 – 10 VAS)	5.2 ± 0.8	6.2 ± 0.8	6.1 ± 0.7	F _{2.0, 28.0} = 1.42 (P = 0.260)	F_{2.39, 33.45} = 20.61 (P < 0.0005)	F _{2.48, 34.65} = 0.35 (P = 0.755)
Alertness (0 – 10 VAS)	5.7 ± 0.6	5.1 ± 0.7	4.9 ± 0.6	F _{1.6, 22.1} = 1.46 (P = 0.252)	F_{3.0, 42.0} = 17.46 (P < 0.0005)	F _{4.0, 56.5} = 0.57 (P = 0.685)
Stanford Sleepiness	3.0 ± 0.3	3.4 ± 0.3	3.4 ± 0.3	F _{2.0, 28.0} = 2.59 (P = 0.097)	F_{2.1, 29.7} = 15.54 (P < 0.0005)	F _{3.3, 46.0} = 1.18 (P = 0.329)
POMS						
Vigour	6.1 ± 3.6	5.2 ± 3.9	5.3 ± 3.8	F _{2.0, 28.0} = 1.72 (P = 0.197)	F_{2.4, 34.2} = 18.43 (P < 0.0005)	F _{4.3, 60.7} = 1.23 (P = 0.309)
Anger	1.2 ± 2.2	1.8 ± 0.9	0.9 ± 1.4	F _{1.4, 17.7} = 0.92 (P = 0.383)	F _{1.4, 18.6} = 2.44 (P = 0.127)	F _{2.4, 31.2} = 1.19 (P = 0.324)
Tension	0.6 ± 0.2	0.9 ± 0.4	0.7 ± 0.2	F _{1.4, 19.1} = 0.68 (P = 0.466)	F _{1.4, 19.1} = 1.95 (P = 0.177)	F _{3.4, 46.9} = 0.72 (P = 0.559)
Calm	5.5 ± 0.7	5.1 ± 0.8	5.0 ± 0.7	F _{2.0, 26.0} = 0.75 (P = 0.481)	F _{1.9, 24.3} = 1.94 (P = 0.167)	F _{3.3, 43.3} = 1.08 (P = 0.370)
Happiness	4.9 ± 0.7	4.1 ± 0.7	4.3 ± 0.7	F _{2.0, 26.0} = 0.93 (P = 0.407)	F_{2.3, 29.8} = 4.98 (P = 0.011)	F _{3.9, 51.2} = 0.98 (P = 0.428)
Confusion	1.3 ± 0.4	1.8 ± 0.6	1.0 ± 0.3	F _{1.3, 16.6} = 1.25 (P = 0.293)	F_{1.7, 22.5} = 5.51 (P = 0.014)	F _{3.4, 44.2} = 1.17 (P = 0.333)
Depression	1.0 ± 0.4	1.6 ± 0.5	1.0 ± 0.3	F _{2.0, 26.0} = 1.14 (P = 0.335)	F _{1.5, 19.8} = 2.98 (P = 0.085)	F _{3.2, 41.4} = 1.26 (P = 0.300)
Fatigue	5.6 ± 2.0	6.8 ± 0.9	6.1 ± 0.8	F _{2.0, 28.0} = 1.24 (P = 0.305)	F_{2.3, 32.4} = 14.54 (P < 0.0005)	F _{2.9, 40.3} = 0.43 (P = 0.726)
Stroop test						
Incongruent/Total	60.2 ± 2.3	62.8 ± 2.0	66.8 ± 2.7	F_{1.5, 21.5} = 5.81 (P = 0.015)	F _{1.4, 19.1} = 1.66 (P = 0.217)	F _{2.8, 39.8} = 1.13 (P = 0.346)
Incongruent/Errors	1.2 ± 0.2	1.1 ± 0.2	1.1 ± 0.2	F _{2.0, 28.0} = 0.54 (P = 0.591)	F_{3.0, 42.0} = 2.98 (P = 0.042)	F _{3.7, 51.5} = 1.28 (P = 0.289)
Congruent/Total	105.6 ± 3.9	108.7 ± 4.1	110.5 ± 3.2	F_{2.0, 28.0} = 3.47 (P = 0.045)	F _{2.2, 31.0} = 0.85 (P = 0.447)	F _{4.3, 59.9} = 0.38 (P = 0.833)
Congruent/Errors	1.0 ± 0.2	0.7 ± 0.1	0.8 ± 0.2	F _{2.0, 28.0} = 0.87 (P = 0.431)	F _{2.4, 33.0} = 1.35 (P = 0.274)	F _{3.9, 54.5} = 0.34 (P = 0.844)

5.3.12 Incongruent errors

There was no main effect for condition (*P* = 0.59; see Table 5.2), but there was a significant main effect for time-of-day (*P* = 0.04, η^2_p = 0.18). From 07:00 h (1.4 ± 0.3; 95% CI = 0.9 - 2.0) to 11:00 h (1.0 ± 0.2; 95% CI = 0.7 –

1.3) there were less errors recorded, however errors increased at 14:00 h (1.0 ± 0.2 ; 95% CI = 0.6 – 1.5) and 17:00 h (1.1 ± 0.2 ; 95% CI = 0.7 – 1.5).

5.3.13 Congruent total

There was a significant main effect of condition ($P = 0.045$, $\eta^2_p = 0.20$; pairwise comparisons show that the lowest total score was in the PSR₀ condition (105.6 ± 3.9 ; 95% CI: 97.3 – 113.9) whereas PSR₆₀ achieved the highest total score (110.5 ± 3.2 ; 95% CI: 103.7 – 117.3). Yet there was no significant main effect of time of day or any interactions between ‘condition and time of day’ (Table 5.2).

5.3.14 Congruent errors

There was no significant main effect of condition or time of day ($P = 0.431$, $P = 0.274$; respectively). There were also no significant interactions for ‘condition and time-of-day’.

5.3.15 Actimetry variables

There was no significant main effect of condition for any actimetry variables (fell asleep, woke up, actual sleep time, sleep efficiency and fragmentation index). There was also no significance main effect between nights for any actimetry variables other than ‘Time in bed’ ($P = 0.009$, $\eta^2_p = 0.39$). No significant interactions of ‘condition and night’ were identified (see Table 5.3).

Table 5.3: Mean \pm SD, F values and *P* values for all actimetry and Waterhouse questionnaire variables measured in the study. **Bold** values indicate significant figures ($P < 0.05$).

Actimetry Variables	PSR ₀	PSR ₃₀	PSR ₆₀	Significance Condition	Significance Night	Interaction (Condition*Night)
Fell asleep (h:mm)	02:10 \pm 0:23	02:31 \pm 0:24	02:27 \pm 0:17	F _{1.9, 26.6} = 1.69 (P = 0.205)	F _{1.0, 14.0} = 0.50 (P = 0.491)	F _{2.0, 28.0} = 0.04 (P = 0.966)
Time in bed (h:mm)	04:15 \pm 0:32	04:16 \pm 0:29	04:19 \pm 0:17	F _{1.4, 20.2} = 0.18 (P = 0.769)	F_{1.0, 14.0} = 9.09 (P = 0.009)	F _{1.4, 19.2} = 1.30 (P = 0.284)
Woke up (h:mm)	06:18 \pm 0:46	06:30 \pm 0:36	06:24 \pm 0:14	F _{2.0, 28.0} = 3.11 (P = 0.060)	F _{1.0, 14.0} = 1.55 (P = 0.233)	F _{1.3, 18.4} = 1.78 (P = 0.201)
Actual sleep time (h:mm)	03:22 \pm 0:27	03:23 \pm 0:28	03:26 \pm 0:17	F _{2.0, 28.0} = 0.26 (P = 0.770)	F _{1.0, 14.0} = 0.04 (P = 0.848)	F _{2.0, 28.0} = 2.41 (P = 0.108)
Sleep Efficiency (%)	80.24 \pm 2.06	79.88 \pm 2.28	78.37 \pm 2.63	F _{2.00, 28.00} = 0.33 (P = 0.721)	F _{1.00, 14.00} = 2.45 (P = 0.140)	F _{1.10, 15.37} = 1.47 (P = 0.247)
Fragmentation Index (%)	29.55 \pm 2.91	24.02 \pm 3.59	23.16 \pm 2.60	F _{2.00, 28.00} = 1.35 (P = 0.275)	F _{1.00, 14.00} = 0.118 (P = 0.737)	F _{2.00, 28.00} = 0.53 (P = 0.597)
Waterhouse Questionnaire						
Q1: How easily did you get to sleep?	0.3 \pm 3.3	0.3 \pm 4.2	-1.3 \pm 3.2	F _{1.5, 21.1} = 1.82 (P = 0.191)		
Q2: What time did you get to sleep?	1.9 \pm 2.9	1.7 \pm 3.7	1.7 \pm 3.8	F _{2.0, 28.0} = 0.35 (P = 0.966)		
Q3: How well did you sleep?	0.5 \pm 2.7	-0.7 \pm 2.2	0.6 \pm 2.5	F _{2.0, 24.6} = 2.33 (P = 0.116)		
Q4: What was your waking time?	-3.1 \pm 1.5	-3.5 \pm 2.1	-3.5 \pm 1.9	F _{2.0, 28.0} = 0.50 (P = 0.612)		
Q5: How alert did you feel after 30 minutes of waking?	-2.3 \pm 2.6	-2.3 \pm 2.0	-0.6 \pm 3.2	F_{2.0, 28.0} = 3.40 (P = 0.048)		

5.4 Discussion

Following two consecutive nights of 4 h partial sleep restriction (PSR) from 02:30 to 06:30 h, a post lunch nap (30 or 60-min at 13:00 h), did not improve evening performance (maximal or submaximal; Table 5.1) compared to no nap, in a cohort of resistance trained males. Research on PSR and submaximal performance is scarce, however the findings disagree with those of Brotherton et al. (2019) who employed a similar protocol [2 nights PSR (3 h, 03:30 to 06:30 h), evening sub-maximal weightlifting], and population in terms of strength conditioned (>2 years), sleep habits (~ 8 h) and age (22.7 ± 2.5 versus 21.6 ± 1.6 years). Where the opportunity to nap for 0 min versus 60 min showed an increase in grip strength (2.1 %), bench press (8.3 % for AP, 6.6 ms⁻¹ for PV), leg press (4.6 % for AP) where $P < 0.05$.

The first fundamental difference between this study and Brotherton et al. being the control condition (N) wherein the participants slept 7.5 h (N, retiring at 23:00 and waking at 06:30 h). Sleep restriction resulted in a decrease in maximal grip strength (2.7 %), bench press (AP 11.2 %, average force [AF] 3.3 % and PV 9.4 %) and inclined leg press variables (AP 5.7 %) when compared to N, using the MuscleLab linear encoder (Brotherton et al. 2019). In the current study there was only conditions of sleep restriction, therefore it is not possible to compare the current findings to a normal sleep schedule. This could partially explain the lack of effect between a nap and no nap.

The second fundamental difference is the sleep restriction protocols (4 versus 3 h) which represent a 50 *versus* 37.5 % reduction of the participants habitual sleep duration. To the best of the authors knowledge, no research investigating a potential dose effect of PSR on submaximal weightlifting performance has been published, where with more exposure to sleep loss there is a greater impact on performance (Silva et al. 2021; Walsh et al. 2021). As such, there may be a cut off where in the current study 50% of habitual sleep taken for two nights is tolerated and the homeostatic drive is not affected by a nap of 30 or 60-mins. Belenky et al. (2003) employed four sleep conditions (3, 5, 7 and 9 h per night, 7 consecutive nights) and reported that 5 and 7 h sleep per night reduced performance but stabilised after day two. Those restricted to 3 h had continual performance reductions for the 7-day duration. They concluded approximately 4 h sleep per night is the minimum to achieve a state of equilibrium, that enables an individual to maintain a ‘stable’ level of alertness and performance. However, <4 h may result in decrements. Although this agrees with the hypothesised ‘dose response’ of sleep restriction, Belenky et al. (2003)

measured cognitive function opposed to a physical performance test. Habitual sleep of participants was also not reported. Therefore, participants may have been achieving insufficient sleep pre study and therefore less sensitive to the restriction protocol.

In the current study physical performance was tested in the morning and evening and observed greater values in the afternoon for hand grip strength (5.6 % and 3.9 %, respectively), bench press (7.6 % for AP) and back squat [4.4 % (AP), 3.6 % (AV), 5.1 % (PV); $P < 0.05$]. Despite no 'normal' sleep condition findings agree with Robertson et al. (2018), who conducted exercise at 07:30 and 17:30 h. With low to high masses on the bar they reported diurnal variation in submaximal measures with increases in AF and PV from morning to evening in bench press (2.5 and 12.7 %) and back squat (1.9 and 8.3 % difference). This is consistent with existing literature that has shown greater muscle force output is aligned with the daily peak of core temperature between 15:00-18:00 h (Edwards et al. 2013; Reilly and Waterhouse, 2009). This data demonstrates that independent of the participants sleep schedule prior to performance, diurnal variation can be detected for submaximal performance.

As expected, a significant main effect for load was present where AP, AV, D, PV were highest when load on the bar was lowest (40 % 1RM; Table 5.1). However, tPV increased when there was greater load against the movement, as it typically takes longer for the participant to generate and produce power to perform the movement (Robertson et al. 2018; Brotherton et al. 2019; Figure 5.1 and 5.2). The findings are consistent with fundamental force-velocity properties of skeletal muscle that have been demonstrated previously during complex movements, using linear encoders and force platforms systems (Ammar et al. 2018). A significant interaction was also observed for time- of-day and load for back squat values of AV, PV and tPV ($P = 0.032$; $P = 0.022$; $P = 0.004$, respectively). Both AV and PV were 9.6 % and 15.3 %, respectively, greater at the highest load (80% of 1RM) in the evening compared to morning. To achieve performance enhancement, it is suggested that athletes should train at loads comparable to maximal power output, approximately 70 – 80 % of 1RM, hence the reason the protocol employed 40, 60 and 80 % 1RM (Ammar et al. 2018). Previous literature has suggested that skill-orientated lifts with a high cognitive component (such as bench press) may be more affected by sleep loss compared to lifts such as leg press. Deterioration in cognitive tasks after 13:00 h has been attributed to **i)** an increase in circulation of catecholamines in the blood hence increased arousal, **ii)** the homeostatic drive due to time awake and/or mental fatigue (Reilly and Edwards, 2007; Carrier and Monk, 2000). These findings influenced the choice of exercise as

back squat has a greater cognitive component when compared to inclined leg press. It was anticipated that submaximal back squat would be inhibited under conditions of sleep restriction.

To explain the underlying processes of sleep regulation, mathematical models based on physiological processes, have been developed to account for circadian, ultradian and homeostatic components. These models address: 1) the homeostatic component accountable for greater sleep propensity, during waking and the dissipation during sleep; 2) circadian processes, independent of prior sleep, that define the alternating periods from low to high sleep propensity; 3) ultradian processes that occurs within the sleep episode and represents shifts from nonREM to REM sleep (Borbély and Achermann, 1992; Brotherton et al. 2019). Models representing these processes have been extensively reviewed and become an important approach for experiments investigating sleep restriction, cognitive performance, and napping (Borbély and Achermann, 1992; Rempe et al. 2010). These conceptual frameworks readily explain that as the homeostatic component increases cognitive function is inhibited. This may explain the greater test errors in the PSR₀ condition as those retiring for a nap were able to dissipate the accumulated homeostatic pressure and lower sleep propensity post nap.

In contrast to previous research, intra-aural temperature differed between conditions however this may be due to method of measurement error, as intra-aural is not considered as robust as rectal and gut sites. Previous literature has shown alertness and fatigue are closely influenced by body temperature and time since awake. Alertness and temperature show a causal link with fatigue producing inverse values to these (Edwards and Waterhouse, 2009). A transient fall in temperature and alertness would be expected around 12:00-14:00 h, often referred to as the 'post lunch dip', where post nap ratings of alertness increase, and tiredness decreases (Waterhouse et al. 2007). In agreement, intra-aural temperature peaked at 17:00 h, coinciding with greater muscle force production (Robertson et al. 2018). Tiredness values were lowest at 14:00 h whereas alertness values increased from 14:00 to 17:00 h. Higher alertness at 17:00 h may have contributed to greater evening performance as mood and motivation have shown to be contributing factors to muscle force output (Brotherton et al. 2019). The outcomes may have been influenced by sleep inertia, due to immediate post nap measures, however sleep inertia is less likely to develop after a 30-min nap (Hilditch et al. 2017b).

For cognitive function, significantly greater total scores for the incongruent and congruent Stroop tasks in PSR₃₀ and PSR₆₀ compared to PSR₀ were observed (Table 5.2). Of the two nap conditions PSR₆₀ achieved the highest scores, where longer nap duration correlated with greater test accuracy. More test errors occurred in the morning (07:30 h) for the incongruent test rather than congruent task, this corresponds with higher alertness in the early morning. Mood states changed across the day with vigour and happiness highest at 17:00 h whereas confusion and fatigue decreased throughout the day. Following sleep restriction, time since awake is greater which corresponds with elevated fatigue and impaired function (Monk, 2012). These results suggest that implementing a nap can counteract symptoms of fatigue by reducing sleep propensity and improving cognitive variables such as fewer test errors. Cognitive variables follow a circadian rhythm, with function typically lower in the morning and peaking in the evening (Van Dongen and Dinges, 2000; Munnilari et al. 2024). The improvement in cognitive ability post nap is often said to be dependent on sleep stages that occur, as the quantity of slow wave sleep and rapid eye movement (REM) may differ dependent on nap duration (Ficca et al. 2010).

5.4.1 Limitations

Throughout the experimental conditions dietary intake was not recorded and therefore it is difficult to ensure consistency across the study. Lack of dietary control was highlighted in the mixed methods review of the thesis (See Chapter 2, Table 2.3) and will be addressed in later chapters. This consideration is necessary as following sleep restriction individuals typically consume significantly greater caloric intake, which is associated with altered leptin and ghrelin hormone responses. It is possible that participants may have consumed greater food intake during the study that may have influenced outcomes. Another limitation was the absence of a control condition (like Brotherton et al. 2019); however, the current protocol was already demanding for those that participated. Previous research by the working group has reported diurnal variation after employing normal sleep protocols, hence the decision to not include a habitual sleep condition. Lastly, an objective measure of sleep was not taken during the nap at 13:00 h. If sleep had been assessed using polysomnography or actigraphy greater insight would have been available on key sleep variables such as sleep duration, sleep efficiency and sleep architecture. Measurement of sleep via polysomnography was conducted later in the thesis (see Chapter 7).

5.4.2 Conclusion

Results obtained from this study in a population of healthy recreationally (Tier 2) active males, indicates that 4 h of PSR, for two consecutive nights, did not have a significant effect on maximal grip strength and submaximal measures of bench press and back squat (AV, AP, tPV, PV, D; $P > 0.005$). Implementing a nap at 13:00 h did not improve submaximal performance compared to no nap. Despite this, a 30 and 60-min nap at 13:00 h can improve mood state, executive function and reduce tiredness. As reported previously, neurobehavioral deficits are often reported following sleep restriction. It is hypothesised that a nap opportunity provides temporary relief from excessive tiredness and allows the accumulation of homeostatic pressure to dissipate, however the exact mechanisms are yet to be investigated (see Chapter 7). These findings hold important questions regarding the optimal nap duration.

Chapter 6:

Investigating the dose response between 3 versus 4 hours of partial sleep restriction on performance outcomes?

This chapter explores the hypothesised dose response between different durations of sleep restriction and effects this may have on performance outcomes.

6.1 Introduction

Sleep is a state of reduced responsiveness to internal, external stimuli and is characterised by numerous physiological processes that are critical for human life (Banks and Dinges, 2007). For the consequences of inadequate sleep and the host of associated symptoms please refer to Chapter 2 (section 2.5) of the mixed methods review where this is discussed further.

Compared to total sleep deprivation, the occurrence of sleep restriction (SR), can be far more frequent in general and athletic populations (Reynolds and Banks, 2010; Smithies et al. 2021). At a physiological level sustained durations of wakefulness can increase sleep pressure/need, that is only dissipated during sleep. When considering changes in sleep architecture, differences are noticeable after only 1-2 nights of SR (Banks and Dinges, 2007). Protocols that have employed 4 h of SR per night have reported decreases in all sleep stages except slow wave sleep (SWS). However, when SR is maintained over multiple days, rapid eye movement (REM) sleep may occur earlier to compensate for the lack of REM (Belenky et al. 2003). If and when recovery sleep happens a REM sleep rebound may occur alongside a shorter sleep latency (Carskadon and Roth, 1991).

Acute sleep loss can have a negative impact on an array of exercise types, with those requiring a greater skill component being the most sensitive (Van Dongen et al. 2003). Studies focusing on muscular strength, with a short time-on-task and minimal cognitive component, report no effect of sleep restriction on singular bouts of maximal exercise (Craven et al. 2022). Cognitive deficits are apparent following SR, with slower response times and greater performance lapses following 3 h of SR for 7 nights compared to 5 h and 7 h of SR per night (Belenky et al. 2003). These findings are consistent following 4 h of SR for 5 nights, with reported increases in psychomotor vigilance lapses and longer reaction times that were cumulative across the day (08:00-20:00 h; Banks et al. 2010). In agreement with Horne's hypothesis (Horne, 1988), there is potentially dose response whereby ≥ 4 h of sleep per night is tolerable for the brain to maintain stable (yet lower than normal) performance, but any less is below the bodies physiological limit.

Unfortunately, SR in athletes is sometimes unavoidable and therefore effective interventions are required. Several have been proposed in the literature such as nutritional and pharmacological strategies (Hatia et al. 2024). For

athletes, their schedules may not allow for extended nocturnal sleep therefore naps may be the most suitable. Naps have no associated cost, are non-invasive alternative and increase the total sleep time within a 24-h period (Cunha et al. 2023). There is still insufficient evidence to suggest the optimal nap duration, however naps of 10-60 minutes have shown to improve subjective responses, cognitive and physical performance following acute SR. Naps > 30 minutes in duration may not present immediate benefits due to subsequent sleep inertia, that may take 30-60 minutes to dissipate (Brooks and Lack, 2006). If athletes can allow 30-60 min post nap to recover from potential sleep inertia, naps of 30-90 min appear to provide greater recovery benefits (Mesas et al. 2022).

Therefore, the purpose of the present study was to 1) determine whether there is a dose response between 3 versus 4 h of sleep restriction on measures of strength and cognitive function; 2) investigate the effectiveness of a 30 min and 60-min nap when compared to no nap on evening performance.

6.2 Methods

6.2.1 Participants

Eighteen males, as identified by sex and gender (refer to General Methods Chapter 3, Table 3.1) participated in the current study. Participants were separated, by random allocation, into two groups of 3 h (n = 9) or 4 h (n = 9) of sleep restriction per night for two consecutive nights (refer to Table 3.1 for participant characteristics). For the study inclusion criteria, refer to Chapter 3.1: General methods and details of the pre-screening process can be found in Chapter 3.1.2. Verbal explanation of the experimental procedures was provided with the study aims and any possible risks of participating outlined, followed by written consent. Experimental procedures were approved and conducted in accordance with the University Human Ethics Committee and complied with the Declaration of Helsinki (Ethics code: M19_SPS_140 and 22/SPS/061).

6.2.2 Research Design

Participants were required to attend the laboratory on seven occasions. Prior to testing participants completed (i) 7-day habitual sleep recording using actimetry (Motionware 8, CamnTech) (ii) 7-day sleep diary (iii) 5-day habitual food diary. All habitual data was collected two weeks before experimental conditions to ensure

participants maintained healthy sleep and food habits prior to testing. The first three laboratory visits were conducted in the University Strength and Conditioning Performance Unit where participants completed one repetition max (1RM) for bench press and back squat, followed by two familiarisation sessions. Visits were separated by 7 days to ensure adequate recovery, with a further 7-day period between the final familiarisation and first experimental condition. Details of the familiarisation sessions are explained in Chapter 3, section 3.2.2. Prior to all experimental conditions participants were partially sleep restricted at their own home for two consecutive nights retiring to sleep at 02:30 or 03:30 and rising at 06:30 dependent on the group (SR₃ or SR₄) they were assigned to. In the 24 h before each testing condition participants were asked to refrain from any vigorous physical exercise and avoid alcoholic or caffeine containing drinks. Participants were also asked to consume no food in the hour before testing sessions and refrain from screen use (i.e. television, mobile phone) in the hour before retiring to sleep. All conditions were counterbalanced by order of administration and participants were allocated into groups equally based on their physical ability via a stacking method using each individual 1RM values for bench press and back squat (Monk and Leng, 1982).

Upon entering the laboratory on the third day for testing, participants would either have no nap (N₀), 30-min nap (N₃₀) or a 60-min nap (N₆₀) commencing at 13:00 h. For the no nap condition (N₀), the protocol differed between the two groups (SR₃ and SR₄). Participants in SR₃ remained in the sleep laboratory and in bed for the 60-minute duration and were only allowed to read or listen to music, with no access to screens or devices. To ensure the participant remained awake a researcher stayed in the laboratory to monitor the participant but they did not engage in conversation. Participants in SR₄ were allowed to leave the sleep laboratory but had to remain on campus for this duration and return to the laboratory at the end of the 60 mins. At the end of each nap opportunity participants were asked if they ‘managed to sleep’, as detailed in the previous study (Chapter 5). Throughout the 3-day testing period researchers regularly communicated with participants via direct text message and asked participants to remain on campus for the testing day to ensure compliance. For the two nap conditions, participants were given the opportunity to sleep and required to remain in bed in the University sleep laboratory until a member of the research team re-entered the laboratory at the end of the allocated time.

6.2.3 Measurements

Before testing commenced, participants were required to complete a 1RM for bench press and back squat in the university gym, as outlined in Chapter 3: General Methods. Following this, two familiarisation sessions took place, approximately one week after the 1RM session, to ensure participants were physically capable of performing the lifts and were ran through the questionnaires and cognitive tests they would be completing during the study. The first experimental condition then took place in the fortnight after these sessions. After two consecutive nights of sleep restriction, participants arrived at the laboratory at 07:00 h for the first testing session and returned at 11:00, 13:00-14:15h and 17:00 h for all remaining data collection. The majority of participants lived in close proximity to the university therefore they were able to wake up and attend the laboratory within a 30-minute timeframe. For those who had to travel into the laboratory and required further time we provided 1 hour and delayed the initial testing session to 07:30 h. At each time point (07:00, 11:00, 14:00 and 17:00 h) participants had to provide responses for ratings of mood (Profile of Mood States questionnaire; Terry et al. 2003), sleepiness (Stanford sleepiness questionnaire), tiredness and alertness (0-10 cm VAS) and the Stroop task (General Methods 3.3.4). At 17:00 h following completion of questionnaires and cognitive tests, participants completed an exercise session (refer to Chapter 3.3: General Methods for exercise protocol).

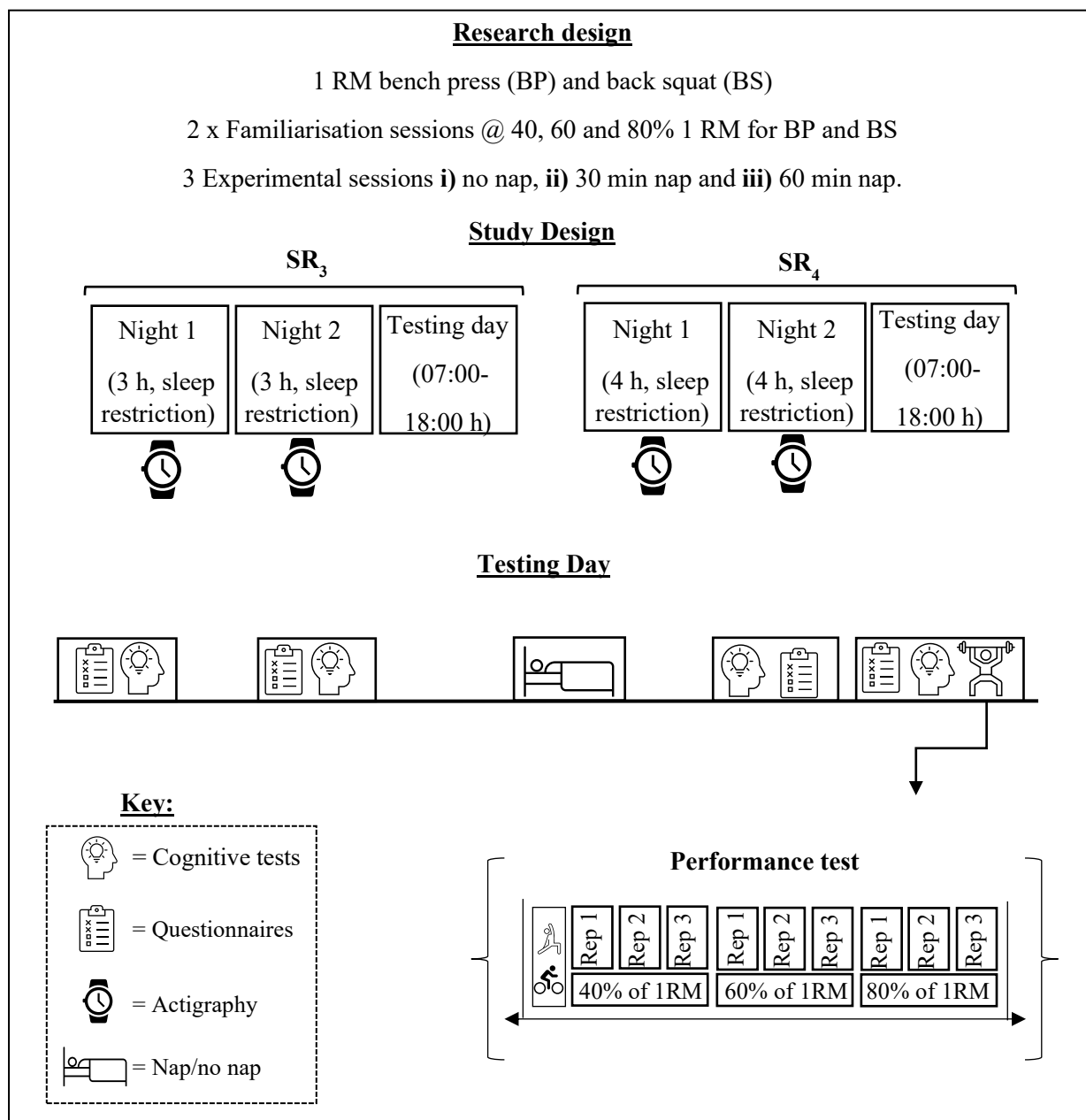


Figure 6.1 Study schematic, outlining the research design, study design and testing day protocol.

6.2.4 Statistical Analysis

As stated in Chapter 3, section 3.4 we conducted within subject factors for nap conditions and cognitive tests with between subject factors tests for differences between the two groups (SR₃ and SR₄). To clarify we did not run between group differences for any of the baseline data such as sleep diary entries and actigraphy data. This pre-screening information was collected to confirm participants were habitually sleeping within the NSF guidelines of 7-9 hours per night. Please refer to Chapter 3, section 3.4 of General Methods for detail of the statistical analysis.

6.3 Results

6.3.1 Performance measures (measured at 17:00 h)

Mean SD values and the results from the ANOVA statistical analysis are shown in Tables 6.1 and 6.2. Statistical significance of the results is also represented in Figures 6.2 and 6.3.

6.3.2 Grip strength (left and right hand)

There was no significant main effect for sleep restriction group on hand grip values (Table 6.1). There was also no significant effect for experimental nap condition on left- or right-hand grip strength values, such that regardless of nap length grip strength values were the same ($P = 0.719$, $P = 0.641$; respectively).

6.3.3 Bench press

There was no significant main effect for group for all performance variables. However, there was a significant main effect for group for RPE ($P < 0.02$, $\eta^2_p = 0.30$) and RPE Muscle Fatigue ($P < 0.001$, $\eta^2_p = 0.58$), where for both variables' the values were higher for SR₃ compared to SR₄ (RPE: 12.8 versus 11.8; RPE Muscle Fatigue: 12.5 versus 10.5, respectively). There was no significant main effect for nap condition for all bench press performance variables (see Table 6.1). However, there was a significant main effect for "load" for all performance variables ($P < 0.001$, see Table 6.1). For AP, AV, PV and D, values were highest at 40% of 1RM and decreased as the load on the bar increased to 80% of 1RM (Table 6.1 and 6.2). There was a significant main effect of condition for RPE Muscle Fatigue ($P < 0.001$, $\eta^2_p = 0.56$), however not for RPE and RPE Breathing. Pairwise comparisons identified that RPE Muscle fatigue values were significantly higher ($P < 0.001$) in N₃₀ condition compared to N₆₀ (12.5, 95%: 11.8-13.1 versus 10.7, 95% CI: 10.2 - 11.1, respectively). As anticipated, there was a significant main effect of load on RPE, RPE Muscle fatigue and RPE Breathing values with the lowest values at 40% and highest values at 80% of 1RM. There was no significant interaction for "condition and load" for any variable, whereby values across all three conditions decreased in the same manner across the three loads (see Figure 6.2).

6.3.4 Back squat

There was a significant main effect between the groups for RPE and RPE Muscle Fatigue, however not for any back squat performance measures (Table 6.1). Pairwise comparisons showed that RPE values were significantly higher for those in SR₄ compared to SR₃ for two consecutive nights ($P = 0.046$). This was the same for RPE Muscle Fatigue, where pairwise comparisons revealed values were significantly higher following 4 h of SR compared to 3 h of SR ($P < 0.001$). There was no significant main effect of experimental condition for all back squat performance variables ($P = 0.064$, $\eta^2_p = 0.16$; Table 6.1). There was a significant main effect for “load” on the bar for all performance variables, as anticipated AP, AV, PV and D were highest at 40% and lowest at 80% of 1RM ($P < 0.0005$). No significant interactions for “condition and load” were present. There was a significant main effect of condition for RPE Muscle fatigue ($P < 0.001$, $\eta^2_p = 0.64$), yet not for RPE and RPE Breathing values. As expected, there was a significant main effect of load on RPE, RPE Breathing and RPE Muscle fatigue whereby perceived exertion was lowest at 40% of 1RM and highest at 80% of 1RM.

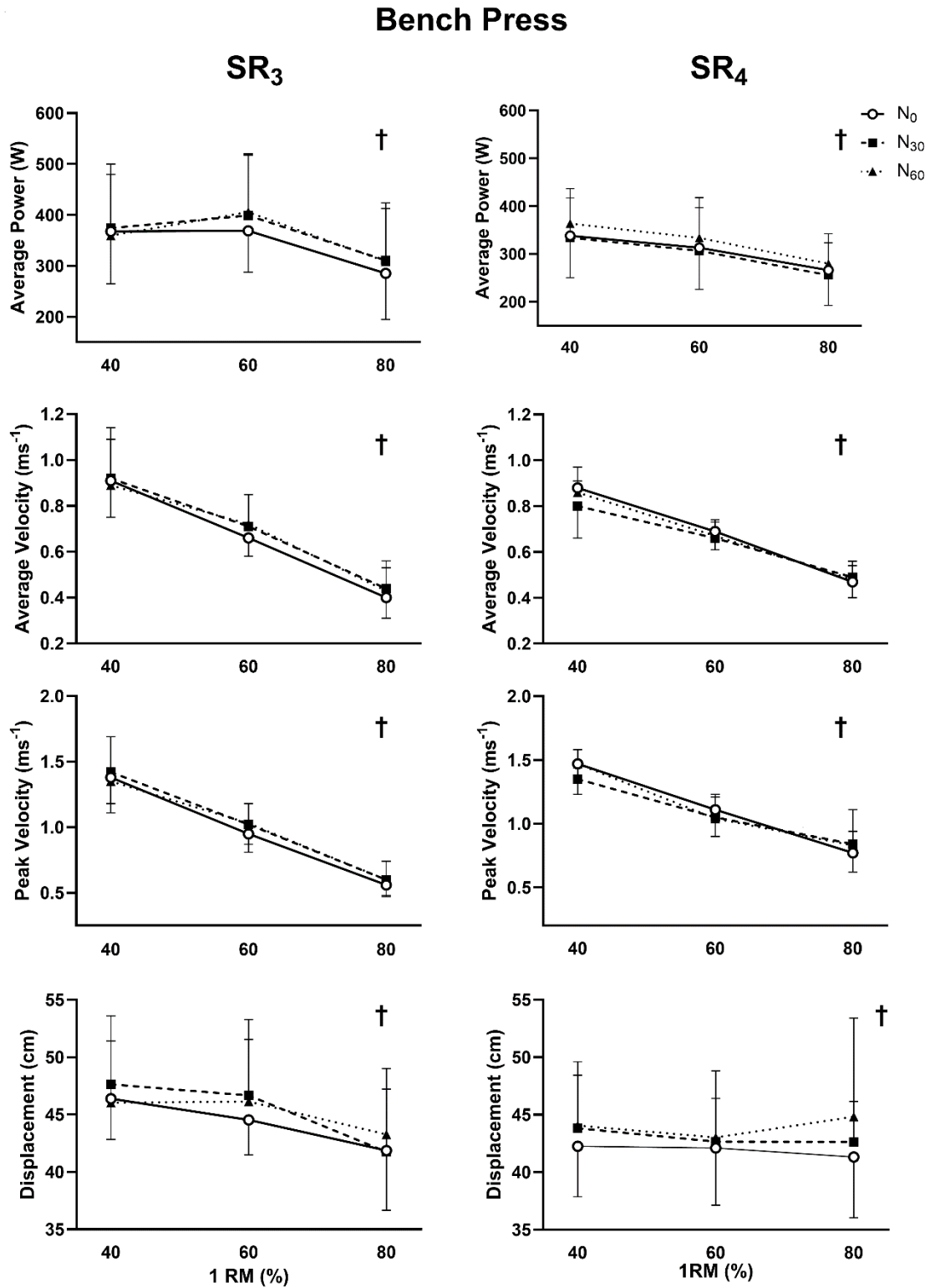


Figure 6.2. Mean \pm SD values for each performance variable for evening (17:00 h) submaximal performance at 40, 60 and 80% of 1RM for bench press. † icon denotes significant effect for 'load on the bar' ($P < 0.05$).

Back Squat

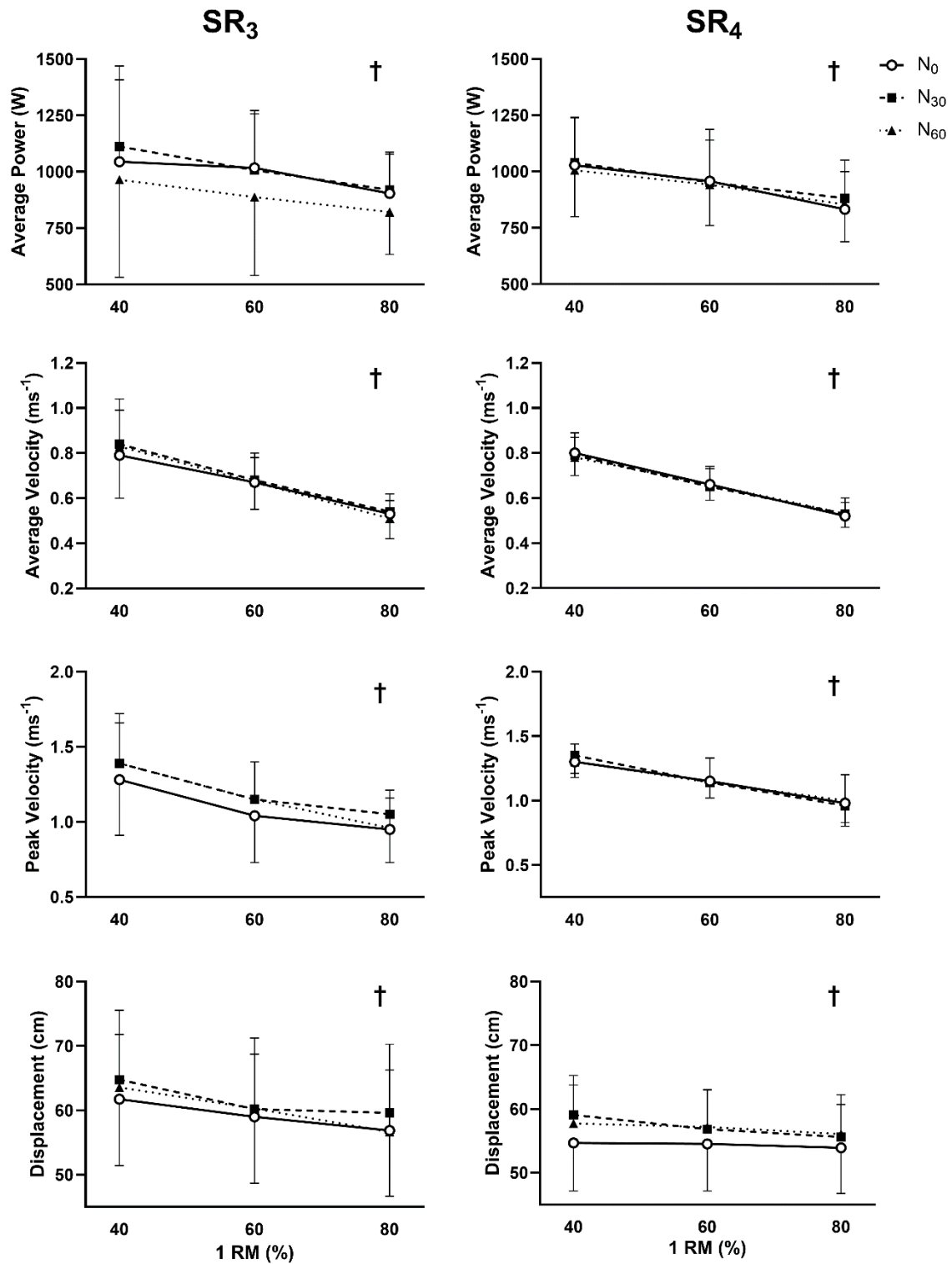


Figure 6.3. Mean \pm SD values for each performance variable for evening (17:00 h) submaximal performance at 40, 60 and 80% of 1RM for back squat. † icon denotes significant effect for 'load on the bar' ($P < 0.05$).

Table 6.1. F values, P values and effect sizes (ES) as partial eta squared are presented for performance variables measured at 17:00 h. Bold indicates significance ($P < 0.05$).

Variable	Significance Group	Significance Condition	Significance Load	Interaction (Condition*Load)
Grip strength (N)				
Left				
	$F_{1,0, 16,0} = 0.73$ ($P = 0.405$), ES = 0.04	$F_{1,0, 16,0} = 0.64$ ($P = 0.436$), ES = 0.04		
Right				
	$F_{2,0, 31,9} = 0.45$ ($P = 0.641$), ES = 0.03	$F_{2,0, 32,0} = 0.33$ ($P = 0.719$), ES = 0.02		
Bench press				
Average power (W)	$F_{1,0, 16,0} = 1.09$ ($P = 0.313$), ES = 0.06	$F_{2,0, 32,0} = 1.27$ ($P = 0.296$), ES = 0.07	$F_{1,2, 18,8} = 22.16$ ($P < 0.001$), ES = 0.58	$F_{3,0, 48,3} = 0.77$ ($P = 0.519$)
Displacement (cm)	$F_{1,0, 16,0} = 0.80$ ($P = 0.386$), ES = 0.05	$F_{2,0, 32,0} = 1.24$ ($P = 0.304$), ES = 0.07	$F_{1,5, 24,4} = 10.13$ ($P = 0.001$), ES = 0.39	$F_{2,4, 38,2} = 1.51$ ($P = 0.232$)
Average velocity (ms^{-1})	$F_{1,0, 16,0} = 0.05$ ($P = 0.833$), ES = 0.00	$F_{1,4, 22,9} = 0.03$ ($P = 0.929$), ES = 0.00	$F_{1,2, 19,3} = 148.18$ ($P < 0.001$), ES = 0.90	$F_{2,7, 42,7} = 1.41$ ($P = 0.254$)
Peak velocity (ms^{-1})	$F_{1,0, 16,0} = 3.31$ ($P = 0.088$), ES = 0.17	$F_{1,2, 19,0} = 0.05$ ($P = 0.859$), ES = 0.00	$F_{1,3, 21,0} = 248.33$ ($P < 0.001$), ES = 0.94	$F_{2,4, 38,1} = 0.66$ ($P = 0.550$)
Back squat				
Average power (W)	$F_{1,0, 16,0} = 0.04$ ($P = 0.848$), ES = 0.00	$F_{1,3, 20,1} = 1.86$ ($P = 0.188$), ES = 0.10	$F_{1,1, 18,0} = 12.11$ ($P = 0.002$), ES = 0.43	$F_{2,5, 39,5} = 0.68$ ($P = 0.544$)
Displacement (cm)	$F_{1,0, 16,0} = 1.12$ ($P = 0.307$), ES = 0.07	$F_{2,0, 32,0} = 3.00$ ($P = 0.064$), ES = 0.16	$F_{1,6, 25,1} = 21.21$ ($P < 0.001$), ES = 0.57	$F_{4,0, 64,0} = 1.37$ ($P = 0.255$)
Average velocity (ms^{-1})	$F_{1,0, 16,0} = 0.12$ ($P = 0.738$), ES = 0.01	$F_{2,0, 32,0} = 0.36$ ($P = 0.700$), ES = 0.02	$F_{1,2, 18,6} = 111.50$ ($P < 0.001$), ES = 0.88	$F_{2,7, 42,5} = 0.31$ ($P = 0.793$)
Peak velocity (ms^{-1})	$F_{1,0, 16,0} = 0.00$ ($P = 0.959$), ES = 0.00	$F_{1,5, 23,4} = 1.45$ ($P = 0.247$), ES = 0.08	$F_{1,3, 20,1} = 108.90$ ($P < 0.001$), ES = 0.87	$F_{2,8, 44,4} = 0.45$ ($P = 0.703$)

6.4 Cognitive function and psychological responses (measured at 07:30, 11:00, 14:00 and 17:00

h)

6.4.1 Stroop Black Ink - Total

There was no significant main effect of condition, time-of-day or group and there were no interactions between condition and time of day (Table 6.2).

6.4.2 Black Ink - Error

There was a significant main effect for group with greater test errors in SR₄ (0.9 ± 0.2 , 95% CI: 0.6-1.3) compared to SR₃ (0.4 ± 0.2 , 95% CI: 0.0-0.7; $P = 0.016$). There was no significant effect for condition or time-of-day ($P = 0.930$ and $P = 0.559$, respectively).

6.4.3 Incongruent - Total

There was no significant main effect for group, experimental condition or time-of-day and no interactions between condition and time-of-day, see Table 6.2.

6.4.4 Incongruent - Errors

There was no significant main effect for group, experimental condition or time-of-day or group ($P = 0.582$, $P = 0.091$ and $P = 0.382$, respectively).

6.4.5 Congruent - Total

There was no significant main effect for group or experimental condition ($P = 0.095$ and $P = 0.956$). However, there was a significant effect for time-of-day ($P = 0.041$) with the lowest total score at 07:00 h (104.4 ± 3.6), the highest score achieved at 11:00 h (112 ± 2.8), followed by a decrease at 14:00 h (108.4 ± 3.6) and a final increase in total score at 17:00 h (110.7 ± 3.0). There were no significant interactions between condition and time-of-day (Table 6.2).

6.4.6 Congruent - Errors

There was no significant main effect for group, however there was a significant main effect for condition ($P = 0.047$, $\eta^2_p = 0.21$), with the greatest number of errors in N_0 (1.0 ± 0.2) compared to N_{30} (0.7 ± 0.1) and N_{60} (0.7 ± 0.1). There was no significant main effect for time-of-day and no interactions between condition and time-of-day ($P = 0.651$).

6.4.7 Profile of mood state

There was no significant main effect for group or napping condition for all mood states, see Table 6.2. Despite this, there was a significant effect for time-of-day for states of vigour, anger, confusion, depression and fatigue. Vigour was lowest at 07:00 h (3.4 ± 0.6) and highest at 17:00 h (5.6 ± 0.6), with the remaining mood states (anger, confusion, depression and fatigue) being highest at 07:00 h and decreasing across the day. There were no interactions between condition and time-of-day for any mood state (Table 6.2).

6.4.8 Stanford sleepiness and Waterhouse questionnaire

There was no significant main effect for group or condition for the Stanford sleepiness scale. Although there was a significant effect for time-of-day ($P < 0.001$, $\eta^2_p = 0.40$), with the highest sleepiness score at 07:00 h (4.3 ± 0.3 , 95% CI: 3.6-4.9) and gradually decreasing across the day with the lowest value at 17:00 h (3.1 ± 0.2 , 95% CI: 2.6-3.7). No interactions were found between condition and time-of-day. For the Waterhouse questionnaire, there was no significant effect for group for all responses. A significant main effect for condition for Question 3 (“How well did you sleep?”) was evident, with more perceived waking episodes in the N_{60} condition; but no significant of condition for all remaining questions.

Table 6.2. F values and P values for Profile of Mood States, Stroop test, Stanford sleepiness and Waterhouse questionnaire across group, condition and time-of-day (TOD). Effect sizes (ES) are presented as partial eta squared. **Bold** values indicate significant figures.

Variables	Significance Group	Significance Condition	Significance TOD	Interaction (Condition*TOD)
Profile of Mood States				
Vigour	$F_{1.0, 16.0} = 0.33$ ($P = 0.575$), ES = 0.02	$F_{2.0, 32.0} = 0.47$ ($P = 0.630$), ES = 0.03	$F_{2.4, 38.5} = \mathbf{12.41}$ ($P < \mathbf{0.001}$), ES = 0.44	$F_{4.4, 70.0} = 2.15$ ($P = 0.077$)
Anger	$F_{1.0, 16.0} = 0.06$ ($P = 0.803$), ES = 0.00	$F_{1.5, 24.7} = 0.56$ ($P = 0.534$), ES = 0.03	$F_{2.3, 36.9} = \mathbf{4.43}$ ($P = \mathbf{0.015}$), ES = 0.22	$F_{2.7, 43.9} = 0.71$ ($P = 0.537$)
Tension	$F_{1.0, 16.0} = 1.55$ ($P = 0.231$), ES = 0.09	$F_{1.2, 18.8} = 1.64$ ($P = 0.219$), ES = 0.09	$F_{2.0, 32.1} = 2.71$ ($P = 0.082$), ES = 0.15	$F_{3.7, 59.1} = 0.49$ ($P = 0.726$)
Calm	$F_{1.0, 16.0} = 3.72$ ($P = 0.072$), ES = 0.19	$F_{2.0, 32.0} = 0.05$ ($P = 0.954$), ES = 0.00	$F_{2.5, 40.0} = 0.79$ ($P = 0.486$), ES = 0.05	$F_{3.8, 60.2} = 1.01$ ($P = 0.409$)
Happiness	$F_{1.0, 16.0} = 2.64$ ($P = 0.124$), ES = 0.14	$F_{2.0, 32.0} = 0.04$ ($P = 0.962$), ES = 0.00	$F_{3.0, 48.0} = 2.46$ ($P = 0.074$), ES = 0.13	$F_{4.4, 70.8} = 0.93$ ($P = 0.457$)
Confusion	$F_{1.0, 16.0} = 1.10$ ($P = 0.310$), ES = 0.06	$F_{1.6, 25.1} = 0.42$ ($P = 0.616$), ES = 0.03	$F_{1.9, 30.7} = \mathbf{5.59}$ ($P = \mathbf{0.009}$), ES = 0.26	$F_{3.7, 58.8} = 0.95$ ($P = 0.435$)
Depression	$F_{1.0, 16.0} = 0.05$ ($P = 0.819$), ES = 0.00	$F_{2.0, 32.0} = 0.43$ ($P = 0.653$), ES = 0.03	$F_{2.3, 37.4} = \mathbf{5.50}$ ($P = \mathbf{0.006}$), ES = 0.26	$F_{3.1, 49.8} = 1.08$ ($P = 0.367$)
Fatigue	$F_{1.0, 16.0} = 0.78$ ($P = 0.392$), ES = 0.05	$F_{2.0, 32.0} = 0.32$ ($P = 0.725$), ES = 0.02	$F_{2.3, 36.9} = \mathbf{8.04}$ ($P < \mathbf{0.001}$), ES = 0.33	$F_{3.1, 48.9} = 0.72$ ($P = 0.549$)
Stroop task				
Black ink-Total	$F_{1.0, 16.0} = 1.55$ ($P = 0.231$), ES = 0.09	$F_{2.0, 32.0} = 0.02$ ($P = 0.982$), ES = 0.00	$F_{2.3, 37.1} = 1.53$ ($P = 0.229$), ES = 0.09	
Black ink-Errors	$F_{1.0, 16.0} = \mathbf{7.22}$ ($P = \mathbf{0.016}$), ES = 0.31	$F_{1.9, 30.4} = 0.07$ ($P = 0.930$), ES = 0.00	$F_{2.5, 40.0} = 0.65$ ($P = 0.559$), ES = 0.04	
Incongruent-Total	$F_{1.0, 16.0} = 0.95$ ($P = 0.344$), ES = 0.06	$F_{1.9, 30.2} = 0.77$ ($P = 0.465$), ES = 0.05	$F_{2.2, 35.8} = 2.51$ ($P = 0.090$), ES = 0.14	
Incongruent-Error	$F_{1.0, 16.0} = 0.32$ ($P = 0.582$), ES = 0.02	$F_{1.5, 24.6} = 2.82$ ($P = 0.091$), ES = 0.15	$F_{3.0, 48.0} = 1.04$ ($P = 0.382$), ES = 0.06	
Congruent-Total	$F_{1.0, 16.0} = 3.14$ ($P = 0.095$), ES = 0.16	$F_{2.0, 32.0} = 0.05$ ($P = 0.956$), ES = 0.00	$F_{3.0, 48.0} = \mathbf{2.96}$ ($P = \mathbf{0.041}$), ES = 0.16	
Congruent-Error	$F_{1.0, 16.0} = 0.27$ ($P = 0.611$), ES = 0.02	$F_{1.3, 20.4} = \mathbf{4.12}$ ($P = \mathbf{0.047}$), ES = 0.21	$F_{1.9, 30.5} = 2.01$ ($P = 0.153$), ES = 0.11	
Stanford Sleepiness	$F_{2.0, 32.0} = 2.02$ ($P = 0.150$), ES = 0.03	$F_{1.0, 16.0} = 0.42$ ($P = 0.526$), ES = 0.11	$F_{1.8, 28.2} = \mathbf{10.65}$ ($P < \mathbf{0.001}$), ES = 0.40	
Waterhouse				
Q1	$F_{1.0, 16.0} = 4.10$ ($P = 0.061$), ES = 0.20	$F_{2.0, 32.0} = 0.40$ ($P = 0.672$), ES = 0.03		
Q2	$F_{1.0, 16.0} = 0.13$ ($P = 0.721$), ES = 0.01	$F_{1.4, 22.2} = 1.54$ ($P = 0.235$), ES = 0.09		
Q3	$F_{1.0, 16.0} = 0.35$ ($P = 0.565$), ES = 0.02	$F_{2.0, 32.0} = \mathbf{3.74}$ ($P = \mathbf{0.035}$), ES = 0.19		
Q4	$F_{1.0, 16.0} = 1.54$ ($P = 0.233$), ES = 0.09	$F_{2.0, 32.0} = 1.98$ ($P = 0.155$), ES = 0.11		
Q5	$F_{1.0, 16.0} = 0.02$ ($P = 0.896$), ES = 0.01	$F_{2.0, 32.0} = 0.41$ ($P = 0.666$), ES = 0.03		

Table 6.3. F values and P values for actimetry variables and Waterhouse questionnaire variables measured in the study. **Bold** values indicate significant figures ($P < 0.05$).

Actimetry Variables	Significance Group	Significance Condition	Significance of Night	Interaction (Condition*Night)
Sleep latency (h:m)	$F_{1.0, 16.0} = 3.75$ ($P = 0.071$)	$F_{1.3, 20.4} = \mathbf{5.97}$ ($P = \mathbf{0.018}$)	$F_{1.0, 16.0} = 0.73$ ($P = 0.405$)	$F_{2.0, 32.0} = \mathbf{5.90}$ ($P = \mathbf{0.007}$)
Sleep Efficiency (%)	$F_{1.0, 16.0} = 1.39$ ($P = 0.255$)	$F_{2.0, 32.0} = 1.05$ ($P = 0.361$)	$F_{1.0, 16.0} = 1.64$ ($P = 0.219$)	$F_{1.2, 18.8} = 1.46$ ($P = 0.247$)
Fragmentation Index (%)	$F_{1.0, 16.0} = 0.01$ ($P = 0.932$)	$F_{2.0, 32.0} = 0.39$ ($P = 0.684$)	$F_{1.0, 16.0} = 0.10$ ($P = 0.761$)	$F_{2.0, 32.0} = 1.40$ ($P = 0.260$)
Actual sleep time (h:m)	$F_{1.0, 16.0} = \mathbf{51.95}$ ($P < \mathbf{0.001}$)	$F_{2.0, 32.0} = 0.56$ ($P = 0.575$)	$F_{1.0, 16.0} = 3.00$ ($P = 0.102$)	$F_{1.4, 22.7} = \mathbf{5.33}$ ($P = \mathbf{0.021}$)

6.5 Actimetry variables

There was a significant main effect for group for actual sleep time ($P < 0.001$, $\eta^2_p = 0.19$) due to the greater sleep opportunity in SR₄ compared to SR₃ but not differences for the remaining variables. A significant main effect for condition for sleep latency was found ($P = 0.018$, $\eta^2_p = 0.27$) with shorter time to sleep onset in N₀ compared to N₃₀ and N₆₀. No difference for condition was reported for sleep efficiency, fragmentation index and actual sleep time (Table 6.3). No significant effect between night 1 and 2 of sleep restriction for all actimetry variables suggesting sleep was relatively similar. For interactions between condition and nights of sleep restriction sleep latency on night two was significantly shorter, which is likely due to the accumulation of sleep loss and greater sleep propensity.

6.6 Discussion

There was no dose response observed between experimental groups (SR₃ and SR₄) for grip strength, bench press or back squat when measured at 17:00 h. Regarding the effectiveness of a nap, no improvement in evening performance was reported following a 30- and 60-min nap at 13:00 h when compared to employing no nap in a sleep restricted state.

Previous studies that have employed sleep restriction of 3 h per night and assessed maximal and submaximal performance, have reported impaired grip strength, bench press and leg strength in similar populations (Brotherton et al. 2019; Reilly and Piercy, 1994). A potential explanation for this difference is the lack of a control condition in the current study. Without a control, where participants obtain habitual sleep in addition to sleep restriction, the differences between the two groups (SR₃ and SR₄) are likely minimal in comparison. For perceptual changes, RPE and RPE muscle fatigue values were greater for the SR₄ group when performing bench press and back squat. The findings demonstrate that participants were able to retain submaximal performance despite greater perceived efforts. It is possible that some individuals exert greater effort to compensate for sleep loss and try to mask the negative effect on performance (Axelsson et al. 2008; Drummond et al. 2000). These outcomes may also be influenced by factors such as motivation or individual variability in susceptibility to sleep restriction. Based on the pre-screening responses to the Composite Questionnaire, participants reported high flexibility opposed to rigidity and were more 'vigorous' (specifically SR₃). This suggests they were able to adjust better to sleep restriction and easily overcome drowsiness. These subjective responses are typically associated with lower age and male individuals (Marcoen et al. 2015).

The few studies that have investigated a dose response to sleep restriction predominantly investigate psychomotor performance opposed to physical performance (Banks et al. 2010; Van Dongen et al. 2003). Cote et al (2009) and Belenky et al (2003) both employed 3 versus 5 h of sleep restriction per night and reported greater performance impairments in the 3 h condition, with more lapses in performance and slower reaction times. These findings support the hypothesis that the minimum sleep duration required to maintain stable cognitive performance is approximately 4 h per night, with any less resulting in unavoidable decrements (Belenky et al. 2003). Although this may not align with the 'inflection point' hypothesis, actimetry data showed that SR₄ participants averaged only $3:25 \pm 0:03$ h:mm of sleep per night. Both groups achieved below the proposed threshold of 4 h per night, suggesting that future studies should use protocols with $>$ or $<$ 4 h of sleep per night to account for sleep onset duration.

No differences were found between experimental conditions (N₀, N₃₀ and N₆₀) for maximal and submaximal strength at 17:00 h (Table 6.1). Although napping protocols are recommended to counter sleep loss, the impact on maximal and submaximal performance remains inconclusive. Other studies that have assessed maximal strength following acute SR, agree and report no change following a post lunch nap (Waterhouse et al. 2007). However, submaximal strength, particularly lower limb exercises, may be impaired following acute SR and

restored after a 60-min nap (Brotherton et al. 2019; Ajjimaporn et al. 2020). Timing a nap that coincides with the ‘post lunch dip’ increases the tendency to nap along with a shorter sleep latency, due to the peak in sleep propensity that is further amplified following sleep loss. Naps have therefore been hypothesised to reduce the adverse effects of SR and restore exercise performance compared to no nap. Hence why greater cognitive function and submaximal weightlifting performance would be expected following a 30- or 60-min nap. Differences in the literature may be due to variations in duration and timing of the nap period, as well as the time interval between the nap ending and the exercise task (Boukhris et al. 2019). Previous studies that report no benefits from napping attribute it to sleep inertia. However, in the current study the exercise session was scheduled 3 h post nap to allow time for sleep inertia to dissipate as the effects of sleep inertia have shown to subside within 45 min to 2 h (Brooks and Lack, 2006). A current limitation was the absence of an objective sleep measurement during the nap as the sleep obtained cannot be adequately quantified. Given that the benefits of a nap are often determined by the duration and sleep architecture (i.e. greater SWS is proposed to facilitate cellular restitution) conclusions cannot be drawn on the quantity or quality of the naps (Fushimi et al. 2008; Botonis et al. 2021). Future studies should objectively measure sleep using polysomnography or actigraphy to evaluate the mechanism of action of a nap that may lead to improvements in physical and cognitive performance.

As anticipated, there was an effect for load on the bar with peak values at 40% and lowest at 80% of 1RM for all variables, consistent with force velocity properties of skeletal muscle (Ammar et al. 2018; see Figure 6.2). In contrast, average power did not peak at 80% of 1RM, the explanation for this is unclear but likely due to sleep restriction. For perceived exertion, ratings were lowest following the 60-min nap which may infer the nap was effective at restoring responses to muscle fatigue. A recent study that examined strength (via maximal voluntary contraction) following a 40- and 90-min nap without sleep restriction, reported reduced muscle soreness (assessed by the delayed onset muscle soreness questionnaire) compared to no nap (Boukhris et al. 2020). Following sleep restriction, perceived muscle fatigue/soreness may be more exacerbated as sleep loss has been associated with changes in pain perception (Faraut et al. 2015).

The literature suggests there may be a dose dependent effect on cognitive performance, where greater sleep loss increases performance ‘lapses’ (Anholá and Polo-Kantola, 2007). In contrast the current findings report greater

test errors after 4 h of SR compared to 3 h (SR₃: 0.4 ± 0.2 , 95% CI: 0.0-0.7; SR₄: 1.0 ± 0.2 , 95% CI: 0.6-1.3). Since both experimental groups achieved < 4 h of sleep per night all participants experienced similar durations of sleep restriction. For the Stroop task, SR₄ had higher total scores in the congruent condition. This may suggest that participants were able to complete the task at a faster pace but incurred greater test errors due to performance lapses and reduced vigilance. Overall, this confirms that even acute bouts of sleep restriction can impair cognitive function in healthy populations, as previously shown (Van Dongen et al. 2003). Irrespective of these conclusions there is no consensus on the effect of sleep loss on executive function, particularly when employing the Stroop test. Compared to the psychomotor vigilance test (PVT), the Stroop task requires a short time on task and minimal bouts of attention and vigilance; two key functions impaired by sleep loss (Cunningham et al. 2018). Participants may also experience a Stroop learning effect and develop ‘reading suppression responses’ that may mask effects of sleep restriction (Sagaspe et al. 2006). This could explain the lack of significance as all participants were familiarised on two occasions with this test before beginning experimental conditions.

As anticipated subjective sleepiness and negative mood states (fatigue, confusion, tension and anger) increased at 07:00 h with a further increase after 12:00 h, likely due to a natural propensity to sleep in the early afternoon where sleepiness increases and alertness declines (Monk, 2012). In addition to the major peak in sleep propensity that typically occurs at nighttime there is a secondary increase in the afternoon accompanied by reduced alertness and greater sleepiness hence the term ‘post lunch dip’ (Bes et al. 2009). Positive mood states followed an inverted manner, with lowest values at 07:00 h and decreasing further from 14:00-17:00 h. Despite a time-of-day effect, no differences were observed between nap lengths, suggesting the nap was ineffective at restoring perceptual values. Alternatively, the acute nature and degree of sleep restriction may not have been severe enough to impact performance. This disagrees with existing literature, that reports a midday nap can reduce fatigue and improves mood in athletes, likely due to multiple physiological and psychological mechanisms (Hsouna et al. 2019; Botonis et al. 2021). A possible explanation for these outcomes is the duration and/or quality of the naps as prior studies have found greater slow wave sleep during a nap enhances cognitive function and alertness (Ong et al. 2020). Without objective measures of sleep, it is difficult to confirm this hypothesis. If participants struggled to initiate sleep or experienced frequent awakenings due to the laboratory setting it is possible they remained in lighter sleep stages (N1 and N2). However, this is merely speculation and cannot be confirmed.

6.6.1 Conclusion

There was no dose response for sleep loss on strength performance measures (either maximal or submaximal, with different cognitive components). Such that both independent groups had similar values. However, there was a dose response and significant group effect for measures of cognitive function (Stroop task). This contributes to existing literature and confirms that cognitive performance is impaired to a greater extent than physical performance under acute bouts of sleep restriction. As stated previously, the lack of a dose response for maximal and submaximal strength measures and subjective responses is likely due to both groups achieving similar sleep durations in the 3 versus 4 h opportunity. Future studies should also aim to evaluate the effectiveness of naps in both biological sexes, as recent findings suggest females may respond differently to sleep loss than males. Other populations should also be explored such as those with different individual preferences (such as habitual versus non habitual nappers) or athletes from a range of sports to provide an in-depth assessment on nap interventions after sleep loss.

6.6.2 Limitations

This study identified limitations particularly regarding the absence of objective sleep monitoring during the nap period which prevented the assessment of sleep architecture and the ability to confirm whether participants entered slow wave sleep; particularly during the 60-minute nap. This restricted the ability to determine whether sleep inertia contributed to cognitive function and perceptual response outcomes. To address this limitation, the following Chapter (Chapter 7) incorporated PSG monitoring into the study protocol during the nap periods. Additional data collection points were also introduced with testing immediately post nap and 45 minutes post nap to assess the presence and dissipation of sleep inertia. These adjustments were informed by the issues encountered in the current study.

Chapter 7:

Investigating the mechanisms of action of a nap following two nights of partial sleep restriction, which nap duration is optimal?

The data from this study was presented at the European College of Sport Science Conference, Glasgow 2024. This chapter explores a novel approach by measuring temperature change and sleep architecture across the duration of a diurnal nap.

7.1 Introduction

Naps are frequently referred to as a period less than 50% of the major sleep period that occur in addition to nocturnal sleep (Lastella et al. 2021). Individuals may indulge in a nap to prepare for or in response to sleep loss, others may nap out of habit (Milner and Cote, 2009). According to the Sleep Foundation, 81 % of the US population have taken a nap within the previous 3 months, with 7 % napping daily (Sleep Foundation, 2023). Napping can provide immediate benefits such as increased alertness, improved motor performance and mood regulation (Lou et al. 2024). The extent of these benefits may be influenced by factors such as prior sleep, time and duration of the nap and individual characteristics such as age, sex, genetics (Milner and Cote, 2009). It has been well documented that the best opportunity to nap is in line with the circadian dip in alertness and temperature when there is a greater tendency to sleep, between 13:00 and 17:00 h (Monk, 2012). The optimal length of a nap is still yet to be alluded to, with prior research employing nap durations anywhere from 5 to 60 min. The potential disadvantage of longer naps is sleep inertia that may result in a decline in performance upon waking (Faraut et al. 2015).

Thermoregulatory mechanisms are fundamental to sleep. As the body prepares for sleep, heat loss occurs via vasodilation leading to a resultant increase in distal and proximal temperatures (Nicol, 2019). After lights out core body temperature decreases, due to relaxation and eye closure, resulting in shorter sleep onset latency as heat re-distributes from the core to the shell (Gilbert et al., 2004). The differences between nocturnal sleep and a ‘nap’ make it difficult to extrapolate the reduction of core body temperature during an 8 h sleep to that of a nap < 75 min. Following sleep restricted, circadian rhythms, such as changes in body temperature, may be masked due to the homeostatic drive of greater sleep pressure (Kräuchi et al., 2006). There is limited research on temperature change during a nap and therefore it is unknown how temperature may differ following sleep restriction.

Sleep loss is still neglected as a public health issue with little progression into investment, policy change and evidence-based support (Leong and Chee, 2023). The effects of acute sleep restriction on performance and psychological variables are discussed further in Chapter 2, section 2.5 of the review. Considering these effects, student athletes are a population that are often overlooked in research, yet they face unique challenges. The burden of academic pressures and sport specific stressors may be overwhelming to manage (Blumert et al. 2007).

Therefore, the purpose of the present study was to determine the mechanisms of action of a 30- and 60-min nap compared to a control condition of no nap (SR₀), by measuring skin and rectal temperature and sleep architecture

following sleep restricted (3 h per night for 2 nights). Further aims were to investigate the effect of a nap opportunity on exercise performance and the time-of-day effects on mood state, cognitive function and subjective values of alertness and sleepiness. It was hypothesised that a longer nap of 60 minutes would provide an opportunity to obtain greater NREM and REM sleep during the nap period compared to a 30-minute nap and no nap. More restorative sleep would translate to performance enhancement and restored alertness; however, these improvements would not be present immediately post waking due to the likely occurrence of sleep inertia.

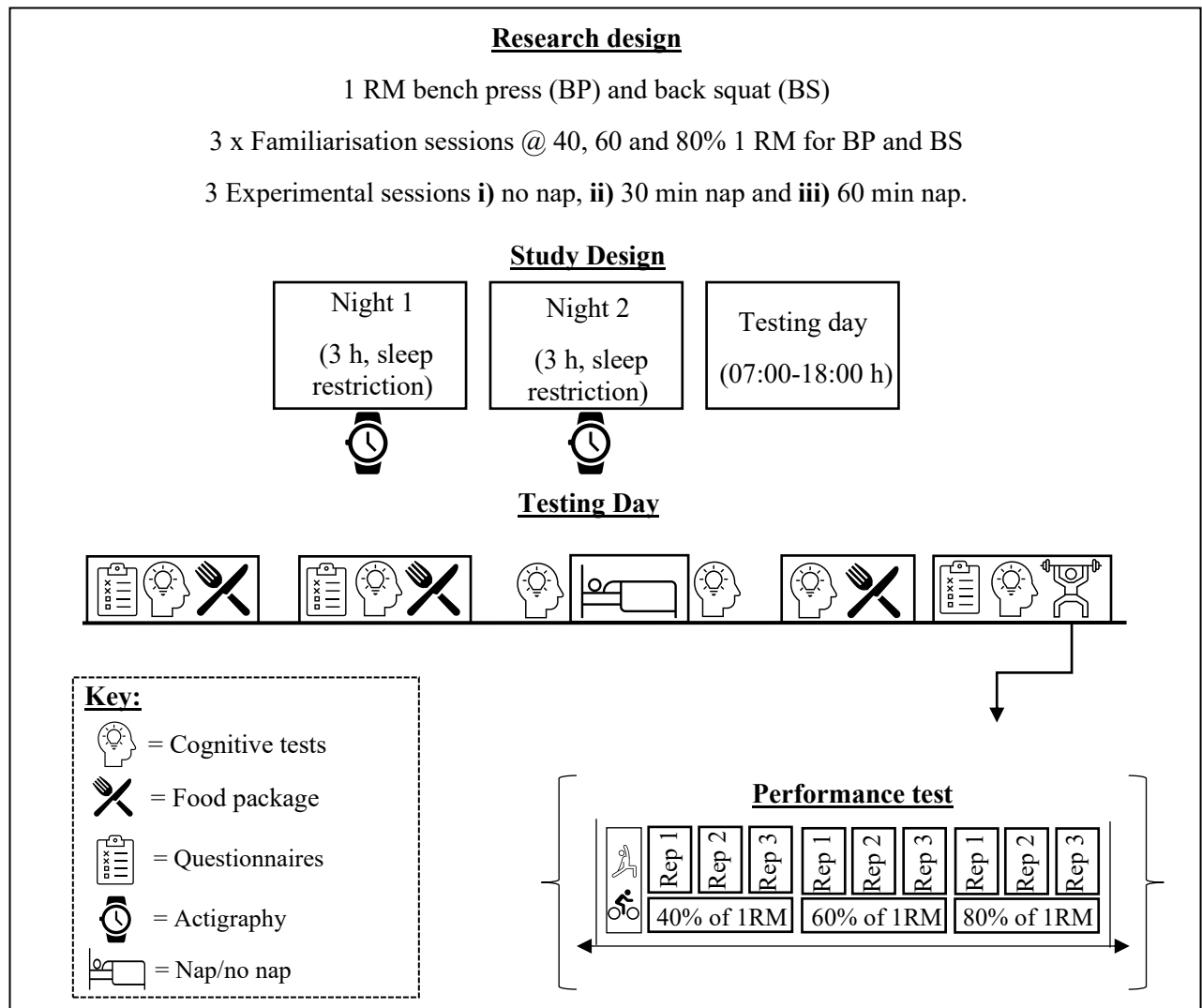
7.2 Methods

7.2.1 Participants

Eleven males as identified by sex and gender (refer to Table 3.1 for characteristics), participated in the study (Ethics code: 22/SPS/061).

7.2.2 Research Design

The three experimental conditions all followed two consecutive nights of partial sleep deprivation at the participants home, retiring at 03:30 h and waking at 06:30 h. Experimental conditions consisted of reclining 1) at 45 degrees in a hospital bed for 60 min remaining awake and able to read in ambient lighting of 250 Lux (SR_0) from 13:00 to 14:00 h, 2) in a supine position for 30 min in complete darkness and attempt to sleep (SR_{30}) from 13:00 to 13:30 h, 3) in a supine position for 60 min in complete darkness and attempt to sleep (SR_{60}) from 13:00 to 14:00 h. This session took place in the university sleep laboratory and the participants were asked to remain in bed until the session finished. All experimental sessions were counterbalanced in order of administration to minimise any potential learning effects (Monk and Leng, 1982).



had finished. The contents of the meals provided were standardised across all participants, the only difference being the quantity of food which was relative to each individual's body mass. At 12:00 h participants arrived at the laboratory for the afternoon session for all experimental conditions to allow sufficient time to prepare and kit up the participant with polysomnography for 13:00 h and placement of skin thermistors. During the SR₀ condition participants were required to remain awake in the sleep laboratory in the bed at a 45-degree angle. Participants were kitted with PSG across all conditions to standardise experimental conditions. For participants in the SR₀ condition they were instructed to remain in bed for the full 60 minute duration but allowed to read or listen to music. A researcher remained in the room throughout this period to ensure the participant did not fall asleep. To maintain consistency with the nap conditions, the researcher did not engage in verbal communication however they regularly checked on the participant using non-verbal cues such as a thumbs up gesture or a nod. Lights were dimly lit at 150 lux to mimic similar conditions to SR₃₀ and SR₆₀. Once the nap protocol had finished, all equipment was removed from the participant.

7.2.4 Appetite and mood ratings

For ratings of appetite and mood, participants completed a series of 8 questions on a 0-100 mm scale with 0 being 'Not at all' and 100 being 'Extremely'. To answer each question, they were asked to draw a vertical line on the scale in relation to how they felt. These questions were asked at four time points across the day (07:00, 11:00, 14:00 and 17:00 h) and included statements such as 'I feel hungry', 'I have a desire to eat something savoury' and 'I feel physically tired'. The scale used has been previously validated in nutritional studies.

7.2.5 Core and Skin Temperature

To measure skin temperature participants were fitted with 7 skin thermistors (Grant Instruments, Cambridge, UK), attached using tape. Thermistors were placed on the following locations on the body: Infraclavicular fossa, midpoint of the forearm, midpoint of the back of the hand, 1 cm above the navel, thigh, calf and the arch of the foot (Ramanathan, 1964). To measure core body temperature a rectal probe was inserted 10 cm past the anal sphincter. Both skin (T_{skin}) and rectal (T_{rec}) temperature values were recorded for 5 min before the nap and for the duration of the nap/rest period.

7.2.6 Polysomnography

Sleep was recorded using polysomnography with an ambulatory monitor (SOMNOMedics, GmbHTM, Germany). Details of the PSG are outlined in Chapter 3, section 3.3.1 of General methods.

7.2.7 Cognitive function

In addition to the Stroop test, an additional cognitive test was employed to assess attentional response using Gorilla software. The cognitive tests were conducted at six time points across the testing day: 07:00, 11:00, 12:45 (pre-nap), 13:30/14:00 (post-nap), 14:15/14:45 (45 min post-nap) and 17:00 h (prior to exercise protocol).

7.2.8 Statistical Analysis

For the following variables a generalised mixed model (GMM) with gamma distribution was conducted: Polysomnography, temperature at sleep onset, hunger scales, Stroop test and dot probe outputs. For rapid eye movement (REM) and NREM 3 sleep variables a GMM with Poisson distribution was ran to account for 0 values. Fixed effects included experimental condition (SR₀, SR₃₀, SR₆₀) and time of day effects, with random effects accounting for individual differences. For the remaining variables (exercise performance, temperature values, POMS, Stanford sleepiness and Waterhouse questionnaire) two-way ANOVA with repeated measures with two factors for experimental condition and time of day were conducted. Please refer to Chapter 3, section 3.4 of General Methods for further detail of the statistical analysis.

7.3 Results

7.3.1 Performance variables (measured at 17:00 h)

7.3.2 Grip strength

Left- and right-hand grip strength values were similar across conditions with no significant main effect for condition ($P = 0.765$, $P = 0.960$, respectively).

7.3.3 Bench Press

Performance variables showed no main effect for experimental condition with average power, average velocity and peak velocity values remaining similar across conditions. Load on the bar had a significant effect on all performance variables with average velocity, displacement and peak velocity achieving greatest values at 40% of 1RM and then decreasing with greater load ($P < 0.001$, see Table 7.1). For average power the greatest values were at 60% of 1RM ($P = 0.010$). No interactions were found between condition and load (see Table 7.1). For ratings of perceived exertion (RPE) there was a significant main effect for condition for RPE breathing ($P < 0.001$) only, with significantly greater values in the no nap condition (SR_0). There was a significant effect for load for RPE, RPE breathing and RPE muscle fatigue values ($P < 0.001$) with highest values reported at maximum load (80% of 1RM). An interaction was found between condition and load with greater RPE breathing values reported at 80 % of 1RM across all conditions.

7.3.4 Back Squat

There was no significant main effect for experimental condition for all back squat performance variables (see Table 7.1). Similar to bench press, load on the bar had a significant effect on average power, average velocity, displacement and peak velocity, with greatest values at 40% followed by a continual decrease in all variables at 60 and further at 80% of 1RM ($P < 0.05$). There were no significant interactions between condition and load. There was no significant effect for condition for RPE values, though a significant effect for load was identified for RPE, RPE Breathing and RPE Muscle fatigue ($P < 0.001$). No interactions were found between condition and load on the bar ($P > 0.05$).

Table 7.1: F values and P values for all performance variables measured in the study and rating on perceived exertion (RPE) for bench press and back squat. **Bold** values indicate significant values ($P < 0.05$).

Variable	Mean SD		Condition	Load	Interaction
	SR ₀	SR ₃₀	SR ₆₀		
Grip Strength (N)					
Left	39.4 ± 2.6	39.0 ± 2.7	39.5 ± 2.6	F _{1,5, 15,4} = 0.20 (P = 0.765)	
Right	42.6 ± 2.8	42.7 ± 3.0	42.4 ± 2.8	F _{2,0, 20,0} = 0.40 (P = 0.960)	
Bench Press					
Average Power (W)	341.0 ± 28.1	359.5 ± 32.2	356.1 ± 31.0	F _{1,2, 12,4} = 0.76 (P = 0.426)	F _{2,9, 29,1} = 0.85 (P = 0.476)
Displacement (cm)	44.0 ± 1.2	44.9 ± 1.8	44.7 ± 1.8	F _{1,5, 15,2} = 0.60 (P = 0.057)	F _{2,3, 23,4} = 1.95 (P = 0.159)
Average Velocity (ms ⁻¹)	0.6 ± 0.1	0.7 ± 0.1	0.7 ± 0.1	F _{1,1, 11,4} = 0.66 (P = 0.451)	F _{3,2, 32,0} = 0.98 (P = 0.421)
Peak Velocity (ms ⁻¹)	0.9 ± 0.1	1.0 ± 0.1	1.0 ± 0.1	F _{1,2, 11,8} = 1.32 (P = 0.283)	F _{3,2, 31,7} = 1.25 (P = 0.305)
RPE (6-20)	11 ± 0	11 ± 0	10 ± 0	F _{1,9, 19,4} = 0.29 (P = 0.744)	F _{2,1, 51,5} = 0.41 (P = 0.683)
RPE Breathing (6-20)	13 ± 0	10 ± 0	10 ± 0	F_{2,0, 20,0} = 51.37 (P < 0.001)	F_{1,9, 18,8} = 21.16 (P < 0.001)
RPE Muscle Fatigue (6-20)	10 ± 0	10 ± 0	10 ± 0	F _{2,0, 20,0} = 0.83 (P = 0.452)	F _{1,8, 18,0} = 1.42 (P = 0.266)
Back Squat					
Average Power (W)	982.9 ± 71.3	1000.0 ± 72.1	930.7 ± 77.8	F _{1,2, 12,2} = 0.70 (P = 0.447)	F _{2,6, 26,2} = 0.87 (P = 0.455)
Displacement (cm)	60.2 ± 3.1	61.3 ± 3.0	60.4 ± 2.4	F _{2,0, 20,0} = 0.39 (P = 0.684)	F _{4,0, 40,0} = 2.11 (P = 0.098)
Average Velocity (ms ⁻¹)	0.7 ± 0.1	0.7 ± 0.1	0.7 ± 0.1	F _{2,0, 20,0} = 0.63 (P = 0.543)	F _{2,6, 26,4} = 1.45 (P = 0.252)
Peak Velocity (ms ⁻¹)	1.1 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	F _{1,3, 12,6} = 2.73 (P = 0.119)	F _{2,7, 27,1} = 1.54 (P = 0.230)
RPE (6-20)	12 ± 0	12 ± 0	13 ± 0	F _{2,0, 20,0} = 1.18 (P = 0.327)	F _{1,6, 15,6} = 1.25 (P = 0.303)
RPE Breathing (6-20)	12 ± 0	11 ± 0	11 ± 0	F _{2,0, 20,0} = 0.76 (P = 0.481)	F _{2,5, 24,9} = 0.35 (P = 0.751)
RPE Muscle Fatigue (6-20)	12 ± 0	11 ± 0	11 ± 0	F _{2,0, 20,0} = 2.12 (P = 0.147)	F _{2,8, 28,1} = 1.47 (P = 0.245)

7.4 Physiological variables (measured 13:00-14:00 h)

7.4.1 Core body temperature across the nap duration

There was no significant main effect for condition for core body temperature ($F_{2,0, 20,0} = 0.91$, $P = 0.419$). There was a significant main effect for time ($F_{1,7, 17,4} = 55.68$, $P < 0.001$; Figure 6.2), with pairwise comparisons revealing a gradual decrease in temperature across the duration of the nap. There was no significant interaction between condition and time ($F_{2,4, 15,9} = 1.54$, $P = 0.233$).

7.4.2 Distal temperature across the nap duration

There was no significant effect for condition for distal temperature ($F_{1,3, 13,0} = 0.19$, $P = 0.736$). However, there was a significant main effect for time ($F_{1,7, 17,5} = 13.67$, $P < 0.001$). Distal temperature gradually increased across the nap duration and peaked at 75% of the nap duration ($32.33 \pm 0.73^{\circ}\text{C}$). There was no interaction between condition and time ($F_{2,5, 25,1} = 1.53$, $P = 0.234$).

7.4.3 Proximal temperature across the nap duration

There was no significant effect for condition for proximal temperature ($F_{1,6, 15,7} = 1.31$, $P = 0.289$). There was a significant effect for time point ($F_{1,8, 18,0} = 17.13$, $P < 0.001$) with proximal temperature gradually increasing across the nap duration with the lowest value at 0 min ($30.32 \pm 0.15^{\circ}\text{C}$) and peak values at 100% of nap duration ($31.30 \pm 0.18^{\circ}\text{C}$). However, there was a significant main effect for time ($F_{1,5, 15,1} = 80.57$, $P < 0.001$). There was a no significant interaction between condition and time ($F_{2,2, 21,7} = 1.33$, $P = 0.288$).

7.4.4 Trec-Tskin

There was no significant effect for condition ($F_{1,3, 13,0} = 0.75$, $P = 0.437$). There was a significant effect for time point ($F_{1,4, 14,2} = 13.40$, $P = 0.001$) with a drop in temperature gradient from 0% ($4.40 \pm 0.23^{\circ}\text{C}$) to 75% ($2.69 \pm 0.20^{\circ}\text{C}$) of the nap duration followed by a slight increase at 100% of nap duration (3.13 ± 0.48). However, there was no significant interaction between condition and time ($F_{1,8, 18,3} = 0.81$, $P = 0.452$).

7.4.5 Core body temperature change from start of the nap to sleep onset

There was a significant main effect for condition ($X^2 = 4.76$, $df = 1.00$, $P = 0.029$). Pairwise comparisons revealed that rectal temperature changed at a greater rate from wake to sleep onset in SR₃₀ compared to SR₆₀ (MD: -0.21°C , 95% CI: -0.40 to -0.02°C). There was a no significant effect for time point ($X^2 = 1.33$, $df = 1.00$, $P = 0.248$) or interaction between condition and time point ($X^2 = 0.34$, $df = 1.00$, $P = 0.559$).

7.4.6 Distal temperature

There was no significant main effect for condition ($X^2 = 0.18$, $df = 1.00$, $P = 0.673$). There was a no significant effect for time point ($X^2 = 1.15$, $df = 1.00$, $P = 0.284$) and interaction between condition and time point ($X^2 = 0.23$, $df = 1.00$, $P = 0.632$).

7.4.7 Proximal temperature

There was no significant main effect for condition ($X^2 = 0.00$, $df = 1.00$, $P = 0.953$). There was a no significant effect for time point ($X^2 = 1.89$, $df = 1.00$, $P = 0.169$) and interaction between condition and time point ($X^2 = 0.71$, $df = 1.00$, $P = 0.399$; see Figure 6.3).

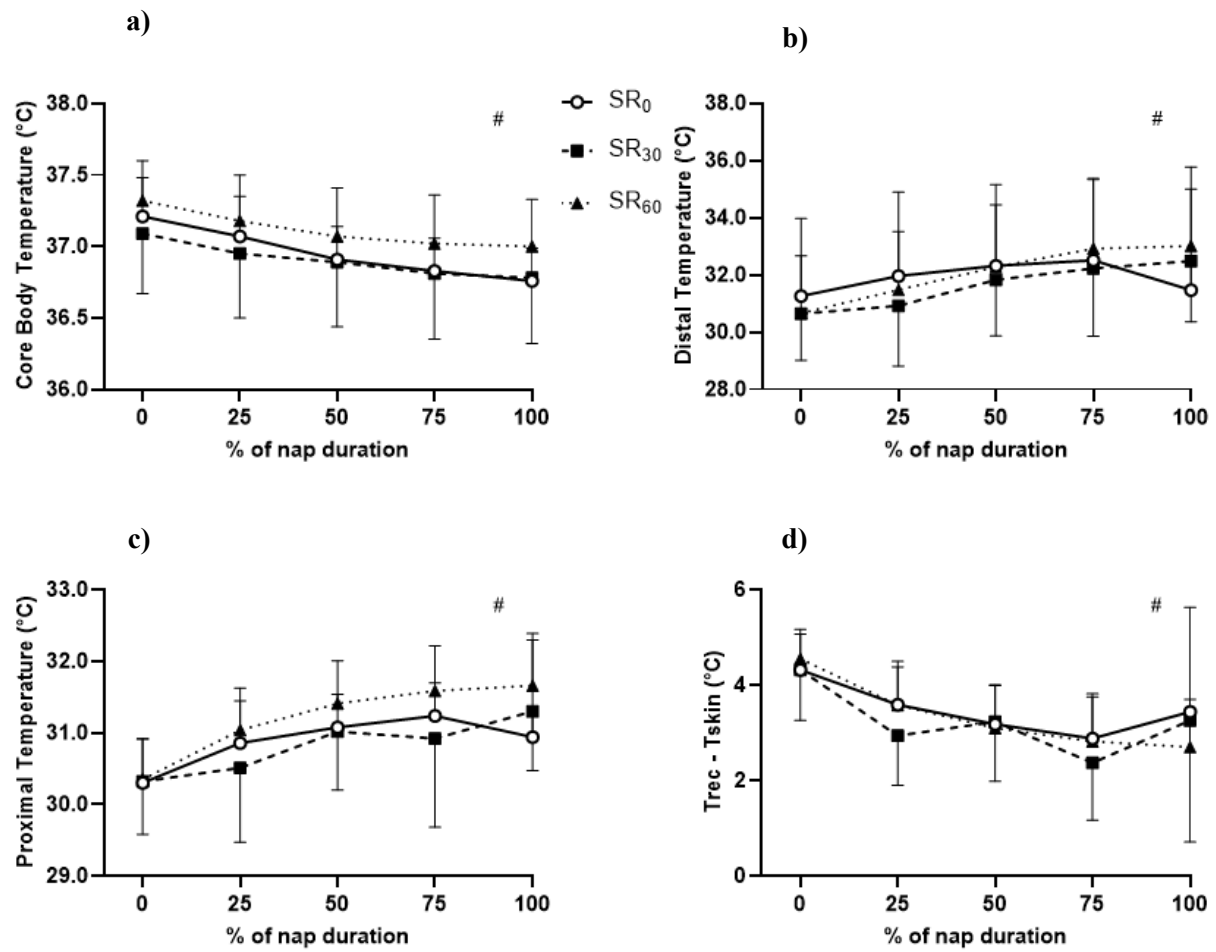
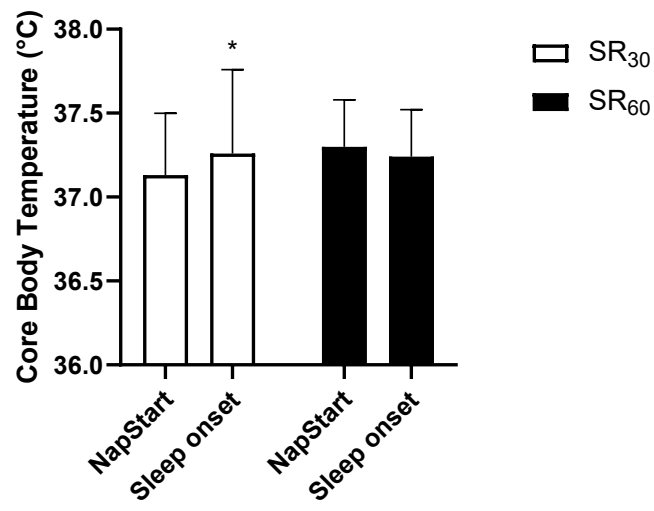
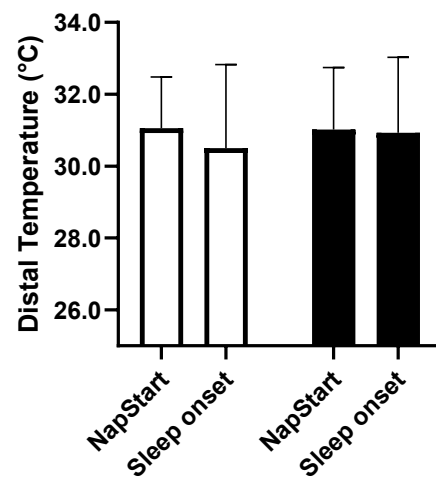


Figure 7.2. (a) Mean values for core body temperature (b) distal temperature (c) proximal temperature and (d) Trec-Tskin (rectal temperature minus total skin temperature), across percentage of nap duration for the three experimental conditions. # indicates a significant ($P < 0.05$) effect for time point.

a)



b)



c)

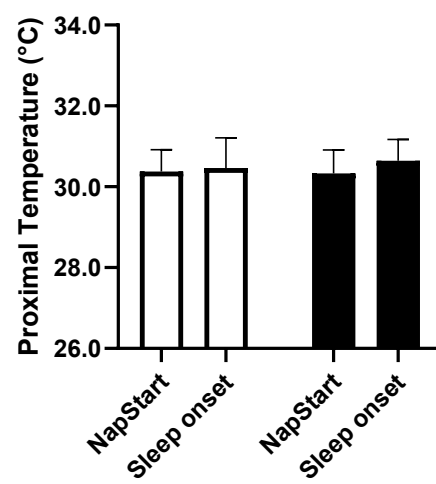


Figure 7.3. (a) Mean values for core body temperatures **(b)** distal temperature and **(c)** proximal temperature at the start of the nap session and at sleep onset for the nap conditions (SR₃₀ and SR₆₀). * Indicates significance difference between temperature from nap start to sleep onset ($P < 0.05$).

Polysomnography

7.4.8 Total sleep time

The model explains 91.4% of the variance due to fixed effects (marginal $R^2 = 0.914$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition ($X^2 = 61.4$, $df = 1.00$, $P < 0.001$). Pairwise comparisons report greater sleep duration during 60-min nap compared to 30-min nap (MD: 24:28, 95% CI: 18:22 – 30:37 mm:ss).

7.4.9 Sleep efficiency

There was no significant effect for condition ($P = 0.748$) with similar sleep efficiency values between the two experimental conditions (see Table 7.2).

7.4.10 Wake bouts

The model explains 10.9% of the variance due to fixed effects (marginal $R^2 = 0.109$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant effect for condition ($X^2 = 5.70$, $df = 1.00$, $P = 0.017$), with greater awakenings during the 60-min nap when compared to the 30-min nap (MD: 03:10, 95% CI: 00:55 - 06:03 mm:ss).

7.4.11 Sleep latency

The model explains 36.9% of the variance due to fixed effects (marginal $R^2 = 0.369$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition for ($X^2 = 4.67$, $df =$

1.00, $P = 0.031$). Pairwise comparisons reveal that sleep latency was longer during the 60-min nap compared to 30-minute nap (MD: 02:35, 95% CI: 00:18 – 04:10 mm:ss).

7.4.12 NREM 1

The model explains 1% of the variance due to fixed effects (marginal $R^2 = 0.001$) and 100% when including random effects (conditional $R^2 = 1.000$). There was no significant effect for condition ($P = 0.897$) with NREM 1 sleep duration relatively similar across the two experimental conditions.

7.4.13 NREM 2

The model explains 14% of the variance due to fixed effects (marginal $R^2 = 0.140$) and 100% when including random effects (conditional $R^2 = 1.000$). There was no significant effect for condition, with NREM 2 sleep duration relatively similar across the two experimental conditions.

7.4.14 NREM 3

The model explains 33.4% of the variance due to fixed effects (marginal $R^2 = 0.334$) and 99.9% when including random effects (conditional $R^2 = 0.999$). There was a significant main effect for condition for NREM 3 sleep ($P < 0.001$), with pairwise comparisons reporting greater durations of N3 in the 60-min nap compared to 30-minute nap.

7.4.15 REM

The model explains 28.9% of the variance due to fixed effects (marginal $R^2 = 0.289$) and 99.1% when including random effects (conditional $R^2 = 0.991$). There was a significant main effect for condition ($X^2 = 14.3$, $df = 1.00$, $P < 0.001$). Pairwise comparisons report greater durations of REM sleep during the 60-min (M: 04:60 \pm 11:20 mm:ss, 95% CI: 0 - 1 minutes) nap compared to 30-minute nap (M: 00:11 \pm 00:33 mm:ss, 95% CI: 0 - 34 min).

Table 7.2: Summary of the polysomnography variables measured during the nap durations. Mean and standard deviations (SD) are presented for SR₃₀ and SR₆₀ conditions. Chi-squared (χ^2), degrees of freedom (df) and P value are provided for significance between conditions.

Variable	Mean \pm SD		χ^2	Condition	
	SR ₃₀	SR ₆₀		df	P
Sleep Efficiency (%)	80.2 \pm 19.2	82.0 \pm 17.1	0.10	1.00	0.748
Sleep Latency (mm:ss)	03:25 \pm 01:49	05:04 \pm 03:14	2.80	1.00	0.094
Total Sleep Time (mm:ss)	24:48 \pm 05:19	48:43 \pm 10:05	61.40	1.00	< 0.001
Awake (mm:ss)	07:23 \pm 06:50	11:42 \pm 10:05	5.70	1.00	0.017
REM (mm:ss)	00:11 \pm 00:33	05:00 \pm 11:20	14.30	1.00	< 0.001
N1 (mm:ss)	09:27 \pm 8:40	07:27 \pm 03:26	0.02	1.00	0.897
N2 (mm:ss)	11:26 \pm 06:48	18:03 \pm 10:25	2.97	1.00	0.085
N3 (mm:ss)	06:23 \pm 05:57	18:43 \pm 13:04	2464.00	1.00	< 0.001

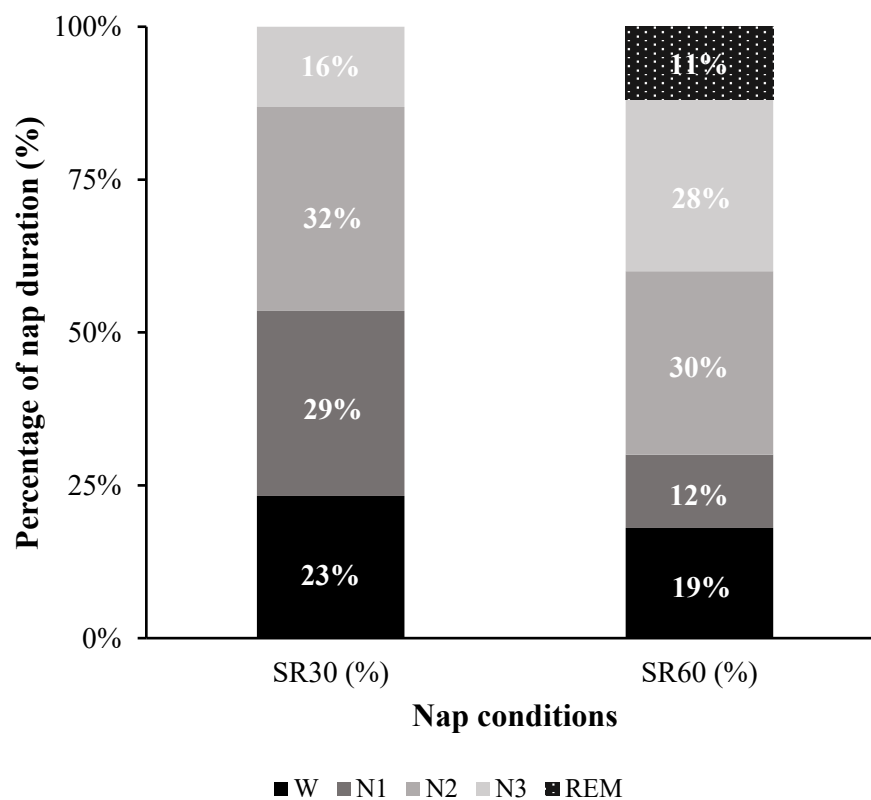


Figure 7.4. Stacked bar chart displaying the duration of each sleep stage as a percentage, in the 30 (SR₃₀) and 60 minute (SR₆₀) napping conditions.

7.5 Cognitive function (measured at 07:00, 11:00, 12:45, 14:00, 14:45 and 17:00 h)

7.5.1 Stroop task Black ink – Total

The model explains 7.8% of the variance due to fixed effects (marginal $R^2 = 0.078$) and 40.6% when including random effects (conditional $R^2 = 0.406$). There was no significant main effect for condition for the total score on the black ink test. However, there was a significant time of day effect ($P < 0.001$), with an increase in total score between 14:00 and 17:00 h. There were no significant interactions between condition and time-of-day for total score (see Table 7.3).

7.5.2 Blank ink - Errors

The model explains 39.9% of the variance due to fixed effects (marginal $R^2 = 0.399$) and 99.9% when including random effects (conditional $R^2 = 0.999$). There was no significant effect for condition or time-of-day ($P = 0.160$ and $P = 0.562$, respectively). There was no significant interaction between condition and time-of-day effects ($P = 0.709$).

7.5.3 Congruent– Total

The model explains 39.9% of the variance due to fixed effects (marginal $R^2 = 0.399$) and 99.9% when including random effects (conditional $R^2 = 0.999$). There was no significant main effect of condition or time of day for total score on the word reading test ($P = 0.705$, $P = 0.157$, respectively). There was also no significant interaction between condition and time-of-day factors for total score ($P = 0.412$, see Table 7.3).

7.5.4 Congruent - Errors

The model explains 51.9% of the variance due to fixed effects (marginal $R^2 = 0.519$) and 100% when including random effects (conditional $R^2 = 1.000$). There was a significant main effect for condition ($P = 0.014$), with post-hoc tests reporting greater errors in SR_0 compared to SR_{30} condition ($P = 0.010$). There was no significant time-of-day effect ($P = 0.092$) and no interaction between condition and time of day for number of errors ($P = 0.460$).

7.5.5 Incongruent- Total

The model explains 24.5% of the variance due to fixed effects (marginal $R^2 = 0.245$) and 100% when including random effects (conditional $R^2 = 1.000$). There was no significant main effect of condition for total score in the incongruent test ($P = 0.431$). However, a significant time-of-day effect was found for total score ($P = 0.004$), with the lowest total achieved at 11:00 h (63.5 ± 10.4), followed by an increase at 13:00 h (67.8 ± 11.2) and 14:00 h (69.1 ± 10.8), with a slight reduction at the remaining two time points (68.6 ± 8.9 and 67.5 ± 9.1). No interactions between condition and time of day were reported for total score (see Table 7.3).

7.5.6 Incongruent - Errors

The model explains 32.9% of the variance due to fixed effects (marginal $R^2 = 0.329$) and 99.9% when including random effects (conditional $R^2 = 0.999$). There was no significant effect for condition for errors in the incongruent test ($P = 0.909$). There was no significant time-of-day effect for incongruent errors ($P = 0.147$). There was no significant interaction between condition and time of day effects (see Table 7.3).

Table 7.3: Summary of the cognitive tests measured across experimental conditions and time of day (TOD). Chi-squared (χ^2), degrees of freedom (df) and P value are provided. **Bold** values indicate significant figures ($0.1 < P > 0.05$).

Variable	Mean \pm SD			Condition			TOD			Interaction (Condition*TOD)		
	SR ₀	SR ₃₀	SR ₆₀	χ^2	df	P	χ^2	df	P	χ^2	df	P
Dot Probe time	23 \pm 9	23 \pm 10	22 \pm 7	2.61	2.00	0.271	60.20	5.00	< 0.001	26.87	10.00	0.003
Stroop Test												
Black Ink Total	118 \pm 16	118 \pm 17	117 \pm 16	0.81	2.00	0.668	24.67	5.00	< 0.001	6.66	10.00	0.757
Black Ink Errors	0 \pm 1	0 \pm 0	1 \pm 1	3.67	2.00	0.160	3.91	5.00	0.562	7.18	10.00	0.709
Congruent Total	107 \pm 16	110 \pm 19	107 \pm 16	0.70	2.00	0.705	7.99	5.00	0.157	10.34	10.00	0.412
Congruent Errors	1 \pm 2	1 \pm 1	1 \pm 1	8.60	2.00	0.014	9.48	5.00	0.092	9.78	10.00	0.460
Incongruent Total	66 \pm 9	67 \pm 10	68 \pm 11	1.68	2.00	0.431	17.45	5.00	0.004	7.66	10.00	0.662
Incongruent Errors	1 \pm 1	1 \pm 1	1 \pm 1	0.19	2.00	0.909	8.17	5.00	0.147	2.84	10.00	0.985

7.5.7 Dot Probe Test

The model explains 84.6% of the variance due to fixed effects (marginal $R^2 = 0.846$) and 99.7% when including random effects (conditional $R^2 = 0.997$). There was no significant effect for condition for measures of attention ($P = 0.271$), yet there was a significant time of day effect ($P < 0.001$). Attentional responses times were highest at 07:00 h (30.5 ± 13.3 s) and decreased across the course of the day with the quickest response times recorded 45 min post ‘nap period’ (19.5 ± 5.36 s) and 17:00 h (20.2 ± 5.26 s). A significant interaction between condition and time-of-day factors was observed ($P = 0.003$, Table 7.3)

7.6 Subjective responses

7.6.1 Appetite responses

There was a significant main effect for condition for ‘feelings of fullness’ (Q2; $P = 0.005$, Figure 7.4), with those in SR₀ (45.9 ± 12.6) reporting greater fullness compared to SR₃₀ (39.2 ± 17.5). There was no significant effect for condition for the remaining questions (Q3-8). There was a significant time-of-day effect for ‘feelings of fullness’

($P = 0.006$) with higher values at 14:00 and 17:00 h compared to 07:00 h (MD: 5.2, respectively). A significant effect for time of day for 'feeling energetic' ($P = 0.007$) was identified, with lower energy levels at 07:00 compared to 17:00 h (MD: 7.2). There was a significant interaction between condition and time-of-day for 'feeling energetic' (Q7; $P = 0.011$).

7.6.2 Caffeine Withdrawal Questionnaire (CWQ)

There was no significant main effect for any factors of the CWQ between conditions when measured at 07:00 h on the testing day ($P > 0.05$, see Table 7.4). The CWQ was completed at four time points across the day, however due to missing data points we only included responses from 07:00 h in the study analysis.

7.6.3 Profile of Mood State

There was no significant main effect for condition for all mood states. However, mood states such as vigour, anger, confusion and depression did significantly differ across the four time points. For positive feelings of vigour, reported values were lowest at 07:00 h (4.1 ± 0.8) and gradually increased across the day with greatest values at 17:00 h (5.5 ± 0.7). In an inverted manner, negative mood states of anger, confusion and depression were highest at 07:00 h and reduced across the day, other than confusion which further increased between 14:00-17:00 h (Figure 7.5). No significant interactions were reported between condition and time of day.

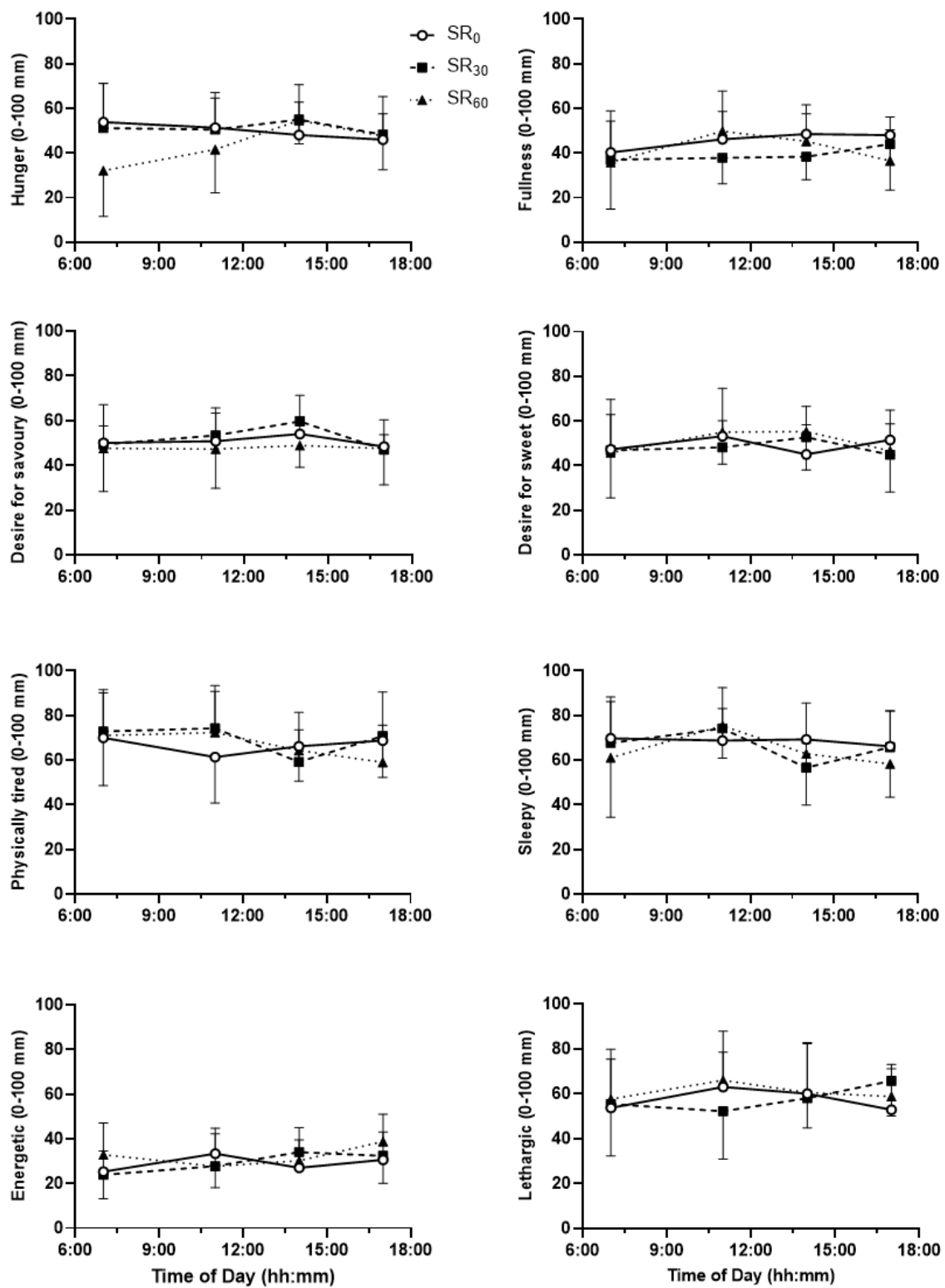


Figure 7.5. Mean and SD (standard deviation) values from responses to the ‘Hunger Scale’ survey assessing appetite ratings at four time points across the testing day for each condition.

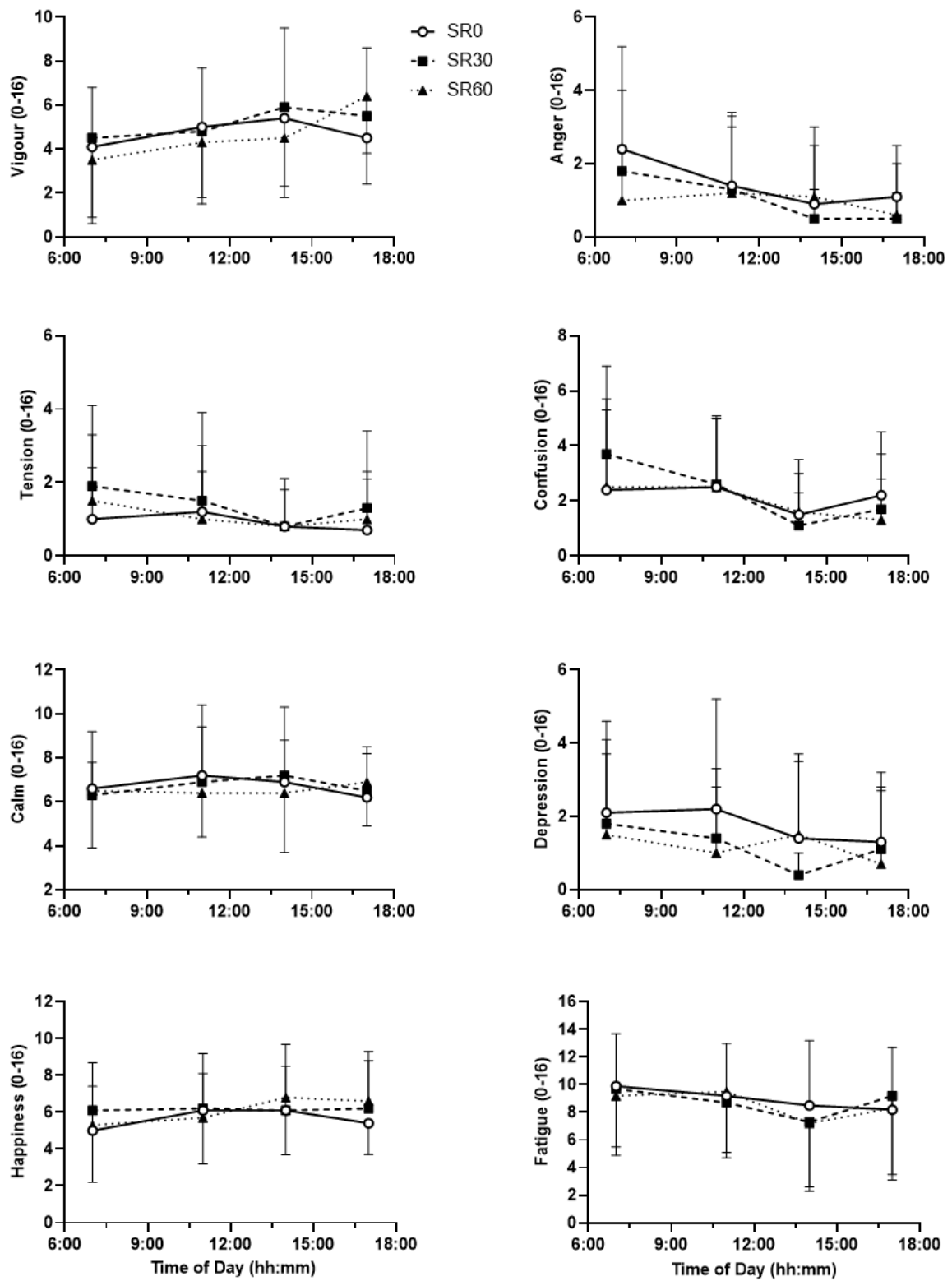


Figure 7.6. Mean and SD (standard deviation) values for mood states assessed using the Profile of Mood States questionnaire at four time points across the testing day for each condition.

7.6.4 Stanford Sleepiness

Subjective sleepiness did not significantly differ between conditions ($P = 0.155$), however there was a significant time of day effect ($P = 0.011$, Table 7.4). Sleepiness was reported to be highest at 07:00 and 11:00 h but gradually decreased across the day with lowest values at 17:00 h.

Table 7.4: Summary of the Stanford Sleepiness, Waterhouse Questionnaire responses and Caffeine withdrawal questionnaire. Mean and SD (standard deviations) are presented for each condition as well as F values and P values for all variables. **Bold** values indicate significant figures ($0.1 < P > 0.05$).

Variables	Mean \pm SD			Condition	TOD	Interaction (Condition*TOD)
	SR ₀	SR ₃₀	SR ₆₀			
Stanford Sleepiness	4 \pm 0	4 \pm 0	4 \pm 0	F _{1.4, 14.0} = 2.21 (P = 0.155)	F_{1.8, 18.2} = 6.08 (P = 0.011)	F _{3.4, 33.8} = 1.96 (P = 0.132)
Waterhouse						
Q1: How easily did you get to sleep?	2 \pm 1	1 \pm 1	1 \pm 1	F _{2.0, 20.0} = 0.53 (P = 0.597)		
Q2: What time did you get to sleep?	3 \pm 1	2 \pm 1	3 \pm 1	F _{1.4, 14.3} = 0.28 (P = 0.686)		
Q3: How well did you sleep?	1 \pm 1	1 \pm 1	1 \pm 1	F _{2.0, 20.0} = 0.03 (P = 0.971)		
Q4: What was your waking time?	-3 \pm 1	-3 \pm 0	-3 \pm 1	F _{1.5, 14.9} = 0.88 (P = 0.407)		
Q5: How alert did you feel after 30 min of waking?	-2 \pm 1	-2 \pm 1	-2 \pm 1	F _{1.2, 11.7} = 0.54 (P = 0.504)		
Caffeine Withdrawal Questionnaire						
Factor 1: Drowsiness/Fatigue	8.7 \pm 3.7	9.5 \pm 2.6	9.3 \pm 3.2	F _{1.3, 11.4} = 0.29 (P = 0.657)		
Factor 2: Decreased alertness	7.7 \pm 2.9	8.6 \pm 2.4	7.9 \pm 2.4	F _{2.0, 18.0} = 0.61 (P = 0.552)		
Factor 3: Mood disturbances	4.2 \pm 3.0	3.8 \pm 2.5	3.6 \pm 2.8	F _{2.0, 18.0} = 0.30 (P = 0.742)		
Factor 4: Decreased sociability	2.9 \pm 2.6	3.3 \pm 2.8	3.3 \pm 2.8	F _{1.0, 9.0} = 0.23 (P = 0.642)		
Factor 5: Nausea/upset stomach	0.5 \pm 0.7	0.4 \pm 0.7	0.8 \pm 1.3	F _{1.1, 9.6} = 0.52 (P = 0.501)		
Factor 6: Flu like feelings	2.9 \pm 2.0	2.2 \pm 2.3	2.6 \pm 2.6	F _{1.3, 12.0} = 0.94 (P = 0.379)		
Factor 7: Headache	1.2 \pm 1.1	0.6 \pm 1.0	0.9 \pm 0.9	F _{1.5, 13.9} = 0.73 (P = 0.466)		

7.6.5 Waterhouse

Subjective responses for questions 1-5 did not differ between conditions (Table 7.4).

7.7 Actigraphy

There was no significant main effect for condition and no significant effect for night for all actigraphy variables ($P > 0.05$, Table 7.5). A significant interaction was found between condition and night for time in bed with greater time in bed on night two of the sleep restriction protocol compared to night 1 for SR₀ and SR₃₀.

Table 7.5: Mean \pm SD, F values and P values for all actimetry variables measured in the study. **Bold** values indicate significant figures ($P < 0.05$).

Actimetry Variables	SR ₀		SR ₃₀		SR ₆₀		Condition	Night	Interaction (Condition* Night)
	N1	N2	N1	N2	N1	N2			
Time in bed (hh:mm)	03:12 \pm 0:30	03:34 \pm 00:32	03:14 \pm 0:14	03:24 \pm 00:26	03:13 \pm 0:33	03:12 \pm 0:21	F _{2.0, 20.0} = 0.47 (P = 0.632)	F _{1.0, 10.0} = 3.19 (P = 0.104)	F_{2.0, 19.7} = 5.22 (P = 0.016)
Actual sleep time (hh:mm)	02:36 \pm 0:26	03:02 \pm 00:39	02:41 \pm 0:19	02:52 \pm 00:27	02:31 \pm 00:38	02:33 \pm 00:30	F _{2.0, 20.0} = 1.09 (P = 0.355)	F _{1.0, 10.0} = 4.44 (P = 0.061)	F _{2.0, 20.0} = 2.18 (P = 0.142)
Sleep latency (mm:ss)	00:12 \pm 0:13	00:10 \pm 00:15	00:09 \pm 0:14	00:06 \pm 00:10	00:12 \pm 0:14	00:12 \pm 0:13	F _{1.5, 15.4} = 0.64 (P = 0.503)	F _{1.0, 10.0} = 0.23 (P = 0.640)	F _{2.0, 20.0} = 0.06 (P = 0.940)
Sleep Efficiency (%)	81.7 \pm 8.6	84.6 \pm 10.6	83.1 \pm 7.8	84.4 \pm 10.4	78.3 \pm 16.8	79.6 \pm 11.3	F _{2.0, 20.0} = 0.91 (P = 0.420)	F _{1.0, 10.0} = 0.60 (P = 0.457)	F _{2.0, 20.0} = 0.06 (P = 0.938)
Fragmentation Index (%)	24.9 \pm 12.5	19.5 \pm 13.8	21.7 \pm 12.6	26.1 \pm 18.6	24.4 \pm 25.2	25.1 \pm 14.2	F _{2.0, 20.0} = 0.07 (P = 0.931)	F _{1.0, 10.0} = 0.01 (P = 0.941)	F _{2.0, 20.0} = 0.93 (P = 0.409)

7.8 Discussion

This is one of the first studies to simultaneously measure sleep architecture and temperature changes during a diurnal nap, following sleep restriction. While no significant changes in diurnal, proximal or core body temperature between conditions were observed, there were noticeable changes across the nap duration. Polysomnography data revealed SR₆₀ had greater durations of REM and slow wave sleep (N3), despite greater awakenings, compared to SR₃₀. Interestingly physical performance at 17:00 h remained unchanged, with cognitive function and perceptual responses being similar, however time of day effects were observed. These findings suggest a longer nap opportunity allows for more restorative sleep but does not necessarily translate to greater performance outcomes, even compared to no nap.

It is well established that circadian cycles of temperature and sleep are closely related, with sleep onset occurring on the descending portion of the core body temperature (CBT) curve (Gradisar et al. 2004). Recent findings suggest that skin temperature, opposed to CBT, may play a primary role in sleep activation as changes to skin blood flow support heat dissipation from the core. The supine position during a nap, combined with an inactive state, reduce heat production allowing blood vessels to vasodilate and increase both distal and proximal temperatures (Kräuchi et al. 2001). In the current study, temperature changed across the nap duration supposedly due to the redistribution of heat from the core to the shell shortly after sleep onset. As anticipated core body temperature decreased throughout the nap, with greater distal and proximal temperatures. A similar pattern occurred in the no nap condition, despite the absence of sleep, which may be due to relaxation and reduced sympathetic tone or dimmed lighting that may have blunted melatonin secretion (Krauchi et al. 2006). Despite this, changes to skin temperature precede sleep onset suggesting they are not solely sleep related. In addition, there was a significant decrease in temperature from wake to sleep onset in SR₃₀ along with a shorter sleep latency, though this was no significant, compared to SR₆₀. When assessing temperature gradient ($T_{rec} - T_{skin}$) the most significant drop in temperature occurred between the start of the nap session and 75% of the nap duration; at which point temperature plateaued until the session ended. This is one of the few studies to measure core body and skin temperature during no nap and nap durations. Due to the complexity of sleep and temperature networks it remains a challenge to determine their relationship. It is thought that the thermoregulatory system is independent of the sleep homeostat therefore conditions of sleep restriction have little to any effect on thermoregulatory responses.

Following sleep restriction individuals often engage in ‘recovery naps’ to reduce homeostatic pressure and alleviate the negative effects of sleep loss. The architecture of recovery naps distinctly differs from those after normal nocturnal sleep, primarily due to an elevation in slow wave sleep (SWS; Mantua and Spencer, 2017). In the current study, the 60-minute nap contained significantly greater SWS compared to the 30-minute nap (mean difference: 12:20 h:mm). As SWS is considered the most ‘restorative’ sleep stage, a longer nap would hypothetically enhance alertness and reduce sleepiness compared to no nap. Despite this, no differences between conditions were found, though a time-of-day effect for sleepiness was observed with a gradual decline across the day. Positive feelings of vigour increased across the testing day with negative mood states of anger, confusion and depression following an inverse manner (Figure 7.5). Improvements in mood state would typically be expected following a nap compared to no nap, as shown in previous studies. However, the reason for no effect in the current study remains unclear (Scott et al. 2006; Souissi et al. 2020).

Naps that contain SWS have also been associated with improved cognitive function, although these benefits vary between individual’s dependent on their state-trait qualities. Despite occurrence of SWS, no difference was found between conditions but a time-of-day effect for cognitive function was observed. Attentional response, measured using the dot probe test, was slower immediately post-nap but improved when re-tested 45 min later. The delayed response likely reflects sleep inertia, which is present following longer naps (> 30 min) and reduces an individual’s ability to perform. Occurrence of sleep inertia and the time it takes to dissipate are greater when the quantity of SWS is longer (Dutheil et al. 2021). Interestingly, similar test outcomes were reported in the no nap condition, suggesting that the opportunity to ‘relax’ in a reclined position with dimmed lighting and low stimulation might restore alertness. However further research is needed to confirm this. For other markers of cognitive function, there was a decline in performance post nap for the Stroop task (blank ink test). However, 45 min post nap the total score significantly improved and remained elevated 3 h later. For the incongruent Stroop task performance did not recover when re-tested 45 min and 3 h post nap, possibly due to task difficulty. Consistent with the current findings a 5 h SR study observed no cognitive improvements following a 30-minute nap, even after re-testing at 35 minutes post nap (Tietzel and Lack, 2001). The authors suggested additional post nap testing should be extended for several hours to account for lingering effects of inertia. Individual differences may also explain the variability in outcomes with prior research indicating habitual nappers may experience greater nap related benefits compared to no nappers (Ru et al. 2019).

Although SWS is important it remains unclear whether the benefits of a nap occur due to specific sleep stages or simply total sleep duration. The role of REM sleep during a nap remains largely unknown as typically little to no REM occurs in a nap (Mantua and Spencer, 2017). However, in this study, the 60-min nap contained significantly greater REM sleep compared to the 30-min nap, possibly due to a REM rebound following sleep restriction (Milner and Cote, 2009). Previous findings indicate that longer total sleep time and more REM during a daytime nap may enhance working memory, however this was not observed following sleep restriction (Lau et al. 2015). Further research is needed to determine objective measures of sleep during a nap and to understand the role of sleep stages on specific cognitive functions.

The effects of sleep loss on muscle strength remain inconclusive (Lastella et al. 2021). In the current study, maximal and submaximal performance did not differ between conditions at 17:00 h. In agreement, other studies show no effect of sleep loss on resistance training, although some of these studies employed total sleep deprivation opposed to restriction (Meney et al. 1998; Goh et al. 2001; Blumert et al. 2007; Gallagher et al. 2023). Studies that have reported strength deficits following sleep restriction found performance to be worse in the afternoon compared to the morning likely due to prolonged wakefulness (Souissi et al. 2013). However, the current study only measured performance in the afternoon, so cannot elaborate on this. Overall, these findings suggest that muscle strength following sleep loss may depend on exercise type, training status and environment. Prolonged and exhaustive exercise may also exaggerate the negative effect of sleep loss on performance (Craven et al. 2022). Moreover, studies with longer nap durations (90 minutes) that contained both SWS and REM have shown to result in greater performance (Souabni et al. 2021). This may suggest that a 60-min nap may not have been sufficient to improve outcomes. Motivation and perceived exertion also influence maximal performance. Participants in the no nap condition reported higher perceived exertion for breathing that may be due to extended wakefulness and heightened sleep pressure (Table 7.1). The lack of a control condition limits comparison to other studies, as noted in previous work (Gallagher et al. 2023). Future research should use objective sleep measures and explore the interaction between sleep loss and resistance training on skeletal muscle to better inform exercise recommendations following inadequate sleep.

Dietary intake is another factor that may influence performance outcomes following sleep restriction. In the current study dietary intake was controlled and standardised, as suggested in Chapter 2, Table 2.3 for future research considerations. Total calorie intake of the food packages provided were relative to the individuals body mass. These procedures were followed to reduce the impact of diet consumption on study outcomes. Previous research has shown that sleep restriction can lead to individuals consuming greater calorie intake as a means of sustaining energy (Maloney et al. 2023). More palatable foods that are high in fats and carbohydrates are often chosen, linking chronic sleep loss to weight gain (Spaeth et al. 2013; Nedeltcheva et al. 2009). Appetite ratings appear to increase following SR but more so when early awakening is imposed rather than delayed bedtimes (McNeil et al. 2017; Maloney et al. 2023). In this study appetite ratings were recorded across the day and found participants to feel greater ‘fullness’ in the SR₃₀ condition. Fullness was also greater in the afternoon compared to 07:00 h, due to consumption of food across the day (Figure 7.4). Changes in leptin and ghrelin concentrations are thought to contribute to appetite ratings though the literature remains mixed (Spiegel et al. 2004). Regarding caffeine consumption participants were required to abstain throughout the three-day protocol. To assess for potential withdrawal effects participants were required to complete the ‘Caffeine Withdrawal Questionnaire’ (CWQ) on the testing day following two days of abstinence. Across conditions at 07:00 h participants reported relatively similar symptom responses, with the mean values suggesting that participants did not experience severe withdrawal following 48 h of abstinence (Table 7.4). To further reduce the likelihood of withdrawal, habitual caffeine consumption was recorded in pre-screening to ensure participants were low to moderate caffeine consumers. For athletic populations these factors are extremely important as changes to their nutritional intake and metabolism could significantly impact their performance outcomes.

7.8.1 Conclusion

The current study is among the first to explore the physiological mechanisms of a nap following sleep restriction. Temperature data followed the expected pattern and longer nap opportunities allowed for greater occurrences of SWS and REM sleep. However, naps did not enhance performance compared to no nap. The findings may suggest that simply relaxing in an environment with reduced stimulation (i.e. no screen use) may be sufficient to maintain or restore cognitive function and alertness, though further research is needed. Participant flexibility and vigour, confirmed through pre-screening, may explain the lack of differences between conditions as these traits are linked

to an ability to cope better with sleep loss. However, state-trait factors and chronotype are often overlooked in study pre-screening making it difficult to compare participant populations across studies.

7.8.2 Limitations

The study was designed with rigorous controls including dietary intake, familiarisation of protocol, extensive pre-screening and use of gold standard methods. Nonetheless using actigraphy to assess nocturnal sleep limited the ability to measure sleep architecture. The delayed bedtime protocol may have reduced SWS, as SWS dominates the first half of the night. As a result, the 60-min nap may not have been sufficient to recover the SWS deficits from the two nights prior, explaining the lack of performance improvement. The author acknowledges some limitations to the study such as a small sample size which may have led to underpowered statistics. The absence of a control condition also makes it difficult to compare the findings to those of normal sleep conditions.

Chapter 8:

Synthesis of findings

8.1 Achievement of aims and objectives

The primary aim of the thesis was to examine the effects of acute sleep restriction on exercise performance, cognitive function and perceptual responses while exploring nap interventions and investigating methods of sleep measurement. Across the experimental studies, the aim was to provide insight on the optimal timing and duration of naps and to broaden the understanding of the physiological mechanisms influencing the effectiveness of naps. In addition, the validity of sleep measurement methods was investigated to inform guidelines and recommendations for general and athletic populations. Collectively, the findings and recommendations from each chapter aim to support future research in sleep restriction and chronobiology, addressing gaps in the literature.

Aim 1- To review existing literature on the effects of sleep restriction on exercise performance and explore secondary factors such as cognitive function, perceptual responses and time-of-day effects.

The review identified current gaps in the literature such as cognitive function being more sensitive to sleep loss than physical outcomes. It appears that the extent of these effects depends on the type of task being executed. Tasks requiring sustained attention and aerobic or anaerobic capacity, such as time to exhaustion tests or repeated sprints, appear more vulnerable than strength-based tasks. The timing of restriction protocol, also influences the outcomes, with early awakening resulting in greater impairment than delayed bedtime. This is also the case for time-of-day effects with morning performance exhibiting less effects than afternoon sessions. Both early awakening protocols and afternoon scheduling extend the time since awake, increasing the accumulation of homeostatic pressure and heightening the consequences of sleep loss. These findings highlight the importance of considering task type and timing of restriction protocols.

Aim 2- To investigate the validity of polysomnography compared to surrogate sleep devices and assess the differences between of objective and subjective measurements in the laboratory versus the participants home setting.

Consumer sleep devices may pose as a promising option in the absence of actigraphy or PSG, for obtaining basic sleep variables such as total sleep time, time in bed and sleep efficiency. Yet significant limitations were evident for detecting sleep stages due to the absence of EEG brain activity measurement, which largely restricts the accuracy and ability of these devices to measure such variables. Other challenges remain such as battery life, data

loss and device misuse (Chaudry et al. 2020). Laboratory monitoring demonstrated that a familiarisation night is strongly advised as it facilitates adjustment to the environment, which is reflected in the data with greater sleep quality from night one to night two. Surprisingly participants also slept better in the laboratory compared to the home setting with longer sleep durations, fewer awakenings and a higher sleep efficiency.

Aim 3- i) To determine the physiological and psychological effects of partial sleep restriction (4 h sleep per night, over two consecutive nights) and investigate the effectiveness of a 30 versus 60-minute nap at 13:00 h.

Acute sleep restriction, impaired cognitive function more than maximal or submaximal performance in healthy individuals. Providing a nap opportunity at 13:00 h enhanced cognitive outcomes with both 30- and 60-min naps (PSR₃₀ and PSR₆₀) showing benefits. These findings suggest that naps allows temporary relief from excessive tiredness and allows the accumulation of homeostatic pressure to dissipate. However, the exact mechanisms of how a nap opportunity translates to enhanced functioning remain unclear (see Chapter 6). These findings hold important questions regarding the optimal nap duration.

Aim 4- To investigate the dose response between 3 versus 4 h of sleep restriction across two consecutive nights on measures of physical performance, cognitive function and perceptual responses.

No significant differences were observed between 3 versus 4 h of sleep restriction on measures of exercise performance. This is likely due to similar total sleep times being achieved by the two groups, as confirmed by actigraphy. However differences between groups were observed for cognitive function with slightly more errors for the Stroop task following 4 h sleep per night opposed to 3 h. This reaffirms that cognitive function remains highly sensitive to acute bouts of sleep restriction. Nap interventions did not improve perceptual responses or performance outcomes compared to no nap. Emphasising the need for future studies to obtain objective sleep measurements during naps and exploration of individual preferences such as habitual versus non habitual nappers.

Aim 5- To determine the mechanisms of action of a nap regarding temperature regulation and sleep architecture following two nights of 3 h per night sleep restriction. A further aim was to investigate the effectiveness of 30 versus 60 min nap on psychological measures and submaximal performance at 17:00 h.

Having observed mixed findings from the prior experimental studies, further insight into the physiological processes of a nap was warranted. Objective measures of sleep confirm that the longer nap duration (60 min) provided a greater opportunity for REM and slow wave sleep to occur. However, the occurrence of these ‘restorative’ sleep stages did not translate to enhanced performance outcomes compared to shorter naps or no nap nap (N_{30} and N_{60} , respectively). These findings suggest that allowing an individual to rest in an environment free of distractions, such as in the no nap condition, provided similar benefits to having a nap.

8.2 Practical applications and recommendations

This area of research warrants further investigation to provide more clarity on the effects of sleep restriction on different exercise types and across more diverse populations, such as those of different ages, biological sex and genetics. Despite this there are several insights from the experimental investigations that will further contribute to this field of research and aid future recommendations. The proposed guidelines support future studies to conduct rigorous research designs that will allow for comparable findings between studies and more transparency in study methodologies.

Napping Interventions:

- Recovery is essential for athletes, especially following sleep loss. A 60-minute nap may provide more opportunity to enter restorative sleep stages that will favour athletes if they have the time available in their schedule.
- When embedding a nap into an individual’s schedule, coaches should ensure there is sufficient time post nap to recover from potential sleep inertia, which can impair functioning for up to 60 minutes following a nap of > 30 minutes, dependent on sleep architecture.
- The findings from this thesis do not allow for confirmation of the ‘optimal’ nap protocol. However, when longer naps are not feasible, short naps (< 30 min) or the opportunity to rest in an environment without disturbances may provide relief from the effects of sleep restriction.

Sleep and Performance Considerations:

- Sleep need and responses to sleep loss vary greatly between individuals due to variations in trait-like differences, genetics, biological sex, age and lifestyle factors. More in depth pre-screening is required to

provide extensive insight and improve the study design. Reporting participant characteristics also enables more accurate comparisons between study populations.

- Methodological considerations are important when planning a research study, especially factors such as caffeine intake, fitness level, timing of restriction protocol (as detailed in Table 2.3). By controlling for such factors, it may reduce the applicability to real life situations in some scenarios, yet failing to measure or consider them will likely impact study outcomes.
- Understanding whether a dose response exists is critical to identify the minimum sleep threshold required to maintain performance (i.e. critical threshold of < 3 h sleep per night).

Strength and Cognitive Tasks:

- Tasks requiring high attention and fast reaction time (such as combat and racket sports) are more vulnerable to sleep restriction. A 60 min post lunch nap can mitigate the effects of sleep loss, by reducing excessive fatigue and potentially enhancing function in such tasks. However, sports that require short time on task with minimal bouts of sustained attention may not seek any greater benefit from having a nap.
- The Stroop test is a tool widely used in sleep studies for assessing cognitive function, particularly processing speeds and cognitive flexibility. The task is easy to employ and is effective for assessing the impact of sleep restriction on functioning, with participants exhibiting greater errors and slower response speeds. Studies should aim to employ cognitive tasks that relate directly to the chosen sport.

Sleep Measurement:

- Consumer sleep devices are not yet appropriate for research and clinical application. To achieve values similar to that of polysomnography different approaches need to be taken that encompass measurement of brain activity such as EEG signals.
- Combining objective and subjective measures of sleep are essential to capturing the true quality and perception of an individual's sleep. There are often differences between these values hence neither method should be employed alone if possible.
- Actigraphy and wearable consumer devices allow measurement over longitudinal periods and may be able to determine basic measures of sleep such as total sleep time, wake bouts and sleep efficiency. Despite this, it is important that athletes do not rely on information from consumer sleep devices, as the

majority have not been validated in athletes. The outcomes may therefore cause unnecessary stress and intensify poor perception of sleep.

8.3 Limitations of the thesis:

In the final study (Chapter 7) the criteria were tailored to recruit female participants however none showed interest to participate. These recruitment challenges restricted the generalisability to male athletes only.

Throughout Chapters 5, 6 and 7 only delayed bedtime restriction protocols were employed. The reason being that delayed bedtime is common for many sports where training or competition is scheduled in the evening. However, this does not accommodate for sports that require early awakening, where athletes are required to rise early for training sessions or competitions, for example in swimming or triathlon. The effects following early awakening have found to be more detrimental on performance therefore investigating both restriction types would have allowed for further exploration.

The study population consisted of healthy, trained individuals opposed to elite level athletes. Due to the nature and commitment of the protocols it was difficult to recruit higher calibre athletes. The findings may not directly relate to national and international level athletes as their characteristics greatly differ to those who are trained and/or recreationally active. The population was also relatively narrow regarding age range and biological sex, with all participants being males aged between 20-30 years old.

Habitual sleep conditions were not included throughout the studies in this thesis predominately due to time constraints, with the study protocols taking around 8-9 weeks from start to completion for each participant. This limits the ability to compare between restriction and normal sleep schedules; however, it is commonly acknowledged that athletes rarely obtain 'normal' sleep.

8.4 Overall recommendations for future research:

- Develop sleep optimisation programmes that are tailored to individual athletes to suit their preferences and the specific characteristics of their sport. These programmes would provide coaches and support staff with useful guidelines to aid their athlete in achieving optimal recovery that is adapted to their schedule.

- Adopt standardised frameworks when developing study designs for the pre-screening process as discussed in Chapter 2, mixed methods review and accommodate for key considerations such as those discussed in Table 2.3.
- Investigate the effects of sleep restriction in female athletes as there are established sex differences in sleep physiology, particularly across certain stages of the menstrual cycle. The current lack of studies limits our understanding of how females respond to sleep loss and whether these responses differ from males, highlighting the need for investigation to guide evidence-based decisions.
- Explore more strength-based performance tasks, as much of the existing literature focuses on similar lifts such as bench press, back squat or leg press. Other compound lifts commonly performed in competition, such as the ‘snatch’ and ‘clean and jerk’ are complex movements with many technical elements. Attentional focus significantly influences the execution of these lifts, suggesting certain movements may be more significantly affected by sleep loss.
- Nap interventions should be investigated further, specifically using objective measures of sleep to develop a greater understanding of sleep architecture of daytime naps, following sleep loss. This would further build on the findings from Chapter 6 and the limited studies to date that have employed polysomnography during nap periods.
- Continue to conduct more validation studies comparing consumer sleep devices against actigraphy and polysomnography to inform use of these devices in research and the applied setting, as the demand for these devices continues to grow.

Chapter 9:

References

References

- Abdelmalek S, Chtourou H, Aloui A, Aouichaoui C, Souissi N and Tabka Z (2013). Effect of time of day and partial sleep deprivation on plasma concentrations of IL-6 during a short-term maximal performance. *European Journal of Applied Physiology*, 113, pp.241-248.
- Ajjimaporn A, Ramyarangsi P and Siripornpanich V (2020). Effects of a 20-min nap after sleep deprivation on brain activity and soccer performance. *International Journal of Sports Medicine*, 41(14), pp.1009-1016.
- Alhola P and Polo-Kantola P (2007). Sleep deprivation: Impact on cognitive performance. *Neuropsychiatric Disease and Treatment*, 3(5), pp.553-567.
- Altun İ, Cınar N and Dede C (2012). The contributing factors to poor sleep experiences in according to the university students: A cross-sectional study. *Journal of Research in Medical Sciences: The Official Journal of Isfahan University of Medical Sciences*, 17(6), pp.557.
- Ameen MS, Cheung LM, Hauser T, Hahn MA and Schabus M (2019). About the accuracy and problems of consumer devices in the assessment of sleep. *Sensors*, 19(19), pp.4160.
- Ammar A, Riemann BL, Masmoudi L, Blaumann M, Abdelkarim O and Hökelmann A (2018). Kinetic and kinematic patterns during high intensity clean movement: searching for optimal load. *Journal of Sport Sciences*, 36(12), pp.1319-1330.
- Arnal PJ, Thorey V, Debellemanni E, Ballard ME, Bou Hernandez A, Guillot A, Jourde H, Harris M, Guillard M, Van Beers P and Chennaoui M (2020). The Dreem Headband compared to polysomnography for electroencephalographic signal acquisition and sleep staging. *Sleep*, 43(11), pp.zsaa097.
- Axelsson J, Kecklund G, Åkerstedt T, Donofrio P, Lekander M and Ingre M (2008). Sleepiness and performance in response to repeated sleep restriction and subsequent recovery during semi-laboratory conditions. *Chronobiology International*, 25(2-3), pp.297-308.
- Axelsson J, Ingre M, Kecklund G, Lekander M, Wright Jr KP and Sundelin T (2020). Sleepiness as motivation: a potential mechanism for how sleep deprivation affects behaviour. *Sleep*, 43(6), pp.zsz291.
- Bailey MP, Derks G and Skeldon AC (2018). Circle maps with gaps: Understanding the dynamics of the two-process model for sleep–wake regulation. *European Journal of Applied Mathematics*, 29(5), pp.845-868.
- Baati H, Chtourou H, Moalla W, Jarraya M, Nikolaidis PT, Rosemann T and Knechtle B (2020). Effect of angle of view and partial sleep deprivation on distance perception. *Frontiers in Psychology*, 11, pp.201.
- Bambaeichi E, Reilly T, Cable NT and Giacomoni M (2005). The influence of time of day and partial sleep loss on muscle strength in eumenorrheic females. *Ergonomics*, 48(11-14), pp.1499-1511.
- Banks S and Dinges DF (2007). Behavioural and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine*, 3(5), pp.519-528.
- Banks S, Van Dongen HP, Maislin G and Dinges DF (2010). Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. *Sleep*, 33(8), pp.1013-1026.
- Baroni A, Bruzzese JM, Di Bartolo CA and Shatkin JP (2016). Fitbit Flex: an unreliable device for longitudinal sleep measures in a non-clinical population. *Sleep and Breathing*, 20, pp.853-854.
- Belenky G, Wesensten NJ, Thorne DR, Thomas ML, Sing HC, Redmond DP, Russo MB and Balkin TJ (2003). Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: A sleep dose-response study. *Journal of Sleep Research*, 12(1), pp.1-12.

Berryhill S, Morton CJ, Dean A, Berryhill A, Provencio-Dean N, Patel SI, Estep L, Combs D, Mashaqi S, Gerald LB and Krishnan JA (2020). Effect of wearables on sleep in healthy individuals: a randomized crossover trial and validation study. *Journal of Clinical Sleep Medicine*, 16(5), pp.775-783.

Bes F, Jobert M and Schulz H (2009). Modeling napping, post-lunch dip, and other variations in human sleep propensity. *Sleep*, 32(3), pp.392-398.

Besedovsky L, Lange T and Born J (2012). Sleep and immune function. *Pflügers Archiv-European Journal of Physiology*, 463(1), pp.121-137.

Bianchi MT (2018). Sleep devices: wearables and nearables, informational and interventional, consumer and clinical. *Metabolism*, 84, pp.99-108.

Birk, T.J. and Birk, C.A., 1987. Use of ratings of perceived exertion for exercise prescription. *Sports Medicine*, 4(1), pp.1-8.

Blumert PA, Crum AJ, Ernsting M, Volek JS, Hollander DB, Haff EE and Haff GG (2007). The acute effects of twenty-four hours of sleep loss on the performance of national-caliber male weightlifters. *Journal of Strength and Conditioning Research*, 21(4), pp.1146–1154.

Borbély AA (1982). A two-process model of sleep regulation. *Human Neurobiology*, 1(3), pp.195-204.

Borbély AA and Achermann P (1992). Concepts and models of sleep regulation: an overview. *Journal of Sleep Research*, 1(2), pp.63-79.

Botonis PG, Koutouvakis N and Toubekis AG (2021). The impact of daytime napping on athletic performance—a narrative review. *Scandinavian Journal of Medicine & Science in Sports*, 31(12), pp.2164-2177.

Boukhris O, Abdessalem R, Ammar A, Hsouna H, Trabelsi K, Engel FA, Sperlich B, Hill DW and Chtourou H (2019). Nap opportunity during the daytime affects performance and perceived exertion in 5-m shuttle run test. *Frontiers in Physiology*, 10, pp.779.

Boukhris O, Trabelsi K, Ammar A, Abdessalem R, Hsouna H, Glenn JM, Bott N, Driss T, Souissi N, Hammouda O and Garbarino S (2020). A 90 min daytime nap opportunity is better than 40 min for cognitive and physical performance. *International Journal of Environmental Research and Public Health*, 17(13), pp.4650.

Brooks A and Lack L (2006). A brief afternoon nap following nocturnal sleep restriction: which nap duration is most recuperative? *Sleep*, 29(6), pp.831-840.

Brotherton EJ, Moseley SE, Langan-Evans C, Pullinger SA, Robertson CM, Burniston JG and Edwards BJ (2019). Effects of two nights partial sleep deprivation on an evening submaximal weightlifting performance; are 1 h power naps useful on the day of competition? *Chronobiology International*, 36(3), pp.407-426.

Bruyneel M, Sanida C, Art G, Libert W, Cuvelier L, Paesmans M, Sergysels R and Ninane V (2011). Sleep efficiency during sleep studies: results of a prospective study comparing home-based and in-hospital polysomnography. *Journal of Sleep Research*, 20(1pt2), pp.201-206.

Buysse DJ, Reynolds III CF, Monk TH, Berman SR and Kupfer DJ (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), pp.193-213.

Carrier J and Monk TH (2000). Circadian rhythms of performance: new trends. *Chronobiology International*, 17(6), pp.719-732.

Carskadon MA and Roth T (1991). Sleep restriction.

Carskadon MA and Dement WC (2005). Normal human sleep: an overview. *Principles and Practice of Sleep Medicine*, 4(1), pp.13-23.

Cauldron Science (2016) *Gorilla Experiment Builder*. Available at: <https://gorilla.sc>

Chae KY, Kripke DF, Poceta JS, Shadan F, Jamil SM, Cronin JW and Kline LE (2009). Evaluation of immobility time for sleep latency in actigraphy. *Sleep Medicine*, 10(6), pp.621-625.

Chase JD, Roberson PA, Saunders MJ, Hargens TA, Womack CJ and Luden ND (2017). One night of sleep restriction following heavy exercise impairs 3-km cycling time-trial performance in the morning. *Applied Physiology, Nutrition, and Metabolism*, 42(9), pp.909-915.

Chattu VK, Manzar MD, Kumary S, Burman D, Spence DW and Pandi-Perumal SR (2018). The global problem of insufficient sleep and its serious public health implications. In *Healthcare*, 7(1), pp. 1.

Chaudhry FF, Danieleto M, Golden E, Scelza J, Botwin G, Shervey M, De Freitas JK, Paranjpe I, Nadkarni GN, Miotto R and Glow P (2020). Sleep in the natural environment: a pilot study. *Sensors*, 20(5), pp.1378.

Chinoy ED, Cuellar JA, Huwa KE, Jameson JT, Watson CH, Bessman SC, Hirsch DA, Cooper AD, Drummond SP and Markwald RR (2021). Performance of seven consumer sleep-tracking devices compared with polysomnography. *Sleep*, 44(5), pp.zsaa291.

Chrisholm DM, Collis ML, Kulak LL, Davenport W and Gruber N (1975). Physical activity readiness. *British Columbia Medical Journal*, 17, pp. 375-378.

Cohen J (1988). *Statistical Power Analysis for the Behavioural Sciences*. 2nd ed. Lawrence Erlbaum Associates.

Cook C, Beaven CM, Kilduff LP and Drawer S (2012). Acute caffeine ingestion's increase of voluntarily chosen resistance-training load after limited sleep. *International Journal of Sport Nutrition and Exercise Metabolism*, 22(3), pp.157-164.

Cote KA, Milner CE, Smith BA, Aubin AJ, Greason TA, Cuthbert BP, Wiebe S and Duffus SE (2009). CNS arousal and neurobehavioral performance in a short-term sleep restriction paradigm. *Journal of Sleep Research*, 18(3), pp.291-303.

Craven J, McCartney D, Desbrow B, Sabapathy S, Bellinger P, Roberts L and Irwin C (2022). Effects of acute sleep loss on physical performance: a systematic and meta-analytical review. *Sports Medicine*, 52(11), pp.2669-2690.

Crivello A, Barsocchi P, Girolami M and Palumbo F (2019). The meaning of sleep quality: a survey of available technologies. *IEEE access*, 7, pp.167374-167390.

Cullen T, Thomas G, Wadley AJ and Myers T (2019). The effects of a single night of complete and partial sleep deprivation on physical and cognitive performance: A Bayesian analysis. *Journal of Sports Sciences*, 37(23), pp.2726-2734.

Cunha LA, Costa JA, Marques EA, Brito J, Lastella M and Figueiredo P (2023). The impact of sleep interventions on athletic performance: a systematic review. *Sports Medicine-Open*, 9(1), pp.58.

Cunningham JE, Jones SA, Eskes GA and Rusak B (2018). Acute sleep restriction has differential effects on components of attention. *Frontiers in Psychiatry*, 9, pp.499.

Daaloul H, Souissi N and Davenne D (2019). Effects of napping on alertness, cognitive, and physical outcomes of karate athletes. *Medicine and Science in Sports and Exercise*, 51(2), pp.338-345.

- Davenne D (2009). Sleep of athletes—problems and possible solutions. *Biological Rhythm Research*, 40(1), pp.45-52.
- Dean B, Hartmann T, Wingfield G, Larsen P and Skein M (2023). Sleep restriction between consecutive days of exercise impairs sprint and endurance cycling performance. *Journal of Sleep Research*, 32(5), pp.e13857.
- De Zambotti M, Claudatos S, Inkelis S, Colrain IM and Baker FC (2015). Evaluation of a consumer fitness-tracking device to assess sleep in adults. *Chronobiology International*, 32(7), pp.1024-1028.
- De Zambotti M, Cellini N, Goldstone A, Colrain IM and Baker FC (2019). Wearable sleep technology in clinical and research settings. *Medicine and Science in Sports and Exercise*, 51(7), pp.1538.
- De Zambotti M, Rosas L, Colrain IM and Baker FC (2019). The sleep of the ring: comparison of the ŌURA sleep tracker against polysomnography. *Behavioral Sleep Medicine*, 17(2), pp.124-136
- De Zambotti M, Goldstein C, Cook J, Menghini L, Altini M, Cheng P and Robillard R (2024). State of the science and recommendations for using wearable technology in sleep and circadian research. *Sleep*, 47(4), pp.zsad325.
- Doherty R, Madigan SM, Nevill A, Warrington G and Ellis JG (2021). The sleep and recovery practices of athletes. *Nutrients*, 13(4), pp.1330.
- Drummond SP, Brown GG, Gillin JC, Stricker JL, Wong EC and Buxton RB (2000). Altered brain response to verbal learning following sleep deprivation. *Nature*, 403(6770), pp.655-657.
- Dutheil F, Chambres P, Honde N, Pereira B and Moustafa F (2021). Napping and cognitive performance: A systematic review. *Journal of Clinical Sleep Medicine*, 17(5), pp. 1251–1265.
- Edwards BJ and Waterhouse J (2013). Athletic Performance; Effects of Sleep Loss. In: Kushida C. (ed.) *The Encyclopaedia of Sleep*, 1, pp. 320-326.
- Edwards BJ, Gallagher C, Pullinger SA, de Mello MT and Walsh NP (2021). Athletic performance; Effects of sleep loss, Reference Module in Neuroscience and Biobehavioural Psychology, Elsevier.
- Edwards BJ, Adam RL, Gallagher C, Germaine M, Hulton A.T, Pullinger SA and Chester NJ (2024). In Males with Adequate Dietary Needs Who Present No Sleep Disturbances, Is an Acute Intake of Zinc Magnesium Aspartate, Following Either Two Consecutive Nights of 8 or 4 h of Sleep Deprivation, Beneficial for Sleep and Morning Stroop Interference Performance?. *Behavioural Sciences*, 14(7), pp.622.
- Edwards BJ, Boyle BB, Burniston JG, Doran DA, Doran D, Giacomoni M, Mahon E and Pullinger SA (2025). Evidence of a circadian variation in 10-km laboratory running time-trial performance, where a standardised approach has been employed. *Chronobiology International*, pp.1-15.
- Elmenhorst EM, Elmenhorst D, Luks N, Maass H, Vejvoda M and Samel A (2008). Partial sleep deprivation: impact on the architecture and quality of sleep. *Sleep Medicine*, 9(8), pp.840-850.
- Evenson KR, Goto MM and Furberg RD (2015). Systematic review of the validity and reliability of consumer-wearable activity trackers. *International Journal of Behavioural Nutrition and Physical Activity*, 12, pp.1-22.
- Faraut B, Léger D, Medkour T, Dubois A, Bayon V, Chennaoui M and Perrot S (2015). Napping reverses increased pain sensitivity due to sleep restriction. *PloS one*, 10(2), pp.e0117425.
- Faraut B, Nakib S, Drogou C, Elbaz M, Sauvet F, De Bandt JP and Léger D (2015). Napping reverses the salivary interleukin-6 and urinary norepinephrine changes induced by sleep restriction. *The Journal of Clinical Endocrinology & Metabolism*, 100(3), pp.E416-E426.
- Ficca G, Axelsson J, Mollicone DJ, Muto V and Vitiello MV (2010). Naps, cognition and performance. *Sleep Medicine Reviews*, 14(4), pp.249-258.

- Fullagar HH, Skorski S, Duffield R, Hammes D, Coutts AJ and Meyer T (2015). Sleep and athletic performance: the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise. *Sports Medicine*, 45(2), pp.161-186.
- Fullagar HH and Bartlett JD (2016). Time to wake up: individualising the approach to sleep promotion interventions. *British Journal of Sports Medicine*, 50(3), pp.143-144.
- Fushimi A and Hayashi M (2008). Pattern of slow-wave sleep in afternoon naps. *Sleep and Biological Rhythms*, 6(3), pp.187-189.
- Gallagher C, Green CE, Kenny ML, Evans JR, McCullagh GD, Pullinger SA and Edwards BJ (2023). Is implementing a post-lunch nap beneficial on evening performance, following two nights partial sleep restriction? *Chronobiology International*, 40(9), pp.1169-1186.
- Garbarino S, Lanteri P, Bragazzi NL, Magnavita N and Scoditti E (2021). Role of sleep deprivation in immune-related disease risk and outcomes. *Communications Biology*, 4(1), pp.1304.
- George AA, Bansal R, Majhi D and Narula A (2024). A Study on Sleep Quality and Mental Fatigue among College Students. *International Journal of Interdisciplinary Approaches in Psychology*, 2(12), pp.220-230.
- Gilbert SS, van den Heuvel CJ, Ferguson SA and Dawson D (2004). Thermoregulation as a sleep signalling system. *Sleep Medicine Reviews*, 8(2), pp.81-93.
- Goh QY, Lin H and Teo C (2001). Sleep deprivation and physical performance: Effects on maximal strength and endurance. *Sleep Research*, 12, pp. 42-50.
- Goldstein C (2020). Current and Future Roles of Consumer Sleep Technologies in Sleep Medicine. *Telehealth in Sleep Medicine An Issue of Sleep Medicine Clinics, E-Book: Telehealth in Sleep Medicine An Issue of Sleep Medicine Clinics, E-Book*, 15(3), pp.391-408.
- Gradisar M and Lack L (2004). Relationships between the circadian rhythms of finger temperature, core temperature, sleep latency, and subjective sleepiness. *Journal of Biological Rhythms*, 19(2), pp.157-163.
- Groeger JA, Lo JC, Santhi N, Lazar AS and Dijk DJ (2022). Contrasting effects of sleep restriction, total sleep deprivation, and sleep timing on positive and negative affect. *Frontiers in Behavioural Neuroscience*, 16, pp.911994.
- Gupta L, Morgan K and Gilchrist S (2017). Does elite sport degrade sleep quality? A systematic review. *Sports Medicine*, 47, pp.1317-1333.
- Hachana Y, Attia A, Chaabène H, Gallas S, Sassi RH and Dotan R (2012). Test-retest reliability and circadian performance variability of a 15-s Wingate Anaerobic Test. *Biological Rhythm Research*, 43(4), pp.413-421.
- HajSalem M, Chtourou H, Aloui A, Hammouda O and Souissi N (2013). Effects of partial sleep deprivation at the end of the night on anaerobic performances in judokas. *Biological Rhythm Research*, 44(5), pp.815-821.
- Halsen SL (2019). Sleep monitoring in athletes: motivation, methods, miscalculations and why it matters. *Sports Medicine*, 49(10), pp.1487-1497.
- Hatia M, Loureiro N, Ribeiro J, Moeda F, Melo M, Tocha J, Schonenberger A, Correia C and Braz AS (2024). A Narrative Review of the Impact of Sleep on Athletes: Sleep Restriction Causes and Consequences, Monitoring, and Interventions. *Cureus*, 16(12).
- Herberger S, Aurnhammer C, Bauerfeind S, Bothe T, Penzel T and Fietze I (2025). Performance of wearable finger ring trackers for diagnostic sleep measurement in the clinical context. *Scientific Reports*, 15(1), pp.9461.

- Hilditch CJ, Dorrian J and Banks S (2017). A review of short naps and sleep inertia: do naps of 30 min or less really avoid sleep inertia and slow-wave sleep? *Sleep Medicine*, 32, pp.176-190.
- Hilditch CJ and McHill AW (2019). Sleep inertia: current insights. *Nature and Science of Sleep*, pp.155-165.
- Horne J (1988). *Why we sleep: the functions of sleep in humans and other mammals*. Oxford University Press.
- Hsouna H, Boukhris O, Abdessalem R, Trabelsi K, Ammar A, Shephard RJ and Chtourou H (2019). Effect of different nap opportunity durations on short-term maximal performance, attention, feelings, muscle soreness, fatigue, stress and sleep. *Physiology & Behaviour*, 211, pp.112673.
- Hutchinson JC and Tenenbaum G (2019). Perceived effort and exertion.
- Iber C, Redline S, Gilpin AMK, Quan SF, Zhang L, Gottlieb DJ, Rapoport D, Resnick HE, Sanders M and Smith P (2004). Polysomnography performed in the unattended home versus the attended laboratory setting—Sleep Heart Health Study methodology. *Sleep*, 27(3), pp.536-540.
- Jarraya M, Jarraya S, Chtourou H, Souissi N and Chamari K (2013). The impact of partial sleep deprivation on the diurnal variations of cognitive performance in trained subjects. *Procedia-Social and Behavioural Sciences*, 82, pp.392-396.
- Juliano LM, Huntley ED, Harrell PT and Westerman AT (2012). Development of the caffeine withdrawal symptom questionnaire: caffeine withdrawal symptoms cluster into 7 factors. *Drug and Alcohol Dependence*, 124(3), pp.229-234.
- Johns MW, Thornton C and Doré C (1976). Heart rate and sleep latency in young men. *Journal of Psychosomatic Research*.
- Johns MW (1991). A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*, 14(6), pp. 540-545.
- Juliff LE, Halson SL and Peiffer JJ (2015). Understanding sleep disturbance in athletes prior to important competitions. *Journal of Science and Medicine in Sport*, 18(1), pp.13-18.
- Kelly JM, Strecker RE and Bianchi MT (2012). Recent developments in home sleep-monitoring devices. *International Scholarly Research Notices*, (1), pp.768794.
- Keyes KM, Maslowsky J, Hamilton A and Schulenberg J (2015). The great sleep recession: changes in sleep duration among US adolescents, 1991–2012. *Pediatrics*, 135(3), pp.460-468.
- Khemila S, Abdelmalek S, Romdhani M, Souissi A, Chtourou H and Souissi N (2021). Listening to motivational music during warming-up attenuates the negative effects of partial sleep deprivation on cognitive and short-term maximal performance: effect of time of day. *Chronobiology International*, 38(7), pp.1052-1063.
- Killgore WD (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research*, 185, pp.105-129.
- Kline CE (2020). Sleep quality. In *Encyclopedia of Behavioural Medicine*, pp. 2064-2066.
- Kräuchi K (2001). The thermoregulation of sleep onset. *Chronobiology International*, 18(2), pp. 181–198.
- Kräuchi K, Knoblauch V, Wirz-Justice A and Cajochen C (2006). Challenging the sleep homeostat does not influence the thermoregulatory system in men: evidence from a nap vs. sleep-deprivation study. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 290(4), pp.R1052-R1061.
- Krystal AD and Edinger JD (2008). Measuring sleep quality. *Sleep Medicine*, 9, pp.S10-S17.
- Ko PRT, Kientz JA, Choe EK, Kay M, Landis CA and Watson NF (2015). Consumer sleep technologies: a review of the landscape. *Journal of Clinical Sleep Medicine*, 11(12), pp.1455-1461.

- Kujawa K, Olpińska-Lischka M and Maciaszek J (2020). The influence of 24-hour sleep deprivation on the strength of lower limb muscles in young and physically fit women and men. *Sustainability*, 12(7), pp.2762.
- Landolt HP (2008). Sleep homeostasis: a role for adenosine in humans? *Biochemical Pharmacology*, 75(11), pp.2070-2079.
- Lastella M, Roach GD, Halson SL and Sargent C (2015). Sleep/wake behaviours of elite athletes from individual and team sports. *European Journal of Sport Science*, 15(2), pp.94-100.
- Lastella M, Halson SL, Vitale JA, Memon AR and Vincent GE (2021). To nap or not to nap? A systematic review evaluating napping behavior in athletes and the impact on various measures of athletic performance. *Nature and Science of Sleep*, pp.841-862.
- Le Bon O (2020). Relationships between REM and NREM in the NREM-REM sleep cycle: a review on competing concepts. *Sleep Medicine*, 70, pp.6-16.
- Lee JM, Byun W, Keill A, Dinkel D and Seo Y (2018). Comparison of wearable trackers' ability to estimate sleep. *International Journal of Environmental Research and Public Health*, 15(6), pp.1265.
- Leong RL and Chee MW (2023). Understanding the need for sleep to improve cognition. *Annual Review of Psychology*, 74(1), pp.27-57.
- Lim SE, Kim HS, Lee SW, Bae KH and Baek YH (2023). Validation of Fitbit Inspire 2™ Against Polysomnography in Adults Considering Adaptation for Use. *Nature and Science of Sleep*, pp. 59-67.
- Loddo G, La Fauci G, Vignatelli L, Zenesini C, Cilea R, Mignani F, Cecere A, Mondini S, Baldelli L, Bisulli F, Licchetta L, Mostacci B, Guaraldi P, Giannini G, Tinuper P and Provini F (2021). The Arousal Disorders Questionnaire: a new and effective screening tool for confusional arousals, sleepwalking and sleep terrors in epilepsy and sleep disorders units. *Sleep Medicine*, 80, pp. 279-285.
- Lou S, Hu S, Chen Y, Zhao K, Zhou Y, Hou J, Liang L and Sun H (2024). Benefits of napping habits in healthy adults: Maintaining alerting performance and cortisol levels change within 90 min of habitual napping time. *Sleep Medicine*, 119, pp.214-221.
- Maloney S, Houston A and Forbes P (2023). The impact of napping on athletic performance: A meta-analysis. *Journal of Sports Sciences*, 41(2), pp.189–202.
- Mantua J, Gravel N and Spencer RM (2016). Reliability of sleep measures from four personal health monitoring devices compared to research-based actigraphy and polysomnography. *Sensors*, 16(5), pp.646.
- Mantua J and Spencer RM (2017). Exploring the nap paradox: are mid-day sleep bouts a friend or foe? *Sleep Medicine*, 37, pp.88-97.
- Marcoen N, Vandekerckhove M, Neu D, Pattyn N and Mairesse O (2015). Individual differences in subjective circadian flexibility. *Chronobiology International*, 32(9), pp.1246-1253.
- Marino M, Li Y, Rueschman MN, Winkelman JW, Ellenbogen JM, Solet JM, Dulin H, Berkman LF and Buxton OM (2013). Measuring sleep: accuracy, sensitivity, and specificity of wrist actigraphy compared to polysomnography. *Sleep*, 36(11), pp.1747-1755.
- McCarthy ME and Waters WF (1997). Decreased attentional responsivity during sleep deprivation: orienting response latency, amplitude, and habituation. *Sleep*, 20(2), pp.115-123.
- McKay AK, Stellingwerff T, Smith ES, Martin DT, Mujika I, Goosey-Tolfrey VL, Sheppard J and Burke LM (2021). Defining training and performance caliber: a participant classification framework. *International Journal of Sports Physiology and Performance*, 17(2), pp.317-331.

- McNeil J, Forest G, Hintze L and Lafrance G (2017). The effects of sleep restriction on cognitive and physical performance. *Journal of Sleep Research*, 26(5), pp.622–629.
- Mejri MA, Hammouda O, Zouaoui K, Chaouachi A, Chamari K, Rayana MCB and Souissi N (2014). Effect of two types of partial sleep deprivation on Taekwondo players' performance during intermittent exercise. *Biological Rhythm Research*, 45(1), pp.17-26.
- Mejri MA, Yousfi N, Mhenni T, Tayech A, Hammouda O, Driss T, Chaouachi A and Souissi N (2016). Does one night of partial sleep deprivation affect the evening performance during intermittent exercise in Taekwondo players? *Journal of Exercise Rehabilitation*, 12(1), pp.47.
- Mendonça F, Mostafa SS, Morgado-Dias F, Ravelo-Garcia AG and Penzel T (2019). A review of approaches for sleep quality analysis. *Ieee Access*, 7, pp.24527-24546.
- Meney C, Waterhouse J, Atkinson G and Reilly T (1998). The effect of sleep deprivation on physical performance: A meta-analysis. *Journal of Human Kinetics*, 12(3), pp.273–288.
- Mesas AE, de Arenas-Arroyo SN, Martinez-Vizcaino V, Garrido-Miguel M, Fernández-Rodríguez R, Bizzozero-Peroni B and Torres-Costoso AI (2023). Is daytime napping an effective strategy to improve sport-related cognitive and physical performance and reduce fatigue? A systematic review and meta-analysis of randomised controlled trials. *British Journal of Sports Medicine*, 57(7), pp.417-426.
- Milner CE and Cote KA (2009). Benefits of napping in healthy adults: impact of nap length, time of day, age, and experience with napping. *Journal of Sleep Research*, 18(2), pp.272-281.
- Mitchell M and Werkhaven JA (2020). Cost-effectiveness of polysomnography in the management of pediatric obstructive sleep apnea. *International Journal of Pediatric Otorhinolaryngology*, 133, pp.109943.
- Monk TH and Leng VC (1982). Time of day effects in simple repetitive tasks: some possible mechanisms. *Acta Psychologica*, 51(3), pp.207-221.
- Monk TH (2012). Sleep and human performance. *The Oxford Handbook of Sleep and Sleep Disorders*, pp.95-109.
- Montgomery-Downs H, Insana S and Bond J (2012). Movement toward a novel activity monitoring device. *Sleep Breath*, 16, pp. 913–917.
- Mougin F, Bourdin H, Simon-Rigaud ML, Didier JM, Toubin G and Kantelip JP (1996). Effects of a selective sleep deprivation on subsequent anaerobic performance. *International Journal of Sports Medicine*, 17(02), pp.115-119.
- Mougin F, Bourdin H, Simon-Rigaud ML, Nhu UN, Kantelip JP and Davenne D (2001). Hormonal responses to exercise after partial sleep deprivation and after a hypnotic drug-induced sleep. *Journal of Sports Sciences*, 19(2), pp.89-97.
- Mukherjee S, Patel SR, Kales SN, Ayas NT, Strohl KP, Gozal D and Malhotra A (2015). An official American Thoracic Society statement: the importance of healthy sleep. Recommendations and future priorities. *American Journal of Respiratory and Critical Care Medicine*, 191(12), pp.1450-1458.
- Munnilari M, Bommasamudram T, Easow J, Tod D, Varamenti E, Edwards BJ, Ravindrakumar A, Gallagher C, Pullinger SA (2024). Diurnal variation in variables related to cognitive performance: a systematic review. *Sleep and Breathing*, 28(1), pp. 495-510.
- Myktyyn IJ, Sajkov D, Neill AM and Mc Evoy RD (1999). Portable computerized polysomnography in attended and unattended settings. *Chest*, 115(1), pp.114-122.

- Nedeltcheva AV, Kilkus JM and Imperial J (2009). Sleep curtailment is accompanied by increased intake of calories from snacks. *American Journal of Clinical Nutrition*, 89(5), pp. 126–133.
- Nelson KL, Davis JE and Corbett CF (2022). Sleep quality: An evolutionary concept analysis. In *Nursing Forum*, 57, pp. 144-151.
- Netzer NC, Stoohs RA, Netzer CM, Clark K and Strohl KP (1999). Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Annals of Internal Medicine*, 131(7), pp.485-491.
- Newell J, Mairesse O, Verbanck P and Neu D (2012). Is a one-night stay in the lab really enough to conclude? First-night effect and night-to-night variability in polysomnographic recordings among different clinical population samples. *Psychiatry Research*, 200(2-3), pp.795-801.
- Nicol F (2019). Temperature and sleep. *Energy and Buildings*, 204, pp.109516.
- Omiya K, Akashi YJ, Yoneyama K, Osada N, Tanabe K and Miyake F (2009). Heart-rate response to sympathetic nervous stimulation, exercise, and magnesium concentration in various sleep conditions. *International Journal of Sport Nutrition and Exercise Metabolism*, 19(2), pp.127-135.
- Ong JL, Lau TY, Lee XK, van Rijn E and Chee MW (2020). A daytime nap restores hippocampal function and improves declarative learning. *Sleep*, 43(9), pp.zsaa058
- Owens J, Au R, Carskadon M, Millman R, Wolfson A, Braverman PK, Adelman WP, Breuner CC and Levine, DA (2014). Insufficient sleep in adolescents and young adults: an update on causes and consequences. *Pediatrics*, 134(3), pp.e921-e932.
- Paryab N, Taheri M, H'Mida C, Irandoust K, Mirmoezzi M, Trabelsi K, Ammar A and Chtourou H (2021). Melatonin supplementation improves psychomotor and physical performance in collegiate student-athletes following a sleep deprivation night. *Chronobiology International*, 38(5), pp.753-761.
- Patel M, Mendpara A, Satapathy SK, Thumar M, Patel R and Mohapatra R (2024), December. Sleep Stages Classification Based on Diverse Resting-State Multi-Channel EEG Using Deep Learning. In *2024 IEEE 8th International Conference on Information and Communication Technology (CICT)*, pp. 1-6.
- Patel AK, Reddy V, Shumway KR and Araujo JF (2024). Physiology, sleep stages. In *StatPearls [Internet]*. StatPearls Publishing.
- Portier F, Portmann A, Czernichow P, Vascaut L, Devin E, Benhamou D, Cuvelier A and Muir JF (2000). Evaluation of home versus laboratory polysomnography in the diagnosis of sleep apnea syndrome. *American Journal of Respiratory and Critical Care Medicine*, 162(3), pp.814-818.
- Pullinger SA, Adhikari R, Bawari B, Pérez Armendáriz ML, Manathanath A, Bommasamudram T, Gallagher C, Bampouras TM and Edwards BJ (2025). Sleep quality, behaviour, disorders prevalence and chronotype in elite junior and senior athletes. *Biological Rhythm Research*, pp.1-17.
- Ramanathan NL (1964). A new weighting system for mean surface temperature of the human body. *Journal of Applied Physiology*, 19(3), pp.531-533.
- Ravindra PN and Kutty BM (2012). Functional importance of sleep: An overview. *Indian Journal of Sleep Medicine*, 7(2), pp.42-44.
- Reilly T and Deykin T (1983). Effects of partial sleep loss on subjective states, psychomotor and physical performance tests. *Journal of Human Movement Studies*, 9(4), pp.257-270.
- Reilly T and Piercy M (1994). The effect of partial sleep deprivation on weight-lifting performance. *Ergonomics*, 37(1), pp.107-115.
- Reilly T and Edwards B (2007). Altered sleep–wake cycles and physical performance in athletes. *Physiology & Behaviour*, 90(2-3), pp.274-284.

- Reilly T and Waterhouse J (2009). Sports performance: is there evidence that the body clock plays a role? *European Journal of Applied Physiology*, 106, pp.321–332.
- Rempe MJ, Best J and Terman D (2010). A mathematical model of the sleep/wake cycle. *Journal of Mathematical Biology*, 60(5), pp.615-644.
- Rentz LE, Ulman HK and Galster SM (2021). Deconstructing commercial wearable technology: contributions toward accurate and free-living monitoring of sleep. *Sensors*, 21(15), pp.5071.
- Reynolds AC and Banks S (2010). Total sleep deprivation, chronic sleep restriction and sleep disruption. *Progress in Brain Research*, 185, pp.91-103.
- Roberts SS, Teo WP, Aisbett B and Warmington SA (2019). Extended sleep maintains endurance performance better than normal or restricted sleep. *Med Sci Sports Exercise*, 51(12), pp.2516-2523.
- Robertson CM, Pullinger SA, Robinson WR, Smith ME, Burniston JG, Waterhouse JM. and Edwards BJ (2018). Is the diurnal variation in muscle force output detected/detectable when multi-joint movements are analysed using the musclelab force–velocity encoder? *Chronobiology International*, 35(10), pp.1391-1401.
- Romdhani M, Hammouda O, Chaabouni Y, Mahdouani K, Driss T, Chamari K and Souissi N (2019). Sleep deprivation affects post-lunch dip performances, biomarkers of muscle damage and antioxidant status. *Biology of Sport*, 36(1), pp.55-65.
- Romdhani M, Souissi N, Chaabouni Y, Mahdouani K, Driss T, Chamari K and Hammouda O (2020). Improved physical performance and decreased muscular and oxidative damage with postlunch napping after partial sleep deprivation in athletes. *International Journal of Sports Physiology and Performance*, 15(6), pp.874-883.
- Romdhani M, Souissi N, Moussa-Chamari I, Chaabouni Y, Mahdouani K, Sahnoun Z, Driss T, Chamari K and Hammouda O (2021). Caffeine use or napping to enhance repeated sprint performance after partial sleep deprivation: why not both?. *International Journal of Sports Physiology and Performance*, 16(5), pp.711-718.
- Romyn G, Lastella M, Miller DJ, Versey NG, Roach GD and Sargent C (2018). Daytime naps can be used to supplement night-time sleep in athletes. *Chronobiology International*, 35(6), pp.865-868.
- Ru T, Chen Q, You J and Zhou G (2019). Effects of short midday nap on habitual nappers' alertness, mood and mental performance across cognitive domains. *Journal of Sleep Research*, 28(3), pp.12638.
- Rundo JV and Downey III R (2019). Polysomnography. *Handbook of Clinical Neurology*, 160, pp.381-392.
- Sadeh A (2011). The role and validity of actigraphy in sleep medicine: an update. *Sleep Medicine Reviews*, 15(4), pp.259-267.
- Sagaspe P, Sanchez-Ortuno M, Charles A, Taillard J, Valtat C, Bioulac B and Philip P (2006). Effects of sleep deprivation on Color-Word, Emotional, and Specific Stroop interference and on self-reported anxiety. *Brain and Cognition*, 60(1), pp.76-87.
- Saletin JM, Hilditch CJ, Dement WC and Carskadon MA (2017). Short daytime naps briefly attenuate objectively measured sleepiness under chronic sleep restriction. *Sleep*, 40(9), pp.zsx118.
- Samson DR (2021). Taking the sleep lab to the field: Biometric techniques for quantifying sleep and circadian rhythms in humans. *American Journal of Human Biology*, 33(6), pp.e23541.
- Sargent C, Lastella M, Halson SL and Roach GD (2014). The impact of training schedules on the sleep and fatigue of elite athletes. *Chronobiology International*, 31(10), pp.1160-1168.

- Sargent C, Lastella M, Halson SL and Roach GD (2021). How much sleep does an elite athlete need? *International Journal of Sports Physiology and Performance*, 16(12), pp.1746-1757.
- Scarpina F and Tagini S (2017). The stroop color and word test. *Frontiers in Psychology*, 8, pp.557.
- Scott JPR, McNaughton LR and Polman RCJ (2006). Effects of sleep deprivation on physical performance and cognitive function. *Journal of Sports Sciences*, 24(8), pp.781–789.
- Scott H, Lack L and Lovato N (2020). A systematic review of the accuracy of sleep wearable devices for estimating sleep onset. *Sleep Medicine Reviews*, 49, pp.101227.
- Silva AC, Silva A, Edwards BJ, Tod D, Souza Amaral A, de Alcântara Borba D, Grade I and Túlio de Mello M (2021). Sleep extension in athletes: what we know so far - A systematic review. *Sleep Medicine*. 7, pp.128-135.
- Silvani MI, Werder R and Perret C (2022). The influence of blue light on sleep, performance and wellbeing in young adults: A systematic review. *Frontiers in physiology*, 13, p.943108.
- Simpson NS, Gibbs EL and Matheson GO (2017). Optimizing sleep to maximize performance: implications and recommendations for elite athletes. *Scandinavian Journal of Medicine & Science in Sports*, 27(3), pp.266-274.
- Sinnerton S and Reilly T (1992). Biomechanics and Medicine in Swimming: *Swimming Science VI*, pp.399.
- Smith CS, Reilly C and Midkiff K (1989). 'Evaluation of three circadian rhythm questionnaires with suggestions for an improved measure of morningness', *Journal of Applied Psychology*. 74(5), pp. 728-738.
- Smithies TD, Toth AJ, Dunican IC, Caldwell JA, Kowal M and Campbell MJ (2021). The effect of sleep restriction on cognitive performance in elite cognitive performers: a systematic review. *Sleep*, 44(7), pp.zsab008.
- Souabni M, Hammouda O and Nedelec M (2021). The effects of napping on athletic performance: A systematic review. *Frontiers in Physiology*, 14, pp. 10000707.
- Souissi N, Souissi M, Souissi H, Chamari K, Tabka Z, Dogui M and Davenne D (2008). Effect of time of day and partial sleep deprivation on short-term, high-power output. *Chronobiology International*, 25(6), pp.1062-1076.
- Souissi N, Chtourou H, Aloui A, Hammouda O, Dogui M, Chaouachi A and Chamari K (2013). Effects of time-of-day and partial sleep deprivation on short-term maximal performances of judo competitors. *The Journal of Strength & Conditioning Research*, 27(9), pp.2473-2480.
- Souissi M, Abdelmalek S, Bou Dhiba D, Theodoros Nikolaidis P, Ben Awicha H, Chtourou H and Sahnoun Z (2015). Morning caffeine ingestion increases cognitive function and short-term maximal performance in footballer players after partial sleep deprivation. *Biological Rhythm Research*, 46(5), pp.617-629.
- Souissi M, Chikh N, Affès H and Sahnoun Z (2018). Caffeine reversal of sleep deprivation effects on alertness, mood and repeated sprint performances in physical education students. *Biological Rhythm Research*, 49(5), pp.746-760.
- Souissi W, Hammouda O, Ayachi M, Ammar A, Khcharem A, de Marco G, Souissi M and Driss T (2020). Partial sleep deprivation affects endurance performance and psychophysiological responses during 12-minute self-paced running exercise. *Physiology & Behaviour*, 227, pp.113165.
- Spiegel K, Tasali E, Penev P and Van Cauter E (2004). Brief sleep curtailment increases hunger and caloric intake. *Annals of Internal Medicine*, 141(11), pp.846–850.
- Sweeney EL, Peart DJ, Kyza I, Harkes T, Ellis JG and Walshe IH (2020). Impaired insulin profiles following a single night of sleep restriction: the impact of acute sprint interval exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, 30(2), pp.139-144.

- Stickgold R (2005). Sleep-dependent memory consolidation. *Nature*, 437(7063), pp.1272-1278.
- Svensson T, Madhawa K, Hoang NT, Chung UI and Svensson AK (2024). Validity and reliability of the Oura Ring Generation 3 (Gen3) with Oura sleep staging algorithm 2.0 (OSSA 2.0) when compared to multi-night ambulatory polysomnography: A validation study of 96 participants and 421,045 epochs. *Sleep Medicine*, 115, pp.251-263.
- Tamaki M, Nittono H, Hayashi M and Hori T (2005). Examination of the first-night effect during the sleep-onset period. *Sleep*, 28(2), pp.195-202.
- Terry PC, Lane AM and Fogarty GJ (2003). Construct validity of the Profile of Mood States—Adolescents for use with adults. *Psychology of Sport and Exercise*, 4(2), pp.125-139.
- Tester NJ and Foss JJ (2018). Sleep as an occupational need. *The American Journal of Occupational Therapy*, 72(1), pp.7201347010p1-7201347010p4.
- The Sleep Charity (2024). *Dreaming of Change: A Manifesto for Sleep*. The Sleep Charity. Available at: <https://thesleepcharity.org.uk/get-involved/sleep-manifesto-2024/>
- Thun E, Bjorvatn B, Flo E, Harris A and Pallesen S (2015). Sleep, circadian rhythms, and athletic performance. *Sleep Medicine Reviews*, 23, pp.1-9.
- Tietzel AJ and Lack LC (2001). The recuperative value of brief and long naps for alertness and performance. *Journal of Sleep Research*, 10(3), pp. 197–204.
- Trotti LM (2017). Waking up is the hardest thing I do all day: Sleep inertia and sleep drunkenness. *Sleep medicine reviews*, 35, pp.76-84.
- Van Dongen HP and Dinges DF (2000). Circadian rhythms in fatigue, alertness, and performance. *Principles and Practice of Sleep Medicine*, 20, pp.391-399.
- Van Dongen HP, Vitellaro KM and Dinges DF (2005). Individual differences in adult human sleep and wakefulness: Leitmotif for a research agenda. *Sleep*, 28(4), pp.479-498.
- Van Dongen HP, Maislin G, Mullington JM and Dinges DF (2003). The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep*, 26(2), pp.117-126.
- Vardar SA, Öztürk L, Kurt C, Bulut E, Sut N and Vardar E (2007). Sleep deprivation induced anxiety and anaerobic performance. *Journal of Sports Science & Medicine*, 6(4), pp.532.
- Vyazovskiy VV (2015). Sleep, recovery, and metaregulation: explaining the benefits of sleep. *Nature and Science of Sleep*, pp.171-184.
- Walsh NP, Halson SL, Sargent C, Roach GD, Nédélec M, Gupta L, Leeder J, Fullagar HH, Coutts AJ, Edwards BJ and Pullinger SA (2021). Sleep and the athlete: narrative review and 2021 expert consensus recommendations. *British Journal of Sports Medicine*, 55(7), pp.356-368.
- Waterhouse J, Atkinson G, Edwards B and Reilly T (2007). The role of a short post-lunch nap in improving cognitive, motor, and sprint performance in participants with partial sleep deprivation. *Journal of Sports Sciences*, 25(14), pp.1557-1566.
- Watson A, Johnson M and Sanfilippo J (2020). Decreased sleep is an independent predictor of in-season injury in male collegiate basketball players. *Orthopaedic Journal of Sports Medicine*, 8(11), p.2325967120964481.

Wells ME and Vaughn BV (2012). Poor sleep challenging the health of a nation. *The Neurodiagnostic Journal*, 52(3), pp.233-249.

Wüst LN, Capdevila NC, Lane LT, Reichert CF and Lasauskaite R (2024). Impact of one night of sleep restriction on sleepiness and cognitive function: A systematic review and meta-analysis. *Sleep Medicine Reviews*, pp.101940.