Measuring, monitoring, and improving sleep variables: its application to professional football players

A thesis submitted in partial fulfilment of the requirements for a degree of Doctor of Philosophy

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I. Abstract

After several papers reported that Whole Body Cryotherapy (WBC) can improve objective and subjective markers of sleep, supported by anecdotal reports of post-exposure sleepiness from players at Southampton FC (SFC; PhD sponsor), the original aim of this thesis was to elucidate the effect of WBC on sleep in professional football players. However, after the UK COVID-19 lockdowns, WBC was not considered covid safe and, therefore, sleep became the central theme. Sleep plays an important role in the maintenance of both physiological and psychological homeostasis. During sleep, the release of human growth hormone and other anabolic hormones peak, inflammatory processes are modulated, and memories and skills are consolidated. Therefore, sleep is considered integral to athletic recovery and player well-being. Despite this, professional football players regularly present with sub-optimal sleep duration and/or quality. However, the factors associated with sleep variability are not fully understood, and there is no consensus on what the optimal level of sleep for athletes is. Therefore, this thesis conceptualised the following research questions: (1) What is known about the quality and duration of sleep amongst professional footballers? (2) What factors affect sleep in professional football players, specifically at SFC? (3) What are suitable and effective ways of improving sleep in professional football players? These questions were addressed across 2 systematic reviews (Chapters 2 & 4), an interventional study (Chapter 3), an observational cohort study (Chapter 5), a method agreement study (Chapter 6), and finally a case study (Chapter 7).

Chapter 3 presents a study that aimed to (1) investigate the effect of a WBC applied across an in-season microcycle on the objective and subjective sleep quality in under-18 (U18) professional footballers, and (2) determine the effect of WBC on game-day inflammation, testosterone, and cortisol. Unfortunately, this study was curtailed by the COVID lockdowns. Nevertheless, novel findings were reported. Specifically, whilst objective sleep data were not significantly different between groups, players who received WBC during the microcycle preceding a competitive fixture, reported a greater sense of alertness following wake, as determined by the Leeds Sleep Quality Index. Whilst these results are subjective, they could also be indicative of improved sleep architecture following WBC. However, considering objective sleep was determined from wrist-worn activity monitors without the capability to detect sleep stages, this cannot be known with certainty.

In Chapter 4, a scoping review of observational studies was performed that suggested that professional football players' mean sleep duration, sleep latency, and wake after sleep onset (WASO), were all within recommended guidelines (these same reference limits were also used for Chapter 4). This conclusion was made on the basis that over 63% of the included studies reported means that were above the lower reference boundary for sleep duration. Despite this, several papers reported error bars that exceeded the reference limits, suggesting that suboptimal sleep remains common among individual players. In Chapter 5, an observational study was performed on under-18 professional SFC players, and the results matched what was observed from the scoping review in Chapter 4. Specifically, whilst sleep duration on matchday+1 (the day proceeding matchday) presented with a beta estimate (derived from linear mixed models) of 400mins, the remaining day types presented with sleep durations of above 420mins, the lower end of the reference limits. Nevertheless, in this study, confidence intervals breached the reference limits, therefore, further suggesting that suboptimal sleep occurs in this population. In tandem, results from Chapter 4 and Chapter 5 potentially indicate that group-level interventions are unnecessary. Rather, practitioners may find it more efficient to target support to players who report sleep disturbances.

The scoping review presented in Chapter 4 also suggested that professional football players' sleep was also more variable compared to age-matched controls and several factors (e.g. scheduling variables) were associated with disrupted sleep. Chapter 5 builds on these findings by demonstrating for the first time that scheduled start time (the time players were scheduled to arrive at training or for a fixture) was associated with the amount of sleep that U18 players attained. Specifically, for every hour increase in start time, player sleep duration increased by an estimated 19.1mins (CI:9.4–28.79; $p<0.001$). This occurred in tandem with an 18mins (CI:9.3–26.6; $p<0.001$) later wake time, per hour increase in scheduled start time. It is not clear to what magnitude start time would have to be extended to generate increases in player performance, secondary to increased sleep duration. However, considering the player's age from this study (age: 17.3 ± 0.7yrs), a later start time may befit their intrinsic chronotype and, therefore, support the players by reinforcing their natural sleep habits.

Whilst data from Chapter 5 support the notion that scheduling variables are associated with sleep in U18 professional footballers, they also suggest that sleep is not meaningfully associated with external workload. Global positioning and accelerometry data were collected and collated across 1-day, 7-day, and 28-day periods. For every 100m increase in high-speed running (>5.5 $m \cdot s^{-1}$), sleep onset and wake time were extended by 4.68min (CI:2.78—6.58mins) and 3.38mins (CI: 1.27—5.5mins), respectively. However, considering that workload had no significant effect on total sleep duration, the changes to wake time and sleep onset time should not concern practitioners.

In Chapters 3, 5, and 7, objective sleep monitoring was completed using ReadiBand wrist-worn activity monitors. Though, it was acknowledged that these devices cannot readily link objective sleep quality and performance, and players' data could be missing due to poor band adherence. Therefore, another approach was trialled where the effect that inadequate sleep has on cognitive variables that are sensitive to sleep loss was determined, rather than measuring sleep directly. Consequently, this thesis also assessed the use of a novel virtual reality eye-tracking device that could rapidly administer an oculomotor task which was reported to be sensitive to total sleep deprivation. However, to be efficacious in a footballing environment, the device would have to demonstrate sensitivity to the daily fluctuation of sleep. Target radial variation (a measure of spatial accuracy) was found to be significantly correlated with perceived daytime sleepiness ($r=0.33$, $p=0.005$), however, no further relationships were observed between oculomotor function, psychometric vigilance, daytime sleepiness, and sleep metrics. In a retrospective analysis on a second data set from military personnel (that was included to augment the original analysis), only psychomotor vigilance, and not oculomotor function, were associated with the total amount of sleep achieved. This suggested that this device would not be efficacious in a footballing environment as a replacement for sleep monitoring.

Following the research presented in Chapters 4 and 5, it was surmised that a bespoke approach to sleep intervention would be more efficacious than team-based interventions. To this end, a framework was conceptualised in collaboration with a multidisciplinary team from SFC (Chapter 7). Next, a player was referred to the scheme after reporting excessive night time awakenings. After consultation, the player completed several subjective questionnaires to assess sleep quality (Pittsburgh Sleep Quality Index), insomnia severity (Insomnia Severity Index), and daytime sleepiness (Epworth Sleepiness Scale) followed by a period of objective sleep monitoring. The sleep monitoring confirmed excessive nighttime awakenings and based on the responses from the initial consultation, a sleep hygiene intervention was applied tailored to the players' responses during the initial consultation. Results revealed improved subjective sleep quality, insomnia severity, and nighttime awakenings. Whilst a case study cannot establish causality, it does provide a potential framework for practitioners looking to provide targeted sleep interventions.

Conclusions:

- In general, professional football players' sleep quantity, latency, and WASO is within available population-based reference limits.
- Scheduling variables, and not workload variables, are associated with activity monitor-derived objective sleep metrics in professional football players.
- Scheduled start time is associated with the amount of sleep that professional U18 football players receive.
- An oculomotor task does not have the requisite sensitivity to detect acute sleep loss in professional football players.
- A bespoke sleep intervention strategy can be efficacious in an applied footballing environment for players reporting sleep disruption.

II. Acknowledgements

This PhD was completed during the Covid 19 pandemic and lockdown, which rendered the original research theme mute. This also affected the resources available to support subsequent research. If it were not for the strong support, encouragement, and mentoring provided by my supervisory team, St Mary's University, Twickenham, and Southampton FC, then I would not have been in this position.

Firstly, I to Professor Charles Pedlar, whose enthusiasm was contagious. Whenever a fresh challenge presented itself, you were on hand with activity monitors or oculomotor tests. You always knew the right person, at the right time. This, alongside your invaluable guidance and experience, was invaluable to the success of the thesis.

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To Charlotte Willbourne, who smiled politely as I ranted about sleep, cold immersion, and physiology for almost 4 years.

I would also like to thank Jonny Woodhouse, Dr Amy Spencer, Dr Greg Clarke, and the rest of the U18 coaching staff for facilitating several studies throughout this thesis.

III. Declarations

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

Manuscripts that have been published based on work from this thesis

1. Luke Edinborough, Stewart Bruce-low, Jessica Hill, Jonathon Woodhouse, Mark Jarvis, Charles Pedlar (2023). *Day Type and Start Time May Influence Sleep in Adolescent Professional Football Players*. Int J Sports Med. DOI: 10.1055/a-1974-5441
2. Edinborough L, Hill J, Jarvis M, Bruce-Low S, Pedlar CR. A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study. J Sports Sci. 2023 May 14:1-8. doi: 10.1080/02640414.2023.2213032.

Oral Presentations

3. Luke Edinborough, Stewart Bruce-low, Jessica Hill, Jonathon Woodhouse, Mark Jarvis, Charles Pedlar (2021). *Influence of scheduling on objective sleep metrics in professional U18 footballers: a longitudinal observational study.* The British Association of Sport and Exercise Sciences 2021 annual conference.
4. Luke Edinborough, Stewart Bruce-low, Jessica Hill, Jonathon Woodhouse, Mark Jarvis, Charles Pedlar (2021). *Influence of scheduling on objective sleep metrics in professional U18 footballers: a longitudinal observational study.* St Mary's University, Twickenham, Festival of Research Conference

Invited Talks

5. A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study. ORRECO

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VII. List of abbreviations

Abbreviation	Definition	Abbreviation	Definition
ACC	Acceleration	L	Liter
AFS	Awakenings following sleep	LCL	Lower confidence limit
AM	Away match	LDH	lactate dehydrogenase
BAM+	Brief assessment for mood	LH	luteinizing hormone
BFSleep	Behavior following sleep	LMM	Linear Mixed Model
BFW	Behavior following waking	LSEQ	Leeds sleep evaluation questionnaire
BIC	Bayesian Information Criterion	MD	Match day
BL	Baseline	MDT	multidisciplinary team
CA	California	MEQ	Morning eveningness questionnaire
CAT	Catalase	MODREC	Ministry of Defence Research Ethics Committee
CD	conjugated dienes	NM	Night match
CI	Confidence intervals	NREM	Non-rapid eye movement
CK	Creatine Kinase	NSA	No scheduled activity
CL	Confidence limits	NSF	National sleep foundation
CMJ	Counter movement jump	PBC	Partial body cryotherapy
CON	Control group	PECO	participant, exposure, control, outcomes
COSMOS-E	Conducting Systematic Reviews and Meta-Analyses of Observational Studies of Etiology	PRISMA	Preferred Reporting Items for Systematic reviews and Meta-Analyses
CRP	C-reactive protein	PRISMA-ScR	Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews
CRYO	Whole-body cryotherapy group	PSG	Polysomnography
CV	coefficient of variation	PSQI	Pittsburgh sleep quality index
CWI	Cold water immersion	PVT	psychomotor vigilance task
DALDA	Daily Analysis of Life Demands for Athletes	QS	Quantity of sleep
DEC	Decelerations	R^2	Coefficient of determination
DM	Day match	REM	Rapid eye movement
DOMS	Delayed onset muscle soreness	REST-Q-Sport	Recovery-Stress Questionnaire for Athletes
EEG	Electroencephalography	RNCD	Royal Navy Clearance Diver
EIMD	Exercise induced muscle damage	RNS	Reactive nitrogen species
EPL	English Premier league	ROS	Reactive Oxygen species
ES	Effect sizes	RPE	Ratings of perceived exertions
ESS	Epworth sleepiness scale	RR	relative risk
ETHS	Eye tracking headsets	RT	Reaction time
EU	European Union	SD	Standard deviation
FA	Football Association	SFC	Southampton FC
FC	Football Club	SHI	Sleep Hygiene Index
GD	Game day	SOD	superoxide dismutase
GH	Growth hormone	SOL	sleep onset latency
GnRH	Gonadotropin-releasing hormone	SWS	Slow wave sleep
GPx	glutathione peroxidase	TBARS	thiobarbituric acid reactive substances
GPS	Global Positioning System	TD	Training days
GTS	getting to sleep	TNF-α	Tumor necrosis factor- alpha
Hg	Mercury	TV	Television
HM	Home match	U17	Under 17
HRV	Heart rate variability	U18	Under 18
HSR	High speed running	U21	Under 21
I^2	Percentage of variance	U23	Under 23
ICAM-1	intercellular adhesion molecule 1	UCL	Upper confidence limit
IGF-1	Insulin-like growth factor	UEFA	Union of European Football Associations
IL	interleukin	UK	United Kingdom
ISI	Insomnia severity index	USA	United States of America
KO	Kick off	VAS	Visual Analogue Scale
L	Liter	WASO	Wake after sleep onset
LCL	Lower confidence limit	WBC	Whole-body cryotherapy

VIII. Thesis introduction

I. Background

Contemporary football involves periods of low-intensity movements interspersed with high-intensity accelerative and decelerative actions [1–3]. The result is substantial disrupted physiological [4–7] and psychological [56–59] homeostasis, and the onset of exercise-induced muscle damage that can be measured in the days after exercise [12]. Considering that professional football players are required to perform up to 60 competitive fixtures per season [12], practitioners and researchers have invested great amounts of investigative interest in recovery strategies aimed at re-establishing pre-exercise function [13], maintaining athletic performance [14], and reducing injury risk [15].

Optimal sleep quantity and/or quality is considered an essential element to athletic recovery [14] and to the maintenance of physiological [16] and psychological [17] homeostasis. During sleep, the release of human growth hormone and other anabolic hormones peak, inflammatory processes are modulated, and memories and skills are consolidated. Furthermore, recovery from muscle-damaging exercise has been observed to be impaired in the presence of sleep restriction [18], and sleep extension has been observed to improve elements of physiological and psychological wellbeing after competition [19].

Despite a well-documented relationship between sleep, recovery, and performance, athletes have been observed to have suboptimal sleep compared to age-matched controls [20], with professional football players presenting with significantly greater sleep onset latency variability compared to non-athletic comparators. However, the factors associated with sleep disruption in these populations are not fully understood, nor is the optimal approach to sleep monitoring and intervention.

This thesis was first instigated to assess the use of Whole-Body Cryotherapy (WBC) in professional football players. After several papers reported that WBC can improve objective and subjective markers of sleep, supported by anecdotal reports of post-exposure sleepiness from players at Southampton FC (SFC; PhD sponsor), this thesis initiated studies which aimed to elucidate the effect of WBC on sleep in professional football players. However, after the UK COVID-19 lockdowns, WBC was not considered a covid safe therapy, and the English Football Association prohibited its use. Therefore, considering the aforementioned information and the work completed thus far, sleep became the central theme.

II. Thesis aim and objectives

Initially, this thesis aimed to answer the following questions:

1. What are the optimal exposure frequency and timing of WBC within the professional microcycle at Southampton FC?
2. Can WBC be used as an ergonomic sleep a id for professional football players?

After covid, adaptions were made, and the following thesis aims were conceptualised:

1. What is known about the quality and duration of sleep amongst professional footballers?
2. What factors affect sleep in professional football players, specifically at SFC?
3. What are suitable and effective ways of improving sleep in professional football players?

These aims were addressed across 2 systematic reviews (Chapters 2 & 4), an interventional study (Chapter 3), an observational cohort study (Chapter 5), a method agreement study (Chapter 6), and finally a case study (Chapter 7). See Figure 1 for a schematic overview of the thesis.

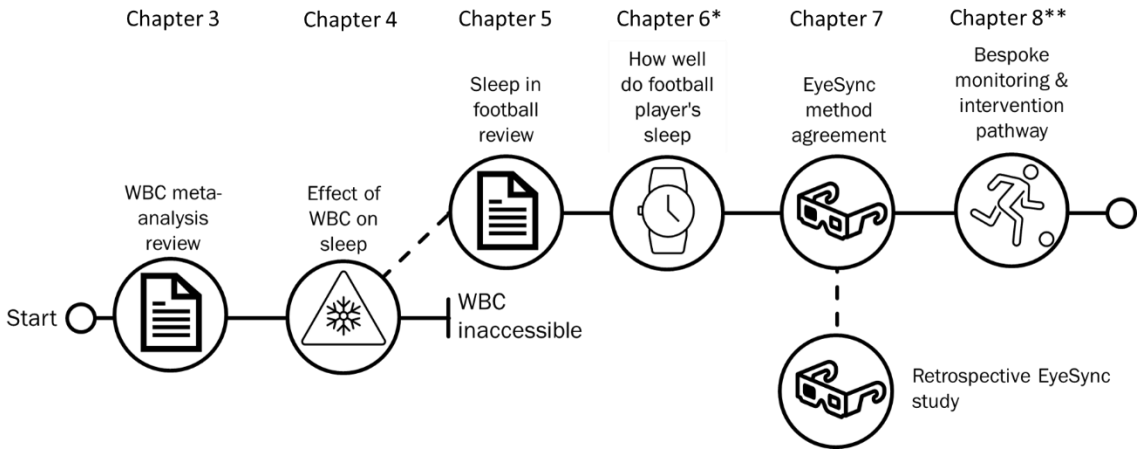

Figure 1: PhD thesis schematic. *Published in the International of Sports Medicine **Published in the Journal of Sports Science

III. Specific aims

The specific aim of this thesis are as follows:

1. Examine the use and frequency of post-exercise WBC, compared to passive recovery, on markers of inflammation, redox, and variables related to post-exercise fatigue **(Study 1)**
2. Investigate the effects of WBC, applied across an in-season microcycle on the objective and subjective sleep quality in under 18 (U18) professional footballers, and determine the effect of WBC on game-day inflammation, testosterone, and cortisol **(Study 2)**
3. Examine what is known about sleep quality and quantity, in relation to published norms, and identify the main literature themes concerning barriers to optimal sleep in full-time, professional footballers **(Study 3)**
4. Assess the influence of scheduling and workload variables on objective sleep markers in professions football players **(Study 4)**
5. Investigate if a virtual reality eye-tracking smooth pursuit assessment is sensitive to day-to-day variation in sleep metrics, and assess if the test can detect the presence of sleep loss in a military training environment with prescribed sleep deprivation **(Study 5)**
6. Trial an individualised sleep monitoring and intervention strategy aimed at improving the subjective and objective sleep in a professional U18 football player reporting suboptimal sleep **(Study 6)**

Chapter 1

1. The demands of elite football and the role of sleep in recovery and performance (Literature review: part 1)

1.1. Physiological and psychological demands of football

To understand the role and importance of sleep in athletic recovery and wellbeing in professional football players, it is logical first to understand the effects of competitive fixtures and training on subsequent physiological disruption.

Therefore, the purpose of this section is to explore the physiological and psychological demands of football before implicating sleep in the recovery process.

1.1.1. Match and Training demands

The physiological demands of modern association football (football) have increased over recent decades [21,22]. The appropriate evaluation of player work rate is, therefore, necessary for sport scientists to monitor athlete condition and implement appropriate training and recovery regimes. With the advent of global positioning system (GPS) and multiple-camera tracking technologies, large sets of data quantifying the external loads experienced by players in elite football have been generated [23]. This has enabled an increasingly accurate evaluation of player activity [24], work rate [25], load, and injury risk [15].

Elite male footballers cover a distance of 9 to 14km per competitive fixture. This is largely dominated by low-intensity activity, with intermittent high-intensity movements interspersed throughout a game [1–3]. One study conducted in the English Premier League observed that the distance travelled at high-intensity running (speed >19.8 $km \cdot h^{-1}$) can exceed 3000m [26] and Barnes et al. [27] highlighted that sprint distance has increased by approximately 30% between the 2006/07 and 2012/13 seasons, emphasising the growing physical demand placed on footballers.

Further research has also highlighted inter-positional and inter-game variability in the amount of work completed at high intensity. Di Salvo et al. [28] examined the within-position differences in physical performance in Premier League and Championship (English tier-two league) players across multiple seasons. In both leagues, the greatest distance covered at sprint speeds (>25.2 $km \cdot h^{-1}$) was completed by wide midfielders, followed by attackers and wide defenders, with central defenders covering the least distance. Likewise, Dellal et al. [29] also observed greater high-intensity running (21-24 $km \cdot h^{-1}$, multiple-camera match analysis system) in wider positions and additionally noted differences when central midfielders were analysed based on their tactical roles (i.e. attacking or defensive). Moreover, whilst the total distance travelled appears unaffected by formation, attacking players performed more high-intensity work in 4-3-3 compared to 4-4-2 and 4-5-1 formations [30]. This is also possession dependant, with teams performing more high-intensity running in possession when utilising a 4-5-1 formation [31].

In addition to large amounts of high-intensity running across match play, research has highlighted considerable deceleration work within football. A meta-analysis demonstrated that footballers perform more high-intensity (>2.5m · s^{-2}) decelerations compared to other popular team sports [12]. This too is position-specific, with wider midfielders performing more decelerations and changes of directions throughout a game [24,32]. Furthermore, the number of high-intensity actions has also been associated with increased injury risk. Bowen et al. [33] used accelerometer and GPS data to characterise the relationship between acute:chronic workload ratio and injury risk. Amongst the findings, increased acceleration work across a three-week period was most associated with increased overall injury risk.

Match and/or training activities may also differ in professional (full-time, contracted player, with no other training or work obligations) players representing different age group teams (e.g., U18, U23, 1st team) from the same club. However, whilst senior 1st teams have frequently been analysed, there are somewhat limited data on U23 and U18 teams [24]. This may be due to a limited number of monitoring units [34], or differences in tactics and longer-term strategic factors (i.e., maturation status of players or preparing younger players for senior football compared to preparing senior players to be competitive) that make direct comparisons across age groups mute.

In one study, the match demands of U18, U23, and 1st team professional players representing an English football club were compared across a season [35]. Results suggested that U18 players completed significantly ($p<0.001$) less HSR (>5.5 $m \cdot s^{-1}$) distance and high-intensity burst distance (defined as acceleration (≥4.0 $m \cdot s^{-2}$), deceleration (≤4.0 $m \cdot s^{-2}$), or impact (≥11G) activities completed in succession separated by 20 s or less) compared to U23 and 1st team players. However, in both cases, the effect size was revealed to be *small* (Cohen's D: 0.2-0.6). Considering the effect sizes, it is not clear if the significant differences in match activities between age groups elicits a meaningful response to the severity of EIMD, injury risk, or potential sleep disruption. It is also noteworthy that the authors elected to group accelerative, decelerative, and impact activities. Declarative actions are more associated with the onset of EIMD, compared with acceleration [36], whereas some analysis suggests that accelerations are more greatly associated with non-contact injury risk [33]. It may be useful to understand how these specific variables differ over different age groups so that recovery strategies (e.g., whole-body cryotherapy or sleep support) can be better tailored.

In a similarly designed study, the same analysis was applied to U18 and 1st team players representing a professional club in Switzerland [35]. In this instance, accelerations and decelerations were analysed independently. Compared to the 1st team, the U18 team performed a significantly lower number of decelerations (≤4.0 $m \cdot s^{-2}$) per game (1st: 33.7 ± 9.5, U18: 27.3 ± 8.1), although no significant difference was detected in the number of accelerations (≤4.0 $m \cdot s^{-2}$) per game (1st: 19.4 ± 6.7, U18: 18.5 ± 6.8).

This suggests that 1st team players may be at greater risk of EIMD, and potential sleep disruption compared to U18 players.

It is challenging to compare the data between these two studies directly [35,37]. Firstly, the variables of note in this discussion are reported in incompatible ways (i.e., analysing accelerations and decelerations independently compared to grouping them with impacts). Furthermore, the two studies are set in different countries and different clubs which may implement differing tactics, playstyles, and development targets. This may be presented in differing match actions. Nonetheless, these studies still highlight the fact that football players of all age groups experience considerable external load, including decelerative loading. Therefore, players likely experience notable EIMD and wider physiological disruption [38].

Regardless of the inter-positional and potential inter-team (e.g., U18, U23, 1st team) heterogeneity, the prevalence of deceleration work in football implicates a high degree of mechanical loading during competitive fixtures [38]. Significant demand is placed on a player's ability to repeatably absorb decelerative forces through eccentric muscular contraction, in turn causing sarcomere disruption and exercise-induced muscle damage (EIMD) [39].

1.1.2. Validity of external load measures

There is a range of available external load metrics that can be derived from both accelerometry (e.g., number of high-speed decelerations and accelerations) and GPS (e.g., total distance and distances performed at a predefined velocity) units, and the consensus is that these metrics can give a valid indication of daily or accumulated load alongside peak match demand [40,41]. The devices themselves have also been validated against gold-standard methodology. Specifically, devices show good agreement for peak speed and distance covered with radar guns and runs over predefined distances, respectively [40,42]. Furthermore, their reliability has been consistently observed [42,43]. However, the specific validity of individual external load variables may depend on the post-exercise physiological disruption that sport scientists wish to monitor. For example, the number of high-speed decelerations is associated with the onset and severity of EIMD [4–7] whereas the number of accelerations has been associated with an increased risk of non-contact injury [33]. Therefore, the validity of the individual external load measure may depend on the explicit element of physiology that a practitioner wishes to monitor.

Selecting an appropriate external load metric to monitor potential sleep disruption is not unequivocal. Although this is discussed in greater detail later in this review, a conclusive relationship between external load and sleep quality in football players has not been established [44–46], despite studies that highlight a potential association between sleep and external load in other sports [47]. However, the

limited number of studies that have assessed the potential for a relationship in football have, thus far, only utilised subjective [44,45] or activity monitor [46] based sleep monitoring which cannot elucidate sleep architecture. This indicates the need for further exploratory observational studies in professional footballers to provide greater clarity on any potential meaningful association.

Several studies that have sought to study potential relationships between external workload and sleep have utilised high-speed running (HSR) distance (running speeds $m \cdot s^{-2}$) as a global measure of total workload [44–46]. Whilst this approach discounts activity at other speeds, or specific associations between sleep and greater speeds, previous research has reported large correlations ($R > 0.6$) between HSR and other external load metrics (e.g., total distance) [46]. However, HSR in isolation may not encompass all aspects of physiological disruption that may impact EIMD severity, sleep propensity, or sleep quality/quantity. The number of decelerations has been linked to EIMD severity [4–7], and the associated DOMs, may impact sleep (i.e., pain/discomfort during nocturnal movements). Likewise, considering evidence linking the number of accelerations with non-contact injury risk [33], and separate evidence, albeit limited, highlighting a potential link between sleep quality and injury [48], it is logical to include these actions in any future exploratory analysis in football players.

It is not the purpose of this literature review to make recommendations and influence the method by which Southampton FC analyses its data. Rather, it looks to establish if their current practises have the requisite validity to give an accurate indication of a player's external load so subsequent analysis can determine if there is a statistically significant association between external load and sleep.

1.1.3. Exercise-induced muscle damage in football

The eccentric muscular contractions, resulting from the notable number of high-intensity decelerations, and changes of direction in contemporary football, likely causes EIMD [24,49]. Metabolic processors also likely contribute to primary EIMD [50], however, eccentric mechanical loading is considered to be the primary driver of EIMD onset and severity [36,51]. The importance of eccentric loading can be attributed to the fact eccentric muscular contractions recruit fewer motor units compared to concentric contractions of the same force [36,51]. Consequently, greater mechanical stress is placed on fewer muscle fibres resulting in structural and physiological disruption of those fibres. Specifically, during lengthening, sarcomeres can stretch non-uniformly until the actin and myosin filaments of the contractile apparatus no longer overlap. This can result in the sarcomere "popping" phenomenon [52] and increases the tension on the non-contractile structural proteins of the contractile unit. In turn, this can result in further disruption to the ultrastructure of the muscle fibre and contributes to the subsequent EIMD [52].

EIMD typically presents with oedematous swelling, an influx of intramuscular proteins and enzymes in blood, delayed onset muscle soreness (DOMS), impaired muscular function, and further inflammatory processes that may exacerbate the initial muscular damage [36]. Considering the complexity and scale of mechanisms surrounding EIMD, its onset and severity are challenging to determine non-invasively in football players [36]. Rather, EIMD can be assessed indirectly by sampling the levels of intramuscular proteins in blood, repeatedly, over time [7], measuring muscle function [6], or subjectively monitoring a player's perception of DOMs [53] in the proceeding time after the initial damage. Considering the impact that EIMD may have on a player's comfort, injury risk, and performance, it is not surprising that there is a plethora of studies that have sought to characterise EIMD in professional senior football players [4–7]. However, it is important to note that there is a scarcity of data that has also examined the onset and severity of EIMD in adolescent players. Whilst it is likely that EIMD will remain consistent over differing age groups, there may be elements relating to a player's maturation status that may alter the time-course of EIMD symptom severity and recovery. Nevertheless, it is evident that professional football players do withstand significant EIMD that can be sampled over the proceeding days [4–7].

The effects of a competitive fixtures on markers of EIMD have been examined extensively [4–7] in adult male professional football players. Creatine Kinase (CK) is a commonly measured blood marker of EIMD. In muscle, CK catalyses the reaction where adenosine diphosphate donates a phosphate ion to create adenosine triphosphate [4]. As a result of EIMD, muscle cell integrity is degraded and CK leaks into peripheral blood, where it is sampled to track the time-course of EIMD recovery [36]. Varley et al. [7] sampled CK after two competitive fixtures and saw a significant increase, compared to pre-match levels, and CK had not normalised after 60 hours. Likewise, an earlier study [12] observed that, in second-division football players, CK had not returned to baseline by 72 hours post-game.

Inter-game variation in CK has also been highlighted with one study observing differences of up to 41% in a between-game analysis of CK activity. Authors suggested that variations in high-intensity actions likely resulted in differences in CK levels, however, the study did not quantify changes of direction, distance covered, or any other measure of in-game activity, so these findings should be interpreted with caution [54]. Nevertheless, CK is highly variable with factors including age, ethnicity, muscle mass, hydration, exercise intensity, and fitness affecting levels in blood [4,5,36]. It has been suggested that CK has more validity in detecting the presence of EIMD rather than the magnitude [36]. More robust conclusions might be obtained if within-athlete CK activity is examined, however, CK activity might also be considered alongside other markers of EIMD to gain a more complete gauge of EIMD severity and recovery.

For example, while Nedelec et al. [55] were not able to run correlations between CK and match activity due to technical issues, perceptions of muscle soreness were strongly correlated with the number of sprint actions of less than 5m, at 48 and 72 hours post-match, in professional footballers. Whilst the accelerations likely contributed to the severity of EIMD, it is likely that the deceleration phase of the sprint generated damage to the ultrastructure of the muscle, causing nociceptor stimulation, and pain [36].

Alongside the appearance of intramuscular milieu in blood, and the onset of soreness, EIMD is also associated with the reduction of muscular performance. This likely impacts the players ability to perform in training, or in competitive fixtures [55]. For example, after a match, the magnitude of the reduction in countermovement jump (CMJ) performance and knee extensor torque were correlated with the number of directional changes within a fixture. Moreover, while peak power output, as determined by a CMJ, does not appear to have the same within-game variation compared to CK activity [6], correlations have been detected between CK and power output, suggesting that both are valid in determining the severity of post-game EIMD. This supports the idea that player performance is affected by the onset, and potentially the severity, of EIMD. Observations of reduced muscle function combined with perceptual indices, (e.g., DOMS) indicate that football induces significant levels of EIMD, and it is likely that its severity is dictated by the number of high-intensity match actions, chiefly decelerations, that a player completes. Up to 120 hours might be necessary for players to fully recover from a professional game, necessitating a need for comprehensive recovery strategies, especially during periods of fixture congestion, where up to three games might be played in a 7 day period.

1.1.4. Inflammation and reactive oxygen species.

After EIMD, an immune response is triggered that mediates the subsequent repair and adaptation processes [36,56]. The term ‘inflammation’ is often used to characterise this response and it consists of a cascade of leukocytes, pro-inflammatory macrophages, and anti-inflammatory macrophages that have a multitude of cellular and transcriptional effects associated with damaged muscle break-down, tissue repair, and muscle plasticity [56]. Inflammatory proteins may also have an influence on sleep in humans, and act as hypnogenic compounds [57]. Much of what is understood about the inflammation response to EIMD is derived from animal models that have elicited muscle damage through unloading/loading paradigms [56]. However, histological observations in humans also offer insight into the time-course of inflammation in humans.

Muscle biopsies sampled directly after muscle-damaging exercise in cohorts of healthy males suggest that leukocytes, predominately neutrophils, accumulate immediately after EIMD onset [58,59]. These then transmigrate to sites of muscle damage and break down damaged tissue through phagocytosis and the release of proteolytic enzymes. In turn, this generates substances that are readily turned into reactive

779 nitrogen (RNS) and oxygen species (ROS) [56]. The time-course of the subsequent influx of
780 inflammatory proteins is heavily dependent on the intensity and unfamiliarity of the initial exercise, as
781 well as the pre-exercise state of muscle [51,56,60,61]. In humans subjected to 'severe' muscle-damaging
782 exercise, leukocyte levels have been observed to remain above baseline for up to three weeks post-
783 damaging stimulus [60] and myofiber necrosis has been observed after electrostimulation [61].
784 However, this level of physiological disruption is not likely under normal exercise conditions and
785 research suggests that leukocyte levels typically peak within 24 hours post-EIMD [62], and disappear
786 rapidly from repairing muscle fibres [56].

787 After the initial neutrophil invasion, pro-inflammatory macrophages, notably tumour necrosis factor-α
788 (TNF-α) and interleukin-1 (IL-1) [63,64] begin to accumulate approximately 1 to 4 hours post muscle-
789 damaging exercise [56]. These proteins also have phagocytosis effects and initiate further downstream
790 inflammatory proteins [65]. Between 4 and 24 hours post-muscle damage, anti-inflammatory
791 macrophages can be observed in both muscle biopsies and in the extracellular space [56]. These anti-
792 inflammatory macrophages, for example IL-10, are associated with myogenin expression, and myotube
793 formation and initiate other key repair transcription factors associated with repair and muscle plasticity
794 [66].

795 The release of anti-inflammatory proteins marks the commencement of the muscle repair and adaptation
796 phase of the inflammatory process. Much of what is understood about inflammations' mechanistic role
797 in adaptation and muscle plasticity is derived from knock-out animal models, where specific genetic
798 mutations have been deselected [67]. However, mice and rodents, that constitute much of the animal
799 models, are more metabolically active and their inflammatory processes may be quicker compared to
800 exercising humans [68]. Nevertheless, murine models' rich genetic diversity enables researchers to
801 obtain specific gene mutations that enable them to model a multitude of proteins, cellular processes,
802 and diseases in a manner that cannot be repeated ethically in humans [67]. Such investigations have
803 elucidated the roles that specific inflammatory proteins have in muscle plasticity, secondary to EIMD.
804 For example, mouse model investigations suggest interleukin (IL) -6 (IL-6) is essential for myoblast
805 proliferation [46]. Likewise, studies in cultured murine myoblasts examined in vitro have demonstrated
806 that low levels of IL-1β significantly impair myogenesis [70]. Further, in rodents subjected to hindlimb
807 suspension and then reloading to cause muscle damage, IL-10 was observed to be a critical mediator of
808 muscle repair and regeneration through its impact on myogenin transcription factors [71].

809 It is clear that the inflammatory process is axiomatic to the homeostatic processes that mediates the
810 breakdown, repair, and adaptation of damaged muscle, secondary to EIMD [72]. However, the same
811 inflammatory process can also acerbate the primary damage [73]. Toumi and Best [73] proposed that
812 the breakdown of damaged muscle tissue through neutrophil-mediated phagocytosis generates

substances readily turned into ROS, including superoxide and hydrogen peroxide. ROS and RNS have an inherently unstable chemical structure, hosting one or more unpaired electrons within their atomic orbitals. This results in a significantly reactive radical [74]. Under repeated or severe exercise, ROS and RNS production may initiate further phagocytosis and generate oxidative stress can damage cellular proteins, lipids, and deoxyribonucleic acid, in turn, exacerbating the initial damage and disrupting remodeling [75]. See Figure 2 for a schematic overview relationship between EIMD, inflammation and ROS.

Inflammation presents in a biphasic dose-response relationship, synonymous with exercise-induced hormesis [72,76]. Whilst it is essential to the EIMD recovery and adaptive remodelling process, the production of RNS and ROS can exacerbate the initial damage. Therefore, it is essential to manage this process in players to ensure a balance between the restorative and muscle-damaging components associated with inflammation. Practitioners employ several methodologies to manage inflammation and support EIMD recovery, and sleep may be a primary modulator with holistic systemic effects [57].

Notably, sleep, inflammation, and immunity share a two-way relationship. Not only do certain inflammatory proteins act as sleep-initiating hypnogenic compounds, but pro-inflammatory proteins peak during the night in humans [57], suggesting that the sleep state is a key regulator of the inflammatory process. Sleep is further associated with the anabolic compounds which drive key anabolic processes that are associated with EIMD recovery and muscle plasticity [57]. These factors are reviewed later in this chapter.

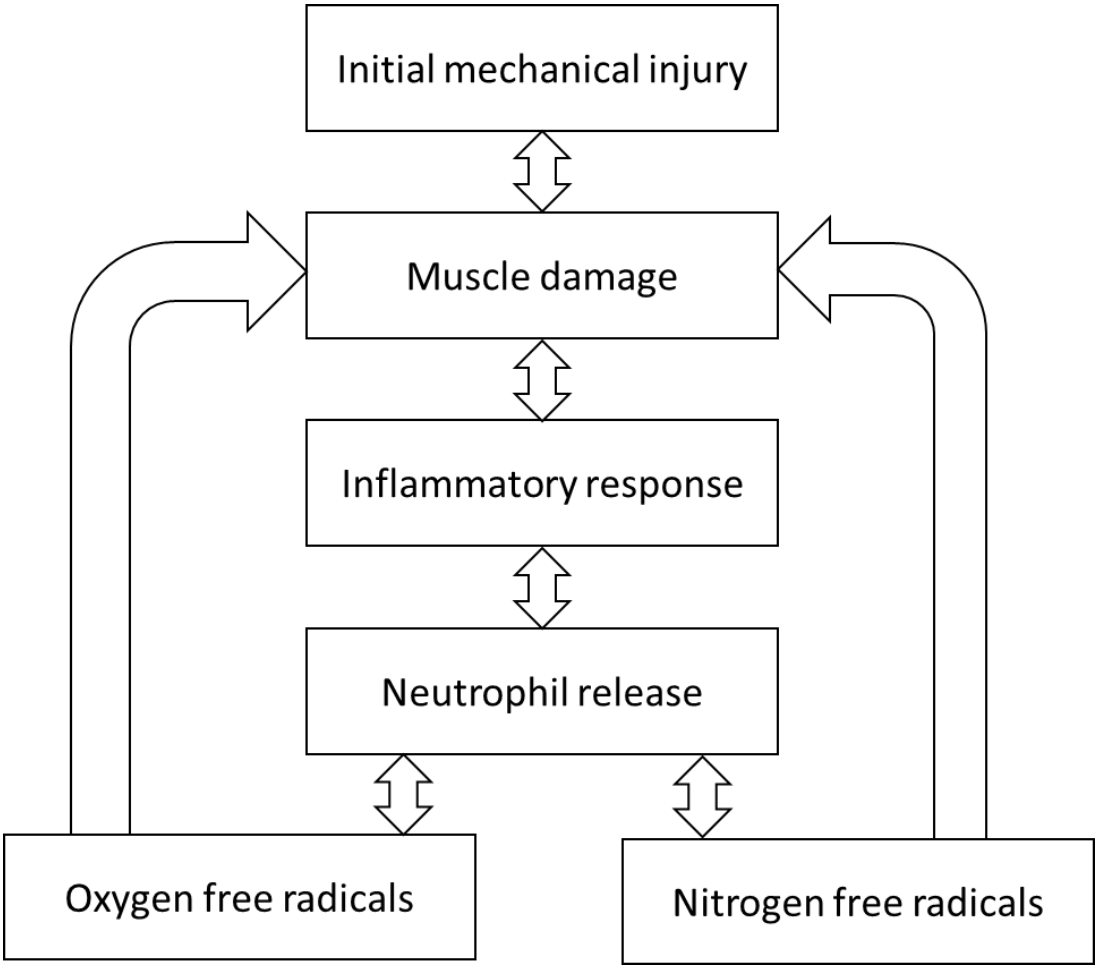

Figure 2: The relationship between the inflammatory response to mechanical injury and further muscle damage, adapted from Toumi and Best [73]. The initial mechanically induced damage produces myofibril tearing and inflammatory cell infiltration. Neutrophils may promote further damage through the release of oxygen-free radicals and lysosomal proteases and elastases [73].

Several antioxidative mechanisms preserve optimal reactive oxidant balance and limit oxidative stress, however, these can be outpaced by repeated EIMD, even in an adapted muscle [77]. The influx of neutrophils, other leukocytes, and pro and anti-inflammatory macrophages in addition to other inflammatory proteins can be measured in blood to determine the magnitude of the inflammatory response.

1.1.5. Inflammatory response to competition in football

Romagnoli et al. [78] saw a 3-fold increase in neutrophil levels after a competitive fixture in Italy's Serie A. Levels peaked at 30 minutes post-game and had not returned to pre-game levels after 48 hours. This occurred in tandem with an influx of CK demonstrating an inflammatory response occurring alongside EIMD. Another study in players competing in secondary divisions in Portugal saw less modest increases in neutrophil levels after a competitive fixture and levels had returned to pre-game

levels by 24 hours post-game [79]. Differences in the immune reaction across the two studies can be attributed to the potential variances in match intensity or state of residual fatigue upon entering into the study.

After the neutrophil invasion, an influx of interleukins has also been observed after competitive fixtures. IL-6 is a pro-inflammatory cytokine and is commonly used as a marker of inflammation following the initial immune response. IL-6 has been observed to be significantly increased immediately after a competitive fixture, normalising by 13 hours post-game [80]. Compared with basketball, volleyball, handball and a non-exercising control, Souglis et al. [81] noted that footballers experienced the greatest increase in IL-6 and wider inflammatory markers (e.g. TNF-α). These results also coincide with research that suggests that footballers experience greater highspeed decelerations compared to other sports [24], indicating greater eccentric work. Romagnoli et al [82] saw a similar response in IL-6 after a professional football game, with levels peaking 30 minutes post-game and returning to pre-match values by 24 hours post-game. Therefore, a greater acute-phase inflammatory response is expected.

Further inflammatory markers are also commonly sampled in the hours and days post-competitive fixture to indicate the presence and magnitude of the inflammatory response in football players [83]. For example, C-reactive protein (CRP) is hepatic in origin and is produced in response to IL-6 and TNF-α. During inflammation, its primary purpose appears to stimulate tissue factor production and clear tissue debris [65]. In elite-level footballers, Souglis et al. sampled CRP immediately, 13 and 37 hours post-game [51]. Although levels were increased at 13 hours, they returned to baseline levels at 37 hours. Likewise, Romagnoli et al [82] saw elevated levels at 24hrs post-fixture, compared to baseline, however, levels had normalised by 48hrs. A further study looked to characterise the inflammatory time course in football players up to 144-hours post fixture [84]. Their results concurred with previous research [51]. CRP peaked at 24 hours, and no significant difference was observed beyond 48 hours post-competition, compared to the control. However, despite no significant differences from 48 hours to 144 hours, results still presented with substantial heterogeneity (as evidenced by the magnitude of the error bars). Whilst, a larger study power might have generated significant results, a more recent study with a larger sample size also noted no significant difference 2 days post-fixture [51]. In line with data that indicates positional differences in match activity, the same study also noted greater CRP levels in midfield players.

Some research has highlighted CRP as a valid measure of player load, particularly when multiple games are played across a single week [85]. In 23 players from an under-20s team competing in São Paulo's first division, one study tracked endocrine and inflammatory markers during a 7-day period where 3 games, each separated by 48 hours, were played. Each variable was correlated with the number of GPS-recorded high-intensity actions completed by players and, whilst significant moderate to weak

correlations were detected in IL6 and IL1β after all three games, only CRP presented with a strong correlation after game 2 ($r=0.71$, $p<0.01$) and game 3 ($r=0.79$, $p<0.01$) in addition to a moderate correlation after game 1 ($r=0.59$, $p<0.59$) [50]. Considering the severity of EIMD and injury risk is most likely causally linked with the amount of high-intensity work completed by players [6,24,33], this suggests that CRP is a valid measure of player load, particularly when multiple games are played across a microcycle.

Furthermore, CRP is readily sampled in both relatively small (~25µl) quantities of blood and serum using point-of-care assay devices, without the need to transport samples to a laboratory via a cold-chain. This enables a valid and convenient method of obtaining CRP levels without altering the players' normal routine. However, care must be taken in the interpretation of CRP. As discussed, it is released as part of the inflammatory cascade [85], but, its production is agnostic of the source of inflammation. As a result, CRP may be elevated due to a respiratory virus (e.g., the common cold [86]), or autoimmune disease (e.g., Asthma [87]). Considering these factors alongside residual inflammation from a previous bout of physical activity, research needs to be mindful of a plethora of factors when deciding at what point in the microcycle CRP should be sampled. Following typical workload tapers that occur in the days before a fixture, the theoretical most rested state of a football player is the hours before a competitive fixture. Likewise, the most fatigued state is in the hours post-fixture. All other factors aside, these points provide two potential windows in which a valid repeatable measure can be obtained.

1.1.6. Psychological impact

Psychological demands of football incorporate a multitude of factors that can impact wellbeing, anxiety, motivation, football-specific skill execution, and performance, with some suggesting that perceptual responses may be an early indicator of fatigue [56–59]. Several self-reported athlete-specific assessment tools have been utilised throughout the literature. Tests, including the Daily Analysis of Life Demands for Athletes (DALDA) [88], Recovery-Stress Questionnaire for Athletes (REST-Q-Sport) [89], and the Brief assessment of Mood (BAM+) [90], incorporate multiple components to assess mood state and perceived recovery. Whereas simpler tests self-report DOMs, or individual Likert scales for mood, wellbeing, stress and sleep [91].

The perception of workload is also a consideration and proved to be a powerful tool. A recent review has highlighted those subject assessments benefit from simplicity and links with an athlete's physiological and psychological status have contributed to its longevity [92]. An older study recorded significant correlations ($r = 0.50$ to 0.85) between post-training session ratings of perceived exertions (RPE) and several heart-rate-based indices of fatigue in u18 footballers [93]. More recently, a similar trend was observed between morning subjective fatigue and heart rate variability in a similar cohort

[91]. This demonstrates that the demands of competitive football also perturbed the psychological homeostasis of the players.

1.1.7. Recovery methods

Within the performance sciences, recovery can be defined as a multifaceted, physiological, psychological and time-relative process that re-establishes pre-exercise function [13]. This process can be supported by strategies designed to modify the physiological and psychological side effects of match play so that a more efficient recovery is achieved [13,94]. A plethora of these strategies have received in-depth investigative interest and are briefly reviewed herein.

1.1.7.1. Nutrition and hydration

The purpose of a post-football nutritional strategy is initially to replace glycogen and to rehydrate. It has been long held that cellular hydration supports protein turnover through the activation of anabolic pathways [95]. However, current hydrational strategies may already be sufficient for most team sport athletes. Research has demonstrated that athletes can recover from 2% of body mass loss of water through the intake of fluid, specifically with a Na^+ concentration of 61 mmol/L, within 6 hours of competition [96,97]. Consequently, so long as an appropriate rehydration strategy is observed, dehydration is unlikely to be a limiting factor in recovery. Likewise, the consumption of high glycaemic index carbohydrates at regular intervals maximises muscle glycogen resynthesis [96,97].

Sufficient protein intake is also required to support EIMD repair [98]. The most effective quantity and type of protein have been subject to some debate and football-specific data are scarce [99], however, around 20g of protein post-exercise appears sufficient to stimulate protein synthesis [100]. Compared to consuming carbohydrates or protein on their own, the co-ingestion of both substances has been shown to improve symptoms of EIMD, including creatine kinase levels and muscle function, despite no differences in anabolic signalling or glycogen metabolism [100,101].

Aside from macronutrients, several other vitamins, and antioxidant-rich supplements have been purported to enhance recovery [102,103]. Vitamins, specifically C and E, can stabilise ROS and are termed antioxidants. A recent meta-analysis [74] reviewed the role of the vitamins C and E in EIMD recovery, however, there was too much variability in blood markers of EIMD to make firm conclusions. In football-specific studies, De Oliveria et al. [104] reported inhibited oxidative stress, characterized by reduced lipid peroxide activity, in football players who received high-dose vitamin supplementation supplantation for 7-days before and after an exercise stressor, compared to a placebo control. Nevertheless, CK activity, vertical jump and sprint performance were not significantly different between groups, suggesting no effect on EIMD. It may be possible that the effect of vitamin supplementation on EIMD is negligible over short-duration interventions, and longer terms studies are

required to observe a statistically significant effect. In support, one longitudinal study supplemented vitamin C and E across a season and significant differences were only observed at the end of the season rather than at predetermined sampling points throughout the study [105]. While this suggests that vitamin supplementation can improve markers of oxidative stress, its effect on EIMD, and therefore athletic recovery appears muted.

1.1.7.2. Compression garments

Compression garments were initially used in the treatment of inflammatory conditions within clinical settings [106] and have been implemented widely among athletes to facilitate recovery from EIMD [79]. Nonetheless, there are few studies investigating their use in professional football players. A recent systematic review of recovery methods in footballers [108] highlighted just two studies that used compression garments in semi-professional footballers [81,82], with no data from contracted, full-time professionals. Nevertheless, compression garments worn during and for 3 days post-game (7 hours per day) failed to improve CK and lactate dehydrogenase (LDH), compared to a control group, in regional and national players. In the wider literature, the effectiveness of compression garments is equivocal [111], and the mechanism of support is yet to be fully elucidated. However, the addition of pressure is thought to reduce the space available for swelling to occur as well as positively affecting venous return, wider hemodynamic, and lymphatic drainage [112].

An 'ideal' compressive force of 17.3 and 15.1 mmHg has been suggested for the calves and quadriceps, respectively [113]. However, it has been highlighted that the pressure received from a garment may differ between individuals, potentially due to anthropometric disparities, and many may not receive adequate stimuli [86]. This in turn may account for the equivocal results between studies [111]. In support of this, Hill et al. [107] compared the influence of two garments that provided a mean pressure of 8.1 ± 1.3 mmHg and 14.8 ± 2.2 mmHg at the thigh level, respectively. Although there was no difference in CK, CRP, and myoglobin, muscle function was significantly improved with the higher-pressure garment. This suggests that pressure is an important modulator when prescribing compression garments.

1.1.7.3. Cold Immersion and cryotherapies

The aim of cold immersion and cryotherapies is to reduce tissue temperature to induce a therapeutic response. The reported benefits include analgesia, a reduction in tissue metabolism, and a reduction in inflammation post-EIMD [115,116] several cryotherapy methodologies are now available to practitioners, including the local application of ice packs, cold water-immersion, whole-body cryotherapy and partial body-cryotherapy. Definitions can be found in Table 1.

Table 1: Examples and definitions of cryotherapies

Cryotherapy	Description	Temperature	Duration
Cold water immersion (CWI) [117]	Neck-down, or waist-down immersion in cold water	<15°C	5 to 25 mins
Whole-body cryotherapy (WBC) [118]	Extremely cold air for short periods while wearing minimal clothing (slippers, socks, shorts, gloves, hat and face mask), in specially designed chambers.	-110°C to -160°C	120 to 240 secs
Partial-body cryotherapy (PBC)	Extremely cold air for short periods while wearing minimal clothing (slippers, socks, shorts, gloves, hat and face mask), in cabins with the head exposed.	-110°C to -190°C	120 to 240 secs
Local ice application [115]	Application of crushed ice (or similar) directly to tissue.		5 to 15 mins

Local ice application is used to reduce oedema and promote analgesia following tissue trauma. After a review, Bleakley and Hopkins [115] concluded that a tissue temperature <13°C is sufficient to decrease nerve receptor sensitivity, firing rate and muscle spasm. Other cryotherapies have recorded temperatures of 5.3 ± 3.0°C on the surface of the legs [119], suggesting a cold-induced analgesic effect is possible without the direct application of ice.

WBC is used as a recovery aid in elite sports settings, despite limited evidence of its effectiveness [90,92,93]. The limited number of investigations to date have reported no [116,122,123], mixed [124], or beneficial [125–127] effects of post-exercise WBC on inflammatory and wider EIMD markers.

CWI is a more established cryotherapy that is also used in an attempt to reduce inflammatory markers; several studies have suggested efficacy in football [128]. Compared to static stretching, one study found that CWI combined with active recovery significantly improved recovery from EIMD in academy footballers playing for a Premier League club [128]. Yet, no significance was found between active recovery and CWI. This suggests that CWI can be effective, but its efficacy is similar to strategies that are relatively less sophisticated and potentially better tolerated.

1.1.7.4. Massage

Massage describes the mechanical manipulation of body tissues with rhythmical pressure to promote health and well-being [129]. Massage is a commonly utilised recovery strategy with 78% of French professional football teams reporting regular use of numerous massage techniques including effleurage, petrissage, tapotement, friction, and vibration [130]. However, despite its widespread use, evidence suggests only moderate physiological benefits, and there is a scarcity of studies investigating its effectiveness in football players [103,131]. Studies have reported that massage had no effect on the removal of metabolic by-products, including H^+ and La^-, and did not modulate peripheral blood flow [104]. A meta-analysis of 22 trials did note modest improvement in muscle performance recovery but found that the effect sizes were greater in non-athletic, compared to athletic, populations and found that

shorter (5-12 min) treatments appeared more effective [133] [101]. Nevertheless, this meta-analysis also highlights the inconsistencies between the results in the included studies [133]. This in turn may be due to the challenges in controlling the massage pressure of the masseuse and body composition of the receiver.

Interestingly, massage also appears to have a role in supporting psychological recovery. Not only is a more pronounced effect on the perception of DOMS recovery compared with objective markers of EIMD [134], but, the association between massage and positive mood states is long established [135]. The mechanisms supporting perceptual and mood enhancement secondary to massage are unclear, however, it might be related to the social interaction between the athlete and masseur leading to sympathetic withdrawal or a placebo. That said, the overall balance of the research suggests that massage can be an effective method to aid psychological recovery.

1.1.7.5. Active Recovery

Active recovery involves structured activity performed at low intensity for a short period (15 to 20mins). Common modalities include running, swimming, or cycling [100,136]. In the acute stages of exercise recovery, the effects of active recovery are long-established. Several studies have noted an increased rate of La- removal immediately after active recovery, suggesting an increase in blood flow leading to La^- oxidation [109–111]. In professional footballers competing in Spain, active recovery improved countermovement jump performance compared to those who completed passive recovery [140]. This study was not randomised and had no crossover element; therefore, results should be interpreted with caution. However, these results are corroborated by data collected from a randomised trial of professional footballers competing in Italy. In a comparison of recovery strategies, active recovery was more effective than water immersion and passive rest for reducing muscle pain, after preseason training [141]. This suggests, at the very least, a subjective effect on EIMD.

1.1.7.6. Stretching

Stretching is primarily used to increase range of motion, decrease musculofascial stiffness and is used frequently for injury prevention [100]. In a study on professional football teams, 50% of clubs surveyed reported using stretching as a recovery strategy [100]. However, stretching does not appear to be efficacious in enhancing recovery after exercise. A review of 12 studies completed by the Cochrane group found that post-exercise stretching had little to no effect on muscle soreness, noting consistent results across studies [114]. In footballers representing an English Premier League academy, a static stretching protocol was implemented post-match. Elevated CK levels, oedema, DOMS and reduced countermovement jumps confirmed the presence of EIMD. However, stretching was unable to produce

any changes in markers of EIMD at 48 hours post-match, suggesting a limited beneficial effect in footballers [143].

1.1.7.7. Sleep

Sleep and sleep in football is a central theme within this thesis and is reviewed in detail later in this thesis. In the interim, a brief outline of sleep relevance in football recovery is provided here.

Sleep is an essential, and multiphasic event that contributes to physiological and psychological health. Footballers are subject to physiological and psychological stressors (training/competition stress, DOMS, extreme lighting) that can negatively influence sleep and, in general, have been associated with suboptimal sleep quality compared to age-matched controls [20,46]. However, despite several authors commenting that further research is required, little progress has been made regarding the acute and chronic effects of reduced sleep in professional footballers and athletes in general [100,144].

What is clear, is that sleep facilitates vital metabolic and immune processors [100,144]. During sleep, anabolic hormones are released, which in turn promotes protein synthesis, peripheral muscular repair and plasticity [14]. Dattilo et al. [14] postulated that a reduction in testosterone and human growth hormone excretion, secondary to sleep restriction, can negatively affect athletic recovery. Athletes exposed to post-game sleep deprivation have recorded greater levels of CRP and CK compared to controls [18,145]. While this still requires elucidation, the concept that sleep and physiological recovery are synonymous is clear.

The link between sleep and cognitive health is better understood. The demands of competitive fixtures impose a psychological toll and sleep is recognised to be a key modulator in the stress-recovery continuum [146]. Athletes exposed to sleep deprivation after competition report reduced performance in sport-specific skills [147], while sleep disruption due to travel significantly affects stress-recovery scores [18].

1.2. Sleep and recovery in professional football

Humans spend approximately one-third of their life in a state of sleep [148]. Sleep is not merely defined as an absence of wakefulness, but is an active, regulated and metabolically distinct state [148], characterised by a reversible perceptual disengagement from the environment [149]. The overarching reason for sleep is not clear, however during sleep, a plethora of homeostatic processors that are essential to health and well-being are up regulated [149].

In this section, the role of sleep in relation to recovery will be reviewed, followed by an overview of sleep physiology, sleep monitoring, and ways sleep might be improved in professional footballers.

1.2.1. Sleep physiology: mechanisms regulating the sleep/wake cycle

Sleep-wake regulation is generally explained through a two-process model termed Process S and Process C [151]. Process S represents sleep homeostasis, or sleep debt, and is associated with the accumulation of sleep-promoting substances that accumulate during wakefulness [150]. As Process S-associated substances reach an upper boundary, sleep onset is initiated. Likewise, as substances dissipate towards a lower boundary, wakefulness commences [150]. This boundary oscillates throughout the day (Figure 3).

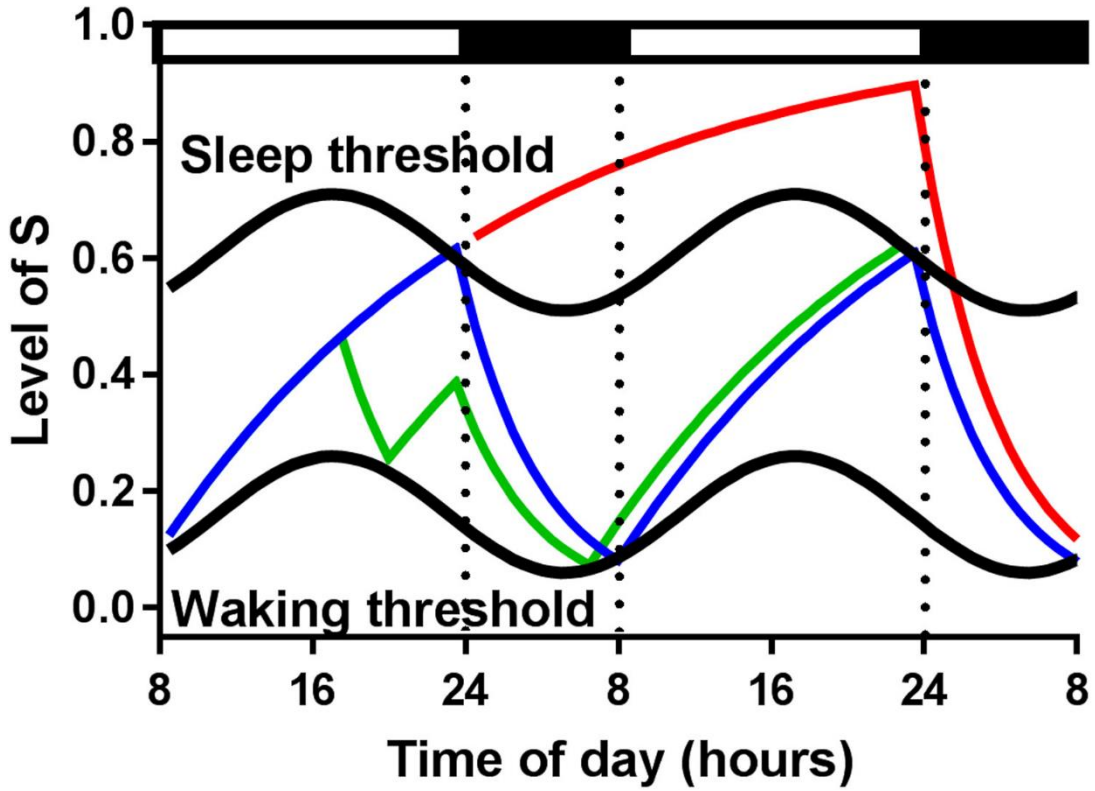

Figure 3: A simplified simulation of Process S. The normal sleep/wake timing is indicated by black and white bars, respectively. The blue line indicates the baseline condition with 8 hours of sleep and 16 hours of waking. During the time period that the blue line increases the model is awake. When it reaches the upper threshold (the upper sinusoidal black line) the model goes to sleep and the line decreases. This process continues until it reaches the lower threshold, and the model awakens again. The green line indicates the effects of a 2h nap starting around 18:00 followed by a normal night of sleep. The red line indicates sleep deprivation (40h of continuous waking by skipping a night) and recovery sleep during the following night. Note that the model assumes that naps and sleep deprivations have no effect on circadian regulation on the next day. Taken from DeBoer, 2018 [150].

The accumulation of sleep-promoting substances was first identified in animal studies where sleep was induced in rested controls by the transfusion of cerebral spinal fluid from sleep-deprived subjects [152]. Subsequent investigations have sought to identify specific compounds and their respective mechanistic interactions that inhibit sleepiness and/or wakefulness [127]. Brown et al. [153] proposed that sleep-inducing factors should fulfil the following criteria: 1. Administration induces sleep, 2. Levels of the substrate should increase with sleep propensity, and 3. Substances should act on brain regions that are involved with sleep.

Adenosine has been strongly implicated as a clear wakefulness inhibitor whose kinetics appear synonymous with Process S [127]. The hypnogenic effects of adenosine were initially elucidated in felines [128] and further research has highlighted that the administration of adenosine or adenosine agonists can induce sleepiness and reduce cognitive function [155,156]. Adenosine levels have also been observed in a dose-dependent manner with time spent awake [157]. Consequently, accumulation tracks sleep propensity. Neuro-stimulants including caffeine, often recreationally consumed in the form of coffee, and other substances (e.g., theophylline) actively work as adenosine receptor antagonists, blocking adenosines sleep-promoting effect [153]. Interestingly, caffeine is often consumed before competition by athletes who seek to benefit from its stimulating effect to support performance [16]. However, this may also impair their ability to sleep and recover, particularly after night games [16]. Nevertheless, whilst there is a vast evidence base implicating the waking accumulation of adenosine in the homeostatic onset of sleep (Process S), much of the data is in animal studies. Nevertheless, adenosine remains strongly implicated across all mammalian species.

Other substances have also been associated with Process S, including nitrous oxide and prostaglandin D2, however, mechanistic pathways remain somewhat unknown [153]. Cytokines, normally associated with inflammation, also appear to have a notable role in sleep regulation [158]. In humans, IL1 administration results in fatigue and sleepiness [158], and levels of IL1 and TNF-α appear to track sleep propensity, peaking at sleep onset [159]. This further demonstrates the propensity of sleep with inflammation and EIMD recovery. Furthermore, in rodent models, ribonucleic acid expressions of IL1 and TNF-α demonstrate a diurnal pattern [160,161]. This suggests that recovery modalities with purported anti-inflammatory actions (e.g., WBC [116], tart cherry juice ingestion [162]) may also modulate sleep regulatory behaviour. This has received some attention in the literature. For example, WBC has been reported to reduce the number of nocturnal movements in physically active males. However, the results are conflicting [163]. Likewise, tart cherry juice has anti-inflammatory actions and was able to improve sleep, although, whether this was related to inflammatory protein modulation or other mechanisms (e.g., naturally occurring melatonin) is unknown [162].

Process C dictates the daily rhythm of sleep. Under this process, sleep onset is initiated through several circadian processors driven by a series of endocrine-controlled homeostatic actions mediated by the hypothalamus [164]. Circadian activity actively synchronises to an approximate 24-hour cycle [165]; however, individuals entrain differently depending on exogenous and endogenous signals. The primary exogenous stimuli are light/dark signals passing through the retinohypothalamic tract to the suprachiasmatic nucleus of the anterior hypothalamus. Decreases in light lead to increased secretion of melatonin from the pineal gland. Melatonin, in turn, transmits time information to other homeostatic processors associated with sleep onset [131,164]. These induce the physiological changes associated with sleep onset, including increased vagal tone and parasympathetic activity, reduced heart rate, and a

reduction in core temperature [166,167]. Increased light signals close to bedtime, for example from electronic device use, inhibit melatonin production, in turn, down-regulating Process C and affecting subsequent sleep onset [131,168]. While it is unknown if device use in footballers is greater than that of the general population, sleep hygiene interventions that limit phone use have been successful in improving sleep quality in highly trained amateur footballers [169]. Consequently, electronic device use might inhibit sleep onset in footballers as well as the general population [131,168].

Endogenously, how an individual's circadian activity is entrained to a 24-hour system is subject to individualised factors that differ from person-to-person [138]. The result can be described by way of a chronological phenotype, or chronotype, which reflects the phase of entrainment of an individual [165,170]. An individual's chronotype can be quantified by determining the point of mid-sleep on nights when there are no work or additional pressures affecting sleep or wake time. By determining the point of mid-sleep in this manner, it is hypothesised that sleep onset is more likely to occur in line with their chronotype [165]. However, chronotype is more traditionally assessed on a continuous scale using specially validated questionnaires (e.g., Morningness-Eveningness Questionnaire (MEQ) or the Munich Chronotype questionnaire and categorised based on a person's 'morningness' or 'eveningness' [170]. Morning types prefer waking and sleeping earlier, whereas evening types preference a later wake and sleep onset time; these are also colloquially termed larks and owls, respectively. An individual's chronotype extends beyond sleeping preferences and is further reflected in a range of physiological and cognitive processors that are subjected to circadian pressures, including differences in glycaemic control [171], appetite [144], alertness [145], and academic performance (in adolescent students) [145].

There is clear evidence indicating that chronotype varies across ages. In a large-scale cross-sectional study (n= 53,689), Fischer et al [165] modelled the point of mid-sleep (time measure of chronotype) on work-free days and determined that peak lateness occurred during late adolescence, approximately 104 mins later than the lifespan average [165], before transitioning to an earlier time throughout an individual's 20s, 30s and 40s [165]). While the data presented in a near-normal distribution, indicating very late and very early chronotypes across all ages, results still demonstrated a clear relationship between age and chronotype [165]. It follows that circadian sleep pressures may differ across ages, and this may need to be reflected in how professional footballers' start times are scheduled across age groups.

In adolescent students in the USA (age: 13 to 18yrs), scheduling a later school start time resulted in longer sleep durations [173], reductions in daytime sleepiness [174], reductions in motor vehicle accidents, and improved academic performance [175]. Whilst the factors that influence sleep behaviour in professional footballers may be different in similarly aged general populations, adjusting start time may improve sleep in adolescent professionals.

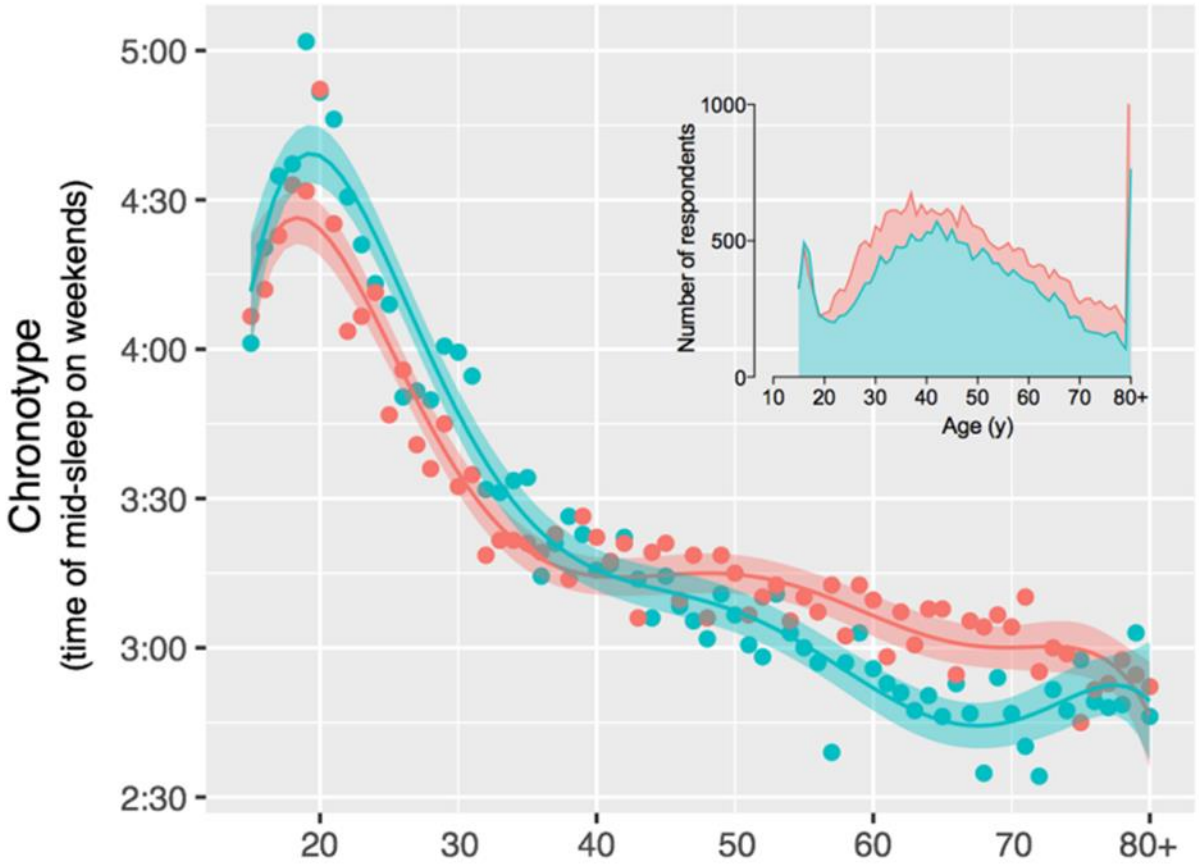

Figure 4: Graph showing the mean ± SD chronotype by age. The inlay represents the number of responses by age. Blue represents males, pink represents females. Taken from Fischer et al [165].

Thermoregulation also appears causatively associated with sleep onset, with a further impact on sleep quality and architecture [176]. Approximately 2 hours before sleep, core temperatures begin to decline under circadian (Process C) control [177]. The reduction is caused by increased peripheral, notably distal [178], vasodilation that shunts warm central blood to the periphery where body heat can be dissipated into the ambient environment [176]. As a result, a decrease in core, and increase in peripheral temperature is observed before sleep is initiated [176,179]. Changes in distal vasodilation and reductions in core temperatures track melatonin release and are consequently considered a circadian process associated with Process C [176]. The temporal relationship between sleep onset and core temperature can be modified by exogenous manipulation. For example, in 8 healthy participants, the administration of melatonin supplementation at 1300 increased distal skin temperature, and decreased core (rectal) temperature out of phase of their normal diurnal rhythm [180], and the blockade of melatonin release through the application of harsh light nullified the circadian temperature change [181].

Immersion in hot water prior to, but not immediately before, sleep has been shown to increase sleep depth and decrease sleep latency [176]. This has been termed the 'warm bath effect' and does not appear

immediately compatible with the circadian cooling role in sleep [176]. The mechanistic pathways require elucidation, nevertheless, heating before sleep may augment distal vasodilation which, in turn, may have a direct impact on sleep or, alternatively, may further facilitate the conduction of heat from the core [182]. Van Someren et al. [182] proposed that changes in core and skin temperature could modulate neuron activity in sleep-regulating areas of the brain. To test this, sleep was objectively assessed (PSG) while participants wore a water-cooled whole-body thermal suit during sleep that was capable of selectively and independently cooling distal and/or proximal areas of the body. The data suggested that a 1°C increase in proximal skin temperature shortened sleep latency by 2.68mins (CI: 1.34 – 4.03mins) [183]. It should be noted that this relationship was only revealed through a regression analysis that was performed post hoc, nevertheless, the results solidified the relationship between thermoregulation and sleep onset.

Another study combined data from two interventional protocols (total n= 20) where participants were free to initiate sleep independently from any external cues or zeitgebers (external time cues), in order to evaluate the role of heat loss in sleep initiation. Compared to core and distal skin temperature, results revealed that the distal-to-proximal temperature gradient was the strongest variable in predicting sleep onset latency [179]. This implicates sleep-wake states as a major driver of thermoregulation, and not just a consequence of circadian processors [177]. It also suggests that sleep onset is linked to the thermoregulatory response that dissipates heat from the core, rather than changes in core temperature itself. specifically sleep onset may be associated with a feedback loop secondary to peripheral vasodilation. This is further demonstrated by studies [184] where ice was ingested before sleep. Although core temperature declined, sleep was not initiated. Instead, alertness increased alongside vasoconstriction [184].

The vasodilation that facilitates the movement of core heat to the surrounding environment is caused by the decreased sympathetic drive to the vessels of the periphery [185]. This is suggestive of a general reduction of sympathetic and an increase in parasympathetic drive that commences approximately 2 hours prior to sleep [167], occurring in tandem with melatonin release [131,164] and core temperature reductions [177]. Parasympathetic drive can be readily quantified through indirect assessment of the vagal control of the heart using heart rate variability (HRV) analysis [186]. Through this methodology, a clear circadian pattern has been observed across several demographics that accumulates in increasing vagal (parasympathetic) signals as sleep onset approaches [166,187,188], this is also accompanied by a concurrent reduction in heart rate [166] (Figure 5). In turn, this implies that sleep onset requires an autonomic nervous shift towards parasympathetic predominance [185]. Disruption, or augmentation, of this process may affect sleep.

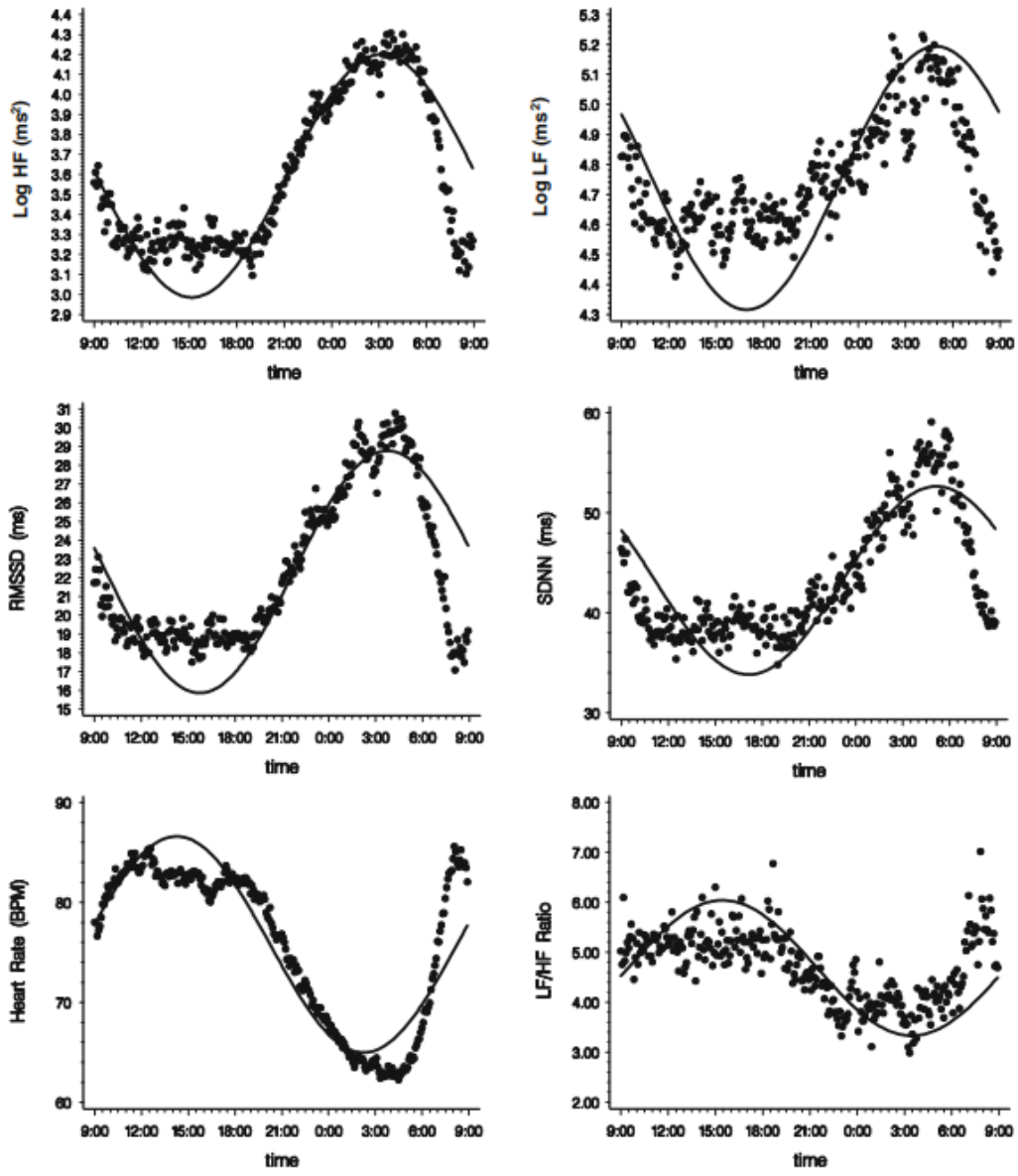

Figure 5: The Circadian rhythm of heart rate variability variables overlaid with time-specific segment averages. Overall periodic curve derived from random-effects meta-analysis (solid line). Time-specific segment average values (Dots). Taken from [166].

1.2.2. Sleep physiology: sleep architecture

Once sleep is initiated, it can be defined as a reversible behavioural state of perceptual disengagement from the environment and its onset is marked by distinct electrical changes within the brain [149]. However, sleep itself is not a homogeneous state [149]. Distinct phases of sleep can be identified through the measurement of action potentials across the brain using electroencephalography (EEG) [149] and the structure and organisation of sleep, termed sleep architecture, can be described. Normal sleep has two distinct phases, non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is further divided into stages N1, N2, and N3, each representing the relative depth of sleep [149]. Previously, N3 was subdivided and referred to stages 3 and 4, respectively. However, stages 3

and 4 were combined considering the difficulty in interpreting the stages [189]. Examples of EEG recordings for each stage of sleep can be found in Figure 6.

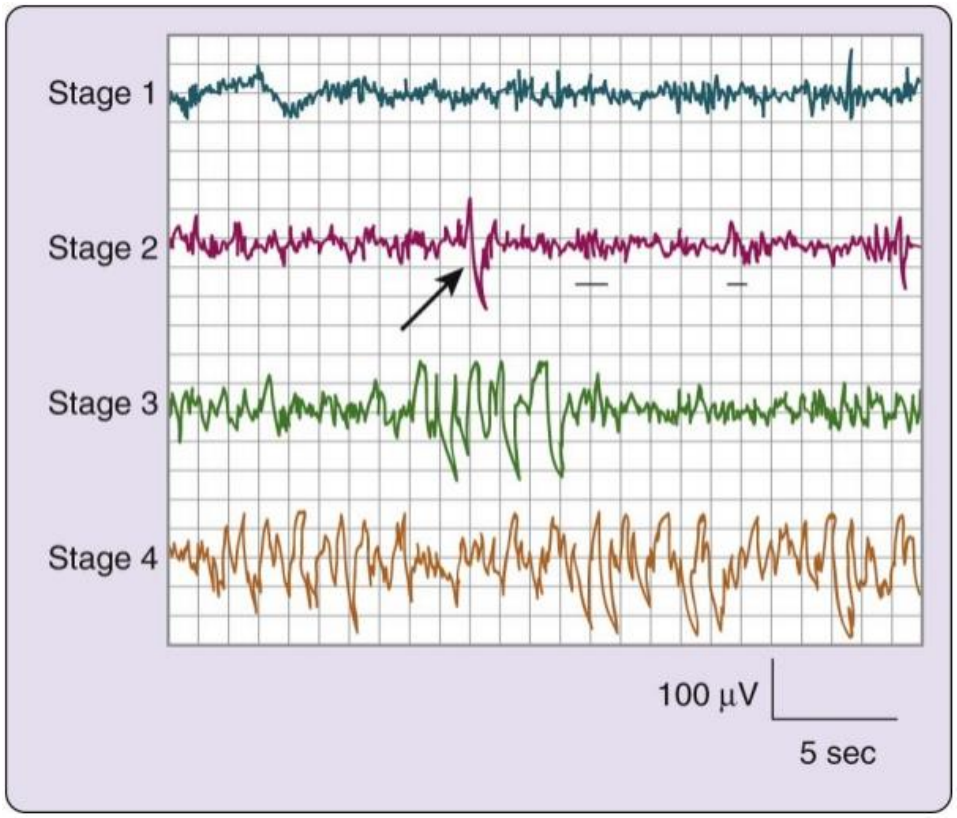

Figure 6: Electroencephalogram (EEG) characteristics of each of the 4 stages of non-rapid eye movement sleep. The four electroencephalogram tracings depicted here are from a 19-year-old female volunteer. Each tracing was recorded from a referential lead (C3/A2) recorded on a Grass Instruments Co. (West Warwick, R.I.) Model 7D polygraph with a paper speed of 10 $m \cdot s^{-1}$, time constant of 0.3 sec, and 1/2 -amplitude high-frequency setting of 30 Hz. The arrow denotes the presence of a K-complex and the horizontal line denotes sleep spindles. Taken from Carskadon and Dement (2011) [149].

NREM stage 1 (N1) is marked by the transition of recurrent alpha waves to mixed frequency waves. This sleep stage typically lasts less than 10 minutes and serves as a transition from wakefulness to sleep [149] (Figure 6). When Sommers et al. [190] recorded sympathetic afference by monitoring the interneural nervous activity of muscle blood vessels alongside cardiovascular measures, entry into NREM sleep was associated with a significant reduction in heart rate and mean blood pressure. This suggests increasing parasympathetic predominance on entry into sleep and is synonymous with the circadian autonomic pattern [166]. Sommers et al. [190] did not report a significant change in sympathetic activity during N1, nevertheless, the transition into stage occurs when the distal to core temperature gradient is at its maximal [177].

NREM Stage 2 (N2) is characterised by the presence of sleep spindles, spontaneous rhythmic bursts of EEG activity, and k-complexes, small positive signals on either side of a larger negative wave [161], and is further associated with reduced heart rate, blood pressure, and core temperature compared to

wakefulness [139,149,162]. As sleep persists, the length of each successive N2 increases, eventually contributing to approximately 45 to 55% of total sleep duration [149].

In contrast, stage 3 (N3) only contributes to 3 to 8% of sleep, yet it is distinguishable by increased slow-wave activity [149]. Stage N3 has the highest arousal threshold of all the NREM of sleep and is characterised by increased high-voltage, slow-wave activity on the EEG [151]. N3 is termed slow-wave-sleep (SWS) and is marked by a reduction in sympathetic output [190]. As participants entered SWS sleep Sommers et al. noted significantly reduced sympathetic bust frequency and amplitude, compared to waking, from neurons controlling vessels in the lower limb vascular.

The relative stages of sleep are also sensitive to temperature fluctuations [176,177]. Using a theromosuit in 8 healthy subjects, the warming of proximal skin increased the proportion of slow wave sleep from 18.0 ± 3.6% to 25.9 ± 6.1% at the expense of lighter sleep states and nocturnal awakenings [191]. This occurred with a concurrent reduction in core temperature, suggesting that the mechanistic pathway in this case may be related to a feedback loop involving vasodilation and sleep-regulating parts of the brain [176,177]. Previously, suggestions have been made that the most efficacious way to predict sleep latency and NREM sleep depth is to induce distal vasodilation without increasing core temperature, rather than direct action on core temperature [184].

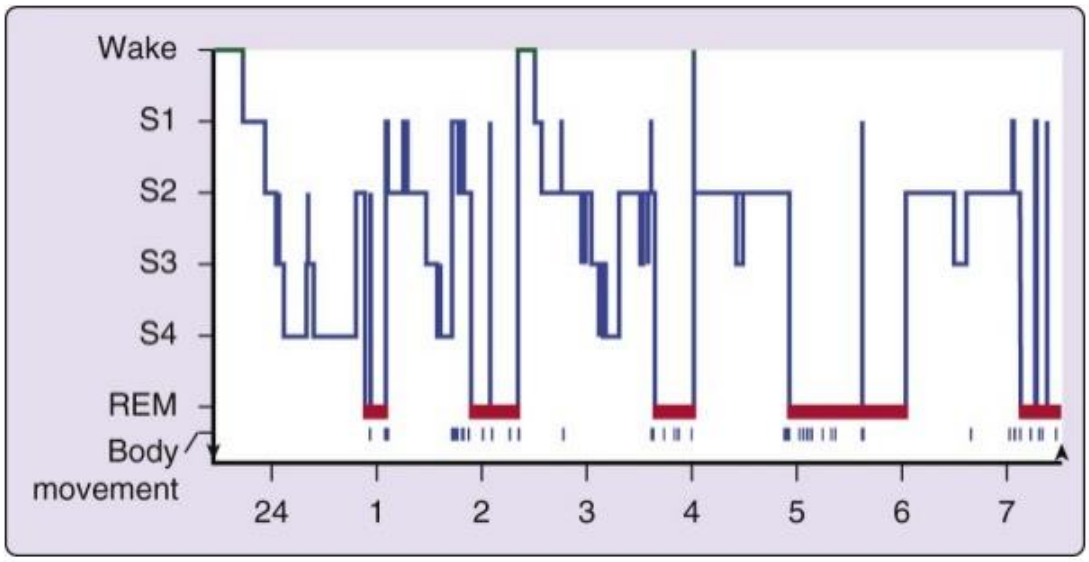

Figure 7: Progression of sleep states across one single night in a normal volunteer. This graph was based on an encephalogram, electrooculogram and electromyogram and assessed in 30 second epochs to derive the stages of sleep. Taken from Carskadon and Dement [149]. REM (rapid eye movement).

In healthy individuals, NREM (stages 1 to 4) and REM sleep alternate in a cyclical manner. The first cycle lasts between 70 and 100 minutes, and each subsequent cycle lasts, on average, between 90 and 120 minutes (Figure 7). During sleep, several well-documented physiological changes occur, these are summarised in Table 2.

The final stage of sleep is termed REM sleep and is characterised by the presence of low-voltage, mixed-frequency brain activity, complete muscle atonia, and bursts of rapid eye movements [149]. The initial phase of REM sleep may only last up to 5 min, however, subsequent bouts become progressively longer as sleep persists. Whilst REM sleep may occur during the first half of the night, it features predominantly more in the latter half [149]. REM sleep provides several essential cognitive tasks including functions relating to learning, motor skill, and memory consolidation [192,193]. Unlike NREM sleep, REM sleep presents with brain waves and autonomic activity that is more similar to that of wakefulness [149]. The notable difference between REM sleep and wakefulness is the state of atonia (absence of muscle tone) that prevents people from moving while dreaming [149].

Table 2: Physiological changes during Non-rapid eye movement and rapid eye movement.

Physiological Process	NREM	REM
Brain activity	Decreases from wakefulness	Increases in motor and sensory areas, while other areas are similar to NREM
Heart rate	Slows from wakefulness	Increases and varies compared to NREM
Blood pressure	Decreases from wakefulness	Increases (up to 30 percent) and varies from NREM
Sympathetic nerve activity	Decreases from wakefulness	Increases significantly from wakefulness
Muscle tone	Similar to wakefulness	Absent
Blood flow to brain	Decreases from wakefulness	Increases from NREM, depending on brain region
Respiration	Decreases from wakefulness	Increases and varies from NREM, but may show brief stoppages; coughing suppressed
Airway resistance	Increases from wakefulness	Increases and varies from wakefulness
Body temperature	Is regulated at lower set point than wakefulness; shivering initiated at lower temperature than during wakefulness	Is not regulated; no shivering or sweating; temperature drifts toward that of the local environment
Sexual arousal	Occurs infrequently	Greater than NREM

NREM (non-rapid eye movement)
REM (rapid eye movement)

1.2.3. Methods of assessing sleep quality, sleep quantity.

Sleep assessment methods can be categorised as objective or subjective. Objective measures utilise technologies and predictive algorithms to measure sleep quality and quantity. Some methodologies go further to provide detailed information on sleep architecture and nocturnal physiology [194]. Subjective measures use sleep diaries and questionnaires to determine perceived sleep quality and quantity and further assessments provide highlight the presence of insomnia or daytime sleepiness [194–196].

1.2.3.1. Objective sleep assessment

Polysomnography (PSG) is an objective method and is largely considered to be the gold standard of sleep quality assessment with the capability to provide an in-depth analysis of the structure and quality of sleep [194]. Polysomnography has had limited use in athletes. It can be complex and comparatively invasive with participants having to undergo extensive instrumentation to observe brainwave activity, muscle tone, eye movement, expired gas analysis, breath patterns, and cardiac indices [167]. Nevertheless, using polysomnography, researchers have highlighted short-term reductions in REM sleep and persistent disordered breathing in U17 footballers who participated in a training camp at 3600m above sea level [196]. This demonstrates the ability of polysomnography to produce a detailed study of athlete's sleep quality and architecture. Although portable polysomnography technologies are available [196], analysis is normally completed in specialised sleep laboratories. The unfamiliar sleep environment can reduce the validity and few sleep laboratories can accommodate large numbers of people over consecutive nights [197]. This makes it challenging to extensively utilise in team sport environments, nevertheless, it remains the gold standard method to measure sleep.

Wrist-actigraphy devices can also provide objective information on sleep in professional football players [20], and there is a growing literature base where they have been used to elucidate sleep quality in football players. Whilst these devices can estimate similar metrics to PSG (e.g., Wake after sleep onset (WASO), sleep duration, sleep onset latency, etc.), they provide data by interpreting nocturnal movements with proprietary algorithms, rather than encephalography [198,199]. This means that wrist-actigraphy devices cannot provide information regarding sleep architecture, therefore, the effect of scheduling variables, workload, and other factors on the relative depth of sleep cannot be ascertained from wrist-worn activity monitors alone [167]. Nevertheless, where PSG requires instrumentation that may alter a player's normal bedtime routine, or remove them entirely from their normal sleeping space, activity monitors remain a valid alternative that can collect objective sleep data relatively non-evasively compared to PSG.

Activity monitors are typically worn on the athlete’s wrist [197], and research has demonstrated high levels of agreement between these devices and PSG [198,199] when interpreting nocturnal metrics. In one validity study, 34 healthy non-athletes wore a range of 6 research grade and commercial wrist-accelerometery devices while sleep was also assessed using PSG [199]. Participants engaged in 2 nights of normal sleep, and a third night where sleep was purposefully disrupted. Compared to PSG, high epoch-to-epoch sensitivity (all $\geq$0.93) was observed across all sleep metrics (sleep duration, sleep efficiency, sleep latency, WASO) [199]. However, comparisons relating to sleep depth were mixed and did not show acceptable agreement with PSG, suggesting wrist-accelerometery is a valid assessment of two-stage sleep (i.e., assessing whether the wearer is in a state of wakefulness or sleep). Additionally, in 11 participants, when two wrist-accelerometers were worn concurrently, both devices demonstrated

93% agreement with each other over a 7 day period, demonstrating reliability [200]. Whilst wrist-worn activity monitors may provide a valid alternative to PSG that can provide objective information on participants' sleep, they may be limited by the internal algorithm used to estimate sleep metrics. Considering that each band's respective algorithm is proprietary, and therefore unique, it limits direct comparisons between bands and each algorithm must be validated against PSG. Furthermore, the accuracy of the band will be reliant on the quality of the predictive algorithm[199,201]. Moreover, it has been highlighted that periods of inactivity, such as sedentary time during travel, can be registered as periods of sleep. The raw data can be manually screened and corrected in some devices; however, this increases the risk of potential biases. Furthermore, unlike polysomnography, the stages of sleep cannot be measured [199,202]. Nevertheless, in combination with subjective assessments, their validity in providing objective sleep data and application in field research have rendered wrist-accelerometery highly efficacious in team-athlete sleep analysis [20,197,202].

1.2.3.2. Subjective sleep assessment

Subjective measures of sleep quality are less technologically sophisticated; however, they can provide a valid assessment of sleep quality and several questionnaires have been trialled in athletic populations [202,203]. They are suitable for field research in team environments and investigations have shown good reliability and validity between subjective and objective measures of sleep quality [202]. That said, they rely on truthful and subjective feedback from athletes that limits the confidence in which conclusions can be made.

Various subjective methods have received investigative interest. The Leeds Sleep Evaluation Questionnaire (LSEQ) is commonly used to assess subjective sleep quality and has been used in athletic populations [204]. The LSEQ uses ten 100mm visual analogue scales (VAS) to assess four sleep quality metrics that are largely synonymous with wrist-accelerometery measures. They include ease of getting to sleep, quality of sleep, awakenings following sleep onset and behaviour following wake. Participants are asked to mark the VAS where the midpoint represents the norm before any intervention. The Pittsburgh Sleep Quality Index (PSQI) also assesses sleep quality, but over a 19 item self-reported questionnaire. The PSQI assesses sleep quality over a 1 month period and therefore is not suited to shorter interventions. Scores of ≥5 on the PSQI indicated sub-optimal quality [205]. Further self-reported questionnaires do not assess sleep quality per se but do look to subjectively quantify related variables. For example, The Insomnia Severity Index (ISI) and Epworth Sleepiness Scale (ESS) have also been utilised to investigate clinically relevant insomnia and daytime sleepiness, respectively [206].

1.2.3.3. Other methods of assessing sleep

Sleep restriction also has profound effects on psychomotor abilities which can be measured through several methodologies, as a surrogate to typical objective and subjective assessments. The Psychomotor Vigilance Task (PVT) has commonly been utilised to assess psychomotor degradation after sleep restriction or deprivation [207]. In one study, 160 adults completed the PVT every 2 hours whilst staying awake for 24 hours. Results showed a clear detriment to performance as sleep restriction continued [207], demonstrating that the PVT is sensitive to sleep deprivation.

Since its inception, the PVT has evolved. The most recent iteration of the PVT involves responding to a randomised visual stimulus on a touch-screen computer tablet by tapping the screen. Further, research has also sort to highlight the most valid and sensitive PVT-derived metric in regards to sleep disruption [208]. Still, the PVT takes 10 minutes to complete, leaving it susceptible to lapses in concentration, limiting its validity in some cases [209]. One study evaluated the validity and sensitivity of 3- and 5-minute versions of the PVT against the standard 10 minute version. However, results showed that the shorter versions were not comparable in response speed, lapses, or errors, concluding that the 10min version remains the gold-standard [209].

Other tests also have the potential to give practitioners information on how their athletes have slept the night before, but are quicker than the 10 minute PVT. If these tests are demonstrated to be valid and reliable, then they may give practitioners a practical objective tool to assess how their athletes have slept, and potentially perform and recover, in a point-of-care manner. Research investigating the sensitivity of oculomotor function to sleep fluctuations is building momentum [210,211]. When a moving target is visually tracked, spatial and temporal predictions are used to circumvent the neural delay required for visuomotor processing. Specifically, the cognitively predicted path of the object must be synchronised with the true moving target during continuous tracking [212]. This ability to track an object in space, as well as time, is considered a function of attention [213]. In turn, attention, particularly sustained attention, is susceptible to sleep deprivation [214].

A small number of studies have investigated the sensitivity of a 3 min oculomotor smooth pursuit test to sleep restriction and sleep deprivation [210,211]. Originally developed to assess mild traumatic brain injury, the smooth pursuit test requires participants to visually track a target as it follows a predictable circular path. Eye-tracking software then determines the accuracy with which the target is tracked in both space and time [210,211]. In military personal subject to 26 hours of sleep deprivation, the smooth pursuit test revealed increases in tangential and radial variability, suggesting a loss of ability to predict the target in both time and space, respectively [211]. These results are collaborated by later research that found degradation of binocular coordination after sleep deprivation [215], demonstrating that oculomotor function is affected by sleep deprivation and a smooth pursuit test can detect it.

However, the majority of the research thus far has been collected from a military sample, undergoing total and extended sleep deprivation [211,215]. For the technology to have a wider impact, particularly in sports, future research needs to elucidate the influences of sleep restriction in athletes.

1.2.4. The relationship between sleep and exercise performance

The effects of sleep on physiological performance have been extensively researched using sleep deprivation (defined here as a complete absence of sleep) research designs [216,217], although, the results of such studies are somewhat equivocal in terms of the magnitude of change [16,217]. Nevertheless, the majority of studies have observed cognitive and/or physiological deficits as a consequence of sleep loss, albeit in laboratory settings [218–224]. However, such protocols deprive participants of sleep for periods that exceed 24 hours. Whilst these studies have demonstrated reductions in repeated sprint performance [218] and power [219], a complete lack of sleep is not the common reality faced by the majority of athletes [20].

Further studies have investigated the effect of sleep restriction (defined here as a reduction of total sleep time) on performance which, arguably, has more ecological validity. Eleven healthy participants completed force-velocity and Wingate tests after a normal night's sleep (control), and after two sleep restriction protocols that either delayed sleep onset by 4 hours or woke participants 4 hours early [219]. Both sleep restriction protocols significantly affected anaerobic performance, indicating that sleep restriction impairs function. In a similar research design, 12 male Judo athletes, competing in national championships, completed a handgrip strength test, maximal voluntary contraction of the elbow flexors, and a Wingate test before and after sleep restriction [220]. Morning re-tests yielded no significant differences. However, when participants were re-assessed in the evening, lower limb power output was significantly impaired in participants that awoke 4 hours early. A similar effect was also observed in 10 nationally competitive male taekwondo athletes, where 4 hours of morning sleep restriction similarly impaired anaerobic performance [221]. Alongside sleep restrictions, results may be influenced by chronotype disruption. Whilst this is conjecture, all participants from these studies [219–221] were in their late adolescence (>20 years), therefore, their chronotypes are likely to be approaching peak lateness [165]. Consequently, they may better withstand later bedtimes compared with earlier wake times. Nevertheless, these studies demonstrate that sleep disruption has the potential to disrupt physiological measures of exercise performance.

Although arguably more ecologically valid than total sleep deprivation, 4 hours of sleep restriction, as used in the aforementioned studies [219–221], may only be experienced by professional football players in specific situations, for example, during and after travel [225–227]. However, this is unlikely to be the norm. Nevertheless, studies have also observed significant performance decrements in participants that have been subjected to more modest levels of sleep restriction [217]. For example, in professional

rugby athletes, players who received <6hrs of sleep the night before performed significantly less total (self-selected) load across bench press, squat and bent over row exercises compared to players who received >8hrs sleep [228].

Not all studies agree with the results discussed thus far. Blumert et al. [229] exposed national-calibre male collegiate weightlifters to a maximal weightlifting protocol after 24 hours of total sleep deprivation and observed no significant difference compared to a normal night's sleep. Similarly, athletes (undefined) who had their bedtime delayed until 3 am (with a consistent wake time) experienced no significant decline in peak power, mean power output, and peak velocity compared to a reference night where participants followed their normal routine [230]. The reason for the disparity between studies is not clear. However, the effects of sleep loss are a highly variable phenomenon. Notwithstanding the interindividual differences in the physiological and cognitive responses to sleep loss in the general population [231], studies have also reported more prominent intraindividual variation in sleep efficiency and onset latency in professional footballers, as well as wider athletic populations [20], compared to age-matched non-athletic controls [46]. Considering both the variation in how professional players sleep, and the response to sleep loss, it may be more logical to prescribe individualised intervention to athletes reporting sleep loss, compared to more wholesale, team-based interventions [232].

Nevertheless, there is sufficient evidence to suggest that sleep loss disadvantages performance [16,217,233]. One review of studies investigating the effect of loss on resistance exercise performance concluded that inadequate sleep could impair maximal strength, notably in the absence of motivational strategies during exercise performance [233]. Another review suggested that aerobic performance may be more sensitive to sleep loss, compared to anaerobic performance [217]; citing studies observing reductions in yo-yo intermittent recovery test performance after just 4 hours of sleep restriction [221].

Alongside studies demonstrating that sleep restriction/deprivation can negatively impact performance [16,217,233], there are also compelling data suggesting that sleep extension can positively affect performance [221]. Sleep extension involves implementing a strategy that directly increases sleep duration, normally by mandating a waketime and sleep time or by setting sleep duration targets [221]. In varsity tennis players [234], significant improvements in daytime sleepiness occurred in tandem with enhanced serve accuracy when athletes slept for 9hrs, compared to when participants slept for less than 7hrs. Furthermore, a mean sleep duration increase of 110.9 ± 79.7 min was significantly associated with improved sprint times in colligate level basketball [235]. Similar improvements have also been observed in college-level swimming performance after a sleep extension intervention. Indeed, where sleep extension has been applied, studies have observed significant improvements in elements of wellbeing, technical performance or physiological performance [234–236]. However, the primary demographic studied are varsity level athletes with no studies on full-time professional athletes [234–236]. Further,

where extensions have been applied, the increase in sleep duration has exceeded 60 mins [234–236], with some studies mandating up to 2 hours of sleep extension [235]. Consequently, considering professional football players scheduling, training, playing and travel commitments [96,100], the magnitude of sleep extension necessary to mediate performance improvements may be unfeasible. Nevertheless, the fact that sleep extension can potentially improve performance highlights its link with sleep.

Whilst the influence of sleep loss and/or extension has been well investigated, the vast majority of the literature investigates the effect of acute sleep manipulation on performance [16,217,233], therefore, there is little information on the effect of longer-term sleep loss on athletic performance or recovery [217,233]. What is understood is that sleep duration may be related to all-cause mortality. One meta-analysis suggested that sleep duration has a U-shaped relationship with cardio-vascular events, with both habitual short and long sleep duration associated with an increased risk of all-cause mortality [237], and a more recent large prospective cohort study with follow-up (n=380k) revealed significant associations between consistent poor sleep and all-cause mortality [238]. Nevertheless, Research investigating the effect of persistently reduced or suboptimal sleep on physiological recovery and/or performance is lacking. However, there is a growing body of evidence linking chronic sleep quality and injury risk [239].

1.2.5. Sleep and injury risk

Injuries impose substantial tolls on both professional football players and their clubs [240,241]. Injury prevalence in football is higher than in many other team sports [241] with some research suggesting that a typical squad of 25 players may sustain approximately 50 injuries per season [240]. Subsequent research has also linked the time loss through injury to overall league position. Specifically, a significant correlation ($p=0.001$, $r = -0.44$) was observed between the time spent injured during the season and the place difference between their predicted (according to player value) and actual final league positions [242]. Furthermore, the analysis suggested that for every 136 days lost to injury (across the team) equated to 1 league point, and every 271 days lost equated to 1 league place. Notwithstanding the money spent on wages while a player is injured, points and league positions lost to injury represent a major financial liability to professional football teams [242]. Whilst there is a multitude of factors and confounders related to injury onset and severity [33], there is a growing body of data that suggests sleep quantity and quality may be associated with injury onset; although, the causative mechanisms are unknown [239].

In a follow-up survey conducted on adolescents aged 15 to 19 (n= 1773), insufficient sleep was associated with the prevalence of low back pain 2 years later [243]. These results have also been expanded upon in longitudinally designed studies using student-athletes. Milewski et al [244] monitored

112 students (mean age: 15.2 ± 1.5yrs) across multiple sports over 21 months, recording a total of 250 injuries. Although analysis revealed that the number of hours of sleep per night (relative risk: 0.8, p= 0.006) and strength training (relative risk: 2.0, p= 0.01) independently predicted injury onset, the strongest predictor was receiving <8 hours of sleep (relative risk: 2.1, p= 0.01). Similarly, when 496 adolescent athletes were longitudinally monitored over 52 weeks as part of a larger athlete screening project [245], increases in load and intensity occurring in tandem with decreases in total sleep volume were significantly associated with increased injury risk. These results have also been replicated in adult endurance athletes (n= 95, mean age: 42 ± 10 yrs) where analysis suggested that a mean sleep quantity of <7 hours over 14 days significantly predicted new injury risk, although training load was not observed to be related to injury onset [246]. These studies consistently link suboptimal sleep volume with increased injury risk, however, the knowledge base as a whole is limited by a lack of data from elite or professional adult football athletes. Furthermore, subjective sleep diaries, or sleep recall methods, have been used to assess sleep. Consequently, results maybe be confounded by sleep overestimation and other potential biases [247–249].

Whilst studies investigating the relationship between objectively assessed sleep quality and quantity in professional footballers are scarce, what is available supports what has previously been discussed [244–246]. In a prospective cohort of 23 elite football players competing at the highest level in Brazil, Silva et al. [48] used wrist-accelerometry to objectively monitor sleep over 10 days. Injury rate, injury severity, and time loss to injury were then collated over the ensuing 6-month period to determine any relationship between the sleep data and later injury occurrence. Results revealed that sleep efficiency (R^2=0.44) and WASO (^{R2}= 0.30) accounted for 44% and 30% of the total variance in the total number of injuries sustained. It is not surprising that both WASO (time spent awake after sleep onset) and sleep efficiency (per cent of time spent asleep in bed not sleeping) presented with similar relationships, considering the interrelated nature of the two variables. Sleep efficiency further accounted for 24% (R^2=0.24) and 47% (R^2=0.47) of the variation in time lost to injury and injury severity, respectively, reaffirming a probable link between sleep and athletic injury.

This study is not without its limitations. Primarily, its analysis links a relatively short period of sleep monitoring with a longer injury monitoring with no simultaneous observation of both sleep and injury [48]. Consequently, sleep and injury risk cannot be causatively associated due to unaccounted common confounders associated with both sleep and subsequent injury, unless it is speculatively assumed that sleep remained constant over the 6 month injury monitoring period. Therefore, results should be interpreted appropriately. In footballers, sleep and injury rates have been monitored simultaneously elsewhere, albeit only in case study form [250]. In a 31 year old professional fullback playing at the highest level in France, researchers objectively (wrist-accelerometry) and subjectively (sleep diary) monitored sleep during a preseason baseline period and then continuously across a period that contained

15 competitive fixtures [250]. During this period, 3 injuries (moderate groin tear, moderate hamstring strain, major ankle sprain) were sustained, equating to a total of 23 days of time loss. Analysis indicated that sleep metrics were altered during the 7-day period and the night before injury occurrence. During baseline, sleep efficiency was reported as 90 ± 3% whereas sleep efficiencies of 74%, 66%, and 79% were reported the night before each injury, figures that fall below what is considered normal (85%) [251]. This supports the findings of previous studies suggesting that a reduction in sleep efficiency is related [48]. Moreover, compared to baseline (18 ± 13mins) substantial sleep latencies of 118mins, 159mins, and 73mins, respectively, were also reported on the night before injury occurrence. This further supports a relationship between the player's sleep and injury occurrence. The baseline was measured over 5 days, and no in-season measures were presented. Consequently, it is not known if sleep on nights preceding injury was different compared to nights preceding no injury. Nevertheless, the case study does support a link between sleep and injury, and it is clear that further investigation is warranted.

1.2.6. Sleep and anabolic signalling pathways

Mechanistically, the pathways that describe the role of sleep in athletic recovery, performance, and injury require elucidation [252]. However, what is understood is that sleep has an encompassing role in several hormonal, regulatory, and cerebral homeostatic processors that are heavily implicated in athletic recovery [14]. Dattilo et al [14] proposed that periods of suboptimal sleep quantity or quality can restrict muscle recovery by limiting anabolic and catabolic endocrine systems, specifically, the release of human growth hormone (GH), insulin-like-growth factors (IGF), testosterone, and cortisol that are known to be significantly mediated by specific sleep stages [253,254].

It is long established that the hypothalamic-pituitary facilitated release of human growth hormone (GH), an anabolic substrate [255,256], increases during sleep, in a sleep-stage dependant manner [253]. During one study, sleep architecture was recorded in 8 participants while blood was drawn every 30mins and sampled for human GH. Results demonstrated a clear relationship with NREM sleep with an approximate increase of 27 μg/ml GH in blood that coincided with the first phase of slow-wave sleep. When sleep was delayed by 3 hours, the spike in GH was similarly delayed until the first NREM phase, indicating that the secretion is SWS dependant, and not circadian [253]. Human GH is also released in a pulsatile manner across the 24-hour cycle in response to exercise, blood sugar levels, and protein ingestion [257,258] (Figure 8). However, subsequent studies demonstrated that over 95% of GH is secreted during NREM sleep [257–259].

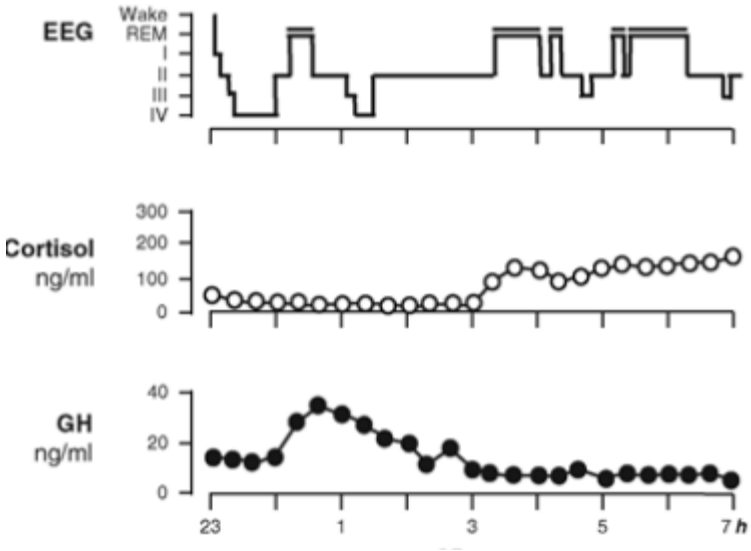

Figure 8: Release of Cortisol and GH (Growth Hormone) by sleep stage as measured by EEG [189]

In response to GH stimulation, hepatic-originated IGFs are produced and enter circulation [255,256]. IGFs have a range of metabolic, mitogenic, and anabolic cellular responses [260] and their effects include satellite cell activation, proliferation, survival, and differentiation, myotube plasticity, regulation of protein synthesis, muscle hypertrophy, and neuronal myelinisation [261–263]. Accordingly, the intrinsic relationship between sleep, the hypothalamic-pituitary axis, and the anabolic signalling that is essential for EIMD and repair is self-evident. However, IGFs are also involved in an intricate feedback mechanism whereby it inhibits GH gene expressions and actively stimulates the secretion of somatostatin, a peptide that acts as an antagonist to GH-releasing hormone (an upstream activator of human GH release) [264]. In humans, exogenous supplementation of GH-releasing hormone can stimulate NREM sleep [265], which implicates the GH-IGF-somatostatin pathway as a self-limiting system. To be precise, the release of GH is associated with NREM sleep, but the down steam products of GH limit substances that can initiate NREM sleep. Therefore, the focus may be better placed on ensuring athletes receive optimal slow-wave sleep, rather than attempting to enforce a sleep duration that is above what is normally expected.

Further to GH release, in males, the hypothalamus and pituitary are implicated in the nocturnal production of testosterone, a major anabolic hormone, from the testis [14,266,267]. Secondary to the release of gonadotropin-releasing hormone (GnRH) from the hypothalamus, the pituitary secrets luteinizing hormone (LH) [266,267], which, in turn, acts on the testis to produce testosterone. Like GH and IGFs, this system has an intricate feedback mechanism, where testosterone metabolites inhibit GnRH and LH production [268].

Like GH, testosterone kinetics have been intrinsically linked to specific sleep stages. Lubroshitzky et al [254] monitored sleep architecture while determining nocturnal LH and testosterone levels in 6

healthy participants. Results showed a clear increase in testosterone and LH levels on sleep initiation, before peaking at the first bout of REM sleep. Testosterone levels then remained at that level until waking. Subsequent studies have confirmed the relationship between testosterone and the first REM sleep [269,270]. One study used a sleep fragmentation protocol of 7 mins sleep 13 min awake, repeated 72 times over 24 hrs, that prevented REM sleep and observed a notably reduced testosterone curve [226]. Likewise, in another study, results indicated that testosterone levels still peaked to coincide with the first REM phase despite bedtime being delayed by 8 hours (from 2300 to 0700), compared to a control [270]. Both studies [269,270] observed small evening-time increases in testosterone regardless of intervention, suggesting that its secretion is partly circadian, nevertheless, results demonstrated that testosterone is a sleep-mediated hormone. Considering the role of testosterone as a major anabolic endocrine, this, in turn, provides a logical mechanistic link between sleep and athletic performance [14].

It is likely that testosterone kinetics are similar in adolescent males compared to adults, however, much of data thus draw is drawn from adult populations [269,270]. Nevertheless, the onset of puberty in males is associated with an approximate 26-fold increase in testosterone levels which drives anabolic processors during maturation. As discussed, adolescence is also associated with changes in sleep architecture and a chronotype that favours later sleep onsets and wake times compared to other age groups [165]. It is not clear if there is a mechanistic link between changes in testosterone kinetics and in adolescent populations [271]. However, the link between sleep and testosterone is clear, and considering the importance of testosterone in adolescent maturation, practitioners may wish to ensure optimal sleep is achieved in their academy players.

1.2.7. Sleep and barriers to sleep in professional football players

It has not been established what the minimum quality or quantity of sleep is required by elite football players, nor in the wider athletic community [131]. Most studies are designed to compare sleep quality on game days to a '*typical*' training day, which acts as the control. This is justified as the day type most removed from competition and travel, and as the most numerous day type [272–277]. However, this cannot provide a robust measure in which to centre results due to continued potential stressors that may affect sleep in professional footballers. Therefore, it is challenging to make assumptions about the day-to-day sleep quality of footballers. However, athletes, in general, present with sub-optimal sleep patterns [20]. Using wrist-accelerometry, Leeder et al. [20] studied sleep hygiene in 46 athletes (canoeing n=11, diving n=14, rowing n=10 and speed skating n=11) and compared the data to 20 healthy, non-athletic aged-matched controls. Sleep was monitored for 4 nights during an out-of-season training phase with periods involving long-haul flights excluded. Compared to the control group, athletes had greater sleep latency (time to sleep onset), time in bed, time awake, restlessness/fragmentation and reduced sleep efficiency despite no statistically significant differences in total sleep time. The cause of the variation is likely multifaceted, nevertheless, this demonstrates, that

whilst athletes might receive the recommended amount of sleep, total quality may be poorer. Research suggests that this may hold in professional footballers. Academy players representing professional clubs presented with poorer sleep efficiency compared to age-matched controls, that were selected from a local university, after a 6-day monitoring period [46]. Results also suggested the footballers had more variable sleep latency and sleep efficiency after standard deviations were statistically compared between groups. These observations are also broadly comparable with latter studies in profession female footballing cohorts [278]. The cause of this is not entirely understood, nevertheless, it is likely multifaceted. The purpose of this section is to review the potential factors that may affect sleep in professional football players.

1.2.7.1. Sleep quality and variability in professional players

Cross-sectional studies utilising subjective questionnaires also highlight suboptimal sleep. One investigation conducted on footballers competing in Qatar used multiple subjective assessments to measure sleep quality (PSQI), clinically relevant insomnia (ISI) and daytime sleepiness (ESS). The results were telling. Of the 111 footballers assessed, 76 breached the PSQI threshold for poor sleep quality, whilst 30 presented with clinical insomnia, and 25 reported excessive daytime sleepiness [178]. Other studies suggest that suboptimal sleep is not as widespread. In footballers playing in The Netherlands top tier competition, one investigation recorded values of 3.6 ± 2.42 in the PSQI [279]. Whilst participants were categorised as having adequate sleep by this methodology, the standard deviation suggests players approached the threshold for clinically sub-optimal sleep. This study did not use further sleep assessments; therefore, it is not certain if players are presenting with excessive sleepiness or insomnia. It is also important to note that environmental and cultural factors between athletes competing in Qatar [206] and Europe [279] might impact sleep.

Sleep in professional footballers may present with greater variability than in aged-matched non-athletic controls [46,278]. This is discussed in greater detail in later sections; however, one source of inter-individual variation may be the player's respective chronotypes. Although some research suggests that football player's chronotype distribute (e.g., morning, intermediate, evening) is not significantly different from age-matched controls [280], chronotype approaches peak lateness during late adolescence, before a gradual decline [165]. Consequently, how payers entrain to the 24-hour cycles may change as the player continues through their career.

1.2.7.2. Night-time and evening matches

Evening and night-time fixtures are commonplace in elite football [131,274,281] and involve playing competitive fixtures in stadia that are equipped with floodlit illumination equivalent to ≈2000 lux [131]. Fullagar et al. [274] examined sleep after night-time fixtures, daytime fixtures and on training days in

16 football players competing in the topflight German and Dutch leagues. Results suggested no significant difference between training and daytime match days, however, after night matches total sleep quantity was reduced by approximately 200 minutes. Players also reported later bedtimes and wake times in addition to increased sleep latency compared to training days. Post-game logistical factors and media commitments are highly likely to be a factor in the later bedtime, whilst greater sleep latency can be explained by increased exercise and environmental arousal reducing parasympathetic outflow [282], the results of the fixture may also exacerbate psychological barriers to sleep onset. Players also subjectively rated significantly less restful sleep after night games [274]. Players reported adequate sleep on training days and after daytime fixtures, however, it is important to note that players might be accustomed to sub-optimal sleep patterns, therefore report what is relative to them. Thus, actual sleep quality might be suboptimal and subjective reporting is not sensitive enough to report this.

Physical activity close to habitual bedtime during night games might also affect sleep quality. However, research is equivocal regarding the effect of evening physical activity with some studies showing no or a beneficial effect on sleep [276,283], and others recording negative effects [284,285]. One suggestion is that the intensity of exercise close to bedtime dictates the magnitude of sleep disruption. Oda and Shirakawa [286] subjected healthy participants to exercise between 2120 and 2200 at a heart rate reserve of 80%, 60%, or a rested control. Their analysis demonstrated increased bedtime arousal and sleep latency (+14 minutes) when participants exercised at 80% of heart rate reserve, compared to other conditions. Further, heart rate at bedtime was significantly increased and reduced high-frequency heart rate variability, suggesting inhibited parasympathetic nervous output. Whilst it may be intuitive to suggest that exercise should aid in sleep, it has also been suggested that prolonged high-intensity activity might increase sympathetic nervous tone and/or blunt parasympathetic drive, antagonising sleep [282]. As discussed, footballers cover considerable distances and undergo a substantial number of high-intensity actions, compared to other sports [12]. The result is a notable onset of EIMD symptoms and homeostatic disruption [24]. In tandem with stadium lights, noise and emotional factors close to bedtime [131], a state of arousal that affects a footballer's ability to sleep and recover is to be expected.

The physical activity associated with match play will also induce changes in core temperature [287], potentially antagonising the circadian temperature cascade that accompanies sleep initiation [167]. Moreover, competition-induced DOMS, secondary to significant EIMD, might impact restfulness during sleep [288]. Furthermore, studies have highlighted that elite footballers are habitual caffeine consumers, consuming the stimulant for both pleasure and as an ergonomic aid [289,290]. Caffeine is well known to non-selectively antagonise adenosine receptors in the brain [291], in turn, disrupting sleep homeostasis and arousal regulation [292]. One longitudinal study typically shows that caffeine consumption reduced the length of slow-wave sleep and increases the time spent in NREM stage 1, the number of awakenings, and sleep latency [291].

Further to the factors already presented, night-matches involve playing competitive fixtures in stadia that are equipped with floodlit illumination equivalent to ≈2000 lux [131]. Light exposure can inhibit melatonin production, reducing the circadian signals (Process C) that initiate sleep [293]. Previous work has stated that just 1000 lux or more is sufficient to affect sleep [294], therefore, it is likely that the intensity of the light can disrupt the circadian initiation of sleep (process C). Whilst the lux level during night matches may be considerable, players may still be exposed to increased levels of light away from night games, which in turn may affect sleep. One study suggested that approximately 80% of players surveyed use electronic devices or watch television before bed which likely interrupts circadian melatonin production [131,168]. In a cross-sectional study (n=9846), the frequency of electronic device use was revealed to be significantly negatively correlated with sleep duration in non-athletic adolescent teenagers [168], and investigations have observed improved sleep latency in interventions that have limited electronic device use [169]. It is not known if electronic device use is notably greater in football players compared to the general population. Regardless, if a player uses electronic devices close to bedtime, then it is not unreasonable to assume sleep disruption follows.

1.2.7.3. Travel

The effect of travel has also been observed to be a meaningful and statistically significant sleep disruptor in professional players. One study analysed the effect of short-haul domestic travel on professional football players in Australia, over 12 matches (6 home, 6 away). The results were largely equivocal, most likely due to low study power (n=6), however, there was evidence of increased sleep latency after away matches, potentially resulting in a disrupted routine during periods of travel [18]. In another, better powered, study [225], 15 elite male footballers were observed as they engaged in 18 hours of international air travel. Over the investigative period, sleep duration and efficiency were reduced significantly compared to baseline on travel days and after matches, with no additional effect on sleep on training days. This suggests that sleep disruption during travel is limited to the actual travel day and can likely be attributed to logistics and arousal of travel.

1.2.7.4. Circadian misalignment

A player's intrinsic chronotype coupled with, travel demands, and inconsistent schedules [96,131] may also give rise to a phenomenon known as circadian misalignment, also colloquially termed social-jet lag [295]. Specific to footballers, this may occur when player's schedules (e.g., night games, or days off) dictate playing or training commitments that interfere with their normal sleep behaviour. One consequence of this may manifest altered and suboptimal sleep behaviour during the nights following the initial event. The prevalence of social jet lag has not been investigated in professional footballers, nor has its potential effect on subsequent performance. However, social jet lag has been proposed as a factor that may influence sleep in adolescent players [46]. In a study that investigated sleep across a

microcycle, investigators noted reduced sleep duration on matchday+1 (MD+1), compared to other days. Considering that MD+1 was a recovery day, which allowed players substantially more time to socialise, the authors suggested that subsequent changes in sleep may be due to circadian misalignment and social jet lag. Whilst this cannot be proven, other studies have suggested prevalent social jetlag amongst adolescents [296] and adults [297]. Therefore, it can be reasonable surmised that professional footballers may encounter circadian misalignment in light of inconsistent scheduling and night matches.

As chronotype reaches peak lateness during late adolescence, before falling throughout a person's 20s, 30s, and beyond. This may mean that circadian misalignment may manifest itself differently throughout a player's career. For example, if travel is scheduled during the evening, this may influence the sleep of someone whose chronotype preferences an earlier bedtime, compared to someone who preferences a later one. The subsequent effect on sleep, and sleepiness across subsequent days, may differ. This may also influence the optimal scheduling for individual players across their playing schedules.

1.2.7.5. External workload

In athletes, several studies have suggested links between workload and subsequent sleep metrics [47], consequently, a player's workload may be a factor influencing the amount of sleep that they achieve. However, the data are equivocal. For example, in profession rugby league players that were monitored during preseason, the number of acceleration/decelerations demonstrated a significant and positive relation relationship with objectively derived sleep efficiency. Although the effect size was small (effect size= 0.15), this suggests that players who engaged in more changes in velocity experienced improved sleep. The cause of this relationship remains unknown, although authors suggested a perceptual response associated with a perceived sense of effort [47]. Contrastingly, in trained endurance athletes who were monitored before and during an intensified training period, the analysis suggested that increased workload was associated with reduced sleep duration and efficiency [298]. Notably, participants demonstrated a progressive decline in sleep efficiency and sleep duration over the 3-week overreaching training block which may be related to the accumulation of mild muscular fatigue, although, causation at not be inferred based on the data available [298].

In football players, a meaningful relationship between external workload and sleep metrics is yet to be established. In senior English Premier League players, 1, 2, 3, and 4-day accumulated high-intensity running (classified as total distance accumulated at speeds greater than $4 m \cdot s^{-1}$) were not found to be significantly associated with perceived sleep quality [44,45], suggesting objective measures of workload are not associated with subjective measures of sleep. However, in professional youth players, Whitworth-Turner et al [275] reported a significant relationship between total high-speed running distance (distance accumulated at speeds greater than $5.5 m \cdot s^{-1}$) and subsequent objectively derived WASO, time in bed, and sleep duration sleep metrics. While differences in how the workload was

classified, and how sleep was measured, may account for discrepancies between studies, Whitworth-Turner et al [275] still reported only trivial increases in WASO, time in bed, and sleep duration per every 100m increase in high-speed running distance. Whilst this data cannot rule out a substantial relationship between sleep and external workload, it does suggest that the magnitude of potential sleep disruption in response to workload may not be sufficient to concern practitioners and coaches. Nevertheless, polysomnography investigations would be better placed to confirm this. Furthermore, data collected across different macro cycles may also better elucidate any potential relationships.

1.2.8. Methods to improve sleep in football players

There is a plethora of novel and more traditional strategies available to improve sleep quality in footballers, athletes and the general population [216]. These range from sleep extension [234] and sleep hygiene strategies [169] to more indirect methods, like whole-body cryotherapy [299] and showers before bedtime [300]. Interestingly, many of these strategies revolve around countering the disruption to the two-process model or augmenting it.

1.2.8.1. Sleep extension through scheduling

Sleep extension in non-athletes is well investigated and involves tasking participants to reach a target total sleep duration, or time in bed, that is greater than what is normally experienced [216]. This is normally applied in research settings, however, research in athletic populations is limited and there are a scarcity of data on footballers. In varsity tennis players [234], improvements in daytime sleepiness occurred in tandem with serve accuracy after participants were asked to extend sleep to 9 hours per night. In collegiate basketball players, sprint times significantly increased after a mean sleep duration increase of 110.9 ± 79.7 min [235]. Similarly, PVT scores have improved in military personnel after they have undergone sleep extension [301]. Nevertheless, sleep extension interventions may not be applicable in professional sporting environments. In cases where sleep extension has resulted in a significant performance benefit [234,235], sleep extension durations >90 mins have been utilised. Consequently, implementing sleep extension strategies of a similar magnitude is likely to be incompatible with the training, scheduling, and family commitments that professional footballers may face. However, a form of sleep extension may be achieved through the manipulation of the scheduling variables that coaches have a substantial element of control over, for example, start time (the time players are scheduled to arrive for training or competition). This could be particularly pertinent for academy professional players whose biological chronotype (the intrinsic entrainment of an individual's circadian system to a 24-hour cycle) is expected to be later compared to senior players [165]. Biological chronotype varies across the lifespan, peaking during late adolescence [165]. Consequently, it follows that sleep scheduling considerations for professionals in their late teens or early 20s.

In adolescents in the USA, a regression analysis from a cross-sectional survey of 2454 students (age: 12 to 19yrs) demonstrated that for every 1-hour extension in start time, sleep duration increased by 34.8 mins [255]. Likewise, using data from the American Time Use Survey [303], researchers observed a 25.2 min extension to sleep duration per 1-hour increase to start time. These results have been replicated in subsequent studies [173], and other investigations have reported further benefits when start time has been extended in American students. For example, in a retrospective analysis, Borloase et al. [174] found that ESS scores were improved when start time was extended from 09:00 am to 10:30 am, most likely as a result of an increased window in which to sleep. Further analysis also suggests that a later start time is significantly associated with a reduction in motor vehicle accidents in adolescent drivers in the USA [175], and there is growing evidence suggesting that later start times are beneficial to this age group [175].

Extending start time in professional footballers, notably in adolescent academy players whose chronotype better suits later start times, may represent an indirect method of applying a sleep extension strategy. The commitments of academy players representing a professional may differ from the general population, and it is not known whether the more levels of sleep extensions associated with later start times will manifest in a tangible performance benefit, such as is observed when >90mins of sleep extension has been applied in varsity athletes [234,235]. Nevertheless, considering it is a low-tech and practically cost-negligible intervention, it is worth investigating.

1.2.8.2. Sleep hygiene

Sleep hygiene strategies were initially developed for the treatment of moderate insomnia and are defined as a set of behavioural and environmental initiatives intended to promote healthy sleep [304]. In a review of sleep hygiene, Irish et al. [304] concluded that many strategies are supported by plausible physiological or psychological mechanisms, however, research around their actual efficacy is limited by vague, inconsistent recommendations and limited guidance. The authors also highlighted that research is focused on acute effects in laboratory settings. Nevertheless, in 98 national representative youth athletes (mean age: 18 ± 3yrs), significant correlations were observed between sleep hygiene and PSQI scores ($r = 0.45$, $p<0.001$) [305] and sleep hygiene education has been successful in improving sleep metrics in national representative netball players [261].

Sleep hygiene strategies aimed at reducing evening light exposure, caffeine intake, and alcohol consumption have also been suggested for football players [281]. Experimentally, some of these strategies are efficacious in football players [169]. After two-night games, Fullager et al. [169] placed highly-trained amateur players in a dimly lit bedroom and prohibited electronic device use 15 to 30 minutes before bedtime. Compared to the control (players are free to make their own decisions), results

revealed significantly greater sleep duration and fewer wake episodes. The improvements can be attributed to the reduction in artificial illumination levels (relative to the control), preserving Process C

1.2.8.3. Thermoregulation: the 'warm bath effect'

There have been attempts to improve sleep quality in the general population, particularly as a treatment for insomnia, by augmenting the circadian thermoregulatory process in the lead-up to, and during sleep, taking advantage of the so-called 'warm bath effect' [307]. The overarching physiological mechanism that underpins this phenomenon was introduced in section 2.1. Physiological mechanisms regulating the sleep-wake cycle. In brief, using a theromosuit to apply heat to proximal and distal sections of the body to induce vasodilation, Raymann et al [183] were able to reduce sleep onset latency by 3.09 min (95% CI: 1.91 to 4.28). Subsequent application of the theromosuit during sleep increased the time spent in slow-wave sleep at the expense of wakefulness and lighter NREM sleep [191]. Whilst the additional slow-wave sleep may support the athletic recovery process, donning a full-body suit, similar in appearance to a wet suit, may be impractical if used each night and is likely best suited for research, i.e., to elucidate the mechanistic physiology. Water-based passive cooling (e.g., hot/warm bath or shower) is a far more common and tolerable method to induce vasodilation close to bedtime, in an effort to improve sleep metrics. One meta-analysis pooled the results of 13 studies (median n= 13) that assessed the effect of water-based passive cooling before bed on subsequent sleep [307]. Analysis revealed a trend that suggested a shower or bath 1 to 2 hours before bed improved sleep latency, sleep duration, quantity of slow-wave sleep, and sleep efficiency; yet only sleep latency and sleep efficiency demonstrated significance. Sleep latency presented with the largest effect size ($Z= 2.58$; $p=0.01$) with reports indicating an average 8.6min reduction in the time taken to fall asleep.

One study has also observed significant benefits in football players when water-based passive cooling has been applied before sleep. In 11 professional (full time, contracted) academy football players (mean age: 18 ± 1yrs), Whitworth-Turner et al. [300] applied a warm shower 20 minutes before bedtime and, compared to the control condition (no shower), sleep latency was significantly reduced from 24 ± 15mins to 17 ± 15mins. Sleep efficiency was also significantly improved (control: 94 ± 3%, shower 96 ± 3%) in a trend that is observed elsewhere in the literature [307]. This suggests that a hot/warm bath or shower may be a suitable intervention to improve sleep onset latency and efficiency in football players when implemented as part of a sleep hygiene strategy.

1.2.8.4. Thermoregulation: application of cold

Perhaps counter-intuitively considering the warm bath phenomenon, cold immersion has also been investigated as a potential sleep aid. The effects of several cooling methods have been investigated,

with varying effects [163,299,308,309] and, where they have been effective, the underlying mechanistic response is unclear. As a consequence, it is challenging to optimise specific methodologies.

As previously highlighted the application of cold therapies is increasingly being used as a recovery modality within professional sport, and there have been several, but inconsistent, reports of improved sleep after its use [163,299,308,309]. Most notably, the application of WBC, subjecting athletes to extremely cold air (-110°C to -160°C) for short periods (120 to 240 secs) while wearing minimal clothing (slippers, socks, shorts, gloves, hat and face mask), has been investigated as am ergonomic sleep aid. After evening exercise, Douzi et al. [299] reported fewer nocturnal movements in healthy participants who had received post-exercise WBC. Likewise, sleep disruption associated with increased training intensity was attenuated by WBC in Olympic synchronised swimmers [308]. Further, in academy players representing a professional club, increased testosterone was observed in players who received post-exercise WBC. The investigators suggested that this may be a result of improved sleep, however, sleep was not monitored as part of this study [310], so this cannot be confirmed. Contrastingly, no benefit was observed in professional rugby players who received WBC after a competitive B team fixture compared to a control (no intervention) or compared to when participants slept on a high-heat capacity mattress; designed to support conductive heat transfer from the body [309]. Similarly, when highly-trained cyclists engaged in a 4-week high-intensity cycling intervention (3 sessions per week) over 4 weeks, post-exercise WBC failed to significantly affect objective sleep quality [163].

The reasons for the disparities between studies are not clear. Both single and multiple exposures reported significant [299,308] and non-significant results [163,309], suggesting a dose-response is not apparent. Other factors might include the timing of WBC relative to bedtime, confounders from applied studies, or the exposure temperature. However, in the studies available, there is no clear pattern to confirm or reject these hypothesises [163,299,308,309]. This indicates that further investigation is required to discover key variables that instigate a beneficial response.

The use of WBC as an ergonomic sleep aid in football is under-investigated. However, one study utilising PBC, a similar modality to WBC where the head is not directly exposed, did report a significant response in 9 professional footballers competing in the French second-tier [311]. Players randomly engaged in 4 protocols, a control (no PBC), a 180-second exposure, a 90-second exposure, and two 90 seconds exposures separated by 5 minutes at room temperature. The reporting of the wrist-accelerometry data was atypical, with authors electing to report the amount of movement through the x, y, and z-axis rather than the predicted sleep variables. Nevertheless, results indicated significantly less nocturnal movement after the 180-second exposure protocol suggesting players may have slept better.

How WBC might support sleep is also not clear, and it is interesting to note that a similar relationship between sleep and CWI has not been identified [312]. Several potential underlying mechanisms have been discussed, including thermoregulatory or inflammatory pathways. However, the most compelling evidence suggests that sleep maybe supported through parasympathetic activation, secondary to WBC exposure. In 25 healthy males, a 3-minute (-120°C) WBC exposure induced a reduction in heart rate and significantly increased HRV metrics associated with increased parasympathetic afference [313]. These results have been replicated elsewhere [314,315] with further studies noting a stronger parasympathetic output after WBC compared to PBC [314]. Hausswirth et al. [314] also observed a greater reduction in skin temperature after WBC, compared to PBC, which occurred with a greater release of noradrenaline; the catecholamine responsible for cold-induced vasoconstriction response [272]. Consequently, WBC may increase parasympathetic outflow by baroceptor stimulation secondary to increased central blood volume after cold-induced vasoconstriction [313][314,315], alongside trigeminal nerve stimulation [119,317,318]. The antinomic response to WBC may then support the circadian increase in parasympathetic afference associated with sleep onset [139].

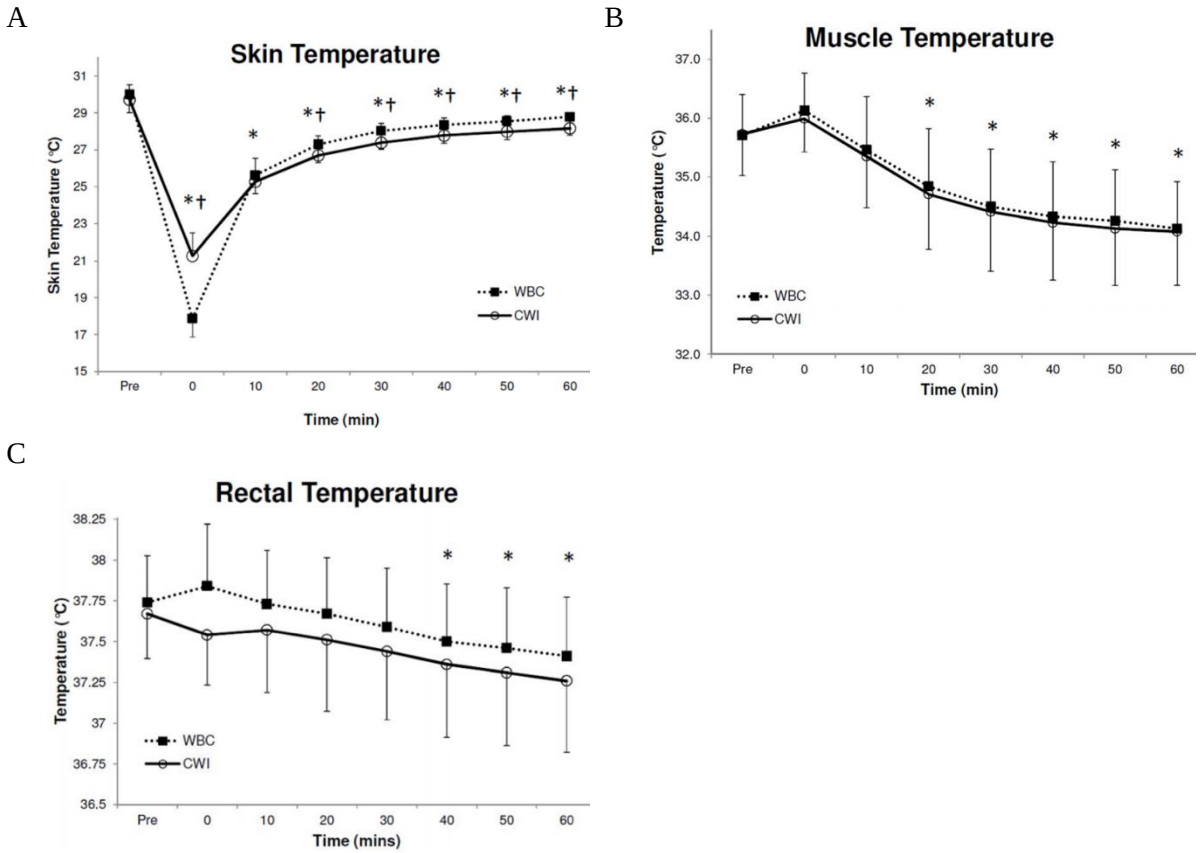

Figure 9: Skin temperature (A), Muscle temperature (B) and Rectal temperature (C) before and after whole-body cryotherapy (WBC) and cold water immersion (CWI). *significant difference from pre. †significant difference between conditions. Taken from [319].

WBC may also benefit sleep through the reduction of core temperature, mimicking the circadian drop that is also associated with sleep onset [176–178]. WBC initially induces a small increase in core temperature as warm blood from the periphery is shunted to the core, followed by a steady decline over the next 60-minutes, following a long-observed phenomenon where core temperature and muscle temperature continues to fall after the participant has been removed from the cold stimuli (Figure 9B and C), termed thermal afterdrop [320,321]. Afterdrop was initially thought to be caused by cold venous return, secondary to vasodilation upon rewarming [278,279]. However, observational data suggests that the phenomenon is in part, or solely, a conductive mechanism that is a thermodynamic inevitability in any model where the core is warmer relative to its shell [323]. Nevertheless, the data indicate a clear reduction in core temperature. Nevertheless, this is yet to be linked to sleep in research. Previous attempts at reducing core temperature through the ingestion of ice close to bedtime have not been able to support sleep onset or depth [184]. Instead, while the core temperature was reduced, this occurred alongside vasoconstriction and increased alertness, with no modulation to sleep onset.

WBC was initially introduced into sporting settings to attenuate inflammation secondary to EIMD [115,116], and an alternative explanation as to why WBC may support sleep is its possible influence on inflammatory proteins with anti- or pro-somnogenic properties [324]. As discussed, IL1 and TNF-α are primary pro-somnogenic inflammatory proteins whose levels demonstrate a diurnal pattern and track sleep propensity [158–161]. However, they are also considered pro-inflammatory proteins [324], and practitioners may recommend WBC specifically to limit pro-inflammatory action. Pournot et al. [127] exposed 11 well-trained runners to a simulated trail run, with downhill segments designed to cause EIMD. They then received daily WBC or passive recovery for four days. Results indicated reduced IL-1 and increased IL-1ra concentrations after WBC, suggesting WBC does not support pro-somnogenic inflammatory proteins. TNF-α and IL-10 (a further somnogenic interleukin) were also sampled although there was no significant change, which is also a trend observed elsewhere [116]. In another study, Ziemann et al. [126] saw reductions in TNF-α after WBC was applied daily across a microcycle in professional tennis players during a post-competition recovery camp. Considering that IL-1 and TNF-α have observationally meaningful somnogenic actions [158–161], and it is likely that WBC reduces levels in blood, results thus far suggest that any WBC sleep benefit occurs despite its anti-inflammatory actions, rather than a direct mechanistic result.

1.3. Summary and general aims

In summary, this literature review highlights the substantial amount of workload a professional football player performs throughout a competitive fixture. Specifically, research has demonstrated that the magnitude of declarative, and other high-intensity actions, causes EIMD and physiological disruption that can take days to normalise. The EIMD subsequently initiates an inflammatory cascade that is

marked by the recruitment of neutrophils, macrophages, and inflammatory proteins [63,64]. These substances are implicated in the healing process and the transcription factors that are associated with muscle plasticity and adaptive remodelling. However, the inflammatory process also generates substances that are readily converted into ROS and RNS that can outpace anti-oxidative mechanisms and exacerbate the original muscular damage. Therefore, a large amount of investigative research has been given to methodologies that can support the inflammatory process and augment the recovery in professional football players.

Sleep is essential to this recovery process. During sleep, memories and skills are consolidated, inflammation is modulated, and anabolic substrate production is increased. Football player's sleep presents with greater variability compared to age-matched, non-athletic controls. Furthermore, professional players encounter several barriers to restful, restorative sleep as part of their normal competitive scheduling. These include the overall effect of night matches, scheduling variables, and potentially workload. Therefore, to fully support the sleep and recovery of professional football players research should investigate the barriers to sleep in professional footballers, methods to monitor good sleep in this demographic, and approaches to improving sleep in a professional footballing environment. Accordingly, the following study aims were conceptualised (Figure 1):

1. Examine the use and frequency of post-exercise WBC, compared to passive recovery, on markers of inflammation, redox, and variables related to post-exercise fatigue **(Study 1)**
2. Investigate the effects of WBC, applied across an in-season microcycle on the objective and subjective sleep quality in under 18 (U18) professional footballers, and determine the effect of WBC on game-day inflammation, testosterone, and cortisol **(Study 2)**
3. Examine what is known about sleep quality and quantity, in relation to published norms, and identify the main literature themes concerning barriers to optimal sleep in full-time, professional footballers **(Study 3)**
4. Assess the influence of scheduling and workload variables on objective sleep markers in professions football players **(Study 4)**
5. Investigate if a virtual reality eye-tracking smooth pursuit assessment is sensitive to day-to-day variation in sleep metrics, and assess if the test can detect the presence of sleep loss in a military training environment with prescribed sleep deprivation **(Study 5)**
6. Trial an individualised sleep monitoring and intervention strategy aimed at improving the subjective and objective sleep in a professional U18 football player reporting suboptimal sleep **(Study 6)**

Chapter 2

2. Post-exercise whole-body cryotherapy and recovery: a systematic review and meta-analysis (Literature part 2)

2.1. Abstract

Objective: To examine the use and frequency of post-exercise whole-body cryotherapy (WBC) on exercise recovery. ***Design:*** A meta-analysis and systematic review. Change-score data were analysed assuming a random-effects model and sub-grouped by the number of exposures. ***Data Sources:*** Web of knowledge, PubMed, MEDLINE and SPORTDiscus to May 2021. ***Eligibility criteria for selecting studies:*** post-exercise WBC in healthy participants; measured variables relating to recovery from exercise; were available in English full-text; compared WBC to a passive control; and presented data in a manner suitable for meta-analysis. If all criteria were met, but the data could not be synthesised for a meta-analysis, then the study was included in the systematic review. ***Results:*** 11 studies were identified, encompassing 139 participants (81 males, 31 females, 27 not stated, mean age 18 to 26.7 years) ranging from healthy participants to Olympic athletes. Risk of bias factors included low-powered studies, inadequate description of participants, and no randomisation. Creatine kinase (CK) activity, delayed onset muscle soreness (DOMS), muscle function, cortisol, testosterone, and interleukin-6 (IL-6) were subject to meta-analysis. Sleep, inflammation, and redox-related biomarkers were reviewed qualitatively. Only multiple WBC exposures showed a beneficial effect on CK activity, DOMS, muscle function, or cortisol. Single exposures beneficially affected testosterone, IL-1, and IL-1 receptor agonist. No effect was detected for IL-6 and the effect on sleep is unclear. ***Summary/Conclusions:*** Multiple WBC exposures are more likely to provide a beneficial effect on muscular performance, CK activity, and DOMS. Single exposures might be adequate to increase testosterone, reduce inflammation and support sleep.

2.2. Introduction

Whole-body cryotherapy (WBC) is used as a recovery aid in elite sport settings, despite limited evidence of its effectiveness [118,120,121]. It involves subjecting athletes to extremely cold air (-110°C to -160°C) for short periods (120 to 240 secs) while wearing minimal clothing (slippers, socks, shorts, gloves, hat and face mask), in specially designed chambers [118]. WBC is purported to enhance athletic recovery and alleviate symptoms of exercise-induced muscle damage (EIMD), caused by the mechanical stress placed on sarcomeres during strenuous exercise [36]. EIMD is characterised by oedematous swelling, increased intramuscular milieu in blood, delayed onset muscle soreness (DOMS), diminished muscular function, and an inflammatory response that exacerbates the initial muscular damage [36]. WBC has previously been used to attenuate inflammation in arthritic populations [325], however, its effectiveness in relieving inflammation after EIMD is less clear.

Cryotherapies are generally used to reduce tissue metabolism and induce analgesia, with some researchers proposing WBC mediates reductions in intercellular adhesion molecule-1 (ICAM-1), which in turn lessens the transmigration of inflammatory proteins to sites of muscle damage [115,116]. However, the limited number of investigations to date have reported no [116,122,123], mixed [124], or beneficial [125–127] effects of post-exercise WBC on inflammatory and wider EIMD markers, in addition to a possible effect on redox balance [326–328]. Further reports suggest that WBC might be efficacious in reactivating the parasympathetic autonomic nervous system after exercise [314,315], in turn improving recovery through sleep [163,299,308,309].

The discrepancies between studies limits the confidence in the recommendations that can be made available to practitioners. Heterogeneity in study outcomes might be explained by methodological disparities and, whilst several authors have reviewed WBC and its efficacy [118,121,329,330], a specific analysis of the effect these disparities have on outcomes via meta-analysis is incomplete. Notably, investigations differ on the number of WBC exposures applied after muscle-damaging exercise [122,123,125,126,310]. An exploratory analysis of the influence of WBC exposure frequency can highlight where future research is required and enable practitioners to better understand where potential benefits of WBC could be found, and in what time frame.

Therefore, the purpose of this investigation is to conduct a rigorous meta-analysis and systematic review, with a specific sub-group analysis on exposure frequency, investigating the use of post-exercise WBC, compared to passive recovery, on markers of EIMD, inflammation, redox, and variables related to post-exercise fatigue and recovery in healthy and athletic populations.

2.3. Methodology

This study was reported following the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement [331]. This was a systematic review and meta-analysis of published studies; therefore, ethical approval was not required.

2.3.1. Search strategy

Trials that used WBC as a therapeutic aid for recovery were identified following a search of the databases PubMed, MEDLINE, Web of Knowledge and SPORTDiscus. In conjunction with Boolean Logic commands, the search terms whole-body cryostimulation, whole-body cryotherapy, cryo* chamber AND recovery, athlete, exercise, fatigue, sleep, redox and inflammation were used. See Table 3 for the complete search strategy. Peer-reviewed academic papers from the start of records until May 2021 and their references screened for additional studies. Results were imported to reference management software (Mendeley Elsevier, Amsterdam, Netherlands) and duplicates were removed.

Table 3: Full search strategy

Database	Search terms
Web of Science	(“whole body cryostimulation” OR “whole body cryotherapy” OR “cryo* chamber”) AND (“recovery” OR “athlete” OR “exercise” OR “fatigue” OR “sleep” OR “inflam*” OR “cortisol” OR “testosterone” OR “redox” OR “oxidative stress”) 1900-01-01 2022-10-31
PubMed	(“whole body cryostimulation” OR “whole body cryotherapy” OR “cryo* chamber”) AND (“recovery” OR “athlete” OR “exercise” OR “fatigue” OR “sleep” OR “inflam*” OR “cortisol” OR “testosterone” OR “redox” OR “oxidative stress”) 1900/1/1 to 2022/10/31
SportDiscuss	(“whole body cryostimulation” OR “whole body cryotherapy” OR “cryo* chamber”) AND (“recovery” OR “athlete” OR “exercise” OR “fatigue” OR “sleep” OR “inflam*” OR “cortisol” OR “testosterone” OR “redox” OR “oxidative stress”) Published Date Start month: January Start year: 1900 — End month: October End year: 2022

2.3.2. Eligibility criteria

Studies were excluded if they did not compare the effect of post-exercise WBC to a passive control in non-clinical participants, did not sample a metric of recovery (as previously described), if there was no passive control, or if the study population was clinical. Studies were also excluded from meta-analysis if they did not yield enough information to accurately estimate the mean change score and standard deviation. If this was the case, yet they met all other criteria, then they were included in the systematic review only. Further, studies that only applied WBC before exercise were excluded. No specific restrictions were set on exposure temperature and duration. Study suitability was assessed independently by two authors (LE and JH).

2.3.3. Data extraction

Change from baseline scores were extracted from studies that assessed the effects of WBC versus control by one author (LE) and independently confirmed by a second (JH). Standardised mean effect sizes (ES) were calculated from pre–post-change scores between WBC and control groups, using the standard deviation of those changes (SD_{change}). Measures of CK and inflammatory proteins were obtained via venous or capillary sampling. Measures of DOMS were obtained via Likert or visual analogue scales. Measures of muscle function were derived from the analysis of maximal isotonic, isokinetic or isometric torque in addition to countermovement jumps (CMJ). Testosterone and cortisol measures were obtained via venous blood or saliva sampling.

A meta-analysis was only performed if at least three data sets from unrelated research groups were identified. Several studies reported data from CMJ with either hands-on-hips or where arms were permitted to swing. Where studies reported both, only power derived from countermovement jumps with hands-on-hips was considered for analysis.

Change scores were extracted or calculated from the included studies. Where SD_{change} was not reported, values were calculated using the following equation [332]:

$$SDchange = \sqrt{(SD2baseline + SD2final - (2 \times Corr \times SD2baseline \times SD2final))}$$

Where $SD_{baseline}$ represents the baseline SD, SD_{final} represents the post-intervention SD and Corr represents a correlation coefficient. A conservative correlation coefficient of 0.5 was used in all cases [332], this has been used elsewhere [333]. To assess the impact of this relatively arbitrary number, a sensitivity analysis was completed where the main analysis was repeated using a correlation coefficient of 0.25 and 0.75 to determine if the results were influenced [331,332]. Where data were presented in graphs, ImageJ software (NIH, USA) was used to estimate data from figure images. Where only median and confidence limits (CL) were presented, change scores were calculated only if the paper expressly

stated that the data met the assumption of normality. If it did not, then the data were excluded from the meta-analysis and considered qualitatively. Results were assessed with the I^2 statistic, quantifying the percentage of variability in effect size (ES) from heterogeneity, rather than chance. I^2 thresholds were interpreted following Cochrane guidance (0% to 40%: unimportant; 30% to 60%: moderate; 50% to 90%: substantial; 75% to 100%: considerable heterogeneity [332]. Where I^2 fell between two boundaries, the most severe interpretation was assumed.

Meta-analysis data were grouped by variable, then by the duration after exposure in which they were sampled. These were: <1 hour, 1 to 24 hours, then 24, 48, 72, 96, 120, 144 and 168 hours post-exposure. Data were then sub-grouped into single exposures (one exposure per study arm) and multiple exposures (more than 1 exposure over consecutive days).

2.3.4. Risk of Bias

Risk of bias was reported using the Cochrane Collaboration online risk assessment tool [332] where a series of signalling questions were used to assess potential bias.

2.3.5. Statistical Analysis

Data were analysed using RevMan statistical software package (version 5.0; The Nordic Cochrane Centre, The Cochrane Collaboration, Copenhagen, 2011). Standardized mean ES and 95% confidence intervals (CIs) were reported as (ES [LCL, UCL]), where LCL and UCL represent the lower and upper 95% confidence limits, respectively. Subgroup differences were presented as P values with χ^2 scores, while the likelihood of independent results was presented as P values alongside corresponding Z scores. The threshold values for standardised changes were as follows: <0.2 (trivial), 0.2 (small), 0.5 (moderate) and 0.8 (large) [334]. The threshold for statistical significance was set at $P<0.05$, and changes were deemed very likely beneficial if the 95% CI cleared the threshold for the smallest worthwhile change [335]. Effects were deemed unlikely beneficial if the 95% CI extended across the threshold for the smallest worthwhile change.

2.4. Results

1233 studies were identified through database searches. After 303 duplicates were removed, 930 abstracts were screened with 888 subsequently excluded. 32 potential studies were assessed for eligibility. Of these studies, 21 were excluded due to: 1. no English full text available; 2. no passive control group; 3. no exercise; 4. if studies described partial body cryotherapy (PBC) rather than WBC; or 5. if data could not be synthesised in a manner suitable for the meta-analysis. If the data met all other assumptions, then the studies were considered for the systematic review (Figure 10).

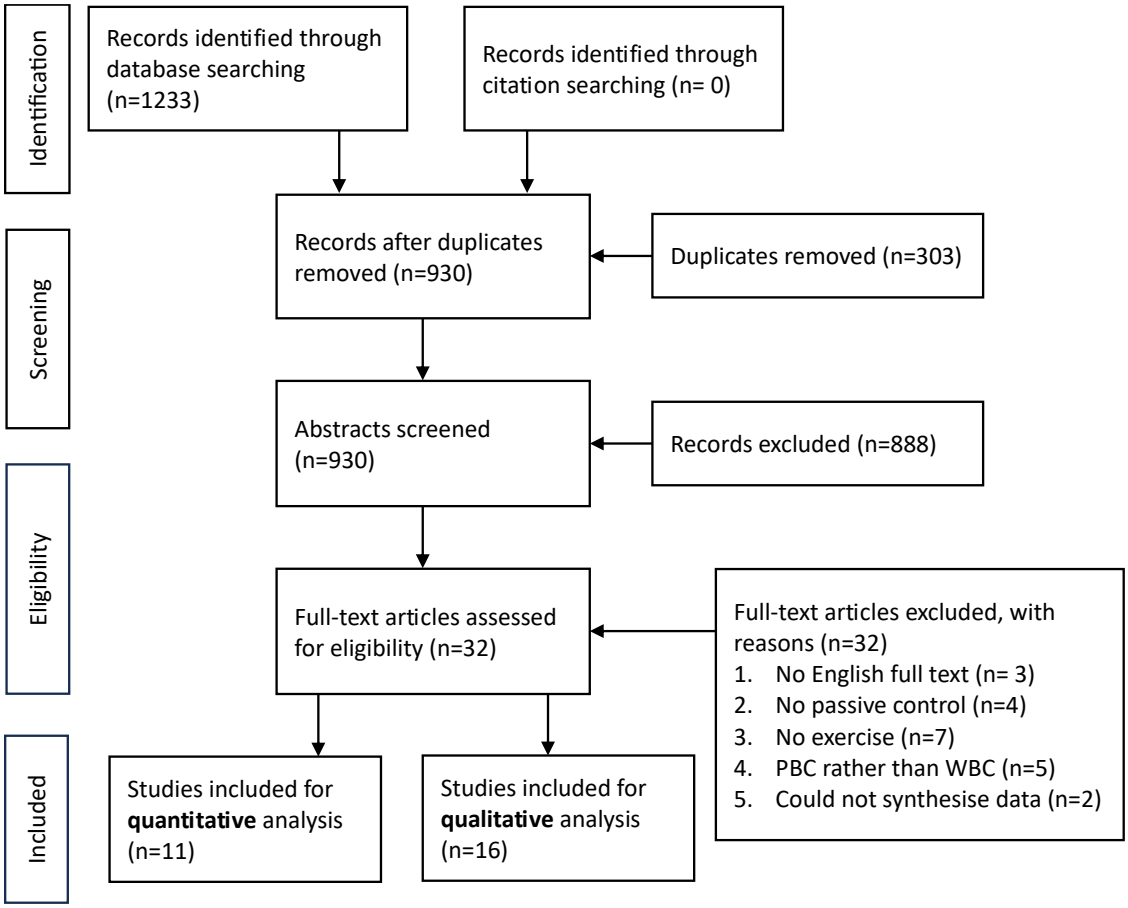

Figure 10: Search results schematic

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2061 11 studies met the inclusion criteria for the meta-analysis, including the following variables: CK,
2062 DOMS, muscle function, cortisol, testosterone and IL-6. Sensitivity analysis revealed that a correlation
2063 coefficient of 0.5 was considered sufficiently robust for the present analysis, with 0.25 and 0.75 not
2064 altering the significance of the results. six additional (total 16) studies were then considered for a
2065 systematic review, encompassing sleep quality, inflammation and antioxidant activity in addition to
2066 those included in the meta-analysis (Table 4).

Table 4: Study Information for investigation included in the meta-analysis and the systematic review.

Study Author (s), Year	Participants n, training status/level of competition (as stated in source) and sport, sex, intervention age, control age (if different)	Study Design	Exercise Protocol	WBC Protocol Time (s), temperature, exposures	Outcome measure	Sampling time points in reference to first exposure.
Creatine Kinase						
Russell et al., 2017 [310]	14, Professional youth football players, male, 18 ± 2	Randomised repeated measures	15 x 30m sprints with 10m deceleration zone	30/ 120, 60°C/135°C, 1 exposure	CK	<1 hours,1 to 24 hours, 24 hours.
Hausswirth et al., 2011 [124]	9, well trained runners, male, 31.8 ± 6.5	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	CK	<1hour, 24 hours, 48 hours
Ziemann et al., 2012 [126]	6 per group, high-ranking national level tennis players, male, 23 ± 3.0, 20 ± 2.0	Independent groups	Tennis recovery camp	30/180, -60°C/-120°C, 2 exposures per day for 5 days	CK	144 hours
Mila-Kierzenkowska et al., 2011 [125]	9, Polish Olympic Kayak team, f, 23.9 ± 3.2	Repeated measures	10 day microcycle	30/180, -60°C/ -120 to -140°C, 2 per day for 10 days	CK	144 hour, 168 hours
Wozniak et al., 2007 [336]	21, Olympic team Kayakers, Sex not stated, 24.6 ± 4.3	Repeated measures	10 day microcycle	-120 to -140°C, 3 times per day for 10 days	CK	144 hours, 168 hours
Wozniak et al., 2013 [327]	6, international-level rowers, sex not stated, 26.7 ± 3.6	Repeated measures	6 day microcycle	10 to 20/ 180, -60°C/ -125 to -150°C	CK	72 hours, 144 hours
Ziemann et al., 2013 [337]	9 per group, physically active, males, 21.7 ± 0.9, 22.0 ± 2.0	Independent groups	Step up/step down exercise (30 minutes)	20 to 30/ 180, -60°C/-110°C, 2 times per day for 5 days	CK	120 hours (protocol included second exercise, only 120 hours post exposure met the criteria)
Delayed onset muscle soreness						
Russell et al., 2017 [310]	14, Professional youth football players, male, 18 ± 2	Randomised repeated measures	15 x 30m sprints with 10m deceleration zone	30/ 120, 60°C/135°C, 1 exposure	Pain at rest	<1 hours, 1 to 24 hours, 24 hours

Hausswirth et al., 2011 [124]	9, well trained runners, male, 31.8 ± 6.5	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	Pain at rest	<1hour, 24 hours 48 hours
Costello et al., 2013 [330]	9, healthy adults, male and Female, 21.2 ± 2.1	Independent groups	Eccentric knee extensions	20/180, -60°C /-110 °C, 1 exposure	Pain at rest	24 hours, 48 hours, 72 hours
Muscle function						
Russell et al., 2017 [310]	14, Professional youth football players, male, 18 ± 2	Randomised repeated measures	15 x 30m sprints with 10m deceleration zone	30/ 120, 60°C/135°C, 1 exposure	Power (CMJ)	<1 hours, 1 to 24 hours, 24 hours
Jaworska et al., 2018 (m) [338]	10, university volleyball, male, Age not specified	Independent groups	2 week volley ball training with sports specific and power sessions	180, -110 °C, daily exposure with weekends off (10 in total)	Power (CMJ)	168+ hours
Jaworski et al., 2018 (f) [338]	10, university volleyball, female, Age not specified	Independent groups	2 week volley ball training with sports specific and power sessions	180, -110 °C, daily exposure with weekends off (10 in total)	Power (CMJ)	168+ hours
Costello et al., 2012 [319]	9, healthy adults, male and female, 21.2 ± 2.1	Independent groups	Eccentric knee extensions	20/180, -60°C /-110 °C, 1 exposure	Torque (knee extensor)	24 hours, 48 hours, 72 hours
Hausswirth et al., 2011 [124]	9, well trained runners, male, 31.8 ± 6.5	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	Torque (knee extensor)	1 hour,24 hours, 48 hours
Cortisol						
Russell et al., 2017 [310]	14, Professional youth football players, male, 18 ± 2	Repeated measures	15 x 30m sprints with 10m deceleration zone	30/ 120, 60°C/135°C, 1 exposure	Cortisol	<1 hour, 1 to 24 hours, 24 hours
Ziemann et al., 2012 [126]	6 per group, high-ranking national level tennis players, male, 23 ± 3, 20 ± 2.0	Independent groups	Tennis recovery camp	30/180, -60°C/-120°C, 2 exposures per day for 5 days	Cortisol	144 hours
Mila-Kierzenkowska et al., 2011 [125]	9, Polish Olympic Kayak team, female, 23.9±3.2	Repeated measures	10day microcycle	30/180, -60°C/ -120 °C to -140°C, 2 per day for 10 days	Cortisol	144 hours, 168 hours
Wozniak et al., 2013 [327]	6, international-level rowers, sex not stated, 26.7 ± 3.6	Repeated measures	6day microcycle	10 to 20/ 180, -60°C/ -125 to -150°C	Cortisol	72 hours, 144 hours
Wozniak et al., 2007 [336]	21, Olympic team Kayakers, Sex not stated, 24.6 ± 4.3	Repeated measures	10day microcycle	-120 to -140°C, 3 times per day for 10 days	Cortisol	144 hours, 168 hours

Schaal et al., 2015 [308]	10, national level Synchronised swimmers, female, 20.4 ± 0.4	Randomised repeated measures	1week microcycle	180 in coldest, -10°C /-60°C/-110°C	Cortisol	186 hours
Testosterone						
Russell et al., 2017 [310]	14, Professional youth football players, male, 18 ± 2	Repeated measures	15 x 30m sprints with 10m deceleration zone	30/ 120, 60°C/135°C, 1 exposure	Testosterone	1 to 24 hours, 24 hours
Ziemann et al., 2012 [126]	6 per group, high-ranking national level tennis players, male, 23 ± 3, 20 ± 2.0	Independent groups	Tennis recovery camp	30/180, -60°C/-120°C, 2 exposures per day for 5 days	Testosterone	144 hours
Krueger et al., 2019 [116]	11, healthy endurance trained, male, 25.9 ± 2.	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	Testosterone	<1 hour.
Interleukin 6						
Jaworska et al., 2018 [338]	20, university volleyball, 10 male, 10 females, Age not specified	Independent groups	2week volley ball training with sports specific and power sessions	180, -110 °C, daily exposure with weekends off (10 in total)	IL-6	168+ hours
Krueger et al., 2019 [116]	11, healthy endurance trained, male, 25.9 ± 2.	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	IL-6	<1 hour (protocol included second exercise, only 120 hours post exposure met the criteria)
Ziemann et al., 2012 [126]	6 per group, high-ranking national level tennis players, male, 23 ± 3, 20 ± 2.0	Independent groups	Tennis recovery camp	30/180, -60°C/-120°C, 2 exposures per day for 5 days	IL-6	144 hours
Studies for review						
Schaal et al., 2015 [308]	10, national level Synchronised swimmers, female, 20.4 ± 0.4	Randomised repeated measures	1 week microcycle	180 in coldest, -10°C /-60°C/-110°C	Bedtime, time asleep, sleep latency, sleep efficiency.	data averaged across week.
Douzi et al., 2018 [299]	22, physically active, male, 28.5 ± 7.3	Randomised repeated measures	Standardised repeated high intensity exercise	(forced convection WBC-2.3m s^{-1} wind speed) 30/180, 24°C/-40°C	Sleep time accelerometery	Single night post exercise

Broatch et al. 2019 [163]	11 per group, recreational athletes (triathlon or cycling), male, 37 ± 9, 37 ± 8	Independent groups	4 week interval training, 3 x per week (12 total), cycling	180 in coldest, -10°C /-60°C/-110°C, 3 exposures per week for 4 weeks (after exercise)	Bedtime, time asleep , sleep latency, sleep efficiency, moving time	Data averaged across 4 week period and compared to a control week
Aloulou et al., 2020 [309]	19, under-23 rugby union forwards and backs, 20.6 ± 1.3, 20.8 ± 1.0	Randomised repeated measures	Professional rugby union game	180, -110°C	Time asleep, sleep latency, sleep efficiency, wake after sleep onset	Single night post exercise
Pournot et al., 2011 [127]	11, well-trained runners, male, 31.8 ± 6.5	Randomised, cross over	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	IL-1, IL10, TNF-α, CRP	<1 hour, 1 hour, 24 hours, 48 hours 72 hours, 96 hours.
Krueger et al., 2019 [116]	11, healthy endurance trained, male, 25.9 ± 2.	Randomised repeated measures	Simulated trail run	180 in coldest, -10°C /-60°C/-110°C, 1 exposure	IL10, CRP, ICAM-1.	<1 hour (protocol included second exercise, only 120 hours post exposure met the criteria)
Ziemann et al., 2012 [126]	6 per group, high-ranking national level tennis players, male, 23 ± 3, 20 ± 2.0	Independent groups	Tennis recovery camp	30/180, -60°C/-120°C, 2 exposures per day for 5 days	TNF-α	144 hours
Wozniak et al., 2011 [327]	6, international-level rowers, sex not stated, 26.7 ± 3.6	Repeated measures	6 day microcycle	10 to 20/ 180, -60°C/ -125 to -150°C	SOD, CAT, GPx, CD, TBARS	72 hours, 144 hours
Wozniak et al., 2007 [326]	21, Olympic team Kayakers, Sex not stated, 24.6 ± 4.3	Repeated measures	10 day microcycle	-120 to -140°C, 3 times per day for 10 days	SOD, CAT, GPx, CD, TBARS	144 hours, 168 hours
Mila-Kierzenkowska et al., 2009 [328]	9, Polish Olympic Kayak team, female, 23.9 ± 3.2	Repeated measures	10 day microcycle	30/180, -60°C/ -120 to -140°C, 2 per day for 10 days	SOD, CAT, GPx, CD, TBARS	144 hours, 168 hours

Creatine kinase (CK), countermovement jump (CMJ), interleukin-6 (IL-6), interleukin-1 (IL-1), interleukin-10, tumour necrosis factor alpha (TNF-α), C-reactive protein (CRP), intercellular adhesion molecule-1 (ICAM-1), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), thiobarbituric acid reactive substances (TBARS).

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2.4.1. Assessment of bias

No identified studies used a blind, randomised crossover design. Other sources of bias included: sex not stated (2 studies), using the same baseline for both control and intervention conditions, no randomisation (4) not listing method of randomisation (if any) (4) and stating that participants were assigned to control due to cold sensitivity (1) (Figure 11).

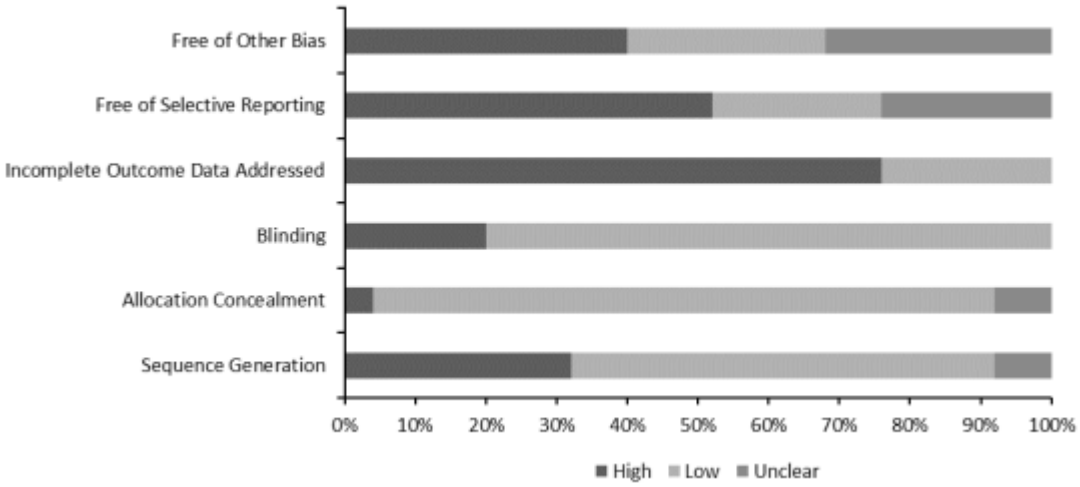

Figure 11: Percentage risk of bias for the included studies

2.4.2. Effect of whole-body cryotherapy on exercise-induced muscle damage

2.4.2.1. Muscle function

From four studies, 10 data points were extrapolated (n=61; 38 male, 14 female, 9 sex not stated; mean age: 23.7 years) [124,310,330,338]. The muscle damage interventions included sprints with decelerations (1), simulated trail running (1), eccentric knee extensions (1), and a varsity volleyball training microcycle (1) (Table 4). No overall statistically significant effect was detected (Z=1.27, P=0.21). After sub-grouping, single exposures showed no statistically significant effect (Z=0.48, P=0.63) and substantial and significant heterogeneity remained (I^2=71%, P=0.005). After multiple exposures, a significant effect was detected that favoured WBC (Z=2.50, P=0.01). However, there was significant heterogeneity (I^2=78%, P=0.004; Figure 12).

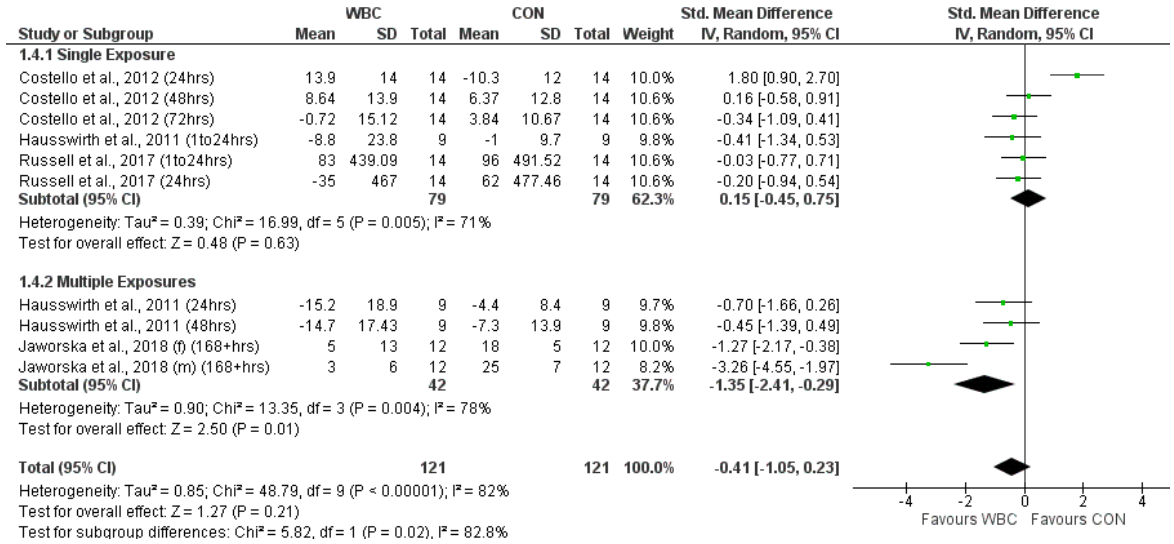

Figure 12: Forest plot illustrating the effect of whole-body cryotherapy on muscle function at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.2.2. Delayed onset muscle soreness

From three studies, 10 data points were extrapolated (*n*= 72; 28 males, 4 females, 9 sex not stated; mean age 23.7 years) [124,310,330]. Investigations included sprints with a deceleration phase (1), simulated trail running (1), and eccentric knee extension (1) (Table 4). No overall statistically significant effect was detected (Z=1.39, P=0.17) and heterogeneity was non-significant (I^2=21%, P=0.25). No significant effect was detected in the single exposure group (Z=0.27, P=0.79), however, a statistically significant effect was detected for multiple exposures with a large effect size favouring WBC for multiple exposures (Z=2.54, P=0.01). In both cases, heterogeneity remained minor (Figure 13).

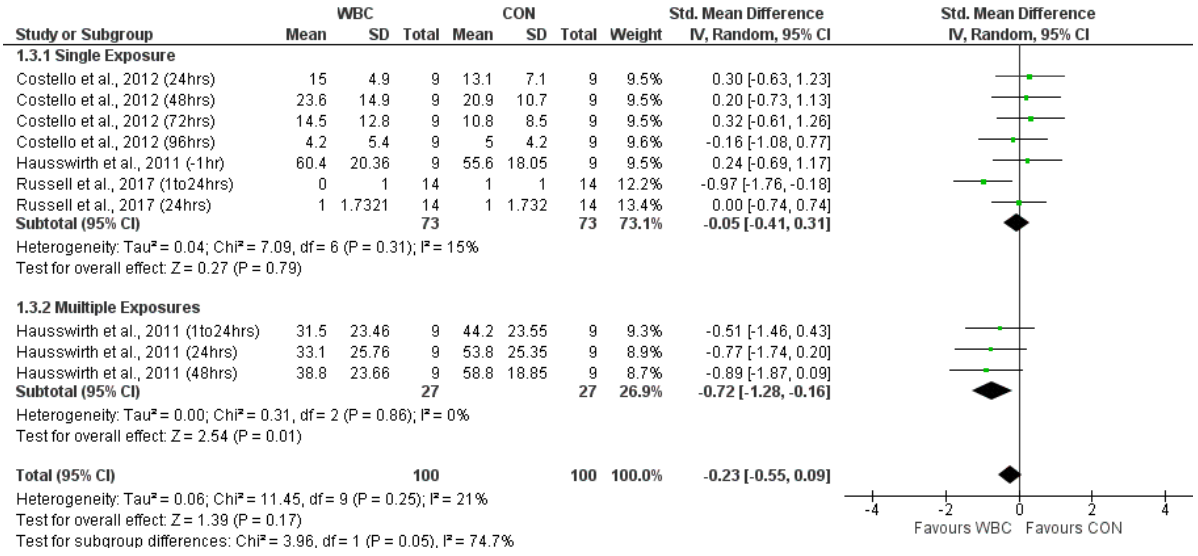

Figure 13: Forest plot illustrating the effect of whole-body cryotherapy on delayed onset muscle soreness at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.2.3. Creatine kinase activity

From 7 studies, 12 data points were extrapolated (n=88; 44 male, 9 female, 36 sex not stated; mean age 24.6 years) [124–126,310,327,336,337,339]. Studies included sprints with a deceleration phase (1), simulated trail running (1), a tennis-specific recovery microcycle (1), an Olympic kayak training microcycle (2), an Olympic rowing training microcycle (1), step-up task (1). Results approached the significance threshold of p<0.05, but failed to breach it (Z=1.95, P=0.50). No significant effect (Z=1.34, P=0.18) or heterogeneity (P=0.48, I^2= 0%) was detected in the single exposure subgroup. For multiple exposures, a significant effect was detected favouring WBC (Z=2.61, P=0.009), and considerable and significant heterogeneity remained (I^2=77%, P<0.0001; Figure 14).

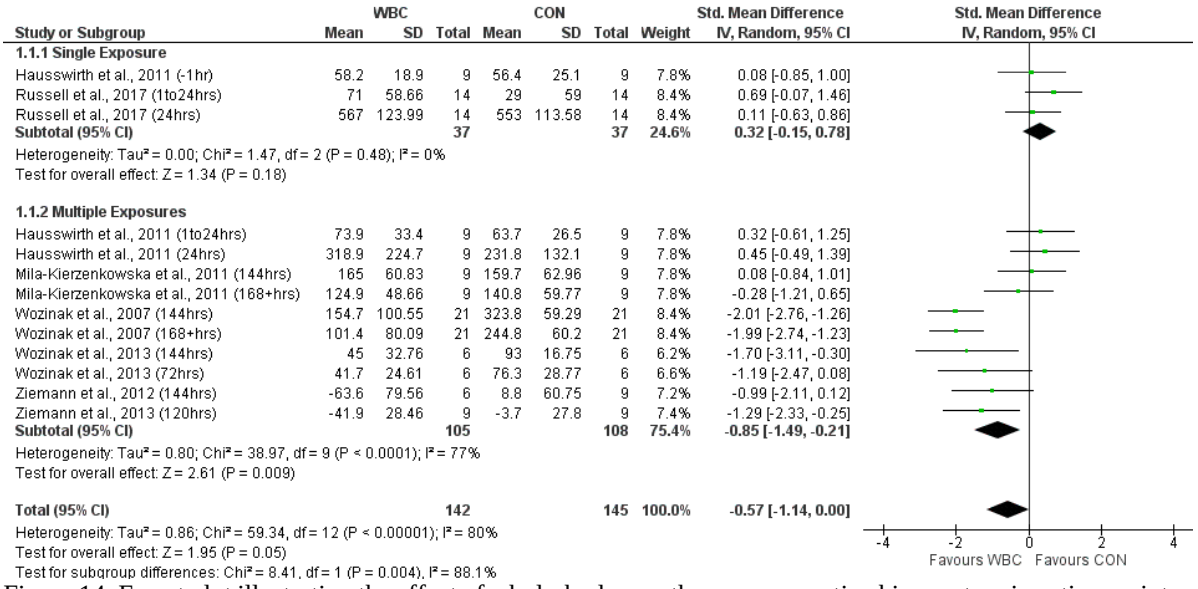

Figure 14: Forest plot illustrating the effect of whole-body cryotherapy on creatine kinase at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.3. Inflammation

2.4.3.1. Interleukin-6

IL-6 was the only marker of inflammation to be quantitatively analysed. Four data points from three studies were extracted (*n*=43; 33 male, 10 female; mean age 24 years) [116,338,340]. Studies included a simulated trail run (1), a tennis-specific recovery microcycle (1) and a varsity-level volleyball training microcycle (1). Analysis revealed no significant effect (single exposure; Z=0.56, P=0.57, multiple exposure; Z=1.14, P=0.25). Multiple exposures resulted in substantial heterogeneity (I^2=70%) that approached significance (P= 0.07) (Figure 15).

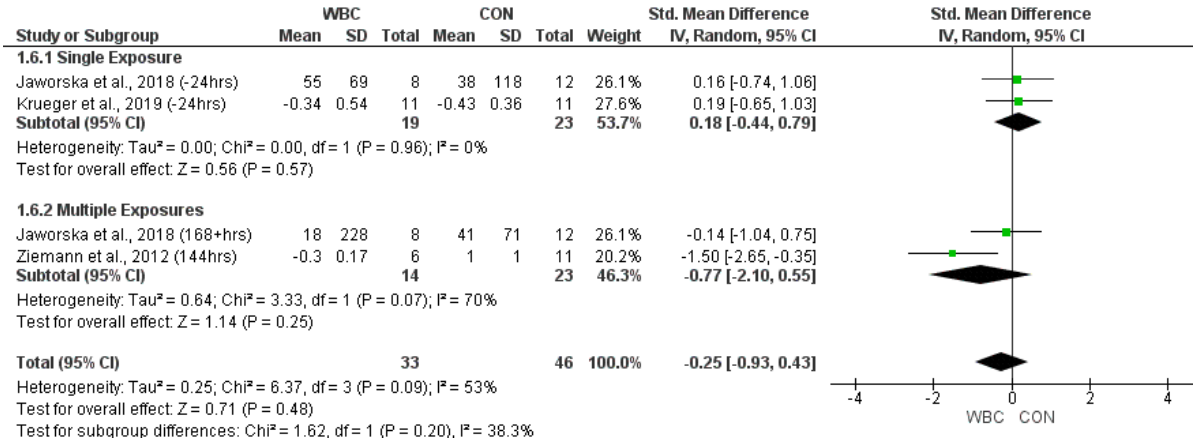

Figure 15: Forest plot illustrating the effect of whole-body cryotherapy on interleukine-6 at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.3.2. Other inflammatory proteins

Further markers of inflammation were considered qualitatively (*n*= 44; all male, mean age 26.9 years). Two laboratory-based studies investigated WBC effect on anti-inflammatory markers [116,127]. In one study, participants experienced a single exposure, the other two multiple exposures. Interleukin receptor agonist (IL-1ra) was increased, whereas interleukin-10 (IL-10) was unchanged. Four studies investigated the effect of WBC on pro-inflammatory markers. Two were laboratory-based using well-trained participants [116,127] and two were field studies completed on athletes [126,338]. Three of the four studies used multiple exposures and one used a single exposure. Levels of tumour necrosis factor-alpha (TNF-α) were decreased in one study [126] and not affected significantly in another [127]. C-reactive protein (CRP) levels were likewise decreased in one study [127], with no changes observed in another [116]. One further study also identified a lower level of IL-1 [127] (Table 4).

2.4.4. Endocrine biomarkers

2.4.4.1. Cortisol

From 7 studies, 11 data points were extrapolated *(n*=72; 26 male, 19 female, 27 sex not stated; mean age: 22.7 years) [116,125,126,308,310,327,336]. Studies exposed participants to sprints with a deceleration phase (1), a tennis-specific recovery microcycle (1), an Olympic kayak microcycle (2), an Olympic rowing microcycle (1), Olympic synchronised swimming microcycle (1), running (1). A statistically significant effect was detected favouring WBC, with a large effect size (Z=2.42, P=0.02). Subsequent subgroup analysis showed that single exposures had no statistically significant effect (Z=0.3, P=0.77) with low heterogeneity (I^2=13%). In multiple exposures, a large and significant effect

was detected that benefitted WBC (Z=3.34, P=0.0008). Heterogeneity was moderate, yet insignificant (I^2=33%, P=0.17; Figure 16).

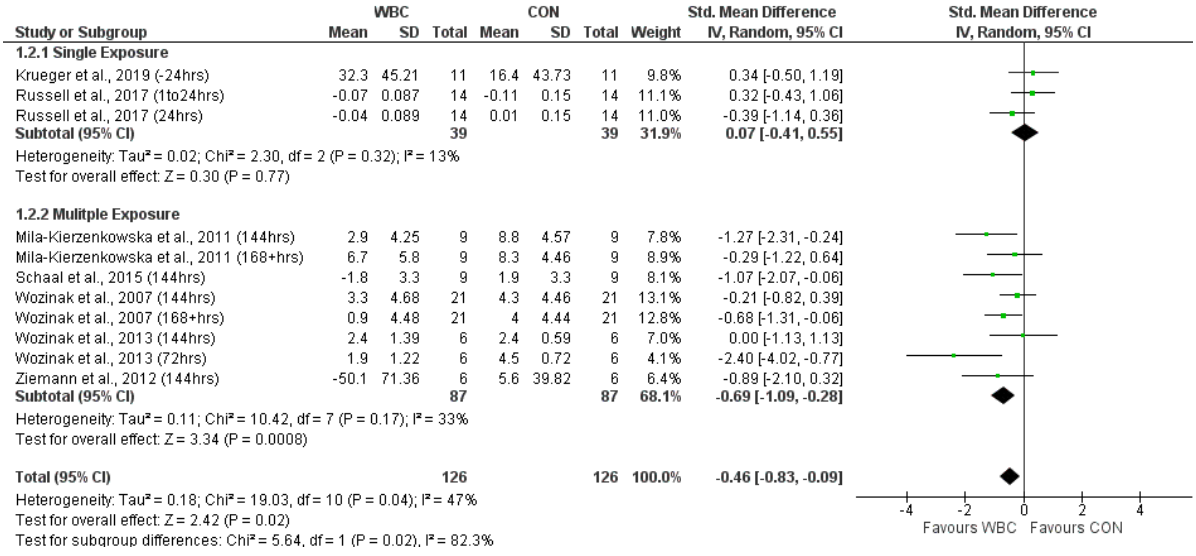

Figure 16: Forest plot illustrating the effect of whole-body cryotherapy on cortisol at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.4.2. Testosterone

Four data points were extrapolated from three studies that reported testosterone (*n*=27*,* all male; mean age 21.8 years) [116,310,340]. Investigations implemented sprints with a deceleration phase (1), simulated trail run (1), and tennis-specific recovery microcycle (1). Moderate non-statistically significant heterogeneity (I^2=46%, P= 0.14) was detected and a statistically significant effect was demonstrated favouring WBC (Z=2.26, P=0.02). It was decided not to sub-group considering the low power for multiple exposures (1 datapoint, *n*=6; Figure 17).

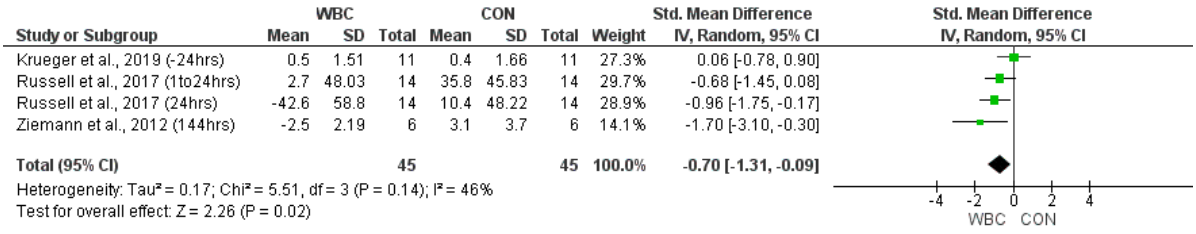

Figure 17: Forest plot illustrating the effect of whole-body cryotherapy on testosterone at various time points post-exposure. The square boxes represent the standardised mean effect for each data set with lines demonstrating 95% CIs. The size of the boxes represents the weight of the dataset. The diamond represents the overall standardised mean effect. The forest plot is sub-grouped into single (top) and multiple (bottom) exposures. Effect considered significant at P<0.05.

2.4.3. Redox biomarkers

Three studies from the same group examined redox biomarkers (*n*= 72 [sex not stated], mean age 24.3 years) [326–328]. All studies applied WBC across a microcycle (rowing (2), kayaking (1)) in international athletes. In all cases, the antioxidant enzymes catalase (CAT), superoxide dismutase (SOD) and glutathione peroxidase (GPx) were sampled in addition to the markers of lipid peroxidation, including, thiobarbituric acid reactive substances (TBARS) and conjugated dienes (CD) (Table 4). All studies reported improvement in redox balance, however, one observed no further improvement after continued WBC use [341].

2.4.4. Sleep quality

Four studies investigated the effect of WBC sleep quality (*n*=73; 63 male, 10 female; mean age 25.4 years) [163,299,308,309]. Participants engaged in professional under-23 rugby matches (1), high-intensity running (1), Olympic synchronised swimming microcycle (1) and high-intensity cycling (1). Although the number of sleep quality studies identified met the prerequisite for quantitative analysis, data could not be synthesised in a manner suitable for meta-analysis from at least three studies. Therefore, sleep quality was evaluated qualitatively. All studies used wrist-actigraphy technologies. One study reported the number of movements per minute by axis (*x, y, z*) during sleep, and the remaining three used algorithms to estimate time asleep, sleep onset latency and efficiency. Two studies also reported sleep and wake time [163,308], and one reported waketime after sleep onset (WASO) [309]. Of the four studies, two studied professional athletes in the field [308,309] and two utilised recreational athletes in laboratory-based trials [163,299]. Two studies used single WBC exposures [299,309], and two utilised multiple exposures [163,308] (Table 4). Two studies reported a significant and beneficial effect on metrics of sleep quality [299,308].

2.5. Discussion

This meta-analysis and systematic review investigated WBC as a post-exercise recovery strategy, with a subgroup analysis on exposure frequency. The primary findings of this study are that multiple exposures applied daily for at least 4 consecutive days significantly improved symptoms of EIMD (e.g., muscle function, CK, and DOMS), whereas single exposures did not. Furthermore, whilst multiple exposures also had a beneficial effect on cortisol, testosterone levels were significantly increased after just one exposure. No significant effect was detected for IL-6, although, markers of IL-1 and IL-1ra were improved. This analysis also highlighted significant heterogeneity across the data set. This likely due to the limited number of studies in this area to date, and the diverse range exercise and WBC exposure regimes used (Table 4).

2.5.1. Exercise induced muscle damage

2.5.1.1. Muscle function

Muscle function has been described as a fundamental indicator of EIMD [342]. The more frequent exposures to WBC may have promoted the removal of metabolites via the cold pressor response [124] to a greater extent than single exposures. This may have contributed to the alleviation of pain during movement [343], in turn, enhancing muscle function recovery. This could also be mediated by a placebo effect; though this has not been investigated directly and remains conjecture. Two studies have concluded that WBC was not more beneficial than a placebo amino acids supplement [122,123]. However, both studies used an unconventional WBC protocol at -85°C. One mathematical model suggests that a temperature of -130°C is required to influence muscle recovery, based on 3-minute exposures [344], but this is also likely to be dependent on the chamber design. Nevertheless, temperatures of ≤-110°C were exclusively used in all pooled muscle function investigations considered in this meta-analysis [124,310,330,338]. Therefore, it is not clear whether -85°C provides a valid and comparable therapeutic stimulus.

2.5.1.2. Creatine kinase

CK is used extensively as a marker of muscle damage in blood [36] and multiple WBC exposures significantly reduced CK activity. Benefits were exclusively observed in athletic training camps, where at least 5 daily exposures were applied across a microcycle [125,126,327,336]. Studies that utilised fewer exposures appeared not to impact CK activity significantly [124,310], suggesting that more than 4 exposures are required to reduce secondary muscle fibre breakdown [124,345] or muscle fibre permeability. Covariates are challenging to control for in applied studies and might be a factor in the substantial heterogeneity that remained after subgrouping (I^2=77%). It is important to note that CK is known to be highly variable [4] and factors including exercise modality, intensity, training status, and sex could potentially influence results [4,36,346]; irrespective of the number of WBC exposures. Nevertheless, the limited number of studies available for analysis is likely to be the major cause of heterogeneity in this instance. Considering the sensitivity of CK, it is likely that studies were insufficiently powered to make firm conclusions.

2.5.1.3. Delayed onset muscle soreness

Costello et al. [330] previously reviewed the influence of WBC and PBC on DOMS and found no statistically significant effect, albeit with low study numbers. Whilst the present meta-analysis and the review presented by Costello et al. [330] took similar approaches, they differ in their eligibility criteria. Studies that described a PBC device in their methodologies were not included in this meta-analysis, as WBC and PBC are reported to trigger different physiological responses [314]. Considering the severely limited number of included mstudies (n=3), robust conclusions cannot be made at this time. However,

the results follow the same trend as muscle function and CK, alluding to the fact that multiple exposures are required to maximise any therapeutic response. Reductions in DOMS might be attributed to a cold-induced analgesic effect [115,329] resulting from decreased receptor sensitivity, firing rate and muscle spasm when skin temperature falls below 13°C [115]. The exposed surfaces of the legs have been recorded at 5.3 ± 3.0°C [119], suggesting a cold-induced analgesic effect is plausible.

Whilst the results of the meta-analysis indicate multiple exposures are more effective in EIMD recovery, some studies have observed benefits to EIMD after one single exposure. Hausswirth et al. [124] recorded improvements in both DOMS and muscle function after one exposure, while CK remained unaffected. It has been suggested that the within-athlete variability of CK has greater validity in determining the presence of EIMD as opposed to the absolute magnitude [36], therefore these findings should be interpreted accordingly. A placebo, short-term perceptual response or an analgesic effect might account for the improvement in DOMS and muscle function. Especially considering that DOMS and muscle function were improved just one hour after exposure.

2.5.2. Inflammation

2.5.2.1. IL-6

IL-6 was the only inflammatory protein that met the prerequisites for a meta-analysis. Neither single nor multiple WBC exposures resulted in a significant effect on IL-6. Another study, not included in the quantitative analysis due to the absence of numerical data, supports this with the authors stating that no significant changes were observed in IL-6 [127].

2.5.2.2. TNF-α, CRP, and IL-1

It was theorised that the cold pressor response would induce a reduction in intercellular adhesion molecule-1 (ICAM-1) that transmigrates cytokines to sites of EIMD, however, this has not been consistently observed [116]. Studies have investigated TNF-α and, while Pournot et al. [127] observed no changes after 4 WBC sessions in 4 days, Ziemann et al. [126] did see reductions in TNF-α after WBC was applied daily across a microcycle in professional tennis players. Both studies utilised multiple exposure protocols, however, Ziemann et al. [126] collected data during a post-competition recovery camp. Therefore, residual levels of TNF-α from competition might account for some differences between studies.

CRP is used as an acute phase marker of systemic inflammation [347] and was investigated in two studies [116]. Krueger et al. [127] reported no changes in CRP compared to control after one exposure. Pournot et al. [127], however, did see a significant change in CRP at 24 hours post-exercise in addition to a benefit to IL-1 activity. Blood was sampled before the second exposure and, therefore, cannot be

attributed to exposure frequency. Although, where Pournot et al. [127] sampled at 24 hours, Kruger et al. [116] sampled directly after exposure, and as such, the difference in sampling points might account for differences in the outcomes.

Pournot et al. [127] exposed 11 well-trained runners to a simulated trail run, with downhill segments designed to cause EIMD. They then received daily WBC or passive recovery for four days. One-hour post-recovery, researchers observed greater levels of IL-1ra compared to the control. IL-1ra typically peaks within the first hour post-exercise [348] and counters the pro-inflammatory actions of IL-1. This was not seen at subsequent time points during the proceeding four days of treatment, suggesting WBC is most effective when applied directly after exercise. Pournot et al. [127] also considered the anti-inflammatory cytokine IL-10 and, although not numerically represented, the authors commented that it was not affected significantly, which is also supported by others [116].

2.5.3. Endocrine markers

2.5.3.1. Cortisol

Cortisol presented with the same trend as DOMS, muscle function and CK, with reduced levels after multiple exposures. Cortisol plays a multifaceted role in exercise recovery. It is principally catabolic [349], responsible for liberating amino acids for muscle plasticity and adaptive remodelling [350]. However, cortisol also competes for receptor space with the anabolic testosterone [350,351], and a hyper-corticoid state is indicative of over-training or fatigue [350]. Increased cortisol release, secondary to both exercise and cold exposure [350,352,353], might increase the inhibitory effect on testosterone, limiting any testosterone-linked therapeutic response. It is possible that with familiarisation, the stress response alleviates. This has not been investigated directly and, therefore, remains speculation.

2.5.3.2. Testosterone

Overall, WBC increased testosterone concentrations after exercise. Subgrouping was not completed in this instance since only one data point considered multiple exposures, where n=6. Nevertheless, studies demonstrated increased testosterone compared to no intervention, irrespective of the number of exposures [126,310]. This suggests WBC can provide beneficial improvements to endocrinological status and is perhaps mechanistically linked to improvements in EIMD. In one exception, Krueger et al. [116] observed no differences in testosterone between groups when 11 participants received either WBC or a passive control after high-intensity running. However, this study included a second ramped bout of exercise after WBC, meaning only measures taken immediately after WBC met the inclusion criteria, allowing very little time for testosterone levels to react to WBC.

2.5.4. Sleep

It is commonly stated that sleep quality is axiomatic to the recovery process [354–356]. Heart rate variability (HRV) investigations have demonstrated WBCs efficacy in increasing vagal-mediated cardiac control, suggesting WBC can augment post-exercise parasympathetic reactivation and support sleep quality [313,314]. The studies that have investigated the effect of WBC on sleep are varied [163,299,308,309], with both single and multiple exposures reporting significant [299,308] and non-significant effects [163,309]. Whilst the number of exposures might still influence results, it is not an apparent factor in the limited number of studies available to date. One recent study has measured reduced noradrenaline after five successive WBC exposures suggesting physiological autonomic habituation to WBC that might impact sleep quality [316]. However, WBC applied daily over a 14-day microcycle in Olympic standard synchronised swimmers were still sufficient to positively influence sleep [308]. Both Schaal et al. [308] and Douzi et al. [299] reported better sleep quality after participants received evening WBC (~1900 and ~2030, respectively), yet, Allou et al. did not record a difference when under-23 rugby players received WBC at a similar time (~2130) [309]. This makes it unclear if the timing of WBC in relation to bedtime is a factor. In markers of EIMD, greater success has been observed when WBC has been utilised in applied studies [125,126,327,336]. However, the same conclusion cannot be drawn in sleep quality with applied studies reporting both a sleep benefit [308] and no effect [309]. The same pattern is apparent in the two more laboratory-based investigations [163,299]. None of the research to date suggests that WBC negatively affects sleep [163,299,308,309], nevertheless, whilst it remains possible that sleep can be positively affected by WBC, further studies are required.

2.5.5. Redox balance

EIMD and inflammation lead to, or occurs in tandem with, increased reactive oxygen species (ROS) and exacerbated EIMD (58,59). Several antioxidative mechanisms counter ROS, though, these can be outpaced by repeated EIMD, even in an adapted muscle, leading to a state of oxidative stress [77]. In 20 national-level kayakers, concentrations of SOD and GPx were attenuated by WBC by day 6. However, concentrations were not different from the control group by day 10 [326]. This was accompanied by reductions in TBARS and CD. A later study on female kayakers [328] mostly concurs with previous data [326]. Finally, a study of a similar design measured antioxidant enzymes after 3 and 6 days of WBC [327], rather than 6 and 10 days [326], in international-level rowers. Results indicate reduced enzymes at 6 compared to 3 days [328]. Overall, the data suggest that there might be no additional benefit after 6 days of WBC on redox balance. All authors reporting redox data propose that a homeostatic adaptation occurs in response to WBC that supports the antioxidant balance.

WBC and other cryotherapies (cold-water immersion (CWI), local ice application, etc.) might reduce tissue metabolism [115,116,329]. In turn, limiting secondary tissue damage and injury risk [36]. However, the associated substrates also activate signalling pathways which ultimately regulate transcription factors that drive muscle adaptive remodelling [72]. Whilst the prevention of inflammatory proteins and ROS during competition congestion is of interest to practitioners, they should also be aware of potential negative effects on muscle plasticity and adaptation. A growing body of evidence has highlighted that post-exercise CWI can attenuate muscular adaptation to resistance training [357]. However, in response to endurance and high-intensity cycling, CWI has no, or a slightly beneficial effect on training outcomes [358]. WBC is yet to receive the same investigative interest. Nevertheless, after a 4-week high-intensity cycling intervention (3 sessions weekly), post-exercise WBC did not significantly influence peak aerobic power, oxygen uptake, time to exhaustion or substrate utilisation, compared to the control [359]. Further research is needed to determine the effect on muscular strength and mass. Although this indicates that practitioners should consider training aims as well as schedules before WBC application.

2.5.6. Practical implications and future research

This review suggests that four or more WBC exposures are required to impact upon EIMD recovery. Therefore, practitioners should schedule multiple exposures across a microcycle. Practitioners should also beware that WBC might influence sleep quality, although data here are limited and further studies are needed. Further research is also needed on WBC effect on muscle plasticity, anabolic signalling and adaptation to exercise. There are also several intra and inter-individual factors that require elucidation.

2.5.7. Conclusions

In conclusion, the strength of the current body of literature is poor, with a small number of studies presenting with low power. In the investigations available, a fairly large number of exercises are considered, limiting sport-specific recommendations that can be made to athletes and practitioners. However, the meta-analysis indicates that multiple exposures, applied across a microcycle can improve EIMD recovery. This might be attributed to a reduced stress response over successive WBC exposures, or, adaptive inflammatory and redox balance responses. There might also be a benefit to sleep after one exposure that subsequently impacts endocrine and other markers of recovery.

Chapter 3

3. The effect of whole-body cryotherapy on sleep quality and game-day endocrine and inflammatory markers in U18 professional football players: A descriptive pilot study

This crossover-designed study was unfortunately curtailed by the Covid-19 pandemic and lockdown restrictions. Consequently, only the first phase was completed. The resultant independent group analysis is presented here.

3.1. Abstract

No studies have investigated the use of whole-body cryotherapy (WBC) applied consecutively on the prior to a competitive fixture. This may be particularly pertinent as some clubs may currently schedule WBC in this manner with no evidence to suggest efficacy. Therefore, this study aimed to investigate the effect of WBC applied across an in-season microcycle on objective and subjective sleep quality and game-day inflammation and endocrine markers in U18 professional footballers (n=17, 17.4 ± 0.6yrs). On two consecutive game days (GD1 and GD2), Players were sampled for salivary testosterone, salivary cortisol and capillary high-sensitivity C-reactive protein (hsCRP). Players then either received WBC (CRYO; n=9) or no WBC (CON; n=8) over 4 consecutive days preceding GD2. During this period, sleep was monitored objectively (activity monitor) and subjectively (Leeds Sleep Evaluation Questionnaire). Within and between-group comparisons were made between GD1 and GD2 for the inflammation and endocrine markers. Between-group differences amongst sleep metrics were compared by day (Day 1 to 4) and by week. Testosterone levels decreased from GD1 to GD2 in both the CON (GD1: 401.5 ± 162 pg/ml, GD2: 315.4 ± 123.8 pg/ml, p=0.031) and CRYO (GD1: 592.9 ± 146 pg/ml, 352.9 ± 146.1 pg/ml, p=0.028) groups. However, there was no significant between-group difference in the change scores (p>0.05). There was also no significant within or between-group difference for cortisol, hsCRP, or objective sleep metrics (p>0.05). Although, players in the CRYO group reported better behaviour following wake (CRYO: 62 ± 11) compared to the control group (CON: 49 ± 17, p=0.001) =This study suggests that WBC applied during an in-season microcycle does not affect testosterone, cortisol, hsCRP, or objective sleep metrics. However, Players who received WBC felt more alert after and thus WBC may be used to increase the perception of alertness in professional U18 football players.

3.2. Introduction

Sleep plays a pivotal role in physiological [14] and psychological homeostasis [192,193,360]. Therefore, it is considered central to athletic performance and recovery [14]. However, football players, sleep metrics present with significant inter/intra-player variation [275] and several factors have been highlighted that may affect sleep, including day type (training day, match day, etc.,) [275], travel [226,277], night matches [273,361], and fixture results [362]. Therefore, there is an interest in methodologies that support sleep in football players [363–365]. Whole-body cryotherapy (WBC; a 2-3 min whole body exposure to -110°C to -160°C air wearing minimal clothing in specially designed chambers) was initially developed to attenuate inflammation [118,366], however, WBC has recently emerged as a novel therapy that may support sleep in athletes [367].

The mechanism in which WBC may support sleep is unclear, although, it may be related to an augmented post-exposure parasympathetic response [299,313], with studies demonstrating that WBC increases heart rate variability metrics associated with increased vagal tone [299,313]. However, reports examining the ability of WBC to improve sleep are equivocal [299,308,359,368]. In Olympic swimmers, daily WBC significantly attenuated sleep disruption during a training camp [308], likewise, participants who received post exercise WBC objectively recorded less nocturnal movements compared to those who did not [299]. Contrastingly, post-exercise WBC failed to significantly influence sleep metrics in healthy males engaged in a 4-week high-intensity interval cycling intervention [359], and post-game WBC afforded no significant sleep benefit to professional rugby players [368].

Nevertheless, in adolescent professional footballers [310], post-exercise WBC has been observed to increase testosterone levels. This may be related to improved sleep [14]; however, an empirical link is yet to be established [310]. Regardless, testosterone is an anabolic steroid that is essential to protein synthesis, turnover, repair, and athlete recovery [14] and its levels have speculatively been suggested to be an indicator of athletic preparedness [369]. Likewise, the post-exercise inflammatory response is axiomatic to the recovery process [72,76], yet excessive inflammation can exacerbate exercise-induced muscle damage and prolong recovery [72,76]. Some studies have reported that WBC can attenuate post-exercise inflammation, in turn, abating secondary muscle damage [116,367,370]. However, there has been no research examining the effect of WBC in professional football players and studies have applied WBC during an in-season period [299,308,359,368]. Furthermore, no research has investigated the use of multiple WBC exposures applied across a microcycle, during the lead-up to a competitive. This may be particularly pertinent as some clubs may be currently scheduling their WBC in this manner, due to other scheduling commitments, with no evidence to suggest efficacy.

Therefore, this study provides the first data on sleep and WBC in professional football players by the aims of this study were to (1) investigating the effects of a WBC applied across an in-season microcycle

on the objective and subjective sleep quality in under 18 (U18) professional footballers, and (2) determining the effect of WBC on game-day inflammation, testosterone, and cortisol.

3.3. Methodology

3.3.1. Participants

After informed consent, 17 under 18 (U18) professional footballers (17.4 ± 0.6yrs) from an English Premier League academy were recruited for this study. All procedures were approved by the St Mary's University, Twickenham, ethics review board and were conducted in accordance with the Declaration of Helsinki and Nuremberg Code.

3.3.2. Experimental procedure

This study was conducted during an in-season microcycle (Table 5), between two consecutive game days (GD1 and GD 2), spaced 7 days apart. On GD1 players reported to the training ground and provided capillary blood (serum hsCRP) and saliva samples (testosterone and cortisol) before travelling by coach for an away fixture. Players were then randomly assigned to either the intervention group (CRYO, n=9) or the control group (CON, n=8). Four days before GD2 players in the CRYO group commenced 4 days (Day 1 to 4) of WBC (one exposure per day) using a specially designed liquid nitrogen-cooled chamber (CryoAction, Wrocław, Poland) situated at the training ground. This WBC regime was chosen as it was synonymous with what is commonly scheduled for the first team. The WBC exposure took place at the end of each training day between 1500 and 1600. After ensuring that their skin was dry and free of treatment oils, players wore minimal clothing (shorts, socks, clogs, mask, gloves, and a hat covering the ears) and entered the prechamber (-60°C) for 30sec before moving to the main chamber (-135°C) for 150sec. Players in the CON group remained seated in the changing rooms during the treatment. On GD2, players reported for repeat capillary blood and saliva sampling. Objective and subjective sleep data were collected for Day 1 to 4 inclusive. Blood and saliva measures were taken at the same time to avoid circadian variation (Figure 18).

Table 5: Typical in-season week for the U18 footballers involved in this study

Day	AM	PM
Monday (TD)	Education	Training
Tuesday (TD)	Training	Gym training/ Injury prevention/ technical skills training/ analysis
Wednesday (TD)	Gym training/ Injury prevention, technical skills training/ analysis	Education
Thursday (TD)	Education	Training
Friday (MD-1)	Training	Team meeting
Saturday (MD)		Matchday
Sunday (MD+1)	Off/ rest day	

Training day (TD)
Matchday minus one (MD-1)
Matchday plus one (MD+1)
Matchday (MD)

Players were subsequently excluded from parts of the analysis for the following reasons: moved team, unable to provide a biological sample and/or technical error. Final numbers for each variable are as follows: Objective and subjective sleep analysis (n=15, CON=8, CRYO=7), saliva (n=10, CON=5, CRYO=5), hsCRP (n=15, CON=6, CRYO=8).

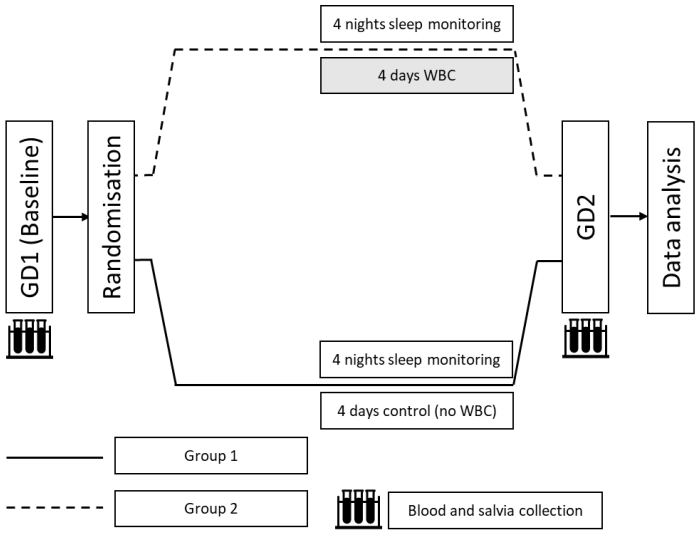

Figure 18: The experimental protocol that was followed. The original proposal included a washout and a crossover arm; however, it was curtailed due to COVID-19 lockdown restrictions. GD (Gameday), WBC (Whole-body Cryotherapy).

3.3.3. Sleep monitoring

Sleep was objectively monitored using a ReadiBand (Fatigue Science, Vancouver BC, Canada) wrist-worn activity monitor. Nocturnal movements detected by the device are converted by built-in algorithms to predict participant sleep quantity, sleep quality, awakenings per hour, total awakenings, wake after sleep onset (WASO), sleep latency, sleep onset time and wake time. Player's mean weekly sleep duration

was also collated based on whether they achieved the minimum quantity of sleep (420mins) according to published recommendations from the National Sleep Association (NSA; a not-for-profit organisation based in the USA) [371].

ReadiBands have demonstrated good accuracy compared to the gold-standard sleep-plethysmography (93%), and good inter-device reliability [198,372]. Participants were given the same device, after their assigned intervention (CON or CRYO), and asked to wear it on their non-dominant wrist. The devices were then collected the proceeding morning at ~0830 (on arrival at the training ground) and synched to cloud-based software.

Subjective sleep quality was assessed using the Leeds Sleep Evaluation Questionnaire (LSEQ). Upon arrival to the training ground, participants were asked to mark 10 100mm visual analogue scales (VAS) that assessed the ease of getting to sleep (GTS), quality of sleep (QS), and awakenings following sleep onset (AFS) and behaviour following waking (BFW). The midpoint represented the present feeling before the intervention. Scores were represented in mm.

3.3.4. Serum hsCRP and saliva endocrines

Blood and saliva samples were collected on the morning of GDs at a consistent time (between 0730 and 0830) upon arrival to the training ground. It was decided to only sample on GDs as it represented, theoretically, the most rested and repeatable point of the microcycle, as players workload was deliberately tapered in preparation of game day. After surface preparation, capillary blood was drawn using a single-use lancet (ACCU-CHEK Safe-T-Pro Plus, Indiana, USA) and collected using untreated serum separation microvettes (Microvette CB300, Sarstedt Inc, Nümbrecht, Germany). Samples were then allowed to clot at room temperature for 30 minutes before centrifugation. 25µl of serum was then aliquoted into reagent kits and serum hsCRP levels were analysed using a point-of-care analyser (Eurolyser CUBE, Eurolyser Diagostica, Austria).

Saliva was collected via passive drool without stimulation. Samples were deposited into cryovials (SalivaBio Cryogenic Vials, SalivaBio, USA), refrigerated at 4 to 6°C, and then frozen at -80°C within 3 hours of collection. They were then thawed, vortexed, and centrifuged at 1500g for 15 minutes. Samples were analysed, in duplicate, using testosterone and cortisol (high sensitivity) enzyme immunoassay kits (Salimetrics, PA, USA), respectively, following the manufacturer's protocol. Optical densities were read on a plate reader (ASYS Expertplus plate reader, Biochrom, Germany) at 450nm with a secondary filter correction at 492nm. A standard curve was generated with each plate using a standard of a known sample dilation and a 4-parameter non-linear regression curve was fitted to convert to µg/dL.

3.3.5. External load assessment

Workload could not be controlled between groups, therefore, Global positioning system (GPS) data, routinely collected by coaching staff, was used to determine any differences. Participants donned a vest that placed a GPS and accelerometry unit (Viper V.2, StatSports, Ireland) between the scapulae. The unit sampled GPS and accelerometry data at 10 Hz and 100 Hz, respectively. The data were downloaded using specialist software (Viper, V.2.1.3.0) for analysis. High-speed running (HSR; total distance (m) covered at running speeds >5.5m·s^{-1}), total number of accelerations (ACC; an increase in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}) and total number of decelerations (DEC; a decrease in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}).

3.3.6. Statistical analysis

A Shapiro–Wilk test was used to determine normality. Differences in activity monitor sleep data, LSEQ, and GPS workload between groups were assessed using an independent t-test or Mann-Whitney U (normality dependent). Within-group differences between GD1 and GD2 were assessed using paired sample t-tests. Change scores between GD1 and GD2 were calculated and differences between groups were assessed using an independent t-test. Pearson's correlations were performed between day 4 objective and subjective sleep metrics and saliva endocrine samples. To assess assay reliability, Pearson's correlations were performed between duplicate samples. All data were analysed using the R statistical environment and $p<0.05$ was considered statistically significant for all tests.

3.4. Results

Data are presented as mean ± standard deviation. No significant difference was found between groups for HSR, ACC or DEC ($p>0.05$). Pearson's correlations revealed that only objective sleep efficiency and WASO had a significant relationship ($R=0.87$, $p=0.032$) (Figure 19).

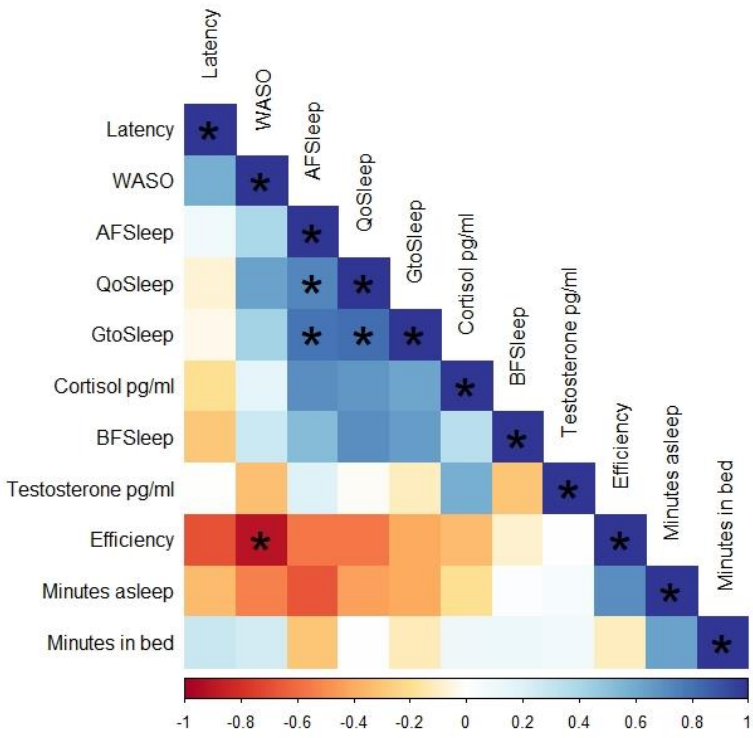

Figure 19: Pearson's correlation matrix between day 4 sleep objective and subjective sleep variables and salivary endocrine samples. Dark blue indicates a very positive relationship, dark red represents a very negative relationship. * indicates a statistically significant relationship. WASO (wake after sleep onset), AFSleep (awakenings following sleep), QoSleep (Quality of sleep), GtoSleep (Ease of getting to sleep), BFSleep (Behaviour following sleep)

3.4.1. Sleep monitoring

There was no significant interaction between CON or CRYO for weekly mean sleep latency (CON: 27.3 ± 23.1 mins, CRYO: 23.3 ± 25.5 mins; p=0.15), WASO (CON: 37.8 ± 35.9 mins, CRYO: 33.0 ± 24.1 mins, p=0.87), MiB (CON: 483.6 ± 55.4 mins, CRYO: 506.1 ± 80.0 mins; p=0.33), sleep duration (CON: 399.8 ± 55.6mins, CRYO: 419.4 ± 58.6mins; p=0.2), or sleep efficiency (CON: 82.8 ± 7.3%, CRYO: 83.4 ± 8.2%; p=0.76). Likewise, there were no significant differences between CON and CRYO on individual days in sleep latency, WASO, MiB, sleep duration, or sleep efficiency (p>0.05) (Figure 20). According to analysis, 66.67% of players in the CRYO group achieved ≥420mins sleep, compared to 25% of the CON group (Figure 21).

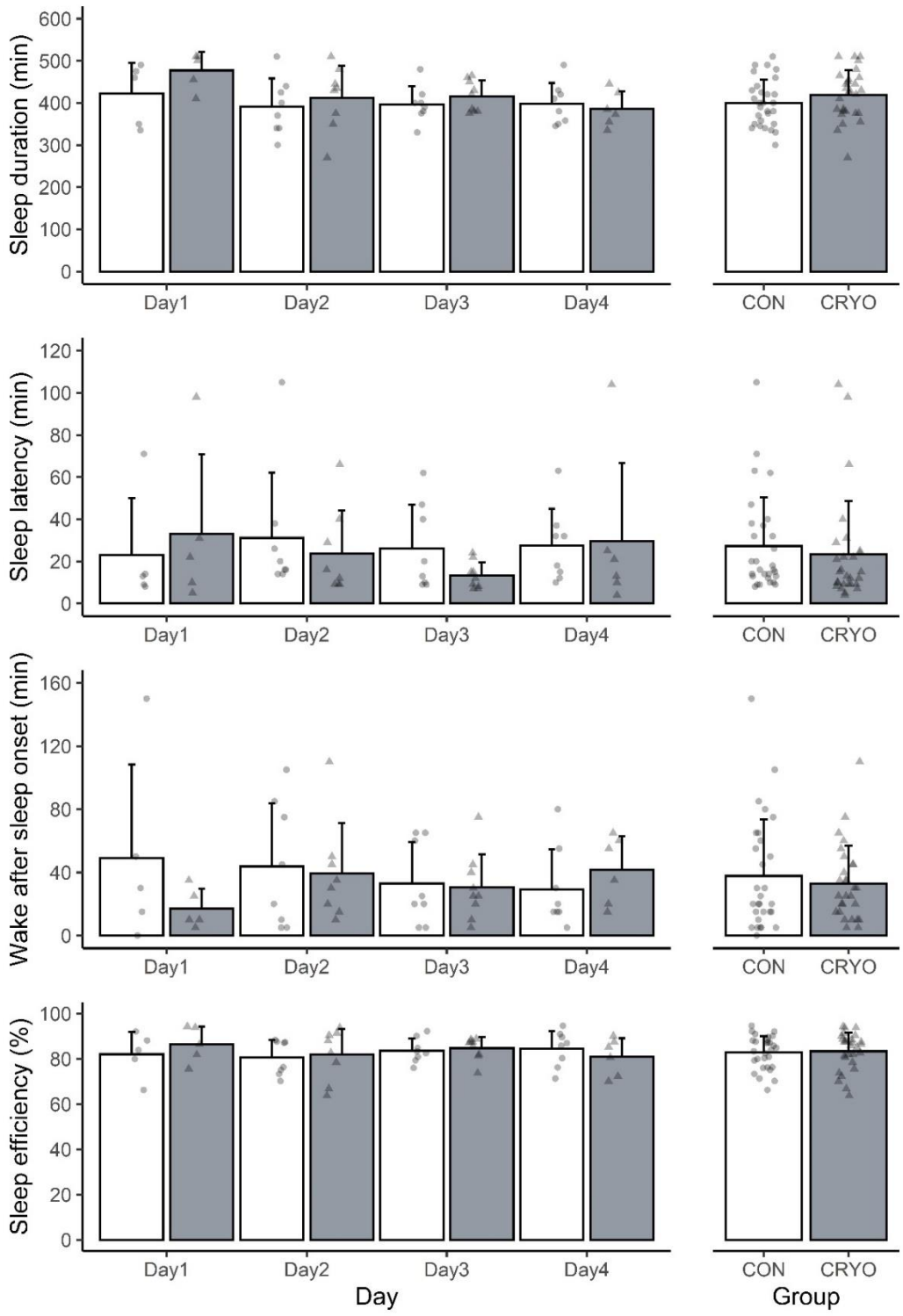

Figure 20: Activity monitor-derived sleep metrics displayed by day (left) and weekly mean (right). CON is shown in white and CRYO in grey.

2504

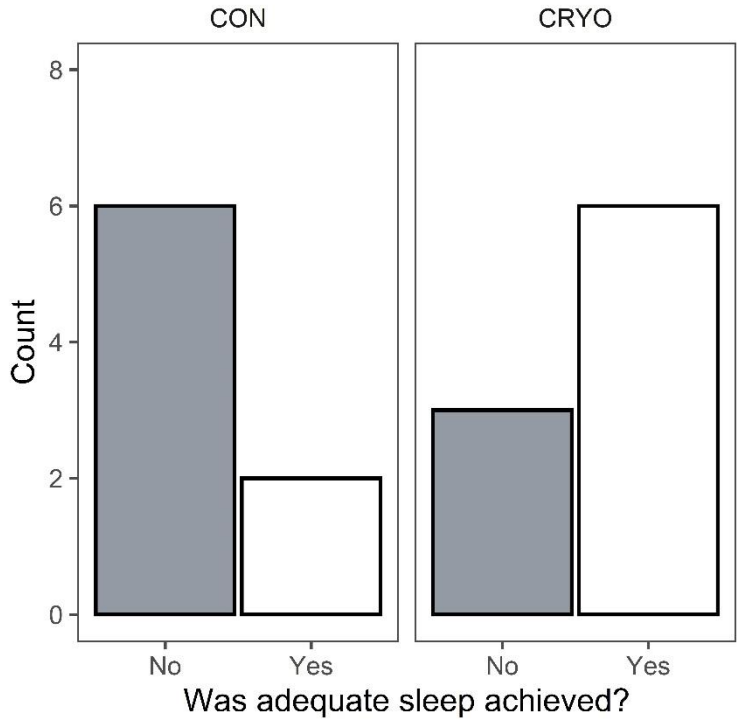

Figure 21: Number or players whose mean weekly sleep ≥420 minutes or higher.

Perceived BFW in the CON group was significantly lower compared to the CRYO group (CON: 49 ± 17, CRYO: 62 ± 11; p=0.001). No significant differences were observed between groups for GTS (CON: 45 ± 11, CRYO: 49 ± 17; p=0.33), QS (CON: 46 ± 13, CRYO: 51 ± 18; p=0.25), or AFS (CON: 51 ± 16, CRYO: 57 ± 17, p=0.17). By individual day, AFS was significantly higher (p=0.048) in the CRYO group (59 ± 11mm) compared to CON (48 ± 10mm) on day 2. Likewise, BFW was significantly higher (p=0.014) in the CRYO group (57 ± 10mm) compared to CON (53 ± 19) on day 3. There were no further significant differences in perceived sleep metrics (Figure 22).

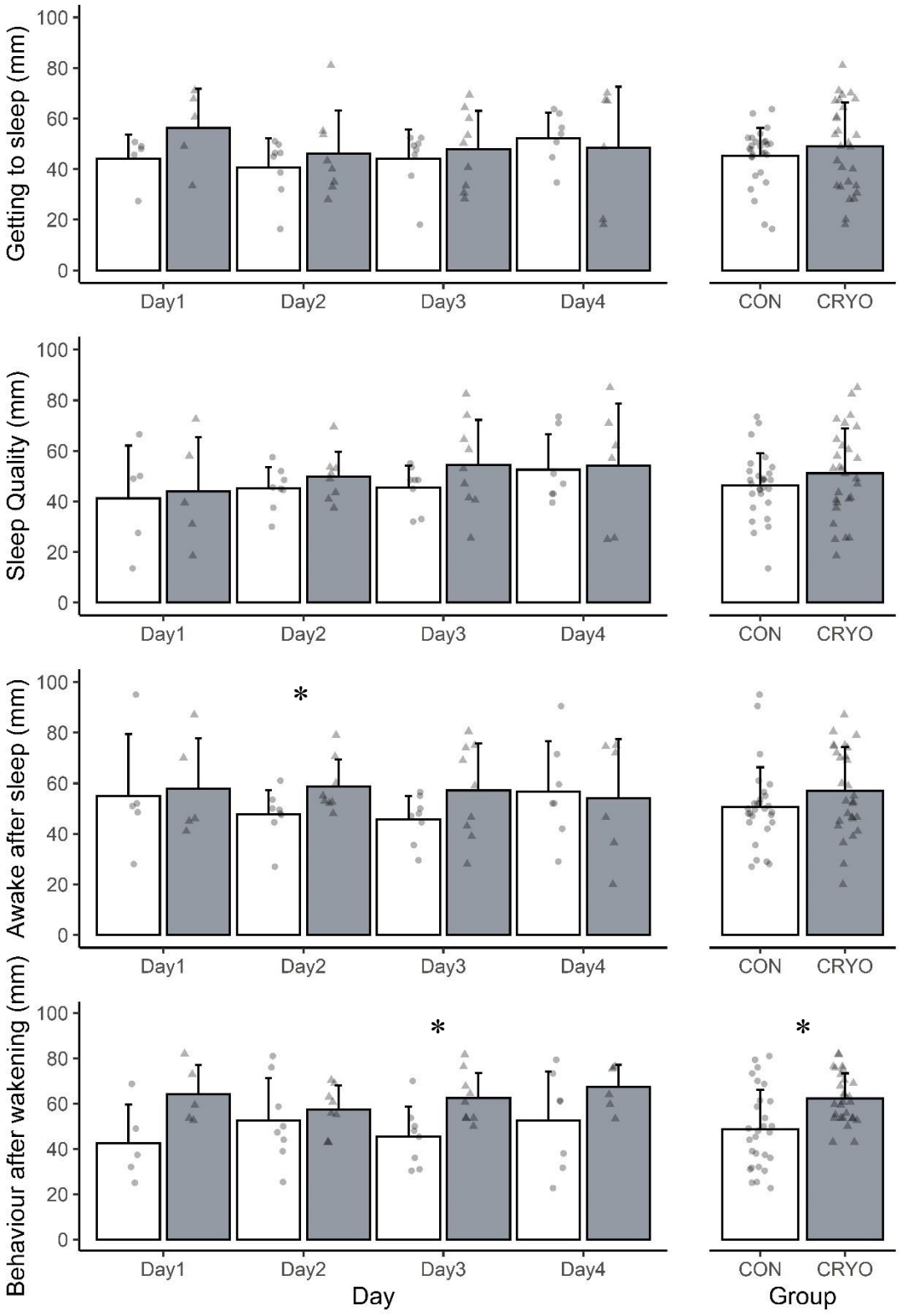

Figure 22: Leeds sleep evaluation questionnaire results displayed by day (left) and weekly mean (right). CON is shown in white and CRYO in grey. *Indicates significance between groups ($p<0.05$).

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3.4.2. Saliva endocrines and serum hsCRP

Inter-assay agreement between duplicate samples were high (testosterone: R^2= 0.94, cortisol: R^2= 0.84). No within-group significant differences in cortisol levels were detected between GD1 and GD2 in both the CON (GD1: 120 ± 40.1 pg/ml, GD2: 107.9 ± 24.4 pg/ml; p=0.525) and the CRYO groups (GD1: 184.3 ± 71.6 pg/ml, GD2:167.3 ± 68.5 µg /ml, p=562). However, testosterone significantly decreased from GD1 to GD2 in both the CON (GD1: 401.5 ± 162 pg/ml, GD2: 315.4 ± 123.8 pg/ml, p=0.031) and CRYO groups (GD1: 592.9 ± 146 pg/ml, 352.9 ± 146.1 pg/ml, p=0.028). When the change scores (differences between GD1 and GD2) were compared between groups, no significant difference was revealed in cortisol (CON: -12.3 ± 39.5 pg/ml, CRYO: -16.9 ± 59.9 pg/ml, p=0.89), or testosterone (CON: -86.1 ± 59.9 pg/ml, CRYO: -239.3 ± 157.9 pg/ml). Whilst the mean change score between GD1 and GD2 for testosterone trended towards a reduction from GD1 to GD2, results did not reach the significance threshold (p=0.097) (Figure 23A&B).

Likewise, there was no within-group significant difference in hsCRP levels between GD1 and GD2 in both the CON (GD1: 0.55 ± 0.053 mg/L, GD2: 0.59 ± 0.13, p=0.584) and the CRYO groups (GD1: 0.66 ± 0.2, GD2: 0.62 ± 0.21, p=0.834). There was also a significant difference when the hsCRP change scores (differences between GD1 and GD2) were compared between groups (CON: 0.048 ± 0.13, CRYO: -0.039 ± 0.29, p=0.695) (Figure 23C).

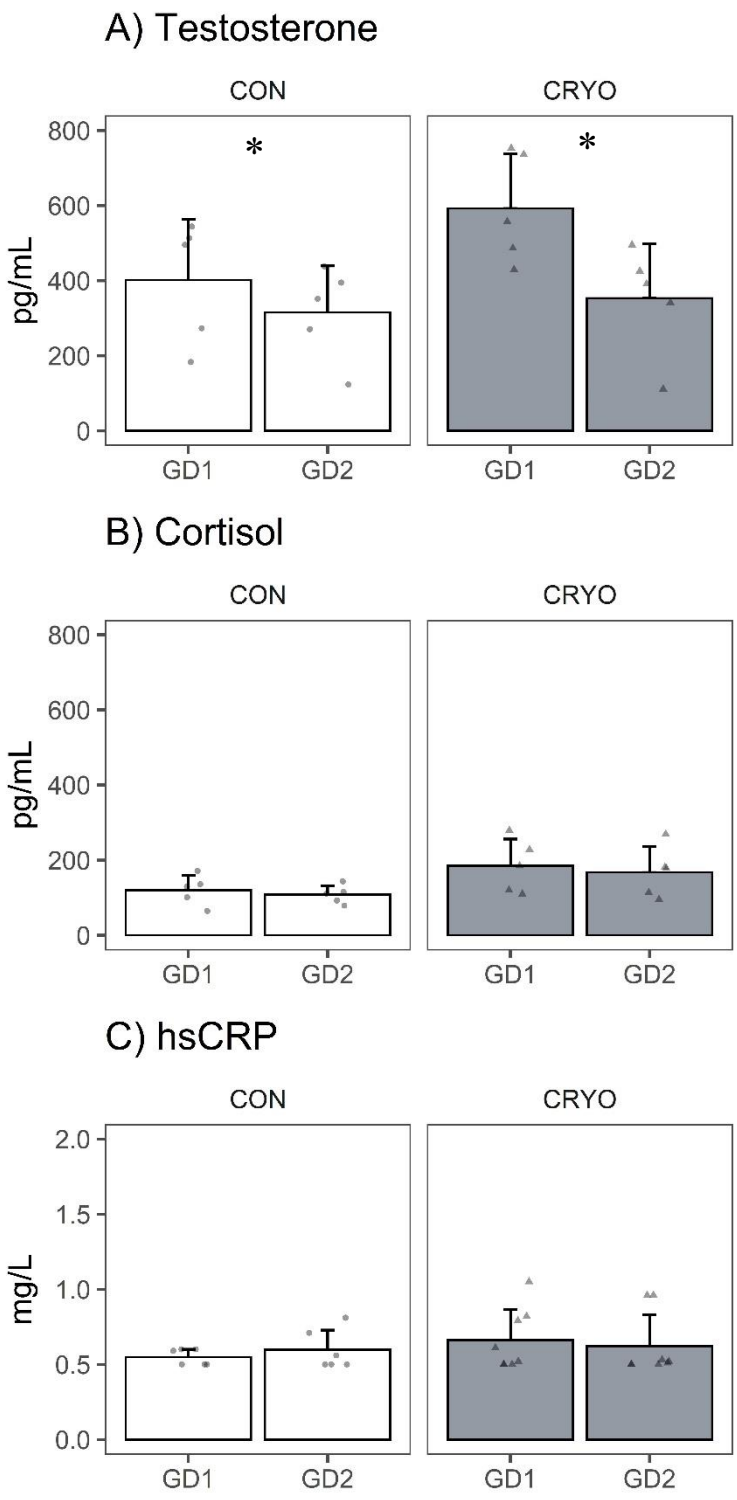

Figure 23: Cortisol (A), testosterone (B), and high sensitivity C-reactive protein (hsCRP; C) on GD1 (Game day 1) and GD2 (Game day 2). *Indicates a significant difference between game days ($p<0.05$).

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3.5. Discussion

The purpose of this study was to investigate the effect of daily WBC exposures on the objective and subjective sleep quality in U18 professional footballers, during an in-season period and assess any subsequent effect on game-day testosterone and cortisol. This is the first time a study of this type has been completed in professional football players and was originally designed as a crossover study; however, the second phase was interrupted by the United Kingdom national lockdown and restrictions. The primary finding of this study is that WBC does not impact objective sleep quality, although, there were improvements in perceptions of behaviour following wake.

There was no significant difference in activity-derived sleep metrics between groups when data were analysed by day or by week. The literature base is conflicting, nevertheless, these results are in agreement with other studies. In one report [359], post-exercise WBC, compared to a passive control, had no significant effect on activity monitor sleep metrics when healthy males engaged in high-intensity cycling over a 4-week intervention. Likewise, in professional rugby players, polysomnography recordings suggested that post-fixture WBC provided no significant benefit to sleep metrics, compared to no treatment [368].

In contrast, some studies reported improved sleep after WBC exposure. In Olympic synchronised swimmers, Schaal et al [308] reported reductions in objective sleep duration during an intensified training week. However, when participants received daily WBC, objective sleep disruption was significantly attenuated. In trained healthy males who were subject to evening exercise, WBC resulted in less activity monitor derived nocturnal movements compared to a passive control [299]. A beneficial, dose-related decrease in nocturnal movements has also been reported in professional football players in response to partial-body cryotherapy (PBC) [311]. However, although the therapies are similar, investigations have highlighted different thermoregulatory and physiological responses between WBC and PBC [313,314]. Therefore, it is not clear if they mediate a synonymous therapeutic response.

The reasons for the variance between studies are not clear, and a plethora of unknown confounders may account for the disparities between investigations. Such factors may include the number of exposures, the timing of WBC relative to bedtime, differences in exposure temperature, or intra/interindividual variation in sleep metrics. However, in the studies available, there is no clear pattern to confirm or reject these variables [299,308,359,368]. It is also important to note that the present study applied WBC during an in-season microcycle, during the lead-up to a competitive fixture. Other studies that have examined WBC effect in athletes have monitored sleep during the night proceeding a competitive fixture [368], or during a pre-event training camp [308]. Consequently, there may be factors relating to workload and psychological stressors that may contribute to differences between studies.

Despite no significant difference in sleep metrics, this study reports that 66.67% of players in the CRYO group achieved a weekly mean sleep duration that was equal to or above the minimum threshold suggested by the NSA [371], compared to 25% of the CON group. Whilst this cannot be robustly used to support WBC as a sleep aid, it does tentatively imply a potential benefit and provides pilot data; although, the lack of a cross-over arm severely limits the analysis. This is further supported by the results of the subjective LSEQ, where players reported a significantly greater mean weekly perception in BFW in the CRYO group, compared to the CON group. BFW is a collated score that considers perceptions of alertness on waking, alertness while completing the LSEQ, and balance/ coordination on waking. Practically, this suggests that players who received WBC perceived feeling more alert immediately after waking up which, in turn, suggests that WBC may have supported restorative sleep in those players.

Only one other study has reported subjective makers after WBC, and results are largely in agreement with the present study. Using the Spiegel's questionnaire, Douzi et al [299] reported significantly improved self-reported sleep quality after participants received post-exercise WBC, compared to when they did not. Notably, the component that assessed morning mood state was significantly higher after WBC which supports the increased BFW that was reported here. Whilst this may be symptomatic of an improved overall sleep quality resulting in improved alertness, mechanistic studies have also reported increases in dopamine, a neurotransmitter associated with feelings of well-being, pleasure, and motivation, after ~15 minutes post-WBC [314]. Consequently, the improved alertness following wake may be due to the latent influence of dopamine. However, studies have only noted relatively small dopamine increases compared to controls (Cohen's d= 0.28 ± 0.33) and its release is sympathetically mediated [314]. Although the initial cold immersion response to WBC is primarily initiated by sympathetic pathways, studies have noted post-WBC parasympathetic predominance that persists for at least 6hrs post-exposure [299,313]. Consequently, it is not clear if the dopamine response is sufficient to alter mood the morning after an exposure. Research that examines this further may enable practitioners to better position WBC within the training day and microcycle. However, WBC chambers are large and have expensive installation and operating costs, and other ergonomic sleep aids may offer greater value and efficacy. Aloulou et al [368] reported no significant effect of post-game WBC on polysomnography readings in professional rugby players. Yet, in the same study, a thermal mattress (a mattress designed to support the dissipation of heat during sleep) mediated reductions in WASO, and improved sleep architecture (as determined by polysomnography) compared to a control. Further, in semi-professional footballers [365], a simple sleep hygiene strategy that limited device use and light exposure before bedtime resulted in significantly increased sleep duration after competitive fixtures. Consequently, practitioners may want to invest their resources into other sleep strategies before utilising WBC as purely a sleep aid.

This study sampled saliva on GD1 and GD2, justified by the fact that it is theoretically the most rested and repeatable point in the microcycle (eg. players training would taper for GD). Results indicated that testosterone statistically decreased from GD1 to GD2 in both the CON and CRYO groups, however, when the changes scores were compared, there was no statistical difference between CON and CRYO. Nor was testosterone significantly correlated with objective or subjective sleep metrics. While the decreases in testosterone from GD1 to GD2 may represent differences in the physiological and/or psychological profile across the preceding microcycle [369], overall, this study suggests that WBC applied daily across the microcycle has no effect on GD testosterone levels. Other studies have also observed no significant effect of WBC on testosterone. For example, in healthy males who engaged in muscle-damaging exercise, post-exercise WBC did not significantly alter testosterone kinetics compared to a passive control condition [373].

These results may be isolated. During a recovery camp for high-level tennis players, Zieman et al [126] reported that testosterone levels in athletes were higher in those who received WBC. The higher frequency of WBC exposures (2 times per day for 5 days) may account for the differences in results, although, another study has observed increases after a single exposure [310]. Academy football players representing a professional club completed who completed a muscle-damaging exercise regime (sprints with deceleration phase), post-exercise WBC resulted in significantly higher testosterone levels when sampled at both 2 and 24 hrs post-exercise [310]. Both these studies utilised WBC in a recovery capacity, either during a mid-season recovery camp [126] or immediately after a bout of muscle-damaging exercise [310]. The present study used WBC during an in-season microcycle on the days preceding a competitive fixture. During this time, the workload will most likely be in taper in preparation for performance. Therefore, different interactions between WBC and exercise intensity may account for the differences between studies.

No differences were observed in cortisol levels between GD1 and GD2 in both the CON or the CRYO group, nor were there any significant differences in change scores between groups. This indicates that daily WBC utilised during the days leading up to a competitive fixture does not significantly modulate cortisol in professional players. Considering the players were potentially tapering for GD, the lack of response may be due to the absence of an exercise induce stimulus sufficient to stimulate cortisol production. That said, Russel et al [310] also did not observe a significant on cortisol after academy footballers representing a professional club received WBC immediately after performing muscle-damaging exercise. Further, no effect of WBC was observed in high-ranking tennis players [126] or Olympic synchronised swimmers [308] who were engaged in a recovery camp and an Olympic preparation camp, respectively. Contrastingly, another study reported significantly greater cortisol levels, compared to baseline, in rowers by day 6 of an Olympic training camp. However, there was no such change in athletes who received daily WBC [336]. This study, although, subjected players to 3

WBC sessions per day for the duration of the training camp. Therefore, the higher frequency may have resulted in a statistically significant response.

This study reports no significant effect of WBC on GD hsCRP, suggesting WBC used daily 4 days before a competitive fixture does not impact acute phase inflammation. CRP is often used alongside other markers (e.g., creatine kinase, delayed onset muscle soreness) to assess exercise-induced muscle damage severity and recovery [81]. Therefore, the lack of change in hsCRP may be due to the fact that players are in a relatively rested state in preparation for their fixture. Other studies have investigated the effect of post-exercise WBC on CRP; however, results are mixed. Pournot et al [127] instigated muscle damage through a running exercise with downhill segments, followed by either WBC or a passive control. Significantly lower CRP levels in were observed at 1hr post-exercise in the WBC group and remained significantly lower compared to the control at 96hrs post-exercise. In contrast, after high intensity running (without downhill segments), Kruger et al [373] reported that WBC had no significant impact on CRP, compared to a passive control. Yet, in this study, CRP was not observed to increase from baseline until 24 hours post-exercise. Therefore, differences in exercise modality, and the resultant effect on CRP kinetics, may have contributed to the differences between studies.

This study has several limitations. Most notably, the lack of a crossover phase severely limits the strength of the conclusions that can be made. In its present form, within-participant comparisons cannot be made and so the residual analysis cannot account for any intra-individual variation. Further, where the majority of other studies apply WBC in a post-exercise capacity or during a specialised training camp, the present study is set during an in-season microcycle, and this limits robust comparisons with other reports. Nevertheless, this study remains relevant as it mimicked what was currently being applied within the club.

In conclusion, WBC applied daily 4 days before a competitive fixture appeared not to affect objective sleep metrics, however, players who received WBC reported better behaviour following wake. This may suggest that WBC can be used to improve subjective readiness on game days. Despite this, no significant differences were observed in cortisol, testosterone, or hsCRP. Consequently, WBC used during the taper phase of a microcycle does no impact on anabolic/catabolic endocrine function or inflammatory state. WBC may be used to increase the perception of alertness in professional U18 football players.

Chapter 4

4. How well do professional football (soccer) players sleep? A systematic scoping review of observational studies (Literature review part 3)

After the outbreak of the COVID 19 pandemic, the whole-body cryotherapy chamber was not considered covid safe. Therefore, the original proposed scheme of work could not be completed. Subsequently, the central theme of this thesis refocussed to measuring, monitoring, and improving sleep in professional football players. This chapter consolidates that change and forms a foundation for further work.

4.1. Abstract

There is a growing literature base surrounding sleep in professional football (soccer) players, yet, despite the number of observational studies on the subject, there have been no systematic reviews. The aim of this scoping review was to describe what is known about sleep in full-time professional footballers and identify the main investigative themes concerning factors that may influence sleep in this population. From inception until November 2022, Web of Knowledge, PubMed, and SPORTDiscus were searched, and observational studies were included if they reported objective or subjective sleep data in professional footballers. Of the included studies (n= 1495, 84% male, age: 23.0 ± 3.4 years), 33 used subjective methodologies, 6 utilised objective, and 6 used both in mixed method designs. Sleep duration, wake after sleep onset, and sleep onset latency scores across studies were within guidelines, however, error scores suggest suboptimal scores are common. The variability could be a result of psychological factors associated with matchdays, workload, competitive scheduling, or intraindividual confounders. Scheduling factors and their effect on sleep were identified as a primary literature theme across the literature base with night matches, compared to training days, and travel was highlighted as factors that may influence sleep. The effect of workload on sleep has also received notable investigative interest, although there was little to substantiate a meaningful relationship. Overall, this review highlights that sleep disruption is common, however, players mean sleep is within guidelines

4.2. Introduction

Sleep loss protocols have demonstrated impairments in anabolic signalling [14], cognitive function [374], motor skill acquisition, and memory consolidation [192,193,360]. From an athletic standpoint, this implies that sleep disruption can hinder physiological and psychological recovery and performance [14,375]. Despite its perceived importance, sleep quality in athletes is generally considered suboptimal compared to aged-matched controls [20]. This may also be true in football (soccer) players [46,376] who are regularly exposed to factors that may disrupt sleep [363,377], and often present with significant inter/intra-variation. Therefore, an understanding of the factors that affect sleep in this population is warranted.

Practitioners have access to a number of research- and commercial-grade tools that can support the assessment of sleep behaviour in their players [378]. Wearable [273,274,379,380] (eg. wrist-worn activity-monitors) and nearable [16,381] (e.g., bedside devices) technologies provide an accessible method to objectively monitor players sleep outside of the laboratory and, whilst there is a tendency for such devices to misinterpret sleep markers relative to the gold-standard polysomnography (PSG) [378], validated devices have been used to assess the influence of factors including travel [226,277] and day type [275] on sleep in footballers. Several subjective methodologies are also available enabling cross-sectional and longitudinal evaluation of player's sleep [206,382]. Whilst perceptions can be biased by mood, memory and other factors [247–249], subjective methodologies facilitate an inexpensive evaluation of players perceived sleep quality (36).

The application of sleep assessment tools in the published literature is becoming more frequent [16], with increasing amounts of data examining the quality [383], quantity [226,277], and factors that may affect sleep in professional footballers [274]. Despite this, the aetiology of sleep disruption in football is not clear [7,46,354] and there no study has systematically collated the available data from professional football players. Therefore, the purpose of this study was to describe what is known about sleep quality and quantity, in relation to published norms [371], and identify the main literature themes concerning barriers to optimal sleep by systematically examining observational studies that have monitored sleep in full-time, professional footballers. Due to the lack of commonality between methodological elements in observational studies, a scoping review approach was judged to be the most appropriate review method.

4.3. Methodology

This systematic scoping review of observational studies was performed following guidance from the *Conducting Systematic Reviews and Meta-Analyses of Observational Studies of Etiology* (COSMOS-E [384]) and *Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping*

Reviews (PRISMA-ScR; Figure 24 [385]). The research questions were shaped using a participant, exposure, control, outcomes (PECO) framework [384]).

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
TITLE			
Title	1	Identify the report as a scoping review.	111
ABSTRACT			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	112
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	114
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	114
METHODS			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	na
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	114
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	114
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	114
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	114
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	114
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	114
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).	116
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	116

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
RESULTS			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	117
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	111
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	116
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	table 7
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	7
DISCUSSION			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	126
Limitations	20	Discuss the limitations of the scoping review process.	throughout
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	136
FUNDING			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	na

JBI = Joanna Briggs Institute; PRISMA-ScR = Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews.
* Where *sources of evidence* (see second footnote) are compiled from, such as bibliographic databases, social media platforms, and Web sites.
† A more inclusive/heterogeneous term used to account for the different types of evidence or data sources (e.g., quantitative and/or qualitative research, expert opinion, and policy documents) that may be eligible in a scoping review as opposed to only studies. This is not to be confused with *information sources* (see first footnote).
‡ The frameworks by Arksey and O'Malley (6) and Levac and colleagues (7) and the JBI guidance (4, 5) refer to the process of data extraction in a scoping review as data charting.
§ The process of systematically examining research evidence to assess its validity, results, and relevance before using it to inform a decision. This term is used for items 12 and 19 instead of "risk of bias" (which is more applicable to systematic reviews of interventions) to include and acknowledge the various sources of evidence that may be used in a scoping review (e.g., quantitative and/or qualitative research, expert opinion, and policy document).

Figure 24: Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews checklist

4.3.1. Search strategy

From inception until April 2022, Web of Knowledge, PubMed, and SPORTDiscus were searched using a glossary of search terms that included "Football", "Soccer", "Sleep" and terms relating to objective and subjective sleep assessment, in conjunction with Boolean logic terms (Table 6). Articles were exported to reference management software (Mendeley, London, UK) and duplicates were removed. The remaining cases were screened independently by two authors (LE, CP). Any disagreements were reconciled with a third author (JH).

Table 6: Chapter 4 search strategy

Database	Search terms and Boolean logic
Web of knowledge	(“soccer” OR “football”) AND (“Sleep*”) AND (“quality” OR “quantity” OR “hygiene” OR “actigraphy” OR “polysomnography” OR “subjective” OR “objective” OR “sleep diary” OR “questionnaire” OR “survey”) Start: 1900 End: 2022-11-30
PubMed	(“soccer” OR “football”) AND (“Sleep*”) AND (“quality” OR “quantity” OR “hygiene” OR “actigraphy” OR “polysomnography” OR “subjective” OR “objective” OR “sleep diary” OR “questionnaire” OR “survey”) Custom range: from 1000/1/1 to 2022/11/30
SPORTDiscus	(“soccer” OR “football”) AND (“Sleep*”) AND (“quality” OR “quantity” OR “hygiene” OR “actigraphy” OR “polysomnography” OR “subjective” OR “objective” OR “sleep diary” OR “questionnaire” OR “survey”) Start: blank End: Nov 2022

4.3.2. Eligibility criteria and data extraction

Studies were included if they monitored sleep objectively or subjectively in professional footballers (full-time contracted athletes, with no additional work or education) using an observational design. Studies were excluded if there was no within-study comparison (e.g., training days versus match days), or sleep metrics were not reported in standardised units (e.g. minutes or results from questionnaires, e.g. the Pittsburgh Sleep Quality Index (PSQI)). Case studies on a single participant were also not eligible. No eligibility criteria were placed on competitive or playing phase, sex/gender, or geographical location.

4.3.3. Data Extraction

Data were extracted and collated based on emerging themes, developed by highlighting trends in the literature. If reported, data relating to sleep duration, sleep onset latency (SOL), and wake after sleep onset (WASO) were extracted for data visualisation purposes using R statistical environment (The R Foundation for Statistical Computing; ggplot2 [386])

4.3.4. Risk of bias

Risk of bias (RoB) was assessed for each study according to the domains and guidance described in the COSMOS-E [384] and supported by the RoB of exposures [387]. The bias domains were confounding variable bias, participant selection bias, outcome measurement bias, exposure measurement bias, missing data bias, and information bias. Signalling questions were used to guide assessments and are listed in Chapter 4 supplementary materials (Appendix 1: Chapter 4 supplementary materials).

4.4. Results

A total of 1103 studies were identified through database searches and, after duplicates were removed (n= 473 studies), 525 studies were excluded following title and abstract screening. The remaining 105 studies were assessed for relevance and 60 were excluded due to: not observational, not professional players, contained an intervention, and no quantitative sleep data. Subsequently, 45 studies were included for analysis (Figure 25). Furthermore, the following themes emerged that described factors influencing sleep: match days, night matches, intra and inter-microcycle variation, inter-season variation, long-haul travel, and external workload.

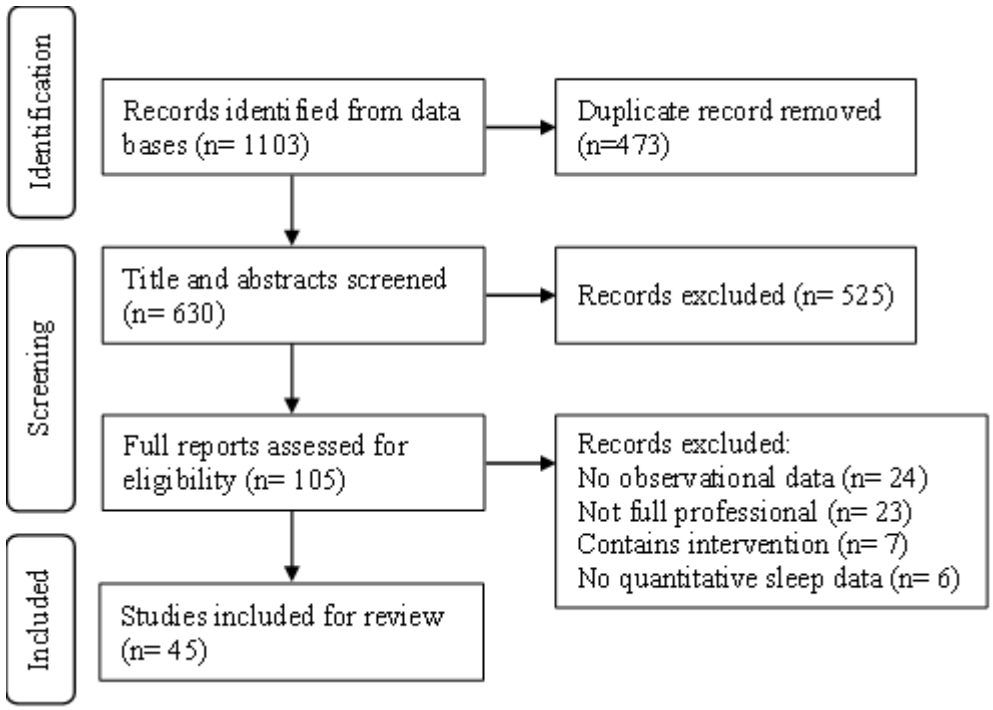

Figure 25: Study selection flow chart

4.4.1. Study characteristics

Of the 45 studies included (n= 1495, 84% male, age: 23.0 ± 3.4 years), 34 studies involved players from senior 1st teams (n= 1348, 83% male, age: 24.4 ± 2.5 years), 2 studies included players from U23 teams (n= 20, 100% male, age: 20.3 ± 0.8 years), and 9 studies were set in professional academies (n= 127, 100% male, age: 18.1 ± 0.6 years). By location, 27 were set in European leagues (n= 633, 94% male, age: 22.6 ± 3.7 years), 8 in Australian leagues (n= 374, 65% male, age: 24.1 ± 2.5 years), 10 in Middle Eastern leagues (n= 371, 84% male, age: 22.8 ± 2.7 years), and two were set in South America (n=117, 100% male, age: 25.8 ± 0.8 years). Thirty-three studies used only subjective monitoring (n=1308, 84% male, age: 23.7 ± 3.4 years), 6 studies used only objective monitoring (n=98, 82%, age: 20.3 ± 3.4), and a further 6 studies combined both objective and subjective assessments (n= 61, 100% male, 23.1 ± 3.6) (Table 7)

Table 7: Included studies that met the eligibility criteria describing sleep variables in professional football players.

Study, design	Participant details Setting, training phase	Observation length and frequency	Sleep assessment method	Outcome variables	Primary Analysis	Primary findings
Abbott et al 2020a [272], longitudinal	U23, Age: 20.0 ± 1.0 years, n= 10, m, England, in-season period	35 competitive matches across 1 season, post-game	Brief assessment for mood (BAM+)	Subjective: Sleep quality DOMS Fatigue Mood Stress	Differences in feelings of wellness after games in relations to season progress, match result, match location and quality of opposition	Sleep not affected by negative match results. Better sleep quality in early and midseason compared to late season
Abbott et al. 2018 [362], longitudinal	U23, Age: 19.5 ± 1.2 years, n= 11, m, England, in-season period	17 competitive matches, daily	In-house questionnaire including sleep assessment	Subjective: Sleep quality DOMS Fatigue Mood Stress	Subjective feelings of wellness (including sleep) before and after competitive matches in relation to opposition quality, result and distance to fixture.	Subjective sleep was worse after away matches and losses
Ballesio et al 2021 [388], cross-sectional	Senior, Age: 25.0 ± 6.7 years, n= 210, m, Italy, in-season period	One off observation	ISI	Related metrics	Relationship between psychological factors and ISI	Significant correlations between variables and ISI
Carriço et al. 2018 [273], longitudinal	Professional footballers, Age: 26.3 ± 4.7 years, n= 25, m, Portugal, in-season period	Season long, 3 training days, then every game	Activity monitor	Objective: Bedtime Wake time Time in bed Sleep duration SOL Sleep efficiency WASO	Effect of match scheduling (eg. Away and home, day and night) on subsequent sleep.	Significant differences in key sleep variables between TD, HM and DM
Costa et al 2022 [389]	Academy players, Age: 17.9 ± 0.4 years, n= 13, m, Portugal, pre-season	16 days, daily	Activity monitor	Objective: Sleep duration Sleep efficiency Subjective: Sleep quality	Comparison of single and dual occupancy rooms on sleep	Reduced sleep quantity in dual occupancy rooms compared to single

Delaval et al 2022 [390], longitudinal	Professional football players, age: 24.2 ± 4.7 yrs, n= 46, France (Ligue 1), in-season	2 seasons, daily	Hooper questionnaire	Related metrics	Relationship between recover metrics (including sleep) and non-contact injury	No relationship between **Subjective** sleep and injury occurrence
Douchet et al 2021 [391], longitudinal	Professional football players, Age: 24.2 ± 2.3 years, n= 12, f, France, in-season	2 weeks, weekly	Hooper questionnaire	Related metrics	Effect of "heavy" and "low" intensity weeks	Sleep quality was rated significantly worse at the end of the "heavy" week, with no change during "low" week.
Evans et al 2022 [392], longitudinal	Elite youth football players, Age:18 ± 1 years, n= 16, m, England, in-season	36 matches, daily	Wellness questionnaire	Subjective: Sleep quality	Explore efficacy of wellness scores to detect post-match fatigue	Pre-match sleep scores associated with number of accelerations and decelerations
Fernandes et al 2022 [383], longitudinal	Professional football players, Age:24.6 ± 2.3 years, n= 10, f, Portugal, in-season	7 months, daily	Hooper questionnaire		Quantify internal and external intensities across a microcycle	no significant change in sleep across micro cycle
Fessi and Moalla 2018 [393], longitudinal	Professional footballers, Age: 25.6 ± 3.6 years, n= 12 Qatar, in-season	2 seasons, post competitive fixture, pre-recovery.	7 point psychometric questionnaire (including sleep quality)	Subjective: RPE Sleep quality Fatigue	Match result on outcome variables.	Reduced perceived sleep quality following competitive defeat
Fessi et al. 2016 [394], longitudinal	Professional footballers, Age: 23.7 ± 3.2 years, n= 17 Qatar, Pre- and in-season period	Season long, pre training and competitive fixture	Hooper questionnaire	Related metrics	Comparison between pre- and in-season periods.	Greater perceived sleep quality in the pre-season phase compared to in-season.
Fitzpatrick et al. 2019 [395], longitudinal	Youth soccer players, Age: 17.5 ± 0.5 years, n= 12 England, in-season	2 weeks	Subjective wellness (including sleep)	Sleep quality	Reproducibility of wellbeing metrics (including sleep quality) over two weeks.	Subjective sleep quality was not reproducible across two consecutive weeks
Fowler et al 2014 [396], longitudinal	Professional footballers, Age (CI):23.4 (19.9-25.9), n = 6, m, Australia, in-season	12 matches (2 days pre-match, match day, two days post-match)	Activity monitor Likert scale	Objective: Sleep duration Bedtime Wake time SOL Sleep efficiency Wake episodes WASO	Acute effects of short-haul travel during a micro cycle on sleep	no significant differences between home and away matches for sleep

				Subjective: Sleep quality		
Fowler et al. 2015 [227], longitudinal	Professional football players, Age (CI): 27.0 years (25.0–29.0), m, Australia, in-season	12 matches (2 days pre-match, match day, two days post-match)	Activity monitor, sleep diary	Objective: Sleep duration	Effect of northbound travel on sleep duration and jet-lag	Sleep negatively affected on travel days
Fowler et al. 2017 [397], longitudinal	Professional football players, Age: 26 ± 4 years, m, Australia, in-season	1 week prior to, and 5 days post long-haul travel	Sleep diary	Subjective: Bedtime Wake time SOL Sleep duration WASO	Effects of long-haul air travel from Australia to Brazil on **Subjective** jet-lag, sleep and wellness responses in professional football players	Sleep responses affects by east bound long haul travel
Fullagar et al 2016a [277], longitudinal	Professional footballers, Age: 25.5 ± 4.9 years, n= 15 Netherlands, pre-season	10 days with 3 day baseline	Activity monitor BL measures completed by survey	Objective: Sleep duration Bedtime Wake time SOL Sleep efficiency Wake episodes WASO	Sleep quality after outbound and return flights and match days	Sleep duration affected by long-haul travel and night matches
Fullagar et al. 2016b [274], longitudinal	Professional football players, Age: 25.9 ± 7.5 years, n= 16, m, Germany and Netherlands, in-season	3 weeks, daily	Sleep and sporting activity questionnaire Sleep diary	Subjective: Bedtime Wake time SOL Sleep duration WASO Restfulness Nap duration	Sleep quality after training days, day matches and night matches	Reduction in sleep duration and a later bedtime after NM after TD and DM
Jorquera-Aguilera et al. 2021 [398], cross-sectional	Professional football players, Age: 25 ± 5.3 years, m, n= 94, Chile, Primera Division	Single observation	Sleep diary PSQI	Sleep duration SOL Bedtime Related metrics	Comparison of sleep quality between four Primera Division clubs	Mean PSQI was <5 and no significant dif. was reported between clubs.

Khalladi et al 2019 [206], cross-sectional	Professional footballers, Age: 23.7 ± 4.8 years, n= 111, m Qatar, in-season microcycle	14 days, daily	PSQI ISI ESS	Related metrics	Frequency and percentage of players that reached the clinical threshold of the respective tests.	High prevalence (68.5%) of sleep disorders in longitudinal.
Kilic et al 2021 [399], cross-sectional	Professional football players, Age: m24.3 ± 4.8 years, f22.8 ± 4.0years, n=281, m=149, f=132, Australian A- and W-Leagues, respectively	Single observation	Athlete sleep screening questionnaire	Related metrics	Prevalence of disrupted sleep between male, female, and former football players	Sig. more prevalent disruption in former players compared to male current players
Lastella et al 2019 [226], longitudinal	Professional footballers, Age:25.2 ± 3.2 years, n=7, m, Australia, Asian Champions League and related travel	19 days, daily	Activity monitor Sleep diaries	Objective: Bedtime Wake time Time in bed Sleep duration SOL Sleep efficiency Subjective: Bedtime Wake time Time in bed Sleep duration SOL Sleep efficiency	Assess sleep metrics before and during a period of international travel.	Compromised sleep patterns during travel
Lozano et al 2022 [400], longitudinal	Professional football players, age: 25.37 ± 3.60 yrs, n= 31, Spain, in-season	1 season, daily	Hooper questionnaire	Related metrics	Effect of microcycle length on perceived wellness (including sleep)	No significant relationship between length of microcycle and sleep
Mateus et al. 2021 [401], longitudinal	Professional football players, Age: 26.1 ± 3.9 years, n= 13, m, Spain, Segunda División (Spanish second division)	16 weeks, daily	Customised wellness questionnaire	Subjective: Sleep duration scale (1 to 10 scale) Perceive sleep quality	Relationship between perceived sleep and training sessions organised by intensity and activity	No relationship was observed

Moalla et al. 2016 [402], longitudinal	Professional footballers, Age: 25.7 ± 2.6 years, n= 14, pre-season and in-season	16 weeks, daily	Hooper questionnaire	Related metrics	Relationship between Hooper index and internal load	Significant correlation between training load and sleep
Nédélec et al. 2019 [361], longitudinal	Professional football players, Age: 26.0 ± 4.6 years, n= 20 (12 training days, 7 night games), m, France, in-season	12 training days + 5 night games over 3 week period, 6.1 ± 3.2 nights per player	Activity monitor Sleep diary	Objective: Bedtime Wake time Time in bed Sleep duration SOL Sleep efficiency Subjective: Sleep quality	Sleep quality of training days compared to night matches (n= 7).	Time in bed and sleep duration we decreased after NM compared to TD
Nobari et al. 2021 [403], longitudinal	U17, Age: 16.1 ± 1.4 years, n= 21, m, Country not stated, pre- and in-season	One season, daily	Hooper Questionnaire	Related metrics	Perceived sleep quality across meso-cycles and perceived sleep quality by positions	Sig. greater perceived sleep quality during early-season compared to mid-season. No sig. for playing position.
Noon et al. 2015 [404], longitudinal	U17 to U21 academy players, Age:17 ± 1 years, n= 14, England, pre- and in-season	One season, 1 to 4 times per week	Subjective wellbeing questionnaire that includes sleep quality	Related metrics	Comparison between pre-season and three in-season training blocks.	Decrease in sleep quality and other wellbeing metrics over the season.
Noor et al 2021 [405]	Professional footballers, Age: 26.4 ± 4.1 years, n= 37, m, Australian, in-season	42 days, daily	Hooper questionnaire	Related metrics	Effect of match day load on self-reported fatigue profiles during congested and non-congested periods.	Reduced post-match sleep quality/quantity in 2 match microcycles
Oliveira et al. 2021 [406], longitudinal	Professional football players, Age: 28 ± 2.8 years, n=9, EU, UEFA Champions league	One season, daily	Hooper Questionnaire	Related metrics	Quality of oppositions, match location, and location on sleep when two matches were played in a 7 day period	High values of sleep quality on the day following and away-win against top-level opponent
Oliveira et al. 2022 [407]	Professional football players, age: 26.2 ± 3.5 yrs, n=17, Europe, in-season	10 mesocycles (months), daily	Hooper questionnaire	Related metrics	Variation of sleep across mesocycles, positions, and starters/non-starters	Significant difference between starters and non-starters during the first mesocycle

Olivera et al [408]	Professional football players, Age: 26.3 ± 4.3 years, n= 18, m, Portugal, in-season	39 weeks, daily	Hooper questionnaire	Related metrics	Changes across mesocycle and microcycle	Differences across microcycle but not mesocycle
Robey et al. 2013 [276], longitudinal	Professional football players, Age: 18.5 ± 1.4 years, n= 12, m, Australia, in-season, regular eastward travel (one time zone)	7 weeks, Tues to Thurs, inclusive, only (3 nights each week)	Activity monitor	Objective: Bedtime Wake time Sleep duration SOL Sleep efficiency WASO Subjective: RPE Rating of fatigue Rating of recovery	Sleep quality after training, and on rest days.	No differences between sleep quality and quantity on training and rest days.
Saidi et al [409]	Professional football players, Age: 20.9 ± 0.8 years, n= 14, m, Tunisia, in-season	12 weeks, 3 times per week	Hooper questionnaire	Related metrics	Changes in wellness in relation to changes in training and match exposure	Sleep was unaffected by changes in load
Selmi et al [410]	Professional football players, Age:25.0 ± 1 years, n=15, Tunisia, Pre-season	2 weeks, daily	Hooper questionnaire	Related metrics	Sleep response to an intensified training period	no significant change
Selmi et al. 2020 [411], longitudinal	Professional football players, Age: 24.0 ± 1 years, n= 15, m, Tunisia, pre-season	6 weeks, daily	Hooper questionnaire	Related metrics	Examine the change in perceived sleep quality after a period of intensified training	No significant effect of intensified training on sleep
Silva et al 2021 [412]	Professional football players, Age: 18.8 ± 0.4 years, n= 20, Portugal	2 weeks, daily	Sleep Diary		Effect of weekly variations in training intensity on youth soccer players	Correlations between pre-training sleep quality and session RPE and workload variables
Silva et al 2020 [413], observational	Professional football players, Age: 26.5 ± 5.2 years, n=20	10 days	Activity-monitor	Related metrics	Relationships between a 10 day sleep metrics on injury occurrence over the subsequent 6 months	negative correlation between sleep efficiency and injury characteristics

Springham et al. 2021 [382], longitudinal	Professional football players, age: 18 ± 3.8 years, n= 18, m, England, English Championship, pre- and in-season	One season, daily	self-reported measures (including sleep quality)	Perceived sleep quality (1 to 5 scale)	Longitudinal changes in sleep quality	Improvement in sleep as season persisted, compared to pre-season
Thomas et al. 2021 [376], longitudinal	Professional football players, age: 24.9 ± 2.8 years, n=18, f, England, English Women's Super League, in-season	4 weeks, daily	Activity monitor	Objective Bed time Wake time Time in bed Sleep duration SOL Number of awakenings WASO Efficiency	Mean sleep and sleep variation in both athletes compared to non-athletic controls	Significantly greater time in bed, sleep duration, SOL, and more variable bedtime than age-match controls.
Thorpe et al. 2015 [44], longitudinal	Professional football players, Age: 19.1 ± 0.6 years, n= 10, m, England, in-season	17 days, daily	In-house questionnaire (1 to 7) assessing sleep quality, muscle soreness and fatigue	Subjective Sleep quality	Partial correlations and general linear models between sleep quality and workload	Trivial and non-significant relationship between workload and Subjective sleep quality
Thorpe et al. 2016 [414], longitudinal	Professional football players, Age: 27 ± 5.1 years, n= 29, m England, in-season	Median 3 weeks per player, 6 days per week (not MD	In-house questionnaire (1 to 7) assessing sleep quality, muscle soreness and fatigue	Subjective Sleep quality	Differences between day activity (eg, MD+1, MD-1, TD)	Greatest Subjective sleep quality on MD-1, lowest on MD +1
Thorpe et al. 2017 [45], longitudinal	Professional football players, Age: 19.1 ± 0.6 years, n= 10, m England, in-season	17 days, daily	In-house questionnaire (1 to 7) assessing sleep quality, muscle soreness and fatigue	Subjective Sleep quality	Accumulated workload (total high speed running) and Subjective fatigue metrics (including sleep quality)	No correlation between 2, 3 and 4 day accumulated workload and sleep quality.
Whitworth-Turner et al. 2018 [381], longitudinal	Academy players, Age: 19 ± 1 years, n= 12, m United Kingdom, in-season	6 days, daily	Objective electroencephalogram	Objective Lights out time Wake time Time in bed Sleep duration SOL Number of awakenings WASO Efficiency	Mean sleep and sleep variation in both athletes compared to non-athletic controls	Greater but more varied sleep in football players compared to non-athletic controls. Greater latency in soccer players

Whitworth-Turner et al. 2019 [275], longitudinal	Professional football players, age: 18 ± 1 years, n= 10, m, England, in-season	2 weeks, nightly	Bedside device	Objective: Lights out time Wake time Time in bed Sleep duration SOL Number of awakenings WASO	Magnitude of effect of high-speed distance (>5.5 $m \cdot s^{-1}$) and training schedule (eg. MD, MD+1, MD-2, etc.) on sleep quality	Reduction in sleep duration on MD+1 compared to TD. High-speed distance was associated with increases in total sleep duration
Yadroudi et al [415], cross-sectional	Professional football players, Age: 21.82 ± 4.44 years, m n=91, f n= 61, Jordan	Single observation	Modified PSQI	Subjective: Related metrics	Off-season and in-season sleep patterns relationship with injury occurrence	Reduced sleep quantity associated with more injuries

BL (Baseline), SOL (sleep onset latency) WASO (wake after sleep onset), GPS (global positioning data), RPE (ratings of perceived exertion), PSQI (Pittsburgh Sleep Quality Index), ISI (insomnia severity index), ESS (Epworth sleepiness scale, CI (confidence interval), DOMS (delayed onset muscle soreness), RPE (rating of perceived exertion), MD (match days), TD (training days), HM (home match), REM (rapid eye movement), M (male), F (female). Age presented as mean ± SD (unless stated).

4.4.2. Study quality and risk of bias

All studies were, overall, judged to exhibit moderate to serious RoB. Serious RoB was notable in the confounding measurement domain with studies failing to measure and account for interindividual (e.g., chronotype, family responsibilities) and external (travel duration, country setting) confounders that may feasibly interact with sleep outcomes in an unknown manner and to an unknown extent. Although, the authors accept that such factors are synonymous with observational studies in applied settings and, in some cases, are not readily quantified. Full RoB assessment can be found in the Chapter 4 supplementary material (10.1.1) and is summarised in Figure 26.

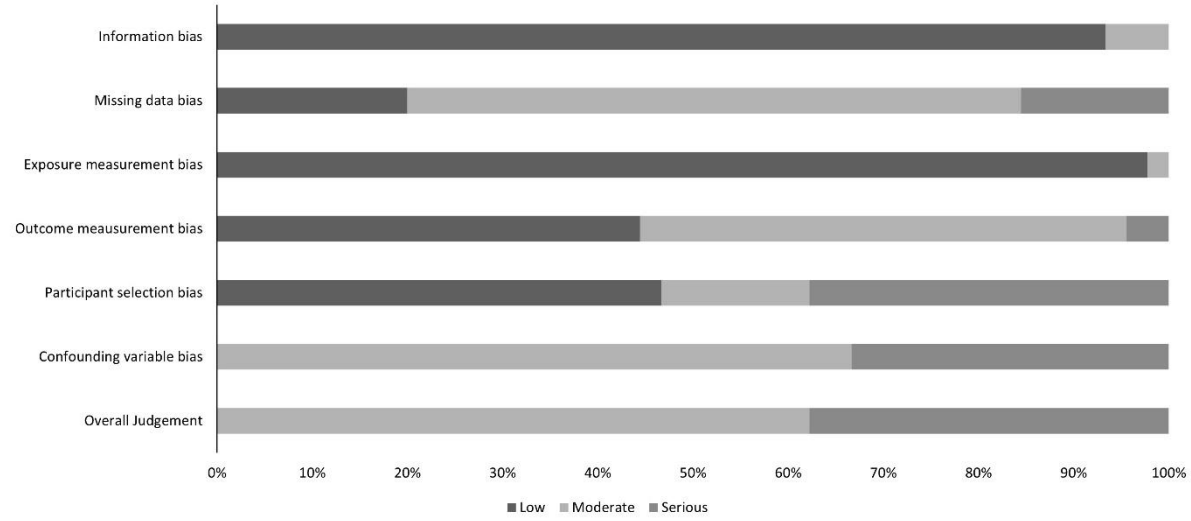

Figure 26: Results from risk of bias assessment

4.5. Discussion

The purpose of this study was to describe what is known about sleep quality and quantity, in relation to published norms [371], and identify the main literature themes concerning barriers to optimal sleep by systematically examining observational studies that have monitored sleep in fulltime, professional footballers. Subjective methods constituted the primary form of sleep assessment with 37 studies total (88%, 6 in tandem with objective methods) utilising sleep diaries or scales. Research has highlighted that subjective methods can be limited by mood, memory and other factors [249], potentially introducing biases to the data set. Eleven studies (28%) studies used activity-monitors or bedside devices to observe sleep. The devices and respective algorithms used varied across studies making direct comparisons challenging [199,416]. The studies were predominantly conducted in male professionals (83%), with relatively fewer studies focusing on females.

A conclusive appraisal of sleep quality in footballers is challenging based on current research. No sleep data were reported in footballers away from their normal playing and training schedule. Most studies

used training days (TD) as a baseline or control [206,272–277,381,417], justified as the most removed from competition and travel, and as the most numerous day type. This review took the same stance; however, we accept that TD does not constitute a robust baseline due to the continued psychological and physiological pressures associated with professional football. Furthermore, while factors associated with reduced sleep quality and quantity have been highlighted, it is acknowledged that results may be influenced by unknown and unaccounted confounders and should be interpreted accordingly. The primary findings are that professional football players sleep values were mostly within guidelines [371], however, players sleep remained variable and suboptimal in some regards. Furthermore, the respective influence of scheduling factors and workload on sleep was a primary investigative theme within the literature base with scheduling factors appearing to influence sleep in professional players.

4.5.1. Sleep characteristics

4.5.1.1. Sleep duration

The NSF recommends between 7 and 9 hours of sleep per night for both adults (26 to 64 years) and young adults (18 to 25 years) [371]. Sleep duration was reported in 11 studies [46,206,272–274,277,376,380,398,413,418] (Figure 27), with 9 studies reporting means that were within recommendations for sleep duration [371]. The extracted data were also not dissimilar to the mean sleep duration of a prospective study of British adults (7.04 ± 1.55hrs; n=2000) [419]. This trend has been observed in athletes previously [20]. In one comparison, athletes sleep duration was not significantly different compared to age-match controls, despite significantly reduced sleep quality [20]. Data suggests that footballers, in general, achieve adequate sleep, however, it is not clear what constitutes 'optimal' sleep for footballers, compared to the general population [281].

All five of the studies that monitored sleep subjectively (using sleep diaries or questionnaires) reported mean durations greater than 7 hours [206,272,274,277,398], with one reporting greater than 9 [272] (Figure 27). In general, studies utilising subjective methodologies reported greater sleep durations than those that used objective activity-monitors to assess sleep. This supports previous research that suggests that subjective assessments tend to overestimate sleep duration [247,248]. Further, subjective assessments can be limited by mood, memory and other biases [249]. Despite this, subjective and objective assessments do correlate (sleep duration, $r=0.62$, $p< 0.0001$) [247], indicating that sleep diaries are still suitable when investigating changes in sleep quantity between conditions.

The two studies that did not report adequate sleep used objective wrist-accelerometry in male senior players (mean age ≥26 years) [273,380]. However, it is not clear why the respective cohorts failed to meet sleep recommendations. Age may be a factor, with older players at an increased likelihood of habitual consumption of stimulants [131], family responsibilities, and earlier chronotype compared with adolescent players [165]. Although the majority of the objective studies that reported adequate sleep

used young (under 23 years) or Academy male teams (mean age ≤19 years) [46,275,418], one study in senior female players (mean age: 23.2 ± 4.5) did report sufficient sleep [376]. However, there is not enough data to speculate on the role age has on professional players sleep and sleep behaviour. Consequently, further research would allow practitioners to better understand the potential need for targeted sleep interventions.

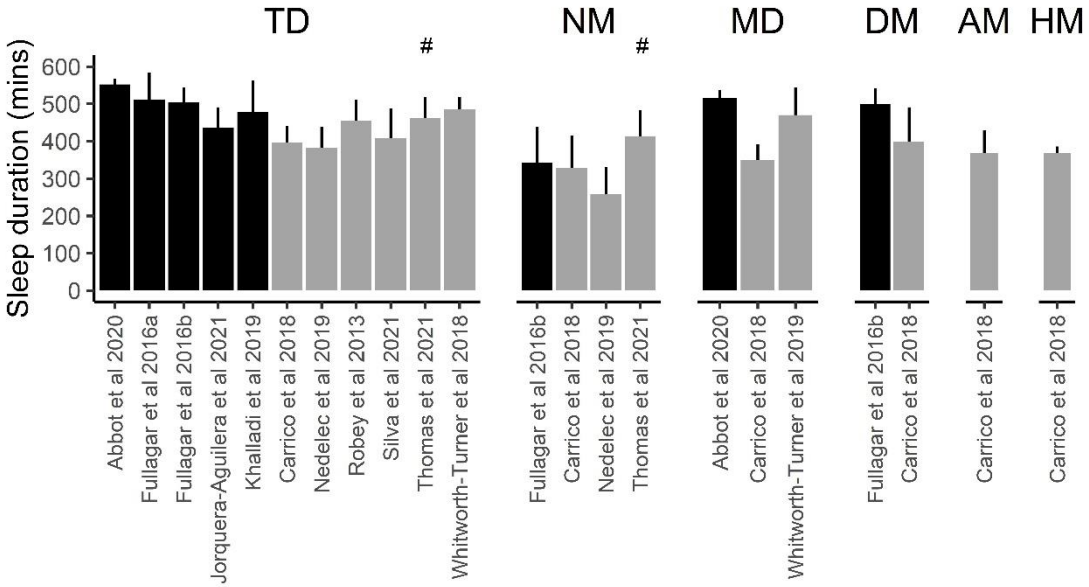

Figure 27: Mean sleep duration ± standard deviation for the included studies (where reported). TD (Training days), NM (Night match), MD (Matchdays), DM (Day match), AM (Away match), HM (Home match). Black bars indicate subjective data, grey bars indicate objective data. # Indicates a female cohort.

4.5.1.2. Sleep onset latency

The NSF suggests that a SOL score of <30 mins is appropriate for adults [371]. Two studies reported a mean SOL score over 30 mins [48,380] (Figure 28). However, several studies report standard deviations (SD) that breach the threshold [273,277,361,381].

Irrespective of the recommendations, SOL appears extended compared to non-athletic populations. Two similar studies observed significantly greater sleep latencies in academy players and female professionals, respectively, compared to age-match controls [376,381]. This observation unique to professional football. Leeder et al [20] compared activity monitor derived sleep metrics between athletes (but not footballers) and age-matched controls. While the athlete's SOL remained within guidelines, albeit variable (18.2 ± 16.5mins), it was still significantly extended compared to non-athletes (5.0 ± 2.5mins).

It is not clear why footballers may experience extended SOL. Sleep onset is a multifaceted, circadian and endocrine process primarily driven by a reduction of light/dark signals passing through the retinohypothalamic tract [420]. Electronic device use close to bedtime can inhibit SOL through increased light signals [168]. Although (to the author's knowledge) it is not known if device use is greater in footballers, sleep hygiene interventions that limit artificial light exposure have been successful in improving sleep quality in footballers [169], albeit only highly-trained amateur players. Increased pain during movement secondary to exercise-induced muscle damage (EIMD) [36], or disrupted post-exercise autonomic/ thermoregulatory circadian processors [166,167,282] might also be contributory to extended SOL.

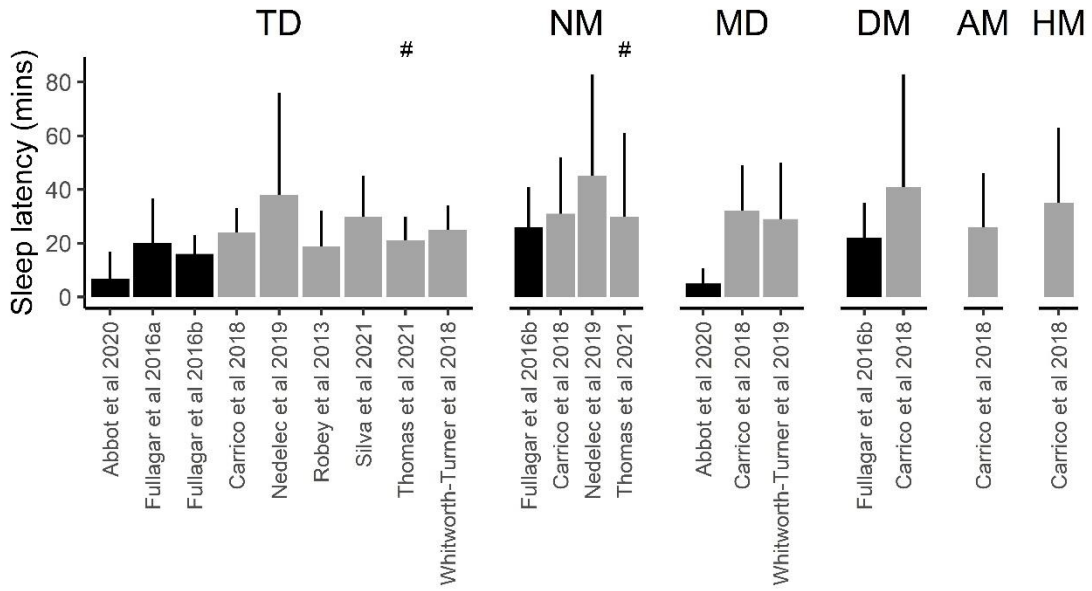

Figure 28: Mean sleep onset latency ± standard deviation for the included studies (where reported). TD (Training days), NM (Night match), MD (Matchdays), DM (Day match), AM (Away match), HM (Home match). Black bars indicate subjective data, grey bars indicate objective data. # Indicates a female cohort.

4.5.1.3. Wake After Sleep Onset (WASO)

WASO (total time awake between bedtime and time of final awakening) was assessed by 5 studies objectively [273,276,376,381,413], and 3 subjectively [272,274,277]. The results were variable. WASO from the subjective assessments were typically less than the objective, which fits with previously examined trends suggesting that self-reported WASO is underestimated compared to activity monitors [247]. That said, activity monitors rely on proprietary algorithms that interpret nocturnal movements to predict WASO. As with sleep duration and SOL, the quality of estimation is dependent on the algorithm and research suggests that activity monitors consistently underestimate WASO compared to PSG and agreement between devices can vary [199,416]. WASO data should therefore be interpreted with caution. Polysomnography is required to definitively confirm WASO in football players.

Thomas et al [376] recorded extended WASO scores in female footballers representing an English Women's Super League Club. A large cohort meta-analysis of non-athletes (n=68,604) [421] suggested females experience greater WASO, however, significant differences were not observed until >50 years. Unfortunately, additional studies reporting WASO in female footballers were not identified, therefore, it is not known if the reported data are truly representative of this population. In male players, Carriço et al. [273] observed a WASO of 30 ± 16 mins whereas Whitworth-Turner et al [46] only observed 12 min, with both studies utilising objective methods. The variation might be attributed to the eight-year difference between the mean ages of the respective studies. WASO can increase with age, however, meaningful changes do not present until greater than approximately 30 years and the magnitude of difference between the studies would suggest other covariates are apparent [422]. This might include the sensitivity of the respective devices, or the algorithm used to interpret periods of wakefulness [199,416].

Recommendations suggest that WASO duration of less than 20mins is appropriate for ages 14 to 64 years. Five of the eight studies report scores of <20mins [46,273,275,276,376], however, the reported variance suggests that WASO above 20mins is common. One study highlighted greater WASO in footballers compared to non-athletic controls, supporting the observation that footballers experience reduced sleep quality compared to non-athletic populations [381].

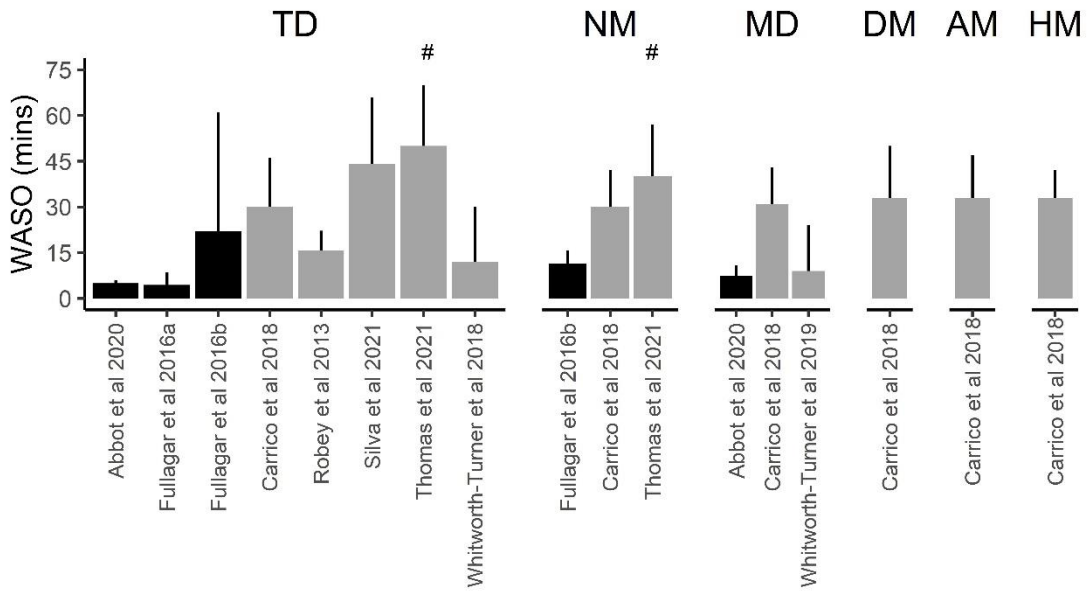

Figure 29: Mean wake after sleep onset ± standard deviation for the included studies (where reported). TD (Training days), NM (Night match), MD (Matchdays), DM (Day match), AM (Away match), HM (Home match). Black bars indicate subjective data, grey bars indicate objective data. # Indicates a female cohort.

4.5.1.4. Sleep assessment questionnaires in cross-sectional studies

Five studies circulated questionnaires to professional cohorts that assess clinically relevant sleep disorders or quality [206,388,398,399,423]. Kilic et al [399] used an athlete psychological strain questionnaire in a large cohort (n=281) and highlighted a 12% and 33% prevalence of sleep disturbance for males and females, respectively. Khalladi et al. [206] circulated PSQI questionnaires in players competing in the Stars League (Qatar) and reported a 68.5% (n=111) incidence rate for poor sleep quality (PSQI score ≥5). Results are corroborated by data from Chilean professionals who reported mean PSQI scores of 4.75 ± 2.29 [398]. Khalladi et al. [206] notes that the extreme heat, socialising norms and Islamic practices (first prayer with sunrise) in the Middle East may exacerbate sleep issues compared to western teams, however, a Dutch study reported a PSQI of 3.6 ± 2.4 [423], suggesting suboptimal sleep quality may also exist among European players.

Data also suggests moderate levels of subclinical insomnia in Qatar's Star League and in Italian players with a reported prevalence of 27% and 32%, according to the Insomnia Severity Index (ISI) criterion [206,388]. Furthermore, 22.5% reported excessive daytime sleepiness according to the Epworth Sleepiness Scale (ESS; ≥8) [206]. Another study in a similar cohort reported a mean score of 5.2 ± 5 and 6.1 ± 5 for the ISI and ESS, respectively; potentially indicating a more serious issue [423].

4.5.2. Scheduling factors and sleep

Scheduling factors relate to the time and location that training, fixtures, and other commitments professional footballers may encounter, are positioned within their normal routine. Some factors, including match location and kick-off time, been highlighted as a major investigative theme within the literature.

4.5.2.1. Matchdays

In total, seven studies analysed the effect of matchdays (MD) on sleep [273–275,361,362,376]. Four studies assessed sleep objectively and three subjectively. In all cases, TDs were used for comparative baselines.

4.5.2.2. Night matches

The influence of night matches (NM; kick-off times after 1800 hours) was investigated in four studies, and sleep disruption is evident across several studies (Figure 27) [273,274,361,376]. Using self-reported sleep diaries, male footballers representing top-flight clubs in Germany and the Netherlands [274] reported mean sleep duration reductions >3hrs after NM. Results are corroborated by wrist activity-monitor studies in other top-flight European leagues [273,361,376], albeit with mean sleep loss limited

to approximately one hour. Differences in sleep assessment method may explain the differences in sleep loss data, however, the post-game travel time relative to each country may also be a factor.

In all cases, the reduction in sleep quantity occurred in tandem with later sleep-onset times [273,274,361,376]. This is likely to be secondary to a plethora of factors, including later kick-offs compared to TD commitments, hyperarousal, consumption of so-called pre-match performance stimulants [131,251,424], in addition to post-game media, team, and recovery commitments that may push sleep onset time back [131,424].

Regardless, data suggests that night matches directly or indirectly reduce sleep quality in a period where recovery is paramount. The effect this disruption has on performance is unknown, however, Fullagar et al. [274] reported reduced perceptions of wellbeing and stress/recovery balance following NMs compared to day matches (DMs) and TDs.

4.5.2.3. Day matches

DM (KO before 1800 hours) appear to have limited influence on sleep metrics. Carriço et al. [273] did note significantly later bedtimes and wake times after DMs, compared to TDs, in 25 professional players competing in Portugal. However, objectively derived sleep duration, was unchanged. These results were similarly observed subjectively in professional players elsewhere in European top-flight leagues [274], and in professional youth players [381]. Although, the latter [381] also reported no significant disruption to normal bedtimes or waketimes, in contrast to the studies in senior players [273,274]. The youth players were staying in halls of residence and, therefore, may have kept to a stricter regime. Overall, the evidence suggests that day matches are not associated with changes in sleep.

4.5.2.4. Long-haul travel

Players engage in international and domestic travel to attend scheduled training camps and/or competitive fixtures. Four studies were identified that measured sleep patterns during long-haul travel (defined here as air travel >7hrs) [226,227,277,397]. In studies that have monitored players during westbound (4 time zones) [277] and eastbound travel (11 time zones) [397], sleep duration reductions were limited to the day of travel only. This suggests that the travel itself is a primary cause of disruption, and not the circadian disturbance of traversing time zones [425–427]. That said, the studies cannot provide evidence to conclusively demonstrate if the if post-travel sleep was restorative. After eastbound travel, players self-reported jetlag symptoms that persisted for at least 5 days [397], a trend observed in other athletes [425–427].Studies have shown more notable jetlag symptoms after eastward, compared to westward, travel owing to the more rapid circadian realignment after a phase delay [425–427].

Northbound travel does not necessarily require time zone changes, therefore, any circadian disruption to sleep may be less of a factor. Lastella et al monitored sleep in layers travelling northward for the Asian Champions League and noted that sleep duration was approximately 3.6 hours less on travel days compared to non-travel days [226]. However, excluding MD, sleep duration was similar to what was experienced in the athletes' own home (7.0 ± 1.6 hours) and at the travel destination (7.0 ± 2.1 hours). In a similar study [227], a reduction in sleep duration was observed on the day before travel, rather than the travel day, although, this is possibly due to the differences in departure times between studies. Nevertheless, sleep duration on non-travel and non-game days remained similar to sleep recordings taken in the footballer's home. In the data available to date, it appears that sleep disruption is limited to the day of travel, rather than the relocation. Future studies should place emphasis on the overall quality of sleep after long-haul travel and asses if subsequent sleep is restorative.

4.5.3. Sleep variation

4.5.3.1. Intra and inter-microcycle variation in sleep metrics

Three studies assessed the variation in sleep across a microcycle [275,383,414]. Male academy footballers presented with greater objective sleep duration on MD-2, MD-1, and MD compared to MD+1 [275], highlighting heterogeneous sleep across different day types. Likewise, using subjective monitoring (7 point scale), Thorpe et al. [414] observed a similar pattern in players competing in the EPL. Conversely, in Female professionals observed over 7 months, no significant differences in perceived sleep quality were found between MD-5, -4, -2, and MD [383]. The reasoning for the disparity is unclear. There are several confounders that could feasibly introduce variability throughout the microcyle, including travel [226,227,277,397], social jet lag [295], potentially workload [401,409,411], or other scheduling variables [274]. Speculatively, players also might report better sleep during the night before the match in an effort to increase the likelihood of being involved on MD.[226]

Sleep may also vary between microcycles. In U18 footballers playing for an EPL Academy (n=12), a moderate decrease in subjective sleep quality 24 hours post-MD was reported [395]. However, the same decrease was not reproduced in the following week, indicating inter-microcycle variation. As discussed, several factors can affect perceptions of sleep, nevertheless, it is possible that the subjective sleep reporting was not sensitive enough to detect changes [247–249,395]. That said, sleep is subject to normal day-to-day variation. This has been observed in non-athletic populations [381,428,429], however, there is evidence that this is exacerbated in footballers. When the objectively derived standard deviations of sleep metrics from professional players were compared to age-matched controls, tests revealed significantly greater levels of variation in SOL, efficiency, and bedtime [46,376]. Consequently, it is plausible that any inter or intra-microcycle heterogeneity is a result of the intra-individual variation present in footballers.

4.5.3.2. Inter-season variation in sleep

No studies assessed inter-season sleep variation objectively, however, three studies did use Likert-type scales [382,403,404]. The results are variable and conflicting. In players representing an EPL Academy [404], perceived sleep quality reduced as the season persisted, with the latter two blocks significantly reduced compared to the first in-season block, and all in-season blocks significantly lower than pre-season. This occurred in tandem with increases in stress levels and muscle soreness which could have been contributory to a decrease in perceived sleep quality. Contrastingly, two other studies recorded increases in sleep quality towards the later mesocycles, compared to pre-season [382,403]. The reasons for the discrepancies are not clear. Each study uses different scales in which to judge perceived sleep, rendering direct comparisons mute and perceptions may be influenced by the success of the team as a whole [249,362,406]. Season-long objective studies are required to fully characterise the variability of in-season sleep.

4.5.4. Influence of workload on sleep

Several studies have investigated the influence of player workload on sleep [44,45,275,391,392,401,408,409,412]. The Hooper index [391,409,410] and other Likert scales [44,45,392,401] constitute the primary method to assess sleep in relation to external load. Although, there is little to substantiate a clear relationship. Douchet et al. [391] observed that perceived sleep quality was reduced after a heavy intensity microcycle, compared to a lighter intensity microcycle, in female professional players competing in France. However, similar studies across both youth [44,45] and senior [401,409,411] professional demographics have reported no significant relationships. Further, no studies have associated cumulative workload with perceived sleep quality. In 10 EPL players, monitored over 17 days, significant relationships were found between fatigue and total high-speed running (>4m·s^{-1}) [44], suggesting players reacted to changes in workload, however, perceived sleep was not affected [44]. In the same cohort, a retrospective analysis assessed the influence of daily accumulated loads on subjective sleep quality [45]. Yet, the relationship between perceived sleep quality and 2-, 3-, and 4-day accumulated total high-speed distance remained trivial and non-significant.

The Hooper and similar scales may lack the requisite sensitivity to adequately assess any effect of workload on sleep, and more sophisticated sleep diary or objective methodologies may be required. Sleep diary analysis revealed significant correlations (r=0.205) between sleep duration and total distance in 20 youth professional players who were monitored over 2 weeks [412], potentially suggesting a relationship. Likewise, another study observed a significant relationship between total high-speed distance (>5.5 m·s^{-1}) and objectively derived sleep metrics in 10 English academy players [275]. Nevertheless, the effect sizes were small to trivial, with every 100m increase in high-speed distance equating to an additional 1-min, 10-mins, and 10-mins for WASO, time in bed, and sleep

duration, respectively. The study also reported that sleep was sensitive to day type (e.g., MD, MD+1 etc), therefore, the changes could be a consequence of tapering workload and adjusted sleep behaviour.

Although there is little evidence to support a substantial relationship between external workload and sleep, further research is needed. Specifically, investigations that assess the impact of workload on subsequent sleep architecture in footballers would enable far greater understanding.

4.5.5. Other related factors

The influence of other related on sleep metrics have also been investigated, including match result (win/lose), match location (home/away), fixture congestion, quality of opposition, and single compared with dual occupancy rooms. However, research within these areas is scarce, therefore, any insights are limited to speculation. Nevertheless, emerging investigative trends that may meaningfully impact applied practice are highlighted here.

Match location (home/away) can feasibly impact sleep due to the presence of post-game travel commitments. In one season-long study, objectively derived bedtimes and wake times were later after away matches compared to home and TDs, however, objective sleep duration was unaffected [273]. Another study suggested that subjective sleep quality was reduced after an away match and also suggested that subjective sleep was also negatively associated with a loss, or after playing a team positioned higher in the league [362]. In this study, more games were lost compared to winning against higher-quality teams which may be a confounding factor. A further study [406] noted a better perception of sleep after a positive result against teams rated more highly, supporting the notion that mood state may be contributory, however, this may also be related to workload.

Noor et al [405] observed the effects of fixture congestion on self-reported markers of fatigue during international fixtures and reported reduced perception of sleep during acute congestion (2 matches in <4 days) compared to no match days. As before, this may be related to the effect of workload, but may also be influenced by the psychological demands of international competitions. Also, one study reported that objective sleep duration and subjective sleep quality were lower in professional youth players that shared a room during a training camp, compared to when they slept in individual rooms [389].

Only one study investigated the influence of Ramadan on sleep in practising Muslim professional players [430]. Results suggested a reduction in sleep duration, however, no studies have been completed outside of the Middle East, where cultural differences and the extreme heat may impact sleep behaviour compared to other leagues [206,430]. Finally, player's sleep may also be affected by altitude, and, although reductions in the quantity of slow-wave sleep and sleep duration have been observed in young

players engaged in a 19-day training camp at altitude (3600m) (mean age: 15.6 ± 0.5 years) [196], this research has not been repeated in professional players.

4.5.6. Limitations

Firstly, this scoping review used the themes highlighted in the literature to structure the subsequent discussion. Therefore, it cannot comment on other confounders that are yet to receive investigative interest nor can it be known how comprehensive this review is. Furthermore, many studies reviewed in this report used Likert scales to assess sleep which may not have been sensitive enough to detect any meaningful change. However, considering that this was a scoping review, it is important to include these studies. Finally, this scoping review was not registered before its commencement.

4.5.7. Conclusions and recommendations

Results suggest that professional football players sleep duration is within national recommendations and published norms. However, practitioners should be aware of variable WASO and SOL scores among players, and interventions targeting these may be valuable. This scoping review suggests that scheduling and workload variables are primary research themes within the literature, with scheduling highlighted as a factor that affects sleep in professional players. This is potentially more notable after NM, but not DM, possibly secondary to media and travel commitments. Match scheduling is typically out of the control of coaches, therefore, proactively adjusting the start time on MD+1 might provide an opportunity to increase sleep duration. Travel in general, whether a result of NM, away matches, or long-haul travel, was highlighted as a potential barrier to sleep quantity. Consequently, team commitments should be scheduled in a way to protect the physiological and cognitive performance of the players, and potentially their longer-term health.

Chapter 5

5. Day type and start time may influence sleep in adolescent professional football players

Publications associated with this chapter:

6. Luke Edinborough, Stewart Bruce-low, Jessica Hill, Jonathon Woodhouse, Mark Jarvis, Charles Pedlar (2021). *Influence of scheduling on objective sleep metrics in professional U18*

footballers: a longitudinal observational study. The British Association of Sport and Exercise Sciences 2021 annual conference.

7. Luke Edinborough, Stewart Bruce-low, Jessica Hill, Jonathon Woodhouse, Mark Jarvis, Charles Pedlar (2023). *Day Type and Start Time May Influence Sleep in Adolescent Professional Football Players*. Int J Sports Med. DOI: 10.1055/a-1974-5441

This project was also fed back to the players involved in the study my way of a video designed to be viewed on a mobile screen (please follow the QR code).

Password: LE_4_PhD123

5.1. Abstract

This study assessed if scheduling (start time and day type) and workload variables influenced sleep markers (activity monitor) in professional academy footballers (n=11; 17.3 ± 0.7yrs) over a 10-week in-season period. Separate linear mixed regressions were used to describe the effect of start time on the previous nights sleep, and the effect of day type (matchday, matchday+1) and workload on subsequent sleep. Workload variables were modelled by day (day), 7-day (acute), and 28-day (chronic) periods. Sleep duration following matchday+1 (400mins; 95%CI:368—432) was significantly reduced compared to all other day types($p<0.001$). Sleep onset time following matchday (00:35; CI:00:04—01:12) and wake time on matchday+1 (09:00; CI:08:37—09:23) were also significantly later compared to all other day types ($p<0.001$). Sleep duration (19.1mins; CI:9.4–28.79), wake time (18mins; CI:9.3–26.6), and time in bed (16.8mins; CI:2.0–31.5) were significantly increased per hour delay in start time. When no activity was scheduled sleep duration (37mins; CI:18.1—55.9), sleep onset (42.1mins; CI:28.8–56.2), and wake times (86mins; CI:72–100) were significantly extended, relative to a 09:00 start time. Day, acute, and chronic workloads were associated with sleep onset and wake times only. Scheduled start times were associated with changes in sleep duration, therefore, delaying start times may increase sleep in this population.

5.2. Introduction

Sleep monitoring methodologies in observational studies have highlighted several factors that may influence sleep in professional football players. Notwithstanding the significant inter/intra-individual variation [275], studies have also reported differences according to day type (e.g., matchday (MD), MD+1) [275], and reduced sleep quality or quantity after night matches [273,361], and travel [226,277]. Consequently, there is growing evidence to suggest that competitive scheduling contributes to sleep disruption in footballers. As biological chronotype (the intrinsic entrainment of an individual's circadian system to a 24-hour cycle) approaches peak lateness during late adolescence, approximately 104 mins later than the lifetime average [431], it follows that scheduling considerations for adolescents and senior players should differ.

Start time (the time players are scheduled to arrive for training or competition) is a consideration that coaches arguably have more control over than other scheduling elements. This could be particularly pertinent for professional academy (full-time, contracted) players whose chronotype may support a delayed start time [175,431]. In adolescent students in the USA (13 to 18yrs), later school start times have been associated with longer sleep durations, reduced daytime sleepiness, and improved academic performance [175,431]. Professional academy players commitments vary compared to the general population, consequently, the influence start time has on professional academy footballers sleep is not known.

Workload may also influence sleep [275], with both workload [33] and suboptimal sleep [48] linked to increased injury risk. Yet, reports investigating the impact of workload on subsequent sleep are equivocal. In professional rugby league players, higher acceleration/deceleration counts resulted in greater sleep efficiency [47], whereas intensified training in endurance athletes resulted in reduced sleep duration and efficiency [298]. However, in football a substantial relationship is yet to be presented. In English Premier League (EPL) players, no significant link was revealed between total distance covered above >4m·s^{-1} and subsequent perceived sleep quality [44,45], and, while another study [275] did observe a significant relationship between distance high speed running (>5.5 m·s^{-1}; HSR) and sleep duration, effect sizes were trivial.

Therefore, the aims of this study were to 1) assess how start time may influence sleep the night before, and how day type may influence subsequent sleep; and 2) assess how workload may influence subsequent sleep in 18year old (U18) professional footballers.

5.3. Materials and methods.

5.3.1. Participants

Eleven male U18 outfield professional (full-time, contracted) footballers playing for a category 1 EPL academy participated in this study (17.3 ± 0.7yrs; 178.6 ± 7.4cm, 74.8 ± 8.4kg). Players were excluded if they had previously self-reported any clinical sleep issues to the club's medical team. Fourteen players were initially recruited but 3 were excluded from the analysis due to lack of adherence (n=2), and technology failure (n=1). All players were living at home or with host families throughout the duration of the study and travelled to training via their own means or a minibus service provided by the club. Informed participant and parental consent were obtained before data collection and this study was approved by the ethics committee at St Mary's University, Twickenham.

5.3.2. Experimental design

This was a longitudinal, observational study which spanned a 10-week in-season period during the 20/21 season and, therefore, subject to National and Football Association COVID-19 regulations. However, players continued their normal uninterrupted competitive schedule throughout the study. The study included 9 matches (66.7% home) and all kick-offs were before 1300. A typical training week is described in Table 8. Player sleep was monitored objectively using activity monitors (ReadiBand, Fatigue Science, Vancouver BC, Canada). Data were then categorised by day type (activity of the day, relative to match day, eg MD, MD+1) and start time (the time players were scheduled to arrive at the training ground). Throughout training and matches, players workload was quantified using the Global positioning system (GPS) and accelerometry (Viper V.2, StatSports, Ireland) data routinely collected by the club. This has been validated against radar gun over predefined distances [42] . Periods of injury/illness were excluded.

Table 8: Typical in-season week for the U18 footballers involved in this study

Day	AM	PM
Monday (TD)	Education	Training
Tuesday (TD)	Training	Gym training/ Injury prevention/ technical skills training/ analysis
Wednesday (TD)	Gym training/ Injury prevention, technical skills training/ analysis	Education
Thursday (TD)	Education	Training
Friday (MD-1)	Training	Team meeting
Saturday (MD)		Matchday
Sunday (MD+1)	Off/ rest day	

Training day (TD)
Matchday minus one (MD-1)
Matchday plus one (MD+1)
Matchday (MD)

5.3.3. Sleep monitoring

Players wore activity monitors on their non-dominant wrists. Nocturnal movements were then used to estimate time-in-bed, sleep duration, sleep quality, wake after sleep onset (WASO), sleep latency and sleep onset time. ReadiBands have demonstrated good inter-device reliability and accuracy compared to polysomnography [198,199]. The devices were synced to cloud-based software by training staff who also requested and logged information on naps. Activity monitors can interpret sedentary periods (e.g., travel) as sleep, therefore, any periods where the device registered sleep before 21:30 were removed after self-reported naps were accounted for. Activity monitors were worn for an average of 52% of nights that they were requested to be worn (Table 9). Forgetfulness was most often cited for non-adherence. Players who wore the devices for less than 14 days were excluded (n= 2).

5.3.4. Start time and day type

Separate statistical models were generated for start time and day type. The day types were training day (TD, a normal training day), match day (MD, a day in which a competitive fixture is played), pre-match training day (MD-1, a normal training day the day before a MD) and post-match day (MD+1, the day after MD). As the players scheduled day off, no start time was available for MD+1. Therefore, to elucidate the complete influence of start time on sleep metrics, two separate start time models were generated. First, start time was coded as a categorical variable with no scheduled activity (NSA) imputed as the start time for MD+1. Start time was then analysed under the following categories: 08:00, 08:15, 09:00, 09:30, 10:00, 11:15, NSA. Data were compared against a 09:00 start time as the most frequent start time. Second, NSA was excluded from the dataset and start time was modelled continuously.

An individual's chronotype can be quantified through their mid-sleep point on work-free days [431]. As MD+1 had no scheduled activity, it was assumed that players were more likely to initiate sleep on MD and wake on MD+1 without any influence from scheduling demands [431]. The authors accept that an accurate chronotype may not be calculated due to the effects of MD exertion on sleep drivers, nevertheless, the lack of scheduling on MD+1 provides a proxy for when sleep is supposed to occur naturally to estimate chronotype. Consequently, for reference purposes only, chronotype was calculated as the midpoint between sleep onset on MD and the wake time on MD+1 [431].

5.3.5. External load

GPS data were used to quantify workload during training and matches. The players donned a vest that placed a GPS and accelerometry unit between the scapulae. The unit sampled GPS and accelerometry data at 10 Hz and 100 Hz, respectively, and was downloaded using specialist software (Statsports APEX). To assess the influence of workload on sleep metrics, HSR distance (total distance (m) covered at running speeds >5.5m·s^{-1}; HSR) was used as a global measure of external load, as per previous research [44,45,275] and due to its association with injury occurrence in U18 footballers [33]. Additionally, high-speed decelerations (a decrease in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}, DEC), and high-speed accelerations (an increase in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}; ACC) were included due to their links with muscle damage and possible pain that may disrupt sleep during nocturnal movements [36]. Each variable was sampled by day (day), accumulated 7day (acute), and accumulated 28day (chronic). High chronic (relative risk (RR): 2.14; p=0.003) and acute (RR:1.73; p=0.029) HSR has been associated with increased overall injury risk in a similar cohort (U18 footballers, 17.3±0.9yrs) [33]. HSR is reported per 100m. DEC and ACC are reported per 10 actions.

5.3.6. Statistical analysis

Linear mixed modelling (LMM) were performed for all analysis with activity monitor-derived sleep metrics imputed as the dependant variable and random slopes and intercepts generated for each individual [432]. To assess differences in sleep according to day type, a regression was performed with Bonferroni *post hoc*. The mid-point of sleep between MD sleep onset and MD+1 wake time was derived from this model. Separate regressions were performed for start time viewed continuously (excluding NSA), and categorically. Finally, the influence of DEC, ACC, and HSR was assessed through separate multiple regressions with day, acute, and chronic workloads as the predictor variables. All data were analysed using the R statistical environment (The R Foundation for Statistical Computing) in Rstudio (Boston, USA). Blank code can be found in the Appendix (Appendix 2: Chapter 5 supplementary materials). All data are presented with estimates and 95% confidence intervals (CI), and $P<0.05$ was considered statistically significant.

5.4. Results

Data from 402 nights were collected. Multiple regressions require data from all predictor variables to be available. This reduced the data available for the workload models (Table 9).

Table 9: Total number of observations per linear mixed model

Variable	Number of observations	Observations per participant (mean ± SD, min, max)
Day type	402	36.5 ± 11.7, 18, 56
TD	265	
MD-1	52	
MD	33	
MD+1	52	
Start time (categorical)	402	36.5 ± 11.7, 18, 56
08:00	10	
08:15	7	
09:00*	244	
09:30	28	
10:00	67	
11:15	8	
NSA	38	
Start time (continuous)	364	33.1 ± 10.1, 16, 49
08:00	10	
08:15	7	
09:00	244	
09:30	28	
10:00	67	
11:15	8	
Workload	250	22.7 ± 7.8. 14, 38

TD (training day)
MD (match day)
NSA (no scheduled activity)
* Used as reference start time

5.4.1. Day type and start time

Sleep duration ($p<0.001$) was significantly reduced following MD+1 (400mins, CI:368—432) compared to all other day types (TD: 430mins, CI:400—459, $p=0.007$; MD: 456mins, CI:422—490, $p<0.001$; MD-1:433mins, CI:401—465, $p=0.03$). Time-in-bed was significantly longer ($p=0.009$) following MD (570mins, CI:535—605mins) compared to MD+1 (506, CI:476—537mins; $p=0.005$) and TD (529, CI:505—552; $p=0.047$). Sleep onset time was significantly later ($p<0.001$) following MD (00:35, CI:00:04—01:12) compared with all other day types (MD-1: 23:47, CI:23:17—00:14, $p<0.001$; MD+1:00:03, CI:23:33—00:29, $p=0.009$; TD: 23:56, CI:23:27—00:29, $p<0.001$). Wake time was significantly later on MD+1 (09:00, CI:08:37—09:23mins) compared with all other day types (TD: 07:44, CI:07:26—08:01, $p<0.001$; MD-1: 07:38, CI:07:16—07:58, $p<0.001$; MD: 07:42, CI:07:20:38—08:04, $p<0.001$) (Figure 30). Based on the available data from MD (n=33), mid-sleep point (chronotype) is estimated at 04:46 ± 00:44, (CI: 04:19–05:13).

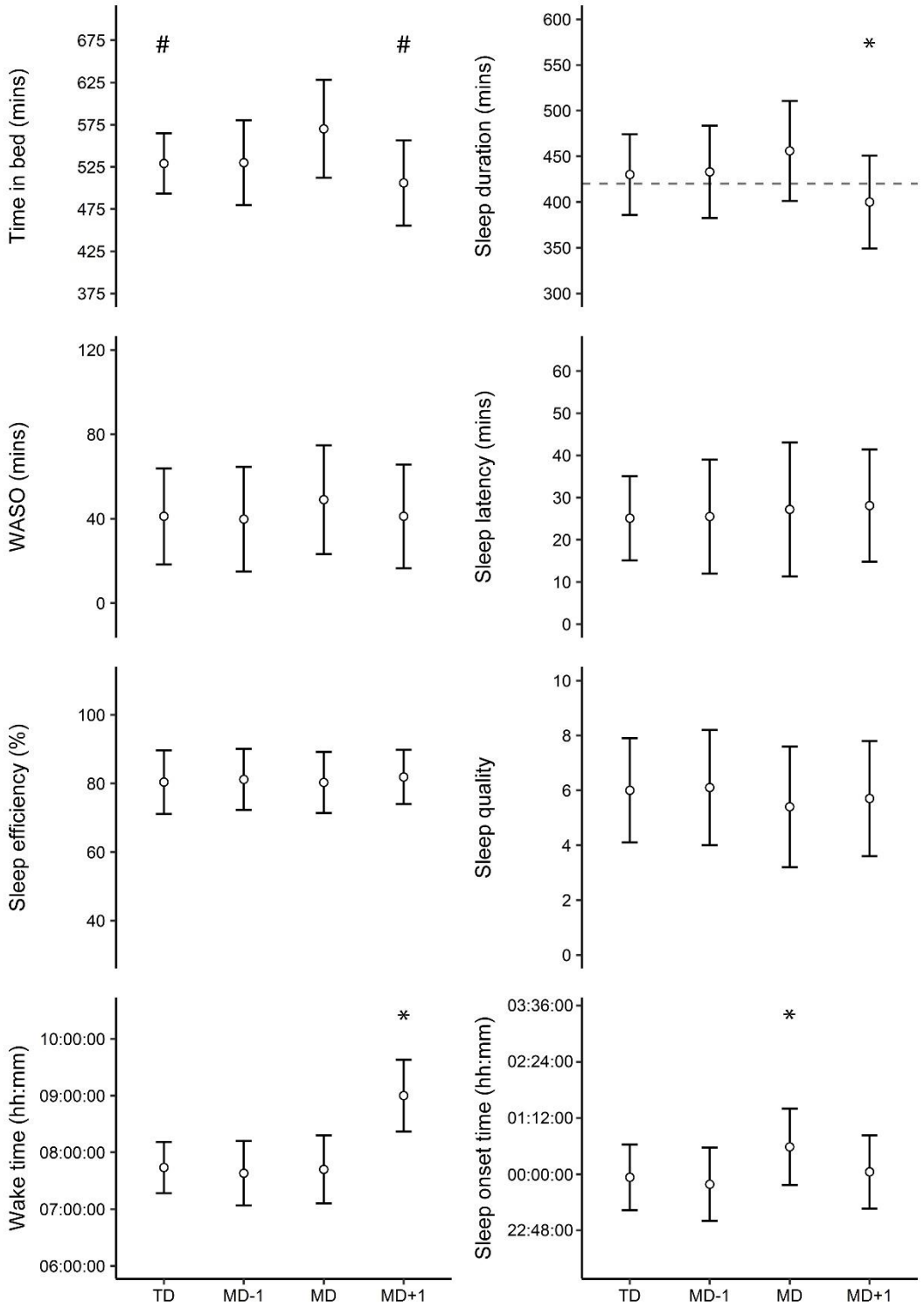

Figure 30: Estimated marginal means ± 95% confidence intervals for activity monitor derived sleep metrics across the 4-day types. For reference, the dashed line on sleep duration represents 420 mins. Training day (TD), Matchday (MD), the day before MD (MD-1), day after MD (MD+1), time awake after sleep onset (WASO). Number of observations: TD (265), MD-1 (52), MD (33), MD+1 (52). *Significantly different from all other day types ($p<0.05$). #significantly different from MD ($p<0.05$)

3237

3238

When start time was analysed continuously, time in bed (16.8mins, CI:2–31.5; p=0.026), sleep duration (19.1mins, CI:9.4–28.79; p<0.001), and wake time (18mins, CI:9.3–26.6; p<0.001) significantly increased per hour delay in start time. Relative to a 09:00 start time, sleep duration was extended during the night preceding all other start times, with the exception of a 11:15 start time (09:30: 31.7mins, CI: 9.51–53.96, p= 0.0052; 10:00: 17.7mins, CI: 2.72 – 32.67, p=0.0198; and NSA: 37mins, CI: 18.1–55.9, p<0.001). Compared to the reference 09:00 start time, wake time was later than on all other start times, with the exception of 11:15 (09:30: 38mins, CI: 14–62, p<0.001; 10:00: 22min, CI: 14–0.30, p=0.001; and NSA 86mins, CI:72–100, p<0.001). Sleep onset time was also significantly later the night before NSA (42mins, CI:29–55; p<0.001) compared to all other start times. Time-in-bed (45mins, CI:17–73; p=0.002) and WASO (7.4mins, CI:0.2–14.6; p=0.044) the night before NSA were significantly greater than on 09:00 start time days. Sleep latency on 10:00 start time days (-8.5mins, CI: -14.5– -2.6; p=0.006) was significantly reduced compared to 09:00 start time days (Figure 31 and Figure 32).

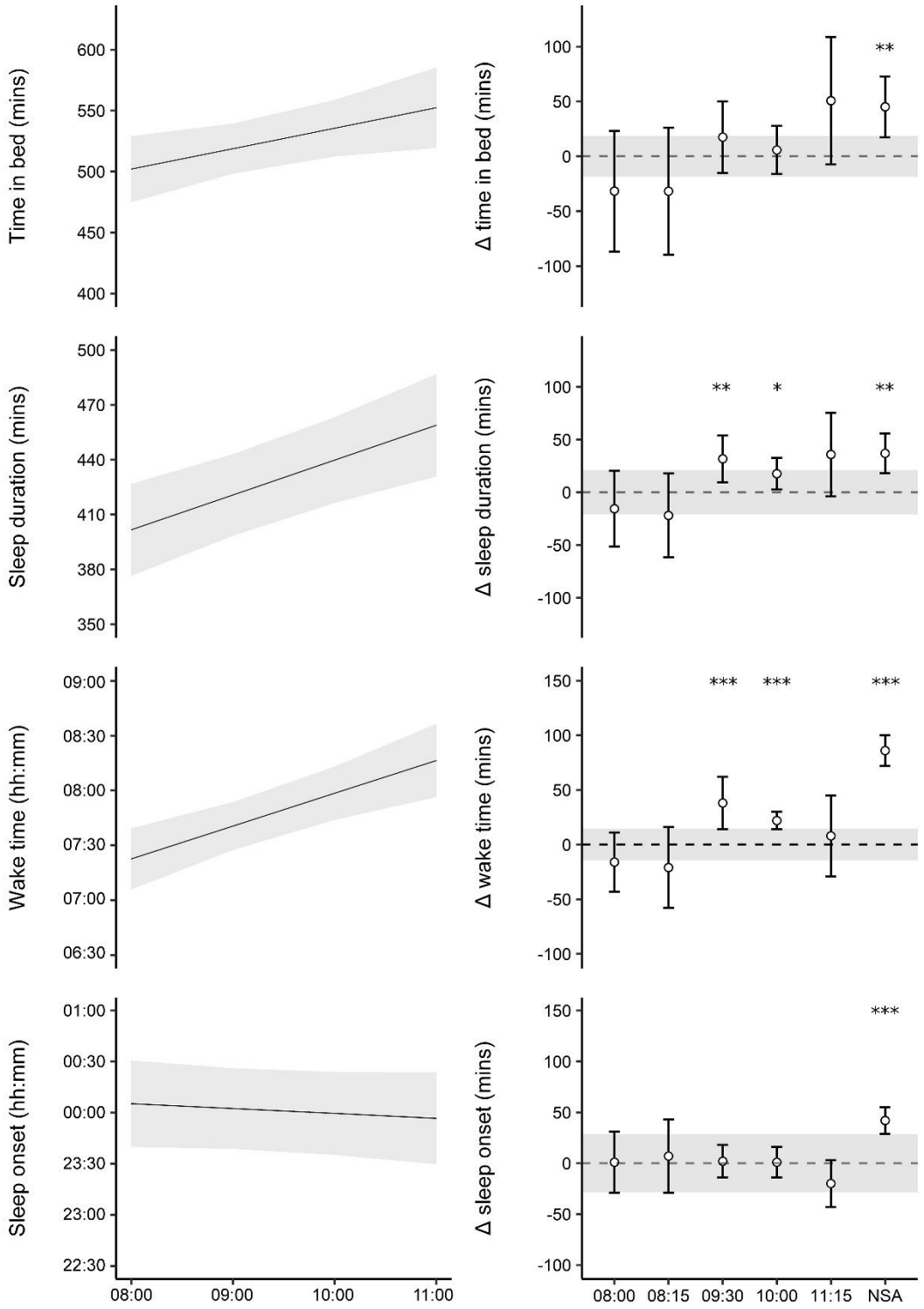

Figure 31: Data visualisation for the continuous start time model (left) and categorical start time model (right) for time in bed, sleep duration, wake time, and sleep onset. Data are presented as beta estimates ± 95% confidence intervals (grey area). No scheduled activity (NSA). 08:00 (10), 08:15 (7), 09:00 (244), 09:30 (28), 10:00 (67), 11:15 (8), NSA (38). * *p<0.05* ** *p<0.01* *** *p<0.001*

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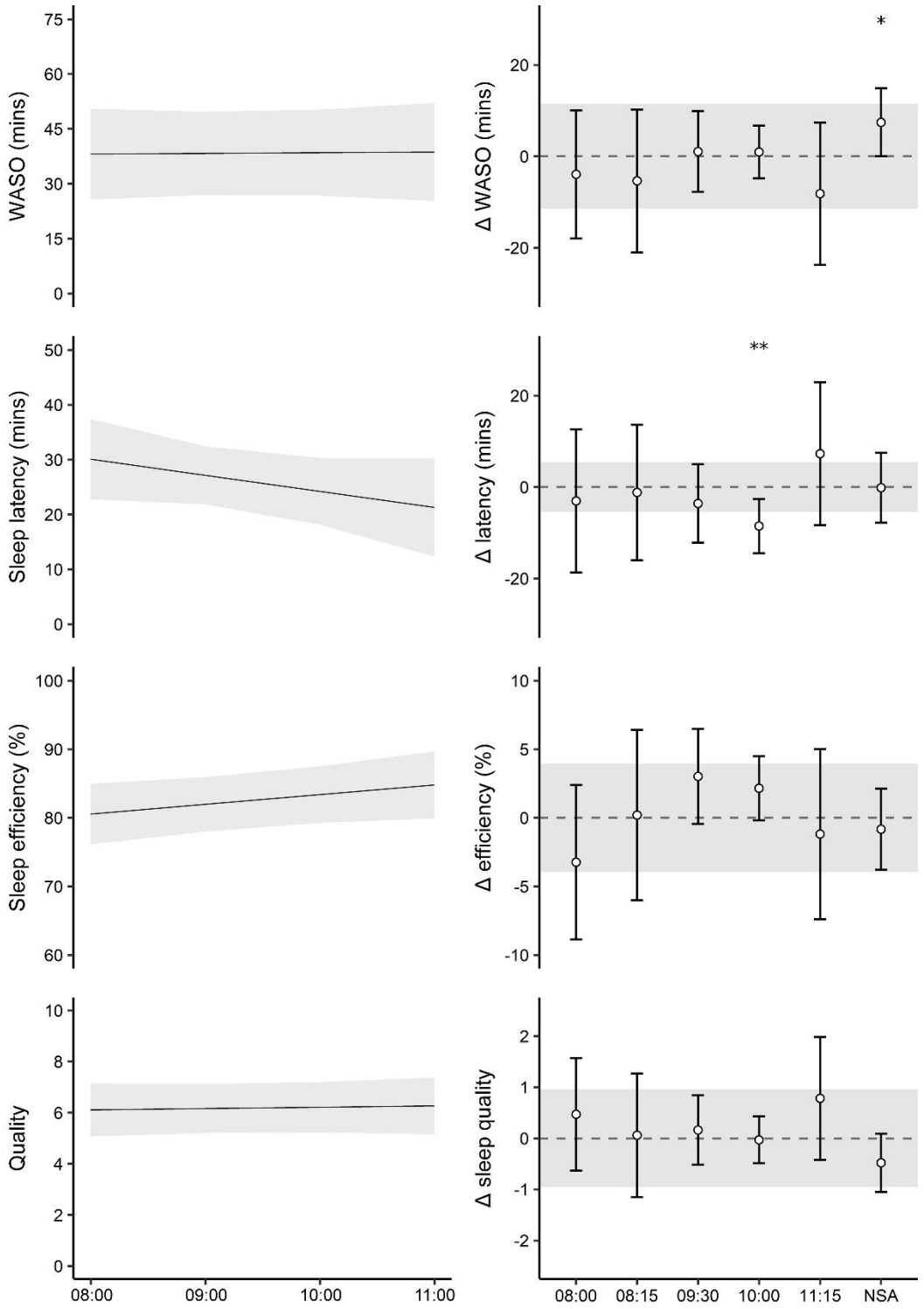

Figure 32: Data visualisation for the continuous start time model (left) and categorical start time model (right) for wake after sleep onset (WASO), sleep latency, sleep efficiency, and quality. Data are presented as beta estimates ± 95% confidence intervals (grey area). No scheduled activity (NSA). 08:00 (10), 08:15 (7), 09:00 (244), 09:30 (28), 10:00 (67), 11:15 (8), NSA (38). * p<0.05 ** p<0.01 *** p<0.001

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5.4.2. Workload

Each 100m increase in Day HSR resulted in a 4.48 min (CI:2.78–6.58min; p<.001) later sleep onset time and a 3.38min (CI:1.27–5.5mins; p=0.002) later wake time the following morning. Contrastingly, each 100m increase in acute HSR accounted for a 1.22min (CI:-2.27– -0.17; p=0.024) earlier sleep onset time. Each 100m increase in chronic HSR also accounted for a 2.58mins (CI:-4.87–-0.3; p=0.027) earlier sleep onset time and a 4.13mins (CI:-6.58– -1.68; p=0.001) earlier wake time. For every 10 DEC and 10 ACC, modelling revealed that sleep onset time was 0.9min (CI:-1.7– -0.1; p=0.004) and 1.32min (CI:-2.2– -0.42; p=0.026) earlier, respectively (Table 10). There was no significant change in sleep duration as a result of workload.

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Table 10: Results from the linear mixed multiple regression models for each activity monitor derived sleep metric with day (1 day workload), acute (accumulated 7 day workload), chronic (accumulated 28 day workload), workloads for high-speed distance, high-speed accelerations, and high-speed deceleration as the predictor variables. Beta values represent the estimated outcome change per unit change of the predictor and are presented with 95% confidence intervals.

	Latency (mins)	WASO (mins)	Quality	Time in bed (mins)	Sleep duration (mins)	Efficiency (%)	Sleep Onset time (mins)	Wake time (mins)
Predictor				**High-speed running (100m)**				
Day	-0.64 (-1.62 – 0.33)	-0.16 (-1.25– 0.94)	0.03 (-0.06– 0.12)	-2.27 (-5.92 – 1.38)	-1.37 (-4.14– 1.40)	0.10 (-0.30 – 0.50)	4.68*** (2.78– 6.58)	3.38** (1.27–5.5)
Acute	0.24 (-0.28 – 0.76)	-0.04 (-0.66 – 0.57)	-0.01 (-0.06– 0.04)	-0.17 (-2.17 – 1.83)	0.31 (-1.22 – 1.84)	0.10 (-0.12 – 0.32)	-1.22* (-2.27– -0.17)	-0.15 (-1.32–1.27)
Chronic	-0.14 (-1.18 – 0.90)	0.54 (-0.81 – 1.88)	-0.09 (-0.20– 0.02)	2.45 (-1.46– 6.36)	-1.71 (-4.96 – 1.54)	-0.43 (-0.91 – 0.05)	-2.58* (-4.87– -0.3)	-4.13*** (-6.58– -1.68)
				High-speed accelerations (10 occurrences)				
Day	-0.05 (-1.34 – 1.24)	-0.04 (-1.47 – 1.39)	-0.04 (-0.16 – 0.07)	-1.32 (-6.28 – 3.64)	-2.35 (-6.07 – 1.37)	-0.22 (-0.76 – 0.33)	-0.4 (-3.13–2.32)	-2.65 (-5.67–0.38)
Acute	0.16 (-0.21 – 0.52)	0.31 (-0.11 – 0.74)	-0.02 (-0.05 – 0.01)	0.07 (-1.35 – 1.48)	0.2 (-0.88 – 1.28)	0.05 (-0.11 – 0.21)	-0.9* (-1.7– -0.1)	-0.65 (-1.32–0.22)
Chronic	-0.23 (-0.84 – 0.38)	-0.21 (-0.93 – 0.51)	0.02 (-0.04 – 0.08)	-0.64 (-2.86 – 1.58)	-0.74 (-2.56 – 1.07)	0.04 (-0.23 – 0.31)	0.23 (-1.12–1.58)	-0.97 (-2.4– 0.47)
				High-speed decelerations (10 occurrences)				
Day	-0.05 (-1.34 – 1.24)	-0.04 (-1.47 – 1.39)	-0.04 (-0.16 – 0.07)	-1.32 (-6.28 – 3.64)	-2.35 (-6.07 – 1.37)	-0.22 (-0.76 – 0.33)	1.67 (-1.38–4.71)	-1.47 (-4.9– 1.97)
Acute	0.16 (-0.21 – 0.52)	0.31 (-0.11 – 0.74)	-0.02 (-0.05 – 0.01)	0.07 (-1.35 – 1.48)	0.2 (-0.88 – 1.28)	0.05 (-0.11 – 0.21)	-1.32** (-2.2– -0.42)	-0.72 (-1.72–0.27)
Chronic	-0.23 (-0.84 – 0.38)	-0.21 (-0.93 – 0.51)	0.02 (-0.04 – 0.08)	-0.64 (-2.86 – 1.58)	-0.74 (-2.56 – 1.07)	0.04 (-0.23 – 0.31)	0.68 (-0.6– 1.98)	-0.57 (-1.97– 0.85)

Day (1 day workload), acute (accumulated 7 day workload), chronic (accumulated 28 day workload), Wake after sleep onset (WASO). * p<0.05, ** p<0.01, *** p<0.001.

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5.5. Discussion

This explorative longitudinal study assessed whether day type, start time, and workload accounted for any variability in activity monitor-derived sleep metrics in U18 professional footballers.

To the author's knowledge, this is the first study to examine the influence of start time on sleep variables in this population. Analysis suggests that start time is a significant factor in the amount of sleep achieved by U18 footballers, with an estimated sleep extension of 19.1mins (CI: 9.4–28.79) per hour delay in start time. This also occurred in tandem with later wake times (18mins, CI:9.3–26.6), with no significant change to sleep onset times ($p>0.05$). To some extent, start time is likely to be related to day type, for example, the scheduled start time on MDs may depend on travel or kick-off time, however, start time is still a manipulatable variable, notably on TDs where coaches may have greater control.

Despite sleep extensions, it is not clear to what magnitude start time would have to be manipulated to produce a meaningful well-being or performance benefit. Whilst sleep extension protocols in athletes are limited to the collegiate level, studies have demonstrated improvements in daytime sleepiness and performance. However, extensions of ≥90mins were used [433]. The required magnitude of start time manipulation to generate synonymous levels of sleep extension may be unfeasible. Nevertheless, similar levels of sleep extension have also been reported in a cross-sectional study in American High Schools (13 to 18yrs) where each 30mins delay in school start time yielded 12mins of additional sleep [173]. Further studies have linked extensions to school start time with reductions in daytime sleepiness and improved academic performance [175]. Therefore, delaying start time may support adolescent footballers by increasing the available window for sleep. This may also be strengthened by encouraging earlier sleep onset times, although, this may not be supported by their intrinsic chronotype [431].

The players studied (17.3 ± 0.7yrs) presented with a similar mid-sleep point (04:46 ± 00:44) as a similarly aged non-athletic population (17yrs, n=458, 04:35 ± 02:14)[431]. Whilst it is acknowledged that the chronotype calculation cannot be robust due to the unknown inference of MD, it does follow that the players may benefit from a later start time [431].

Coaches should also be aware that player sleep habits may differ as a result of days off. In the present study, sleep onset time was later on the nights preceding NSA (42.1mins, CI:28.8–56.2), occurring alongside later wake times (86mins, CI:72–100) and an extended sleep duration start time (37mins, CI: 18.1–55.9), relative to a 09:00, on NSA. The change may be due to players electing to use their free time to engage in social activities and/or delay sleep in anticipation of their day off. Regardless, the change may generate circadian misalignment as players subsequently readjust sleep behaviour to coincide with training schedules; a phenomenon termed *social jetlag* [295].

WASO on NSA days was also longer (7.4min, CI:0—14.8) compared to a 09:00 start time. The reasoning is not clear; however, this may be due to increased electronic device use or social jetlag [168,295]. Sleep latency the night before a 10:00 start time was also lower with no obvious explanation. It may be related to pre-MD nerves with a 10:00 start more likely associated with MD, rather than TD. Later start times may have exhibited a similar trend if a greater number of data points were available (11:15, n=8).

Sleep duration was shorter following MD+1 in comparison to all other day types. These findings are in line with other results in similarly aged footballing cohorts [275]. The reduction may be a result of a reduced workload on MD+1 as a rest day. However, we were unable to monitor workload on MD+1 as it was exclusively the players day off (i.e., they did not train or play), so this cannot be assessed. Alternatively, without the presence of scheduling pressures, players may have chosen to modulate their sleep and social activities resulting in circadian misalignment [275,295] and reduced sleep on MD+1 [275].

Only sleep onset and wake times were associated with workload, however, results are conflicting. We report that for every 100m increase in day HSR, sleep onset and wake time are extended by 4.68min (CI:2.78—6.58mins) and 3.38mins (CI: 1.27—5.5mins), respectively. Yet, chronic HSR appeared to have the opposite effect, with every 100m increase resulting in an earlier sleep on onset time (-2.58mins, CI: -4.87— -0.3mins) and waketime (-4.13mins, CI: -6.58— -1.68mins). This may suggest a different interaction between day and chronic workloads on subsequent sleep, however, sleep duration was not affected.

The current study does not rule out any influence of workload on sleep. Activity monitors interpret nocturnal movements to infer sleep metrics [198,199]. Polysomnography studies in footballers would be needed to conclusively determine if workload affects sleep architecture. Results are not dissimilar to other studies. In English Premier League players, 1, 2, 3, and 4-day accumulated high-intensity running (classified as total distance >4m·s^{-1}) were not associated with perceived sleep quality [44,45]. However, in professional youth players, Whitworth-Turner et al [275] reported a significant relationship between total HSR (>5.5 m·s^{-1}) and subsequent objective sleep metrics. While differences in how workload was classified, and how sleep was measured, may account for discrepancies between studies, Whitworth-Turner et al [275] still reported only trivial increases in WASO, time in bed, and sleep duration per every 100m increase in HSR.

This study is limited by players' adherence to wearing their devices, as results may be biased against periods of non-adherence. Furthermore, this study was completed during the COVID-19 pandemic. Whilst data collection was not interrupted by lockdowns there may have been a latent effect of lockdowns on behaviour and chronotype [434]. This study also did not record any subjective measures;

thus, it is unclear if participants perceived an effect to the investigated variables. This data may also not reflect the sleep behaviours of other academy cohorts or senior players with differing schedules and pressures.

5.5.1. Conclusions

In conclusion, start time appeared to influence the total sleep duration that the U18 professional footballers obtained, in tandem with changes in wake times. Further interventional studies are needed to determine any effect on performance or well-being. Day type was also associated with sleep, with MD+1 exhibiting reduced sleep duration, and this may be attributable to a form of social jetlag. Commensurate with previous reports, there was little evidence to suggest that workload affected activity monitor-derived sleep metrics.

Chapter 6

6. Sensitivity to sleep loss: a Method Agreement study between three fatigue-related measures.

This chapter presents a method agreement study and an retrospective analysis that assess a novel oculomotor assessment that may be suitable to detect sleep loss in a professional sporting environment. However, during period in which this study was conducted, Southampton FC still had tightened access to its players due to the COVID-19 pandemic. Considering the potential financial and performance implications of a COVID-19 outbreak within a team, the English Premier League maintained greater precautions for a longer time than the public. Therefore, this study utilises volunteers from St Mary's University, Twickenham, and Royal Navy Divers for the method agreement and retrospective analysis portions of the study, respectively. This gave an opportunity to study within a population that is in a similar age range and one that experiences contextual factors that limits the amount time available for sleep.

6.1. Abstract

There is growing research suggesting that a smooth pursuit oculomotor assessment may be sensitive to changes in sleep and may support the assessment of sleepiness in athletes. Therefore, the aims of this study were (1) to investigate if an eye-tracking smooth pursuit assessment is sensitive to day-to-day variation in quality and quantity, and (2) to assess if the test can detect sleep loss in footballing environments. This study presents data from a Method Agreement study in 14 healthy participants (Part 1) and a Retrospective Analysis from 9 Royal Navy Clearance Divers (RNCD) (Part 2). **Part 1:** In the Method Agreement study, participants completed a smooth pursuit oculomotor task, a psychometric vigilance task (PVT), and the Epworth Sleepiness scale (ESS) for 5 consecutive days while reporting subjective sleep metrics (sleep diary), in free-living conditions. Associations between the subjective sleep metrics and the outcome variables were assessed using linear mixed model regression analysis and correlations determined the strength of any relationships between the outcome metrics. No significant associations were revealed between the subjective sleep metrics and the smooth pursuit, PVT, or ESS outcome variables ($p>0.05$). However, smooth pursuit radial variation and ESS global scores were significantly correlated ($r=0.33$, $p=0.0049$). **Part 2:** In the Retrospective Analysis, the RNCD's completed a baseline week followed by a Fatigued Phase (lasting 1 week) which generated significant sleep loss (Baseline: 7.08 ± 1.05 hrs; Fatigue Phase: 4.33 ± 1.05 hrs). Objective sleep metrics were monitored across both phases and participants completed the smooth pursuit oculomotor task and PVT each morning. Smooth pursuit mean phase error ($p=0.049$) as well as PVT mean reaction time ($p<0.001$), mean reaction time standard deviation ($p=0.030$), and median reaction time ($p<0.001$) were significantly impaired during the Fatigue phase compared to the Baseline phase. Subsequent regression analysis revealed that the PVT mean reaction time ($p<0.001$) and median reaction time ($p<0.001$), but not PVT mean reaction time standard deviation ($p=0.131$) or smooth pursuit mean phase error ($p=0.121$), were significantly associated with sleep duration. Overall, results suggest that the smooth pursuit assessment did not have the requisite sensitivity to detect daily fluctuations in sleep quality, nor was it sensitive to the magnitude of sleep loss experienced by the RNCD. Further research should investigate the relationship between oculomotor function and sleep to elucidate the most sensitive metrics to sleep loss.

6.2. Introduction

Sleep is considered essential to the maintenance of normal cognitive [17] and physiological homeostasis [16]. However, professional footballers encounter several factors that may affect their quantity and quality of sleep, including day type [275], travel [226,277], night matches [273,361], and fixture results [362]. Therefore, the development of non-invasive performance measures that are sensitive to sleep loss, or reductions in sleep quality, would be useful to practitioners to assess the sleepiness state of their players and assess athletic readiness. Assessments including the psychomotor vigilance task (PVT-10 [209]) have previously been shown to be sensitive to sleep loss [207], however, this requires participants to remain engaged throughout the 10-minute assessment. Nonetheless, there is growing research that suggests that a smooth pursuit oculomotor assessment may be sensitive to reductions in sleep quality/quantity and, consequently, may be well-placed to provide coaches with an objective assessment of player sleep state [212,215,435].

Smooth pursuit eye movements enable humans to maintain visual acuity whilst tracking a target [436]. Whilst this process may appear relatively simple, there are complex spatial and temporal predictions that circumnavigate the visuomotor processing delay between the target moving and the eye adjusting its position to maintain the target's image on the fovea [212,436]. In short, these predictions allow the eye and target to be synchronised during continuous tracking [212,436]. However, these processors are also sensitive to sleep loss and circadian misalignment. Consequently, performance on a smooth pursuit task may assist the assessment of sleep state in footballers [437], especially considering such tasks are time efficient (~3 mins) and can be completed using novel eye tracking headsets (ETHS) [212,215,435].

Research has demonstrated clear reductions in smooth pursuit performance after sleep deprivation. In a military sample, investigators noted a significant decline in eye-tracking performance after 20hrs and 24hrs of total sleep deprivation, compared to a well-rested state [211]. Likewise, in participants that maintained wakefulness for approximately 26hrs, authors report significantly reduced visuomotor precision. However, they also noted an adaption in predictive mechanisms as participants performed significantly more corrective saccades (rapid eye movements used to relocate the target) when sleep-deprived, compared to baseline measures [438], highlighting a measurable pattern in sleep deprived participants. A further investigation that utilised a similar sleep deprivation protocol noted that sleep loss generated significantly greater gaze position variability in the horizontal, and not tangential, direction. This indicates that spatial acuity was significantly affected, whilst temporal indices were preserved [210]. Further studies have also observed reduced binocular coordination during the smooth pursuit of total sleep loss [437].

Whilst these studies indicate that smooth pursuit performance is sensitive to sleep loss, the majority of studies thus far have focused on military populations exposed to total sleep deprivation (>24hrs).

Although the sleep of professional footballers has been reported to be variable [46,278], and suboptimal [279], total sleep deprivation is not the reality faced by footballers [273]. Consequently, for smooth pursuit performance to be efficacious in applied environments, tests have to show sensitivity to daily fluctuations in sleep quality. Unfortunately, the COVID-19 pandemic limited access to the players at Southampton FC, as the English Premier League placed controls to limit the risk of transmission. Therefore, in lieu of professional football players, participants of a similar age were recruited, and data was analysed in tandem with a cohort of trainee Royal Navy Divers who were subjected to contextual factors that limited the time available for sleep. Viewed together, this gave an opportunity to assess the utility of a novel eye-tracking smooth pursuit assessment in an applied environment in populations who experience similar contextual factors as a professional footballing cohort.

Therefore, the aims of this study were (1) to investigate if an eye-tracking smooth pursuit assessment is sensitive to day-to-day variation in sleep metrics, and (2) to assess if the test can detect the presence of sleep loss in a military training environment with prescribed sleep deprivation.

6.3. Methodology

There were two protocols in this study (Figure 33). The first (Part 1) represents a Method Agreement analysis between a novel oculomotor smooth pursuit test, the PVT-10, and a subjective assessment of daytime sleepiness (Method Agreement). The second (Part 2) is a Retrospective Analysis on smooth pursuit performance and PVT-10 data collected across a baseline and a fatiguing week in military divers (Retrospective Analysis).

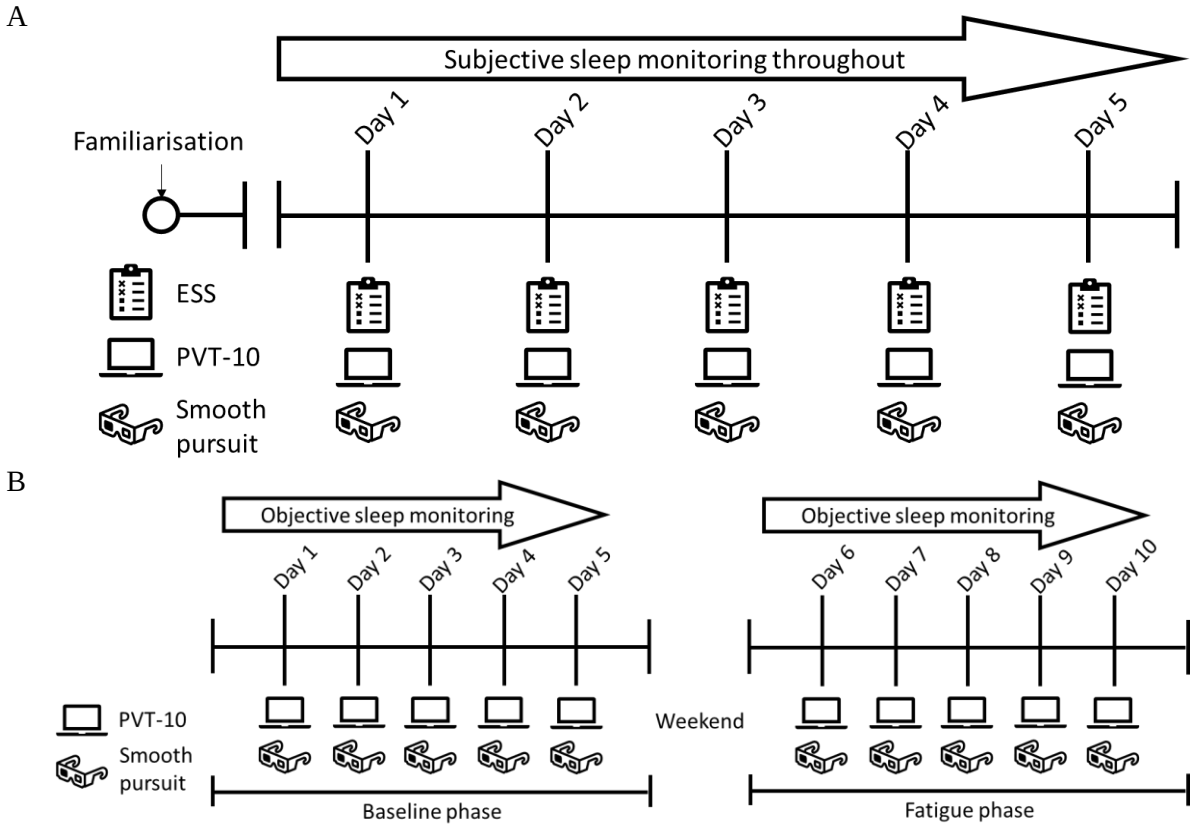


Figure 33: Protocol schematics for (A) the Method Agreement, and (B) the Retrospective Analysis.

6.3.1. Methodology Part 1: Method agreement

6.3.1.1. Method Agreement: Participants

Fourteen (m=9, f=5) participants were recruited for this study (age: 27.5 ± 4.4yrs, weight: 75.9 ± 15kg, height: 173.2 ± 10.9cm). Therefore, this study was identically powered to other method agreement studies utilising the PVT-10 [209]. Participants were included if they were aged between 18-35 and free from any diagnosed sleep issues. Throughout the research period, participants were asked to maintain their normal dietary and exercise habits but refrain from caffeine until after the testing sessions. Participants were familiarised with all procedures before the start of the study. Informed consent was obtained from each participant before data collection and ethical approval was provided faculty ethics board at St Mary's University, Twickenham.

6.3.1.2. Method Agreement: Procedure

After a familiarisation session, participants reported to the lab on 5 consecutive days for testing. Testing was completed between 08:00 am and 11:00 am and each participant attended a consistent time slot. Participants completed the smooth pursuit oculomotor test using an ETHS headset (EyeSync®, ThinkSync, Palo Alto, CA), the PVT-10 using a laptop computer, and a paper version of the Epworth Sleepiness scale (ESS). The order of the tests was randomised by assigning a number to each test and

then using a random number generator (random.org) to select the tests. All tests were completed in an isolated area to prevent distractions. Each morning on wake participants completed the Consensus Sleep Diary [439] where they self-reported the time they got into bed, the time they attempted sleep, waketime, the time they got out of bed, sleep onset latency, number of nighttime awakenings, time spent awake after sleep onset, and subjective sleep quality. The sleep diary was formatted as an online form and sent to each participant the night prior to each testing session.

6.3.2. Methodology Part 2: Retrospective analysis

6.3.2.1. Retrospective Analysis: Participants

Eight male Royal Navy Clearance divers (RNCD) volunteered to participate in this research (age: 29 ± 3yrs, height: 182 ± 6cm, weight: 81.8 ± 4.8kg). All participants provided written informed consent following a written and verbal brief of all the procedures, at least 24 hours before the first day of data collection. Ethical approval was provided by the Ministry of Defence Ethics Committee (MODREC) (Protocol number: 2088/MODREC/21) and the data were retrospectively analysed with permission.

6.3.2.2. Retrospective Analysis: Procedure

This study design consisted of two 5 day (Monday - Friday) periods separated by a weekend and was situated during weeks 11 and 12 of the RNCD training course, respectively. Week 1 (Baseline Phase) involved a mix of scheduled low-level training, classroom lessons, maintenance and scheduled dives all occurring within normal working hours (08:00-16:00hrs). Week 2 (Fatigued phase) was scheduled to simulate the high intensity of Fleet operations by requiring personnel to complete repeated dives or periods of standby during both day and night (extended working hours: 08:00-00:00 hours), leading to significant sleep loss (Table 11). Participants were expected to be in a relatively non-fatigued state during the Baseline Phase, and relatively fatigued (loss of sleep) during the Fatigued Phase.

During both the Baseline Phase and the Fatigue Phase, participants reported to a classroom at 08:00hrs for testing, which consisted of the smooth pursuit oculomotor test using an ETHS headset (EyeSync®, ThinkSync, Palo Alto, CA) and the PVT-10 using a laptop computer. Both tests took place in a secluded area, away from any distractions. Sleep was measured objectively using a ReadiBand wrist-actigraphy device (Fatigue Science Inc., Canada) whereby nocturnal movements detected by the device are converted by built-in algorithms to predict participant sleep quantity, sleep quality, awakenings per hour, total awakenings, wake after sleep onset (WASO), sleep latency, sleep onset time and wake time. ReadiBands have demonstrated good inter-device reliability and accuracy compared to polysomnography [198,199]. Bands were given to participants 3 days before the Baseline Phase, and they were asked to wear them continuously (except for dives). Data from the bands was synced to cloud-based software using a proprietary iPad application.

6.3.3. Methodology general procedures

6.3.4. Smooth pursuit test

The Method Agreement and the Retrospective Analysis both followed the same protocol for the smooth pursuit test. Participants sat with their elbows on a table and held the ETHS headset to their eyes. Participants were instructed to place their thumbs or part of their hands on their faces/heads to stabilise the device. The EyeSync® device consisted of virtual reality goggles embedded with infra-red eye-tracking sensors that determined ocular movements and predicted gaze position and velocity using proprietary algorithms. The researcher ensured that the headset was correctly positioned by asking the participant to confirm that the target and text were in focus and that at least three tracking lights were evident around each pupil (as displayed in the software's calibration interface). On the commencement of the smooth pursuit test, the EyeSync® device performed a short calibration sequence that consisted of tracking a red dot across a white background as it moved to predefined positions.

During the smooth pursuit task, participants were asked to observe and track a red target against a black background as it moved around the screen in a predictable circular pattern and velocity. The test assessed the participant's gaze location in relation to the target and characterised accuracy through the following metrics: Mean phase error (MeanPhErr; mean gaze location relative to the target), Radial variance (RadVar; a measure of spatial variability), and tangential variance (TanVar, a measure of timing variability). After each assessment, data were synced with an iPad tablet via the proprietary software and manually transferred to spreadsheet format. Participants used the same EyeSync® device throughout the respective studies.

6.3.5. Psychomotor vigilance task

The PVT-10 [208,440] was completed in an isolated area free of distractions. The test was performed on a laptop computer with a separate high-sensitivity gaming mouse (Logitech G203, Logitech, Newark, USA), as per the manufacturer's guidance [440]. On the commencement of the test, the participant was presented with a black screen, then a red counter would appear at randomised (2-10 seconds) intervals. The participant would then react by clicking a mouse button once as quickly as possible with their dominant hand. This would continue for 600 seconds (10 mins). On each successful response, the reaction time (RT) would be displayed in milliseconds. If a response was made prior to the stimulus, a 'false start' message was displayed.

6.3.6. Statistical analysis

To determine the most appropriate statistical model for the data, the Bayesian Information Criterion (BIC) was computed for a model that kept the intercept fixed for each participant (general linear model), and a model that allowed random intercepts for each participant (linear mixed model). Then the BIC

was compared between models for each outcome to determine which model was the most appropriate fit. This is an accepted method of model selection [441,442]. Results indicated that 87% of the models that allowed random intercepts fitted the data better than those that kept intercepts fixed. Consequently, linear mixed models were generated for all subsequent regressions.

In the Method Agreement study, the influence that subjective sleep metrics had on smooth pursuit, PVT-10, and ESS scores were assessed using linear mixed regression models with random slopes and intercepts for each participant. After the assumption of normality was violated (Shapiro-wilk test), a Spearman's correlation was performed between the outcome variables for the smooth pursuit assessment, PVT-10, and ESS to assess the significance of any relationships. The strength of the relationship was interpreted using predefined guidelines [443] .

In the Retrospective Analysis, smooth pursuit and PVT-10 scores across each respective phase were averaged and a linear mixed model was created with mean smooth pursuit and PVT-10 scores from each phase inputted as the outcome variable, and Baseline Phase and Fatigue Phase inputted as a categorical predictor variable. Finally, a second linear mixed model was generated that assessed the influence of objective sleep metrics on smooth pursuit and PVT-10 scores.

All data were analysed using the R statistical environment (The R Foundation for Statistical Computing) in Rstudio (Boston, USA). All data are presented with estimates and 95% confidence intervals (CI), and $P<0.05$ was considered statistically significant.

6.4. Results

Descriptive statistics for the Method Agreement and the Retrospective Analysis are displayed in Table 11.

Table 11: Mean ± SD and coefficient of variation (CV) for all performance metrics in both the Method Agreement and the Retrospective Analysis

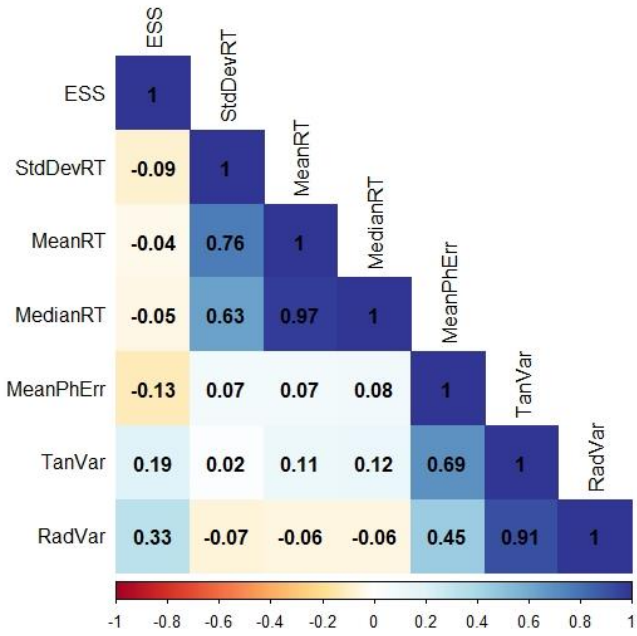
Method Agreement				
Variable	Mean ± SD	CV		
Tangential variation	1.24 ± 0.80	0.64		
Radial variation	0.91 ± 0.49	0.54		
Mean phase error	0.66 ± 4.17	6.35		
PVT-10 mean reaction time	234.70 ± 23.98	0.10		
PVT-10 mean reaction time SD	52.08 ± 17.78	0.34		
PVT-10 median reaction time	223.96 ± 20.48	0.09		
ESS	4.54 ± 3.84	0.85		
Subjective sleep duration (hrs)	7.07± 0.92	0.13		
Subjective sleep efficiency (%)	87.71 ± 7.03	0.08		
Subjective sleep onset latency (mins)	15.71 ± 11.58	0.74		
Subjective WASO (mins)	13.54 ± 23.28	1.72		
Retrospective Analysis				
	Baseline		Fatigue	
Variable	Mean ± SD	CV	Mean ± SD	CV
Tangential variation	1.40 ± 0.60	0.43	1.48 ± 0.84	0.56
Radial variation	1.02 ± 0.26	0.25	1.06 ± 0.42	0.40
Mean phase error	0.58 ± 0.27	0.47	0.74 ± 0.47	0.64
PVT-10 mean reaction time	267.27 ± 40.53	0.15	314.26 ± 73.64	0.23
PVT-10 mean reaction time SD	84.54 ± 66.92	0.79	110.80 ± 80.42	0.73
PVT-10 median reaction time	248.41 ± 28.21	0.11	286.27 ± 53.97	0.19
Objective sleep duration (hrs)	7.08 ± 1.05	0.15	4.33 ± 1.05	0.24
Objective sleep efficiency (%)	86.17 ± 12.35	0.14	89.23 ± 9.71	0.11
Objective sleep onset latency (mins)	41.63 ± 122.10	2.93	12.11 ± 21.42	1.77
Objective WASO (mins)	23.28 ± 23.85	1.02	13.21 ± 21.22	1.61

Psychometric vigilance task (PVT)
Wake after sleep onset (WASO)
Stand deviation (SD)
Coefficient of variation (CV)

6.4.1. Method Agreement

In total 70 data points were collected, per outcome, across this study. Linear mixed model analysis revealed no significant associations ($p>0.05$) between the subjective sleep metrics and the smooth pursuit performance metrics, ESS scores, or the PVT-10 performance metrics (Table 12). When the strength of any relationship between smooth pursuit performance metrics, ESS scores, and the PVT-10 performance metrics was tested using Spearman's correlation, results suggested a significant moderate relationship between the ESS global score and Radial variation ($r=0.33$, $p=0.0049$). There were no further significant between-test relationships detected ($p>0.05$) (Figure 34).

A



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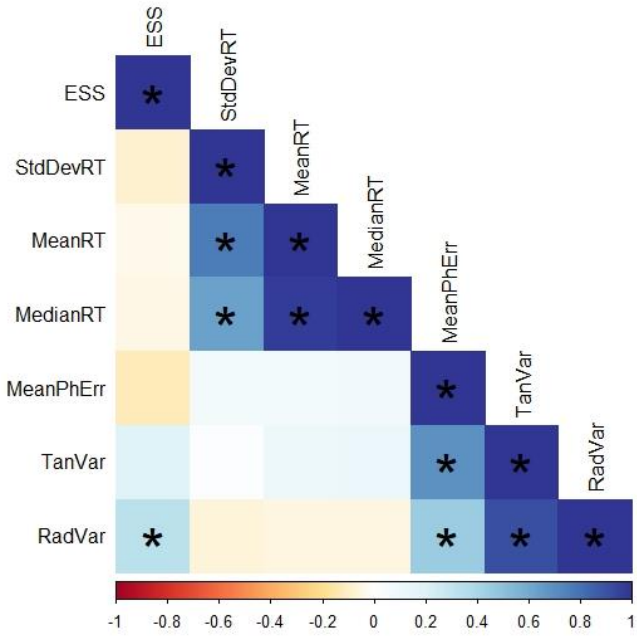

Figure 34: Non-parametric Spearman's correlation matrix for (**A**) the strength of the relationship (correlation coefficient; r); and (**B**) the location of significant relationships between the smooth pursuit, psychometric vigilance task, and the ESS outcome variables. *significant difference ($p<0.05$) *ESS (Epworth sleepiness scale), StdDevRT (PVT-10 reaction time standard deviation), MeanRT (mean reaction time), MedianRT (median reaction time), MeanPhErr (Mean phase error), TanVar (Tangential variation), RadVar (radial variation).*

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Table 12: Results from the linear mixed model regression between the subjective sleep metrics (predictor variable) and the performance metrics (outcome variable) in the Method Agreement study. Results show the change per unit sleep metric and 95% CI.

	Smooth pursuit metrics			Sleepiness scale	PVT-10 metrics		
Predictor variable	Tangential variation	Radial Variation	Mean phase error	ESS global score	Mean reaction time	Mean reaction time SD	Median reaction time
Sleep duration (hrs)	0.07 (-0.07 – 0.22)	0.05 (-0.02 – 0.13)	0.00 (-0.84 – 0.84)	-0.09 (-1.05 – 0.88)	-0.37 (-5.54 – 4.81)	-1.86 (-5.64 – 1.92)	0.55 (-3.67 – 4.77)
Sleep efficiency (%)	0.00 (-0.02 – 0.02)	0.00 (-0.01 – 0.01)	-0.03 (-0.14 – 0.07)	-0.05 (-0.17 – 0.08)	0.19 (-0.48 – 0.86)	0.27 (-0.22 – 0.77)	0.05 (-0.50 – 0.60)
Sleep onset latency (mins)	0.00 (-0.01 – 0.01)	0.00 (-0.00 – 0.01)	0.01 (-0.05 – 0.07)	0.05 (-0.02 – 0.12)	-0.21 (-0.57 – 0.16)	-0.19 (-0.45 – 0.07)	-0.08 (-0.37 – 0.22)
WASO (mins)	0.00 (-0.01 – 0.00)	0.00 (-0.00 – 0.00)	-0.02 (-0.05 – 0.02)	0.00 (-0.04 – 0.04)	0.04 (-0.17 – 0.26)	-0.06 (-0.22 – 0.10)	0.06 (-0.11 – 0.23)

Psychometric vigilance task (PVT)
Wake after sleep onset (WASO)
Standard deviation (SD)

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6.4.2. Retrospective Analysis

There was a significant difference (p=0.006) in mean nightly sleep duration between the Baseline Phase (7.08 ± 1.05 hrs) and the Fatigue Phase (4.33 ± 1.05 hrs).

Linear mixed model comparisons on the smooth pursuit performance data suggested that MeanPhErr scores were significantly (p=0.0499) impaired (represented by higher scores) during the Fatigue Phase (0.74 CI:0.56 – 0.92) compared to the Baseline Phase (0.58 CI:0.39 – 0.76). However, no other smooth pursuit performance metric was significantly altered by the phase (p>0.05) (Figure 35). There was also no significant relationship observed between smooth pursuit performance and objective sleep duration (Figure 36), nor any other objective sleep metric.

Further regression analysis on the PVT-10 performance data suggested that MeanRT (Baseline: 267.27ms CI:228.39 – 306.14, Fatigue: 314.26ms CI:275.39 – 353.14; p<0.001), MedianRT (Baseline: 248.41 CI:220.67 – 276.15, Fatigue: 286.27ms CI:258.53 – 314.01; p<0.001), and StdDevRT (Baseline: 84.54ms CI: 38.26 – 130.83, Fatigue: 110.80ms CI: 64.51 – 157.09; p=0.030) were significantly impaired (also represented by higher scores) as a result of the Fatigue Phase, compared to the Baseline Phase. Subsequent linear mixed regressions between PVT-10 performance and objective sleep metrics suggested a significant relationship between objective sleep duration and MeanRT (p<0.001) and MedianRT (p<0.001), respectively. Results suggest that for each hour increase in sleep duration MeanRT decreases (improves) by 12.51ms (CI: -18.67 – -6.36). Likewise, MedianRT decreases (improves) by 10.37ms (CI: -14.62 – -6.12) (Figure 36). There were no further significant associations between PVT-10 performance metrics and any other objective sleep metrics (p>0.05).

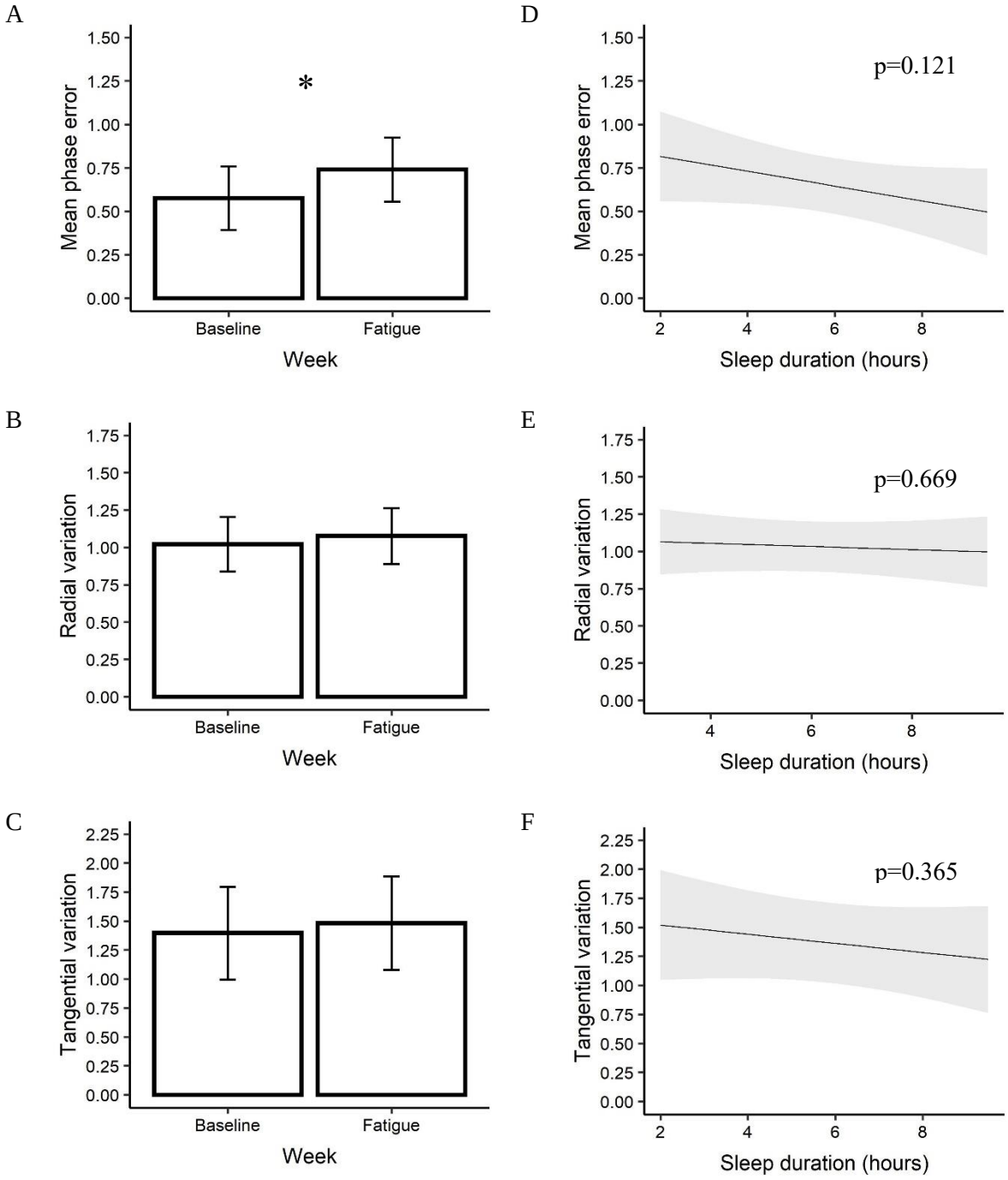

Figure 35: Visual representation of the linear mixed models from the Retrospective Analysis with the smooth pursuit performance data as the outcome variable. Plots show beta estimates with the phase as the outcome variable (A, B, C), and with objective sleep duration as the outcome variable (D, E, F). Error bars and shaded area represent 95% confidence intervals respectively. **significant difference between Baseline Phase and Fatigue Phase ($p<0.05$).*

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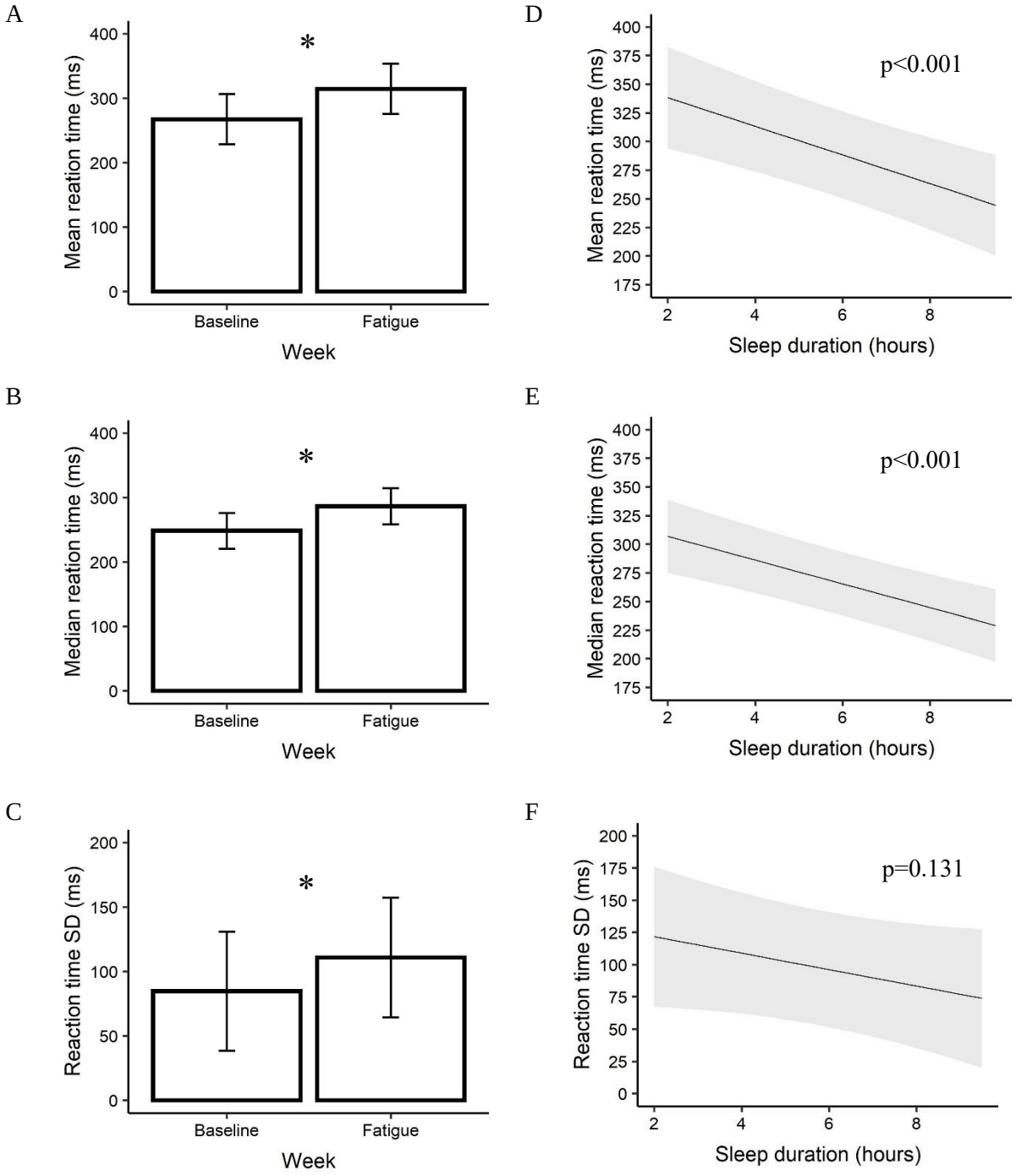

Figure 36: Visual representation of the linear mixed models from the Retrospective Analysis with the psychometric vigilance task performance data as the outcome variable. Plots show beta estimates with the phase as the outcome variable (A, B, C), and with objective

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6.5. Discussion

This study presents data from two unrelated data sets and its purpose was to investigate if an eye-tracking smooth pursuit assessment was sensitive to day-to-day variation in sleep metrics and if the test can detect the presence of sleep loss in a military training environment with prescribed sleep deprivation.

6.5.1. Method Agreement: discussion

Results from the Method Agreement study suggest that the smooth pursuit assessment, the PVT-10, and the ESS, all lack the requisite sensitivity to detect day-to-day fluctuations in subjective sleep duration, sleep efficiency, sleep onset latency, and WASO. The variation in sleep quantity and quality experienced by the participants in the Method Agreement study may not have been sufficient to mediate changes in the assessments investigated during this study. The results presented here suggest that the assessments tested in this study have little fidelity in assessing the sleep state of professional football players.

Smooth pursuit assessments have previously been shown to be sensitive to sleep loss. In a sample of healthy military volunteers, significant impairment of tangential variation was detected after 20hrs of wakefulness [438]. Likewise, following a similar protocol, another study similarly observed significant deficits in radial variation in addition to tangential variation after 24hrs of sleep loss [210,438]. However, these studies are limited by their sampling frequency with no smooth pursuit assessments being recorded before at least 20hrs of wakefulness [210,438]. Therefore, smooth pursuit performance may be sensitive to sleep loss, but the magnitude of sleep loss required to mediate performance reductions remains unknown. Nevertheless, the present study presents data suggesting that the day-to-day variation in sleep is not sufficient to reduce smooth pursuit performance. Therefore, future studies employing a gradual sleep loss protocol may further elucidate the requisite magnitude of sleep loss to illicit reductions in oculomotor function.

PVT-10 results from the Method agreement study also suggest that PVT performance metrics are not sensitive to day-to-day fluctuations in sleep, however, sleep deprivation and sleep restriction studies have highlighted significant PVT deficits after sleep loss [207]. When healthy participants were restricted to 6hrs or 4hrs of sleep per night for 14-days, or a 3-day total sleep deprivation protocol, the analysis suggested a cumulative dose-dependent reduction in PVT performance [17], with the total sleep deprivation group reporting the greatest performance reduction. However, in participants that received 8hrs of sleep per night, performance remained significantly affected throughout the duration of the study. Whilst this reaffirms the fact that PVT-10 performance is sensitive to sleep loss, it also highlights that PVT-10 deficits may not be sensitive to daily fluctuations in sleep quality and/or quantity and a

sleep loss paradigm that instils deficits that are greater than what is normally experienced is required before performance decrements are observed.

Despite previous studies demonstrating that both PVT [17,207] and the smooth pursuit [210,438] assessments experience performance deficits in response to sleep restriction or deprivation, this study suggests that the outcome variables from each respective assessment are not correlated. Therefore, it is not likely that the smooth pursuit assessment and the PVT-10 can be used interchangeably to assess sleep status. These results may be indicative of the fact that the participants in this study did not experience sleep loss outside of their normal variation, consequently, smooth pursuit and PVT performance variation may not have reached a sufficient magnitude where correlations could be detected. Alternatively, this may also suggest that the neuro-cognitive processors governing oculomotor and psychomotor function may be unrelated in the absence of sleep loss. However, based on this data, it remains unclear if the mechanisms underpinning the respective performance decrements are related.

Interestingly, a significant relationship ($r=33$, $p=0.0049$) was detected between ESS global scores and the radial variation metric of the smooth pursuit assessment. The ESS was developed to reliably assess the presence of excessive daytime sleepiness [444,445], whereas radial variability reflects spatial accuracy during visual tracking [212]. Research has highlighted declines in radial variability scores in the presence of at least 20hrs of sleep deprivation, however, a significant association was not revealed between self-reported sleep diary metrics and smooth pursuit radial variation in the present study. However, in the absence of objective sleep assessment, or a reliable way to determine sleep architecture, higher ESS may reflect a greater sense of restorative sleep, therefore, a moderately strong [443] correlation between ESS and radial variation may suggest that the latter is, in turn, related to overall sleep quality. This is a speculative notion, and these results must not be over analysed. There were no significant associations revealed through linear mixed model regressions between the sleep diary metrics and ESS score or radial variations, and any number of unexpected confounders may influence both oculomotor functions and perceptions of daytime sleepiness. Nevertheless, if future research can better define a relationship between subjective ESS and objective radial variation during a smooth pursuit task, then it may give practitioners a reliable way to objectively assess athlete sleep state and readiness.

6.5.2. Retrospective Analysis: discussion

In the Retrospective Analysis, results indicated that both PVT-10 and smooth pursuit performance were impaired during the Fatigue Phase, compared to the Baseline Phase. Specifically, tests revealed performance declines in smooth pursuit mean phase error as well as PVT-10 mean reaction time, reaction time standard deviation, and median reaction time. This suggests that an element, or series of elements, associated with the Fatigue Phase can impact the neuro-cognitive processors that govern

oculomotor and psychomotor functioning. Factors that may affect these processors are likely multifaceted [17,437], however, this study reports that subsequent linear mixed model regression analysis between the objective sleep metrics and the performance outcomes suggested that sleep duration across both phases was significantly associated with the PVT-10 mean and median reaction time, but not with smooth pursuit mean phase error. Considering that mean sleep duration during the Fatigue Phase (4.33 ± 1.05 hrs), was significantly ($p=0.006$) less than the Baseline Phase (7.08 ± 1.05 hrs), this suggests that the reduction in sleep duration mediated a portion of the performance decline that was observed in the PVT-10 metrics during the Fatigue Phase, but not the smooth pursuit performance metrics.

Previous research has investigated the effect of sleep loss on PVT performance in controlled studies that mostly account for the influence of external variables [17,207]. However, the Retrospective Analysis represents a combination of increased workload and sleep loss. Considering data was taken from RNCD as they complete their normal training routine, the respective influence of the two variables cannot be separated and individually determined. Nevertheless, whilst the Method Agreement analysis suggests that the PVT-10 test lacks the requisite sensitivity to detect normal fluctuations in sleep quality, the Retrospective Analysis supports the notion that sleep loss can cause deficits in PVT performance. The data may also suggest that the PVT has greater sensitivity to sleep restriction compared to the smooth pursuit assessment. However, the mean sleep duration during the Fatigue Phase of the Retrospective Analysis (4.33 ± 1.05 hrs) is still not representative of what is normally experienced by professional footballers [46,273,278], or the wider athletic community [20], with the possible exception of night matches [446], or long-haul travel [277]. Consequently, such a test would have limited utility in assessing the sleep state of professional players as their normal sleep patterns may not present with sufficient variation to elicit changes in PVT-10 performance. Furthermore, the PVT assessment has been described as a sustained-attention reaction time task [17], and, indeed, the PVT-10 used in both the Method Agreement and Retrospective Analysis portions of the current study takes 600 seconds to complete, leaving it susceptible to lapses in concentration, limiting its fidelity and utility. [209]. In professional and semi-professional Australian basketball players, shorter 3 and 5-minute variants of the PVT were compared to the PVT-10 [209]. However, the respective variants presented with significant differences in mean reaction time, total lapses, and total errors compared with the PVT-10 leading the authors to conclude that the three variants cannot be used interchangeably. Therefore, whilst the PVT-10 may be used to detect the magnitude of psychomotor deficits following sleep loss its practical utility may be limited.

The Retrospective Analysis reports that Smooth Pursuit mean phase error was significantly higher during the Fatigue week compared to the Baseline week, however, regression analysis did not reveal a significant relationship between mean phase error and the objective sleep metrics. Therefore, this study

suggests that mean phase error may not be affected by changes in sleep quantity and the observed change was mediated through another mechanism. Research suggests that smooth pursuit mean phase error is relatively resistant to sleep restriction, with studies in healthy military volunteers reporting no significant change in mean phase error compared to baseline after 20hrs [210,438], 24hrs [210,438], and 26hrs [438] of wakefulness. Therefore, the results from the Retrospective Analysis support what has previously been published.

The decline in mean phase error may be related to increases in workload, however, workload was not quantified as part of this study. Regardless, to the author's knowledge there is no precedent in the literature to suggest that any smooth pursuit performance metric would be affected by workload. In one study, student-athletes performed smooth pursuit assessments before and after training and the results suggested no significant difference between conditions [435]. In this investigation [435], the intensity of the respective training sessions were not described, therefore, the extent participants were fatigued is not known. Consequently, the influence of workload on smooth pursuit metrics clarify its prospective utility.

Mean phase error is formed by the angle between the gaze position and the target if it was fixed at 12 o'clock and is a measure of temporal-spatial gaze accuracy [435]. Scores can be either positive, which indicates that the mean gaze position was ahead of the target, or negative, which indicates that mean gaze position was behind the target [435]. It is interesting to note that in both the Method Agreement (0.66 ± 4.17) and the Retrospective Analysis (0.58 ± 0.27) the overall mean for smooth pursuit mean phase error was recorded as positive, which signals that the mean gaze position was anticipatory across both portions of the analysis. In studies involving military personnel [211], and student-athletes, [435] overall mean phase error has reported negative scores. The disparity between scores is not clear. The RNCD analysed in the Retrospective Analysis were already 11 weeks into the RNCD training course at the start of this study, therefore, residual fatigue and/or sleep may have influenced tracking behaviour. However, the same cannot be stated for the Method Agreement study. Other possibilities may include a learning effect or familiarisation with the testing procedures, regardless, further research is needed to guide the interpretation of the smooth pursuit metrics.

The Method Agreement analysis is limited by the use of a subjective methodology to assess sleep. Although the sleep diary is a valid and reliable method in which to gauge sleep [439], subjective measures have been reported to underestimate sleep variables in comparison to objective methods [247] and may be sensitive to internal biases [249]. Considering the Retrospective Analysis utilised wrist-actigraphy, the use of different sleep monitoring methodologies across both portions of the analysis means that results involving sleep metrics cannot be directly compared. Finally, the Retrospective

Analysis is limited by small sample size (n=9) and results may be influenced by unaccounted factors arising from the preceding weeks of RNCD training.

6.5.3. Conclusions

In conclusion, this study assessed if a novel ETHS smooth pursuit test and the PVT-10 were sensitive to daily fluctuations in sleep and if they could be used to assess sleep state in real-life ecological environments. Smooth Pursuit radial variation was significantly correlated with perception of daytime sleepiness, however, results suggested that both tests lack the requisite sensitivity to daily sleep fluctuations, although, the PVT-10 may be sensitive to sleep loss during a fatiguing training phase.

Chapter 7

7. A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study.

Publications associated with this chapter:

1. Edinborough L, Hill J, Jarvis M, Bruce-Low S, Pedlar CR. A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study. J Sports Sci. 2023 May 14:1-8. doi: 10.1080/02640414.2023.2213032.

Appendix 5: Publication associated with Chapter 7

7.1. Abstract

This case study reports on a professional football player (age: 17.6years) who was referred for sleep monitoring and intervention after reporting excessive night-time awakenings. The player undertook a series of subjective sleep assessments and objective sleep monitoring (activity monitor). Based on the data presented, a sleep hygiene intervention was prescribed. Numerical comparisons were made between pre-intervention (Pre) and post-intervention (Post) values. Objective values were also compared to reference data from a similarly aged professional cohort from the same club (n=11). Wake episodes per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9; -43%) and wake after sleep onset (WASO; Pre: 74.3 ± 31.8 mins, Post: 50.0 ± 22.8 mins, -33%) were improved from Pre to Post. Compared to the reference data, mean wake episodes per night (Pre: 7.9 ± 3.0, reference: 4.6 ± 2.6; -42%) and WASO (Pre: 74.3 ± 31.8 mins, reference: 44.3 ± 36.5 mins; -40%) were all lower compared to Pre levels.. Whilst causality cannot be proven, we observed multiple sleep metrics improving following an intervention. This provides a potential framework for practitioners looking to provide targeted sleep assessment and intervention.

7.2. Introduction

During competitive fixtures, professional football players engage in considerable amounts of high-intensity running and decelerations that can result in exercise-induced muscle damage and physiological disruption [7,24]. Numerous recovery methodologies are employed to mitigate the symptoms of exercise-induced muscle damage and restore muscle function [16], however, adequate sleep remains a pivotal factor in the restoration of both physiological and psychological homeostasis [16]. Nevertheless, studies have highlighted suboptimal sleep quality in football players [423], and observational studies have reported several factors that may influence sleep quality or quantity in footballers, including day type (e.g., match day, training day, start time etc.) [447], and/or travel commitments [226].

Practitioners have a diverse range of methodologies at their disposal that are reported to support sleep in footballers. These range from mindfulness [448], behavioural [448,449], or nutritional [16] interventions to more novel cryotherapy [364] and thermoregulatory [368] techniques. Interventions that support sleep hygiene have also gained prominence [449] and refer to the practice of adhering to behaviours that facilitate sleep while avoiding behaviours that interfere with sleep. For example, warm showers before bed reduced sleep onset latency in academy football players [450] (control: 24 ± 15mins, intervention: 17 ± 15min), and one meta-analysis suggested that the ingestion of melatonin-rich foods before bedtime may improve sleep quality scores in adolescents [451]. In semi-professional footballers, a sleep hygiene strategy that maintained a dimly lit and cool room close to bedtime and limited electronic device use 30 minutes before lights-out successfully improved sleep duration (d= 1.5) [365]. Similarly, a sleep hygiene intervention that focused on generic practical sleep habit guidance [452], followed by an individualised session was successful in improving sleep latency (~30mins) in healthy professional cricket players who had not previously reported sleep issues [232].

Sleep is a highly variable phenomenon. Notwithstanding the interindividual differences in the physiological and cognitive responses to sleep loss [231], studies have also reported more prominent intraindividual variation in sleep efficiency and onset latency in professional footballers, as well as wider athletic populations [20], compared to age-matched non-athletic controls [46]. The cause of the variation is likely multifaceted, nevertheless, individual differences in chronotype and habitual tendencies render the prescription of generic sleep recommendations illogical [453]. Consequently, an individualised approach developed in consensus with a multidisciplinary team (MDT) may be more suitable compared to team-wide interventions [232].

To the author's knowledge, there have been no reports examining the use of individualised interventions on professional athletes reporting sleep issues. Therefore, this case study reports on the results of an individualised monitoring and intervention strategy aimed at improving the subjective and objective

sleep in a professional U18 football player who was referred after reporting perceived excessive night-time awakenings and excessive night-time sweating.

7.3. Methods

7.3.1. Participant

The participant (age: 17.6yrs, height: 174cm, weight: 73kg), was a professional (full-time, contracted) footballer representing a category one English Premier League Academy. He played primarily as a central attacking midfielder and was referred for sleep monitoring and bespoke intervention after reporting perceived excessive night-time awakenings and perceived excessive night-time sweating to a member of the psychology team. Written informed consent was obtained before data collection, and this study was approved by the ethics committee at St Mary's University, Twickenham.

7.3.2. Case study procedure

Following referral, the procedures for the case study were agreed by an MDT (Figure 37) and were based on a sleep optimisation flow chart published in a consensus statement [16]. The player attended a consultation and underwent an objective sleep monitoring period before the MDT analysed the data and formulated a bespoke intervention. Finally, the player received the intervention and attended a debrief to ascertain its success and determine if any further support was needed. The purpose of this approach was to ensure that the player received the appropriate individualised support. The duration of each phase was dependent on the player's schedule and the nature of their bespoke intervention (Figure 37). In this instance, the MDT analysed and collaboratively formed the intervention package 14 days after the initial consultation and the intervention was delivered after 48 hours. The final debrief took place 28days after the delivery of the intervention. All phases took place in-season, and the player continued their normal playing and training schedule throughout.

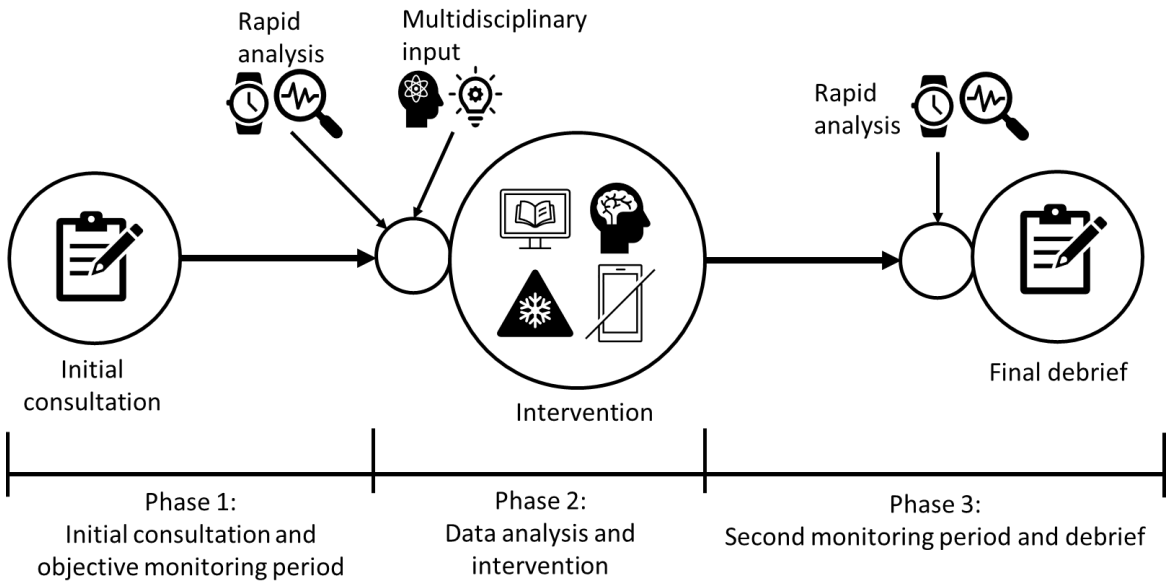

Figure 37: Case study schematic. Multidisciplinary input was provided by a panel consisting of a sports psychologist, a clinical psychologist (with a background in sleep referral), a strength and conditioning coach, and a sports physiologist.

7.3.3. Subjective and objective sleep monitoring

To assess changes in the player's perceived sleep quality, insomnia severity, and daytime sleepiness, the player completed the Pittsburgh Sleep Quality Index (PSQI [454]), Insomnia Severity Index (ISI [455]), and Epworth Sleepiness Scale (ESS [456]), respectively, during both the initial consultation and the final debrief. To gain holistic insights, the global score of each assessment was considered alongside individual components. If the player scored a component negatively, then this triggered further conversation around that topic. Furthermore, the player also completed the Morningness-Eveningness Questionnaire (MEQ [457]) and the Sleep Hygiene Index (SHI [458]) to assess chronotype and sleep hygiene, respectively. These assessments were chosen based on the MDT experience.

The player was also given a wrist-worn activity monitor (ReadiBand, Fatigue Science, Vancouver BC, Canada) that detected nocturnal movements and used proprietary algorithms to estimate sleep quantity, awakenings per hour, total awakenings, wake after sleep onset (WASO), and sleep latency. The player was given the activity monitor during the initial consultation and asked to wear it as frequently as possible on his non-dominant wrist. The data was synced to cloud-based software via Bluetooth, and a tablet computer was used to examine the status of the activity monitor. This enabled the player to continue their normal schedule without interruption. If it required charging, then the activity monitor was collected from the player, charged, and returned later the same day. ReadiBands have demonstrated good inter-device reliability and accuracy compared to polysomnography [198,199]. The player was

objectively monitored for a total of 28 days and was only able to provide data from training days due to activity monitor adherence. All data provided was at least 1 day removed from competition.

The player's objective data was compared to data collected from a sample of U18 professional players (n=11; 17.3 ± 0.7yrs) from the previous year's cohort who were monitored using the same devices over a 10-week in-season period (reference data; [447]). Considering the player in this study was only able to provide data on nights proceeding training days, only data from training days were included in the analysis from the reference data. The authors do not claim that the reference data is an example of good sleep for this population. Nevertheless, it does provide a proxy to establish what is normally experienced by players of the same demographic.

7.3.4. Bespoke sleep intervention

The intervention was formed collaboratively by the MDT. The meeting took 25 minutes and included a short case review of the baseline data and an open discussion. Potential interventions that were discussed included sleep hygiene education, mindfulness and/or cognitive therapy, and a thermal mattress to support nocturnal heat dissipation [368]. All members of the MDT unanimously agreed that an individualised sleep hygiene education session, followed by further evaluation and intervention (if appropriate) would be the most efficacious, cost-effective, and quickest intervention to deploy.

The sleep hygiene intervention session took place 48 hours after the collaborative MDT meeting in the form of an informal presentation that covered the physiology of sleep initiation and evidence-based techniques to support sleep onset, as well as a discussion on their bedtime habits and evidence-based behaviours that supported sleep. The session content was tailored to the player based on the data collected from the initial consultation and advised on a regular bedtime routine, melatonin-rich foods, and showers before bed.

This session was provided by a sports physiologist with 3 years of experience in sleep research. Generalised sleep hygiene advice was also provided based on published recommendations [16,57,452,459]. This guidance had previously been shown to improve sleep in professional athletes [232] and specific emphasis was placed on elements, raised during the consultation, that the MDT thought would have a targeted impact. A summary of the bespoke sleep hygiene strategy can be found in Table 13. The final debrief took place 28 days after the delivery of the intervention.

Table 13: Summary of the individualised and general advice provided to the player as part of their sleep hygiene strategy.

Targeted advice Player response		Strategy	Justification
1	The player reported getting into bed hours (e.g., to watch television) before attempting to sleep and was noted as having a moderate evening chronotype.	Advised player not to get into bed until he intended to sleep and to attempt sleep when he is tired.	This can reinforce a regular sleep routine and sleep onset attempts will occur during periods when melatonin release increases [16].
2	The player typically showered in the morning or after training (approx. 1500 to 1700).	Advised to have a warm shower or bath within one hour of getting into bed. No specific temperature was advised as this could not feasibly be determined within the player's home. The player was advised to self-select a temperature that they perceived to be appropriate.	A warm shower before bed can improve sleep onset latency and may support the thermoregulatory process associated with sleep onset [450].
3	The players' secondary sleep complaints included night-time sweats.	Advised maintaining a cool sleeping environment. Methods discussed included opening widows and modulating central heating	Sleep onset has a thermoregulatory component. A cool sleeping environment may support this [365].
4	The player mentioned melatonin-rich foods (walnuts, almond milk) were in his most recent nutrition plan when several examples were presented.	Suggested consuming melatonin-rich foods, in line with their nutrition plan, closer to bedtime.	Melatonin initiates processes that are associated with sleep onset and depth [451].
Additional general advice [16,57,452,459]			
1	Don’t go to bed until you are sleepy. If you aren’t sleepy, get out of bed and do something else until you become sleepy.		
2	Regular bedtime routines/rituals help you relax and prepare your body for bed (reading, warm bath, etc.).		
3	Try to get up at the same time every morning (including weekends and holidays).		
4	Try to get a full night’s sleep every night and avoid naps during the day if possible (if you must nap, limit to 1 h and avoid napping after 15:00 p.m.).		
5	Use the bed for sleep and intimacy only; not for any other activities such as TV, computer, or phone use, etc		
6	Avoid caffeine if possible (if caffeine is consumed, avoid after lunch)		
7	Avoid alcohol if possible (if must use alcohol, avoid right before bed).		
8	Avoid blue light emitted from screens at least 2 h before bed (smartphones, laptop, monitors).		
9	Meditation/ mindfulness may be helpful		

7.3.5. Analysis

Comparisons were made between Pre and Post-scores, as well as between Pre and Post-scores and the reference data.

7.4. Results

7.4.1. Pre-intervention observations

The SHI raised several areas of concern including, going to bed with psychological stress, using the bed for other activities rather than sleep or intimacy (e.g., sitting in bed watching television), and thinking or planning when in bed. During the consultation, the player also reported spending a large amount of time in the evening watching television or using electronic devices (Table 14). The player was rated as having poor sleep quality (PSQI: 22) and moderate insomnia (ISI: 15). Components that related to sleep onset latency, wake after sleep onset, feeling too hot, daytime sleepiness, enthusiasm, and overall sleep quality were rated most negatively. The MEQ suggested that the player's chronotype was a moderate evening type.

The player provided 7 days of objective sleep data after the initial consultation. The days were not consecutive, and all recorded nights proceeded training days. The objective supported what was reported by the player. Specifically, the activity-monitor reported mean awakenings per night, awakening per hour, WASO, and sleep efficiency that was greater than the reference data (Figure 38).

Table 14: Sleep hygiene index responses. A self-reported assessment of sleep hygiene behaviours [458].

	Component	Response
1	I take daytime naps lasting two or more hours	Frequently
2	I go to bed at different times from day to day.	Sometimes
3	I get out of bed at different time from day to day.	Sometimes
4	I exercise to the point of sweating within 1 hour of going to bed.	Rarely
5	I stay in bed longer than I should two or three times a week.	Rarely
6	I use alcohol, tobacco, or caffeine within 4 hours of going to bed or after going to bed.	Never
7	I do something that may wake me up before bedtime (for example: play video games, use the internet, or clean).	Frequently
8	I go to bed feeling stressed, angry, upset, or nervous.	Sometimes
9	I use my bed for things other than sleeping or sex (for example: watch television, read, eat, or study)	Always
10	I sleep on an uncomfortable bed (for example: poor mattress or pillow, too much or not enough blankets).	Never
11	I sleep in an uncomfortable bedroom (for example: too bright, too stuffy, too hot, too cold, or too noisy)	Sometimes
12	I do important work before bedtime (for example: pay bills, schedule, or study).	Rarely
13	I think, plan, or worry when I am in bed.	Frequently
	Global Score	24

7.4.2. Post-intervention observations

The player's Post-PSQI score improved compared to Pre- (Pre: 22, Post: 9), however, both remained above the threshold for '*poor'* sleep quality (>5). Components relating to sleep latency and WASO (Pre: once or twice a week, Post: less than once a week), and feeling too hot (Pre: three or more times a week, Post: less than once a week) were improved (Table 15). ISI classification was reduced from moderate

insomnia to sub-threshold insomnia (Pre: 15, Post: 8). Components relating to sleep latency and WASO were both reduced from '*Moderate'* to '*Mild'*, and the player's perceived satisfaction of his current sleep pattern improved from '*Dissatisfied'* to '*Satisfied'* (Table 16). Finally, the player's ESS classification also improved from '*moderate'* to '*mild'* daytime sleepiness (Pre: 15, Post: 11; Table 17). During the final debrief, the player self-reported a reduction in night-time awakenings and improved, but not absent, perceived night-time sweating.

Table 15: Pre and Post-PSQI responses. The PSQI is a self-rated questionnaire which assesses sleep quality and disturbances over a 1-month time interval [454].

	Component	Pre-	Post
1	When have you usually gone to bed?	22:00	23:00
2	How long (in minutes) has it taken you to fall asleep each night?	25 minutes	18 minutes
3	When have you usually gotten up in the morning?	07:00	07:00
4	How many hours of actual sleep do you get at night?	7hrs	8hrs
5	During the past month, how often have you had trouble sleeping because you…		
5a	Cannot get to sleep within 30 minutes	Once or twice a week	Less than once a week
5b	Wake up in the middle of the night or early morning	Once or twice a week	Less than once a week
5c	Have to get up to use the bathroom	Once or twice a week	Not during the past month
5d	Cannot breathe comfortably	Less than once a week	Not during the past month
5e	Cough or snore loudly	Not during the past month	Not during the past month
5f	Feel too cold	Less than once a week	Not during the past month
5g	Feel too hot	Three or more times a week	Less than once a week
5h	Have bad dreams	Once or twice a week	Less than once a week
5i	Have pain	Not during the past month	Not during the past month
6	During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?	Not during the past month	Not during the past month
7	During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?	Once or twice a week	Less than once a week
8	During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?	Once or twice a week	Less than once a week
9	During the past month, how would you rate your sleep quality overall?	Once or twice a week	Fairly good
	Global score	22	9

PSQI (Pittsburgh Sleep Quality Index)

Table 16: Pre and Post-ISI responses. The ISI is an instrument to assess the severity of both nighttime and daytime components of insomnia [455].

	Component	Pre-	Post
1	Difficulty falling asleep	Moderate	Mild
2	Difficulty staying asleep	Moderate	Mild
3	Problems waking up too early	Severe	Moderate
4	Problems waking up too early	Dissatisfied	Satisfied
5	How noticeable to others do you think your sleep problem is in terms of impairing the quality of your life?	Somewhat	A little
6	How worried/distressed are you about your current sleep problem?	A little	A little
7	To what extent do you consider your sleep problem to interfere with your daily functioning (e.g., daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) currently?	Somewhat	A little
	Global score	15	8

ISI (Insomnia Severity Index)

Table 17: Pre and Post-ESS. The ESS is a self-reported questionnaire which provides a measurement of the subject's genera level of daytime sleepiness [456].

Situation	Pre-	Post
Sitting and reading	3	2
Watching TV	2	1
Sitting inactive in a public place	1	1
As a passenger in a car for an hour without a break	2	1
Lying down to rest in the afternoon when circumstances permit	3	3
Sitting and talking to someone	1	1
Sitting quietly after lunch without alcohol	1	1
In a car, while stopped for a few minutes in traffic	2	1
Global score	15	11

ESS (Epworth Sleepiness Scales)

The player provided 7 and 8 nights of objective data for Pre and Post, respectively. From Pre to Post, the player's WASO (Pre: 74.3mins ± 31.9mins, Post: 50.0mins ± 22.8mins, -33%), sleep latency (Pre: 12.6mins ± 6.5mins, Post: 8.9mins ± 1.3mins, -29%), sleep efficiency (Pre: 79.2% ± 6.0%, Post: 85.3% ± 5.4%, 8%), awakenings per hour (Pre: 1.2 ± 0.5, Post: 0.6 ± 0.2, -50%), and awakening per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9, -43%) all improved. Compared to the reference data, WASO (Pre: 74.3mins ± 31.8mins, reference: 44.3mins ± 36.5mins, -40%), awakenings per hour (Pre: 1.2 ± 0.5, reference: 0.7 ± 0.4, -42%), awakenings per night (Pre: 7.9 ± 3.0, reference: 4.6 ± 2.6, -42%) were greater at Pre, whereas Post scores only presented with seemingly trivial differences compared to the reference data (Figure 38 and Table 18).

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Table 18: Means ± SD for Pre, Post, and Reference data alongside Pre, Post, and Reference percentage change. Negative/ positive values indicate the direction of change.

	Pre	Post	Reference	Pre vs Post	Pre vs Reference	Post vs reference
Sleep duration (mins)	394.3 ± 53.0	419.4 ± 57.4	433.4 ± 68.0	6%	10%	3%
MiB (mins)	497.4 ± 51.6	491.1 ± 56.6	533.0 ± 81.5	-1%	7%	9%
WASO (mins)	74.3 ± 31.8	50 ± 22.8	44.3 ± 36.5	-33%	-40%	-11%
Sleep latency (mins)	12.6 ± 6.5	8.9 ± 1.2	23.6 ± 26.1	-29%	87%	165%
Sleep efficiency (%)	79.2 ± 6	85.3 ± 5.4	81.9 ± 10.3	8%	3%	-4%
Awakenings per hour	1.2 ± 0.5	0.6 ± 0.2	0.7 ± 0.4	-50%	-42%	17%
Awakenings per night	7.9 ± 3	4.5 ± 1.9	4.6 ± 2.6	-43%	-42%	2%

Wake after sleep onset (WASO)

3916

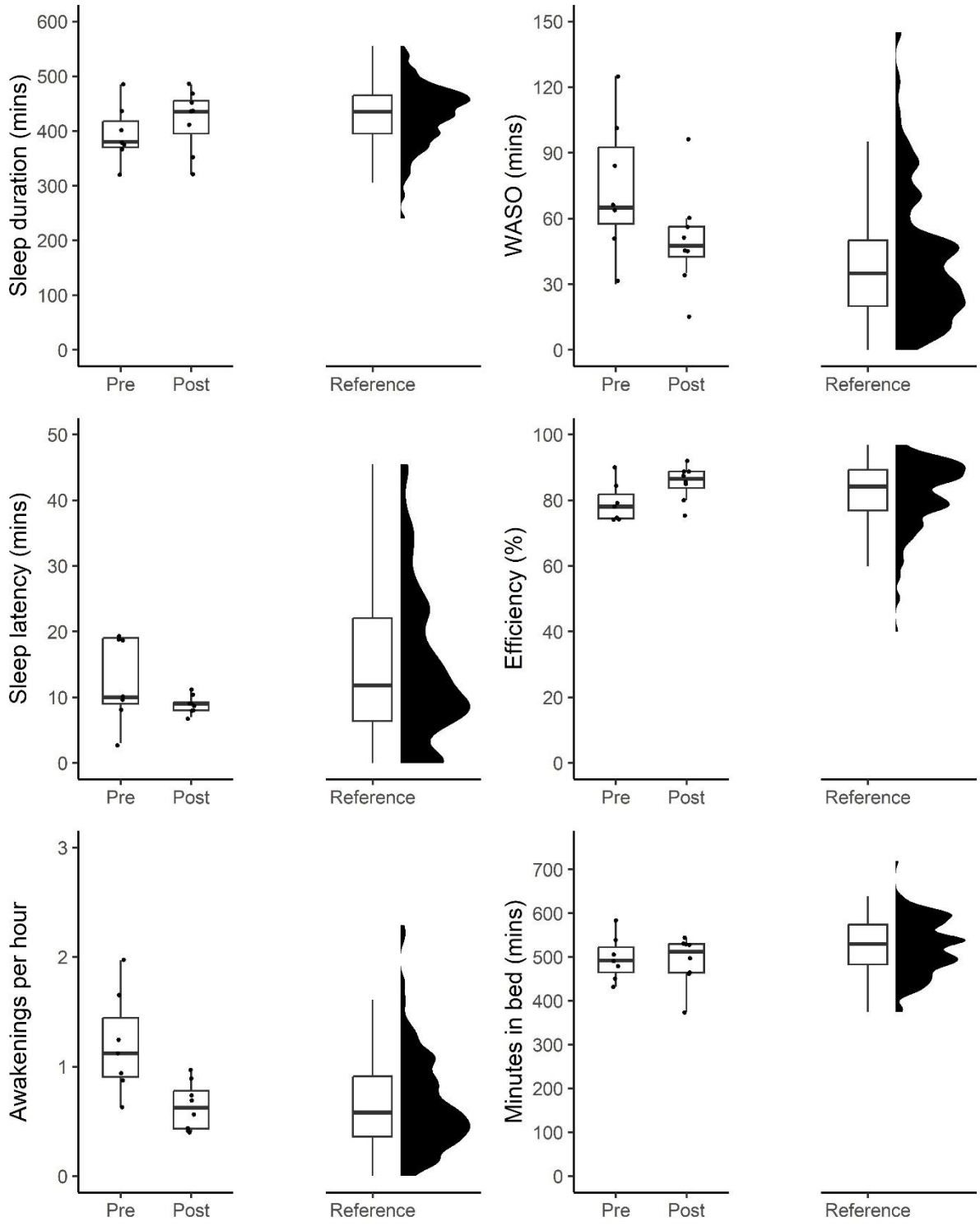

Figure 38: Box and whisker plots for Pre, Post, and the reference data. The reference data is shown alongside a cloud plot to highlight distribution. Outliers have been removed from the box and whisker plots.

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7.5. Discussion

The primary finding of this study is that the player's primary and secondary sleep complaints were improved after a bespoke sleep hygiene strategy. Notably, the player's awakenings per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9, -43%) and awakenings per hour (Pre: 1.2 ± 0.5, Post: 0.6 ± 0.2, -50%) improved from Pre to Post. Furthermore, Post data for awakenings per night and awakenings per hour was more similar to the reference data compared to Pre, suggesting that the players sleep was more in line with reference norms. Whilst this case study cannot definitively say that the sleep hygiene strategy mediated the improvements to objectively and subjectively rated sleep metrics (i.e., causality), we observed a positive response to the intervention across several sleep and sleep-related variables, indicating better sleep. It is important to note, nonetheless, that the player's objective data presented with relatively large CI (Figure 38). Whilst the large CI may be due to a low number of data points or the inherently variable nature of sleep [46], this may also indicate that the stated response could be in the opposite direction. However, considering the subjective and the objective data overall suggest a beneficial response, it is likely that a positive effect was observed.

Research has highlighted that sleep hygiene in athletes may be sub-optimal [460]. In one study, a sample of professional team sport players (n=184) scored lower on the SHI compared to a cohort of age-matched controls (n=101). Notably, athletes scored significantly lower in components relating to bedtime/wake time regularity, sleep environment, and nap behaviour suggesting that athletes, in general, may benefit from sleep hygiene interventions.

There is little data examining the effectiveness of personalised or individualised sleep hygiene interventions in athletic populations [232]. However, the limited amount of data that has been collected aligns with this case study. In international standard cricket players (n=9) [232], a one-on-one education session resulted in significantly improved activity-monitor derived sleep latency, which also like caused an improvement in sleep efficiency (+5%). In this case study, sleep efficiency improved by a similar magnitude. However, in this instance, improved WASO scores were likely the primary driver. Results from more generalised, group-based sleep hygiene interventions have also reported improved sleep, with positive results reported in both professional rugby league players [461] and non-professional football players [462]. Furthermore, in highly trained footballers [365], a sleep hygiene strategy that directly restricted ambient light, limited electronic device use, and controlled room temperature (~17°C) resulted in significantly improved post-fixture sleep duration compared to a control.

Where previous research has observed benefits to sleep duration [365,461], sleep efficiency [232], and sleep onset latency [232,462], this case study also observed a benefit to WASO, awakenings per hour, and awakenings per night, which appears unique in the literature base thus far. However, the studies involving professional or elite athletes [365,461] have excluded participants that have reported historic

sleep issues, whereas this case study investigated a professional player that was specifically referred after reporting excessive night-time awakenings. Therefore, this case study may have observed improvements in WASO, awakenings per hour, and awakenings per night because the player's scores were already suboptimal, compared to other age-matched footballers.

Alongside improvements to objective sleep metrics, this case study also reports improved PSQI, ISI, and ESS scores after the sleep hygiene intervention. Whilst the ESS rates the perception of sleepiness at the time of completion [456], the PSQI [454] and ISI [455] give a more general interpretation. Components relating to sleep onset latency, night-time awakenings, and overall sleep quality, in addition to issues with daytime sleepiness and enthusiasm were perceived to improve. Together with the objective data, this may suggest that the player perceived a benefit to their daytime functioning. Similar results have also been observed in professional cricket players [232] and non-professional footballers [462] who received a sleep hygiene intervention.

It is challenging to deduce which element, or combination of elements, of the sleep hygiene intervention mediated changes to the player's objective and subjective sleep metrics. During the final debrief, the player inferred that he perceived the consumption of melatonin-rich foods (specifically walnuts and other nuts), a shower before bed, and a more regular bedtime routine were notably beneficial. Walnuts are considered to be melatonin-rich and randomised placebo-controlled trials suggest that consumption of walnut-derived peptides can significantly improve PSQI scores in adolescent and elderly populations [451]. Whilst research is still emerging, it does indicate that the consumption of walnuts close to bedtime may increase melatonin and aid in sleep initiation. There is a more established research base surrounding the use of warm baths or showers close to bedtime to aid sleep, particularly regarding sleep initiation. This has been observed in professional adolescent football players [450], where the application of a warm shower 20 minutes before bedtime resulted in significantly improved sleep efficiency and sleep onset latency. Whilst it is beyond the scope of this case study to investigate the effectiveness of individual components on the player's sleep, this case study suggests that a combined approach is efficacious.

This case study used a combination of subjective (PSQI, ISI, ESS) and objective measures (wrist-activity monitors) to gain a holistic view of the player's sleep. However, the efficacy of such an approach should be questioned. The player was referred because they self-reported sleep disruption. This was subsequently discussed in the initial consultation and confirmed through both subjective and objective monitoring. However, the sleep assessments did not reveal anything new that the player had not already verbally stated. Therefore, if data from the initial consultation was viewed in isolation, then the sleep hygiene intervention could have been applied in the first instance, without the need for a period of objective monitoring. However, subjective assessments are potentially limited by subjective biases,

although, one advantage of utilising wrist-activity monitors is their ability to reconcile the subjective assessments. Compared to polysomnography, activity monitors have demonstrated validity [199] and their use in research has helped to elucidate several factors that may influence sleep in professional players [275]. Therefore, whilst objective measures offered little additional information compared to the subjective assessments, it did offer an opportunity to collaborate the data.

This case study has several limitations. Firstly, this was not a controlled study with a suitable comparator, thus results can neither support nor refute the efficacy of an individualised sleep hygiene intervention in professional football players reporting sleep issues. Nevertheless, it offers a potential guide to the decision-making process and provides a real-world example framework for sport science and medicine professionals when they encounter sleep issues within their practice. Further, whilst the intervention was formulated by an MDT with a wealth of applied experience and on the guidance of the data available, its formulation is still likely influenced by subjective individual biases. Therefore, the most efficacious intervention may not have been applied. Also, this case study did not monitor or re-evaluate sleep after the final debrief and it is not known if sleep metrics continued to improve or relapsed, nor was it able to elucidate sleep architecture. Finally, while the player also identified night sweats as a sleep complaint, this could not be objectively determined so did not form a central part of the discussion.

7.6. Conclusions

In conclusion, this case study applied an individualised sleep hygiene intervention to a player who was referred after reporting excessive night-time awakenings and night-time sweats. The player's subjective and objective sleep metrics subsequently improved. Whilst this case study cannot definitively say the intervention caused the changes to the sleep metrics, a player reported excessive nighttime awakenings, an intervention was applied, and then the player reported improvement. This case study provides a potential framework for coaches and sports practitioners who may encounter reported sleep issues as part of their practice.

Chapter 8

8. General discussion and conclusions

8.1. Introduction

The final iteration of this PhD thesis was influenced by the COVID-19 pandemic and subsequent restrictions. Initially, this PhD was formed in collaboration with Southampton FC and St Mary's University, Twickenham, to investigate the effectiveness and best-practise use of WBC in professional football players. This included exploring its efficacy, reported benefits, such as improved sleep quality, and how the therapy can impact performance in a professional environment. Accordingly, studies were conceptualised to aid in the understanding and utilisation of this poorly understood recovery modality. Firstly, a study was completed which aimed to conduct a rigorous meta-analysis and systematic review of studies that examined the use of post-exercise WBC, compared to passive recovery, on markers of EIMD, inflammation, redox and variables related to post-exercise fatigue and recovery in healthy and athletic populations.

Secondly, an applied cross-over designed study was enacted which aimed to (1) investigate the effect of a WBC applied across an in-season microcycle on the objective and subjective sleep quality in under 18 (U18) professional footballers, and (2) determine the effect of WBC on game-day inflammation, testosterone, and cortisol. However, the English Premier League (EPL) and Football Association (FA) decided to postpone the season shortly before the start of the second phase of this study. Subsequently, the EPL and FA declared that WBC chambers were not COVID-safe, therefore, the focus of this PhD was moved away from WBC and sleep became a more central theme. This change was formed in collaboration with St Mary's University, Twickenham, and Southampton FC and brought together the resources and technologies available to all parties. Subsequently, new research questions were formed that adhered to the overarching performance goals of Southampton FC. The new research questions were defined as:

1. What is known about the quality and duration of sleep amongst professional footballers?
2. What factors affect sleep in professional football players, specifically at SFC?
3. What are suitable and effective ways of improving sleep in professional football players?

The purpose of this chapter is to review the main findings of this thesis and discuss the applied impact of the research.

8.2. PhD narrative and summary of the main findings

To satisfy the stated aims, a combination of meta-analyses, systematic literature reviews, and interventional and longitudinal studies were completed alongside a final case study. The primary findings of each chapter are summarised herein.

8.2.1. Chapter 2 (Post-exercise whole-body cryotherapy and recovery: a systematic review and meta-analysis).

In the studies examined, there was not the requisite data to form robust conclusions regarding the efficacy of WBC; and the mechanism behind a successful stimulus remains largely unknown. Nevertheless, subsequent subgrouping suggested that multiple exposures applied across a microcycle were able to elicit a beneficial repose to some key markers of exercise-induced muscle damage. Whereas single exposures did not. These insights consequently informed how WBC was applied at Southampton FC. Specifically, players were encouraged to use the WBC chamber once a day for at least 3 consecutive days.

The meta-analysis also highlighted several studies that investigated the effect that WBC may have on sleep. Whilst the studies presented with conflicting results, the reports were consistent with anecdotal evidence from Southampton FC where players reported feeling sleepy, or described a perceived benefit to sleep, after exposure. These factors provided stimulus for the first experimental chapter.

8.2.2. Chapter 3 (The effect of whole-body cryotherapy on sleep quality and game-day endocrine and inflammatory markers in U18 professional football players)

This study was curtailed by the COVID-19 restrictions. Nevertheless, novel findings were reported. Specifically, data suggested that WBC had no significant influence on objective sleep markers, however, subjective sleep quality was greater in players who received WBC compared to those who did not. As part of this study, match-day testosterone, cortisol, and c-reactive protein (CRP) were also sampled to determine whether WBC affected their levels on match day. These markers were chosen because of the relationship between testosterone, cortisol and sleep [14], and CRP as an indicator of systemic inflammation. However, this study did not observe a statistical relationship between players who received WBC (CRYO) and players who did not (CON) for testosterone (CON: -86.1 ± 59.9 pg/ml, CRYO: -239.3 ± 157.9 pg/ml), cortisol (CON: -12.3 ± 39.5 pg/ml, CRYO: -16.9 ± 59.9 pg/ml, p=0.89), and CRP (CON: 0.048 ± 0.13, CRYO: -0.039 ± 0.29, p=0.695), suggesting that WBC did not mediate any changes in this instance.

8.2.3. Chapter 4 (How well do professional football (soccer) players sleep? A systematic scoping review of observational studies)

Academically, the purpose of this study was to describe what is known about sleep quality and quantity and identify the main literature themes concerning barriers to optimal sleep by systematically examining observational studies that have monitored sleep in full-time, professional footballers. Regarding the PhD thesis narrative, this chapter also supported refocusing the central theme from WBC to sleep in professional footballers. Due to the lack of commonality between methodological elements between

observational studies, a scoping review approach was judged to be the most appropriate review method. Results indicated that professional football players' mean sleep duration was within guidelines, however, sleep may be more variable compared to age-matched controls. It also highlighted that scheduling variables (e.g., kick-off time, home compared to away, travel) were associated with overall sleep metrics in professional football players. Therefore, these observations formed the basis of subsequent chapters.

8.2.4. Chapter 5 (Day type and start time may influence sleep in adolescent professional football players)

This chapter built on the conclusions presented in chapter 4, and to the author's knowledge, was the first study to investigate start time, and assess its association with objective sleep metrics, in professional football players. Accordingly, this study aimed to assess how start time may influence sleep the night before, how day type may influence subsequent sleep, and assess how workload may influence subsequent sleep in under 18 (U18) professional footballers. This study also provided specific insights to Southampton FC on the effect of scheduling variables (that may be unique to this club) on their players. Results suggested that start time appeared to influence the total sleep duration that the U18 professional footballers obtained (an additional 19.1mins per hour extension to start time), and that day type was also associated with sleep, with MD+1 exhibiting reduced sleep duration. There was also little evidence to suggest that workload affected activity monitor-derived sleep metrics.

8.2.5. Chapter 6 (Sensitivity to sleep loss: a Method Agreement study between three fatigue-related measures)

The EyeSync virtual reality eye-tracking smooth pursuit task was reported to be sensitive to fluctuations in sleep quality and research has demonstrated sensitivity to total (>24hour) sleep deprivation trials [210,438]. However, this chapter suggested that the device does not have the requisite sensitivity to detect the magnitude of sleep variations normally experienced by professional football players.

Considering the limitations of chapter 5, specifically that the player's adherence to wearing the bands was sub-optimal, it is postulated that the device may engage the players whilst giving them feedback on a performance metric that is associated with sleep. However, research that examines the sensitivity of the EyeSync virtual reality eye-tracking device to sleep loss has only been on military populations exposed to 24 to 26 hours of total sleep loss. Whilst football players sleep variables may be more variable compared to age-matched controls [46,278,447], total sleep deprivation is not the reality faced by the majority of football players [447]. Consequently, the aims of this study were (1) to investigate if a virtual reality eye-tracking smooth pursuit assessment is sensitive to day-to-day variation in sleep metrics, and (2) to assess if the test can detect the presence of sleep loss in applied environments. This

was achieved by running a method agreement study on university students and completing a retrospective analysis in a sample of Royal Navy Divers. Despite previous research suggesting that this device was sensitive to complete sleep deprivation, the primary finding from this study was that it lacked the requisite sensitivity to be useful in applied environments to detect sleep loss more synonymous with what is realistically experienced by professional football players.

8.2.6. Chapter 7 (A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study.)

The objective of this chapter was to bring together the approaches that have been observed throughout this thesis to implement a mixed-method sleep monitoring and intervention pathway, in collaboration with a multidisciplinary team, to test its ecological validity. Furthermore, this study also supported the consolidation of some of the primary themes developed through the production of the thesis. Therefore, this case study reports on a professional U18 football player who was referred for bespoke sleep monitoring and intervention after reporting perceived excessive night-time awakenings and excessive night-time sweating. Sleep is a highly variable phenomenon [231], consequently, an individualised approach was considered more logical compared to the prescription of generic sleep recommendations [453]. After consultation and qualitative and quantitative sleep monitoring, a sleep hygiene intervention was applied that was tailored to the player's responses. Whilst this study cannot imply causation, a player reported a sleep issue, an intervention was prescribed, and the player subsequently reported improved sleep, as measured by a set of well-established tools (e.g., Pittsburg sleep quality index, insomnia severity index, Epworth sleep quality scale, and research grade activity monitors)

The impact of this study is multifaceted. It supports the notion, that a bespoke approach is viable in players reporting sleep issues and it provides a framework for practitioners to engage with. However, to the player in question, this engagement supersedes any academic conclusions and has a direct impact on his sleep, well-being and, potentially, his performance. Therefore, this case study may have the largest utility from a personal applied standpoint.

8.3. Discussion of main findings

8.3.1. The quality and duration of sleep among professional footballers

In chapter 4, a review of observational studies indicated that professional football players, overall, achieve at least 7 hours of sleep on training days, the minimum recommended quantity according to the NSF [371]. This was evidenced by 82% of included studies reporting means above 7 hours. Similarly, 63% of observational studies also reported that mean sleep onset latency and mean WASO scores were within published guidelines [371]. Nevertheless, the reported variance in the included studies indicates that suboptimal sleep is present in professional players and direct comparisons between professional

players and age-matched controls also suggest greater variation [20]. These conclusions were made by analysing the data only from training days. In this review, training days were used as a proxy for baseline data since they are typically the most numerous day type, and they are the most removed from competition stressors. However, it is noted that training days cannot provide a true baseline because of the continued training, workload, and competition-related factors that may impact physiological or psychological variables that may antagonise restorative sleep. Therefore, this data does not provide evidence to suggest that sleep in this demographic is typically sufficient. Firstly, it is not clear what constitutes sufficient sleep in athletes, although there is some evidence to suggest increased injury risk secondary to sleep disruption [463,464]. Furthermore, the results from chapter 4 also suggest that sleep on training days is more variable compared to age-matched controls and the reported error bars suggest that suboptimal sleep (according to non-athletic recommendations) is common.

Therefore, whilst results highlight the notion that sleep in professional football players is largely within published guidelines, it also suggests that it is more variable compared to age-matched controls. Whilst the source of the additional heterogeneity warrants further investigation, the increased inter and intra-population variability suggests that some players, but not all, experience suboptimal sleep. Therefore, these results suggest that practitioners should avoid the prescription of generic team-based sleep interventions, and focus on highlighting individual players who are not in receipt of optimal sleep or feel dissatisfied with their overall sleep quality. Through this method, interventions can be applied where they will have the largest impact and it avoids layering support on players already in receipt of apparently sufficient sleep quality and quantity.

8.3.2. The effect of scheduling variables on sleep in professional football players.

This thesis supports the notion that scheduling factors can affect sleep in professional football players. In chapter 4, a scoping review was performed that suggested scheduling factors (the time and location that training, fixtures, and other commitments professional footballers may encounter, are positioned within their normal routine) were a primary literature theme regarding sleep in professional football players, and there was consistent evidence highlighting the impact that these variables can have on sleep. For example, notwithstanding the significant inter/intra-individual variation [275], studies have also reported differences according to day type (e.g., matchday (MD), MD+1) [275], and reduced sleep quality or quantity after night matches [273,361], and travel [226,277]. Furthermore, other research has highlighted the impact start time may have on sleep in adolescent students [175,431], whose chronological phenotype is typically later than the lifetime average [431]. However, start time has not been investigated as a factor that may impact sleep in professional adolescent football players who may have differing commitments compared to non-athletic populations. The subsequent chapter builds on this notion. Specifically, Chapter 5 [447] reveals, for the first time, that the scheduled start time (the time players are scheduled to arrive for training or competition) is significantly associated with sleep

duration in professional U18 football players. To the author's knowledge, this was the first time that sleep and start time have been modelled in a study of this type and, therefore, provides unique insight into the variables that may affect sleep in adolescent professional players.

Analysis suggests that start time is a significant factor in the amount of sleep achieved by U18 footballers, with an estimated sleep extension of 19.1mins (CI: 9.4–28.79) per hour delay in start time. This also occurred in tandem with later wake times (18mins, CI:9.3–26.6), with no significant change to sleep onset times ($p>0.05$). To some extent, start time is likely to be related to day type, for example, the scheduled start time on matchdays may depend on travel or kick-off time. However, start time is a manipulatable variable, notably on training days where coaches may have greater control. This highlights the applied benefit of these results. In previous chapters (Chapters 2 and 3) little evidence was provided that WBC can benefit sleep. What was observed was an equivocal selection of results from studies that investigated WBC as an ergonomic sleep aid in elite athletes in Chapter 3, and a potential subjective benefit to the sense of alertness upon wake in Chapter 4. Whilst Chapter 4 was curtailed by the national lockdown, the fact remains that clubs have to make a substantial financial investment in a therapy that may, or may not, provide a meaningful benefit. Conversely, data from this thesis demonstrates that sleep duration can be extended simply by extending the scheduled start time.

It is not clear to what magnitude start time would have to be manipulated to produce a meaningful well-being or performance benefit. The effect of sleep extension on athletes has only been applied at the collegiate level where studies have demonstrated improvements in daytime sleepiness and performance. However, extensions of ≥90mins were used [433]. Consequently, the required magnitude of start time manipulation to generate synonymous levels of sleep extension may be unfeasible. Nevertheless, similar levels of sleep extension have also been reported in a cross-sectional study in American High Schools (13 to 18yrs) where each 30mins delay in school start time yielded 12mins of additional sleep [173]. Further studies have linked extensions to school start time with reductions in daytime sleepiness and improved academic performance [175]. Therefore, delaying start time may support adolescent footballers by increasing the available window for sleep.

Based on these results, practitioners may wish to permanently schedule later start times for their adolescent athletes to promote a sleep pattern that is more suited to the intrinsic chronotype of their age [431]. This may be more logical than extending start time every time a coach wishes to increase the sleep duration within their squad. However, practitioners should note that such an approach may be counterintuitive considering the intra- and inter-variable nature of sleep [465] and may add inconsistency to a player's sleep schedule which is contrary to most sleep hygiene advice [16,57,459].

These results also have limitations. Specifically, this data may also not reflect the sleep behaviours of other academy cohorts or senior players with differing schedules, pressures, or chronotypes. However,

this data does provide direct evidence to coaches at Southampton FC that the sleep in their adolescent players can be extended by scheduling a later start time and highlights a manipulatable scheduling factor.

8.3.3. The effect of workload variables on sleep in professional football players.

Chapters 3 and 4 suggest that scheduling factors are associated with sleep variation in professional football players, however, they did not lend credence to the notion that workload affects objective sleep metrics in the same demographic. In chapter 5, objective sleep metrics were modelled against day, 7day accumulated (acute) and 28day accumulated (chronic) high-speed running (total distance (m) covered at running speeds >5.5m·s^{-1}; HSR), high-speed decelerations (a decrease in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}, DEC), and high-speed accelerations (an increase in speed for at least half a second with maximum deceleration in the period of at least 0.5m·s^{-2}; ACC). Whilst the threshold for significance was reached ($p<0.05$) for some variables, the magnitude of effect was arguably not meaningful. For example, Chapter 5 reports that for every 100m increase in day HSR, sleep onset and wake time are only extended by 4.68min (CI:2.78—6.58mins) and 3.38mins (CI: 1.27—5.5mins), respectively. Moreover, despite the reported changes to wake and sleep onset time, there was no significant change to sleep duration, so these results are unlikely to be of any concern to practitioners.

These results were collated during a 10-week in-season period, consequently, workload may have remained relatively stable throughout. Therefore, it remains plausible that sleep may still be influenced by larger changes in workload, compared to the variation in workload that is present during the in-season phases. Few studies have investigated changes in sleep quality across season phases in professional players, and what does exist remains somewhat contentious. Douchet et al. [391] observed that perceived sleep quality was reduced after a heavy-intensity microcycle, compared to a lighter-intensity microcycle, in female professional players competing in France. However, similar studies across both youth [44,45] and senior [401,409,411] professional demographics have reported no significant relationships. The cause of the disparities is not clear but may be related to the relative change in intensity across studies affecting the underlying sleep architecture, resulting in a perceived change in subjective sleep quality. Alternatively, where these studies used subjective methodologies, perceptual biases may have influenced results [247–249].

It is important to note that the data from this chapter was collected using activity-monitor wrist-actigraphy which interprets nocturnal movements through proprietary algorithms to estimate periods of wakefulness and sleep [199,416]. Consequently, these devices are unable to provide detailed information on sleep architecture [199,416]. Therefore, whilst changes in sleep duration, sleep onset latency, sleep efficiency, and WASO were not determined to have been influenced by workload in the

study presented in Chapter 5, that is not to say that the underlying sleep architecture was not influenced by changes in in-season workload. Further elucidation may be provided by determining changes in sleep architecture relative to changes in workload. This can normally be achieved through polysomnography (PSG). However, this requires relatively invasive instrumentation which may, in turn, alter a player's regular sleep routine and detract from the applied nature of this thesis. Next-generation smart wearables may have the capacity to elucidate the presence of REM/N-REM sleep and may present an interesting alternative to PSG if such devices are validated. Considering sleep architectures' link with hormonal and anabolic signalling, this future research may support the optimisation of athletic recovery in professional players.

These results are highly specific to the U18 players at Southampton FC. Other clubs with differing technical approaches may have systems that present with larger fluctuations in external workload, therefore, these results may not be readily transferred to other academy players. Nevertheless, the data presented in Chapter 5 suggests that workload is not a factor influencing sleep in the professional U18 players at Southampton FC and, overall, the results from this thesis suggest that practitioners should not be concerned about fluctuations in workload affecting sleep in their players.

8.3.4. Point-of-care measurement of sleep

The EyeSync a virtual reality eye-tracking device was purported to be sensitive to sleep loss and, therefore, may have provided a novel, interactive, and performance-centred tool to provide biological feedback to players regarding their sleep [210,438]. Previous research is limited to military samples where studies have demonstrated that smooth performance was reduced after >24hrs of total sleep deprivation [210,438]. However, whilst the sleep of professional footballers has been reported to be variable [46,278], total sleep deprivation is not the reality faced by footballers [273]. Consequently, for smooth pursuit performance to be efficacious in applied environments, tests have to show sensitivity to daily fluctuations in sleep quality. In Chapter 6, a method agreement study and retrospective analysis of data collected from Royal Navy Divers were conceptualised to test this hypothesis. However, results suggest that the EyeSync virtual reality eye-tracking device does not have the requisite sensitivity to detect daily fluctuations in sleep metrics that are normally experienced by professional football players. Considering that other studies on military personnel have observed degradations in smooth pursuit performance after 24hrs of total sleep deprivation [212,215,435], the studies from this thesis would suggest that smooth pursuit performance changes occur at greater levels of sleep loss than was reported in the studies in this thesis.

Chapter 6 also suggests that a psychometric vigilance task (PVT) lacks the requisite sensitivity to detect the sleep fluctuations that are normally experienced by football players. However, there was evidence that the PVT had greater sensitivity to sleep loss than the smooth pursuit test. In the retrospective

analysis portion of chapter 6, whilst both the PVT and the smooth pursuit performance were reduced during a 'fatiguing' week compared to the control, linear mixed modelling revealed that only the variability in PVT scores was associated with objective sleep metrics. These results should not be over-interpreted and do not suggest that the PVT is more suited to assessing sleep state in athletes compared to the smooth pursuit test. Firstly, the study has limitations, notably the sample size (n=9) and the likelihood of sustained fatigue from previous weeks' training impacting upon results. Secondly, the PVT takes 10 minutes to perform and requires the participant to remain engaged throughout [209]. This may limit its utility in applied environments where player time and buy-in may be limited. Regardless, the amount of sleep loss participants experienced in this study is not synonymous with what is normally experienced by professional players [273]. One exception may be after night games, where several papers have reported notable sleep loss [273,361,376]. However, night games are predictable events and practitioners may find it more efficient to use a subjective methodology(e.g., Leeds sleep evaluation questionnaire) to assess the quality of sleep the night before, rather than a 10-minute objective test.

This may also have been apparent in Chapter 7. Here a player reported sleep disruption and subsequently underwent a period of objective sleep monitoring, however, nothing additional was learned from the objective sleep monitoring that was not first revealed through the initial consultation and subjective assessments. It should be noted that this player was already aware and had reported sleep disruption. If another player was not aware or open about difficulties with their sleep, then a period of objective screening may have been useful. For this reason, further research should continue to investigate a point-of-care test that is sensitive to sleep loss. If such a test can be related to performance, then it could be used to educate players regarding the importance of sleep.

8.3.5. Bespoke sleep intervention framework

Chapter 7 provided a framework for a bespoke sleep monitoring and intervention pathway in professional football, and other athletic, environments. The pathway was based on guidance that was presented in a consensus statement that was published in the British Journal of Sports Medicine [16]. This study presented a case of an U18 professional football player who was referred after reporting perceived excessive night-time awakenings and night sweats. After receiving an individualised sleep hygiene intervention, both subjective and objective sleep measures reported improvements.

The case study cannot demonstrate causality between the intervention and improved sleep metrics. Nevertheless, a player reported sleep issues, the sports science and medicine staff intervened, and the player's sleep subsequently improved. Despite this, a major strength of the study is the inclusion of reference data from Chapter 4. Considering this data came from players of comparable age from the same academy, it acted valid comparator in which to assess the player's sleep scores, and any subsequent improvements. The data from Chapter 4 may not represent optimal sleep for this population, because it

is not clear what constitutes optimal sleep in professional football players. However, what it does offer is comparator data from a highly similar reference population who are not reporting perceived sleep issues.

Previous studies have observed efficacy after implementing bespoke sleep hygiene interventions in professional athletes [232], however, there are few examples of studies involving bespoke interventions in football players reporting sleep issues. Whilst a sleep hygiene intervention was not a predetermined intervention, the case study suggests that a bespoke sleep monitoring and intervention is a logical approach. Considering that sleep is a highly variable phenomenon [20], impacted by a multitude of factors including chronotype and habitual tendencies [453], the approach presented in this study is arguably more logical than team-based interventions. Therefore, chapter 7 provides a potential framework for practitioners who may wish to implement a similar scheme for their athletes.

8.3.6. Whole-body cryotherapy and sleep

Initially, WBC was the primary theme of this thesis. The meta-analysis and systematic review presented in Chapter 2 considered the influence of post-exercise WBC on sleep and subsequently reviewed 4 studies. The data was somewhat mixed, with two studies suggesting efficacy and two studies suggesting no effect. The reason for the disparity remains unclear and in the data available, the number of exposures, exposure time in relation to bedtime, level of the athlete (e.g., highly trained vs Olympic level), and duration of study were not obviously connected to the success, or lack thereof, of the intervention. Nevertheless, players at Southampton FC anecdotally reported improved subjective sleep quality after WBC exposure and no research was identified that investigated the use of WBC in professional football players as an ergonomic sleep aid. Therefore, sleep in professional footballers was also introduced as a theme and a study was designed to test the hypothesis that WBC could support the sleep of professional football players. This study was originally a cross-over designed study, however, COVID lockdowns prevented its completion and sleep in professional football players took a more central theme. Nevertheless, this thesis still provides insight into the role of WBC on sleep in professional athletes. Results from the subsequently curtailed study suggested that players who received WBC did report a significantly improved sense of alertness the morning after WBC exposure using subjective measures (e.g., Leeds sleep evaluation questionnaire), despite no significant impact on objective activity-monitor sleep markers. Since activity monitors only estimate time asleep from nocturnal movements the subjective improvements in alertness may represent an effect on the underlying sleep architecture. However, this is not clear.

These results are in partial agreement with the wider literature base. In both Olympic standard synchronised swimmers and recreationally active male participants, evening WBC appeared to significantly support sleep quality compared to control conditions. Furthermore, Douzi et al also

reported an improved sense of alertness after WBC [299]. However, Aloulou et al. [309] did not record a difference when under-23 rugby players received WBC at a similar time (~2130). Furthermore, in well-trained cyclists, post-exercise WBC did not significantly impact sleep quality throughout a 4-week high-intensity exercise intervention [359]. The cause of the disparity between studies remains unclear, although, in the limited data thus far, exposure frequency, competitive level, and time of exposure in relation to bedtime appear not to be factors that predict a therapeutic stimulus in this instance. Nevertheless, whilst the conclusions of this study are severely limited by the lockdown-mandated curtailment, this thesis suggests that WBC may support players by improving their sense of alertness the morning after an exposure.

If practitioners wish to improve player sleep and perception of alertness, then WBC may present a valid option, albeit with limited investigative support. However, there are potentially more efficacious methods of doing so rather than investing in a WBC chamber. Perhaps a good example can be drawn from Chapter 7, which details a case study of a single player reporting sleep issues. In this study, a bespoke sleep hygiene intervention was applied which elicited notable improvements to both subjective and objective sleep metrics. Whilst participants from studies are not directly comparable despite being similar ages, due to participants from Chapter 3 not reporting sleep issues, it does suggest that practitioners should explore less expensive options before investing in a WBC chamber simply to improve sleep. Although, it should be noted that the WBC chamber is purported to offer wider-reaching benefits than just ergonomic sleep aid [366].

8.3.7. Limitations

The primary finding of this thesis is that start time is significantly associated with the amount of sleep U18 professional footballers receive. However, this thesis also provides evidence that workload is not significantly associated with activity monitor-derived sleep metrics. As discussed, activity monitors measure nocturnal movements and then use proprietary algorithms to estimate sleep onset time, wake time, WASO, sleep onset latency, time-in-bed, and total sleep duration [198,199]. However, activity monitors do not possess the ability to assess further physiological metrics, specifically ones that are associated with REM/NREM. Therefore, these findings are limited by methodologies' inability to elucidate sleep architecture in participants. It remains credible that changes in workload may still be associated with changes in sleep architecture, respiratory rate, heart rate variability, and thermoregulation. Considering sleeps association with anabolic signalling, disruption to these factors may still affect athletic recovery.

Furthermore, whilst research-grade activity monitors are considered a reliable and valid method of sleep monitoring, polysomnography (PSG) is the gold standard due to its ability to measure a range of

physiological metrics associated with wakefulness and sleep states, including respiratory, cardiovascular, and brain wave activity.

Furthermore, activity monitor sleep assessments cannot elucidate sleep architecture, consequently, important information regarding the depth of sleep would be missed by relying on activity monitors alone. That said, PSG requires extensive instrumentation and is relatively invasive. Therefore, its use may inadvertently disrupt the normal sleep pattern of the players, detracting from the applied impact.

This thesis also heavily relied on U18 teams as participants. This may limit the conclusions that can be transposed from these studies to more senior players. For example, in Chapter 5 it was suggested that U18 players may benefit from later start times due to their intrinsic chronotype favouring later bedtimes and wake times compared to other ages. This would, in turn, suggest that more senior players may not benefit from later start times as their intrinsic chronotype may transition to an earlier phenotype.

Moreover, data from all experimental Chapters presented within this thesis were collected during in-season periods. Differing workloads [391], perceived psychological stress/recovery balance [274], and team performance [393] may have influenced results throughout this thesis.

8.3.8. Future research

This thesis highlights several avenues for future research. Firstly, commercially available wearable devices that can noninvasively measure heart rate variability and skin temperature have recently received investigative interest [466]. If subsequent research demonstrates reliability and validity, then such devices may provide a greater depth of understanding of how scheduling and workload variables impact the sleep architecture in professional players, and the wider athletic base, without them undergoing relatively more invasive instrumentation that may impact upon their normal routine.

Furthermore, this thesis provides information that suggests that start time is significantly associated with sleep duration in U18 professional players. The players' intrinsic biological chronotype may be better suited to later bedtimes and wake times, however, as players age beyond late adolescence and their chronotype moves towards an earlier phenotype, then any benefit of a later state time may dissipate [431]. Therefore, future research may wish to investigate the impact of scheduling variables across age groups. This would allow teams to better focus on the need for sleep support at specific stages of players' careers.

Similarly, further research should be dedicated to factors that may impact player sleep throughout their career. For example, it is well established that new parents experience a drop in sleep quality and/or quantity as a result of parental responsibilities [467] Therefore, understanding the magnitude and nature

of any sleep disruption in professional players may allow clubs to support players well fair by targeting sleep interventions for players who are likely to need sleep support.

8.3.9. Conclusions

The original purpose if this thesis was to assess the use of WBC as a recovery aid in professional football players. This included a study that tested the hypothesis that WBC could be used as an ergonomic sleep aid in professional football players. However, due to the influence of COVID, sleep in football players became the primary theme of this thesis. Nevertheless, this thesis adds to the literature base with several key findings that were developed over 5 investigative chapters. Therefore, the primary conclusions of this thesis are as follows:

- For the first time, start time is significantly associated with the amount of sleep that U18 professional football players receive. This is a new novel finding that was demonstrated in this population for the first time.
- In the same population, workload was not observed to be significantly associated with activity monitor-derived sleep metrics.
- Scheduling variables were also noted as being a consistent factor that influences sleep variables in professional players.
- The smooth pursuit oculomotor test, as performed on the EyeSync eye-tracking device, did not have the requisite sensitivity to detect day-to-day fluctuation in sleep loss.
- After meta-analysis, this thesis suggests that multiple WBC exposures are more successful at eliciting a therapeutic response to symptoms of EIMD, compared to single exposures
- WBC may support perceived alertness the morning after exposure and, whilst activity-derived sleep metrics were unchanged, this may be due to improved sleep.
- Finally, a bespoke sleep hygiene intervention may have improved sleep in a U18 professional player, and this thesis provides a potential framework for practitioners to consider should they encounter an athlete experiencing sleep issues.

Furthermore, this thesis highlights potential avenues for future research. Notably, the next generation of wearable technologies reportedly can measure heart rate variability and skin temperature. If subsequent investigations determine these devices to be reliable and valid, then greater information can be gained regarding the sleep properties of professional athletes in response to workload, scheduling, and other stimuli. Considering sleeps relationship with inflammatory, endocrine, and psychological homeostasis, then these tools may allow practitioners to highlight players in need of support and intervention with much greater fidelity compared to what is available.

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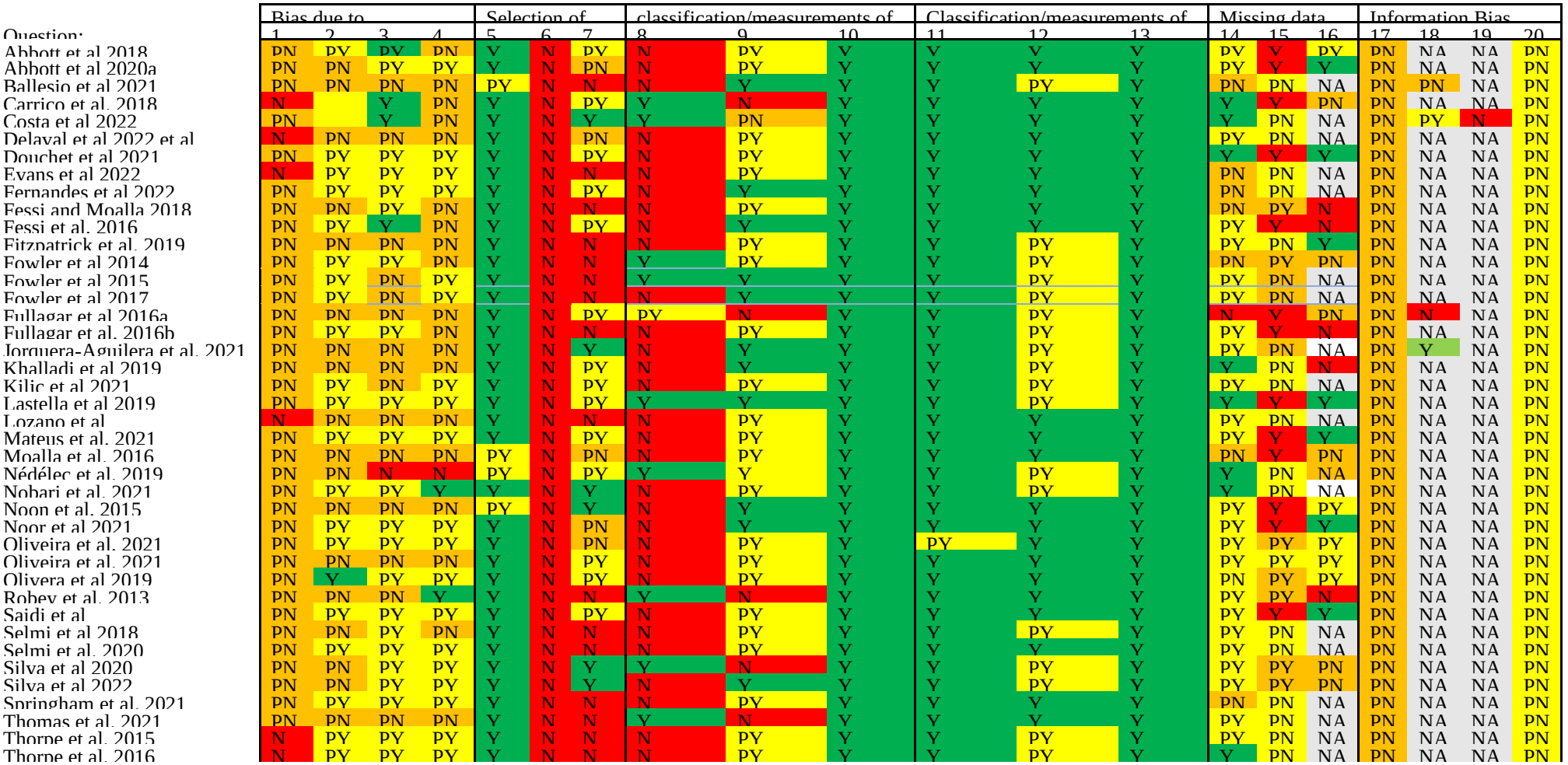
10. Appendices

10.1. Appendix 1: Chapter 4 supplementary materials

10.1.1. Risk of bias assessment




Thorpe et al. 2017 N PY PY PY Y N N N PY Y Y PY Y PY PN NA PN NA NA PN
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Signalling questions:

1. Inclusion of intraindividual factors that can feasibly affect sleep as covariates (e.g., age, chronotype, sleep related issues, internal load, etc)
2. Inclusion of external factors that can feasibly affect sleep as covariates (e.g., country, match location, training location, season period [if monitoring across micro/meso cycles], external workload, etc)
3. Were these variables measured reliably and validly?
4. Were appropriate methods or method design employed to account for all the important confounding variables?
5. Was selection into the study related to both the exposure (Pro football) and outcome (sleep)
6. Was any statistical method used to select the participants (e.g., randomly selected)?
7. Was there a well-defined inclusion/exclusion criterion that clearly accounted for the level of competition, periods of injury, and adherence to monitoring?
8. Was sleep objectively measured using a validated, reliable, research grade device?
9. Was sleep subjectively measured using a recognised questionnaire or diary?
10. Was sleep sampled at a consistent time point throughout the monitoring period?
11. Was the exposure (professional football) well defined? (e.g., level of competition, season phase, number of games, number of training sessions, country settings)
12. Was the exposure (monitoring) duration sufficient to draw robust conclusions?
13. Was the exposure consistent for all players?
14. Were outcome data available for all, or nearly all (interpreted as enough to be confident of the findings), participants?
15. Were any participants, or any individual data point, excluded or missing?
16. Was the reason for missing data clear and obvious?
17. Were outcome assessors unaware of the exposure status of study participants?
18. Were the methods of outcome assessment comparable across exposure groups (if applicable)?
19. Was the definition of case status/control status applied without knowledge of exposure status (if applicable)?
20. Was data collection on exposure status unaffected by knowledge of the outcome or risk of the outcome?

10.2. Appendix 2: Chapter 5 supplementary materials

10.2.1. Blank R coding

```
## Key ##
#df  dataframe
## packages
{library(readxl)
library(emmeans)
library(sjstats)
library(lme4)
library(lmerTest)
library(MuMIn)
library(sjPlot)
options(scipen = 999)}

## linear mixed model anova, repeat for each sleep variable ##
LMM_ANOVA <- lmer(df$sleep_variable ~ as.factor(df$day_type) + (1|df$ID)) ###linear model DV
predicted by the IV
summary(LMM_ANOVA) ### summary of model
anova(LMM_ANOVA)                              ###show model as anova
eta_sq(LMM_ANOVA, partial = TRUE)                   ### partial eta sq
r.squaredGLMM(LMM_ANOVA)                            ### Rsq
emmeans(LMM_ANOVA, list(pairwise ~ day_type), adjust = "bonferroni")  ###post hoc

## Linear mixed model multiple regression for external work load ##
LMM_mRegression <-  lmer(df$sleep_variable ~  df$acute+ df$chronic + as.numeric (df$Ratio) + (1|
df$ID)) ### linear model
summary (LMM_mRegression) ###  summary of model
tab_model(LMM_mRegression) ### out put model as HTML table

## Linear mixed model multiple regression for start time ###
##### set factors #####
df$Start_time <- factor(df$Start_time,
                        levels = c("09:00:00",
                                   "08:00:00",
                                   "08:15:00",
                                   "09:30:00",
                                   "10:00:00",
                                   "11:15:00",
                                   "NSA"
##### contrasts and dummy coding ######
`08:00 vs 09:00`<-  c(0,1,0,0,0,0,0)
`08:15 vs 09:00`<-  c(0,0,1,0,0,0,0)
`09:30 vs 09:00`<-  c(0,0,0,1,0,0,0)
`10:00 vs 09:00`<-  c(0,0,0,0,1,0,0)
`11:15 vs 09:00`<-  c(0,0,0,0,0,1,0)
`NSA vs 09:00`<-  c(0,0,0,0,0,0,1)

contrasts(df$Start_time) <-
  cbind(`08:00 vs 09:00`,
        `08:15 vs 09:00`,
        `09:30 vs 09:00`,
        `10:00 vs 09:00`,
        `11:15 vs 09:00`,
```

```
5519          `NSA vs 09:00`)
5520    ##### regression #####
5521    LMMstart_time <-
5522      lmer(df$Sleep_variable ~  df$Start_time + (1|ID))
5523    summary (LMMstart_time)
5524    tab_model(LMMstart_time)
5525
```

Appendix 3: Chapter 6 supplementary materials
Blank R coding

```
##### Bayesian Information Criterion comparison for General and linear mixed models

GLM_Varible 1_BIC <-
  gls(Variable 1 ~ 1,
      data = DF,
      method = "ML")

LMM_ Varible 1_BIC <-
  lme(Variable 1  ~ 1 ,
      data = DF,
      random = ~1|ID,
      method = "ML")

anova(GLM_Varible 1_BIC,
LMM_ Variable 1_BIC)

# Key#
Variable 1: Variable
DF: dataframe
ID: Identifier

####### LLM for comparator variables

Name of model <-
  lmer(outcome_variaible~input__variable + (1|ID),
       data = master)

summary(Name of model)

ID: Identifier
```

10.3. Appendix 4: Publication associated with Chapter 5

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10.4. Appendix 5: Publication associated with Chapter 7

Journal of Sports Sciences

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A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study

Luke Edinborough, Jessica Hill, Mark Jarvis, Stewart Bruce-Low & Charles R Pedlar

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SPORTS PERFORMANCE

A bespoke sleep monitoring and sleep hygiene intervention improves sleep in an U18 professional football player: A case study

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ABSTRACT

This case study reports on a professional football player (age: 17.6 years) who was referred for sleep monitoring and intervention after reporting excessive night-time awakenings. The player undertook a series of subjective sleep assessments and objective sleep monitoring (activity monitor). Based on the data presented, a sleep hygiene intervention was prescribed. Numerical comparisons were made between pre-intervention (Pre) and post-intervention (Post) values. Objective values were also compared to reference data from a similarly aged professional cohort from the same club ($n = 11$). Wake episodes per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9; −43%) and wake after sleep onset (WASO; Pre: 74.3 ± 31.8 mins, Post: 50.0 ± 22.8 mins, −33%) were improved from Pre to Post. Compared to the reference data, mean wake episodes per night (Pre: 7.9 ± 3.0, reference: 4.6 ± 2.6; −42%) and WASO (Pre: 74.3 ± 31.8 mins, reference: 44.3 ± 36.5 mins; −40%) were all lower compared to Pre levels. Whilst causality cannot be proven, we observed multiple sleep metrics improving following an intervention. This provides a potential framework for practitioners looking to provide targeted sleep assessment and intervention.

ARTICLE HISTORY
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KEYWORDS
Recovery; objective; subjective; wrist-actigraphy; athlete

Introduction

During competitive fixtures, professional football players engage in considerable amounts of high-intensity running and decelerations that can result in exercise-induced muscle damage and physiological disruption (Harper et al., 2019; Varley et al., 2017). Numerous recovery methodologies are employed to mitigate the symptoms of exercise-induced muscle damage and restore muscle function (Walsh et al., 2021), however, adequate sleep remains a pivotal factor in the restoration of both physiological and psychological homoeostasis (Walsh et al., 2021). Nevertheless, studies have highlighted suboptimal sleep quality in football players (Rijken et al., 2016), and observational studies have reported several factors that may influence sleep quality or quantity in footballers, including day type (e.g., match day, training day, start time etc.) (Edinborough et al., 2022), and/or travel commitments (Lastella et al., 2019).

Practitioners have a diverse range of methodologies at their disposal that are reported to support sleep in footballers. These range from mindfulness (Murawski et al., 2018), behavioural (Biggins et al., 2019; Murawski et al., 2018), or nutritional (Walsh et al., 2021) interventions to more novel cryotherapy (Douzi et al., 2019) and thermoregulatory (Aloulou et al., 2020) techniques. Interventions that support sleep hygiene have also gained prominence (Biggins et al., 2019) and refer to the practice of adhering to behaviours that facilitate sleep while avoiding behaviours that interfere with sleep. For example, warm showers before bed reduced sleep onset latency in academy football players (Whitworth-Turner et al., 2017) (control: 24 ± 15 mins, intervention: 17 ± 15 min), and one meta-analysis suggested that the ingestion of melatonin-rich foods before bedtime may improve sleep quality scores in adolescents (Yeh et al., 2022). In semi-professional footballers, a sleep hygiene strategy that maintained a dimly lit and cool room close to bedtime and limited electronic device use 30 minutes before lights-out successfully improved sleep duration (d = 1.5) (Fullagar et al., 2016). Similarly, a sleep hygiene intervention that focused on generic practical sleep habit guidance (McCloughan et al., 2014), followed by an individualised session was successful in improving sleep latency (~30 mins) in healthy professional cricket players who had not previously reported sleep issues (Driller et al., 2019).

Sleep is a highly variable phenomenon. Notwithstanding the interindividual differences in the physiological and cognitive responses to sleep loss (Nedelec et al., 2018), studies have also reported more prominent intraindividual variation in sleep efficiency and onset latency in professional footballers, as well as wider athletic populations (Leeder et al., 2012), compared to age-matched non-athletic controls (Whitworth-Turner et al., 2018). The cause of the variation is likely multifaceted, nevertheless, individual differences in chronotype and habitual tendencies render the prescription of generic sleep recommendations illogical (Fullagar & Bartlett, 2016). Consequently, an individualised approach developed in consensus with a multidisciplinary team (MDT) may be more suitable compared to team-wide interventions (Driller et al., 2019).

To the author's knowledge, there have been no reports examining the use of individualised interventions on professional athletes reporting sleep issues. Therefore, this case study

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reports on the results of an individualised monitoring and intervention strategy aimed at improving the subjective and objective sleep in a professional U18 football player who was referred after reporting perceived excessive night-time awakenings and excessive night-time sweating.

Methods

Participant

The participant (age: 17.6 yrs, height: 174 cm, weight: 73 kg), was a professional (full-time, contracted) footballer representing a category one English Premier League Academy. He played primarily as a central attacking midfielder and was referred for sleep monitoring and bespoke intervention after reporting perceived excessive night-time awakenings and perceived excessive night-time sweating to a member of the psychology team. Written informed consent was obtained before data collection, and this study was approved by the ethics committee at St Mary's University, Twickenham.

Case study procedure

Following referral, the procedures for the case study were agreed by an MDT (Figure 1) and were based on a sleep optimisation flow chart published in a consensus statement (Walsh et al., 2021). The player attended a consultation and underwent an objective sleep monitoring period before the MDT analysed the data and formulated a bespoke intervention. Finally, the player received the intervention and attended a debrief to ascertain its success and determine if any further support was needed. The purpose of this approach was to ensure that the player received the appropriate individualised support. The duration of each phase was dependent on the player's schedule and the nature of their bespoke intervention (Figure 1). In this instance, the MDT analysed and collaboratively formed the intervention package 14 days after the initial consultation and the intervention was delivered after 48 hours. The final debrief took place 28 days after the delivery of the intervention. All phases took place in-season, and the player continued their normal playing and training schedule throughout.

Subjective and objective sleep monitoring

To assess changes in the player's perceived sleep quality, insomnia severity, and daytime sleepiness, the player completed the Pittsburgh Sleep Quality Index (PSQI (Buysse et al., 1989)), Insomnia Severity Index (ISI (Bastien et al., 2001)), and Epworth Sleepiness Scale (ESS (Kendzerska et al.,)), respectively, during both the initial consultation and the final debrief. To gain holistic insights, the global score of each assessment was considered alongside individual components. If the player scored a component negatively, then this triggered further conversation around that topic. Furthermore, the player also completed the Morningness-Eveningness Questionnaire (MEQ (Natale et al., 2006)) and the Sleep Hygiene Index (SHI (Mastin et al., 2006)) to assess chronotype and sleep hygiene, respectively. These assessments were chosen based on the MDT experience.

The player was also given a wrist-worn activity monitor (ReadiBand, Fatigue Science, Vancouver BC, Canada) that detected nocturnal movements and used proprietary algorithms to estimate sleep quantity, awakenings per hour, total awakenings, wake after sleep onset (WASO), and sleep latency. The player was given the activity monitor during the initial consultation and asked to wear it as frequently as possible on his non-dominant wrist. The data was synced to cloud-based software via Bluetooth, and a tablet computer was used to examine the status of the activity monitor. This enabled the player to continue their normal schedule without interruption. If it required charging, then the activity monitor was collected from the player, charged, and returned later the same day. ReadiBands have demonstrated good inter-device reliability and accuracy compared to polysomnography (Chinoy et al., 2021; Driller et al., 2016). The player was objectively monitored for a total of 28 days and was only able to provide data from training days due to activity monitor adherence. All data provided was at least 1 day removed from competition.

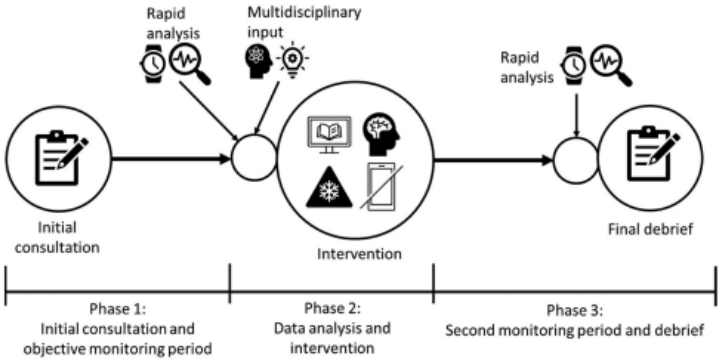

Figure 1. Case study schematic. Multidisciplinary input was provided by a panel consisting of a sports psychologist, a clinical psychologist (with a background in sleep referral), a strength and conditioning coach, and a sports physiologist.

The player's objective data was compared to data collected from a sample of U18 professional players ($n = 11$; 17.3 ± 0.7 yrs) from the previous year's cohort who were monitored using the same devices over a 10-week in-season period (reference data (Edinborough et al., 2022)). Considering the player in this study was only able to provide data on nights proceeding training days, only data from training days were included in the analysis from the reference data. The authors do not claim that the reference data is an example of good sleep for this population. Nevertheless, it does provide a proxy to establish what is normally experienced by players of the same demographic.

Bespoke sleep intervention

The intervention was formed collaboratively by the MDT. The meeting took 25 minutes and included a short case review of the baseline data and an open discussion. Potential interventions that were discussed included sleep hygiene education, mindfulness and/or cognitive therapy, and a thermal mattress to support nocturnal heat dissipation (Aloulou et al., 2020). All members of the MDT unanimously agreed that an individualised sleep hygiene education session, followed by further evaluation and intervention (if appropriate) would be the most efficacious, cost-effective, and quickest intervention to deploy.

The sleep hygiene intervention session took place 48 hours after the collaborative MDT meeting in the form of an informal presentation that covered the physiology of sleep initiation and evidence-based techniques to support sleep onset, as well as a discussion on their bedtime habits and evidence-based behaviours that supported sleep. The session content was tailored to the player based on the data collected from the initial consultation and advised on a regular bedtime routine, melatonin-rich foods, and showers before bed.

This session was provided by a sports physiologist with 3 years of experience in sleep research. Generalised sleep hygiene advice was also provided based on published recommendations (Halson, 2014; Vitale et al., 2019; McCloughan et al., 2014; Walsh et al., 2021). This guidance had previously been shown to improve sleep in professional athletes (Driller et al., 2019) and specific emphasis was placed on elements, raised during the consultation, that the MDT thought would have a targeted impact. A summary of the bespoke sleep hygiene strategy can be found in Table 1. The final debrief took place 28 days after the delivery of the intervention.

Analysis

Comparisons were made between Pre and Post-scores, as well as between Pre and Post-scores and the reference data.

Results

Pre-intervention observations

The SHI raised several areas of concern including, going to bed with psychological stress, using the bed for other activities rather than sleep or intimacy (e.g., sitting in bed watching television), and thinking or planning when in bed. During the consultation, the player also reported spending a large amount of time in the evening watching television or using electronic devices (Table 2). The player was rated as having poor sleep quality (PSQI: 22) and moderate insomnia (ISI: 15). Components that related to sleep onset latency, wake after sleep onset, feeling too hot, daytime sleepiness, enthusiasm, and overall sleep quality were rated most negatively. The

Table 1. Summary of the individualised and general advice provided to the player as part of their sleep hygiene strategy.

Targeted advice			
Player response		Strategy	Justification
1	The player reported getting into bed hours (e.g., to watch television) before attempting to sleep and was noted as having a moderate evening chronotype.	Advised player not to get into bed until he intended to sleep and to attempt sleep when he is tired.	This can reinforce a regular sleep routine and sleep onset attempts will occur during periods when melatonin release increases (Walsh et al., 2021).
2	The player typically showered in the morning or after training (approx. 1500 to 1700).	Advised to have a warm shower or bath within one hour of getting into bed. No specific temperature was advised as this could not feasibly be determined within the player's home. The player was advised to self-select a temperature that they perceived to be appropriate.	A warm shower before bed can improve sleep onset latency and may support the thermoregulatory process associated with sleep onset (Whitworth-Turner et al., 2017).
3	The players' secondary sleep complaints included night-time sweats.	Advised maintaining a cool sleeping environment. Methods discussed included opening widows and modulating central heating	Sleep onset has a thermoregulatory component. A cool sleeping environment may support this (Fullagar et al., 2016).
4	The player mentioned melatonin-rich foods (walnuts, almond milk) were in his most recent nutrition plan when several examples were presented.	Suggested consuming melatonin-rich foods, in line with their nutrition plan, closer to bedtime.	Melatonin initiates processes that are associated with sleep onset and depth (Yeh et al., 2022).
Additional general advice (Halson, 2014; Vitale et al., 2019; McCloughan et al., 2014; Walsh et al., 2021)			
1	Don't go to bed until you are sleepy. If you aren't sleepy, get out of bed and do something else until you become sleepy.		
2	Regular bedtime routines/rituals help you relax and prepare your body for bed (reading, warm bath, etc.).		
3	Try to get up at the same time every morning (including weekends and holidays).		
4	Try to get a full night's sleep every night and avoid naps during the day if possible (if you must nap, limit to 1 h and avoid napping after 15:00 p.m.).		
5	Use the bed for sleep and intimacy only; not for any other activities such as TV, computer, or phone use, etc		
6	Avoid caffeine if possible (if caffeine is consumed, avoid after lunch)		
7	Avoid alcohol if possible (if must use alcohol, avoid right before bed).		
8	Avoid blue light emitted from screens at least 2 h before bed (smartphones, laptop, monitors).		
9	Meditation/mindfulness may be helpful		

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Table 2. Sleep hygiene index responses. A self-reported assessment of sleep hygiene behaviours (Mastin et al., 2006).

	Component	Response
1	I take daytime naps lasting two or more hours	Frequently
2	I go to bed at different times from day to day.	Sometimes
3	I get out of bed at different time from day to day.	Sometimes
4	I exercise to the point of sweating within 1 hour of going to bed.	Rarely
5	I stay in bed longer than I should two or three times a week.	Rarely
6	I use alcohol, tobacco, or caffeine within 4 hours of going to bed or after going to bed.	Never
7	I do something that may wake me up before bedtime (for example: play video games, use the internet, or clean).	Frequently
8	I go to bed feeling stressed, angry, upset, or nervous.	Sometimes
9	I use my bed for things other than sleeping or sex (for example: watch television, read, eat, or study)	Always
10	I sleep on an uncomfortable bed (for example: poor mattress or pillow, too much or not enough blankets).	Never
11	I sleep in an uncomfortable bedroom (for example: too bright, too stuffy, too hot, too cold, or too noisy)	Sometimes
12	I do important work before bedtime (for example: pay bills, schedule, or study).	Rarely
13	I think, plan, or worry when I am in bed.	Frequently
	Global Score	**24**

MEQ suggested that the player's chronotype was a moderate evening type.

The player provided 7 days of objective sleep data after the initial consultation. The days were not consecutive, and all recorded nights proceeded training days. The objective supported what was reported by the player. Specifically, the activity-monitor reported mean awakenings per night, awakening per hour, WASO, and sleep efficiency that was greater than the reference data (Figure 2).

Post-intervention observations

The player's Post-PSQI score improved compared to Pre- (Pre: 22, Post: 9), however, both remained above the threshold for *"poor"* sleep quality (>5). Components relating to sleep latency and WASO (Pre: once or twice a week, Post: less than once a week), and feeling too hot (Pre: three or more times a week, Post: less than once a week) were improved (Table 3). ISI classification was reduced from moderate insomnia to sub-threshold insomnia (Pre: 15, Post: 8). Components relating to sleep latency and WASO were both reduced from *"Moderate"* to *"Mild"*, and the player's perceived satisfaction of his current sleep pattern improved from *"Dissatisfied"* to *"Satisfied"* (Table 4). Finally, the player's ESS classification also improved from *"Moderate"* to *"Mild"* daytime sleepiness (Pre: 15, Post: 11; Table 5). During the final debrief, the player self-reported a reduction in night-time awakenings and improved, but not absent, perceived night-time sweating.

The player provided 7 and 8 nights of objective data for Pre and Post, respectively. From Pre to Post, the player's WASO (Pre: 74.3 mins ±31.9 mins, Post: 50.0 mins ±22.8 mins, −33%), sleep latency (Pre: 12.6 mins ±6.5 mins, Post: 8.9 mins ±1.3 mins, −29%), sleep efficiency (Pre: 79.2% ± 6.0%, Post: 85.3% ± 5.4%, 8%), awakenings per hour (Pre: 1.2 ± 0.5, Post: 0.6 ± 0.2, −50%), and awakening per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9, −43%) all improved. Compared to the reference data, WASO (Pre: 74.3 mins ±31.8 mins, reference: 44.3 mins ±36.5 mins, −40%), awakenings per hour (Pre: 1.2 ± 0.5, reference: 0.7 ± 0.4, −42%), awakenings per night (Pre: 7.9 ± 3.0, reference: 4.6 ± 2.6, −42%) were greater at Pre, whereas Post scores only presented with seemingly trivial differences compared to the reference data (Figure 2 and Table 6).

Discussion

The primary finding of this study is that the player's primary and secondary sleep complaints were improved after a bespoke sleep hygiene strategy. Notably, the player's awakenings per night (Pre: 7.9 ± 3, Post: 4.5 ± 1.9, −43%) and awakenings per hour (Pre: 1.2 ± 0.5, Post: 0.6 ± 0.2, −50%) improved from Pre to Post. Furthermore, Post data for awakenings per night and awakenings per hour was more similar to the reference data compared to Pre, suggesting that the players sleep was more in line with reference norms. Whilst this case study cannot definitively say that the sleep hygiene strategy mediated the improvements to objectively and subjectively rated sleep metrics (i.e., causality), we observed a positive response to the intervention across several sleep and sleep-related variables, indicating better sleep. It is important to note, nonetheless, that the player's objective data presented with relatively large CI (Figure 2). Whilst the large CI may be due to a low number of data points or the inherently variable nature of sleep (Whitworth-Turner et al., 2018), this may also indicate that the stated response could be in the opposite direction. However, considering the subjective and the objective data overall suggest a beneficial response, it is likely that a positive effect was observed.

Research has highlighted that sleep hygiene in athletes may be sub-optimal (Cameron et al., 2021). In one study, a sample of professional team sport players ($n = 184$) scored lower on the SHI compared to a cohort of age-matched controls ($n = 101$). Notably, athletes scored significantly lower in components relating to bedtime/wake time regularity, sleep environment, and nap behaviour suggesting that athletes, in general, may benefit from sleep hygiene interventions.

There is little data examining the effectiveness of personalised or individualised sleep hygiene interventions in athletic populations (M. W. Driller et al., 2019). However, the limited amount of data that has been collected aligns with this case study. In international standard cricket players ($n = 9$) (M. W. Driller et al., 2019), a one-on-one education session resulted in significantly improved activity-monitor derived sleep latency, which also like caused an improvement in sleep efficiency (+5%). In this case study, sleep efficiency improved by a similar magnitude. However, in this instance, improved WASO scores were likely the primary driver. Results

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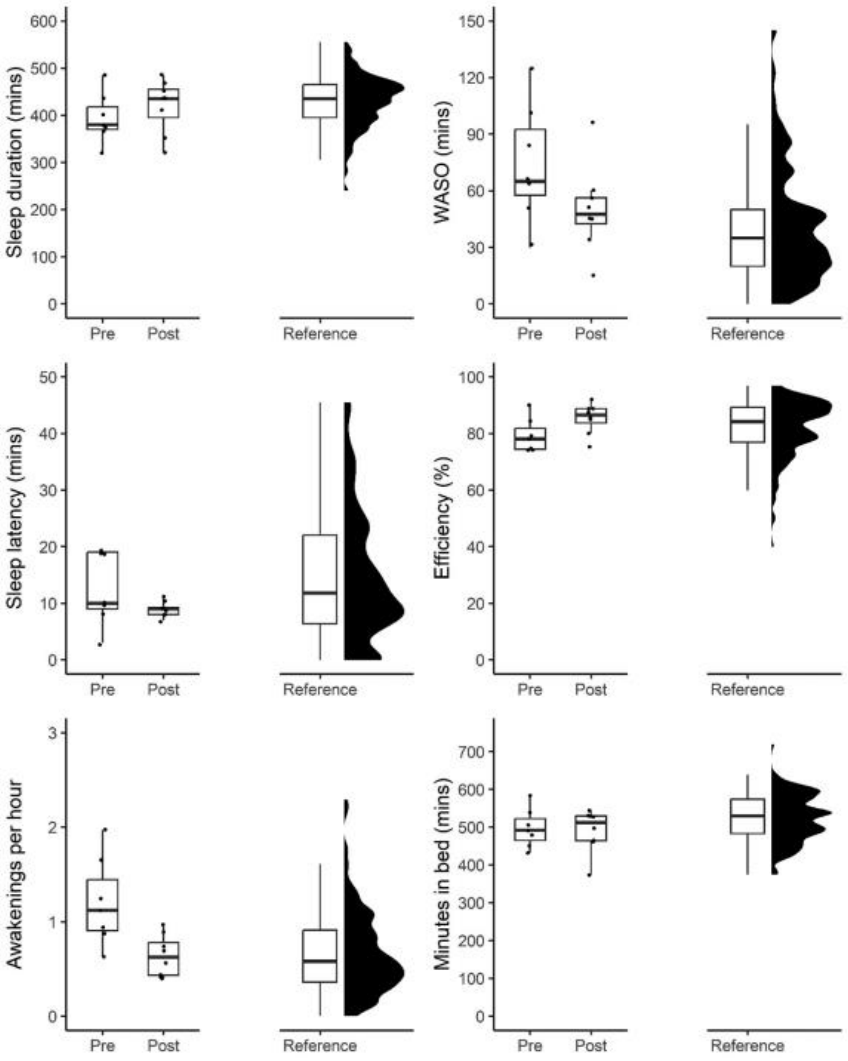

Figure 2. Box and whisker plots for Pre, Post, and the reference data. The reference data is shown alongside a cloud plot to highlight distribution. Outliers have been removed from the box and whisker plots.

from more generalised, group-based sleep hygiene interventions have also reported improved sleep, with positive results reported in both professional rugby league players (Caia et al., 2018) and non-professional football players (Vitale et al., 2019). Furthermore, in highly trained footballers (Fullagar et al., 2016), a sleep hygiene strategy that directly restricted ambient light, limited electronic device use, and controlled room temperature (~17°C) resulted in significantly improved post-fixture sleep duration compared to a control.

Where previous research has observed benefits to sleep duration (Caia et al., 2018; Fullagar et al., 2016), sleep efficiency (Driller et al., 2019), and sleep onset latency (J. A. Vitale et al., 2019; Driller et al., 2019), this case study also observed a benefit to WASO, awakenings per hour, and awakenings per night, which appears unique in the literature base thus far. However, the studies involving professional or elite athletes (Caia et al., 2018; Fullagar et al., 2016) have excluded participants that have reported historic sleep issues,

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Table 3. Pre and Post-PSQI responses. The PSQI is a self-rated questionnaire which assesses sleep quality and disturbances over a 1-month time interval (Buysse et al., 1989).

	Component	Pre-	Post
1	When have you usually gone to bed?	22:00	23:00
2	How long (in minutes) has it taken you to fall asleep each night?	25 minutes	18 minutes
3	When have you usually gotten up in the morning?	07:00	07:00
4	How many hours of actual sleep do you get at night?	7hrs	8hrs
5	During the past month, how often have you had trouble sleeping because you . . .		
5a	Cannot get to sleep within 30 minutes	Once or twice a week	Less than once a week
5b	Wake up in the middle of the night or early morning	Once or twice a week	Less than once a week
5c	Have to get up to use the bathroom	Once or twice a week	Not during the past month
5d	Cannot breathe comfortably	Less than once a week	Not during the past month
5e	Cough or snore loudly	Not during the past month	Not during the past month
5f	Feel too cold	Less than once a week	Not during the past month
5g	Feel too hot	Three or more times a week	Less than once a week
5h	Have bad dreams	Once or twice a week	Less than once a week
5i	Have pain	Not during the past month	Not during the past month
6	During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?	Not during the past month	Not during the past month
7	During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?	Once or twice a week	Less than once a week
8	During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?	Once or twice a week	Less than once a week
9	During the past month, how would you rate your sleep quality overall?	Once or twice a week	Fairly good
	Global score	**22**	**9**

Note: *PSQI (Pittsburgh Sleep Quality Index).*

Table 4. Pre and Post-ISI responses. The ISI is an instrument to assess the severity of both night-time and daytime components of insomnia (Bastien et al., 2001).

	Component	Pre-	Post
1	Difficulty falling asleep	Moderate	Mild
2	Difficulty staying asleep	Moderate	Mild
3	Problems waking up too early	Severe	Moderate
4	Problems waking up too early	Dissatisfied	Satisfied
5	How noticeable to others do you think your sleep problem is in terms of impairing the quality of your life?	Somewhat	A little
6	How worried/distressed are you about your current sleep problem?	A little	A little
7	To what extent do you consider your sleep problem to interfere with your daily functioning (e.g., daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) currently?	Somewhat	A little
	Global score	**15**	**8**

Note: *ISI (Insomnia Severity Index).*

Table 5. Pre and Post-ESS. The ESS is a self-reported questionnaire which provides a measurement of the subject's genera level of daytime sleepiness (Kendzerska et al., 2014).

Situation	Pre-	Post
Sitting and reading	3	2
Watching TV	2	1
Sitting inactive in a public place	1	1
As a passenger in a car for an hour without a break	2	1
Lying down to rest in the afternoon when circumstances permit	3	3
Sitting and talking to someone	1	1
Sitting quietly after lunch without alcohol	1	1
In a car, while stopped for a few minutes in traffic	2	1
Global score	**15**	**11**

Note: *ESS (Epworth Sleepiness Scales).*

whereas this case study investigated a professional player that was specifically referred after reporting excessive night-time awakenings. Therefore, this case study may have observed improvements in WASO, awakenings per hour, and awakenings per night because the player's scores were already suboptimal, compared to other age-matched footballers.

Alongside improvements to objective sleep metrics, this case study also reports improved PSQI, ISI, and ESS scores after the sleep hygiene intervention. Whilst the ESS rates the perception of sleepiness at the time of completion (Kendzerska et al., 2014), the PSQI (Buysse et al., 1989) and ISI (Bastien et al., 2001) give a more general interpretation. Components relating to sleep onset latency, night-time awakenings, and overall sleep quality,

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Table 6. Means ± SD for Pre, Post, and Reference data alongside Pre, Post, and Reference percentage change. Negative/positive values indicate the direction of change.

	Pre	Post	Reference	Pre vs Post	Pre vs Reference	Post vs reference
Sleep duration (mins)	394.3 ± 53.0	419.4 ± 57.4	433.4 ± 68.0	6%	10%	3%
MiB (mins)	497.4 ± 51.6	491.1 ± 56.6	533.0 ± 81.5	−1%	7%	9%
WASO (mins)	74.3 ± 31.8	50 ± 22.8	44.3 ± 36.5	−33%	−40%	−11%
Sleep latency (mins)	12.6 ± 6.5	8.9 ± 1.2	23.6 ± 26.1	−29%	87%	165%
Sleep efficiency (%)	79.2 ± 6	85.3 ± 5.4	81.9 ± 10.3	8%	3%	−4%
Awakenings per hour	1.2 ± 0.5	0.6 ± 0.2	0.7 ± 0.4	−50%	−42%	17%
Awakenings per night	7.9 ± 3	4.5 ± 1.9	4.6 ± 2.6	−43%	−42%	2%

Note: Wake after sleep onset (WASO).

in addition to issues with daytime sleepiness and enthusiasm were perceived to improve. Together with the objective data, this may suggest that the player perceived a benefit to their daytime functioning. Similar results have also been observed in professional cricket players (Driller et al., 2019) and non-professional footballers (Vitale et al., 2019) who received a sleep hygiene intervention.

It is challenging to deduce which element, or combination of elements, of the sleep hygiene intervention mediated changes to the player's objective and subjective sleep metrics. During the final debrief, the player inferred that he perceived the consumption of melatonin-rich foods (specifically walnuts and other nuts), a shower before bed, and a more regular bedtime routine were notably beneficial. Walnuts are considered to be melatonin-rich and randomised placebo-controlled trials suggest that consumption of walnut-derived peptides can significantly improve PSQI scores in adolescent and elderly populations (Yeh et al., 2022). Whilst research is still emerging, it does indicate that the consumption of walnuts close to bedtime may increase melatonin and aid in sleep initiation. There is a more established research base surrounding the use of warm baths or showers close to bedtime to aid sleep, particularly regarding sleep initiation. This has been observed in professional adolescent football players (Whitworth-Turner et al., 2017), where the application of a warm shower 20 minutes before bedtime resulted in significantly improved sleep efficiency and sleep onset latency. Whilst it is beyond the scope of this case study to investigate the effectiveness of individual components on the player's sleep, this case study suggests that a combined approach is efficacious.

This case study used a combination of subjective (PSQI, ISI, ESS) and objective measures (wrist-activity monitors) to gain a holistic view of the player's sleep. However, the efficacy of such an approach should be questioned. The player was referred because they self-reported sleep disruption. This was subsequently discussed in the initial consultation and confirmed through both subjective and objective monitoring. However, the sleep assessments did not reveal anything new that the player had not already verbally stated. Therefore, if data from the initial consultation was viewed in isolation, then the sleep hygiene intervention could have been applied in the first instance, without the need for a period of objective monitoring. However, subjective assessments are potentially limited by subjective biases, although, one advantage of utilising wrist-activity monitors is their ability to reconcile the subjective assessments. Compared to polysomnography, activity monitors have demonstrated validity (Chinoy et al., 2021) and their use in research has helped to elucidate several factors that may influence sleep in professional players (Whitworth-Turner et al., 2019). Therefore, whilst objective measures offered little additional information compared to the subjective assessments, it did offer an opportunity to collaborate the data.

This case study has several limitations. Firstly, this was not a controlled study with a suitable comparator, thus results can neither support nor refute the efficacy of an individualised sleep hygiene intervention in professional football players reporting sleep issues. Nevertheless, it offers a potential guide to the decision-making process and provides a real-world example framework for sport science and medicine professionals when they encounter sleep issues within their practice. Further, whilst the intervention was formulated by an MDT with a wealth of applied experience and on the guidance of the data available, its formulation is still likely influenced by subjective individual biases. Therefore, the most efficacious intervention may not have been applied. Also, this case study did not monitor or re-evaluate sleep after the final debrief and it is not known if sleep metrics continued to improve or relapsed, nor was it able to elucidate sleep architecture. Finally, while the player also identified night sweats as a sleep complaint, this could not be objectively determined so did not form a central part of the discussion.

In conclusion, this case study applied an individualised sleep hygiene intervention to a player who was referred after reporting excessive night-time awakenings and night-time sweats. The player's subjective and objective sleep metrics subsequently improved. Whilst this case study cannot definitively say the intervention caused the changes to the sleep metrics, a player reported excessive night-time awakenings, an intervention was applied, and then the player reported improvement. This case study provides a potential framework for coaches and sports practitioners who may encounter reported sleep issues as part of their practice.

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10.5. Appendix 6: Ethics

10.5.1. Chapter 3

St Mary's University Twickenham London

02 September 2019 SMEC_2018-19_054

Luke Edinborough (SHAS): 'Effect of a 5-day whole-body therapy course on sleep quality in u18 professional athletes'

Dear Luke

University Ethics Sub-Committee

Thank you for re-submitting your ethics application for consideration.

I can confirm that all required amendments have been made and that you therefore have ethical approval to undertake your research.

Yours sincerely

Matthew James
Acting Chair, Ethics Sub-Committee

Cc Jessica Hill

St Mary's University, Waldegrave Road, Strawberry Hill, Twickenham, London TW1 4SX
Switchboard 020 8240 4000, Fax 020 8240 4255, www.stmarys.ac.uk

St Mary's University Twickenham London
Faculty of Sport, Health & Applied Science

5/11/2020

Dear Mr Edinborough,

Re. **Longitudinal Monitoring of sleep quality in u18 footballers**

Thank you for submitting your ethics application for consideration.

I can confirm that your application has been considered by the SHAS Ethics Committee and that ethical approval is granted. Please find attached your signed approval form.

Yours sincerely,

Dr Phil Price

Faculty of SHAS Ethics Committee

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5607 10.5.3. Chapter 6

St Mary's University Twickenham London

Approval Sheet

(This sheet must be signed at all relevant boxes)

Name of proposer(s)	Luke Edinborough
Name of supervisor(s)	Dr Charles Pedlar, Dr Jessica Hill
Programme of study	PhD
Title of project	Method agreement between oculomotor assessment and Psychomotor Vigilance Task in relation to daily sleep variation.

Supervisors, please complete section 1. If approved at level 1, please forward a copy of this Approval Sheet to the Faculty Ethics Representative for their records.

SECTION 1: To be completed by supervisor (for student research projects).			
☐ Approved at Level 1. ☒ Refer to Faculty Ethics Representative for consideration at Level 2 or Level 3.			
Name of Supervisor:	Charles Pedlar		
Signature of Supervisor:	C. Pedlar	Date:	28.04.21

SECTION 2: To be completed by Faculty Ethics Representative.			
☒ Approved at Level 2. ☐ Level 3 consideration is required by Ethics Sub-Committee.			
Name of Faculty Ethics Representative:	Elaine Mullally		
Signature of Faculty Ethics Representative:	E Mullally	Date:	23.02.22

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5609 10.5.4. Chapter 7

St Mary's University Twickenham London
Sport, Allied Health & Performance Sciences

25 February 2022

Dear Luke Edinborough,

Re. **Sleep monitoring case study series**

Thank you for submitting your updated revised ethics application for consideration.

I can confirm that your application has been considered by the SAHPS Ethics Committee and that ethical approval is granted. Please find attached your signed approval form.

Yours sincerely,

Jamie North

Faculty of SAHPS Ethics Committee

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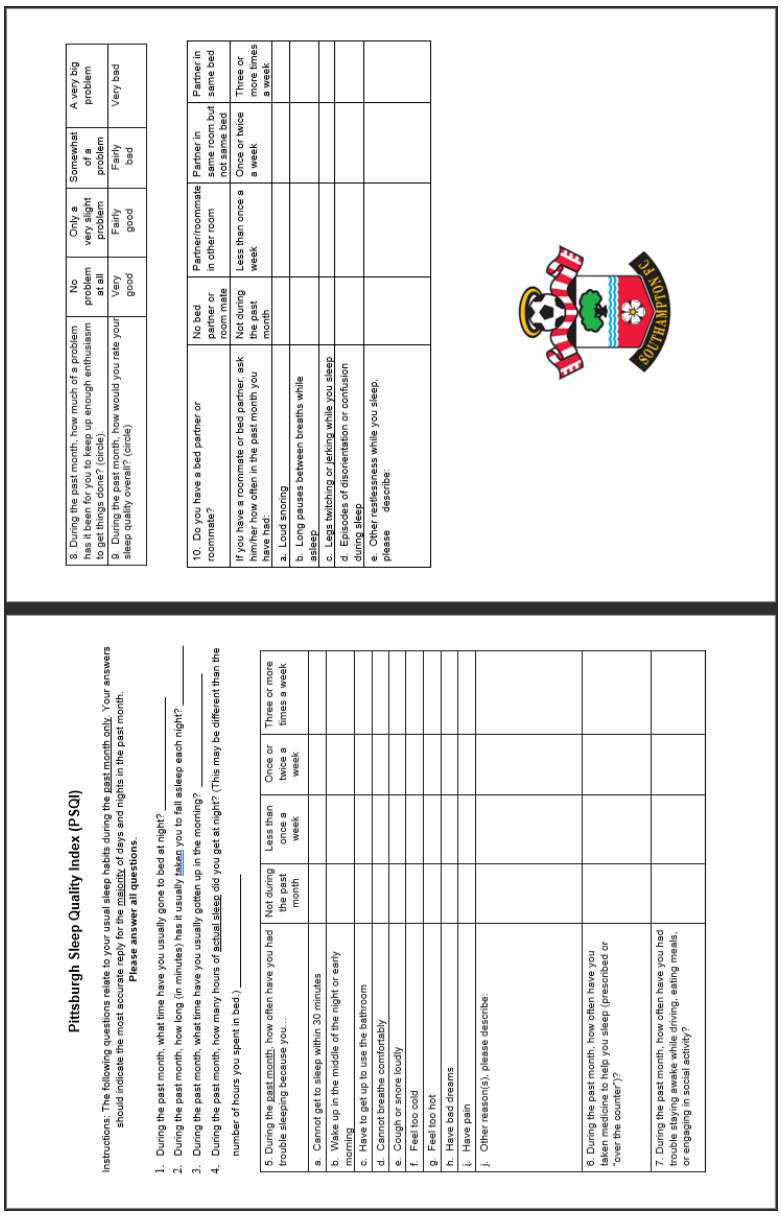
10.6. Appendix 7: Questionnaires and forms

10.6.1. Pittsburgh sleep quality index



Pittsburgh Sleep Quality Index (PSQI)

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

1. During the past month, what time have you usually gone to bed at night? _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? _____
3. During the past month, what time have you usually gotten up in the morning? _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				
6. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				

8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done? (circle).	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
9. During the past month, how would you rate your sleep quality overall? (circle)	Very good	Fairly good	Fairly bad	Very bad

10. Do you have a bed partner or roommate?	No bed partner or room mate	Partner/roommate in other room	Partner in same room but not same bed	Partner in same bed
If you have a roommate or bed partner, ask him/her how often in the past month you have had:	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Loud snoring				
b. Long pauses between breaths while asleep				
c. Legs twitching or jerking while you sleep				
d. Episodes of disorientation or confusion during sleep				
e. Other restlessness while you sleep, please describe:				

SOUTHAMPTON FC

10.6.2. Insomnia severity index

Insomnia Severity Index

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. When you have your total score, look at the 'Guidelines for Scoring/Interpretation' below to see where your sleep difficulty fits.

For each question, please CIRCLE the number that best describes your answer.

Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).

Insomnia Problem	None	Mild	Moderate	Severe	Very Severe
1. Difficulty falling asleep	0	1	2	3	4
2. Difficulty staying asleep	0	1	2	3	4
3. Problems waking up too early	0	1	2	3	4

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

Very Satisfied	Satisfied	Moderately Satisfied	Dissatisfied	Very Dissatisfied
0	1	2	3	4

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

Not at all Noticeable	A Little	Somewhat	Much	Very Much Noticeable
0	1	2	3	4

6. How WORRIED/DISTRESSED are you about your current sleep problem?

Not at all Worried	A Little	Somewhat	Much	Very Much Worried
0	1	2	3	4

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

Not at all Interfering	A Little	Somewhat	Much	Very Much Interfering
0	1	2	3	4

Guidelines for Scoring/Interpretation:

Add the scores for all seven items (questions 1 + 2 + 3 + 4 + 5 +6 + 7) = _____ your total score

Total score categories:
0–7 = No clinically significant insomnia
8–14 = Subthreshold insomnia
15–21 = Clinical insomnia (moderate severity)
22–28 = Clinical insomnia (severe)

Used via courtesy of www.myhealth.va.gov with permission from Charles M. Morin, Ph.D., Université Laval

5621 10.6.3. Epworth sleepiness scale

Epworth Sleepiness scale

The Epworth Sleepiness Scale is widely used in the field of sleep medicine as a subjective measure of a patient's sleepiness. The test is a list of eight situations in which you rate your tendency to become sleepy on a scale of 0, no chance of dozing, to 3, high chance of dozing. When you finish the test, add up the values of your responses. Your total score is based on a scale of 0 to 24. The scale estimates whether you are experiencing excessive sleepiness that possibly requires medical attention.

How Sleepy Are You?

How likely are you to doze off or fall asleep in the following situations? You should rate your chances of dozing off, not just feeling tired. Even if you have not done some of these things recently try to determine how they would have affected you. For each situation, decide whether or not you would have:

- No chance of dozing = 0
- Slight chance of dozing = 1
- Moderate chance of dozing = 2
- High chance of dozing = 3

Write down the number corresponding to your choice in the right hand column.

Situation	Chance of snoozing
Sitting and reading	
Watching TV	
Sitting inactive in a public place (eg., theatre or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car, while stopped for a few minutes in traffic	
Name	
Day (eg., mon, tue, etc.)	

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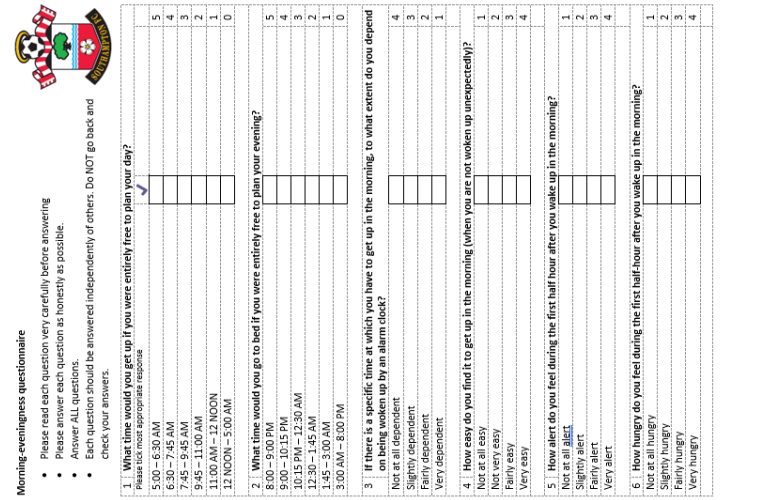
5624 10.6.4. Morning eveningness questionnaire



Morning-eveningness questionnaire

- Please read each question very carefully before answering
- Please answer each question as honestly as possible.
- Answer ALL questions.
- Each question should be answered independently of others. Do NOT go back and check your answers.

1	**What time would you get up if you were entirely free to plan your day?** Please tick most appropriate response	✓
	5.00 – 6:30 AM	5
	6.30 – 7:45 AM	4
	7:45 – 9:45 AM	3
	9:45 – 11:00 AM	2
	11:00 AM – 12 NOON	1
	12 NOON – 5:00 AM	0
2	**What time would you go to bed if you were entirely free to plan your evening?**	
	8:00 – 9:00 PM	5
	9:00 – 10:15 PM	4
	10:15 PM – 12:30 AM	3
	12:30 – 1:45 AM	2
	1:45 – 3:00 AM	1
	3:00 AM – 8:00 PM	0
3	**If there is a specific time at which you have to get up in the morning, to what extent do you depend on being woken up by an alarm clock?**	
	Not at all dependent	4
	Slightly dependent	3
	Fairly dependent	2
	Very dependent	1
4	**How easy do you find it to get up in the morning (when you are not woken up unexpectedly)?**	
	Not at all easy	1
	Not very easy	2
	Fairly easy	3
	Very easy	4
5	**How alert do you feel during the first half hour after you wake up in the morning?**	
	Not at all alert	1
	Slightly alert	2
	Fairly alert	3
	Very alert	4
6	**How hungry do you feel during the first half-hour after you wake up in the morning?**	
	Not at all hungry	1
	Slightly hungry	2
	Fairly hungry	3
	Very hungry	4
7	**During the first half-hour after you wake up in the morning, how tired do you feel?**	
	Very tired	1
	Fairly tired	2
	Fairly refreshed	3
	Very refreshed	4
8	**If you have no commitments the next day, what time would you go to bed compared to your usual bedtime?**	
	Seldom or never later	4
	Less than one hour later	3
	1-2 hours later	2
	More than two hours later	1
9	**You have decided to engage in some physical exercise. A friend suggests that you do this for one hour twice a week and the best time for him is between 7:00 – 8:00 am. Bearing in mind nothing but your own internal "clock", how do you think you would perform?**	
	Would be in good form	4
	Would be in reasonable form	3
	Would find it difficult	2
	Would find it very difficult	1
10	**At what time of day do you feel you become tired as a result of need for sleep?**	
	8:00 – 9:00 PM	5
	9:00 – 10:15 PM	4
	10:15 PM – 12:45 AM	3
	12:45 – 2:00 AM	2
	2:00 – 3:00 AM	1
11	**You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last for two hours. You are entirely free to plan your day. Considering only your own internal "clock", which ONE of the four testing times would you choose?**	
	8:00 AM – 10:00 AM	4
	11:00 AM – 1:00 PM	3
	3:00 PM – 5:00 PM	2
	7:00 PM – 9:00 PM	1
12	**If you got into bed at 11:00 PM, how tired would you be?**	
	Not at all tired	1
	A little tired	2
	Fairly tired	3
	Very tired	4
13	**For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which ONE of the following are you most likely to do?**	
	Will wake up at usual time, but will NOT fall back asleep	4
	Will wake up at usual time and will doze thereafter	3
	Will wake up at usual time but will fall asleep again	2
	Will NOT wake up until later than usual	1

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14	**One night you have to remain awake between 4:00 – 6:00 AM in order to carry out a night watch. You have no commitments the next day. Which ONE of the alternatives will suite you best?**		
	Would NOT go to bed until watch was over		1
	Would take a nap before and sleep after		2
	Would take a good sleep before and nap after		3
	Would sleep only before watch		4
15	**You have to do two hours of hard physical work. You are entirely free to plan your day and considering only your own internal "clock" which ONE of the following time would you choose?**		
	8:00 AM – 10:00 AM		4
	11:00 AM – 1:00 PM		3
	3:00 PM – 5:00 PM		2
	7:00 PM – 9:00 PM		1
16	**You have decided to engage in hard physical exercise. A friend suggests that you do this for one hour twice a week and the best time for him is between 10:00 – 11:00 PM. Bearing in mind nothing else but your own internal "clock" how well do you think you would perform?**		
	Would be in good form		1
	Would be in reasonable form		2
	Would find it difficult		3
	Would find it very difficult		4
17	**Suppose that you can choose your own work hours. Assume that you worked a FIVE hour day (including breaks) and that your job was interesting and paid by results). Which FIVE CONSECUTIVE HOURS would you select?**		
	5 hours starting between 4:00 AM and 8:00 AM		5
	5 hours starting between 8:00 AM and 9:00 AM		4
	5 hours starting between 9:00 AM and 2:00 PM		3
	5 hours starting between 2:00 PM and 5:00 PM		2
18	**At what time of the day do you think that you reach your "feeling best" peak?**		
	5:00 – 8:00 AM		5
	8:00 – 10:00 AM		4
	10:00 AM – 5:00 PM		3
	5:00 – 10:00 PM		2
	10:00 PM – 5:00 AM		1
19	**One hears about "morning" and "evening" types of people. Which ONE of these types do you consider yourself to be?**		
	Definitely a "morning" type		6
	Rather more a "morning" than an "evening" type		4
	Rather more an "evening" than a "morning" type		2
	Definitely an "evening" type		0

Name:

10.7. Appendix 8: Declaration of Originality

Students are reminded that the work that they submit for assessment must be their own. Please read the following statements and sign and date at the bottom of this form to show that you have complied:

1. This thesis and the work to which it refers are the results of your own efforts. Any ideas, data or text resulting from the work of others (whether published or unpublished) are fully identified as such within the work and attributed to the originator in the text, bibliography or footnotes.

2. This thesis has not been submitted in whole or in part for any other academic degree or professional qualification at this or any other institution.

3. Any chapters that describe the outcomes of joint research should be clearly identified as such with a statement inserted as a footnote on the first page and contributors named. Significant data, images or text resulting from the input of other researchers should be identified as such and attributed to the persons concerned by means of a footnote within the chapter.

4. It is usual to acknowledge the help and guidance of others who have assisted you during your research and preparation of your thesis. Such acknowledgements do not replace or obviate the need for individual attribution as discussed in points 1 and 3.

5. The University reserves the right to submit electronic versions of your draft documents for assessment of plagiarism using electronic detection software such as 'turnitin'. In addition, whether or not drafts have been so assessed, the University reserves the right to require an electronic version of the final document (as submitted) for assessment.

SIGNED:……L Edinborough

PRINT NAME:……LUKE EDINBROUGH

DATE:…04/07/2023