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Maxime Chauvineau. Sleep in elite athletes: from the impact of training load to bedding optimization for the Paris 2024 Olympic Games. Life Sciences [q-bio]. Université Paris Cité, 2024. English. NNT : . tel-04669550

HAL Id: tel-04669550

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Submitted on 8 Aug 2024

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Université Paris Cité

École doctorale 566 : Sciences du Sport, de la Motricité et du Mouvement Humain

Laboratoire Sport, Expertise et Performance – EA 7370

Sleep in elite athletes: from the impact of training load to bedding optimization for the Paris 2024 Olympic Games

Le sommeil du sportif de haut niveau : de l'impact de la charge d'entraînement à
l'optimisation de la literie en vue des Jeux Olympiques de Paris 2024

Par Maxime CHAUVINEAU

Thèse de doctorat en Sciences du Sport et du Mouvement Humain

Dirigée par Gaël GUILHEM

Co-encadrée par Mathieu NEDELEC et François DUFORÉZ

Présentée et soutenue publiquement le 19 juin 2024

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Preface

The research in this PhD thesis was conducted in the Sport, Expertise and Performance Laboratory at the French Institute of Sport (INSEP, Paris, France). Funding was provided by COFEL Industries and its Bultex company, specializing in bedding design (CIFRE contract no. 2020/0967).

This adventure began almost 6 years ago, when I first passed through the gates of the French Institute of Sport (INSEP) to pursue my Master's degree. These years have been filled with collaborations, teamwork, and extensive sharing, which have allowed the completion of this PhD thesis. In these few words, I would like to thank everyone who contributed in any way to the present work and supported me throughout this journey.

My first words are addressed to Dr. **Charli Sargent** and Prof. **Stéphane Perrey** for accepting to report this thesis, as well as Prof. **Shona Halson**, Prof. **Nathalie Pattyn** and Prof. **Greg Roach** for accepting to examine it. It is a great honour to benefit from your expert opinion and I am looking forward to discussing through this work together.

I would also like to thank Prof. **Shona Halson**, Dr. **Antonio Jesús Morales-Artacho**, and Dr. **Grégory Dupont** for our valuable exchanges and your support during my supervising PhD committee.

Je voudrais sincèrement remercier mes encadrants, **Mathieu Nédélec**, **Gaël Guilhem** et **François Duforez**, qui m'ont accompagné pendant ces trois ans.

Mathieu, tu m'accompagnes depuis mon stage de Master 1 où tu m'as fait découvrir ce monde mystérieux et magique qu'est le sommeil. Quel chemin parcouru ! Tu m'as d'abord donné l'opportunité de mener un projet de recherche qui consistait à plonger des sportifs dans l'eau froide avec la tête sous l'eau. En y repensant c'était un peu fou quand même ! Puis tu m'as pris sous ton aile et tu m'as partagé tes connaissances et ton savoir-faire en t'accompagnant sur les différentes missions d'ASP avec les fédérations. Je tiens à te remercier sincèrement pour la confiance que tu m'as accordée tout ce temps. J'ai énormément appris à tes côtés et je t'en serai toujours reconnaissant.

Gaël, je te remercie d'avoir accepté de diriger ce travail, même s'il sortait un peu de tes sentiers battus. Tu as souvent dit que tu n'étais pas l'expert du sujet, mais tu as été d'un soutien incommensurable pendant ces trois années. Un grand merci pour ta rigueur, ton dévouement professionnel au quotidien, tes idées à foison et tes nombreux questionnements qui m'ont toujours fait réfléchir sous un angle différent. Grâce à toi je suis fier du travail accompli.

François, je me souviens m'avoir accueilli à l'Hôtel-Dieu pour me faire découvrir la médecine du sommeil. Je n'avais pas été déçu de l'après-midi passé à tes côtés avec des patients dépressifs ! « La vraie vie », comme tu me l'avais dit. Ton approche transversale et surtout humaine pour appréhender le sommeil du sportif et clinique est une source d'inspiration. Je suis loin d'avoir fini d'apprendre à tes côtés ! Merci pour tous les conseils et les encouragements.

Je tiens à remercier le groupe COFEL Industries et l'équipe Bultex pour son soutien financier et humain, sans lequel cette thèse n'aurait pas pu être réalisée. Cette thèse CIFRE a été une expérience professionnelle et personnelle extrêmement enrichissante.

Zohra, je ne te remercierai jamais assez pour ton soutien sans faille ces trois dernières années et jusqu'à ces quelques dernières heures d'écriture. Je t'associe pleinement à l'encadrement de cette thèse. J'ai pris conscience du défi qui représente de concilier le monde de la recherche, du sport de haut niveau et un industriel, et grâce à ton engagement et ta passion, nous avons pu le relever. Merci infiniment pour tout.

Je souhaite également remercier toute l'équipe de COFEL Industries et du SommeilLab Bultex et particulièrement **Luis, Nathalie, Francis**, et **Jérémy** qui contribuent au « bien dormir pour mieux vivre », une phrase qui est au cœur de mon travail et de ma motivation au quotidien. Je garderai longtemps en souvenir les moments de partage notamment ceux passés à Amboise (et mon entrée fracassante avec quelques heures de retard...). La course Paris-Versailles restera un souvenir marquant, avec ce repas riche en lipides et en boissons pas tout à fait recommandées avant une course, mais cela ne nous a pas empêché de battre tous les records !

Je remercie tous les **sportifs** ayant participé aux expérimentations pour leur temps et leur implication. **Sébastien Le Garrec**, merci à toi et à ton équipe du pôle médical de l'INSEP pour les (très) nombreuses visites d'inclusion. Je tiens à remercier **Bertrand** (alias Beber), **Gaëtan** (alias la Get) et **Julien** avec qui la collaboration avec la Fédération Française de Rugby a été un

souvenir marquant. **Alexandre**, un grand merci également pour ton aide à la lecture des enregistrements de polysomnographie.

Je remercie chaleureusement l'**INSEP** et particulièrement les **pôles Hospitalité** et **Patrimoine** pour leur aide logistique et leur disponibilité quand il a fallu stocker la tonne de surmatelas. Merci à **Isabelle Amaudry**, **Bruno Loubière** et le **pôle Communication et Audiovisuel**. Merci à l'**IRMES** pour toutes ces heures passées à transpirer le midi. J'adresse également mes remerciements au duo de choc incarné par **Jean-François Robin** et **Adrien Marck**.

Enzo, comment te remercier... Tu as su m'aider à regonfler mes pneus, mes jambes et ma tête quand ils étaient crevés plusieurs matins, entre la boue de la forêt de Ferrière, Nantes et le Mont Saint-Michel. Tu as été un grand frère pour moi tout au long de cette thèse, et je t'en suis éternellement reconnaissant. Merci pour ton amitié. Je te suis également reconnaissant pour l'opportunité et ton aide pour cette dernière étude de thèse en collaboration avec le pôle France badminton de l'**INSEP**. Je suis très heureux d'avoir réalisé cette étude avec toi. Merci également à **Thierry** et les **entraîneurs** pour leur confiance.

J'associe les membres du **laboratoire SEP** et du **Pôle Performance** à ces remerciements qui m'ont accompagné durant ces années de thèse. Plus particulièrement **Bertrand**, **Alexandre**, **Frank**, **Fabrice**, **Claire**, **Hélène**, **Eve**, **Anne-Claire**, **Antonio**, **Giuseppe**, **Antoine**, **Jean**, **Isa**, **Alexis**, **Sinéad**, **Bruno**, **Caro** et **Anaïs**. Enfin, **Julie**, merci pour ta gentillesse et ton aide précieuse au quotidien.

Florane, ces deux années à travailler avec toi sur le sommeil ont été un réel plaisir. Je ris encore en repensant à ta tête rouge comme une tomate en train de transporter les surmatelas ! Je te souhaite tout le meilleur sur la côte Atlantique.

Canelle, un grand merci pour ton soutien statistique. Merci pour tous les footings qui ont parfois été un peu boiteux du côté de l'allée Royale !

Anis, je te remercie de m'avoir initié au monde du sommeil pendant mon Master. Merci pour ton aide et tes nombreux conseils.

Il m'est impossible de ne pas mentionner mes collègues du RDJ, avec qui nous partageons au quotidien les doutes et les frustrations qui animent la vie d'un doctorant. Merci pour nos

discussions scientifiques, les innombrables séances de sport, ainsi que pour tous les moments de joie passés dans et en dehors des murs de l'INSEP. **Vincent, Quentin, Simon, Rafaël, Giuseppe** et tic et tact (**Johan** et **Remi**), merci pour l'amitié qui nous unit. Merci également aux anciens doctorants, et particulièrement à **Ben, Adèle, Valentine, Pauline, Joffrey, Mildred** et **Allison** qui m'ont partagé leur expérience et de nombreux conseils. **Jérôme**, ça a été un grand plaisir de travailler avec toi et l'équipe de France féminine de hockey-sur-glace pendant que tu terminais ta thèse et que je commençais à peine la mienne. Merci aux post-docs **Mathilde, Charli** (« *le sommeil ça requinque, j'te le dis* »), **Benjamin, Robin, Janne** pour votre bonne humeur et votre soutien. Vous êtes tous bien évidemment plus que des collègues.

Merci à mes camarades du Master SEPHN, notamment **François** et **Nico** avec qui j'ai passé mes premières longues soirées studieuses à l'INSEP. **Coline**, je tenais aussi à te remercier pour ton soutien et ton amitié.

Je remercie mes amis de toujours, en particulier **Romain, Gab, Dorian, Quentin, Emmie** et **Théo**. Cette aventure a commencé avec vous sur les bancs de l'école, autour de notre passion pour le sport qui nous réunissait. Je souhaitais en ces quelques mots vous exprimer toute l'amitié que j'ai pour vous.

Amandine, tu es arrivée dans mon quotidien vers la fin de cette aventure, mais tu as assisté à la partie la plus intense. Ton soutien dans cette dernière ligne droite a été extrêmement précieux. Merci pour ton écoute et de m'avoir maintenu à flot tant de fois. Merci pour tout, merci d'être toi.

Enfin, je réserve ces dernières lignes à ma **famille** : mes grands-parents, mes tantes, mon oncle, mes cousins et mon petit frère. **Maman, Papa**, vous avez toujours cru en moi, je ne vous remercierai jamais assez de m'avoir tant donné. Je ne suis pas très bon pour exprimer ce que je ressens, mais sachez tout l'amour que je vous porte.

Publications and conference communications

Published peer-reviewed articles related to the thesis

Chauvineau, M., Pasquier, F., Poirier, C., Le Garrec, S., Duforez, F., Guilhem, G., and Nedelec, M. (2023). Higher training loads affect sleep in endurance runners: Can a high-heat-capacity mattress topper mitigate negative effects? *Journal of Sports Sciences*, 1–12. <https://doi.org/10.1080/02640414.2023.2285574>

Chauvineau, M., Pasquier, F., Duforez, F., Guilhem, G., and Nedelec, M. (2023). Increased training load promotes sleep propensity and slow-wave sleep in endurance runners: can a high-heat-capacity mattress topper modulate this effect? *Journal of Sleep Research*, e14132. <https://doi.org/10.1111/jsr.14132>

Chauvineau, M., Mathieu, B., Boissard G., Piscione J., Duforez, F., Guilhem, G., and Nedelec, M. Morning appointment time rather than training load affects sleep during a moderate-intensified training camp in young elite rugby union players. *Under review*

Chauvineau, M., Hollville, E., Duforez, F., Guilhem, G., and Nedelec, M. Effect of a high-heat-capacity mattress topper on sleep in elite athletes during a summer training period: does one size fit all? *Under review*

Conference communications related to the thesis

Chauvineau M., Pasquier F., Le Garrec S., Duforez F., Guilhem G., and Nedelec M. (2022). Effet de la manipulation de la charge d'entraînement et d'un surmatelas à haute conductivité thermique sur le sommeil de coureurs à pied. Le Congrès du Sommeil, Lille, France.

Chauvineau, M., Pasquier, F., Duforez, F., Guilhem, G., and Nédélec, M. (2023). La charge d'entraînement favorise l'architecture du sommeil chez des coureurs à pied : intérêt d'un surmatelas à haute conductivité thermique. Le Congrès du Sommeil, Lille, France.

Chauvineau, M., Mathieu, B., Boissard, G., Piscione, J., Duforez, F., Guilhem, G., and Nédélec, M. (2023). Impact d'un stage d'entraînement sur les rythmes veille-sommeil et l'architecture du sommeil de jeunes joueurs de rugby de haut niveau. Le Congrès du Sommeil, Lille, France

Chauvineau, M. (2023). Le sommeil du sportif de haut niveau : de l'impact de la charge d'entraînement à l'optimisation de la literie en vue des Jeux Olympiques de Paris 2024. Ateliers CIFRE « Ma recherche j'en parle », Sport et Innovation, Centre National du Football, Clairefontaine-en-Yvelines, France.

Published peer-reviewed articles outside of the scope of the thesis

Chauvineau, M., Pasquier, F., Guyot, V., Aloulou, A., and Nedelec, M. (2021). Effect of the Depth of Cold Water Immersion on Sleep Architecture and Recovery Among Well-Trained Male Endurance Runners. *Frontiers in Sports and Active Living*, 3, 659990. <https://doi.org/10.3389/fspor.2021.659990>

Nédélec, M., **Chauvineau, M.**, and Guilhem, G. (2022). On the Road to Camarón: The Sleep of an Ultra-Endurance Athlete Cycling 10,000 km in 24 Days. *International Journal of Environmental Research and Public Health*, 19(8), 4543. <https://doi.org/10.3390/ijerph19084543>

Nédélec, M., **Chauvineau, M.**, and Martinent, G. (2024). Development and evaluation of the psychometric properties of a new measure of athlete insomnia: Insomnia in Response to Sports-related Stress Test questionnaire. *European Journal of Sport Science*. <https://doi.org/10.1002/ejsc.12095>

Pasquier, F., **Chauvineau, M.**, Castellini, G., Gianola, S., Barger, S., Vitale, J., and Nedelec, M. Does body cooling facilitated by bedding compared to control condition improve sleep? A systematic review and meta-analysis. *Under review*

Conference communications outside of the scope of the thesis

Chauvineau, M., Pasquier, F., Guyot, V., Aloulou, A., and Nedelec, M. (2021). Effect of the Depth of Cold Water Immersion on Sleep Architecture and Recovery Among Well-Trained Male Endurance Runners. 26th European College of Sport Science, Virtual Congress.

Nedelec M., **Chauvineau M.** (2021). Sur la route de Camerone : le sommeil d'un sportif d'ultra-endurance pédalant 10 000 km en 23 jours. Le Congrès du Sommeil, Lille, France.

Chauvineau, M., Pasquier, F., Guyot, V., Aloulou, A., and Nedelec, M. (2021). Effect of the Depth of Cold Water Immersion on Sleep Architecture and Recovery Among Well-Trained Male Endurance Runners. 19^{ème} congrès de l'Association des Chercheurs en Activités Physiques et Sportives, Montpellier, France.

Chauvineau, M. (2022). Intérêts de l'immersion en eau froide et d'un matelas à haute conductivité thermique sur le sommeil des sportifs. CNRS, Groupement de Recherche (GDR), Sport et Activité Physique, Virtual.

Nédélec, M., **Chauvineau, M.**, and Martinent, G. (2023). Development and evaluation of the psychometric properties of a new measure of athlete insomnia: Insomnia in Response to Sports-related Stress Test questionnaire. 28th European College of Sport Science, Paris, France.

Perez, J., Brocherie, F., Bornert-Antelo, T., Mrhar, Z., Nedelec, M., and **Chauvineau, M.** (2023). Is quality of mattress improving ice hockey players' recovery during congested travel schedule? 20^{ème} congrès de l'Association des Chercheurs en Activités Physiques et Sportives, Reims, France.

List of abbreviations

A.U.	Arbitrary unit	MnPO	Median preoptic nucleus
CBT	Core body temperature	N1	NREM 1
CI	Confidence interval	N2	NREM 1
DALDA	Daily Analysis of Life Demands for Athletes	NREM	Non-rapid eye movement
DOMS	Delayed-onset muscle soreness	POAH	Preoptic-anterior hypothalamus
EEG	Electroencephalogram	PSG	Polysomnography
EMG	Electromyogram	PSQI	Pittsburgh Sleep Quality Index
EOG	Electrooculogram	REM	Rapid eye movement
ES	Effect size	SCN	Suprachiasmatic nucleus
ESS	Epworth Sleepiness Scale	SD	Standard deviation
Est.	Estimate	SOL	Sleep onset latency
FIRST	Ford Insomnia Response to Stress Test	sRPE	Session rating of perceived exertion
GABA	γ -aminobutyric acid	SSI	Spiegel Sleep Inventory
GH	Growth hormone	SWC	Smallest-worthwhile change
GPS	Global positioning system	SWS	Slow-wave sleep
HMT	High-heat-capacity mattress topper	TIB	Time in bed
HR	Heart rate	TNF-α	Tumor necrosis factor alpha
HSD	High-speed distance	TST	Total sleep time
IL	Interleukin	VLPO	Ventrolateral preoptic nucleus
ISI	Insomnia Severity Index	$\dot{V}O_{2peak}$	Maximal oxygen consumption
LMT	Low-heat-capacity mattress topper	WASO	Wake after sleep onset
MEQ	Morningness-Eveningness Questionnaire		

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Introduction

Behind the apparent simplicity of going to bed and falling into the arms of Morpheus lies a complex organization leading to sleep. As described by J. Allan Hobson (1989, 2005), sleep is not simply the absence of waking, but a dynamic behaviour with a special activity of the brain, controlled by elaborate and precise systems in and for the brain and the whole body. Although why we sleep remains elusive and a scientific enigma (Frank, 2006; Krueger et al., 2016), sleep is defined as a non-negotiable biological state necessary to sustain human life, alongside air, food and water, to which humans devote around one-third of their lives. Sleep is crucial in proper biopsychosocial development, and for physical and mental health, cognitive functioning, and good quality of life (Grandner and Fernandez, 2021). Nevertheless, insufficient sleep is currently one of the most commonly reported health complaints, and is associated with a range of adverse effects on health (Grandner, 2022). In this regard, the American Academy of Sleep Medicine and the Sleep Research Society urge that sleep is a priority public health and societal issue (Luyster et al., 2012; Buysse, 2014).

In elite sport, the importance of sleep assumes another dimension. Practicing sport at the highest level entails highly demanding physiological and psychological requirements to succeed internationally, amidst dealing with various external stressors (e.g., travel, media, career pressure) and personal life constraints. This requires effective management of the recovery-stress balance to maintain the athlete's physical and mental health, well-being, and performance (Kenttä and Hassmén, 1998; Kellmann et al., 2018). Sleep is increasingly recognized as the foundation of athlete recovery and a crucial component in preparing for competition (Halson and Juliff, 2017; O'Donnell et al., 2018a). It is currently receiving particular interest from athletic/sporting organizations and the scientific community (Venter, 2014; Lastella et al., 2020a). In this context, numerous studies have sought to characterize the sleep of elite athletes, whether during training phases or competitions (Gupta et al., 2017; Roberts et al., 2019b). The picture is not rosy: **the prevalence of sleep complaints and inadequate sleep has been reported to be high in the elite athlete population**. Frequent travel, unfamiliar sleeping environments, varied training and competition schedules, and social and personal life obligations (e.g., family, work, study commitments) are well-identified contributory factors to sleep disturbances in athletes (Walsh et al., 2020).

Among these factors, **the substantially high training load imposed to prepare for major competitions is a potential disruptor of sleep in elite athletes that is not yet fully understood.** A dichotomy still remains. On the one hand, practicing regular physical exercise is recommended by the World Health Organization for improving both the quantity and quality of sleep in the general population (Bull et al., 2020). Numerous studies have demonstrated its benefits in improving the quantity and quality of sleep by reducing nocturnal awakenings, the time to fall asleep and the internal structure of sleep (architecture) towards a deeper sleep (Uchida et al., 2012; Kredlow et al., 2015; Park et al., 2021). On the other hand, some studies report sleep disturbances in elite athletes when exposed to excessive training loads (Roberts et al., 2019b; Walsh et al., 2020). Maladaptive response to training, i.e., prolonged decline in performance and/or increased perceived fatigue leading to overreaching/overtraining state, is usually accompanied by sleep complaints (Kellmann, 2010; Meeusen et al., 2013; Schwellnus et al., 2016). The vast majority of studies that impose large increases in training load have described experiencing more restless sleep, accompanied by a reduction in both sleep quantity and efficiency (i.e., time asleep as a percentage of time in bed) (Roberts et al., 2019b). Nonetheless, the literature is limited, with most information from studies using subjective or actigraphy measures for assessing sleep quantity and quality. Such methods do not provide the intricate neurophysiological features and internal structure of sleep typically achieved through polysomnography, which encompasses crucial information regarding sleep quality (McCarter et al., 2022). Using such an approach would provide insight into **how athletes' sleep architecture responds to varying training loads.** In this view, increased training load leads to physiological and psychological stress and may disrupt the organization and life around training (e.g., increased number and duration of training sessions), which can affect sleep. However, **their role in the potential detrimental effect of an overload on athletes' sleep remains unknown.** While such investigations conducted in real-life settings may face various logistical and operational challenges, in particular when performed in elite sport setup, they would definitely contribute to a better understanding of **how training load affects sleep and whether cofounding factors are involved.** This could offer guidance for practitioners in managing training programs and loads for the benefit of athletes' sleep, well-being, and performance.

At a time when sleep is a matter of health and society, developing practical strategies that can enhance it is highly necessary considering its importance in performance, well-being and health (Walsh et al., 2020). Several pharmacological and non-pharmacological sleep-enhancing strategies have been studied. Some are considered effective for improving sleep, such as

cognitive-behavioural therapy, sleep education and hygiene, and dietary manipulation (Pedlar et al., 2023). However, they have the limitation of requiring individual engagement, may not be short-term effective solutions, nor practical in high-pressure situations that occur in elite sport setting. Bedding is our nightly support for sleep, and is largely little studied. Due to the close, reciprocal relationship between sleep and the body's temperature regulation systems (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019), the thermal properties of bedding have been identified as a key factor of sleep quality (Troynikov et al., 2018). In this context, recent works have investigated the benefits of a high-heat-capacity bedding system on sleep in healthy volunteers (Kräuchi et al., 2018; Herberger et al., 2020, 2024) or elite rugby union players (Aloulou et al., 2020b). Sleeping on this bedding, consisting of a foam mattress coated with a gel sheet on top, has been shown to enhance the slow and continuous removal of body heat during sleep through conductive heat transfer. This improvement in heat dissipation has been associated with enhanced sleep quality and subjective sleep comfort. Nevertheless, these studies were conducted from an acute perspective, i.e., a single night. **The putative relevance of this bedding strategy when used chronically during a typical training context in athletes is currently unknown.** There is also no evidence that **this strategy can mitigate the potential detrimental effect of high training load on sleep.**

Furthermore, increasing and intensified exposure to high temperatures due to climate change is a major current issue (Patz et al., 2014; Watts et al., 2018) affecting both the general population and elite athletes. Sleep quantity and quality are highly sensitive to night-time ambient temperature (Lan et al., 2017), to the extent that predictions suggest that by 2099, rising temperature may erode 50–58 h of sleep per person-year (Minor et al., 2022). Given its interesting thermal properties, **investigating the effectiveness of a high-heat-capacity bedding system in athletes exposed to elevated temperatures is part of the effort to sustainably preserve global public health and human well-being.** Additionally, given the inter-individual variability of sleep (Nedelec et al., 2018) and the adaptive response to heat (Racinais et al., 2012; Corbett et al., 2018), describing individual responses to this strategy in this context is particularly relevant for achieving more precise guidance in prescribing individualized sleep interventions (Fullagar and Bartlett, 2016).

The aim of this doctoral thesis is to deepen our understanding of the **impact of training load on sleep** and to explore the **effectiveness of a high-heat-capacity bedding system on athletes' sleep within a typical training context.** Special attention will be paid on assessing sleep architecture

and the use of objective and subjective sleep monitoring methods to encompass the concepts of sleep quantity and quality. The experimental part of this work is structured around three studies conducted *in situ*, within athletes' training contexts, utilizing descriptive, controlled, and interventional designs.

This manuscript consists of four main chapters. The **first chapter** includes a literature review on the importance of sleep for athletes and the role of training load as a potential modulator of sleep. We will also explore the relationship between sleep and the body's temperature regulation systems, as well as studies investigating thermal strategies to influence sleep. The **second chapter** will provide detailed information on the general methodology associated with experimental studies. The **third chapter** will focus on the experimental contributions and present the findings of the three studies conducted within the framework of this PhD thesis. Finally, the **fourth and last chapter** will discuss the overall results, leading to research perspectives and practical applications of the present work.

Part 1 – Sleep: the bedrock of the athlete recovery

“Sleep is extremely important, probably more important than any other recovery routines that you could do [...]. I try to get those eight or nine hours of beauty sleep. I think that helps a lot with recovery, feeling good, feeling fresh” – Novak Djokovic

In the context of sport, recovery is now considered an integral part of the training process (Mujika et al., 2018; Skorski et al., 2019). It enables an athlete to restore the internal homeostasis and return to or exceed their previous performance levels, the recovery period being the time needed to achieve these adaptations (Hauswirth and Mujika, 2013). Sleep is recognized as the foundation of recovery and is essential in athlete performance, health, and well-being (Halson and Juliff, 2017; O'Donnell et al., 2018a). The first part of this review defines sleep, its functions and its importance in the athlete recovery, and provides an overview of elite athletes' sleep.

1. Human sleep physiology and its regulation

1.1. Internal structure of sleep

1.1.1. Characteristics for measuring and defining sleep

Behaviourally, sleep can be defined as a naturally recurring and reversible state in which the sleeper experiences perceptual disengagement and unresponsiveness to the environment (Carskadon and Dement, 2017; Grandner and Fernandez, 2021). For many years, sleep was considered as an inactive state of the brain (Hobson, 2005; Pelayo and Dement, 2017). In 1928, when the psychiatrist Hans Berger recorded brain electrical activity, changes in cortical rhythms, as recorded by electroencephalogram (EEG), were observed between wakefulness and sleep. This discovery marked the first major step towards a modern understanding of human sleep. Further progress in the 1950s and 1960s revealed the regular alternation of the two basic sleep states: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. Healthy human sleep begins with NREM sleep, followed by REM sleep, usually occurring 80 min or more after sleep onset. A cyclical alternation of NREM and REM sleep then occurs in an ultradian rhythm, with an average NREM-REM cycle period of around 90 to 120 min throughout the night (Achermann and Tarokh, 2014). The changes in oscillatory activity in the EEG (frequency and amplitude), coupled with electromyographic (EMG) and electrooculographic (EOG) signals are typically monitored via a polysomnography (PSG) recording (detailed in page 80), allowing to distinguish three NREM sleep stages (NREM 1–N1, NREM 2–N2, and NREM

3–N3) from REM sleep. By recording these physiological parameters, it is possible to determine sleep architecture, which refers to the internal structure of sleep and the time-course of sleep stages (hypnogram, **Figure 1–top**). The cortical oscillations present during sleep can also be summarized using a fast Fourier transform to obtain the time-course of EEG power density spectra (spectrogram, **Figure 1–bottom**), which provides an additional overview of the internal structure of sleep (Dijk and Landolt, 2019).

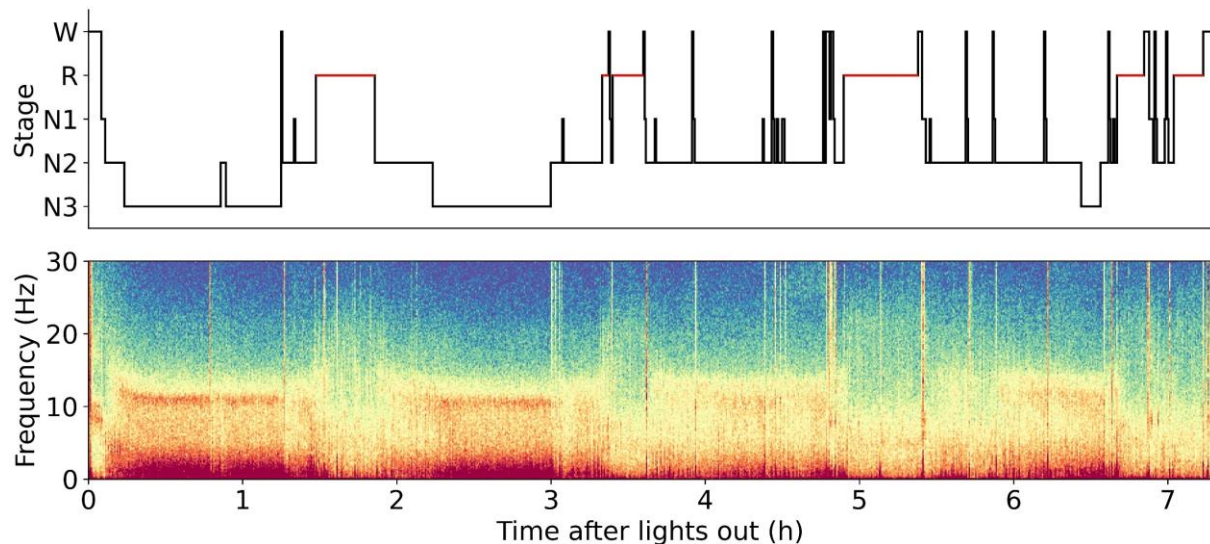


Figure 1. Sleep architecture of an athlete (23 years old) assessed in the present thesis. Top, time-course of sleep stages (hypnogram); bottom, full-night spectrogram of the central EEG channel (C3-M2). Warmer colors indicate higher spectral power in the 0-30 Hz frequency range. Spectrogram allows to easily identify the NREM-REM sleep cycles and overall sleep architecture. *Abbreviations:* W, wake; R, REM sleep; N1–N3, NREM sleep stages 1–3. Figure produced using the “yasa” Python package, v0.6.3 (Vallat and Walker, 2021).

1.1.2. Sleep stages

In this PhD thesis, the 2017 American Academy of Sleep Medicine (Berry et al., 2017) criteria were employed for scoring sleep and associated events, according to the four brain electrical frequency bands relevant for sleep staging: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz) and beta (>13 Hz). The following section describes sleep stages as they can be observed in healthy young adults, in line with the population studied in the present PhD thesis.

N1 sleep initiates sleep for only a few minutes (Carskadon and Dement, 2017). The EEG signal is of low amplitude and mixed frequency, predominantly composed of theta rhythms (beginning to disappear from the alpha rhythm that characterizes the relaxed waking state). This sleep stage is marked by the presence of vertex sharp waves lasting <0.5 s, predominantly in the central and occipital regions, by slow eye movements usually lasting >0.5 s, and by a gradual decline in EMG activity (**Figure 2–top left**). N1 sleep is the lightest sleep stage during which the sleeper can be easily awakened and is a transition sleep stage throughout the night, which

accounts for about 2–5% of total sleep duration. An increase in the amount and percentage of N1 sleep may reflect a more sensitive and discontinuous sleep (Ohayon et al., 2004).

N2 sleep is also composed of a low-amplitude and mixed-frequency EEG signal (mainly theta rhythms) and is characterized by the occurrence of two typical EEG waveforms: sleep spindles and K-complexes (**Figure 2–top right**). Sleep spindles are distinct sinusoidal waves with frequencies ranging from 11 to 16 Hz, lasting between 0.5 and 1.5 s. K-complexes are biphasic sharp waves predominantly in the frontal region, with a negative and then a positive phase lasting ≥ 0.5 s and with a typical amplitude >75 μV . EOG and EMG activity are similar to that of N1 sleep. A more intense stimulus is required to wake the sleeper, and its overall proportion is about 45–55% of sleep duration (Ohayon et al., 2004).

N3 sleep, often referred to as slow-wave sleep (SWS), is defined by the presence of synchronized slow waves (0.5–2 Hz) of high-voltage (>75 mV), among a predominance of delta rhythms (**Figure 2–bottom left**). The power densities of delta rhythms obtained by spectral analysis are typically referred to as slow-wave activity (SWA) (Léger et al., 2018). The amount of SWS and SWA can be considered as indicators of “sleep depth” or “sleep intensity” (Achermann and Tarokh, 2014), and are linked to the length of prior time awake, i.e., sleep homeostasis (see page 22). During SWS, a greater stimulus than for N1 or N2 is usually required to generate an awakening. The EOG activity is similar to the EEG rhythms. The proportion of SWS is most prominent in the first part of the night, before progressively declining (**Figure 1**). Across the night, SWS account for 10–25% of sleep duration (Ohayon et al., 2004).

REM sleep can be described as an “activated brain in a paralysed body” (Carskadon and Dement, 2017). REM sleep is distinguished from NREM by low-amplitude and high-frequency EEG patterns, concurrently with muscle atonia, and episodic bursts of rapid conjugate eye movements lasting <0.5 s (**Figure 2–bottom right**). Muscle atonia is due to blocking of the cortico-spinal pathways by the brain stem, resulting in a suppression of muscle tone, visible in the EMG (Siegel, 2017). Saw-tooth theta waves may also appear during REM sleep. REM occurs with a higher proportion in the second half of the night, usually accounting for 20–25% of sleep duration (Ohayon et al., 2004).

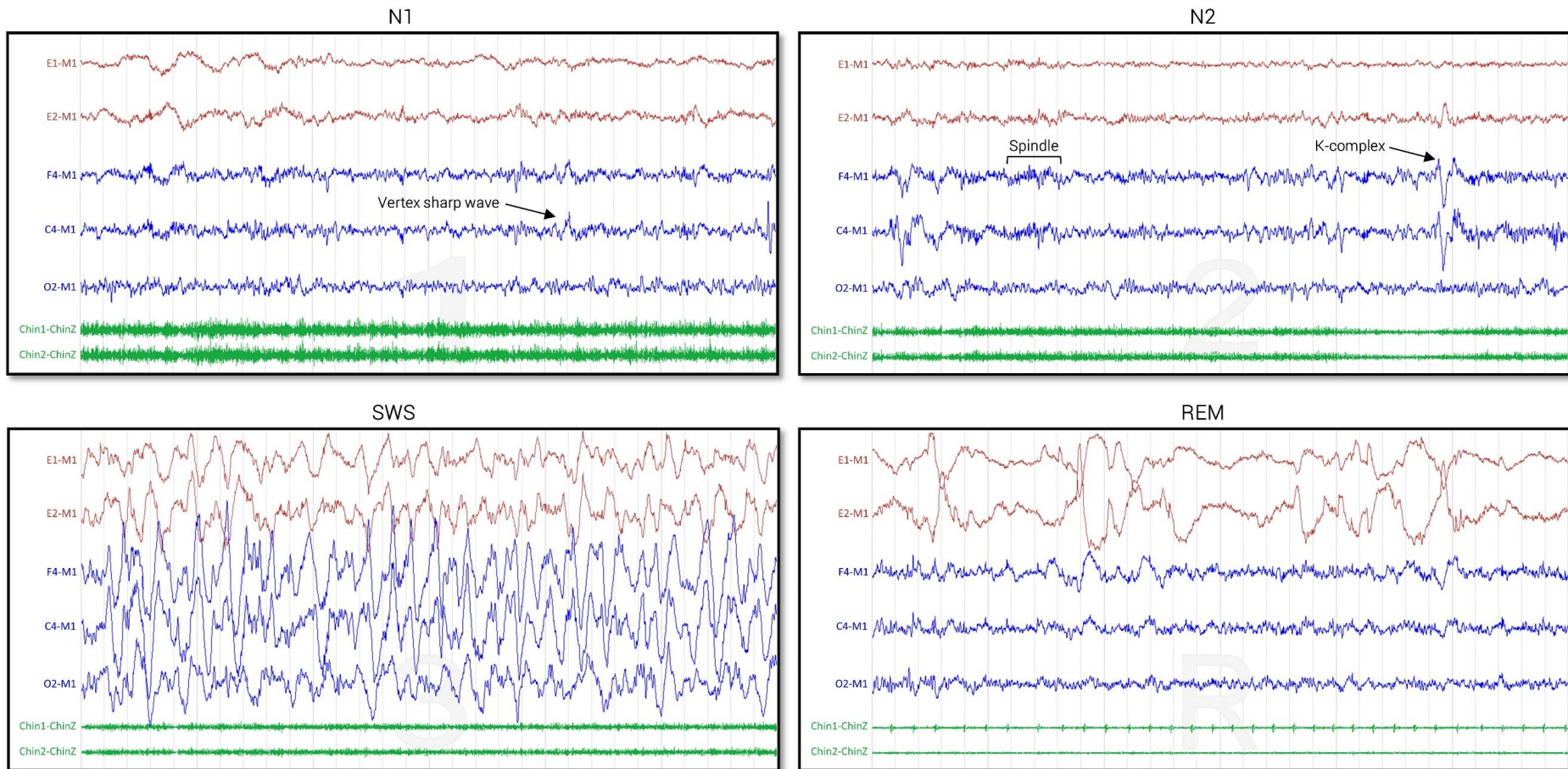


Figure 2. Electrooculographic (EOG, red), electroencephalographic (EEG, blue) and electromyographic (EMG, green) signals in 30-s epochs according to sleep stages. *Notes:* This screenshot of the Noxturnal software represents a polysomnography recording of an athlete (23 years old) assessed in the present PhD thesis. Signals are shown with a gain of 100 $\mu\text{V}/\text{cm}$. *Abbreviations:* N1–N2, non-rapid eye movement (NREM) sleep stages 1–2; SWS, slow-wave sleep; REM, rapid eye movement.

1.2. Sleep-wake regulation systems

Since the 1950s, scientific research has made considerable progress in understanding the neurobiological and regulatory mechanisms of human sleep (Frank, 2006). The most prominent model of sleep regulation is the “two-process model” (Figure 3), first described by Alexander Borbély in 1982. This model considers sleep propensity as the result of the mutual interaction of a homeostatic process (Process S) and a circadian process (Process C), with time-courses derived from physiological and behavioural variables (Borbély, 1982; Borbély et al., 2016).

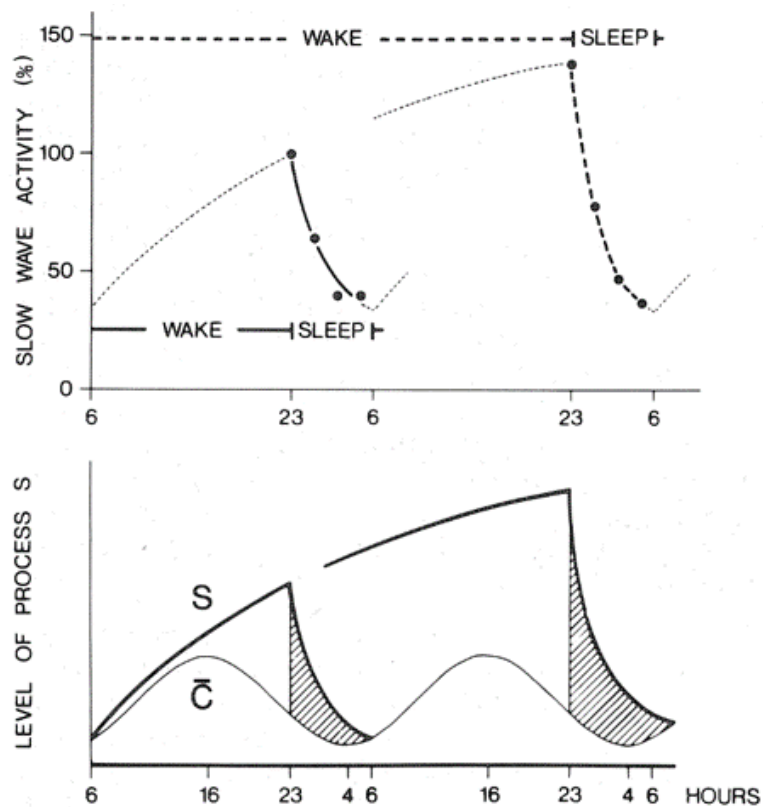


Figure 3. The two-process model. The above part shows the exponential decline in slow-wave activity (SWA) over sleep cycles (100% = value of first cycle) on a typical night (solid line) and after sleep deprivation (dashed line). Dotted curve illustrates the exponential increase of SWA propensity during waking period. Lower part represents the time course of Process C and S, intercepted with sleep periods (dashed area). Sleep propensity is highest when the distance between the Process S and Process C is greatest. Figure from Borbély (1982).

1.2.1. Circadian process

Process C refers to the circadian biological clock (from Latin *circa* meaning “about” and *dies* meaning “day”), a system for the temporal organization of numerous physiological functions (Czeisler and Buxton, 2017). This system is characterized by an endogenous rhythmicity near 24 h that runs independently from daily periodic changes in the environment, but can be

entrained by synchronizers (i.e., *zeitgebers*) so that its activity is synchronized with the 24-h period of the earth's rotation cycle (Gronfier, 2014; Leger et al., 2018).

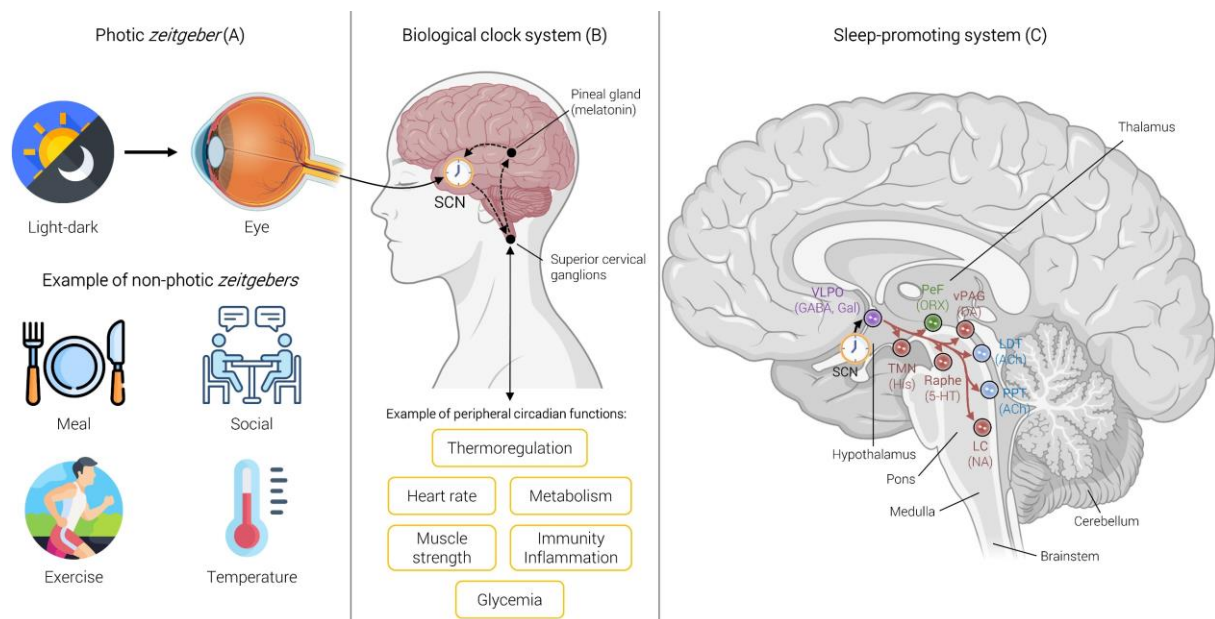


Figure 4. Schematic representation of circadian clock regulatory pathways and the sleep-promoting system. (A-B) Light-dark cycle directly entrains the suprachiasmatic nucleus (SCN) together with non-photoc *zeitgebers*. At night, photic information is transmitted from the eye to the SCN, then to the pineal gland. Melatonin is secreted, which modulates the SCN itself (feedback effects). A network of brain and peripheral clocks are then synchronized by SCN, leading to circadian rhythms of physiologic, metabolic, and hormonal parameters. (C) The SCN projects to neurons in the ventrolateral preoptic nucleus (VLPO), who promotes sleep via inhibitory actions of the main components of the wake-promoting areas. Adapted from Saper et al. (2005), Hickie et al. (2013) and Grosbellet and Challet (2017).

Clock function originates from within each cell, building a circadian program network in tissues, organs, and the entire organism (Koronowski and Sassone-Corsi, 2021). The top-down master circadian pacemaker is located in mammals above the optic chiasma, in the suprachiasmatic nucleus (SCN) of the hypothalamus (**Figure 4**) (Roenneberg and Mellow, 2016). Many physiological and behavioural rhythms are regulated and synchronized by the SCN, including the sleep/wake cycle, body temperature, feeding, neuroendocrine and autonomic systems (detailed from page 66) (Saper et al., 2005; Deboer, 2018). The SCN regulates sleep-wake rhythms by transmitting the circadian signal via a rostral projection to the sleep-promoting areas, i.e., the ventrolateral preoptic nucleus (VLPO) of the preoptic-anterior hypothalamus (POAH). The light-dark cycle is the primary *zeitgeber* of the SCN. Through the photosensitivity (intensity and wavelength) of the melanopsin ganglion cells in the retina, that projects to the SCN via the retinohypothalamic tract, the SCN rhythm period and phase are aligned with the external environment (Grosbellet and Challet, 2017). As night approaches, a neural output signal, generated by the SCN, induces the synthesis of melatonin in the pineal gland, an important endogenous sleep regulator hormone (**Figure 4**) (Zisapel, 2018). The release of

melatonin provides a temporal signal to peripheral organs and the SCN itself through feedback effects, activating sleep-promoting pathways and attenuating wake-promoting pathways (detailed below) (**Figure 4**). Additionally, melatonin release plays a pivotal role in sleep-related thermoregulatory processes, contributing to heat loss from the body (detailed from page 66). This results in a sharp increase in sleep propensity, usually occurring within 2 h after the onset of endogenous melatonin production (Cajochen et al., 2003). Conversely, light acts to inhibit melatonin release, which promotes wakefulness (Guardiola-Lemaître and Quera-Salva, 2011; Grosbellet and Challet, 2017; Zisapel, 2018). The environment provides additional temporal cycling cues for the SCN, including the sleep-wake cycle, meal timing, social routines, physical exercise and environmental temperature (Saper et al., 2005; Czeisler and Buxton, 2017; Grosbellet and Challet, 2017).

1.2.2. Homeostatic process

Process S refers to a progressive increase in sleep propensity during time awake and its dissipation during sleep (Borbély and Achermann, 1999). The amount of SWS and SWA are key indicators of sleep homeostasis, characterized by a progressive decline in their amount as sleep progresses (**Figure 1**) (Achermann and Tarokh, 2014; Deboer, 2018). After sleep debt, i.e., partial (sleep restriction) or total sleep deprivation, the body attempts to recover the amount lost by a subsequent "rebound" of sleep, marked by an increase in subsequent sleep duration, if possible, and both the amount of SWS and SWA (**Figure 3**) (Dijk et al., 1990; Dijk, 2009).

The homeostatic mechanisms are regulated by neuroanatomical and neurochemical systems in the brain (Saper et al., 2005; Murillo-Rodriguez et al., 2012; Deboer, 2018). On the one hand, neurons located primarily in the VLPO of the POAH promote sleep propensity by releasing neurotransmitters such as γ -aminobutyric acid (GABA) that inhibit multiple wake-promoting regions, including the basal forebrain, posterior hypothalamus, and brainstem (**Figure 4**). On the other hand, wakefulness is maintained by these wake-promoting regions via neurons expressing neurotransmitters such as acetylcholine, norepinephrine, orexin, glutamate, and histamine, which inhibit the GABAergic neurons in the VLPO. These mutually inhibitory processes have been described as a bistable, "flip-flop" switch circuit (Saper et al., 2005; Murillo-Rodriguez et al., 2012; McGinty and Szymusiak, 2017; Szymusiak, 2018). GABA release depends in particular on the concentration of adenosine in the brain, a breakdown product of adenosine triphosphate, which has been identified as a homeostatic accumulator of the need to sleep (Landolt, 2008). As waking progresses and/or energy demand increases, adenosine expands in

the brain extracellular space and binds to adenosine receptors (e.g., A₁ or A_{2a}), activating the GABAergic neurons in VLPO and promoting sleep propensity (Deboer, 2018). Other hypnogenic substances released in the brain as a result of neuronal/glial activity have been identified, such as Interleukin-1 β (IL-1 β) and tumor necrosis factor alpha (TNF- α), which also promote sleep propensity (Krueger et al., 2016). In this regard, it is assumed that the quality of sleep depends, in part, on the prior quality of wakefulness (Dijk and Landolt, 2019). Thus, the highly physical demand experienced during training play an important role in modulating elite athletes' sleep (detailed from page 50).

2. Importance of sleep for elite athletes

2.1. Theoretical functions of sleep

Although recent progress has contributed to characterize how humans sleep, the fundamental question of why we sleep remains a scientific enigma (Halson, 2008; Krueger et al., 2016). Several possible interrelated theories of sleep functions have been proposed, which suggest reciprocal modulation between sleep and the body (Frank, 2006; Frank and Heller, 2019). These functions can help understand why insufficient sleep may have detrimental effects on athletic performance.

2.1.1. Somatic theory

The first theory is the somatic one, which proposes that sleep facilitates the body's anabolic and restorative processes through its interaction with the endocrine and immune systems (Frank, 2006). Sleep is accompanied by a downregulation of the hypothalamic-pituitary-adrenal axis (a major neuroendocrine system) and sympathetic nervous system axis, leading to a marked increase in anabolic hormones such as testosterone, growth hormone (GH), prolactin and insulin-like growth factor 1 (Czeisler and Buxton, 2017; Chennaoui et al., 2020, 2021; Morrison et al., 2022). These hormones affect the rates of muscle protein synthesis and are regulators of muscle tissue growth (Rooyackers and Nair, 1997). For instance, the release of GH, secreted by the pituitary gland, is mainly observed during SWS (Takahashi et al., 1968; Obal and Krueger, 2004), and is closely related, temporally and quantitatively, with relative delta power of the sleep EEG, i.e., SWA (Gronfier et al., 1996), although their causal relationship is still debated (Léger et al., 2018). Conversely, some evidence demonstrates that sleep debt induces an anabolic resistance by reducing these muscle protein synthesis (Lamon et al., 2021), and may lead ultimately to muscle atrophy (Morrison et al., 2022). Cortisol is a hormone secreted

by the adrenal glands, with the highest concentrations typically measured in the early morning upon waking, declining throughout the afternoon and evening (Morgan and Tsai, 2015). However, this hormone is involved in muscle protein degradation, and just one night of sleep deprivation is enough to increase its plasma concentration by 21% in healthy adults (Lamon et al., 2021). Given that intensified training typically leads to muscle tissue damage (Paulsen et al., 2012), sleep may play a critical role in optimizing the repair of exercise-induced muscle damage in athletes and for achieving the desired training-induced skeletal muscle adaptations (e.g., hypertrophy) (Dattilo et al., 2011; Morrison et al., 2022).

Additionally, sleep and inflammatory responses are well-known to be bidirectionally linked (Irwin, 2019). On the one hand, circulating levels of anti-inflammatory cytokines such as IL-10 are higher during the daytime, whereas proinflammatory cytokines (e.g., IL-1 β , IL-6, TNF- α , and IL-12) typically peak during sleep (Bollinger et al., 2010; Léger et al., 2018). Pro-inflammatory endocrine release appears to be associated particularly with the onset of SWS during the early nocturnal sleep phase (Besedovsky et al., 2012). On the other hand, some evidence suggests that increased proinflammatory cytokines (in particular IL-1 β , TNF- α) play a role in the homeostatic regulation of sleep by potentially promoting the amount of NREM sleep and suppressing REM sleep (Besedovsky et al., 2019). This may have implications in the context of sports, given the cascade of inflammatory responses led by the immune system following exercise-induced muscle damage (Paulsen et al., 2012), as observed in endurance running (Nieman et al., 2014), which may be further accentuated by collisions typical of contact sports such as rugby (Naughton et al., 2018). This strengthens the interdependence of sleep and the preceding waking phase. However, it is important to note that these relationships have been exclusively assessed in a lab-based context, primarily using animal models, and the extent to which these mechanisms operate in athletes remains hypothetical.

2.1.2. Housekeeping theory

The second theoretical function of sleep is housekeeping, suggesting that sleep restores and repairs neural substrates, contributes to metabolic and cellular energy conservation, and detoxifies substances accumulated during wakefulness (Frank, 2006; Frank and Heller, 2019). This theory assumes that sleep contributes to the activation of enzymes and the expression of genes that only occur during sleep, aiming for the synthesis of synaptic proteins. These processes may play an important role in regulating cellular processes during sleep, such as memory consolidation and cognition (Tudor et al., 2016).

The metabolic energy conservation theory is supported by several studies showing that brain glucose consumption, the major brain energy reserve, is approximately twice as high during waking as during SWS (Krueger et al., 2016). This reduced glucose consumption during SWS compared with wakefulness results in an increase in brain glucose concentration, which would coincide with hormonal changes decreasing insulin sensibility (Morselli et al., 2012; Léger et al., 2018). Recently, it has been observed that the coupling between slow-wave oscillations (<1 Hz) and NREM sleep spindles could be an important predictor of next-day fasting blood glucose levels in humans (Vallat et al., 2023). This theory of restoring energy reserves has been demonstrated among athletes whose sleep loss has stunted muscle glycogen re-synthesis, which may explain in part the subsequent impairment of athletic performance (Skein et al., 2011). In contrast, circulating blood glucose concentration appears to remain unaffected by sleep restriction when SWS is maintained, highlighting the importance of this sleep stage in metabolic conservation (Kontou et al., 2022). This may be especially significant for endurance athletes, whose availability of muscle glycogen increases exercise capacity and performance (Impey et al., 2020).

Moreover, sleep may also assist brain detoxification. There is some evidence to suggest that sleep in mice drives the clearance of metabolic waste products in the brain accumulated during wakefulness via the glymphatic system (Xie et al., 2013). An experimental study in humans demonstrated *in vivo* that one night of sleep deprivation impairs the elimination of metabolic waste from the brain, an effect not alleviated by a further night's sleep (Eide et al., 2021). Specifically, SWA and slow-wave oscillations (<1 Hz) have been suggested as predictors of the clearance of brain-damaging metabolic waste, such as β -amyloid, whose accumulation has been associated with cognitive aging and Alzheimer's disease (Ngo et al., 2020; Winer et al., 2020). In the context of sport, the detoxifying function of sleep in the brain may have implications, particularly in contact sports, due to the potential accumulation of metabolic waste products (e.g., β -amyloid) associated with collision-based actions, which can reciprocally disrupt the glymphatic system and sleep (Asken et al., 2016; Aloulou et al., 2020b), although this remains a hypothesis.

2.1.3. Cognitive theory

The third theoretical function of sleep is cognitive, and proposes that sleep contributes to higher-order functions such as learning, memory, and synaptic plasticity (Frank, 2006; Frank and Heller, 2019). Numerous studies conducted from the 1990s demonstrated the role of sleep

in long-term memory consolidation in humans, as evidenced by learning-related brain activity in neuroimaging data during sleep (Cousins and Fernández, 2019; Hoedlmoser et al., 2022). Nowadays, there is a growing evidence in favour of the *active systems consolidation theory*, which proposes a replay of new memories, involving a dialogue between the hippocampus and cortex, to promote brain synaptic plasticity (Diekelmann and Born, 2010; Klinzing et al., 2019). This hypothesized sleep function is essential for athletes given the importance of memory for athletic, tactical and cognitive development and performance (Watson, 2017). The contribution of sleep to learning is also crucial for athletes, for both cognitive and motor skills. For instance, studies have demonstrated that subjects training a motor skill task in the morning experienced ~20% improvement in motor speed without loss of accuracy following a night of sleep, while an equivalent period of wakefulness provides no significant additional benefit (Walker et al., 2002).

Specific sleep stages and EEG features (e.g., slow-wave oscillations, sleep spindles) have been identified as exerting different functions in memory consolidation and synaptic plasticity (Diekelmann and Born, 2010; Peigneux et al., 2017). A *dual-process hypothesis* has been proposed, suggesting that SWS preferentially benefits declarative memory (explicit = semantic and episodic), while REM sleep is particularly important for non-declarative memory (implicit = procedural skills and emotional). From the perspective of EEG features, memory consolidation would be strengthened by facilitated synaptic plasticity in cortical pyramidal cells when sleep spindles are coupled with slow-oscillations (Klinzing et al., 2019; Schreiner et al., 2021; Hahn et al., 2022). In addition, Strauss et al. (2022) showed that sleep spindles are associated with memory consolidation only when NREM sleep is followed by REM sleep, providing insights into why NREM and REM sleep alternate cyclically, a question still unanswered (Frank and Heller, 2019).

2.2. Role of sleep in athletic performance

2.2.1. Impact of insufficient sleep on performance

Based on the aforementioned theoretical functions of sleep, it is understood that sleep plays a crucial role in various restorative processes and cognitive development. Therefore, if sleep holds importance for athletes, there should be evidence indicating that insufficient sleep is detrimental to athletic performance.

The adverse effects of sleep loss on performance appear to be unequivocal, affecting athletic performance (e.g., strength and endurance), neurocognitive function (e.g., attention and

memory), as well as potentially increasing the risk of physical health issues (e.g., illness and injury) (Fullagar et al., 2015; Simpson et al., 2017; Craven et al., 2022). Craven et al. (2022) conducted a meta-analytical review on the effects of acute sleep loss on various exercise tasks, including various protocols from early/late sleep restriction to total sleep deprivation. While all exercise tasks were affected by sleep loss, the magnitude of these effects varied across exercises. Tasks demanding a high cognitive component for execution appeared to be the most sensitive to sleep loss (mean performance percentage change: -20.9%), followed by strength-endurance tasks (i.e., resistance tasks with ≥ 2 repetitions or > 5 seconds sustained contraction; -9.9%), anaerobic power tasks (i.e., activities lasting ≤ 6 seconds performed at maximum effort; -6.3%), high-intensity interval exercise tasks (i.e., near-maximal effort with brief periods of recovery; -6.2%), endurance tasks (i.e., continuous exercise lasting ≥ 120 seconds; -5.6%), speed/power endurance tasks (i.e., maximal continuous exercise lasting 6–90 seconds; -2.9%), and peak strength tasks during a single effort (-2.9%).

However, it is important to note that the amount (e.g., deprivation/restriction) and type (e.g., early restriction/late restriction) of sleep loss incurred are determining factors in the magnitude of the effect that sleep loss has on athletic performance (Fullagar et al., 2015). Sleep restriction refers to a period during which sleep is limited to ≤ 6 h per night, often resulting from delayed sleep onset, earlier than normal waking, or fragmented sleep, characterized by one or more nocturnal awakenings. Total sleep deprivation refers to a period of sustained wakefulness lasting longer than 24 h (Lastella et al., 2014; Halson and Juliff, 2017). The magnitude of the effect of sleep loss on performance appears to be greater when individuals experience either sleep deprivation or late restriction, with the impact being more pronounced the longer the period of wakefulness prior to exercise (i.e., afternoon or evening) (Craven et al., 2022).

The diverse magnitudes of the effects of sleep loss across exercise tasks may result from the observation that cognitive aspects of performance could be more susceptible to sleep loss, both in terms of magnitude and duration, compared to physical components (Fullagar et al., 2015). Most studies on sleep loss have reported alterations in mood states (e.g., irritability, bad mood, mental fatigue), increased sleepiness, depression, and confusion, which can affect both motivation and perceived exertion as primary outcomes of sleep loss (Fullagar et al., 2015; Romdhani et al., 2019). These effects may subsequently impair cognitive processing abilities such as reaction time, decision-making, memory, and learning (Van Dongen et al., 2003). Although less unequivocal, some physiological responses to exercise may also be altered by a lack of sleep, such as cardiorespiratory variables during incremental exercise (e.g., heart rate,

minute ventilation) (Dean et al., 2023), sympathetic and parasympathetic cardiovascular modulation, pro-inflammatory cytokines responses (Dáttilo et al., 2020) and muscle glycogen concentration (Skein et al., 2011, 2013). These alterations may alter some aspects of physical performance (Fullagar et al., 2015). Thus, disciplines that require both high cognitive function and physical reliance with repeated exercise bouts (e.g., rugby union, racket sports) are likely to be most affected by acute sleep loss (Craven et al., 2022; Fullagar et al., 2023). Furthermore, given the significant motivational component of prolonged endurance exercises, such as endurance running, the altered mood state and increased perceived exertion due to sleep loss can also be detrimental to these disciplines. Roberts et al. (2019a) reported that compared with normal sleep (i.e., ~7 h), endurance cycling time-trial performance was unaffected after one night but impaired (by ~3%) after two nights of sleep loss (i.e., <5 h of sleep per night). After sleep loss, mood and psychomotor vigilance were impaired, and for a given power output, perceived exertion was higher. These authors suggest that the decline in endurance performance induced by sleep loss may result, in part, from increased perceived mental fatigue, reduced pain tolerance, or lower motivation (Roberts et al., 2019a). Finally, decreased sleep has been proposed as a risk factor for injury in various populations of athletes (Milewski et al., 2014; Watson et al., 2020, 2021; Hamlin et al., 2021; Ochoa-Lácar et al., 2022), further reinforcing the assertion that inadequate sleep is detrimental to elite athletes.

However, while evidence suggests that sleep loss impairs physical and cognitive functioning, it remains unclear to what extent this impacts elite athletes, as most studies did not examine elite athletes' sleep loss under real-life conditions (Fullagar et al., 2015; Craven et al., 2022). The few studies examining the effects of real-life partial sleep restriction (i.e., not forced sleep loss) on athletic performance have shown that single bouts of maximal exercise are not significantly affected (Bonnar et al., 2018). Conversely, sport-specific technical skills, reaction time, memory and mood stability are more likely to be impaired (Vitale et al., 2017, 2021). In addition, there are also limited number of studies that have examined the impact of mild, chronic sleep loss on exercise and sports performance in elite athletes (Sargent et al., 2021), which have been reported to be detrimental to many cognitive tasks (e.g., vigilance, reaction time, rate of errors on a response time task), as observed in untrained adults (Belenky et al., 2003; Van Dongen et al., 2003). Finally, there is much more to consider than just the impact of sleep loss on cognitive or physical performance. Poor sleep quality (defined in page 30) may also be detrimental to athletic performance (Craven et al., 2022). Nevertheless, the evidence of a link between cognitive or physical performance and indicators of physiological and perceived measured of

sleep quality are limited (McCarter et al., 2022). This is mainly because only a few studies simultaneously include physiological and subjective measures of sleep quality and performance.

2.2.2. Performance benefits of sleep extension

Unlike sleep loss, some studies have explored the benefits of extending the sleep period on performance (Silva et al., 2021; Gwyther et al., 2022). Sleep extension refers to scheduling a longer sleep opportunity than normal, usually a window of 9–10 h for sleepers who typically get 7–9 h per night (Walsh et al., 2020; Silva et al., 2021). In collegiate basketball players and using a pre-post intervention design, Mah et al. (2011) demonstrated that a sleep extension program, consisting of a minimum of 10 h in bed per night for 5–7 weeks, increased sleep duration from 6.7 ± 1.0 h before the intervention (below the recommended 7 h, see below) to 8.5 ± 1.3 h. This longer sleep duration was associated with significant improvements in reaction time, sprint times, and free-throw shooting accuracy compared with before the sleep extension program. Similarly, improvements in prolonged cycling time-trial performance (Roberts et al., 2019a), in the accuracy of tennis service (Schwartz and Simon, 2015) and reaction time in rugby players (Swinbourne et al., 2018) have been also reported following three days, one week and three week of sleep extension, respectively. In addition to its effect on athletic performance, these studies reported benefits of extending sleep to reduce perceived fatigue, improve mood, vigour and psychomotor vigilance (Mah et al., 2011; Roberts et al., 2019a).

It is important to note that the performance benefits of sleep extension vary across studies, mainly depending on the sleep characteristics of the athletes (e.g., chronotype, sleep pattern, and potential current sleep debt) as well as the parameters of the sleep extension intervention (e.g., the gap between the current amount of sleep and whether the intervention is acute or chronic) (Silva et al., 2021). The performance benefits of prolonged sleep duration thus emphasize that some athletes do not obtain sufficient sleep to achieve peak performance, notably when falling below the *National Sleep Foundation* recommendations (see below) (Mah et al., 2011). Nevertheless, the performance benefits following improvements in sleep quality are unclear, and research in this area is warranted to explore more than just the impact of sleep quantity on athletic performance.

Overall, the aforementioned studies on sleep restriction/deprivation and extension highlight the sensitivity of both physical and cognitive performance, as well as perceived fatigue and mood,

to current sleep patterns. This emphasizes the crucial role of adequate sleep in achieving peak athletic performance. The following section provides information on indicators and recommendations for adequate sleep.

2.2.3. What are the indicators of adequate sleep?

The *National Sleep Foundation* established recommendations for the quantity and quality of sleep according to age (Hirshkowitz et al., 2015b; Ohayon et al., 2017). **Table 1** provides the definition and associated recommendations of the main indicators of sleep quantity and quality used in the present PhD thesis. An 18-member multidisciplinary panel, comprising sleep experts, developed these recommendations on the basis on a systematic review of medical and scientific research regarding how appropriate are the indicators for health and performance (Hirshkowitz et al., 2015a). The recommended sleep duration for young adults (18–25 years) and adults (26–64 years) is 7–9 h (Hirshkowitz et al., 2015b) (**Figure 5**). Regarding the quality of sleep, the recommendations concern markers of sleep continuity, i.e., ability to initiate and maintain sleep during the night (e.g., sleep onset latency [SOL], wake after sleep onset [WASO], sleep efficiency), macro-architecture (i.e., distribution of sleep stages) and fragmentation (e.g., number of stage-shifts, awakenings, cortical arousals and limb movement disturbances) (Ohayon et al., 2017) (**Table 1**).

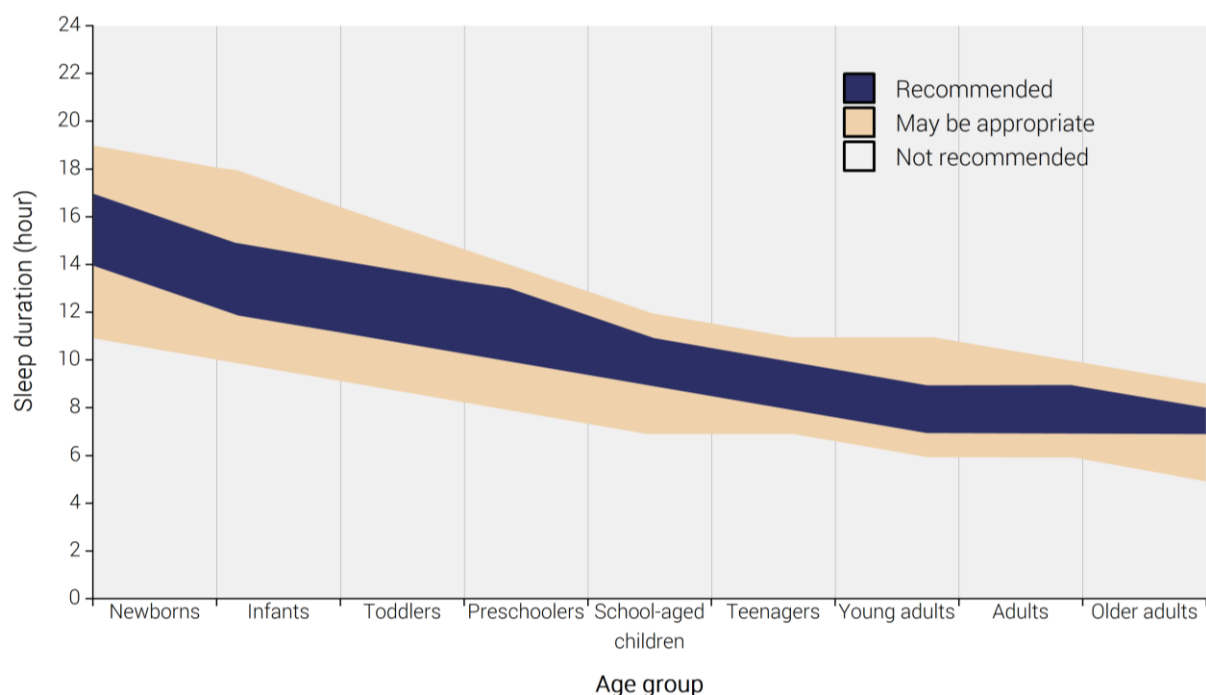


Figure 5. *National Sleep Foundation* recommendations for sleep duration across the life-span. *Notes:* newborns, 0–3 months; infants, 4–11 months; toddlers, 1–2 years; preschoolers, 3–5 years; school-aged, 6–13 years; teenagers, 14–17 years, young adults, 18–25 years; adults, 26–64 years; older adults, ≥65 years. Adapted from (Hirshkowitz et al., 2015b).

Table 1. Definition of sleep variables and associated recommendations for healthy adults.

Sleep variable	Definition	Recommendations			
Time in bed (TIB, hour)	Time between bedtime and get-up time	-	-	-	
Sleep continuity	Total sleep time (TST, hour)	<6 >10	6–7 9–10	7–9	
	Sleep onset latency (SOL, min)	>45	31–45	≤30	
	REM latency (min)	-	-	-	
	Wake after sleep onset (WASO, min)	>40	21–40	≤20	
	Sleep efficiency (%)	<75	75–84	≥85	
Sleep architecture	Time in each sleep stage (%)	Time spent in each of sleep stages expressed as a percentage of TST:			
	N1	≥20	36–20	≤5	
	N2	>81	≤81	-	
	SWS	≤5	6–16 >20	16–20	
	REM	>40	<21 31–40	21–30	
Sleep fragmentation	Sleep stage shifts (events per hour)	Number of changes from one sleep stage to another, expressed per hour of TST	-	-	-
	Awakenings >5 min (events)	Number of awakening episodes longer than 5 min	≥4	2–3	≤1
	Cortical arousals (events per hour)	Number of sudden changes in electroencephalographic frequency (alpha, theta, and/or frequency >16 Hz) lasting at least 3 s and with the presence of at least 10 s of prior sleep, expressed per hour of TST	-	-	-
	Limb movements (events per hour)	Number of increases of ≥8 μV in the activity of the tibial electromyogram lasting between 0.5 and 10 s, expressed per hour of TST	-	-	-

Notes: Recommendations are based on the *National Sleep Foundation* (Hirshkowitz et al., 2015b; Ohayon et al., 2017). Red = inappropriate, orange = unclear, green = appropriate, - = no recommendations. *Abbreviations:* N1–N2, non-rapid eye movement (NREM) sleep stages 1–2; SWS, slow-wave sleep; REM, rapid eye movement. Adapted from Mathias et al. (2018).

Nevertheless, due to its multifaceted nature, the unifying definition and measurement of sleep quality remain elusive and abstract concepts, challenging the establishment of “gold standard” criterion measures (McCarter et al., 2022). This challenge is further supported by the weak correlation between physiological measures of sleep (e.g., sleep stages, EEG features) and subjective measures of sleep quality. Only the amount of SWS, better sleep efficiency and fewer nocturnal awakenings seem to be associated with better perceived sleep and fewer somatic complaints, suggesting both SWS and sleep continuity are key determinants of sleep quality (McCarter et al., 2022). SWS remains the most predictive indicator of sleep quality for daytime performance, as its amount has been shown to have moderate correlations with performance levels during tasks requiring vigilance, motor speed, and executive function. REM sleep is also associated with better executive function and motor performance and may play a crucial role in learning and memory consolidation (McCarter et al., 2022). Consequently, these sleep stages represent important considerations when investigating the elite athletes’ sleep.

The weak associations between physiological and perceived measures of sleep quality and performance can be attributed to the fact that traditional sleep staging may be insufficient for estimating sleep quality. Specific neurophysiologic features such as sleep meso- (i.e., EEG spectral features) and micro-structure (i.e., architectural features of slow-oscillations, sleep spindles), along with the estimation of the probability of transition from one sleep stage to another, can provide additional information on sleep quality (Andrillon et al., 2020). Moreover, these general benchmarks of quantity and quality of sleep apply to the general population and may not be adapted to elite athletes. Considering the high metabolic activity associated with training and/or competition, as well as the need to consolidate what was learned from training, it has been suggested that athletes may require a different quantity and/or quality of sleep to ensure more complete recovery (Davenne, 2009; Bird, 2013). Therefore, although these recommendations serve as a good starting point to characterize their sleep, the ideal sleep quantity and quality for athletes still remains to be elucidated (Halson and Juliff, 2017).

3. Overview of sleep in elite athletes: far from dreamlike

It is now well-documented that sleep is essential for athletes’ health, providing important psychological and physiological functions that determine well-being, cognitive and physical performance. Nevertheless, several systematic reviews and/or meta-analyses have issued warnings about the high prevalence of sleep inadequacy among elite athletes (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020). This statement results from numerous studies

describing the sleep of elite athletes using subjective and objective sleep measures by comparing them to the general population or to recommended guidelines.

3.1. Growing interest for athletes' sleep

Sleep research in athletes is relatively recent, showing a substantial growth since the 2010s (Lastella et al., 2020a). It is important to note that investigations on sleep in athletes are mainly conducted on healthy, trained volunteers, and studies including elite athletes are underrepresented (Figure 6). The challenges associated with exposing this population to sleep protocols, which may risk altering their training habits and/or negatively impacting their performance, may explain this trend. An important aspect of the evolution of sleep research in athletes is the transition of athlete sleep monitoring methods from the laboratory to the field (Halson et al., 2019). Recent technological advances in sleep monitoring, such as portable polysomnography devices, research-grade actigraphy or wearable sleep technologies (see from page 80), have facilitated the implementation of protocols, notably *in situ*, in this population, which were previously mainly restricted to clinical sleep research (de Zambotti et al., 2019, 2023). In addition, this trend follows the exponential increase in sport science publications over the last few decades, reflecting the growing interest in developing the importance of physical exercise for the general population, protecting the athletes' health and providing athletes and coaches with scientific evidence in their quest for performance. In this aim, the interest of scientific community and athletic/sporting organisations in understanding the critical role of

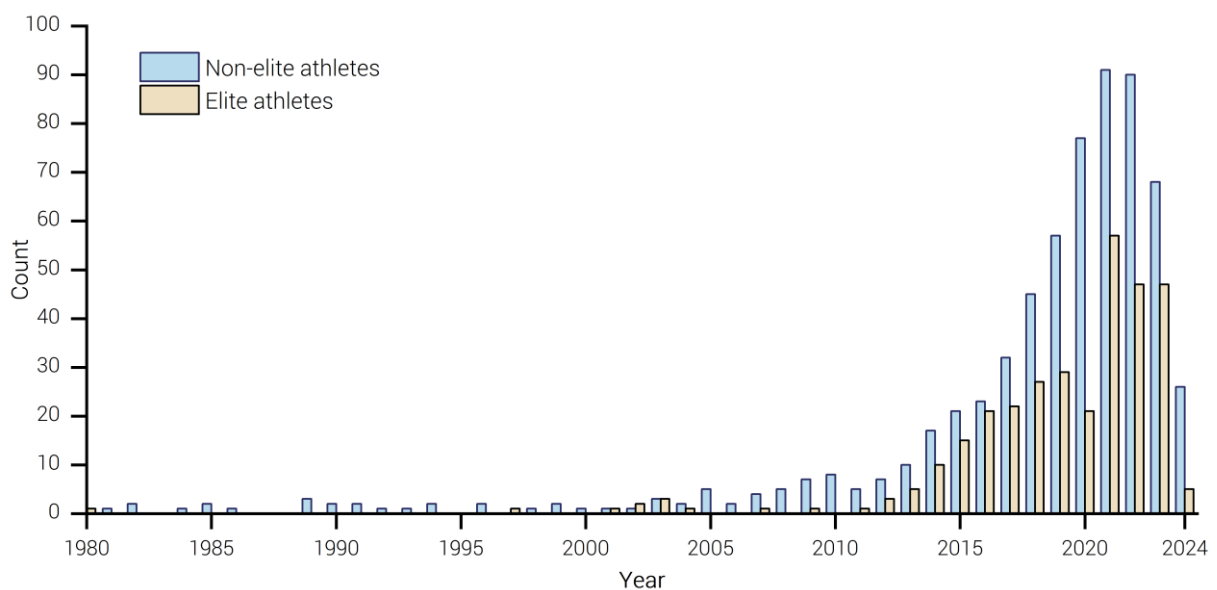


Figure 6. PubMed research and the corresponding number of publications from 1980 to 2024 on sleep in both non-elite and elite athletes, identified using key words adapted from Lastella et al. (2020). *Note:* research updated in March 2024.

sleep for athletes' health, recovery and performance progressively increased (Lastella et al., 2020a).

Venter (2014) asked to 890 athletes to prioritize different recovery strategies, including recovery strategies associated with sociological factors, such as prayer and going out with friends. Sleep was ranked first, followed by blood flow regulation strategies and social activities, represented by time spent with friends and family (Venter, 2014). Nédélec et al. (2013) questioned 32 practitioners in charge of recovery strategies in French professional soccer teams on the strategies they implemented. Similarly, sleep was considered as an effective recovery strategy (95% of practitioners), just like nutrition/hydration (97%) (Nédélec et al., 2013). However, when our laboratory surveyed elite athletes from the French Institute of Sport (532 out of 580, i.e., 92%), 52% were unsatisfied by their amount of sleep, and sleep was identified as the first expected element if they had an extra hour in their day (Burlot et al., 2016). These surveys illustrate a paradox: on the one hand, the importance now accorded to sleep by athletes and their coaches, and on the other hand, a number of elite athletes are concerned and dissatisfied with their sleep. The question here is: "How do elite athletes sleep and what sport-specific factor(s) can contribute to sleep disturbances?". Describing their sleep in real sport settings and identifying the contributory factors that modulate it may further prioritize the research questions that need to be addressed toward strategies to mitigate sleep disturbances (Bishop, 2008).

3.2. Elite athletes' sleep characteristics

3.2.1. Prevalence of sleep complaints

Previous works show that 13 to 78% of elite athletes encounter sleep difficulties during their career (Gupta et al., 2017). This range may be at least partly related to the variety of declarative sleep measures employed. Out of 2067 French elite and junior athletes practicing various sport disciplines at the national or international level, 21.5% declared, over the last 6 months, experiencing sleep difficulties lasting more than 15 days, with the majority having difficulty falling asleep (13.4%) (Schaal et al., 2011). In an exploratory observational 1-year follow-up study in 107 professional ice-hockey players, 22% reported often sleeping problems during the off-season, and this number increased (46%) during the competitive season, resulting in 80% of athletes experiencing sleeping problems either in off- and competitive season (Tuomilehto et al., 2017). Long self-estimated SOL (>30 min) and quality of sleep were identified as the greatest contributors of sleep problems, and were accompanied with a high proportion of players who

perceived unrefreshing sleep. In addition, 36% of players stated that they took sleep medication on at least one night per week to counteract sleep difficulties.

However, the limitation of these studies is they do not use clinically validated sleep questionnaires. The Pittsburgh Sleep Quality Index (PSQI) is the most commonly used questionnaire to assess general perceived sleep quality in both clinical and research settings (see page 88) (Mollayeva et al., 2016), and has been used in a number of studies investigating sleep in athletes (Halsón et al., 2021b). Using this method, the clinical prevalence of “poor sleep quality” (global PSQI score ≥ 5) in elite athletes has been reported to range from 39 to 65% in various sports (Swinbourne et al., 2016; Drew et al., 2018; Aloulou et al., 2021; Halsón et al., 2021b; Suppiah et al., 2021; Anderson et al., 2024). Using a more conservative PSQI threshold (PSQI ≥ 8), the prevalence of highly perceived sleep quality disturbance varies from 22 to 26% (Samuels, 2008; Swinbourne et al., 2016). The high scores in PSQI seem to be mainly influenced by long SOL and poor perceived sleep quality (Halsón et al., 2021b). Nevertheless, although this questionnaire allows for clinical diagnosis of “good” and “poor sleepers”, it has not been validated in elite athletes and concerns were raised regarding the use of this questionnaire in this population, particularly given the reported high scores (Samuels et al., 2016). The Athlete Sleep Screening Questionnaire is a self-reported athlete-specific sleep-screening questionnaire. Using this questionnaire, 25.1% of Canadian national team athletes from 23 sports have been identified to present clinically relevant sleep disturbances that required further clinical sleep assessment (Bender et al., 2018). In 58 Irish international athletes, Biggins et al. (2019) reported that 41% had a mild clinical sleep problem and 16% had a moderate clinical sleep problem. Athletes with a clinically significant sleep problem were more likely to report poor sleep hygiene, more general health complaints, and mood disturbance.

Moreover, daytime sleepiness can be considered as a common consequence of inadequate sleep. The Epworth Sleepiness Scale (ESS) is a clinically questionnaire commonly used to assess sleepiness (Johns, 1991) (see page 89). Given the high prevalence of sleep complaints in elite athletes, it is likely that daytime sleepiness is also prevalent. Indeed, using the ESS, clinical symptoms of daytime sleepiness have been reported in 22–28% of elite athletes (Swinbourne et al., 2016; Drew et al., 2018). Similarly, Aloulou et al. (2021) observed mild to severe excessive daytime sleepiness in French adolescent elite athletes, especially in swimming, cycling, soccer, and handball.

Overall, these findings highlight that, according to subjective methods, sleep complaints and/or sleep dissatisfaction are relatively common among elite athletes.

3.2.2. Prevalence of inadequate sleep quantity and quality

Actigraphy-based sleep

Initial studies on sleep in elite athletes reported that insufficient sleep may be more prevalent in elite athletes than non-athlete population, although only a few studies included non-athlete controls (Walsh et al., 2020). Leeder et al. (2012) compared the sleep of 47 Olympic athletes from various sports (canoeing, diving, rowing, speed skating) during a typical out of competition training phase to the sleep of 20 non-athlete controls, using actigraphy (details on the method of actigraphy are given from page 82). Although there were non-significant differences in TST ($06:55 \pm 00:43$ h vs $07:11 \pm 00:25$ h in controls), elite athletes appeared to sleep poorer compared to gender and age-matched non-athlete controls, characterized by a significantly longer SOL (+13.2 min), elevated WASO (+27 min), and lower sleep efficiency (-8.1%).

Based on a systematic review and meta-analysis, studies in various sports have subsequently confirmed that elite athletes are often unable to achieve the recommended 7–9 h of sleep and 85% of sleep efficiency (**Table 1**) (Gupta et al., 2017; Roberts et al., 2019b). Australian athletes from various individual and team sports ($n = 124$) obtained on average $06:42$ h of sleep per night during a typical out of competition training phase (Lastella et al., 2015b). The same observation has been reported in several sports (Sargent et al., 2014b) such as swimming (Sargent et al., 2014b; Gudmundsdottir, 2020), rugby (Caia et al., 2018; Swinbourne et al., 2018) and badminton (Suppiah et al., 2015). In the few studies that reported average TST >7 h, the prevalence of inadequate sleep remains high. In detail, Knufinke et al. (2018) observed that, while the average sleep efficiency was $88.5 \pm 5.5\%$ in 98 elite athletes, 22.3% of the night had sleep efficiency $\leq 85\%$, accompanied by elevated values for WASO (mean \pm SD: 33 ± 17 min; 7.4% ≥ 60 min). This strengthens the picture of restless sleep in athletes, even when they achieve the recommended sleep duration (Knufinke et al., 2018). Similarly, in elite track-and-field athletes, 18.8% were poor-sleepers, with a sleep efficiency $<85\%$, and 31.3% were short-sleepers, with a TST <7 h. Finally, Whitworth-Turner et al. (2018) showed that youth soccer players engaged in a full-time academy experienced longer SOL, lower sleep efficiency, and higher intra-individual variability for both variables compared to non-athlete controls, despite having longer TST.

The *National Sleep Foundation's* recommendations are useful to identify potential deficiencies in habitual sleep duration at a broad level, but it is important to keep in mind that they may not apply to elite athletes and do not consider the individual differences in sleep need. To date, there is limited information regarding the sleep needs of elite athletes. Sargent et al. (2021) compared the self-assessed sleep need of elite athletes with habitual actigraphy-based sleep duration.

The average subjective sleep need reported by 175 Australian elite athletes in various individual and team sports was 8.3 h. This aligns with the 7–9 h of sleep recommended for nearly 80% of current athletes, providing an initial indication of the sleep duration needed by elite athletes. However, they slept an average of 6.7 h, with 71% not meeting their self-assessed sleep need by 1 h or more, and only 3% obtaining enough sleep to fulfil that need.

Consequently, considering the detrimental effect of sleep loss on athletic performance and the potential performance benefits of sleep extension (see from page 26), we can speculate that a high proportion of athletes may not be obtaining sufficient sleep required for achieving peak performance.

Sleep architecture

Much of the research examining athletes' sleep quantity and/or quality has been derived from actigraphy measurements. Consequently, data are lacking concerning sleep architecture in elite athletes, mainly due to the difficulties in implementing polysomnography in the field and the limited availability of wearable tools for monitoring sleep stages (see page 80). Using one-channel EEG recordings, Knufinke et al. (2018) observed proportions of light sleep (N1 + N2 sleep; $51.4 \pm 8.5\%$) and REM sleep ($27.4 \pm 7.4\%$) that align with the recommendations of the *National Sleep Foundation* and fall within normal healthy ranges (see from page 17). Deep sleep (i.e., SWS; $21.0 \pm 7.7\%$) was higher than the recommended range, i.e., 16–20%. Similar findings were observed in professional cross-country mountain bikers' athletes, whose proportion of SWS reached $21.2\% \pm 3.4$ (Garbellotto et al., 2022). Some evidence suggests that elite athletes often exhibit higher proportions of SWS compared to non-athletes, potentially due to the hypothesized restorative functions of this sleep stage and their elevated need for recovery (Vlahoyiannis et al., 2021). Considering the importance of sleep architecture in defining sleep quality, the very few available data on sleep architecture among elite athletes represents a primary limitation in current studies characterizing athletes' sleep.

3.2.3. Inter-individual variability of sleep in elite athletes

In addition to inadequate sleep, elite athletes' sleep is characterized by high inter-individual variability, especially according to the discipline and gender (Nedelec et al., 2018). It has been observed that athletes from individual sports exhibit shorter TST (-30 min) and lower sleep efficiency (-0.5%) than athletes from team sports (Lastella et al., 2015b). Consistent results were reported in another study involving elite Australian athletes, which found that athletes in

individual sports slept for shorter durations and tended to have earlier sleep onset and wake-up times compared to those in team sports. Notably, rugby union players reported the latest sleep onsets (Sargent et al., 2021). Among Olympic-level track-and-field athletes, Vitale et al. (2023) found that long-distance runners (i.e., race walkers and middle-distance runners) had earlier bedtime (-26 min) and get-up time (-37 min), but lower sleep efficiency (-3%) and longer SOL (+12 min) compared to athletes in short-term disciplines. Moreover, among 128 elite adolescent athletes across 9 different sports, swimmers exhibited increased sleep fragmentation compared to athletes in other sports, highlighting specific sleep disturbance factors inherent to the discipline (Aloulou et al., 2021).

Differences in sleep habits and preferences may also vary depending on the discipline. Chronotype is an individual's circadian phase preference towards "morningness" or "eveningness" (Vitale and Weydahl, 2017). Evidence supports that a high proportion of circadian morning types are involved in sports with early morning training schedules (e.g., swimming, triathlon, endurance running) (Lastella et al., 2016; Vitale et al., 2019a), resulting in earlier bedtime and get-up times. Notably, athletes with evening chronotypes in these sports may experience more sleep difficulties (Bender et al., 2019).

Some individual characteristics may also influence sleep. Sleep patterns can differ between male and female athletes. Studies have reported that, despite similar sleep duration, female elite athletes exhibit higher sleep efficiency (+2.4%) compared to males across various sports (Leeder et al., 2012). In elite track-and-field, female experience longer TST (+15 min) and higher sleep efficiency (+3%), along with shorter SOL (-12 min) and earlier bedtime (-11 min) than male (Vitale et al., 2023b). Similarly, using polysomnography, Silva et al. (2019) found that male athletes had generally poorer sleep pattern, characterized by longer SOL (+15.5 min), higher WASO (+9.9 min), and lower sleep efficiency (-6.3%) compared to female athletes from the Brazilian Olympic team during preparation for the Rio 2016 Olympic Games. Over two consecutive months in junior endurance athletes, female obtained longer TST and higher sleep efficiency than male (Hrozanova et al., 2021b).

Additionally, considering that sleep is influenced by numerous endogenous factors such as sleep requirements, individual responses to pre-competitive stress, and athletes' chronotype, as well as exogenous factors (detailed below), some athletes may be more sensitive to sleep disturbances than others, and may manifest at different periods across the season (Gupta et al., 2017; Nedelec et al., 2018). For instance, Halson et al. (2021a), reported that increased stress state and perceived stress prior to the Rio 2016 Olympic Games were associated with poorer

sleep in elite athletes, particularly increased sleep disturbances and increased daytime dysfunction. Based on a profile approach, Nédélec et al. (2020) showed that athletes with high levels of conscientiousness and neuroticism personality profile reported worse sleep quality and higher levels of insomnia in response to stress than those with more stress-adaptive profiles. Indeed, poor sleep is associated with perseverative cognition (e.g., worry or anxiety about performance or success) and hyperarousal (increased neurobiological and psychological stress during sleep) (Hrozanova et al., 2021a). In addition, sleep habits may vary depending on the athlete's chronotype and sleep need. Very high inter-individual differences in elite athletes have been reported in sleep needs (Sargent et al., 2021). In summary, the high inter- and intra-individual variability in sleep characteristics underscores the necessity for individualized sleep strategies and interventions to promote sleep, taking into account the individual responses of elite athletes (Fullagar and Bartlett, 2016).

3.3. Contributing factors for sleep inadequacy

Despite the importance of sleep for athletes, practicing sport at the highest level imposes a significant challenge to elite athlete's sleep (Nedelec et al., 2018). They face several sporting and societal/environmental factors that may contribute to the high prevalence of sleep complaints and inadequate sleep (Savis, 1994; Walsh et al., 2020; de Blasiis et al., 2021; Cook and Charest, 2023). Societal/environmental factors may refer to pervasive use of smartphones and electronic devices, social media, family or work/study commitments, nutritional habits, seasonal phase or sleep environment (**Figure 7**). While it is difficult to distinguish the influence of sport-related from societal/environmental stressors, it is important to recognize that both can affect athletes' sleep (Halsen, 2016). Here, we will focus on the three main sport-specific factors identified as contributing to sleep disturbance in elite athletes: travel, competition, and training (**Figure 7**) (Gupta et al., 2017; Nedelec et al., 2018; Walsh et al., 2020; Cook and Charest, 2023).

3.3.1. Travel, competition and unfamiliar environments

Elite athletes are often required to travel short distances (<3 h) and long distances (>3 h) to their training or competition locations, which may involve crossing of numerous time zones. This creates a unique challenge for sleep, primarily due to the disruption of the circadian system (i.e., jet lag) and travel fatigue, which can result in a range of symptoms including daytime fatigue,

decreased concentration and alertness, sleep disruption, and gastrointestinal disturbances (Waterhouse et al., 2007; Janse van Rensburg et al., 2021).

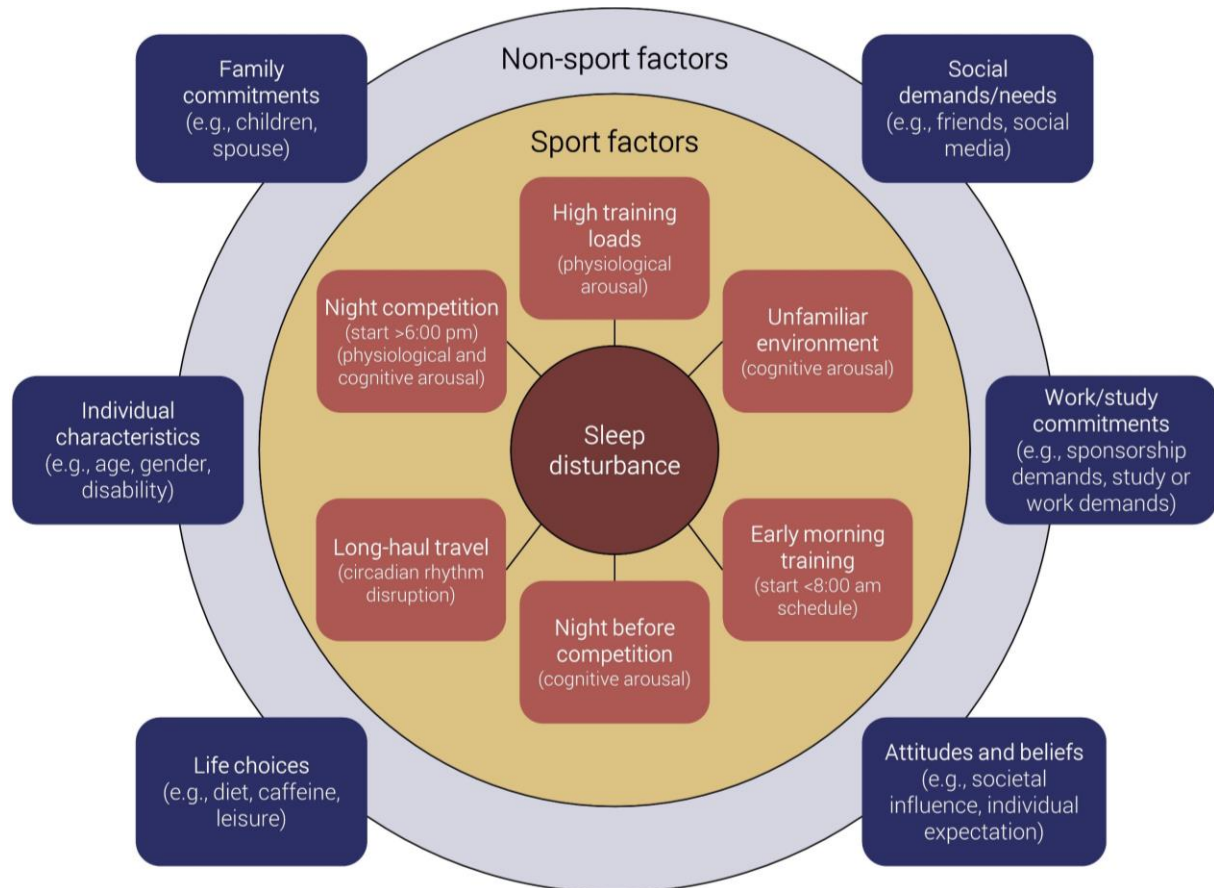


Figure 7. Potential non-sport (i.e., societal/environmental; blue) and sport-specific (red) factors contributing to sleep disturbances in elite athletes. Adapted from Walsh et al. (2020).

With regard to competition, the timing of competition, the problematic congestion of competition schedules, and the psychological distress associated with competition have the potential to negatively impact sleep (Cook and Charest, 2023). Poor sleep is common in elite athletes prior to major competitions, often due to nervousness and pre-competition thoughts (Juliff et al., 2015; Andrade et al., 2021; Sargent et al., 2022; Sim et al., 2024). The night of competition is also affected and typically results in later bedtime and a reduction in TIB, TST and sleep efficiency (Nédélec et al., 2019; Roberts et al., 2019b; Sanders et al., 2024). For example, sympathetic hyperactivity (Kivlighan and Granger, 2006), elevated heart rate and core body temperature (Aloulou et al., 2020a), muscle pain/soreness (Hainline et al., 2017a), and increased circulating cortisol (O'Donnell et al., 2018b) may persist after competition and can contribute to affect sleep (Fullagar et al., 2023).

Elite athletes are also frequently exposed to unfamiliar sleep environments, such as hotels or base camps, during competitions (Bishop, 2004) or training camps (Pitchford et al., 2017).

Changes in sleep location has been identified as an important factor likely to affect sleep (Nedelec et al., 2018). Approximately 28% of elite German athletes reported that an unfamiliar environment disrupted their sleep on the night before competition (Erlacher et al., 2011). In an observational crossover trial, Pitchford et al. (2017) compared the sleep characteristics of 19 elite Australian Rules football players during an 8-day preseason home training period to an 8-day training camp period with a similar training load and schedule. The change in sleep environment during the camp resulted in a longer TIB (+34 min) due to earlier bedtime (-45 min) with no change in wake-up time and sleep duration. However, there was an increase in WASO (-27.6 min), which translated into a lower sleep efficiency (-6%) compared to being at home. Similarly, a reduction in TST (-85 min), TIB (-53 min), and sleep efficiency (-8%) were reduced during a 2-week high-intensity training camp compared with home in 31 professional rugby league players (Thornton et al., 2017). Impaired sleep in unfamiliar environments may result from the athlete's lack of adaptation to the environment, known as "first-night effect" (Suetsugi et al., 2007).

Nevertheless, numerous confounding factors contributing to sleep disturbances may occur to unfamiliar sleep environments such as training camps, including noises both inside and outside the room (Öhrström and Skånberg, 2004), sleeping on unaccustomed or poor-quality bedding (Jacobson et al., 2009; Antonino Vitale et al., 2019), sharing a bedroom with a teammate (Costa et al., 2023b), altitude/hypoxia or heat (Roberts et al., 2019b; Hrozanova et al., 2021c; Vitale et al., 2022). Training camps are also specific periods during which elite athletes undergo repeated strenuous training sessions, exposing them to high training loads and altered schedules (Nedelec et al., 2018). While we are aware of their involvement, the contribution of these confounding factors is currently poorly understood in the context of elite sport.

3.3.2. Training schedules and load

Factors inherent in training that can disrupt sleep can include the timing of training and the extent of training load (Walsh et al., 2020). On the one hand, some studies have demonstrated that morning schedule strongly determines the prior sleep duration across various sports (Sargent et al., 2014a, 2014b; Steenekamp et al., 2021). For instance, in some sports like swimming, triathlon or endurance running, training sessions are frequently scheduled early in the morning. Sargent et al. (2014a) monitored the sleep of 7 elite Australian swimmers over 14 days, during which training started at 6:00 for 10 days. The swimmers obtained on average of 5.4 h of sleep, well below the recommended 7–9 h. In another study from various sports,

Sargent et al., (2014b) observed that for every hour that the start time of training was advanced from 8:00 to 5:00, TST was reduced by 6 min (7 am), 48 min (6 am) and 102 min (5 am). Similar sleeping patterns have been reported in New Zealand adolescent athletes when they are exposed to early morning training (Steenekamp et al., 2021). Early morning training sessions may also result in increased intra-individual TST-variability (Gudmundsdottir, 2020). A consistent finding among these studies was that athletes attempted to compensate for waking up earlier on training days by adjusting their bedtime earlier. However, this adjustment was often insufficient considering the training start time, possibly due to social or family commitments in the evening or the inability to fall asleep earlier (Sargent et al., 2014b). These studies emphasize that the planning of the day and the timing of training sessions can influence sleep duration. This consideration is particularly important during training camps, where athletes are often subjected to congested schedules (Nedelec et al., 2018).

On the other hand, a growing body of studies suggests that the sleep of elite athletes may be sensitive to manipulated training loads. Specifically, excessive training loads and/or maladaptive response to training (overreaching, overload) are usually accompanied by sleep complaints and inadequate sleep (Meeusen et al., 2013; Hauswirth et al., 2014; Roberts et al., 2019b). This raises a dichotomy, as regular physical exercise is recommended by the World Health Organization for improving both the quantity and quality of sleep in the general population (Bull et al., 2020). The potential detrimental effect of high training load on athletes' sleep is not yet fully understood, particularly concerning its impact on sleep quality and architecture, as well as its role in among other sport-specific factors contributing for sleep disturbances. The following section will focus on training load as a potential sleep modulator in elite athletes and examine the extent to which it has been identified as a contributing risk factor for sleep disturbances in this population.

Key Points

- Sleep is regulated by the interaction between a **homeostatic** (i.e., prior accumulated wakefulness and energy demands) and **circadian process** (i.e., endogenous pacemaker driven by *zeitgebers* such as light, physical exercise and temperature).
- The **internal structure of sleep** provides important **restorative effects** on the immune and the endocrine systems, **recovery** of the nervous and metabolic cost **imposed by the waking state**, and **cognitive development**. These functions of sleep underline its **importance for the athletes' health and their performance**.
- **Sleep loss** may affect **athletic performance**, especially in disciplines requiring high cognitive and physically reliance with repeated exercise bouts.
- A growing body of evidence suggests a **high prevalence of sleep complaints and inadequate sleep quantity and/or quality in elite athlete populations**, characterized by usual sleep duration <7 h, difficulties in maintaining sleep, sleep dissatisfaction, unrefreshing sleep, long sleep onset latencies, daytime sleepiness and daytime fatigue.
- Numerous potential **societal/environmental and sport-specific factors likely to disturb elite athletes' sleep** have been identified, related to **competition** (e.g., night before and of competition), **travel** (e.g., jet-lag, travel fatigue) and **unfamiliar sleep environments** (e.g., hotel, base camp), and **training** (e.g., timing of training, high training loads).
- The potential detrimental effect of **high training load on athletes' sleep is not yet fully understood** and requires further investigation regarding its impact on **sleep quality**, as well as its **contribution among other sport-specific factors** that may disturb sleep.

Part 2 – Training load: a potential sleep modulator in elite athletes

The previous section outlined the importance of sleep for athletes' health and performance and provided evidence of a high prevalence of inadequate quantity and/or quality of sleep among elite athletes. High training load has been identified as a sport-specific factor likely to worsen athletes' sleep. This section presents 1) the concept of training load and the various responses to training; and 2) the potential effects of training load on athletes' sleep.

1. Training concept and potential responses to training

1.1. Theoretical training concept

The primary goal of training is to develop specific sport skills to improve performance (Impellizzeri et al., 2019; French and Torres Ronda, 2022). During a training session, athletes experience physiological and psychological stress, disrupting homeostasis across various cardiovascular, metabolic, hormonal, neuromuscular, and cognitive systems. These alterations often lead to motor or cognitive fatigue (*negative* training effects), characterized by a transient decline in performance and an increased perception of tiredness, weakness, weariness, or exhaustion, known as *acute fatigue* (Skorski et al., 2019; Behrens et al., 2023). In response to this stress, changes in homeostasis provide a stimulus for the body that triggers physiological and biochemical adaptations (*positive* training effects). With adequate rest following the training session, the body may experience an increase in capacity or compensation (**Figure 8**). These adaptations occur during the phase of recovery and enable the body to adapt its capacities to cope with the subsequent exposure to stress (Cunanan et al., 2018).

It is possible to distinguish the *acute* (negative/positive) and *chronic* (negative/positive) effects of training (Jeffries et al., 2022) (**Figure 9**). For example, exercise-induced muscle damage from a single bout of exercise may initially reduce force-generating capacity (*negative acute* effect). It can subsequently stimulate the activation of various cell types to facilitate post-exercise muscle tissue regeneration, conferring a protective effect against potential subsequent strenuous exercise (*positive acute* effect) (Lavender and Nosaka, 2008; Peake et al., 2017). *Chronic* training effects can be considered as the more prolonged effects of training over weeks or months, resulting from the cumulative effects of each short-term exercise bout. From a physiological perspective, this may result in musculoskeletal adaptations such as increased muscle volume and mitochondrial density or modified muscle contractile protein composition

(*positive chronic* effect) (Coffey and Hawley, 2007). Conversely, a prolonged maladaptive response to training, such as prolonged fatigue may have a *negative chronic* effect on performance (detailed from page 49) (Meeusen et al., 2013). The training effects can be assessed using several methods related to performance (e.g., graded-exercise test), physiological measures (e.g., maximal oxygen consumption [$\dot{V}O_{2peak}$]), and subjective measures such as validated questionnaires or scales (e.g., the Profile of Mood States, the Daily Analysis of Life Demands for Athletes [DALDA], and the Total Recovery Scale) (Halson, 2014a; Jeffries et al., 2022).

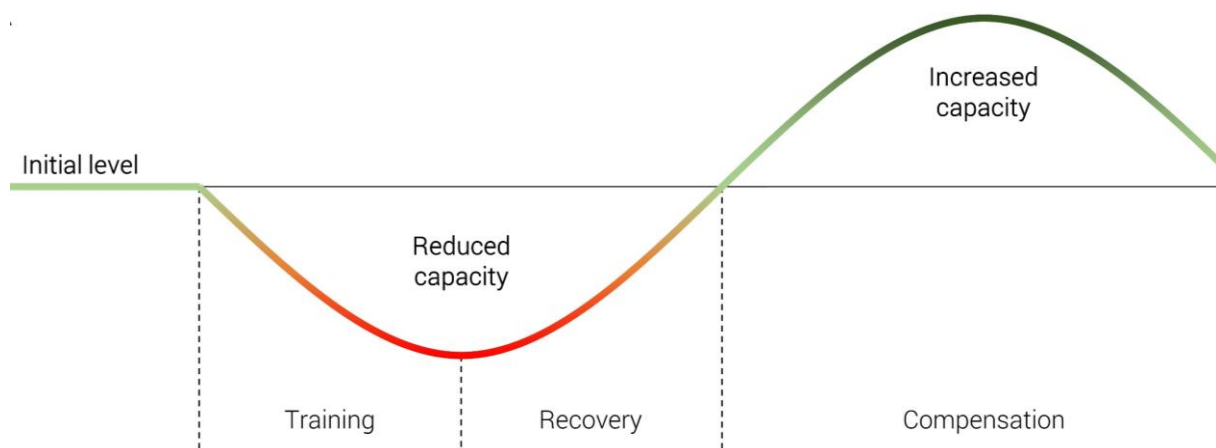


Figure 8. Theoretical response to training. Adapted from Hausswirth and Mujika (2013), Meeusen et al. (2013) and Soligard et al. (2016).

To describe these training effects and the relationship with performance, Banister et al. (1975) has proposed the “fitness-fatigue” model. This model considers athletic performance as the outcome of the difference between negative responses (i.e., increased *fatigue*) and positive responses (i.e., increased *fitness* or adaptation of the body) to training (Morton et al., 1990; Morton, 1997). Therefore, training effects and performance output can be grasped by considering the balance between the adaptative response to training and the training stimulus (**Figure 9**). First, the physiological responses to training are directly dependent on the body's capacity to recover from a training session, emphasising once again the importance of restorative sleep after training. Second, the training effects are dependent on the training stimulus (i.e., prescribed training). This includes the nature, intensity, and duration of the training, which have been proposed as input variables to define the training load concept (see below) (Impellizzeri et al., 2019). Third, individual characteristics (e.g., genetics, anthropometric, psychological traits and states, health, training history) and contextual factors (e.g., environmental, social, cultural conditions) in which the exercise is performed also facilitates or constrains the components of the training process (Kenttä and Hassmén, 1998; Impellizzeri et

al., 2019). Finally, it is important to note that these relationships are bidirectional (Jeffries et al., 2022). Indeed, a positive training response (e.g., increased adaptation of the body) influences individual characteristics by improving the body's ability to adapt to training. This enables more effective recovery from subsequent stress (Hauswirth and Mujika, 2013; Cunanan et al., 2018). Conversely, a negative training response (e.g., increased fatigue) can subsequently influence the body's response to training in the following training session (Jeffries et al., 2022).

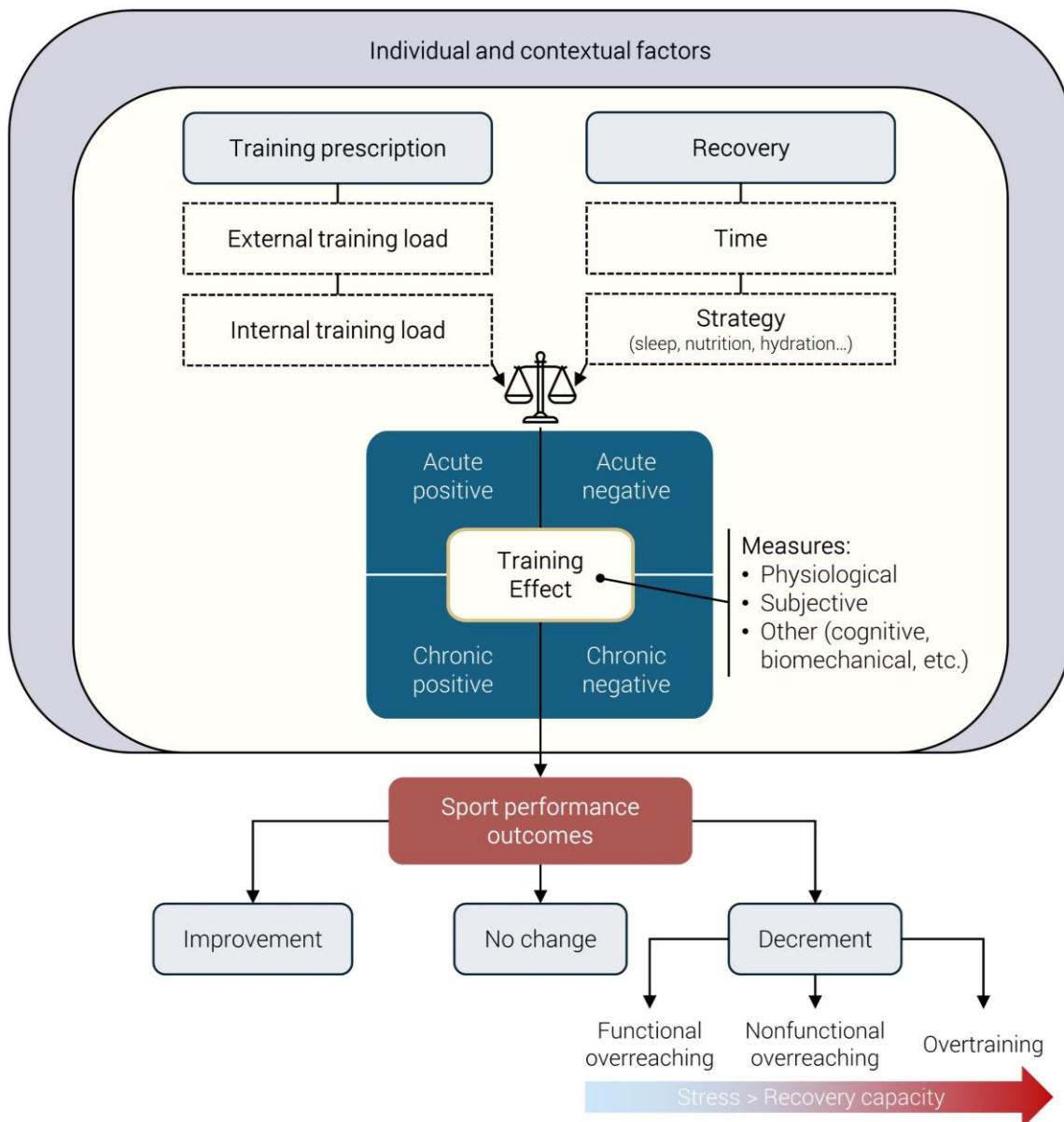


Figure 9. Theoretical framework of training and different responses to training. Adapted from Jeffries et al. (2022).

1.2. Training load concept and quantification

As described previously, the psychophysiological stress imposed by training is a critical determinant of athletes' training responses. Prescribing training aims to manipulate the

organization, nature, quality and quantity of exercise, to elicit the desired psychophysiological and musculoskeletal adaptations. Training load has been described as a high-order multidimensional construct reflecting the amount of physical training that is actually done and experienced by the athletes, including external and internal subdimensions (Impellizzeri et al., 2022; Jeffries et al., 2022).

1.2.1. External training load

External training load is defined as the physical/mental effort performed by the athlete during the training. It can be observed and measured by quantifying what the athlete does. Measures of external training load are specific to the nature of training and the discipline (Impellizzeri et al., 2019). These measures can include, for example, training duration, distance covered, power output, velocity, acceleration, neuromuscular function (e.g., jump and sprint tests, peak force), movement repetition counts, or training frequency/time (Halson, 2014a; Soligard et al., 2016). In the last decades, there has been a proliferation of more sophisticated (micro)technology to detail information about external load (Cardinale and Varley, 2017). For example, the use of portable micro-electromechanical systems on the field, which contain a global positioning system (GPS) coupled with integrated microsensor technology (accelerometer, magnetometer, with or without gyroscope sensors), has become a growing measurement tool in team sports such as rugby union for describing individual time-motion activity (Naughton et al., 2023). These tools allow for easy quantification of the frequency, intensity, and duration of locomotor (velocity, distance covered, and high-intensity efforts, i.e., acceleration/deceleration, of the body centre of mass) and collision actions. The accuracy and reliability of these tools to measure these metrics have been demonstrated as “acceptable” in team sports (Varley et al., 2012; Rampinini et al., 2015; Scott et al., 2016; MacLeod et al., 2018).

1.2.2. Internal training load

Internal training load encompasses all the psychophysiological responses the body initiates to cope with the demands imposed by the external load (Kellmann et al., 2018; Impellizzeri et al., 2019). It can be described using psychological measures such as rating of perceived exertion (RPE) and physiological measures like heart rate, $\dot{V}O_2$ or lactate concentration. The RPE scale, developed by Borg (1982), whose one version is a 1–10 scale (CR-10) assesses the psychophysiological stress perceived by the athlete during exercise (Borg et al., 1985). Subsequently, Foster et al. (2001) proposed to multiply RPE of the session (sRPE) by the

duration of a training session (in minutes) to provide a measure of global training load ($sRPE \times$ duration). While $sRPE \times$ duration offers the advantage of providing a global indicator of training load, it does not provide any indication of the more localized physiological effects of training load. However, studies have validated its use in the field as a valid, reliable and very useful indicator of training load via its strong correlation with other internal load measures such as heart rate and external load variables like distance covered, high-speed distance in various individual and team sports, and the number and the frequency of collisions in team-based contact sports (Haddad et al., 2017).

It is important to note that the external and internal subdimensions of training load may vary based on individual or contextual factors (**Figure 9**). For the same external load, the corresponding internal load varies between the athletes due to multiple factors such as training and recovery status, nutrition, health, psychological status, and genetics, which also leads to inter-individual variability in training adaptive processes (Mann et al., 2014; Vellers et al., 2018; Impellizzeri et al., 2019). For example, high training loads have been associated with an increased risk of illness in non-elite athletes, whereas this negative relationship seems to be less evident in elite athletes who demonstrate greater adaptive and recovery capacity to high training load (Schwellnus et al., 2016). Similarly, an athlete's characteristics can change throughout a season and a career, leading to variations in internal load over time despite consistent external load (intra-individual variability). This highlights the importance of manipulating training load on an individual approach to achieve the desired adaptations, and regularly monitoring it to assess an athlete's response to training (Halson, 2014a).

1.3. Training periodization and responses to training

1.3.1. Training periodization

When the body undergoes repeated bout of psychophysiological stress, long-term training adaptations may occur. One of the associated challenges for coaches and athletes is to design and follow a training program that induces optimal training adaptations, culminating in peak performance during major competitions. Training periodization seeks to plan a short- and long-term training program by manipulating training load (nature, volume, intensity, frequency) and recovery (time, modalities) (Mujika et al., 2018). The typical model to achieve peak training adaptations and performance is to skilfully alternate phases of increase (overload, i.e., stress >

recovery) and phases of decrease (taper, i.e., recovery > stress) in training load (Casado et al., 2022).

Overload aims to temporarily increase psychophysiological stress (from days to a few weeks, *chronic negative* effects) to maximize mild- to long-term adaptations to training. Conversely, the following taper period aims to reduce the detrimental impact of training by reducing the training load and removing residual fatigue, while the biochemical and physiological adaptations elicited during the previous overload are strengthened by appropriate recovery (*chronic positive* effects) (Mujika et al., 2004, 2018; Aubry et al., 2014). In their meta-analysis, Bosquet et al. (2007) established that the most efficient tapering strategy leading for significant performance improvements involved a gradual 41–60% reduction in training volume over a 2-week period, without altering training intensity or frequency. On average, this tapering approach resulted in a substantial performance gain of ~2.0%, which is considerable in the context of high-performance sport (Joyce and Lewindon, 2022). Indeed, the smallest performance improvement considered to have a substantial impact on the likelihood of winning a medal in top competition (i.e., smallest-worthwhile change– SWC) has been estimated to ~1/3 of the within-athlete variation in performance, as measured by the coefficient of variation between competitions (Hopkins et al., 1999). This corresponds to ~0.5–1% changes in performance for middle- and long-distance running, i.e., 1500 m to marathon (Hopkins and Hewson, 2001).

Individual sport athletes, like endurance runners, often achieve peak performance through months of consistent training. Therefore, a periodization strategy involving overload and taper phases is considered relevant and commonly used among these athletes (Mujika et al., 2018; Casado et al., 2022). However, team sports such as rugby union can face substantial challenges in implementing such periodization, especially in elite or professional context. Factors such as extended periods of competition, frequent matches (sometimes more than once a week over several weeks), and frequent travel hinder medium- to long-term periodization (Nédélec et al., 2012). In this context, elite/professional sporting teams often undergo high-intensity preseason training camps involving high training loads to maximize positive physiological adaptations before the competitive season begins (Buchheit et al., 2013; Thornton et al., 2017).

1.3.2. Different responses to training

Based on aforementioned training periodization, positive long-term training effects are generally achieved when high training loads are prescribed before an appropriate taper phase. Consequently, athletes may tend to balance on the thin line between doing right and doing too

much (Kellmann, 2010; Meeusen et al., 2013; Roete et al., 2021). When overloaded, athletes may experience acute perceived fatigue and/or decline in performance. When the balance between overload and adequate recovery is disrupted, leading to the accumulation of fatigue without the capacity for athlete to recover from a training stimulus, maladaptive responses to training can occur. Athletes can develop a continuum of prolonged fatigue, which manifests in various states, including functional overreaching, non-functional overreaching, or overtraining syndrome (**Figure 9**) (Halson and Jeukendrup, 2004; Meeusen et al., 2013).

A functional overreaching is defined as a short-term decrement in performance capacities with or without related physiological and psychological signs and symptoms of maladaptation, and the restoration of performance may occur from about 5 to 7 days. This state of fatigue is sometimes desired to reach a supercompensation through a long period of recovery (Mujika, 2010). Nevertheless, some evidence shows that greater gains in performance can be achieved when overload is followed by a taper phase without the presence of functional overreaching, supporting the interest of overloading prior to tapering as long as fatigue is not too high, i.e., only acute fatigue (Aubry et al., 2014; Bretonneau et al., 2024). At this fatigue state, a non-functional overreaching state may occur when overload continues, or recovery is inadequate. Performance decrements, along with psychological (e.g., decreased vigour, increased fatigue) and hormonal disturbances can persist for extended periods, and their restoration may require several weeks to months. Following this, an extreme fatigue state known as overtraining syndrome may occur, distinguished from non-functional overtraining by a much longer recovery period (several months to more than a year to recover) (Halson and Jeukendrup, 2004; Meeusen et al., 2013).

Several confounding factors contributing to overreaching/overtraining have been identified, including inadequate nutrition, increased risk of illness, and psychological disturbances. Additionally, these maladaptive responses to training are often accompanied by sleep disorders (Kellmann, 2010; Meeusen et al., 2013; Schweltnus et al., 2016). The following section focuses on the impact of training load on athletes' sleep.

2. Training load as a sleep modulator in athletes

2.1. Is training beneficial or detrimental for sleep?

In the section Sleep-wake regulation systems (from page 20), we underlined that sleep propensity and sleep architecture (e.g., SWS and SWA) depend, in part, on the prior accumulated wakefulness and the restorative needs of the body. It is thus tacitly assumed that the quality of

sleep relates, at least in part, to the quality of wakefulness (Dijk and Landolt, 2019). In this respect, physical exercise (i.e., planned, structured, non-competitive physical activity defined by its frequency, intensity, and duration) is recommended by academic sleep associations as a low-cost, easily administered, and non-pharmacologic intervention for improving sleep (Passos et al., 2012; Yang et al., 2012). It has been associated with a reduction in the prevalence of insomnia symptoms and a higher likelihood of sleeping within the recommended 7–9 hours (Bjornsdottir et al., 2024).

Several systematic reviews and meta-analysis have demonstrated the benefits of a single bout of physical exercise on TST, SOL, sleep efficiency, WASO and SWS in healthy individuals, and that regular exercise induces more salient effects (Kubitz et al., 1996; Youngstedt et al., 1997; Driver and Taylor, 2000; Youngstedt, 2005; Uchida et al., 2012; Kredlow et al., 2015). For instance, Park et al. (2021) showed significantly increased delta power in SWS together with increased SWS stability following 60 min of vigorous exercise compared with no exercise in healthy young men. Among an adolescent population, Master et al. (2019) observed a positive dose-response relationship between physical exercise and actigraphy-based sleep. Interestingly, engaging in more daily moderate-to-vigorous physical activity than an individual's average was associated with earlier sleep onset, longer TST, and higher sleep efficiency. Conversely, days with more sedentary behaviour than an individual's average were linked to delayed sleep onset and wake-up time, as well as shorter TST (Master et al., 2019).

Various pathways have been proposed to explain how exercise could promote sleep, although the underlying mechanisms are still uncertain (**Figure 10**) (Youngstedt, 2005; Buman and King, 2010; Chennaoui et al., 2015; Kredlow et al., 2015). The acute and regular rise in body temperature induced by exercise has been proposed as a hypothesis for how sleep propensity could be favoured, as the down-regulation of body temperature following exercise may contribute to this effect (Youngstedt, 2005). The associated heat loss may promote sleep by activating interrelated sleep-promoting and thermoregulation brain regions (detailed from page 66). In addition, acute and regular exercise induce immune, endocrine and metabolic changes, such as marked increase in pro-inflammatory cytokines concentration, growth hormone, prolactin and melatonin secretion, and decrease of plasma cortisol, all likely to favour sleep (see from page 23) (Chennaoui et al., 2015). Regular exercise may also promote sleep through increased cardiac vagal modulation, characterized by an increase in parasympathetic and decrease in sympathetic activities during sleep (Burgess et al., 1997). This vagal modulation could enhance parasympathetic control, thereby promoting sleep (Uchida et al., 2012;

Chennaoui et al., 2015). Additionally, it has been suggested that regular physical activity, especially outdoors and in sunlight exposure, may promote sleep by acting as a *zeitgeber* for the SCN, contributing to the regulation of sleep-wake rhythms (see from page 20) (Youngstedt, 2005). Finally, it is well-established that regular exercise has beneficial effects on mood and anxiety, with antidepressant effects (Youngstedt, 2005). These improvements in mood are linked to elevated levels of brain-derived neurotrophic factors induced by exercise, which may indirectly enhance sleep quality (Uchida et al., 2012; Chennaoui et al., 2015). The positive impact of exercise on sleep, particularly through mood enhancement, can promote the adherence to regular physical activity (Chennaoui et al., 2015).

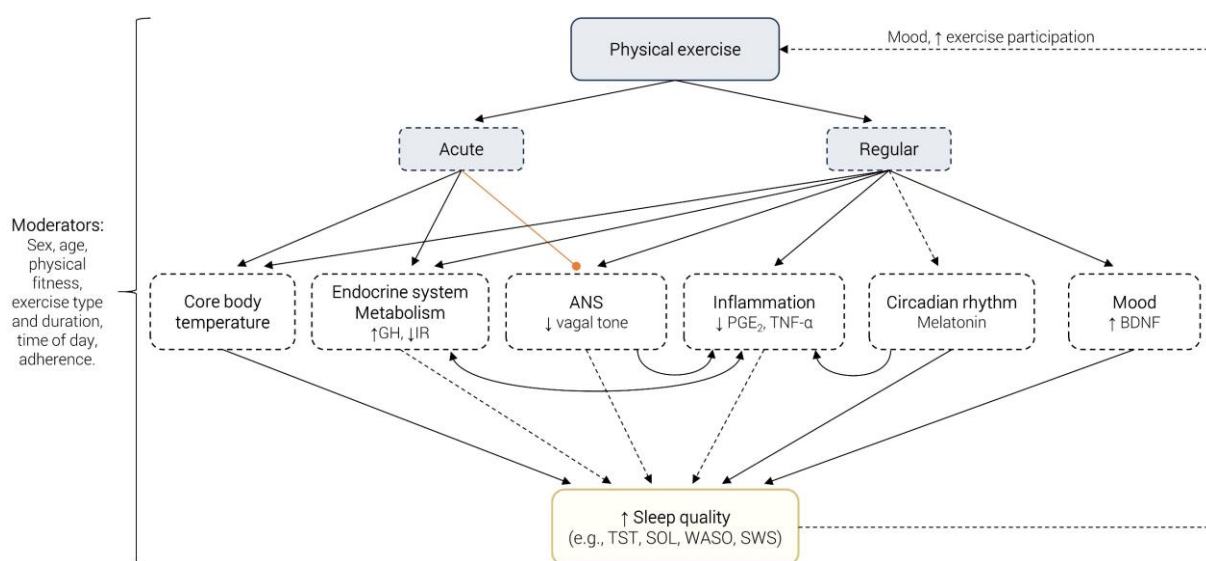


Figure 10. Potential effects of acute and regular exercise on sleep. *Notes:* solid arrow, very likely direct link; dotted arrow, likely direct link; orange line, inhibiting action. *Abbreviations:* GH, growth hormone; IR, insulin resistance; ANS, autonomic nervous system; PGE₂, prostaglandin E₂; TNF-α, tumor necrosis factor alpha; BDNF, brain-derived neurotrophic factor; TST, total sleep time; SOL, sleep onset latency; WASO, wake after sleep onset; SWS, slow-wave sleep. Adapted from Uchida et al. (2012), Chennaoui et al. (2015) and Kredlow et al. (2015).

Nevertheless, unlike their non-athlete peers, elite athletes follow rigorous, well-planned training programs, including intensified training periods. Given the high volume and/or intensity of training, if the dose-response relationship between physical exercise and sleep is linear, we might expect elite athletes to have outstanding sleep. However, high training loads have been identified as a potential sport-specific risk factor for sleep disturbances in athletes (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020). This raises a dichotomy regarding the benefits of physical exercise on healthy individuals' sleep. In this regard, Falck et al. (2021) proposed an inverted U-shaped dose-response relationship between training load and sleep. This model suggests that increased training load promotes sleep until a "tipping point", beyond which

maladaptive responses to training occur, leading to disturbed sleep (**Figure 11**). However, this model is currently theoretical and requires experimental validation among varied population (elite vs non-elite athletes) across different rates of training load, specific physical demands, and traumatic component, as well as various contexts to encompass confounding factors likely to affect sleep.

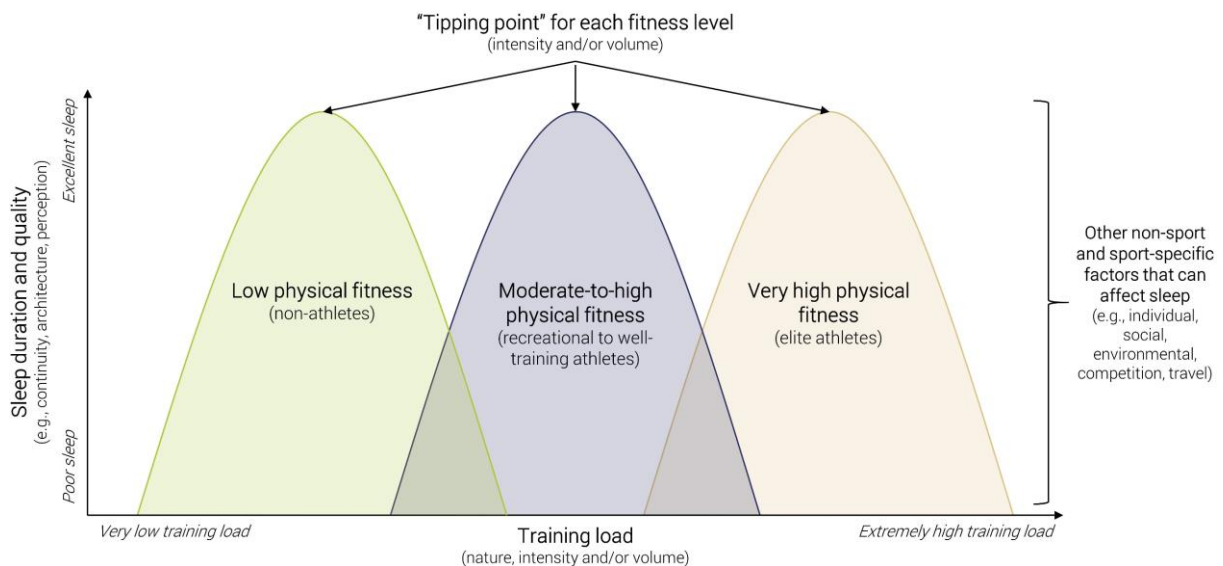


Figure 11. The theoretical inverted U-shaped model, suggesting a dose-response relationship between training load (nature, volume and/or intensity) and sleep (duration and/or quality) proposed by Falck et al. (2021). This model suggests a positive effect of physical exercise on sleep until a “tipping point”, beyond which maladaptive responses to training occur (overreaching), and sleep is disturbed. This “tipping point” for when maladaptive responses to training occurs is dependent of the athlete’s physical fitness for an imposed training load. Other non-sport and sport-specific factors may impact the sleep-training load relationship, such as individual attributes, social/environmental factors, competition and travel (see from page 39). Adapted from Falck et al. (2021).

2.2. Impact of training load on athletes’ sleep

2.2.1. Overview of observational and interventional studies

In situ observational studies

Some observational studies, i.e., *in situ* monitoring of sleep and training load, report changes in athletes’ sleep in response to increased training load (**Table 2**). In 11 young elite female basketball players undergoing a 14-day training camp, it was observed that days with the highest training load resulted in subsequent impairments in sleep efficiency, inferred from actigraphy measurements, compared with days with a moderate (high vs moderate, training load: +106%; sleep efficiency: -0.7%) and low training load (high vs low, training load: +333%; sleep efficiency: -2.7%) (Lastella et al., 2020b). In addition, a reduction in TST was observed in rugby union players during a week of high training load compared with a week of low training

load (high *vs* low, training load: +50%; TST = -33 min) (Conlan et al., 2022). Similarly, Kölling et al. (2016) monitored sleep of 55 young rowers during a 4-week training camp. A reduction in TST was reported during the first two weeks when the training load was particularly high (up to 4 training sessions per day), while a reduction in training frequency during the last two weeks was accompanied by an increase in TST. Impairments in TST and sleep efficiency were also observed during high-intensified training camp compared with usual training regime home periods in rugby league (Thornton et al., 2017) and water-polo (Botonis and Toubekis, 2023).

Several observational studies have also reported sleep disturbances occurring in response to increases in day-to-day training load (**Table 2**). Among 128 high-level adolescent athletes from various sports, the relationship between daily training load, assessed using sRPE \times duration, and subsequent sleep (monitored via actigraphy) was investigated (Aloulou et al., 2021). Each additional 100 A.U. in sRPE \times duration resulted in subsequent earlier bedtime (-2.5 min), shorter TST (-1.2 min), poorer perceived sleep quality (-0.1 A.U.) and higher sleep fragmentation index (+0.1 A.U.). These findings are in agreement with alterations in the subjective and actigraphy-based sleep quantity, efficiency, WASO and/or SOL in response to higher day-to-day sRPE \times duration, as reported in gymnasts (Dumortier et al., 2018), soccer (Costa et al., 2021; Figueiredo et al., 2021) and various sports (de Blasiis et al., 2021). Interestingly, it was observed that the more a rugby league player's sRPE \times duration differed (whether increasing or decreasing) from the average individual sRPE \times duration, the more TST decreased (Pitchford et al., 2017). This supports the hypothesis that sleep is disturbed when there is an unusual increase in training load.

Interventional studies

The detrimental effects of elevated training loads on sleep have also been confirmed in some interventional studies that experimentally manipulated training load (**Table 2**). Lastella et al. (2015) evaluated the impact of a 3-week simulated Grand Tour on sleep compared with a 1-week low-moderate training period (+104% in training volume) in highly-trained cyclists. A progressive decline in TST and sleep efficiency was observed during the Grand Tour compared with baseline, alongside impairments in mood and general well-being. Similar findings were reported in well-trained cyclists undergoing a 9-day high intensified training period compared with a baseline training period (+153% and +146% in averaged training volume and intensity), who showed a progressive reduction in sleep efficiency and increase in sleep fragmentation index (Killer et al., 2017). In elite synchronized swimmers, a 2-week intensified training period

(+25% in sRPE × duration) resulted in decreases in TST (-21 ± 7 min), sleep efficiency ($-1.9 \pm 0.8\%$), and an increase in SOL ($+11 \pm 5$ min), compared with a normal training week (Schaal et al., 2015). Finally, Hausswirth et al. (2014) investigated whether a 3-week overload period (+30% in training volume) may disturb sleep in well-trained triathletes. Triathletes were diagnosed as either functionally overreached, characterized by reduced performance and high perceived fatigue following the overload compared with baseline, or acutely fatigued, where performance either maintained or increased after the overload, with or without increased perceived fatigue. Results revealed an impairment in TST ($-7.9 \pm 6.7\%$) and sleep efficiency ($-1.6 \pm 0.7\%$) in functionally overreached triathletes during the overload compared with baseline, which was reversed during a subsequent taper phase. Overall, these findings suggest that athletes may experience alterations in sleep quantity and/or quality when exposed to excessive, accumulated and/or unusual changes in training load.

In contrast, it is important to note that conflicting results can be found elsewhere (**Table 2**). Some studies observed improvements in sleep quantity and/or quality (Netzer et al., 2001; Hague et al., 2003; Caia et al., 2017; Saidi et al., 2019; Whitworth-Turner et al., 2019; Ferreira et al., 2023), while other noted no significant effect (Knufinke et al., 2018; Walsh et al., 2019; Sekiguchi et al., 2021; Costa et al., 2023a; Surąła et al., 2023). Some studies have been conducted during a period of gradual training reduction (Surąła et al., 2023), a taper period (Walsh et al., 2019; Costa et al., 2023a) or with an underrepresentation of daily high training loads (Knufinke et al., 2018). Moreover, the few improvements in sleep quantity or quality were found after a single day of training (Netzer et al., 2001; Hague et al., 2003; Saidi et al., 2019). These results question the dose-response relationship between the accumulation and/or intensification of training load and sleep. Only Ferreira et al. (2023) observed extended actigraphy-based TST, higher sleep efficiency and a reduction in SOL during a 1-week overload (+38% in sRPE × duration) compared with a 1-week usual training period in youth soccer players. However, the authors warned that the players had no constraints in the morning, which resulted in a later wake-up time that could conceal a potential detrimental effect of the overload on sleep (Ferreira et al., 2023). In addition, these studies explored the impact of training load on sleep during the regular competitive season (Caia et al., 2017; Whitworth-Turner et al., 2019; Sekiguchi et al., 2021) or training camp inducing changes in sleep environment or altered schedules (Pitchford et al., 2017; Thornton et al., 2017; Botonis and Toubekis, 2023), thereby raising the hypothesis of additional confounding factors to training load, such as travel, competition/training schedules, and unfamiliar sleep environments (detailed from page 39).

Overall, when we interest solely to studies that manipulated training load without including confounding factors, artistic swimmers (Schaal et al., 2015), triathletes (Hauswirth et al., 2014), cyclists (Lastella et al., 2015a; Killer et al., 2017), basket-ball (Lastella et al., 2020b) and rugby (Leduc et al., 2019; Conlan et al., 2022) had reduced TST and/or sleep efficiency when they are exposed to periods (i.e., from 1 to 3 weeks) of large increases in training load (i.e., from 25 to 330%) (Figure 12).

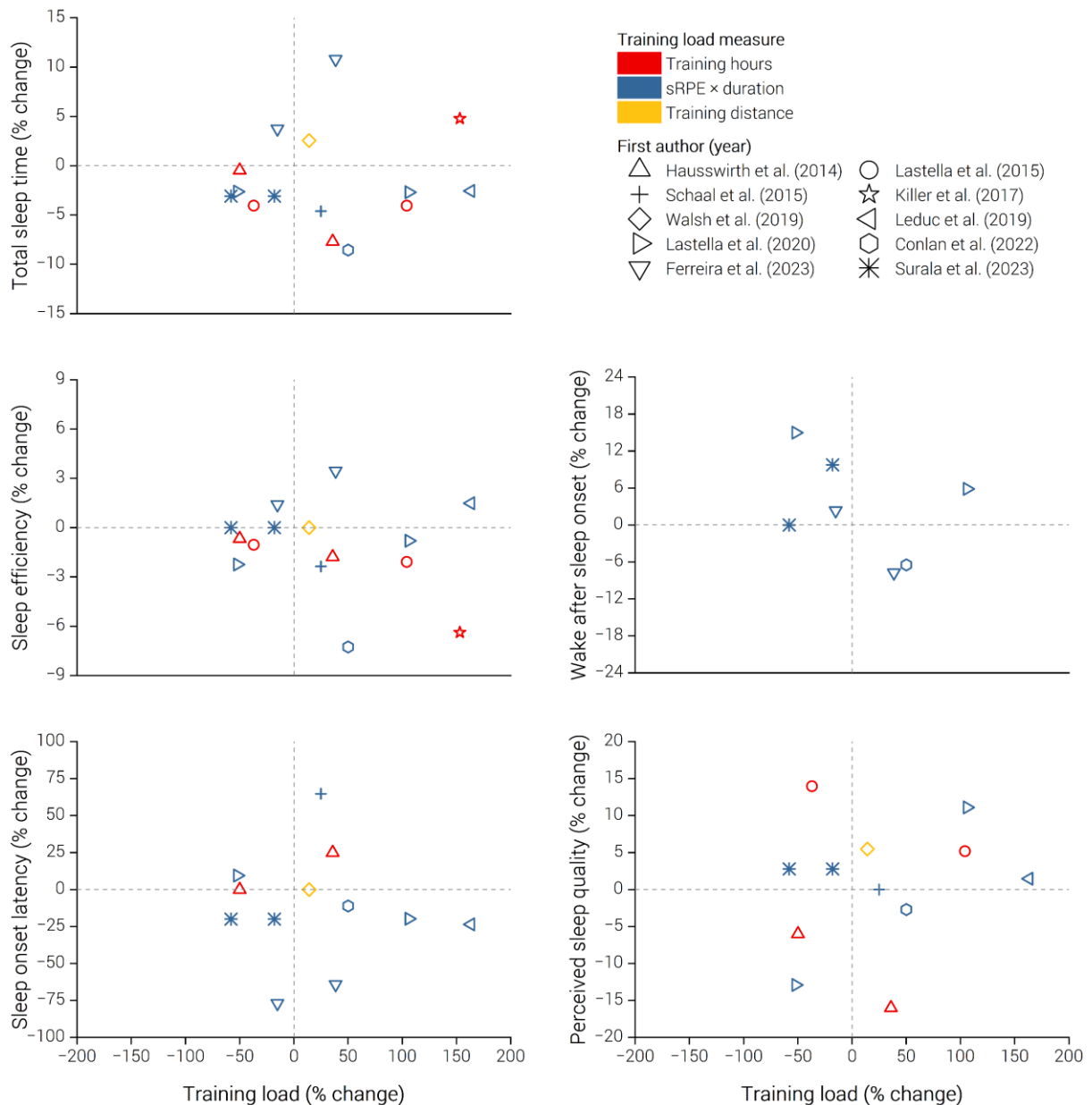


Figure 12. Relationship between sleep parameters and relative change in training load explored in previous studies. *Notes:* Included studies were those that manipulated load over training periods with limited confounding factors (e.g., change in sleep environment or competitive season). *Abbreviations:* sRPE × duration, session-rating of perceived exertion × training session duration.

Table 2. Studies examining the effects of training load on sleep in athletes using an interventional or observational design.

Authors	N (female)	Age	Level (McKay's classification ¹)	Discipline	Sleep monitoring	Training load measure	Protocol	Training load increase (%)	Main findings
<i>Interventional studies</i>									
Driver et al. (1994)	8 (0)	32-42	Fit athletes (2)	Triathlon	PSG	Distance	1-night of no-exercise vs 15-km run vs marathon vs ultra-triathlon	-	Ultra-triathlon vs other exercise: ↑ WASO and REM latency, ↓%REM
Netzer et al. (2001)	13 (0)	24	Highly-trained (3)	Street cyclists	PSG	Distance	1-night after maximal competition exercise vs 1-night after non-training day	-	Night of competition vs non training: ↑ REM latency, %REM
Hague et al. (2003)	15 (8)	22 ± 3	Fit healthy, ~15 ± 4 training hours/week (2)	-	PSG	-	1-night after normal training day vs 1 night after non-training day	-	Night of training vs non training day: ↑SWS and SWA, ↓REM
Hauswirth et al. (2014)	27 (0)	37 ± 6	Well-trained (3)	Triathlon	ACT, SUB	Training hours, frequency and intensity (HR)	3-week usual training (baseline) and overload, 2-week taper	Overload vs baseline: +35% (volume) Taper vs baseline: -50%	Overload vs baseline (OR athletes): ↓ TST, SE, time immobile
Lastella et al. (2015)	21 (0)	22 ± 3	National/International (3-4)	Cycling	ACT, SUB	Distance, training hours, speed	7-day baseline, 3-week simulated grand tour, 10-day recovery	Simulated grand tour vs baseline: +104% (volume) Recovery vs baseline: -37%	Simulated grand tour vs baseline: ↓ TST, SE and ↑ activity score
Schaal et al. (2015)	10 (10)	20 ± 0	International (4)	Artistic swimming	ACT, SUB	Training hours, sRPE × duration	7-day baseline, 2-week HI training	HI vs baseline: +25% (intensity)	Intensified training vs baseline: ↓ TST, SE and ↑ SOL
Killer et al. (2017)	13 (0)	25 ± 6	Highly-trained (3)	Cycling	ACT	Training hours, HR	2-week baseline, 9-day HI training	HI vs baseline: +153% (volume), 146% (intensity)	HI vs baseline: ↑ TIB and fragmentation index, and ↓ SE
Saidi et al. (2019)	20 (0)	14–17	National (3)	Rugby union	Sensewear Armband Pro	HR	1-night of regular training day vs 1-night of HI training day	HI day vs regular training day: +22.1%	HI vs regular training day: ↑ TST, SOL, SE and ↓ WASO
<i>Observational studies</i>									
Taylor et al. (1997)	7 (100)	19 ± 2	National (3)	Swimming	PSG	-	3-month interval: onset, peak training and taper	-	Peak vs Taper: ↓ light sleep, ↑ SWS, ↑ no. movements Onset vs Taper: ↑ SWS, ↑ no. movements

¹McKay et al. (2022)

Kölling et al. (2016)	55 (25)	18 ± 1	National (3)	Rowing	Sleep logs, ACT	No. training	2-week intensified then reduced training periods	-	Intensified vs low reduced load periods: ↓ TST
Caia et al. (2017)	7 (0)	24 ± 2	Elite national team (4)	Rugby league	ACT, SUB	sRPE × duration	2-week of preseason and in competitive season	Pre- vs competitive period: +80%	Preseason vs competitive season: ϕ Sleep ↑ sRPE × duration = ↑ TIB, TST, SE, ↓ SOL
Pitchford et al. (2017)	19 (0)	22 ± 4	Professional (4)	Australian Football	ACT, SUB	sRPE × duration	8-day home and camp periods	Camp vs home: +11%	Home: ϕ relationship TL-sleep Camp: ↑ sRPE × duration = ↓ TST
Thornton et al. (2017)	31 (0)	25 ± 4	Professional (4)	Rugby league	ACT, SUB	Training hours, distance, HSD (GPS)	7-day home period vs 13-day HI training camp	HI camp: +70% (volume), +24% (intensity)	HI camp vs home: ↓ TST, SE, perceived sleep and ↑ WASO ↑ Distance and training hours = ↓ TST
Thornton et al. (2018)	14 (0)	26 ± 3	Professional (4)	Rugby league	ACT, SUB	sRPE × duration, distance, HSD, Acc/Dec load (GPS)	16-week preseason period	-	↑ Acc/Dec load = ↑ SE, TST ↑ Distance and HSD = ↓ TST and SE
Knufinke et al. (2018)	98 (56)	19 ± 3	National/International (3-4)	Individual and team sports	ACT, one-channel EEG headband	sRPE	7 days of regular training	-	ϕ Sleep
Dumortier et al. (2018)	26 (26)	15 ± 4	Elite national team (4)	Artistic gymnastic	Sleep logs	sRPE × duration	9-week training period	-	↑ sRPE × duration = ↓ TST
Whitworth-Turner et al. (2019)	10 (0)	18 ± 1	Academy/National (3)	Soccer	One-channel EEG headband	sRPE × duration, high-speed distance (GPS)	2-week in competitive season	-	↑ HSD = ↑ TIB, TST and WASO ↑ sRPE × duration = ϕ sleep
Leduc et al. (2019)	9 (0)	28 ± 5	International (4)	Rugby sevens	ACT, SUB	sRPE × duration, distance, HSD, no. accel/decel (GPS)	Highest vs lowest intensified weeks during a 6-week preseason	Highest vs lowest: +163%	High vs low = ↓ TIB, TST and subjective sleep quality ↑ Distance = ↑ perceived sleep quality ↑ sRPE × duration = ↓ fragmentation index ↑ HSD = ↑ fragmentation index

Walsh et al. (2019)	12 (3)	21 ± 2	National/ International (3-4)	Swimming	ACT	Distance, intensity zones ([La])	2-week preparation training and taper phases and 3 to 7-day competition and rest phases	Taper vs training: -12% (volume)	Taper vs training: ϕ Sleep
Lastella et al. (2020)	11 (11)	17 ± 1	Elite national team (4)	Basket-ball	ACT, SUB	No. training, training hours, sRPE × duration	Low, moderate and high daily training load	Low vs moderate: -52% High vs moderate: +106%	High vs low: ↓ SE High vs moderate: ϕ Sleep Moderate vs low: ϕ Sleep
Lalor et al. (2020)	38 (0)	22 ± 3	Elite (4)	Australian Football	ACT, sleep logs	PlayerLoad, speed, distance (GPS)	2-week preseason	-	Maximum speed and PlayerLoad are associated with sleep characteristics
Aloulou et al. (2020)	19 (0)	21 ± 1	Highest national (4)	Rugby union	PSG	HSD (GSP), no. collisions (video)	Sleep after an official match	-	↑ Collisions = ↑ light sleep (N1 sleep + N2 sleep) ↑ HSD = ↑ SWS and ↓ light sleep
Hrozanova et al. (2020)	56 (19)	17–19	Club (2-3)	Winter sports	Somnofy	TRIMP	61-day training period	-	↑ TRIMP = ↓ REM
Aloulou et al. (2021)	128 (39)	15 ± 2	National/ International (3-4)	Various sports	ACT, SUB	sRPE × duration	Typical ~7.3 days in season period	-	↑ sRPE × duration = ↓ TST and perceived sleep quality, ↑ fragmentation index
Figueiredo et al. (2021)	18 (0)	15 ± 0	Elite national team (4)	Soccer	ACT	sRPE × duration	3 training days and 2 match days	-	↑ sRPE × duration = ↓ TST
Sekiguchi et al. (2021)	60 (0)	18-23	Academy/ National (3)	Soccer	Sleep logs	Acute:chronic work load from TRIMP	1-year season	-	ϕ Sleep
Costa et al. (2021)	34 (34)	21 ± 2	National/ International (3-4)	Soccer	ACT, SUB	HR, TRIMP, sRPE × duration	2-week in-season competitive period	-	↑ TRIMP and sRPE × duration = ↓ TST and SE
de Blasiis et al. (2021)	15 (8)	22 ± 2	International (4)	Various sports	ACT, SUB	sRPE × duration	Typical, in-season training period (20 ± 1 days) free from competition	-	↑ sRPE × duration = ↑ WASO and SOL
Conlan et al. (2022)	26 (0)	25 ± 4	Professional (4)	Rugby league	ACT, SUB	sRPE × duration	1-week low and high training load, match week	High vs low: +50%	High vs low: ↓ TST

Vachon et al. (2022)	18 (0)	19 ± 1	Elite national team (4)	Rugby union	Sleep questionnaire: PSQI	sRPE × duration, distance, HSD (GPS)	3-day off followed by 3-week preseason overload and 7-day taper	Overload vs taper: +147%	Overload vs season off: ↑ poor sleep in AF players Overload vs season off: ↑ good sleep in F-OR players
Leduc et al. (2022)	23 (0)	21 ± 1	Academy/National (3)	Rugby union	ACT	Distance, HSD (GSP), sRPE × duration, no. collision (video)	13 matches (i.e., 83 player-match observations)	-	↑ Collisions = ↑ wake-up time
Costa et al. (2023)	15 (10)	13 ± 1	Club (2-3)	Swimming	ACT	sRPE × duration, distance	3 weeks of taper phase	-	ϕ Sleep
Ferreira et al. (2023)	13 (0)	16 ± 1	Trained (2)	Soccer	ACT	PlayerLoad, sRPE × duration	1-week usual training (baseline), overload and taper periods	Overload vs baseline: +38% Taper vs baseline: -15%	Overload vs baseline: ↑ TST, SE and ↓ SOL Taper vs baseline: ↓ SOL ↑ PlayerLoad = ↑ TST ↑ sRPE = ↑ TST and SE
Surata et al. (2023)	18 (8)	21 ± 4	National /International (3-4)	Swimming	ACT, sleep logs, SUB	sRPE × duration, distance	3-week training reduction	Week 2 vs week 1: -18% Week 3 vs week 1: -58%	Week 2 vs week 1: ϕ Sleep Week 3 vs week 1: ϕ Sleep
Botonis and Toubekis (2023)	8 (0)	29 ± 4	International (4)	Water-polo	ACT	sRPE × duration	3-day pre-camp vs 5-day HI camp vs 8-day congested period of training and competition	Camp vs pre-camp: +185%	Camp vs pre-camp: ↓ TST, ↓ SE
Apinis-Deshaies et al. (2023)	50 (0)	24 ± 4	Professional (4)	Ice-hockey	Sleep logs, SUB	PlayerLoad, on-ice load (OIL = sum Acc)	1-year season	-	↑ OIL.min ⁻¹ = ↓ TST
Frytz et al. (2023)	53 (0)	14 ± 1	Academy/National (3)	Soccer	ACT, ECG device, sleep logs and SUB	Intensity (0-100 VAS)	3-year season	-	↑ Training intensity = ↑ WASO

Notes: ϕ, no significant relationship/effect. *Abbreviations:* PSG, polysomnography; ACT, actigraphy; SUB, subjective; EEG, electroencephalogram; ECG, electrocardiogram; SWS, slow-wave sleep; REM, rapid eye movement; SWA, slow-wave activity; HR, heart rate; OR, overreaching; F-OR, functional-overreaching; AF, acute fatigue; TST, total sleep time; SE, sleep efficiency; SOL, sleep onset latency; WASO, wake after sleep onset; TIB, time in bed; sRPE × duration, session-rating of perceived exertion × training session duration; Acc, acceleration; Dec, deceleration; HI, high-intensity; HSD, high-speed distance; TRIMP, training impulse; VAS, visual analog scale.

2.2.2. Potential mediators of the relationship between high training load and sleep

Some pathways have been suggested to explain how high training load may affect sleep. As described above, training disrupts homeostasis across various cardiovascular, metabolic, hormonal, neuromuscular, and cognitive systems.

At first glance, intensified training can lead to muscle tissue damage, triggering an inflammatory response led by the immune system (Paulsen et al., 2012). Sleep and inflammatory responses are bidirectionally linked (Irwin, 2019). Proinflammatory cytokine release typically peaks during sleep, including cytokines such as IL-1 β and TNF- α . Their activation due to training-induced muscle damage can initially modulate sleep architecture by increasing NREM sleep and decreasing REM sleep (Besedovsky et al., 2012). In cases of severe immune activation, a concurrent decrease in both NREM and REM sleep, along with other symptoms of sleep disruption may occur (e.g., fragmentation, nonrestorative sleep, daytime sleepiness) (Besedovsky et al., 2019). This could have implications for endurance runners who may experience substantial muscle damage and systemic inflammation during an overload training period (Nieman et al., 2014). Athletes in team-based contact sports such as rugby union may also face these physiological perturbations due to concurrent high-intensity intermittent running (e.g., high-speed runs, changes of direction, rapid accelerations and decelerations) and combative efforts (e.g., tackling, static holds, scrums, rucks, and mauls) (Naughton et al., 2018). However, this dose-response relationship is currently derived from animal models (Besedovsky et al., 2019), and it remains unclear to what extent inflammatory responses and immune activation induced by elevated training demands directly impact athletes' sleep (Hauswirth et al., 2014).

Muscle damage and associated inflammatory response are also well-known to induce pain and delayed-onset muscle soreness (DOMS) (Paulsen et al., 2012; Mizumura and Taguchi, 2016; Hainline et al., 2017b). They may be especially important in contact-sports due to collision-based actions (Naughton et al., 2018). The bidirectional relationship between pain and sleep is well-documented (Herrero Babiloni et al., 2020). Pain may disturb sleep, and disrupted sleep may exacerbate perceived pain (Haack et al., 2020; Stroemel-Scheder et al., 2020). Sleep-wake regulation and nociception share common neuroanatomic and molecular substrates (Bjurstrom and Irwin, 2016), which are modulated, in particular, by inflammation, psychological state (e.g., affect, mood, pain catastrophizing), endocrine substances (e.g., dopamine, melatonin), and activated anatomical pain-related brain areas (Finan et al., 2013; Herrero Babiloni et al., 2020). Some evidence has shown that nociceptive stimuli can affect both sleep

quantity and architecture by fragmenting sleep or disturbing the continuity of sleep stages, leading to transitions from deep to light sleep (Bjurstrom and Irwin, 2016; Herrero Babiloni et al., 2020). Pain and DOMS are common symptoms of excessive training loads and can be considered as potential contributors to disturbed sleep in overloaded athletes (Hainline et al., 2017a). Some studies have also suggested that DOMS induced by excessive loads results in physical discomfort and may lead to difficulties in remaining immobile during sleep in an attempt to get comfortable, thus resulting in restless sleep (Taylor et al., 1997; Hausswirth et al., 2014; Sargent et al., 2016). However, these studies provided anecdotal suggestions, and the putative evidence of pain as a mediator in the detrimental impact of high training load on sleep remains unclear.

Furthermore, intensified training can lead to hormonal disruptions and changes in sympathovagal balance (Roberts et al., 2019b). It has been demonstrated that increased training load results in a rise in circulating cortisol in endurance runners (Balsalobre-Fernández et al., 2014), and strenuous training may result in sympathetic hyperactivity (Iellamo et al., 2002). These endocrine and autonomic changes may prevent the normal downregulation of the human stress systems through their interaction with the hypothalamic-pituitary-adrenal and sympathetic-adrenal-medullary axes, which are required for healthy sleep (Chennaoui et al., 2015). In this regard, cortisol release has been shown to affect both NREM and REM sleep and lead sleep fragmentation due to activation of the hypothalamic-pituitary-adrenal axis (Hirotsu et al., 2015). Additionally, Schaal et al. (2015) observed a negative correlation between changes in resting salivary α -amylase concentration, a marker of sympathetic activity, and sleep efficiency in artistic swimmers. Heavy training may also elevate body temperature, which can counteract the necessary heat loss for adequate sleep (see from page 66), especially when the training session is close to bedtime (Aloulou et al., 2020a).

Moreover, although there are currently limited available data, it is important to consider the psychological and sociological constraints that arise from increased training loads as potential contributors to sleep disturbance caused by excessive load. Indeed, some evidence shows that overloading result in mood disturbances and increased mental stress (Meeusen et al., 2013), which has been associated with a reduction in sleep duration and sleep efficiency among junior winter endurance athletes (Hrozanova et al., 2020). Increasing the volume of training inevitably requires changes in personal and/or professional schedules and commitments. The changes in the organization of training and lifestyle around training can lead to additional stress, resulting in confounding factors in the relationship between sleep and training load. For

instance, this may alter sleep habits, due to earlier morning or later evening training sessions. Additional studies should be conducted on the psychological and sociological factors which may be involved in the relationship between training load and sleep.

Therefore, when considering these disruptions, it becomes evident that extensive training loads may affect athletes' sleep. In **Figure 13**, we propose a model outlining potential physiological and psychosociological mechanisms underlying sleep disturbance when exposed to excessive training load. Nonetheless, it is worth to mention that the etiological mechanisms previously mentioned have been little studied, and thus, are still not fully understood. The extent to which they interact with each other (cross-effect) is also not known. Consequently, whether the effect of high training load on sleep is direct or indirect remains a topic of current debate.

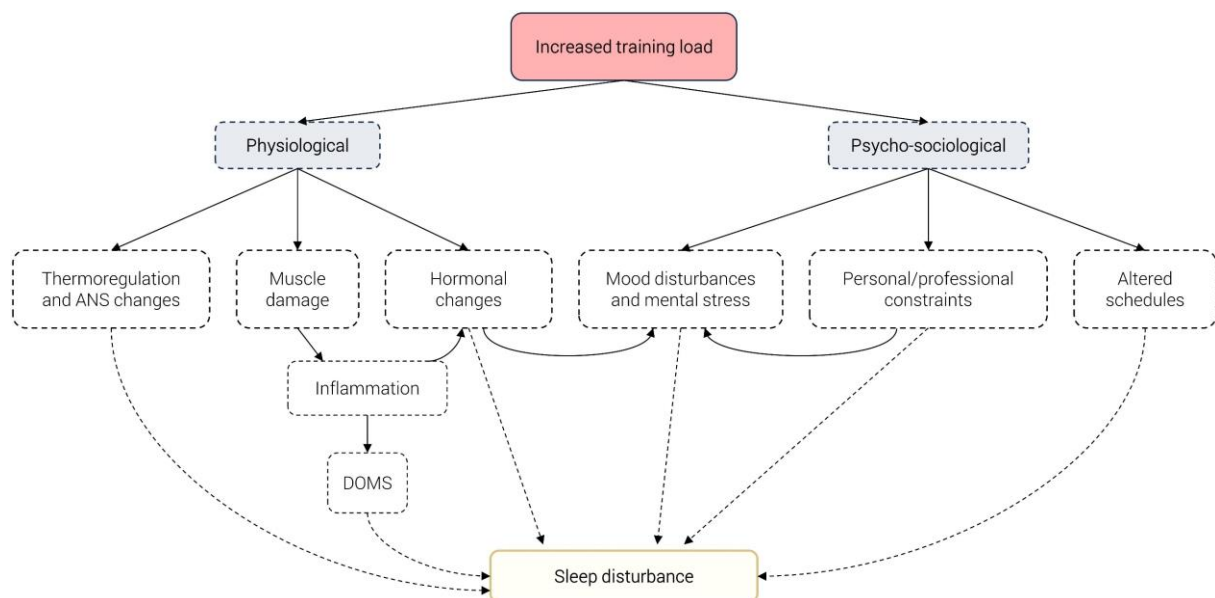


Figure 13. Theoretical model of the contribution of both physiological and psycho-sociological factors in the potential detrimental effect of increased training load on sleep in athletes. *Notes:* solid arrow, evidence-based link; dotted arrow, unexplored link. *Abbreviations:* ANS, autonomic nervous system; DOMS, delayed-onset muscle soreness.

2.2.3. Limitations and important unexplored areas

While there is a growing body of studies investigating the relationship between training load and sleep in athletes, the majority of these studies have relied on actigraphy and/or subjective methods to monitor sleep (**Table 2**). They represent easy-to-use and cost-effective methods to assess individual sleep-wake patterns (detailed from page 82), but do not measure the internal structure of sleep (e.g., sleep stages, cortical arousals). Such measures are critical in determining sleep quality. Studies employing polysomnography have only explored the acute

effect (i.e. a single training day) of training load on subsequent sleep architecture (Driver et al., 1994; Netzer et al., 2001; Hague et al., 2003; Aloulou et al., 2020b).

To the best of our knowledge, only one study has examined sleep architecture in response to various training periods. At 3-month intervals, Taylor et al. (1997) observed higher proportions of SWS along with a greater number of movements during one-night polysomnography at the onset of the season (medium-intensity training) and during an overload period (high-intensity training) compared to a taper period (low training volume) in elite female swimmers. These findings support both the dose response relationship between training and the restorative physiological function of SWS, as well as the potential for restless sleep when exposed to excessive training loads. Nevertheless, this study only involved seven elite athletes practicing a non-weight-bearing discipline and was not replicated in other sports, where training demands and associated psychophysiological stress could differ. It was demonstrated that intensified endurance running induces a larger extent of muscle damage, inflammatory responses and DOMS compared with non-weighted-bearing discipline such as cycling (Nieman et al., 2014). The high-intensity intermittent and combative nature of rugby union may further exacerbate the level of muscle damage, associated inflammatory response, and DOMS, particularly due to collision-based actions (Tavares et al., 2017; Naughton et al., 2018, 2021). These combative actions have been shown to contribute to disturbances in sleep architecture (Aloulou et al., 2020b). Therefore, further research is needed on the impact of high training loads on sleep architecture in other sports, such as in these disciplines that involve diverse external and internal training demands.

Finally, most of the studies assessed training load using sRPE \times duration (**Table 2**). Monitoring additional internal and external measures of training load can offer further insights into the impact of specific physical demands encountered during training on sleep. For instance, increased acceleration/deceleration demands imposed on rugby league players were associated with longer TST and increased sleep efficiency, while higher high-velocity ($>4 \text{ m}\cdot\text{s}^{-1}$) distance was found to be detrimental to TST and sleep efficiency (Thornton et al., 2018). Conversely, improvements in TST and WASO were found following greater high-velocity ($>5.5 \text{ m}\cdot\text{s}^{-1}$) distance in youth soccer players (Whitworth-Turner et al., 2019) and shorter TST was observed following increases in daily distance covered during a rugby league training camp (Thornton et al., 2017). Similarly, Leduc et al. (2019) found improved perceived sleep quality and decreased sleep fragmentation with greater distance covered and sRPE \times duration in elite rugby seven players, respectively. They also noted increased sleep fragmentation as high-velocity

distance was higher. However, once more, these studies monitored sleep using actigraphy, and to date, only one study has highlighted the varied effects of the combative and high-intensity intermittent demands of a rugby union match on subsequent sleep architecture, using single-night polysomnography (Aloulou et al., 2020b). Every 100-m increase in high-velocity ($>5 \text{ m}\cdot\text{s}^{-1}$) running distance was associated with a subsequent reduction in the proportion of light sleep (i.e., N1 + N2 sleep, -1.2%), in favour of higher proportions of SWS (+1.1%). Conversely, for every 10 additional collisions, the SWS proportion decreased (-1.9%) and the light sleep proportion increased (+2.9%). The putative effect of varied training demands in rugby union (e.g., high-intensity intermittent running and collision-based actions) on sleep architecture during a period of cumulative training load has not yet been investigated. This could provide guidance to practitioners on managing training load and imposed specific physical demands, for example in preparation for a match, for the benefit of restorative sleep.

Key Points

- Practicing sport at the highest level requires accurate **management of training loads** to achieve **peak performance**, particularly via periods of **intensified and/or accumulated training (overload)**.
- Despite the recognized sleep benefits of physical exercise in general population, numerous studies have reported **alterations in sleep-wake patterns** (notably sleep duration and efficiency) when athletes are exposed to periods of **increased training load**.
- However, this sport-specific issue is **not yet fully understood**.
- The impact of **excessive training load on sleep** has mainly been assessed through inferred **subjective and/or actigraphy** measurements. **Sleep architecture**, critical in determining **sleep quality**, has received little attention. Thus, it remains elusive how it responds to **various training loads** and **specific physical demands**.
- Increased training load imposes **physiological** and **psychological stress** and may disrupt the **organization and lifestyle around training**. It remains **unclear** to what extent they contribute to sleep disturbances in athletes when exposed to elevated training loads.
- Additional investigations in these unexplored areas could provide **guidance for practitioners** on **managing training load and physical demands** for the benefit of **quality sleep**.

Part 3 – A bedding strategy susceptible to mitigate sleep disturbances in athletes?

Despite the importance of sleep for athletes, previous sections have highlighted evidence of a high prevalence of inadequate sleep quantity and/or quality among elite athletes. High training loads are a potential sport-specific stress factor likely to exacerbate athletes' sleep problems. Consequently, developing practical strategies that can promote athletes' sleep is highly required. The thermal properties of bedding have been identified as a major determinant of the sleep quality, yet their effects on sleep, particularly in athletes, have received limited scientific attention (Troynikov et al., 2018). The following section 1) presents evidence of an interdependent and pivotal relationship between sleep and thermoregulation; and 2) focuses on a sleep strategy based on the thermal properties of bedding designed to manipulate thermoregulatory processes.

1. Sleep and thermoregulation

Who has never experienced sleep difficulties during heat events such as in summer? It is well-documented that sleep is sensitive to even mild changes of night-time ambient temperature (in addition to humidity and air flow) (Carskadon and Dement, 2017; Lan et al., 2017). In some controlled laboratory studies, it has been demonstrated that deviating from a thermoneutral zone in ambient temperature, characterized by thermal discomfort (hot or cold), reduces the amount of sleep, SWS and REM sleep, as well as delayed SOL and increased wakefulness (Haskell et al., 1981; Okamoto-Mizuno et al., 2005; Pan et al., 2012; Lan et al., 2014). The ambient temperature dependence of sleep is currently an important issue, particularly considering the increasing and intensified exposure to high temperatures due to climate change, which has become a major concern (Patz et al., 2014; Watts et al., 2018). In a large study investigating the relationship between meteorological factors and human sleep, Minor et al. (2022) predicted that by 2099, the end of the current century, warmer night-time outdoor temperature may result in the loss of 50–58 h of sleep per person per year. These authors also found no evidence of short-term acclimatization of sleep to varying temperatures. These findings offer evidence of the interdependence between sleep and body temperature regulation.

1.1. A circadian-coupled and mutually dependent relationship

Sleep-wake rhythms and thermoregulation processes are strongly and reciprocally linked to both neurophysiological and behavioural mechanisms (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019). This relationship is considered especially important for initiating and maintaining sleep (Okamoto-Mizuno and Mizuno, 2012). The core body temperature (CBT) exhibits a circadian rhythm that reflects the balance between the heat-producing homeothermic core (i.e., brain, the abdominal cavity including inner organs), and the heat-loss-regulating poikilothermic periphery (i.e., peripheral tissues such as fat, skin, and skeletal muscles). The latter allows heat to be dissipated by thermal exchange towards the ambient environment (Krauchi and Deboer, 2010; Szymusiak, 2018). As shown in **Figure 14–bottom**, CBT is temporally aligned with sleep-wake rhythms, characterized by elevated daytime (its acrophase typically observed in the late afternoon or early evening) and depressed night-time temperature, reaching its nadir in the early morning hours. Sleep is more likely to occur during the circadian phase of CBT decline, typically 4–6 h before the CBT minimum, when heat loss-effector processes are maximal (Kräuchi, 2007; Okamoto-

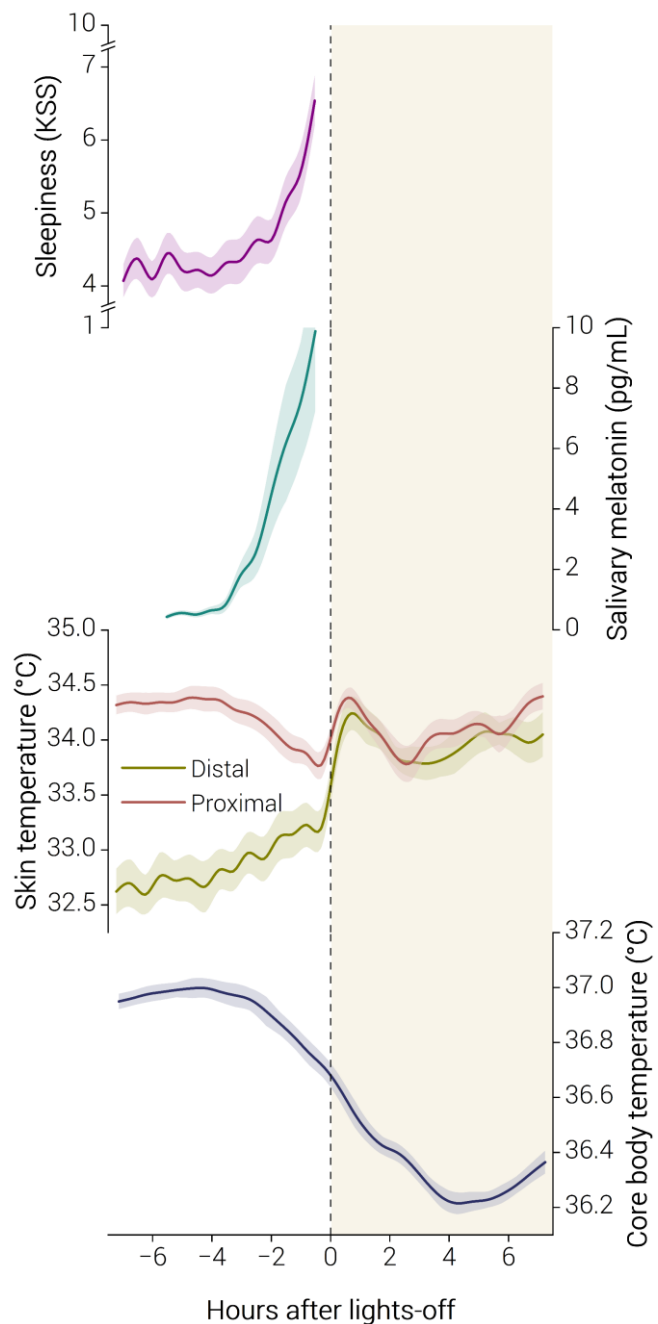


Figure 14. Time course of core body temperature, distal (hands and feet) and proximal (forehead, thigh, infraclavicular area and abdomen) skin temperatures, salivary melatonin concentration, and subjective rating of sleepiness in a baseline 7.5 h constant routine followed by a 7.5 h sleep period (yellow area). Data are presented as mean (\pm standard error) in 30-min bins in 18 healthy men. Mean sleep onset latency: 12 ± 4 min. *Abbreviations:* KSS, 1-10 Karolinska sleepiness scale. Adapted from Kräuchi et al. (2000).

Mizuno and Mizuno, 2012; Szymusiak, 2018). Conversely, awakening is more prevalent during the rising phase of CBT.

The circadian phase of CBT decline usually begins 2–3 h before bedtime and is accompanied by increased distal (e.g., hands and feet) and decreased proximal (e.g., thigh, infraclavicular region, stomach, forehead) skin temperatures. These changes in body temperature result from a convective redistribution of heat from the core to the periphery to evacuate heat to the ambient environment, through vasodilatation of capillaries and arteriovenous anastomoses, particularly dense in distal skin regions (Kräuchi et al., 2000; Kräuchi, 2007; Okamoto-Mizuno and Mizuno, 2012). At the same time, a progressive increase in sleepiness occurs in parallel with salivary melatonin concentrations. When sleep occurs, CBT continues to fall slowly and distal and proximal skin temperatures further increase for 1–2 h. This coincides with the highest prevalence of SWS and SWA (Krauchi and Deboer, 2010; Kräuchi and Deboer, 2017). Additionally, it has been observed that some pre-bedtime sleep-permissive behaviours such as lying down on bed, turning off the lights, practicing mind and/or muscle relaxation, and simple knowing the bedtime, lead to more blood flow from the core to the skin, lower CBT and finally sleep promotion (Rogers et al., 2007; Krauchi and Deboer, 2010).

1.2. Interconnected neural control of sleep and body temperature

The coupling mechanisms between sleep-wake rhythms and thermoregulation processes are clocked in the brain by the master circadian pacemaker, i.e., the suprachiasmatic nucleus (SCN) of the hypothalamus, and controlled by neurons located in the preoptic-anterior hypothalamus (POAH) (McGinty and Szymusiak, 2017), as illustrated in **Figure 15**. The POAH is recognized as a sleep-promoting and thermoregulatory control site, especially via its ventrolateral preoptic nucleus (VLPO) and median preoptic nucleus (MnPO) (McGinty and Szymusiak, 2001; McGinty et al., 2001). The POAH receives and processes information of body temperature from numerous thermo-sensitive neurons (mainly warm-sensitive), and activates thermo-effector responses via the autonomic nervous system, such as sweating, shivering and changes in vasomotor tone, to maintain CBT within reference thresholds (Romanovsky, 2018). These thresholds delimit a thermoneutral narrow zone, defined as the range in which CBT regulation is achieved solely by controlling sensible heat loss, i.e., vasoconstriction and vasodilatation are the only autonomic thermoeffector responses to regulate CBT, without regulatory changes in either metabolic heat production or evaporative heat loss, and which includes the zone of thermal comfort (Krauchi and Deboer, 2010; Romanovsky, 2018). This thermoneutral zone is

adjusted according to a circadian rhythm by the SCN, which transmits the circadian signal via a rostral projection to the POAH (Kräuchi and Deboer, 2017).

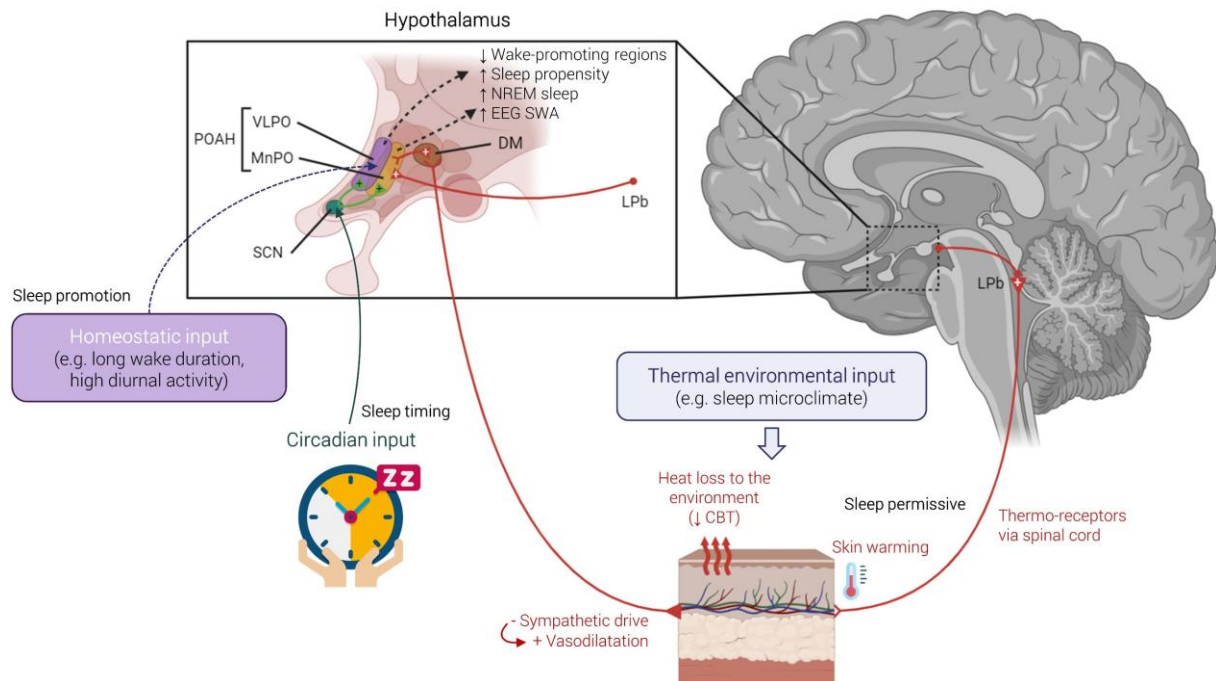


Figure 15. Summary of the interactions between the thermoregulatory and sleep regulatory areas in the POAH, and thermosensitive and effector pathways. Sleep and thermoregulatory mechanisms depend on input from three competing mechanisms: the homeostatic and circadian processes, and thermal environmental input. The SCN synchronizes sleep-wake rhythms and thermoeffector responses controlled by POAH by promoting sleep during the circadian phase of CBT decline (i.e., heat loss), and waking during the elevated CBT phase (not shown). *Abbreviations:* +, promotional actions; -, inhibitory actions; POAH, preoptic-anterior hypothalamus; SCN, suprachiasmatic nucleus; CBT, core body temperature; VLPO, ventrolateral preoptic nucleus; MnPO, median preoptic nucleus; GABA, γ -aminobutyric acid; DM, dorsomedial; LPb, lateral parabrachial; SWA, slow-wave activity. Adapted from McGinty and Szymusiak (2017), Szymusiak (2018) and Harding et al. (2019).

As bedtime approaches, SCN drives neurons in the VLPO, increasing sleep propensity (see from page 20). In parallel, the SCN activates the warm-sensitive neurons in the MnPO, increasing both sleep propensity (subsets of these neurons may project to ascending arousal systems and are GABAergic) and peripheral dilatation of arteriovenous anastomoses. This peripheral vasodilatation is permitted by a reduction in the activation of noradrenergic vasoconstrictor tone via the inhibition of the sympathetic premotor neurons in the ventral medial medulla and the dorsomedial part of the hypothalamus (McGinty and Szymusiak, 2017; Harding et al., 2020). Peripheral vasodilatation favours the convective redistribution of heat from the core to the periphery via blood flow, thus facilitating heat loss (and consequently CBT decline) to the environment through the distal skin surface. In response to distal skin warming, warm-sensitive neurons in the MnPO receive input from thermoreceptors in the skin via the spinal cord and

relays the information to the lateral parabrachial nucleus in the pons, resulting in feedforward activation of warm-sensing and sleep-promoting neurons, which facilitates transitions from wakefulness to sleep (Szymusiak, 2018). The onset of sleep increases the discharge of warm-sensing and sleep-promoting neurons in the VLPO and the MnPO, expanding the heat loss-effector processes and decline in CBT (Harding et al., 2019). Concurrently, this promotes NREM sleep and SWA by inhibiting wake-promoting regions in the brain. In addition, the SCN-driven melatonin release prior bedtime may also play a crucial role in sleep-related thermoregulatory processes, by acting on SCN itself (feedback effects), which in turn activates the heat loss-effector processes, and thus promotes sleep (Kräuchi et al., 2006; Lok et al., 2019).

2. Potential strategies to promote sleep

2.1. Warming or cooling sleep strategy?

The previous section highlighted that body heat loss, resulting in an increase in distal skin temperature and a decline in CBT, is both a predeterminant of sleep propensity and a consequence of sleep onset. In this respect, many studies have attempted to manipulate body temperature to promote heat loss and sleep (Romeijn et al., 2012; Te Lindert and Van Someren, 2018). In a systematic review and meta-analysis, Haghayegh et al. (2019) reported that skin warming using a warm shower or bath 1–2 h before bedtime can reduce SOL, most likely by the rate of change in CBT, i.e., heat loss. Similarly, Raymann et al. (2005, 2007, 2008) manipulated the skin temperature using a thermo-suit worn before and during sleep. A subtle skin warming (+0.4–0.8°C around a mean of 35°C), whilst not altering CBT, has been shown to reduce SOL, promote SWS, and mitigate WASO.

Conversely, Togo et al. (2007) demonstrated that a slow and mild reduction in ambient temperature by 2°C through the night increased the amount of SWS and the rise in SWA, by lowering and delaying the minimum of CBT. This may be explained by the fact that the onset of SWS is accompanied by a reduction in peripheral vascular resistance (Te Lindert and Van Someren, 2018), and by feedback effects, facilitating heat loss which may finally promote SWS (Szymusiak, 2018). This mild reduction in ambient temperature was within a thermoneutral range (i.e., 29.5°C, without quilt and ticking, and semi-nude), which did not have detrimental effects on other sleep parameters, such as REM sleep that is more sensitive to the thermal environment caused by reduced tonic muscle tone and increased peripheral vasoconstriction (Szymusiak, 2018; Te Lindert and Van Someren, 2018).

Overall, these findings support the importance of increased heat loss to promote the initiation, depth and continuity of sleep. Nevertheless, these studies were conducted under strictly-controlled conditions within a thermal comfort zone, which can be difficult to implement in real-life conditions (Raymann et al., 2008). For instance, the warming strategy may not be relevant during elevated ambient temperatures considering the impairment of sleep comfort, quantity, and quality in these conditions (Lan et al., 2017; Minor et al., 2022). This situation is common in real life, especially among elite athletes, as they are regularly exposed to unusual sleeping environments due to travel, sometimes experiencing high ambient temperatures (see from page 39). Major international sporting events, such as the Summer Olympic Games, are often held during the hottest months of the year, posing a significant challenge for athletes' sleep (Racinais et al., 2023). Therefore, developing a practical, home-friendly strategy that regulates body temperature within a thermoneutral comfort range, favouring heat loss without inducing counter-regulation in various thermal conditions, could benefit the sleep of elite athletes.

2.2. Sleep microclimate and high-heat-capacity bedding system

Although a slightly cool ($\sim 19\text{--}21^\circ\text{C}$) sleep environment is usually recommended for adequate sleep, the sleep microclimate has been proposed as a key factor which determines sleep-related thermoregulation mechanisms, thermal comfort, and sleep quality (Raymann et al., 2005; Troynikov et al., 2018; Harding et al., 2019). The sleep microclimate is defined as the thermal

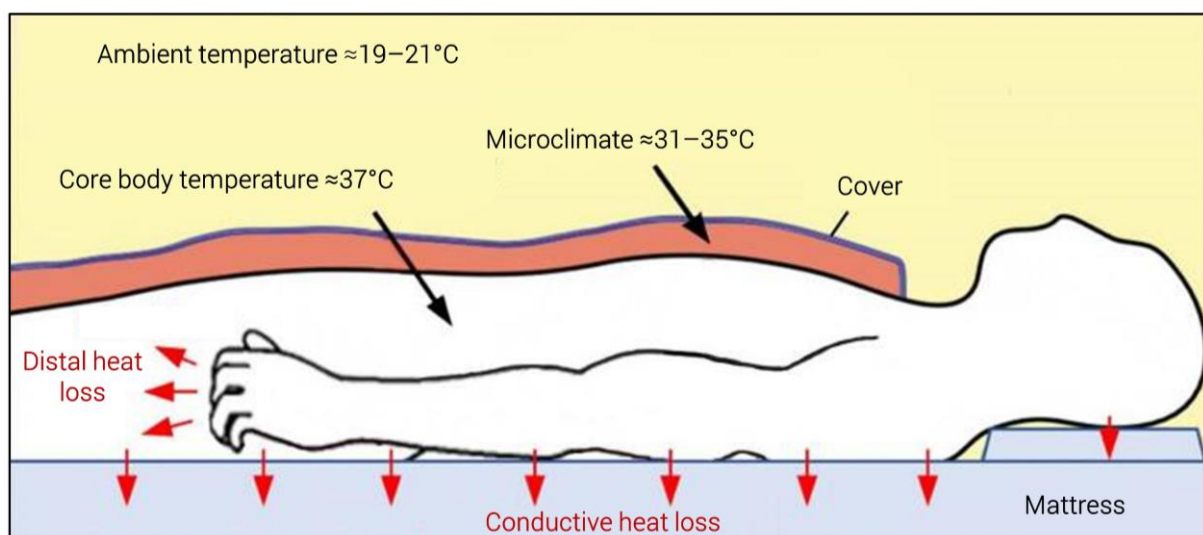


Figure 16. Thermal heat exchanges between a human and the sleep environment. The sleeper uses the bedding system (e.g., mattress, cover, pillow) to create a warm sleeping microclimate, which activates thermoregulatory mechanisms triggering convective peripheral heat loss via vasodilatation (mainly in distal skin regions), and the sleep-promoting systems. Body heat loss is conveyed by conduction to the bedding system, also determining the microclimate during sleep according to its thermal resistance properties. A high-heat-capacity bedding system may promote slow and continuous evacuation of body heat during the night. Adapted from Harding et al. (2019) and Herberger et al. (2024).

environment that surrounds the sleeper's skin, mainly determined by the heat exchange between the insulating bedding system (mattress, blanket, pillow and sleepwear) and the body (**Figure 16**). It is recommended to maintain the sleep microclimate at $\sim 31\text{--}35^\circ\text{C}$ to maintain CBT in a thermoneutral zone, while facilitating heat loss (Harding et al., 2019). Nevertheless, ambient temperature is not the only factor determining the sleep microclimate and heat exchanges with the environment. Other factors have been suggested such as humidity, air flow, barometric pressure, thermal conductivity of bedding materials, and body contact area with these materials (Romanovsky, 2018). Thus, the thermal properties of bedding have been identified as important for achieving heat loss and quality sleep (Troynikov et al., 2018).

Although bedding is our nightly support for sleep, it has received little attention in research. The thermal conductance, in opposition to thermal resistance, is defined as the ability of a material or system to conduct heat, from the hottest to the coldest part. Nowadays, bedding industries offer a wide range of products with varying insulating properties. In this respect, the effectiveness of a high-heat-capacity bedding system on sleep has recently been investigated in healthy male volunteers (Kräuchi et al., 2018; Herberger et al., 2020). Sleeping on this bedding system, consisting of a foam mattress coated with a gel sheet on the top, enhanced the slow and continuous removal of body heat through lower CBT

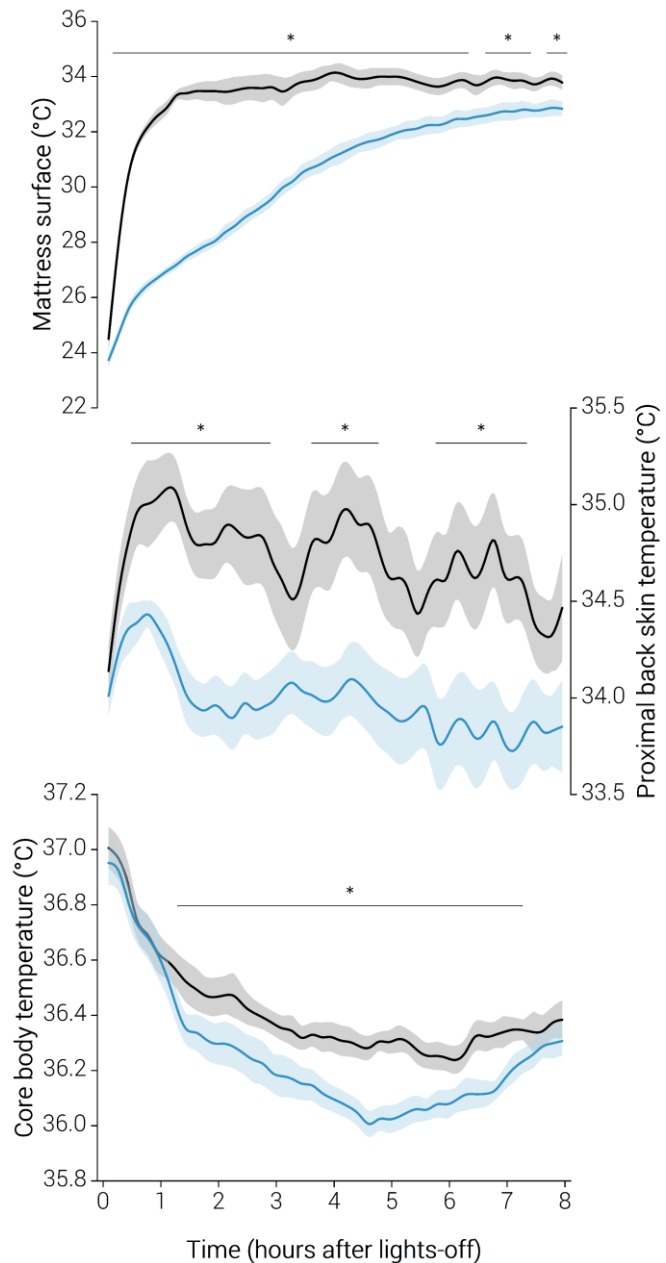


Figure 17. Time courses (mean \pm standard error, 10 min-bins) of mattress surface temperature (monitored below the ticking), proximal back skin temperature (i.e., averaged temperature of back shoulders and spinal cross), and core body temperature during 8 h after bedtime in 15 healthy young men, according to two mattress types (blue, high-heat-capacity; black, low-heat-capacity). *Significant difference between mattress types, $p < 0.05$. Adapted from Kräuchi et al. (2018).

($\approx -0.14^{\circ}\text{C}$) and proximal back skin temperatures ($\approx -0.55^{\circ}\text{C}$) during sleep compared with a 100% foam mattress, which may be explained by differences in insulating properties (**Figure 17**). These findings were associated with an improvement in subjective sleep comfort, and an increase in SWS proportion and standardized EEG slow-wave energy (as time integral of SWA in the delta range, i.e., 0.8–4.3 Hz). In addition, increased EEG slow-wave energy was significantly correlated with the reduction in proximal back temperature (1st half of the night, $r = -0.47$) and CBT (2nd half of the night, $r = -0.46$) (Kräuchi et al., 2018; Herberger et al., 2020). A similar reduction in CBT associated with an improvement in SWS using this bedding strategy was observed in postmenopausal women (Reid et al., 2021). Based on these three studies, Herberger et al. (2024) reported that this bedding strategy provides an increase of $\sim 7.5 \pm 21.6$ min in nocturnal SWS for 7.5 h of sleep. These sleep benefits are particularly observed in individuals experiencing greater body heat loss during sleep, given that those with minimal heat loss show no significant changes in sleep architecture. Additionally, mediation analysis revealed that the decline in CBT does not directly affect SWS. Instead, SWS is influenced through the increased CBT–proximal back skin temperature gradient, which reflects the amount of heat transfer from the body core to the periphery (Herberger et al., 2024). This finding supports the evidence suggesting that heat loss enhances the depth of sleep.

According to these results and in the framework of a PhD thesis in our laboratory, Aloulou et al. (2020) investigated the effect of a similar high-heat-capacity mattress topper (HMT) on sleep architecture after an elite rugby union match. The HMT reduced the mean nocturnal CBT (-0.14°C), mitigated WASO (-10.5 min), and increased the proportion of REM sleep (+2.8%) compared with a control mattress composed of 100% foam. However, further investigations are needed to determine the sleep benefits of this bedding strategy in real-life settings, particularly among elite athletes. First, the aforementioned studies were conducted from an acute perspective, i.e., a single night, and the long-term implications of using this bedding strategy among athletes remain unknown. Second, considering the increasing temperatures due to climate change, which is a major current concern, and the intriguing thermal properties, further investigations are required to understand its sleep benefits under varying thermal conditions. Finally, given the high inter-individual variability of sleep (see section Variability of Sleep among Elite Athletes) and the adaptive response to heat (Racinais et al., 2012; Corbett et al., 2018), additional studies on individual responses to this strategy are warranted. Such studies would provide more precise guidance for prescribing individualized sleep interventions (Fullagar and Bartlett, 2016).

Key Points

- In the final part of the literature review, we highlighted the **interdependent relationship between sleep regulation and body temperature**. This is illustrated by the additional **sleep difficulties** experienced when exposed to **elevated ambient temperatures**, such as in summer.
- Favours **heat loss** as night approaches and during sleep is especially important to **initiate, maintain and promote the depth of sleep**.
- A **high-heat-capacity bedding system** has been identified as a strategy to enhance the slow and continuous **removal of body heat** during sleep. Initial studies have reported improvements in **subjective sleep comfort** and **sleep quality**, particularly through an increase in the amount of **SWS**.
- However, to date, there is **no evidence of the sleep benefits** of this bedding strategy **for athletes when used chronically**, nor when used in **high ambient temperatures** such as in summer. The potential **inter-individual sleep responses** have not yet been investigated.

Research questions and objectives

On the one hand, physical exercise, whether acute or chronic, strongly influences human sleep. High training loads are potential contributory factors to the high prevalence of sleep complaints and inadequate sleep among elite athletes (Walsh et al., 2020). Most studies that imposed extensive overload periods (>25% increase in training load) have described experiencing more restless sleep, accompanied by a reduction in both sleep quantity and efficiency (Roberts et al., 2019b). Nonetheless, the literature in this population is limited, with most information derived from studies using subjective or actigraphy measures to assess sleep. **Further exploration of the internal structure of sleep is required to provide a more comprehensive understanding of the relation between training load and sleep architecture and, in turn sleep quality.** Such investigations conducted in real-life settings may face various logistical and operational challenges, particularly when performed in elite sport setup, whose influence on sleep remains unknown. Increased training load leads to physiological and psychological stress and may disrupt the organization and life around training (e.g., increased number and duration of training sessions), which can be confounding factors in assessing the impact of training load on sleep. **Assessing training load and sleep within the specific context a congested training program would contribute to a better understanding of how training load affects sleep and whether confounding factors are involved.** Further exploring the impact of training load on athletes' sleep may offer crucial insights for practitioners to adapt training programs and loads, thereby benefiting athletes' sleep, well-being, and performance.

On the other hand, strategies striving to promote sleep in athletes is also of prime importance considering its importance in performance, well-being and health (Walsh et al., 2020). Although bedding is our nightly support for sleep, it is rarely studied. The thermal properties of bedding have been identified as a key factor of sleep quality (Troynikov et al., 2018) due to close, reciprocal relationship between sleep and thermoregulation (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019). In this context, a high-heat-capacity bedding system has recently been suggested as a potential practical strategy to promote heat loss, and in turn, sleep quality. Nevertheless, **the putative relevance of this bedding strategy when used chronically during a typical training context in athletes is currently unknown.** Considering the role of training load as a potential modulator of sleep, **investigating how a high-heat-capacity mattress topper (HMT) can mitigate the potential detrimental effect of high training load on sleep would provide crucial information on the relevance of such practical strategy for elite athletes.**

Moreover, increasing and intensified exposure to high temperatures due to climate changes is a current issue for both elite athletes and the general population (Patz et al., 2014; Watts et al., 2018). Since sleep is highly sensitive to elevated night-time temperatures (Minor et al., 2022), **the relevance of this cooling strategy in athletes exposed to elevated temperatures should be investigated in order to preserve their quantity and quality of sleep.** Moreover, given the inter-individual variability of sleep (Nedelec et al., 2018) and the adaptive response to heat (Racinais et al., 2012; Corbett et al., 2018), describing individual responses to this strategy in this context is required for achieving more precise guidance in prescribing individualized interventions (Fullagar and Bartlett, 2016). Exploring such a sleep-promoting strategy in elite athletes will provide insights into its relevance in the context of preparation for the summer Olympic Games. Additionally, testing its applicability in athletes may offer further benefits for the global public, particularly at a time when sleep is recognized as a matter of health and society.

The experimental part presented in this PhD thesis aims to deepen our understanding of the impact of training load on athletes' sleep and to explore the effectiveness of a high-heat-capacity bedding system on athletes' sleep within a typical training context.

Elite team-based contact sports often involve training camps to prepare for major competitions and the competitive season. The often-hectic schedule, alongside with the varied daily training loads experienced by players during these camps, as well as unaccustomed schedules and commitments surrounding training sessions, can concurrently affect sleep (Pitchford et al., 2017; Thornton et al., 2017). *Study 1* aimed to **evaluate the sleep-wake patterns and sleep architecture of young elite rugby union players during a 10-day training camp, taking into account the potential influence of daily training load and the varied morning appointment time.** We hypothesized that early morning appointments may impair sleep duration and that increased daily training load may disturb subsequent sleep-wake patterns and promote sleep architecture.

In individual sport athletes such as endurance runners, it is well recognized that training periodization over several weeks, involving phases of increase (overload) and decrease (taper) in training load, is beneficial and commonly used to achieve peak performance. Therefore, the aim of *study 2* was to (i) **investigate the sleep responses to a training program including a 2-week usual training regimen, overload training, and taper periods in well-trained endurance**

runners, and (ii) **determine whether a high-heat-capacity bedding system, when used chronically, can mitigate the potential detrimental effect of overloading on sleep.** As actigraphy and polysomnography assess different dimensions of sleep, the results of this study are presented in two parts. *Study 2-A* focused on the effect of self-reported training load and HMT on daily actigraphy-based sleep variables and subjective assessments, allowing a day-to-day approach, while *study 2-B* examined the polysomnography-based sleep architecture in response to these training periods and HMT. It was hypothesized that (i) an overload training period negatively affects sleep continuity, (ii) promotes the depth of sleep, and (iii) that sleeping on HMT may mitigate this negative effect by facilitating heat loss.

Lastly, summer period can be particularly decisive for elite athletes, with intensified trainings to prepare for the following season, or sometimes with the presence of major competitions (e.g., summer Olympic Games). Thus, *study 3* **aimed to evaluate the effectiveness of a high-heat-capacity bedding system on elite badminton players' sleep during a summer training period.** We carefully examined the potential individual sleep responses to this strategy, taking into account the sleep patterns of each player. It was hypothesized that this cooling bedding strategy may favour sleep quantity and quality in comparison to a control bedding.

General experimental methodology

This section outlines the primary methodology employed within the framework of this PhD thesis. We will offer detailed information on participants, study designs, and the common methods employed within studies. Further elaboration on each experimental method will be provided within respective study sections. All the experimental methodology presented below have been approved by the local ethic committee (approval reference: 21.00586.000020, ID-RCB: 2021-A01607-34) and in accordance with the Declaration of Helsinki (1964: revised in 2001). Data processing and statistical analyses were conducted using R version 4.2.0 to 4.3.2 (R Core Team, 2023) within R Studio (Posit Team, 2024), and some figures were performed using OriginPro software (OriginLab Corporation, 2024).

1. Participant characteristics and study designs

A total of 61 volunteers aged over 18 years old respectively participated in our three studies (**Table 3**). They received comprehensive verbal and written explanations regarding the purpose of the studies and the experimental procedures, and provided written informed consent. **Studies 1 and 3** were conducted among an elite athlete population engaged full-time in their discipline. The participants in **study 2** were well-trained recreational athletes, managing a full-time professional or academic activity alongside their training program. Inclusion and exclusion criteria are specified within respective study sections. All provided their written informed consent to participate after comprehensive verbal and written explanations of the protocol.

The three experiments were conducted *in situ*, i.e., within athletes' training contexts. **Study 1** was observational and involved 26 youth elite male rugby union players during a 10-day training camp as part of a national team convocation. **Study 2** employed a matched group single-blinded design. Twenty-one trained male endurance athletes performed a 6-week training program in their usual training facilities and were assigned into two groups based on the mattress topper used: a high-heat-capacity mattress topper (HMT, $n = 11$, interventional bedding condition) or a low-heat-capacity mattress topper (LMT, $n = 10$, control bedding condition). Participants were not informed of the presence of another group, nor were they given any information about mattress toppers. In **study 3**, 14 elite badminton players (6 males and 8 females) participated in a randomized, single-blinded, crossover and counterbalanced-order protocol, which consisted of two 1-week conditions: sleeping on HMT and sleeping on LMT. Participants were blind to the type of mattress topper used, nor were they given any information about it.

Table 3. Summary of the experimental studies and sleep monitoring tools used.

	Study 1	Study 2	Study 3
Participants			
N (female)	26 (0)	21 (0)	14 (8)
Age (years)	18.7 ± 0.3	27.2 ± 4.8	23.6 ± 4.2
Discipline	Rugby union	Endurance running	Badminton
Performance level	Tier 4: elite/international	Tiers 2–3: trained/developmental to highly trained/national	Tiers 4–5: elite/international and world class
Experimental design	Observational	Matched group single-blinded	Randomized, single-blinded, crossover, counterbalanced order
Sleep monitoring	Actigraphy, EEG headband, sleep diary and questionnaires	PSG, actigraphy, sleep diary and questionnaires	PSG, actigraphy, sleep diary and questionnaires

Notes: Age is presented as mean ± SD. Performance level was defined according to the guidelines proposed by McKay et al. (2022). *Abbreviations:* PSG, polysomnography.

2. Mattress topper properties

In **studies 2** and **3**, the sleep benefits of the HMT were investigated in comparison with the LMT. Both mattress toppers sized 90 × 200 × 6 cm (Bultex, Noyen-sur-Sartre, France) (**Figure 18**). The HMT consisted of a 40 kg.m⁻³ density, 100% foam, covered on the top with a high-heat-capacity gel sheet (density of 1000 kg.m⁻³; dimensions of 70 × 120 × 2.5 cm). The LMT comprised solely of the same 100% foam material. Both mattress toppers were covered with a ticking composed of 99% of polyester and 1% of elastane. In **study 2**, the ticking had a density of 540 g.m², resulting in thermal resistances of 1.18 m².K.W⁻¹ for HMT and 1.63 m².K.W⁻¹ for LMT. In **study 3**, the density of the ticking was reduced to 370 g.m², leading to thermal resistances of 1.01 m².K.W⁻¹ for HMT and 1.45 m².K.W⁻¹ for LMT. This adjustment in thermal resistance aimed to enhance the heat exchange between the body and the bedding system, following the results of **study 2**. Thermal resistance measurements were carried out by an independent laboratory specializing in building thermal assessment (Centre scientifique et technique du bâtiment, CSTB, Champs-sur-Marne, France). Participants kept their own blanket, pillow and sleepwear throughout the studies.

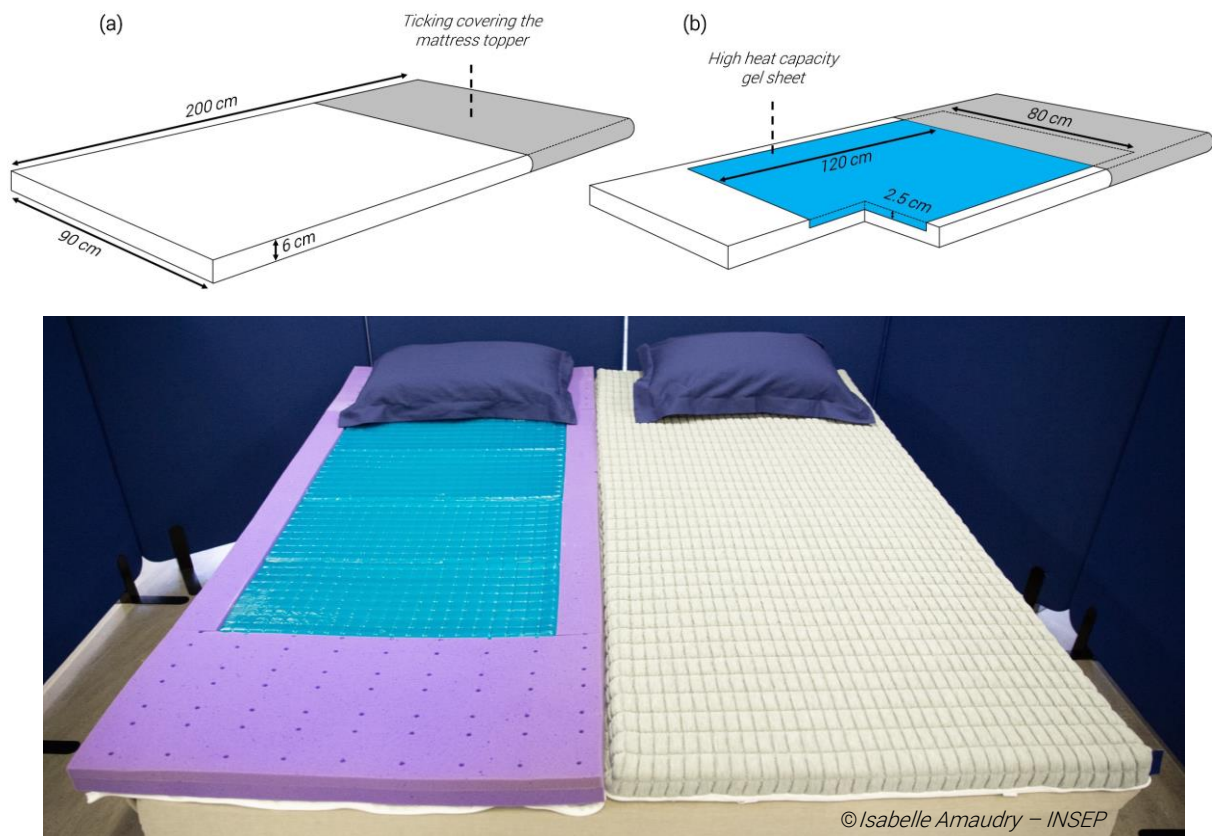


Figure 18. Figure illustrating the design of the low- (a) and high- (b) heat-capacity mattress topper. Picture represents the high-heat-heat-capacity mattress topper without (left) and with (right) the ticking.

3. Sleep monitoring

3.1. Polysomnography

3.1.1. Principle, relevance and limitation

Polysomnography (PSG) is the “gold standard” method for assessing sleep architecture and sleep disorders (American Academy of Sleep Medicine, 2014; Berry et al., 2017). It typically monitors sleep-related physiological signals including eye movement, brain activity, heart rate, muscle activity, oxygen saturation, breathing rate and body movements. PSG allows for the determination of the internal structure of sleep, i.e., NREM (N1, N2 and SWS) and REM sleep stages. However, PSG is an expensive (~US\$17k, plus software), time-consuming, invasive, complex and typically stationary laboratory-based technique that requires a qualified sleep technician to set up and manually score the sleep data. This method is also difficult to access and unsuitable for longitudinal monitoring in athletes (Halsom, 2019; Driller et al., 2023). Another disadvantage of PSG is the so-called “*first-night effect*”, which assumes that sleep architecture is disturbed during the first sleep assessment (Agnew et al., 1966). Nowadays, advanced technologies enable portable PSG devices to monitor sleep *in situ*, i.e., at home, in a familiar sleep environment while maintaining sleep habits, which is a promising method for elite athletes

that do not wish to attend a laboratory (Halson, 2019; Driller et al., 2023). Nevertheless, some elite athletes still appear to be more affected by the "first-night effect" than others in home-based PSG settings (Hof Zum Berge et al., 2021), suggesting at least two monitoring nights when investigating interventions that might influence sleep architecture in this population. Thus, in **study 2-B**, a night of familiarization with a portable home-based PSG equipment was performed in the same home environment as for the experimental nights. PSG was also used to ensure compliance with participant inclusion/exclusion criteria. In **study 3**, a reduced-montage PSG was performed without cannula and thermistor to minimize inconvenience in elite athletes.



Figure 19. Polysomnography montage using a portable device (Nox A1, ResMed, USA).

3.1.2. Technology used and data processing

In **studies 2-B** and **3**, PSG recordings were obtained using a portable device (Nox A1; ResMed, San Diego, California, USA). The same qualified practitioner equipped the participants with the PSG equipment. The montage included six EEG channels placed according to the international 10–20 electrodes placement system (F3-M2, F4-M1, C3-M2, C4-M1, O1-M2 and O2-M1), left and right EOG channels, chin (mentalis and submentalis) and bilateral tibial EMG channels, an electrocardiogram, and two respiratory impedances for rib cage and abdominal wall motion (**Figure 19**). The sleep data were scored using Noxturnal software (version 6.0; ResMed, San Diego, CA, USA) by one trained specialist (>3000 analyses in the last 10 years) who was blinded to the aim of the studies. The 2017 American Academy of Sleep Medicine (Berry et al., 2017) criteria were employed to establish the individual hypnograms from 30-s epochs scored as

wakefulness, N1 sleep, N2 sleep, SWS or REM sleep. Cortical arousals, leg movements and respiratory events were also identified, as defined in **Table 1** (page 31).

3.1.3. Sleep parameters

The continuity of sleep (TST, SOL, REM latency, WASO, sleep efficiency), its architecture (time and proportion spent in each sleep stage) and fragmentation (sleep stage shifts, awakenings >5 min, cortical arousals, limb movements) were the main PSG-based sleep parameters used in the present experimental studies. To further estimate the continuity and discontinuity of sleep architecture in **study 2-B**, homogeneous first-order Markov chain modelling were computed from each hypnogram. This assumes that the probability of a future sleep stage depends only on the current sleep stage (Kim et al., 2009). The Markov chain transition probability $Pr_{i \rightarrow j}$ from sleep stage i to sleep stage j over the whole sleep period was calculated as follows:

$$Pr_{i \rightarrow j} = (N_{i \rightarrow j} / T_i) \times 100,$$

where $N_{i \rightarrow j}$ is the number of transitions from sleep stage i to sleep stage j , and T_i is the total number of sleep stage i (Kishi et al., 2018). Models were computed using the “createSequenceMatrix” function of the “markovchain” R package, and the results are presented as transition matrices.

3.2. Actigraphy

3.2.1. Principle, relevance and limitation

Actigraphy is a non-invasive, practical, relatively low-cost (~US\$1000 per unit, plus software) activity monitor. It is typically worn on the nondominant wrist and records movement as a function of time using an accelerometer to identify whether an individual is awake or asleep for each 60-s period. This research-grade activity monitor has been validated against PSG for sleep-wake detection, making it suitable for both longitudinal (~2 weeks) research and clinical purposes (Sadeh, 2011; Meltzer et al., 2012; Smith et al., 2018; Liguori et al., 2023). It is important to note that actigraphy measures different dimensions of sleep compared with PSG. Although PSG measures various sleep-related physiological signals forming the basis of objective determination of sleep stages, actigraphy measures limb movement to infer wakefulness and sleep. Therefore, this method has limited capacity to infer sleep stages due to the lack of physiological signals, and cannot be used to assess sleep architecture (Yuan et al., 2024).

3.2.2. Technology used and data processing

In the framework of the present experimental studies, we used the MotionWatch 8 (CamNtech Ltd, Cambridge, UK) activity monitor. Raw acceleration data were processed by a specific algorithm (MotionWare Software; version 1.3.17; CamNtech Ltd, Cambridge, UK) following four stages (**Figure 20**): 1) filtering of the channel perpendicular to the watch (i.e., Z-axis, bandwidth: 3–11 Hz); 2) the peak acceleration (either positive or negative) during each second is recorded; 3) this is compared to a minimum “not moving” threshold of approximately 0.1g (values below this threshold are ignored); and 4) the result from each second is summed over a 60-s epoch. This sum is then compared with the sleep-wake sensitivity threshold chosen to score the epoch as “awake” or “asleep”.

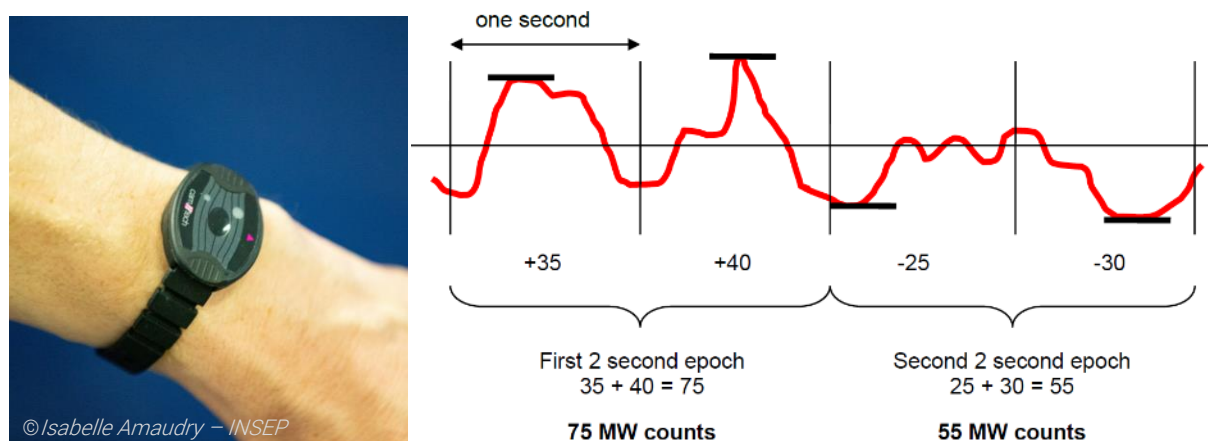


Figure 20. Left, MotionWatch 8 device and right, an illustration of data processing (refer to text, from CamNtech user guide). *Notes:* The red line corresponds to the time-course of recorded acceleration data from the Z-axis. Peak acceleration during each second is detected (indicated by the horizontal black bar), and then summed over a 60-s epoch.

3.2.3. Sleep parameters

Actigraphy provides objective measures of the sleep period, i.e., from bedtime to get-up time, which are not derived from the actigraphy data processing. Bedtime is the self-reported clock time when the participant attempts to sleep, while get-up time is when they stop attempting to sleep, both indicated by a marker button. Then, the sleep timing data are entered into the software to estimate sleep-wake patterns from the aforementioned actigraphy data processing as follows: TIB (min), the time between bedtime and get-up time; TST (min), the time spent asleep, determined from sleep onset to sleep offset, minus any waking time; WASO (min), the amount of time spent awake between sleep onset and sleep offset; sleep efficiency (%), TST expressed as a percentage of TIB; and SOL (min), the time between bedtime and sleep onset.

When the self-reported bedtime and/or get-up time did not match that of the sleep diary (see page 87), the night was excluded.

Importantly, actigraphy has been suggested as a reliable and accurate alternative to PSG for measuring these sleep parameters in an elite athlete population, but requires a threshold adjustment for detecting sleep (Sargent et al., 2016; Fuller et al., 2017). On the one hand, Sargent et al. (2016) reported that a high threshold (>80 activity counts = wake) provides the best combination of agreement (89.5%), sensitivity (91.9%) and specificity (67.0%), and generates the smallest mean biases for TST (-18 min), SE (+2%) and WASO (+4.1 min) compared with PSG in individual-sport athletes. On the other hand, a medium sleep-wake threshold (>40 activity counts = wake) demonstrates the smallest mean bias, i.e., 8.5 min for total sleep time, 1.8% for sleep efficiency and -4.1 min for wake after sleep onset, when compared with PSG in team-sport athletes (Fuller et al., 2017). Accordingly, a medium threshold was applied to the rugby union population in **study 1**, and a high threshold was applied to the population of individual-sport athletes recruited in **studies 2-A** and **3**.

3.3. EEG headband

As presented above, the research-grade tools for assessing sleep suffer from several limitations such as qualified expertise, significant costs and/or time-consumption. Actigraphy can compensate for this, but cannot assess sleep architecture. Since the early 2010s, the sleep-measurement market has been expanded by a growing number of consumer wearable sleep trackers. These devices claim to determine sleep-wake patterns and sleep stages, while being less expensive, more user-friendly and requiring no specific expertise, since sleep data processing and analysing is automatic (Driller et al., 2023). Wearable sleep trackers, combined with improved machine learning algorithms, offer promising advances in our understanding of sleep, in particular in free-living conditions (de Zambotti et al., 2019, 2023). However, it is important to note that, to date, many of these sleep monitors, such as Oura smart ring (ŌURA, Oulu, Finland), Whoop band (WHOOP, Boston, Massachusetts, USA), present insufficient scientific evidence for sleep staging because they do not record brain cortical activity (Depner et al., 2020). In addition, access to raw data and to the proprietary algorithms is not always possible (de Zambotti et al., 2019, 2023). Therefore, to assess the sleep architecture of rugby union population in **study 1** *in situ*, i.e., during a training camp, we decided to use a reduced-montage dry-EEG wireless headband (EEG headband, Dreem 3, Paris, France).

3.3.1. Principle, relevance and limitation

The EEG headband provides access to raw brain cortical activity data on a dedicated platform. This device proposes a cheaper (~US\$1500) and user-friendly alternative to PSG for sleep-related physiological signals acquisition and automatic sleep stage detection using a deep learning algorithm. This device has been demonstrated to have the capacity to measure sleep architecture similarly to sleep specialists (detailed below), which might therefore be of interest in longitudinal sleep studies in athletes in ecological conditions. However, it is important to note that in 2023, after the completion of **study 1**, the company was bought out, which raises questions about the long-term use of this device in a research setting, access to data and reproducibility of studies (de Zambotti et al., 2019, 2023). In addition, we also intended to use the EEG headband device in **study 2**, but some participants were reluctant to comply, given the longer duration of the protocol, i.e., 6 weeks. This tool can be uncomfortable for some users (maybe in part due to anatomic differences), and can sometimes move during sleep, resulting in artefacts that require additional processing to select the best-quality recordings (detailed below).

3.3.2. Data processing

Four types of physiological signals are recorded in real time: (1) brain cortical activity via five EEG dry electrodes with high-consistency silicone with soft, flexible protuberances yielding five derivations (F7, F8, Fp1, O1, O2 according to the International 10–20 system; 250 Hz with a 0.4–35 Hz bandpass filter), and (2-4) movements, head position and breathing frequency by a 3D accelerometer located over the headband (**Figure 21**). Following the recording, raw data are transferred via Bluetooth to a dedicated mobile application or via Wi-Fi to the sponsor's servers, where a proprietary algorithm enables automatic sleep stage analysis.

Automatic sleep stage analysis enables in two phases: (1) feature extraction in 30-s epochs from the relative EEG spectral power computed in frequency bands relevant for sleep analysis ($\lambda = 0.5\text{--}4$ Hz, $\theta = 4\text{--}8$ Hz, $\alpha = 8\text{--}14$ Hz, and $\beta = 15\text{--}30$ Hz), and (2) classification to establish a hypnogram from 30-s epochs scored as wake or sleep stages (N1, N2, N3 or REM). The signal acquisition of the EEG headband and the performance of its embedded automatic sleep staging algorithms have been validated by the manufacturer's scientific team compared to the consensus of five independent sleep specialists using a medical-grade polysomnography (Arnal et al., 2020). Automatic sleep-staging classification performed by the EEG headband was

similar (overall accuracy: $83.5 \pm 6.4\%$) to the inter-scoring accuracy achieved with PSG by sleep specialists (overall accuracy: $86.4 \pm 8.0\%$).

The EEG headband recordings were then processed as follows to select the best-quality recordings, using a similar approach from Knufinke et al. (2018): (1) bedtime and get-up time were adjusted according to actigraphy and sleep diaries as reference; (2) if there were missing epochs at the beginning and end of the night, they were manually added as “undefined”; (3) all “undefined” epochs prior to sleep onset were manually scored as “wake”; (4) REM sleep epochs within the first 30 min of the recording without succeeding SWS were manually scored as “undefined” to be consistent with the normative range of REM sleep latency (49.5–278.5 min; (Mitterling et al., 2015)); and (5) recordings containing more than 15% “undefined” epochs were excluded.



Figure 21. Illustration of the EEG headband and the location of sensors: (A) 2 EEG frontal electrodes, (B) 1 ground EEG electrode, (C) 2 occipital EEG electrodes, (D) bone conduction speaker for audio output, and (E) 3D accelerometer.

3.3.3. Sleep parameters

Based on this data processing and similar to PSG, the continuity of sleep (TST, SOL, REM latency, WASO, sleep efficiency), its architecture (time and proportion spent in each sleep stage) and fragmentation (sleep stage shifts) were calculated as defined in **Table 1** (page 31).

4. Self-report measures

This section presents the questionnaires and declarative measures used in the three experimental studies, which have been performed using online software for conducting surveys (Limesurvey GmbH, 2003) and the Smartabase player-management system (Fusion Sport, 2022).

4.1. Sleep diaries and questionnaires

Sleep diaries and questionnaires represent easy-to-use and cost-effective self-assessment methods to determine the individual's sleep profile and sleep hygiene. They are often used as the first sleep diagnostic test (Ibáñez et al., 2018), by encompassing, for instance, perceived sleep quality and sleep complaints, preferences and habits. Therefore, the following sleep diary and questionnaires were employed in the present experimental studies, as inclusion/exclusion criteria to constitute a homogeneous population (i.e., **study 2**) and to characterize participants (i.e., **studies 1** and **3**). It is important to note that they do not provide information on sleep architecture or sleep-wake patterns. We used them in conjunction with objective tools (PSG, actigraphy and/or EEG headband) to provide a more comprehensive overview of sleep quantity and quality.

4.1.1. Sleep diary and Spiegel Sleep Inventory

A French sleep diary was completed every day for assessing sleep-wake patterns, daytime routines and subjective information (Carney et al., 2012; Anderson, 2018). Upon waking, participants informed bedtime, estimated time asleep and get-up time. They also informed any daytime napping and lifestyle factors such as caffeine and alcohol consumption, meals and the use of electronic devices. An electronic version of the sleep diary has been shown to perform like a paper sleep diary while facilitating data processing and analysis (Tonetti et al., 2016).

Daily perceived sleep quality was completed in all experimental studies within 30 min upon waking using the *Spiegel Sleep Inventory* (SSI). It is composed of six questions scored from 1 to 5 about sleep initiation, the quality and duration of sleep, nocturnal awakenings, dreams, and feeling refreshed in the morning. The overall score is the sum of the six items, and a higher score indicates a better sleep quality. Few data are available on the psychometric validity of the SSI among athletes, but it is a very simple and easy-to-use scale that is often used to assess the presence of insomnia (Léger et al., 2006). The SSI was used in all experimental studies by including it in the sleep diaries.

4.1.2. Sleep questionnaires

Pittsburgh sleep quality index (PSQI)

The PSQI is a 19-item self-report questionnaire that assesses 7 components of the sleep quality and disturbances in adults over the last month: perceived sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, the use of sleep medications, and daytime dysfunction. The global score ranges from 0 to 21, and a score ≥ 5 indicates “poor sleep” quality (Buysse et al., 1989), although a more conservative score ≥ 8 is sometimes used (Samuels, 2008). The PSQI is considered as the most rigorously validated tool used as first sleep diagnostics (Manzar et al., 2018). It has been suggested that this questionnaire may overestimate sleep disturbances due to a high proportion (~50%) of athletes’ score ≥ 5 on the PSQI indicating “poor sleep” (Samuels, 2008; Gupta et al., 2017; Halson et al., 2021b). The PSQI does not consider the impact of sport-specific factors likely to affect sleep such as frequent travels, high training loads and stress associated with competition over the season (Walsh et al., 2020), which is critical in our sport-specific context. In this respect, the Athlete Sleep Screening Questionnaire (Samuels et al., 2016; Bender et al., 2018) and the Athlete Sleep Behaviour Questionnaire (Driller et al., 2018) have been developed elsewhere to specifically examine sleep in athletes with a reduced number of questions, and to guide a follow-up strategy based on responses to specific items. However, we did not use these questionnaires in our experimental studies, as only Athlete Sleep Behaviour Questionnaire was validated in French-language (Baize et al., 2023), but after initiating our experiments. Consequently, the use of the French version of the PSQI (Blais et al., 1997) was favoured throughout the experimental studies by its current usage in both research and practice, and the availability of normative data provided in individual and team sport athletes (Halson et al., 2021b).

Morningness-eveningness questionnaire (MEQ)

The MEQ consists of 19 self-assessed questions that assesses an individual’s preferences for completing activities at various times of the day to determine chronotype. The chronotype classification is based on the sum of the scores for each question, as follows: 16–30 = definitely evening type; 31–41 = moderately evening type; 42–58 = neither type; 56–69 = moderately morning type; and 70–86 = definitely morning type (Horne and Ostberg, 1976). The French version of this questionnaire was used in the present experimental studies (Bessot and Margas, 2024).

Epworth sleepiness scale (ESS)

The *Epworth Sleepiness Scale* (ESS) is a self-administered questionnaire that assesses a general daytime sleepiness based on retrospective reports of the likelihood of dozing off or falling asleep in 8 standardized daily situations. For each situation, respondents are asked to rate their usual chances of dozing off or falling asleep on a 0 to 3 scale. The global score ranged from 0 to 24, with scores ≥ 10 reflecting excessive daytime sleepiness (Johns, 1991). Daytime sleepiness is not necessarily a sleep problem, but can be viewed as a proxy consequence of sleep disturbance, as it is common in unhealthy sleep (Lok and Zeitzer, 2021). The French version of the ESS was used in the present experimental studies (Kaminska et al., 2010).

Ford insomnia response to stress test (FIRST)

The FIRST is a self-rated 9-item scale used to assess an individual's likelihood of sleep disruption due to common specific stressful situations and more broadly described periods of stress occurring during the day or evening (e.g., after getting bad news during the day, after a stressful experience in the evening). Items are scored on a 4-point Likert scale, and the total score ranges from 9 to 36; higher scores indicate higher levels of sleep hyperarousal (Drake et al., 2004). The French version of FIRST was employed (Chen et al., 2015). One limitation of the FIRST questionnaire is its inclusion of items that inquire about the likelihood of sleep disruption due to common stressful situations, yet it lacks specific information tailored to athletes. In this regard, we developed and validated the Insomnia in Response to Sports-related Stress Test (IRSST) questionnaire, a new specific instrument aimed at sensitively measuring vulnerability to sport-specific stressful situations among elite athletes (Nédélec et al., 2024). However, since the validation study was published after the initiation of our experiments, we did not use this questionnaire in the present experimental studies.

Insomnia severity index (ISI)

The French version of the *Insomnia severity index* (ISI) was also used to evaluate insomnia complaint severity (Bastien et al., 2001). The ISI is a 7-item self-report questionnaire assessing the nature, severity, and impact of insomnia over the last month: severity of sleep onset, sleep maintenance, and early morning awakening problems, sleep dissatisfaction, interference of sleep difficulties with daytime functioning, noticeability of sleep problems by others, and distress caused by the sleep difficulties. Respondents rate their experiences with insomnia using a 5-point Likert scale, from 0 to 4, yielding a total score ranging from 0 to 28 interpreted

as follows: 0–7 = absence of insomnia; 8–14 = sub-threshold insomnia; 15–21 = moderate insomnia; and 22–28 = severe insomnia (Morin et al., 2011).

4.2. Daily subjective wellness ratings

Subjective wellness ratings were collected upon waking. In **studies 1** and **3**, participants were asked to rate their perceived general fatigue, general pain (including DOMS) and stress using a visual 0–10 analog scale via a sliding cursor (from “no pain”, “no fatigue” or “no stress” to “unbearable pain”, “very, very tired” or “very, very stressed”). In **study 2-A**, participants were asked to rate their muscle soreness, general fatigue and stress using the Hooper 1-7 scale (Hooper and Mackinnon, 1995).

4.3. Thermal and bedding perception

In studies assessing the effectiveness of HMT on sleep (i.e., **studies 2-B** and **3**), perceived thermal comfort (on a scale from -2 “very uncomfortable” to +2 “very comfortable”) and sensation (on a scale from -3 “cold” to +3 “hot”) (Zhang and Zhao, 2008) during the night were recorded upon waking. This was performed following the usual training, overload and taper periods in **study 2-B** (**Figure 28**, page 138), and each morning in **study 3**.

In **study 3**, perceived bedding comfort was reported on the morning of the second night of each condition using a visual analog scale via a sliding cursor ranged from 0 (“not at all comfortable”) to 10 (“very, very comfortable”). On the morning of the last night of each condition, players reported the perceived effectiveness of bedding by answering the question “Did the mattress topper you used this week improve your sleep?” on a 7-point Likert-type scale ranging from 1 (“strongly disagree”) to 7 (“strongly agree”).

5. Training load quantification

5.1. Internal training load

In **studies 1** and **2**, the sRPE was recorded within 30 min from the end of each training session using the modified CR-10 scale (Foster et al., 2001), which participants were all accustomed. The daily internal training load (A.U.) was calculated by multiplying sRPE and the duration of each training session within a day (sRPE × duration). On rest days, internal training load was scored as 0. In **study 2**, the weekly training load (A.U.) was determined as the sum of the daily training loads for each week.

5.2. External training load

To record individual time-motion activity in **study 1**, rugby union players wore a portable micro-electromechanical system (mass: 67 g, size: 88 × 50 × 19 mm, MinimaxXTM S4, Catapult Innovations, Australia) during each rugby training and match session. This tool contains a 10-Hz GPS, a 100-Hz triaxial accelerometer, a gyroscope and a magnetometer. The micro-electromechanical system was positioned in a Lycra vest on the upper thoracic spine between the scapulae. To avoid any inter-device variability, players kept the same micro-electromechanical system throughout the training camp (Coutts and Duffield, 2010). Total and relative distance covered per minute, at low- (<18 km.h⁻¹) and high-speed (HSD, >18 km.h⁻¹), and high-intensity acceleration ($\geq 2.5 \text{ m.s}^{-2}$) and deceleration ($\leq -2.5 \text{ m.s}^{-2}$) were used to assess the locomotor load according to previously-established thresholds (Lacome et al., 2017; Harper et al., 2019). These external training load metrics represent the most important components associated with rugby union-related metabolic and neuromuscular fatigue (Naughton et al., 2021). The accuracy and reliability of GPS units to measure these movement patterns have been demonstrated as “acceptable” in team sports (Varley et al., 2012; Rampinini et al., 2015; Scott et al., 2016). Finally, collision events have been detected from the micro-electromechanical system, which have been shown to be accurate (93%), sensitive (94%), and specific (93%) in identifying collision events in professional rugby union players (MacLeod et al., 2018). On rest days, external training loads metrics were scored as 0.

6. Temperature measurements

In studies assessing the effectiveness of HMT on sleep (i.e., **studies 2 and 3**), CBT and bedroom ambient temperatures were measured.

6.1. Core body temperature

At least three hours before bedtime, participants ingested a radio telemetric pill (BodyCap; e-Celsius® Performance) to continuously record the nocturnal CBT with a sampling rate of 1/min. The sensor is reliable and valid, with a mean difference of -0.08°C compared to reference hot water bath temperatures within physiological ranges (33–44°C), a low standard error of measurement (0.04°C), an intraclass correlation coefficient of 1.00, and a mean agreement between test and retest trials of $-0.01 \pm 0.03^\circ\text{C}$ (Bongers et al., 2018). The nocturnal CBT data were averaged and displayed in 10-min time bins.

6.2. Bedroom ambient temperature

The measurement of the ambient temperature was performed using an iButton (DS 1922L, iButtonLink, Whitewater, USA; resolution 0.0625°C; sampling rate: 4 values per hour) placed on the wall 1 m above the bed. The average temperature from bedtime to getting up in the morning was calculated for each night.

Study 1

Morning appointment time rather than training load affects sleep during a moderate-intensified training camp in young elite rugby union players

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Associated publication

Under review

Associated communications

Chauvineau, M., Mathieu, B., Boissard, G., Piscione, J., Duforez, F., Guilhem, G., and Nedelec, M. (2023). Impact d'un stage d'entraînement sur les rythmes veille-sommeil et l'architecture du sommeil de jeunes joueurs de rugby de haut niveau. *Congrès du sommeil, Lille, France. **Poster communication***

Abstract

This investigation aimed to evaluate sleep of young rugby union players during a 10-day training camp accounting for the potential influence of prior daily training load and the morning appointment time. Twenty-six elite male under-20 rugby union players were monitored each day during a 10-day training camp including two exhibitions matches. Sleep was assessed using actigraphy, a reduced-montage dry-electroencephalographic headband device and sleep diaries, training load and subjective fatigue, pain and stress were evaluated using GPS trackers and 10-score visual analog scales, respectively. Overall, the prevalence of nights with sleep duration <7 h (30.3%), WASO >40 min (77.8%) and sleep efficiency <85% (43.4%) was high. Linear mixed-effects models revealed that for every additional 100-m covered at high-speed >18 km.h⁻¹, subsequent sleep duration was promoted ($\beta = +4.9$ min, $p < 0.05$) and the number of sleep stage shift was reduced ($\beta = -1.1$, $p < 0.05$). Later bedtime increased perceived fatigue ($p < 0.05$). The shortest sleep duration (06:52 \pm 00:34 h) occurred on the day of Match 1, when the morning appointment was the earliest, i.e., from 7:30 to 8:30. Sleep duration (+17.2 min, $p = 0.01$) and efficiency (+2.1%, $p < 0.01$) were highest when morning appointment was scheduled at 8:00-9:00 compared with 8:30-9:30. This study supports that training camp is a vulnerable period for sleep, but a controlled and not excessive training load did not contribute to disturb sleep. Coaches should be aware of the negative impact of earlier and/or unusual morning appointment time on sleep, especially in proximity to a match.

Keywords: athletes, training program, recovery, team sports, sleep architecture

Introduction

Rugby union is an intermittent team-sport, characterised by periods of high-intensity running activities (e.g., high-speed runs, changes of direction, rapid accelerations and decelerations) and collision-based actions (e.g., tackling, static holds, scrums, rucks and mauls) interspersed with low intensity activities (Till et al., 2020). This high-intensity intermittent and combative nature of rugby leads to notable muscle damage resulting in biochemical, metabolic and neuromuscular alterations that increase physiological fatigue, reduce subjective wellness, and can impair athletic performances (Tavares et al., 2017; Naughton et al., 2018, 2021). To adequately prepare for such substantial physical demand, elite rugby union teams undergo training camps that expose them to repeated strenuous training sessions. In this context, adequate sleep is essential to facilitate player recovery given its well-established physiological and restorative functions (Halson and Juliff, 2017; O'Donnell et al., 2018a).

Despite this importance of sleep for athletes, training camps have been identified as a vulnerable period for sleep in team-based contact sports, with some evidence for impairments in sleep-wake patterns, i.e., TST, WASO and/or sleep efficiency (Pitchford et al., 2017; Thornton et al., 2017). One of the putative reasons for such alterations in sleep during training camps is the high training load that players can experience (Roberts et al., 2019b; Walsh et al., 2020). In Australian rules football players, Pitchford et al. (2017) showed that daily increases in training load, assessed using the sRPE \times training duration, were detrimental to subsequent sleep duration only during a training camp, and not during a similar training period at home. Sleep duration was also negatively correlated with prior daily total distance covered during a rugby league training camp (Thornton et al., 2017). However, the negative impact of increased training load on sleep is not consistent across studies, as some evidence has shown that the rugby-specific physical demands encountered during training (e.g., total distance covered and HSD, acceleration/deceleration demands, collisions) can have varying effects on sleep-wake patterns (Thornton et al., 2018; Leduc et al., 2019). Moreover, to the best of our knowledge, only one study has highlighted the varied effect of the combative and high-intensity intermittent demands of rugby union on sleep architecture, i.e., the internal structure of sleep, using a single night PSG (Aloulou et al., 2020b). Increased HSD ($>18 \text{ km}\cdot\text{h}^{-1}$) was associated with subsequent lower light sleep in favour to higher SWS proportions and, conversely, increased number of collisions resulted in higher light sleep (N1 + N2 sleep) proportions. However, the putative effect of varied daily training demands on sleep architecture in young elite rugby union players throughout a training camp has not currently been investigated. Forward players are more likely

to experience a larger number of collisions than back players (Naughton et al., 2020). The difference in sleep characteristics between playing positions in young elite rugby union players is yet unclear. It is also unclear how potential changes in sleep may affect wellness, such as perceived fatigue, pain, and stress, in this population.

Another important consideration for sleep disturbances is the often-hectic training camp program (Nedelec et al., 2018). Camp situations are often accompanied by a change in sleeping environment and unaccustomed schedules, with several training sessions a day, sometimes exhibition matches, and commitments outside of training (e.g., meetings, working sessions and collective lunches, travels), which may affect sleep behaviours, i.e., bedtime and get-up time (Pitchford et al., 2017; Thornton et al., 2017). Some studies have demonstrated that morning appointment times strongly determines the prior sleep duration in various sports (Sargent et al., 2014a, 2014b; Steenekamp et al., 2021). However, no study has explored how sleep of young rugby union players is affected by morning appointment time during a training camp, while accounting for the potential effect of training load on sleep. Answering this question would provide some rationale for practitioners to adapt the training camp program.

The present study aimed to evaluate sleep-wake pattern and sleep architecture of young rugby union players during a 10-day training camp accounting for the potential influence of prior daily training load and the morning appointment time. We hypothesized that early morning appointment may impair sleep duration, and that higher daily training load may both disturb subsequent sleep-wake patterns and promote sleep architecture.

Materials and methods

Participants

Twenty-six elite male under-20 rugby union players, i.e., 14 forwards (mean \pm SD; age: 18.8 ± 0.2 years; body mass = 104.7 ± 8.7 kg; height = 188.2 ± 7.5 cm) and 12 backs (age: 18.6 ± 0.4 years; body mass = 84.4 ± 7.8 kg; height = 178.7 ± 5.3 cm), from various clubs, participated in the protocol. Players were classified at “Elite/International Level” (Tier 4) according to the guidelines proposed by McKay et al. (2022), as they were all competing at the highest national French level from the national team. Players were excluded ($n = 0$) in the case of medical circumstances influencing training frequency, taking medication for sleep disorders, and travel involving crossing of several time zones in the 2 weeks preceding the protocol.

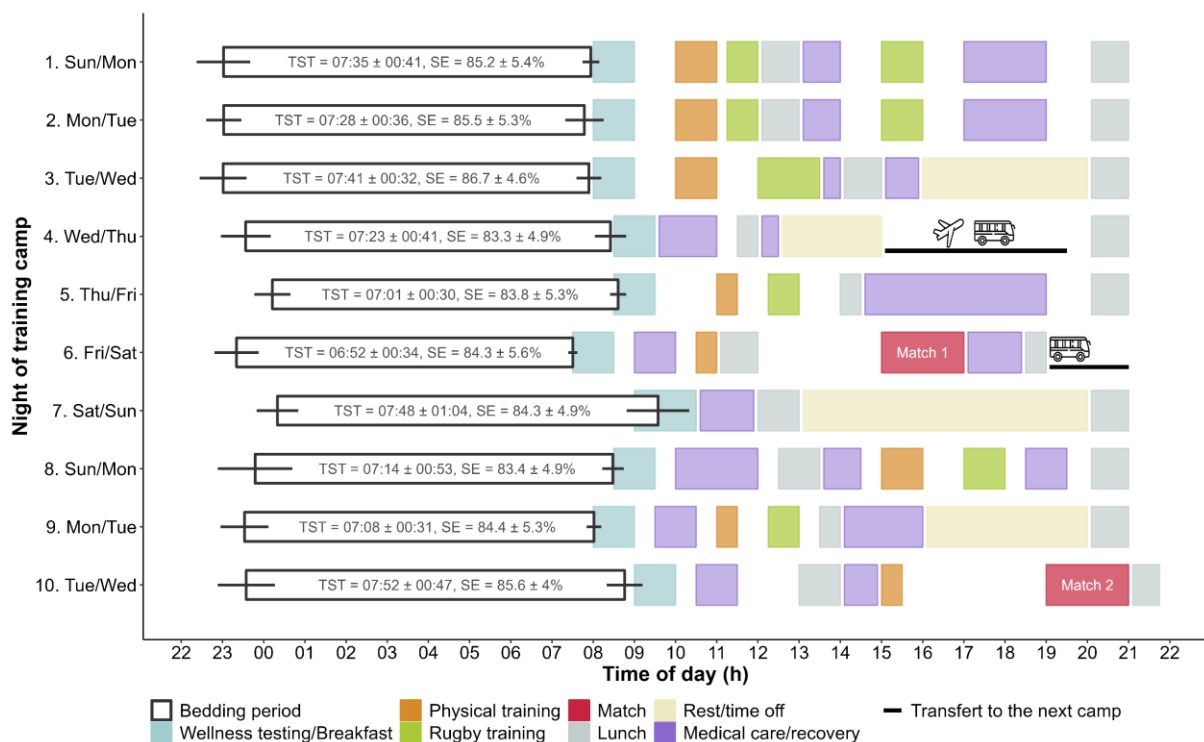


Figure 22. Sleep-wake patterns according to the training camp timeline. *Notes:* Each line represents a 24-h training camp day from 22:00 to 22:00. White bars indicate the mean \pm SD start and end times of bedding periods. Coloured bars indicate the day's scheduled events. *Abbreviations:* TST, total sleep time (h:min); SE, sleep efficiency. Data are reported as mean \pm SD.

Training camp

Sleep, training load and subjective ratings were monitored on a daily basis during a 10-day training camp as part of a national team convocation, interspersed with two exhibition matches (days 6 and 10). The training camp took place in April 2023 in a familiar camp for days 1–4, then two different locations days 5–6, and for days 7–10, with no change in time zone. The first journey was completed by plane and bus (4 h), and the second by bus only (2 h). The program was developed by the squad's coaches (**Figure 22**) and consisted of 6 training days (days 1–3, 4, 8–9), two rest days (4, 7), and 2 exhibition match days (days 6, 10), kicking off at 15:00 and 19:00, respectively. The training program for days 1–3 focused on high-intensity training sessions including collisions. Then, a reduction in intensity and number of training sessions was emphasised, interspersed with the two exhibition matches. At the same time, the number of rest and time off periods increased.

Players slept in shared twin rooms with separate beds, and the pairing remained unchanged throughout the training camp. Players had free choice of bedtime. Each morning they had a 1-hour window, i.e., morning appointment time, during which they completed the subjective ratings of sleep quality, general pain, fatigue and stress, and ate breakfast. Following the lunch break, players had a rest period (from 30 min to 1 h) and the possibility of taking a nap. Players

were not restricted from consuming caffeine, alcohol, or sleep medication throughout the camp.

At the start of the training camp, all players received a wristwatch actigraphy worn from before going to bed to getting up in the morning to record sleep-wake patterns and an electronic sleep diary in which to report the daily subjective ratings. No data were monitored after the second match, which marked the end of the training camp and the return of the players to their respective clubs. To assess sleep architecture, 13 forward players also received an EEG headband, with the first night considered as a familiarization (Hof Zum Berge et al., 2021) and not included in further analysis. We chose to equip only forward players because they are more likely to complete a larger number of collisions than back players (Naughton et al., 2020), which may subsequently influence sleep architecture (Aloulou et al., 2020b). All players were asked to complete the following sleep questionnaires: (a) the MEQ to identify the individual's chronotype (Horne and Ostberg, 1976), (b) the ESS to assess daytime sleepiness (Johns, 1991), (c) the PSQI to evaluate the perceived sleep quality (Buysse et al., 1989), (d) the FIRST to measure the vulnerability of sleep disturbance in response to common stressors (Drake et al., 2004), (e) and the ISI to assess severity of insomnia symptoms (Morin et al., 2011).

Statistical analysis

Data processing and statistical analyses were conducted using R software (version 4.3.2). The proportion of nights outside the National Sleep Foundation's recommendations (Hirshkowitz et al., 2015b; Ohayon et al., 2017) were reported as percentage. The between-playing positions comparisons in sleep questionnaire scores and average sleep data over the training camp were conducted using a Student *t*-test comparison. The chi-squared test was used to compare chronotype proportions between the playing positions. No obvious deviations from normality of data distribution and the homogeneity of variance were reported using the Shapiro–Wilk and Levene tests, respectively. Sleep data (i.e., sleep-wake patterns, sleep architecture and SSI score) were analysed using linear mixed-effects models (“lmer” function of the “lme4” R package) to consider the potential missing data and the non-independence of observations at the level of players including the player as random effect. A first linear mixed-effects model was performed to assess the relationships between sleep data (i.e., sleep-wake patterns and SSI score) and the following variables as fixed effects: the morning appointment time (categorical predictor) and the prior training load metrics as continuous predictors (i.e., HSD, number of collisions, number of high-intensity accelerations and high-intensity decelerations). A separate

linear mixed-effects model was used to evaluate the relationships between sleep architecture among forward players and the prior training load covariates, adjusted for the morning appointment time. Variance inflation factor was used to assess collinearity issues between variables, and all variance inflation factors were <6 , indicating a low collinearity. The significance of the morning appointment time and the playing positions effects was determined using the “mixed” function from the “afex” R package, using type 2 test. Post-hoc Student *t*-test pairwise comparisons were conducted when a significant main effect was reported, using least square means contrasts, with the “Tukey” method for alpha adjustments and a Kenward-Roger adjustment for the degrees of freedom calculation (“emmeans” function of the “emmeans” package). Differences were assessed using effect sizes (ES), and interpreted using the following criteria: $<0.2 = \textit{trivial}$, $0.2-0.49 = \textit{small}$, $0.5-0.79 = \textit{moderate}$, and $\geq 0.8 = \textit{large}$ (Cohen, 1988). A second linear mixed-effects model was fitted to evaluate the relationships between sleep data (i.e., sleep-wake patterns, sleep architecture and SSI score) and subsequent subjective ratings, including general fatigue, pain intensity and stress as fixed effects. For each linear mixed-effects model, the normality of residuals was visually checked using a Q–Q plot, and no obvious deviations from normality were revealed. The descriptive data are reported as the mean \pm SD, and linear mixed-effects models results are presented as the estimate (Est.), 95% confidence interval (CI) and p-value. The level of significance was set at $p < 0.05$.

Results

The 10-day training camp resulted in a total of 198 out of 260 (i.e., 76.2%) possible actigraphy-based sleep recordings, with missing data due to participants forgetting to wear the actigraphy of some nights or conflicts between recorded bedtimes and wake-up times and those in the sleep diary. A total of 45 out of 117 (i.e., 38.5%) possible EEG headband-based sleep recordings was obtained. Missing data were due to mismatches with bedtimes and get-up times from actigraphy and sleep diaries, recordings containing more than 15% “undefined” epochs, or players not wearing the EEG headband. One player did not present actigraphy data and two other players did not show EEG headband data throughout the training camp, and their sleep data were not included in further analysis.

The mean \pm SD scores from sleep questionnaires were below the cut-off suggesting sleep complaints (ESS: 9.5 ± 4.4 A.U.; PSQI: 3.5 ± 1.4 A.U.; FIRST: 16.5 ± 4.8 A.U.; ISI: 4.9 ± 3.9 A.U.), with no significant differences between playing position (all $p > 0.51$). One player reported perceived “poor sleep quality” and one other player reported “moderate insomnia” based on

global PSQI score ≥ 5 and ISI score ≥ 15 , respectively. Excessive daytime sleepiness (ESS score ≥ 10) was observed in 10 players. The Morningness–Eveningness Questionnaire revealed that players were either definitely morning type ($n = 1$), moderately morning type ($n = 6$), neither type ($n = 15$) or moderately evening type ($n = 2$) with no significant differences between playing position ($p = 0.25$). Morning appointment time varied from 7:30–8:30 ($n = 1$), 8:00–9:00 ($n = 4$), 8:30–9:30 ($n = 3$) to 9:00–10:00 ($n = 2$) (**Figure 22**). Daily changes in training load metrics are shown in **Table 4**.

Sleep during the training camp

Average sleep data over the training camp are presented in **Table 5**. Overall, 30.3% of nights showed a TST < 7 h, 77.8% had WASO > 40 min, 43.4% had sleep efficiency $< 85\%$, and 20.7% had SOL > 30 min. The average TST was > 7 h for all nights (mean \pm SD: 07:24 \pm 00:44 h), except for the night prior to Match 1, during which the players obtained 06:52 \pm 00:34 h of sleep, alongside the earliest get-up time (7:30 \pm 00:06, **Figure 22**). No significant differences between playing positions were observed in any sleep-wake patterns, except for TIB, which was longer for the back players than for the forward players (+18.6 min, ES = 0.49, $p = 0.04$). Linear mixed-effects models revealed, irrespective of the prior training load covariates, a significant main effect of the morning appointment time on prior bedtime ($F_{(3, 171)} = 29.4$, $p < 0.001$), get-up time ($F_{(3, 169)} = 137.2$, $p < 0.001$), TIB ($F_{(3, 171)} = 11.8$, $p < 0.001$), TST ($F_{(3, 174)} = 10.1$, $p < 0.001$) and sleep efficiency ($F_{(3, 171)} = 3.6$, $p = 0.01$, **Table 6**). **Figure 23** presents the post-hoc analyses of changes in sleep-wake patterns based on the morning appointment time. No significant main effect of the morning appointment time was found for the other sleep variables.

Relationships between sleep and prior daily training load and subjective ratings

Table 6 and **Table 7** provides an overview of the potential relationships between sleep and prior training load and subjective ratings covariates, independently from the morning appointment time. Every additional 100-m in HSD was associated with subsequent increases in TIB ($\beta = +4.5$ min [95% CI, 0.5 to 8.5 min], $p < 0.05$) and TST ($\beta = +4.9$ min [0.6 to 9.2 min], $p < 0.05$), as well as a reduction in the number of sleep stage shift per hour of sleep ($\beta = -1.1$ [-2.1 to -0.2], $p < 0.05$). Each additional 1 A.U. in general fatigue was associated with a later bedtime ($\beta = +4.3$ min [0.3 to 8.3 min], $p < 0.05$), a higher WASO ($\beta = +1.7$ min [0.2 to 3.1 min], $p < 0.05$), and a lower SSI score ($\beta = -0.6$ A.U. [-0.8 to -0.4 A.U.], $p < 0.001$). No significant relationships were observed between sleep and the other training load and subjective ratings covariates.

Table 4. Changes in training load metrics throughout the training camp.

Day	Total distance (m)	Relative distance (m.min ⁻¹)	HSD (m)	Collisions (n)	HIA (n)	HID (n)	Perceived training load (A.U)
1. Mon	2934.4 ± 1189.5	39.8 ± 16.1	170.9 ± 137.1	11.5 ± 7.3	17.7 ± 9.9	13.3 ± 6.8	449.3 ± 174.9
2. Tue	3620.2 ± 417.9	57.6 ± 6.6	441.0 ± 174.7	12.5 ± 5.8	17.9 ± 8.8	10.3 ± 6.0	551.4 ± 62.7
3. Wed	3710.5 ± 487.9	49.6 ± 7.8	370.8 ± 209.9	15.4 ± 6.0	19.3 ± 9.3	13.9 ± 7.1	702.5 ± 93.9
4. Thu	Rest						
5. Fri	2569.7 ± 315.3	56.5 ± 6.9	264.3 ± 70.8	7.7 ± 3.4	11.0 ± 6.0	7.3 ± 3.9	341.5 ± 64.6
6. Sat (Match)	5738.8 ± 1154.4	65.7 ± 6.7	599.5 ± 272.7	22.4 ± 8.2	28.2 ± 13.0	20.4 ± 10.9	660.1 ± 135.7
7. Sun	Rest						
8. Mon	3382.4 ± 467.7	42.3 ± 5.9	161.5 ± 101.2	5.8 ± 5.0	18.0 ± 12.5	12.0 ± 8.5	545.4 ± 95.7
9. Tue	2673.5 ± 202.1	59.4 ± 4.3	253.5 ± 75.8	7.8 ± 4.1	14.8 ± 6.5	8.3 ± 4.1	355.4 ± 82.1
10. Wed (Match)	5601.1 ± 1495.2	65.8 ± 7.5	516.7 ± 265.0	23.3 ± 12.3	31.5 ± 14.2	23.0 ± 11.5	674.4 ± 128.5

Abbreviations: HSD, High-speed distance >18 km.h⁻¹; HIA, High-intensity acceleration ≥2.5 m.s⁻²; HID, High-intensity deceleration ≤-2.5 m.s⁻²; A.U., Arbitrary unit. Data are reported as mean ± SD.

Table 5. Descriptive statistics (mean \pm SD) of sleep data over the training camp according to playing positions.

	Overall	Backs	Forwards	Playing position differences
<i>Sleep-wake patterns</i>				
Bedtime (h:min)	23:29 \pm 00:44	23:24 \pm 00:43	23:32 \pm 00:44	ES = -0.23, p = 0.38
Get-up time (h:min)	08:13 \pm 00:36	08:18 \pm 00:33	08:09 \pm 00:37	ES = 0.27, p = 0.18
TIB (h:min)	08:44 \pm 00:41	08:54 \pm 00:38	08:37 \pm 00:42	ES = 0.49, p = 0.04
TST (h:min)	07:24 \pm 00:44	07:27 \pm 00:44	07:21 \pm 00:43	ES = 0.24, p = 0.32
WASO (min)	56.7 \pm 19.7	60.2 \pm 22	54.2 \pm 17.5	ES = 0.29, p = 0.59
Sleep efficiency (%)	84.7 \pm 5.1	83.8 \pm 5.7	85.4 \pm 4.5	ES = -0.26, p = 0.47
SOL (min)	20.3 \pm 20.6	21.8 \pm 22.6	19.2 \pm 18.9	ES = 0.12, p = 0.51
SSI score (A.U.)	22.8 \pm 3.1	23.3 \pm 3.3	22.3 \pm 2.9	ES = 0.41, p = 0.14
<i>Sleep architecture</i>				
Light sleep (N1 + N2 - %)	–	–	51.0 \pm 6.6	–
SWS (%)	–	–	24.1 \pm 6.8	–
REM (%)	–	–	25.0 \pm 5.0	–

Abbreviations: TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset; SOL, sleep onset latency; SSI, Spiegel sleep inventory; A.U., arbitrary unit; N1–N2, NREM sleep stages 1–2; SWS, slow wave sleep, REM, rapid eye movement sleep stage; ES, effect size: <0.2 = *trivial*, 0.2–0.49 = *small*, 0.5–0.79 = *moderate*, and \geq 0.8 = *large*.

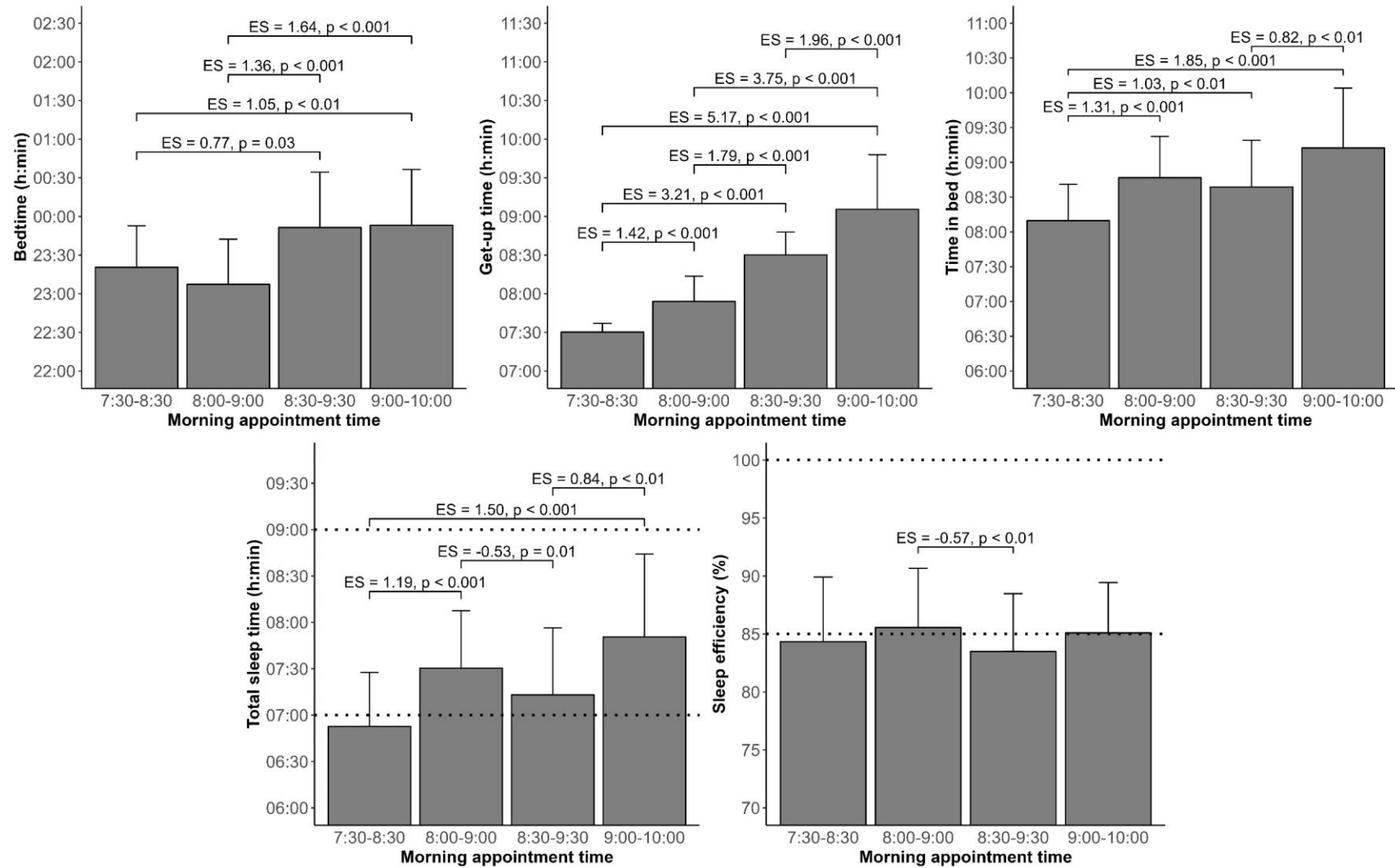


Figure 23. Changes in sleep-wake patterns (mean \pm SD) according to the morning appointment time. *Notes:* Dotted lines denote the range of recommended sleep duration and sleep efficiency. *Abbreviations:* ES, effect size: <0.2 = trivial, 0.2–0.49 = small, 0.5–0.79 = moderate, and \geq 0.8 = large.

Table 6. Linear mixed-effects models evaluating the relationship between the daily training load metrics, subjective ratings, and sleep-wake patterns.

	Bedtime (min)		Get-up time (min)		Time in bed (min)		Total sleep time (min)		Wake after sleep onset (min)		Sleep efficiency (%)		Sleep latency (min)	
<i>Fixed effects</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>
<i>Training load</i>														
HSD (×100-m)	2.5 (-6.3 – 1.4)	0.21	1.9 (-0.4 – 4.2)	0.10	4.5 (0.5 – 8.5)	0.03	4.9 (0.6 – 9.2)	0.03	0.8 (-0.8 – 2.4)	0.31	0.3 (-0.2 – 0.8)	0.21	-0.8 (-2.9 – 1.4)	0.49
Collisions (n)	-0.8 (-1.9 – 0.3)	0.16	-0.1 (-0.7 – 0.6)	0.80	0.6 (-0.6 – 1.7)	0.34	0.3 (-0.9 – 1.5)	0.59	0.2 (-0.2 – 0.7)	0.29	-0.0 (-0.2 – 0.1)	0.69	-0.1 (-0.7 – 0.6)	0.85
HIA and HID (n)	0.3 (-0.2 – 0.8)	0.28	-0.0 (-0.3 – 0.3)	0.87	-0.2 (-0.7 – 0.3)	0.39	-0.3 (-0.9 – 0.2)	0.21	0.0 (-0.2 – 0.2)	0.69	-0.0 (-0.1 – 0.0)	0.41	-0.1 (-0.3 – 0.2)	0.63
sRPE × duration (×100 A.U.)	0.5 (-3.2 – 4.3)	0.78	-0.7 (-2.9 – 1.5)	0.53	-1.4 (-5.4 – 2.5)	0.48	-0.8 (-5.0 – 3.4)	0.71	-0.6 (-2.1 – 0.8)	0.39	0.0 (-0.5 – 0.5)	0.97	0.5 (-1.6 – 2.7)	0.63
Morning appointment time (main effect)		<0.001		<0.001		<0.001		<0.001		0.27		0.02		0.41
<i>Subjective ratings</i>														
General fatigue (A.U.)	4.3 (0.3 – 8.3)	0.04	2.6 (-0.7 – 5.9)	0.12	-1.5 (-5.4 – 2.4)	0.45	-3.0 (-7.0 – 1.0)	0.14	1.7 (0.2 – 3.1)	0.02	-0.2 (-0.7 – 0.2)	0.34	-0.2 (-2.0 – 1.7)	0.86
General pain intensity (A.U.)	1.0 (-4.4 – 6.3)	0.73	3.8 (-0.8 – 8.3)	0.11	3.0 (-2.2 – 8.2)	0.26	2.7 (-2.8 – 8.2)	0.34	0.1 (-1.6 – 1.8)	0.92	-0.0 (-0.6 – 0.5)	0.88	0.1 (-2.5 – 2.7)	0.93
General stress (A.U.)	0.2 (-6.9 – 7.2)	0.96	-2.6 (-8.4 – 3.2)	0.37	-2.9 (-9.8 – 4.1)	0.42	1.0 (-6.1 – 8.1)	0.79	-2.5 (-5.0 – -0.0)	0.05	0.6 (-0.2 – 1.4)	0.15	-1.7 (-4.9 – 1.5)	0.29

Abbreviations: Est., estimate; CI, confidence interval; HSD, high-speed distance >18 km.h⁻¹; HIA, high-intensity acceleration ≥2.5 m.s⁻²; HID, high-intensity deceleration ≤-2.5 m.s⁻²; sRPE × duration, session-rating of perceived exertion × training session duration; A.U., arbitrary unit.

Table 7. Linear mixed-effects models evaluating the relationship between the daily training load metrics, subjective ratings, and sleep architecture and SSI score.

<i>Fixed effects</i>	Light sleep (%)		N2 (%)		SWS (%)		REM (%)		Sleep stage shifts (/h)		SSI score (A.U.)	
	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>	Est. (95% CI)	<i>p</i>
<i>Training load</i>												
HS (×100-m)	-1.3 (-3.52 – -0.9)	0.25	-0.8 (-2.7 – 1.2)	0.42	0.3 (-1.7 – 2.2)	0.78	0.9 (-0.7 – 2.6)	0.25	-1.1 (-2.1 – -0.2)	0.03	-0.1 (-0.3 – 0.2)	0.73
Collisions (n)	0.0 (-0.4 – 0.5)	0.85	-0.0 (-0.4 – 0.4)	0.96	-0.0 (-0.4 – 0.4)	0.92	-0.0 (-0.3 – 0.3)	0.93	0.2 (-0.0 – 0.4)	0.08	-0.0 (-0.1 – 0.1)	0.96
HIA and HID (n)	0.0 (-0.3 – 0.3)	0.91	0.0 (-0.2 – 0.3)	0.77	-0.1 (-0.3 – 0.1)	0.40	0.0 (-0.2 – 0.2)	0.76	-0.0 (-0.2 – 0.1)	0.52	0.0 (-0.0 – 0.1)	0.25
sRPE × duration (×100 A.U. A.U.)	0.6 (-1.1 – 2.2)	0.48	0.3 (-1.1 – 1.7)	0.62	0.2 (-1.3 – 1.6)	0.82	-0.5 (-1.7 – 0.7)	0.40	0.3 (-0.5 – 1.0)	0.46	-0.1 (-0.4 – 0.1)	0.35
Morning appointment time (main effect)		0.70		0.58		0.69		0.17		0.52		0.62
<i>Subjective ratings</i>												
General fatigue (A.U.)	0.4 (-1.3 – 2.1)	0.60	0.4 (-1.1 – 1.8)	0.61	-0.9 (-2.4 – 0.5)	0.21	0.4 (-0.9 – 1.7)	0.50	0.3 (-0.5 – 1.1)	0.51	-0.6 (-0.8 – -0.4)	<0.001
General pain intensity (A.U.)	0.5 (-1.6 – 2.7)	0.62	0.5 (-1.4 – 2.3)	0.61	0.3 (-1.7 – 2.2)	0.79	-0.7 (-2.4 – 1.0)	0.40	0.2 (-0.9 – 1.3)	0.69	0.2 (-0.2 – 0.5)	0.31
General stress (A.U.)	-0.9 (-3.0 – 1.1)	0.35	-0.7 (-2.4 – 1.0)	0.43	0.6 (-1.2 – 2.5)	0.49	0.2 (-1.3 – 1.7)	0.79	-0.4 (-1.4 – 0.6)	0.42	0.1 (-0.2 – 0.5)	0.49

Abbreviations: Est., estimate; CI, confidence interval; SWS, slow-wave sleep; REM, rapid eye movement; SSI, Spiegel sleep inventory HSD, high-speed distance >18 km.h⁻¹; HIA, high-intensity acceleration ≥2.5 m.s⁻²; HID, high-intensity deceleration ≤-2.5 m.s⁻²; sRPE × duration, session-rating of perceived exertion × training session duration; A.U., arbitrary unit.

Discussion

The present study revealed 1) a high prevalence of insufficient quantity and fragmented sleep during the training camp, 2) higher distance covered at high-speed favours subsequent sleep duration and continuity of sleep architecture, and 3) sleep duration and sleep efficiency are affected by changes in the morning appointment time. The first issue identified by our study was the high prevalence of nights characterized by a TST <7 h (30.3%), WASO >40 min (77.8%) and sleep efficiency <85% (43.4%), i.e., outside the National Sleep Foundation's recommendations for healthy young adults (Hirshkowitz et al., 2015b; Ohayon et al., 2017). Previous studies also observed an impairment in sleep-wake patterns, i.e., TST, WASO and/or sleep efficiency, among players undergoing a training camp in team-based contact sports (Pitchford et al., 2017; Thornton et al., 2017). Factors such as unfamiliar sleeping environment, training load (Pitchford et al., 2017), sharing a bedroom with a teammate (Costa et al., 2023b), and unaccustomed and congested schedules (e.g., several training sessions a day, meetings, working sessions and collective lunches, travels) have been identified as potential reasons why camp can be a vulnerable period for athletes' sleep (Nedelec et al., 2018). However, the players' sleep was not monitored in their usual sleep environment before the camp, so we cannot conclude whether the training camp altered their sleep patterns or if the players are simply poor sleepers.

In contrast to our hypothesis, training load did not contribute to disturb sleep during the camp. Present findings indicated that, irrespective of the morning appointment time effect, every additional 100-m of HSD was associated with an increase in subsequent TIB (+4.5 min) and TST (+4.9 min). A novelty of this study is the introduction of the EEG headband, which revealed a reduction in the number of sleep stage shift following higher daily HSD. These findings support the hypothesis that higher running demands promote sleep propensity and continuity of sleep architecture (Aloulou et al., 2020b; Chauvineau et al., 2023a). Conversely, previous studies reported negative relationships between training load and sleep in team-based contact sports (Pitchford et al., 2017; Thornton et al., 2017, 2018). One explanation for this discrepancy may be related to the training load achieved and/or the training period. Thornton et al., (2017) observed a *moderate* negative within-subject correlation between prior running total distance and TST ($r = -0.3 \pm 0.2$), when rugby league players were exposed to a 2-week preseason high-intensity training camp aimed to maximize adaptations before the competitive season. In our study, running demands were clearly below (daily mean \pm SD, total distance: 3023.1 ± 1981.9 m vs 6225.3 ± 1595.1 m in Thornton et al., (2017)), mainly because the camp program was

designed to prepare players for two exhibition matches in a congested period of time (i.e., 5 days) and without accumulating excessive fatigue. Nevertheless, training load in the present study was higher to those previously reported among under-20 rugby-union players over an intensified international tournament (Lacome et al., 2018). An inverted U-shaped, dose–response relationship model between training load and quantity and/or quality has been proposed (Falck et al., 2021). This model refers to a positive effect of increased load on sleep until a "tipping point", beyond which acute fatigue or overreaching occurs, and sleep is disturbed. It is therefore possible that the training load was not high enough to disrupt sleep. This supports the benefits of not excessive and controlled training load for sleep (Uchida et al., 2012; Kredlow et al., 2015). In addition, Aloulou et al. (2020) observed a negative impact of the number of collisions (assessed via video analysis, $n \approx 16$) experienced by players during an official rugby-union match on subsequent sleep architecture. Our results revealed no significant relationship between the number of collisions and subsequent sleep, likely due to a reduced number of collisions during the present training camp (daily mean \pm SD, $n = 10.7 \pm 9.9$). Future studies are required to further investigate how various physical demands may affect sleep (especially its architecture), when rugby-union players are exposed to high training loads, e.g., during an intensified preseason or tournament preparation period.

Another major finding from this study was that sleep-wake patterns changed according to the morning appointment time. Some studies have demonstrated that morning schedule strongly determines the prior sleep duration (Sargent et al., 2014b, 2014a; Steenekamp et al., 2021). From various sports, Sargent et al. (2014b) observed that the earlier the training start time from 8:00 to 5:00, the less sleep athletes obtained the night before. Although the morning training sessions were scheduled for a later time in the present study (starting from 10:00), players had appointments scheduled between 7:30 and 10:00, depending on the day, to start the day collectively. The earliest scheduled morning appointment, i.e., 7:30–8:30, was associated with the shortest average sleep duration ($06:52 \pm 00:34$ h), which falls below the recommendations for healthy young adults (Ohayon et al., 2017). This sleep reduction is explained by the fact that players did not compensate for having to wake up earlier by going to bed earlier. On the one hand, most social engagements are scheduled in the evening, which could limit the extent to which they can advance their bedtime. On the other hand, it is possible that sleep propensity was not enough for players to fall asleep earlier. Sleep propensity is driven by the interaction between a circadian process, which determines the sleep-wake cycle, and a homeostatic process, which reflects the pressure for sleep that increases during wakefulness (Borbély et al.,

2016). Sleep onset is most favourable during the circadian sleep phase and when sleep propensity is highest. Consequently, going to bed earlier in prevention of an earlier wake-up may not ensure the preservation of sleep duration. This earlier morning appointment occurred the night before Match 1. Considering the importance of sleep for athlete preparation (Halson and Juliff, 2017; O'Donnell et al., 2018a) and the negative impact of sleep restriction on athletic performance (Craven et al., 2022), advancing the morning appointment time just before a match may not be conducive to achieving peak performance during a match. This issue is particularly important as poor sleep is common prior to competitions in team-based contact sports, often due to nervousness and pre-competition thoughts (Sargent et al., 2022; Sim et al., 2024). Furthermore, one unexpected result of the present study was that delaying the morning appointment time led to players getting up later, but did not necessarily guarantee an increase in sleep duration or sleep efficiency. The highest average sleep duration was obtained when appointment time was scheduled at 9:00–10:00. Nevertheless, sleep duration (+17.2 min) and sleep efficiency (+2.1%) were most favourable when morning appointment was scheduled at 8:00–9:00 compared with 8:30–9:30. This difference may be imputable to a greater delay in bedtime (+44.1 min) than in get-up time (+36.1 min) at the 8:30–9:30 appointment compared with 8:00–9:00. Players may have taken advantage of a later morning appointment to enjoy a longer free evening, which had a detrimental effect on sleep duration and efficiency. Our results also indicate that delaying bedtime resulted in subsequent increased perceived fatigue. These results support the importance of the regularity of sleep habits, which has been demonstrated as an important factor for greater sleep duration and efficiency in elite team sport athletes (Halson et al., 2022).

Limitations

Some limitations need to be considered. The descriptive nature of the current rugby team and training camp makes it difficult to extrapolate the results to other disciplines or training situations. However, exploring this type of period in elite athletes is very challenging, and the use of such a sleep methodology makes the results unique and remains of major interest in the context of high performance. In addition, we did not monitor sleep during a usual training home period. We cannot exclude the potential effect of an unfamiliar sleep environment on the present results, as the training camp took place in three different locations, nor can we discount the extent to which the training load was unusual, which are important factors likely to affect sleep (Pitchford et al., 2017; Roberts et al., 2019b). It is also important to note that this study

was carried out during a national team convocation, with putative high stakes for subsequent selection for major competitions. Although both exhibition matches were won, they may have led to additional nervousness that could affect sleep (Juliff et al., 2015). Players did not provide any data on naps despite some of them napped, which would have been relevant considering its benefit on subsequent sleep during a camp (Thornton et al., 2017). Finally, there was missing EEG headband data, resulting in a lower statistical power. In this respect, linear mixed-effects models were used to assess the relationship between training load and sleep at the individual level while dealing with missing data (Harrison et al., 2018).

Practical applications

When designing camp schedules, coaches should be aware of the negative impact of earlier and/or unusual morning appointment time on sleep, especially the day before or the day of a match. In particular, consistent morning appointment time should promote players' sleep quantity and quality, and prevent subjective fatigue. It could be useful to implement sleep-hygiene training courses for players, to promote consistent sleep timing and sleep quantity and quality during this compromised period for sleep.

Conclusion

The present study highlights the high prevalence of fragmented sleep during a moderate-intensified training camp, supporting the evidence that such a period is vulnerable for sleep in team-based contact sports. Daily training load had no detrimental effects on sleep. In contrast, higher running demands promoted sleep duration and the continuity of sleep architecture. These results may reflect that controlled and not excessive training load promotes sleep. Inversely, timing of the morning appointment may substantially affect sleep-wake pattern. Earlier and/or unusual morning appointment time reduced sleep duration and sleep efficiency, due to inappropriate bedtime.

Study 2-A

Higher training loads affect sleep in endurance runners: can a high-heat-capacity mattress topper mitigate negative effects?

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Associated publication (Appendix 1)

Chauvineau, M., Pasquier, F., Poirier, C., Le Garrec, S., Duforez, F., Guilhem, G., & Nedelec, M. (2023). Higher training loads affect sleep in endurance runners: Can a high-heat-capacity mattress topper mitigate negative effects? *Journal of Sports Sciences*, 1–12. <https://doi.org/10.1080/02640414.2023.2285574>

Associated communications

Chauvineau, M., Pasquier, F., Le Garrec, S., Duforez, F., Guilhem, G., & Nedelec, M. (2022). Effet de la manipulation de la charge d'entraînement et d'un surmatelas à haute conductivité thermique sur le sommeil de coureurs à pied. *Congrès du sommeil, Lille, France. Poster communication*

Abstract

This study investigates the impact of a training program on the sleep of endurance runners and the benefits of chronically using a high-heat-capacity mattress topper (HMT). Twenty-one trained male athletes performed a 2-week usual training regimen, sleeping on a low-heat-capacity mattress topper (LMT), followed by 2-week overload and taper periods. From overload, participants were assigned into two groups based on the mattress topper used: HMT (n = 11) or LMT (n = 10). Irrespective of the group, overload increased *general stress* and *stress-reaction symptoms* evaluated by questionnaires, with no decline in performance on a graded-exercise treadmill test, the majority of participants being “non-overreached” (n = 14). From a daily perspective, each additional 100 A.U. in training load, assessed using the session rating of perceived exertion, was associated with an impairment in subsequent sleep efficiency ($\beta = -0.2\%$; $p < 0.01$), WASO ($\beta = +0.4$ min; $p < 0.05$) and SOL ($\beta = +0.5$ min; $p < 0.05$), which was unaffected by HMT use. Practitioners should be aware that sleep can be vulnerable during excessive training loads, and of the need to implement individualised strategies to mitigate sleep disturbance in this context. Further studies should be conducted on the potential benefits of the HMT among athletes in various training/environmental conditions.

Keywords: recovery, athletes, training program, performance, innovative bedding system, muscle soreness

Introduction

Athletes seek to follow a well-controlled training program to achieve peak performance during the most important competitions of the season. The training periodization aims to accurately manage phases of increase (overload period) and decrease (taper period) in training load (Mujika et al., 2018) which can cause variations in the athlete's training state between negative ("fatigue") and positive ("fitness") responses (Banister et al., 1975). The physiological, psychological, and performance benefits of manipulating training load throughout the competition preparation are well established for endurance athletes (Bosquet et al., 2007; Mujika et al., 2018). However, an imbalance between training load and recovery leads to training maladaptation and increases the risk of excessive fatigue, overtraining, illness, injury and underperformance (Meeusen et al., 2013). Therefore, adequate management of the recovery process is necessary to minimize training-induced fatigue and to promote training adaptations (Mujika et al., 2018).

Sleep is recognized by the scientific community as providing a number of important metabolic, hormonal and cognitive restorative functions (Halson and Juliff, 2017). Several studies have demonstrated the beneficial effects of acute and chronic physical activities on sleep quantity and quality in healthy individuals (Kredlow et al., 2015). On the one hand, high training loads have been identified as a sport-specific factor that may impair sleep among athletes (Roberts et al., 2019b; Walsh et al., 2020). A systematic review with meta-analysis proposed that an increase of more than 25% in training load, compared with a usual training period, is likely to reduce TST and/or sleep efficiency (Roberts et al., 2019b). Previous studies have suggested that muscle fatigue, DOMS and increased sympathetic activity induced by high training loads may all contribute to sleep disturbance (Hauswirth et al., 2014; Schaal et al., 2015; Sargent et al., 2016), which may reflect maladaptation to training (Roberts et al., 2019b). However, these studies included subelite to elite endurance athletes in non-weight-bearing sports (i.e., triathlon, swimming, cycling). This precludes drawing clear conclusions about prolonged running exercises inducing greater muscle damage resulting from such stretch-shortening cycle activities (Nieman et al., 2014). Exercise-induced muscle damage and subsequent DOMS and inflammation are likely to affect sleep (Finan et al., 2013), which suggests a more profound effect of muscle damage induced by high training loads on sleep in endurance runners. On the other hand, the purpose of tapering following high training loads is to reduce physiological and psychological stress and fatigue (Mujika et al., 2018), making sleep a central component of the recovery and adaptations sought during this period. Consequently, there is an interest in

optimizing sleep among endurance runners exposed to high training loads and during taper periods to enhance training adaptations and performance.

Sleep regulation and the circadian rhythm of core body temperature are closely related (Krauchi and Deboer, 2010). Sleep initiation is most likely to occur when the decline in CBT is maximal, as a result of skin warming and body heat loss at the periphery (Krauchi and Deboer, 2010). Sleeping on a high-heat-capacity gel sheet on the top of a foam mattress, which enhances the slow and continuous removal of body heat during sleep by conductive heat transfer, has shown to increase SWS and improve subjective sleep comfort from an acute perspective (Kräuchi et al., 2018; Herberger et al., 2020). Aloulou et al. (2020) observed a reduction in WASO and a higher REM sleep proportion when sleeping on a similar gel sheet after an elite rugby union match. Nonetheless, the putative relevance of this cooling strategy for endurance runners during an overload and taper periods is currently unknown.

The aim of the present study was to investigate the impact of an overload training and taper period on sleep among well-trained endurance runners; as well as the effect of a high-heat-capacity mattress topper (HMT) on sleep during these two training periods. We hypothesized that an overload training period negatively affects sleep, and that sleeping on HMT may mitigate this negative effect.

Methods

Participants

A call for participation was sent to various regional track and field clubs. Forty-four runners responded to the call. The inclusion criteria were to have been competing for ≥ 4 years and training ≥ 5 times a week with a required performance level < 38 min per 10 km to include runners from 'Trained/Developmental' to 'Highly Trained/National' levels (McKay et al., 2022). Six participants did not meet these requirements and were therefore excluded. The following exclusion criteria were then checked before the start of the study: (a) any contraindication to high-intensity training based on an examination by a medical doctor including a detailed medical history and an electrocardiogram at rest ($n = 2$ exclusions); (b) actigraphy or PSG-confirmed sleep disorders, such as sleep apnea (apnea-hypopnea index > 10), periodic limb movement syndrome, hypersomnia, insomnia, circadian sleep rhythm disorders, or narcolepsy ($n = 2$ exclusions); (c) sleep complaints (i.e., global PSQI scores ≥ 5 ; Buysse et al. 1989), extreme morning or evening chronotype (i.e., MEQ scores < 31 or > 69 ; Horne and Ostberg 1976), and excessive daytime sleepiness (i.e., ESS scores ≥ 10 ; Johns 1991) assessed by questionnaires

(n = 4 exclusions) (d) shift workers (n = 4 exclusions); (e) daily consumption of alcoholic beverages, more than 300 mg of caffeine per day and the use of prescribed medications. A total of 26 well-trained male endurance runners (mean \pm SD; age = 27.2 ± 4.8 years; height = 175.4 ± 5.6 cm; body mass = 67.6 ± 7.0 kg) completed the inclusion and exclusion criteria and participated in this study.

Experimental design

The study was conducted from September 2021 to March 2022. An overview of the study design is presented in **Figure 24**. The sleep of each participant was continuously monitored using actigraphy for a period of 6 weeks, divided into three 2-week training periods, using a protocol adapted from Hauswirth et al. (2014). The daily internal training load was also calculated by multiplying the sRPE by the duration of each training session within a day. In the first 2 weeks (baseline), participants followed a training program based on their usual training regimen. The following 2 weeks (overload), participants were asked to increase the duration of each training session by 40% in an attempt to overreach them (Hauswirth et al., 2014). In the last 2 weeks of the protocol (taper), the participants reduced the duration of each training session of the usual training regimen by 50% while maintaining the training intensity in an attempt to maximise post-taper performance gains (Bosquet et al., 2007). They completed the training sessions in their own training facility and the same researcher daily checked the correct sequence of training sessions by SMS and/or phone calls. Before the 6-week training follow-up, each participant came to the laboratory to get familiar with the testing battery including: (a) surveys, and (b) a graded-exercise test. At the end of baseline (Test 1), overload (Test 2) and taper (Test 3), participants performed the testing battery. In order to limit dietary influences on test results, participants were asked to follow standardised nutritional guidelines (quantity and content for food and drink) 48h prior to and during the day of the testing battery, as well as abstaining from physical activity 24h prior to the testing battery. Compliance with these instructions was checked upon arrival for each testing battery.

Participants slept in the same bedroom in their home throughout the protocol. During baseline, all participants slept on a low-heat-capacity mattress topper (LMT). At the start of overload (the day after Test 1) and according to a matched group single-blinded experimental design, participants were allocated into two groups based on the mattress topper used until the end of the protocol: HMT (n = 13, experimental group) and LMT (n = 13, control group).

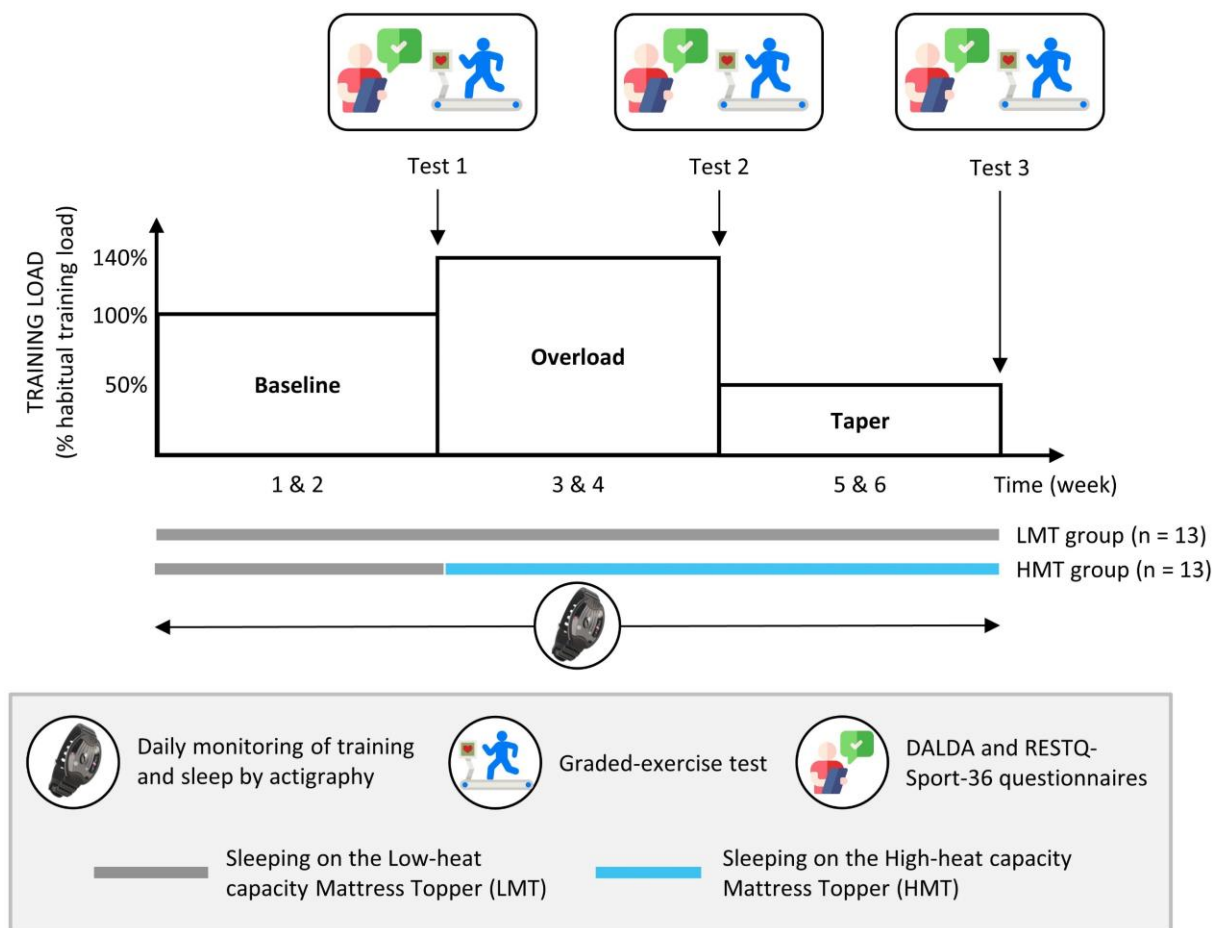


Figure 24. Overview of the experimental design.

Fatigue markers following training periods

Declarative assessments

Before the graded-exercise test, participants were asked to complete the short French questionnaire version of the Recovery-Stress Questionnaire for Athletes (RESTQ-Sport-36; Kellmann and Kallus 2016), and the Daily Analysis of Life Demands for Athletes (DALDA; Rushall 1990) to assess their recovery-stress levels and stress-reaction symptoms.

Performance testing

A graded-exercise test was performed until exhaustion to determine the performance and $\dot{V}O_{2peak}$ on a treadmill (Saturn 300/100r, h/p/cosmos Sports & Medical gmbh, Germany). The exercise started with 5 min of warm-up at 10 km.h⁻¹ followed by an increase of 1 km.h⁻¹ every 2 min until volitional exhaustion with a slope fixed at +1% (Jones and Doust, 1996; Hausswirth et al., 2011). Oxygen uptake, carbon dioxide output, and ventilation were continuously monitored using a breath-by-breath analyser (Quark CPET, Cosmed, Rome, Italy). Heart rate (HR) was continuously recorded with a heart rate monitor (HRM-Dual, Garmin, Olathe, KA, USA). $\dot{V}O_{2peak}$

was estimated by the highest 20-s average value. Performance was calculated as follows: $\text{Performance} = S_{\text{compl}} + 25 \times (t/120)$ where S_{compl} is the speed of the last successful 2-min step and t is the number of seconds in the last step.

Assessment of individual training responses

The participants who decreased performance in a larger extent than the smallest worthwhile change (SWC) at Test 2 were diagnosed as “overreached” (Meeusen et al., 2013). The SWC was calculated as 0.3 of the individual coefficient of variation between the familiarisation and Test 1 (Hopkins et al., 2009), during which training load was not manipulated (baseline). The participants who maintained or increased performance at Test 2 were considered “non-overreached”.

Statistical analysis

We used a non-inferiority sample size calculation to estimate the required sample size to detect potential differences in sleep efficiency between baseline and overload. Based on previous research from our lab (Hauswirth et al., 2014), it was necessary to include 10 participants to achieve significance and power criteria ($1 - \beta$) of 0.05 and 90%, respectively (G*Power program, version 3.1.9.7). Considering the longitudinal design and the fact that participants were allocated into two distinct groups, we chose to increase the number of participants to 13 in each group to account for potential drop out. All statistical procedures were performed using R software (version 4.2.0). The normality of the data distribution and the homogeneity of variance were assessed using the Shapiro-Wilk and Levene tests, respectively.

Three linear mixed-effects models (“lmer” function of the “lme4” R package) were used in accordance with the dependent-tested variables and the research question (**Table 8**). Linear mixed-effects models extend traditional linear models by combining fixed and random effects to consider the non-independence of observations at the level of individuals. For the second linear mixed-effects model, training load was specified as a random slope by adding it to the participant random effect to test the hypothesis that the strength of the effect (slope) of training period on fatigue markers, sleep variables and DOMS varies depending on the training load achieved by each participant. For the first and second linear mixed-effects models, when a significant effect of a fixed-effect (i.e., group, time and/or group \times time interaction) was reported, multiple post-hoc Student t-test pairwise comparisons were conducted using least square means contrasts with the Bonferroni correction for alpha adjustments (“emmeans” function of

the “emmeans” package). The effect sizes were calculated to interpret the magnitude of the difference in means values between the fixed-effects, and were interpreted using the following criteria: $<0.2 = \textit{trivial}$, $0.2-0.6 = \textit{small}$, $0.6-1.2 = \textit{moderate}$, $1.2-2.0 = \textit{large}$, and $>2.0 = \textit{very large}$ (Hopkins et al., 2009).

To determine whether the association between daily training load and sleep variables was mediated by DOMS, general fatigue or stress, separate mediation analyses were performed using the PROCESS function (Model 4, version 4.2; Hayes 2022). The models selected were those in which the mediator was associated with both daily training load (univariate regression) and sleep variable (by fixing the effect of daily training load using multiple regression). A 5,000 bootstrapping procedure was performed to estimate the 95% bias-corrected nonparametric CIs. When a 95% CI did not include zero, it indicated that the indirect effect was statistically significant. If both the indirect and direct effect were significant, then the mediating effect was considered partial, otherwise it was full if only indirect effect was significant.

Results are expressed as the mean \pm SD, except for the linear mixed-effects models and mediation models for which the results are presented as the estimate (β) \pm standard error and the 95% CI. For each linear mixed-effects model, marginal R^2 and conditional R^2 are provided as measures of the proportion of variance explained by the fixed effects(s) alone and both the fixed and random effects, respectively. The level of significance was set at $p < 0.05$.

Table 8. Linear mixed-effects models' details used for each dependent variable according to the research question.

Objective	Dependent variables	Fixed effect(s)	Random effect	Formula
To compare fatigue markers following training periods and weekly change in training session details for each group.	<ul style="list-style-type: none"> - <u>Fatigue markers</u>: RESTQ-Sport-36 and DALDA items, $\dot{V}O_{2peak}$, HR_{peak} and MAS - <u>Training session details</u>: weekly training load and training volume, number of training sessions, mean sRPE per training session 	Group, time, and group × time interaction	The participant	$var \sim time + group + time \times group + (1 participant)$
To evaluate the weekly change in daily-monitored measures for each group.	<ul style="list-style-type: none"> - <u>Sleep variables</u>: bedtime, get-up time, TIB, TST, WASO, sleep efficiency, SOL, SSI score - DOMS 	Group, time, and group × time interaction	The participant and the slope (daily training load)	$var \sim time + group + time \times group + (daily\ training\ load participant)$
To assess the relationship between daily training load, mattress topper type and subsequent sleep.	<ul style="list-style-type: none"> - <u>Sleep variables</u>: TIB, TST, WASO, sleep efficiency, SOL, SSI score 	Daily training load, mattress topper type (categorical predictor), and daily training load × mattress topper type interaction	The participant	$var \sim daily\ training\ load + mattress\ topper\ type + daily\ training\ load \times mattress\ topper\ type\ interaction + (1 participant)$

Notes: Linear mixed-effects formulas were fitted with the dependent variable (var) on the left of a '~' operator and the fixed effect(s) separated by '+' operators on the right. Interaction between fixed effects is indicated by '×'. Random effect is distinguished by '|'. *Abbreviations:* RESTQ-Sport-36, Recovery-Stress Questionnaire for Athletes; DALDA, Daily Analysis of Life Demands for Athletes; MAS, maximal aerobic speed; $\dot{V}O_{2peak}$, peak oxygen uptake; HR_{peak} , peak heart rate; sRPE, rating of perceived exertion of the session; TIB, time in bed; TST, total sleep time; SOL, sleep onset latency; WASO, wake after sleep onset; SSI, Spiegel Sleep Inventory; DOMS, delayed-onset muscle soreness.

Results

Five participants dropped out the protocol because of injury (n = 2), illness (n = 1), and for personal/professional reasons (n = 2), and were excluded from further analysis. Participants were classified at level 2 '*Trained/Developmental*' (n = 7) and at level 3 '*Highly Trained/National*' (n = 14) according to the guidelines proposed by McKay et al. (2022). No significant differences ($p > 0.05$) in participants characteristics were reported between groups. A total of 777 actigraphy-based night sleep data (88.1%), 859 daily training load data (97.4%), and 772 subjective ratings data (87.5%) out of 882 were recorded. A main effect of time was found ($p < 0.001$) for weekly training load, number of training sessions, training volume and sRPE. No main effect of group and group \times time interaction was reported ($p > 0.05$). For all these variables, post-hoc analyses between weeks independently from the group are shown in **Table 9**.

Impact of training periods on fatigue markers and sleep

Declarative assessments

A main effect of time was found for the RESTQ-Sport-36 and DALDA items related to *disturbed breaks* ($p < 0.05$), *emotional exhaustion* ($p < 0.05$), *injury* ($p < 0.05$), *sources of stress* ($p < 0.05$) and *stress-reaction symptoms* ($p < 0.01$; **Table 10**). No main effect of group and group \times time interaction was observed on overall RESTQ-Sport-36 and DALDA items ($p > 0.05$). Independently from the group, the score for *disturbed breaks* item was higher at Test 2 than Test 1 ($p < 0.05$; $d = 0.84$) and Test 3 ($p < 0.05$; $d = 0.79$). An increase in score for *injury* ($p = 0.05$; $d = 0.77$) and *emotional exhaustion* ($p < 0.05$; $d = 0.83$) items, and greater number of "worse than normal" *stress-reaction symptoms* ($p < 0.01$; $d = 1.01$) were noted at Test 2 compared with Test 1. A *moderate* greater number of "worse than normal" *sources of stress* was observed ($p = 0.05$; $d = 0.78$) at Test 3 compared with Test 1.

Performance testing

There was no main effect of group, time and group \times time interaction ($p > 0.05$) for $\dot{V}O_{2peak}$, HR_{peak} and the ambient environment (mean \pm SD: $22.4 \pm 1.1^\circ\text{C}$ and $39.8 \pm 7.0\%$ relative humidity). A main effect of time ($p < 0.01$) was reported for performance, but no main effect of group and group \times time interaction ($p > 0.05$) was noted (**Table 10**). Irrespective of the group, a non-significant *moderate* decrease at Test 2 ($-0.18 \text{ km}\cdot\text{h}^{-1}$; $p = 0.15$; $d = -0.63$) and a *small* increase in performance at Test 3 ($+0.16 \text{ km}\cdot\text{h}^{-1}$; $p = 0.32$; $d = 0.53$) were observed compared with Test

1 (mean \pm SD: $18.6 \pm 1.1 \text{ km}\cdot\text{h}^{-1}$). A significant *moderate* increase ($+0.34 \text{ km}\cdot\text{h}^{-1}$; $p < 0.01$; $d = 1.16$) in performance was observed at Test 3 compared with Test 2.

Individual training responses

Seven of the twenty-one participants decreased in performance of at least the SWC (0.41% of Test 1) at Test 2 ($17.6 \pm 1.4 \text{ km}\cdot\text{h}^{-1}$) compared with Test 1 ($18.4 \pm 1.4 \text{ km}\cdot\text{h}^{-1}$), and were therefore diagnosed as “overreached”. The fourteen other participants maintained or increased their performance at Test 2 ($18.8 \pm 1.0 \text{ km}\cdot\text{h}^{-1}$) compared with Test 1 ($18.7 \pm 1.0 \text{ km}\cdot\text{h}^{-1}$), followed by a supercompensation in performance at Test 3 ($19.1 \pm 1.1 \text{ km}\cdot\text{h}^{-1}$), and were considered as “non-overreached”.

Daily-monitored sleep measures and DOMS

No main effect of group and group \times time interaction was observed on sleep variables and DOMS ($p > 0.05$). There was no main effect of time for bedtime, get-up time, TIB, TST, sleep efficiency and SOL ($p > 0.05$), but a main effect of time was found for WASO ($p < 0.05$), SSI score ($p < 0.01$) and DOMS ($p < 0.001$). As shown in **Figure 25**, irrespective of the group, WASO significantly increased during the first week of taper ($+4.1 \text{ min}$; $p < 0.05$; $d = 0.41$) compared with the second week of baseline (mean \pm SD: $23.8 \pm 9.6 \text{ min}$), while SSI score was higher during the first week of taper (mean \pm SD: $22.7 \pm 2.0 \text{ A.U.}$) compared with the first (-1.1 A.U. ; $p < 0.05$; $d = -0.38$) and second week (-1.5 A.U. ; $p < 0.01$; $d = -0.48$) of baseline. DOMS was higher during the second week of overload (mean \pm SD: $3.2 \pm 0.8 \text{ A.U.}$) compared with the first week (-0.6 A.U. ; $p < 0.001$; $d = -0.55$) and second week (-0.4 A.U. ; $p < 0.05$; $d = -0.40$) of baseline, and the first (-0.7 A.U. ; $p < 0.001$; $d = -0.61$) and second week (-0.6 A.U. ; $p < 0.01$; $d = -0.48$) of taper.

Table 9. Weekly training session details during baseline, overload and taper periods in the two experimental groups.

	Baseline		Overload		Taper	
	<i>Week 1</i>	<i>Week 2</i>	<i>Week 3</i>	<i>Week 4</i>	<i>Week 5</i>	<i>Week 6</i>
Weekly training load (A.U.)***	1391.5 ± 522.2	1495.2 ± 843.2	2120.0 ± 735.9 ^{a,b}	1937.4 ± 806.4 ^{a,b}	976.6 ± 566.8 ^b	938.1 ± 699.6 ^{a,b}
LMT	1427.9 ± 494.3	1317.8 ± 378.8	2139.2 ± 494.8	1697.3 ± 835.1	1037.7 ± 620.8	865.0 ± 568.9
HMT	1358.4 ± 568.4	1656.5 ± 1110.2	2102.5 ± 928.5	2155.6 ± 750.2	921.1 ± 537.3	1004.6 ± 823.1
Weekly training volume (h)***	6.0 ± 2.3	5.9 ± 2.8	8.0 ± 2.9 ^{a,b}	6.9 ± 2.9	4.7 ± 2.6	4.0 ± 2.5 ^{a,b}
LMT	6.1 ± 2.2	5.6 ± 1.7	8.4 ± 3.0	6.4 ± 2.9	5.2 ± 3.4	4.2 ± 2.5
HMT	5.9 ± 2.4	6.3 ± 3.6	7.6 ± 2.9	7.3 ± 3.0	4.3 ± 1.6	3.9 ± 2.6
Number of training sessions***	5.3 ± 1.6	5.0 ± 1.6	6.1 ± 1.8 ^b	5.2 ± 1.7	4.9 ± 1.5	4.1 ± 1.6 ^a
LMT	5.2 ± 1.6	4.9 ± 0.9	6.2 ± 1.5	4.6 ± 1.3	4.9 ± 1.4	4.3 ± 1.2
HMT	5.4 ± 1.7	5.2 ± 2.0	6.0 ± 2.0	5.7 ± 1.9	4.8 ± 1.6	4.0 ± 1.9
Mean sRPE per training session***	2.6 ± 0.9	2.7 ± 0.9	3.3 ± 1.0 ^{a,b}	2.9 ± 1.0	2.1 ± 0.7 ^b	2.0 ± 0.8 ^{a,b}
LMT	2.6 ± 0.8	2.8 ± 0.6	3.4 ± 1.0	2.8 ± 1.2	2.0 ± 0.7	2.1 ± 0.8
HMT	2.5 ± 0.9	2.6 ± 1.1	3.2 ± 0.9	3.1 ± 0.9	2.1 ± 0.7	2.0 ± 0.8

Notes: Data are presented as mean ± SD. Significant main effect of week: ***p < 0.001. Independently from the group, ^asignificantly different from Week 1, and ^bsignificantly different from Week 2. *Abbreviations:* HMT, High-heat-capacity Mattress Topper group; LMT, Low-heat-capacity Mattress Topper group; A.U., arbitrary unit; sRPE, session Rating of Perceived Exertion.

Table 10. Mean values \pm SD of performance testing and declarative assessments after baseline, overload and taper periods for LMT and HMT groups.

	Test 1 (baseline)	Test 2 (overload)	Test 3 (taper)
<i>Performance (km.h⁻¹) **</i>	18.6 \pm 1.1	18.4 \pm 1.3	18.8 \pm 1.2 ^a
LMT	18.5 \pm 1.2	18.3 \pm 1.5	18.6 \pm 1.4
HMT	18.7 \pm 1.1	18.5 \pm 1.1	19.0 \pm 1.0
<i>$\dot{V}O_{2peak}$ (mL.min⁻¹.kg⁻¹)</i>	61.9 \pm 5.4	61.8 \pm 4.5	60.8 \pm 4.4
LMT	62.5 \pm 6.6	60.8 \pm 5.1	60.1 \pm 5.0
HMT	61.2 \pm 4.1	62.9 \pm 3.7	61.5 \pm 3.9
<i>HR_{peak} (bpm)</i>	186.1 \pm 9.7	186.2 \pm 8.2	186.9 \pm 9.0
LMT	188.6 \pm 7.7	188.9 \pm 6.9	189.6 \pm 8.1
HMT	183.5 \pm 11.1	183.2 \pm 8.8	184.6 \pm 9.5
RESTQ-Sport			
<i>General stress</i>	2.6 \pm 0.5	2.5 \pm 0.7	2.5 \pm 0.8
LMT	2.7 \pm 0.4	2.5 \pm 0.8	2.7 \pm 0.9
HMT	2.5 \pm 0.5	2.4 \pm 0.6	2.3 \pm 0.6
<i>Social stress</i>	1.8 \pm 0.7	2.1 \pm 0.8	2.1 \pm 0.9
LMT	1.9 \pm 0.7	2.1 \pm 0.6	2.4 \pm 1.1
HMT	1.8 \pm 0.8	2.1 \pm 1.0	1.8 \pm 0.6
<i>Fatigue</i>	2.6 \pm 0.8	2.9 \pm 0.9	2.5 \pm 0.8
LMT	2.6 \pm 0.6	2.7 \pm 0.6	2.5 \pm 0.7
HMT	2.6 \pm 1.0	3.1 \pm 1.1	2.5 \pm 0.9
<i>Disturbed breaks*</i>	1.9 \pm 0.4	2.5 \pm 1.0 ^a	1.9 \pm 1.2 ^b
LMT	1.9 \pm 0.5	2.5 \pm 1.1	2.2 \pm 1.5
HMT	1.9 \pm 0.5	2.5 \pm 1.0	1.7 \pm 0.8
<i>Emotional exhaustion*</i>	1.5 \pm 0.3	2.0 \pm 0.7 ^a	1.9 \pm 0.9
LMT	1.6 \pm 0.4	2.0 \pm 0.6	2.2 \pm 1.1
HMT	1.4 \pm 0.3	2.0 \pm 0.8	1.6 \pm 0.4
<i>Injury*</i>	2.6 \pm 0.7	3.2 \pm 0.6 ^a	2.7 \pm 1.1
LMT	2.5 \pm 0.7	3.2 \pm 0.7	3.0 \pm 1.4
HMT	2.7 \pm 0.8	3.2 \pm 0.6	2.4 \pm 0.6

Table 11 (suite). Mean values \pm SD of performance testing and declarative assessments after baseline, overload and taper periods for LMT and HMT groups.

	Test 1 (baseline)	Test 2 (overload)	Test 3 (taper)
Social relaxation	4.1 \pm 0.7	3.8 \pm 1.0	3.8 \pm 0.9
LMT	4.2 \pm 0.7	3.8 \pm 0.9	3.7 \pm 0.9
HMT	4.0 \pm 0.7	3.8 \pm 1.2	3.9 \pm 0.9
General well-being	4.4 \pm 0.5	4.3 \pm 0.7	4.4 \pm 0.8
LMT	4.3 \pm 0.5	4.2 \pm 0.6	4.1 \pm 1.0
HMT	4.5 \pm 0.5	4.3 \pm 0.8	4.7 \pm 0.4
Sleep quality	4.1 \pm 0.8	4.0 \pm 0.8	4.1 \pm 0.9
LMT	3.9 \pm 0.9	4.0 \pm 0.7	4.0 \pm 1.0
HMT	4.3 \pm 0.6	4.1 \pm 0.9	4.3 \pm 0.9
Being in shape	3.9 \pm 0.6	3.8 \pm 0.7	4.0 \pm 1.0
LMT	4.1 \pm 0.6	3.9 \pm 0.8	4.1 \pm 1.2
HMT	3.8 \pm 0.6	3.8 \pm 0.7	4.0 \pm 0.8
Personal accomplishment	4.1 \pm 0.7	4.0 \pm 0.7	3.8 \pm 0.8
LMT	4.1 \pm 0.8	3.9 \pm 0.8	3.7 \pm 0.9
HMT	4.1 \pm 0.6	4.0 \pm 0.8	3.9 \pm 0.7
Self-efficacy	3.8 \pm 0.7	3.8 \pm 0.7	3.8 \pm 1.0
LMT	3.8 \pm 0.8	3.7 \pm 0.6	4.0 \pm 1.2
HMT	3.8 \pm 0.7	3.9 \pm 0.9	3.5 \pm 0.8
DALDA			
Sources of stress - number of "worse than normal"*	0.8 \pm 0.9	1.5 \pm 1.4	1.6 \pm 1.3a
LMT	0.9 \pm 1.1	1.3 \pm 1.3	1.8 \pm 1.5
HMT	0.7 \pm 0.8	1.6 \pm 1.6	1.4 \pm 1.1
Stress-reaction symptoms - number of "worse than normal"*	2.0 \pm 2.0	5.0 \pm 3.7a	3.8 \pm 4.5
LMT	2.2 \pm 2.3	5.4 \pm 4.1	4.0 \pm 5.5
HMT	1.9 \pm 1.8	4.7 \pm 3.4	3.6 \pm 3.4

Notes: Significant main effect of time (test): **p < 0.01. Independently from the group, ^asignificantly different from Test 2. *Abbreviations:* HMT, High-heat-capacity Mattress Topper group; LMT, Low-heat-capacity Mattress Topper group; $\dot{V}O_{2peak}$, peak oxygen uptake; HR_{peak} , peak heart rate.

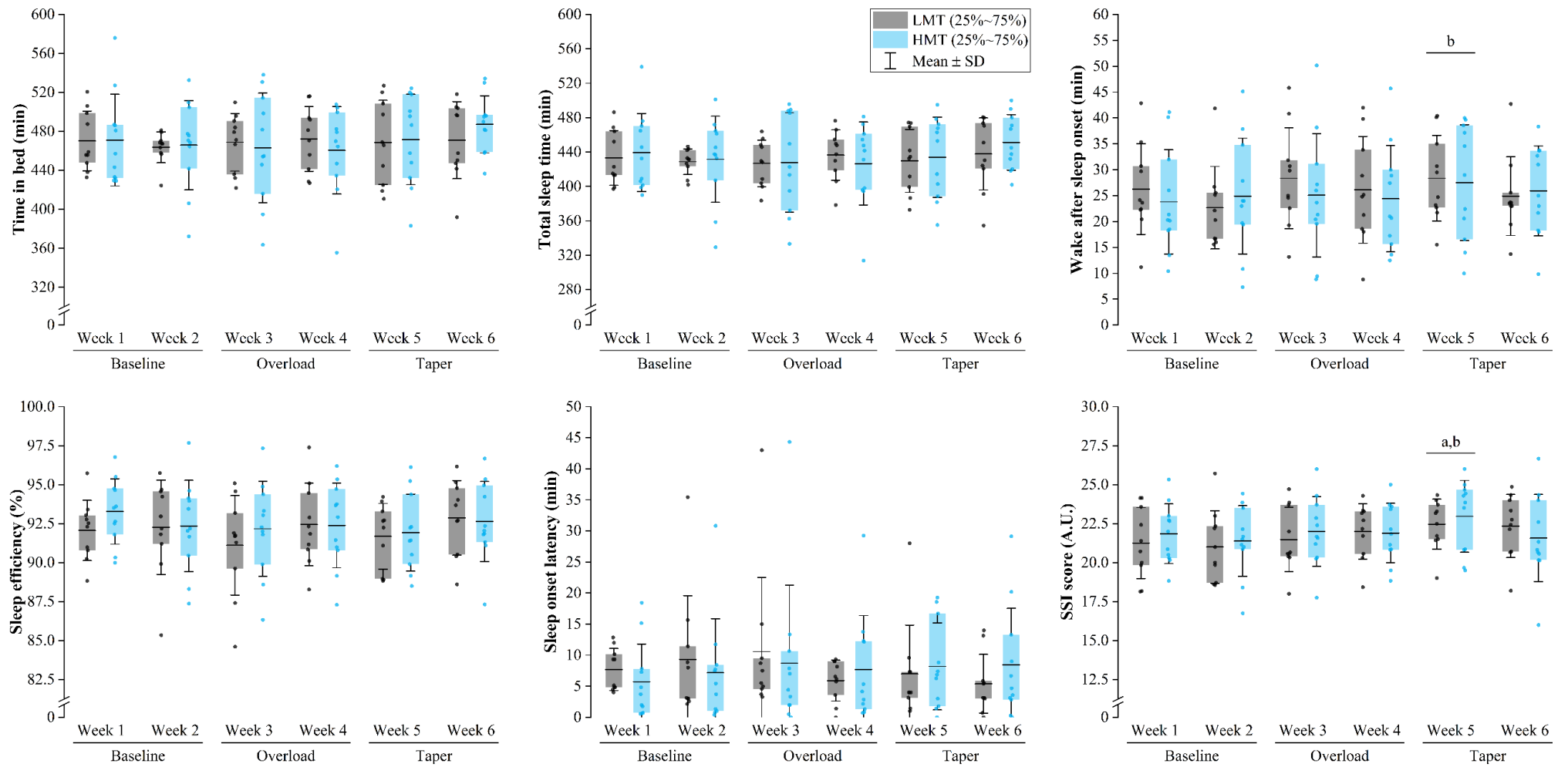


Figure 25. Weekly average of sleep variables during baseline, overload and taper periods for the LMT and HMT groups. *Abbreviations:* SSI, Spiegel sleep Inventory; A.U., arbitrary unit. *Notes:* Independently from the group, ^asignificantly different from week 1 ($p < 0.05$), and ^bsignificantly different from week 2 ($p < 0.05$).

Table 12. Linear mixed-effects model assessing the relationships between daily training load and subsequent sleep variables, and the effect of mattress topper type.

Fixed effects	Time in bed (min)		Total sleep time (min)		Sleep efficiency (%)		Wake after sleep onset (min)		Sleep onset latency (min)		SSI score (A.U.)	
	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>	<i>Est. ± SE (95% CI)</i>	<i>p-value</i>
Intercept	470.1 ± 7.6 (455.2 – 485.0)	<0.001	437.3 ± 7.8 (422.0 – 452.5)	<0.001	93.0 ± 0.5 (92.0 – 94.0)	<0.001	24.3 ± 2.0 (20.4 – 28.1)	<0.001	5.9 ± 1.5 (2.9 – 8.9)	<0.001	21.7 ± 0.4 (21.0 – 22.5)	<0.001
LMT	<i>Reference</i>											
HMT	3.1 ± 6.9 (-10.5 – 16.7)	0.66	-1.2 ± 6.8 (-14.7 – 12.2)	0.86	-0.7 ± 0.5 (-1.6 – 0.2)	0.12	1.9 ± 1.3 (-0.7 – 4.4)	0.16	2.8 ± 1.6 (-0.3 – 5.9)	0.07	0.6 ± 0.4 (-0.1 – 1.3)	0.11
Daily training load (x100 A.U.)	-0.6 ± 1.1 (-2.7 – 1.5)	0.57	-1.6 ± 1.1 (-3.7 – 0.4)	0.12	-0.2 ± 0.1 (-0.4 – -0.1)	0.001	0.4 ± 0.2 (0.1 – 0.8)	0.03	0.5 ± 0.2 (0.1 – 1.0)	0.03	0.0 ± 0.1 (-0.1 – 0.1)	0.90
Daily training load (x100 A.U.) × HMT interaction	-0.4 ± 1.6 (-3.5 – 2.8)	0.82	0.2 ± 1.6 (-2.9 – 3.4)	0.88	-0.1 ± 0.1 (-0.1 – 0.3)	0.48	-0.1 ± 0.3 (-0.7 – 0.5)	0.74	1.0 ± 0.1 (-1.1 – 0.3)	0.25	-0.1 ± 0.1 (-0.2 – 0.1)	0.50
Random Effects												
N	21		21		21		21		21		21	
Observations	777		777		777		777		777		772	
Marginal R ² /	0.001 /		0.004 /		0.021 /		0.010 /		0.010 /		0.005 /	
Conditional R ²	0.223		0.242		0.237		0.393		0.181		0.203	

Abbreviations: Est., estimate; SE, standard error; CI, confidence interval; A.U., arbitrary unit; HMT, high-heat-capacity mattress; LMT, low-heat-capacity mattress; SSI, Spiegel sleep inventory.

Relationships between daily training load, mattress topper type and subsequent sleep

Table 12 provides an overview of the relationships between daily training load and subsequent sleep variables adjusted for mattress topper type. For every additional 100 A.U. in training load, we found subsequent lower sleep efficiency ($\beta = -0.2\%$; 95% CI: -0.4 to -0.1 %; $p < 0.01$; Figure 26-A), higher WASO ($\beta = +0.4$ min; 95% CI: 0.1 to 0.8 min; $p < 0.05$; Figure 26-B) and an increase in SOL ($\beta = +0.5$ min; 95% CI: 0.1 to 1.0 min; $p < 0.05$; Figure 26-C). No significant differences between HMT and LMT, and no daily training load \times HMT interaction were observed ($p > 0.05$) in any sleep variables.

Mediation analysis of the relationship between daily training load and subsequent sleep

Univariate regressions showed that daily training load was not significantly associated with general

fatigue and stress ($p > 0.05$) and thus were not included in further analyses. A significant and positive effect of prior daily training load on DOMS was observed ($p < 0.001$). After controlling for daily training load, multivariate regressions showed that DOMS was associated with WASO ($p < 0.01$), but not significantly with the other sleep variables ($p > 0.05$). Figure 27 contains the results of the mediating effect of DOMS in the relationship between daily training load and WASO. Total effect model including the mediator (DOMS) predicted significant variance in WASO ($R^2 = 0.02$; $p < 0.01$). The results revealed a negative significant indirect effect via DOMS (for additional 100 A.U. in daily training load, $\beta = -0.2 \pm 0.1$ min; 95% CI: -0.3 to -0.0 min), with a

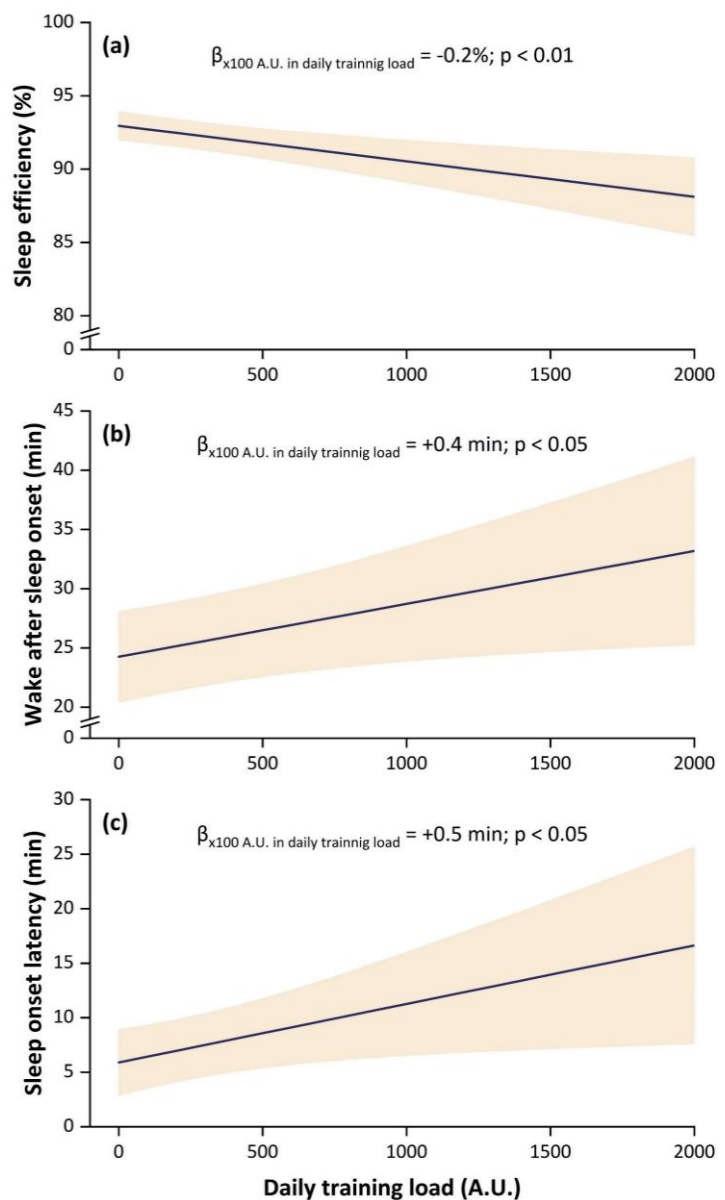


Figure 26. The relationship (and 95% confidence interval) between daily training load and subsequent (a) sleep efficiency, (b) wake after sleep onset, and (c) sleep onset latency.

positive significant direct effect after controlling DOMS effect (for additional 100 A.U. in daily training load, $\beta = 0.8 \pm 0.2$ min; 95% CI: 0.4 to 1.2 min; $p < 0.001$), i.e., DOMS partially mediates the relationship between daily training load and WASO through a suppression effect.

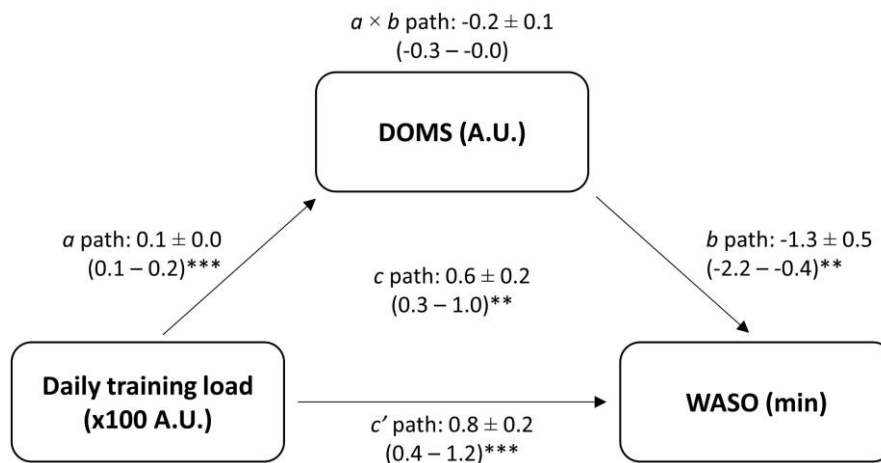


Figure 27. Mediating effect of delayed-onset muscle soreness (DOMS) in the relationship between daily training load and subsequent wake after sleep onset (WASO). *A*, *b*, *c* and *c'* are regression coefficient with *c* = total effect, *c'* = direct effect, and *a* × *b* = indirect effect. Data are presented as unstandardized coefficients ± standard errors (and bootstrapped 95% CI). *A* × *b* indirect effect was considered significant when 95% CI did not include zero. NS, not significant ($p > 0.05$); ** $p < 0.01$; *** $p < 0.001$.

Discussion

Three major findings emerged from this study: 1) no change in the weekly averaged sleep variables was observed throughout the overload and taper periods; 2) from a day-to-day approach, higher daily training load was associated with an impairment in subsequent sleep efficiency, WASO and SOL; and 3) HMT did not mitigate the negative effect of higher training load on sleep.

Impact of training periods on sleep

In contrast to our hypothesis, the first important finding is that overload did not alter the weekly averaged sleep variables. A first explanation may be related to the training load achieved during the overload. Roberts et al. (2019) suggested that sleep disruption occurs when training load increases by more than 25%. Specifically, studies that reported sleep disruption during an overload period increased training volume and/or intensity between +25% and +70% for 2-3 weeks in artistic swimmers, triathletes and rugby players (Hauswirth et al., 2014; Schaal et al., 2015; Thornton et al., 2017), and up to extreme increases (>140%) in cyclists for 1-3 weeks (Lastella et al., 2015a; Killer et al., 2017). Although the current average increase in weekly

training load was $\approx +40\%$ during overload compared with baseline, as required to participants, we observed important inter-individual differences (from +10% to +170%), which may have resulted in individual sleep responses. However, potential individual sleep responses cannot be observed with group means, which justifies the interest of future studies to determine the effect of high training loads at individual level. These inter-individual differences in increased training load may be explained by the different possibilities for each individual to train more than their usual training regimen. Some participants exceeded 10 h of training per week, including one accustomed to running 120 km per week, making it more difficult to increase training load by 40% compared with those training less. In addition, they had to adapt their daily planning to increase the training volume while dealing with personal constraints and work/study schedules. The combination of increased training load and personal/professional constraints may explain the higher scores observed following overload for the general stress items (i.e., *disturbed breaks* and *emotional exhaustion*) of the RESTQ-Sport-36, as well as the higher prevalence for *stress-reaction symptoms* measured by the DALDA. This finding supports the fact that overload effectively increased mental stress (Meeusen et al., 2013).

In contrast, overload did not result in significant physical stress as we did not observe a decrease in performance on the graded-exercise test following overload compared to baseline. The level of accumulated fatigue has been suggested to interact with sleep, with sleep disturbances frequently observed in overreached athletes (Walsh et al., 2020). In the present study, the majority of participants ($n = 14$) were “non-overreached”, which may explain the lack of sleep disruption observed during overload among the present population. These participants also demonstrated an improvement in performance following taper compared with baseline, reflecting a positive adaptation to training (Meeusen et al., 2013). This result supports the interest of overloading prior to tapering provided that the fatigue level is not too high (Aubry et al., 2014). In addition, the effect of accumulated training load over a training period, and consequently level of fatigue, may result in a progressive change in sleep, possibly with some delay and a possible peak effect. The quite modest increase (+4 min) in WASO observed during the first week of taper compared to the second week of baseline may be explained by this potential delay effect of overload, or conversely, by a lack of training during tapering since a minimum of physical activity is recommended for adequate sleep quality (Kredlow et al., 2015). Future investigations should be conducted with an individualised approach to describe the time-course of the effect of an accumulated training load on sleep, and conversely its reduction especially during a taper period prior to a competition.

Relationships between daily training load and subsequent sleep

Although we did not observe alterations in sleep during the overload period considered as a whole, the second important finding was the negative relationship between training load achieved on a daily basis and subsequent sleep. For each additional 100 A.U. in training load, we observed a significant reduction in subsequent sleep efficiency (-0.2%) as well as an increase in WASO (+0.4 min) and SOL (+0.5 min). These results are consistent with previous studies reporting a reduction in TST and/or sleep efficiency in response to day-to-day variation in internal training load in various sports (Dumortier et al., 2018; Lastella et al., 2020b; Aloulou et al., 2021; Costa et al., 2021). Intensified running endurance trainings are likely to induce muscle damage and/or pro-inflammatory response, which may result in DOMS (Nieman et al., 2014). Consistently, the second week of overload resulted in an increase in DOMS compared to baseline. We observed that DOMS in the morning partially mediated the relationship between daily training load and subsequent impairment in WASO through a suppression effect. This indicates that higher training load induced greater DOMS which in turn was related to decreased WASO. This result contradicts the theory that pain and sleep are reciprocally and negatively linked (Finan et al., 2013). However, this finding should be taken with caution since the current mediation analysis does not provide causal evidence. Moreover, the aetiology mechanisms of DOMS may differ from other types of pain (Hainline et al., 2017b), which may affect sleep differently, hence the interest to explore the impact of DOMS on sleep in various sports.

It has been shown that increased daily mental strain, as a mental component of stress, is associated with a subsequent decrease in sleep duration and sleep efficiency among junior winter endurance athletes (Hrozanova et al., 2020). This may have been particularly apparent during overload in the present study since higher general stress scores and prevalence of *stress-reaction symptoms* were observed. Furthermore, it has been suggested that the predominance of the cardiac sympathetic activity and increased circulating cortisol following intensive endurance training may affect the normal down-regulation of the body's stress systems necessary for adequate sleep (Roberts et al., 2019b). This may have contributed to the observed increase in SOL and WASO after higher training load. Additional studies should be conducted on the physiological, psychological and sociological factors which may be involved in the relationship between training load and sleep. Finally, some studies in rugby players have shown that the training components defining training load, may be positively or negatively associated with sleep (Roberts et al., 2019b; Aloulou et al., 2020b). As training load was

quantified only by a subjective approach in the present study, further studies are recommended to better characterise training load and its effects on sleep, particularly in endurance runners. Taken together, performance and well-being of athletes can be affected by excessive load, particularly as sleep is impaired by high training loads (Campbell et al., 2021). Thus, special attention should be allocated to sleep when training periodization involves excessive training loads, especially among elite athletes for whom the prevalence of lack of sleep (i.e., <7 h) and insufficient sleep requirement are frequently observed (Walsh et al., 2020). Nevertheless, the magnitude of the effect of high training load on subsequent sleep is quite modest and should be tempered. Indeed, even at excessive daily training load (e.g., +1000 A.U.), the subsequent impairment in sleep efficiency, SOL and WASO predicted by the linear mixed-effects models in the present study remain within the limits of National Sleep Foundation's recommendations (Ohayon et al., 2017).

Effects of high-heat-capacity mattress topper on sleep

This study is the first to assess the sleep benefit of chronic HMT use. Our hypothesis was that sleeping on HMT may mitigate the negative effect of high training loads on sleep. This hypothesis was not confirmed since the observed negative relationship between daily training load and subsequent sleep was not significantly modified by the use of HMT. Additionally, no significant difference was observed in sleep variables between the HMT and LMT groups throughout the protocol. Previous studies have shown the interest of high-heat-capacity mattress to improve sleep quality, with various positive outcomes on sleep i.e. a higher SWS, a reduction in WASO and/or an increase in REM proportion (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020). The discrepancies with the present study could be explained by the difference in the population studied and/or the experimental protocols, as sleep was monitored on a daily basis by actigraphy, in contrast to previous studies that assessed sleep on a single night by PSG. Future studies are required to assess the potential chronic effect of HMT on sleep architecture. Moreover, the properties of HMT differed from those of the other studies, as the tested high-heat-capacity gel sheet was on the top of either a mattress (Kräuchi et al., 2018; Herberger et al., 2020) or a mattress topper (Aloulou et al., 2020b). The thickness of the high-heat-capacity gel sheet also differed since it was 2.5 cm in the present study and 2 cm (Kräuchi et al., 2018; Herberger et al., 2020) or 4 cm (Aloulou et al., 2020b) in the previous studies, which may have resulted in different thermal behaviour. Different high-heat-capacity gel sheet thicknesses and materials could be tested on the top of mattress vs. mattress topper

in further studies to determine the best bedding system design to remove body heat and promote sleep. Moreover, the present study was conducted among healthy sleepers and from September to March. We do not exclude that HMT could be more beneficial for poor sleepers and in a hot environment, and/or in conditions where sleep may be also disrupted (e.g., training camp, following night competition, unfamiliar sleeping environment, during long-haul travel; Walsh et al. 2020). Additional studies should be conducted in this direction.

Limitations

Some limitations need to be acknowledged. First, the participants of this study completed the training sessions in their own training facility; although we checked daily that the training sessions had been carried out correctly, we do not exclude the possibility that training location (indoors, track, or overland) and environmental conditions (temperature, humidity) could have affected sleep. To avoid any climatic season effect, participants were allocated in LMT or HMT groups in a counterbalanced order. Considering the climatic season (i.e., summer, autumn and winter) in which each participant accomplished the protocol did not modify the linear mixed-effects models results. Second, we only manipulated the duration of training sessions; increased training intensity could have result in more muscle damage and a distinct impact on sleep. Third, as this study was conducted in a domestic environment, the sleep recording method did not allow to monitor sleep architecture on a daily basis. Additional research on the effect of excessive and/or accumulated training load on sleep architecture will provide a better understanding of how sleep adapts and responds to high training loads.

Practical applications

Practitioners should allocate a special attention to sleep with an individualised approach, considering the level of fatigue and training response, to manage training periodization involving excessive training loads, such as during a training camp. Preventing sleep disruptions in this context may promote training adaptation and performance improvement. The use of HMT did not improve sleep compared with LMT nor interfered with sleep. The interest of the present sleep hygiene strategy should be primarily assessed on an individual basis.

Conclusion

The present study highlighted that the overload and taper periods did not alter the weekly averaged sleep variables in well-trained endurance runners, which may be due to inter-individual differences into the training load effectively achieved throughout the period and/or training responses. However, from a day-to-day approach, higher daily training load was associated with an impairment in subsequent sleep efficiency, WASO and SOL. The chronic use of HMT did not mitigate the negative effect of high training loads on sleep, and future studies should be conducted to explore the potential benefits of HMT in athletes with sleep disturbances and/or exposed to inadequate sleep conditions.

Study 2-B

Increased training load promotes sleep propensity and slow-wave sleep in endurance runners: can a high-heat-capacity mattress topper modulate this effect?

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Associated publication (Appendix 2)

Chauvineau, M., Pasquier, F., Duforez, F., Guilhem, G., & Nedelec, M. (2023). Increased training load promotes sleep propensity and slow-wave sleep in endurance runners: can a high-heat-capacity mattress topper modulate this effect? *Journal of Sleep Research*, e14132. <https://doi.org/10.1111/jsr.14132>

Associated communications

Chauvineau, M., Pasquier, F., Duforez, F., Guilhem, G., & Nedelec, M. (2023). La charge d'entraînement favorise l'architecture du sommeil chez des coureurs à pied : intérêt d'un surmatelas à haute conductivité thermique. *Congrès du sommeil, Lille, France. Poster communication*

Abstract

The present study aimed to (1) investigate sleep architecture in response to an overload training and taper periods among endurance runners, and (2) assess the sleep benefits of a high-heat-capacity mattress topper (HMT). Twenty-one trained male endurance runners performed a 2-week usual training regimen (baseline) followed by 2-week overload and taper periods. From overload to the end of the taper period, they were assigned into two groups based on the mattress topper used: HMT (n = 11) or a low-heat-capacity mattress topper (LMT, n = 10). Training load was assessed daily using the session rating of perceived exertion. Following each period, sleep was monitored by PSG, and nocturnal CBT was recorded throughout the night. Irrespective of the group, awakening episodes >5 min decreased following overload compared with baseline (-0.48 , $p = 0.05$). Independently of mattress topper, each 100 A.U. increase in 7-day training load prior to PSG recording were associated with higher SWS proportion ($\beta = +0.13\%$, $p = 0.05$), lower SOL ($\beta = -0.49$ min, $p = 0.05$), and a reduction in the probability of transition from N1 sleep to wakefulness ($\beta = -0.12\%$, $p = 0.05$). Sleeping on HMT did not affect any sleep variable compared with LMT. Increased training loads promote SWS and sleep propensity, highlighting the adaptative nature of sleep to diurnal activity and the role of sleep in physiological recovery. Further studies are required on the potential benefits of HMT on sleep architecture among athletes.

Keywords: recovery, athletes, training program, polysomnography, thermoregulation, sleep architecture

Introduction

Regular participation in physical activity provides various health benefits through exercise-induced musculoskeletal, metabolic and cardiorespiratory adaptations (Paffenbarger and Lee, 1998). Previous research demonstrates the beneficial effects of regular physical activity on sleep quantity and quality in healthy individuals (Kubitz et al., 1996; Kredlow et al., 2015; Park et al., 2021). Sleep is increasingly recognized as a crucial component in athlete preparation by providing a number of metabolic, hormonal and cognitive restorative functions (Halson and Juliff, 2017; O'Donnell et al., 2018a). It has notably been suggested that SWS, a component of NREM sleep, is associated with increased secretion of growth hormone and plays an important role in recovery process via muscle repair and adaptation (Halson, 2014b; Léger et al., 2018; O'Donnell et al., 2018a).

However, practicing sport at the highest level requires high volumes of training, which in turn poses a significant challenge to athlete's sleep (Nedelec et al., 2018). Insufficient sleep quantity and/or quality are frequently observed among elite athletes (Walsh et al., 2020), which may be detrimental to endurance performance (Craven et al., 2022). A growing body of scientific evidence highlights the negative impacts of excessive and/or accumulated training loads (overload) on sleep (Roberts et al., 2019b; Walsh et al., 2020; Falck et al., 2021). Previous studies have suggested several potential factors that may contribute to sleep disturbance during an overload period, such as DOMS, increased sympathetic activity, and depressed immunity (Hauswirth et al., 2014; Schaal et al., 2015; Sargent et al., 2016). Most of the aforementioned studies used wristwatch actigraphy to examine the impact of an overload period on sleep. Such a method does not provide comprehensive measurement of sleep stages, which are typically assessed using PSG. Taylor et al. (1997) observed higher SWS proportions alongside a greater number of movements using one-night PSG at the onset of the season (medium-intensity training) and an overload period (high-intensity training) compared with a taper period (low training volume) in elite female swimmers, which supports both the restorative physiological function of SWS and the potential negative impact of excessive training loads on sleep, respectively. To the best of our knowledge, no study determined the impact of an overload period on sleep architecture among endurance runners. Prolonged running exercises induce larger extent of muscle damage, DOMS and inflammation response (Nieman et al., 2014) compared with non-weight bearing activities such as swimming, which may suggest an important effect of overloading on sleep in endurance runners (Halson, 2014b). Furthermore, the traditional hypnogram-based features (time spent in sleep stages) used by Taylor et al.

(1997) do not provide information on the internal structure of sleep and may be insufficient for assessing its quality (McCarter et al., 2022). Estimating the probability of transition from one sleep stage to another can provide additional information on sleep quality.

Based on aforementioned findings, developing practical strategies for the optimization of athletes' sleep is highly required. The thermal properties of bedding can be manipulated to optimize sleeping microclimate and favour sleep by influencing thermoregulatory processes (Troynikov et al., 2018). Sleep initiation and the high prevalence of SWS in the first sleep cycles are intimately coupled with the circadian decline phase of CBT, which is mediated by distal skin warming and heat loss from the periphery (Van Someren, 2006; Krauchi and Deboer, 2010). Sleeping on a high-heat-capacity gel sheet on the top of a foam mattress has been shown to enhance the slow and continuous removal of body heat during sleep by conductive heat transfer, leading to an increased SWS proportion and improved subjective sleep comfort from an acute perspective, i.e. a single night (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020). However, the relevance of chronically using of this bedding strategy for endurance runners exposed to high training loads is currently unknown.

The aims of the present study were to (1) examine the sleep architecture in response to 2-week overload training and taper periods among endurance runners, and (2) to assess the effectiveness of a high-heat-capacity mattress topper (HMT) on sleep. We hypothesized that higher training loads may increase SWS proportion while disturbing sleep continuity, and that sleeping on HMT may mitigate this negative effect.

Methods

Participants

Twenty-six well-trained male endurance runners [mean \pm SD; age = 27.2 \pm 4.8 years; height = 175.4 \pm 5.6 cm; body mass = 67.6 \pm 7.0 kg; peak oxygen uptake ($\dot{V}O_{2peak}$) = 61.9 \pm 5.4 mL.min⁻¹.kg⁻¹] volunteered to participate in this study. All participants have been examined by a medical doctor including an electrocardiogram at rest to exclude any contraindication to high-intensity training. The inclusion criteria were: (a) competing for ≥ 4 years, (b) training ≥ 5 times per week, and (c) having a performance level below 38 min over a 10-km race. The exclusion criteria were checked at the start of the study as follows: (a) actigraphy or PSG-confirmed sleep disorders, such as sleep apnea (apnea-hypopnea index > 10), periodic limb movement syndrome, hypersomnia, insomnia, circadian sleep rhythm disorders, or narcolepsy; (b) sleep complaints [i.e., PSQI score ≥ 5 (Buysse et al., 1989)], extreme morning or evening chronotype [i.e. MEQ score

< 31 or > 69 (Horne and Ostberg, 1976)], and excessive daytime sleepiness [i.e., ESS score >10 (Johns, 1991)] (c) shift workers; (d) daily consumption of alcoholic beverages, more than 300 mg of caffeine per day and the use of prescribed medications. All the participants recruited were recreational athletes, carrying out a full-time professional/study activity in addition to their training program.

Experimental design

The protocol was conducted from September 2021 to March 2022. An overview of the study design is presented in Figure 28. Participants followed a 6-week training program divided into three 2-week periods during which training volume was manipulated. The instructions were to increase/decrease the duration of each training session while maintaining the intensity and number of sessions per week. The first period consisted of two weeks (baseline) based on their usual training regimen. During the second 2-week period (overload), participants were asked to increase the duration of each training session by 40% in an attempt to elicit functional overreaching (Meeusen et al., 2013; Hausswirth et al., 2014). During the third 2-week period (taper), the duration of each training session was reduced by 50% of the usual training regimen in order to maximise post-taper performance gains (Bosquet et al., 2007; Mujika, 2010). Participants performed training sessions in their usual training facilities, on their own ($n = 9$) or with a coach ($n = 12$). The same investigator daily checked that training sessions were carried out correctly. Before the 6-week training program, each participant performed a night of familiarization with the PSG equipment in the same home environment as for the experimental nights to avoid any first night effect (Hof Zum Berge et al., 2021). At the end of baseline (Test 1), overload (Test 2) and taper (Test 3), participants came to the laboratory and the same qualified practitioner equipped them with the PSG equipment. They were instructed to go to bed and get up at similar times between Test 1, Test 2 and Test 3, according to their sleep habits assessed daily by actigraphy during each training period. Given the potential interest of HMT and not manipulating training load on CBT, nocturnal CBT was continuously recorded only at Test 1 and Test 2. In order to limit dietary influences on test results, participants were asked to follow standardised nutritional guidelines (quantity and content for food and drink) 48 h prior to the night with PSG, as well as abstaining from any training session 24 h prior to the night with PSG. Compliance with these instructions was checked upon arrival for each test.

All participants slept in the same bedroom in their home throughout the protocol. During baseline, all used a low-heat-capacity mattress topper (LMT). According to a matched group

single-blinded experimental design, participants were allocated into two groups from the start of overload (i.e., the day after Test 1) to the end of the protocol based on the mattress topper used: HMT (n = 13) or LMT (n = 13). Participants were allocated in LMT or HMT groups in a counterbalanced order to avoid any climatic season effect, and according to their usual training regimen and individual characteristics (i.e., age, height, body mass and $\dot{V}O_{2peak}$). No significant differences ($p > 0.05$) in participants characteristics at baseline were reported between groups.

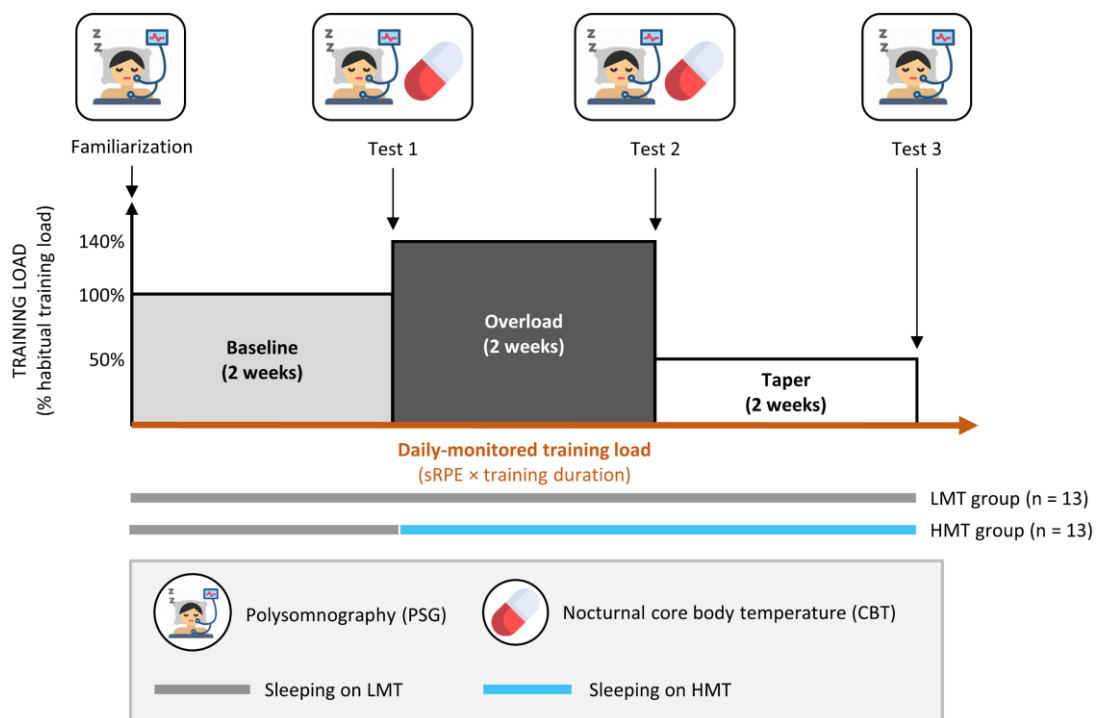


Figure 28. Design of the experimental protocol. *Abbreviations:* HMT, high-heat-capacity mattress topper; LMT, low-heat-capacity mattress topper; sRPE, session rating of perceived exertion.

Data and statistical analysis

Based on previous research from our lab (Hauswirth et al., 2014), we used a non-inferiority sample size calculation to estimate the required sample size to detect potential differences in sleep efficiency between baseline and overload. It was necessary to include 10 participants to achieve significance of 0.05 and power criteria ($1 - \beta$) of 90%, respectively (G*Power program, version 3.1.9.7). Considering the longitudinal design and the allocation of participants into two distinct groups, we chose to increase the number of participants to 13 in each group to account for potential drop out. Data processing and statistical analyses were performed using R software (version 4.2.2). Linear mixed-effects models (“lmer” function of the “lme4” R package) were used to consider the non-independence of observations at the level of individuals. Variance inflation factor (VIF) was used to assess collinearity issues between fixed-effect variables, and models with a low VIF (score < 5) were selected. Then, the structure of the models

(i.e., with and without fixed-effects interaction) was tested, and the best fitting-model was selected according to the smallest Akaike Information Criterion (AIC). The residuals for each model were visually inspected and no obvious deviations from homoscedasticity and normality were revealed. **Table 13** provides an overview of the three linear mixed-effects models used according to the dependent variables tested and the research question.

For the first and third linear mixed-effects models, the significance of the fixed-effects was determined using the “mixed” function from the “afex” R package, using type 2 tests. When a significant effect of a fixed-effect was reported, multiple *post-hoc* Student t-test pairwise comparisons were conducted using least square means contrasts with the Bonferroni correction for alpha adjustments (“emmeans” function of the “emmeans” package). The effect sizes (ES) with associated 95% confidence intervals (CI) were calculated to interpret the magnitude of the difference in means values between the fixed-effects, and were interpreted using the following criteria: $< 0.2 = \textit{trivial}$, $0.2-0.6 = \textit{small}$, $0.6-1.2 = \textit{moderate}$, $1.2-2.0 = \textit{large}$, and $> 2.0 = \textit{very large}$ (Hopkins et al., 2009).

Results are expressed as the mean \pm SD, except for the linear mixed-effects models for which results are presented as the estimate (β) and the 95% CI. The level of significance was set at $p < 0.05$.

Results

Five participants were excluded from the protocol because of injury ($n = 2$), illness ($n = 1$), or for personal/professional reasons ($n = 2$) at the start of the protocol. Twenty-one participants (HMT, $n = 11$ and LMT, $n = 10$) were included. Polysomnographic data from one participant (HMT group) were discarded at Test 3 because of inconsistent bedtimes and wake-up times compared with Test 1 and Test 2. Another participant (HMT group) did not perform Test 3 due to Covid-19 infection. Participants were classified at level 2 ‘*Trained/Developmental*’ (HMT, $n = 3$; LMT, $n = 4$) and at level 3 ‘*Highly Trained/National*’ (HMT, $n = 8$; LMT, $n = 6$) according to the guidelines proposed by McKay et al. (2022). A main effect of training week was found ($p < 0.001$) on weekly training load, number of training sessions, training volume and sRPE. No main effect of group and group \times training week interaction was reported for these variables ($p > 0.24$). For training start times, there was no main effect of training week and group \times training week interaction ($p > 0.22$), but a main effect of condition was observed ($p = 0.02$). Independently from the training week, HMT group trained 02:24 h later than LMT group ($p = 0.02$; ES = 1.41 [95% CI, 0.24 to 2.58]). For all these variables, *post-hoc* analyses between weeks independently from the group are shown in **Table 14**.

Table 13. Linear mixed-effects models' details used for each dependent variable according to the research question.

	Objective	Dependent variables	Fixed effect(s)	Random effect
LMM #1	To compare the weekly change in training session details for each group and evaluate the effect of training periods and between-group differences on sleep.	- <u>Training session details</u> : weekly training load and training volume, number of training sessions, mean sRPE per training session, training start times - <u>Sleep</u> : bedtime, get-up time, PSG-based sleep variables, perceived thermal comfort and sensation, and bedroom air temperature	Group, training week, and group × training week interaction	The participant
LMM #2	To assess the effect of the training load over the 7-day period prior to PSG recording according to the mattress topper.	- <u>Sleep</u> : PSG-based sleep variables	The training load over the 7-day period prior to PSG recording and the mattress topper	The participant
LMM #3	To evaluate between-group differences on nocturnal CBT kinetics following training periods.	- CBT	Group, training period (baseline and overload) and 10-min time interval (main effects and interactions)	The participant and the slope (7-h time points in 10-min time bins)

Abbreviations: CBT, core body temperature; LMM, linear mixed-effects model; PSG, polysomnography; sRPE, session rating of perceived exertion.

Table 14. Weekly training session details during baseline, overload and taper periods in the two experimental groups.

	Baseline		Overload		Taper	
	<i>Week 1</i>	<i>Week 2</i>	<i>Week 3</i>	<i>Week 4</i>	<i>Week 5</i>	<i>Week 6</i>
Weekly training load (A.U.)***	1391.5 ± 522.2	1495.2 ± 843.2	2120.0 ± 735.9 ^{a,b}	1937.4 ± 806.4 ^{a,b}	976.6 ± 566.8 ^b	938.1 ± 699.6 ^{a,b}
LMT	1427.9 ± 494.3	1317.8 ± 378.8	2139.2 ± 494.8	1697.3 ± 835.1	1037.7 ± 620.8	865.0 ± 568.9
HMT	1358.4 ± 568.4	1656.5 ± 1110.2	2102.5 ± 928.5	2155.6 ± 750.2	921.1 ± 537.3	1004.6 ± 823.1
Weekly training volume (h)***	6.0 ± 2.3	5.9 ± 2.8	8.0 ± 2.9 ^{a,b}	6.9 ± 2.9	4.7 ± 2.6	4.0 ± 2.5 ^{a,b}
LMT	6.1 ± 2.2	5.6 ± 1.7	8.4 ± 3.0	6.4 ± 2.9	5.2 ± 3.4	4.2 ± 2.5
HMT	5.9 ± 2.4	6.3 ± 3.6	7.6 ± 2.9	7.3 ± 3.0	4.3 ± 1.6	3.9 ± 2.6
Number of training sessions***	5.3 ± 1.6	5.0 ± 1.6	6.1 ± 1.8	5.2 ± 1.7	4.9 ± 1.5 ^b	4.1 ± 1.6 ^a
LMT	5.2 ± 1.6	4.9 ± 0.9	6.2 ± 1.5	4.6 ± 1.3	4.9 ± 1.4	4.3 ± 1.2
HMT	5.4 ± 1.7	5.2 ± 2.0	6.0 ± 2.0	5.7 ± 1.9	4.8 ± 1.6	4.0 ± 1.9
Mean sRPE per training session***	2.6 ± 0.9	2.7 ± 0.9	3.3 ± 1.0 ^{a,b}	2.9 ± 1.0	2.1 ± 0.7 ^b	2.0 ± 0.8 ^{a,b}
LMT	2.6 ± 0.8	2.8 ± 0.6	3.4 ± 1.0	2.8 ± 1.2	2.0 ± 0.7	2.1 ± 0.8
HMT	2.5 ± 0.9	2.6 ± 1.1	3.2 ± 0.9	3.1 ± 0.9	2.1 ± 0.7	2.0 ± 0.8
Mean training start time [#]	14:26 ± 04:26	14:23 ± 04:26	14:47 ± 04:27	14:46 ± 04:27	14:06 ± 04:28	14:07 ± 04:27
LMT	12:52 ± 04:25	13:11 ± 04:26	13:32 ± 04:27	13:34 ± 04:25	12:59 ± 04:27	13:04 ± 04:25
HMT	15:58 ± 03:24	15:39 ± 04:25	16:03 ± 04:24	15:51 ± 04:26	15:08 ± 04:29	15:19 ± 04:28

Note: Data are presented as mean ± SD. Significant main effect of week: *** $p < 0.001$. Independently from the group, ^asignificantly different from Week 1, and ^bsignificantly different from Week 2. Significant main effect of group: [#] $p < 0.05$. *Abbreviations:* HMT, high-heat-capacity mattress topper group; LMT, low-heat-capacity mattress topper group; sRPE, session rating of perceived exertion.

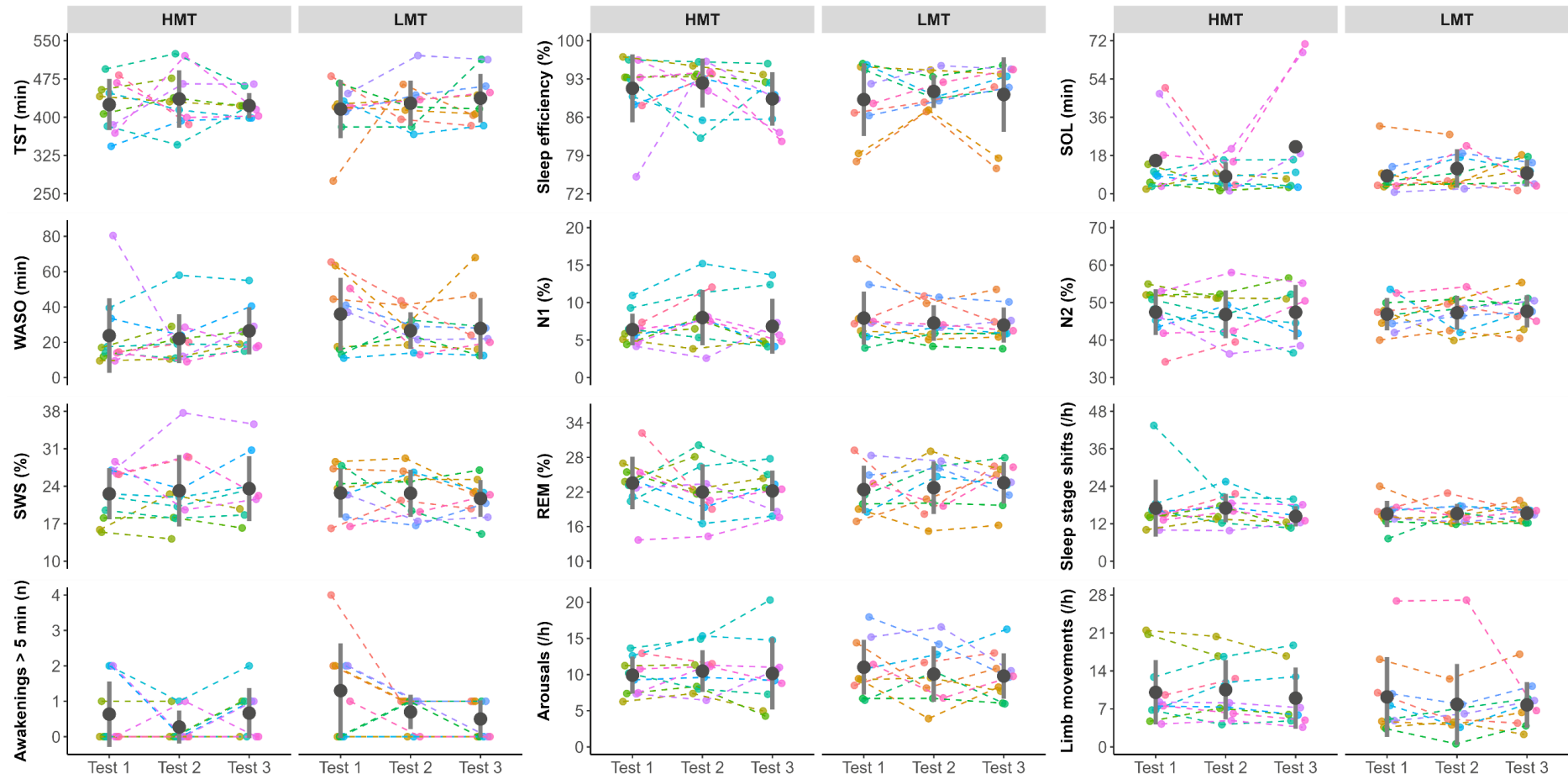


Figure 29. Mean \pm SD polysomnography (PSG)-based sleep data between HMT and LMT groups following baseline (Test 1), overload (Test 2) and taper (Test 3). *Notes:* The coloured dots represent the values for each participant across the tests. *Abbreviations:* HMT, high-heat-capacity mattress topper; LMT, low-heat-capacity mattress topper; TST, total sleep time; SOL, sleep-onset latency; WASO, wake after sleep onset; N1–N2, non-rapid eye movement (NREM) sleep stages 1–2; SWS, slow-wave sleep; REM, rapid eye movement.

Effects of training periods and mattress topper on sleep and temperature

Polysomnography

There was no main effect of group, training period or group \times training period interaction ($p > 0.07$) for bedtime, get-up time, cortical arousals, limb movements as well as the following sleep variables: TST, SE, SOL, WASO, the number of sleep stage shifts and the N1, N2, N3 and REM sleep proportions (**Figure 29**). We observed a main effect of training period for the number of awakenings >5 min ($p = 0.04$), and significant group \times training period interactions for wake-to-wake ($p = 0.01$) and wake-to-N1 ($p < 0.01$) transition probability. Independently from the group, the number of awakenings >5 min was *moderately* lower at Test 2 than Test 1 (-0.48 ; $p = 0.05$; $ES = -0.77$ [-1.41 to -0.13]). As shown in **Figure 30**, only the HMT group showed a *moderate* increase in the probability of transition from wakefulness to wakefulness at Test 3 compared with Test 1 ($+14.4\%$; $p = 0.09$; $ES = 1.03$ [0.09 to 1.98]) and Test 2 ($+16.5\%$; $p = 0.04$; $ES = 1.18$ [0.23 to 2.14]). A *moderate* decrease was observed only in the HMT group in the probability of transition from wakefulness to N1 at Test 3 compared with Test 1 (0.13% ; $p = 0.08$; $ES = -1.05$ [-1.99 to -0.10]) and Test 2 (-0.15% ; $p = 0.05$; $ES = -1.14$ [-2.09 to -0.19]). No main effect of group, training period or group \times training period interaction was observed for the other sleep stage-transition probabilities ($p > 0.06$).

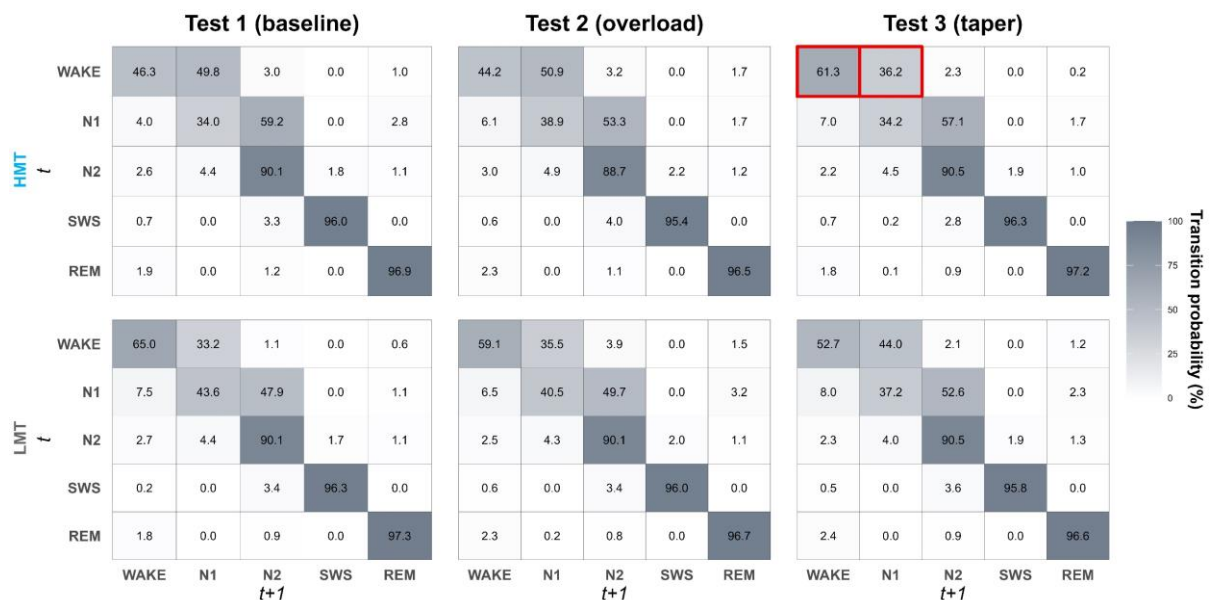


Figure 30. Probability matrices of sleep stage transitions following baseline (Test 1), overload (Test 2) and taper (Test 3) periods for HMT and LMT groups. *Notes:* Each cell presents the transition probability from a sleep stage at time t to another sleep stage at time $t + 1$. Red-squares show significant post hoc differences from Test 2 ($p < 0.05$). *Abbreviations:* HMT, high-heat-capacity mattress topper group; LMT, low-heat-capacity mattress topper group; N1–N2, non-rapid eye movement (NREM) sleep stages 1–2; SWS, slow-wave sleep; REM, rapid eye movement.

Considering the training load during the last 7-day period prior to PSG recording, each additional 100 A.U. increase in training load was associated with lower SOL ($\beta = -0.49$ min [-0.99 to 0.00 min]; $p = 0.05$; **Figure 31-A**), higher SWS proportion ($\beta = +0.13\%$ [0.00 to 0.25%]; $p = 0.05$; **Figure 31-B**), and a reduction in the probability of transition from N1 sleep to wakefulness ($\beta = -0.12\%$ [-0.23 to -0.00%]; $p = 0.05$; **Figure 31-C**). There was neither a significant relationship between the training load prior to PSG recording and any of the other sleep variables, nor a significant difference between HMT and LMT in any sleep variables ($p > 0.08$).

Temperature measurements

No main effects of group, training period or group \times training period interaction ($p > 0.06$) were found for the bedroom ambient temperature (mean \pm SD: $20.6 \pm 2.1^\circ\text{C}$) as well as perceived

thermal comfort and sensation during the night. For the 7 h mean nocturnal CBT, there were a main effect of training period and group \times training period interaction ($p < 0.001$; **Figure 32**), but no main effect of 10-min time interval, group, group \times 10-min time interval interaction and training period \times 10-min time interval interaction ($p > 0.87$). Similar mean nocturnal CBT was reported at Test 1 between the HMT and LMT groups ($p = 0.11$). For the LMT group, mean nocturnal CBT was *moderately* higher ($+0.132^\circ\text{C}$; $p < 0.001$; ES = 0.93 [0.74 to 1.12]) at Test 2 than Test 1, while a *trivial* reduction was observed in the HMT group between Test 2 and Test 1 (-0.025°C ; $p = 0.02$; ES = -0.18 [-0.33 to -0.02]).

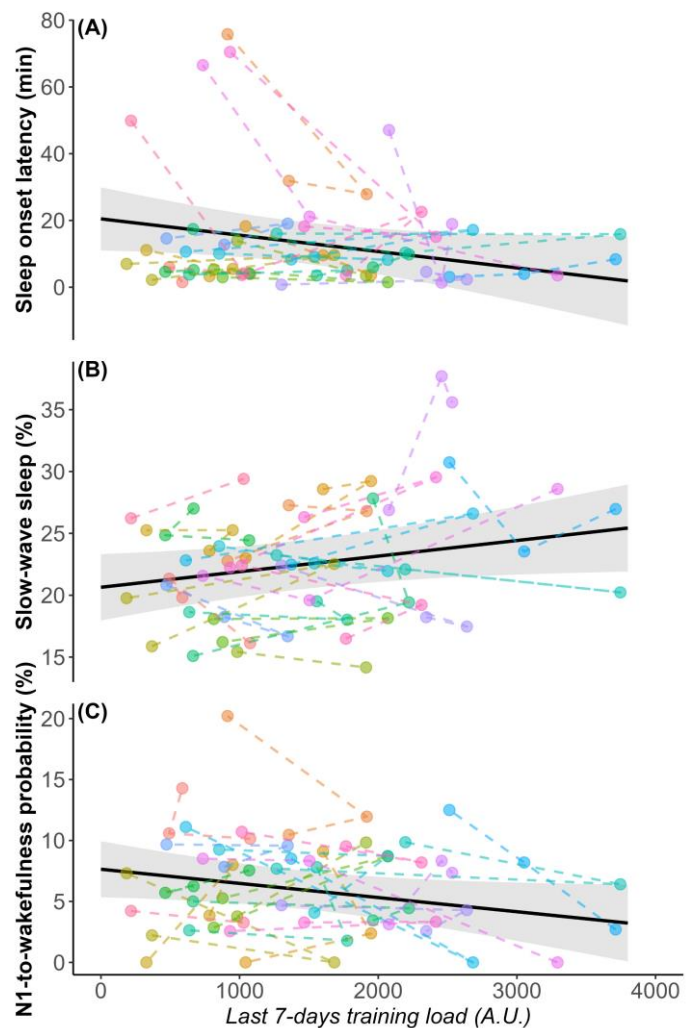


Figure 31. Relationships (and 95% confidence interval [CI]) between the training load over the 7-day period prior to polysomnography recording and subsequent: (a) SOL; (b) SWS; and (c) the probability of transition from N1 sleep to wakefulness. The dots and coloured dashed lines represent the values for each participant according to the order of training periods: baseline (Test 1), overload (Test 2) and taper (Test 3).

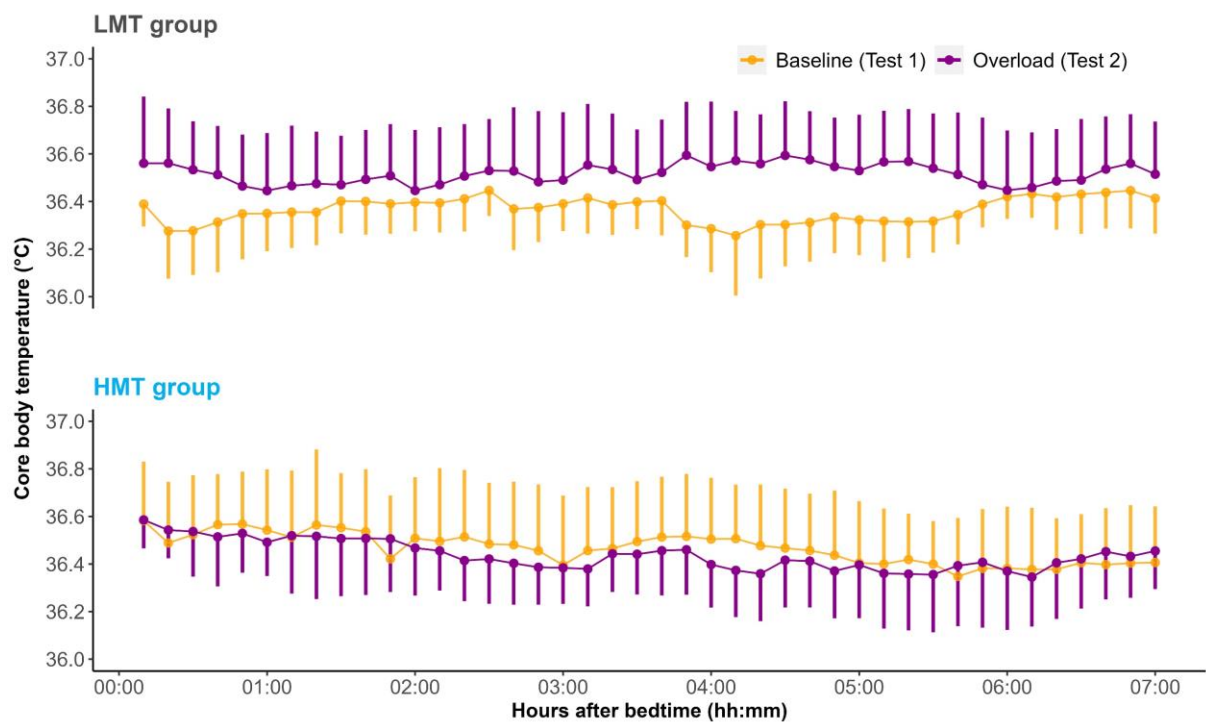


Figure 32. Mean \pm SD changes in core body temperature (CBT) over 7 h after bedtime for each group according to training period: baseline (Test 1) and overload (Test 2).

Discussion

The aims of this study were to examine the impact of training load manipulation (i.e., overload and taper), and to assess the influence of the chronic use of a high-heat-capacity mattress topper (HMT) on sleep architecture in endurance runners. The main findings showed that: 1) higher prior 7-day training loads were associated with an increase in SWS proportion, a reduction in SOL and an improvement in sleep continuity; and 2) HMT did not affect any sleep variable compared with LMT.

Impact of training load on sleep

According to the literature, we hypothesized that high training loads may promote SWS while disturbing sleep continuity. The present findings partially confirmed our hypotheses, given that each 100 A.U. increase in training load over the 7-day period prior to PSG recording were associated with an increase in SWS proportion (+0.13%), a reduction in SOL (-0.49 min), without any alteration of TST, sleep efficiency, WASO, sleep stages distribution, number of sleep stage shifts, cortical arousals and limb movements. A number of studies demonstrated that regular exercise can promote SWS and SOL in healthy populations (Kubitz et al., 1996; Kredlow et al., 2015; Park et al., 2021). Among elite female swimmers, Taylor et al. (1997) reported an increase in SWS proportions using one-night PSG at the onset of the season and during an overload compared with a taper period. In endurance athletes, studies are currently limited, but there is

some evidence for greater amount of SWS following a heavy training or competition (Driver and Taylor, 2000; Chennaoui et al., 2015; Vlahoyiannis et al., 2021). A novelty of the present study is that SWS and SOL are also promoted by the day-to-day accumulation of training load in well-trained endurance runners. This finding was also observed in an ultra-endurance cyclist engaged in a multiday ultra-endurance event, who experienced a progressive day-to-day increase in the amount of SWS (Nédélec et al., 2022). Slow-wave sleep provides a restorative function to the body essential for recovery in athlete through energy conservation, anabolic hormone release (e.g., growth hormone), cleaning of cerebral metabolites accumulated during the waking period and regulation of the immune/inflammatory system (Léger et al., 2018). Slow-wave sleep, together with SOL, acts as the primary driving mechanism in the homeostatic sleep process and is a reliable indicator of sleep need (Borbély et al., 2016). Based on the evidence that prolonged running exercises induce large extents of muscle damage, DOMS and inflammation response (Nieman et al., 2014), it is likely that the daily accumulation of training load maximized the sleep pressure and/or increased the physiological restorative demands promoting SWS and sleep propensity, which supports the adaptative nature of sleep to physical diurnal activity (Halson, 2014b; Léger et al., 2018; Vlahoyiannis et al., 2021). In addition, we observed that higher prior training loads were associated with lower probability of transition from N1 to wakefulness, which may reflect a better sleep continuity. We also noted a reduction in the number of awakenings >5 min, irrespective of the group, following the overload compared with baseline. Taken together, these results suggest that higher training load may have facilitated sleep continuity throughout the night, which in turn may have contributed to increased SWS proportion.

Nevertheless, it is widely recognized that high training loads may be detrimental to the sleep of athletes (Roberts et al., 2019b; Walsh et al., 2020; Falck et al., 2021). Several studies have reported a progressive decline in TST and/or sleep efficiency when endurance athletes are exposed to excessive and/or accumulated training loads (Hauswirth et al., 2014; Lastella et al., 2015a; Kölling et al., 2016), or in response to day-to-day variations in training load (Lastella et al., 2020b; Aloulou et al., 2021; Figueiredo et al., 2021). All of these studies monitored sleep using wristwatch actigraphy that records movements over time to estimate sleep/wake, suggesting that high training loads may cause more movements during sleep. These movements can result in cortical arousals, which may cause changes in sleep stage from a deep one to a light one, with the possibility of awakening as the final outcome (Ohayon et al., 2017). However, actigraphy does not measure actual sleep measures (e.g. sleep stages, cortical

arousals) which are critical determinants of sleep quality (Halson, 2019; McCarter et al., 2022). In addition, Falck et al. (2021) proposed an inverted U-shaped, dose-response relationship between training load and sleep, i.e. increased training load promotes sleep until a 'tipping point' beyond which acute fatigue or overreaching occurs, and sleep is disturbed. In the present study, participants were asked to suddenly increase the training volume during overload, i.e., from one day to the next. We can speculate that a gradual increase in training volume may have resulted in different sleep results. Future studies are required on the effects of various levels of accumulated training loads on sleep architecture.

Effects of high-heat-capacity mattress topper on sleep and temperature

We hypothesized that HMT would improve sleep quality by increasing conductive body heat loss. This hypothesis was not confirmed since no significant difference in any sleep variable was observed between HMT and LMT. It is well documented in the literature that sleep initiation occurs in close phase with the increase in distal skin regions temperature, leading to the subsequent reduction in CBT (Krauchi and Deboer, 2010). In this respect, previous studies have successfully manipulated CBT by thermal interventions in an attempt to promote sleep. When compared to a low-heat-capacity mattress, these studies report that sleeping on a high-heat-capacity mattress, from an acute perspective (i.e. a single night), had positive effects on sleep quality (i.e., higher SWS and REM proportions and/or reduction in WASO) in healthy young men (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020). These studies also noted a decrease in mean nocturnal CBT (from -0.11 to -0.16 °C) when sleeping on this bedding strategy. In the present study, the HMT group showed a trivial reduction (-0.025 °C) in mean nocturnal CBT following the overload compared with baseline, while a moderate increase (+0.132 °C) was observed in the LMT group. We can speculate that sleeping on HMT may have mitigated the overload-induced increase in CBT, without reducing CBT enough to induce any positive effect on sleep. Excessive training loads have been demonstrated to increase circulating cortisol, sympathetic activity and systemic inflammation (Roberts et al., 2019b), and we can assume that overload may have affected the normal downregulation of CBT during sleep. However, these findings should be interpreted with caution, as the intensity and the time of the day are important factors that may lead to different effects of exercise on circadian rhythms (e.g., CBT), and subsequent sleep (O'Connor et al., 1998; Aloulou et al., 2020a; Thomas et al., 2020). In the present study, participants performed very few training sessions (n = 3) close to bedtime, i.e., ending ≤ 1 h before bedtime. Future studies are required on the effect of excessive training loads on circadian rhythms (e.g., CBT) and subsequent sleep.

Limitations

Although we daily checked that training sessions had been effectively carried out, inter-individual differences in training load were observed, which may have resulted in inter-individual variability in sleep responses. Training load in week 4 was similar to week 1 in the LMT group, likely due to a lower number of training sessions performed by some athletes. There was also missing polysomnographic data from two participants in the HMT group at Test 3, resulting in a lower statistical power. In this respect, linear mixed-effects models were used to assess the relationship between training load and sleep at the individual level while dealing with missing data (Harrison et al., 2018). We only quantified training load using a subjective approach (i.e., sRPE), whereas other training metrics (e.g., distance covered, time spent in intensity zones) would have allowed to assess the dissociated effect of training components on sleep, as reported elsewhere (Roberts et al., 2019b; Aloulou et al., 2020b). Moreover, several factors not assessed in the present study may contribute to sleep disturbances during an overload period, such as muscle damage and DOMS, exercise-induced inflammation, depressed immune response, mood changes, increased stress, anxiety and sympathetic activity (Roberts et al., 2019b; Walsh et al., 2020). In addition, we did not use common sleep questionnaires following training periods, which would have provided additional information on the effect of manipulating training load on sleep perception. One originality of the present study was to monitor sleep using polysomnography at the end of each training period. However, this did not capture the daily response of sleep architecture to training load fluctuations, and further investigations should be conducted in this respect to provide more precise information for coaches on the effect of training load variations on sleep architecture.

Conclusion

The present study highlighted that higher accumulated training loads promote SWS and sleep propensity with some evidence of better sleep continuity. Such load-mediated effects may reflect the adaptative nature of sleep to diurnal activity and the role of sleep in physiological recovery. The present findings support overloading as an effective training manipulation to improve sleep quality in healthy individuals. In contrast to our hypothesis, the chronic use of HMT did not influence sleep quality. Additional research should be conducted on the potential benefits of HMT presenting different thermal properties or in various thermal environments on sleep architecture.

Study 3

Effect of a high-heat-capacity mattress topper on sleep in elite badminton players during a summer training period: does one size fit all?

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Associated publication

Under review

Abstract

The present study evaluated the effectiveness of a high-heat-capacity mattress topper (HMT) on sleep quantity and quality in elite badminton players during a summer training period. In a randomized, single-blinded and counterbalanced order, 14 world-class level badminton players completed two 1-week conditions: sleeping on HMT and sleeping on a low-heat-capacity mattress topper (LMT). Sleep was assessed daily using actigraphy and sleep diaries, and perceived sleep quality, thermal comfort and sensation were evaluated each morning. CBT was assessed continuously for two consecutive nights in each condition. Among 7 players, a single-night PSG was performed in each condition to evaluate sleep architecture. Despite a significant reduction in CBT (HMT *vs* LMT: -0.037°C , $p < 0.001$), sleeping on HMT did not affect any sleep variable, nor perceived sleep quality, thermal comfort or sensation (all $p > 0.05$) compared with LMT. Magnitude-based inference revealed a *most likely* increase in sleep efficiency (ES = 1.49 [90% CI: 0.67 to 2.31]) and an *almost certainly* decrease in WASO (ES = -2.04 [-2.60 to -1.47]) among players experiencing more restless sleep when they slept on HMT compared with LMT. The HMT did not influence elite badminton players' sleep during a summer training period. However, the present work provides perspective about potential greater benefits from sleeping on HMT for athletes experiencing more restless sleep. Further research on the effectiveness of this sleep strategy with an individual approach should be conducted to guide practitioners in prescribing this strategy.

Keywords: polysomnography, sleep architecture, individual sport, thermoregulation, recovery, bedding

Introduction

Sleep is recognised as essential in athlete performance and well-being (Samuels, 2008). It provides important restorative neurophysiological and cognitive functions, promotes memory consolidation, hormonal and emotional regulation, metabolic functions, modulation of the immune system, and plays a pivotal role in training-related stress-recovery balance (Halson and Juliff, 2017; O'Donnell et al., 2018a). However, a large body of studies suggested a high prevalence of sleep complaints and inadequate sleep quantity and/or quality in elite athletes when compared to recommended guidelines (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020) and/or self-reported sleep needs (Sargent et al., 2021).

It is well accepted that human sleep comfort, quantity, and quality are sensitive to the thermal environment (Okamoto-Mizuno and Mizuno, 2012; Lan et al., 2014, 2017). Thermoregulation and sleep regulatory systems are strongly linked by both neurophysiological and behavioural processes (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019). The initiation of sleep and the prevalence of SWS, a component of NREM sleep, are more likely to occur during the maximal rate of the circadian decline phase of CBT, which results from increased skin blood flow, skin warming, and body heat loss from the core to the environment (Okamoto-Mizuno and Mizuno, 2012; Szymusiak, 2018).

Nevertheless, elevated ambient night-time temperatures may alter the sleep-related circadian decline in CBT and heat loss. Deviation from thermoneutral conditions, i.e. the range of ambient temperatures where CBT is maintained only through regulating dry heat loss (Romanovsky, 2018), has been shown to result in poorer subjective sleep quality, increased WASO, longer SOL, and reduced amounts of SWS (Lan et al., 2014). In a large study investigating the relationship between meteorological factors and human sleep, Minor et al. (2022) reported that the likelihood of insufficient sleep is largest during the summer months when ambient temperatures are highest, with no evidence of short-term acclimatization of sleep. Furthermore, at a time of increasing and intensified exposure to high temperatures due to climate changes (Patz et al., 2014; Watts et al., 2018), this study also warns that in 2099, the projected rising night-time temperature may erode 50–58 h of sleep per person-year (Minor et al., 2022). Sport practice is not spared by this global warming issue. We can tacitly assume that elevated ambient temperatures, especially during summer, combined with increased body temperature induced by heavy training, may interfere with the body's heat loss during sleep (Aloulou et al., 2020a), thereby amplifying the risk of inadequate sleep among elite athletes. This period can be particularly decisive for elite athletes, involving intensified training sessions to prepare for

major competitions, such as the summer Olympic Games. An International Olympic Committee consensus has emphasized the importance of designing a sleep-promoting environment for Olympic athletes (Reardon et al., 2019). In this context, practical strategies that may help to regulate the body temperature and, in turn, mitigate the potential adverse effect of summer temperature on sleep should receive particular attention.

Among these strategies aiming at facilitating thermoregulation, thermal properties of bedding have been identified as a key factor for achieving heat loss and quality sleep (Troynikov et al., 2018). Sleeping on a high-heat-capacity gel sheet on the top of a foam mattress from an acute perspective, i.e., a single night, has been demonstrated to enhance the slow and continuous removal of body heat during sleep by conductive heat transfer, leading to an increased SWS proportion and improved subjective sleep comfort in healthy sleepers (Kräuchi et al., 2018; Herberger et al., 2020, 2024). However, conflicting results have been observed in athletes. Recent works report reduced WASO and a higher REM sleep proportion in elite rugby players when they slept a single night on a similar high-heat-capacity bedding strategy (Aloulou et al., 2020b). Inversely, no significant chronic sleep benefit was found in well-trained endurance runners exposed to high training loads (Chauvineau et al., 2023a, 2023b). These studies were conducted at a bedroom night-time ambient temperature ranging from 20°C to 23°C, which precludes to determine the relevance of such bedding strategy in nocturnal warmer temperatures encountered during the summer. Furthermore, describing individual responses to this strategy would also seem particularly important in elite athletes (Bonnar et al., 2018), given the inter-variability of sleep (Nedelec et al., 2018) and heat sensitivity (Racinais et al., 2012).

The aim of the present study was to evaluate the effectiveness of a high-heat-capacity mattress topper (HMT) on both objective and perceived sleep quantity and quality, as well as thermal comfort, in elite badminton players during a summer training period. We hypothesized that HMT promotes the objective and perceived sleep quality compared with a control bedding condition.

Materials and methods

Participants

A total of 14 international to world-class level badminton players from the French Badminton Association (8 females and 6 males), participated in the study (mean \pm SD; age: 23.6 \pm 4.2 years; body mass = 69.1 \pm 7.3 kg; height = 175.8 \pm 6.7 cm). Players were housed either at the training centre where this protocol took place (French Institute of Sport – INSEP, Paris, France; n = 8)

or at their personal residence (n = 6). Players were considered as elite/international (i.e., Tier 4, n = 12) or world class level (i.e., Tier 5, n = 2) according to the classification by McKay et al. (2022), as they were all currently competing in Badminton World Federation World Tour. Players were excluded (n = 0) in the case of medical circumstances influencing training frequency, taking medication for sleep disorders, and travel involving crossing of several time zones in the 2 weeks preceding the protocol.

Experimental design

The study was carried out during summer over a 2-week typical, in-season training period, between July and September 2023. Players had neither vacation nor competition during this period. According to a randomized, single-blinded, crossover and counterbalanced order design, the protocol consisted of two 1-week conditions: sleeping on HMT and sleeping on a low-heat-capacity mattress topper (LMT, control bedding condition). An overview of the study design is presented in **Figure 33**. Players were blind to the type of mattress topper used, and received no information about it.

At the start of the protocol, they completed the following sleep questionnaires: (a) the MEQ to identify the individual's chronotype (Horne and Ostberg, 1976), (b) the ESS to assess the daytime sleepiness (Johns, 1991), (c) the PSQI to evaluate the perceived sleep quality (Buysse et al., 1989), (d) the FIRST to measure the vulnerability of sleep disturbance in response to common stressors (Drake et al., 2004), (e) and the ISI to assess the severity of insomnia symptoms (Morin et al., 2011).

They also wore a wristwatch actigraphy from before going to bed to getting up in the morning to daily monitor the sleep-wake patterns, and filled an electronic sleep diary to report the daily subjective ratings upon waking (i.e., perceived sleep quality, thermal comfort and sensation, bedding comfort and effectiveness of bedding). Night-time bedroom temperature was continuously recorded throughout the study. The CBT was monitored during two consecutive nights between the second and fourth night of each condition. Together with CBT, a subgroup of players (5 females, 2 males) performed a one-night PSG in their own sleep environment to assess sleep architecture, i.e., the internal structure of sleep. The same qualified practitioner equipped them with the PSG equipment at ~19:30. During the protocol, training volume, the number and the timing of training sessions each player performed were noted.

Players were requested not to modify their usual diet and sleep habits, and to keep their own blanket, pillow, and sleepwear throughout the protocol. They were not restricted from

consuming caffeine, alcohol, or sleep medication throughout the protocol. Because the players were free from training and sleeping outside their homes during the weekend, we focused solely on the nights spent on the mattress toppers and preceding a training day, i.e., from Sunday evening to Friday morning.

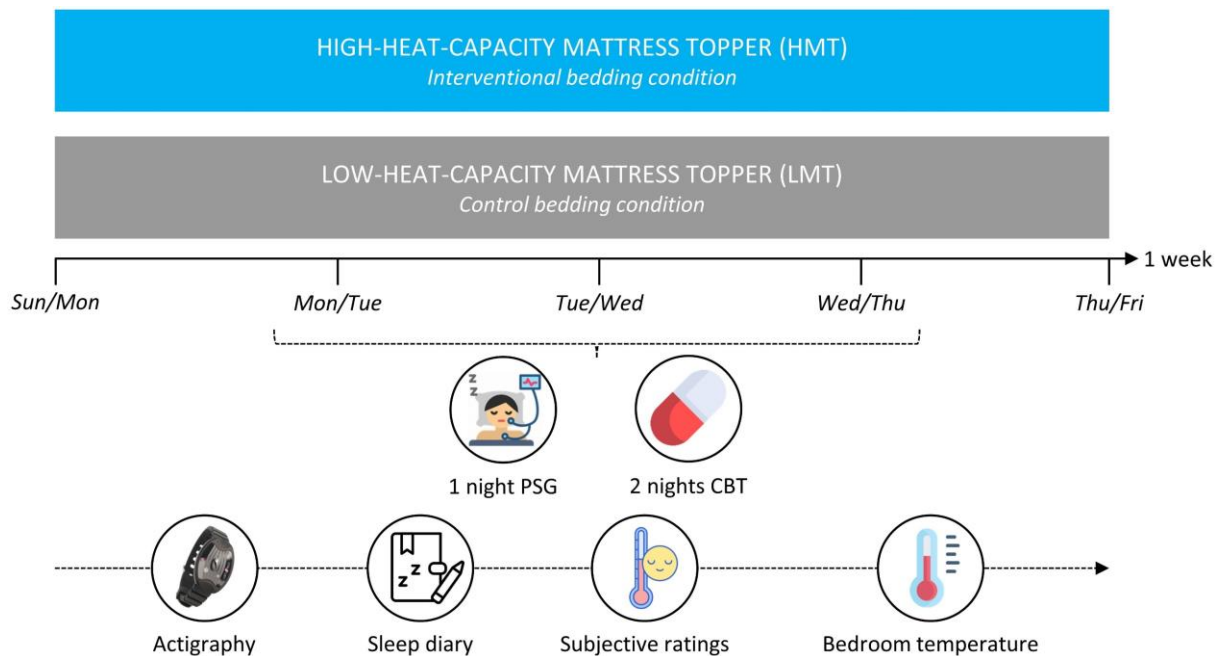


Figure 33. Design of the experimental protocol. *Abbreviations:* PSG, polysomnography; CBT, nocturnal core body temperature. *Notes:* The CBT was monitored during two consecutive nights between the second and fourth night of each condition, alongside a one-night PSG ($n = 7$ players). Subjective ratings = perceived thermal comfort and sensation.

Individual sleep patterns

For actigraphy-based TST, sleep efficiency, WASO and SOL, the median value was calculated in the LMT condition, during which the thermal properties of bedding were not manipulated. The median determined a cut-off separating the 7 players with the most adequate values from the 7 players with the least for the same variable.

Data and statistical analysis

Data processing and statistical analyses were conducted using R software (version 4.3.2). The normality of the distribution of training volume, the timing of training sessions, bedroom temperature, actigraphy-based sleep parameters and subjective ratings was checked using the Shapiro-Wilk test. All data were normally distributed except perceived stress and thermal comfort as well as the perceived effectiveness of bedding. The Student paired t test or Wilcoxon

test were applied to test the null hypothesis for no difference between HMT and LMT, depending on the normality of data distribution.

To assess between-condition differences on night-time CBT kinetics, a linear mixed-effects model ("lmer" function of the "lme4" R package) was fitted to consider the non-independence of observations at the level of individuals. The model included the player as a random effect nested within the monitored CBT night (categorical predictor: 1 and 2), and the condition and 10-min time interval (main effects and interactions) as fixed effects. The normality of the model residuals was visually checked using a Q–Q plot, and no obvious deviations from normality were revealed. The significance of the categorical fixed-effects was determined using the "mixed" function from the "afex" R package, using type 2 tests. When a significant effect was reported, *post-hoc* Student t-test pairwise comparisons were conducted using least square means contrasts with the Bonferroni correction for alpha adjustments ("emmeans" function of the "emmeans" package). Results are expressed as the mean \pm SD and the level of significance was set at $p < 0.05$.

Between-condition differences in PSG-inferred variables were assessed using a magnitude-based inference approach ("smd_test" function of the "mbir" R package). This approach was adopted to indicate the magnitude of the change in data within small sample sizes, which is typically more relevant to provide evidence-based decisions in sports medicine and exercise sciences (Hopkins, 2022). We also used a magnitude-based inference approach to compare the changes in actigraphy-based sleep parameters between the HMT and LMT conditions according to individual sleep patterns, i.e., the group with the most adequate values and the group with the least adequate values. Based on the Shapiro-Wilk test, no relative values for each sleep patterns deviated from the normality. The effect sizes (ES) were calculated to interpret the magnitude of the difference in means values, and were interpreted using the following criteria: $<0.2 = \textit{trivial}$, $0.2-0.6 = \textit{small}$, $0.6-1.2 = \textit{moderate}$, $1.2-2.0 = \textit{large}$, and $>2.0 = \textit{very large}$ (Hopkins et al., 2009). Quantitative chances of greater or smaller changes than the smallest worthwhile change (SWC, defined as $0.2 \times \text{ES}$) were evaluated qualitatively as follows: $<1\% = \textit{almost certainly not}$, $1-5\% = \textit{most unlikely}$, $5-25\% = \textit{probably not}$, $25-75\% = \textit{possibly}$, $75-95\% = \textit{likely}$, $95-99.5\% = \textit{most likely}$, $>99.5\% = \textit{almost certainly}$. If the probability of the effect being substantially positive or negative were both $>5\%$, then the effect was interpreted as *unclear*.

Results

Sleep questionnaires revealed a global ESS score >10 among 10 out of 14 players (female, n = 6/8; male, n = 4/6), which is an indicator of excessive daytime sleepiness. The PSQI scores were ≥ 5 among 10 out of 14 players (female, n = 7/8; male, n = 3/6), indicating poor global sleep quality. Furthermore, 9 out of 14 players (female, n = 7/8; male, n = 2/6) reported a FIRST score ≥ 18 , indicating high levels of sleep hyperarousal. The global ISI scores were also high in 6 out of 14 players (female, n = 5/8; male, n = 1/6) who reported *sub-threshold insomnia*, and in 1 female player who stated *moderate insomnia*. No player had an extreme morning or evening chronotype (i.e., scores <31 and >69).

Training volume, bedroom temperature and wellness ratings

No significant difference between HMT and LMT conditions was observed for averaged training volume (HMT: 24.9 ± 1.6 h, LMT: 23.9 ± 2.3 h, $p = 0.66$, ES = 0.37), the start of the first (HMT: $9:03 \pm 00:11$, LMT: $9:05 \pm 00:11$, $p = 0.20$, ES = -0.12) and the end of the last training of the day (HMT: $17:42 \pm 00:23$, LMT: $17:46 \pm 00:23$, $p = 0.55$, ES = -0.17), as well as the night-time bedroom temperature (HMT: $26.0 \pm 1.7^\circ\text{C}$, LMT: $26.7 \pm 1.6^\circ\text{C}$, $p = 0.32$, ES = 0.25, min–max: 22.4–30.7°C). There was also no significant difference between conditions in perceived general fatigue ($p = 0.90$, ES = -0.04) and pain ($p = 0.79$, ES = -0.07). Perceived stress was significantly higher in HMT than LMT condition ($p < 0.05$, ES = 0.81).

Sleep, thermal and bedding perception

Actigraphy, SSI score, and thermal and bedding perception

Regarding actigraphy data, bedtime, get-up time, TIB, TST, sleep efficiency, WASO, SOL and SSI score did not differ significantly between the conditions (all $p > 0.08$, **Table 15**). There was no significant difference between conditions in subjective ratings, including SSI score, perceived thermal comfort and sensation, as well as perceived bedding comfort and effectiveness of bedding (all $p > 0.21$).

Polysomnography

Regarding PSG data, magnitude-based inferences revealed *unclear* differences between HMT and LMT conditions in bedtime, get-up time, TIB, TST, SOL, WASO, sleep efficiency, N1, N2 and SWS proportions, sleep stage shifts and cortical arousals. Only REM sleep proportion was *likely* lower (ES = -0.57 [90% CI: -1.29 to 0.16]) in HMT than LMT (**Table 16**).

Table 15. Actigraphy data and subjective ratings in LMT and HMT conditions.

	LMT (n = 14)	HMT (n = 14)	p-value	Effect size
Actigraphy				
Bedtime (h:min)	23:21 ± 00:28	23:33 ± 00:27	0.08	0.51 (<i>small</i>)
Get-up time (h:min)	07:48 ± 00:25	07:52 ± 00:25	0.46	0.20 (<i>small</i>)
TIB (min)	506.6 ± 26.3	498.5 ± 32.6	0.24	-0.33 (<i>small</i>)
TST (min)	460.4 ± 26.7	455.4 ± 37.3	0.49	-0.19 (<i>trivial</i>)
Sleep efficiency (%)	90.9 ± 3.3	91.3 ± 2.6	0.47	0.20 (<i>small</i>)
WASO (min)	28.0 ± 12.7	26.5 ± 7.8	0.39	-0.24 (<i>small</i>)
SOL (min)	15.7 ± 10.0	13.4 ± 8.5	0.28	-0.30 (<i>small</i>)
Subjective ratings				
SSI score	19.2 ± 2.8	18.9 ± 3.5	0.62	-0.14 (<i>trivial</i>)
Thermal comfort (A.U.)	0.9 ± 0.5	0.9 ± 0.5	0.60	-0.18 (<i>trivial</i>)
Thermal sensation (A.U.)	1.1 ± 1.0	0.7 ± 1.4	0.31	-0.29 (<i>small</i>)
Bedding comfort (A.U.)	7.1 ± 1.0	7.4 ± 0.9	0.21	0.35 (<i>small</i>)
Bedding effectiveness (A.U.)	3.8 ± 1.3	4.5 ± 1.0	0.44	0.32 (<i>small</i>)

Abbreviations: HMT, high-heat-capacity mattress topper condition; LMT, low-heat-capacity mattress topper condition; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset; SOL, sleep onset latency; A.U., Arbitrary unit; SSI, Spiegel sleep inventory.

Core body temperature

A main effect of 10-min time interval ($p < 0.001$) and condition ($p < 0.001$) was observed for CBT. There was no significant 10-min time interval \times condition interaction ($p = 0.97$, **Figure 34**). A *small* reduction in average CBT during the sleep period ($\Delta\text{HMT-LMT} = -0.037^\circ\text{C}$, $p < 0.001$, $\text{ES} = -0.27$) was noted in HMT compared with LMT.

Individual sleep patterns

The change in actigraphy-based sleep parameters between HMT and LMT conditions based on individual sleep patterns are presented in **Figure 35**. It is *most likely* that there was a greater positive change in sleep efficiency ($\text{ES} = 1.49$ [90% CI: 0.67 to 2.31]) when sleeping on HMT among players below 91.6% compared to those above it. An almost certainly greater positive

change was observed in WASO (ES = -2.04 [-2.60 to -1.47]) when sleeping on HMT among players above 27.1 min compared to those below it. *Unclear* change between sleep patterns was noted for TST (median cut-off = 465.6 min) and SOL (median cut-off = 15.6 min).

Table 16. Polysomnography data in LMT and HMT conditions.

	LMT (n = 7)	HMT (n = 7)	ES [90% CI]
Bedtime (h:min)	23:37 ± 00:38	23:36 ± 00:31	-0.04 [-0.87 to 0.78]
Get-up time (h:min)	7:46 ± 00:31	7:56 ± 00:34	0.26 [-0.26 to 0.78]
TIB (min)	488.8 ± 35.4	500.7 ± 45.6	0.25 [-0.30 to 0.79]
TST (min)	442.3 ± 29.1	452.2 ± 40.8	0.24 [-0.20 to 0.68]
Sleep efficiency (%)	90.6 ± 4.2	90.5 ± 6.3	-0.01 [-0.67 to 0.65]
WASO (min)	22.0 ± 12.6	29.8 ± 30.7	0.28 [-0.22 to 0.78]
SOL (min)	23.1 ± 20.0	17.4 ± 17.7	-0.26 [-1.05 to 0.54]
N1 sleep (%)	5.1 ± 1.2	5.3 ± 2.1	0.07 [-0.48 to 0.63]
N2 sleep (%)	47.2 ± 4.0	48.8 ± 4.4	0.32 [-0.30 to 0.94]
SWS sleep (%)	22.7 ± 3.5	24.0 ± 6.8	0.20 [-0.40 to 0.81]
REM sleep (%)	25.0 ± 3.2	22.0 ± 5.6	-0.57 [-1.29 to 0.16]*
Sleep stage shifts (events per hour)	11.2 ± 2.1	11.7 ± 2.2	0.20 [-0.30 to 0.69]
Cortical arousals (events per hour)	9.6 ± 2.0	10.8 ± 3.2	0.37 [-0.28 to 1.01]

Abbreviations: HMT, high-heat-capacity mattress topper condition; LMT, low-heat-capacity mattress topper condition; TST, total sleep time; WASO, wake after sleep onset; SOL, sleep onset latency; N1–N2, NREM sleep stages 1–2; SWS, slow wave sleep, REM, rapid eye movement sleep stage. *Notes:* *Likely difference.

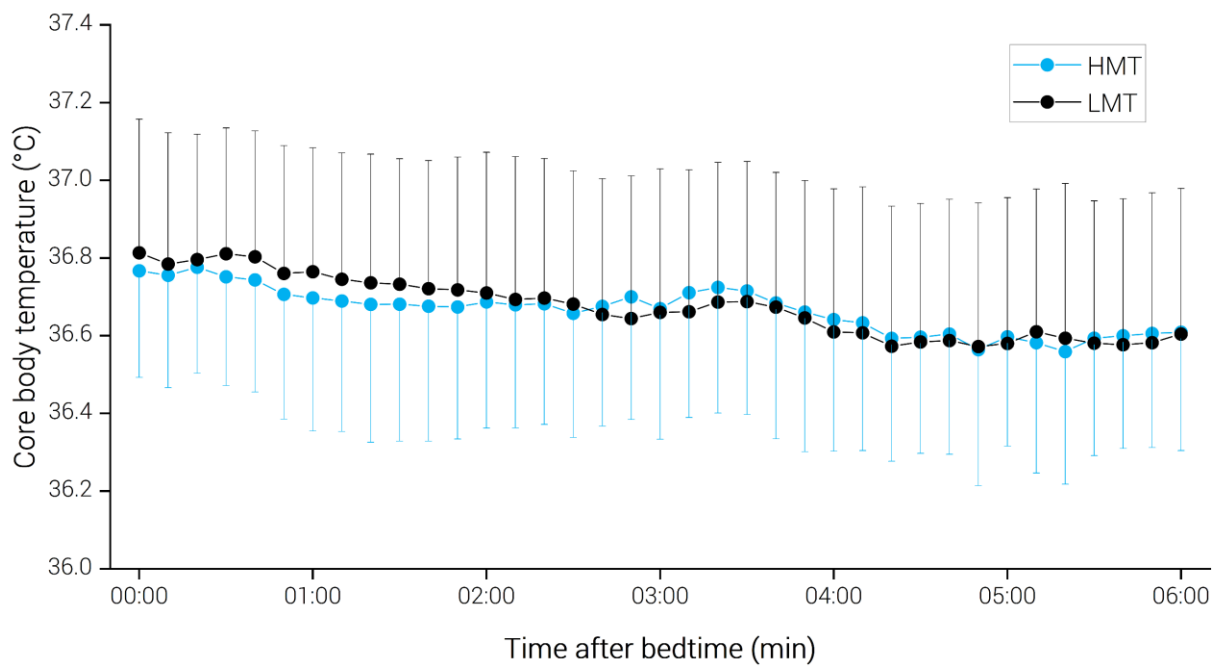


Figure 34. Mean \pm SD changes in core body temperature (CBT) over 6 h after bedtime in HMT and LMT conditions.

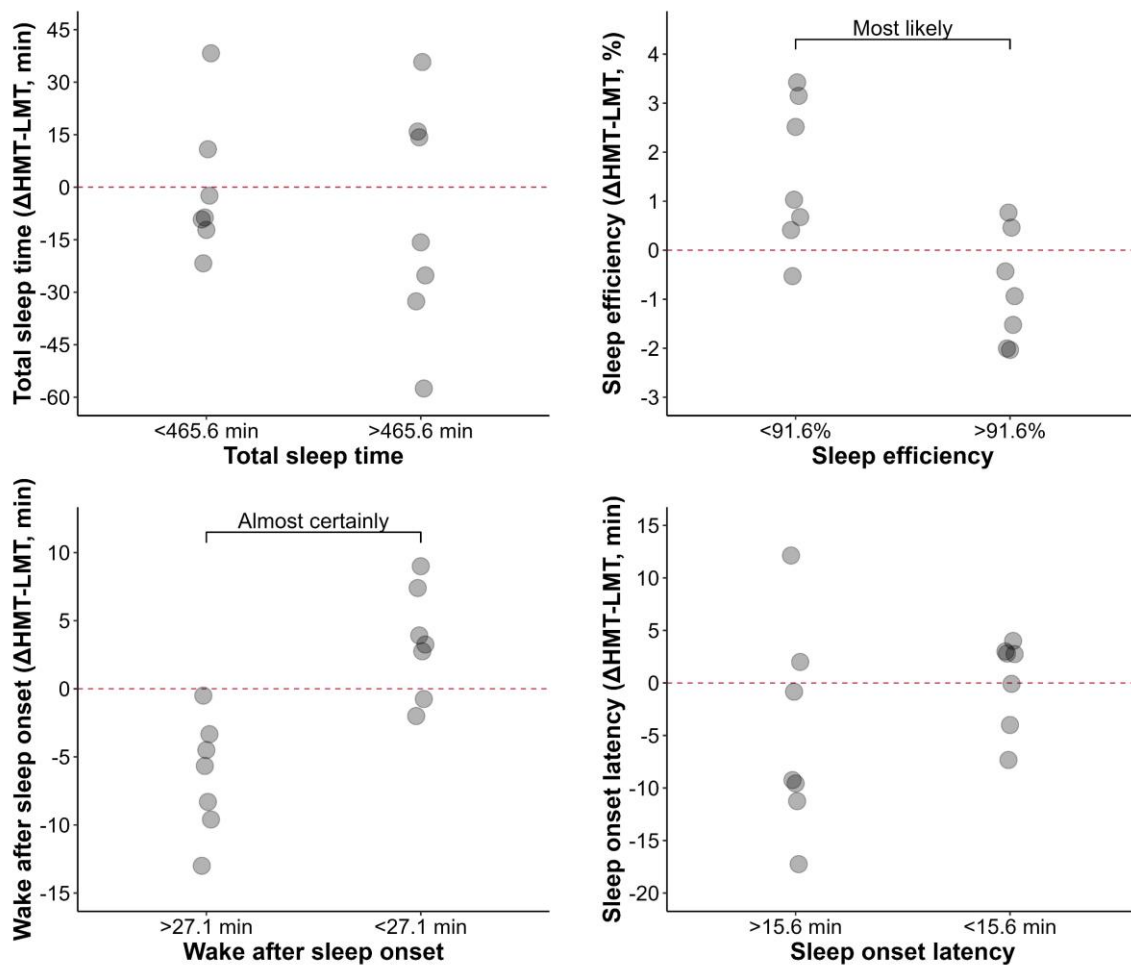


Figure 35. Changes in actigraphy-based sleep parameters when sleeping on HMT compared to when sleeping on LMT, according to individual sleep patterns. *Notes:* The red dotted line represents no difference between the two conditions.

Discussion

The present study aimed to assess the effectiveness of HMT on sleep quantity and quality in elite badminton players during a summer training period. The main findings revealed that (i) HMT did not significantly affect any sleep variables, nor subjective ratings on thermal and bedding perception compared with LMT, and (ii) players with poorer WASO and/or sleep efficiency demonstrated improved sleep efficiency and reduced WASO when sleeping on HMT compared to players who had better WASO and/or sleep efficiency.

The first finding from our study was a high level of subjective complaints related to sleep and sleepiness in the present elite badminton player population. This result is consistent with numerous previous studies that reported a high prevalence of sleep complaints in elite athletes from various sports (Gupta et al., 2017; Aloulou et al., 2021; Halson et al., 2021b). The sleep questionnaires were completed at the beginning of the present protocol, specifically during the summer training period. Summer is acknowledged as a critical period for insufficient sleep, attributed to elevated night-time temperatures and disruptions in circadian rhythms associated with longer sunlight days (Quante et al., 2019; Suzuki et al., 2019; Minor et al., 2022). Additionally, playing badminton at the highest level involves high training/competition demands, including multiple training sessions and/or matches per day, frequent long-haul travel associated with unfamiliar sleeping environment, competition anxiety, along with personal life obligations such as family, work, study commitments, and social demands. These factors have been identified as candidate risk factors for compromised sleep in athletes (Walsh et al., 2020), and could contribute, along with elevated summer temperatures, to sleep dissatisfaction. However, the average TST was ≥ 7 h, sleep efficiency $\geq 85\%$, WASO ≤ 40 min, and SOL ≤ 30 min which did not deviate from the National Sleep Foundation's recommendations for young adults (Hirshkowitz et al., 2015b; Ohayon et al., 2017). Only two players slept on average less than 7 h per night, and two others had a WASO > 40 min. This makes it difficult to assert the presence of sleep disturbances in the present population, or it may reflect that sleep needs were not satisfied (Sargent et al., 2021).

Effect of HMT on sleep and temperature

To our knowledge, this study is the first to assess the sleep benefit of HMT use during elevated summer temperatures within athletes' training contexts. We hypothesized that sleeping on HMT may promote both the quantity and quality of sleep in this specific situation. However, our hypothesis was not confirmed, since no difference between the HMT and LMT conditions was

found in any sleep variables. The use of HMT did not result in any changes in thermal comfort and sensation, nor did it affect the perception of bedding comfort and effectiveness compared to LMT. Only REM sleep proportions was *likely* lower (-3.0%) when sleeping on HMT compared with LMT. It has been suggested that REM sleep is more sensitive to the thermal environment than NREM sleep stages due to reduced tonic muscle tone and increased peripheral vasoconstriction (Szymusiak, 2018; Te Lindert and Van Someren, 2018).

Initial investigations assessing the interest of a high-heat-capacity bedding system from an acute perspective, i.e., a single night, have reported positive effects on sleep quality (i.e., higher SWS and REM sleep proportions and/or reductions in WASO) in healthy young men (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020). These studies also reported a reduction in mean nocturnal CBT from -0.11 to -0.16°C when sleeping on this bedding strategy. In the present study, a small reduction in mean CBT (-0.037°C) was observed in the HMT compared with the LMT condition. Previously, we also observed no significant chronic sleep benefits using HMT in well-trained endurance runners exposed to varying training loads despite a trivial reduction (-0.025°C) in mean nocturnal CBT (Chauvineau et al., 2023a, 2023b). Contradictory results between studies prevent to assert a consistent sleep benefit of HMT in all contexts for all individuals. In the present study, players reported a mean \pm SD thermal sensation of 0.9 ± 1.2 A.U., indicating a slightly warm perception during sleep throughout the study. Deviation from a comfortable thermoneutral zone may result in the activation of thermoregulatory responses (e.g., sweating, shivering, and changes in vasomotor tone) to regulate CBT and facilitate heat loss during sleep (Krauchi and Deboer, 2010; Romanovsky, 2018). We can assume that HMT may have mitigated the adverse effects of summer temperatures on the normal downregulation of CBT during sleep, without reducing CBT enough to induce a substantial positive effect on sleep. Given the increasing and intensifying exposure to high temperatures due to climate change (Patz et al., 2014; Watts et al., 2018), which is likely to further impact sleep, additional studies should be conducted to assess the effectiveness of further reducing the thermal resistance of HMT to promote heat loss and sleep.

Effect of HMT according to sleep patterns

Another important result of the present study was the observed difference in sleep benefits when sleeping on HMT between players with poorer WASO and sleep efficiency (independently), compared to those with better values. In detail, all players with high WASO in LMT condition experienced an improvement when sleeping on HMT. The rationale for such individual

responses has received little attention, but we can hypothesize that they may be related to inter-individual differences in sleep, heat sensitivity, and preferences. First, the high prevalence of sleep complaints suggests that some players experienced sleep difficulties when starting the protocol. It can be easier to promote sleep in poorer sleepers than in good sleepers because of a ceiling or maximum threshold of adequate sleep, beyond which further improvement may not be possible (Youngstedt, 2005). The use of HMT may have provided further advantages to players experiencing higher sleep difficulties. Second, there is inter-individual variability in the physiological adaptive responses to heat (Racinais et al., 2012; Corbett et al., 2018), which may have influenced the effects of HMT on sleep. Recent evidence shows that individuals experiencing greater heat loss during sleep also demonstrate a significant increase in SWS and decrease in REM sleep using a high-heat-capacity bedding system. Conversely, those with small heat loss did not exhibit significant changes in sleep architecture (Herberger et al., 2024). Body fat can vary among athletes both between and within sports (Zemski et al., 2015) and has an insulating effect that contributes to important inter-individual differences in thermoregulation (Zhang et al., 2001). Although this individual characteristic was not considered in the present study, it is possible that some players had better abilities to dissipate heat during sleep and benefit more from HMT to promote sleep. Finally, differences in individual sleep benefits when using HMT may result from personal preferences. Sleep quality and comfort are strongly linked with the mechanical and firmness characteristics of the bedding system, with spinal alignment and contact pressure being the predominant predictors (Radwan et al., 2015; Caggiari et al., 2021; Vitale et al., 2023a). Due to individual anthropometric differences, it is possible that some players may have been better suited to HMT than others. Additionally, the preferred sleeping position may have influenced the contact surface with the bedding system, thereby affecting the capacity for heat exchange between the sleeper and HMT (Tan et al., 2015). Further studies should be devoted to a high-heat-capacity bedding system, taking into account sleep patterns and preferences, as well as individual ability to dissipate heat during sleep. This could provide more precise guidance for prescribing such a sleep-promoting strategy with an individual approach (Fullagar and Bartlett, 2016).

Limitations

There are some limitations that need to be considered when interpreting the present findings. This study was conducted in real-sport settings which faced some logistical and operational challenges. Bedroom temperature was not controlled and varied substantially, i.e., from 22.4 to

30.7°C, although no significant difference between conditions was noted. This variation may have affected both the intra-individual variability of sleep and the thermal properties of bedding conditions. Due to practical constraints, PSG was conducted with only seven players, with a single-night assessment per condition in a paired design. Exploring sleep architecture in elite athletes poses significant challenges. In this respect, we employed magnitude-based inference as a pragmatic approach to making decisions regarding PSG data and sleep patterns comparison, given the applied nature of the present study and to deliver perspectives for future research (Hopkins, 2022).

Conclusion

The present study highlights that the use of HMT did not influence sleep quantity and quality, nor subjective ratings of thermal and bedding perception during a summer training period. However, potential greater benefits from sleeping on HMT were observed for athletes experiencing more restless sleep. Further research should explore the potential benefits of HMT with an individualized approach, considering individual sleep patterns, preferences, and ability to dissipate heat during sleep. This will provide more precise guidance regarding the relevance of prescribing this sleep strategy on an individual basis.

General discussion

The main objective of the present PhD thesis was to deepen our understanding of the impact of training load on athletes' sleep and to explore the relevance of a high-heat-capacity bedding system in this population. To do so, we employed *in situ* experimental designs that incorporated both objective and subjective measures of sleep to encompass concepts of sleep quantity and quality. The implementation of EEG-based analysis of sleep architecture within typical training contexts among elite athletic population represents the original contribution of the present work. Our findings showed that: i) **in a context of controlled training load during an elite rugby union training camp, covering high-speed distance promoted sleep duration and the continuity of sleep architecture. Sleep duration and efficiency were sensitive to the morning appointment time, with a negative impact when it was earlier than usual;** ii) **over a 6-week training program consisting of a usual training period, overload, and tapering, increasing the weekly internal training load enhanced the proportion of SWS, reduced SOL, and improved the continuity of sleep architecture. Conversely, increasing the daily internal training load led to an impairment in subsequent sleep efficiency, WASO and SOL, as inferred from actigraphy measurements;** iii) **the chronic use of a high-heat-capacity mattress topper (HMT) did not provide any sleep benefits across varied training loads or during a summer training period. Nevertheless, it showed potential sleep benefits in athletes experiencing more restless sleep.**

In this final section, we will develop **four main lines of discussion**. First, we will discuss the sleep patterns of the elite athletes studied in the present work, and the **extent to which training load can impact their sleep**. We will address potential contributors to this relationship. Second, we will examine the **effectiveness of HMT within the training contexts studied**. We will also consider potential individual responses to this strategy, along with its potential application in other sporting contexts. Third, we will present **strengths, consistent barriers** to investigating athletes' sleep, and **practical implications** of the present work. In the fourth and last line, we will consider **research perspectives** and propose **practical recommendations** arising from the present findings.

1. A consistent picture of sleep inadequacy in elite athletes

Upon initiating the current research, a growing body of studies suggest a high prevalence of sleep complaints and inadequate sleep quantity and/or quality among the elite athlete population (Gupta et al., 2017; Roberts et al., 2019b). Numerous studies have demonstrated the adverse effects of sleep loss on sport-specific performance (Fullagar et al., 2015; Craven et al., 2022). Conversely, some evidence suggests that athletes may experience performance benefits by extending their sleep, i.e., increasing their sleep opportunity beyond normal (Silva et al., 2021; Gwyther et al., 2022). This emphasizes the crucial role of sleep for athletes and suggests that a high proportion of them may not obtain the adequate sleep required for achieving peak performance. In this regard, our research primarily focused on elite athletes (**studies 1 and 3**) to deepen our understanding of their sleep and what factors affect sleep in their typical sporting context. During a 10-day training camp in young elite rugby union players (**study 1**), we observed a high prevalence of nights characterized by a TST <7 h (30.3%), WASO >40 min (77.8%) and sleep efficiency <85% (43.4%). Elite badminton players in **study 3** reported experiencing a high level of subjective complaints related to sleep and sleepiness, supporting the evidence for certain sleep difficulties in elite athlete population (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020).

It is important to note that actigraphy-based sleep variables and sleep questionnaires differed within these two studies. Despite a high prevalence of inadequate sleep during the camp (**study 1**), only one of the 26 rugby union players experienced “poor sleep quality” and one other reported “moderate insomnia” based on PSQI score ≥ 5 and ISI score ≥ 15 , respectively. Conversely, the high prevalence of perceived sleep difficulties for badminton players in **study 3** were not accompanied by inadequate actigraphy-based sleep values. Numerous studies do not show agreement between objective and subjective measures of sleep in healthy subjects (Baker et al., 1999; Lockley et al., 1999; Forner-Cordero et al., 2018). In the present work, we used sleep questionnaires to assess athletes' perception of their sleep before participating in the protocol. These discrepancies may result from the fact that we did not objectively assess sleep simultaneously, and that sleep questionnaires, such as the PSQI, evaluate perceived sleep over prior weeks or months, potentially introducing recall bias. On the one hand, rugby union players underwent a training camp in **study 1**, which may have contributed to disruptions in sleep patterns among rugby union players compared to a typical training period at home (Pitchford et al., 2017). On the other hand, the sleep dissatisfaction reported by badminton players (**study 3**) may indicate that sleep needs were not met when starting the protocol. This may be

attributed to the timing of the protocol during a vulnerable summer period for sleep (Suzuki et al., 2019; Minor et al., 2022), as well as the high physiological and psychological demands of training and competition, frequent long-haul travels, competition anxiety, and personal life obligations (Walsh et al., 2020). These factors are all inherent to competing badminton and rugby union at the highest level.

Overall, despite discrepancies between sleep monitoring methods, the picture that some elite athletes experience inadequate and/or dissatisfied sleep persists in the present experimental studies. Considering its importance for the athletes' health and their performance (Halson and Juliff, 2017; O'Donnell et al., 2018a), we aimed to further understand the potential sleep disturbance factors associated with practicing sport at the highest level and to investigate the effectiveness of a practical sleep strategy in mitigating these disruptions.

2. Impact of training load on athletes' sleep

In the literature review (from page 53), we have seen that excessive and/or accumulated increased training load (overload) may contribute to sleep disturbances in elite athletes (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020). However, this evidence is mainly derived from actigraphy-based and/or subjective approaches. This sport-related issue was thus not yet fully understood, particularly concerning: 1) the impact of manipulating training load and specific physical demands on sleep architecture, which is critical in determining sleep quality, and 2) the associated physiological and psychological changes, as well as the organization and life around training, in determining whether training load affect athletes' sleep.

2.1. Is training load beneficial or detrimental for athletes' sleep?

In the context of a rugby union training camp (**study 1**), we reported a positive influence of daily increased high-speed (>18 km.h⁻¹) distance covered on subsequent sleep duration and continuity of sleep architecture, as indicated by a reduction in the number of sleep stage shifts. Similarly, the weekly accumulation of training load in endurance runners subsequently increased the proportion of SWS, reduced SOL, and enhanced the continuity of sleep architecture, by reducing the probability of transition from N1 sleep to wakefulness (**study 2-B**). The number of awakenings >5 min following the overload was also reduced compared with the 2-week usual training regimen. Our findings are consistent with Taylor et al. (1997), who reported increased SWS proportions, assessed using a single-night PSG, in elite female swimmers when training load is higher (at the onset of the season and during an overload)

compared to a taper period. Nevertheless, this study only involved seven elite athletes practicing a non-weight-bearing discipline and did not consider the external and internal training load achieved. No study had neither been conducted in sports involving high-intensity intermittent and combative efforts such as rugby union, nor in endurance running, which are prone to induce a greater extent of muscle damage, inflammatory responses, and DOMS, likely to affect sleep (see from page 61), compared to swimming (Nieman et al., 2014; Tavares et al., 2017; Naughton et al., 2018). To the best of our knowledge, the present experimental studies provide the first evidence of a positive influence of accumulating running demand during a typical training period on propensity, depth, and stability of athletes' sleep architecture. Based on the "two-process model" of sleep regulation (Borbély et al., 2016), and the restorative theoretical function of sleep (Frank and Heller, 2019), sleep propensity and the amount of SWS adapt to the body's needs for recovery. That is, the body attempts to recover following training by increasing sleep duration, if possible, and by shifting and enhancing the stability of sleep architecture towards a deeper, more restorative sleep (Kredlow et al., 2015; Dijk and Landolt, 2019; Park et al., 2021). The findings of the present thesis support the adaptive nature of sleep architecture in response to athletes' need for recovery after training.

However, several previous studies across various sports have suggested that prolonged significant increases in training load (overload period, >25% compared to usual training) can have a detrimental effect on athletes' sleep, as inferred from actigraphy measurements (Roberts et al., 2019b). Unlike **study 2-B**, which examined sleep architecture after each training period, the initial aim of **study 2-A** was to compare sleep across the 2-week usual training regimen, overload, and taper training periods. This is why we employed easy-to-use actigraphic measurements to daily assess sleep-wake patterns. No significant changes were noted in actigraphy data between these training periods (**study 2-A**). Sleep disorders are particularly observed alongside extreme fatigue states, such as overreaching or overtraining (Kellmann, 2010; Meeusen et al., 2013; Schweltnus et al., 2016). In our study, two-thirds of participants were diagnosed as "non-overreached", while the remaining tier experienced overreaching. The underrepresentation of maladaptive responses to overload could explain the preservation of sleep-wake patterns across the training program. In the context of **study 1**, the training camp program was designed to prepare rugby union players for two exhibition matches in a congested period of time (i.e., 5 days) without accumulating excessive fatigue. It is possible that players did not experience a maladaptive response to training due to moderately intensified training load, which may not have been high enough to disrupt sleep. Our findings support the

interest of increasing training load as long as fatigue is not too high in a training program, and considering the level of fatigue and training responses to preserve sleep, well-being and promote performance (Aubry et al., 2014; Bretonneau et al., 2024). Moreover, the overload period (**study 2**) and the training camp (**study 1**) were quite short. Hausswirth et al. (2014) observed a significant reduction in sleep duration only from the third week of an overload training period (+35% in training volume) compared to a usual training regimen. We cannot exclude that a longer overload period or a longer training camp would have ultimately led to sleep disruption in the present work.

To further elucidate the dose-response effect of training load on sleep, it is crucial to consider the individual sleep patterns, adjusting for the training load achieved by each athlete. Indeed, a statistical period comparison approach, as mentioned earlier, does not account for the inter-individual variability in the weekly training load achieved, as observed in **study 2** (ranging from +10% to +170% during overload compared to baseline). Linear mixed-effects models are a more flexible and better-suited method for the present ecological training context (Boisgontier and Cheval, 2016; Harrison et al., 2018). Such models revealed impairments in actigraphy-based sleep efficiency, WASO, and SOL following increases in the daily training load of endurance runners (**study 2-A**). Additionally, we further detailed the relationship between daily training load and actigraphy data within each training period. For each additional 100 A.U. in training load, WASO ($\beta = +0.5$ min; 95% CI: 0.1 to 1.0 min; $p < 0.05$) and sleep efficiency ($\beta = -0.2\%$; 95% CI: -0.4 to -0.0%; $p < 0.05$) are significantly impaired only during the overload period, and not during the baseline and taper period (all $p > 0.07$). Sleep duration and SOL were not significantly associated with the prior daily training load, regardless of the training period (all $p > 0.09$). These results differ from the aforementioned positive influence of increased running demands on sleep architecture (**studies 1** and **2-B**) and are aligned with previously mentioned studies suggesting that sleep is vulnerable during overload periods (Roberts et al., 2019b; Walsh et al., 2020). These supplementary findings from **study 2-A** offer deeper insights into whether increased training load leads to restless sleep, i.e., when training load increases within a training phase where it is unusually high (overload). They also support the inverted U-shaped dose-response model proposed by Falck et al. (2021), suggesting that increased training load is detrimental to sleep beyond a “tipping point”. The present results thereby encourage practitioners to devote special attention to sleep when athletes are exposed to overload training periods, considering the level of fatigue and training response to preserve both the quantity and

quality of sleep. This is crucial to maintaining the stress-recovery balance and achieving long-term training adaptations.

Nevertheless, it is important to distinguish between actigraphy and EEG-based measurements, which assess different dimensions of sleep. Actigraphy sleep parameters derive from the application of scoring algorithms to raw wrist movement data over time, making actigraphy quite limited in assessing sleep quality. This differs from EEG-based measurements, which records brain cortical activity (in addition to EOG-EMG signals for polysomnography) to evaluate sleep quantity and quality (Halson, 2019). Thus, our findings suggest that increased daily training load, particularly during overload periods, leads to increased restlessness during sleep (**study 2-A**). Considering the improvements in sleep propensity, depth, and stability of sleep architecture observed following increased training load (**studies 1 and 2-B**), it prompts the question of whether restlessness during sleep adversely affects sleep quality, restorative sleep, and consequently training adaptations. This also raises the question of the dilemma between the quantity (actigraphy) and quality (polysomnography) of collected data and the choice of measurement tool to assess sleep in relation to factors that may influence it. It is also important to note that despite the significance of increased daily training load on subsequent actigraphy parameters, the magnitude is quite modest. For instance, linear mixed-effects models from **study 2-A** estimate that adding 25 min to an usual training session lasting 60 min, rated as "somewhat hard" (i.e., 4 on the CR-10 scale (Borg et al., 1985; Foster et al., 2001)), would result in a subsequent reduction of 0.2% in sleep efficiency, and an increase of 0.4 and 0.5 minutes in WASO and SOL, respectively. The significance of such modest effect results from the number of measured observations, which has a significant impact on detecting relationships in linear mixed-effects models (Harrison et al., 2018). For comparison, the linear mixed-effects models in **study 2-A** included 777 daily recordings of actigraphy and training load data for 21 athletes over ~42 days, whereas the number for polysomnography data in **study 2-B** for the same athletes was 61. Consequently, restlessness during sleep following elevated training loads should be tempered, particularly considering the involvement of multitude other factors likely to affect athletes' sleep, such as frequent travels, sleep environment, training schedules as well as life around training (see below).

Some areas of uncertainty persist. First, the previous statistical approach considered only the absolute data, and it is possible that increased restlessness during sleep is more closely associated with an increased rate of change in training load. Second, in **study 2** we asked participants to suddenly change their training load from one period to the next. The sleep

response to a progressive (linear or non-linear) variation in training load compared to an abrupt change remains unclear. Third, the individual responses to training may suggest potential inter-individual sleep response to increased training load, which have been not investigating using the present statistical approaches. It is possible that athletes with poor sleep quantity and/or quality would have less ability to recover from the imposed load and would therefore be more sensitive, over time, to load variation compared to those with better sleep. Fourth, the present experimental work does not examine the sleep response to the whole possible range of training load and in turn to extremely low and high training loads. On the one hand, considering the importance of maintaining regular physical exercise for adequate sleep quantity and quality (Kredlow et al., 2015), it is possible that athletes may experience poorer sleep when they substantially reduce training load, such as during a pre-competitive tapering period. Impairments in sleep quantity and/or quality could be detrimental to maximizing the removal of residual fatigue after an overload period and before a competition. Only a significant quite modest increase in WASO (+4 min) was observed during the first week of taper compared to the second week of the usual training regimen period (**study 2-A**). On the other hand, ultra-endurance events present athletes with a significant number of stressors that may include sleep restriction (Lahart et al., 2013; Netzer et al., 2020) coupled with emotional disturbances and insufficient energy intake (Knechtle et al., 2005; Hulton et al., 2010). Outside of the scope of the present thesis, we investigated the sleep of an ultra-endurance cyclist engaged in a simulated 10,000 km tour, using a reduced-montage dry-EEG wireless device (Dreem headband, Paris, France). Main results showed a reduction in sleep duration per night throughout the tour (mean \pm SD: 05:13 \pm 00:30 h) compared to prior the tour (07:00 \pm 1:00 h), accompanied by a profound change in sleep architecture, notably with an increase in SWS proportion (46 \pm 7% vs 27 \pm 5%) (Nédélec et al., 2022). These results suggest that sleep architecture adapts to the body's needs for recovery towards deeper, more restorative sleep to promote recovery, even when athletes faced extreme physical demands that restrict sleep quantity.

2.2. Confounding factors to consider when exploring the impact of training load on sleep

Another original contribution of the current PhD thesis was to evaluate the impact of training load on sleep within typical training contexts, accounting for the organization of training and life around training. In **study 1**, we investigated whether changes in the mandatory morning

appointment time during a training camp could affect the sleep of elite rugby union players. The morning schedule is a strong modulator of prior sleep duration. Some studies have demonstrated shorter sleep durations when the morning training is earlier than usual (Sargent et al., 2014b, 2014a; Steenekamp et al., 2021). In **study 1**, the appointments were scheduled between 7:30 and 10:00, depending on the day. On the one hand, we observed that the earliest scheduled appointment (i.e., 7:30–8:30) was associated with the shortest previous average sleep duration during the training camp ($06:52 \pm 00:34$ h). This sleep reduction is explained by the fact that players did not compensate for having to wake up earlier by going to bed earlier. This shortest sleep duration occurred the night prior to the first match of the training camp. Considering the importance of sleep for athlete preparation and the negative impact of sleep restriction on subsequent athletic performance (see from page 23), advancing the morning appointment time just before a match may not be conducive to achieving peak performance during the match. This is particularly relevant as poor sleep is common prior to competitions in team-based contact sports, often due to nervousness and pre-competition thoughts (Sargent et al., 2022; Sim et al., 2024). On the other hand, some morning appointments were scheduled later by coaches to allow players longer periods of sleep.

Unexpectedly, delaying the morning appointment time led to players getting up later, but did not necessarily guarantee an increase in sleep duration and sleep efficiency. Although the highest average sleep duration was obtained when the appointment time was scheduled between 9:00 and 10:00, sleep duration (+17.2 min) and sleep efficiency (+2.1%) were most favourable when morning appointments were scheduled between 8:00–9:00 compared to 8:30–9:30. The Morningness–Eveningness Questionnaire revealed that players were either definitely morning type ($n = 1$), moderately morning type ($n = 6$), neither type ($n = 15$) or moderately evening type ($n = 2$). It is possible that some morning appointment times suited some players better than others due to their individual chronotype and associated preferred sleep schedules. Overall, these findings demonstrate that the sleep-wake patterns of elite rugby players are sensitive to the schedules of training camps, substantially more so than the impact of training load on sleep. The congested schedule, coupled with evening social engagements that may be extended by players when the following morning appointment is delayed, can interfere with individual circadian sleep regulation and act as important barriers to maintaining adequate sleep patterns during a congested training context. Special attention should therefore be allocated to sleep schedule regularity to promote both sleep quantity and quality (Halsom et al., 2022), particularly during a vulnerable training period for sleep (e.g., training camp, overload).

Our findings emphasize the importance of carefully planning the schedule and training program with the benefit of sleep to achieve the desired training adaptations and peak performance. However, we cannot ignore that coaches and athletes may face a dilemma between a congested training schedule period and the necessity of rest and adequate sleep periods. This leads athletes to balance on the thin line between doing right and doing too much. Training is recognized by athletes as the primary element for improving performance (Joncheray et al., 2021). However, environmental features and life around training, such as sleep, social and personal time, form part of the athletes' life balance (Burlot et al., 2018) and contribute to their sporting and personal development and mental health (Henriksen et al., 2010, 2023; Hauser et al., 2022). Being aware of the overall demands placed on athletes during a congested training period is crucial for managing training demand (load), rest and sleep periods to ensure their health, well-being and performance. In this regard, similar to the periodization of training, periodizing recovery, rest, and life around training is highly required in this context (Mujika et al., 2018).

Congested and heavy training periods, such as a training camp and overload, may induce mood disturbances and psychological distress (Meeusen et al., 2013). In **study 2**, the overload period resulted in higher experienced levels of mental stress, including disturbed breaks and emotional exhaustion, as well as a higher prevalence of stress-reaction symptoms compared to the usual training regimen, as measured by the RESTQ-Sport-36 and DALDA questionnaires. It has been shown that increased daily mental strain, as a mental component of stress, is likely to interfere with sleep duration and sleep efficiency among junior winter endurance athletes (Hrozanova et al., 2020). Mental stress induced by overloading may represent an additional confounding and explanatory factor for the observed restlessness during sleep in such a period (**study 2-A**). In our study, some participants usually exceeded 10 h of training per week, including one individual who typically ran 120 km per week. All of them carried out a full-time professional/study activity in addition to the imposed training program. The combination of increased training load and personal/professional constraints experienced by endurance runners could have posed a significant organizational challenge. Some of them added training sessions before or after their daily professional/student activities. In the context of workers in the general population, a more congested work schedule coupled with higher mental strain typically leads to sleep disturbances (Åkerstedt et al., 2002; Åkerstedt et al., 2015; Linton et al., 2015). In this respect, we can assume that overloading may contribute to restlessness during sleep, partly due to altered schedules and associated mental stress.

Additionally, beside organizational and psychological alterations resulting from increased training load, intensified training can disrupt numerous physiological pathways also potentially impacting sleep (see from page 61). Athletes often experience muscle soreness induced by heavy or unaccustomed training (Paulsen et al., 2012; Mizumura and Taguchi, 2016; Hainline et al., 2017b), and it has been suggested that they may lead to difficulties in remaining immobile during sleep in an attempt to get comfortable, thus resulting in restless sleep (Taylor et al., 1997; Hausswirth et al., 2014; Sargent et al., 2016). Unexpectedly, based on mediation analyses, we observed that muscle soreness perceived in the morning partially mediated the increase in WASO following increased daily training load, through a suppression effect. This indicates that higher training load induced greater muscle soreness which in turn was related to mitigate WASO (**study 2-A**). These findings do not provide evidence of the contribution of muscle soreness to increased restlessness during sleep following increased training load in endurance runners. Pain is recognized as an important modulator of sleep in general population, by fragmenting sleep and altering the continuity of sleep stages (Finan et al., 2013; Herrero Babiloni et al., 2020). However, the etiological mechanisms of muscle soreness that athletes may experience can differ from pain (Hainline et al., 2017b). No changes in sleep architecture, assessed using polysomnography, were observed following an unaccustomed eccentric exercise compared to concentric exercise in healthy young men (Breus et al., 2000). In the present thesis, we did not control or manipulate muscle soreness, given the *in situ* experimental design, which prevents us from drawing conclusions about the influence of muscle soreness as a confounding factor on the impact of training load on sleep. Additional studies should be conducted using objective and standardized methods to assess muscle soreness, such as using a pressor algometer to measure pressure sensitivity (Singh et al., 2011), in order to further elucidate their impact on athletes' sleep. It could also be relevant to investigate the impact of different types of pain (i.e., nociceptive, neuropathic and nociplastic/algopathic/nocipathic pain) that athletes may experience on sleep, which could facilitate clinical understanding and treatment (Hainline et al., 2017b).

Furthermore, we reported in **study 2-B** a higher CBT following the overload (+0.132°C) compared to the usual training period in the control group. It is possible that overload may affect the normal downregulation of CBT during sleep and contribute to restless sleep when exposed to excessive training loads. Heavy training close to bedtime may interfere with the natural body's heat loss during sleep, which is detrimental to sleep quality (Aloulou et al., 2020a). However, in **study 2**, participants performed very few training sessions ($n = 3$) close to

bedtime, i.e., ending ≤ 1 hr before bedtime. Therefore, a potential overload-induced increase in CBT remains hypothetical.

Finally, in addition to the positive effects of high-intensity running demands on subsequent sleep duration and continuity of sleep architecture in rugby union players, we also explored whether collision-based actions affect sleep during a training camp (**study 1**). Aloulou et al. (2020b) demonstrated that the number of collisions experienced by rugby union players during a match impaired subsequent sleep architecture by reducing SWS and increasing light sleep proportions. While theoretical, it has been suggested that repetitive head impacts sustained during such activities may lead to an accumulation of metabolic waste products (e.g., β -amyloid) which can disrupt the glymphatic system and consequently sleep (Jaffee et al., 2015; Asken et al., 2016). Our results revealed no significant relationship between the number of collisions and subsequent sleep, both inferred from actigraphy and EEG headband analysis. Discrepancies with previous study may result from the reduced daily number of collisions during the present training camp ($n = 10.7 \pm 9.9$) compared to those during an official match (Aloulou et al., 2020b, $n \approx 16$). In addition, the magnitude/intensity of the collisions was not monitored due to the difficulty of such investigations within an ecological design. Sport-related concussions typically lead to sleep disturbances in team-based contact sports (Jaffee et al., 2015; Leddy et al., 2023), but no concussions were reported throughout the training camp (**study 1**). In our study, players were equipped with instrumented mouthguards during each training, in which an accelerometer and angular rate sensor are embedded, providing insight into head acceleration frequency and magnitude during collision events (Tierney et al., 2021; Tooby et al., 2023). Such instruments have recently been introduced by World Rugby in competitions that use the premium Head Injury Assessment (HIA) protocols (World Rugby, 2023). Unfortunately, due to missing data in both the instrumented mouthguard and sleep data, these measures were not analysed in **study 1**. Further studies assessing sleep architecture in combination with information about the intensity of collision-based actions during a typical training period and matches, notably via instrumented mouthguards, are warranted.

3. Is a high-heat-capacity bedding system relevant for elite athletes?

3.1. Effect of a high-heat-capacity bedding system within typical training contexts

To the best of our knowledge, no study has reported the effects of a high-heat-capacity bedding system when used chronically during a typical training period. The present work is also the first to investigate the relevance of this strategy on the sleep of athletes exposed to elevated training

loads and during a typical summer training period. Our findings from **studies 2** and **3** showed that HMT did not confer benefits for sleep-wake patterns and sleep architecture in these situations. This conflicts with previous pioneer works that have shown positive effects on sleep quality (i.e., higher SWS and REM proportions and/or reduction in WASO) in healthy young males when sleeping on a high-heat-capacity bedding system over a single night (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020).

In the present PhD thesis, the choice of a bedding strategy was motivated by the fact that it serves as our nightly support for sleep, requires no personal involvement, and can therefore be used in any situation. This led us to consider it as a practical sleep strategy for elite athletes, given the numerous high-pressure sporting situations that challenge their sleep (Nedelec et al., 2018). From the thermoregulation perspective, the choice of studying HMT was based on the theory that promoting nocturnal heat loss, which leads to a slow, continuous decline in CBT, may enhance sleep quality (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019). Although we observed a significant reduction in averaged nocturnal CBT when sleeping on HMT compared to a low-heat-capacity mattress topper (LMT, control bedding condition) (**study 2-B**: $\approx -0.03^{\circ}\text{C}$; **study 3**: $\approx -0.04^{\circ}\text{C}$), the decline in CBT was substantially less than that reported in previous studies (from -0.11 to -0.16°C) (Kräuchi et al., 2018; Aloulou et al., 2020b; Herberger et al., 2020). The reductions in CBT observed in our studies are within the measurement error of the telemetric pill (standard error = 0.04°C ; Bongers et al., 2018). We can assume that the current HMT may not have effectively promoted the decline in CBT and heat loss to facilitate sleep.

We first explain these discrepancies by the fact that both **studies 2** and **3** were conducted in a context that potentially altered the normal downregulation of CBT during sleep and body heat loss. The 2-week overload period in **study 2-B** led to an increase in mean nocturnal CBT among the group of endurance runners who slept exclusively on LMT ($\approx +0.13^{\circ}\text{C}$), while runners sleeping on HMT showed a reduction in mean nocturnal CBT (-0.03°C) following the overload. It is possible that sleeping on HMT may have mitigated the overload-induced increase in CBT but not enough to induce any significant positive effect on sleep. However, caution is advised regarding this hypothesis, as the evidence for a nocturnal elevation in CBT in response to an overload period remains hypothetical. Regarding **study 3**, averaged nocturnal bedroom temperature was $26.3 \pm 1.9^{\circ}\text{C}$, ranging from 22.4 to 30.7°C . These summer temperatures were accompanied by a slightly warm perception reported by the players during sleep, without significant differences when sleeping on HMT compared with LMT. This suggests that the

thermal environment of **study 3** deviated from thermoneutral conditions in both conditions. In this regard, when adding the ambient temperature as a fixed effect to the linear mixed-effects model testing the between-condition differences in nocturnal CBT kinetics, the mean nocturnal CBT increased by 0.011°C (95% CI: 0.006 to 0.016°C , $p < 0.001$) for each additional 1°C increase in night-time bedroom temperature. Consequently, it is possible that HMT may have mitigated the detrimental effects of summer temperatures on the normal downregulation of CBT during sleep, without reducing CBT enough to induce any positive effects on sleep. In this regard, as part of a research project funded by the French Research Agency for the Paris 2024 Olympic Games, the effectiveness of HMT has been studied in 12 healthy males in a hot ambient environment (32°C) under well-controlled laboratory conditions. Sleeping on HMT had a protective effect in the hot environment, significantly increasing sleep duration ($+21 \pm 16$ min), sleep efficiency ($+5 \pm 4\%$), REM duration ($+13 \pm 5\%$), and reducing WASO (-11 ± 4 min) compared with a control mattress, as inferred from polysomnography. These effects were associated with lower average night-time skin temperature ($-0.22 \pm 0.06^{\circ}\text{C}$) and CBT ($-0.21 \pm 0.02^{\circ}\text{C}$) (not published).

A second reason for discrepancies with previous research relates to the properties of HMT, which differed from those of the bedding system used in earlier investigations. The thickness of the high-heat-capacity gel sheet was 2.5 cm in **studies 2** and **3** (see **Figure 18**), whereas it was reported to be 2 cm (Kräuchi et al., 2018; Herberger et al., 2020) and 4 cm (Aloulou et al., 2020b) elsewhere. Importantly, the manufacturer who designed the bedding systems remained consistent across previous studies, but differed from the one involved in the present work. It is therefore possible that the thermal characteristics vary depending on the manufacturer. Thermal resistance measurements of HMT and LMT have been performed by an independent laboratory specializing in building thermal assessment (see page 79). The evidence of differences in thermal properties was confirmed, as the results revealed $\sim 30\%$ lower thermal resistance for HMT compared to LMT in **studies 2** and **3**. Unfortunately, the thermal resistance values for the previously tested bedding systems are not available, which prevents us from making any robust comparisons. Following **study 2**, the density of the ticking covering HMT was reduced by $\sim 31\%$ in **study 3** to further enhance the heat exchange between the body and the bedding system. The thermal resistance was reduced by $\sim 14\%$ in **study 3** compared to **study 2** (see page 79). Nevertheless, despite the decrease in thermal resistance of HMT in **study 3**, the observed reduction in average nocturnal CBT (-0.037°C) between HMT and LMT may not have been enough to provide any sleep benefits. Consequently, this raises the question of the

dose-response relationship between the thermal resistance of HMT, heat-loss effects, and sleep benefits. Future studies are needed to determine the best bedding system design to promote athletes' sleep in various thermal and training conditions.

Finally, although we did not report a significant effect of HMT on sleep assessed over the entire night, we cannot exclude the possibility of varying effects of HMT on sleep depending on the progress of the night. Heat loss is typically greatest from the onset of sleep to 1–2 h after sleep onset, coinciding with the highest amount of SWS (Krauchi and Deboer, 2010). Given that SWS plays a pivotal role in the recovery process via muscle repair and adaptation (Léger et al., 2018), it seems relevant to focus on the first sleep cycles when exploring the effectiveness of a sleep strategy in athletes. In this regard, we conducted a pilot analysis on the changes in sleep architecture over sleep cycles according to HMT and LMT, using polysomnography data from **study 3**. As shown in **Figure 36**, differences in sleep architecture appear more pronounced

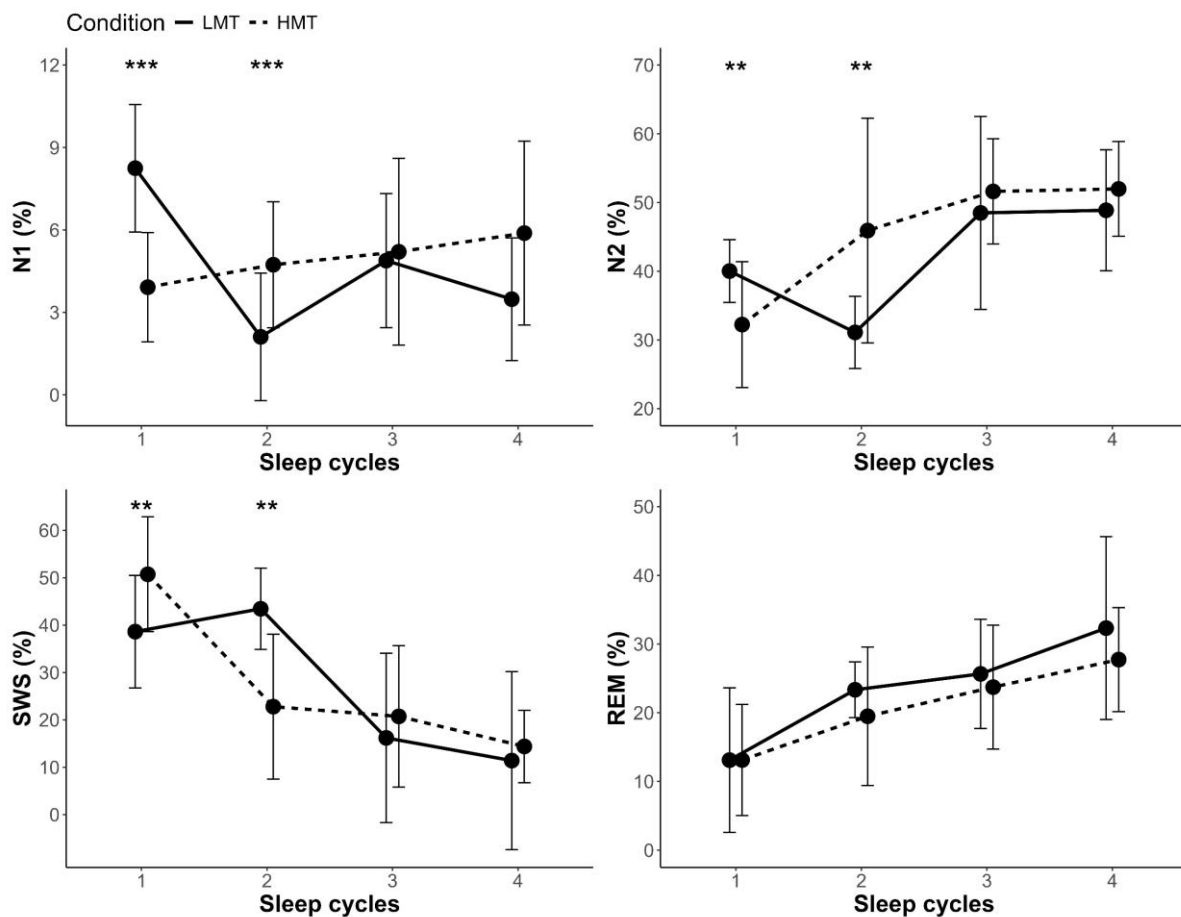


Figure 36. Difference in sleep architecture over the first four sleep cycles according to HMT and LMT conditions. *Notes:* Sleep cycles are detected based on criteria proposed by Feinberg and Floyd (1979), using the “SleepCycles” package for R as described in Blume and Cajochen (2021). We only considered the first four completed sleep cycles of all players, i.e., excluding any incomplete cycles. Sleep stages were expressed as a percentage of total sleep time within sleep cycle. Due to the small sample size ($n = 7$), we employed a magnitude-based inference approach to compare HMT and LMT conditions for each sleep cycle. **most likely, and ***almost certainly difference between conditions. *Abbreviations:* HMT, high-heat-capacity mattress topper condition; LMT, low-heat-capacity mattress topper condition.

during the two first sleep cycles. Notably, there was a *most likely* higher proportion of SWS during sleep cycle 1 when sleeping on HMT compared to LMT (ES = 0.86 [0.35 to 1.37]), and *most likely* lower proportion of SWS during sleep cycle 2 (ES = -1.42 [-2.35 to -0.48]). Nevertheless, it is important to note that these results do not conclude whether sleeping on HMT may affect sleep architecture during the first part of the night compared to LMT. Instead, they provide an example of a more detailed approach to evaluating the effectiveness of a strategy in promoting the quality of a critical part of the night.

3.2. High-heat-capacity bedding system: does it fit all?

Although our initial hypothesis regarding the effectiveness of HMT has not been fully confirmed among overall athletes studied, **study 3** provides perspective about potential sleep benefits for athletes experiencing more restless sleep. Specifically, badminton players with poorer WASO and sleep efficiency (independently) during the control condition, experienced greater positive changes in these sleep variables when sleeping on HMT compared to those with better values. While these initial findings are limited to the studied population and a specific summer training context, they suggest potential individual sleep responses when sleeping on HMT. These responses can result from inter-individual variability of sleep patterns and thermoregulation, as well as individual anthropometric characteristics and personal bedding preferences.

First, **study 2** involved endurance runners with no clinical sleep problems, as assessed by sleep questionnaires for inclusion criteria. Manipulating training load did not substantially compromise sleep, with evidence that sleep architecture and quality were promoted following increased load. In this context, it is possible that implementing HMT did not provide additional sleep benefits due to a ceiling effect of adequate sleep, beyond which further improvement may not be possible with solely HMT. Conversely, the high prevalence of sleep complaints among elite badminton players in **study 3** suggests that some of them experienced sleep difficulties when starting the protocol. It can be easier to observe benefits in poorer sleepers when implementing a sleep strategy (Youngstedt, 2005). The use of HMT may have provided further advantages to players experiencing sleep difficulties in **study 3**. Further investigations should explore the relevance of HMT as a sleep-promoting strategy for those experiencing sleep difficulties considering the high inter-individual variability in sleep patterns among elite athletes (Nedelec et al., 2018).

Second, there is also an important inter-individual variability in the thermo-physiological adaptive responses to the heat (Racinais et al., 2012; Corbett et al., 2018). As a reminder, the

ability to dissipate heat during sleep is crucial for sleep quality (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019). Herberger et al. (2024) demonstrated significant increases in SWS proportions in individuals experiencing greater body heat loss during sleep, when they slept on a high-heat-capacity bedding system. Those with minimal heat loss showed no significant changes in sleep architecture. Body fat has an insulating effect that may restrict heat loss during sleep (Anderson, 1999; Zhang et al., 2001) and can vary among athletes both between and within sports (Zemski et al., 2015). We can hypothesize that athletes with lower body fat and better abilities to dissipate heat during sleep may benefit more from HMT to promote sleep. However, body fat was not considered in the current thesis. Additionally, we monitored nocturnal CBT as an indicator of heat loss during sleep. It has been suggested that the decline in CBT is not directly associated with the amount of SWS. Instead, SWS would depend more on the increased CBT–proximal back skin temperature gradient, reflecting the heat transfer from the body core to the shell (Herberger et al., 2024). Unfortunately, such a measurement could not be performed in the present work due to challenges associated with incorporating additional sensors to sleep monitors within the athlete population.

Third, bedding preferences, sleeping position, and individual anthropometric characteristics may also contribute to hypothesized individual sleep benefits when using HMT. The mechanical characteristics of the mattress are crucial for sleep quality and body comfort, with spinal alignment and body contact pressure being the predominant predictors (Radwan et al., 2015; Caggiari et al., 2021; Vitale et al., 2023a). Some studies have highlighted that a recent bedding, adapted to individual firmness and sleeping position preferences, reduces common musculoskeletal pain and discomfort, and improves sleep quality (Jacobson et al., 2009, 2010). In the present thesis, the mattress toppers studied had identical characteristics for all participants. Some athletes may have been better suited to HMT than others due to their individual anthropometric characteristics and sleeping position preferences. Furthermore, the body surface area in contact with the sleeping surface, primarily determined by the sleeping position, may modulate the heat exchanges between the body and the bedding system, influencing thermal comfort (Tan et al., 2015; Troynikov et al., 2018). In this respect, we can assume that athletes with a larger body contact area with HMT (such as those sleeping on their stomach or back) were in more favourable conditions for heat exchange towards the bedding, and therefore derived more sleep benefits.

Overall, this encourages further research on the effectiveness of a high-heat-capacity bedding system in athletes with an individual approach, particularly considering factors such as sleep

patterns, heat loss abilities during sleep, anthropometric characteristics, and preferred bedding/sleeping positions. Such investigations could help to identify factors that differentiate potential high responders from low responders to this sleep-promoting strategy. This could provide more precise guidance for prescribing bedding environment adapted to the individual characteristics in the context of monitoring and supporting elite athletes' sleep (Fullagar and Bartlett, 2016; Halson, 2019).

3.3. Another possible unexplored usage of a high-heat-capacity bedding system

Elite athletes frequently perform evening competitions and training sessions, which have been identified as an important contributing factor for sleep disturbances in athletes (Gupta et al., 2017; Roberts et al., 2019b; Walsh et al., 2020). It is recognized that such alterations may result in part from elevated post-exercise body temperatures that persist during sleep (Aloulou et al., 2020a; Yue et al., 2022). In **study 2**, participants performed very few training sessions close to bedtime (ending ≤ 1 h before bedtime, $n = 3$), and training sessions ended no later than 18:30 in **study 3**. Given the detrimental effect of heavy evening training or competition on athletes' sleep (Roberts et al., 2019b), which can interfere with the body's heat loss required for quality sleep (Aloulou et al., 2020a), the effectiveness of HMT in these situations warrants further investigation.

Another possible unexplored usage of HMT is its combined effect with popular recovery strategies that act on thermoregulation. Hot and cold post-exercise recovery strategies are commonly used by elite athletes (Nédélec et al., 2013; Venter, 2014; Burlot et al., 2018). When used close to bedtime, they may interfere with the sleep-related thermoregulatory processes by increasing (hot exposure) or decreasing (cold exposure) body temperature (Wilcock et al., 2006; Versey et al., 2013; Stephens et al., 2018). A warm shower or bath, 1–2 h before bedtime, has been shown to improve sleep quality, particularly by reducing SOL and increasing sleep efficiency, with trends towards reduced WASO and increased SWS (Haghayegh et al., 2019). These findings have been associated with an increased heat loss close to bedtime, characterized by a higher rate of decline in CBT. In **studies 2 and 3**, participants were asked to not change their usual post-exercise recovery strategies. We did not collect the recovery strategies used, nor the time at which they showered or bathed. Thus, this raises the question of whether the use of HMT can expand heat loss following the use of a hot exposure post-exercise recovery strategy close to bedtime, and promote sleep quality.

In contrast, cold strategies such as cold water immersion, lead to a reduction in CBT and the activation of thermogenesis mechanisms to maintain CBT within reference thresholds (Ihsan et al., 2016; Romanovsky, 2018). In addition to its primary objective of promoting recovery, if performed close to bedtime, cold exposure could mitigate the increase in body temperature induced by training/competition and prevent it from remaining elevated at bedtime (Nédélec et al., 2015). In this regard, we investigated, prior to the current PhD thesis, the effects of different levels of body immersion in cold water, performed at ~8 PM after a high-intensity, intermittent running exercise, on sleep architecture in endurance runners (Chauvineau et al., 2021). We found that implementing a whole- (including the head) and partial-body cold water immersion could enhance sleep quality compared with an out-of-water control condition by reducing cortical arousals. Additionally, higher SWS proportions during the first part of the night were reported following the whole-body immersion compared with partial immersion. However, whole-body cold water immersion, notably through immersing the head, resulted in decreased parasympathetic activity and compromised thermal sensation and comfort, which may limit its practical application by athletes in the field (Chauvineau et al., 2021). Given the high thermo-sensitivity of the head (Pretorius et al., 2006), exposing the head to a high-heat-capacity pillow containing a gel sheet, similar to HMT, could be a potential strategy for activating the sleep-related heat loss mechanisms, while avoiding thermal stress. A high-heat-capacity pillow would also be a practical and portable strategy to carry in the various sleeping environments faced by athletes (training camp, competition). Furthermore, it has been demonstrated that local cooling applied to larger body sections (back and head) during sleep, by combining a cooling mattress and pillow (via circulated water), provides additional benefits in a hot ambient environment (32°C) on thermal comfort, WASO, and sleep efficiency (Lan et al., 2018). Exploring the relevance of combined high-heat-capacity mattress and pillow following heavy training/competition-induced increased body temperature, or when athletes are exposed to elevated ambient temperatures (e.g., during the summer period), is warranted.

4. Strengths, consistent barriers, and practical implications

4.1. Sleep architecture : an original exploration and persistent challenge

In the present study, the main challenge was to investigate the architecture of athletes' sleep within specific training contexts. Sleep architecture data in elite athletes are very limited (Vlahoyiannis et al., 2021). This is mainly due to the difficulties associated with implementing EEG-based analysis of sleep architecture in the field and the limited availability of validated

wearable tools to monitor sleep stages (de Zambotti et al., 2023). The use of portable polysomnography and reduced-montage dry-EEG wireless devices throughout the experimental studies helped to overcome this obstacle. This allowed us to explore the sleep-related physiological and architectural responses to sporting contexts, which are essential in determining sleep quality. Additionally, we strived to explore how training load and a bedding strategy may affect the sleep architecture of elite athletes in greater detail than the traditional approach of sleep stage distribution, by computing sleep stage transition probabilities through Markov chain modelling. Such analyses can inform on the continuity and discontinuity of sleep architecture, which could determine sleep quality by facilitating the quantity and stability of each sleep stage. Additional studies are needed to better understand the potential impact of enhancing the continuity of sleep architecture on athletic and/or cognitive performance.

Nevertheless, we cannot ignore the presence of consistent barriers in investigating the architecture of elite athletes. Single-night polysomnography recordings were performed in studies **2-B** and **3** due to the expensive, time-consuming, and complex nature of this method. This remains unsuitable for monitoring day-to-day sleep architecture in athletes and may not accurately reflect current sleep, especially in elite athletes who can experience high intra-individual variability in sleep (Nedelec et al., 2018). In recent years, a large number of wearable sleep tools have been developed to analyse sleep architecture in athletes, while being less expensive, more user-friendly, and requiring no specific expertise compared with polysomnography (Driller et al., 2023). However, it is important to note that, to date, many of these sleep monitors present insufficient scientific evidence for sleep staging mainly because they do not record brain cortical activity (Depner et al., 2020; Miller et al., 2022). In **study 1**, we used a portable EEG headband to easily assess the sleep architecture of athletes. Nevertheless, this device could cause discomfort during sleep for certain athletes, which is likely a major cause of low compliance. The high proportion of missing or unused recordings due to insufficient quality (~61.5%) illustrates the challenges of measuring sleep architecture in specific training contexts, despite the use of a user-friendly method. We also intended to use the EEG headband in **study 2** but some participants were reluctant to comply, given the longer duration of the training program, i.e., 6 weeks. It is therefore necessary to persist in our efforts to advance and rigorously validate technology for exploring sleep architecture through EEG analysis in even more practical ways. Advancements in automatic and deep learning models for sleep staging of EEG signals (Phan and Mikkelsen, 2022; Soleimani et al., 2024) could remove this consistent barrier.

4.2. Sleep research: implications both for sports performance and industry

This thesis has been designed to address research questions relevant to both sports performance and industry issues, with the ultimate purpose of disseminating the acquired knowledge for practical applications.

The present findings have provided evidence-based information to support elite athletes in preparing for international competitions. Athletes in the experimental studies received support in the form of individual feedbacks and sleep reports (see examples of reports generated from PSG, actigraphy, and EEG headband in **Appendices 3–5**). An explanation of the findings was first presented to the athletes in person, followed by propositions of individual practical recommendations and interventions to apply. In addition, the results of **study 1** were presented collectively to the rugby union players and the staff members, including the coaches, medical doctors, physiotherapists and managers in charge of organising the training camp. This feedback was designed to raise the importance of sleep for athletes and discussing on practical sleep-promoting strategies during training camps and competition periods. We focused on the observed detrimental effect of earlier morning appointment time on their prior sleep duration. We emphasized the importance of sleep hygiene, regularity, and pre-bedtime routines to mitigate the reduction in sleep duration when waking up earlier. The sleep benefits of single-session and multisession sleep hygiene education in athletes from various sports is well-recognized (Vitale et al., 2019b, 2023b; Gwyther et al., 2022; Pasquier et al., 2023). Unfortunately, we did not have the opportunity to measure the players' sleep after the session to determine whether it had a positive effect. Furthermore, some staff members have expressed interest in receiving support with their sleep. Some of them experienced sleep complaints, shorter and fragmented sleep. Therefore, it would be relevant not to forget the sleep of the staff members and to consider proposing strategies on maintaining their health, well-being, and decision-making during psychologically and physically stressful situations, such as training camps and competition periods (Calleja-González et al., 2021). In addition, the findings of the present thesis were disseminated to numerous coaches, elite athletes, staff members (e.g., medical doctor, physiotherapists), and sporting organizations (e.g., regional training centres). This aligns with the aim of disseminating scientific knowledge to enhance the skills of those contributing to the performance, well-being, and health of athletes.

For the industry partner, Bultex, this work aligns with the aim of developing bedding systems based on scientific evidence. The study of sleep among elite athletes exposed to various stressors serves as a valuable case study for the research and development of innovative

bedding systems. This research informs the industry partner about the effectiveness of a bedding strategy that contributes to the broader effort of promoting sleep, well-being, and overall health in the general population. Moreover, it has identified the limitations and aspects that require further development in this bedding strategy, while proposing scientific perspectives for the design of future bedding systems and the contexts in which their use could be beneficial (see Research perspectives). Additionally, this work provides the company with scientific knowledge about sleep, in a view to respond accurately to the needs of sleepers based on current scientific knowledge.

Overall, this PhD thesis, arising from a scientific collaboration between an industrial partner, a sports organization, and a laboratory, has contributed to addressing scientific, industrial, and sporting issues.

Research perspectives

The first perspective emerging from this work relates to restlessness during sleep when athletes are exposed to excessive training loads. We reported a positive influence of accumulating training load during a typical training period on propensity, depth, and stability of athletes' sleep architecture. It would be interesting to explore the etiological (physiological, psychological, sociological) mechanisms involved in increased restlessness during sleep in response to excessive load and whether they truly impact the restorative functions of sleep. This may help to implement specific strategies to mitigate the restlessness during sleep. To accomplish this, traditional sleep staging may prove insufficient. Novel approaches have emerged to provide further detail on the internal structure of sleep and sleep quality, particularly concerning mesoscopic features extracted from EEG spectra and sleep microstructure (e.g., slow waves and sleep spindles). Recent progress in artificial intelligence and machine learning make such analysis increasingly efficient and relevant (Andrillon et al., 2020).

Additionally, it would be relevant to examine the sleep response to a wider range of training loads and consider how unusual it is, i.e., the rate of change in training load. Moreover, whether increased training load may impact sleep while accounting for individual sleep patterns (e.g., poorer vs good sleepers) would provide guidance to support specific athletes during overload periods.

Future research should also consider various external physical demands and internal training load measures in investigating the relationship between training load and sleep. Identifying training demands that may lead to sleep disturbances or, conversely, may promote sleep, as observed in the current thesis with running demand, can help coaches manage training loads during specific periods that require highly restorative sleep (e.g., overload, pre-competitive periods). For instance, sport-related concussions are a current health issue among athletes with sleep disorders being a common symptom (Jaffee et al., 2015; Leddy et al., 2023). Evidence also showed that even non-concussion collisions could deteriorate sleep architecture (Aloulou et al., 2020b). Further studies are needed, including the evaluation of sleep architecture combined with measurements of the frequency, intensity, and linear/rotational head kinematics of collision-based actions during training and match, such as using instrumented mouthguards.

We also propose scientific perspectives in investigating the effectiveness of a high-heat-capacity bedding system. We suggest further exploration of the design of this bedding strategy

to enhance heat loss during sleep, particularly through the use of lower thermal resistance materials. We recommend to compare the thermal resistance of different bedding component technologies, such as gel sheet, circulated air or water, low thermal resistance foam. Considering the increasing and intensified exposure to high temperatures due to climate change (Patz et al., 2014; Watts et al., 2018), this could be a major perspective.

We also encourage future research considering an individual approach, particularly based on sleep patterns, heat loss abilities during sleep, anthropometric characteristics, and preferred bedding and sleeping positions. We also recommend investigating whether increasing the surface area of the body in contact with a bedding strategy that facilitates heat loss results in greater benefits on sleep (Lan et al., 2018). In this regard, investigating the relevance of both using a high-heat-capacity pillow and mattress is warranted.

Future research should also investigate the relevance of this strategy in contexts that interfere with the sleep-related thermoregulatory processes (e.g., night-time training/competition) and/or combined with post-exercise recovery strategies performed before bedtime, such as warm showers or baths which promote heat loss during sleep. Identifying the criteria that would make this strategy beneficial for athletes may provide more precise guidance for prescribing it in the context of monitoring and supporting elite athletes' sleep.

Practical recommendations

The present work provides practical recommendations to coaches, athletes and sporting organisations. We support the evidence indicating that some elite athletes may experience sleep complaints and inadequate sleep quantity and/or quality. This highlights the importance of conducting rigorous objective and subjective assessments of sleep at different phases of the season (e.g., training camp, overload training phase, summer) to identify vulnerable athletes and propose appropriate support strategies. In this respect, it could be relevant to provide single- and multi-session sleep education programs that have demonstrated efficacy in improving the sleep quantity and quality of elite athletes (Gwyther et al., 2022; Pasquier et al., 2023; Vitale et al., 2023b). This could be achieved by disseminating sport-specific sleep information, such as individual needs, performance impacts, adjustments to training schedules and load, managing life obligations around training, optimizing sleep environment, and encouraging regular sleep and pre-bedtime routines.

Our findings support the adaptive nature of athletes' sleep architecture when exposed to elevated training loads. However, we highlight that the athletes may experience increased restlessness during sleep when the imposed training load becomes excessive (overload), likely due to physiological changes, higher mental strain, coupled with alterations in training organization and life around training. Sleep disorders are notably observed alongside extreme fatigue states, such as overreaching or overtraining (Meeusen et al., 2013). We encourage coaches to devote special attention to sleep when athletes are exposed to overload training periods, considering the level of fatigue and training response. Promoting sleep hygiene strategies (e.g., optimizing sleep environment, encouraging sleep extension and nap opportunities, implementing pre-bedtime routines) during an overload is warranted to promote sleep and, in turn, maintaining the stress-recovery balance, a prerequisite for long-term training adaptations and performance improvement. Additionally, when designing a congested training program (e.g., training camp), we suggest considering the organization of training and life around training, prioritizing regular sleep schedule, and promoting sleep quantity and quality.

Our findings did not demonstrate sleep benefits of a high-heat-capacity bedding system when used chronically during exposure to varied training loads and a summer training period. Nevertheless, we reported potential sleep benefits for athletes experiencing more restless sleep. It would be useful to consider this sleep strategy on an individual basis.

Conclusion

Sleep is critical for health, well-being, recovery and performance of athletes. The present experimental work offers the opportunity to improve our understanding of the extent to which training load affects athletes' sleep and the relevance of a high-heat-capacity mattress topper designed to promote sleep among athletes in various experimental setups. The implementation of EEG-based analysis of sleep architecture within typical training contexts provides crucial information on sleep architecture, and therefore sleep quality. In our humble opinion, this represents the major contribution of the present work.

On the one hand, our findings underline the adaptive nature of sleep architecture in response to training load and athletes' need for recovery, which promotes the propensity, depth, and stability of athletes' sleep architecture. On the other hand, they emphasize the likelihood of increased restlessness during sleep when exposed to excessive training load, potentially due to physiological changes, increased mental strain, coupled with alterations in training organization and life around training. Particular attention to sleep and precise management of training load should be devoted, especially during overload periods, to ensure optimal recovery, long-term training adaptations and performance improvements. Additionally, we demonstrate that the sleep quantity of athletes is putatively affected by morning schedules during training camps, potentially in a larger extent than training load itself. This supports the importance of organizing training and life around training to prioritize regular sleep schedules and favouring sleep quantity and quality.

The chronic use of a high-heat-capacity mattress topper by athletes exposed to varied training loads or during a summer training period did not provide sleep benefits. This is likely due to insufficient body heat loss during sleep. However, this work provides insights into potential sleep benefits in athletes experiencing more restless sleep. This encourages further research into the effectiveness of such a bedding strategy according to an individualized approach.

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Appendices

1. Study 2-A: Associated publication
2. Study 2-B: Associated publication
3. Example of an individual sleep report generated from polysomnography
4. Example of an individual sleep report generated from actigraphy
5. Example of an individual sleep report generated from the EEG headband

Higher training loads affect sleep in endurance runners: Can a high-heat-capacity mattress topper mitigate negative effects?

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ABSTRACT

This study investigates the impact of a training program on sleep among endurance runners and the benefits of chronically using a high-heat-capacity mattress topper (HMT). Twenty-one trained male athletes performed a 2-week usual training regimen, sleeping on a Low-heat-capacity Mattress Topper (LMT), followed by 2-week overload and taper periods. From overload, participants were assigned into two groups based on the mattress topper used: HMT ($n = 11$) or LMT ($n = 10$). Irrespective of the group, overload increased general stress and *stress-reaction symptoms* evaluated by questionnaires, with no decline in performance on a graded-exercise treadmill test, the majority of participant being “non-overreached” ($n = 14$). From a daily perspective, each additional 100 A.U. in training load, assessed using the session rating of perceived exertion, was associated with an impairment in subsequent sleep efficiency ($\beta = -0.2\%$; $p < 0.01$), wake after sleep onset ($\beta = +0.4$ min; $p < 0.05$) and sleep onset latency ($\beta = +0.5$ min; $p < 0.05$), which was unaffected by HMT use. Practitioners should be aware of sleep needs, especially during excessive training loads, whereas implementing individualised sleep strategies. Further studies should be conducted on potential benefits of HMT among athletes in various sleep conditions.

ARTICLE HISTORY

Received 21 June 2023
Accepted 13 November 2023

KEYWORDS

Recovery; athletes; training program; Performance; innovative bedding system; muscle soreness

Introduction

Athletes seek to follow a well-controlled training program to achieve a peak performance during the most important competitions of the season. The training periodization aims to accurately manage phases of increase (overload period) and decrease (taper period) in training load (Mujika et al., 2018) which can cause variations in the athlete’s training state between negative (“fatigue”) and positive (“fitness”) responses (Banister et al., 1975). The physiological, psychological, and performance benefits of manipulating training load throughout the competition preparation are well established for endurance athletes (Bosquet et al., 2007; Mujika et al., 2018). However, an imbalance between training load and recovery leads to training maladaptation and increases the risk of excessive fatigue, overtraining, illness, injury and underperformance (Meeusen et al., 2013). Therefore, adequate management of the recovery process is necessary to minimize training-induced fatigue and to promote training adaptations (Mujika et al., 2018).

Sleep is recognized by the scientific community as providing a number of important metabolic, hormonal and cognitive restorative functions (Halson & Juliff, 2017). Several studies have demonstrated the beneficial effects of acute and chronic physical activities on sleep quantity and quality in healthy individuals (Kredlow et al., 2015). On the one hand, high training loads have been identified as a sport-specific factor that may impair sleep among athletes (Roberts et al., 2019; Walsh et al., 2020). A systematic review with meta-analysis proposed

that an increase of more than 25% in training load, compared with a usual training period, is likely to reduce sleep duration and/or sleep efficiency (Roberts et al., 2019). Previous studies have suggested that muscle fatigue, muscle soreness and increased sympathetic activity induced by high training loads may all contribute to sleep disturbance (Hauswirth et al., 2014; Sargent et al., 2016; Schaal et al., 2015), which may reflect maladaptation to training (Roberts et al., 2019). However, these studies included subelite to elite endurance athletes in non-weight-bearing sports (i.e., triathlon, swimming, cycling). This precludes drawing clear conclusions about prolonged running exercises inducing larger extent of muscle damage resulting from such stretch-shortening cycle activities (Nieman et al., 2014). Exercise-induced muscle damage and subsequent muscle soreness and inflammation are likely to affect sleep (Finan et al., 2013), which suggests a more profound effect of muscle damage induced by high training loads on sleep in endurance runners. On the other hand, the purpose of tapering following high training loads is to reduce physiological and psychological stress and fatigue (Mujika et al., 2018), making sleep a central component of the recovery and adaptations sought during this period. Consequently, there is an interest in optimizing sleep condition among endurance runners exposed to high training loads and during taper periods to enhance training adaptations and performance.

Sleep regulation and the circadian rhythm of core body temperature are closely related (Krauchi & Deboer, 2010).

Sleep initiation is most likely to occur when the decline in core body temperature is maximal, as a result of skin warming and body heat loss at the periphery (Krauchi & Deboer, 2010). Sleeping on a high-heat-capacity gel sheet on the top of a foam mattress, which enhances the slow and continuous removal of body heat during sleep by conductive heat transfer, has shown to increase slow-wave sleep and improve subjective sleep comfort from an acute perspective (Herberger et al., 2020; Kräuchi et al., 2018). Aloulou et al. (2020) observed a reduction in wake after sleep onset and a higher rapid-eye-movement sleep proportion when sleeping on a similar gel sheet after an elite rugby union match. Nonetheless, the putative relevance of this cooling strategy for endurance runners during an overload and taper periods is currently unknown.

The aim of the present study was to investigate the impact of an overload training and taper period on sleep among well-trained endurance runners; as well as the effect of a high-heat-capacity mattress topper (HMT) on sleep during these two training periods. We hypothesized that an overload training period negatively affects sleep, and that sleeping on HMT may mitigate this negative effect.

Methods

Participants

A call for participation was sent to various regional track and field clubs. Forty-four runners responded to the call. The inclusion criteria were to have been competing for ≥ 4 years and training \geq five times a week with a required performance level < 38 min per 10 km to include runners from “Trained/Developmental” to “Highly Trained/National” levels (McKay et al., 2022). Six participants did not meet these requirements and were therefore excluded. The following exclusion criteria were then checked before the start of the study: (a) any contraindication to high-intensity training based on an examination by a medical doctor including a detailed medical history and an electrocardiogram at rest ($n = 2$ exclusions); (b) actigraphy or polysomnography-confirmed sleep disorders, such as sleep apnea (apnea-hypopnea index > 10), periodic limb movement syndrome, hypersomnia, insomnia, circadian sleep rhythm disorders, or narcolepsy ($n = 2$ exclusions); (c) sleep complaints (i.e., global scores ≥ 5 on the Pittsburgh Sleep Quality Index; Buysse et al., 1989), extreme morning or evening chronotype (i.e., scores < 31 or > 69 ; Horne & Ostberg, 1976), and excessive daytime sleepiness (i.e., scores ≥ 10 on the Epworth Sleepiness Scale; Johns, 1991) assessed by questionnaires ($n = 4$ exclusions) (d) shift workers ($n = 4$ exclusions); (e) daily consumption of alcoholic beverages, more than 300 mg of caffeine per day and the use of prescribed medications. A total of 26 well-trained male endurance runners [mean \pm SD; age = 27.2 ± 4.8 years; height = 175.4 ± 5.6 cm; body mass = 67.6 ± 7.0 kg] completed the inclusion and exclusion criteria and participated in this study. All provided their written informed consent to participate after comprehensive verbal and written explanations of the protocol. The study was conducted in accordance with the Declaration of Helsinki (1964: revised in 2001) and was approved by the local ethic committee (approval reference: 21.00586.000020, ID-RCB: 2021-A01607-34).

Experimental design

The study was conducted from September 2021 to March 2022. An overview of the study design is presented in Figure 1. Training and sleep of each participant were monitored continuously for a period of 6 weeks divided into three 2-week training periods, a training protocol adapted from Hausswirth et al. (2014). In the first 2 weeks (baseline), participants followed a training program based on their usual training regimen. The following 2 weeks (overload), participants were asked to increase the duration of each training session by 40% in an attempt to overreach them (Hausswirth et al., 2014). In the last 2 weeks of the protocol (taper), the participants reduced the duration of each training session of the usual training regimen by 50% while maintaining the training intensity in an attempt to maximise post-taper performance gains (Bosquet et al., 2007). They completed the training sessions in their own training facility and the same researcher daily checked the correct sequence of training sessions by SMS and/or phone calls. Before the 6-week training follow-up, each participant came to the laboratory to get familiar with the testing battery including: (a) surveys, and (b) a graded-exercise test. At the end of baseline (Test 1), overload (Test 2) and taper (Test 3), participants performed the testing battery. In order to limit dietary influences on test results, participants were asked to follow standardised nutritional guidelines (quantity and content for food and drink) 48 h prior to and during the day of the testing battery, as well as abstaining from physical activity 24 h prior to the testing battery. Compliance with these instructions was checked upon arrival for each testing battery.

Participants slept in the same bedroom in their home throughout the protocol. During baseline, all participants slept on a Low-heat-capacity Mattress Topper (LMT). At the start of overload (the day after Test 1) and according to a matched group single-blinded experimental design, participants were allocated into two groups based on the mattress topper used until the end of the protocol: High-heat-capacity Mattress Topper (HMT, $n = 13$, experimental group) and LMT ($n = 13$, control group).

Mattress topper properties

The LMT was constituted of a 100% foam (density of 40 kg.m^{-3} ; Bultex, France), whereas the HMT additionally included a high-heat-capacity gel sheet (density of 1000 kg.m^{-3} ; dimensions of $80 \times 120 \times 2.5$ cm) on the top to slowly remove body heat during sleep by conductive transfer (Aloulou et al., 2020; Herberger et al., 2020; Kräuchi et al., 2018). The difference in thermal behaviour between HMT and LMT is related to thermal resistance (HMT: $1.18 \text{ m}^2.\text{K.W}^{-1}$, LMT: $1.63 \text{ m}^2.\text{K.W}^{-1}$ at 28°C). Both mattress topper sized $90 \times 200 \times 6$ cm, and were covered with a ticking (density of 540 g.m^2 , composed of 99% of polyester and 1% of elastane; Figure 2). Participants kept the same bed cover, pillow, sleepwear and bedroom ambient temperature throughout the study.

Fatigue markers following training periods

Declarative assessments

Before the graded-exercise test, participants were asked to complete the short French questionnaire version of the

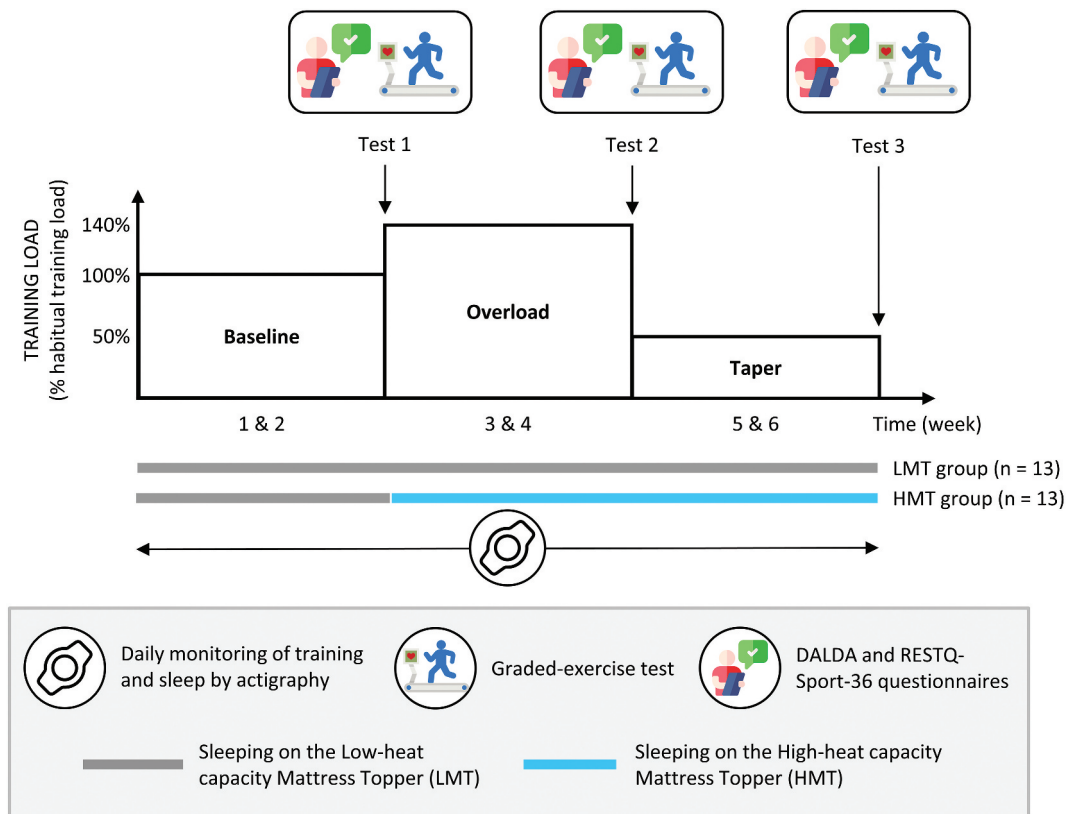


Figure 1. Overview of the experimental design.

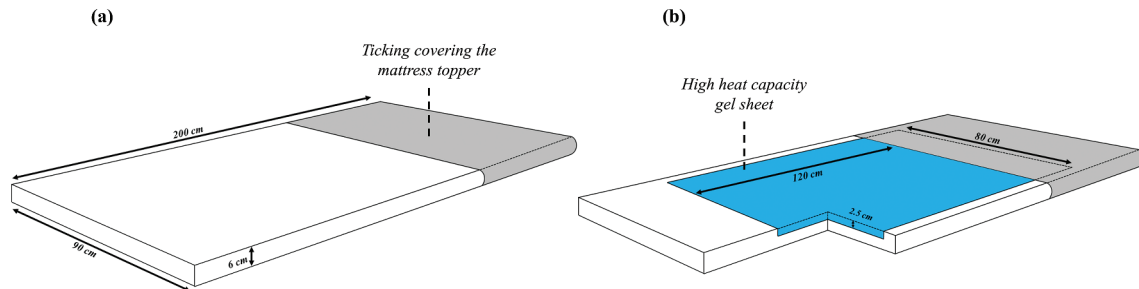


Figure 2. Figure illustrating the properties of mattress topper type: (a) LMT and (b) HMT.

Recovery-Stress Questionnaire for Athletes (RESTQ-Sport-36; Kellmann & Kallus, 2016), and the Daily Analysis of Life Demands for Athletes (DALDA; Rushall, 1990) to assess their recovery-stress levels and stress-reaction symptoms.

Performance testing

A graded-exercise test was performed until exhaustion to determine the performance and peak oxygen uptake ($\dot{V}O_{2peak}$) on a treadmill (Saturn 300/100 r, h/p/cosmos Sports & Medical gmbh, Germany). The exercise started with 5 min of warm-up at $10 \text{ km}\cdot\text{h}^{-1}$ followed by an increase of $1 \text{ km}\cdot\text{h}^{-1}$ every 2 min until volitional exhaustion with a slope fixed at +1% (Hauswirth et al., 2011; Jones & Doust, 1996). Oxygen uptake, carbon dioxide output, and ventilation were continuously monitored using a breath-by-breath analyser (Quark CPET, Cosmed, Rome, Italy). Heart rate (HR) was continuously recorded with a heart rate

monitor (HRM-Dual, Garmin, Olathe, KA, USA). $\dot{V}O_{2peak}$ was estimated by the highest 20-s average value. Performance was calculated as follows: $\text{Performance} = S_{\text{compl}} + 25 \times (t/120)$ where S_{compl} is the speed of the last successful 2-min step and t is the number of seconds in the last step.

Assessment of individual training responses

The participants who decreased performance in a larger extent than the smallest worthwhile change (SWC) at Test 2 were diagnosed as “overreached” (Meeusen et al., 2013). The SWC was calculated as 0.3 of the individual coefficient of variation between the familiarisation and Test 1 (Hopkins et al., 2009), during which training load was not manipulated (baseline). The participants who maintained or increased performance at Test 2 were considered “non-overreached”.

Daily-monitored measures

Training load, muscle soreness, general fatigue and stress

The session rating of perceived exertion (sRPE) was recorded within 30 min from the end of each training session using the modified CR-10 scale (Foster et al., 2001). The daily training load (A.U.) was calculated by multiplying sRPE and the duration of each training session within a day. The weekly training load (A.U.) was determined as the sum of the daily training loads for each week. Each morning, participants were asked to rate their muscle soreness, general fatigue and stress using the Hooper 1–7 scale (Hooper & Mackinnon, 1995).

Sleep analysis

Actigraphy data were collected throughout the study using a bracelet worn on the nondominant wrist (MotionWatch 8; CamNtech Ltd, Cambridge, UK). The recorded data were analysed using MotionWare Software (version 1.3.17; CamNtech Ltd, Cambridge, UK) to identify whether the participant was awake or asleep for each 60-s period. Actigraphy is a low-cost and non-invasive method for longitudinal monitoring sleep-wake patterns that has been shown to generate the smallest mean biases from polysomnography for total sleep time, sleep efficiency and wake after sleep onset in endurance athletes when a high sleep-wake sensitivity threshold (≥ 80 activity counts are scored as wake) is applied (Sargent et al., 2016). A high sensitivity sleep-wake threshold was accordingly applied to the present population of endurance athletes. Actigraphic sleep variables were calculated daily as follow: bedtime (hours:minutes), the self-reported clock time at which the participant went to bed to attempt to sleep; get-up time (hours:minutes), the self-reported clock time at which the participant got out of bed and stopped attempting to sleep; time in bed (min), the time between bedtime and get-up time; total sleep time (min), the time spent asleep, determined from sleep onset to sleep offset, minus any waking time; wake after sleep onset (min), the amount of time spent awake between sleep onset and sleep offset; sleep efficiency (%), the total sleep time

expressed as a percentage of time in bed; and sleep onset latency (min), the time between bedtime and sleep onset.

The Spiegel Sleep Inventory (SSI) was completed each morning throughout the study within 30 min after waking to assess sleep perception. The SSI is a self-administered questionnaire composed of six questions scored from 1 to 5 about sleep initiation, the quality and duration of sleep, nocturnal awakenings, dreams and feeling refreshed in the morning. The overall score is the sum of the six items. Few data are available on the psychometric validity of the SSI among athletes, but it is a very simple and easy-to-use scale that is often used to assess the presence of insomnia (Léger et al., 2006).

Statistical analysis

We used a non-inferiority sample size calculation to estimate the required sample size to detect potential differences in sleep efficiency between baseline and overload. Based on previous research from our lab (Hauswirth et al., 2014), it was necessary to include 10 participants to achieve significance and power criteria ($1 - \beta$) of 0.05 and 90%, respectively (G*Power program, version 3.1.9.7). Considering the longitudinal design and the fact that participants were allocated into two distinct groups, we chose to increase the number of participants to 13 in each group to account for potential drop out. All statistical procedures were performed using R software (version 4.2.0). The normality of the data distribution and the homogeneity of variance were assessed using the Shapiro-Wilk and Levene tests, respectively.

Three linear mixed-effects models ("lmer" function of the "lme4" R package) were used in accordance with the dependent-tested variables and the research question (Table 1). Linear mixed-effects models extend traditional linear models by combining fixed and random effects to consider the non-independence of observations at the level of individuals. For the second linear mixed-effects model, training load was specified as a random slope by adding it to the participant random effect to test the hypothesis that the strength of the effect

Table 1. Linear mixed-effects models' details used for each dependent variable according to the research question.

Objective	Dependent variables	Fixed effect(s)	Random effect	Formula
To compare fatigue markers following training periods and weekly change in training session details for each group.	<ul style="list-style-type: none"> Fatigue markers: RESTQ-Sport-36 and DALDA items, VO_{2peak}, HR_{peak} and MAS Training session details: weekly training load and training volume, number of training sessions, mean sRPE per training session 	Group, time, and group \times time interaction	The participant	$var \sim time + group + time \times group + (1 participant)$
To evaluate the weekly change in daily-monitored measures for each group.	<ul style="list-style-type: none"> Sleep variables: bedtime, get-up time, time in bed, total sleep time, wake after sleep onset, sleep efficiency, sleep onset latency, SSI score Muscle soreness 	Group, time, and group \times time interaction	The participant and the slope (daily training load)	$var \sim time + group + time \times group + (daily\ training\ load participant)$
To assess the relationship between daily training load, mattress topper type and subsequent sleep.	<ul style="list-style-type: none"> Sleep variables: time in bed, total sleep time, wake after sleep onset, sleep efficiency, sleep onset latency, SSI score 	Daily training load, mattress topper type (categorical predictor), and daily training load \times mattress topper type interaction	The participant	$var \sim daily\ training\ load + mattress\ topper\ type + daily\ training\ load \times mattress\ topper\ type\ interaction + (1 participant)$

Abbreviations: RESTQ-Sport-36, Recovery-Stress Questionnaire for Athletes; DALDA, Daily Analysis of Life Demands for Athletes; MAS, maximal aerobic speed; VO_{2peak} , peak oxygen uptake; HR_{peak} , peak heart rate; sRPE, rating of perceived exertion of the session, SSI, Spiegel Sleep Inventory. Linear mixed-effects formulas were fitted with the dependent variable (var) on the left of a "~" operator and the fixed effect(s) separated by "+" operators on the right. Interaction between fixed effects is indicated by " \times ". Random effect is distinguished by "|".

(slope) of training period on fatigue markers, sleep variables and muscle soreness varies depending on the training load achieved by each participant. For the first and second linear mixed-effects models, when a significant effect of a fixed-effect (i.e., group, time and/or group \times time interaction) was reported, multiple post-hoc Student t-test pairwise comparisons were conducted using least square means contrasts with the Bonferroni correction for alpha adjustments ("emmeans" function of the "emmeans" package). The effect sizes were calculated to interpret the magnitude of the difference in means values between the fixed-effects, and were interpreted using the following criteria: $< 0.2 = \text{trivial}$, $0.2\text{--}0.6 = \text{small}$, $0.6\text{--}1.2 = \text{moderate}$, $1.2\text{--}2.0 = \text{large}$, and $> 2.0 = \text{very large}$ (Hopkins et al., 2009).

To determine whether the association between daily training load and sleep variables was mediated by muscle soreness, general fatigue or stress, separate mediation analyses were performed using the PROCESS function (Model 4, version 4.2; Hayes, 2022). The models selected were those in which the mediator was associated with both daily training load (univariate regression) and sleep variable (by fixing the effect of daily training load using multiple regression). A 5,000 bootstrapping procedure was performed to estimate the 95% bias-corrected nonparametric confidence intervals (CIs). When a 95% CI did not include zero, it indicated that the indirect effect was statistically significant. If both the indirect and direct effect were significant, then the mediating effect was considered partial, otherwise it was full if only indirect effect was significant.

Results are expressed as the mean \pm standard deviation (SD), except for the linear mixed-effects models and mediation models for which the results are presented as the estimate (β) \pm standard error (SE) and the 95% CI. For each linear mixed-effects model, marginal R^2 and conditional R^2 are provided as measures of the proportion of variance explained by the fixed effects(s) alone and both the fixed and random effects, respectively. The level of significance was set at $p < 0.05$.

Results

Five participants dropped out the protocol because of injury ($n = 2$), illness ($n = 1$), and for personal/professional reasons ($n = 2$),

and were excluded from further analysis. Participants were classified at level 2 "Trained/Developmental" ($n = 7$) and at level 3 "Highly Trained/National" ($n = 14$) according to the guidelines proposed by McKay et al. (2022). No significant differences ($p > 0.05$) in participants characteristics were reported between groups. A total of 777 actigraphy-based night sleep data (88.1%), 859 daily training load data (97.4%), and 772 subjective ratings data (87.5%) out of 882 were recorded. A main effect of time was found ($p < 0.001$) for weekly training load, number of training sessions, training volume and sRPE. No main effect of group and group \times time interaction was reported ($p > 0.05$). For all these variables, post-hoc analyses between weeks independently from the group are shown in Table 2.

Impact of training periods on fatigue markers and sleep

Declarative assessments

A main effect of time was found for the RESTQ-Sport-36 and DALDA items related to *disturbed breaks* ($p < 0.05$), *emotional exhaustion* ($p < 0.05$), *injury* ($p < 0.05$), *sources of stress* ($p < 0.05$) and *stress-reaction symptoms* ($p < 0.01$; Supplementary Material 1). No main effect of group and group \times time interaction was observed on overall RESTQ-Sport-36 and DALDA items ($p > 0.05$). Independently from the group, the score for *disturbed breaks* item was higher at Test 2 than Test 1 ($p < 0.05$; $d = 0.84$) and Test 3 ($p < 0.05$; $d = 0.79$). An increase in score for *injury* ($p = 0.05$; $d = 0.77$) and *emotional exhaustion* ($p < 0.05$; $d = 0.83$) items, and greater number of "worse than normal" *stress-reaction symptoms* ($p < 0.01$; $d = 1.01$) were noted at Test 2 compared with Test 1. A *moderate* greater number of "worse than normal" *sources of stress* was observed ($p = 0.05$; $d = 0.78$) at Test 3 compared with Test 1.

Performance testing

There was no main effect of group, time and group \times time interaction ($p > 0.05$) for $\dot{V}O_{2\text{peak}}$, HR_{peak} and the ambient environment (mean \pm SD: $22.4 \pm 1.1^\circ\text{C}$ and $39.8 \pm 7.0\%$ relative humidity). A main effect of time ($p < 0.01$) was reported for performance, but no main effect of group and group \times time interaction ($p > 0.05$) was noted (Table 3). Irrespective

Table 2. Weekly training session details during baseline, overload and taper periods in the two experimental groups.

	Baseline		Overload		Taper	
	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6
Weekly training load (A.U.)***	1391.5 \pm 522.2	1495.2 \pm 843.2	2120.0 \pm 735.9 ^{a,b}	1937.4 \pm 806.4 ^{a,b}	976.6 \pm 566.8 ^b	938.1 \pm 699.6 ^{a,b}
LMT	1427.9 \pm 494.3	1317.8 \pm 378.8	2139.2 \pm 494.8	1697.3 \pm 835.1	1037.7 \pm 620.8	865.0 \pm 568.9
HMT	1358.4 \pm 568.4	1656.5 \pm 1110.2	2102.5 \pm 928.5	2155.6 \pm 750.2	921.1 \pm 537.3	1004.6 \pm 823.1
Weekly training volume (h)***	6.0 \pm 2.3	5.9 \pm 2.8	8.0 \pm 2.9 ^{a,b}	6.9 \pm 2.9	4.7 \pm 2.6	4.0 \pm 2.5 ^{a,b}
LMT	6.1 \pm 2.2	5.6 \pm 1.7	8.4 \pm 3.0	6.4 \pm 2.9	5.2 \pm 3.4	4.2 \pm 2.5
HMT	5.9 \pm 2.4	6.3 \pm 3.6	7.6 \pm 2.9	7.3 \pm 3.0	4.3 \pm 1.6	3.9 \pm 2.6
Number of training sessions***	5.3 \pm 1.6	5.0 \pm 1.6	6.1 \pm 1.8 ^b	5.2 \pm 1.7	4.9 \pm 1.5	4.1 \pm 1.6 ^a
LMT	5.2 \pm 1.6	4.9 \pm 0.9	6.2 \pm 1.5	4.6 \pm 1.3	4.9 \pm 1.4	4.3 \pm 1.2
HMT	5.4 \pm 1.7	5.2 \pm 2.0	6.0 \pm 2.0	5.7 \pm 1.9	4.8 \pm 1.6	4.0 \pm 1.9
Mean sRPE per training session***	2.6 \pm 0.9	2.7 \pm 0.9	3.3 \pm 1.0 ^{a,b}	2.9 \pm 1.0	2.1 \pm 0.7 ^b	2.0 \pm 0.8 ^{a,b}
LMT	2.6 \pm 0.8	2.8 \pm 0.6	3.4 \pm 1.0	2.8 \pm 1.2	2.0 \pm 0.7	2.1 \pm 0.8
HMT	2.5 \pm 0.9	2.6 \pm 1.1	3.2 \pm 0.9	3.1 \pm 0.9	2.1 \pm 0.7	2.0 \pm 0.8

Data are presented as mean \pm SD. Abbreviations: HMT, High-heat-capacity Mattress Topper group; LMT, Low-heat-capacity Mattress Topper group; A.U., arbitrary unit; sRPE, session Rating of Perceived Exertion. Significant main effect of week: *** $p < 0.001$. Independently from the group, ^asignificantly different from Week 1, and ^bsignificantly different from Week 2.

Table 3. Mean values \pm SD of performance testing after baseline, overload and taper periods for LMT and HMT groups.

	Test 1 (baseline)	Test 2 (overload)	Test 3 (taper)
Performance (km.h⁻¹) **	18.6 \pm 1.1	18.4 \pm 1.3	18.8 \pm 1.2 ^a
LMT	18.5 \pm 1.2	18.3 \pm 1.5	18.6 \pm 1.4
HMT	18.7 \pm 1.1	18.5 \pm 1.1	19.0 \pm 1.0
VO_{2peak} (mL.min⁻¹.kg⁻¹)	61.9 \pm 5.4	61.8 \pm 4.5	60.8 \pm 4.4
LMT	62.5 \pm 6.6	60.8 \pm 5.1	60.1 \pm 5.0
HMT	61.2 \pm 4.1	62.9 \pm 3.7	61.5 \pm 3.9
HR_{peak} (bpm)	186.1 \pm 9.7	186.2 \pm 8.2	186.9 \pm 9.0
LMT	188.6 \pm 7.7	188.9 \pm 6.9	189.6 \pm 8.1
HMT	183.5 \pm 11.1	183.2 \pm 8.8	184.6 \pm 9.5

Abbreviations: HMT, High-heat-capacity Mattress Topper group; LMT, Low-heat-capacity Mattress Topper group; VO_{2peak}, peak oxygen uptake; HR_{peak}, peak heart rate. Significant main effect of time (test):

** $p < 0.01$. Independently from the group, ^a significantly different from Test 2.

of the group, a non-significant *moderate* decrease at Test 2 (-0.18 km.h^{-1} ; $p = 0.15$; $d = -0.63$) and a *small* increase in performance at Test 3 ($+0.16 \text{ km.h}^{-1}$; $p = 0.32$; $d = 0.53$) were observed compared with Test 1 (mean \pm SD: $18.6 \pm 1.1 \text{ km.h}^{-1}$). A significant *moderate* increase ($+0.34 \text{ km.h}^{-1}$; $p < 0.01$; $d = 1.16$) in performance was observed at Test 3 compared with Test 2.

Individual training responses

Seven of the twenty-one participants decreased in performance of at least the SWC (0.41% of Test 1) at Test 2 ($17.6 \pm 1.4 \text{ km.h}^{-1}$) compared with Test 1 ($18.4 \pm 1.4 \text{ km.h}^{-1}$), and were therefore diagnosed as “overreached”. The fourteen other participants maintained or increased their performance at Test 2 ($18.8 \pm 1.0 \text{ km.h}^{-1}$) compared with Test 1 ($18.7 \pm 1.0 \text{ km.h}^{-1}$), followed by a supercompensation in performance at Test 3 ($19.1 \pm 1.1 \text{ km.h}^{-1}$), and were considered as “non-overreached”.

Daily-monitored sleep measures and muscle soreness

No main effect of group and group \times time interaction was observed on sleep variables and muscle soreness ($p > 0.05$). There was no main effect of time for bedtime, get-up time, time in bed, total sleep time, sleep efficiency and sleep onset latency ($p > 0.05$), but a main effect of time was found for wake after sleep onset ($p < 0.05$), SSI score ($p < 0.01$) and muscle soreness ($p < 0.001$). As shown in Figure 3, irrespective of the group, wake after sleep onset significantly increased during the first week of taper ($+4.1 \text{ min}$; $p < 0.05$; $d = 0.41$) compared with the second week of baseline (mean \pm SD: $23.8 \pm 9.6 \text{ min}$), while SSI score was higher during the first week of taper (mean \pm SD: $22.7 \pm 2.0 \text{ A.U.}$) compared with the first (-1.1 A.U. ; $p < 0.05$; $d = -0.38$) and second week (-1.5 A.U. ; $p < 0.01$; $d = -0.48$) of baseline. Perceived muscle soreness was higher during the second week of overload (mean \pm SD: $3.2 \pm 0.8 \text{ A.U.}$) compared with the first week (-0.6 A.U. ; $p < 0.001$; $d = -0.55$) and second week (-0.4 A.U. ; $p < 0.05$; $d = -0.40$) of baseline, and the first (-0.7 A.U. ; $p < 0.001$; $d = -0.61$) and second week (-0.6 A.U. ; $p < 0.01$; $d = -0.48$) of taper.

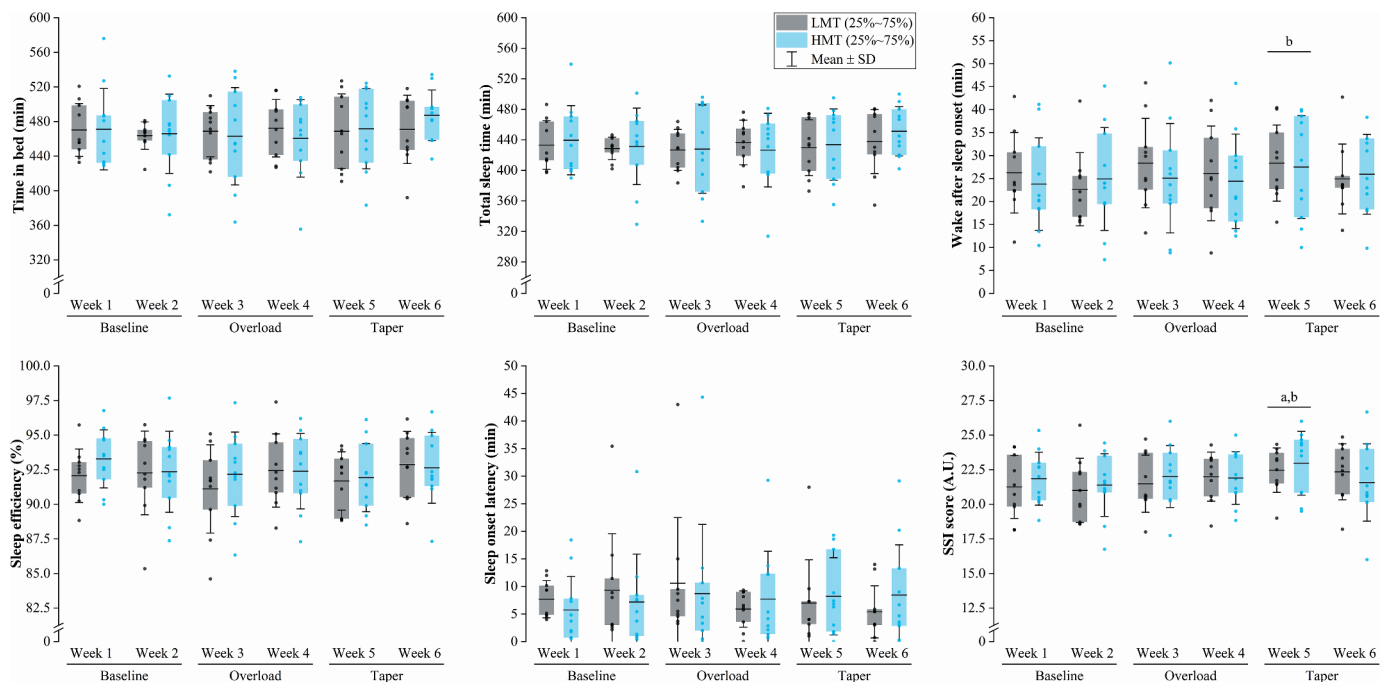


Figure 3. Weekly average of sleep variables during baseline, overload and taper periods for LMT and HMT groups.

Abbreviations: SSI, Spiegel sleep Inventory; A.U., arbitrary unit. Independently from the group, ^a significantly different from week 1 ($p < 0.05$), and ^b significantly different from week 2 ($p < 0.05$).

Relationships between daily training load, mattress topper type and subsequent sleep

Table 4 provides an overview of the relationships between daily training load and subsequent sleep variables adjusted for mattress topper type. For every additional 100 A.U. in training load, we found subsequent lower sleep efficiency ($\beta = -0.2\%$; 95% CI: -0.4 to -0.1% ; $p < 0.01$; Figure 4A), higher wake after sleep onset ($\beta = +0.4$ min; 95% CI: 0.1 to 0.8 min; $p < 0.05$; Figure 4B) and an increase in sleep onset latency ($\beta = +0.5$ min; 95% CI: 0.1 to 1.0 min; $p < 0.05$; Figure 4C). No significant differences between HMT and LMT, and no daily training load \times HMT interaction were observed ($p > 0.05$) in any sleep variables.

Mediation analysis of the relationship between daily training load and subsequent sleep

Univariate regressions showed that daily training load was not significantly associated with general fatigue and stress ($p > 0.05$) and thus were not included in further analyses. A significant and positive effect of prior daily training load on muscle soreness was observed ($p < 0.001$). After controlling for daily training load, multivariate regressions showed that muscle soreness was associated with wake after sleep onset ($p < 0.01$), but not significantly with the other sleep variables ($p > 0.05$). Figure 5 contains the results of the mediating effect of muscle soreness in the relationship between daily training load and wake after sleep onset. Total effect model including the mediator (muscle soreness) predicted significant variance in wake after sleep onset ($R^2 = 0.02$; $p < 0.01$). The results revealed a negative significant indirect effect via muscle soreness (for additional 100 A.U. in daily training load, $\beta = -0.2 \pm 0.1$ min; 95% CI: -0.3 to -0.0 min), with a positive significant direct effect after controlling muscle soreness effect (for additional 100 A.U. in daily training load, $\beta = 0.8 \pm 0.2$ min; 95% CI: 0.4 to 1.2 min; $p < 0.001$), i.e., muscle soreness partially mediates the relationship between daily training load and wake after sleep onset through a suppression effect.

Discussion

Three major findings emerged from this study: 1) no change in the weekly averaged sleep variables was observed throughout the overload and taper periods; 2) from a day-to-day approach, higher daily training load was associated with an impairment in subsequent sleep efficiency, wake after sleep onset and sleep onset latency; and 3) HMT did not mitigate the negative effect of higher training load on sleep.

Impact of training periods on sleep

In contrast to our hypothesis, the first important finding is that overload did not alter the weekly averaged sleep variables. A first explanation may be related to the training load achieved during the overload. Roberts et al. (2019) suggested that sleep disruption occurs when training load increases by more than 25%. Specifically, studies that reported sleep disruption during an overload period increased training volume and/or intensity between + 25% and + 70% for 2–3 weeks in artistic swimmers, triathletes and rugby players (Hauswirth et al., 2014; Schaal

Table 4. Linear mixed-effects model assessing the relationships between daily training load and subsequent sleep variables, and the effect of mattress topper type.

Fixed effects	Time in bed (min)		Total sleep time (min)		Sleep efficiency (%)		Wake after sleep onset (min)		Sleep onset latency (min)		SSI score (A.U.)	
	Est. \pm SE (95% CI)	p-value	Est. \pm SE (95% CI)	p-value	Est. \pm SE (95% CI)	p-value	Est. \pm SE (95% CI)	p-value	Est. \pm SE (95% CI)	p-value	Est. \pm SE (95% CI)	p-value
Intercept	470.1 \pm 7.6 (455.2–485.0)	<0.001	437.3 \pm 7.8 (422.0–452.5)	<0.001	93.0 \pm 0.5 (92.0–94.0)	<0.001	24.3 \pm 2.0 (20.4–28.1)	<0.001	5.9 \pm 1.5 (2.9–8.9)	<0.001	21.7 \pm 0.4 (21.0–22.5)	<0.001
LMT												
HMT	3.1 \pm 6.9 (-10.5–16.7)	0.66	-1.2 \pm 6.8 (-14.7–12.2)	0.86	-0.7 \pm 0.5 (-1.6–0.2)	0.12	1.9 \pm 1.3 (-0.7–4.4)	0.16	2.8 \pm 1.6 (-0.3–5.9)	0.07	0.6 \pm 0.4 (-0.1–1.3)	0.11
Daily training load (x100 A.U.)	-0.6 \pm 1.1 (-2.7–1.5)	0.57	-1.6 \pm 1.1 (-3.7–0.4)	0.12	-0.2 \pm 0.1 (-0.4 – -0.1)	0.001	0.4 \pm 0.2 (0.1–0.8)	0.03	0.5 \pm 0.2 (0.1–1.0)	0.03	0.0 \pm 0.1 (-0.1–0.1)	0.90
Daily training load (x100 A.U.) \times HMT interaction	-0.4 \pm 1.6 (-3.5–2.8)	0.82	0.2 \pm 1.6 (-2.9–3.4)	0.88	-0.1 \pm 0.1 (-0.1–0.3)	0.48	-0.1 \pm 0.3 (-0.7–0.5)	0.74	1.0 \pm 0.1 (-1.1–0.3)	0.25	-0.1 \pm 0.1 (-0.2–0.1)	0.50
Random Effects												
N	21		21		21		21		21		21	
Observations	777		777		777		777		777		772	
Marginal R ² /Conditional R ²	0.001/0.223		0.004/0.242		0.021/0.237		0.010/0.393		0.010/0.181		0.005/0.203	

Abbreviations: Est., estimate; SE, standard error; CI, confidence interval; A.U., arbitrary unit; HMT, high-heat-capacity mattress; LMT, low-heat-capacity mattress; SSI, Spiegel sleep inventory.

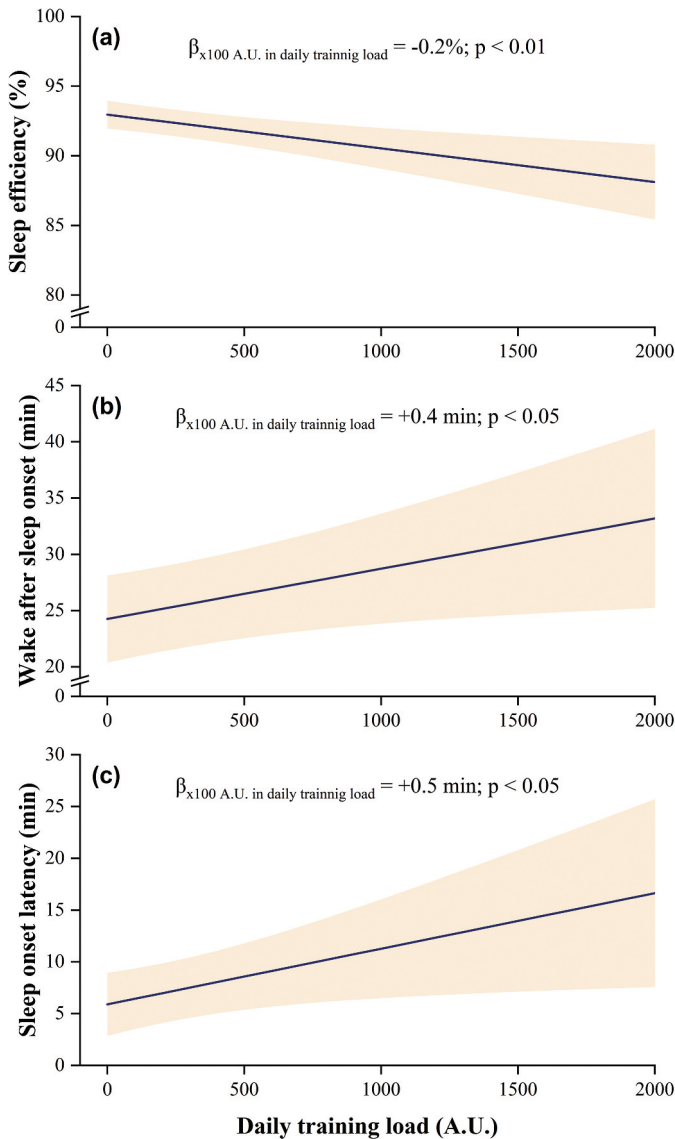


Figure 4. The relationship (and 95% confidence interval) between daily training load and subsequent (a) sleep efficiency, (b) wake after sleep onset, and (c) sleep onset latency.

et al., 2015; Thornton et al., 2017), and up to extreme increases (> +140%) in cyclists for 1–3 weeks (Killer et al., 2017; Lastella et al., 2015). Although the current average increase in weekly training load was ~+40% during overload compared with baseline, as required to participants, we observed important inter-individual differences (from +10% to +170%), which may have resulted in individual sleep responses. However, potential individual sleep responses cannot be observed with group means, which justifies the interest of future studies to determine the effect of high training loads at individual level. These inter-individual differences in increased training load may be explained by the different possibilities for each individual to train more than their usual training regimen. Some participants exceeded 10 h of training per week, including one accustomed to running 120 km per week, making it more difficult to increase training load by 40% compared with those training less. In addition, they had to adapt their daily planning to increase the training volume while dealing with personal constraints and work/study schedules. The combination of increased training load and personal/professional constraints may explain the higher scores observed following overload for the general stress items (i.e., *disturbed breaks* and *emotional exhaustion*) of the RESTQ-Sport-36, as well as the higher prevalence for *stress-reaction symptoms* measured by the DALDA. This finding supports the fact that overload effectively increased mental stress (Meeusen et al., 2013).

In contrast, overload did not result in significant physical stress as we did not observe a decrease in performance on the graded-exercise test following overload compared to baseline. The level of accumulated fatigue has been suggested to interact with sleep, with sleep disturbances frequently observed in overreached athletes (Walsh et al., 2020). In the present study, the majority of participants ($n = 14$) were “non-overreached”, which may explain the lack of sleep disruption observed during overload among the present population. These participants also demonstrated an improvement in performance following taper compared with baseline, reflecting a positive adaptation to training (Meeusen et al., 2013). This result supports the interest of overloading prior to tapering provided that the

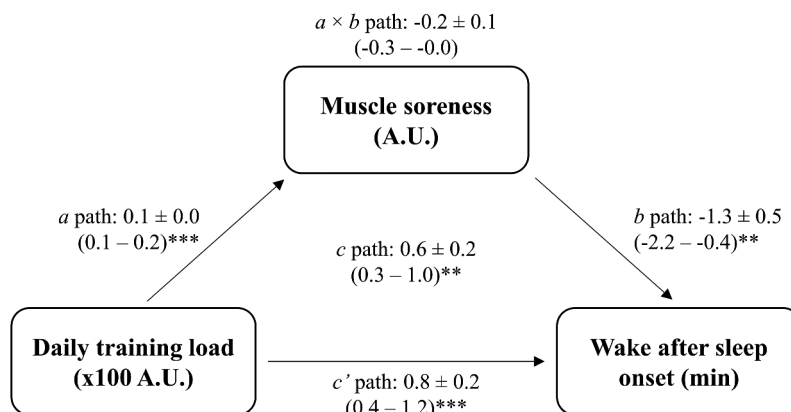


Figure 5. Mediating effect of muscle soreness in the relationship between daily training load and subsequent wake after sleep onset. a , b , c and c' are regression coefficient with c = total effect, c' = direct effect, and $a \times b$ = indirect effect. Data are presented as unstandardized coefficients \pm standard errors (and bootstrapped 95% CI). $a \times b$ indirect effect was considered significant when 95% CI did not include zero. NS, not significant ($p > 0.05$); ** $p < 0.01$; *** $p < 0.001$.

fatigue level is not too high (Aubry et al., 2014). In addition, the effect of accumulated training load over a training period, and consequently level of fatigue, may result in a progressive change in sleep, possibly with some delay and a possible peak effect. The quite modest increase (+4 min) in wake after sleep onset observed during the first week of taper compared to the second week of baseline may be explained by this potential delay effect of overload, or conversely, by a lack of training during tapering since a minimum of physical activity is recommended for adequate sleep quality (Kredlow et al., 2015). Future investigations should be conducted with an individualised approach to describe the time-course of the effect of an accumulated training load on sleep, and conversely its reduction especially during a taper period prior to a competition.

Relationships between daily training load and subsequent sleep

Although we did not observe alterations in sleep during the overload period considered as a whole, the second important finding was the negative relationship between training load achieved on a daily basis and subsequent sleep. For each additional 100 A.U. in training load, we observed a significant reduction in subsequent sleep efficiency (−0.2%) as well as an increase in wake after sleep onset (+0.4 min) and sleep onset latency (+0.5 min). These results are consistent with previous studies reporting a reduction in sleep duration and/or sleep efficiency in response to day-to-day variation in internal training load in various sports (Aloulou et al., 2021; Costa et al., 2021; Dumortier et al., 2018; Michele; Lastella et al., 2020). Intensified running endurance trainings are likely to induce muscle damage and/or pro-inflammatory response, which may result in delayed onset muscle soreness (Nieman et al., 2014). Consistently, the second week of overload resulted in an increase in muscle soreness compared to baseline. We observed that muscle soreness perceived in the morning partially mediated the relationship between daily training load and subsequent impairment in wake after sleep onset through a suppression effect. This indicates that higher training load induced greater muscle soreness which in turn was related to decreased wake after sleep onset. This result contradicts the theory that pain and sleep are reciprocally and negatively linked (Finan et al., 2013). However, this finding should be taken with caution since the current mediation analysis does not provide causal evidence. Moreover, the aetiology mechanisms of muscle soreness may differ from other types of pain (Hainline et al., 2017), which may affect sleep differently, hence the interest to explore the impact of muscle soreness on sleep in various sports.

It has been shown that increased daily mental strain, as a mental component of stress, is associated with a subsequent decreased in sleep duration and sleep efficiency among junior winter endurance athletes (Hrozanova et al., 2020). This may have been particularly apparent during overload in the present study since higher general stress scores and prevalence of *stress-reaction symptoms* were observed. Furthermore, it has been suggested that the predominance of the cardiac sympathetic activity and increased circulating cortisol following intensive endurance training may affect the

normal down-regulation of the body's stress systems necessary for adequate sleep (Roberts et al., 2019). This may have contributed to the observed increase in sleep onset latency and wake after sleep onset after higher training load. Additional studies should be conducted on the physiological, psychological and sociological factors which may be involved in the relationship between training load and sleep. Finally, some studies in rugby players have shown that the training components defining training load, may be positively or negatively associated with sleep (Aloulou et al., 2020; Roberts et al., 2019). As training load was quantified only by a subjective approach in the present study, further studies are recommended to better characterise training load and its effects on sleep, particularly in endurance runners.

Taken together, performance and well-being of athletes can be affected by excessive load, particularly as sleep is impaired by high training loads (Campbell et al., 2021). Thus, special attention should be allocated to sleep when training periodization involves excessive training loads, especially among elite athletes for whom the prevalence of lack of sleep (i.e., < 7 hours) and insufficient sleep requirement are frequently observed (Walsh et al., 2020). Nevertheless, the magnitude of the effect of high training load on subsequent sleep is quite modest and should be tempered. Indeed, even at excessive daily training load (e.g., +1000 A.U.), the subsequent impairment in sleep efficiency, sleep onset latency and wake after sleep onset predicted by the linear mixed-effects models in the present study remain within the limits of National Sleep Foundation's recommendations (Ohayon et al., 2017).

Effects of high-heat-capacity mattress topper on sleep

This study is the first to assess the sleep benefit of chronic HMT use. Our hypothesis was that sleeping on HMT may mitigate the negative effect of high training loads on sleep. This hypothesis was not confirmed since the observed negative relationship between daily training load and subsequent sleep was not significantly modified by the use of HMT. Additionally, no significant difference was observed in sleep variables between the HMT and LMT groups throughout the protocol. Previous studies have shown the interest of high-heat-capacity mattress to improve sleep quality, with various positive outcomes on sleep i.e., a higher slow-wave sleep proportion, a reduction in wake after sleep onset and/or an increase in rapid-eye movement sleep proportion (Aloulou et al., 2020; Herberger et al., 2020; Kräuchi et al., 2018). The discrepancies with the present study could be explained by the difference in the population studied and/or the experimental protocols, as sleep was monitored on a daily basis by actigraphy, in contrast to previous studies that assessed sleep on a single night by polysomnography. Future studies are required to assess the potential chronic effect of HMT on sleep architecture. Moreover, the properties of HMT differed from those of the other studies, as the tested high-heat-capacity gel sheet was on the top of either a mattress (Herberger et al., 2020; Kräuchi et al., 2018) or a mattress topper (Aloulou et al., 2020). The thickness of the high-heat-capacity gel sheet also differed since it was 2.5 cm in the present study and 2 cm (Herberger et al., 2020; Kräuchi

et al., 2018) or 4 cm (Aloulou et al., 2020) in the previous studies, which may have resulted in different thermal behaviour. Different high-heat-capacity gel sheet thicknesses and materials could be tested on the top of mattress vs. mattress topper in further studies to determine the best bedding system design to remove body heat and promote sleep. Moreover, the present study was conducted among healthy sleepers and from September to March. We do not exclude that HMT could be more beneficial for poor sleepers and in a hot environment, and/or in conditions where sleep may be also disrupted (e.g., training camp, following night competition, unfamiliar sleeping environment, during long-haul travel; Walsh et al., 2020). Additional studies should be conducted in this direction.

Limitations

Some limitations need to be acknowledged. First, the participants of this study completed the training sessions in their own training facility; although we checked daily that the training sessions had been carried out correctly, we do not exclude the possibility that training location (indoors, track, or overland) and environmental conditions (temperature, humidity) could have affected sleep. To avoid any climatic season effect, participants were allocated in LMT or HMT groups in a counterbalanced order. Considering the climatic season (i.e., summer, autumn and winter) in which each participant accomplished the protocol did not modify the linear mixed-effects models results. Second, we only manipulated the duration of training sessions; increased training intensity could have resulted in more muscle damage and a distinct impact on sleep. Third, as this study was conducted in a domestic environment, the sleep recording method did not allow to monitor sleep architecture on a daily basis. Additional research on the effect of excessive and/or accumulated training load on sleep architecture will provide a better understanding of how sleep adapts and responds to high training loads.

Practical applications

Practitioners should allocate a special attention to sleep with an individualised approach, considering the level of fatigue and training response, to manage training periodization involving excessive training loads, such as during a training camp. Preventing sleep disruptions in this context may promote training adaptation and performance improvement. The use of HMT did not improve sleep compared with LMT nor interfered with sleep. The interest of the present sleep hygiene strategy should be primarily assessed on an individual basis.

Conclusion

The present study highlighted that the overload and taper periods did not alter the weekly averaged sleep variables in well-trained endurance runners, which is mainly due to inter-individual differences into the training load effectively achieved throughout the period and/or training responses. However, from a day-to-day approach, higher daily training load was associated with an impairment in subsequent sleep

efficiency, wake after sleep onset and sleep onset latency. The chronic use of HMT did not mitigate the negative effect of high training loads on sleep, and future studies should be conducted to explore the potential benefits of HMT in athletes with sleep disturbances and/or exposed to inadequate sleep conditions.

Acknowledgments

The authors are grateful to all the participants for their effort and involvement in this study. Special thanks to Alexandre Dubois for his contribution to the sleep analysis.

Disclosure statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as potential conflicts of interest. The results of the present study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

Funding

MC received a PhD scholarship (CIFRE contract no 2020/0967) funded by Bultex and the French Agency of Research and Technology. The Laboratory Sport, Expertise and Performance is a partner of the French-speaking network ReFORM, recognized as a Research Centre for the Prevention of Injury and Illness and the Protection of Athletes by the International Olympic Committee (IOC). As a member of the IOC Medical Research Network, FIIRST has received funding from the IOC to establish long-term research.

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Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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RESEARCH ARTICLE



Increased training load promotes sleep propensity and slow-wave sleep in endurance runners: Can a high-heat-capacity mattress topper modulate this effect?

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Funding information

PhD scholarship by Bultex, Grant/Award Number: CIFRE contract no 2020/0967

Summary

The present study aimed to: (1) investigate sleep architecture in response to an overload training and taper periods among endurance runners; and (2) assess the sleep benefits of a high-heat-capacity mattress topper. Twenty-one trained male endurance runners performed a 2-week usual training regimen (baseline) followed by 2-week overload and taper periods. From overload to the end of the taper period, they were assigned into two groups based on the mattress topper used: high-heat-capacity mattress topper ($n = 11$) or low-heat-capacity mattress topper ($n = 10$). Training load was assessed daily using the session rating of perceived exertion. Following each period, sleep was monitored by polysomnography, and nocturnal core body temperature was recorded throughout the night. Irrespective of the group, awakening episodes > 5 min decreased following overload compared with baseline (-0.48 , $p = 0.05$). Independently of mattress topper, each 100 A.U. increase in 7-day training load prior to polysomnographic recording was associated with higher slow-wave sleep proportion ($\beta = +0.13\%$; $p = 0.05$), lower sleep-onset latency ($\beta = -0.49$ min; $p = 0.05$), and a reduction in the probability of transition from N1 sleep stage to wakefulness ($\beta = -0.12\%$; $p = 0.05$). Sleeping on a high-heat-capacity mattress topper did not affect any sleep variable compared with a low-heat-capacity mattress topper. Increased training loads promote slow-wave sleep and sleep propensity, highlighting the adaptative nature of sleep to diurnal activity and the role of sleep in physiological recovery. Further studies are required on the potential benefits of high-heat-capacity mattress toppers on sleep architecture among athletes.

KEYWORDS

athletes, polysomnography, recovery, sleep architecture, thermoregulation, training program

1 | INTRODUCTION

Regular participation in physical activity provides various health benefits through exercise-induced musculoskeletal, metabolic and cardiorespiratory adaptations (Paffenbarger & Lee, 1998). Previous research

demonstrated the beneficial effects of regular physical activity on sleep quantity and quality in healthy individuals (Kredlow et al., 2015; Kubitz et al., 1996; Park et al., 2021). Sleep is increasingly recognized as a crucial component in athlete preparation by providing a number of metabolic, hormonal and cognitive restorative functions (Halsom &

Juliff, 2017; O'Donnell et al., 2018). It has notably been suggested that slow-wave sleep (SWS), a component of non-rapid eye movement (NREM) sleep, is associated with increased secretion of growth hormone and plays an important role in the recovery process via muscle repair and adaptation (Halson, 2014; Léger et al., 2018; O'Donnell et al., 2018).

However, practicing sport at the highest level requires high volumes of training, which in turn poses a significant challenge to the athlete's sleep (Nedelec et al., 2018). Insufficient sleep quantity and/or quality are frequently observed among elite athletes (Walsh et al., 2020), which may be detrimental to endurance performance (Craven et al., 2022). A growing body of scientific evidence highlights the negative impacts of excessive and/or accumulated training loads (overload) on sleep (Falck et al., 2021; Roberts et al., 2019; Walsh et al., 2020). Previous studies have suggested several potential factors that may contribute to sleep disturbance during an overload period, such as muscle soreness, increased sympathetic activity and depressed immunity (Hauswirth et al., 2014; Sargent et al., 2016; Schaal et al., 2015). Most of the aforementioned studies used wristwatch actigraphy to examine the impact of an overload period on sleep. Such a method does not allow to provide the most comprehensive measurement of sleep stages, which are typically assessed using polysomnography (PSG). Taylor et al. (1997) observed higher SWS proportions alongside a greater number of movements using 1-night PSG at the onset of the season (medium-intensity training) and an overload period (high-intensity training) compared with a taper period (low training volume) in elite female swimmers, which supports both the restorative physiological function of SWS and the potential negative impact of excessive training loads on sleep, respectively. To the best of our knowledge, no study determined the impact of an overload period on sleep architecture among endurance runners. Prolonged running exercises induce a larger extent of muscle damage, delayed-onset muscle soreness and inflammation response (Nieman et al., 2014) compared with non-weight-bearing activities such as swimming, which may suggest an important effect of overloading on sleep in endurance runners (Halson, 2014). Furthermore, the traditional hypnogram-based features (time spent in sleep stages) used by Taylor et al. (1997) do not provide information on the internal structure of sleep and may be insufficient for assessing its quality (McCarter et al., 2022). Estimating the probability of transition from one sleep stage to another can provide additional information on sleep quality.

Based on the aforementioned findings, developing practical strategies for the optimization of athletes' sleep is highly required. The thermal properties of bedding can be manipulated to optimize sleeping microclimate and favour sleep by influencing thermoregulatory processes (Troynikov et al., 2018). Sleep initiation and the high prevalence of SWS in the first sleep cycles are intimately coupled with the circadian decline phase of core body temperature (CBT), which is mediated by distal skin warming and heat loss from the periphery (Krauchi & Deboer, 2010; Van Someren, 2006). Sleeping on a high-heat-capacity gel sheet on the top of a foam mattress has been shown to enhance the slow and continuous removal of body heat during sleep by conductive heat transfer, leading to an increased SWS proportion and improved subjective sleep comfort from an acute

perspective, i.e. a single night (Aloulou et al., 2020b; Herberger et al., 2020; Kräuchi et al., 2018). However, the relevance of chronic usage of this bedding strategy for endurance runners exposed to high training loads is currently unknown.

The aims of the present study were to: (1) examine the sleep architecture in response to 2-week overload training and taper periods among endurance runners; and (2) to assess the effectiveness of a high-heat-capacity mattress topper (HMT) on sleep. We hypothesized that higher training loads may increase SWS proportion while disturbing sleep continuity, and that sleeping on a HMT may mitigate this negative effect.

2 | METHODS

2.1 | Participants

Twenty-six well-trained male endurance runners [mean \pm SD; age = 27.2 ± 4.8 years; height = 175.4 ± 5.6 cm; body mass = 67.6 ± 7.0 kg; peak oxygen uptake ($\dot{V}O_{2peak}$) = 61.9 ± 5.4 ml min^{-1} kg^{-1}] volunteered to participate in this study. All participants have been examined by a medical doctor including an electrocardiogram at rest to exclude any contraindication to high-intensity training. The inclusion criteria were: (a) competing for ≥ 4 years; (b) training \geq five times per week; and (c) having a performance level below 38 min over a 10-km race. The exclusion criteria were checked at the start of the study as follows: (a) actigraphy or PSG-confirmed sleep disorders, such as sleep apnea (apnea-hypopnea index > 10), periodic limb movement syndrome, hypersomnia, insomnia, circadian sleep rhythm disorders or narcolepsy; (b) sleep complaints [i.e. Pittsburgh Sleep Quality Index ≥ 5 (Buysse et al., 1989)], extreme morning or evening chronotype [i.e. scores < 31 or > 69 (Horne & Ostberg, 1976)], and excessive daytime sleepiness [i.e. Epworth Sleepiness Scale > 10 (Johns, 1991)]; (c) shift workers; (d) daily consumption of alcoholic beverages, more than 300 mg of caffeine per day and the use of prescribed medications. All the participants recruited were recreational athletes, carrying out a full-time professional/study activity in addition to their training program. They provided their written informed consent before the initiation of experiments. The study was conducted in accordance with the Declaration of Helsinki (1964: revised in 2001) and was approved by the local ethic committee (approval reference: 21.00586.000020, ID-RCB: 2021-A01607-34).

2.2 | Experimental design

The protocol was conducted from September 2021 to March 2022. An overview of the study design is presented in Figure 1. Participants followed a 6-week training program divided into three 2-week periods during which training volume was manipulated. The instructions were to increase/decrease the duration of each training session while maintaining the intensity and number of sessions per week. The first period

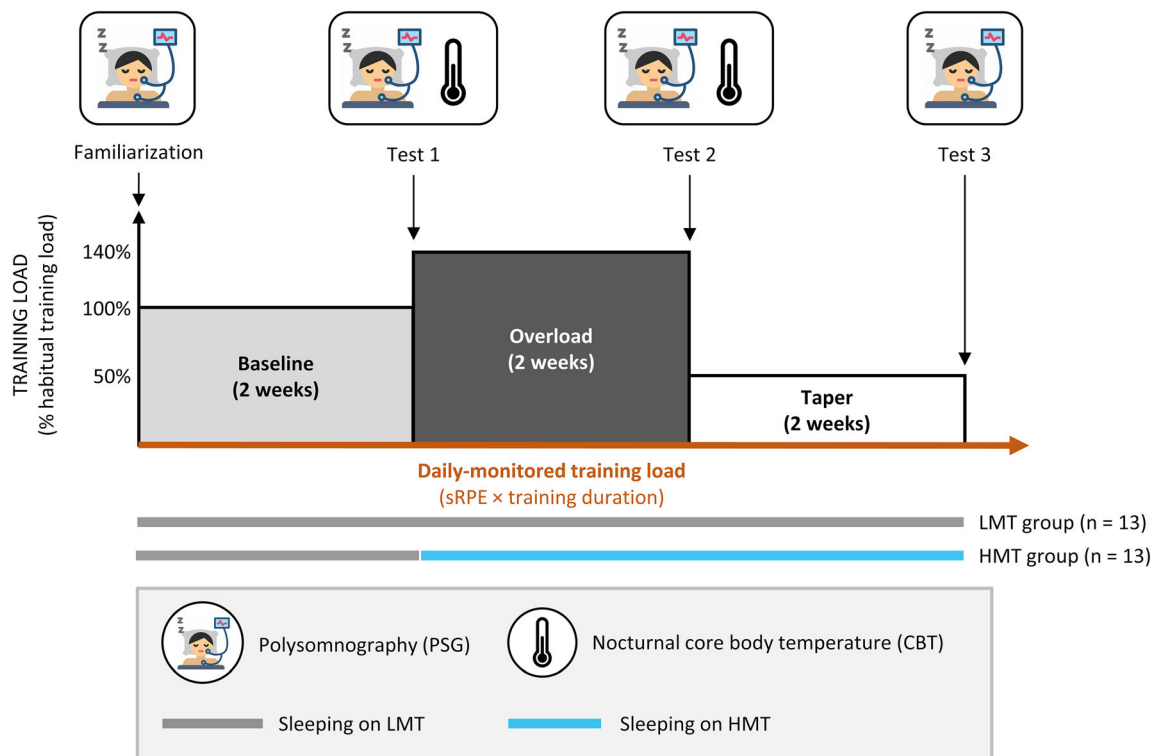


FIGURE 1 Design of the experimental protocol. HMT, high-heat-capacity mattress topper; LMT, low-heat-capacity mattress topper; sRPE, session rating of perceived exertion.

consisted of 2 weeks (baseline) based on their usual training regimen. During the second 2-week period (overload), participants were asked to increase the duration of each training session by 40% in an attempt to elicit functional overreaching (Hauswirth et al., 2014; Meeusen et al., 2013). During the third 2-week period (taper), the duration of each training session was reduced by 50% of the usual training regimen in order to maximize post-taper performance gains (Bosquet et al., 2007; Mujika, 2010). Participants performed training sessions in their usual training facilities, on their own ($n = 9$) or with a coach ($n = 12$). The same investigator daily checked that training sessions were carried out correctly. Before the 6-week training program, each participant performed a night of familiarization with the PSG equipment in the same home environment as for the experimental nights to avoid any first-night effect (Hof Zum Berge et al., 2021). At the end of baseline (Test 1), overload (Test 2) and taper (Test 3), participants came to the laboratory and the same qualified practitioner equipped them with the PSG equipment. They were instructed to go to bed and get up at similar times between Test 1, Test 2 and Test 3, according to their sleep habits assessed daily by actigraphy during each training period. Given the potential interest of HMT and not manipulating training load on CBT, nocturnal CBT was continuously recorded only at Test 1 and Test 2. In order to limit dietary influences on test results, participants were asked to follow standardized nutritional guidelines (quantity and content for food and drink) 48 hr prior to the night with PSG, as well as abstaining from any training session 24 hr prior to the night with PSG. Compliance with these instructions was checked upon arrival for each test.

All participants slept in the same bedroom in their home throughout the protocol. During baseline, all used a low-heat-capacity mattress topper (LMT). According to a matched group single-blinded experimental design, participants were allocated into two groups from the start of overload (i.e. the day after Test 1) to the end of the protocol based on the mattress topper used: HMT ($n = 13$) and LMT ($n = 13$). Participants were allocated in LMT or HMT groups in a counterbalanced order to avoid any climatic season effect, and according to their usual training regimen and individual characteristics (i.e. age, height, body mass and $\dot{V}O_{2peak}$). No significant differences ($p > 0.05$) in participants' characteristics at baseline were reported between groups.

2.3 | Mattress topper properties

The HMT consisted of 100% foam (density of 40 kg m^{-3} ; Bultex, France) with a high-heat-capacity gel sheet (density of 1000 kg m^{-3} ; dimensions of $80 \times 120 \times 2.5 \text{ cm}$) on the top to slowly remove body heat during sleep by conductive transfer (Aloulou et al., 2020b; Herberger et al., 2020; Kräuchi et al., 2018). The LMT consisted solely of the same 100% foam. The different thermal properties are related to thermal resistance (HMT: $1.18 \text{ m}^2 \text{ K W}^{-1}$; LMT: $1.63 \text{ m}^2 \text{ K W}^{-1}$ at 28°C). Both mattress toppers were sized $90 \times 200 \times 6 \text{ cm}$, and were covered with a ticking (density of 540 g m^2 , composed of 99% polyester and 1% elastane; Figure 2). Participants kept the same bed cover, pillow and sleepwear throughout the study.

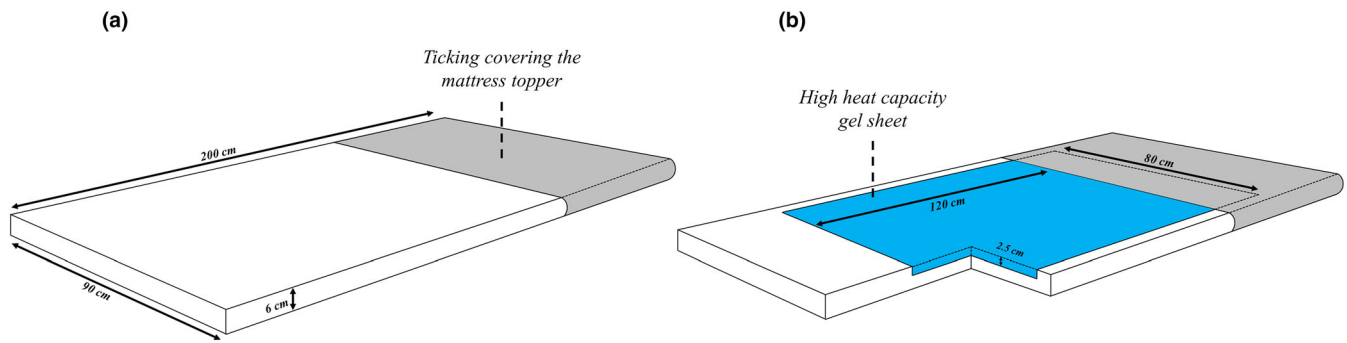


FIGURE 2 Figure illustrating the properties of mattress topper type: (a) low-heat-capacity mattress topper (LMT); and (b) high-heat-capacity mattress topper (HMT).

2.4 | Daily-monitored training load

Participants were asked to answer the question “How was your workout?” using the session rating of perceived exertion (sRPE) within 30 min from the end of each training session using the modified CR-10 scale (Foster et al., 2001). The daily training load was calculated by multiplying sRPE and the duration of each training session within a day. The weekly training load was determined as the sum of the daily training loads for each week, and the average training load per training period was calculated as the average between the 2 weeks of the period.

2.5 | Sleep analysis

2.5.1 | Polysomnography

Polysomnography recordings were obtained using a portable device (Nox A1; ResMed, San Diego, CA, USA). The PSG montage included: six electroencephalographic channels placed according to the international 10–20 electrodes placement system (F3-M2, F4-M1, C3-M2, C4-M1, O1-M2 and O2-M1), left and right electro-oculographic channels, chin (mentalis and submentalis) and bilateral tibial electromyographic channels, an electrocardiogram, and two respiratory impedance for rib cage and abdominal wall motion. The sleep data were scored using Noxturnal software (version 6.0; ResMed, San Diego, CA, USA) by one trained specialist (> 3000 analyses in the last 10 years) who was blinded to the aim of the study. The 2017 American Academy of Sleep Medicine (AASM; Berry et al., 2017) criteria were employed to establish the individual hypnograms from 30-s epochs scored as wakefulness, NREM stage 1 (N1), NREM stage 2 (N2), NREM Stage 3 (SWS) or REM sleep (REM). Cortical arousals, leg movements and respiratory events were also identified. Then, traditional hypnogram-based features were extracted to describe the sleep architecture (Table 1).

To estimate the continuity and discontinuity of sleep architecture, homogeneous first-order Markov chain modelling were computed from each hypnogram, in which the probability of a future sleep stage depends only on the current sleep stage (Kim et al., 2009). The

TABLE 1 Definitions of the PSG-based sleep variables.

Sleep variables	Description
TST (min)	Number of minutes spent in any sleep stage (N1, N2, SWS and REM)
SOL (min)	Number of minutes from lights off to the first epoch of any sleep stage (i.e. N1, N2, SWS and REM)
WASO (min)	Number of minutes spent awake after sleep onset and before final awakening
SE (%)	TST expressed as a percentage of time in bed
N1, N2, SWS and REM (%TST)	Proportion of time spent in each of these sleep stages expressed as a percentage of TST
Sleep stage shifts (events per hr)	Number of changes from one sleep/wake stage to another, expressed per hour of TST
Awakenings > 5 min (events)	Number of awakening episodes longer than 5 min
Cortical arousals (events per hr)	Number of sudden changes in electroencephalographic frequency (alpha, theta and/or frequency > 16 Hz) lasting at least 3 s and with the presence of at least 10 s of prior sleep, expressed per hour of TST
Limb movements (events per hr)	Number of increases of $\geq 8 \mu\text{V}$ in the activity of the tibial electromyogram lasting between 0.5 and 10 s, expressed per hour of TST

Abbreviations: REM, rapid eye movement; SE, sleep efficiency; SOL, sleep-onset latency; SWS, slow-wave sleep; TST, total sleep time; WASO, wake after sleep onset.

Markov chain transition probability $Pr_{i \rightarrow j}$ from sleep stage i to sleep stage j over the whole sleep period was calculated as follows:

$$Pr_{i \rightarrow j} = (N_{i \rightarrow j} / T_i) \times 100,$$

where $N_{i \rightarrow j}$ is the number of transitions from sleep stage i to sleep stage j , and T_i is the total number of sleep stage i (Kishi et al., 2018). Models were computed using the “createSequenceMatrix” function of the “markovchain” R package, and the results are presented as transition matrices.

2.5.2 | Temperature measurements

Core body temperature

Participants ingested a radio telemetric pill (BodyCap; e-Celsius® Performance) at least 3 hr before bedtime to continuously record the nocturnal CBT with a sampling rate of 1 per min. The sensor has been shown to be reliable and valid, with a mean difference of -0.08°C compared with reference hot water bath temperatures within physiological ranges ($33\text{--}44^{\circ}\text{C}$), a low standard error of measurement (0.04°C), an intraclass correlation coefficient of 1.00, and a mean agreement between test and retest trials of $-0.01 \pm 0.03^{\circ}\text{C}$ (Bongers et al., 2018). The nocturnal CBT data were then averaged and displayed in 10-min time bins.

Bedroom air temperature

Bedroom air temperature was measured using an iButton (DS 1922L, iButtonLink, Whitewater, USA; resolution 0.0625°C ; sampling rate: 1 per min) placed on the wall.

Subjective assessments

Perceived thermal comfort (on a scale from -2 “very uncomfortable” to $+2$ “very comfortable”) and sensation (on a scale from -3 “cold” to $+3$ “hot”; Zhang & Zhao, 2008) during the night were recorded for each participant the morning after each test.

2.6 | Data and statistical analysis

Based on previous research from our lab (Hauswirth et al., 2014), we used a non-inferiority sample size calculation to estimate the required

sample size to detect potential differences in sleep efficiency (SE) between baseline and overload. It was necessary to include 10 participants to achieve a significance of 0.05 and power criteria ($1 - \beta$) of 90%, respectively (G*Power program, version 3.1.9.7). Considering the longitudinal design and the allocation of participants into two distinct groups, we chose to increase the number of participants to 13 in each group to account for potential dropout. Data processing and statistical analyses were performed using R software (version 4.2.2). Linear mixed-effects models (LMM; “lmer” function of the “lme4” R package) were used to consider the non-independence of observations at the level of individuals. Variance inflation factor (VIF) was used to assess collinearity issues between fixed-effect variables, and models with a low VIF (score < 5) were selected. Then, the structure of the models (i.e. with and without fixed-effects interaction) was tested, and the best fitting-model was selected according to the smallest Akaike Information Criterion. The residuals for each model were visually inspected, and no obvious deviations from homoscedasticity and normality were revealed. Table 2 provides an overview of the three LMM used according to the dependent variables tested and the research question.

For the first and third LMM, the significance of the fixed-effects was determined using the “mixed” function from the “afex” R package, using type 2 tests. When a significant effect of a fixed-effect was reported, multiple post hoc Student’s *t*-test pairwise comparisons were conducted using least square means contrasts with the Bonferroni correction for alpha adjustments (“emmeans” function of the “emmeans” package). The effect sizes (ES) with associated 95% confidence intervals (CI) were calculated to interpret the magnitude of the difference in means values between the fixed-effects, and were interpreted using the following criteria: $< 0.2 = \text{trivial}$, $0.2\text{--}0.6 = \text{small}$,

TABLE 2 LMM details used for each dependent variable according to the research question.

	Objective	Dependent variables	Fixed effect(s)	Random effect
LMM #1	To compare the weekly change in training session details for each group and evaluate the effect of training periods and between-group differences on sleep	<ul style="list-style-type: none"> Training session details: weekly training load and training volume, number of training sessions, mean sRPE per training session, training start times Sleep: bedtime, get-up time, PSG-based sleep variables, perceived thermal comfort and sensation, and bedroom air temperature 	Group, training week, and group \times training week interaction	The participant
LMM #2	To assess the effect of the training load over the 7-day period prior to PSG recording according to the mattress topper	<ul style="list-style-type: none"> Sleep: PSG-based sleep variables 	The training load over the 7-day period prior to PSG recording and the mattress topper	The participant
LMM #3	To evaluate between-group differences on nocturnal CBT kinetics following training periods	<ul style="list-style-type: none"> CBT 	Group, training period (baseline and overload) and 10-min time interval (main effects and interactions)	The participant and the slope (7-hr time points in 10-min time bins)

Abbreviations: CBT, core body temperature; LMM, linear mixed-effects model; PSG, polysomnography; sRPE, session rating of perceived exertion.

0.6–1.2 = *moderate*, 1.2–2.0 = *large*, and > 2.0 = *very large* (Hopkins et al., 2009).

Results are expressed as the mean \pm standard deviation (SD), except for the LMM for which results are presented as the estimate (β) and the 95% CI. The level of significance was set at $p < 0.05$.

3 | RESULTS

Five participants were excluded from the protocol because of injury ($n = 2$), illness ($n = 1$) or for personal/professional reasons ($n = 2$) at the start of the protocol. Twenty-one participants (HMT, $n = 11$ and LMT, $n = 10$) were included. PSG data from one participant (HMT group) were discarded at Test 3 because of inconsistent bedtimes and wake-up times compared with Test 1 and Test 2. Another participant (HMT group) did not perform Test 3 due to Covid-19 infection. Participants were classified at level 2 “Trained/Developmental” (HMT, $n = 3$; LMT, $n = 4$) and at level 3 “Highly Trained/National” (HMT, $n = 8$; LMT, $n = 6$) according to the guidelines proposed by McKay et al. (2022). A main effect of training week was found ($p < 0.001$) on weekly training load, number of training sessions, training volume and sRPE. No main effect of group and group \times training week interaction was reported for these variables ($p > 0.24$). For training start times, there was no main effect of training week and group \times training week

interaction ($p > 0.22$), but a main effect of condition was observed ($p = 0.02$). Independently from the training week, the HMT group trained 02:24 hr later than the LMT group ($p = 0.02$; ES = 1.41 [95% CI, 0.24–2.58]). For all these variables, post hoc analyses between weeks independently from the group are shown in Table 3.

3.1 | Effects of training periods and mattress topper on sleep and temperature

3.1.1 | Polysomnography

There was no main effect of group, training period or group \times training period interaction ($p > 0.07$) for bedtime, get-up time, cortical arousals, limb movements as well as the following sleep variables: total sleep time (TST), SE, sleep-onset latency (SOL), wake after sleep onset (WASO), the number of sleep stage shifts, and the N1, N2, N3 and REM sleep proportions (Figure 3). We observed a main effect of training period for the number of awakenings > 5 min ($p = 0.04$), and significant group \times training period interactions for wake-to-wake ($p = 0.01$) and wake-to-N1 ($p < 0.01$) transition probability. Independently from the group, the number of awakenings > 5 min was *moderately* lower at Test 2 than Test 1 (-0.48 ; $p = 0.05$; ES = -0.77 [-1.41 to -0.13]). As shown in Figure 4, only the HMT group showed

TABLE 3 Weekly training session details during baseline, overload and taper periods in the two experimental groups.

	Baseline		Overload		Taper	
	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6
Weekly training load (A.U.)***	1391.5 \pm 522.2	1495.2 \pm 843.2	2120.0 \pm 735.9 ^{a,b}	1937.4 \pm 806.4 ^{a,b}	976.6 \pm 566.8 ^b	938.1 \pm 699.6 ^{a,b}
LMT	1427.9 \pm 494.3	1317.8 \pm 378.8	2139.2 \pm 494.8	1697.3 \pm 835.1	1037.7 \pm 620.8	865.0 \pm 568.9
HMT	1358.4 \pm 568.4	1656.5 \pm 1110.2	2102.5 \pm 928.5	2155.6 \pm 750.2	921.1 \pm 537.3	1004.6 \pm 823.1
Weekly training volume (hr)***	6.0 \pm 2.3	5.9 \pm 2.8	8.0 \pm 2.9 ^{a,b}	6.9 \pm 2.9	4.7 \pm 2.6	4.0 \pm 2.5 ^{a,b}
LMT	6.1 \pm 2.2	5.6 \pm 1.7	8.4 \pm 3.0	6.4 \pm 2.9	5.2 \pm 3.4	4.2 \pm 2.5
HMT	5.9 \pm 2.4	6.3 \pm 3.6	7.6 \pm 2.9	7.3 \pm 3.0	4.3 \pm 1.6	3.9 \pm 2.6
Number of training sessions***	5.3 \pm 1.6	5.0 \pm 1.6	6.1 \pm 1.8	5.2 \pm 1.7	4.9 \pm 1.5 ^b	4.1 \pm 1.6 ^a
LMT	5.2 \pm 1.6	4.9 \pm 0.9	6.2 \pm 1.5	4.6 \pm 1.3	4.9 \pm 1.4	4.3 \pm 1.2
HMT	5.4 \pm 1.7	5.2 \pm 2.0	6.0 \pm 2.0	5.7 \pm 1.9	4.8 \pm 1.6	4.0 \pm 1.9
Mean sRPE per training session***	2.6 \pm 0.9	2.7 \pm 0.9	3.3 \pm 1.0 ^{a,b}	2.9 \pm 1.0	2.1 \pm 0.7 ^b	2.0 \pm 0.8 ^{a,b}
LMT	2.6 \pm 0.8	2.8 \pm 0.6	3.4 \pm 1.0	2.8 \pm 1.2	2.0 \pm 0.7	2.1 \pm 0.8
HMT	2.5 \pm 0.9	2.6 \pm 1.1	3.2 \pm 0.9	3.1 \pm 0.9	2.1 \pm 0.7	2.0 \pm 0.8
Mean training start time [#]	14:26 \pm 04:26	14:23 \pm 04:26	14:47 \pm 04:27	14:46 \pm 04:27	14:06 \pm 04:28	14:07 \pm 04:27
LMT	12:52 \pm 04:25	13:11 \pm 04:26	13:32 \pm 04:27	13:34 \pm 04:25	12:59 \pm 04:27	13:04 \pm 04:25
HMT	15:58 \pm 03:24	15:39 \pm 04:25	16:03 \pm 04:24	15:51 \pm 04:26	15:08 \pm 04:29	15:19 \pm 04:28

Note: Data are presented as mean \pm SD. Significant main effect of week: *** $p < 0.001$. Independently from the group, ^asignificantly different from Week 1, and ^bsignificantly different from Week 2. Significant main effect of group: [#] $p < 0.05$.

Abbreviations: HMT, high-heat-capacity mattress topper group; LMT, low-heat-capacity mattress topper group; sRPE, session rating of perceived exertion.

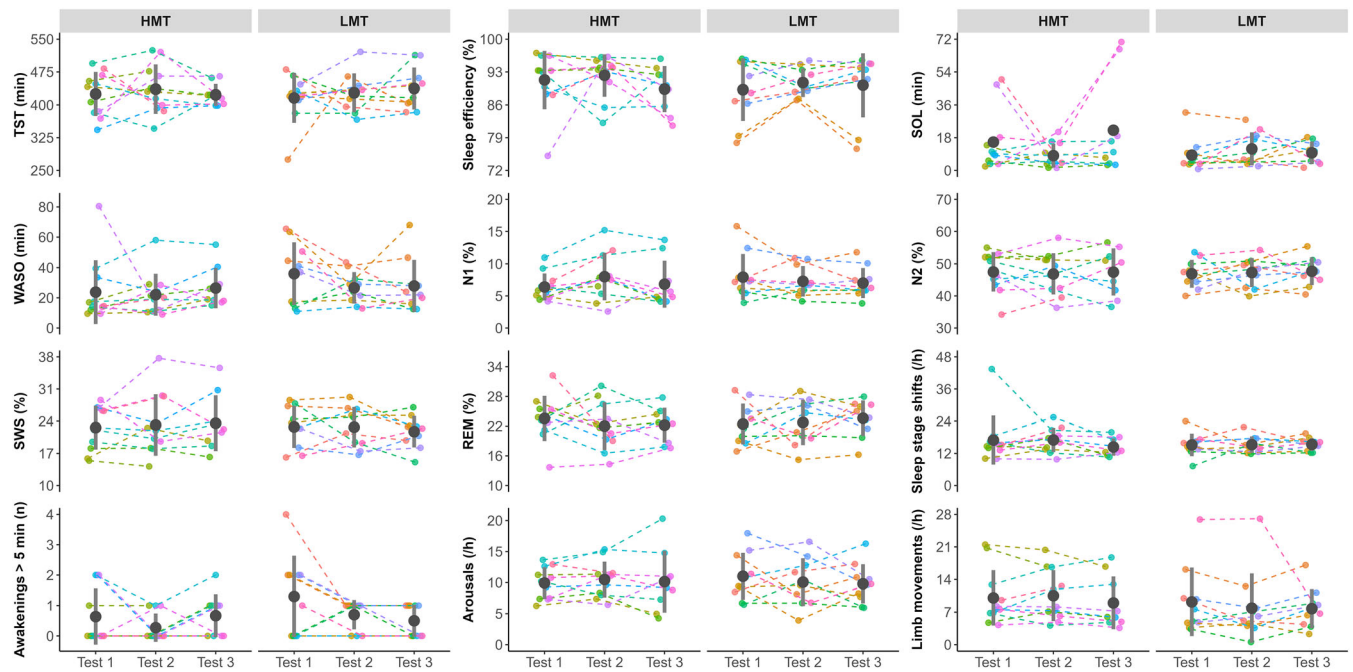


FIGURE 3 Mean ± SD polysomnography (PSG)-based sleep data between HMT and LMT groups following baseline (Test 1), overload (Test 2) and taper (Test 3). The coloured dots represent the values for each participant across the tests. HMT, high-heat-capacity mattress topper group; LMT, low-heat-capacity mattress topper group; SOL, sleep-onset latency; SWS, slow-wave sleep; TST, total sleep time; WASO, wake after sleep onset.

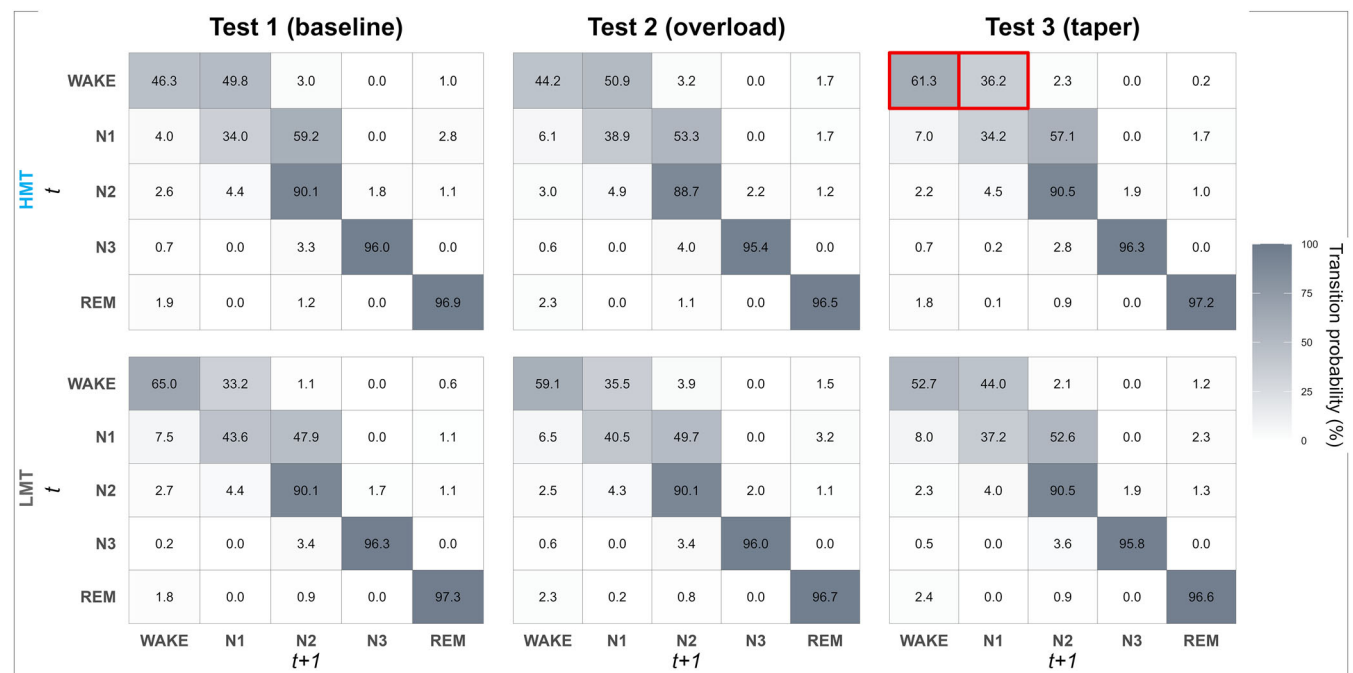


FIGURE 4 Probability matrices of sleep stage transitions following baseline (Test 1), overload (Test 2) and taper (Test 3) periods for HMT and LMT groups. Each cell presents the transition probability from a sleep stage at time t to another sleep stage at time $t + 1$. HMT, high-heat-capacity mattress topper group; LMT, low-heat-capacity mattress topper group. Red-squares show significant post hoc differences from Test 2 ($p < 0.05$).

a moderate increase in the probability of transition from wakefulness to wakefulness at Test 3 compared with Test 1 (+14.4%; $p = 0.09$; $ES = 1.03$ [0.09–1.98]) and Test 2 (+16.5%; $p = 0.04$; $ES = 1.18$ [0.23–2.14]). A moderate decrease was observed only in the HMT

group in the probability of transition from wakefulness to N1 at Test 3 compared with Test 1 (0.13%; $p = 0.08$; $ES = -1.05$ [–1.99 to –0.10]) and Test 2 (–0.15%; $p = 0.05$; $ES = -1.14$ [–2.09 to –0.19]). No main effect of group, training period or group \times training period

interaction was observed for the other sleep stage-transition probabilities ($p > 0.06$).

Considering the training load during the last 7-day period prior to PSG recording, each additional 100 A.U. increase in training load was associated with lower SOL ($\beta = -0.49$ min [-0.99 to 0.00 min]; $p = 0.05$; Figure 5a), higher SWS proportion ($\beta = +0.13\%$ [0.00% to 0.25%]; $p = 0.05$; Figure 5b), and a reduction in the probability of transition from N1 to wakefulness ($\beta = -0.12\%$ [-0.23% to -0.00%]; $p = 0.05$; Figure 5c). There was neither a significant relationship between the training load prior to PSG recording and any of the other sleep variables, nor a significant difference between HMT and LMT in any sleep variables ($p > 0.08$).

3.1.2 | Temperature measurements

No main effects of group, training period or group \times training period interaction ($p > 0.06$) were found for the bedroom air temperature (mean \pm SD: $20.6 \pm 2.1^\circ\text{C}$) as well as perceived thermal comfort and

sensation during the night. For the 7-hr mean nocturnal CBT, there was a main effect of training period and group \times training period interaction ($p < 0.001$; Figure 6), but no main effect of 10-min time interval, group, group \times 10-min time interval interaction and training period \times 10-min time interval interaction ($p > 0.87$). Similar mean nocturnal CBT was reported at Test 1 between the HMT and LMT groups ($p = 0.11$). For the LMT group, mean nocturnal CBT was moderately higher ($+0.132^\circ\text{C}$; $p < 0.001$; ES = 0.93 [0.74 – 1.12]) at Test 2 than Test 1, while a trivial reduction was observed in the HMT group between Test 2 and Test 1 (-0.025°C ; $p = 0.02$; ES = -0.18 [-0.33 to -0.02]).

4 | DISCUSSION

The aims of this study were to examine the impact of training load manipulation (i.e. overload and taper), and to assess the influence of the chronic use of a HMT on sleep architecture in endurance runners. The main findings showed that: (1) higher prior 7-day training loads

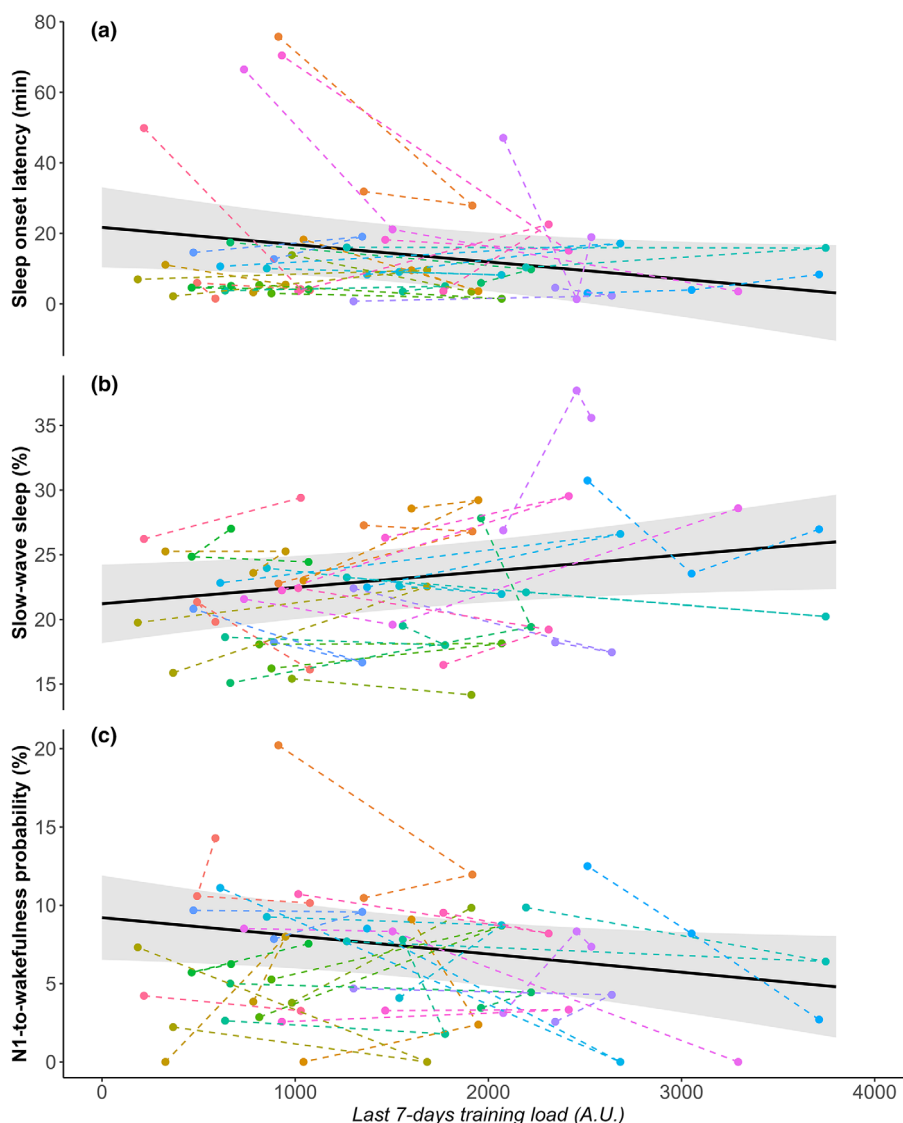


FIGURE 5 Relationships (and 95% confidence interval [CI]) between the training load over the 7-day period prior to polysomnographic (PSG) recording and subsequent: (a) sleep-onset latency (SOL); (b) slow-wave sleep (SWS); and (c) the probability of transition from N1 to wakefulness. The dots and coloured dashed lines represent the values for each participant according to the order of training periods: baseline (Test 1), overload (Test 2) and taper (Test 3).

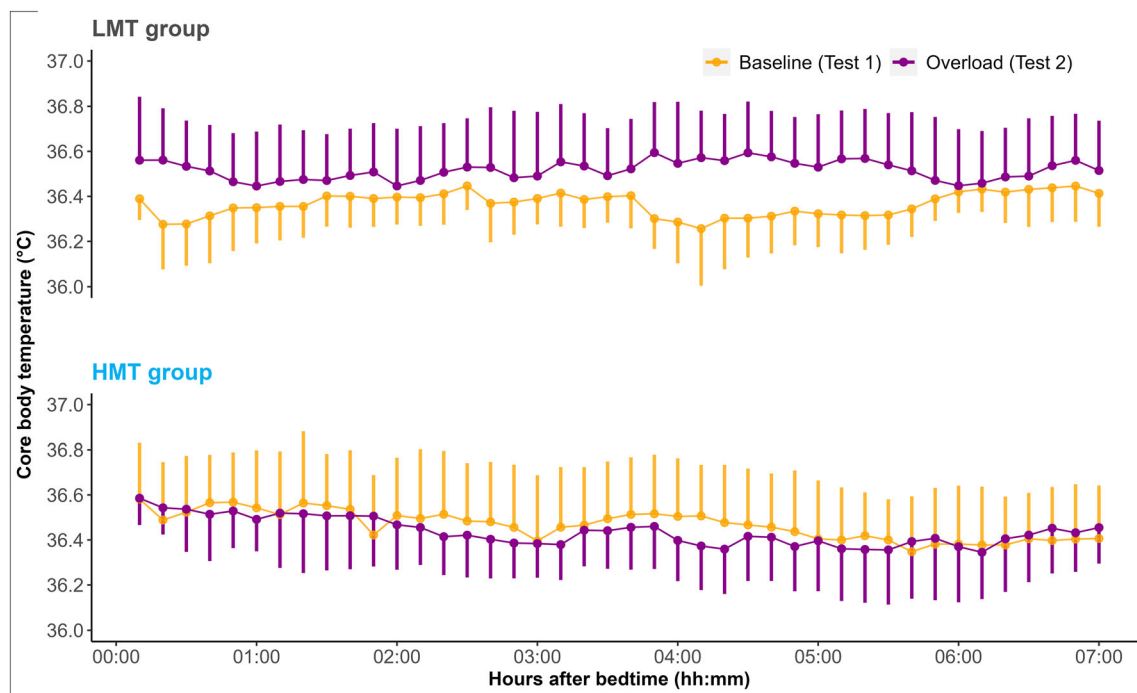


FIGURE 6 Mean \pm SD changes in core body temperature (CBT) over 7 hr after bedtime for each group according to training period: baseline (Test 1) and overload (Test 2).

were associated with an increase in SWS proportion, a reduction in SOL and an improvement in sleep continuity; and (2) HMT did not affect any sleep variable compared with LMT.

4.1 | Impact of training load on sleep

According to the literature, we hypothesized that high training loads may promote SWS while disturbing sleep continuity. The present findings partially confirmed our hypotheses, given that each 100 A.U. increase in training load over the 7-day period prior to PSG recording was associated with an increase in SWS proportion (+0.13%), a reduction in SOL (−0.49 min), without any alteration of TST, SE, WASO, sleep stages distribution, number of sleep stage shifts, cortical arousals and limb movements. A number of studies demonstrated that regular exercise can promote SWS and SOL in healthy populations (Kredlow et al., 2015; Kubitz et al., 1996; Park et al., 2021). Among elite female swimmers, Taylor et al. (1997) reported an increase in SWS proportions using 1-night PSG at the onset of the season and during an overload compared with a taper period. In endurance athletes, studies are currently limited, but there is some evidence for a greater amount of SWS following a heavy training or competition (Chennaoui et al., 2015; Driver & Taylor, 2000; Vlahoyiannis et al., 2021). A novelty of the present study is that SWS and SOL are also promoted by the day-to-day accumulation of training load in well-trained endurance runners. This finding was also observed in an ultra-endurance cyclist engaged in a multiday ultra-endurance event, who experienced a progressive day-

to-day increase in the amount of SWS (Nédélec et al., 2022). SWS provides a restorative function to the body essential for recovery in an athlete through energy conservation, anabolic hormone release (e.g. growth hormone), cleaning of cerebral metabolites accumulated during the waking period and regulation of the immune/inflammatory system (Léger et al., 2018). SWS, together with SOL, acts as the primary driving mechanism in the homeostatic sleep process and is a reliable indicator of sleep need (Borbély et al., 2016). Based on the evidence that prolonged running exercises induce large extents of muscle damage, delayed-onset muscle soreness and inflammation response (Nieman et al., 2014), it is likely that the daily accumulation of training load maximized the sleep pressure and/or increased the physiological restorative demands promoting SWS and sleep propensity, which supports the adaptive nature of sleep to physical diurnal activity (Halson, 2014; Léger et al., 2018; Vlahoyiannis et al., 2021). In addition, we observed that higher prior training loads were associated with lower probability of transition from N1 to wakefulness, which may reflect a better sleep continuity. We also noted a reduction in the number of awakenings > 5 min, irrespective of the group, following the overload compared with baseline. Taken together, these results suggest that higher training load may have facilitated sleep continuity throughout the night, which in turn may have contributed to increased SWS proportion.

Nevertheless, it is widely recognized that high training loads may be detrimental to the sleep of athletes (Falck et al., 2021; Roberts et al., 2019; Walsh et al., 2020). Several studies have reported a progressive decline in sleep quantity and/or SE when endurance athletes are exposed to excessive and/or accumulated training loads

(Hausswirth et al., 2014; Kölling et al., 2016; Lastella et al., 2015), or in response to day-to-day variations in training load (Aloulou et al., 2021; Figueiredo et al., 2021; Lastella et al., 2020). All of these studies monitored sleep using wristwatch actigraphy that records movements over time to estimate sleep/wake, suggesting that high training loads may cause more movements during sleep. These movements can result in cortical arousals, which may cause changes in sleep stage from a deep one to a light one, with the possibility of awakening as the final outcome (Ohayon et al., 2017). However, actigraphy does not measure actual sleep measures (e.g. sleep stages, cortical arousals), which are critical determinants of sleep quality (Halson, 2019; McCarter et al., 2022). In addition, Falck et al. (2021) proposed an inverted U-shaped, dose-response relationship between training load and sleep, i.e. increased training load promotes sleep until a “tipping point”, beyond which acute fatigue or overreaching occurs, and sleep is disturbed. In the present study, participants were asked to suddenly increase the training volume during overload, i.e. from one day to the next. We can speculate that a gradual increase in training volume may have resulted in different sleep results. Future studies are required on the effects of various levels of accumulated training loads on sleep architecture.

4.2 | Effects of HMT on sleep and temperature

We hypothesized that HMT would improve sleep quality by increasing conductive body heat loss. This hypothesis was not confirmed as no significant difference in any sleep variable was observed between HMT and LMT. It is well documented in the literature that sleep initiation occurs in close phase with the increase in distal skin regions temperature, leading to the subsequent reduction in CBT (Krauchi & Deboer, 2010). In this respect, previous studies have successfully manipulated CBT by thermal interventions in an attempt to promote sleep. When compared with a low-heat-capacity mattress, these studies reported that sleeping on a high-heat-capacity mattress, from an acute perspective (i.e. a single night), had positive effects on sleep quality (i.e. higher SWS and REM proportions and/or reduction in WASO) in healthy young men (Aloulou et al., 2020b; Herberger et al., 2020; Kräuchi et al., 2018). These studies also noted a decrease in mean nocturnal CBT (from -0.11 to -0.16°C) when sleeping on this bedding strategy. In the present study, the HMT group showed a trivial reduction (-0.025°C) in mean nocturnal CBT following the overload compared with baseline, while a moderate increase ($+0.132^{\circ}\text{C}$) was observed in the LMT group. We can speculate that sleeping on a HMT may have mitigated the overload-induced increase in CBT, without reducing CBT enough to induce any positive effect on sleep. Excessive training loads have been demonstrated to increase circulating cortisol, sympathetic activity and systemic inflammation (Roberts et al., 2019), and we can assume that overload may have affected the normal downregulation of CBT during sleep. However, these findings should be interpreted with caution, as the intensity and the time of day are important factors that may lead to different effects of exercise on circadian

rhythms (e.g. CBT), and subsequent sleep (Aloulou et al., 2020a; O'Connor et al., 1998; Thomas et al., 2020). In the present study, participants performed very few training sessions ($n = 3$) close to bedtime, i.e. ending ≤ 1 hr before bedtime. Future studies are required on the effect of excessive training loads on circadian rhythms (e.g. CBT) and subsequent sleep.

5 | LIMITATIONS

Although we daily checked that training sessions had been effectively carried out, inter-individual differences in training load were observed, which may have resulted in inter-individual variability in sleep responses. Training load in Week 4 was similar to Week 1 in the LMT group, likely due to a lower number of training sessions performed by some athletes. There was also missing PSG data from two participants in the HMT group at Test 3, resulting in a lower statistical power. In this respect, LMM were used to assess the relationship between training load and sleep at the individual level while dealing with missing data (Harrison et al., 2018). We only quantified training load using a subjective approach (i.e. sRPE), whereas other training metrics (e.g. distance covered, time spent in intensity zones) would have allowed to assess the dissociated effect of training components on sleep, as reported elsewhere (Aloulou et al., 2020b; Roberts et al., 2019). Moreover, several factors not assessed in the present study may contribute to sleep disturbances during an overload period, such as muscle damage/soreness, exercise-induced inflammation, depressed immune response, mood changes, increased stress, anxiety and sympathetic activity (Roberts et al., 2019; Walsh et al., 2020). In addition, we did not use common sleep questionnaires following training periods, which would have provided additional information on the effect of manipulating training load on sleep perception. One originality of the present study was to monitor sleep using PSG at the end of each training period. However, this did not capture the daily response of sleep architecture to training load fluctuations, and further investigations should be conducted in this respect to provide more precise information for coaches on the effect of training load variations on sleep architecture.

6 | CONCLUSION

The present study highlighted that higher accumulated training loads promote SWS and sleep propensity with some evidence of better sleep continuity. Such load-mediated effects may reflect the adaptive nature of sleep to diurnal activity and the role of sleep in physiological recovery. The present findings support overloading as an effective training manipulation to improve sleep quality in healthy individuals. In contrast to our hypothesis, the chronic use of HMT did not influence sleep quality. Additional research should be conducted on the potential benefits of HMT presenting different thermal properties or in various thermal environments on sleep architecture.

AUTHOR CONTRIBUTIONS

Maxime Chauvineau: Conceptualization; investigation; funding acquisition; writing – original draft; methodology; validation; visualization; writing – review and editing. **Florane Pasquier:** Investigation; methodology. **François Duforez:** Conceptualization. **Gaël Guilhem:** Conceptualization; supervision; writing – review and editing; methodology. **Mathieu Nedelec:** Conceptualization; supervision; writing – review and editing; methodology.

ACKNOWLEDGEMENTS

The authors are grateful to all the participants for their effort and involvement in this study. Special thanks to Alexandre Dubois for his contribution to the sleep analysis.

FUNDING INFORMATION

MC received a PhD scholarship (CIFRE contract no 2020/0967) funded by Bultex and the French Agency of Research and Technology. The Laboratory Sport, Expertise and Performance is a partner of the French-speaking network ReFORM, recognized as a Research Centre for the Prevention of Injury and Illness and the Protection of Athletes by the International Olympic Committee (IOC). As a member of the IOC Medical Research Network, FIIRST has received funding from the IOC to establish long-term research programs on the prevention of injuries and illnesses in sport for the protection of athlete health.

CONFLICT OF INTEREST STATEMENT

The authors declare that this work was conducted in the absence of any potential conflicts of interest. The present findings are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

CONSENT TO PARTICIPATE

Informed consent was obtained from all participants included in the study.

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How to cite this article: Chauvineau, M., Pasquier, F., Duforez, F., Guilhem, G., & Nedelec, M. (2023). Increased training load promotes sleep propensity and slow-wave sleep in endurance runners: Can a high-heat-capacity mattress topper modulate this effect? *Journal of Sleep Research*, e14132. <https://doi.org/10.1111/jsr.14132>

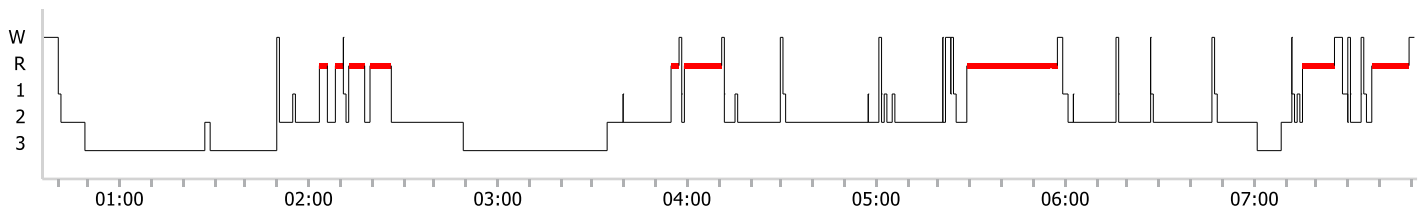
Athlète

Informations

NOM :	Prénom :	Âge :
Taille :	Poids :	Date de naissance :
Fédération :	Sport :	Nuit du :

Résultats

Hypnogramme



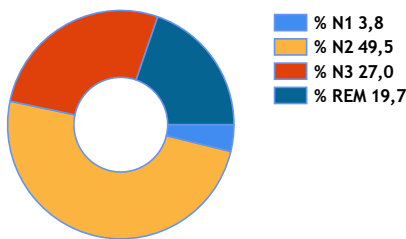
Analyse du tracé

Heure de coucher	Heure de lever	Temps passé au lit	Temps total de sommeil	Efficacité de sommeil	Latence d'endormissement	Latence REM
00:34	07:51	7h 16m	6h 56m	95%	5.1 min	83.5 min

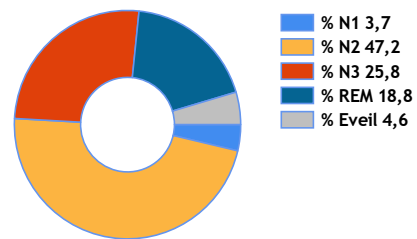
Stades de sommeil

Éveil intra-sommeil	N1	N2	N3	REM
12.5 min	16 min	206 min	112.5 min	82 min

% du Temps Total de Sommeil



% de la Période de Sommeil



Micro-éveils

	Index	Nombre		Index	Nombre
Tous	6.1 /h	42	PLM	0 /h	0
Respiratoires	1.4 /h	10	LM	1.9 /h	13
Spontanés	2.7 /h	19	RERA	0 /h	0

Evènements respiratoires



Paramètres respiratoires

Index Apnées Hypopnées	4 /h	Apnées obstructives	8 soit 1.2 /h
IAH dorsal	5.1 /h	Apnées centrales	0 soit 0 /h
IAH non-dorsal	2.8/h	Apnées mixtes	0 soit 0 /h
Apnée la plus longue	13.4 s	Hypopnées	21 soit 3 /h
Durée moyenne apnées	11.5 s	Durée moyenne hypopnées	25.8 s
Durée totale en apnée	1.5 min	Fréquence respiratoire	14.4 /min

Index de limitation du débit respiratoire : 1,8% du temps d'enregistrement.

Oxymétrie

Index de désaturations	0.4 /h	Nombre de désaturations	3 désats
SpO2 moyenne	94.7 %	SpO2 la plus faible	91 %
Durée SpO2 < 90%	0% soit 0 min	Durée SpO2 ≤ 88%	0% soit 0 min
Pouls moyen	50.9 bpm		
Pouls maximal (0 tachycardies)	88 bpm	Pouls > 100 bpm (0min)	0%
Pouls minimal (0 bradycardies)	39 bpm	Pouls < 40 bpm (0.1min)	0%

Ronflements

Episode de ronflements	13.4%	Volume audio moyen	68.8 dBc
Episode le plus long	4.9 min	Volume audio maximal	83.7 dBc

Mouvements et positions

MPJS	Nombre	Index
MPJS	0	0 /h
Mouvements de jambe	20	2.9 /h

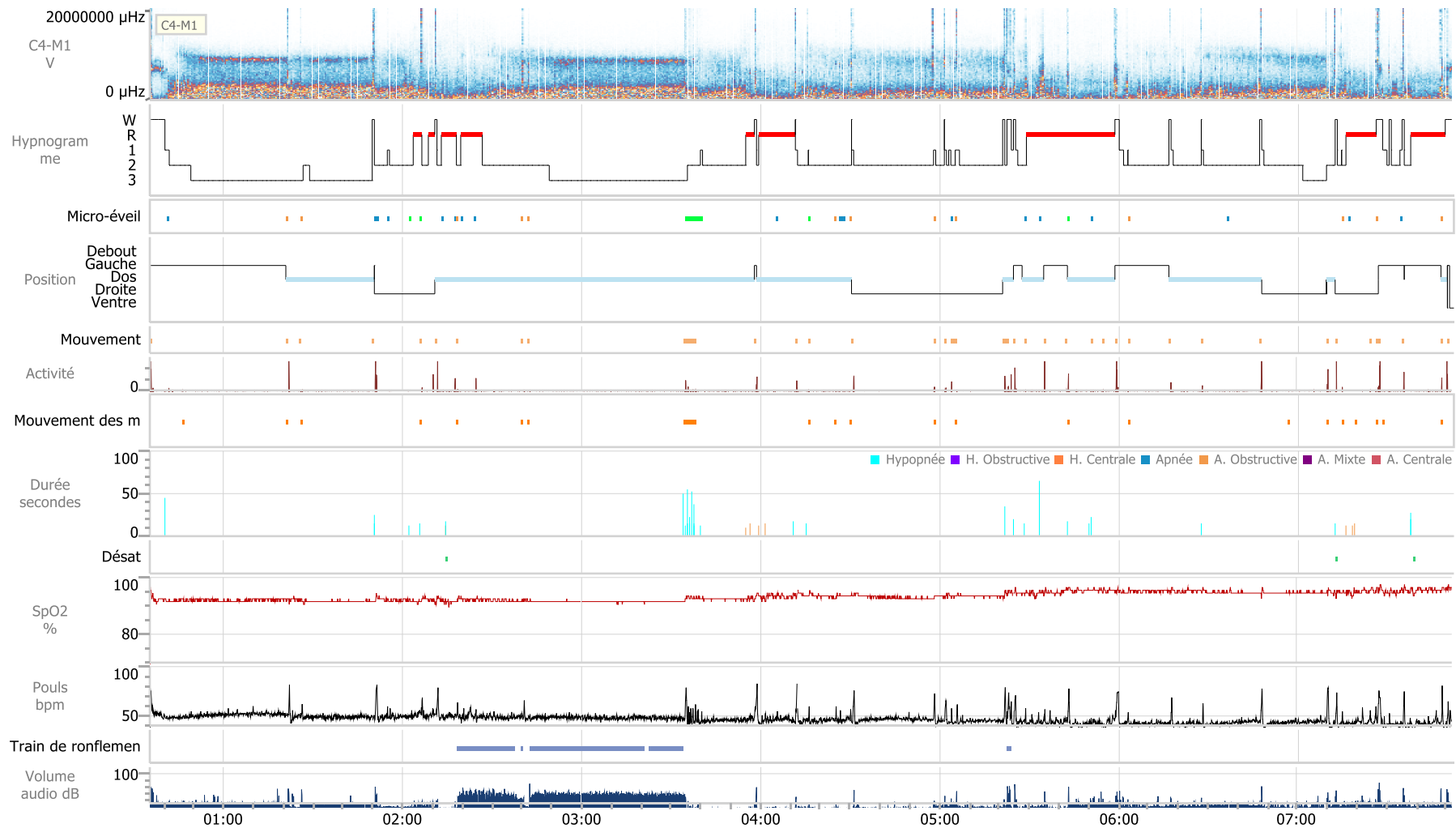
Position et activité

Dorsale	224.2 min	53.8 %
Non-dorsale	192.3 min	46.2 %
Debout	0 min	0 %
Mouvements	4 min	1 %
Données invalides	0 min	0 %

Graphique circulaire des positions :

- % Dos 52,9
- % Ventre 0,3
- % Droite 24,6
- % Gauche 22,2
- % Debous 0,0

Résumé graphique de la nuit



ATHLÈTE

Informations



NOM :

Prénom :

Âge :

Taille :

Masse :

Née le :

Fédération :

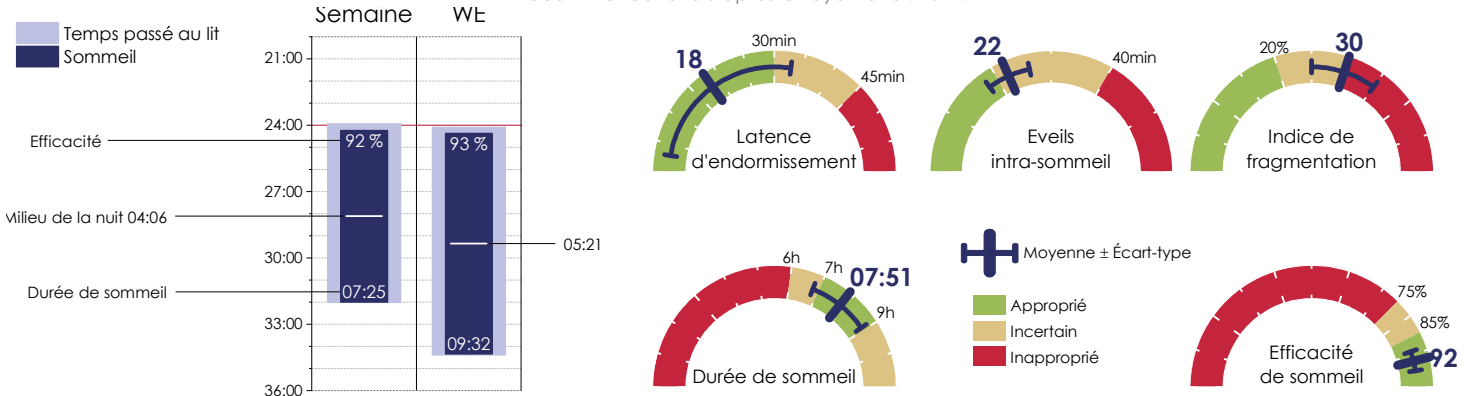
Sport :

Date :

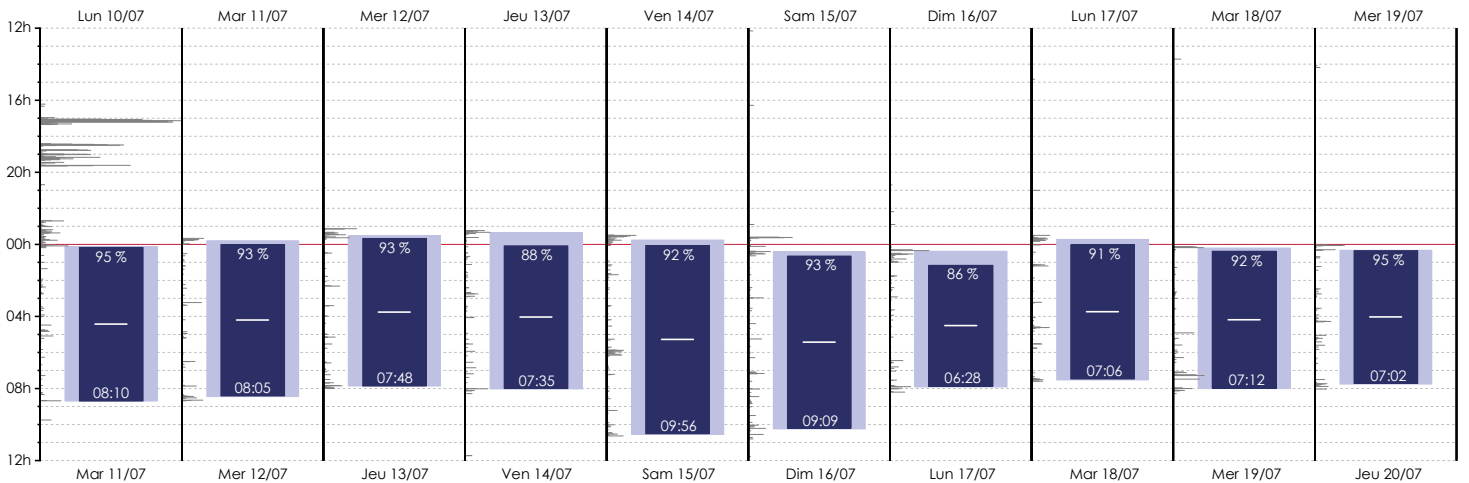
RÉSULTATS

Éléments clés

Données analysées sur 10 nuits, exprimées en moyenne \pm écart type. Seuil d'analyse fixé à 80 (sensibilité faible).
Recommandations d'après Ohayon et al. 2017.

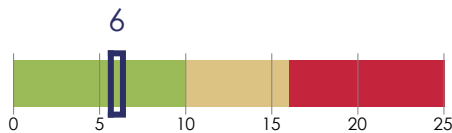


Durée et efficacité du sommeil

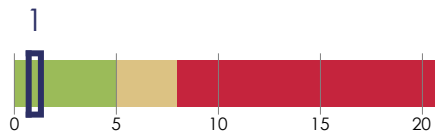


Auto-évaluation du sommeil

Somnolence diurne
(Epworth, d'après Johns 1991)

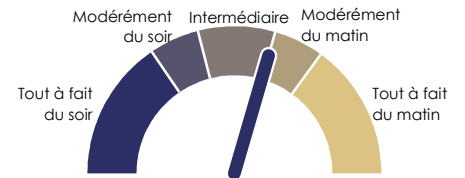


Qualité de sommeil
(PSQI, d'après Buysse et al. 1989)



Chronotype

(d'après Horne et al. 1976)



Commentaires

ATHLETE

Informations



NOM :

Taille :

Fédération :

Prénom :

Masse :

Discipline :

Âge :

Né le :

Période :

RESULTATS

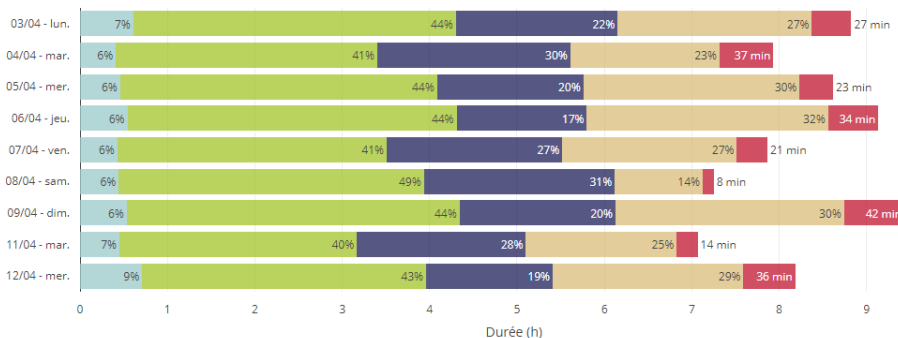
Eléments clés

Données analysées sur 9 nuits, exprimées en moyenne ± écart type. Recommandations d'après Ohayon et al. 2017.

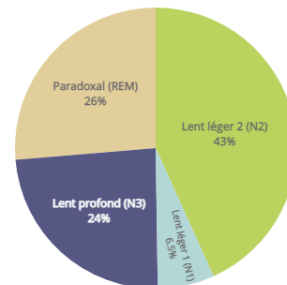
Durée de sommeil	Latence d'endormissement <i>min</i>	Eveils intra-sommeil <i>min</i>	Efficacité de sommeil <i>%</i>	Période(s) d'éveil > 5min <i>n</i>	Proportion scorable <i>%</i>
07h48 ± 00h41	18 ± 8	27 ± 11	90 ± 3	1 ± 1	99 ± 2

Stades de sommeil (% de la durée de sommeil)

■ Eveil ■ Paradoxal (REM) ■ Lent profond (N3) ■ Lent léger 2 (N2) ■ Lent léger 1 (N1)

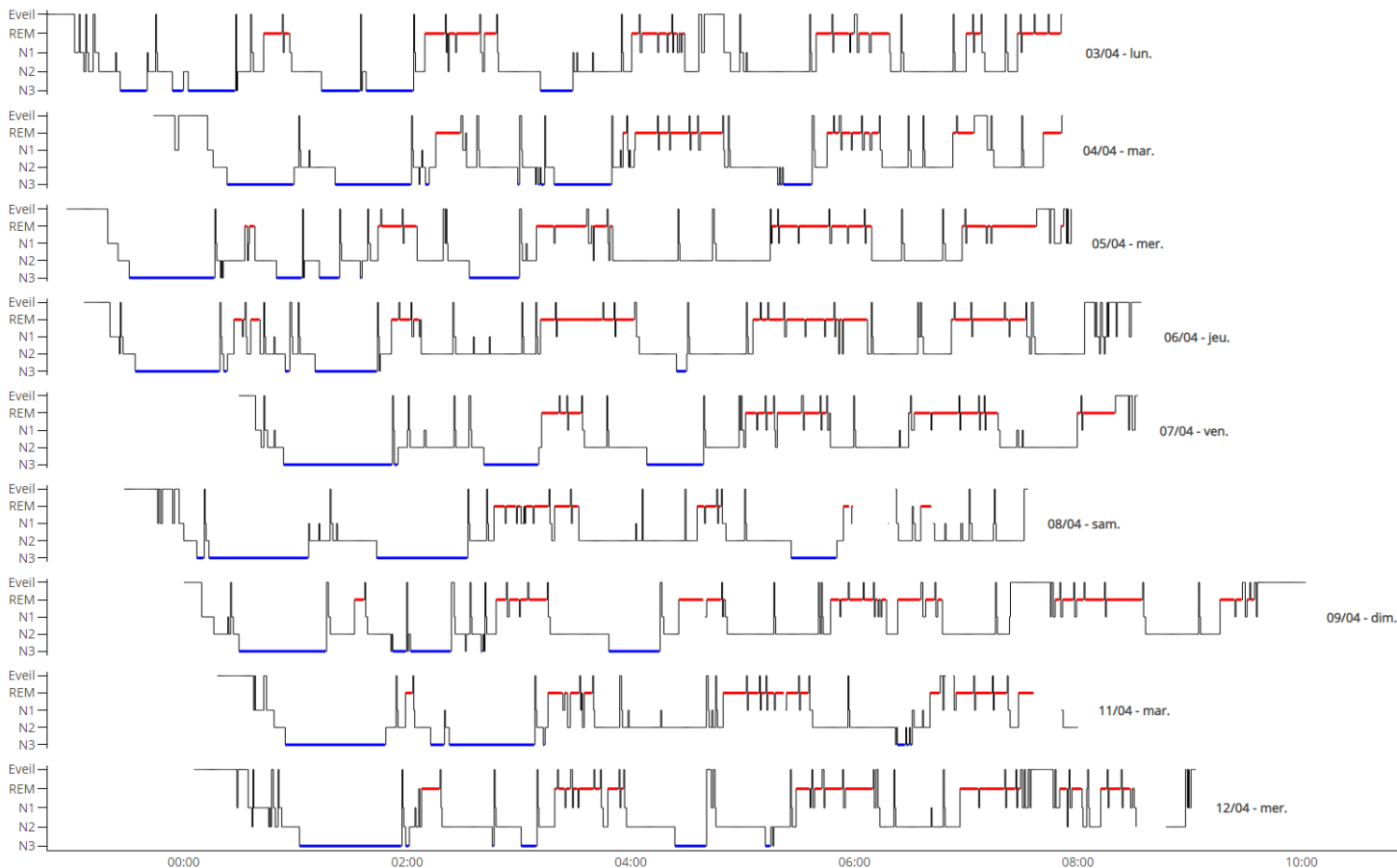


Proportion moyenne des stades de sommeil



Commentaires

Hypnogrammes



Résumé substantiel en français

Le sommeil est essentiel à la santé, au bien-être et à la performance physique et cognitive. Il joue un rôle crucial dans les processus de récupération (Halson and Juliff, 2017; O'Donnell et al., 2018). A cet égard, les sportifs de haut niveau et leur encadrement prennent de plus en plus conscience de son importance dans le processus d'entraînement (Venter, 2014; Lastella et al., 2020). Néanmoins, de nombreux travaux de recherche rapportent une prévalence élevée de plaintes du sommeil et d'un sommeil inadéquat dans cette population (Gupta et al., 2017; Roberts et al., 2019). La pratique du sport au plus haut niveau pose un défi important au sommeil due aux voyages fréquents associés à des environnements parfois non familiers, à des horaires d'entraînement/compétition variés, une anxiété liée à la compétition, combinés aux obligations sociales et de la vie personnelle (e.g., famille, travail, études) (Walsh et al., 2020). Parmi ces facteurs, la charge d'entraînement élevée imposée pour se préparer aux plus grandes compétitions est un potentiel perturbateur du sommeil supplémentaire encore mal compris.

La plupart des études qui ont caractérisé le sommeil des sportifs en réponse à de fortes charges d'entraînement ont décrit un sommeil plus agité, accompagné d'une réduction de la quantité et de la qualité du sommeil (Roberts et al., 2019). Néanmoins, la littérature est limitée principalement à des études évaluant le sommeil en utilisant des méthodes déclaratives et/ou par actimétrie. Ces méthodes ne décrivent pas les caractéristiques neurophysiologiques et la structure interne (architecturale) du sommeil, que l'on obtient à partir d'analyses électroencéphalographiques (EEG), telles que la polysomnographie (McCarter et al., 2022). Bien que ces analyses soient plus complexes à réaliser en situation réelle, elles contiennent des informations cruciales sur la qualité du sommeil. De plus, une forte charge d'entraînement entraîne des changements physiologiques, psychologiques et bouscule l'organisation et la vie autour de l'entraînement (e.g., augmentation du nombre et de la durée des entraînements), pouvant également affecter le sommeil. Leur implication dans les perturbations du sommeil suite à une charge d'entraînement élevée est peu connue. Ainsi, investiguer l'impact de la charge d'entraînement sur le sommeil, notamment son architecture, dans un contexte spécifique d'entraînement permettrait de mieux comprendre comment d'autres facteurs confondants sont impliqués. Les résultats pourraient fournir des informations cruciales aux entraîneurs dans la gestion de la charge et des programmes d'entraînement au profit du sommeil, du bien-être et de la performance des sportifs.

De plus, au regard de l'importance d'une quantité et qualité de sommeil adéquate, il est primordial d'investiguer des stratégies pratiques et non-médicamenteuses permettant de limiter les facteurs de perturbation du sommeil liés à la pratique du sport à haut niveau. En raison de la relation étroite et réciproque entre le sommeil et les systèmes de régulation de la température corporelle (Van Someren, 2006; Krauchi and Deboer, 2010; Harding et al., 2019), les propriétés thermiques de la literie ont été identifiées comme un facteur clé de la qualité du sommeil (Troynikov et al., 2018). Dans ce contexte, des travaux récents ont étudié les bénéfices d'un système de literie à haute conductivité thermique sur le sommeil de volontaires sains (Kräuchi et al., 2018; Herberger et al., 2020, 2024) ou de joueurs de rugby de haut niveau (Aloulou et al., 2020). Ces études ont démontré que dormir sur cette literie, composée d'un matelas en mousse recouvert d'une plaque de gel, facilite la perte de chaleur corporelle pendant le sommeil. Cette meilleure dissipation de la chaleur a été associée à une amélioration de la qualité du sommeil et du confort subjectif du sommeil (Kräuchi et al., 2018; Aloulou et al., 2020; Herberger et al., 2020). Néanmoins, ces études ont été menées pendant une seule nuit, et l'efficacité de cette stratégie lors d'une utilisation chronique dans un contexte d'entraînement est actuellement inconnue. Rien ne prouve non plus que cette stratégie puisse atténuer l'effet potentiellement néfaste d'une charge d'entraînement élevée sur le sommeil.

Enfin, l'exposition accrue à des températures élevées en raison du changement climatique est un problème actuel à la fois pour les sportifs de haut niveau et la population en général (Patz et al., 2014; Watts et al., 2018). La quantité et la qualité du sommeil peuvent être mis à rude épreuve dans ces conditions (Lan et al., 2017; Minor et al., 2022). Ainsi, compte tenu des caractéristiques thermiques d'une literie à haute conductivité thermique, il est nécessaire d'investiguer son efficacité chez des sportifs exposés à des températures élevées, afin de préserver leur quantité et qualité de sommeil. Décrire les réponses individuelles à cette stratégie dans ce contexte est également nécessaire pour parvenir à des orientations plus précises dans la prescription d'interventions individualisées.

L'objectif de ce travail de thèse est d'approfondir la compréhension de l'impact de la charge d'entraînement sur le sommeil et d'explorer l'efficacité d'une literie à haute conductivité thermique sur le sommeil des sportifs dans un contexte d'entraînement habituel. Nous nous sommes concentrés à évaluer l'architecture du sommeil et à utiliser des méthodes objectives et déclarative pour appréhender les concepts de quantité et de qualité du sommeil. La partie

expérimentale de ce travail s'articule autour de trois études menées *in-situ*, dans les contextes d'entraînement des sportifs, en utilisant des designs expérimentaux descriptifs, contrôlés et interventionnels.

La première étude de ce travail de thèse avait pour objectif d'évaluer le sommeil de jeunes joueurs de rugby à XV de haut niveau pendant un stage d'entraînement de 10 jours, comprenant deux matchs amicaux, en tenant compte de l'influence potentielle de la charge d'entraînement quotidienne et de l'heure de rendez-vous du matin sur le sommeil. Les rythmes veille-sommeil ont été évalués quotidiennement par actimétrie et agenda de sommeil tandis que l'architecture du sommeil a été analysée à l'aide d'un dispositif d'électroencéphalographie à montage réduit. La charge d'entraînement externe a été mesurée via des récepteurs GPS comprenant une centrale inertielle miniaturisée et la charge interne a été évaluée en multipliant la perception de l'effort sur l'échelle CR-10 (RPE) par la durée de l'entraînement. La perception de la fatigue, de la douleur et du stress a été recueillie à partir d'échelles visuelles analogiques chaque matin au réveil. La prévalence de nuits avec une durée de sommeil <7 h (30.3%), une durée d'éveil intra-sommeil >40 min (77.8%) et une efficacité du sommeil <85 % (43.4%) était élevée. Des modèles linéaires mixtes ont révélé que pour chaque 100m supplémentaires parcourue à haute vitesse >18 km.h⁻¹, la durée de sommeil était augmentée ($\beta = +4.9$ minutes, $p < 0.05$) et le nombre de changements de stade de sommeil était réduit ($\beta = -1.1$, $p < 0.05$). Un coucher plus tardif augmentait la fatigue perçue le matin ($p < 0.05$). La durée de sommeil la plus courte (06h52 ± 00h34) était le jour du Match 1, lorsque le rendez-vous du matin était le plus tôt, c'est-à-dire de 7h30 à 8h30. La durée de sommeil (+17.2 min, $p = 0.01$) et l'efficacité de sommeil (+2.1%, $p < 0,01$) étaient les plus favorables lorsque le rendez-vous du matin était programmé à 8h00–9h00 par rapport à 8h30–9h30. Cette étude confirme qu'un stage d'entraînement est une période vulnérable pour le sommeil, mais une charge d'entraînement contrôlée et non excessive n'a pas contribué à perturber le sommeil. Les entraîneurs doivent être conscients de l'impact négatif d'un rendez-vous du matin plus tôt et/ou inhabituel sur le sommeil, surtout à proximité d'un match. Une heure de rendez-vous régulière le matin devrait favoriser la quantité et la qualité du sommeil des joueurs et prévenir la fatigue subjective. Il pourrait être également utile de mettre en place un programme d'éducation au sommeil à l'intention des joueurs, afin de favoriser la quantité et la qualité du sommeil au cours de cette période où le sommeil est compromis.

La deuxième étude de ce travail de thèse visait à examiner l'impact d'un plan d'entraînement sur le sommeil chez des coureurs à pied, et l'efficacité d'un surmatelas à haute conductivité thermique (HMT). Pour cela, 21 coureurs à pied bien entraînés en endurance ont réalisé 2 semaines d'entraînement comprenant une charge d'entraînement habituel, en dormant sur un surmatelas à faible capacité thermique (LMT), suivies d'une période de surcharge (augmentation de 40% du volume d'entraînement) puis d'affûtage (diminution de 50% du volume d'entraînement) de 2 semaines, respectivement. A partir de la période de surcharge et jusqu'à la fin du protocole, ils ont été répartis en deux groupes en fonction du surmatelas utilisé : HMT (n = 11) ou LMT (n = 10). Quotidiennement, la charge d'entraînement a été évaluée en multipliant la perception de l'effort sur l'échelle CR-10 (RPE) par la durée de l'entraînement, et le sommeil a été analysé par actimétrie et agenda de sommeil. A l'issue de chaque période d'entraînement, ils ont répondu aux questionnaires RESTQ-Sport-36 et DALDA, et ont réalisé un test de performance incrémental sur tapis de course. Ils ont également dormi une nuit à leur domicile équipé d'une polysomnographie ambulatoire pour évaluer l'architecture du sommeil. La température corporelle centrale a été enregistrée en continu pendant cette même nuit. Les résultats indiquent qu'indépendamment du groupe, la période de surcharge a augmenté les scores *stress général* et *symptômes de réaction au stress* des questionnaires, sans déclin de la performance lors du test incrémental. La majorité des participants a été diagnostiquée comme étant "non-surmenée" (n = 14). Les rythmes veille-sommeil durant les périodes d'entraînement, évalués par actimétrie, n'ont pas été modifiés par la surcharge et la période d'affûtage, en comparaison de la période d'entraînement habituel. Néanmoins, suite à la surcharge, le nombre d'épisodes d'éveil >5 min, évalué par polysomnographie, a diminué par rapport à la période d'entraînement habituel (-0.48, p = 0.05). Afin de prendre en compte la variabilité inter-individuelle de la charge d'entraînement réalisée, des modèles linéaires mixtes ont été utilisés. Au quotidien, chaque augmentation de 100 unités arbitraires de la charge d'entraînement était associée, la nuit suivante, à une détérioration de l'efficacité du sommeil ($\beta = -0.2\%$; p < 0.01), de la durée d'éveil intra-sommeil ($\beta = +0,4$ min ; p < 0,05) et de la latence d'endormissement ($\beta = +0.5$ min ; p < 0,05) évaluées par actimétrie. En décomposant ces relations par période d'entraînement, la relation entre la charge d'entraînement, l'efficacité du sommeil et la durée d'éveil intra-sommeil était significative uniquement pendant la période de surcharge. De manière hebdomadaire, chaque augmentation de 100 unités arbitraires de la charge d'entraînement entraînait une proportion de sommeil lent profond (N3) plus élevée ($\beta = +0.13\%$, p = 0.05), une latence d'endormissement plus courte ($\beta = -0.49$ min, p = 0.05), et une

réduction de la probabilité de transition du sommeil lent léger 1 (N1) vers l'éveil ($\beta = -0.12\%$, $p = 0.05$). Malgré une plus faible température corporelle centrale moyenne au cours de la nuit en utilisant HMT par rapport à LMT (-0.025°C , $p = 0.02$), son utilisation n'a impacté aucune variable de sommeil de manière significative. En conclusion, une charge d'entraînement plus élevée et inhabituelle la journée peut engendrer un sommeil plus agité la nuit suivante, notamment lorsque la charge est exceptionnellement élevée (surcharge). Les entraîneurs devraient accorder une attention particulière au sommeil en privilégiant une approche individualisée, tenant compte du niveau de fatigue et de la réponse à l'entraînement. Nos résultats ont également souligné que l'accumulation de la charge d'entraînement favorise la propension, la profondeur et la stabilité de l'architecture du sommeil des sportifs, soulignant la nature adaptative du sommeil à l'activité diurne et le rôle du sommeil dans la récupération physiologique. De futures études devraient être menées pour explorer les bénéfices potentiels du surmatelas HMT chez des sportifs présentant des problématiques de sommeil et/ou exposés à des conditions peu propices à un sommeil de qualité.

Enfin, **la troisième étude de ce travail de thèse** avait pour objectif d'évaluer l'efficacité d'un surmatelas à haute conductivité thermique (HMT) sur la quantité et la qualité du sommeil chez des joueurs de badminton de haut niveau pendant une période d'entraînement estivale. Quatorze joueurs de badminton de niveau mondial ont été répartis dans deux conditions dans un ordre aléatoire, en simple aveugle et de manière équilibrée : dormir une semaine sur HMT et une autre semaine sur un surmatelas à faible capacité thermique (LMT). Le sommeil a été évalué quotidiennement par actimétrie et agenda du sommeil, et la perception du confort et de la sensation thermique pendant la nuit a été recueillie au réveil. La température nocturne de la chambre a été enregistrée en continu chaque nuit et la température corporelle centrale a été évaluée en continu pendant deux nuits consécutives dans chaque condition. Chez 7 joueurs, une polysomnographie ambulatoire a été réalisée pendant une nuit dans chaque condition pour évaluer l'architecture du sommeil. Le protocole a été réalisé entre juillet et septembre et la température moyenne nocturne de la chambre variait de 22.4 à 30.7°C (HMT : $26.0 \pm 1.7^\circ\text{C}$, LMT : $26.7 \pm 1.6^\circ\text{C}$, $p = 0.32$). Malgré une réduction significative de la température corporelle centrale moyenne au cours de la nuit (HMT vs LMT : -0.037°C , $p < 0.001$), dormir sur HMT n'a pas eu d'effet significatif sur le sommeil de l'ensemble des joueurs, ni sur la perception de la qualité du sommeil, le confort et la sensation thermique ($p > 0.05$) par rapport à LMT. Une approche d'inférence basée sur l'ampleur de l'effet (magnitude-based inference) a révélé une

augmentation très probable de l'efficacité du sommeil (ES = 1.49 [IC à 90 % : 0.67 à 2.31]) et une diminution presque certaine de la durée d'éveil intra-sommeil (ES = -2.04 [-2.60 à -1.47]) en utilisant HMT par rapport à LMT, uniquement chez les joueurs ayant un sommeil plus agité. Cette étude a mis en évidence que HMT n'a pas influencé le sommeil des joueurs de badminton de haut niveau pendant une période d'entraînement estivale. Cependant, ce travail fournit des perspectives sur des bénéfices potentiellement plus importants de cette stratégie chez les sportifs ayant un sommeil plus agité. Cela encourage la poursuite des recherches sur l'efficacité d'une literie à haute conductivité thermique avec une approche individualisée.

Pour conclure, l'originalité de ce travail de thèse réside dans l'évaluation de l'architecture du sommeil à partir d'analyses électroencéphalographiques, telles que la polysomnographie, dans un contexte spécifique d'entraînement. Nos résultats ont montré que la charge d'entraînement influençait positivement la qualité du sommeil, favorisant la propension au sommeil et un sommeil plus profond et plus continu. Néanmoins, une charge d'entraînement excessive (surcharge) peut engendrer un sommeil plus agité, potentiellement dû à des changements physiologiques, à une charge mentale accrue, associés à une modification de l'organisation et de la vie autour de l'entraînement. Ces résultats soulignent l'importance d'accorder une attention particulière au sommeil et d'une gestion précise de la charge d'entraînement, notamment pendant les périodes de surcharge, afin d'assurer une récupération optimale, des adaptations à long terme de l'entraînement et une amélioration des performances. De plus, nous démontrons que la quantité de sommeil des athlètes est affectée par les horaires matinaux lors des camps d'entraînement, beaucoup plus que par la charge d'entraînement. Lors de la conception d'un programme d'entraînement, nous suggérons de considérer l'organisation de l'entraînement et de la vie autour de l'entraînement, en donnant la priorité à un sommeil régulier afin de promouvoir la quantité et la qualité du sommeil.

L'utilisation chronique d'un surmatelas à haute conductivité thermique par des sportifs exposés à une forte charge d'entraînement ou au cours d'une période d'entraînement estivale n'a pas eu d'impact significatif sur le sommeil. Cela est probablement dû à un effet faible sur la perte de la chaleur corporelle pendant la nuit. Ce travail fournit toutefois des perspectives quant à de potentiels bénéfices chez les athlètes connaissant un sommeil plus agité. Cela encourage la poursuite de recherches sur l'efficacité d'une stratégie favorisant la perte de chaleur corporelle au cours de la nuit literie avec une approche individualisée.

Sleep in elite athletes: from the impact of training load to bedding optimization for the Paris 2024 Olympic Games

Abstract: Sleep is essential for the health, well-being, and performance of athletes. However, numerous studies report a high prevalence of inadequate sleep among elite athletes. The high training load imposed to prepare for major competitions is a potential disruptor of sleep in elite athletes that is not yet fully understood. In this context, it is essential to develop strategies to mitigate sleep disturbances in athletes. The aim of this PhD thesis was to deepen our understanding of the impact of training load on athletes' sleep and to explore the effectiveness of a high-heat-capacity mattress topper (HMT), a strategy aimed at promoting body heat loss during sleep, on athletes' sleep within typical training contexts. The first study aimed to assess the sleep of young elite rugby union players during a training camp. In this context, in which training load was controlled and not excessive, the distance covered at high-speed promoted sleep duration and the continuity of sleep architecture. Sleep duration and efficiency were sensitive to the morning appointment time, with a negative impact when it was earlier than usual. The second study aimed to examine the impact of a training program consisting of a usual training period, overload, and tapering, on the sleep of well-trained runners. The effectiveness of HMT on sleep was assessed during this training period. An increase in weekly internal training load improved the proportion of slow-wave sleep, sleep onset latency, and the continuity of sleep architecture. A more restless sleep was observed following an increased daily internal training load, particularly during the overload period. The use of HMT did not significantly affect sleep-wake patterns and sleep architecture compared to control bedding. Finally, the third study of this PhD thesis examined the effectiveness of HMT on the sleep of elite badminton players during a summer training period. Sleeping on HMT did not affect sleep-wake patterns and sleep architecture compared to control bedding. However, a most likely increase in sleep efficiency and an almost certainly decrease in wake after sleep onset were observed using HMT compared to control bedding, only in players with more restless sleep. In conclusion, our results highlight the adaptive nature of sleep architecture in response to recovery needs associated with increased training load. We recommend allocating particular attention to sleep when exposed to excessive and unusual training loads, considering the time and organizational constraints associated with this congested period. Further research on the effectiveness of HMT on athletes' sleep with an individualized approach is warranted.

Keywords: recovery, performance, polysomnography, sleep architecture, thermoregulation, sleep environment, training periodization, fatigue

Le sommeil du sportif de haut niveau : de l'impact de la charge d'entraînement à l'optimisation de la literie en vue des Jeux Olympiques de Paris 2024

Résumé : Le sommeil est essentiel à la santé, au bien-être et à la performance des sportifs. Néanmoins, de nombreuses études font état d'une prévalence élevée de sommeil inadéquat chez les sportifs de haut niveau. La charge d'entraînement élevée imposée pour se préparer aux grandes compétitions est un perturbateur potentiel du sommeil chez le sportif de haut niveau encore mal compris. Dans ce contexte, il est indispensable d'élaborer des stratégies contribuant à limiter les perturbations du sommeil chez le sportif. L'objectif de cette thèse de doctorat était d'approfondir l'impact de la charge d'entraînement sur le sommeil des sportifs et d'explorer l'efficacité d'un surmatelas à haute conductivité thermique (HMT), une stratégie visant à favoriser la perte de chaleur corporelle pendant le sommeil. La première étude avait pour objectif d'évaluer le sommeil de jeunes joueurs de rugby à XV de haut niveau pendant un stage d'entraînement. Dans ce contexte où la charge était contrôlée et non excessive, la distance parcourue à haute intensité a favorisé la durée de sommeil et la continuité de son architecture. La durée et l'efficacité du sommeil étaient sensibles à l'heure de rendez-vous du matin, avec un impact négatif lorsque celui-ci était plus tôt que d'habitude. La deuxième étude visait à examiner l'impact d'un programme d'entraînement comprenant une période habituelle d'entraînement, une surcharge et un affûtage, sur le sommeil de coureurs à pied bien entraînés. L'efficacité de HMT sur le sommeil a été évaluée pendant cette période d'entraînement. Une augmentation de la charge d'entraînement interne hebdomadaire a amélioré la proportion du sommeil lent profond, la latence d'endormissement et la continuité de l'architecture du sommeil. Un sommeil plus agité a été observé suite à une charge d'entraînement interne journalière plus élevée, particulièrement pendant la période de surcharge. L'utilisation de HMT n'a pas eu d'effet significatif sur les rythmes veille-sommeil et l'architecture du sommeil par rapport à une literie contrôlée. Enfin, la troisième étude de cette thèse a examiné l'efficacité de HMT sur le sommeil de joueurs de badminton de haut niveau pendant une période d'entraînement estivale. Dormir sur HMT n'a pas impacté les rythmes veille-sommeil et l'architecture du sommeil par rapport à une literie contrôlée. Cependant, une augmentation très probable de l'efficacité du sommeil et une diminution presque certaine de la durée d'éveil intra-sommeil ont été observées en utilisant HMT par rapport à une literie contrôlée, uniquement chez les joueurs ayant un sommeil plus agité. Pour conclure, nos résultats soulignent la nature adaptative de l'architecture du sommeil face aux besoins de récupération liés à une charge d'entraînement accrue. Nous recommandons de prêter une attention particulière au sommeil lorsque la charge est excessive et inhabituelle, en tenant compte des contraintes temporelles et organisationnelles associées à cette période. Il serait opportun de poursuivre les recherches sur l'efficacité de HMT sur le sommeil du sportif avec une approche individualisée.

Mots clés : récupération, performance, fatigue, polysomnographie, architecture du sommeil, thermorégulation, environnement de sommeil, périodisation de l'entraînement