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Sports Medicine

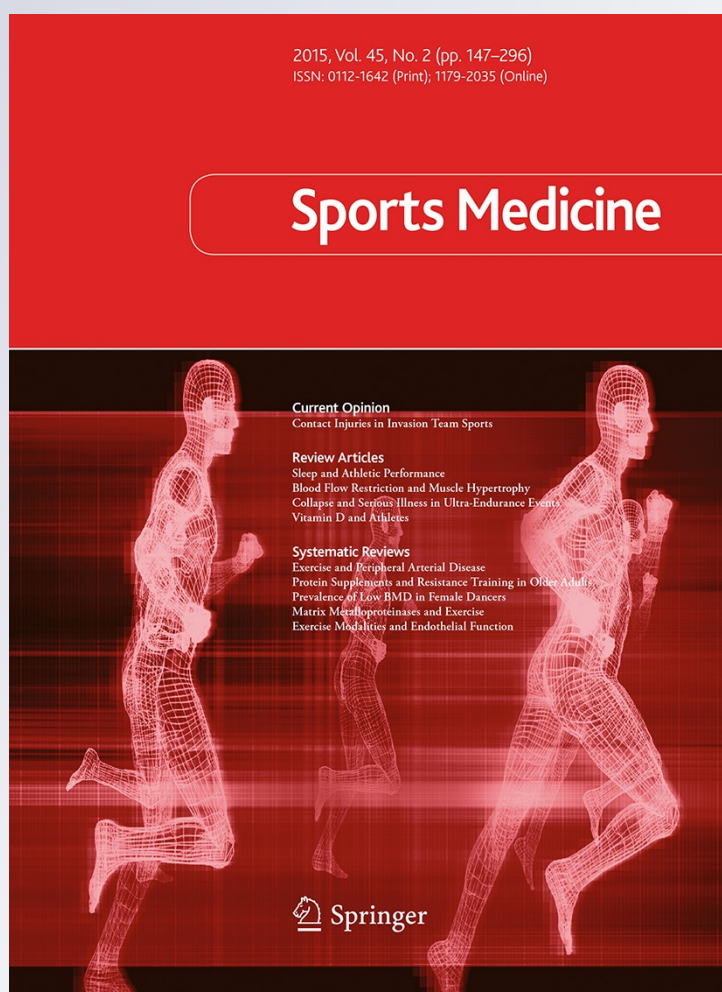
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Sleep and Athletic Performance: The Effects of Sleep Loss on Exercise Performance, and Physiological and Cognitive Responses to Exercise

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Abstract Although its true function remains unclear, sleep is considered critical to human physiological and cognitive function. Equally, since sleep loss is a common occurrence prior to competition in athletes, this could significantly impact upon their athletic performance. Much of the previous research has reported that exercise performance is negatively affected following sleep loss; however, conflicting findings mean that the extent, influence, and mechanisms of sleep loss affecting exercise performance remain uncertain. For instance, research indicates some maximal physical efforts and gross motor performances can be maintained. In comparison, the few published studies investigating the effect of sleep loss on performance in athletes report a reduction in sport-specific performance. The effects of sleep loss on physiological responses to exercise also remain equivocal; however, it appears a reduction in sleep quality and quantity could result in an autonomic nervous system imbalance, simulating symptoms of the overtraining syndrome. Additionally, increases in pro-inflammatory cytokines following

sleep loss could promote immune system dysfunction. Of further concern, numerous studies investigating the effects of sleep loss on cognitive function report slower and less accurate cognitive performance. Based on this context, this review aims to evaluate the importance and prevalence of sleep in athletes and summarises the effects of sleep loss (restriction and deprivation) on exercise performance, and physiological and cognitive responses to exercise. Given the equivocal understanding of sleep and athletic performance outcomes, further research and consideration is required to obtain a greater knowledge of the interaction between sleep and performance.

Key Points

Although sleep is considered critical to optimal performance, many athletes appear to lose sleep prior to competition for various reasons, including noise, light, anxiety, and nervousness.

Whilst there appears sufficient evidence to imply complete sleep deprivation can have significant negative effects on athletic performance, the effects of sleep restriction (partial disturbance of the sleep–wake cycle) are more conflicting; a concerning issue given that athletes are more likely to experience this mode of sleep loss.

The detrimental effect of sleep loss on most aspects of cognitive function remains unequivocal, with only minor conflicting findings present for the extent of the effects of mild sleep restriction, findings that would predictably suggest negative consequences for athletes requiring high neurocognitive reliance.

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1 Introduction

Reoccurring at habitual intervals throughout a 24-h period in humans, sleep is a homeostatically controlled behavioral state of reduced movement and sensory responsiveness [1, 2]. The process of sleep is widely regarded as critical to both cognitive and physiological function [2–7]. In spite of this perceived importance, the consensus regarding the rationale as to why humans sleep remains equivocal, if not robustly debated [2, 8]. Recent studies have shown sleep to regulate key molecular mechanisms (i.e. transcriptional regulatory proteins [1, 9, 10]), and have demonstrated that sleep has an integral role in metabolic homeostasis [11]. Whilst the duration and quality of sleep is manipulated by numerous environmental factors, among them light [12], jetlag [13], and nutrition [14], it has also been shown to be influenced by genetic traits [15, 16]. Notwithstanding the complexity surrounding the need, rationale, and outcome of sleep, it seemingly must serve an important purpose for humans because it has survived so many years of evolution [15].

The ability of humans to cope with physiological and psychological stressors is critical to athletic performance outcomes [17], and is affected by numerous factors, including experience, fitness, motivation, and the natural fluctuation of physiological and behavioral processes across a 24-h period (i.e. sleep–wake cycle, body temperature, hormone regulation [18]). These *circadian rhythms* are primarily controlled by the suprachiasmatic nucleus (SN) within the hypothalamus [2]. However, the SN is unable to always maintain control over these patterns, as humans are highly sensitive to alterations to their natural environment [2, 19], most notably through the light–dark cycle [20]. When athletes encounter disruptions to their environments (e.g. through travel or training/playing at night), endogenous circadian rhythms and normal sleep–wake cycles can become desynchronised [2, 21]. Such perturbations in sleeping patterns can cause an increase in homeostatic pressure and affect emotional regulation, core temperature, and circulating levels of melatonin, causing a delay in sleep onset [22]. Following these periods, there is potential for sleep loss and neurocognitive and physiological performance to be compromised [7, 14, 23, 24]. Thus, since sleep disruption prior to important events is commonly found in elite athletes [25–27], there are numerous instances where the subsequent performance could be compromised [25, 28, 29].

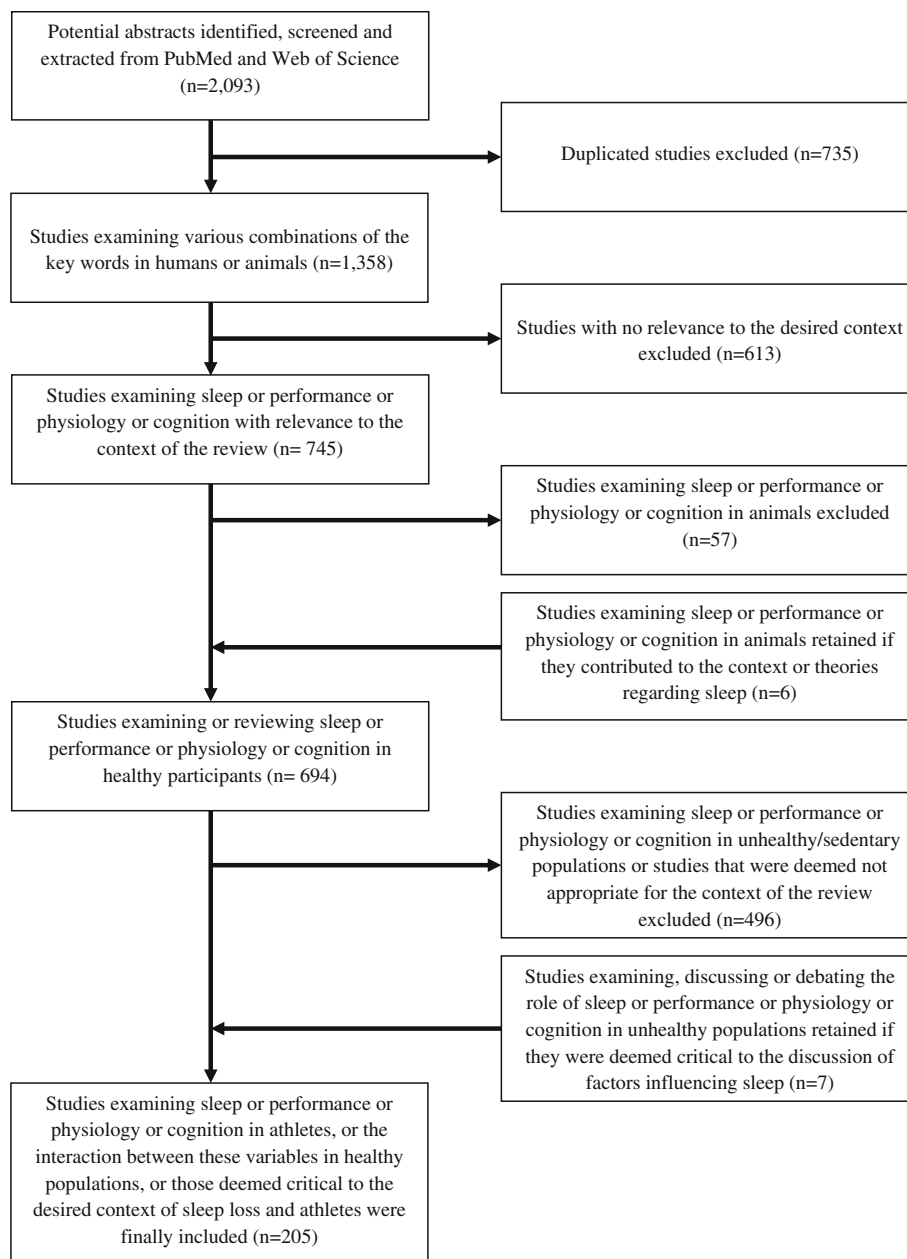
However, due to the complexity of sleep function, the limited availability of athletes to participate in sleep studies, and the variability in the individual requirement for sleep [21, 30], the effects of sleep loss on athletic performance are poorly understood. Furthermore, the increase in

recent literature since past reviews [21, 31, 32] highlights a need to re-evaluate the effects of sleep loss on athletic performance, particularly allowing for a greater focus on sport-specific outcomes. Accordingly, the overall purpose of this review is to examine the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise. As a result, we review the current literature on the theoretical components of sleep and importance for athletes, the quality and quantity of ‘normal’ sleep compared with that of athletes, and the effects of sleep loss on exercise performance and physiological and cognitive responses (including mood) to exercise. In order to accomplish this critical review, a computerized literature search (Fig. 1) was performed over 7 months (August 2013–March 2014) on PubMed and Web of Science for articles within the period January 1960–March 2014. Keywords used in different combinations were ‘sleep’, ‘deprivation’, ‘loss’, ‘restriction’, ‘team’, ‘exercise’, ‘cognition’, ‘physiological’, ‘sport’, ‘athlete’, ‘player’, and ‘performance’. In addition, articles were sourced manually from the reference lists of original manuscripts, and previous critical, systematic, and meta-analytical reviews. The previous work within this field, and the multi-dimensional components of sleep and their role in athletic performance, are duly recognised. Notwithstanding these critical components, their roles are too extensive to be discussed here. The reader is advised to consult previous work regarding the effects of nutrition [14], jetlag [13, 33, 34], and Ramadan [35] on sleep for further detail.

2 The Theoretical Components of Sleep and their Importance for Athletes

A recent review by Frank and Benington [8] identified several theories of the function of sleep, including (1) the restorative effects on the immune and the endocrine systems, (2) a neurometabolic theory suggesting that sleep assists in the recovery of the nervous and metabolic cost imposed by the waking state, and (3) cognitive development, supposing that sleep has a vital role in learning, memory, and synaptic plasticity. An interaction between these theories is likely to contribute to the construct of several stages during sleep [8]. These respective stages not only differ in depth, but also in the frequency and intensity of dreaming, eye movements, muscle tone, regional brain activation, and communication between memory systems [36]. A typical night's sleep is composed of approximately 90-min cycles divided into periods of rapid-eye-movement sleep (REM; associated with dreams), and non-REM sleep (NREM) [37]. NREM sleep is further divided into four different stages (Fig. 2). All stages are classified according

Fig. 1 Flow diagram and results of the literature search to address the aim of the article to evaluate the importance and prevalence of sleep in athletes and review the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise



to parameters such as electrical brain activity, blood pressure, and eye movement [38, 39].

Specifically, the role for NREM sleep is proposed to assist with energy conservation and nervous system recuperation. For example, it has been shown that growth hormone (GH; fundamental to tissue regeneration and growth) is released [40] and oxygen consumption is lowered [41] during phases of NREM sleep. Moreover, NREM sleep seems to be a stimulus for anabolic hormones that increase the synthesis of protein and mobilize free fatty acids to provide energy, thereby preventing amino acid catabolism [42]. Such processes would seem particularly pertinent for athletic populations requiring accelerated

rates of healing to repair peripheral muscular damage [43]. Comparatively, theories of REM sleep have suggested a role for this state in periodic brain activation, localized recuperative processes, and emotional regulation [44]. Especially in the early stages of mammalian life, REM sleep is assumed to be critical in establishing brain connections [44], since neuronal activity in REM sleep is similar to that of waking [45]. Hence, sleep can be defined as an actively regulated process rather than a passive result of diminished waking, and can be seen as a reorganization of neuronal activity [45].

The importance of sleep in athletes has also been discussed in regards to memory consolidation, especially

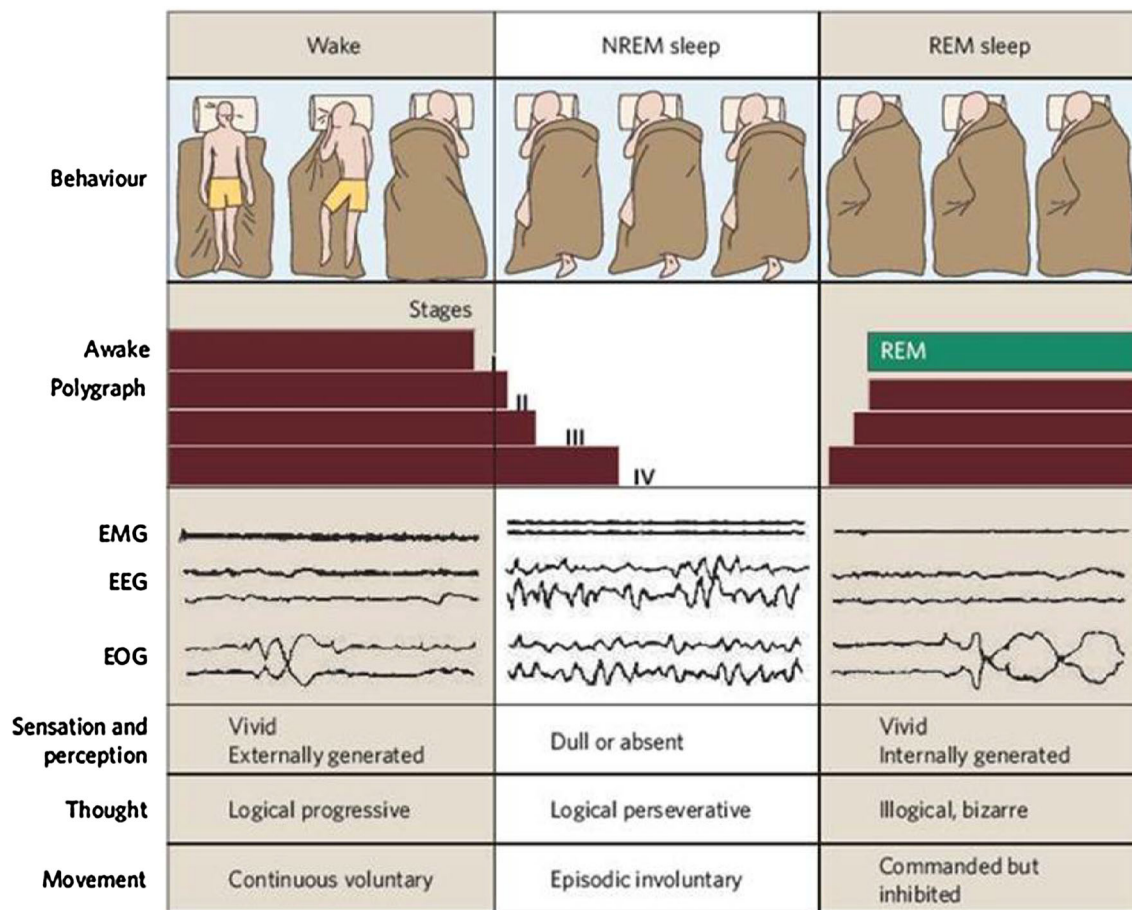


Fig. 2 The behavioral states of humans and phase changes throughout the sleep wake cycle, including states of waking, non-rapid-eye-movement sleep and rapid-eye-movement sleep. The *first row* depicts a visual representation of movements throughout the sleep night. The *second row* illustrates REM sleep and the four stages of NREM sleep. The *third row* includes sample polysomnography tracings (each ~20 s) of an electromyogram, an electroencephalogram, and an electrooculogram to help determine the presence or absence of each

stage. *Rows four, five, and six* portray a range of subjective and objective state variables. Although unable to replicate the sensitivity of these measurement techniques, other sleep indices (i.e. duration, latency) can also be measured by subjective sleep diaries and/or wristwatch actigraphy. Reproduced from Hobson [45], with permission. EEG electroencephalogram, EMG electromyogram, EOG electrooculogram, NREM non-rapid-eye-movement, REM rapid-eye-movement

to motor learning. REM, NREM stage 2, and slow-wave sleep (SWS) have all been implicated in sleep-dependent memory procession [36]. For example, several studies showed improvements in motor task tests after a night of sleep, whereas this was not the case in subjects having an equivalent period of being awake [36, 46–48]. Since sleep loss reduces the overnight improvement in motor learning, it seems that motor task learning may correlate with the amount of specific sleep stages/events, rather than just one specific aspect of sleep [36]. With the ongoing motor learning and cognitive adaptation required for elite athletes to perform [49], combined with the numerous neurocognitive components of many sports [50], it seems that ascertaining an optimal brain state for a range of distinct memory consolidation processes are pertinent for athletes prior to and following competition [49].

3 What is the Quantity and Quality of ‘Normal’ Sleep and how do Athletes Compare?

3.1 What is ‘Normal’ Sleep?

Subjective average total sleep duration has fallen in healthy adults since the mid-twentieth century from approximately 8–9 h per night in 1959 to 7–8 h in 1980 [51]. In a nationwide survey of the USA in 2013, data indicate adults slept for an average of 6 h:51 min on ‘workdays’ and 7 h:37 min on ‘non-workdays’ [52]. A mean 7 h:17 min total sleep time was required for respondents to ‘operate at their best the next day’ [52], which corresponds with the 7–9 h recommended by the National Sleep Foundation for healthy sleep [51–53]. Despite such recommendations, almost one-quarter of adults who have similar sleep durations to these recommendations reported ‘fairly–very bad’

subjective sleep quality [52]. Others have reported that university/college students demonstrate even poorer patterns of sleep than other healthy adults. Many studies indicate that this cohort suffers from chronic sleep problems and disruptions [54–56], with some adolescent athletes sleeping 2 h less than recommended daily sleep volumes [57]. These discrepancies are attributed to the rising melatonin levels of the adolescent cohort [58] and the rapid advances of 21st century technology, prolonging human exposure to light [59–61]. Overall, sleep architecture, quality and quantity varies drastically across individuals and occupations [62], mainly due to a vast array of physiological and cultural differences [63, 64]. Such variety makes the interpretation of generic sleep recommendations (7–9 h, abide by sleep hygiene protocols to optimize sleep quality [51, 52, 65]) difficult, especially for athletes [30].

3.2 Sleep in Athletes

Since both athletes and coaches rate sleep as critical to optimal performance [14, 25], it is peculiar that relatively few studies have investigated the sleep quality and quantity of the athletic cohort. Early research suggests that athletes possess similar or even superior sleep quality and quantity than nonathletic subjects [66, 67], with aerobically fit subjects tending to experience more SWS sleep and longer sleep duration than non-fit controls [68]. However, these findings may have been due to the enduring habitual, genetic, and behavioral patterns of sleep, rather than the greater endurance status per se [15, 69]. Regardless, the longer sleep duration found in certain aerobically fit individuals has been attributed to the restorative and energy conservation theories for sleep (e.g. athletes require greater recovery [69, 70]). Accordingly, some authors suggest athletes should sleep for between 9 and 10 h [71], whilst 7–9 h is recommended as enough for healthy adults [51, 52]. Recent evidence suggests that athletes sleep far less than either of these recommendations [72]. For example, a survey of 890 elite South African athletes showed that three-quarters of athletes reported an average sleep duration of between 6 and 8 h per night [73], while on weekends, 11 % reported sleeping less than 6 h. Moreover, 41 % stated they had problems falling asleep, with these discrepancies attributed to interference by noise and light [25, 74]. Additionally, pre-competition anxiety can also play a role in worsening sleep patterns [26, 75, 76]. For instance, sleep quality [76], efficiency [77], and duration [78, 79] have all been found to dramatically decrease just prior to competition. Juliff et al. [27] found that, within a sample of 283 elite Australian athletes, 64 % reported poor sleep prior to an important competition. The primary reasons for these poor sleep patterns could be due to

nervousness, deteriorations in mood and/or confidence [80], and elevations in physical and mental stress [77].

Recently, Leeder et al. [81] found that Olympic athletes slept for a lower mean total duration (6 h:55 min vs. 7 h:11 min using actigraphy) and had poorer sleep quality than non-athletic controls. Given the short sampling period (4 days), it is difficult to generalize the findings from this study to all athletes; however, there is supportive evidence of training disrupting sleep quality and duration in other athletes. For instance, Taylor et al. [80] reported training volume to alter movements during sleep (greater movements were found; defined as occupying ≥ 4 s of any 20 s epoch within the polysomnographic recording [80]). The effect of training volume on sleep patterns is supported by others [82, 83], with early-morning training severely restricting sleep duration compared with normal (5.4 to 7–8 h) in a group of world-class swimmers [72]. In addition to exercise volume, intensity may also negatively affect sleep, with a recent study reporting increases in sleep onset and physiological excitement following high-intensity exercise conducted prior to bed time (40 min treadmill running at 80 % heart rate reserve commencing at 21 h:20) compared with a non-exercise control condition in active young men [84]. Other possible disruptions of athletes' sleep include altitude, which appears to disrupt REM sleep and impair breathing [85]. Disrupted sleep is also prevalent in numerous extreme adventure and boat sports [86–88]. Despite these findings, further evidence of the sleeping patterns of elite athletes during various scenarios is very rare within the current literature. In summary, the sleep patterns of athletes remain unclear, mainly due to a vast array of physiological differences [63, 64], training [80, 89], and competition [26, 27] stressors. More research is required to assess the sleeping patterns of elite athletes across various scenarios that could potentially influence subsequent performance.

4 Effects of Sleep Loss on Exercise Performance and Physiological and Cognitive Responses

Sleep restriction (SR) occurs when humans fall asleep later or wake earlier than normal; that is, their normal sleep–wake cycle is partially disturbed [90]. In contrast, sleep deprivation (SD) generally refers to extreme cases of sleep loss, whereby humans do not sleep at all for a prolonged period (i.e. whole nights) [90]. The following sections of this article review the effects of sleep loss (restriction and deprivation) on exercise performance (Table 1) and physiological (Table 2) and cognitive (Table 3) responses to exercise. However, due to an abundance of conflicting results, some of the effects of sleep loss on these indices remain uncertain. These varied results are mainly attributed

Table 1 Studies examining the effect of sleep loss (restriction and deprivation) on various parameters of exercise performance

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Performance outcome	Results ^a
Endurance/aerobic					
Azboy et al. [122]	Runners and VB players ^b	25–30 h of SD	Incremental cycling test to exhaustion	Time to exhaustion	↓ in VB players
Hill et al. [120]	14 college students	25–30 h of SD	Incremental cycling test to exhaustion	Total work (kJ) Anaerobic contribution Aerobic contribution	NS NS NS
Martin [94]	8 subjects in 'excellent' health	36 h of SD	Prolonged walking to exhaustion at 80 % VO_{2max}	Time to exhaustion	↓ by ~11 % ^c
Martin and Chen [121]	8 graduate students	50 h of SD	Walking at steady-state then walking to exhaustion	Time to exhaustion	↓
Mejri et al. [98]	10 taekwondo athletes	Partial disruptions at the beginning and end of the night (SR)	YoYo intermittent recovery test level one	Total distance covered	NS
Mougin et al. [95]	7 cyclists	3 h of SR during the night	20 min steady state work (75 % VO_{2max}) on a cycle ergometer followed by an incremental test to exhaustion	Maximal sustained exercise intensity	NS
Oliver et al. [123]	11 recreationally active participants	30 h of SD	30 min pre-load treadmill run at 60 % VO_{2max} then 30 min self-paced treadmill run	Distance ran	↓
Racinais et al. [133]	22 athletes	38 h of SD	Leger and Gadoury shuttle run test	Shuttle run score	NS
Reilly and Deykin [97]	8 trained participants	2.5 h of sleep obtained per night for 3 nights (SR)	Incremental treadmill test to exhaustion	Endurance running performance	NS
Anaerobic					
Abedelmalek et al. [144]	12 footballers	Restricted to 4.5 h for 1 night (SR)	Wingate anaerobic test	Mean power Peak power	↓ ↓ Measured at 18:00
HajSalem et al. [107]	21 judokas	Partial disruptions at the end of 1 night (SR)	Wingate anaerobic test	Mean power Peak power	↓ ↓
Mougin et al. [96]	8 highly trained participants	~4 h of sleep obtained (SR)	Wingate anaerobic test	Mean power Peak power Peak velocity	NS NS NS
Soussi et al. [128]	13 PE students	36 h of SD	Wingate anaerobic test	Maximal power Peak power Mean power	↓ at 36 h ↓ at 36 h ↓ at 36 h

Table 1 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Performance outcome	Results ^a
Soussi et al. [100]	11 PE students	~3–4 h of sleep obtained per night for 2 nights (one at beginning and one at end of night; SR)	Wingate anaerobic test	Maximal power Peak power Mean power Force velocity Mean power	↓ ^d ↓ ↓ ↓ ↓ ^e
Soussi et al. [92]	12 judo competitors	3 h of sleep per night for 2 nights (one at the beginning and one at the end of the night; SR)	Wingate anaerobic test		
Symons et al. [130]	11 volunteers	60 h of SD	Wingate anaerobic test	Peak power Mean power Mean power Peak power	NS NS NS NS
Taheri et al. [196]	18 student athletes	Whole night of SD	Wingate anaerobic test		
Intermittent/RSA					
Skein et al. [126]	10 team-sport athletes	30 h of SD	30 min graded exercise run, 50 min intermittent sprint exercise (15-m maximal sprint per min and self-paced after)	15-m sprint performance	↓
Takeuchi et al. [124]	12 healthy volunteers	64 h of SD	Intermittent treadmill walking at 28 % VO _{2max} and 40 m sprint	40-m sprint performance	NS
Muscular strength					
Bulbulian et al. [127]	24 US Marine Corps	30 h of SD	Walking at low intensity; 45 consecutive maximal reciprocal contraction at a pre-determined isokinetic speed (3.14 rad/s ⁻¹)	Knee extension peak torque Knee flexion peak torque	↓ ↓
HajSalem et al. [107]	21 judokas	Partial disruptions at the end of 1 night (SR)	Muscular strength tests prior to and following a judo match	Handgrip test	NS
Meney et al. [30]	14 healthy participants	Whole night of SD	5 min of self-paced cycling; Muscular strength tests	Self-paced work rate Grip, leg, back strength	NS NS
Reilly and Deykin [97]	8 trained participants	2.5 h of sleep obtained per night for 3 nights (SR)	Muscular strength tests	Isometric handgrip test	NS
Reilly and Piercy [106]	8 healthy participants	3 h of sleep obtained per night for 3 nights (SR)	Maximal and submaximal weight-lifting tasks	Biceps curl Bench press Leg press Dead lift	Submaximal = ↓, maximal = NS Both ↓ Both ↓ Both ↓
Skein et al. [126]	10 team-sport athletes	30 h of SD	Muscular strength tests	MVC (right quadriceps) Voluntary activation	↓ ↓

Table 1 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Performance outcome	Results ^a
Soussi et al. [92]	12 judo competitors	3 h of sleep per night over for 2 nights (one at the beginning and one at the end of the night; SR)	Muscular strengths tests prior to judo combat	Handgrip test MVC (elbow flexors)	↓ ^e ↓
Symons et al. [130]	11 volunteers	60 h of SD	Muscular strength tests	Maximal isometric strength (forearm flexors, leg extensors) Mean torque (endurance) MVC (leg and arm) Rate of force development	NS NS NS NS
Takeuchi et al. [124]	12 healthy volunteers	64 h of SD	Muscular and balance strength tests	Handgrip Balance (stabilometer test) Vertical jump Isokinetic knee extension force	NS NS ↓ ↓
Sport-specific performance					
Edwards et al. [115]	60 differently experienced dart players	3–4 h of sleep obtained (SR)	Dart performance	Mean score Number of zeros Variability of dart score	↓ ↑ ↑
Fröberg et al. [195]	29 Army corporal officers	72 h of SD	Military shooting drills	Number of shots Number of hits	↓ ↓
Goh et al. [145]	14 military service members	Whole night of SD	Military pursuit drills	Drill performance Handgrip test	NS NS
Léger et al. [87]	8 healthy young sailors	2 h of sleep obtained per night	Four Tour de France yacht racing legs (90, 244, 56 and 75 nautical miles, respectively)	Global performance (final official race ranking)	It was found that the “final ranking in the race related to the sleep management strategy of the participants”
Otmani et al. [187]	20 healthy volunteers	4 h of sleep obtained for 1 night (SR)	Simulated car driving protocol	Driving performance measures	NS (except for “number of right edge line crossings” (↓ in alertness))
Reyner and Home [116]	16 tennis players	Delay bedtime 2–2.5 (e.g. ~5 h obtained for 1 night; SR)	Tennis serving drills	Serving accuracy	↓

Table 1 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Performance outcome	Results ^a
Simmerton and Reilly [111]	8 swimmers	2.5 h obtained sleep per night for 4 nights (SR)	Swimming performance test (50 m and 400 m); muscular strength tests	Lap times Back strength Grip strength	NS NS NS

MVC maximal voluntary contraction, NS not significant, PE physical education, RSA repeated sprint ability, SD sleep deprivation, SR sleep restriction, VB volleyball, VO_{2max} maximal oxygen uptake, ↓ and ↑ indicate decrease and increase, respectively

^a All changes signified by ↑ and ↓ were statistically significant ($p < 0.05$)

^b Full text unavailable

^c $p = 0.05$

^d When measurements were obtained at 18:00 and SD was at the end of the night

^e When measurements were obtained at 16:00 and SD was at the end of the night

to differences in exercise protocols, participants' fitness, and the experimental environment. For instance, variations in thermoregulatory responses, habituation to sleep loss and the time of day at which activities are performed have a complex interaction with exercise performance [65, 91, 92], and thus may potentially mask the effects of sleep loss [93]. Furthermore, being unable to blind subjects can potentially result in placebo effects [94].

4.1 Sleep Loss and Exercise Performance

4.1.1 Sleep Restriction and Exercise Performance

Early work from Mougin et al. [95] found no effects of a partially disrupted night's sleep (3 h of sleep loss in the middle of the night) on the maximal sustained exercise intensity during incremental cycle ergometry (20 min at 75 % maximum oxygen uptake [VO_{2max}] followed by 10 W increase every 30 s). The same authors [96] also found no change in mean or peak power or peak velocity during a Wingate cycling test after similar SR compared with normal baseline values in highly trained participants. With regard to more prolonged running exercise modes, Reilly and Deykin [97] reported no decrements in endurance running performance (time to exhaustion) following partial sleep loss (3 h of sleep per night for 3 nights). Furthermore, the total distance covered in a YoYo intermittent-recovery test level one was not different following SR [98]. In contrast to this maintenance of exercise performance, maximal work rate has been found to decrease (~15 W decrease following SR) during incremental cycling to exhaustion (30 min at 75 % VO_{2max} followed by 10 W increase every min [99]). Similarly, mean and peak power during Wingate anaerobic cycle tests have been shown to decrease in students [100], footballers [101], and judo competitors [92] following 4 h of SR for 1 night. Theories on the reasons for this restricted exercise tolerance following SR are attributed to either the impairment of aerobic pathways [102] or perceptual changes (i.e. increased perceived exertion), as physiological responses often remain largely unaltered [94, 103]. Indeed, increases in perceived effort accompanied by a reduction in power output would support neuromuscular causes of fatigue [104], possibly indicating an association between a reduction in central drive and the neural theory of sleep [36, 103, 105]. However, studies investigating perceived effort following SR report mixed results [98, 106, 107], so such theories remain unclear. These conflicting results are attributed to a large body of evidence reporting a vast array of effects on emotional regulation (i.e. mood) following SR [106, 108–111]. Indeed, variations in perceived effort are likely a result of these emotional modifications [112]. Given the widespread use of rating of perceived exertion in

Table 2 Studies examining the effects of sleep loss (restriction and deprivation) on physiological responses to exercise

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Outcome measures	Results ^a
Respiratory/cardiovascular					
Azboy et al. [122]	Runners and VB players ^b	25–30 h of SD	Incremental cycling exercise test to exhaustion	<i>At rest</i> VO ₂ VCO ₂ HR V _E SaO ₂ RQ <i>During exercise</i> HR VO ₂ VCO ₂ RQ SaO ₂ V _E VO ₂	↑ in runners ↑ in both groups NS NS NS NS NS NS NS NS ↓ in both groups NS
Home and Petit [103]	7 physically untrained participants	72 h of SD	40 min (total) cycling at 40, 60, 80 % of VO _{2max}	RPE VO ₂ VCO ₂	↑ NS NS
Martin and Gaddis [158]	6 healthy participants	30 h of SD	8 min of cycling at 25, 50 and 75 % of VO _{2max}	HR V _E BP	NS NS NS
Martin and Chen [121]	8 graduate students	50 h of SD	Walking at steady-state then walking to exhaustion	VO ₂ VCO ₂ HR V _E BP	NS NS NS NS NS
Martin et al. [138]	8 healthy participants	36 h of SD (preceded by 2 nights of partial sleep disruption)	30 min of high-intensity treadmill walking and 3 h of treadmill walking	VO ₂ V _E HR VO ₂ V _E	NS NS NS NS NS
Mejri et al. [98]	10 taekwondo athletes	Partial disruptions at the beginning and end of the night (SR)	YoYo intermittent recovery test level one	HR _{peak} RPE	NS NS
Menev et al. [30]	14 healthy participants	Whole night of SD	5 min of self-paced cycling	HR RPE Self-paced work rate	NS NS NS

Table 2 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Outcome measures	Results ^a
Mougin et al. [102]	7 endurance athletes	Partial disruption during the middle of the night for 1 night (SR)	Submaximal (75 %) cycling test and maximal incremental test on a cycle ergometer	HR Ventilation rate V_E/VO_2 VO_{2max}	↑ at submaximal ↑ at submaximal ↑ at submaximal ↓ at submaximal
Mougin et al. [95]	7 cyclists	3 h of SR during the night	20 min steady state work (75 % VO_{2max}) on a cycle ergometer, followed by incremental test to exhaustion	HR V_E VO_{2peak}	↑ during both phases ↑ during both phases ↓ during incremental
Mougin et al. [96]	8 highly trained participants	4 h sleep obtained for 1 night (SR)	Wingate anaerobic test	V_{Emax} VT VO_{2peak}	NS NS NS
Oliver et al. [123]	11 recreationally active participants	30 h of SD	30 min at 60 % VO_{2max} followed by 30 min self-paced treadmill run	RPE HR VO_2	NS NS NS
Plyley et al. [160]	11 healthy volunteers	64 h of SD	VO_{2max} test, with an additional group completing 1 h of treadmill walking every 3 h	VO_{2max} V_{Emax} RER	↓ ↓ NS
Reilly and Deakin [97]	8 trained participants	2.5 h of sleep obtained per night for 3 nights (SR)	Incremental treadmill test to exhaustion	HR FEV ₁ VC	NS NS NS
Sinnerton and Reilly [111]	8 swimmers	2.5 h obtained sleep per night for 4 nights (SR)	Muscular strength measures; swimming performance test	Lung function	NS
Symons et al. [128]	11 volunteers	60 h of SD	20 min at 75 % VO_{2max} on cycle ergometer; Wingate anaerobic test; Intermittent cycle test; treadmill running at 70–80 % VO_{2max}	HR during 80 % SSE RPE during 80 % SSE BF during 80 % SSE All other respiratory variables	↑ ↑ ↑ NS
Hormonal and immunological					
Abdelmalek et al. [101]	30 footballers	4.5 h obtained for 1 night	4 × 250 m runs on treadmill at 80 % of the personal maximal speed (3 min rest in between sets)	Plasma cortisol Testosterone Growth hormone IL-6 TNF- α IL-6	NS ↑ ↑ ↑ ↑ ↑
Abdelmalek et al. [144]	12 footballers	4 h obtained for 1 night	Wingate anaerobic test	IL-6	↑ Measured at 18:00

Table 2 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Outcome measures	Results ^a
Costa et al. [149]	10 recreationally active participants	30 h of SD	Pre-test: Incremental $\dot{V}O_{2max}$ test to exhaustion; followed by a treadmill time trial Experimental test: Controlled physical activity during the day with a 90 min walk @ 50 % of $\dot{V}O_{2max}$ and a 5 km treadmill time trial	Circulating leukocytes T-lymphocyte subset Bacterially-stimulated neutrophil degranulation Saliva secretory immunoglobulin A Plasma cortisol	NS ^c NS NS NS NS
Goh et al. [145]	14 military service members	Whole night	Military pursuit drills	Melatonin Plasma cortisol	↑ ↑
Martin and Chen [121]	8 graduate students	50 h of SD	Walking at steady-state then walking to exhaustion	Blood lactate Epinephrine Dopamine	NS NS NS
Martin et al. [138]	8 healthy participants	36 h of SD (preceded by 2 nights of partial sleep disruption)	30 min of high-intensity treadmill walking and 3 h of treadmill walking	Plasma cortisol β-endorphins	NS NS
Mougin et al. [95]	7 cyclists	3 h of SR during the night	20 min steady state work (75 % $\dot{V}O_{2max}$) on a cycle ergometer, followed by incremental test to exhaustion	Blood lactate	↑ during both phases
Mougin et al. [96]	8 highly trained participants	~4 h obtained for 1 night (SR)	Wingate anaerobic test	Plasma concentrations of lactate	NS
Mougin et al. [99]	8 well-trained endurance athletes	4.5 h obtained for 2 nights (SR)	30 min steady state cycling at 75 % of $\dot{V}O_{2max}$ then progressive increases to exhaustion	Growth hormone Prolactin Plasma cortisol Catecholamines	NS ↑ ↓ NS
Plyley et al. [160]	11 healthy volunteers	64 h of SD	$\dot{V}O_{2max}$ test, with an additional group completing 1 h of treadmill walking every 3 h	Blood lactate Blood lactate	↑ NS
Soussi et al. [128]	13 physical education students	24 h of SD	Wingate anaerobic test	Blood lactate	NS
Energy substrate storage					
Skein et al. [126]	10 team-sport athletes	30 h of SD	30 min graded exercise run; 50 min intermittent-sprint exercise protocol (15 m maximal sprint every minute and self-paced exercise for remainder of minute)	Muscle glycogen	↓

Table 2 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise protocol	Outcome measures	Results ^a
Thermoregulation					
Martin et al. [138]	8 healthy participants	36 h of SD (preceded by 2 nights of partial SD)	30 min of high intensity treadmill walking and 3 h of treadmill walking	Core temperature	NS
Meney et al. [30]	14 healthy participants	Whole night of SD	5 min of self-paced cycling	Core temperature (tympanic membrane)	NS
Sawka et al. [204]	5 fit participants	33 h of SD	40 min on cycle ergometer (50 % of VO_{2max}) in 28 °C ambient ^c conditions	Core temperature (esophageal) Local sweat rate Chest thermal conductance	NS ↓ ↓

BF breathing frequency, *BP* blood pressure, *FEV₁* forced expiratory volume in 1 second, *HR* heart rate, *HR_{max}* maximal heart rate, *IL* interleukin, *NS* not significant, *RER* respiratory exchange ratio, *RPE* rating of perceived exertion, *RQ* respiratory quotient, *SaO₂* arterial oxygen saturation, *SD* sleep deprivation, *SR* sleep restriction, *SSE* steady state exercise, *TNF* tumor necrosis factor, *VB* volleyball, *VC* vital capacity, *VCO₂* carbon dioxide production, *V_E* minute ventilation, *V_{Emax}* maximal minute ventilation, *VO₂* oxygen uptake, *VO_{2peak}* peak oxygen consumption, *VO_{2max}* maximal oxygen uptake, *VT* tidal volume, ↓ and ↑ indicate decrease and increase, respectively

^a All changes signified by ↑ and ↓ were statistically significant ($p < 0.05$)

^b Full text unavailable

^c Measured at rest

monitoring the training load of elite athletes [113, 114], further research is required to investigate the interaction between these responses to standardized training or match stimuli following sleep loss.

Similar to maximal aerobic demands, a variety of conclusions have been reported for the effects of SR on muscular strength and power. Studies have shown back and grip strength are maintained following SR [93]. In contrast, others have demonstrated 3 h of nocturnal SR to negatively affect both maximal and submaximal weightlifting tasks, with greater effects on the submaximal tasks [106]. Given the high motivational component of weightlifting, this decline in work rate was attributed to the coinciding decline in mood state. However, whilst these perturbations in submaximal work outputs may be due to fluctuations in mood state, or even neurological alterations [104], the central and local muscular fatigue mechanisms behind such outcomes remain unknown [106]. Collectively, these observations indicate that whilst athletes may be able to perform singular, maximal efforts following SR, it is unclear whether they are able to cope with repeated bouts of physical activity such as those required during intensive training or matches [21].

An example of the susceptibility of sport-specific performance following SR in athletes is the reduction in sport-specific skill execution in dart players [115], tennis players [116], and handball goalkeepers [117]. In contrast, swimming performance (lap times) did not differ between SR (2.5 h of sleep per night for 4 nights) and normal sleep for eight trained swimmers [111]. These differing findings could be attributed to the additional cognitive dimension of the aforementioned fine motor skills. For instance, since loss of sleep can result in reductions in decision making abilities and accuracy (see Sect. 4.3), SR would presumably be more likely to affect the performance of sports incorporating a high cognitive reliance (i.e. fine motor movements in the serve accuracy of a tennis player [116]) rather than one involving gross-motor execution (i.e. the stroke rate of a swimmer [111]). Furthermore, since professional sport comprises many environmental components that can influence sleep [14], it has been argued that athletes may be more susceptible to performance decrements following SR than normal healthy participants [81], although this is debated [69, 81, 118, 119].

Overall, the effects of SR on exercise performance are mixed. SR does not appear to affect singular bouts of aerobic performance (neither endurance running nor cycling modes for 20–30 min) or maximal measures of strength, although admittedly conflicting results still exist. A possible reason for this discrepancy is that many studies reporting no effect of SR on endurance exercise have sample sizes less than ten participants (e.g. Reilly and Deykin [97], Mougín et al. [99]; Table 1), making it

Table 3 Studies examining the effects of sleep loss (restriction and deprivation) on cognitive performance and mood state

References	Subjects and fitness status if provided	Sleep intervention	Exercise condition if applicable	Performance measure	Results ^{a,b}
Cognitive performance					
Angus et al. [191]	12 fit young subjects	60 h of SD	NA	Auditory vigilance Logical reasoning Visual search Mental addition Coding RT	↓ ↓ ↓ ↓ ↓ ↑
Axelsson et al. [109]	9 healthy participants	4 h obtained per night for 5 nights	NA	RT	↑
Bonnet [172]	11 healthy adults	Continuous disruption for 2 nights, ~1 h lost per night (SR)	NA	RT	↑
Drummond et al. [185]	44 healthy participants	3.5–4 h obtained per night for 4 nights (SR)	NA	Visual working memory performance Filtering efficiency performance	NS NS
Drummond et al. [185]	44 healthy participants	Whole night of SD	NA	Visual working memory performance Filtering efficiency performance	NS ↓
Grundgeiger et al. [175]	60 first-year university students	25 h of SD	NA	Two prospective memory tasks (more demanding and less demanding combinations of German 'living' and 'non-living' words)	↓ in both
Harrison and Horne [193]	10 trained participants	36 h of SD	NA	Critical reasoning Game involving decision making and innovative thinking	NS ↓
Hurdiel et al. [86]	12 professional competitive sailors	22 ± 30 min, 92 ± 34 min and 172 ± 122 min during the race	150, 300 and 350 nautical mile races	5 min serial reaction time test	↑
Jarraya et al. [117]	12 handball goalkeepers	4–5 h obtained for 2 nights (1 with SR at the start of the night, 1 with SR at the end of the night)	NA	RT Stroop test (selective attention and reading ability) Barrage test (visual-spatial ability and recognition)	↑ ↓ ↓
Khazaie et al. [183]	26 medical residents	<6 h obtained per night for 5 nights (SR)	NA	Wisconsin card sorting test Time perception task Iowa gambling test	NS NS NS

Table 3 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise condition if applicable	Performance measure	Results ^{a,b}
Lucas et al. [199]	9 adventure racers	100 h of SD	96–125 h of adventure racing	Altered stroop test (simple and complex response/decision making)	NS
Olsen et al. [200]	71 army and navy cadets	2.5 h obtained per night for 5 nights (SR)	Combat simulation drills	Defining issues test (moral reasoning)	↓
Rosa et al. [197]	12 healthy participants	40–64 h of SD	NA	Williams word memory test	↓
Scott et al. [192]	6 students	30 h of SD	Rest and cycle ergometry at 50 % VO_{2max} for 20 min every 2 h for 30 h of SD	Tracking task Number cancellation task 2 choice reaction time and simple reaction time	NS NS ↑ at rest
Symons et al. [130]	11 volunteers	60 h of SD	20 min at 75 % VO_{2max} on cycle erg; Wingate anaerobic test; Intermittent cycle test; Treadmill running at 70–80 % VO_{2max} ; Muscular isometric strength tests	RT	NS
Taheri et al. [196]	18 student athletes	Whole night of SD	Wingate anaerobic test	Choice reaction time	↑
Vgontzas et al. [143]	25 normally active participants	6 h per night (2 h less than normal) for 8 nights (SR)	NA	Psychomotor vigilance test	↓
Williamson et al. [194]	39 volunteers from transport industry and the army	17–19 h of SD	NA	RT Mackworth clock (passive vigilance test) Tracking (hand–eye coordination) Dual task (divided attention) Symbol digit test (coding) Spatial memory search Memory and search test	Speed and accuracy for all tasks were generally poorer with results at the end of the SD period equivalent to blood alcohol concentrations of 0.01–0.05
Wimmer et al. [198]	12 undergraduate students	Whole night of SD	NA	Torrence test of creative thinking Trail marking test (attention) Letter recognition task (attention) Working memory performance	↓ ↓ ↓ ↓

Table 3 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise condition if applicable	Performance measure	Results ^{a,b}
Mood state					
Angus et al. [191]	12 fit young subjects	60 h of SD	NA	Subjective fatigue checklist	↑
				Stanford Sleepiness Scale	↑
				Mood state	↓
				Auditory vigilance	↓
				Logical reasoning	↓
				Visual search	↓
				Mental addition	↓
				Coding	↓
				RT	↑
Axelsson et al. [109]	9 healthy participants	4 h obtained per night for 5 nights (SR)	NA	RT	↑
				Karolinka Sleepiness Scale	↑
Bonnet [172]	11 healthy adults	Continuous disruption for 2 nights, ~1 h lost per night (SR)	NA	Clyde Mood Scale	↓
				Stanford Sleepiness Scale	NS
Edwards and Waterhouse [115]	60 differently experienced dart players	3–4 h obtained for 1 night (SR)	Dart throwing	Subjective alertness	↓
				Subjective fatigue	↑
Koboyashi et al. [110]	13 healthy university students	5 h obtained per night for 7 nights (SR)	NA	Subjective sleepiness	↑
Meney et al. [30]	14 healthy participants	Whole night of SD	5 min of self-paced cycling;	<i>POMS</i>	
				Fatigue	↑
				Confusion	↑
				Vigour	↓
Olsen et al. [200]	71 army and navy cadets	2.5 h obtained per night for 5 nights (SR)	Combat simulation drills	Stanford Sleepiness Scale	↑
				Defining issues test (moral reasoning)	↓
Reilly and Piercy [106]	8 healthy participants	3 h obtained per night for 3 nights (SR)	Weight lifting tasks	<i>POMS</i>	
				Fatigue	↑
				Confusion	↑
				Vigour	↓
				Depression	NS
				Anger	NS
				Tension	NS
				Sleepiness	↑
				Perceived effort	↑

Table 3 continued

References	Subjects and fitness status if provided	Sleep intervention	Exercise condition if applicable	Performance measure	Results ^{a,b}
Scott et al. [192]	6 students	30 h of SD	Rest and cycle ergometry at 50 % VO_{2max} for 20 min every 2 h for 30 h of SD	<i>POMS</i> Fatigue Confusion Vigour Depression Tension Anger Tracking task Number cancellation task 2 choice reaction time and simple reaction time	↑ ^c NS ↓ ↑ NS NS NS ↓ ↑ at rest ^c
Sinnerton and Reilly [111]	8 swimmers	2.5 h obtained per night for 4 nights (SR)	Muscular strength measures; Swimming performance test	<i>POMS</i> Fatigue Confusion Vigour Depression Anger Tension	↑ ↑ ↓ ↑ ↑ ↑
Skein et al. [126]	10 team-sport athletes	30 h of SD	30 min graded exercise run, 50 min intermittent sprint exercise (15 m maximal sprint per min and self-paced after)	<i>POMS</i> Liveliness Alertness Energetic Fatigue	↓ NS NS NS
Vgontzas et al. [143]	25 normally active participants	6 h per night for 8 nights	NA	Multiple sleep latency test	↑

NA not applicable, NS not significant, *POMS* Profile of Mood States, *RT* simple reaction time, *SD* sleep deprivation, *SR* sleep restriction, VO_{2max} maximal oxygen uptake, ↓ and ↑ indicate decrease and increase, respectively

^a All changes signified by ↑ and ↓ were statistically significant ($p < 0.05$)

^b Note that, for RT, ↑ represents a slowing down of reaction time

^c Results here are derived from interaction effects. Please refer to the original article for main condition effects and further detail on the role of cognition during exercise following sleep deprivation

difficult to extrapolate the results of these studies due to the underpowered nature of the study. In contrast, sports-specific skill execution, submaximal strength, and muscular and anaerobic power seem to decline following SR. Given these findings, whilst it seems that SR impedes some aspects of athletic performance; it is still not clear whether sleep is critical to performance for *all* athletes who experience small one-off SR periods.

4.1.2 Sleep Deprivation and Exercise Performance

Similar to SR, the effects of total SD on exercise performance are varied [120]. Mean time to exhaustion for

prolonged treadmill walking (80 % of VO_{2max}) is reduced by ~11 % following 36 h of SD [94]. These results are supported by other studies highlighting reduced time to exhaustion (mean ~20 % [121]) during incremental exercise protocols following SD [122]. In addition, mean distance covered has been found to decline (6,224 to 6,037 m) following SD during 30 min of self-paced treadmill running [123]. It appears time to exhaustion decreases because of either perceptual changes or reductions in arousal and impaired muscle fiber coordination (e.g. decreases in vertical jump performance and knee extension torque [124]) following prolonged SD, although the mechanisms behind this are unclear [94]. Indeed, it is

proposed that increased muscular and central fatigue is unlikely to explain decreases in prolonged exercise performance following SD [112]; however, this warrants further investigation.

Despite the popularity of sports that require high intermittent-sprint performance (i.e. team sports [125]), there is a relatively poor understanding of the effect of SD on these activities. Skein et al. [126] recently reported slower mean sprint times and reduced muscle glycogen concentration, voluntary force, and activation during maximal isometric knee extensions, along with an increased perceptual effort following 30 h of SD in ten team-sport athletes [126]. Similarly, several other studies have shown the detrimental effects of SD on muscular strength [30, 124, 127], power [128], and speed [129]. In contrast, Symons et al. [130] reported no effect of 60 h of SD on a range of maximal upper and lower body isometric and isokinetic strength tests. Indeed, several studies have shown that grip strength performance is maintained regardless of the amount of sleep loss [131, 132], and shuttle run scores remain unaffected [133]. Indeed, submaximal strength tasks may be more susceptible to SD than maximal tasks due to the sustained effort required to complete the task, whereby perception of effort could increase exponentially with time to task completion [123]. In addition, differences in reported muscle contractility (i.e. voluntary activation) between studies could be explained by the sensitivity and accuracy of electromyography measurements. Older studies (i.e. Symons et al. [130]; [Table 1]) may have been limited in comparison with the equipment used in recent research [126, 134].

In summary, although the effect of SD on exercise performance remains somewhat unclear, there appears sufficient evidence to imply that SD can have a significant effect on aspects of athletic performance. This seems particularly pertinent for time to exhaustion in running activities lasting longer than 30 min. Nonetheless, whilst these studies reveal important physiological mechanisms, conceptually it is debatable whether the findings are applicable to elite athletic populations given it would be rare for an athlete to endure a night(s) of complete SD.

4.2 Sleep Loss and Physiological Responses to Exercise

4.2.1 Sleep Restriction and Physiological Responses to Exercise

Examples of the susceptibility of physiological responses to exercise following SR are the increase in heart rate, minute ventilation, and plasma lactate concentration during submaximal and maximal exercise after a partially disrupted night's sleep (3 h of sleep loss in the middle of the night) [95]. These responses are attributed to the increased

metabolic demand [135], perceived effort [94], and catecholamine concentrations following SR [136]. This could be interpreted as SR acting as an additional stress to the stress imposed by exercise itself [137]. In contrast, Martin et al. [138] showed that 2 nights of fragmented sleep (eight 'wake up' calls ranging 30–75 min) had no significant effect on heart rate, oxygen consumption, minute ventilation, and core body temperature during 30 min of heavy treadmill walking. Similarly, these findings support other results, suggesting no alterations to physiological responses following SR, i.e. lung function and power unaffected by minor sleep loss [97, 111]. Whilst the error sensitivity across metabolic collection systems could perhaps explain some differences across studies [139–142], these differences are perhaps more attributable to the exercise mode and protocol administered (running [98] vs. cycling [95]; free-paced exercise [111] vs. time to exhaustion [102]).

Although various hormonal concentrations (e.g. plasma cortisol) will typically increase during exercise-induced stress, the interaction between these responses and sleep loss is inconclusive [31]. For instance, there have been reports by some [99, 143], but not all [138, 144, 145] studies that cortisol concentration might be lowered following sleep loss. These varied results are likely attributed to the fact that cortisol secretion is dependent on the timing, intensity, and duration of the stimulus [146] and is highly driven by circadian rhythms [147]. As an example of the sensitivity of hormonal and additionally immune responses to SR and exercise stimuli, GH, prolactin and interleukin (IL)-6 have been shown to increase following SR and four 250-m treadmill runs at 80 % maximum speed [101]. This is supported by findings of next-day increases in IL-6 (threefold) and tumor necrosis factor (TNF)- α (twofold) following SR [148], although others have reported these variables to remain unchanged at rest [149]. Since increases in these pro-inflammatory cytokines (e.g. IL-6; mean $4.11 \pm$ standard deviation 0.99 rising to 5.44 ± 1.1 pg·ml⁻¹ [144] and TNF- α [143] following SR and exercise) might be associated with unfavorable metabolic profiles [143] and inflammatory disease risk [147, 150], there is concern about obtaining sufficient quality and duration of sleep in all individuals from an overall health perspective [14, 143].

4.2.2 Sleep Deprivation and Physiological Responses to Exercise

Energy substrate balance appears vulnerable to sleep loss, with 30 h of SD shown to blunt the full restoration of muscle glycogen stores in team-sport athletes [126]. Without adequate intake, this could hinder the ability of athletes to compete for sustained periods, as muscle glycogen shortage is known to reduce muscle function and

total work capacity [3, 151]. Indeed, energy imbalances are associated with SD, potentially leading to decreased aerobic and anaerobic power production [21, 152]. Prolonged periods of SD (36 h) are further associated with increased sympathetic and decreased parasympathetic cardiovascular modulation, and spontaneous baroreflex sensitivity during sitting and vigilance testing in healthy adults [153]. Since disruptions to the sympathetic–parasympathetic balance are associated with overtraining [154], it is possible these disturbances to the autonomic nervous system following SD could support the development of an over-reaching or over-training status [3, 155]. Indeed, of importance to athletes, maintaining this autonomic balance is critical for producing optimal performance [156]. Notwithstanding this, most [94, 103, 122], but not all [122] studies have reported that SD does not alter cardiorespiratory variables during incremental exercise (e.g. VO_{2max} , minute ventilation). Further to these results, there were no significant effects on cardiorespiratory or thermoregulatory function despite a reduction in distance covered during 30 min of self-paced treadmill running following SD [123]. Taken with other results [94, 123, 157, 158], these findings suggest that SD has minimal effect on cardiorespiratory function during intermittent submaximal exercise, despite observations of a reduction in performance. Oliver et al. [123] hypothesize this could be due to the influence of the perception of effort during the end stages of prolonged high-intensity exercise. Extreme periods of sleep loss (i.e. 100 h without sleep) are more likely to negatively affect cardiorespiratory variables than acute SD (24–36 h) [159].

Similar to the effects of SR, the effects of SD on hormonal and endocrine responses to exercise are unclear. It has been shown that SD (50 h) does not affect blood parameters such as blood lactate, epinephrine, norepinephrine, and dopamine during treadmill walking to exhaustion [121], nor in cases where subjects exercised (28 % VO_2 max for 1 h every 3 h for 64 h of SD) during the SD period (i.e. blood lactate concentration [12.1 vs. 11.8 $mmol \cdot l^{-1}$] [160]). However, such responses are heavily influenced by circadian fluctuations [40], making the effect of SD on these parameters difficult to determine. Interestingly, these two studies [121, 160] and others [138] that reported no differences in hormonal and endocrine responses to exercise following SD used constant exercise protocols, whereas two studies that reported significant changes following SR [95, 99] utilized incremental tests to exhaustion. Thus, the variable load at the end of exercise appears to increase the final stress-related response. The response of blood-cortisol concentrations to SD are similar to those with SR, with inconsistent findings presented [138, 149, 161]. Theoretically, if increased cortisol concentrations do occur [161], this could lead to increased muscle catabolism and a reduction in protein synthesis [3]. As

such, this would lend support to the restorative theory that sleep is required for muscular recovery [162]; however, such hypotheses require further research for clarification. For instance, whilst SD can initially blunt the secretion of GH [163], possibly hindering growth [42] and recovery [162], this deficiency is compensated for by increasing GH secretion during waking hours [164].

4.3 Sleep Loss, Cognitive Performance, and Mood Responses

Numerous studies report that when sleep is reduced to less than 7 h in healthy adults, cognitive performance is poorer in tests for alertness, reaction time, memory, and decision making [23, 109, 165–170]. Heightened levels of sleepiness, depression, confusion, and poorer overall mood states have also been reported [171–174]. Decrements in cognitive performance have previously been attributed to disruptions to pre-frontal cortex functioning, as cognitive deficiencies that occur outside this area of the brain malfunction in qualitatively different ways [169]. Recently, a more universal effect of sleep disruption on cognition has been proposed [175], due to the sensitivity of cognitive performance to both arousal (not limited to pre-frontal activity) and attention in a sleep-disrupted state [166]. The neuroanatomical mechanisms behind this state are intricately complex [176]. For instance, when the quality and quantity of human sleep is reduced, it appears the largest decreases in cerebral metabolism (compared with the awake-rested state) are apparent in the thalamus, cerebellum, and prefrontal, posterior parietal, and temporal cortices [176, 177]. The reduced metabolic rates within these regions have been correlated with decreased cognitive performance [178, 179], highlighting their influence on optimum cognitive functioning [176, 180]. Based on these collective findings, some support suggested sleep benefits from models related to neural mechanisms, rather than peripheral tissues [103].

4.3.1 Cognitive Performance and Mood Responses Following Sleep Restriction

As an example of the sensitivity of cognitive function to sleep disruption, simple reaction time (RT) has been shown to increase in individuals following 1 h of SR for 2 nights [108] and 4 h of SR for 5 nights [109]. In addition, Jarraya et al. [117] found increases in RT and decreases in selective and constant attention in 12 handball goalkeepers following 4–5 h of SR at both the beginning and the end of the night [117]. With RT slower following even minor disruptions to both sleep quality [108] and duration [117], it would seem pertinent for athletes with a high reliance on this cognitive component to ensure optimum sleep conditions prior to competing (e.g. baseball, cricket). This may be particularly

challenging for baseball teams who play more than 80 away matches per season, where sleep conditions will change on an almost daily basis. These recommendations might be extrapolated to a host of individual and team-sport athletes, as many sports also involve critical decision making [181, 182], which is also susceptible following SR [169]. Although the majority of literature supports the impairment of decision making following sleep loss [169], others have reported no effects [183]. Khazaie et al. [183] reported no change in abstract reasoning, time reproduction skills, or decision-making ability in 26 sleep-restricted (<6 h sleep for 5 nights) medical residents. Whilst this was most likely due to a lack of an effect of partial SR on pre-frontal cognition or the interaction between the type of SR and type of cognitive task, it does show that optimum sleep may not always be critical for maintenance of decision-making performance over an acute period.

The understanding of the effect of SR on memory and recall is also equivocal, with some authors reporting decrements in short-term memory following SR [184], whilst others report no change [185]. For instance, Drummond et al. [185] found no changes in visual working memory or filtering efficiency following 3.5–4 h of sleep. Whilst SR is unlikely to affect elite players' memory of *how* a (motor) skill is executed, it could potentially affect the recall and understanding of tactical awareness or positioning. From this perspective, it seems that sufficient sleep should be obtained following training sessions, as the perceptual and motor learning processes continue into and throughout subsequent sleep [186]. Another example of the detrimental effects of SR on cognitive performance is the plethora of evidence that reports poorer mood states after SR, with decreases in vigor along with increases in

depression, sleepiness, and confusion [106, 109, 115, 172, 187]. These negative mood states have been linked to over-reaching and over-training [188–190]. Indeed, this increase in psychological fatigue following SR would appear to create a neurocognitive state not conducive for either engaging in physical activity requiring a high motivational component or employing optimal decision making; however, such concepts still require further substantiation.

4.3.2 Cognitive Performance and Mood Responses Following Sleep Deprivation

The effects of SD on cognitive performance are quite clear, with many studies showing that greater total sleep loss results in poorer overall mood states, with increased fatigue, sleepiness, and confusion, decreased vigor [30, 138, 191] and liveliness [126], and heightened depression [192]. In addition, decreases in logical reasoning, coding, decision making, and filtering efficiency have also been reported [185, 191, 193]. The speed and accuracy at which these tasks are performed are also negatively affected by SD [194, 195]. Moreover, previous studies show that participants perform poorer in tests for auditory vigilance [192], simple and complex RT [191, 192, 196], and memory [175, 194, 197, 198] following complete sleep loss. Limited data are available for cognitive functioning during sporting events, although during extreme sports (i.e. long-haul yacht racing), it appears cognitive impairments present following extensive SD [86]. These findings potentially have severe repercussions for athletic performance (Table 4). Nonetheless, conflicting results do exist, with no significant differences in simple and complex responses to an altered Stroop test for decision making during 96–125 h of

Table 4 Effects of sleep loss on cognitive functioning and possible extrapolations to sport performance (column 1 adapted from Durmer and Dinges [23], with permission)

Effects of sleep loss on cognitive performance	Possible effects on professional athletes
Time pressure increases error rate	More errors in time-affected sports (e.g. shotclock in basketball)
Response time slows	Decreased reaction time could be especially pertinent for sprinters, baseballers, cricketers, goalkeepers, and tennis and handball players
Both short-term recall and working memory performances decline	Effects the messages coaches can deliver to athletes, this will have a flow-on effect on tactical awareness (may be pertinent for teams with set plays e.g. American football, ice hockey, rugby league, basketball, and soccer)
Reduced learning (acquisition) of cognitive tasks	Blunt cognitive-induced training adaptations during periods of high-intensity learning (e.g. players could struggle whilst learning new tactics and formations during the pre-season in sports such as soccer and Australian rules football)
Response perseveration on ineffective solutions is more likely	If an athlete continually tries to perform a task in the wrong manner from a reduced proprioceptive state, this could lead to an increase in injury [3]
Tasks may be begun well, but performance deteriorates as task duration increases	Fatigue can lead to an increase in decision-making errors. Could affect all sports played over prolonged periods (e.g. decathlon, American football, baseball, Australian rules football)
Increased compensatory effort is required to remain behaviorally effective	This would suggest a decrease in time to fatigue, affecting numerous sports that experience intermittent and repeated exercise bouts

adventure racing (~100 h of SD [199]). These differences are most likely attributable to the intra-individual variability in personality and mood state and sleep requirement, in addition to sample size and task familiarity [200]. For instance, Edinger et al. [201] found vastly different responses for sleepiness and mood when investigating the daytime functioning of two players during a 146-h marathon tennis match. Indeed, humans are sometimes unaware of their increasing cognitive deficits and declining neuro-behavioral function following SD [65]. In summary, SD results in relatively unequivocal decrements in most aspects of cognitive function and mood responses.

5 Future Research

Currently, there is insufficient evidence to clarify the importance of sleep for athletes and the effects of sleep loss on exercise performance, alongside physiological and cognitive responses to exercise. Indeed, more research is required to confirm what dimensions of exercise performance are affected by sleep loss, especially those with a focus on repeated bouts of intermittent exercise and sport-specific performance. Admittedly, very little of the current literature has been conducted in team-sport athletes, making the extrapolation of assumptions regarding sleep and performance to team sports difficult. Furthermore, there is little to no statistical analysis in the majority of previous studies with regard to magnitudes of effect, which may cloud some statistical inferences as to the effect on performance with respect to practical relevance [202]. Moreover, the majority of studies that assess the effect of sleep loss on athletic performance are those involving SD, a scenario that is very rare in the real world. For athletes, it

would seem more pertinent in future research to investigate the effect of SR on parameters related to athletic performance. Future research may also focus on the interaction between sleep and acute and chronic training adaptations. Further research is also required to confirm whether reduced sleep in elite athletic populations is associated with illness and injury occurrence, and whether such disturbances can partly explain the over-training state. Preliminary evidence indicates that athletes who are at least functionally over-reached present with sleep disturbances and illness prevalence during high-volume training [203]. From a purely scientific perspective, it is pertinent certain factors are considered in future endeavors when defining the effect of sleep on athletic performance within an experimental protocol [21, 204], including isolating homeostatic and circadian components, utilizing an externally valid competitive event and minimizing the many confounding variables that affect sports performance [205].

6 Practical Recommendations

The following recommendations (Table 5) are based on the literature within this review. However, the authors recognize that given the equivocal findings for most summaries, future research is required to confirm these recommendations. Most importantly, it is recommended to understand the intra-individual differences with regards to sleeping patterns. Practitioners should strive to identify where sleep problems exist, and if necessary employ ethical interventions. If problems persist, these should be dealt with by medical professionals [7]. Whilst there are numerous examples of the interaction between sleep and performance that may aid practitioners, there is little literature

Table 5 Practical recommendations for sporting practitioners

Identify whether sleep problems exist within your athletic population—collect and compare with longitudinal data across a variety of situations and competitions. Where possible, collect performance and/or match data to detect possible associations. There may be instances where there are no sleep issues apparent
If issues are present, identify poor practice; how, when, and why do these issues occur. If problems persist, treat in conjunction with a trained medical professional from the team to improve the quantity and quality of sleep (follow sleep hygiene practice, i.e. no technology 30 min before bedtime, no TV or use of laptops in bed; dark, cool, and quiet rooms)
Understand that the effect of a poor night's sleep (acute sleep restriction) before a match or training may not necessarily affect athletic (exercise) performance. Theoretical principles and limited evidence would suggest it is more likely to affect illness and injury occurrence
Avoid early morning training sessions following sleep disruption where possible, as these can be more detrimental to muscle strength and power performance than late bedtimes
Be aware that poor sleep prior to training could influence motivation and may hinder both cognitive- and physiological-induced training adaptations
Where possible, align training sessions to game times to adjust circadian rhythms. However, such practices have logistical issues and should not be at the risk of the quality of training
Practitioners, where possible, should supplement this understanding of sleep loss and performance with an increased knowledge of the relationship between sleep and recovery. Despite a widely held assumption that sleep is crucial for recovery, the interaction between sleep and recovery remains poorly understood. Limited evidence indicates sleep has a role to play in athletic recovery; however, the mechanisms behind this remain uncertain, so this assumption should be treated with caution

confirming the importance of sleep to physiological and psychological recovery. In particular, evidence of the role and importance sleep plays within the professional sporting environment during various scenarios is lacking. Thus, although sport science personnel and researchers should be aware of the complex effects of sleep loss on athletic performance, such knowledge needs to be supplemented with sufficient understanding of sleep's role in recovery, and possible sleep hygiene strategies to alleviate these issues. Accordingly, future examination of the evidence of sleep and the potential role it may play in recovery for athletes is warranted.

7 Conclusion

Although sleep is generally considered critical for human and athletic performance, there are mixed results regarding objective performance decrements in the current scientific literature. Individual athletes appear to lose sleep just prior to competing or if forced to train at early times; however, evidence for such instances in team sports is lacking. Exercise performance seems to be negatively affected during periods of SD (specifically endurance and repeated exercise bouts), although conflicting results exist for the effect of acute SR, as performance during maximal one-off efforts (in particular for maximal strength) is generally maintained. Possible reasons for these differences could be due to contrasting research designs and statistical power. The effects of sleep loss on physiological responses to exercise could potentially hinder muscular recovery and lead to a reduction in immune defense, although this still remains speculative. The majority of studies focusing on sleep loss and cognitive performance and mood responses have found detriments to most aspects of cognitive function (i.e. RT) and mood stability, results that potentially could hinder the neurocognitive components of many sports. Despite common assumptions around the importance of sleep, the lack of scientific evidence (especially in elite athletes) suggests future research into the examination of sleep and athletic performance is warranted.

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