Sleep and Nutrition Interactions

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Sleep disturbances and short sleep duration are behavioural risk factors for inflammation, associated with increased risk of illness and disease, which can be modified to promote sleep health. For sleep to have a restorative effect on the body, it must be of adequate duration and quality; particularly for athletes whose physical and mental recovery needs may be greater due to the high physiological and psychological demands placed on them during training and competition. Sleep has been shown to have a restorative effect on the immune system, the endocrine system, facilitate the recovery of the nervous system and metabolic cost of the waking state and has an integral role in learning, memory and synaptic plasticity, all of which can impact both athletic recovery and performance.

Keywords: sleep ; athletes ; chrononutrition

1. What is Sleep?

Sleep, in humans, is defined as a complex reversible behavioural state where an individual is perceptually disengaged from and unresponsive to their environment [1]. Sleep architecture has two basic states based on physiological parameters: non-rapid eye movement sleep (NREM) and rapid eye movement (REM) sleep [2]. Sleep stages fall along a continuum from fully awake to deep sleep [3]. NREM has been defined as “a relatively inactive yet actively regulating brain in a moveable body” [2], (p.17). In terms of brain activity, the electroencephalogram (EEG) pattern of NREM sleep is commonly described as synchronous (increasing depth of sleep is indicated by progressive dominance of high voltage, low frequency EEG patterns), with characteristic waveforms (sleep spindles, K-complexes and high voltage waves) [2]. NREM is usually associated with minimal or fragmented mental activity. **Table 1** shows the traditional four stages of NREM which are associated with differing levels of depth of sleep, with arousal thresholds generally lowest in Stage 1 and highest in Stage 4 sleep [2].

Table 1. Characteristics of NREM Sleep. [4]

Stage	Characteristics
1	Sleep is easily discontinued (e.g., noise, a light touch, etc.) Sleep is easily interrupted Key role in the initial wake to sleep transition Transitional stage throughout the sleep cycle
2	More intense stimuli required to produce arousal (e.g., bright light or loud noise) Indicated by K-complexes or sleep spindles in the EEG High voltage slow wave EEG activity will become apparent
3	High voltage (75 μV) slow wave (two cycles per second [cps]) activity that is ≥ 20% but < 50% of EEG activity
4	High voltage slow wave activity is ≥ 50% of EEG activity.

In contrast, REM sleep is defined by EEG activation, muscle atonia (paralysis) and episodic bursts of rapid eye movement [2]. REM sleep is associated with cognitive activity, while brain stem mechanisms inhibit spinal motor neurons limiting

movement. Hence, REM sleep has been defined as “an activated brain in a paralysed body” [2], (p.16). It should be noted that the American Academy of Sleep Medicine (AASM) have recommended alternative terminology for Sleep staging. Wake is referred to as W, NREM sleep is referred to as N and is divided into three stages: N1 – Stage 1, N2 – Stage 2 and N3 – Slow Wave Sleep or Deep Sleep, i.e., Stage 3 and 4 combined; while REM is referred to as R [5].

Sleep health is a multidimensional pattern of sleep-wakefulness adapted to individual, social and environmental demands, which promotes physical and mental wellbeing [6]. Good sleep health is characterised by satisfaction, appropriate timing, adequate duration, high efficiency and sustained alertness during waking hours [6]. Sleep deprivation adversely affects glucose metabolism and neuroendocrine function which can affect carbohydrate metabolism, appetite, energy intake and protein synthesis [1]. These factors may negatively impact an athlete’s nutritional, metabolic and endocrine status impacting athletic performance and recovery [1], (e.g., impaired glucose metabolism could reduce glycogen repletion while impaired protein synthesis could reduce recovery and adaptation from training).

1.1. How and Why Sleep Occurs

The brain is essentially an electrical system with circuits that switch on and off to promote either wakefulness or sleep. Since the arousal and sleep-promoting systems are mutually inhibitory, a sleep switch or ‘flip-flop’ model has been proposed [7]. A flip-flop switch contains mutually inhibitory elements where activity in one of the competing sides shuts down inhibitory inputs from the other side producing two discrete states with sharp transitions [8]. Activation of arousal systems inhibits sleep active neurons facilitating sleep while activation of sleep-promoting neurons inhibits arousal-related neurons reinforcing consolidated sleep episodes providing a mechanism for stabilisation of sleep and waking states [9].

The circadian rhythm in humans has been estimated in young males (24.18 ± 0.04 h; PCV 0.54%) and older adults (24.18 ± 0.04 h; PCV 0.58%), low percentage coefficients of variation and no significant difference between the groups indicated a small range variability in circadian rhythms [10]. Humans however, typically display individual differences in their behaviour (e.g., social activities, daytime activities and sleep). Chronotype is the expression of individual circadian rhythmicity and has been categorised as follows: morning types, intermediate types and evening types [11]. Chronotype is, in part, genetic but cultural and environmental factors also affect an individual’s sleep pattern. Research in the general population has demonstrated that most people are intermediate types (70%) with the remainder being either morning types (14%) or evening types (16%) [12].

Sleep is a dynamic process largely regulated by two factors; the circadian systems and the sleep homeostat. The Two Process Model for Sleep Regulation was developed to illustrate the interaction of the homeostatic sleep drive (sleep pressure or urge to sleep that accumulates during wakefulness) and the circadian system (endogenous timing system) in the timing and duration of sleep [13][14]. The homeostatic process (S) is a function of sleep and waking, while the circadian process (C) is controlled by a circadian oscillator [10]. S increases during waking and declines during sleep and it interacts with C, which is independent of sleep and waking and receives cues (e.g., light) from the environment [13][14]. The suprachiasmatic nucleus (SCN) in the brain is central to this process but secondary clock systems have been identified throughout the body [14].

Process S is an endogenous mechanism, relying on exogenous cues to regulate it to approximately 24 h. Process S represents sleep debt which increases during waking and reduces during sleep within a range that oscillates within a period that is normally entrained to day and night by process C [14]. When S reaches the lower boundary of the range, awakening is triggered and when S reaches the upper boundary sleep is triggered [14]. In terms of process C, the Two-Process Model focuses on time-of-day effects on sleep propensity, specifically that sleep propensity is minimal near midday and is strongly promoted in the early hours of the morning [13]. This circadian rhythmicity in sleep propensity is combined with S by C dictating the threshold values at which S transitions from sleep to wake, and vice versa [13][14]. Core body temperature and melatonin rhythms are markers of C [11]. The SCN has melatonin receptor cells, as darkness falls, melatonin is secreted by the pineal gland making the individual sleepy [15]. Animal studies have demonstrated that exogenous melatonin and ramelteon (an MT1/MT2 melatonin receptor agonist) function as non-photic entrainers, which phase advance the SCN [16]. A Three-Process Model of Sleep Regulation has also been proposed whereby sleepiness and alertness are stimulated by the combined action of a homeostatic process, a circadian process and sleep inertia process, the model has been extended to include sleep onset latency (the length of time of the transition from wakefulness to sleep), sleep length and performance [17].

Sleep has a restorative effect on the immune system and the endocrine system, facilitates the recovery of the nervous and metabolic cost of the waking state and has an integral role in learning, memory and synaptic plasticity (ability of synapses to strengthen or weaken over time) [18][19]. Sleep, particularly slow wave sleep (or N3) early in the night promotes prolactin release, while the anti-inflammatory actions of cortisol and catecholamines are reduced [18]. Acute

sleep deprivation and sleep disturbance (short sleep duration or reduced sleep efficiency) impair adaptive immunity which is associated with reduced response to vaccinations and increased vulnerability to infectious diseases, attributed to reduced growth hormone release during deep sleep and increased sympathetic output [20]. Tumour necrosis factor-α (TNFα) along with other cytokines are considered key to the regulation of sleep in normal physiological conditions [21]. Research has demonstrated that sleep disturbance (i.e. insomnia) and extremes of sleep durations affect risk factors of inflammatory disease and contribute to all-cause mortality [18][22]. Increased levels of circulating inflammatory markers (i.e., C-reactive protein [CRP] and Interleukin-6 [IL-6]) predict body mass gain in older adults [23] and type 2 diabetes [24]. Sleep disturbance is believed to have proximal effects on IL-6, which induces CRP [18], therefore, increases in CRP may be attributed to persistent or severe sleep disturbance. In a recent meta-analysis sleep disturbance (i.e., poor sleep quality, insomnia) was associated with increased levels of IL-6 (ES: 0.20 (0.08–0.31)) and CRP (ES: 0.12 (0.05–0.19)) [18]. Short sleep duration (< 7 h per night) was associated with increased IL-6 (ES: 0.29 (0.05–0.52)), while long sleep duration (> 8 h per night) was also associated with increased IL-6 (ES: 0.11 (0.02–0.20)) but also increased CRP (ES: 0.17 (0.01–0.34)) [18]. Similarly, a meta-analysis of sleep duration and all-cause mortality demonstrated a U-shaped association, whereby long sleep (> 8 h per night) has a 30% (RR: 1.30 (1.22–1.38)) greater risk while short sleep (<7 h per night) has a 12% (RR: 1.12 (1.06–1.18)) greater risk compared to normal sleep reference (7–8 h per night) [25].

Inappropriate timing of lifestyle behaviours can cause disruption to the circadian rhythm, resulting in an altered physiological response (e.g., poor sleep). Lifestyle factors (e.g., caffeine consumption, alcohol consumption and timing of sleep) can cause alterations in environmental cues which may negatively impact circadian rhythms and in turn result in negative physiological consequences [26]. The SCN receives environmental cues such as the light-dark cycle and additional information from other areas of the brain (e.g., when we eat or exercise). Give that Process C can be modified by exogenous cues [27], there is scope for investigation of nutrition interventions to enhance sleep quality and quantity. Similarly, the effect of nutrition interventions that promote athlete recovery on sleep quality and quantity should be investigated.

2. Sleep and Athletes

The classic view of sleep is that it is a recovery process, with the circadian system regulating feelings of sleepiness and wakefulness throughout the day [28]. Cognition, tissue repair and metabolism are critical psychological and physiological factors that contribute to training capacity, recovery and ultimately performance [28]. The relationship between sleep, performance and recovery can be viewed in terms of 3 key factors that affect the recuperative outcome:

- Sleep length (total sleep duration; hours/night, plus naps)
- Sleep quality (i.e., the experience and perceived adequacy of sleep)
- Sleep phase (circadian timing of sleep) [28].

Post-exercise recovery is vital for all athletes. If the balance between training stress and physical recovery is inadequate, performance in subsequent training sessions or competition may be adversely affected [15]. Muscle fatigue or soreness may adversely affect sleep, with inflammatory cytokines linked to disruption of normal sleep [29]. Inadequate recovery can reduce autonomic nervous system (ANS) resources, with an associated reduction in heart rate variability (HRV) and increased resting heart rate [30]. Sleep deprivation is associated with increased catabolic and reduced anabolic hormones which results in impaired muscle protein synthesis [31], blunting training adaptations and recovery.

Sleep disturbances and inadequate sleep duration have been reported in athletic populations. Assessment of the sleep patterns of professional male ice hockey players (*n* = 23) using polysomnography (PSG), demonstrated mean total sleep duration was 6.92 h; 95% CI 6.3–7.5 h [32]. Similarly, sleep was self-reported as the most important recovery modality utilised by South African athletes (*n* = 890; international *n* = 183, national *n* = 474, club *n* = 233) [15]. While a similar study found that 66% (*n* = 416) of elite German athletes (*n* = 632) reported pre-competition insomnia symptomology including difficulty falling asleep, waking during the night and early final waking times [33]. Sleep duration (< 8 h) has been identified as the strongest predictor of injury in adolescent athletes (RR = 2.1; 95% CI: 1.2–3.9) [34]. The Karolinska Athlete Screening Injury Prevention (KASIP) study investigated injury occurrence in Swedish adolescent elite athletes (*n* = 340; 178 males and 162 females) and demonstrated that athletes sleeping >8 h were less likely to suffer an injury (OR: 0.39; 95% CI 0.17–0.96) [35]. The aetiology of sleep disturbances is unclear during periods of intense training, it is unclear whether poor sleep is a symptom of overtraining, or intense training negatively affects sleep and recovery [30]. Sleep also has a pivotal role to play in performance, training adaptations and recovery [1][18]. Given the importance of sleep for athlete recovery, further research is warranted to investigate potential nutritional interventions to promote improved sleep quality and/or duration and recovery in athletes.

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