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## Sleep and Recovery in the Injured Athlete: A Comprehensive Narrative Review

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## **Abstract**

Sleep is increasingly recognized as a fundamental pillar of athletic health, yet its role in the specific context of injury rehabilitation remains under-emphasized in clinical practice. This narrative review synthesizes current literature regarding the bidirectional relationship between sleep and sports injuries. We examine how the neuro-endocrine system, specifically the GH-IGF-1 axis and cortisol-testosterone balance, dictates the speed of tissue remodeling. Furthermore, we analyze the "vicious cycle" of pain-induced sleep fragmentation and sleep-loss-induced hyperalgesia. Finally, we explore the immunological shift during sleep deprivation that promotes chronic inflammation. Our results suggest that optimizing sleep through hygiene, nutrition, and psychological support is essential for accelerating return-to-play timelines and preventing secondary injuries.

**Keywords:** sports injuries, sleep deprivation, athletic rehabilitation, central sensitization, neuroendocrinology

## **1. Introduction: The Biological and Economic Burden of Injury**

Sleep is not merely a passive state of rest but a highly active, homeostatically regulated period of physiological reconstruction. For the elite athlete, whose body is subjected to repetitive mechanical and metabolic stress, sleep serves as the primary window for systemic "resetting." The complexity of sleep architecture, involving the cyclical transition between Non-Rapid Eye Movement (NREM) and Rapid Eye Movement (REM) stages, ensures that both the neurological and musculoskeletal systems undergo repair [1–6].

Sports injuries represent a significant burden, not only for the athlete's health but also for the economic and competitive stability of sports organizations. The loss of a key player can lead to a decrease in team performance and significant financial losses in professional leagues. Despite

this, the focus of rehabilitation is often limited to physical therapy and surgical interventions, neglecting the "third pillar" of recovery: sleep. Experimental evidence underscores that even moderate sleep debt significantly degrades cognitive processing [7], memory consolidation [8], and emotional regulation [9]. These deficits can impair the athlete's ability to engage effectively with complex rehabilitation exercises and maintain the psychological resilience required for long-term recovery.

Furthermore, sleep deficiency interferes with vital restorative mechanisms, including cellular repair and tissue growth [10], while simultaneously disrupting glucose metabolism [11] and compromising immune vigilance [12, 25]. Collectively, these disruptions impair the body's adaptive capacity, significantly hindering the physiological recovery necessary for optimal athletic function.

## **2. Sleep Architecture and the Neuro-Endocrine Basis of Healing**

To understand the impact of sleep on injury rehabilitation, one must examine the specific stages of sleep and their unique contributions to biology. Normal human sleep follows a cyclical pattern of approximately 90-minute intervals. NREM sleep, specifically Stage 3 (Slow-Wave Sleep or SWS), is characterized by high-amplitude delta waves and is widely regarded as the most critical period for physical recovery [1, 29].

### **2.1. The GH-IGF-1 Axis: The Molecular Engine of Repair**

The secretion of Human Growth Hormone (hGH) is arguably the most vital endocrine event for an injured athlete. Czeisler and Klerman identified that GH release is not constant but follows a strict pulsatile rhythm, with the largest pulse occurring immediately after the onset of SWS [10].

**Pulsatile Mechanism:** Up to 70% of the daily GH quota is released during these nocturnal pulses. Van Cauter et al. demonstrated that when sleep is fragmented—common in athletes experiencing post-injury pain—the amplitude of these GH pulses is significantly blunted [30].

**Role in Tissue Regeneration:** GH stimulates the liver and local tissues to produce Insulin-like Growth Factor 1 (IGF-1). This hormone acts as a potent mitogen, stimulating the proliferation of satellite cells in skeletal muscle and the synthesis of collagenous matrix in tendons and ligaments. Without adequate SWS, the body remains in a state of "anabolic failure," where the rate of tissue degradation exceeds the rate of repair [15, 31, 32].

## **2.2. Testosterone-Cortisol Dynamics and Muscle Atrophy**

Injury often leads to localized muscle atrophy due to disuse. The neuro-endocrine environment during sleep determines the severity of this muscle loss.

**Anabolic Support:** Testosterone levels rise during sleep, reaching a peak toward the end of the first REM cycle. Testosterone is essential for maintaining muscle mass and promoting bone mineral density repair [31].

**Catabolic Stress:** Cortisol, the primary catabolic hormone, follows a circadian rhythm where it should reach its nadir (lowest point) around midnight. Spiegel et al. proved that sleep restriction (even to 4 hours) causes evening hypercortisolemia [11]. For an injured athlete, high cortisol levels combined with low testosterone creates a "catabolic storm" that accelerates muscle protein breakdown and inhibits the formation of new contractile fibers [31, 39].

## **3. The Vicious Cycle: Pain, Sleep, and Central Sensitization**

Pain is the most frequently cited barrier to sleep in the clinical setting of sports medicine. For the injured athlete, the inability to achieve restorative sleep is rarely the result of a single factor; rather, it is a consequence of acute physiological trauma combined with neurobiological shifts in how the brain processes noxious stimuli. Acute trauma—such as ligamentous tears, bone fractures, or post-surgical wounds—triggers a continuous stream of nociceptive signals that invade the sleep state, causing frequent micro-awakenings and a catastrophic shift in sleep architecture [9, 25, 33].

### **3.1. Impact on Sleep Micro-Architecture and Continuity**

The primary effect of post-injury pain is the fragmentation of sleep. Nociceptive signals act as internal "alarms" that trigger the ascending reticular activating system (ARAS), forcing the brain out of deep, regenerative stages and into lighter stages of NREM sleep (N1 and N2) or full wakefulness.

**Reduction of Slow-Wave Sleep (SWS):** As discussed in previous chapters, SWS is vital for tissue repair. Pain significantly reduces the total duration of SWS by preventing the brain from sustaining the high-amplitude delta waves necessary for this stage. This results in a state of "non-restorative sleep," where the athlete may be unconscious for 8 hours but fails to achieve the biological depth required for recovery [33].

**Micro-arousals and Adrenergic Drive:** Pain-induced micro-arousals lead to an increase in sympathetic nervous system activity during the night. Instead of the parasympathetic

dominance usually seen in sleep, the athlete remains in a state of low-level "fight or flight," which elevates heart rate and prevents the metabolic downregulation essential for cellular cleaning and repair [9, 25].

### 3.2. The Bidirectional Nature of the Pain-Sleep Interaction

One of the most critical findings in modern pain science is that the relationship between sleep and pain is not a one-way street. While pain undoubtedly disrupts sleep, recent longitudinal studies indicate that **sleep deprivation is an even stronger predictor of next-day pain than the other way around** [33, 34].

This bidirectional link creates a "vicious cycle":

1. The injury causes pain, which fragments sleep.
2. The fragmented sleep increases the athlete's sensitivity to pain the following day.
3. Increased pain leads to higher levels of stress and more sleep disruption.

This cycle can lead to a state where the athlete's recovery stalls, not because the tissue isn't healing, but because the nervous system has become hyper-reactive.

### 3.3. Neurobiological Mechanisms of Central Sensitization

The core of this "vicious cycle" lies in a process called **Central Sensitization**. Sleep restriction, even if moderate, causes a profound dysregulation in the body's endogenous pain-modulating systems.

**Impairment of Descending Inhibitory Pathways:** Under normal conditions, the brain has an "internal pharmacy" that sends signals down the spinal cord to "mute" or dampen incoming pain signals (descending inhibition). Sleep deprivation severely impairs these pathways. As a result, the "volume" of the pain signal reaching the brain is turned up, a phenomenon known as sleep-loss-induced hyperalgesia [34, 35].

**Neurochemical Shifts:** Studies using meta-analytical data [36] suggest that sleep loss alters the balance of neurotransmitters involved in nociception. Specifically, the downregulation of dopamine and the dysregulation of adenosine receptors in the brain make the athlete more vulnerable to spontaneous pain and mechanical allodynia (where even light touch or movement during physical therapy feels painful).

**Pro-inflammatory Priming:** Sleep loss primes the glial cells in the central nervous system to release pro-inflammatory cytokines (such as IL-1 $\beta$  and TNF- $\alpha$ ). These chemicals lower the firing threshold of pain-sensing neurons, effectively "sensitizing" the entire nervous system to the injury site [9, 36].

### **3.4. Clinical Implication: The "Analgesic Ceiling"**

For sports medicine practitioners, understanding this cycle is vital for medication management. Athletes experiencing sleep-loss-induced hyperalgesia often show a reduced response to standard analgesics, including NSAIDs and even opioids. This is because the pain is no longer just "in the tissue" (peripheral), but is being amplified by a sleep-deprived brain (central).

Breaking the cycle requires a dual approach: managing the peripheral pain at the injury site while simultaneously utilizing sleep-promoting interventions (such as cognitive behavioral therapy for insomnia or non-pharmacological sleep hygiene) to restore the brain's natural ability to modulate pain [33, 34]. Without addressing the sleep component, the rehabilitation process may be perceived as failing, leading to psychological frustration and an increased risk of chronic pain development.

**Molecular Mechanisms:** Sleep restriction alters somatosensory processing and reduces the effectiveness of descending inhibitory pathways in the spinal cord. This results in "central sensitization," where the central nervous system becomes hyper-reactive to pain signals [9, 36]. Breaking this cycle is paramount; without addressing sleep quality, analgesic medications may lose efficacy, and the rehabilitation process may be perceived as excessively burdensome by the athlete.

## **4. Immune Function and the Inflammatory Response: The Molecular Mending**

The initial phase of any musculoskeletal injury is characterized by a tightly regulated inflammatory cascade. While inflammation is often viewed negatively in a clinical context, it is a prerequisite for tissue regeneration. Sleep acts as a master-regulator of this process via a complex mechanism known as "sleep-immune crosstalk" [25, 37].

### **4.1. The Circadian Rhythm of Cytokines**

Sleep deprivation induces a profound shift in the timing and magnitude of the immune response. Under normal conditions, the early stages of sleep (SWS) promote a pro-inflammatory environment that is localized to the site of injury, facilitating the clearance of necrotic debris.

**Adaptive vs. Innate Immunity:** Sleep enhances the adaptive immune response by promoting the production of antigen-specific T-cells and regulating the release of Interleukin-12. However, chronic sleep restriction shifts the balance toward the innate immune system, triggering a state of **systemic low-grade inflammation** [12, 38].

**The Pro-inflammatory Storm:** Studies have shown that sleep loss activates cellular inflammatory signaling via the  $\text{TNF-}\alpha$  pathway. This leads to an overproduction of circulating pro-inflammatory cytokines, specifically **Interleukin-6 (IL-6)** and **Tumor Necrosis Factor-alpha (TNF- $\alpha$ )**. For the injured athlete, this means that instead of a localized, constructive inflammatory phase, the body enters a state of systemic stress that can damage healthy adjacent tissues and delay the onset of the proliferative phase [38, 39].

#### 4.2. Impact on the Proliferative and Remodeling Phases

The transition from clearing damage to building new tissue (collagen synthesis and myofibril formation) is highly dependent on a stable immunological environment.

**Inhibition of Mending:** Persistent inflammation, fueled by sleep debt, prevents fibroblasts from effectively synthesizing the extracellular matrix. Experimental evidence indicates that sleep deprivation negatively affects muscle protein synthesis pathways (e.g., the mTOR pathway), skewing the muscle's metabolic environment toward degradation rather than restoration [13, 25].

**Leukocyte Trafficking:** Sleep influences the "homing" of immune cells to the injury site. Without restorative sleep, the recruitment of regenerative macrophages is impaired, leading to the formation of suboptimal, weaker scar tissue which significantly increases the risk of recurrent injury upon return-to-play [15, 28, 39].

### 5. Psychological Load and Circadian Misalignment: The Invisible Barrier

The psychological burden of an injury is often the most significant yet least addressed factor in an athlete's recovery. This load directly interferes with the neurobiology of sleep, creating a state of "cognitive hyperarousal" that is antithetical to the initiation of the sleep state [19, 23, 25].

#### 5.1. The Athletic Identity Crisis and Autonomic Dysregulation

For many elite athletes, their self-worth is inextricably linked to their physical performance. An injury triggers an "identity crisis," resulting in clinical levels of emotional distress and anxiety.

**Sympathetic Dominance:** This anxiety keeps the **Sympathetic Nervous System (SNS)** in a state of dominance long into the nocturnal period. The physiological "switch" to Parasympathetic (vagal) dominance, which is required for heart rate variability (HRV) to increase and sleep to commence, remains stuck. This prevents the athlete from reaching the

deeper, more restorative stages of sleep, even if the total duration of sleep seems adequate [9, 21, 23, 39].

## 5.2. Disruption of the Two-Process Model of Sleep Regulation

According to the "Two-Process Model," sleep is governed by Process S (Homeostatic Sleep Pressure) and Process C (Circadian Rhythm). Injury disrupts both.

**Failure of Process S:** Homeostatic pressure is built through physical and metabolic exertion (the accumulation of adenosine in the brain). A sudden transition to forced inactivity means the athlete does not accumulate sufficient sleep pressure, leading to long sleep onset latency and frequent nighttime ruminations [1, 2, 22].

**The Molecular Clock and Transcriptome:** At a cellular level, myocytes and fibroblasts possess "molecular clocks" that synchronize repair processes. Circadian misalignment—exacerbated by a lack of social "zeitgebers" (like team training sessions) and reduced exposure to natural morning light—leads to a **desynchronization of the transcriptome** [4]. This means the genes responsible for tissue repair may be "turned off" when they should be most active, effectively slowing the biological speed of recovery [3, 4, 14, 35].

| Physiological System                 | Normal Restorative Function during Sleep                                                                      | Pathological Shift due to Sleep Deprivation                                                                       |
|--------------------------------------|---------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------|
| <b>GH-IGF-1 Axis</b>                 | Pulsatile GH release during SWS stimulates IGF-1, promoting protein and collagenous matrix synthesis.         | Blunted GH pulses lead to "anabolic failure," where tissue degradation exceeds the rate of repair.                |
| <b>Testosterone-Cortisol Balance</b> | Testosterone levels rise while cortisol reaches its nadir around midnight, promoting muscle mass maintenance. | Evening hypercortisolemia and low testosterone create a "catabolic storm," accelerating muscle protein breakdown. |

|                                           |                                                                                                            |                                                                                                                                                     |
|-------------------------------------------|------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Pain Modulation</b>                    | Descending inhibitory pathways in the central nervous system dampen incoming nociceptive signals.          | Impairment of descending pathways causes central sensitization and sleep-loss-induced hyperalgesia.                                                 |
| <b>Immune &amp; Inflammatory Response</b> | Enhances adaptive immunity and promotes a localized pro-inflammatory environment to clear necrotic debris. | Shifts toward innate immunity, triggering systemic low-grade inflammation (increased IL-6 and TNF- $\alpha$ ) and delaying the proliferative phase. |

## 6. Monitoring and Multidisciplinary Interventions: Practical Frameworks

To move beyond theoretical understanding, sports medicine must adopt objective, evidence-based interventions that treat sleep as a clinical metric during rehabilitation.

### 6.1. Objective vs. Subjective Monitoring

Relying solely on an athlete's self-report of sleep quality is often misleading, as injured athletes may overestimate their sleep duration.

**Actigraphy and PSG:** While Polysomnography (PSG) is the gold standard, **wrist-worn actigraphy** provides a practical, non-invasive method for longitudinal tracking. It allows clinicians to monitor sleep efficiency and the timing of sleep cycles throughout the multi-week rehabilitation process [17, 21, 24].

**Screening Tools:** The use of validated questionnaires, such as the **Athlete Sleep Behavior Questionnaire (ASBQ)** or the **Pittsburgh Sleep Quality Index (PSQI)**, can help identify athletes who are at high risk for "rehabilitation failure" due to chronic sleep disturbances [21].

### 6.2. Targeted Nutritional Interventions

Nutrition serves as a potent, non-pharmacological modulator of sleep architecture.

**The Tryptophan-Serotonin Pathway:** Consuming high-glycemic-index (GI) carbohydrates in the evening (approx. 4 hours before bed) increases the ratio of tryptophan to large neutral

amino acids in the blood, facilitating the production of serotonin and subsequently melatonin [26, 27].

**Exogenous Melatonin and Tart Cherry:** Supplementing with tart cherry juice provides both natural melatonin and anti-inflammatory polyphenols. Studies have demonstrated that this can reduce systemic markers of muscle damage and improve total sleep duration, making it an ideal "sleep-recovery" cocktail for post-traumatic phases [39].

### 6.3. Environmental and Behavioral Optimization

Clinical environments (like rehabilitation centers or post-surgical wards) are often hostile to sleep. Optimization must include:

**Light Hygiene:** Blocking blue light from electronic devices at least 90 minutes before bed is essential to prevent the suppression of endogenous melatonin [5, 26, 38].

**Cognitive Behavioral Therapy for Insomnia (CBT-I):** For athletes with prolonged recovery timelines, CBT-I has proven more effective than pharmacological aids in addressing the root causes of injury-related insomnia without the risk of grogginess or reduced reaction times the following day [23, 24, 39].

## 7. Discussion

The synthesis of evidence presented in this review underscores a fundamental paradigm shift in sports medicine: sleep must no longer be viewed as a passive consequence of recovery, but as an active, non-negotiable physiological driver of tissue rehabilitation. The bidirectional relationship between sleep and injury creates a complex biological feedback loop that can either accelerate or catastrophically delay an athlete's return to competition.

### 7.1. The "Anabolic Failure" Hypothesis

A central theme emerging from the analysis of the neuro-endocrine data (Chapter 2) is what could be termed "anabolic failure" during sleep deprivation. The synchronization of  $\text{hGH}$  pulses with Slow-Wave Sleep (SWS) is not a biological coincidence but a sophisticated evolutionary mechanism designed to repair the metabolic and mechanical damage incurred during wakefulness.

When an athlete is injured, the demand for  $\text{IGF}\text{-}1$  and protein synthesis increases exponentially to facilitate the bridging of torn ligaments or the remodeling of bone matrix. However, the data from Van Cauter [30] and Dattilo [31] suggest that the very trauma that creates this demand also disrupts the sleep architecture necessary to satisfy it. This paradox

represents a significant challenge in rehabilitation. If the "anabolic window" of the first SWS cycle is missed due to pain or anxiety, the body remains in a catabolic state dominated by cortisol, effectively "starving" the injury site of the hormonal signals required for repair.

## **7.2. The Pain-Sleep Interaction: A Double-Edged Sword**

The most striking clinical finding discussed in this review is the potency of sleep-loss-induced hyperalgesia (Chapter 3). Traditional orthopedic rehabilitation often focuses on the peripheral site of injury—the "hardware" of the body. However, the evidence from Finan et al. [34] suggests that sleep deprivation "reprograms" the central nervous system (the "software"), making it hypersensitive to nociceptive input.

This has profound implications for pharmacological management in sports medicine. If an athlete reports increasing pain despite tissue healing, clinicians might be tempted to increase analgesic dosages. However, the underlying issue may not be the injury itself, but the lack of sleep-mediated endogenous pain inhibition. By treating the sleep deficit rather than just the pain, practitioners can lower the athlete's pain perception more effectively and avoid the side effects of long-term NSAID or opioid use, such as gastrointestinal distress or disrupted sleep architecture.

## **7.3. The Inflammatory Paradox**

Another critical point for discussion is the role of the immune system. While the "PEACE & LOVE" protocol emphasizes managing inflammation, the molecular data (Chapter 4) suggests that sleep is the ultimate regulator of this process. The systemic "low-grade inflammation" triggered by sleep debt is particularly dangerous for elite athletes. Unlike the localized, acute inflammation necessary for healing, systemic inflammation (marked by elevated  $IL\text{-}6$  and  $TNF\alpha$ ) acts as a barrier to the proliferative phase.

This suggests that an athlete "pushing through" a rehabilitation program while sleep-deprived is not just working hard; they are actively working against their own biology. The persistent pro-inflammatory cytokine profile prevents the necessary shift to anti-inflammatory macrophage activity, potentially leading to the formation of "low-quality" scar tissue that lacks the tensile strength of the original fibers.

## **7.4. Psychological Resilience and Athletic Identity**

From a psychological perspective (Chapter 5), the "identity crisis" of the injured athlete cannot be overstated. The loss of daily training removes the primary homeostatic driver for sleep

(adenosine accumulation), while the stress of the injury activates the sympathetic nervous system.

The discussion must highlight that the mental health of an injured athlete and their sleep quality are inextricably linked. A "tired but wired" athlete—one who is physically inactive but mentally hyperaroused—is at the highest risk for developing chronic insomnia. This suggests that mindfulness-based interventions and cognitive behavioral strategies (CBT-I) should be considered as standard parts of the "physical" therapy plan, as they directly facilitate the parasympathetic state required for restorative SWS.

### 7.5. Limitations and Future Directions

Despite the strong physiological rationale presented, this review identifies a significant gap in clinical literature. Most research to date has focused on the impact of sleep on *performance* (e.g., speed, accuracy) or *injury prevention*. There is a surprising lack of large-scale, longitudinal clinical trials that specifically measure the rate of tissue healing (via MRI or ultrasound) in relation to objective sleep metrics in injured athletes.

Furthermore, the impact of "sleep extension" as a therapeutic intervention in post-surgical recovery remains largely untested in the athletic population. Future research should prioritize:

1. Quantifying the direct impact of sleep duration on the rate of bone mineral density recovery and ligamentous tensile strength.
2. Evaluating the efficacy of non-pharmacological sleep aids (e.g., tart cherry juice, magnesium, or weighted blankets) specifically during the acute post-traumatic phase.
3. Developing "sleep-integrated" rehabilitation apps that track both physical therapy progress and sleep efficiency to provide a holistic recovery score.

### 7.6. Practical Recommendations

Based on the synthesized data, the following clinical recommendations are proposed:

- **Screening:** Implement the PSQI or ASBQ at the moment of injury diagnosis.
- **Education:** Educate athletes on the "Anabolic Window of Sleep" to increase compliance with sleep hygiene.
- **Environmental Control:** For hospitalized or home-recovering athletes, strictly limit blue light and noise during the 22:00–06:00 window.
- **Scheduling:** Avoid early-morning physiotherapy sessions for injured athletes to allow for "sleep-in" periods, which maximize the testosterone-heavy REM cycles of the late morning.

## **8. Conclusion**

Sleep is the biological foundation of the healing process. The neuro-endocrine, immunological, and psychological evidence presented in this review confirms that any rehabilitation program that ignores sleep is fundamentally incomplete. By restoring the GH-IGF-1 axis, dampening central pain sensitization, and regulating the inflammatory response, sleep provides the body with the necessary tools to transform a period of injury into a period of successful physiological adaptation. For the modern sports medicine practitioner, "Sleep Hygiene" should be prescribed with the same precision and urgency as any surgical or physiotherapeutic intervention.

### **After conclusions:**

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During the preparation of this work, the authors used generative AI to assist with grammar and stylistic editing to ensure appropriate academic language and for translation into English. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the final content of the manuscript.

## References

1. **Beersma DGM, Gordijn MCM.** Circadian control of the sleep–wake cycle. *Physiol Behav.* 2007;90(2–3):190–5.
2. **Le Meur Y, Duffield R, Skein M.** Sleep. In: Recovery for performance in sport. Champaign: Human Kinetics; 2012.
3. **Reilly T, Waterhouse J.** Sports performance: is there evidence that the body clock plays a role? *Eur J Appl Physiol.* 2009;106(3):321–32.
4. **Archer SN, Laing EE, Moller-Levet CS, et al.** Mistimed sleep disrupts circadian regulation of the human transcriptome. *Proc Natl Acad Sci U S A.* 2014;111(6):E682–91.
5. **Davenne D.** Sleep of athletes—problems and possible solutions. *Biol Rhythm Res.* 2009;40(1):45–52.
6. **Samuels C.** Sleep, recovery, and performance: the new frontier in high-performance athletics. *Neurol Clin.* 2008;26(1):169–80.
7. **Belenky G, Wesensten NJ, Thorne DR, et al.** Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. *J Sleep Res.* 2003;12(1):1–12.
8. **Walker MP, Stickgold R.** It’s practice, with sleep, that makes perfect: implications of sleep-dependent learning and plasticity for skill performance. *Clin Sports Med.* 2005;24(2):301–17.
9. **Haack M, Mullington JM.** Sustained sleep restriction reduces emotional and physical well-being. *Pain.* 2005;119(1-3):56–64.
10. **Czeisler CA, Klerman EB.** Circadian and sleep-dependent regulation of hormone release in humans. *Recent Prog Horm Res.* 1999;54:97–130.

11. **Spiegel K, Leproult R, Van Cauter E.** Impact of sleep debt on metabolic and endocrine function. *Lancet*. 1999;354(9188):1435–9.
12. **Lange T, Perras B, Fehm HL, et al.** Sleep enhances the human antibody response to hepatitis A vaccination. *Psychosom Med*. 2003;65(5):831–5.
13. **Tu H, Li YL.** Inflammation balance in skeletal muscle damage and repair. *Front Immunol*. 2023;14:1133355.
14. **Dubois B, Esculier J.** Soft-tissue injuries simply need PEACE and LOVE. *Br J Sports Med*. 2020;54(2):72-73.
15. **Chennaoui M, Vanneau T, Trignol A, et al.** How does sleep help recovery from exercise-induced muscle injuries? *J Sci Med Sport*. 2021;24(10):982-987.
16. **Milewski MD, Skaggs DL, Bishop GA, et al.** Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. *J Pediatr Orthop*. 2014;34(2):129-133.
17. **Roberts SSH, Teo WP, Warmington SA.** Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis. *Br J Sports Med*. 2019;53(8):513-522.
18. **Grier T, Dinkeloo E, Reynolds M, Jones BH.** Sleep duration and musculoskeletal injury incidence in physically active men and women: a study of U.S. Army Special Operation Forces soldiers. *Sleep Health*. 2020;6(3):344-349.
19. **Nédélec M, Leduc C, Dawson B, et al.** Case study: sleep and injury in elite soccer—a mixed method approach. *J Strength Cond Res*. 2019;33(11):3085-3091.
20. **Smith RS, Efron B, Mah CD, Malhotra A.** The impact of circadian misalignment on athletic performance in professional football players. *Sleep*. 2013;36(12):1999-2001.
21. **Gupta L, Morgan K, Gilchrist S.** Does elite sport degrade sleep quality? A systematic review. *Sports Med*. 2017;47(7):1317-1333.
22. **Lastella M, Vincent GE, Duffield R, et al.** Can sleep be used as an indicator of overreaching and overtraining in athletes? *Front Physiol*. 2018;9:436.
23. **Nédélec M, Halson S, Abaidia AE, et al.** Stress, sleep and recovery in elite soccer: a critical review of the literature. *Sports Med*. 2015;45(10):1387-400.
24. **Kelley GA, Kelley KS.** Exercise and sleep: a systematic review of previous meta-analyses. *J Evid Based Med*. 2017;10(1):26-36.
25. **Besedovsky L, Lange T, Haack M.** The sleep-immune crosstalk in health and disease. *Physiol Rev*. 2019;99(3):1325-1380.

26. Leeder J, Glaister M, Pizzoferro K, Dawson J, Pedlar C. Sleep duration and quality in elite athletes measured using wristwatch actigraphy. *J Sports Sci.* 2012;30(6):541-5. doi: 10.1080/02640414.2012.660188. Epub 2012 Feb 14. PMID: 22329779.
27. Lastella M, Roach GD, Halson SL, Sargent C. Sleep/wake behaviours of elite athletes from individual and team sports. *Eur J Sport Sci.* 2015;15(2):94-100. doi: 10.1080/17461391.2014.932016. Epub 2014 Jul 4. PMID: 24993935.
28. Lastella M, Roach GD, Halson SL, Martin DT, West NP, Sargent C. The impact of a simulated grand tour on sleep, mood, and well-being of competitive cyclists. *J Sports Med Phys Fitness.* 2015 Dec;55(12):1555-64. Epub 2014 Oct 6. PMID: 25286890.
29. Taylor SR, Rogers GG, Driver HS. Effects of training volume on sleep, psychological, and selected physiological profiles of elite female swimmers. *Med Sci Sports Exerc.* 1997 May;29(5):688-93. doi: 10.1097/00005768-199705000-00016. PMID: 9140908.
30. Richmond LK, Dawson B, Stewart G, Cormack S, Hillman DR, Eastwood PR. The effect of interstate travel on the sleep patterns and performance of elite Australian Rules footballers. *J Sci Med Sport.* 2007 Aug;10(4):252-8. doi: 10.1016/j.jsams.2007.03.002. Epub 2007 May 23. PMID: 17524795.
31. Melone MA, Tourny C, Gehlbach BK, Schmidt EL, Lalevée M, L'Hermette M. Prevalence and Risk Factors of Poor Sleep Quality in Collegiate Athletes during COVID-19 Pandemic: A Cross-Sectional Study. *Int J Environ Res Public Health.* 2022 Mar 6;19(5):3098. doi: 10.3390/ijerph19053098. PMID: 35270790; PMCID: PMC8910097.
32. Juliff LE, Halson SL, Peiffer JJ. Understanding sleep disturbance in athletes prior to important competitions. *J Sci Med Sport.* 2015 Jan;18(1):13-8. doi: 10.1016/j.jsams.2014.02.007. Epub 2014 Feb 13. PMID: 24629327.
33. Buckley TM, Schatzberg AF. On the interactions of the hypothalamic-pituitary-adrenal (HPA) axis and sleep: normal HPA axis activity and circadian rhythm, exemplary sleep disorders. *J Clin Endocrinol Metab.* 2005 May;90(5):3106-14. doi: 10.1210/jc.2004-1056. Epub 2005 Feb 22. PMID: 15728214.
34. Rice TB, Dunn RE, Lincoln AE, Tucker AM, Vogel RA, Heyer RA, Yates AP, Wilson PW, Pellmen EJ, Allen TW, Newman AB, Strollo PJ Jr; National Football League Subcommittee on Cardiovascular Health. Sleep-disordered breathing in the National Football League. *Sleep.* 2010 Jun;33(6):819-24. doi: 10.1093/sleep/33.6.819. PMID: 20550023; PMCID: PMC2881715.

35. Dumortier J, Mariman A, Boone J, Delesie L, Tobback E, Vogelaers D, Bourgois JG. Sleep, training load and performance in elite female gymnasts. *Eur J Sport Sci*. 2018 Mar;18(2):151-161. doi: 10.1080/17461391.2017.1389992. Epub 2017 Oct 26. PMID: 29072537.
36. Besedovsky, L., Lange, T. & Born, J. Sleep and immune function. *Pflugers Arch - Eur J Physiol* **463**, 121–137 (2012). <https://doi.org/10.1007/s00424-011-1044-0>
37. Venter, R.E. (2014), Perceptions of team athletes on the importance of recovery modalities. *European Journal of Sport Science*, 14: S69-S76. <https://doi.org/10.1080/17461391.2011.643924>
38. Bonnar, D., Bartel, K., Kakoschke, N. *et al.* Sleep Interventions Designed to Improve Athletic Performance and Recovery: A Systematic Review of Current Approaches. *Sports Med* **48**, 683–703 (2018). <https://doi.org/10.1007/s40279-017-0832-x>
39. Gosselin N, Lassonde M, Petit D, Leclerc S, Mongrain V, Collie A, Montplaisir J. Sleep following sport-related concussions. *Sleep Med*. 2009 Jan;10(1):35-46. doi: 10.1016/j.sleep.2007.11.023. Epub 2008 Jan 28. PMID: 18226956.