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Sleep Loss and Post-Exercise Muscle Recovery: Hormonal, Inflammatory, Metabolic and Circadian Mechanisms with Practical Implications for Athletes

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Abstract

Introduction and aim

Sleep is essential for skeletal muscle repair, endocrine regulation, immune balance, metabolic homeostasis and adaptation to exercise. In athletes and physically active individuals, insufficient sleep may impair post-exercise recovery and reduce physical performance. The aim of this review was to analyze the hormonal, inflammatory, metabolic and circadian

mechanisms through which sleep deprivation may affect skeletal muscle recovery after exercise.

Materials and methods

This narrative review was based on a literature search conducted in PubMed, Scopus, Web of Science and Google Scholar. The analysis included systematic reviews, meta-analyses, randomized and controlled human studies, experimental sleep restriction studies and selected animal or cellular studies that provided relevant mechanistic explanations. Publications concerning sleep deprivation, exercise recovery, skeletal muscle metabolism, hormonal regulation, inflammation, mitochondrial function, circadian rhythm and physical performance were included.

Results

Available evidence indicates that sleep deprivation may disturb the GH/IGF-1 axis, increase cortisol activity, alter testosterone secretion, promote inflammatory dysregulation, reduce myofibrillar protein synthesis, impair mitochondrial function, decrease insulin sensitivity and disrupt skeletal muscle circadian regulation. These mechanisms may contribute to impaired muscle repair, reduced recovery capacity and less favorable training adaptation.

Conclusions

Sleep should be regarded as an active component of post-exercise recovery rather than a passive period of rest. Sleep optimization, sleep extension before anticipated sleep loss, strategic napping, appropriate training load adjustment and adequate nutrition may support recovery in athletes and physically active individuals. Further studies are needed to clarify dose-response relationships and to identify the most effective recovery strategies in real-world sports settings.

Keywords: sleep deprivation; muscle recovery; exercise; growth hormone; cortisol; inflammation; protein synthesis; circadian rhythm; skeletal muscle; athletes

1. Introduction

Sleep is an essential biological process involved in physiological restoration, endocrine regulation, immune function, metabolic homeostasis and tissue repair. In athletes and physically active individuals, sleep represents a key component of post-exercise recovery, during which skeletal muscle remodeling, hormonal secretion, mitochondrial restoration and immune regulation occur [1,2]. Sleep should therefore be understood not only as a passive state of rest, but also as an active biological condition required for adaptation to physical training and maintenance of musculoskeletal health [1,3].

Insufficient sleep is common in modern society and may be particularly relevant in sport. Athletes may experience sleep restriction due to early training sessions, evening competitions, travel, psychological stress, academic or occupational duties and exposure to electronic devices [1,4]. Sleep loss has been associated with impaired endurance capacity, strength, reaction time, motor control and perceived exertion. Systematic reviews and meta-analyses indicate that acute sleep loss may negatively affect several domains of physical performance, including anaerobic power, strength, endurance, speed and sport-specific skills [5,6].

The mechanisms through which sleep deprivation may impair muscle recovery are multifactorial. They include disturbances in endocrine signaling, inflammatory regulation, protein turnover, mitochondrial function, insulin sensitivity, autonomic nervous system activity and circadian biology. These pathways may interact with each other and create a less favorable environment for skeletal muscle repair and training adaptation.

The aim of this review is to summarize the current evidence on the hormonal, inflammatory, metabolic and circadian mechanisms linking sleep deprivation with impaired skeletal muscle recovery after exercise. Particular attention is given to the GH/IGF-1 axis, cortisol and testosterone balance, inflammatory signaling, myofibrillar protein synthesis, mitochondrial function, peripheral circadian clocks and practical implications for athletes, coaches and sports medicine professionals.

2. **Sleep Architecture and Its Relevance to Muscle Recovery**

Normal sleep architecture comprises cyclical alternation between non-rapid eye movement (NREM) and rapid eye movement (REM) sleep stages. Slow-wave sleep (SWS), the deepest stage of NREM sleep, is of particular relevance to muscle recovery due to its association with peak growth hormone (GH) secretion, reduced sympathetic nervous system activity, and decreased cortisol output [7,8].

In normal young adults, approximately 70% of daily GH output occurs during early sleep, in temporal association with the first period of SWS [7]. A recent study published in Cell elucidated the neuroendocrine circuit underlying this phenomenon, demonstrating that GH release is regulated by sleep-wake-dependent activity of hypothalamic neurons expressing GH-releasing hormone (GHRH) and somatostatin (SST). During NREM sleep, moderately increased GHRH activity and decreased SST activity promote GH secretion, while during REM sleep, strong surges of both GHRH and SST activity are observed [9]. This circuit provides the mechanistic basis for the well-established link between sleep and anabolic hormone release.

When training volume and intensity increase, a corresponding increase in SWS is typically observed, suggesting a homeostatic relationship between exercise stress and restorative sleep [8]. However, a fine line exists between the promotion and disruption of sleep; overtraining, psychological stress, and competition schedules can fragment sleep architecture and reduce total sleep duration, thereby compromising the very recovery processes that athletes require [2,4].

3. Hormonal Mechanisms

3.1 The Growth Hormone/IGF-1 Axis

The GH/IGF-1 axis is a principal anabolic pathway governing skeletal muscle growth, repair, and protein synthesis. Sleep is the primary physiological stimulus for pulsatile GH secretion, and disruptions to sleep profoundly affect this axis [7,10].

Sustained sleep deprivation in animal models nearly abolishes high-amplitude GH pulses and suppresses circulating concentrations of both GH and IGF-1 [11]. In rats subjected to prolonged sleep deprivation using the disk-over-water method, GH, IGF-1, and prolactin were all significantly suppressed, while corticosterone remained relatively unaffected, pointing to

hypothalamic mechanisms as the primary locus of disruption [11]. In humans, 25 hours of total sleep deprivation significantly decreased circulating free IGF-1 concentrations, an effect that was reversible with one night of recovery sleep [12]. Importantly, this decrease in IGF-1 correlated negatively with increases in Toll-like receptor 4 (TLR-4) mRNA expression, suggesting a mechanistic link between inflammatory activation and IGF-1 axis suppression during sleep loss [12].

Sleep extension, conversely, has been shown to increase IGF-1 concentrations. In a randomized crossover study, six nights of extended sleep (10 hours time in bed) prior to total sleep deprivation resulted in significantly higher free and total IGF-1 levels at baseline, during 24 hours of sleep deprivation, and after recovery, compared to habitual sleep conditions [13]. These findings suggest that sleep banking may provide a protective buffer for the GH/IGF-1 axis against subsequent sleep loss.

The clinical significance of these hormonal changes for muscle recovery is underscored by the role of local IGF-1 in muscle regeneration. Chennaoui et al. proposed that sleep extension could potentially accelerate recovery from exercise-induced muscle injuries through increasing local IGF-1 and controlling local inflammation [2]. The GH/IGF-1 axis also interacts with the immune system through its anti-inflammatory properties, creating a bidirectional relationship between sleep, hormonal status, and immune function [10].

3.2 Cortisol and the Hypothalamic-Pituitary-Adrenal Axis

Cortisol, the primary glucocorticoid in humans, is a potent catabolic hormone that promotes protein degradation and inhibits protein synthesis in skeletal muscle. Sleep deprivation has been consistently associated with alterations in cortisol secretion patterns, though the magnitude and timing of these changes vary across studies.

Subchronic sleep restriction (five nights of 4 hours time in bed) increased 24-hour urinary free cortisol by 21% compared to normal sleep [14]. Time-of-day analyses have revealed that sleep restriction significantly increases cortisol in the afternoon, particularly in older men, while morning cortisol may be blunted [15,16]. This pattern suggests a time-of-day-dependent uncoupling of hypothalamic-pituitary-adrenal (HPA) axis regulation.

In the context of exercise-induced muscle damage, total sleep deprivation following eccentric exercise resulted in significantly higher cortisol concentrations and an elevated cortisol-to-total testosterone ratio compared to normal sleep conditions [17]. This shift in the cortisol/testosterone ratio is particularly relevant because it reflects the net anabolic-catabolic balance in skeletal muscle. An elevated ratio favors proteolysis and impairs the muscle's capacity for repair and remodeling.

The catabolic effects of elevated cortisol on skeletal muscle are mediated through several molecular pathways. Glucocorticoid signaling activates the ubiquitin-proteasome system through upregulation of two E3 ubiquitin ligases, MAFbx (atrogin-1) and MuRF1, leading to selective degradation of myosin heavy chain proteins [18]. Additionally, elevated cortisol inhibits IGF-1 expression, causing downregulation of the PI3K/Akt pathway, which in turn reduces activation of the mammalian target of rapamycin (mTOR) and p70S6 kinase—key determinants of cell size and ribosome biogenesis [18,19].

3.3 Testosterone

Testosterone is the principal anabolic hormone in males and plays a critical role in stimulating protein synthesis and inhibiting protein degradation in skeletal muscle. Sleep restriction has been associated with reductions in testosterone, though findings are not entirely consistent across studies.

A prospectively randomized crossover study in 35 healthy men demonstrated that overnight sleep deprivation had multiple effects on 24-hour testosterone secretion, with significant reductions in mean concentrations, basal secretion, total and pulsatile secretion, and pulse frequency [15]. These effects were most apparent in older men and occurred in the absence of detectable changes in luteinizing hormone (LH), suggesting a time-of-day-dependent uncoupling of the regulatory control of the testicular axis [15]. When epidemiological and interventional studies are considered collectively, sleep loss and shorter sleep duration are associated with lower morning, afternoon, and 24-hour testosterone [20].

Interestingly, in the study by Dáttilo et al. examining total sleep deprivation after eccentric exercise-induced muscle damage, no significant differences in testosterone levels were detected between sleep-deprived and normal sleep conditions [17]. This discrepancy may reflect differences in the duration and type of sleep manipulation, the concurrent effects of exercise on testosterone secretion, or the relatively short observation period. Nevertheless, the elevated cortisol/testosterone ratio observed in the sleep-deprived condition still indicates a net shift toward catabolism [17].

The reciprocal changes in testosterone and cortisol induced by sleep loss create what has been termed an "anabolic-catabolic imbalance" [20]. Liu and Reddy demonstrated that fixing this testosterone-cortisol balance through a novel dual-hormone clamp mitigated the induction of insulin resistance by sleep restriction, providing proof-of-concept that the metabolic harm from sleep loss can be ameliorated by approaches that do not require sleeping more [20].

4. Inflammatory Mechanisms

4.1 Systemic Inflammatory Markers

The relationship between sleep deprivation and systemic inflammation is complex and appears to depend on the duration, type, and chronicity of sleep loss. A landmark systematic review and meta-analysis of 72 studies ($n > 50,000$) found that sleep disturbance was associated with higher levels of C-reactive protein (CRP; effect size 0. 12) and IL-6 (effect size 0. 20), though neither experimental sleep deprivation nor sleep restriction was significantly associated with acute changes in CRP, IL-6, or TNF-α [21].

However, more recent evidence suggests that the inflammatory response to sleep loss may require sustained exposure. An updated meta-analysis of 35 experimental studies (887 participants) found that multiple nights of partial sleep deprivation (sleep duration reduced to approximately 4. 5 hours for 3 or more nights) were associated with significant increases in IL-6 ($d = 0. 42$) and CRP ($d = 0. 76$), while a single night of total or partial sleep deprivation was not associated with changes in inflammation [22]. This temporal pattern suggests that the upregulation of inflammatory proteins in blood may only manifest following persistent periods of sleep restriction.

In the specific context of post-exercise recovery, Dáttilo et al. found that total sleep deprivation after eccentric exercise-induced muscle damage selectively increased IL-6 without affecting TNF-α, IL-1β, IL-1 receptor antagonist, or IL-10 [17]. The elevation of IL-6

is noteworthy because this cytokine has dual roles: as a pro-inflammatory mediator when chronically elevated, and as a myokine with anti-inflammatory and metabolic regulatory functions when released acutely from contracting muscle. Sleep deprivation-induced IL-6 elevation may therefore disrupt the finely tuned inflammatory cascade required for orderly muscle regeneration.

Cullen et al. further demonstrated that sleep deprivation increased resting IL-6 concentrations, which were associated with lowered blood glucose, increased perception of effort, and impaired exercise performance [23]. Importantly, while resting IL-6 was elevated, the cytokine and neuroendocrine responses to exercise itself were not altered by sleep deprivation, suggesting that the primary impact occurs through changes in the basal inflammatory milieu rather than the exercise-induced response [23].

4.2 Skeletal Muscle Inflammatory Signaling

Beyond systemic markers, sleep loss induces inflammatory changes at the tissue level within skeletal muscle. Saner et al. examined plasma and skeletal muscle inflammatory markers following five nights of sleep restriction and found that while plasma inflammatory cytokines (IL-6, TNF-α, IFN-γ) did not change significantly, skeletal muscle protein content of NFAT1 (nuclear factor of activated T-cells 1) increased specifically in the sleep-restricted group [24]. NFAT1 is a transcription factor involved in immune cell activation and inflammatory gene expression, and its upregulation in muscle tissue suggests local inflammatory priming even in the absence of detectable systemic changes.

Transcriptomic analyses have provided deeper insight into the inflammatory landscape of sleep-deprived muscle. Lin et al. demonstrated that five nights of sleep restriction in healthy young males resulted in increased enrichment of inflammatory and immune response-related gene pathways in skeletal muscle, with concurrent decreased enrichment of pathways associated with mitochondrial function [25]. Notably, performing three sessions of high-intensity interval exercise (HIIE) during the sleep restriction period attenuated these transcriptional changes, with the direction of enrichment in inflammatory pathways occurring in the opposite direction in the exercise group [25].

Acute sleep loss also induces inflammatory gene expression at the whole-blood level. Chennaoui et al. showed that 25 hours of sleep deprivation significantly increased mRNA levels of TNF-α, its soluble receptor sTNF-R1, and TLR-4, while IL-6 mRNA remained unchanged [12]. Changes in TLR-4 expression correlated positively with TNF-α and sTNF-R1 and negatively with circulating free IGF-1, suggesting that TLR-4-mediated inflammatory signaling may contribute to IGF-1 axis suppression during sleep loss [12].

4.3 Inflammation and Muscle Regeneration Phases

Muscle regeneration following exercise-induced injury proceeds through sequential phases: degeneration, inflammation, regeneration, remodeling, and maturation [2]. Each phase requires precisely coordinated inflammatory signaling. The initial inflammatory phase involves neutrophil infiltration and pro-inflammatory macrophage (M1) activation, which clear damaged tissue. This is followed by a transition to anti-inflammatory macrophages (M2) that promote satellite cell proliferation and myofiber regeneration.

Sleep deprivation may disrupt this orderly progression. In a mouse model, Yang et al. demonstrated that 72 hours of sleep deprivation combined with high-intensity exercise resulted in elevated TNF-α levels and increased creatine phosphokinase and aspartate aminotransferase activities, indicating exacerbated muscle impairment [26]. The combination of exercise-induced damage and sleep deprivation appeared to have synergistic deleterious effects, as neither sleep deprivation alone nor exercise alone produced the same degree of muscle injury markers [26].

Animal studies have further revealed that sleep deprivation induces histopathological changes in skeletal muscle, including tissue degeneration, inflammatory infiltrate, and tissue edema, with type I (slow-twitch, oxidative) fibers showing greater susceptibility to damage than type II (fast-twitch, glycolytic) fibers [27]. The increased oxidative damage in type I fibers, evidenced by elevated 8-OHdG nuclear labeling, lipid peroxidation, and lysosomal cathepsin L activity, suggests that the oxidative metabolic profile of these fibers renders them more vulnerable to sleep deprivation-induced injury [27].

5. Metabolic Mechanisms

5.1 Myofibrillar Protein Synthesis

The rate of myofibrillar protein synthesis (MyoPS) is a key determinant of skeletal muscle mass and is central to the repair and remodeling of contractile proteins following exercise. Saner et al. provided the first direct evidence that sleep restriction reduces MyoPS in humans [28]. In their study, five nights of sleep restriction (4 hours time in bed per night) resulted in significantly lower MyoPS rates (fractional synthetic rate 1. 24 ± 0. 21 %/day) compared to normal sleep (1. 53 ± 0. 09 %/day). Critically, performing HIIE during the sleep restriction period maintained MyoPS at control levels (1. 61 ± 0. 14 %/day) [28].

Paradoxically, despite the reduction in MyoPS, no changes were observed in the canonical regulators of protein synthesis (phosphorylated AKT and mTOR) or degradation (FoxO1/3 mRNA and LC3 protein) in any group [28]. This dissociation between functional protein synthesis rates and signaling pathway activation suggests that sleep restriction may affect MyoPS through mechanisms not captured by single-timepoint assessments of these molecular markers, or through alternative regulatory pathways.

In animal models, the effects on protein turnover are more pronounced. Paradoxical sleep deprivation in rats induced muscle atrophy with reductions in both body mass and gastrocnemius muscle mass [18]. The hormonal milieu—characterized by elevated corticosterone and reduced testosterone—was associated with activation of the ubiquitin-proteasome system and downregulation of the PI3K/Akt/mTOR pathway [18,29]. Leucine supplementation during sleep deprivation prevented muscle mass loss and type IIb fiber atrophy, likely through direct stimulation of mTORC1 signaling, although it did not fully normalize the proteolytic environment [29].

5.2 Mitochondrial Function

Mitochondrial respiratory function is essential for skeletal muscle energy production and recovery. Sleep restriction has been shown to impair mitochondrial function in human skeletal muscle. Saner et al. demonstrated that five nights of sleep restriction reduced mitochondrial respiratory capacity by approximately 16 pmol $O_2 \cdot s^{-1} \cdot mg^{-1}$, an effect that was completely

prevented by concurrent HIIE [30]. This reduction in mitochondrial function was associated with impaired glucose tolerance and reduced sarcoplasmic protein synthesis, suggesting interconnected metabolic disturbances [30].

Transcriptomic analyses corroborate these findings. Sleep deprivation alters the skeletal muscle transcriptome by decreasing expression of oxidative phosphorylation pathways and increasing expression of inflammatory pathways [25,31]. Gene set enrichment analyses in sleep-restricted individuals revealed decreased enrichment of gene pathways associated with mitochondrial function, a pattern that was reversed by exercise [25,31].

In Drosophila models, short-term sleep deprivation decreased respiration at both oxidative phosphorylation (OXPHOS) and electron transport system (ETS) capacities, increased reactive oxygen species (ROS) production and lipid peroxidation, and upregulated expression of genes involved in oxidative stress response and apoptosis [32]. While direct translation from invertebrate models to human physiology requires caution, these findings support the concept that mitochondria represent a primary target of sleep deprivation-induced cellular stress.

5.3 Insulin Sensitivity and Glucose Metabolism

Skeletal muscle is the primary site of insulin-mediated glucose disposal, and sleep deprivation-induced insulin resistance has important implications for muscle recovery. Experimental sleep restriction consistently reduces insulin sensitivity, with effects detectable after as few as one night of restricted sleep [31,33].

Subchronic sleep restriction (five nights of 4 hours time in bed) decreased whole-body insulin sensitivity by 25% and peripheral insulin sensitivity by 29%, without significantly affecting hepatic insulin sensitivity [14]. This peripheral insulin resistance was associated with substantial increases in fasting non-esterified fatty acids (62%) and β-hydroxybutyrate (55%), suggesting increased lipolysis and fat oxidation as contributing mechanisms [14]. Stress hormones, including cortisol, metanephrine, and normetanephrine, were modestly elevated and may contribute to insulin resistance through promotion of lipolysis [14].

The relevance of insulin resistance to muscle recovery lies in insulin's role as an anabolic hormone that promotes amino acid uptake and protein synthesis in skeletal muscle. Impaired insulin signaling reduces the muscle's capacity to utilize circulating amino acids for repair and remodeling. Furthermore, suppression of SWS—the sleep stage most closely associated with GH release and reduced sympathetic activity—has been shown to decrease insulin sensitivity by 25%, with the magnitude of insulin sensitivity reduction strongly correlated ($r = 0.89$) with the degree of SWS suppression [31].

5.4 Epigenomic and Transcriptomic Alterations

Emerging evidence suggests that sleep loss may induce tissue-specific epigenomic and transcriptomic changes in skeletal muscle. A single night of sleep deprivation has been shown to alter genome-wide DNA methylation and induce molecular signatures related to inflammation and altered metabolic fuel utilization in human tissues [34]. These findings indicate that even short-term sleep disruption may affect regulatory mechanisms involved in muscle metabolism and adaptation.

Transcriptomic studies also suggest that sleep restriction may influence gene pathways related to inflammation, mitochondrial function and muscle remodeling [25,35]. In healthy young men, sleep restriction was associated with enrichment of inflammatory and immune response-related pathways and reduced enrichment of mitochondrial pathways in skeletal muscle [25]. In postmenopausal women, sleep restriction induced coordinated transcriptional remodeling involving oxidative phosphorylation, myogenesis and immune-related pathways [35].

Although these findings are mechanistically important, their clinical relevance for athletes remains uncertain. Current evidence suggests that epigenomic and transcriptomic changes may contribute to impaired recovery and altered muscle adaptation during sleep loss, but further studies are needed to determine whether these molecular changes translate into measurable differences in strength, hypertrophy, injury risk or long-term training outcomes.

6. Circadian Clock Mechanisms

Skeletal muscle contains peripheral circadian clocks that regulate daily rhythms of protein turnover, substrate utilization, mitochondrial function and gene expression [3,37]. Sleep deprivation and irregular sleep-wake schedules may disrupt these rhythms, leading to impaired temporal coordination of muscle metabolism and recovery. This mechanism may be particularly relevant for athletes exposed to early training sessions, late competitions, travel across time zones or irregular recovery periods.

Experimental studies indicate that the skeletal muscle clock is involved in the regulation of proteolysis, muscle growth and regeneration. Disruption of muscle clock genes may alter protein degradation pathways and contribute to impaired muscle function over time [38]. Circadian regulators such as Per1, Per2 and Bmal1 have also been linked to myogenic differentiation, satellite cell function and muscle repair after injury [39,40].

Satellite cells are essential for skeletal muscle regeneration, and emerging evidence suggests that their activity may be influenced by circadian timing. Animal and cellular studies indicate that disruption of circadian clock genes can impair satellite cell proliferation, differentiation and post-injury muscle repair [39–42]. These findings suggest that sleep loss may affect recovery not only through systemic hormonal and inflammatory changes, but also through direct disruption of local molecular clocks within skeletal muscle.

However, most evidence regarding circadian regulation of muscle repair comes from experimental and preclinical models. Therefore, practical recommendations concerning optimal training time, injury recovery or therapeutic interventions after sleep loss remain limited. Future human studies should evaluate how chronotype, training time, sleep timing and circadian misalignment influence muscle recovery and adaptation in athletes.

7. Autonomic Nervous System Dysregulation

Sleep deprivation is associated with increased sympathetic nervous system activity, which may influence recovery through cardiovascular, metabolic and neuromuscular pathways. Shorter habitual sleep duration has been linked to higher muscle sympathetic nerve activity, suggesting that insufficient sleep may contribute to persistent autonomic activation [43].

Sympathetic overactivity may also promote higher blood pressure, impaired vascular function, systemic inflammation and altered glucose metabolism [14,44].

In the context of exercise recovery, increased sympathetic tone may delay the return to physiological balance after training and may contribute to fatigue, reduced readiness and impaired adaptation. Acute exercise performed after sleep deprivation may further reduce parasympathetic activity and increase inflammatory responses, indicating that training intensity and timing should be considered carefully when sleep loss has occurred [45].

Experimental evidence also suggests that sleep deprivation may affect neuromuscular junction structure and function, which could contribute to impaired motor performance and muscle fatigue [46]. However, this area remains insufficiently studied in athletes, and further research is needed to determine whether such changes have practical relevance for sports performance and recovery.

8. Effects on Body Composition

Sleep deprivation may also influence body composition, which is relevant to muscle recovery and adaptation. In controlled experimental conditions, sleep restriction during caloric deficit has been associated with reduced fat loss and greater loss of fat-free mass compared with adequate sleep opportunity [47]. This suggests that insufficient sleep may make it more difficult to preserve lean tissue during weight reduction.

Population-level studies also indicate associations between poorer sleep quality or abnormal sleep duration and unfavorable body composition, including lower skeletal muscle mass and higher fat mass [48,49]. Although observational data cannot confirm causality, they support the concept that sleep may be an important factor in maintaining muscle mass and metabolic health.

The mechanisms underlying these effects likely include reduced anabolic hormone activity, increased cortisol, impaired insulin sensitivity, decreased myofibrillar protein synthesis and altered tissue-specific gene regulation [14,28,34,47]. For athletes, this may be particularly important during periods of caloric restriction, weight-category preparation or heavy training blocks, when preservation of fat-free mass is a key component of performance and recovery.

9. Countermeasures and Mitigation Strategies

Several strategies may help reduce the negative impact of sleep deprivation on post-exercise muscle recovery. The most important intervention remains adequate sleep opportunity, as sleep is directly involved in endocrine regulation, immune balance, metabolic recovery and skeletal muscle repair. In athletes, sleep duration, sleep quality and sleep timing should therefore be considered part of the recovery process rather than only a lifestyle factor [1–3].

Sleep extension may be useful before anticipated periods of sleep loss, such as travel, early competitions, congested schedules or increased psychological stress. Extending time in bed for several nights before sleep deprivation has been associated with higher IGF-1 concentrations and may provide a partial physiological buffer against subsequent sleep loss [13]. Although this strategy cannot fully prevent the consequences of sleep deprivation, it may be practical for athletes who are exposed to predictable sleep disruption.

Strategic daytime napping is another useful intervention, particularly when nocturnal sleep is insufficient. Systematic reviews indicate that napping may improve short-term physical

performance, endurance, sport-specific skills, reaction time and attention in physically active individuals [51,52]. Longer nap opportunities, especially those lasting approximately 60–90 minutes, may provide greater benefits, although shorter naps may be more feasible during competition or training days. When possible, naps should be scheduled in the early afternoon and should allow sufficient time before exercise to reduce sleep inertia [51–53].

Exercise may also attenuate some of the adverse effects of sleep restriction on skeletal muscle metabolism. Human studies suggest that high-intensity interval exercise performed during periods of sleep restriction may help preserve myofibrillar protein synthesis, mitochondrial function, glucose tolerance and skeletal muscle transcriptomic responses [25,28,30]. However, exercise should not be regarded as a substitute for sleep. Acute high-intensity exercise after sleep deprivation may increase autonomic and inflammatory stress, so training load and intensity should be adjusted according to sleep history, fatigue and recovery status [45].

Nutritional strategies may support recovery during periods of sleep restriction, but evidence remains limited. Adequate protein intake, sufficient energy availability, appropriate carbohydrate intake and hydration may help maintain muscle repair and performance readiness. Leucine supplementation has shown anti-atrophic effects in animal models of sleep deprivation, but human studies are needed before specific supplementation protocols can be recommended [29]. Therefore, nutrition should be used as supportive care rather than as a replacement for adequate sleep.

11. Strength of Evidence

The evidence linking sleep deprivation with impaired post-exercise recovery is heterogeneous and differs across mechanistic domains. The strongest evidence concerns the negative effects of sleep loss on physical performance, perceived exertion, endocrine regulation and glucose metabolism. These outcomes have been evaluated in human experimental studies and systematic reviews, although study protocols differ in sleep loss duration, exercise modality, participant characteristics and timing of measurements [5,6,14,17,23,33].

Moderate evidence supports the role of sleep restriction in shifting the anabolic-catabolic balance toward a less favorable recovery environment. Experimental studies suggest that sleep loss may suppress the GH/IGF-1 axis, increase cortisol activity and alter testosterone secretion, although these responses depend on age, sex, circadian timing and the type of sleep deprivation protocol used [7,11–13,15,17,20]. The cortisol-to-testosterone ratio appears particularly relevant in the post-exercise context, as it reflects the balance between catabolic and anabolic signaling during muscle repair [17,20].

The evidence for inflammatory dysregulation is also moderate but complex. Sustained partial sleep restriction appears more consistently associated with increased circulating IL-6 and CRP than a single night of sleep loss [21,22]. In the context of exercise-induced muscle damage, sleep deprivation may selectively increase IL-6 and alter the basal inflammatory environment, while tissue-level inflammatory signaling may occur even when systemic markers remain unchanged [17,23–25].

Evidence regarding myofibrillar protein synthesis, mitochondrial function, circadian regulation, epigenomic remodeling and neuromuscular junction changes is highly relevant but still more limited. Available human studies suggest that sleep restriction can reduce

myofibrillar protein synthesis and mitochondrial respiratory capacity, while high-intensity interval exercise may attenuate some of these changes [28,30]. However, many molecular mechanisms remain based on small human studies, animal models or cellular research, and their direct translation to athletes requires caution [34,36,38–42,46].

Overall, current evidence supports sleep optimization as a key component of recovery strategies in athletes and physically active individuals. Practical recommendations should distinguish between well-supported interventions, such as adequate sleep opportunity, sleep extension and strategic napping, and emerging approaches, such as targeted supplementation or circadian timing strategies, which require further validation in human sports populations.

12. Practical Implications for Athletes and Coaches

Sleep should be considered an active component of training adaptation and post-exercise recovery. In athletes and physically active individuals, sleep duration, sleep quality and sleep timing should be monitored together with training load, nutrition, perceived exertion, soreness and readiness to perform [1,2,4–6].

Recent sleep history should be taken into account when planning high-intensity training, eccentric loading, resistance exercise, sprint work or repeated competition exposure. A single night of markedly reduced sleep does not necessarily require training cancellation, but it may justify reducing training volume, lowering intensity, extending warm-up and cool-down procedures or prioritizing technical and low-load sessions rather than maximal neuromuscular work.

Repeated sleep restriction over several nights may be more problematic than isolated acute sleep loss, because sustained partial sleep restriction appears more likely to increase inflammatory markers and impair recovery-related processes [21,22]. Coaches and sports medicine professionals should therefore pay particular attention to athletes reporting consecutive nights of poor sleep, especially during periods of intensified training, travel, caloric restriction or psychological stress.

Sleep extension and strategic napping may be useful practical tools when sleep disruption is expected or unavoidable. Extending sleep opportunity before travel, competitions or congested schedules may help reduce the physiological burden of subsequent sleep loss [13]. Daytime naps may support physical and cognitive performance, particularly when nocturnal sleep is insufficient, although they should be planned with enough time before training or competition to reduce sleep inertia [51–53].

Nutritional support may also be important during periods of sleep restriction. Athletes should avoid aggressive caloric restriction when sleep is insufficient, because sleep restriction during energy deficit may increase the risk of fat-free mass loss [47]. Adequate protein intake, sufficient carbohydrate availability, hydration and regular meal timing may help support recovery, although specific supplementation strategies require further validation.

In practical monitoring, simple tools such as sleep diaries, questionnaires, wearable sleep estimates and daily wellness scores may help identify insufficient recovery before measurable decrements in performance occur. Sleep data should be interpreted together with training load,

psychological stress, illness, injury risk, menstrual cycle status and nutritional intake rather than as an isolated variable.

13. Integrative Model and Future Directions

The evidence reviewed in this article supports an integrative model in which sleep deprivation impairs post-exercise muscle recovery through several interconnected pathways. At the systemic level, sleep loss may disturb the GH/IGF-1 axis, increase cortisol activity, alter testosterone secretion and enhance sympathetic nervous system activation. This hormonal and autonomic profile may shift the anabolic-catabolic balance toward protein breakdown, reduced tissue repair and impaired training adaptation [7,11,15,17,18,20,43].

At the inflammatory level, sleep deprivation may promote systemic and local immune dysregulation. Sustained sleep restriction has been associated with increased circulating inflammatory markers, particularly IL-6 and CRP, while experimental studies have also shown changes in inflammatory signaling within skeletal muscle tissue [21,22,24,25]. In the context of exercise-induced muscle damage, such changes may disturb the coordinated inflammatory response required for satellite cell activation, tissue remodeling and effective recovery [2,17,23,26].

At the metabolic and cellular level, sleep loss may reduce myofibrillar protein synthesis, impair mitochondrial respiratory capacity, reduce insulin sensitivity and alter skeletal muscle gene expression [14,28,30,33,34]. These disturbances may limit the ability of skeletal muscle to restore energy balance, repair contractile proteins and adapt to repeated training stimuli. Circadian disruption may further contribute to impaired recovery by disturbing peripheral clocks in skeletal muscle and satellite cells, which normally regulate protein turnover, stem cell activity and inflammatory signaling [3,37–42].

Despite substantial progress, several important gaps remain. Most human experimental studies have been conducted in young, healthy men, while data in women, older adults and clinical populations remain limited. Many protocols also use acute total sleep deprivation or severe sleep restriction, which may not fully reflect the chronic, moderate sleep insufficiency commonly observed in athletes and the general population. Future studies should therefore evaluate dose-response relationships between sleep duration, sleep quality and post-exercise recovery in more diverse populations.

Further research is also needed to determine which countermeasures are most effective. Current evidence supports adequate sleep opportunity, sleep extension before anticipated sleep loss and strategic daytime napping as practical interventions for athletes [13,50–53]. Exercise, particularly high-intensity interval exercise, may attenuate some adverse metabolic and molecular effects of sleep restriction, although the optimal timing, intensity and volume of exercise under sleep-deprived conditions remain unclear [25,28,30,45]. Nutritional strategies, including adequate protein intake and leucine supplementation, are biologically plausible but require stronger evidence from human studies before they can be recommended as specific interventions for sleep deprivation-induced muscle impairment [29].

Overall, sleep deprivation should be understood as a multisystem stressor that affects muscle recovery through hormonal, inflammatory, metabolic and circadian mechanisms. Future research should integrate sleep science, exercise physiology, molecular biology and sports

medicine in order to develop practical, evidence-based recovery strategies for athletes and physically active individuals.

14. Conclusion

Sleep deprivation may impair skeletal muscle recovery after exercise through a complex network of hormonal, inflammatory, metabolic and circadian mechanisms. Suppression of the GH/IGF-1 axis, changes in testosterone secretion, increased cortisol activity and sympathetic overactivation may shift the anabolic-catabolic balance toward a less favorable environment for tissue repair and training adaptation [7,11,15,17,18,20,43].

Inflammatory dysregulation represents another important mechanism. Sustained sleep restriction may increase systemic inflammatory markers, while experimental studies suggest that sleep loss can also modify inflammatory signaling within skeletal muscle tissue [21,22,24,25]. These changes may interfere with the coordinated inflammatory response required for muscle regeneration, particularly after exercise-induced muscle damage [2,17,23,26].

Sleep loss may also affect muscle recovery through metabolic and molecular pathways, including reduced myofibrillar protein synthesis, impaired mitochondrial function, decreased insulin sensitivity and altered gene expression in skeletal muscle [14,28,30,33,34]. Circadian disruption may further compromise recovery by disturbing peripheral clocks involved in protein turnover, satellite cell activity and immune-muscle communication [3,37–42].

Although the current evidence supports the importance of sleep for post-exercise recovery, several limitations should be emphasized. Many studies involve small samples, acute or severe sleep deprivation protocols and predominantly young male participants. Therefore, the findings should be interpreted cautiously and should not be automatically generalized to all athletes, women, older adults or clinical populations.

From a practical perspective, sleep should be regarded as an active component of recovery and training adaptation rather than a passive period of rest. Sleep optimization, sleep extension before anticipated sleep loss, strategic napping, appropriate training load adjustment and adequate nutrition may support recovery in athletes and physically active individuals [13,50–53]. Future studies should clarify the dose-response relationship between sleep loss and muscle recovery, identify sex- and age-specific responses and determine which interventions are most effective in real-world sports settings.

Disclosure

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