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RECOVERY STRATEGIES IN SPORT - A REVIEW OF CURRENT RESEARCH

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ABSTRACT

Introduction and Purpose: Effective recovery is critical for optimizing athletic performance and preventing injury. This review aims to synthesize current research on recovery strategies in sport by examining the physiological, neurological, and psychological mechanisms underlying fatigue and regeneration.
Key findings: The multifactorial nature of fatigue is discussed, including metabolic, central, and peripheral contributors, as well as individual differences related to age and sex. Cellular and systemic regenerative processes are analyzed to understand recovery dynamics. Popular recovery methods such as sports massage, cryotherapy, balneotherapy, and hydrotherapy are evaluated alongside nutritional interventions including protein, carbohydrates, creatine, BCAAs, caffeine, and vitamin D supplementation. The role of sleep and neuropsychological recovery techniques, including breathing exercises, is emphasized. Emerging trends such as active recovery protocols and the application of wearable technology—particularly heart rate variability (HRV) monitoring—are reviewed for their potential to personalize and enhance recovery.
Conclusions: A holistic, individualized approach to recovery that integrates physiological, nutritional, and technological strategies is essential for maximizing athletic outcomes. Despite advances, significant gaps remain in understanding the optimal combination and timing of recovery interventions, highlighting the need for further rigorous research.

KEYWORDS
Recovery Strategies, Post-Exercise Recovery, Fatigue, Cold Water Immersion, Personalized Regeneration, Supplementation

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1. Introduction

Achieving an optimal balance between training load and recovery processes is a critical factor influencing athletic performance. Inadequate recovery management can lead to chronic fatigue, decreased physical capacity, and increased injury risk. Recovery strategies in sport play a crucial role in optimizing athletic performance, preventing injuries, and accelerating return to full function after intense physical exertion. Contemporary scientific literature presents a wide array of recovery approaches, encompassing both physical modalities and active rest techniques. However, despite numerous studies, there remains a need for a systematic review of available scientific evidence to determine the efficacy of specific methods in relation to delayed onset muscle soreness (DOMS), fatigue, muscle damage, and inflammatory markers[1].

A meta-analysis conducted by Dupuy et al., which included 99 randomized controlled trials, examined the effects of various recovery techniques on these parameters. The results indicate that interventions such as massage, compression garments, water immersion, contrast therapy, and cryotherapy significantly reduce DOMS and fatigue, whereas other methods, including stretching and electrical stimulation, showed no significant impact on these outcomes. Additionally, massage and cold exposure were found to be most effective in lowering inflammatory markers such as creatine kinase (CK), interleukin-6 (IL-6), and C-reactive protein (CRP).

Despite promising findings, the authors emphasize the necessity for further research, particularly regarding the long-term effects of individual and combined recovery strategies. It is also critical to consider individual differences such as sex, training status, and type of exercise performed, which may influence the effectiveness of applied recovery interventions.[2]

The objective of the present review is to synthesize current scientific evidence on the efficacy of various recovery methods in sport, considering their impact on physiological parameters and practical applications within training and sports rehabilitation contexts.[1]

2. Methodology

2.1. Literature Search Strategy

A comprehensive literature search was conducted using major electronic databases including PubMed, Scopus, Web of Science, Wiley Online Library, ACS Publications, and SciFinder. The search strategy combined Medical Subject Headings (MeSH) with free-text keywords related to recovery strategies in sport. Boolean operators (AND, OR) were employed to optimize sensitivity and specificity of the search. The search aimed to identify peer-reviewed articles published between 2010 and 2025, with an emphasis on studies from 2018 onward.

2.2. Inclusion and Exclusion Criteria

To ensure scientific rigor and relevance, inclusion criteria comprised peer-reviewed articles, systematic reviews, meta-analyses, and randomized controlled trials (RCTs) published in English. Studies lacking full text, control groups, or those with low methodological quality were excluded. An initial screening of titles and abstracts

was performed to exclude irrelevant records, followed by full-text assessment based on predefined research questions. Quality assessment was conducted following established guidelines such as PRISMA and GRADE.

2.3. Types of Evidence Included

This review synthesizes evidence from high-level medical research, including randomized controlled trials, systematic reviews, and meta-analyses, focusing on the efficacy of various recovery strategies in sport.

2.4. Timeframe and Language

Only articles published in English within the timeframe of 2005 to 2025 were included, with a particular focus on recent evidence from 2018 to 2025.

3. Results of research

3.1 Physiological Basis of Recovery

Key aspects of muscle recovery physiology include high-energy phosphate metabolism, acid-base balance changes within muscles, and the specific metabolic processes occurring during aerobic and anaerobic exercise.[3] Studies employing phosphorus-31 magnetic resonance spectroscopy (^31P-MRS), a non-invasive method to monitor high-energy phosphate levels such as ATP and phosphocreatine (PCr), have demonstrated that intense anaerobic exercise rapidly decreases PCr levels, indicating its utilization as a primary energy source under elevated energy demand[3]. The post-exercise resynthesis of PCr is therefore a critical component in restoring muscle capacity for force and power generation.

Another significant factor influencing recovery is muscle pH changes. During anaerobic exercise, hydrogen ions (H^+) accumulate, leading to intracellular acidification, commonly referred to as muscle acidosis. This phenomenon limits the activity of metabolic enzymes, particularly in glycolysis, and impairs muscle contractile function, contributing to the development of fatigue.[4] A key element of recovery is thus the restoration of acid-base homeostasis, including buffering and removal of excess H^+, which enables the recovery of full contractile function.

Recovery mechanisms also vary depending on the dominant nature of the exercise. Aerobic exercise is associated with lower H^+ accumulation and a more stable profile of high-energy phosphate metabolism, resulting in reduced metabolic fatigue compared to anaerobic efforts.[5] Understanding these differences is crucial for optimizing recovery strategies, which should be tailored to the specific exercise demands of a given sport or training protocol.

3.2 Mechanisms of Fatigue Including Metabolic, Neurological, and Psychological Factors Affecting Physical Performance

According to current literature, exercise-induced fatigue is broadly classified into peripheral and central fatigue.[6] Central fatigue refers to the decline in the central nervous system's (CNS) ability to effectively recruit motor units during physical exertion. The underlying mechanisms include neurochemical changes within the CNS, psychological factors, and individual predispositions.[7] Neural transmission disturbances also play a pivotal role, including insufficient activation of the motoneuron pool, reduced excitatory drive from the motor cortex, and presynaptic inhibition of Ia afferents by group III–IV afferent fibers, which together reduce the excitability of spinal motor neurons. [8]

A crucial component of the pathophysiology of central fatigue involves alterations in the levels of key neurotransmitters, such as serotonin (5-HT), dopamine (DA), and noradrenaline

(NA), all of which modulate perceived exertion, motivation, and willingness to engage in prolonged exercise. An increase in CNS serotonin levels during prolonged physical activity promotes sensations of sleepiness, lethargy, and reduced motivation, forming the basis of the so-called central fatigue hypothesis.[9]

Dopamine is a key neurotransmitter of the central nervous system, playing a crucial role in motivational processes, reward pathways, and the regulation of motor functions.[10] Research indicates that during intense physical exercise, dopamine levels in the CNS decrease, leading to reduced motivation and a diminished ability to sustain physical activity.[11]Experimental data confirm that fluctuations in dopamine concentrations within the CNS significantly modulate the perception of fatigue during exercise. Enhanced dopaminergic transmission fosters motivation and reinforces reward mechanisms, whereas decreased dopaminergic activity can contribute to an increased subjective sense of fatigue and diminished psychophysical performance.[12]

The same study also highlights the role of noradrenaline as a critical neurotransmitter responsible for regulating alertness and attentional mechanisms. Elevated noradrenergic transmission during exercise promotes wakefulness and readiness for action, which may help delay the onset of fatigue during physical activity.[13] These mechanisms represent an essential element of the neurobiological foundations of central fatigue.

Available literature suggests that manipulating noradrenergic system activity influences fatigue perception during exercise. Increased noradrenergic transmission is associated with improved alertness, concentration, and a delayed onset of fatigue. However, excessive stimulation of the noradrenergic system may have the opposite effect, exacerbating fatigue symptoms and negatively affecting psychological comfort during exertion.[13]

A key factor in achieving optimal physical and mental performance is the homeostatic balance between serotonergic, dopaminergic, and noradrenergic activity. Disruption of this balance may lead to earlier onset of fatigue, reduced motivation, and worsened mood. For example, an increased serotonin-to-dopamine ratio may favor the premature development of central fatigue, while maintaining appropriate levels of dopamine and noradrenaline supports motivational function and sustained alertness.[9]

3.3 Psychological Factors and Individual Variability in Exercise-Induced Fatigue

Scientific literature emphasizes the significant role of psychological factors and individual variability in the body's response to physical exertion. According to a systematic review on the impact of mental fatigue (MF) on endurance performance, it has been demonstrated that mental fatigue exerts a substantial negative influence on physical performance.[14] However, the analysis of individual variables, such as sex, age, body mass index (BMI), or training status, did not reveal significant differences in susceptibility to MF. The authors highlight numerous methodological gaps in the available studies, including insufficient reporting of participant characteristics, lack of standardized procedures, and inadequate consideration of variables that may influence individual responses to MF.[15]

Further research is necessary, with precise measurement of both psychological and physiological parameters of participants—such as training level, nutritional status, or psychoregulatory strategies—to effectively elucidate the mechanisms underlying individual differences in the response to mental fatigue.

3.4 Peripheral Fatigue Mechanisms

Peripheral fatigue is defined as a set of processes occurring at the level of muscle fibers, resulting in a decreased ability to generate force[5]. The main mechanisms include excitation–contraction coupling impairment, accumulation of metabolites (including hydrogen ions and inorganic phosphates), and restricted muscle perfusion, leading to hypoxia and impaired muscle function.[16]

Classic works in exercise physiology confirm that a key factor in peripheral fatigue is impaired excitation–contraction coupling, resulting from reduced sensitivity of the muscle's contractile apparatus to calcium ions and diminished efficiency of Ca^{2+} release from the sarcoplasmic reticulum.[17] [18]

The accumulation of H^+ ions leads to a decrease in intracellular pH, which negatively affects the activity of glycolytic enzymes and the function of contractile proteins such as myosin[56]. Additionally, inorganic phosphate (Pi) and ADP can impair muscle contraction by interfering with calcium release and inhibiting actin–myosin interactions, ultimately leading to reduced force production.[19]

During intense muscle contractions, a significant increase in intramuscular pressure is observed, which can mechanically restrict perfusion in capillaries. This leads to local ischemia, reduced oxygen delivery, and metabolite accumulation, thereby accelerating the development of muscular fatigue. [20]

3.5 Exercise-Induced Fatigue: Mechanisms and Specificity

Task Specificity and Variability of Fatigue Mechanisms

Exercise-induced fatigue shows significant variability depending on the specific task performed and individual characteristics of the person studied. A key factor determining the nature of fatigue is the type of muscle contraction involved. Isometric, concentric, and eccentric contractions differ markedly in terms of the mechanisms and dynamics of fatigue development.[21] [16]

Concentric contractions (CON), involving muscle shortening during force generation, often lead to a faster and more pronounced decline in force production compared to eccentric contractions. The etiology of concentric fatigue includes both central mechanisms, such as decreased motor activation, and peripheral mechanisms, including metabolite accumulation within muscle fibers. [22]

Isometric contractions (ISO), where the muscle length remains unchanged, show a slower onset of fatigue compared to concentric contractions. However, their impact on reduced force production may persist for a longer duration.[23] The mechanisms of isometric fatigue involve restricted blood flow to the working muscles, leading to hypoxia and the accumulation of metabolites that impair force generation. [24]

Eccentric contractions (ECC), characterized by muscle lengthening during force generation, are relatively more resistant to fatigue in the short term. Nevertheless, eccentric contractions are associated with a higher risk of structural muscle fiber damage, resulting in delayed onset muscle soreness (DOMS) and increased levels of creatine kinase (CK), a biochemical marker of muscle microtrauma.[25]

Contraction type	The rate of increase in fatigue	Persistence of fatigue	Risk of muscle damage
Contraction	high	medium	low
Isometric	medium	high	low
Eccentric	low	high	high

Fig. 1. *Comparison of muscle contraction types in terms of fatigue*

3.6 The Importance of Contraction Intensity, Speed, and Stabilization for Muscle Fatigue Mechanisms

Both the intensity and speed of muscle contractions are crucial factors influencing the mechanisms of muscle fatigue. A study evaluating the effects of different contraction velocities and intensities on muscle activation during bench press exercise demonstrated that a faster tempo allowed for a greater number of repetitions before reaching muscular failure.[26]Before the onset of fatigue, higher contraction speeds and loads were associated with increased electromyographic (EMG) signal amplitude, indicating greater motor unit recruitment.

However, during faster repetition tempos, a significant decrease in EMG amplitude was observed in the final phase of concentric contraction compared to slower repetitions.[27] This reduction suggests that not all motor units are recruited with equal intensity at higher speeds, potentially temporarily reducing the load on the neuromuscular system. Nevertheless, after fatigue onset, EMG activity increased regardless of movement speed, while a decrease in EMG median frequency indicated progressive muscle fatigue, likely related to metabolite accumulation and impaired neuromuscular transmission.[26]

Biomechanical stability during exercise also influences the rate of fatigue development. Tasks requiring greater stabilizing effort impose higher demands on both the muscular and nervous systems. A study published in the *Journal of Applied Physiology* showed that tasks requiring postural maintenance, such as limb stabilization, led to faster fatigue development compared to tasks focused solely on force production. These findings suggest that the stabilization component significantly affects neuromotor demands and may contribute to earlier onset of fatigue during complex motor tasks. [28]

3.7 Sex- and Age-Related Differences in Susceptibility to Muscle Fatigue

A review of the literature indicates that women generally exhibit greater resistance to muscle fatigue than men during isometric muscle contractions of comparable relative intensity. [29] However, these differences are task-specific and may depend on factors such as contraction type, exercise intensity, muscle group involved, or environmental conditions. Nevertheless, several well-documented physiological mechanisms underlie the observed sex-based discrepancies in muscle fatigue susceptibility.

One of the key differentiating factors is muscle perfusion and the metabolic characteristics of muscle fibers. Women produce lower absolute muscle forces, resulting in lower intramuscular pressure during isometric contractions. Consequently, the mechanical restriction of blood flow within muscle vessels is less pronounced in women, which facilitates sustained oxygen delivery to the muscles and more effective removal of metabolites, thereby delaying fatigue onset. [30] Moreover, women tend to have a higher proportion of type I

(slow-twitch) muscle fibers, which are more fatigue-resistant, better capillarized, and more efficient in oxygen utilization. [31]

Sex-related differences in fatigue susceptibility also involve differing cardiovascular responses, partially attributable to the modulatory influence of estrogens on vascular endothelial function. Estrogens stimulate the synthesis of nitric oxide (NO), a potent vasodilatory factor that promotes vessel relaxation and enhances muscle perfusion.[32] As a result, women demonstrate a greater capacity to maintain or increase blood flow to active muscles during exertion, whereas men more often exhibit vasoconstrictive responses. [33]

Neuromuscular mechanisms also play a crucial role in sex-based differences in fatigue. These include the activation of central nervous system (CNS) motor neurons and afferent nerve fibers sensitive to metabolic byproducts. Women exhibit a smaller reduction in voluntary activation and a lesser decline in force output generated by spinal motor neurons during isometric tasks. [30] Sustained activation of motor neurons originating from cortical and subcortical CNS structures supports prolonged exercise capacity.

In contrast, men's greater absolute force production and higher intramuscular pressures lead to more intense activation of group III and IV afferent nerve fibers, which respond to the accumulation of metabolites such as hydrogen ions (H^+) and ATP. [29] Activation of these fibers generates feedback signals to the CNS, reducing central motor drive and contributing to faster onset of central fatigue.

3.8 Regenerative Processes at the Cellular and Systemic Levels

Skeletal muscle regeneration following physical exercise is a complex process involving both cellular and systemic mechanisms. Satellite cells ($Pax7^+$), the primary population of muscle stem cells, play a pivotal role in the repair of damaged muscle fibers. Following injury or intense exercise, these cells become activated, proliferate, and differentiate into myoblasts, which subsequently fuse with damaged fibers or form new myofibrils, contributing to the structural regeneration of muscle tissue.[34]

Autophagy is another crucial mechanism supporting muscle regeneration. This catabolic process involves the degradation and recycling of damaged organelles and misfolded proteins, facilitating the removal of dysfunctional structures while simultaneously providing precursors for the synthesis of new cellular components and muscle structures.[35] Autophagy is particularly important in the context of exercise-induced adaptation and the repair of microdamage within muscle fibers.

The activation of satellite cells is regulated by numerous signaling pathways and transcription factors. Among them, the p38 MAPK pathway and the transcriptional regulator TAZ are key elements that promote satellite cell proliferation and differentiation, thereby determining the efficiency of regenerative processes.[36] It is noteworthy that the satellite cell population is heterogeneous—various subpopulations differ in their capacities for self-renewal, proliferation, and differentiation, ultimately influencing the dynamics and effectiveness of muscle regeneration. [37]

Regenerative processes may be impaired in cases of excessive training load (overreaching), potentially leading to overtraining syndrome. Chronic high-intensity exercise has been shown to result in reductions in maximal muscle strength and molecular-level alterations, such as impaired contractile function of myofibrils and increased calcium sensitivity—potential compensatory mechanisms in response to reduced contractile force. Additionally, elevated autolysis of calpain-3, a protease responsible for the degradation of structural proteins, has been observed, which may contribute to compromised muscle integrity. [38]

Importantly, exercise-induced autophagy is not limited to skeletal muscle. Activation of this process has been observed in other tissues as well, supporting cellular homeostasis and counteracting the development of systemic dysfunctions. Different types of physical exercise elicit specific autophagic responses across various organs, highlighting the significance of physical activity for overall organismal health. [39]

3.9 Popular Regeneration Strategies

3.9.1 Biological Regeneration

Biological regeneration encompasses a broad range of therapeutic methods aimed at accelerating recovery processes following physical exertion. These strategies are designed to restore homeostasis within the musculoskeletal and nervous systems, minimize the effects of muscle microtrauma, and reduce the accumulation of metabolic by-products associated with fatigue. [2]

Among the most commonly applied biological regeneration methods in sports are cryotherapy, hydrotherapy, massage, and thermotherapy. Each of these interventions exerts specific physiological effects that can influence the dynamics of post-exercise recovery. [40]

3.9.2 Sports Massage

A literature review indicates that sports massage may have beneficial effects on motor skills as well as neurophysiological and psychological mechanisms; however, these effects are moderate and highly dependent on the context of application.[41]Moreover, studies show that massage contributes to improved muscle flexibility and reduction of delayed onset muscle soreness (DOMS), although no clear impact on direct enhancement of physical performance was found. [42] Sports massage may also support the regeneration of skeletal muscles after intense physical exercise, yet further precise research is needed to confirm these properties.

3.9.3 Cryotherapy – Cold Baths, Whole-Body Cryotherapy

The mechanism of cryotherapy is explained by the induction of vasoconstriction of small blood vessels and the reduction of capillary permeability, leading to decreased swelling and inflammatory infiltration in damaged tissues.[43] Subsequently, there is a slowing of nerve conduction, which produces an analgesic effect, as well as a reduction in metabolic rate and oxidative stress in injured tissue, accelerating the restoration of intracellular homeostasis.

Cold Water Immersion (CWI), a form of cryotherapy, involves immersion of the whole body or body parts in cold water typically at 8–15°C for 5–15 minutes, usually after intense exercise or injury.[44] CWI induces strong vasoconstriction and decreases capillary permeability in working muscles, reducing blood flow and promoting the limitation of swelling and inflammation. [45]

According to the "secondary injury" model, cryotherapy limits tissue metabolism increase, protecting healthy structures from edema and ischemia, thereby preventing further damage after the initial injury. [46]

A meta-analysis including 52 studies showed that the use of Cold Water Immersion (CWI) during the recovery period after intense exercise improves muscle strength and power recovery. Additionally, CWI lowers serum creatine kinase (CK) levels and reduces the severity of DOMS within the first 24 hours post-exercise.[44] Comparative studies found that CWI significantly decreases DOMS severity and accelerates recovery compared to passive rest. [44]

Research from 2017 indicates that Cold Water Immersion is more effective in reducing muscle soreness and accelerating recovery of jump performance than Whole-Body Cryotherapy (WBC).[47]

Cryotherapy also serves an analgesic function by slowing nerve conduction. The analgesic mechanism involves inhibition of sensory and motor nerve conduction. [48] The reduction in nerve conduction velocity exceeds 30% with a temperature drop to about 10°C, significantly increasing pain threshold and tolerance to stimuli.

3.9.4 Balneotherapy

A meta-analysis including seven studies with a total of 467 participants demonstrated that balneotherapy leads to reduced bone resorption, while exercises performed in an aquatic environment increase osteocalcin levels and decrease parathyroid hormone (PTH) concentration, indicating beneficial effects on bone metabolism.[49] Moreover, studies showed that balneotherapy combined with physiotherapy yields better outcomes in pain reduction, functional improvement, and quality of life compared to physiotherapy alone. [50]

A systematic review on aquatic therapies for musculoskeletal disorders suggests that water-based exercises produce small but statistically significant benefits in reducing pain and improving functionality in patients with musculoskeletal conditions. However, long-term effects of these interventions require further investigation. [51]

In the context of aquatic therapies, including balneotherapy and spa therapy for chronic low back pain, literature reviews indicate that spa therapy is superior to tap water therapy in reducing pain and dysfunction. It should be noted, however, that the quality of available studies is often low. [52]

3.9.5 Hydrotherapy

Available evidence suggests that hydrotherapy can accelerate cardiovascular recovery and reduce fatigue perception after intense physical exercise.[53] In a randomized controlled trial involving 34 participants after a spinning session, the effects of hydrotherapy were compared with static bed rest. The hydrotherapy group showed a return of heart rate and diastolic blood pressure to baseline values, while these parameters remained elevated in the control group. The improvement in venous return is attributed to the hydrostatic effect — the pressure exerted by water on the body increases venous blood return to the heart, resulting in greater cardiac output, faster heart rate stabilization, and improved metabolite clearance.

Moreover, immersion in water, especially at moderate or low temperatures, has the ability to reduce sympathetic nervous system activity, evidenced by decreased secretion of norepinephrine and adrenaline, which affects blood pressure and heart rate regulation. [54]

The mechanism of hydrotherapy's influence on the autonomic nervous system occurs in two phases. The first phase happens during immersion, where a cold shock activates the sympathetic nervous system, initially increasing catecholamine concentrations, causing peripheral vasoconstriction and elevated blood pressure.[54] The second phase is the adaptation period, dominated by parasympathetic activity. This occurs after a few minutes of immersion or upon exiting the water and involves a baroreceptor mechanism — the increased blood pressure is detected by baroreceptors in the aortic arch and carotid sinuses, leading to reflex inhibition of sympathetic activity and increased vagal tone, resulting in decreased heart rate and blood pressure.[55]

Additionally, face immersion in cold water can activate the diving reflex — a complex physiological response consisting of two components. The first involves stimulation of the trigeminal nerve (V) endings, which send impulses to the cardiac center of the vagus nerve in the medulla oblongata. The second component is vagus nerve (X) activation, releasing acetylcholine (ACh) at parasympathetic cardiac endings. Acetylcholine acts on M2 muscarinic receptors in the sinoatrial (SA) node, causing bradycardia. [55]

As a result of the short-term activation of the sympathetic nervous system, parasympathetic dominance follows, characterized by reduced norepinephrine and adrenaline levels and accelerated homeostasis restoration after exercise. This process leads to improved well-being, reduced fatigue, and stabilization of cardiovascular parameters. [55]

3.10 Nutrition and supplementation

3.10.1 Whey Protein Supplementation and Muscle Recovery

A study evaluating the effect of whey protein (WP) supplementation on muscle function recovery after resistance training observed small to moderate improvements in muscle contractile function up to 96 hours post-exercise compared to a control.[56] Although only about half of the individual studies showed significant effects, a meta-analysis confirmed a statistically significant advantage of whey supplementation over control. It is recommended to consume 20–30 g of whey protein post-exercise to support muscle function recovery.

In the context of muscle injuries, various nutritional strategies are also employed.[57] A literature review suggests a recommended protein intake of 20–40 g (0.25–0.4 g/kg body weight) per meal every 3–4 hours to stimulate muscle protein synthesis (MPS). Additionally, supplementation with essential amino acids (EAA), including leucine, supports anabolic processes and reduces muscle damage markers such as creatine kinase (CK) and lactate dehydrogenase (LDH).[57]

Moreover, consumption of a protein and carbohydrate combination immediately after exercise increases muscle protein synthesis and accelerates muscle glycogen replenishment.

Timing of supplementation relative to training is also critical. In a randomized study, the P0 group ingested a supplement containing 10 g protein, 7 g carbohydrates, and 3 g fat immediately post-exercise, while the control group P2 received the same supplement with a 2-hour delay. The P0 group demonstrated a significant increase in quadriceps muscle volume and dynamic strength improvement by 46% and isokinetic strength by 15%, whereas the P2 group showed only a 36% increase in dynamic strength. [59] These results indicate that immediate post-exercise protein intake promotes better hypertrophy and muscle strength gains compared to delayed supplementation.

A systematic review analyzing 13 randomized controlled trials on whey protein supplementation and muscle recovery after resistance exercise in young healthy adults showed that protein-carbohydrate meals taken immediately post-exercise and repeated daily (typically every 24 hours) resulted in more effective restoration of muscle contractile function compared to control groups receiving placebo or isocaloric meals during the 24–96 hour recovery period.[56] This effect is attributed to the high leucine content in whey protein and rapid amino acid delivery, which activates the mTOR pathway and enhances muscle protein synthesis. [60]

3.10.2 Supplements (Creatine, BCAA, Caffeine, Vitamin D3, Curcumin)

Regarding creatine supplementation, it has been established that creatine can reduce muscle damage as measured by the creatine kinase (CK) marker, although its impact on subjective recovery indicators requires further research. A study analyzing creatine supplementation before and/or after exercise-induced muscle damage (EIMD) evaluated recovery markers such as muscle strength, delayed onset muscle soreness (DOMS), range of motion, inflammation, oxidative stress, and muscle damage markers (e.g., CK). Creatine

supplementation significantly lowered CK levels 48 hours post-exercise, but no significant differences were observed in muscle strength, pain, range of motion, inflammation, or oxidative stress. [61]

In terms of caffeine's effect on recovery, studies have shown that a single dose of 6 mg/kg administered after muscle-damaging exercise significantly reduces DOMS within 24–48 hours post-injury, with a stronger effect observed in men. [62]Additionally, both sexes experienced faster recovery of maximal voluntary isometric contraction (MVIC), with a markedly greater effect in men.[63] Caffeine intake was also associated with higher blood glucose and lactate levels compared to placebo ($p < 0.05$), suggesting improved energy availability during recovery. [63]

The mechanism of caffeine's action is explained by its blockade of adenosine A1 and A2A receptors, leading to reduced fatigue perception and increased catecholamine levels (e.g., adrenaline, noradrenaline), which enhances energy mobilization. Additionally, caffeine improves sarcoplasmic reticulum calcium (Ca^{2+}) handling, contributing to improved muscle contraction quality and faster power recovery. Caffeine also stimulates the Na^{+}–K^{+}–ATPase pump, aiding metabolic balance restoration in muscle tissue. [64]

A 2020 study demonstrated that vitamin D3 supplementation increased 25(OH)D levels, correlating with a significant reduction in CK activity 24 hours post-exercise, as well as decreased troponin and myoglobin levels. Furthermore, reductions in TNF-α cytokine concentrations were observed immediately and 1-hour post-exercise, and IL-6 levels decreased 24 hours after running. These findings indicate vitamin D's protective effects on muscles, evidenced by lower CK levels, and its immunomodulatory role through inhibition of pro-inflammatory cytokines. The active form of vitamin D, $1,25(OH)_2D$, binds vitamin D receptors in muscles, initiating anti-inflammatory and regenerative processes. [65]

Article	Population	Dosage	Duration of supplementation	Impact on regeneration	Biochemical markers levels
Żebrowska et al., 2020 JISSN	Ultramarathon runners	2000 IU/d	3 weeks	- Reduced muscle damage - Faster return of efficiency	↓CK ↓Troponine ↓TNF-α ↓IL-6
Liu et al., 2023 Nutrients	Heatlhy, active adults	5000 IU/d	4 weeks	- Reduced soreness - Reduced inflammation	↓CK ↓LDH ↓IL-6
Owczarek et al.,2020 Health	Physical education students	4000 IU/d	8 weeks	- Inflammation markers reduced after exercise	↓TNF-α ↓IL-6
Ota et al., 2019 front. Physiol.	Physically active men	4000 IU/d	6 weeks	- improved isometric strength recovery	↓CK ↓TNF-α ↓IL-6

Fig. 2. *Study Comparison Chart: Vitamin D and muscle recovery*

Vitamin D3 also effectively reduces oxidative stress and protects muscle tissue proteins and lipids. Compared to placebo, individuals supplemented with vitamin D exhibited significantly lower TBARS (thiobarbituric acid reactive substances) levels—a marker of lipid damage—and reduced carbonylated protein concentrations—indicating protein damage—immediately, 2 hours, and 24 hours post-exercise. [66]

Curcumin supplementation shows potential in reducing muscle damage and inflammation caused by eccentric exercise, attributed to its antioxidant and anti-inflammatory properties. [67]

3.11 Sleep and neuropsychological recovery

3.11.1 Effect of Sleep on Muscle Recovery

A 2019 study on a mouse model investigated the effect of sleep deprivation on muscle recovery after intense physical exercise. Four groups were distinguished: a control group (no exercise, no sleep deprivation), an exercise group (EX), a sleep deprivation group (SD), and an exercise plus sleep deprivation group (EX + SD). After intense exercise, the EX + SD group was deprived of sleep for approximately 72 hours. Markers of muscle damage (including creatine kinase [CK] and aspartate aminotransferase [AST]), muscle function, and recovery progression were monitored.

A significant increase in muscle damage markers was found in the EX + SD group compared to the EX- group. Muscle recovery in the sleep-deprived group was slower, as evidenced by a lower number of regenerating muscle fibers, reduced expression of repair proteins, and delayed return of muscle function, including isometric strength. These results indicate that sleep deprivation significantly inhibits muscle recovery after intense exercise, increasing damage and impairing repair mechanisms. [68] Similar reports suggest that even a short period of sleep deprivation after training may increase injury risk, delay return to full fitness, and negatively affect training adaptation.[69],[70]

Another study hypothesized that insufficient sleep causes hormonal imbalance, leading to increased muscle protein degradation and impaired recovery after exercise, injuries, or conditions such as sarcopenia and cachexia. An increase in cortisol levels in humans and corticosterone in animals—hormones with proteolytic effects promoting protein breakdown—was observed. Concurrently, levels of anabolic hormones such as testosterone and IGF-1 decreased, weakening muscle protein synthesis. Consequently, catabolism outweighs anabolism, resulting in enhanced muscle tissue degradation and impaired recovery[71] .

3.11.2 Neuropsychological Recovery After Exercise

Neuropsychological recovery is the process of restoring and optimizing nervous system and mental functions after physical, psychological, or emotional stressors, especially related to physical exercise, stress, or sleep deprivation.

A randomized controlled trial showed that after intense exercise (85–90% HRmax), there is a significant increase in peak alpha frequency (iAPF) in EEG immediately after and up to approximately 30 minutes post-exercise. iAPF is an indicator of cognitive processing efficiency, a marker of heightened mental arousal, attention, and processing speed. This suggests that physical exercise improves cognitive readiness and that the brain becomes better prepared to process information. Properly dosed exercise may thus support concentration and executive functions immediately after training.

This study also indicates that neuropsychological recovery can be delayed. Sustained elevated iAPF (about 0.5–1 Hz above baseline) after intense exercise reflects a transient state of increased nervous system activation. From a neurobiological perspective, cortical structures responsible for cognitive processing and autonomic control remain hyperactive for 30–60 minutes post-exercise. This may impair sleep initiation and quality after evening training (due to sustained beta activity and decreased theta activity), predispose to hypothalamic–pituitary–adrenal (HPA) axis overload, intensify symptoms of psychological overtraining, and prolong full post-exercise recovery by delaying autonomic nervous system calming and neuroendocrine homeostasis restoration, thereby affecting skeletal muscle restitution and protein synthesis. [72]

3.11.3 Breathing Techniques

The use of deep diaphragmatic breathing (DB) after intense physical exercise has shown beneficial effects on the biochemical profile related to post-exercise recovery. According to the study by Angelucci et al. (2012), this technique leads to a significant increase in biological antioxidant potential (BAP), accompanied by a reduction in reactive oxygen metabolites (ROM), indicating a decrease in oxidative stress. Additionally, a reduction in cortisol levels and an increase in melatonin concentration were observed, suggesting regulation of the hypothalamic–pituitary–adrenal (HPA) axis and support for neurohormonal restitution processes.[73] From the perspective of exercise physiology, these effects imply that DB may serve as an effective non-pharmacological intervention supporting psychophysiological recovery by reducing oxidative load in muscle tissues, accelerating hormonal balance restoration, improving sleep quality and circadian rhythm (in the context of melatonin), and potentially enhancing the organism's adaptive capacity to exercise.[74] Moreover, short-term breathing interventions, particularly the cyclic sighing technique, have been shown to significantly impact physiological and psychological parameters related to post-exercise recovery. Cyclic sighing,

characterized by an extended exhalation phase (e.g., 4–6 seconds inhalation, 8–10 seconds exhalation), compared to other techniques such as box breathing or controlled hyperventilation, demonstrated the highest effectiveness in lowering heart rate, increasing heart rate variability (HRV), and improving subjective mood. Porto et al. (2022) indicate that just five minutes of daily cyclic sighing practice leads to significant changes in autonomic nervous system tone, shifting the balance toward parasympathetic activation. [75] Compared to classic mindfulness interventions, which primarily involve mindful observation of internal experiences without manipulating the breathing rhythm, techniques with prolonged exhalation seem to exert a stronger influence on indicators of physiological recovery.

The mechanisms underlying this difference may include stronger vagus nerve stimulation during the exhalation phase and faster activation of the limbic-parasympathetic axis, supporting neuropsychological restitution and improving conditions for muscle recovery through reductions in heart rate, cortisol, and enhanced tissue perfusion.

3.12 Active Recovery and Regenerative Training

Active recovery, involving low-intensity exercise, mobilization, and stretching, constitutes a critical component of protocols aimed at facilitating muscle homeostasis restoration following high-intensity physical exertion.[76] Numerous studies demonstrate that moderate physical activity, such as light jogging or cycling, enhances blood flow, thereby improving the clearance of metabolic byproducts like lactate and reducing sensations of muscle fatigue. Additionally, stretching, both static and dynamic, positively influences the flexibility of soft tissues and range of motion, potentially mitigating functional limitations and lowering the risk of injury.[77] However, scientific evidence regarding stretching's efficacy in alleviating delayed onset muscle soreness (DOMS) remains inconclusive, with some studies indicating limited benefits in reducing post-exercise muscle pain.[77] Considering current data, active recovery strategies that promote circulation and mobilization are recommended as effective interventions supporting regenerative and adaptive processes within the musculoskeletal system, particularly in the context of high-load athletic training.

3.13 Modern Technologies

Modern technologies play an increasingly important role in optimizing recovery processes in athletes. Fatigue monitoring through heart rate variability (HRV) analysis allows for precise assessment of the autonomic nervous system status and adjustment of training and recovery intensities.[78] Wearable devices such as smartwatches and biometric sensors enable continuous collection of physiological data, facilitating the individualization of training and recovery plans. [79]Additionally, muscle electrostimulation (EMS) is used as a supportive recovery method by increasing blood flow, reducing muscle pain, and accelerating the removal of exercise-induced metabolites. The integration of these technologies supports effective fatigue management and muscle repair processes, which is crucial for improving performance and reducing injury risk. [80]

13.14 Scientific Evidence – Systematic Review

The current state of knowledge regarding the effectiveness of recovery strategies is primarily based on randomized controlled trials (RCTs) and meta-analyses, which represent the highest level of scientific evidence. Systematic literature reviews indicate that methods such as active recovery, massage, cryotherapy, and supplementation demonstrate moderate effectiveness in accelerating muscle recovery and reducing perceived fatigue. [2], [81] However, critical analysis of the quality of available studies reveals considerable heterogeneity in methodology, sample size, and control of confounding factors, which limits the ability to draw definitive conclusions. [82] Furthermore, many studies are characterized by short observation periods and lack standardized recovery protocols, further complicating the interpretation of their results. Therefore, although there is evidence supporting the positive impact of selected recovery strategies, further well-designed research is necessary to fully determine their efficacy and practical application in sports.

3.15 Emerging Trends and Controversies

The field of sports recovery is witnessing rapid development of innovative methods, such as active recovery based on light physical activity and the use of advanced wearable technologies for fatigue monitoring and tailoring individualized training and recovery protocols.[83] Muscle electrostimulation (EMS) and techniques grounded in cellular biology are also gaining popularity as recovery aids. Despite growing interest, many of these methods are still characterized by a limited number of high-quality clinical studies, necessitating

caution in their widespread application and highlighting the need for further research to confirm their efficacy and safety.[84] The literature also emphasizes the significant impact of placebo effects and athlete expectations on both subjective and objective recovery outcomes, complicating the interpretation of research findings and potentially inflating the perceived effectiveness of some interventions.[85] Therefore, future studies should incorporate psychological mechanisms and employ appropriate control methods to rigorously assess the true impact of innovative recovery strategies.

4.1 Discussion - Individual Differences in Recovery

Individual differences in muscle recovery after physical exertion reflect complex interactions between the characteristics of the sport discipline and personal factors. Disciplines dominated by eccentric muscle work, such as long-distance running or soccer, exhibit greater muscle damage and slower recovery compared to sports involving more isometric or dynamic efforts, such as sprinting or weightlifting. [25] Age is a significant determinant of recovery processes—older athletes show reduced muscle protein synthesis capacity and a diminished anabolic response, associated with decreased levels of hormones like testosterone and IGF-1. 86

Sex influences recovery through hormonal and inflammatory differences; women often demonstrate lower levels of muscle damage markers and faster recovery, which may be related to the anti-inflammatory and antioxidant effects of estrogens. [87], [88] Other individual factors such as genetics, training status, nutritional state, and sleep quality and quantity also modulate muscle repair processes, affecting the rate and effectiveness of recovery. [89] Considering these variables is crucial when designing personalized recovery strategies and training programs aimed at maximizing performance and minimizing injury risk.

4.2 Discussion - Practical Implications and Applications

Understanding the mechanisms of performance fatigue and their dependence on task specificity forms the foundation for effective design of training programs that can be precisely tailored in terms of intensity and exercise type to the individual needs and capacities of athletes. [90] In the context of rehabilitation, this knowledge enables the development of effective therapeutic plans aimed at individuals with various neuromuscular disorders, promoting the optimization of the recovery process. [21] Furthermore, applying this knowledge to improve physical performance allows for optimization of training strategies aimed at increasing endurance and delaying the onset of fatigue during exercise. [8]

For coaches, physiotherapists, and athletes themselves, integrating recovery strategies within the overall training plan is crucial, as it supports faster and more efficient restoration of the body after exertion.[91] Additionally, educating athletes about the importance of recovery and its methods is an essential element supporting long-term improvement in sports performance and reduction of injury risk. [83]

5. Conclusions

The literature analysis indicates that exercise-induced fatigue is a complex phenomenon influenced by both central and peripheral mechanisms. The impact of neurotransmitters such as serotonin, dopamine, and noradrenaline plays a key role in regulating motivation, fatigue perception, and maintaining vigilance during physical activity. [9]

Furthermore, peripheral fatigue is associated with impairments in excitation–contraction coupling and accumulation of metabolites such as hydrogen ions and inorganic phosphates, leading to reduced muscle function efficiency.[92] The nature of fatigue also depends on the type of muscle contraction performed—concentric contractions cause faster onset of fatigue compared to isometric or eccentric contractions. [93]

Individual differences, including psychological factors and training status, significantly affect susceptibility to both mental and physical fatigue, necessitating further research that considers these variables. [15]

In sports practice, optimizing recovery strategies should be tailored to the specific demands of the sport discipline and the individual characteristics of the athlete to effectively counteract fatigue and support performance and motivational functions. [83]

6. Summary

A review of current research on recovery strategies highlights a wide range of methods supporting the body's restoration after physical exertion, such as active recovery, manual techniques, supplementation, and modern fatigue-monitoring technologies. Numerous randomized controlled trials and meta-analyses confirm the effectiveness of selected interventions; however, the quality of some studies requires further improvement, and results can be inconsistent, especially in the context of individual differences and the specificity of sports disciplines.

Identified research gaps include, among others, the long-term effects of specific strategies on training adaptations, the molecular mechanisms underlying recovery, and the optimization of protocols that consider diverse individual factors such as age, sex, and training status. Further high-quality studies with placebo control and greater methodological standardization are needed.

The importance of a holistic approach to recovery is emphasized, integrating physiological, psychological, and behavioral aspects, as well as the individualization of recovery strategies. Comprehensive recovery management is key to optimizing performance, reducing injury risk, and improving the quality of life for athletes and physically active individuals.

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