



ZIEMIŃSKI, Adam, GRZEŚKIEWICZ, Katarzyna and MIESZKOWSKI, Jan Paweł. The Impact of Basketball Training and Competition on Oxidative Balance, Inflammation Intensity, and Muscle Damage – Perspectives for Recovery: Perspectives for Recovery. *Journal of Education, Health and Sport*. 2026;95:76261. <https://doi.org/10.12775/JEHS.2026.95.76261>

ARTICLE TIMELINE

Received: 24.09.2026

Accepted: 29.10.2026

Published: 03.11.2026

INDEXING & EVALUATION

MEiN points: 40 Unique ID: 201159

Disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

The journal has been awarded 40 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32318). Unique Journal Identifier: 201159. Scientific disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne z 2019 – aktualny rok 40 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu). © The Authors 2026.

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The Impact of Basketball Training and Competition on Oxidative Balance, Inflammation Intensity, and Muscle Damage – Perspectives for Recovery

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Summary

Basketball demands a complex mix of explosive power, endurance, and physical contact, putting significant mechanical and metabolic stress on players. During competition and intense training, repeated sprinting, jumping, and rapid decelerations trigger acute muscle

damage, temporary inflammation, and an overproduction of reactive oxygen species affecting performance, muscle activity, and health.

Muscle disruption is most commonly tracked via elevated serum levels of myoglobin, creatine kinase, and lactate dehydrogenase, each following a distinct recovery timeline. At the same time, players experience a brief inflammatory response—marked by transient leukocytosis and elevations of inflammatory cytokines (such as IL-6, TNF- α , and IL-10) and C-reactive protein—which generally settles within 24 to 48 hours. Acute exertion also causes a temporary shift in redox status, elevating oxidative stress markers (e.g., MDA, protein carbonyls) and depleting reduced glutathione. However, well-structured training and scheduled rest periods help athletes adapt, ultimately improving their resting antioxidant capacity and lowering basal cortisol levels.

In this review, we examine the post-exercise response of muscle damage, inflammation, and oxidative stress markers in basketball. We also evaluate how practical recovery strategies—such as cold-water immersion, massage, active cool-downs, and targeted nutrition—can reduce fatigue and support faster physiological recovery.

Keywords: basketball training, oxidative balance, inflammation, exercise-induced muscle damage

1. Introduction

Basketball is one of the most popular sports globally, played competitively across condensed seasonal schedules (Calleja-González et al. 2016). A standard game according to FIBA regulations lasts 40 minutes, during which male and female players cover distances between 5 and 6 km at average intensities exceeding 85% of maximal heart rate and above the anaerobic threshold (Kostopoulos et al. 2017; Militello et al. 2021; Davis et al. 2022). The intermittent profile of competitive play features rapid transitions between offensive and defensive possessions, characterized by high-frequency accelerations, eccentric braking, multidirectional changes of direction, maximal jumps, and frequent physical impacts between opponents (Calleja-González et al. 2016).

To satisfy the rapid adenosine triphosphate (ATP) turnover required during high-intensity bursts, skeletal muscle relies extensively on anaerobic glycolysis, supplemented by aerobic metabolism during low-intensity recovery periods between sprints. These high metabolic rates, combined with severe mechanical strain on contracting muscle fibers, trigger a cascade of acute physiological disruptions. At the cellular level, repeated eccentric loading and high-intensity effort cause microtrauma in the sarcolemma and myofibrils, initiate localized

and systemic inflammatory cascades, and generate free radicals via mitochondrial and non-mitochondrial enzymatic pathways (Davis et al. 2022).

Understanding the interaction between muscle damage, cytokine-mediated inflammation, and oxidative stress is vital for managing fatigue, designing periodized training cycles, and implementing evidence-based recovery modalities in elite and competitive basketball players (Bolner et al. 2019; Szydłowska et al. 2025; Cichoń-Woźniak et al. 2024; Calleja-González et al. 2016; Wu et al. 2026).

2. Exercise-Induced Muscle Damage (EIMD) in Basketball

2.1 Mechanics of Sarcolemma Disruption

The movement mechanics inherent to basketball—specifically eccentric muscle contractions during deceleration, landing from jumps, and planting for change-of-direction maneuvers—generate mechanical stress across active sarcomeres. This mechanical strain alters sarcolemma permeability and disrupts intracellular calcium handling, triggering proteolytic degradation and leaking intramuscular proteins into the extracellular space and bloodstream (Wu et al. 2026).

The primary serum biomarkers utilized to evaluate the magnitude of EIMD in basketball include creatine kinase (CK), lactate dehydrogenase (LDH), and MYB ((Davis et al. 2022).

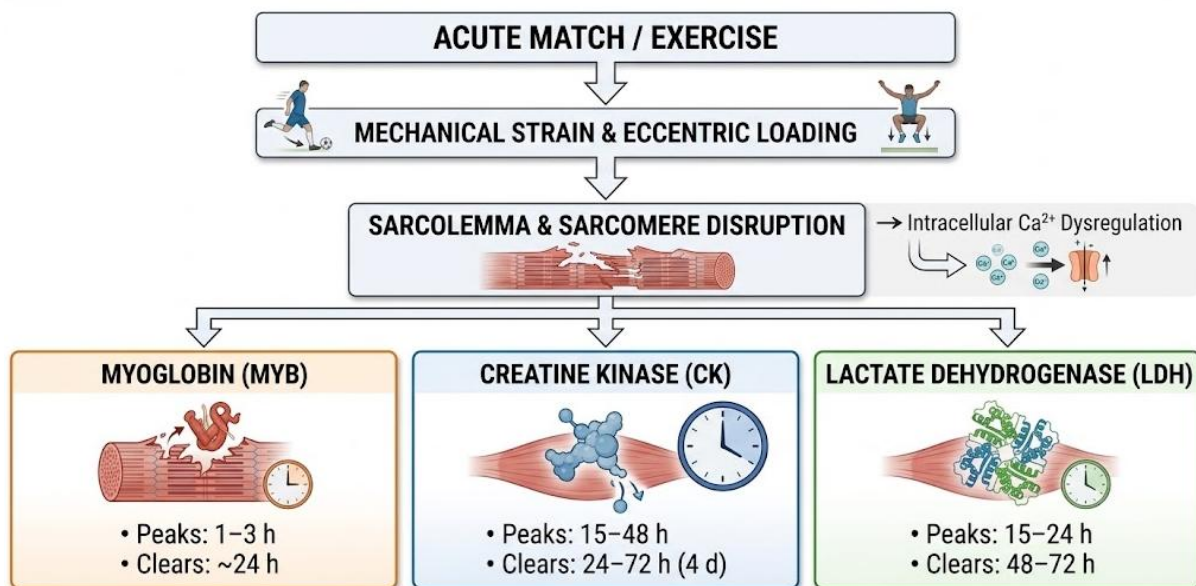


Fig. 1. Mechanism and biomarkers of exercise-induced muscle damage

Source: Own elaboration based on Cichoń-Woźniak et al. (2024), Calleja-González, et al. (2016), and Wu et al. (2026) (visualizations generated using Gemini AI).

2.2 Temporal Kinetics of Muscle Damage Markers

Following an official basketball match or high-intensity simulated game, serum MYB, CK, and LDH activities display distinct time-dependent release and clearance profiles ((Davis et al. 2022; Kostopoulos et al. 2017; KILIÇ et al. 2018) :

- **MYB:** Serum MYB concentrations increase rapidly post-game, reaching peak levels within 1 to 3 hours after physical exertion due to its relatively small molecular weight. MYB levels typically return to baseline resting values within 24 hours post-exercise.
- **CK:** CK activity increases post-contest, reaching peak concentrations between 15 and 48 hours after exercise. CK activity remains elevated for 24 to 72 hours, usually resolving by the fourth day post-match in trained athletes.
- **LDH:** Serum LDH activity rises post-match, peaking at 15 to 24 hours and remaining elevated before returning toward baseline across 48 to 72 hours of recovery (Kostopoulos et al. 2017).

2.3 Individual Variability: Responders vs. Non-Responders

The magnitude of EIMD after competitive play varies significantly among elite basketball athletes. Research categorizes players into distinct physiological subgroups based on serum CK activity changes (Kostopoulos et al. 2017):

- **Responders:** Athletes who demonstrate a marked transient post-game increase in CK activity ranging from 15% to 300% above baseline at 15 hours post-match.
- **Non-Responders:** Athletes who display no significant elevation in post-game CK activity despite participating in the same match demands.

Notably, non-responder players still experience substantial post-game leukocytosis (5% to 85% increases in white blood cell counts), demonstrating that systemic inflammatory activation can occur independently of overt structural muscle membrane damage. Factors influencing this distinction include muscle architecture, genetic variation, training status, and individual exercise experience (Kostopoulos et al. 2017; Mexis et al. 2023; Pernigoni et al. 2024).

2.4 Multi-Day Tournament Kinetics and Sport Comparisons

During congested multi-day tournament schedules (e.g., 3 to 4 games played on consecutive days), serum CK, LDH, and MYB activities increase significantly following the initial contest. However, MYB and CK typically do not reach higher peak levels during subsequent matches on days 2, 3, or 4 compared to the initial post-game peak. This blunted response reflects a combination of active pacing strategies, shortened individual playing times, or early neuromuscular adaptation (the repeated bout effect) (Montgomery et al. 2008; Chatzinikolaou et al. 2014; Doma et al. 2018).

When compared to other elite team ball sports, basketball induces moderate levels of EIMD (Chatzinikolaou et al. 2014). Soccer play generates significantly higher muscle damage indices—producing CK elevations 2 to 3 times greater than basketball—due to continuous high-

speed eccentric running over a larger playing pitch. Volleyball induces the lowest level of muscle damage among court sports, whereas basketball occupies an intermediate position between volleyball and soccer (Chatzinikolaou et al. 2014; Souglis et al. 2015).

3. Intensity and Dynamics of the Inflammatory Response

3.1 Post-Match Leukocytosis and Cell Subsets

A single basketball match triggers acute, systemic immune activation characterized by leukocytosis (Chatzinikolaou et al. 2014). Total circulating white blood cell (WBC) counts increase by approximately 30% post-game and remain elevated for up to 48 hours (Cichoń-Woźniak et al. 2024):

- **Neutrophils:** The delayed phase of post-exercise leukocytosis is predominantly driven by neutrophilia. Mobilized by exercise-induced catecholamine surges and localized tissue injury, circulating neutrophils infiltrate damaged skeletal muscle to clear cellular debris via phagocytosis and localized oxidative bursts. The elevation in neutrophil counts is positively correlated with total playing time during the match (Cichoń-Woźniak et al. 2024).
- **Lymphocytes and Monocytes:** Acute exercise induces transient lymphocytosis and monocytosis immediately post-match, which rapidly shifts to lymphopenia during the delayed recovery phase (15 to 39 hours post-game) (Kostopoulos et al. 2017).
- **Platelets:** Circulating platelet counts display a sustained ~13% increase for up to 39 hours post-match, driven by mechanical compression, high vascular pressure, and hemoconcentration (Kostopoulos et al. 2017; Nowaczyk et al. 2023; Pernigoni et al. 2024)

Measured Marker	Pre-Match Baseline	15 h Post-Match Acute Inflammation	39 h Post-Match Recovery Phase	Primary Physiological Driver
 WBC ($10^3/\mu\text{L}$)	5.1 ± 1.0	6.4 ± 0.8 (+31%)	6.4 ± 1.3 (+29%)	Systemic immune mobilization & tissue repair 
 Neutrophils	Baseline	Elevated	Elevated	Phagocytic clearance of muscle microtrauma 
 Lymphocytes	Baseline	Reduced (%)	Reduced (%)	Post-exercise extravasation & redistribution 
 Platelets ($10^3/\mu\text{L}$)	239 ± 41	270 ± 48 (+13%)	262 ± 47 (+13%)	Shear stress & vascular release 

Fig. 2. Inflammatory and hematological marker response to exercise.

Source: Own elaboration based on Kostopoulos et al. (2017) and Cichoń-Woźniak et al. (2024) (visualizations generated using Gemini AI).

3.2 Cytokine Kinetics and Acute-Phase Response

Skeletal muscle contractions and secondary immune cell activation coordinate the synthesis and release of pro- and anti-inflammatory cytokines (Cichoń-Woźniak et al. 2024):

- **Interleukin-6 (IL-6):** Released from contracting muscle fibers as a myokine, IL-6 increases acutely post-match, acting as an energy sensor that stimulates lipolysis and hepatic glycogenolysis. It concurrently triggers the synthesis of anti-inflammatory mediators (Ringleb et al. 2026).
- **Tumor Necrosis Factor-Alpha (TNF- α) and IL-1 β :** Pro-inflammatory cytokines rise transiently post-game, stimulating vascular cell adhesion molecules and facilitating leukocyte diapedesis into micro-damaged muscle tissue (Ringleb et al. 2026)..
- **Interleukin-10 (IL-10):** Following the initial pro-inflammatory cascade, anti-inflammatory IL-10 increases significantly, acting as an immunological "brake" that suppresses monocyte/macrophage activation, inhibits excessive pro-inflammatory cytokine production, and supports tissue remodeling.
- **C-Reactive Protein (CRP):** Produced by liver hepatocytes in response to circulating IL-6, CRP levels increase post-match, peaking within 15 to 24 hours (up to a 3-fold elevation) before declining over subsequent days as tissue repair resolves (Souglis et al. 2025; Szydłowska et al. 2025) .

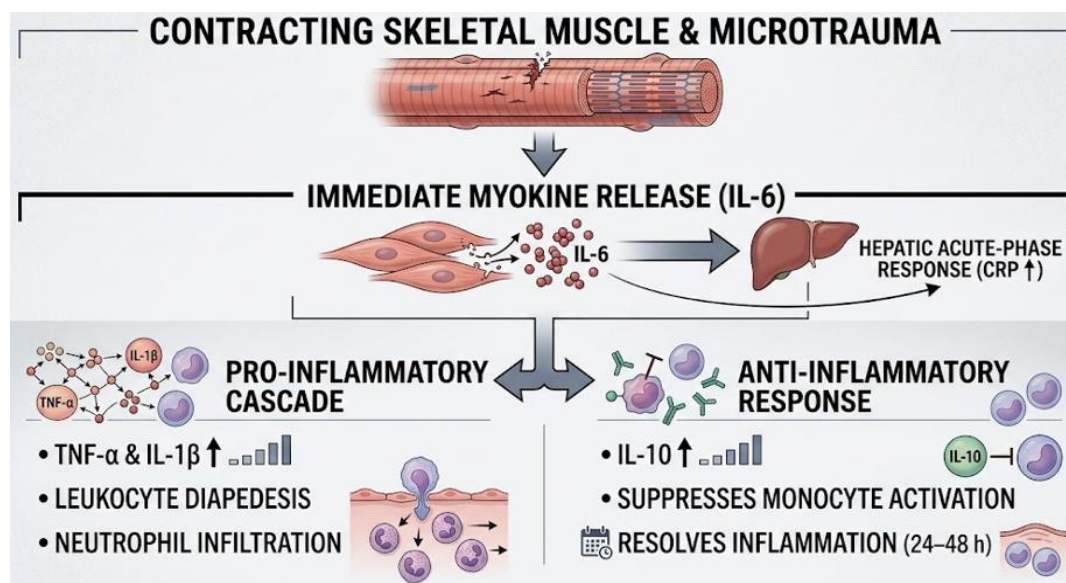


Fig. 3. Contracting skeletal muscle & microtrauma

Source: Own work (visualizations generated using Gemini AI).

3.3 Microcycle Recovery Dynamics

Tracking elite basketball players across a weekly post-match microcycle demonstrates that inflammatory and metabolic alterations are relatively short-lived. Blood lactate, ammonia, cortisol, CRP, CK, and circulating cytokines peak within ~24 hours post-match and subside toward baseline over the subsequent 24 to 48 hours (Chatzinikolaou et al. 2024). Physical performance indicators (jump height, sprint capacity) and muscle soreness (knee flexors and extensors) peak between 24 and 48 hours post-game and fully recover within 48 to 72 hours. Overall, a single basketball match elicits a moderate, brief (~24–48 h) inflammatory response that is considerably less severe than that observed in long-distance endurance running or official soccer matches (Mohr et al. 2016; Pernigoni et al. 2024).

3.4 Impact of Training Status and Rest-Pause Phases

An athlete's resting status and prior training periodization strongly modulate the immunological response to exertion (Cichoń-Woźniak et al. 2024). When trained female basketball players undergo an exhaustive exercise test following a 2-week rest-pause period (consisting of moderate-intensity maintenance workouts), the immune response is markedly regulated:

- Anti-inflammatory **IL-10** increases significantly post-exercise and returns toward baseline during 3 hours of recovery (Cichoń-Woźniak et al. 2024).
- Pro-inflammatory cytokines **IL-1α** and **TNF-α** display no significant post-exercise elevation, while **IL-2** and **IL-13** levels decrease during recovery (Cichoń-Woźniak et al. 2024).

- Post-exercise leukocytosis, lymphocytosis, and granulocytosis occur rapidly but resolve efficiently within 3 hours (Cichoń-Woźniak et al. 2024).

These findings indicate that providing adequate rest-pause phases stabilizes the immune system, preventing chronic low-grade inflammation and enabling athletes to tolerate high-intensity exercise without excessive systemic pro-inflammatory stress.

4. Oxidative Stress and Redox Homeostasis

4.1 Pathways of Free Radical Generation

High-intensity basketball play elevates oxygen consumption and metabolic activity, accelerating the generation of reactive oxygen and nitrogen species (RONS) (Magherini et al. 2019; Baralić et al. 2019). Multiple subcellular sources contribute to RONS production during training and matches:

1. **Mitochondrial Respiration:** Electron leakage at Complexes I and III of the mitochondrial electron transport chain during accelerated aerobic ATP synthesis (Baralić et al. 2019).
2. **Enzymatic Oxidases:** Activation of non-mitochondrial enzymes, including NADPH oxidase (NOX) and xanthine oxidase, stimulated by mechanical strain and muscle membrane disruption.
3. **Phagocytic Respiratory Burst:** Infiltrating neutrophils and macrophages produce superoxide anions and hydrogen peroxide (H_2O_2) during the clearance of damaged muscle tissue (Kozakowska et al. 2015).
4. **Ischemia-Reperfusion & Calcium Overload:** Repeated high-intensity intermittent sprints create transient localized tissue ischemia followed by reoxygenation, disrupting cellular (Ca^{2+}) homeostasis and activating pro-oxidant pathways (Taherkhani et al. 2021).

4.2 Acute Matches vs. Chronic In-Season Adaptation

Acute basketball exertion temporarily overwhelms endogenous antioxidant defenses, inducing transient oxidative stress:

- **Acute Markers:** Capillary blood assessment using the Free Oxygen Radicals Test (FORT) shows significant post-exercise increases in hydroperoxides immediately following a single basketball match or exercise bout. Circulating malondialdehyde (MDA), protein carbonyls (PC), and oxidized glutathione increase for ~24 hours post-

game, accompanied by a transient reduction in reduced glutathione (GSH) (Taherkhani et al. 2021; Meng & Su, 2025)

- **Plasma Antioxidant Mobilization:** Free Oxygen Radical Defense (FORD) values often remain unchanged acutely post-match. This stability is maintained by the rapid mobilization of non-enzymatic antioxidants (ascorbate, uric acid, tocopherol) from tissue stores into plasma to buffer circulating RONS (Wang et al. 2026).

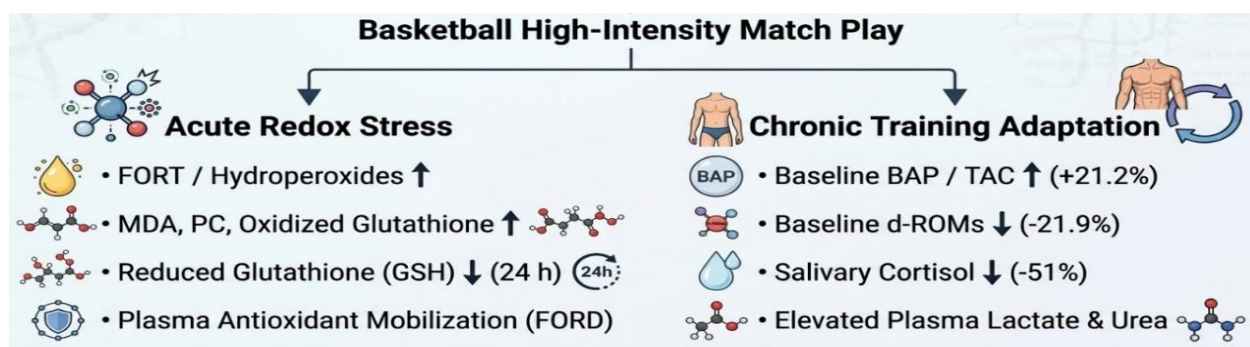


Fig. 4. Physiological effects and adaptations of basketball high-intensity match play

Source: Own elaboration based on Taherkhani et al.(2021), Meng & Su (2025) and Wang et al. (2026). (visualizations generated using Gemini AI).

In contrast to acute exercise stress, chronic in-season basketball training induces favorable adaptations in the endogenous antioxidant defense system (Baralić et al. 2019):

- **Redox Status in Elite Athletes:** Elite female basketball players evaluated during in-season training possess significantly higher resting Biological Antioxidant Potential (BAP, +21.2%) and lower resting reactive oxygen metabolites (d-ROMs, -21.9%) compared to age- and BMI-matched sedentary controls (Militello et al. 2021).
- **Hormonal & Metabolomic Profile:** Trained female basketball players exhibit 51% lower resting salivary cortisol levels alongside higher resting plasma lactate (+460%) and urea (+220%) concentrations. Elevated resting lactate acts as an intracellular buffer and signaling molecule ("lactormone") that supports antioxidant potential and mitochondrial biogenesis without inducing chronic inflammation or overtraining (Militello et al. 2021; Miguel-Ortega et al. 2024).

5. Influencing Factors, Recovery Modalities, and Nutritional Strategies

5.1 Recovery Modalities

Due to condensed competitive schedules (often playing 2 to 3 matches per week), practical recovery interventions are necessary to restore neuromuscular function and attenuate inflammation (Calleja-González et al. 2016):

- **Cold-Water Immersion (CWI):** Immersing the lower limbs or full body in cold water (10.5–12°C for 5–15 minutes, continuously or intermittently, such as 5 times x 1min or 5 times x 2 min significantly reduces post-game delayed onset muscle soreness (DOMS), mid-thigh muscle swelling, and circulating muscle damage markers (CK, MYB). CWI accelerates the recovery of countermovement jump (CMJ) height, repeated sprint ability (RSA), and line-drill performance during multi-day tournaments (Calleja-González et al. 2016).
- **Massage and Stretching:** A 30-minute sports massage administered immediately post-match significantly reduces perceived leg fatigue. Combining sports massage with static stretching yields superior recovery outcomes compared to massage alone, significantly decreasing muscle soreness and accelerating the restoration of repeated-sprint performance (Calleja-González et al. 2016; Davis et al. 2022).
- **Active Recovery (Cool-Down):** Implementing a 10-minute active cool-down protocol post-match (consisting of low-intensity jogging, active breathing, and light stretching) enhances local venous return and accelerates the clearance of blood LDH and MYB back toward resting levels compared to passive rest (Calleja-González et al. 2016).

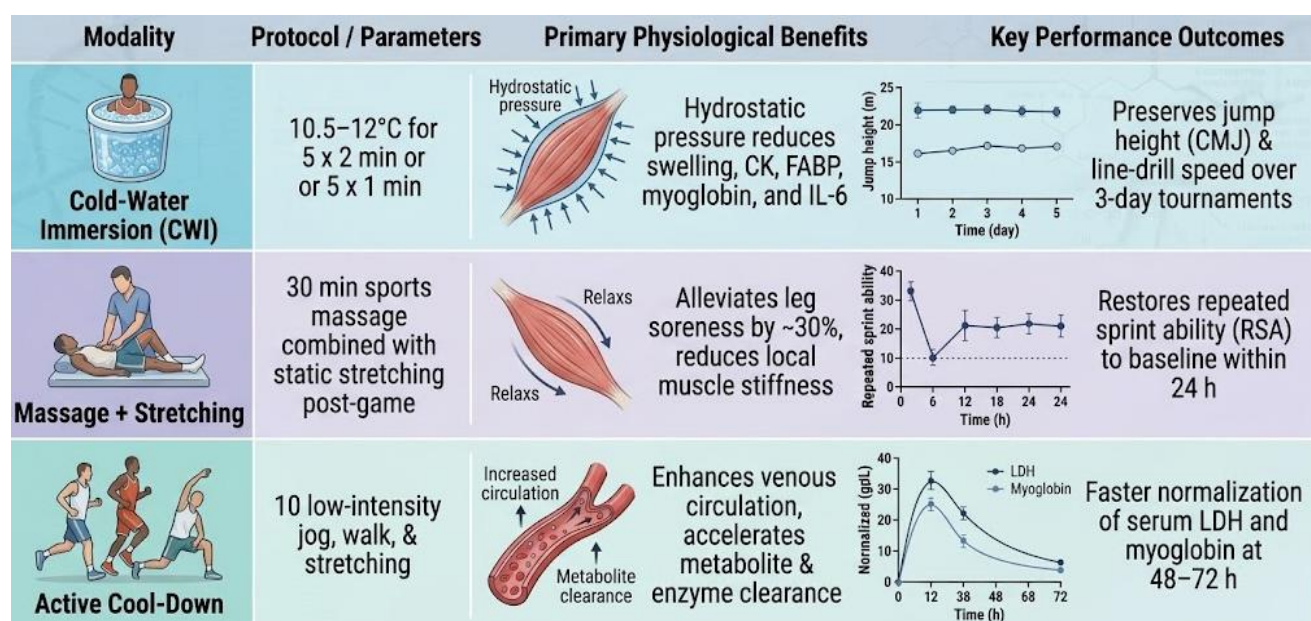


Fig. 5. Models of recovery in basketball (integration).

Source: Own elaboration based on Calleja-González et al. (2016) (visualizations generated using Gemini AI).

5.2 Nutritional Strategies

Nutritional intake directly influences the magnitude of post-game muscle damage, inflammation, and glycogen resynthesis (Kostopoulos et al. 2017):

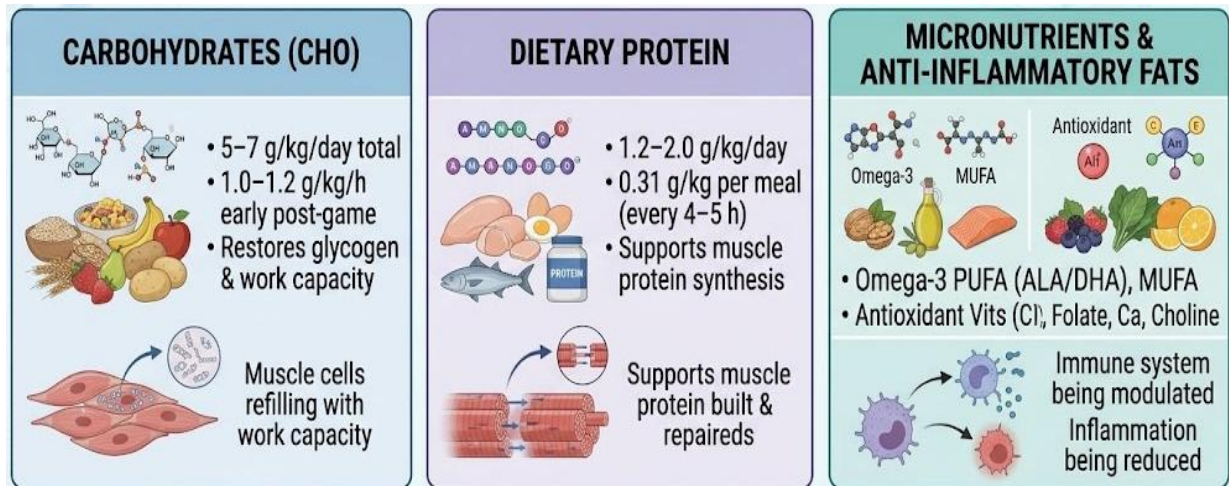


Fig. 6. Post-match recovery nutrition.

Source: Own elaboration based on Kostopoulos et al. (2017) (graphics generated using Gemini AI).

- **Carbohydrate & Protein Timing:** Basketball relies heavily on muscle glycogen. Athletes should consume 5 to 7 g/kg/day of carbohydrates, including 1.0 to 1.2 g/kg/h during early post-game recovery. Consuming protein at 1.2 to 2.0 g/kg/day (distributed as 0.31 g/kg every 4 to 5 hours) provides essential amino acids to stimulate muscle protein synthesis (MPS) and repair eccentric muscle injury (Kostopoulos et al. 2017).
- **Anti-Inflammatory Fatty Acids & Micronutrients:** Match-day and long-term dietary intake of monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA, particularly omega-3 ALA and DHA), folate, calcium, choline, and antioxidant vitamins (C and E) are significantly inversely correlated with post-game WBC, neutrophil, and CK elevations. Conversely, high dietary saturated fat intake augments post-game CK spikes (Kostopoulos et al. 2017; Baralić et al. 2019).
- **Nutraceutical Supplementation:** Long-term administration (45 days) of a protective antioxidant complex (GE132®, containing *Ganoderma lucidum* extract, royal jelly, and resveratrol) in elite female basketball players significantly attenuates acute post-exercise FORT hydroperoxide elevations and lowers resting baseline CK and LDH enzyme activities (Baralić et al. 2019).

6. Conclusion

High-intensity basketball match play and training impose a unique combination of mechanical strain and metabolic fatigue on athletes. These physiological demands trigger a well-defined cascade of acute disruptions, including microstructural muscle damage, systemic inflammation, and transient oxidative stress.

The temporal kinetics of blood biomarkers offer valuable insights into player recovery. Muscle membrane disruption is marked by an early surge in serum myoglobin (peaking within 1–3 hours), followed by more sustained elevations in creatine kinase and lactate dehydrogenase, which generally return to baseline over 24 to 72 hours. Importantly, individual variability in these responses underscores the distinction between "CK responders" and "non-responders," highlighting that muscle damage cannot be assumed to be uniform across a team. Concurrently, match play triggers a transient inflammatory cascade characterized by leukocytosis, neutrophilia, and moderate elevations in IL-6, TNF- α , and CRP, counterbalanced by the anti-inflammatory action of IL-10 to promote tissue resolution within 24–48 hours. At the redox level, acute exertion temporarily overwhelms antioxidant defenses, elevating markers of lipid and protein oxidation. However, chronic periodized training and structured rest-pause phases drive positive adaptations, enhancing resting antioxidant potential (BAP/TAC) and attenuating basal cortisol levels to protect against overtraining.

To mitigate these acute physiological stressors and support consistent performance across congested competitive schedules, practitioners should implement targeted, multi-modal recovery frameworks. Combining cold-water immersion, active cool-downs, and sports massage with stretching helps accelerate waste clearance and alleviate muscle soreness. Furthermore, periodized nutritional support—focusing on timely carbohydrate and protein intake alongside anti-inflammatory fatty acids and antioxidants—provides the biochemical foundation required to optimize muscle repair, restore redox balance, and enhance long-term athletic durability.

Authors' contributions

Study design, supervision of experimental implementation, technical editing of the article, approval of the final version: AZ, KG, JM. Design of the intervention protocol, preparation of graphs, review of sources: AZ, KG. Data analysis: Data collection, statistical analyses, and writing of the initial draft: AZ, KG, JM.

All authors contributed to the approval of the final version of the article and assume responsibility for all aspects of the work.

Declarations

Use of Generative AI in Visual Content Preparation: Conceptual design and data synthesis for Figures 1–7 were performed by the authors. The graphical representations were rendered using the Gemini model (Google AI) and subsequently verified for factual accuracy and biological relevance by the authors.

Availability of data and materials

The bibliographic analyses used in presented publication are available from the corresponding author on reasonable request.

Competing interests

The authors declare no conflict of interest.

Funding

- none

Acknowledgements

- none

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