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Impact of excessive simple sugar consumption on the pathophysiology of muscle injury in athletes

Impacto do consumo excessivo de açúcares simples na fisiopatologia da lesão muscular em atletas

Impacto del consumo excesivo de azúcares simples en la fisiopatología de la lesión muscular en atletas

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Abstract:
Structural integrity and muscle regeneration capacity are essential for athletic performance and recovery. Although carbohydrate intake is widely recommended in sports nutrition to maintain glycogen stores and support energy recovery, excessive and frequent consumption of simple sugars, particularly those found in ultra-processed foods and sugar-sweetened beverages, may contribute to metabolic and inflammatory alterations that compromise skeletal muscle homeostasis. This study aimed to analyze the influence of excessive simple sugar consumption on the pathophysiology of muscle injury in athletes, investigating the potential biochemical, inflammatory, and cellular mechanisms associated with impaired tissue repair, as well as potentially mitigating nutritional strategies. This study consists of a narrative literature review conducted through searches in PubMed/MEDLINE, SciELO, ScienceDirect, MDPI, PLOS ONE, the CAPES Journal Portal, and specialized scientific journals, using standardized descriptors from the MeSH and DeCS vocabularies. The analyzed studies suggest that glycemic overload, particularly when associated with inadequate dietary patterns, may promote a pro-inflammatory environment, alterations in redox balance, and increased oxidative stress, in addition to being associated with changes in biochemical markers such as high-sensitivity C-reactive protein (hs-CRP), creatine kinase (CK), and lactate dehydrogenase (LDH). At the structural level, the accumulation of advanced glycation end-products (AGEs) may promote collagen cross-linking in muscular connective tissues, potentially altering their biomechanical properties and their ability to adapt to mechanical loads. Furthermore, changes in insulin signaling and the satellite cell microenvironment may interfere with myogenic proliferation, protein synthesis, and muscle regeneration. Conversely, nutritional strategies based on a balanced diet, the consumption of foods rich in bioactive compounds, such as polyphenols, and the combination of carbohydrates and proteins may contribute to the modulation of inflammatory responses and muscle recovery. It is concluded that chronic and excessive simple sugar consumption may represent an unfavorable metabolic factor for skeletal muscle homeostasis, although further clinical studies involving athletes are needed to establish its direct influence on muscle injury incidence and recovery time. Therefore, dietary adequacy represents a relevant strategy for supporting muscle health and athletic recovery.

Keywords:
Dietary Sugars. Athletic Injuries. Muscle Damage. Advanced Glycation End-Products. Satellite Cells.

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Resumo:
A integridade estrutural e a capacidade de regeneração muscular são fundamentais para o desempenho esportivo e a recuperação de atletas. Embora o aporte de carboidratos seja amplamente recomendado na nutrição esportiva para a manutenção dos estoques de glicogênio e a recuperação energética, o consumo excessivo e frequente de açúcares simples, especialmente os provenientes de alimentos ultraprocessados e bebidas açucaradas, pode contribuir para alterações metabólicas e inflamatórias capazes de comprometer a homeostase do tecido musculoesquelético. O presente estudo teve como objetivo analisar a influência do consumo excessivo de açúcares simples na fisiopatologia da lesão muscular em atletas, investigando os possíveis mecanismos bioquímicos, inflamatórios e celulares relacionados ao comprometimento do reparo tecidual, bem como estratégias nutricionais potencialmente atenuantes. Trata-se de uma revisão bibliográfica narrativa, realizada por meio de pesquisas nas bases PubMed/MEDLINE, SciELO, ScienceDirect, MDPI, PLOS ONE, Portal de Periódicos CAPES e periódicos científicos especializados, utilizando descritores padronizados dos vocabulários MeSH e DeCS. Os estudos analisados apontam que a sobrecarga glicêmica, especialmente quando associada a padrões alimentares inadequados, pode favorecer um ambiente pró-inflamatório, alterações no equilíbrio redox e aumento do estresse oxidativo, além de estar relacionada a alterações em marcadores bioquímicos, como proteína C-reativa ultrassensível (hs-CRP), creatina quinase (CK) e lactato desidrogenase (LDH). No âmbito estrutural, o acúmulo de produtos finais de glicação avançada (AGEs) pode favorecer a formação de ligações cruzadas no colágeno dos tecidos conjuntivos musculares, potencialmente alterando suas propriedades biomecânicas e sua capacidade de adaptação às cargas mecânicas. Adicionalmente, alterações na sinalização da insulina e no microambiente das células-satélite podem interferir nos processos de proliferação miogênica, síntese proteica e regeneração muscular. Em contrapartida, estratégias nutricionais que priorizam uma alimentação equilibrada, o consumo de alimentos ricos em compostos bioativos, como polifenóis, e a associação de carboidratos e proteínas podem contribuir para a modulação da resposta inflamatória e para a recuperação muscular. Conclui-se que o consumo excessivo e crônico de açúcares simples pode representar um fator metabólico desfavorável à homeostase musculoesquelética, embora sejam necessários estudos clínicos específicos com atletas para estabelecer sua influência direta sobre a incidência de lesões musculares e o tempo de recuperação. Dessa forma, a adequação do padrão alimentar constitui uma estratégia relevante para favorecer a saúde muscular e a recuperação esportiva.

Palavras-chave:
Açúcares da Dieta. Lesões Esportivas. Dano Muscular. Produtos Finais de Glicação Avançada. Células-Satélite.

Resumen:
La integridad estructural y la capacidad de regeneración muscular son fundamentales para el rendimiento deportivo y la recuperación de los atletas. Aunque en nutrición deportiva se recomienda ampliamente el aporte de carbohidratos para mantener las reservas de glucógeno y recuperar la energía, el consumo excesivo y frecuente de azúcares simples, especialmente de alimentos ultraprocesados y bebidas azucaradas, puede contribuir a alteraciones metabólicas e inflamatorias capaces de comprometer la homeostasis del tejido musculoesquelético. Este estudio tuvo como objetivo analizar la influencia del consumo excesivo de azúcares simples en la fisiopatología de la lesión muscular en atletas, investigando los posibles mecanismos bioquímicos, inflamatorios y celulares relacionados con el deterioro de la reparación tisular, así como las estrategias nutricionales que podrían mitigarlo. Se realizó una revisión bibliográfica narrativa mediante búsquedas en PubMed/MEDLINE, SciELO, ScienceDirect, MDPI, PLOS

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ONE, el Portal de Revistas CAPES y revistas científicas especializadas, utilizando descriptores normalizados de los vocabularios MeSH y DeCS. Los estudios analizados indican que la sobrecarga glucémica, especialmente cuando se asocia con patrones alimentarios inadecuados, puede favorecer un entorno proinflamatorio, alteraciones del equilibrio redox y un aumento del estrés oxidativo, además de asociarse con cambios en marcadores bioquímicos como la proteína C reactiva de alta sensibilidad (hs-CRP), la creatina quinasa (CK) y la lactato deshidrogenasa (LDH). En el plano estructural, la acumulación de productos finales de glicación avanzada (AGEs) puede favorecer la formación de enlaces cruzados en el colágeno de los tejidos conjuntivos musculares y alterar sus propiedades biomecánicas y su capacidad de adaptación a las cargas mecánicas. Asimismo, los cambios en la señalización de la insulina y en el microambiente de las células satélite pueden interferir en la proliferación miogénica, la síntesis de proteínas y la regeneración muscular. Por su parte, las estrategias nutricionales que priorizan una alimentación equilibrada, el consumo de alimentos ricos en compuestos bioactivos, como los polifenoles, y la combinación de carbohidratos y proteínas pueden contribuir a modular la respuesta inflamatoria y a favorecer la recuperación muscular. Se concluye que el consumo excesivo y crónico de azúcares simples puede constituir un factor metabólico desfavorable para la homeostasis musculoesquelética, aunque se requieren estudios clínicos específicos en atletas para establecer su influencia directa sobre la incidencia de lesiones musculares y el tiempo de recuperación. Por ello, adecuar el patrón alimentario constituye una estrategia relevante para favorecer la salud muscular y la recuperación deportiva.

Palabras clave:
Azúcares de la dieta. Lesiones deportivas. Daño muscular. Productos finales de glicación avanzada. Células satélite.

2 Introduction

The structural integrity and regenerative capacity of the musculoskeletal system are critical to physical performance and long-term function in athletes who compete in sports with high mechanical demands. Sports nutrition has traditionally emphasized substantial carbohydrate intake to maximize muscle and liver glycogen stores before and after exercise (Burke; Collier; Hargreaves, 2019). Adjusting the glycemic index (GI) of foods consumed during recovery and training directly affects glycogen resynthesis and subsequent performance (Burke; Collier; Hargreaves, 2019; Kaviani *et al.*, 2011). Recent research in metabolic biochemistry and immunology, however, indicates that chronically excessive intake of refined free sugars, particularly sucrose, fructose, and corn syrup, has detrimental effects on the cellular microenvironment that extend beyond daily energy balance, leading to recurrent inflammatory episodes and reduced force-generating capacity (Ma *et al.*, 2022; Hou *et al.*, 2025; Zhu et al., 2024).

Excessive intake of simple sugars strongly modulates systemic and local inflammatory pathways (Ma *et al.*, 2022). Frequent hyperglycemic fluctuations trigger nonenzymatic reactions between reducing sugars and free amino groups on structural proteins (the Maillard

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reaction), leading to the formation and accumulation of advanced glycation end products (AGEs) (Olson *et al.*, 2021). The interaction of AGEs with their specific transmembrane receptor (RAGE) activates intracellular cascades dependent on nuclear factor kappa β (NF-κβ), triggering the overexpression of proinflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and interleukin-1 beta (IL-1β) (Ma *et al.*, 2022; P2Life, 2021).

Studies in athletes have also found direct clinical and laboratory correlations between high intake of free fructose and glucose and significant increases in serum inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP) and adenosine deaminase (ADA) (Hou *et al.*, 2025). This chronic subclinical inflammation occurs alongside mitochondrial overload in myocytes, with increased generation of reactive oxygen species (ROS) and oxidative stress. These changes compromise sarcolemmal integrity and exacerbate post-exercise enzyme leakage, as reflected in elevated creatine kinase (CK) and lactate dehydrogenase (LDH) levels (Baleeiro *et al.*, 2021; Sports Performance Bulletin, 2022). Nutritional strategies using complex food matrices or bioactive compounds and polyphenols, including tart cherry juice, low-fat chocolate milk, honey-sweetened beverages, and carbohydrate supplements combined with protein or whey, have shown the potential to reduce delayed-onset muscle soreness (DOMS), speed functional recovery, and limit increases in these markers (Hemmatti et al., 2024; Fabre et al., 2022; Liang et al., 2022; Molaeikhatabadi et al., 2022; Gao *et al.*, 2024).

At the histological and ultrastructural levels, continued AGE deposition promotes abnormal cross-link formation in types I and III collagen, the main components of the endomysium, perimysium, and epimysium (Olson *et al.*, 2021). This architectural change decreases tissue compliance and viscoelasticity, making muscle fascicles and tendons stiffer and more susceptible to microtears during eccentric contractions and biomechanical overload. Other systemic and behavioral factors, such as unbalanced diets in amateur athletes and oral disease, have also been epidemiologically associated with a higher incidence of musculotendinous injuries in athletes (Monteiro, 2022; Rodrigues et al., 2025). At the same time, glycemic stress and peripheral insulin resistance disrupt the muscle satellite cell niche, impairing the cells' ability to proliferate and differentiate during myogenic repair (Olson *et al.*, 2021; Hou *et al.*, 2025).

The need to integrate the cytological, biochemical, and clinical mechanisms linking athletes' dietary patterns to tissue susceptibility warrants an in-depth literature review to clarify the pathophysiological consequences of glycemic overload in skeletal muscle.

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3 Objectives

3.1 General objective

To analyze how excessive simple sugar intake affects the pathophysiology of muscle injury in athletes by identifying biochemical, inflammatory, and cellular mechanisms associated with impaired tissue repair and examining nutritional strategies that may mitigate this impairment and support recovery.

3.2 Specific objectives

a. To investigate oxidative stress pathways and changes in biomarkers of inflammation and tissue damage (hs-CRP, ADA, CK, and LDH) resulting from dietary glucose and fructose overload in athletes;
b. To describe the formation and accumulation of advanced glycation end products (AGEs) and activation of the AGE/RAGE axis in the muscle extracellular matrix and adjacent connective tissue;
c. To assess the effects of collagen matrix stiffness and glycation on tissue viscoelasticity, muscle strength, and biomechanics during high-intensity exercise;
d. To clarify the cellular effects of glycemic stress on the satellite cell niche and myocyte mitochondrial homeostasis, and the compensatory role of supplementing carbohydrates with proteins or consuming polyphenol-rich foods and functional food matrices in muscle damage and soreness.

4 Review methodology

This qualitative, exploratory study was designed as a narrative literature review to develop an integrated understanding of biomolecular, histopathological, and clinical mechanisms.

The literature search used leading national and international electronic databases and publications in health and biomedical sciences: PubMed/MEDLINE (via the *National Center for Biotechnology Information*, NCBI), SciELO (*Scientific Electronic Library Online*), ScienceDirect/Elsevier, PLOS ONE, MDPI, Frontiers in Physiology, and the CAPES Journal Portal. Specialized journals in sports nutrition and exercise physiology were also consulted,

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including the *Revista Brasileira de Nutrição Esportiva* (RBNE) and the *Journal of Human Kinetics*.

Studies were identified using standardized descriptors from the controlled vocabularies *Medical Subject Headings* (MeSH) and Health Sciences Descriptors (DeCS). The following structured search terms were used:

"Dietary Sugars," "Sugar-Sweetened Beverages," "Fructose," "Glycemic Index," "Skeletal Muscle," "Muscle Damage," "Athletic Injuries," "Muscle Regeneration," "Inflammation," "Advanced Glycation End Products," "Oxidative Stress," "C-Reactive Protein," and "Creatine Kinase."

The following criteria were established to screen and refine the search results:

a) Inclusion criteria:

- Original articles (clinical trials, cohort studies, cross-sectional studies, and *in vivo*/*in vitro* experiments) and systematic or narrative reviews;
- Publications written in English or Portuguese;
- Studies published between 2006 and 2026, with priority given to recent evidence (the last five years) while retaining landmark work on glycogen kinetics;
- Studies that directly investigated the effects of free sugars, sugar-sweetened beverages, or high-glycemic-load diets on inflammatory measures, oxidative stress, myofascial stiffness, tissue damage, gastrointestinal symptoms, or muscle regeneration in athletes, physically active populations, or representative cellular or animal models.

b) Exclusion criteria:

- Duplicate articles across the databases consulted;
- Studies outside the proposed cellular, metabolic, or muscular scope;
- Editorials, letters to the editor, conference abstracts without a published full article, and unindexed gray literature.

5 Development and discussion

The relationship between dietary carbohydrate intake and exercise physiology has historically been defined by the need to resynthesize muscle glycogen after exercise (Burke; Collier; Hargreaves, 2019). The glycemic index of sports bars and supplements consumed

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during recovery significantly affects metabolism and exercise performance the next day (Kaviani *et al.*, 2011). The distinction between the bioenergetic benefits of carbohydrates and their potential to induce tissue pathology lies in the pattern and quality of intake. Moderate, strategically timed consumption around training supports homeostasis, whereas chronic overload with refined free sugars such as sucrose, glucose, and high-fructose corn syrup strongly activates proinflammatory pathways and systemic oxidative stress (Ma *et al.*, 2022).

5.1 Serum biomarkers of muscle damage and systemic inflammatory response

Dietary fructose and sucrose overload significantly increases circulating inflammatory mediators and acute-phase proteins (Ma *et al.*, 2022). In athletes participating in high-impact team sports, excessive consumption of sweetened beverages correlates with elevated plasma levels of high-sensitivity C-reactive protein (hs-CRP) and adenosine deaminase (ADA) (Hou *et al.*, 2025). Elevated hs-CRP reflects chronic subclinical inflammation, which diminishes cellular antioxidant capacity and predisposes skeletal muscle to exaggerated inflammatory responses to the mechanical microtrauma that typically occurs during training (Sports Performance Bulletin, 2022).

Susceptibility to sarcolemmal disruption under mechanical stress also increases markers of cellular leakage. Creatine kinase (CK) and lactate dehydrogenase (LDH), commonly used to monitor training load and recovery, showed slower clearance and higher peaks in individuals exposed to sugar-sweetened beverages (Baleeiro *et al.*, 2021). Interventions combining carbohydrates and proteins, such as native whey protein, have been effective in limiting CK and LDH leakage, reducing muscle damage, and controlling the plasma inflammatory response during intense tournaments such as Rugby Sevens and in endurance runners (Fabre et al., 2022; Liang et al., 2022).

Functional foods or beverages enriched with natural sugars and bioactive compounds also showed favorable effects on muscle soreness and damage. In strength-trained women, a honey-sweetened beverage substantially reduced muscle soreness and accelerated recovery of physical performance after eccentric exercise-induced muscle damage (Hemmatti et al., 2024). Short-term consumption of low-fat chocolate milk similarly reduced delayed-onset muscle soreness (DOMS) and preserved performance in women's collegiate badminton players (Molaeikhatabadi et al., 2022).

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5.2 Advanced glycation (AGEs/RAGE), strength decline, and extracellular matrix changes

At the molecular and ultrastructural levels, repeated hyperglycemic spikes promote the nonenzymatic binding of reducing sugars to long-lived proteins, such as collagen fibers in the perimysium and endomysium (the Maillard reaction), giving rise to advanced glycation end products (AGEs) (Olson *et al.*, 2021). The incorporation of AGEs into the extracellular matrix (ECM) has two principal pathophysiological consequences:

a. **Biomechanical changes and functional decline:** Abnormal intramolecular and intermolecular cross-links form in types I and III collagen, stiffening the tissue architecture. Loss of compliance and viscoelasticity reduces the ability of the muscle–tendon unit to absorb energy during the eccentric phase of contraction, predisposing athletes to acute muscle strains and overuse tendinopathies (Olson *et al.*, 2021; Hou *et al.*, 2025). Longitudinal cohort studies also associate high intake of added sugars and their liquid and solid sources with accelerated decline in handgrip strength and impaired neuromuscular function (Zhu et al., 2024);
b. **Activation of the AGE/RAGE pathway:** The ligand–receptor interaction in myocytes activates the NF-κβ transcription factor pathway, leading to sustained transcription of inflammatory cytokines (TNF-α, IL-6, IL-1β) and induction of endoplasmic reticulum stress, which prevents normal resolution of post-exercise inflammation (Ma *et al.*, 2022; P2Life, 2021).

5.3 Systemic factors, ultra-endurance management, and bioactive food matrices for recovery

Beyond the direct effects of sugar on myocytes, the literature indicates that the overall dietary profiles and lifestyle habits of amateur and semiprofessional athletes, including flag football players, often involve inadequate daily macronutrient intake and supplementation, increasing susceptibility to musculotendinous injuries (Rodrigues et al., 2025). Regular sports participation without adequate oral care has also been associated with a higher prevalence of oral disease, such as periodontitis and dental caries. These conditions act as sources of systemic inflammation and correlate significantly with musculotendinous injury incidence among physically active people (Monteiro, 2022).

During ultra-endurance events such as one-day trail ultramarathons, carbohydrate intake during the race is essential to maintain euglycemia. Systematic reviews indicate, however, that excessive doses or an unbalanced supply of simple sugars contribute to discomfort and severe

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gastrointestinal problems, increasing perceived overall fatigue (Arribalzaga et al., 2021). Compared with traditional sports drinks composed exclusively of refined simple sugars, polyphenol-rich beverages such as tart cherry juice have been shown to improve substrate metabolism, reduce oxidative stress, and optimize functional recovery during and after cycling (Gao *et al.*, 2024).

5.4 Satellite cell impairment and myogenic repair

Muscle regeneration primarily depends on recruiting, activating, and differentiating quiescent satellite cells located between the basal lamina and the myofibrillar sarcolemma. Hyperglycemic environments characterized by tissue insulin resistance and oxidative stress profoundly affect the regenerative niche:

a. Signaling through the Akt/mTOR phosphorylation cascade, essential for protein biosynthesis and compensatory hypertrophy, is attenuated by inflammatory cytokine-mediated serine phosphorylation of insulin receptor substrate 1 (IRS-1);
b. Excess mitochondrial ROS suppress myogenic precursor cell proliferation and disrupt the coordinated expression of myogenic regulatory transcription factors such as Pax7, MyoD, and myogenin, delaying myoblast fusion and repair of damaged myofibrils (Olson *et al.*, 2021).

As a result, myocytes remain in a degenerative state for prolonged periods. This favors replacement of contractile fibers with interstitial fibrosis and disorganized connective tissue, increasing the recurrence of muscle injuries in athletes exposed to high training loads.

6 Final considerations

The findings gathered in this review indicate that sustained excessive consumption of simple sugars is a determining factor in the pathogenesis of muscle injuries and impaired recovery in people engaged in high-performance exercise. Beyond serving as an energy source, an overload of refined glucose, sucrose, and fructose triggers a proinflammatory cellular environment characterized by increased serum biomarkers (hs-CRP, ADA, CK), heightened oxidative stress, loss of strength, and sarcolemmal dysfunction.

Chronic AGE deposition also compromises the biomechanical properties of the extracellular matrix by inducing collagen cross-links, decreasing muscle tissue compliance and increasing vulnerability to mechanical strain. At the same time, impaired satellite cell

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recruitment and related factors such as oral disease and inadequate nutritional intake delay myogenic repair.

These findings underscore the importance of sports nutrition strategies that moderate refined free sugar intake and prioritize complex carbohydrate sources, appropriate carbohydrate-to-protein ratios, and food matrices rich in bioactive compounds, such as honey, chocolate milk, or tart cherry juice. Limiting refined sugars supports general metabolic health and can serve as a preventive measure to reduce recurrent myofascial injuries and optimize tissue regeneration in athletes.

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