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Hypothyroidism and Musculoskeletal Injury Susceptibility in Athletes: A Narrative Review

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Abstract

Background

Thyroid hormones are key elements regulating the body's metabolism, skeletal muscle function and connective tissue homeostasis. Therefore, hypothyroidism may affect exercise performance and regeneration processes. Its role as a factor increasing susceptibility to sports injuries remains insufficiently defined.

Objective

This narrative review aims to evaluate evidence regarding the impact of hypothyroidism on the musculoskeletal system and the risk of sports injuries with attention paid to the underlying physiological mechanisms.

Methods

Studies published from 1986–2026 were searched in the PubMed database. The literature was reviewed using the following keywords: hypothyroidism, skeletal muscle, tendinopathy, exercise performance, athletes, and musculoskeletal injuries. Additional papers were identified by reviewing references of relevant articles.

Results

Available evidence indicates that thyroid hormones regulate multiple processes relevant to musculoskeletal health, including mitochondrial oxidative metabolism, neuromuscular function, and extracellular matrix remodeling. Hypothyroidism has been associated with reduced aerobic capacity, impaired muscle strength, and delayed muscle regeneration after injury. Experimental data also suggest disturbances in tendon homeostasis, which may influence tissue adaptation to mechanical loading. Although these findings provide a plausible link between hypothyroidism and increased injury susceptibility, direct evidence in athletic population remains limited.

Conclusions

Current literature suggests an association between hypothyroidism and impaired musculoskeletal function that may contribute to increased susceptibility to sports-related injuries. However, due to the lack of prospective studies in athletes, hypothyroidism should be considered a potential modifying factor rather than a risk factor for injury. Further sport-specific research is required to clarify its significance in injury prevention and athletic management.

Keywords: hypothyroidism; athletes; musculoskeletal injury; skeletal muscle; tendon; thyroid hormones; exercise physiology; sports medicine

1. Introduction

Hypothyroidism is a clinical syndrome resulting from insufficient production or peripheral action of thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), leading to a generalized reduction in metabolic activity across multiple organ systems. At the cellular level, thyroid hormone deficiency is associated with decreased mitochondrial oxidative capacity, impaired protein synthesis, and altered ion and fluid homeostasis, resulting in systemic metabolic slowing and interstitial tissue changes [3,12].

From a clinical perspective, hypothyroidism presents with a wide range of symptoms, including chronic fatigue, weight gain, reduced cold tolerance, bradycardia, delayed post-exercise recovery, and neuromuscular disturbances. This is particularly relevant in physically active individuals, as these symptoms may directly impair training adaptation, reduce training quality, and negatively affect motivation to engage in physical activity. [6,13].

Thyroid hormones are key regulators of energy metabolism, thermogenesis, and neuromuscular function. They influence skeletal muscle contractility, fiber type composition, mitochondrial biogenesis, and substrate utilization during exercise, which together support optimal exercise performance and resistance to fatigue. Experimental and clinical evidence further suggests that thyroid hormone signaling is involved in skeletal muscle remodeling and regeneration processes, which are critical in response to repetitive mechanical loading and microtrauma in sport [3,12,13].

In addition, emerging evidence indicates that thyroid hormones contribute to connective tissue homeostasis through regulation of collagen turnover and extracellular matrix remodeling within tendons and skeletal muscle. This is of particular relevance in sports medicine, where tendon adaptation and load tolerance depend on balanced extracellular matrix remodeling and mechanical stress responses [1,8].

In the context of sports medicine, optimal endocrine function is essential for maintaining adequate training adaptation, recovery, and resistance to musculoskeletal overload. Even subtle disturbances in thyroid hormone levels may influence fatigue perception, neuromuscular control, and tissue repair capacity, all of which are recognized determinants of injury susceptibility in athletes. From a biomechanical perspective, injury risk is increasingly understood as the result of a dynamic imbalance between external training load and internal tissue capacity, where fatigue, impaired neuromuscular control, and reduced tissue resilience may act as key mediators [9,10].

Despite these biologically plausible mechanisms, the relationship between hypothyroidism and musculoskeletal injury risk in athletic populations remains insufficiently characterized. Most

available evidence is derived from experimental studies or non-athletic cohorts, while direct epidemiological data in athletes are scarce. Therefore, the aim of this narrative review is to evaluate current evidence regarding the impact of hypothyroidism on musculoskeletal function and injury susceptibility in athletes, with emphasis on underlying physiological mechanisms and clinical implications for sports participation and injury prevention [2,6].

2. Biological mechanisms linking thyroid hormones to musculoskeletal integrity

Thyroid hormones are essential regulators of skeletal muscle metabolism, connective tissue homeostasis, and cellular adaptation to mechanical loading. Through genomic and non-genomic mechanisms, triiodothyronine (T3), the biologically active thyroid hormone, modulates mitochondrial biogenesis, oxidative phosphorylation, protein turnover, and the expression of genes involved in muscle growth and repair. Consequently, adequate thyroid hormone signaling is required to maintain the structural and functional integrity of the musculoskeletal system, particularly in physically active individuals exposed to repetitive mechanical stress [3,12,13].

At the cellular level, thyroid hormone deficiency impairs mitochondrial oxidative metabolism and reduces ATP production, leading to diminished energetic efficiency within skeletal muscle. Experimental studies have demonstrated abnormalities in oxidative phosphorylation, reduced activity of mitochondrial enzymes, and altered substrate utilization in hypothyroid muscle, providing a physiological explanation for reduced fatigue resistance and impaired exercise tolerance observed in affected individuals [3]. In addition, thyroid hormones regulate muscle protein synthesis, fiber-type composition, and calcium handling, all of which contribute to normal muscle contractility and neuromuscular performance [12,13].

Beyond their metabolic actions, thyroid hormones play a critical role in skeletal muscle regeneration. Experimental models have shown that hypothyroidism disrupts both myogenic and non-myogenic repair pathways following acute muscle injury, leading to delayed satellite cell activation, impaired myofiber regeneration, and prolonged inflammatory responses [4,5,13]. These findings suggest that thyroid hormone deficiency may reduce the capacity of skeletal muscle to recover from exercise-induced microdamage, an essential component of physiological adaptation to athletic training.

Thyroid hormones also influence connective tissue biology through regulation of extracellular matrix (ECM) turnover and tenocyte activity. Thyroid hormone receptors have been identified in tendon tissue, supporting the concept that thyroid hormones directly regulate collagen synthesis, extracellular matrix remodeling, and tendon homeostasis [1]. Because tendon

adaptation depends on a dynamic balance between collagen degradation and synthesis in response to repetitive loading, endocrine disturbances may impair this adaptive process (8,10). Although direct evidence in athletes is lacking, thyroid dysfunction has been associated with degenerative tendon disorders, including calcific tendinopathy, suggesting that endocrine factors may contribute to impaired tendon resilience [1,7,11].

The interaction between skeletal muscle and connective tissue is particularly relevant in the context of sports injuries. According to the International Olympic Committee consensus on connective tissue and muscle injuries, successful adaptation to training depends on coordinated remodeling of muscle fibers and extracellular matrix in response to mechanical loading [9]. Since thyroid hormones regulate biological pathways involved in both muscle regeneration and extracellular matrix remodeling, hypothyroidism may theoretically reduce the ability of musculoskeletal tissues to adapt to repeated loading, thereby decreasing tissue capacity relative to training demands.

Collectively, current evidence indicates that thyroid hormones regulate multiple biological processes essential for musculoskeletal integrity, including mitochondrial energy production, muscle regeneration, extracellular matrix remodeling, and tendon adaptation. Although these mechanisms provide a strong biological rationale for increased musculoskeletal vulnerability in hypothyroidism, they do not by themselves demonstrate an increased incidence of sports injuries. Instead, they establish the physiological basis for the hypothesis explored in subsequent sections of this review.

3. Functional consequences of hypothyroidism in athletes

3.1 Impaired muscle performance and strength capacity

Evidence from experimental and clinical studies indicates that hypothyroidism is associated with reduced skeletal muscle performance, affecting both strength and power output. Thyroid hormones regulate muscle protein turnover, calcium handling, and contractile protein expression, which are essential for efficient force generation. In hypothyroid states, reduced triiodothyronine (T3) availability leads to decreased contractile efficiency and impaired neuromuscular transmission, contributing to reductions in maximal voluntary strength and muscular endurance [12,13].

Experimental studies on hypothyroid muscle demonstrate altered cellular energetics, including reduced mitochondrial oxidative capacity and impaired ATP production, which limit the ability to sustain high-intensity muscle contractions [3]. These changes may manifest clinically as

reduced strength, early fatigue, and decreased tolerance to resistance-based exercise. Such impairments are particularly relevant in athletic populations, where repeated high-force contractions are required for both performance and injury prevention through neuromuscular control.

3.2 Reduced exercise tolerance and aerobic capacity

Hypothyroidism has been consistently associated with reduced exercise tolerance, primarily due to impaired mitochondrial function and decreased oxidative metabolism. Reduced ATP production and altered substrate utilization lead to earlier onset of fatigue during aerobic activity and diminished endurance performance [3,12].

Clinical observations in individuals with thyroid dysfunction, including those receiving replacement therapy, suggest that exercise capacity may remain suboptimal despite biochemical normalization of thyroid hormone levels [2,6]. This may be reflected in reduced VO_2 tolerance, increased perceived exertion, and impaired recovery kinetics following prolonged exercise.

From a sports performance perspective, these limitations are particularly relevant in endurance-based disciplines such as running, cycling, or rowing, where sustained aerobic metabolism is critical for performance maintenance.

3.3 Impaired recovery and muscle regeneration

A growing body of evidence indicates that thyroid hormones play a key role in skeletal muscle regeneration following injury or intense exercise. Experimental models have shown that hypothyroidism impairs muscle repair by disrupting both myogenic and non-myogenic pathways, including satellite cell activation, inflammatory regulation, and myofiber remodeling [4,5,13].

In hypothyroid conditions, delayed regeneration is associated with prolonged structural and functional recovery of skeletal muscle following injury. This may result in increased susceptibility to cumulative microtrauma during repeated training sessions, as incomplete recovery can reduce tissue readiness for subsequent mechanical loading.

These findings are particularly relevant in sports characterized by high training frequency and repetitive eccentric loading, where efficient muscle repair is essential for maintaining performance and reducing injury risk.

3.4 Functional limitations in athletic populations

In physically active individuals, hypothyroidism is associated not only with reduced performance capacity but also with broader functional limitations affecting training adaptation. Studies in athletic and recreational populations indicate that individuals with primary hypothyroidism report reduced exercise participation, increased fatigue, and perceived limitations in training intensity, even during hormone replacement therapy [2,6].

These functional impairments may influence the athlete's ability to tolerate progressive training loads and maintain consistent training stimuli over time. Inadequate recovery and reduced neuromuscular efficiency may also alter movement quality during fatigue, potentially increasing biomechanical stress on musculoskeletal structures.

From a sport-specific perspective, the functional impact of hypothyroidism may vary depending on the dominant physiological demands of the discipline. Endurance athletes may experience limitations in aerobic capacity, while strength and power athletes may be more affected by reduced force production and neuromuscular efficiency.

These functional impairments may influence the ability to tolerate progressive training loads and maintain optimal movement control under fatigue.

4. Mechanistic links between hypothyroidism and musculoskeletal injury risk in athletes

The relationship between hypothyroidism and musculoskeletal injury susceptibility in athletes can be conceptualized within an integrated load–capacity framework. In this model, injury risk is determined by the interaction between external training load and the internal capacity of musculoskeletal tissues to tolerate and recover from mechanical stress. Thyroid hormones represent a key systemic regulator of this internal capacity, influencing mitochondrial energy production, neuromuscular control, and extracellular matrix remodeling. Consequently, hypothyroidism may reduce tissue tolerance to repetitive loading by simultaneously affecting metabolic, neuromuscular, and connective tissue systems.

From a physiological perspective, thyroid hormone deficiency leads to impaired oxidative metabolism and reduced ATP availability, which may decrease the ability of skeletal muscle to sustain repeated mechanical contractions during training and competition [3,12]. This energy deficit can promote earlier onset of fatigue, which is a well-recognized contributor to altered movement mechanics and reduced neuromuscular control during sport-specific tasks. Fatigue-related deterioration in motor coordination has been identified as a key intrinsic risk factor for both acute and overuse musculoskeletal injuries.

At the neuromuscular level, hypothyroidism is associated with reduced muscle strength, slowed contraction and relaxation kinetics, and impaired neuromuscular efficiency [3,13]. These alterations may negatively affect joint stabilization and movement precision, particularly under conditions of high training load or repetitive high-intensity effort. In dynamic sporting environments, such deficits may increase reliance on compensatory movement strategies, which can elevate localized tissue stress and contribute to injury development.

In parallel, thyroid hormones play a central role in connective tissue adaptation through regulation of collagen synthesis and extracellular matrix turnover within tendons [1,7,8]. Tendon health depends on a dynamic balance between collagen degradation and synthesis in response to mechanical loading. Disruption of thyroid hormone signaling may impair this adaptive response, potentially reducing tendon stiffness regulation and mechanical resilience. This is supported by experimental and clinical evidence linking thyroid dysfunction with degenerative tendon conditions, including calcific tendinopathy [11], although direct causal relationships in athletic populations remain unclear.

Furthermore, thyroid hormone deficiency may impair skeletal muscle regeneration following injury by disrupting both myogenic and non-myogenic repair pathways [4,5,13]. Delayed or incomplete muscle recovery may increase vulnerability to reinjury, particularly in sports characterized by short recovery periods between training sessions or competitions. Inadequate tissue regeneration may also contribute to cumulative microdamage, which is a recognized mechanism in the development of overuse injuries.

From a sports injury epidemiology perspective, the International Olympic Committee consensus on muscle and tendon injuries emphasizes that tissue failure occurs when applied mechanical load exceeds the capacity of muscle–tendon units to adapt and recover [9]. Within this framework, hypothyroidism may be interpreted as a systemic factor that shifts this balance toward reduced tissue capacity rather than acting as a direct injury trigger. This distinction is important, as it suggests that thyroid dysfunction is unlikely to function as an independent causal risk factor, but may instead act as a biological modifier influencing injury susceptibility under conditions of high training load.

Despite these mechanistic considerations, direct clinical evidence linking hypothyroidism with increased injury incidence in athletes remains limited. Existing studies are predominantly derived from non-athletic populations or focus on isolated physiological outcomes rather than sports-related injury epidemiology [6]. As a result, the hypothesized relationship remains biologically plausible but not empirically confirmed in competitive athletic cohorts.

Collectively, current evidence suggests that hypothyroidism may contribute to a reduction in musculoskeletal load tolerance through combined effects on energy metabolism, neuromuscular control, connective tissue adaptation, and recovery processes. In the context of high-performance sport, these alterations may increase susceptibility to both acute and overuse injuries by reducing the functional reserve of musculoskeletal tissues under repetitive mechanical stress.

5. Clinical implications for sports medicine and athlete management

The potential impact of hypothyroidism on musculoskeletal function and injury susceptibility has relevant implications for sports medicine practice, particularly in athlete monitoring, diagnostic evaluation, and training management. Although overt hypothyroidism is relatively uncommon in athletic populations, subclinical thyroid dysfunction or inadequately controlled disease may contribute to persistent, non-specific symptoms such as fatigue, reduced exercise tolerance, delayed recovery, and unexplained reductions in performance capacity. These manifestations frequently overlap with functional overreaching or overtraining syndrome, making clinical differentiation challenging without targeted endocrine assessment.

From a diagnostic perspective, thyroid function testing, including serum thyroid-stimulating hormone (TSH) and free thyroxine (fT4), should be considered in athletes presenting with disproportionate fatigue, recurrent soft tissue injuries, impaired recovery, or sustained decline in performance despite appropriate training modifications. Evidence from sports medicine literature indicates that thyroid disorders, although not highly prevalent, are clinically relevant in athletic populations and may be underdiagnosed in the context of performance-related complaints .

In terms of training management, athletes with hypothyroidism may require individualized adjustments in load prescription, recovery intervals, and periodization strategies. This is particularly relevant given the documented impairments in aerobic capacity, neuromuscular efficiency, and tissue repair processes associated with thyroid hormone deficiency [3, 12, 13]. Depending on the dominant physiological demands of a given sport, the functional consequences may vary. In endurance disciplines such as long-distance running and cycling, reduced mitochondrial oxidative capacity may limit aerobic adaptation and accelerate fatigue onset. In strength and power sports, impaired muscle contractility and force production may reduce performance output. In team sports, where repeated high-intensity efforts and rapid directional changes are required, fatigue-related deterioration in neuromuscular control may increase injury susceptibility. In aesthetic and weight-category sports, even mild hormonal

imbalance may negatively influence recovery dynamics and energy availability, with downstream effects on training consistency.

Thyroid hormone replacement therapy, typically levothyroxine, remains the standard treatment for hypothyroidism and is effective in restoring systemic hormonal balance in most patients. However, evidence suggests that normalization of biochemical parameters does not always fully translate into immediate restoration of exercise performance or neuromuscular function, particularly in physically active individuals [2]. Therefore, return-to-sport decisions should not rely solely on laboratory normalization but should incorporate functional assessment, symptom resolution, and sport-specific performance evaluation.

Close monitoring of subjective fatigue, perceived exertion, training response, and recovery kinetics may provide additional clinically relevant information for optimizing athlete management. This is particularly important during rehabilitation phases, where excessive training progression may increase the risk of reinjury in the context of unresolved metabolic or neuromuscular limitations.

Despite these considerations, sport-specific guidelines addressing the management of athletes with thyroid dysfunction remain limited. Current recommendations are largely extrapolated from general endocrinology practice rather than based on high-quality athlete-centered studies. As a result, interdisciplinary collaboration between endocrinologists, sports physicians, physiotherapists, and coaching staff is essential to ensure safe and effective return-to-performance strategies.

6. Limitations and gaps in the current literature

Despite increasing interest in the role of thyroid hormones in skeletal muscle physiology, tendon biology, and exercise performance, the current body of evidence linking hypothyroidism to musculoskeletal injury susceptibility in athletes remains limited. A major limitation is the scarcity of well-designed prospective epidemiological studies specifically investigating injury incidence in athletic populations with diagnosed thyroid dysfunction. Most available data are derived from general or non-athletic populations, which limits direct applicability to high-performance sport settings.

Another important limitation is the predominance of mechanistic and experimental studies focusing on cellular and molecular pathways, including mitochondrial function, muscle energetics, and connective tissue remodeling [3,8,12,13]. While these studies provide strong biological plausibility for impaired musculoskeletal function, they do not directly assess clinically relevant outcomes such as injury incidence, severity, or time-loss from sport.

In addition, heterogeneity in study populations represents a significant challenge. Differences in the severity of thyroid dysfunction (overt versus subclinical hypothyroidism), treatment status, and adequacy of hormone replacement therapy introduce variability that complicates interpretation of findings. Athletes receiving optimized levothyroxine therapy may demonstrate near-normal physiological function, potentially masking associations between thyroid status and injury risk [2,6].

Sports injuries themselves are multifactorial in nature, resulting from complex interactions between intrinsic and extrinsic factors, including training load, biomechanics, neuromuscular control, previous injury history, and recovery strategies. Within this multifactorial framework, isolating the independent contribution of hypothyroidism remains methodologically challenging.

Finally, there is a lack of longitudinal and sport-specific cohort studies evaluating whether thyroid dysfunction represents an independent risk factor for injury or acts primarily as a modifying physiological condition under increased training stress. This represents a critical gap in the literature and highlights the need for future prospective research in athletic populations.

7. Conclusion

Current evidence indicates that thyroid hormones play a fundamental role in the regulation of skeletal muscle function, energy metabolism, and connective tissue homeostasis, all of which are critical determinants of exercise performance and musculoskeletal integrity. Hypothyroidism, through systemic reductions in thyroid hormone availability, may therefore contribute to impaired mitochondrial function, reduced neuromuscular efficiency, altered muscle contractility, and disrupted extracellular matrix remodeling.

These physiological alterations provide a biologically plausible framework through which hypothyroidism may influence musculoskeletal vulnerability in athletes. In particular, reduced metabolic capacity, impaired motor control, and delayed tissue repair may collectively contribute to an increased susceptibility to both acute and overuse injuries under conditions of repeated mechanical loading.

However, despite strong mechanistic evidence, direct clinical data linking hypothyroidism to injury incidence in athletic populations remain limited. Existing studies are predominantly indirect, derived from experimental models or non-athletic cohorts, and do not allow for definitive causal inference. As a result, hypothyroidism should currently be considered a

potential modifying factor rather than an established independent risk factor for sports-related injuries.

From a sports medicine perspective, recognition of thyroid dysfunction may be relevant in athletes presenting with unexplained performance decline, persistent fatigue, recurrent soft tissue injuries, or delayed recovery. Nevertheless, clinical management should be individualized and based on comprehensive assessment, integrating biochemical, functional, and sport-specific parameters.

Future research should prioritize prospective, sport-specific cohort studies to clarify the relationship between thyroid dysfunction and injury risk, as well as to determine the impact of thyroid hormone replacement therapy on musculoskeletal outcomes in athletic populations.

8. AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

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