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Overtraining Syndrome: Pathophysiology, Diagnostic Challenges and Prevention Strategies – A Narrative Review

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Abstract

Background. Overtraining Syndrome (OTS) represents a complex, maladaptative state characterized by a persistent decline in athletic performance and systemic physiological dysfunction. Despite its prevalence in elite and amateur sports, it remains one of the most challenging conditions to diagnose and treat.

Aim. The study aims to provide a comprehensive review of the current physiological frameworks, diagnostic challenges, and modern management strategies for OTS, while integrating classical theories with emerging research on the gut-brain axis and metabolomics.

Material and methods. A narrative review of academic literature from 1980 to 2024 was conducted using databases such as PubMed and Google Scholar. The selection focused on joint consensus statements, clinical trials, and systematic reviews. Analysis was assisted by AI tools for structural organization and linguistic refinement, ensuring adherence to modern scientific writing standards.

Results. The findings indicate that the pathophysiology of OTS is multifaceted, involving systemic inflammation (the cytokine hypothesis), metabolic depletion (the glycogen hypothesis), and structural changes in intestinal permeability (the gut-brain axis). The review distinguishes between sympathetic and parasympathetic OTS phenotypes, each requiring different monitoring approaches. Modern research highlights a significant clinical overlap between OTS and Relative Energy Deficiency in Sport (RED-S), suggesting that low energy availability is a primary driver of the syndrome. Diagnostic methodologies continue to rely on the exclusion of organic diseases, supplemented by longitudinal monitoring of heart rate variability (HRV) and psychological profiling (POMS).

Conclusions. OTS is a systemic failure of homeostatic regulation rather than a localized muscular issue. Prevention through individualized periodization, optimized energy availability, and psychological monitoring is more effective than current treatment protocols, which remain limited to prolonged rest. Future diagnostic models are expected to leverage metabolomic profiling and AI-driven predictive analysis for early intervention.

Key words: overtraining syndrome, sports medicine, heart rate variability, cytokine hypothesis, RED-S, athlete monitoring.

1. Introduction

Overtraining syndrome (OTS) is defined as a condition marked by a prolonged decline in athletic performance, resulting from a sustained imbalance between training and non-training stressors and inadequate recovery. In the absence of a definitive gold-standard diagnostic test, OTS continues to be classified as a diagnosis of exclusion. A range of endocrine, neurochemical, and metabolic markers, together with psychological, electrocardiographic, electroencephalographic, and immunological alterations, have been proposed as potential diagnostic indicators of overtraining syndrome. These findings underscore the multisystemic and complex nature of the condition [1]. Current research has not consistently demonstrated a clear shift in performance or mood from a healthy state to a sustained overtrained condition, nor has it reliably documented prolonged suppression of athletic performance. These limitations are largely attributable to inconsistent terminology, difficulties in conducting long-term monitoring of physiological and psychological variables, and the absence of a standardized and feasible diagnostic testing protocol, which together contribute to an insufficient evidence base for a comprehensive understanding of overtraining syndrome in athletes [2]. This paper examines the current understanding of overtraining syndrome, with particular emphasis on its pathophysiology, clinical manifestations, diagnostic challenges, and evidence-based strategies for prevention and management.

2. Methods

This manuscript was conducted as a narrative review of the current literature on overtraining syndrome (OTS) in athletes. A comprehensive search of the scientific literature was performed using the PubMed and Google Scholar databases to identify relevant studies published in English.

The search strategy included combinations of the following keywords and Medical Subject Headings (MeSH) terms: “overtraining syndrome,” “athletes,” “non-functional overreaching,” “fatigue,” “training load,” “biomarkers,” “diagnosis,” and “recovery.” Additional articles were identified through manual screening of reference lists from selected publications and relevant review papers to ensure completeness of the literature coverage.

The inclusion criteria comprised original research articles, systematic reviews, narrative reviews, and consensus statements addressing the pathophysiology, clinical presentation, diagnostic approaches, or prevention and management of OTS in human subjects. Studies focusing exclusively on animal models, non-sport-related fatigue conditions, or unrelated medical disorders were excluded.

Priority was given to publications from the last 10-15 years to ensure incorporation of the most up-to-date evidence; however, seminal and highly cited older studies were also included where relevant for conceptual and historical context.

Due to the heterogeneity of study designs, populations, and outcome measures, a qualitative synthesis approach was applied. The findings were organized thematically into key domains, including definitions and classification, pathophysiology, clinical manifestations, diagnostic approaches, differential diagnosis, and prevention strategies.

No ethical approval was required for this study as it is based solely on previously published literature and does not involve human participants or new experimental data.

3.1 Pathophysiology

The pathophysiology of overtraining syndrome (OTS) is a multifaceted process resulting from a cascade of dysfunctions within the neuroendocrine axis, the immune system, and cellular metabolism, which collectively lead to a profound and persistent loss of the body's adaptive capacity to training stress. There are two main etiologic theories: the cytokine hypothesis and the glycogen hypothesis.

According to the cytokine hypothesis, repetitive microtrauma to the musculoskeletal system, coupled with inadequate recovery, triggers a state of chronic, low-grade systemic inflammation [3]. Pro-inflammatory cytokines, specifically interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), act as signaling molecules that transition from local muscle repair to a systemic response. These cytokines interact with the central nervous system, inducing "sickness behavior," which manifests as the hallmark symptoms of OTS: mood disturbances, sleep fragmentation, and anorexia [4].

In contrast, the glycogen hypothesis focuses on the metabolic demands of prolonged, high-intensity training. It posits that the persistent depletion of muscle and liver glycogen stores, often exacerbated by insufficient dietary carbohydrate intake, acts as a primary physiological stressor [5]. This metabolic deficit leads to an increased reliance on oxidation of branched-chain amino acids (BCAAs) for energy, which subsequently raises the plasma concentration of free tryptophan. The resulting shift in the tryptophan-to-BCAA ratio facilitates the transport of tryptophan across the blood-brain barrier, increasing cerebral serotonin synthesis. This neurochemical shift is a key driver of the "central fatigue" and lethargy frequently reported by athletes suffering from OTS [6].

3.2 Clinical Presentation

The clinical manifestation of Overtraining Syndrome (OTS) is characterized by a heterogenous cluster of symptoms that extend far beyond localized muscular fatigue. The cardinal symptom of OTS is a persistent, unexplained decrement in sport-specific performance, which remains unresolved despite a period of regenerative rest [4]. This performance collapse is often accompanied by an increased perceived effort during submaximal training loads and a prolonged recovery time between sessions.

Physiological symptoms of OTS are frequently categorized into autonomic and immunological categories. Athletes often report sleep disturbances, including insomnia and fragmented sleep, which further impair the regenerative process [7]. From an immunological standpoint, OTS is strongly associated with an increased susceptibility to Upper Respiratory Tract Infections (URTI). This "open window" effect is a result of chronic immunosuppression, characterized by reduced salivary immunoglobulin A (sIgA) levels and impaired neutrophil function [8] Additionally, athletes may experience systemic symptoms such as loss of appetite (anorexia), unexplained weight loss, and persistent heavy-headedness.

The psychological and behavioral domain is equally critical for identifying OTS. The term "staleness" is frequently used in the literature to describe the mental exhaustion and lack of motivation (loss of "drive") observed in affected individuals. Objective psychological profiling often reveals a reversal of the "iceberg profile" (a term used to describe a healthy athlete's psychological state), where levels of vigor drop significantly, while scores for tension, depression, anger, and fatigue rise sharply [9]. These neuropsychiatric alterations are thought to be linked to changes in central neurotransmitter levels, particularly serotonin and dopamine, highlighting the systemic nature of the syndrome [10].

3.3 Diagnosis

The diagnosis of Overtraining Syndrome (OTS) remains one of the most significant challenges in sports medicine due to the absence of a single, definitive "gold standard" biomarker. Current clinical practice dictates that OTS must be treated as a diagnosis of exclusion. This requires the systematic elimination of confounding factors such as undiagnosed organic diseases (e.g., thyroid dysfunction, anemia, or chronic infections), caloric insufficiency (Relative Energy Deficiency in Sport - RED-S), and psychological disorders [4]. The primary clinical criterion remains a profound and persistent decrease in sport-specific performance that continues despite a period of rest exceeding two weeks.

From a physiological perspective, monitoring the Autonomic Nervous System (ANS) has emerged as a non-invasive and sensitive diagnostic approach. Heart Rate Variability (HRV) is frequently utilized to assess the balance between sympathetic and parasympathetic activity. A chronic reduction in parasympathetic indices (e.g., RMSSD - Root Mean Square of Successive Differences) or an abnormally elevated resting heart rate often precedes the clinical manifestation of OTS, reflecting an inability of the ANS to maintain homeostasis [11].

Biochemical and hormonal markers provide additional insight into the athlete's state of strain. Historically, the free testosterone-to-cortisol (T:C) ratio was considered a primary indicator of the anabolic-catabolic balance. However, contemporary research suggests that a decrease in the T:C ratio of more than 30% is a marker of intense physiological strain rather than a definitive diagnostic for OTS itself [12]. Furthermore, psychological assessment tools, such as the Profile of Mood States (POMS) and the Recovery-Stress Questionnaire for Athletes (REST-Q-Sport), are increasingly recognized for their predictive value. Clinical data indicate that changes in mood states—specifically increased fatigue, depression, and irritability—often occur before significant physiological or performance decrements become apparent [9].

3.4 Prevention and Management Strategies

The prevention of Overtraining Syndrome (OTS) is a multifaceted process that must be integrated into the athlete’s long-term developmental plan. The cornerstone of prevention is the implementation of a well-structured, individualized periodization program. This approach must ensure a dynamic balance between training stress and recovery, incorporating "unloading" phases where volume and intensity are significantly reduced to allow for physiological supercompensation [4]. Furthermore, the use of longitudinal monitoring—combining objective data like Heart Rate Variability (HRV) with subjective wellness scales—is imperative. Evidence suggests that self-reported measures, such as perceived stress, sleep quality, and muscle soreness, often detect the early onset of non-functional overreaching (NFOR) more reliably than physiological markers alone [13].

Nutritional intervention and sleep hygiene are non-negotiable pillars of prevention. Maintaining high carbohydrate availability is essential to prevent the chronic glycogen depletion that characterizes the metabolic pathway of OTS [5]. Moreover, addressing the risk of Relative Energy Deficiency in Sport (RED-S) by ensuring adequate caloric intake is crucial to maintaining the health of the neuroendocrine axis [14]. Sleep, often described as the most potent recovery tool, must be optimized; chronic sleep deprivation (<7 hours per night) is a significant predictor of both injury and the systemic maladaptation seen in OTS [15].

When OTS is clinically confirmed, the primary and only universally recognized treatment is total rest or a dramatic reduction in training load. The recovery period is highly variable, often requiring several weeks to several months depending on the severity of the neuroendocrine dysfunction [7]. During the initial phase of treatment, the athlete should be removed from the competitive environment to eliminate psychological stressors. The return-to-play protocol must be gradual and strictly monitored, starting with low-intensity active recovery and slowly reintroducing volume only when performance and psychological markers return to baseline levels. Psychological support, including cognitive-behavioral therapy (CBT), may be necessary to address the "loss of identity" and depression that frequently accompany a forced hiatus from sport [10,16].

4. Discussion

The clinical understanding of Overtraining Syndrome (OTS) has undergone a significant paradigm shift in the last five years. Recent evidence suggests that OTS should no longer be viewed as a purely exercise-induced condition, but rather as a systemic maladaptation to a mismatch between total cumulative stress and recovery. A major theme in contemporary discussion is the striking phenotypical and physiological overlap between OTS and Relative Energy Deficiency in Sport (RED-S). Recent studies [17] argue that many cases traditionally diagnosed as OTS are, in fact, driven by Low Energy Availability (LEA). This necessitates a more rigorous nutritional screening during the diagnostic process, as the hormonal suppression (specifically of the hypothalamic-pituitary-gonadal axis) observed in both conditions is nearly indistinguishable [18].

Furthermore, the search for a definitive biomarker has transitioned into the realm of metabolomics and transcriptomics. While traditional markers like creatine kinase or urea have proven unreliable, recent "omics" profiling has identified unique metabolic signatures—specifically alterations in amino acid metabolism and lipid signaling—that differentiate OTS patients from healthy, highly trained athletes [19]. This supports the view that OTS is a state of "metabolic inflexibility," where the body loses its ability to efficiently switch between fuel sources under stress.

Another critical development in the 2022–2024 literature is the recognition of cognitive and psychological load as a primary driver of physical overtraining. Modern athletes face unprecedented levels of "mental fatigue" due to digital connectivity and cognitive demands, which share neurobiological pathways with physical fatigue [20]. This integration of mental and physical stressors has led to the proposal of a "complex systems approach" to monitoring. Instead of looking for a single threshold, researchers now advocate for the use of Artificial Intelligence (AI) and Machine Learning to analyze multivariate data from wearables (e.g., sleep architecture, HRV, and power output) to predict the transition from functional overreaching to OTS before performance collapse occurs [21].

Finally, the discussion must address the limitations of current recovery protocols. While rest remains the primary treatment, recent clinical trials have begun investigating the role of anti-inflammatory nutrition and gut microbiota modulation. Given the "leaky gut" associated with the cytokine hypothesis of OTS, targeting the gut-brain axis represents a novel and promising frontier for both prevention and accelerated recovery in elite populations [22].

3.5 Autonomic Phenotypes: Sympathetic and Parasympathetic OTS

Clinical observations indicate that OTS does not manifest uniformly across different athletic populations; rather, it presents through two distinct autonomic phenotypes depending on the nature of the training stimulus. The sympathetic form (also known as the "Basedowoid" type) is more prevalent in power, sprint, and explosive sports. It is characterized by a state of prolonged sympathetic dominance, manifesting as an elevated resting heart rate, increased blood pressure, insomnia, irritability, and a delayed return to baseline heart rate after exercise [16].

In contrast, endurance athletes typically develop the parasympathetic form (the "Addisonoid" type). This phenotype is more insidious and harder to diagnose, as it manifests through excessive parasympathetic activity. Symptoms include a paradoxically low resting heart rate (bradycardia), low blood glucose levels during exercise, and a blunted catecholamine response to stress [4]. Athletes in this state often appear phlegmatic or depressed. Distinguishing between these two forms is crucial for clinical practitioners, as the sympathetic type often requires a shorter recovery period than the more profound neuroendocrine exhaustion seen in the parasympathetic type.

3.6 The Emerging role of the Gut-Brain Axis

Recent research has highlighted the gut-brain axis as a critical pathway in the development of OTS. Intense physical exertion causes a redistribution of blood flow away from the splanchnic area to the working muscles and skin, leading to intestinal ischemia and increased gut permeability, commonly referred to as "leaky gut" [22]. This structural compromise allows for the translocation of lipopolysaccharides (LPS) and other endotoxins into the systemic circulation, which further exacerbates the systemic inflammatory response described in the cytokine hypothesis [3].

4. Conclusions

Based on the comprehensive review of current literature and recent clinical findings, the following conclusions can be drawn:

Systemic Nature of OTS: Overtraining Syndrome is a complex, multi-systemic disorder that extends beyond localized muscle fatigue. Its pathophysiology is rooted in a cascade of neuroendocrine dysfunctions, chronic systemic inflammation (the cytokine hypothesis), and metabolic maladaptation, necessitating a holistic approach to the athlete's health.

Diagnostic Paradox: Despite decades of research, OTS remains a diagnosis of exclusion. Clinical performance remains the only reliable "gold standard" for diagnosis, although the integration of Heart Rate Variability (HRV) and psychological screening (e.g., POMS) provides significant predictive value for early detection.

The RED-S Overlap: Modern clinical perspectives highlight a critical overlap between OTS and Relative Energy Deficiency in Sport (RED-S). Ensuring adequate energy availability is a primary preventative measure, as many physiological symptoms of overtraining are indistinguishable from those caused by chronic caloric insufficiency.

Priority of Prevention: Given that the recovery from OTS can take months or even years, prevention must be the priority. This is best achieved through individualized periodization, mandatory unloading phases, and the optimization of non-training factors, particularly sleep hygiene and nutritional support.

Future Perspectives: The transition from traditional biochemical markers to advanced "omics" technologies (metabolomics and transcriptomics) offers a promising frontier for identifying definitive biological signatures of OTS. Future monitoring systems should leverage Artificial Intelligence to analyze multivariate data streams, allowing for real-time risk stratification in elite athletes.

Disclosure

The authors declare no conflicts of interest regarding the publication of this paper. Author's contribution:

Conceptualization, B. Głowacka, H. Mruzek; methodology, A. Wesołowska, O. Kałwak; software, Z. Kania-Bonicka, M. Żółtowska; validation, P. Jasak, S. Owczarek and A. Sobótka; formal analysis, O. Dziechciarz, D. Zgodzińska and B. Głowacka; investigation, Barbara Głowacka, O. Dziechciarz, H. Mruzek, A. Wesołowska, Z. Kania-Bonicka, O. Kałwak, M. Żółtowska, P. Jasak, S. Owczarek, A. Sobótka, D. Zgodzińska; resources, H. Mruzek, D. Zgodzińska; data curation, Z. Kania-Bonicka, M. Żółtowska and O. Dziechciarz; writing -original draft preparation, B. Głowacka, A. Wesołowska, O. Kałwak and P. Jasak; writing -review and editing, S. Owczarek, A. Sobótka; visualization, D. Zgodzińska; supervision, B. Głowacka and H. Mruzek; project administration, A. Wesołowska, O. Kałwak; funding acquisition, Not applicable.

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