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OVERTRAINING SYNDROME AND MENTAL HEALTH IN ATHLETES - A LITERATURE REVIEW

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ABSTRACT

Overtraining Syndrome (OTS) is a complex biopsychosocial condition arising from a chronic imbalance between training load and recovery. OTS produces numerous psychiatric symptoms that remain systematically underrecognized in clinical practice. This review examines the neurobiological mechanisms linking OTS to mental health disorders, characterizes its psychiatric symptomatology, clarifies differential diagnosis with primary psychiatric conditions, and synthesizes current evidence on monitoring, prevention, and management. OTS affects numerous elite athletes during their careers, with a relapse rate of 80–90% within three years of recovery. Its neurobiological mechanisms — HPA axis exhaustion, serotonergic receptor dysregulation, neuroinflammatory activation, and autonomic nervous system dysfunction — overlap substantially with those of major depressive disorder, providing a mechanistic basis for the extensive psychiatric comorbidity observed clinically. OTS presents across three dimensions: affective and anxiety symptoms, neurocognitive and somatic impairment, and burnout. Differential diagnosis requires systematic exclusion of primary psychiatric disorders, Relative Energy Deficiency in Sport (RED-S), and organic pathology. Prevention and management form a continuum encompassing psychological monitoring, primary prevention strategies, and — in established OTS — multidisciplinary treatment including psychiatric and psychological interventions. OTS is a biopsychosocial condition whose psychiatric dimension is clinically substantial yet underdiagnosed. Its neurobiological overlap with major depressive disorder warrants integration of psychiatric expertise into the routine evaluation and management of overtrained athletes. The field remains constrained by the absence of validated diagnostic criteria and evidence-based psychological interventions — gaps that sports psychiatry is well-positioned to address.

KEYWORDS

Overtraining Syndrome, Mental Health, Athletes Burnout, Sports Psychiatry, Recovery

CITATION

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1. Introduction

Elite athletic performance demands sustained, high-intensity training regimens that systematically push the boundaries of human physiology. When combined with adequate periods of rest and recovery, such training produces meaningful physiological adaptations and measurable performance gains. However, when the balance between training stimulus and recovery is chronically disrupted, the athlete enters a pathological state known as Overtraining Syndrome (OTS) [1].

OTS is estimated to affect between 20% and 60% of elite athletes at some point during their careers [2]. Elite athletes were long regarded as resilient to mental health disorders by virtue of their physical fitness; however, the evidence now clearly demonstrates that excessive training at the elite level can compromise psychological well-being through overtraining, injury, and burnout [3, 4]. Once regarded as a purely physiological phenomenon, OTS is now recognized as a complex biopsychosocial condition whose psychological manifestations — closely mirroring those of major depressive disorder (MDD), anxiety disorders, and burnout syndrome — create significant diagnostic challenges at the interface of sports medicine and clinical psychiatry [5].

The growing field of sports psychiatry has begun to address these intersections systematically. The first sports psychiatry research concerning OTS appeared in the early 2000s. Armstrong and VanHeest [6] proposed that OTS shares neurobiological mechanisms with depression and psychoneuroimmunological dysregulation. In 2013, the joint consensus statement of the European College of Sport Science (ECSS) and the American College of Sports Medicine (ACSM), authored by Meeusen et al. [1], established the current gold standard for the definition and staging of OTS. The same year, Bär and Markser (2013) argued that sport-specific mental disorders — including OTS-related psychopathology — demand a distinct subspecialty within psychiatry. More recently, international bodies including the International Olympic Committee have formalized consensus

statements on mental health in elite athletes, acknowledging overtraining and burnout as key risk factors for psychological disorders in sport [8]. Despite these advances, OTS remains underdiagnosed and undertreated from a mental health standpoint [9].

From a psychiatric perspective, OTS presents a compelling model for studying the interaction between extreme physical stress, neurobiological dysregulation, and mental health deterioration. Historically, the terminology surrounding OTS has been inconsistent — including terms such as "staleness," "burnout," "unexplained underperformance syndrome (UUPS)," and "paradoxical deconditioning syndrome (PDS)" — which has significantly hampered both research quality and clinical recognition [10]. A 2022 systematic review by Grandou et al. [2] revealed that no published study provided objective evidence simultaneously demonstrating prolonged performance decrements and documented psychological symptoms meeting rigorous research criteria — highlighting a critical evidence gap in the field.

This review therefore aims to map the current state of knowledge on OTS in athletes through the lens of mental health — examining the neurobiological mechanisms that underlie its psychological consequences, characterizing its clinical presentation, clarifying its boundaries with primary psychiatric disorders, and synthesizing the available evidence on screening, prevention, and treatment. Understanding these dimensions is essential not only for sports medicine specialists, but for all clinicians who may encounter competitive athletes in their practice.

2. Methodology

This study is a narrative literature review conducted according to the principles of evidence-based medicine. A structured search was performed between January and April 2025 across four major scientific databases: PubMed/MEDLINE, PsycINFO, Web of Science, and Scopus. The primary search terms used were: "overtraining syndrome," "mental health," "athletes," "burnout," "non-functional overreaching," "depression," "anxiety," "sports psychiatry," "recovery," and "HPA axis." Terms were combined using Boolean operators (AND, OR).

Inclusion criteria were: original research articles, systematic reviews, meta-analyses, and expert consensus statements; publications in English; studies involving adult athletes (age ≥18 years); publications addressing psychological, psychiatric, or neurobiological aspects of OTS or overreaching; publications from 2000 to 2026.

Exclusion criteria were: publications not directly addressing OTS or mental health in athletes; studies limited to purely physiological parameters without mental health outcomes; publications in languages other than English. Following database search, titles and abstracts were screened for relevance, followed by full-text review of eligible articles. Reference lists of included papers were also manually checked for additional relevant sources.

The final review included 31 publications: 5 systematic reviews and meta-analyses, 17 narrative reviews, scoping reviews, and expert opinion articles, 3 original research studies, 2 consensus or position statements, 1 case series, and 1 editorial. One publication predates the 2000 threshold but was retained for its foundational significance as the primary source establishing the link between OTS and depression. Publications were organized thematically and synthesized narratively. Given the exploratory and heterogeneous nature of the available literature, a formal meta-analysis was not performed. Quality assessment of individual studies was conducted informally, with attention to sample size, methodological rigor, and consistency of findings across sources.

3. Results

3.1. Definition, Classification, and Epidemiology of OTS

The ECSS/ACSM joint consensus statement [1] — widely regarded as the gold standard in the field — defines overtraining as a spectrum progressing through three stages. Functional Overreaching (FOR) represents a short-term performance decrement resolving within days to weeks, without severe psychological symptoms; it may ultimately enhance performance after recovery. Non-Functional Overreaching (NFOR) involves performance decrements persisting for weeks to months, accompanied by psychological and hormonal disturbances requiring prolonged recovery. Overtraining Syndrome (OTS) represents the most severe form: a chronic, maladaptive state affecting neurological, endocrinological, immunological, and psychological systems, with recovery potentially requiring months to years and meeting the threshold for clinical intervention [1]. The overlap in clinical, hormonal, and psychological presentation between NFOR and OTS makes differential staging challenging in practice, and the persisting terminological ambiguity across the literature has historically hampered both research quality and clinical recognition of the condition [10, 2].

Epidemiological data indicate that OTS affects between 20% and 60% of elite athletes at some point during their careers, with the highest prevalence reported in endurance sports requiring high training volumes — particularly swimming, triathlon, road cycling, and rowing [2]. These figures may, however, underestimate the true burden, given that many athletes with OTS never seek medical attention and the syndrome was often historically subsumed under the broader term 'unexplained underperformance,' first systematically described by Budgett [19] as a condition of fatigue and declining performance in the absence of organic disease. An estimated 80–90% of athletes who experience OTS relapse within three years of initial recovery, underscoring the chronic and recurrent nature of the condition [9]. The increasing professionalization and density of competition in elite sport — shortening recovery windows — may further drive these rates upward in coming years [18].

3.2. Neurobiological Mechanisms Relevant to Mental Health

Understanding the neurobiological underpinnings of OTS is essential for clinicians approaching this condition from a psychiatric perspective. Several converging pathophysiological pathways link OTS to primary psychiatric disorders [11, 6].

HPA Axis Dysregulation. Under physiological conditions, the hypothalamic-pituitary-adrenal (HPA) axis serves as the body's primary stress-response system: exercise activates the hypothalamus, which stimulates the pituitary to release adrenocorticotropic hormone (ACTH), in turn triggering cortisol secretion from the adrenal cortex. Cortisol mobilizes energy substrates and — critically for mental health — regulates mood, arousal, and emotional reactivity through its receptors in the limbic system and prefrontal cortex. In a well-trained athlete, this response is proportionate and self-limiting, with the axis returning to baseline after adequate recovery [11, 12]. Chronic overtraining disrupts this balance in a characteristic two-phase pattern. In the early stage of non-functional overreaching, the HPA axis is hyperactivated: cortisol levels are elevated as the organism attempts to cope with the relentless training stimulus, much like any sustained biological stressor. However, as training overload persists without sufficient recovery, the axis progressively loses its responsiveness — a state of neuroendocrine exhaustion. In established OTS, the defining finding is therefore not excess cortisol, but rather a blunted ACTH and cortisol response to exercise challenge, a disrupted diurnal cortisol rhythm, and a loss of neuroendocrine flexibility [1, 6]. This hyporesponsiveness has direct psychiatric consequences: because cortisol and the HPA axis are key modulators of mood regulation, stress resilience, and sleep architecture, their dysregulation produces symptoms — low drive, emotional blunting, fatigue, and sleep disturbance — that are clinically indistinguishable from those of atypical depression and chronic fatigue syndrome, both of which are also characterized by HPA axis exhaustion rather than hyperactivation [6, 11].

Serotonergic Dysregulation. During prolonged and intensive exercise, skeletal muscles take up branched-chain amino acids (BCAAs) — valine, leucine, and isoleucine — as an additional fuel source, causing their plasma levels to fall. BCAAs and tryptophan compete for the same carrier protein to cross the blood-brain barrier; as circulating BCAA concentrations decline, plasma free tryptophan gains a relative advantage and enters the brain in greater quantities [6]. There, tryptophan is converted to serotonin (5-HT), resulting in elevated central serotonergic activity. Under normal physiological conditions, this exercise-induced rise in 5-HT contributes to the subjective perception of effort and signals the onset of fatigue — a protective brake preventing overexertion [13]. In the context of chronic overtraining, however, this mechanism becomes maladaptive. Persistently elevated central 5-HT activity — or, as emerging evidence suggests, an increased sensitivity of serotonin receptors that develops in overtrained athletes — produces sustained fatigue, depressed mood, loss of motivation, and diminished drive that persist well beyond the training session itself [13, 6]. Importantly, it is this receptor hypersensitivity, rather than absolute excess of serotonin, that appears to characterise established OTS: well-trained athletes normally develop reduced 5-HT sensitivity as a physiological adaptation, and the loss of this adaptation in OTS may explain why symptoms persist despite rest [13]. This neurochemical profile closely mirrors the serotonergic dysregulation implicated in depressive disorders, providing a further mechanistic basis for the psychiatric overlap between OTS and major depressive disorder [6, 11].

Neuroinflammation and Cytokine Dysregulation. Each bout of intense exercise produces localised muscle damage and activates the innate immune system, triggering the release of pro-inflammatory cytokines — principally interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor alpha (TNF-α) — as part of the physiological repair process. Under conditions of adequate recovery, this inflammatory response is transient and self-limiting: anti-inflammatory cytokines are subsequently released, oxidative stress is resolved, and tissue homeostasis is restored. Moderate, well-periodised exercise therefore fosters a net anti-inflammatory

environment that is broadly protective for both physical and mental health [11]. In chronic overtraining, however, successive training sessions do not allow sufficient time for this resolution to complete. Pro-inflammatory cytokines accumulate in the bloodstream and, crucially, cross the blood-brain barrier — either directly via active transport, or indirectly by signalling through vagal afferents and circumventricular organs — thereby triggering neuroinflammation in the central nervous system [6, 11]. Once in the brain, elevated IL-1, IL-6, and TNF-α act on limbic structures and the prefrontal cortex to produce a stereotyped behavioural response known as sickness behaviour: fatigue, anhedonia, social withdrawal, cognitive slowing, hypersomnia, and loss of appetite [6]. This pattern is not coincidental — it closely mirrors the symptom profile of major depressive disorder, and there is growing evidence that neuroinflammation is a shared pathophysiological mechanism underlying both conditions. In OTS, the sustained pro-inflammatory state thus acts as a direct neurobiological driver of depressive and motivational symptoms, independent of the psychological burden of underperformance [11, 12].

Autonomic Nervous System Dysfunction. Under normal conditions, the autonomic nervous system (ANS) maintains a dynamic balance between its two branches: the sympathetic system, which mobilises the organism for effort and stress, and the parasympathetic system, which governs recovery, restoration, and rest. In healthy athletes, periods of intense training shift the balance toward sympathetic dominance, while recovery phases allow the parasympathetic system to reassert itself — a rhythm that underpins both physical adaptation and psychological wellbeing. Chronic overtraining disrupts this cycle by sustaining sympathetic activation beyond the body's capacity to compensate [12]. In the early stages of overreaching, sympathetic hyperactivation predominates: resting heart rate rises, the body remains in a state of physiological alertness, and recovery between sessions becomes progressively incomplete. As overtraining progresses toward established OTS, the ANS undergoes a paradoxical shift: the exhausted sympathetic system loses its capacity to respond, and parasympathetic tone becomes relatively dominant — yet this is not restorative. Rather, it manifests as pathological fatigue, bradycardia, emotional blunting, and motivational withdrawal, as the system has effectively disengaged from both effort and recovery [11, 6]. The psychiatric consequences of this dysregulation are direct: persistent sympathetic hyperarousal in the early phase generates anxiety, sleep-onset difficulties, and irritability, while the later parasympathetic dominance produces a clinical picture resembling anhedonia and psychomotor retardation — both core features of depressive episodes [6]. Heart rate variability (HRV), which reflects the flexibility of autonomic regulation, is reduced in overtrained athletes, indicating a loss of the adaptive oscillation between the two branches; this reduction has been validated as an objective, non-invasive marker of emerging OTS in longitudinal monitoring [11, 12].

3.3. Psychiatric Symptomatology of OTS

Athletes with OTS develop a constellation of psychiatric symptoms that, taken individually, are non-specific but collectively constitute a recognizable clinical syndrome. A 2025 systematic review by Andrade et al. [14], encompassing 16 studies and 692 athletes, demonstrated that high training volumes consistently intensify negative mood profiles — including increased fatigue, tension, and depression — while reducing vigor and psychological stamina. The 2026 systematic review by Gerber et al. [15] further documented that non-functional overreaching and OTS impair psychological and cognitive functioning in elite athletes across multiple domains. The clinical presentation can be organized around three overlapping dimensions, each directly traceable to the neurobiological mechanisms described above.

The first and most prominent dimension encompasses affective and anxiety symptoms, which reflect the dysregulation of HPA axis function, serotonergic signalling, and autonomic balance outlined in section 3.2. Depressed mood, irritability, emotional lability, anhedonia, and apathy are among the earliest and most consistently reported manifestations of OTS, frequently emerging before objective performance decrements become apparent [5, 9]. These are accompanied by anxiety symptoms — generalized fearfulness, restlessness, and hypervigilance related to performance evaluation — which reflect the sympathetic hyperactivation characteristic of the early overreaching phase [6]. Tracking the trajectory of these affective states over time is clinically valuable: the Profile of Mood States (POMS) iceberg profile, in which vigor scores exceed all negative subscales, characterizes optimal athletic mental health, whereas a progressive inversion of this pattern — with rising fatigue and depression alongside falling vigor — signals the transition toward non-functional overreaching and OTS [16, 13].

The second dimension comprises neurocognitive impairment and somatic symptoms, which are closely linked to the neuroinflammatory and cytokine-mediated pathways described in section 3.2. Sleep disturbances — including insomnia, non-restorative sleep, and disrupted circadian rhythm — are both a consequence of

OTS and an independent trigger that accelerates its progression, creating a self-reinforcing cycle of inadequate recovery [17]. Cognitive deficits — reduced concentration, impaired working memory, slowed processing speed, and diminished executive function — follow directly from the action of pro-inflammatory cytokines on prefrontal circuits and from the disrupted sleep architecture that undermines memory consolidation [15]. These are accompanied by somatic features including appetite loss, weight changes, gastrointestinal disturbances, and chronic fatigue that persists despite extended rest — the latter reflecting the sustained pro-inflammatory state and HPA axis hyporesponsiveness that characterize established OTS [11].

The third dimension — athlete burnout — represents the psychosocial layer of OTS symptomatology and is conceptually distinct from, yet clinically intertwined with, the preceding biological mechanisms. Burnout in athletes is defined by three core features: emotional exhaustion, depersonalization from sport, and a reduced sense of accomplishment [20]. Burnout symptoms have shown a significant cross-temporal increase between 1997 and 2019 [21], and their co-occurrence with OTS is frequent and diagnostically challenging. Importantly, certain personality characteristics — particularly perfectionism and fear of failure — may constitute vulnerability factors that predispose athletes to both OTS and burnout simultaneously [22]. The relationship between the two conditions is likely bidirectional: the biological exhaustion of OTS depletes the emotional and motivational resources needed to sustain athletic engagement, while the cognitive and emotional features of burnout — avoidance of rest, concealment of symptoms, continued training despite deterioration — may perpetuate the overtraining cycle [18].

3.4. Differential Diagnosis

OTS is a diagnosis of exclusion whose symptom profile overlaps substantially with several primary psychiatric and medical conditions. Table 1 summarises the key shared and distinguishing features of the most clinically relevant differential diagnoses [5, 9, 13, 23, 24, 18].

Table 1. Differential diagnosis of Overtraining Syndrome. FBC = full blood count; TSH = thyroid-stimulating hormone; EBV = Epstein-Barr virus; ECG = electrocardiogram.

Condition	Key shared features with OTS	Key distinguishing features
Major Depressive Disorder (MDD)	Depressed mood, anhedonia, fatigue, sleep disturbance, cognitive impairment, loss of motivation; shared HPA axis dysregulation, serotonergic changes, neuroinflammation	In OTS: dysfunction primarily sport-specific, improves with rest; in MDD: pervasive social/occupational impairment, typically worsens with enforced rest; MDD requires psychiatric treatment regardless of training status
Athlete Burnout	Emotional exhaustion, reduced motivation, performance decline, depersonalization from sport; frequent co-occurrence	Burnout is primarily a psychosocial response to chronic stress, not a physiological maladaptation to training load; no objective neuroendocrine or inflammatory markers; bidirectional relationship with OTS
Anxiety and Trauma-Related Disorders	Sleep disruption, autonomic dysregulation, restlessness, impaired concentration, performance decrements	Identifiable psychological trigger (competition pressure, traumatic event, relationship stress); symptoms present independent of training load; no performance-specific pattern of onset
Relative Energy Deficiency in Sport (RED-S)	Fatigue, mood disturbance, impaired performance, hormonal dysregulation; shared hypothalamic-pituitary origin; 86% of OTS studies show low energy availability markers	RED-S is driven by low energy/carbohydrate availability; nutritional assessment and correction are primary interventions; may coexist with or precipitate OTS
Organic disorders (to exclude)	Fatigue, performance decline, mood changes, sleep disturbance (common to many organic conditions)	Specific laboratory findings: anaemia (FBC), hypothyroidism (TSH), infectious mononucleosis (EBV serology), adrenal insufficiency (cortisol), cardiac disease (ECG, troponin). OTS is a diagnosis of exclusion — organic causes must be systematically ruled out first

Among the conditions listed, the distinction between OTS and major depressive disorder warrants particular clinical attention. As detailed in section 3.2, the two share remarkably similar neurobiological substrates — HPA axis hyporesponsiveness, serotonergic dysregulation, and neuroinflammatory activation — to the extent that some authors have proposed a common pathophysiological origin [6]. In practice, the most useful clinical discriminator remains the domain of dysfunction: impairment in OTS is primarily sport-specific and performance-bound, whereas MDD produces pervasive cognitive, social, and occupational dysfunction independent of athletic context. Furthermore, while rest typically leads to improvement in OTS, the depressed athlete frequently reports worsening of mood and energy with inactivity [5]. However, this distinction is not always clear-cut, particularly in athletes with pre-existing depressive vulnerability, where OTS may unmask or precipitate a primary depressive episode requiring independent psychiatric treatment.

3.5. Prevention and Management

Prevention and management of OTS are best understood as a continuum rather than two separate stages. Given the documented 80–90% relapse rate within three years of initial recovery, effective management does not end with the resolution of acute symptoms — it extends into sustained monitoring, structured return to sport, and active relapse prevention [9]. The following subsections address this continuum across three phases: early monitoring and detection, primary prevention, and treatment of established OTS.

3.5.1. Monitoring and Early Detection

Systematic psychological monitoring is the most effective tool for interrupting the progression from functional overreaching to NFOR and established OTS, given that affective changes reliably precede objective performance decrements by days to weeks [25]. Regular administration of validated instruments — the POMS, the RESTQ-Sport, or the brief Training Distress Scale — enables prospective tracking of mood trajectory across training cycles and should be integrated into routine sports medicine practice rather than reserved for symptomatic athletes [5, 16, 26]. The POMS energy index — calculated as the difference between fatigue and vigor subscale scores — has demonstrated particular sensitivity to training stress and has been used to guide load modifications; in competitive swimmers, POMS-guided training adjustments reduced burnout incidence from 10% to zero [26]. Daily RPE monitoring provides a complementary frontline signal: an overtrained athlete characteristically reports elevated RPE for a given absolute workload, flagging maladaptation early and cost-effectively [1, 26]. For athletes with psychiatric comorbidity or suspected depressive and anxiety symptoms, standard psychiatric instruments — the PHQ-9 and GAD-7 — should be incorporated into routine assessment. At the physiological level, heart rate variability (HRV) has emerged as a promising non-invasive objective marker of autonomic dysregulation in NFOR and OTS, providing a measurable correlate of the subjective monitoring data [11, 12].

3.5.2. Primary Prevention

Primary prevention of OTS rests on a small number of evidence-informed principles that must be applied consistently across the athlete's training environment. Systematic periodization of training — alternating high-load phases with structured recovery periods — is the cornerstone of prevention; training diaries incorporating daily RPE allow coaches and physicians to adjust volume and intensity in response to mood deterioration or performance plateau before NFOR becomes established [26]. Education is equally foundational: athletes must understand that the paradoxical urge to increase training in response to declining performance is itself an early warning sign of overreaching, and that rest is a performance-enhancing strategy rather than a failure. Coaching staff education is critical in this regard, as coaches are typically the first to observe behavioural and performance changes [5, 31]. Nutritional adequacy — particularly maintaining energy availability above the threshold associated with RED-S — is a key modifiable risk factor; even chronic energy deficits of 300–400 kcal/day can become clinically significant over months of intensive training, and adequate carbohydrate availability must be prioritized during high-intensity blocks [23, 29]. Sleep hygiene warrants explicit clinical attention, as inadequate sleep functions both as a consequence and an independent trigger of OTS, creating a self-reinforcing cycle; education on sleep scheduling, sleep environment optimization, and avoidance of stimulants before sleep should be a standard element of athlete health consultations [17]. Finally, integration of mental health programs — including mindfulness-based stress reduction, cognitive-behavioural skills training, and access to sports psychologists and psychiatrists — into elite sports organizations provides the psychological scaffolding that makes all other prevention strategies sustainable [31].

3.5.3. Treatment of Established OTS and Relapse Prevention

Treatment of established OTS requires a patient-centred, multidisciplinary approach. No randomized controlled trials specifically evaluating OTS treatment have been published; recommendations are therefore derived from expert consensus, case series, and extrapolation from related conditions [12, 27]. The primary intervention is reduction or cessation of competitive training. Complete rest, however, may exacerbate mood disturbances — particularly anxiety and depression — in athletes for whom exercise serves as a primary coping mechanism; in such cases, relative rest with low-intensity activity and clear expectations may be preferable, with the choice guided by the athlete's psychiatric risk profile and intrinsic motivation [13]. Return to sport should follow a structured, three-phase protocol — complete cessation, introduction of low-intensity active recovery, then progressive rebuilding of training volume before intensity — monitored throughout by a multidisciplinary team comprising a sports medicine physician, registered dietitian, sport-specific coach, and mental health professional [27]. A real-world illustration of this principle is provided by Solli et al. [28], who documented the individualized, multidisciplinary process enabling the world's best cross-country skier to recover sustainably from a prolonged period of underperformance.

When OTS co-occurs with or precipitates a primary depressive or anxiety disorder meeting diagnostic criteria, psychiatric treatment should follow standard clinical guidelines independently of the athletic context [9]. Cognitive-behavioural therapy (CBT) approaches are particularly well-suited to addressing the maladaptive cognitions that maintain the overtraining cycle — perfectionism, fear of detraining, and catastrophizing around performance decrements — while mindfulness-based interventions support recovery awareness and emotional self-regulation [31]. When pharmacotherapy is considered for co-occurring MDD, clinicians must be aware that serotonergic agents — SSRIs and SNRIs — can reduce exercise performance; shared decision-making with the athlete is therefore essential [9]. Nutritional rehabilitation — restoring adequate energy availability and optimizing macronutrient composition — is a mandatory treatment component wherever RED-S overlap is identified; in athletes with concurrent eating disorder pathology, specialist psychotherapy should be initiated alongside nutritional rehabilitation [23, 29]. Given the 80–90% relapse rate within three years, treatment planning must explicitly include a relapse prevention phase: regular psychiatric and psychological follow-up after initial recovery, continued psychometric monitoring of mood, and a graduated re-exposure to competitive stress with pre-agreed triggers for reassessment [9, 12].

4. Discussion

The most fundamental challenge revealed by this review is the persistent methodological fragility of the OTS literature. As demonstrated by Grandou et al. [2], no published study has simultaneously met rigorous criteria for objective performance decrements and documented psychological symptoms over the required timeframe — meaning that the very condition this review addresses lacks a validated operational definition. This is not a minor limitation: without consensus diagnostic criteria, prevalence estimates spanning 20–60% carry wide uncertainty, cross-study comparisons are unreliable, and the evidence base for any clinical recommendation rests on a foundation of terminological inconsistency. The heterogeneity is compounded by the ethical impossibility of experimentally inducing OTS, which constrains research to observational and retrospective designs, and by the fact that most available studies describe overreached rather than truly overtrained athletes. Standardization of diagnostic criteria — informed by psychiatric nosology and incorporating prospectively validated psychometric thresholds — remains the single most important methodological priority for the field.

Several clinically significant questions remain unresolved and point to a research agenda that is largely unexplored. The directionality of the relationship between OTS and major depressive disorder is perhaps the most pressing: does chronic neuroendocrine and neuroinflammatory stress from overtraining precipitate or unmask depressive episodes in vulnerable individuals, or does subclinical depressive diathesis — through its effects on recovery behaviour, sleep, and motivation — increase susceptibility to OTS? These hypotheses are not mutually exclusive, and their investigation requires prospective longitudinal designs with rigorous psychiatric assessment at multiple time points, which have not yet been conducted [6]. Equally underexplored are the roles of athletic identity, perfectionism, and compulsive exercise tendencies in maintaining the overtraining cycle after initial recovery — constructs well-characterized in clinical psychology but almost entirely absent from OTS research. The high relapse rate of 80–90% within three years [9] strongly suggests that biological recovery alone is insufficient and that these psychological perpetuating factors must be directly targeted, yet no validated psychological intervention for OTS relapse prevention currently exists.

From a broader perspective, OTS represents an underutilized naturalistic model for studying the relationship between sustained biological stress and the emergence of psychopathology. The convergence of HPA axis exhaustion, serotonergic receptor dysregulation, and neuroinflammatory activation in a defined, measurable population of athletes offers a unique opportunity to investigate mechanisms shared with major depressive disorder and chronic fatigue syndrome in a context where the precipitating stressor is quantifiable and temporally defined. Sports psychiatry is well-positioned to lead this research agenda — not only by developing sport-specific screening protocols and validating psychological interventions, but by contributing to the broader understanding of stress-induced psychopathology. Realising this potential will require sustained interdisciplinary collaboration between sports medicine, psychiatry, psychology, nutritional science, and exercise physiology, as well as institutional recognition that the mental health of elite athletes is a clinical priority, not an afterthought.

5. Conclusions

Overtraining Syndrome is a biopsychosocial condition whose psychiatric dimension — encompassing depressive symptoms, anxiety, cognitive impairment, sleep disturbance, and burnout — is clinically substantial yet systematically underrecognized. The neurobiological mechanisms driving these manifestations, particularly HPA axis exhaustion, serotonergic receptor dysregulation, and neuroinflammatory activation, are shared with primary psychiatric disorders, most notably major depressive disorder, and provide a mechanistic rationale for integrating psychiatric expertise into the routine evaluation and management of overtrained athletes.

Effective clinical care requires a multidisciplinary approach spanning early psychological monitoring, structured prevention, individualized return-to-sport planning, and active relapse prevention. The field is currently constrained by the absence of validated diagnostic criteria and evidence-based psychological interventions — gaps that sports psychiatry is uniquely positioned to address. Prospective longitudinal research, randomized intervention trials, and the development of sport-specific psychiatric protocols represent the most urgent research priorities.

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