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NARRATIVE REVIEW

Neurological Aspects of Overtraining Syndrome in Competitive Dance: A Narrative Review

a narrative review

HIGHLIGHTS

- Overtraining syndrome (OTS) in competitive dance is a neurobiological condition extending well beyond peripheral musculoskeletal fatigue, involving HPA-axis dysregulation, autonomic imbalance, monoaminergic depletion, and neuroinflammation.
- Competitive dance combines extreme physical effort with precise proprioceptive, rhythmic, and artistic demands, creating a uniquely vulnerable neurocognitive environment for OTS development.
- Heart rate variability (HRV) monitoring, validated psychometric instruments such as the RESTQ-Sport, and

neurocognitive screening are practical tools for early identification of dancers at risk of OTS.
▸ Disrupted sleep architecture, suppressed BDNF expression, and impaired motor memory consolidation contribute to performance decline and impaired motor engram execution in affected dancers.
▸ Effective prevention and management require a multidisciplinary approach integrating sports medicine, neurology, and psychology, with training-load periodization, sleep optimization, targeted nutrition, and psychological interventions.

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ABSTRACT

BACKGROUND AND AIM: Overtraining Syndrome (OTS) is a maladaptive physiological and psychological response to prolonged high-intensity exercise without adequate recovery. Although musculoskeletal and endocrinological consequences of this condition are relatively well-documented, the neurological aspects of OTS remain underexplored. This narrative review aims to synthesize available evidence on the neurobiological mechanisms underlying OTS with special emphasis on the unique neurological demands of sport dance.

MATERIALS AND METHODS: A narrative literature review was conducted using PubMed, Scopus, and Google Scholar databases. Search terms included: overtraining syndrome, competitive dance, sport dance, central fatigue, HPA axis, autonomic nervous system, serotonin, dopamine, neuroinflammation, cognitive function, and proprioception. Articles published between 2000 and 2025 in English were included. Studies pertaining to general athlete populations were included when dance-specific data were unavailable.

RESULTS: OTS in competitive dance involves dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis, autonomic nervous system dysregulation, depletion of monoaminergic neurotransmitters (serotonin, dopamine), and neuroinflammatory processes. Dance-specific neurological vulnerabilities include impaired proprioception, disrupted motor engram execution, cerebellar and basal ganglia fatigue, and sleep-dependent motor memory consolidation deficits.

CONCLUSIONS: The neurological profile of OTS in competitive dance is complex and multidimensional. The crucial aim is early identification of the problem using validated psychometric tools, heart rate variability monitoring, and neurocognitive screening. A multidisciplinary approach — integrating sports medicine, neurology, and psychology — is essential for both prevention and treatment.

KEYWORDS overtraining syndrome; competitive dance; sport dance; neurological aspects; HPA axis; central fatigue; autonomic nervous system; dopamine; serotonin; cognitive function; proprioception; heart rate variability

1. INTRODUCTION

Competitive dance represents one of the most physiologically and neurologically demanding sport forms. It includes disciplines such as Standard and Latin ballroom dancing, both combining extreme physical effort with the simultaneous execution of precise technical, artistic, and rhythmic tasks, which is distinct from conventional endurance or strength sports. For this reason, competitive dance places unique and multifaceted requirements on the central nervous system (CNS) [1]. Weekly training volumes in elite sport dance often exceed 20–30 hours, with training intensity varying from moderate aerobic work to near-maximal anaerobic bursts during competition routines [2].

The concept of Overtraining Syndrome (OTS) is defined as a chronic maladaptive response originating from an imbalance between training load and recovery. Typically, OTS manifests as persistent performance decline, fatigue, mood disturbances, and hormonal dysregulation [3]. The primary differences compared with the temporary state of non-functional overreaching (NFOR) are a longer time course of recovery (weeks to months) and the depth of systemic involvement [4]. Epidemiological estimates suggest that OTS affects between 20% and 60% of elite athletes across disciplines during their careers [5].

The neurological dimension of OTS has received relatively little attention in spite of growing awareness of this dysfunction. The great majority of research focuses on endocrine biomarkers (mainly cortisol), immunological parameters, and performance standards, although the neurobiological basis — consisting of dysregulated neurotransmission, autonomic imbalance, neuroinflammation, and impaired motor cognition — remains insufficiently characterized. Particularly the context of competitive dance is still underexplored [6].

Competitive dance presents a uniquely vulnerable neurocognitive environment for OTS development. The marked susceptibility to the cascading dysfunction characteristic of the disorder is induced by reliance on refined proprioceptive feedback, cerebellar-mediated rhythmic synchronization, dopaminergic motivation circuits, and sleep-dependent motor memory consolidation [7,8]. The psychosocial pressures intrinsic to the discipline augment this vulnerability: aesthetic evaluation by judges, close partner dependency, and early sport specialization — often beginning in childhood — with implications for neurodevelopmental trajectories.

The aim of this narrative review is to systematically describe the neurological mechanisms underlying OTS and to contextualize them within the specific physiological, biomechanical, and psychosocial framework of competitive dance. Understanding the neurological dimension of OTS in this population is an essential step towards developing evidence-based prevention, monitoring, and rehabilitation strategies.

2. METHODS

This article constitutes a narrative literature review. A systematic search of PubMed, Scopus, and Google Scholar databases was conducted in February–March 2025. The following keywords and their combinations were used: overtraining syndrome, OTS, competitive dance, sport dance, ballroom dance, central fatigue, HPA axis dysregulation, autonomic nervous system, neurotransmitter depletion, serotonin fatigue, dopamine exercise, neuroinflammation athletes, proprioception fatigue, motor memory sleep, heart rate variability overtraining, cognitive function sport, BDNF exercise. Only articles published in English between 2000 and 2025 were considered. Due to the scarcity of OTS research specific to competitive dance, articles investigating professional dancers, artistic athletes, and general sport populations were also included where relevant. The narrative design of this review does not permit quantitative synthesis or meta-analysis; conclusions are therefore qualitative in nature and reflect the weight of available evidence.

3. COMPETITIVE DANCE — PHYSIOLOGICAL AND NEUROLOGICAL DEMANDS

3.1. Training Characteristics and Physical Load

Competitive ballroom dance is classified as an intermittent high-intensity sport, characterized by sequences of intensive anaerobic effort alternating with active aerobic recovery phases [2]. Research indicates that training volumes in elite-level dancers regularly exceed 20–30 hours per week, with peak heart rates approaching 95% of maximum during competition routines and post-exertion lactate concentrations comparable to those observed in middle-distance runners. This combination of metabolic and neuromuscular stress places substantial cumulative demand on the central nervous system.

Competitive dance is unique particularly due to the dual nature of this sport — it requires both athletic performance and artistic expression, which generates an exceptionally high cumulative training load. Beyond physical conditioning, dancers must continuously refine choreography, technique, and partner coordination, often within long, late-evening practice schedules that conflict with circadian regulation of recovery and sleep.

3.2. Neurocognitive Demands Specific to Dance

The neurological demands of competitive dance are qualitatively distinct from those of most other sports. At the cortical level, dance engages a distributed network encompassing the motor cortex, supplementary motor area, premotor cortex, cerebellum, basal ganglia, and prefrontal cortex. These regions cooperate to control rapid postural adjustments, complex limb coordination, and real-time integration of auditory, visual, and proprioceptive input.

The precise regulation of body posture, weight transfer, rotational balance, and partner coupling all depend on intact proprioceptive signaling from muscle spindles, Golgi tendon organs, and joint mechanoreceptors. Vestibular input is critical for spatial orientation during spins and rotations, while cerebellar processing supports temporal precision and rhythmic entrainment with music.

Furthermore, competitive dancers develop motor engrams, defined as highly automatized motor programs created through thousands of hours of repetitive practice. These engrams are consolidated primarily during slow-wave and REM sleep, and their reliable retrieval underlies the apparently effortless execution of complex choreographies. Disruption of either learning or consolidation processes therefore translates directly into performance decline.

4. DEFINITIONS AND EPIDEMIOLOGY OF OVERTRAINING SYNDROME

4.1. Classification and Diagnostic Criteria

The European College of Sport Science (ECSS) and the American College of Sports Medicine (ACSM) distinguish three stages along the progressive spectrum of maladaptation to training load: Functional Overreaching (FOR), Non-Functional Overreaching (NFOR), and Overtraining Syndrome (OTS) [4]. FOR represents a short-term decrement in performance that resolves within days of recovery and is often used as a planned training stimulus. NFOR involves a more pronounced decrement that may persist for weeks. OTS represents the most severe and prolonged form, characterized by months of impaired performance and systemic symptoms.

With regard to diagnostics, OTS remains a diagnosis of exclusion. A major challenge in sports medicine is the absence of a single validated biomarker or gold-standard diagnostic test [4]. Current diagnostic approaches rely on a combination of clinical history, performance decrement documentation, symptom profiles, and the exclusion of medical conditions that can mimic OTS, including anemia, thyroid dysfunction, depressive disorders, and infectious diseases.

4.2. Prevalence and Dance-Specific Context

Researchers estimate that OTS prevalence across sports ranges from 20% to 60% of elite athletes across their careers [5]. However, dance-specific epidemiological data remain extremely limited. The key risk factors include high training volume, rapid increases in training load, insufficient recovery, concurrent psychosocial stressors, and inadequate nutrition or sleep. In competitive dance, these factors are often compounded by early sport specialization, perfectionistic culture, and aesthetic pressure regarding body composition [18,20].

5. NEUROBIOLOGICAL MECHANISMS OF OTS

5.1. Hypothalamic-Pituitary-Adrenal (HPA) Axis Dysregulation

The hypothalamic-pituitary-adrenal (HPA) axis comprises one of the primary neuroendocrine systems through which the body responds to physiological and psychological stress [13]. During acute exercise, activation of the HPA axis results in transient release of corticotropin-releasing hormone, adrenocorticotropic hormone, and cortisol, mobilizing energy substrates and modulating immune function. In healthy adaptation, this response is followed by efficient downregulation during recovery.

In the early stages of OTS, called the hypersympathetic phase, HPA-axis activity is elevated, producing chronically high cortisol levels [3]. Prolonged hypercortisolism exerts neurotoxic effects on the hippocampus, prefrontal cortex, and amygdala, impairing neurogenesis, synaptic plasticity, and emotional regulation. In later stages, the axis may transition into a hyporesponsive state, with blunted cortisol responses to stress and persistent fatigue.

There are neurobiologically significant parallels between HPA-axis dysfunction in OTS and major depression [14]. Both conditions involve disruption of glucocorticoid receptor sensitivity, altered feedback regulation, and downstream effects on monoaminergic transmission, which may help explain the substantial symptomatic overlap between athletes with OTS and patients with clinical depression.

5.2. Autonomic Nervous System Dysregulation

The autonomic nervous system (ANS) serves as the primary rapid-response regulator of physiological adaptation to training stress [4]. Healthy athletes have lower resting heart rates and higher heart rate variability (HRV), reflecting predominance of parasympathetic tone at rest and efficient sympathetic mobilization during effort. With progression toward OTS, this balance shifts: resting heart rate may increase, while HRV declines, signaling diminished parasympathetic reactivation.

Heart rate variability (HRV) is a measure of beat-to-beat variation in cardiac interval that reflects autonomic tone. It is considered one of the most practical and sensitive non-invasive markers of autonomic balance in athletes. Sustained reductions in HRV over consecutive days, particularly in the morning, are increasingly recognized as an early warning sign of non-functional overreaching and developing OTS.

Autonomic dysregulation in OTS also manifests as disrupted thermoregulation, altered gastrointestinal motility, impaired immune modulation, and reduced adaptability of cardiovascular responses during effort and recovery, all of which directly affect performance and well-being in competitive dancers.

5.3. Monoaminergic Neurotransmission: Serotonin and Dopamine

The monoaminergic hypothesis of OTS, formulated primarily by Meeusen and colleagues, suggests that a crucial role in the development of central fatigue is played by chronic training-induced depletion of brain serotonin and dopamine [6,8]. Increased free-tryptophan transport across the blood-brain barrier during prolonged exercise enhances serotonergic activity, which may contribute to perceived effort, mood disturbances, and reduced motivation.

Dopamine plays a vital role in motivation, reward processing, psychomotor arousal, attentional focus, and movement initiation [7,8]. The characteristic 'exercise-induced euphoria' is partly mediated by dopaminergic activation, while chronic depletion can produce anhedonia, motor slowing, and reduced training drive — symptoms that are clinically prominent in athletes with established OTS.

The interdependence of serotonergic and dopaminergic systems is essential: serotonergic projections from the dorsal raphe nucleus inhibit dopaminergic release in the substantia nigra pars compacta, meaning that chronic serotonergic excess in OTS can secondarily suppress dopaminergic motivation circuits.

5.4. Neuroinflammation and the Immune-Neural Interface

Chronic high-intensity training without adequate recovery activates the innate immune system, leading to sustained elevation of pro-inflammatory cytokines including interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor-α (TNF-α) [11]. Although acute exercise-induced cytokine release supports adaptive remodeling, chronic elevation can cross the blood-brain barrier and influence central nervous system function.

The gut-brain axis as a pathway through which intensive exercise affects neurological function is attracting increasing interest. Approximately 90% of the body's serotonin is synthesized in the gastrointestinal tract, and dysbiosis induced by repeated stress, altered nutrition, and exercise-related changes in gut permeability may contribute to systemic inflammation and neurobehavioral symptoms observed in OTS.

5.5. Sleep Architecture and BDNF-Mediated Neuroplasticity

The crucial period for neurological restoration and motor memory consolidation is sleep. Both slow-wave sleep (SWS) and REM sleep are critical. SWS supports synaptic homeostasis and metabolic waste clearance, while

REM sleep plays a key role in consolidation of procedural and emotional memory. Brain-derived neurotrophic factor (BDNF) modulates these restorative processes by supporting synaptogenesis and neuronal survival.

This neurorestorative cycle is deeply disrupted in OTS. First, elevated cortisol and cytokine levels suppress BDNF expression; then sympathetic overactivation delays sleep onset and reduces SWS proportion. The resulting impairment of motor memory consolidation is particularly detrimental in competitive dance, where automated motor programs underpin nearly every performance.

6. DANCE-SPECIFIC NEUROLOGICAL MANIFESTATIONS OF OTS

6.1. Proprioceptive and Somatosensory Dysfunction

Proprioception is the continuous, unconscious monitoring of body segment position, velocity, and loading. It is exceptionally important in competitive dance. The execution of technically demanding figures — pirouettes, lifts, partner contact — relies on millisecond-level proprioceptive integration with motor output.

In dancers, OTS-related proprioceptive deterioration may manifest in practice as subtle but performance-critical deficits, including instability during rotational figures (e.g., pirouettes, spins), reduced precision of foot placement, deteriorated partner coupling, and increased risk of microtraumatic injuries.

6.2. Motor Program Disruption and Cerebellar Fatigue

Highly practiced dance routines are encoded as automated motor programs (engrams) and are stored in a distributed network involving the basal ganglia, cerebellum, and supplementary motor area. The basal ganglia coordinate sequence selection and timing, while the cerebellum fine-tunes movement execution and rhythmic precision.

The reliable execution of stored motor programs is impaired in OTS through depletion of dopamine and disruption of cerebellar metabolic reserves. The affected dancer may experience unexplained 'forgetting' of well-trained choreography, increased step errors, and reduced fluency of transitions between figures.

6.3. Musical and Rhythmic Processing Deficits

Mandatory synchronization of movement with music is a defining characteristic of sport dance. It is a complex function that recruits the auditory cortex, basal ganglia, cerebellum, and supplementary motor area. Dopaminergic transmission is integral to beat perception and the predictive timing of motor output.

In the context of OTS, dopaminergic depletion may therefore manifest as subtle but measurable deterioration in musical timing — an increased beat-phase error, reduced consistency of step timing, or impaired anticipation of accents — all of which directly compromise judging scores in competitive evaluation.

6.4. Cognitive Fatigue, Attention, and Executive Function

Competitive dance places significant demands on executive cognitive functions: working memory (for choreographic sequencing), selective attention (for spatial awareness and partner interaction), and cognitive flexibility (for real-time adjustments to errors or partner behavior). These functions depend critically on prefrontal cortical integrity.

OTS may cause prefrontal hypofrontality, defined as a reduction in prefrontal cortical activation resulting from dopaminergic insufficiency and neuroinflammatory disruption. It impairs all of the above cognitive domains, producing the clinical picture of slowed thinking, reduced concentration, and diminished decision-making accuracy that competitive dancers may describe as 'mental fog'.

6.5. Psychological Manifestations: Mood, Motivation, and Identity

The psychological dimension of OTS in competitive dance is inseparable from its neurological one. A direct consequence of dopaminergic depletion in the mesolimbic reward pathway is anhedonia — the reduced ability to derive pleasure from previously rewarding activities — which in dancers may manifest as loss of joy in performance and training.

The convergence of HPA-axis dysregulation, monoaminergic depletion, and neuroinflammation explains the depressive symptomatology in OTS, including low mood, fatigue, sleep disturbance, and cognitive impairment. In adolescent dancers, in whom artistic identity is often closely tied to self-concept, these symptoms can carry particularly serious psychological consequences.

7. DIAGNOSIS AND MONITORING

7.1. Clinical Symptom Profile

There is a characteristic constellation of neurological and psychological symptoms of OTS in competitive dance. Key symptoms include persistent fatigue unresponsive to rest, sleep-onset insomnia and non-restorative sleep, deterioration in motor precision and musical timing, anhedonia, increased emotional lability, and progressive performance decline despite continued training effort.

7.2. Validated Assessment Tools

Several psychometric instruments have been validated for OTS screening in athletes. The RESTQ-Sport (Recovery-Stress Questionnaire for Athletes) provides a multidimensional profile of recovery and stress states. The Profile of Mood States (POMS) is also widely used to monitor mood trajectory across training cycles, with increasing depression and decreasing vigor scores often preceding overt performance decline [19].

7.3. Neurophysiological Biomarkers

The most practical neurophysiological biomarker currently available for monitoring autonomic status in athletes is heart rate variability (HRV) [4]. Daily HRV measurement using validated applications (e.g., chest-strap or finger-pulse devices) enables longitudinal tracking of parasympathetic recovery and provides objective data complementing subjective wellness questionnaires. Sustained reductions in morning HRV over consecutive days, combined with self-reported fatigue, should prompt training-load adjustment.

8. PREVENTION AND MANAGEMENT

8.1. Training Load Monitoring and Periodization

In the prevention of OTS in competitive dance, systematic monitoring and management of training load play a key role. A practical framework for identifying dangerous load spikes that precede overreaching and injury is the acute:chronic workload ratio (ACWR) [12]. Structured periodization with planned deload weeks, individualized adjustment to the competitive calendar, and clear separation of high- and low-intensity sessions are essential components of preventive practice.

8.2. Sleep and Neurological Recovery

As sleep plays a pivotal role in neurological restoration and motor memory consolidation, sleep optimization is a non-negotiable component of OTS prevention in competitive dance. Dancers should be instructed in basic sleep-hygiene principles: consistent sleep–wake schedules, limitation of evening light exposure, and avoidance of caffeine and intensive training late in the evening [16,17].

8.3. Nutritional Support for Neurotransmitter Systems

The monoaminergic neurotransmitter systems vulnerable in OTS can be supported by targeted nutritional strategies. Branched-chain amino acid (BCAA) supplementation reduces free-tryptophan transport across the blood-brain barrier and may attenuate exercise-induced central serotonergic activity. Adequate carbohydrate intake supports cerebral metabolism and dopaminergic function, while sufficient energy availability is essential to prevent the compounded risk of Relative Energy Deficiency in Sport (RED-S) [20].

8.4. Psychological and Neuropsychological Interventions

Early psychological interventions may be exceptionally helpful in managing OTS. Mindfulness-based stress reduction (MBSR) and biofeedback techniques targeting HRV coherence have demonstrated efficacy in modulating autonomic balance and emotional reactivity in athletes. Cognitive-behavioral interventions can address perfectionistic cognitions and performance-related anxiety that often coexist with OTS in competitive dance environments.

9. CONCLUSIONS

Overtraining Syndrome in competitive dance is a complex neurobiological condition with a multidimensional pathophysiology that extends well beyond peripheral physiological exhaustion. The neurological mechanisms — including HPA-axis dysregulation, autonomic imbalance, monoaminergic depletion, neuroinflammation, and disrupted sleep-dependent neuroplasticity — interact to produce the characteristic clinical picture of persistent fatigue, performance decline, cognitive impairment, and mood disturbance.

In the context of competitive dance, neurological vulnerability to OTS is intensified by the unique demands of this discipline: integration of proprioceptive precision, cerebellar rhythm processing, dopaminergic motivation, and prefrontal executive control. Early identification through validated psychometric tools, HRV monitoring, and neurocognitive screening — combined with a multidisciplinary approach integrating sports medicine, neurology, and psychology — is essential for effective prevention and management.

The development of dance-specific OTS diagnostic protocols incorporating neurocognitive and rhythmic-motor assessment domains should be prioritized in future research. Longitudinal neuroimaging studies and standardized monitoring frameworks will be required to translate current mechanistic understanding into actionable clinical practice for competitive dancers.

DISCLOSURE

Author Contributions

Conceptualization: Anna Ignatowicz, Gabriela Grylowska. Methodology: Anna Paluch, Michał Tryba. Formal analysis: Anna Ignatowicz, Michał Tryba. Investigation: Mateusz Banasik, Marcin Rebizant. Data curation: Gabriela Grylowska, Katarzyna Gunia. Writing — original draft preparation: Anna Ignatowicz, Katarzyna Gunia, Aleksandra Adamczyk. Writing — review and editing: Andrzej Palak, Agata Żak. All authors have read and agreed to the published version of the manuscript.

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Declaration of Generative AI and AI-Assisted Technologies

In preparing this work, the authors used Claude Sonnet (Anthropic) to assist in drafting and language editing of the text. After using this tool, the authors reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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