Review

Exploring Autonomic Dysfunction in Overtraining: An Updated Narrative Review

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Exploring Autonomic Dysfunction in Overtraining: An Updated Narrative Review

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Abstract: Background: Overtraining syndrome (OTS) is a maladaptive response to prolonged training stress with insufficient recovery, leading to persistent performance decline and physiological dysfunction. Despite its recognition in sport and clinical contexts, the precise mechanisms underlying OTS remain poorly understood, researched, and diagnosis is often reliant on subjective clinical judgment. **Purpose**: This narrative review explores the autonomic imbalance hypothesis as a potential framework for understanding the physiological basis of OTS, emphasizing its relevance to cardiovascular, neuroendocrine, and gastrointestinal dysregulation. **Methods**: A non-systematic, narrative approach was used to conceptualize foundational literature on the topic. Targeted searches of PubMed and Google Scholar were conducted using keywords such as "overtraining syndrome", "autonomic nervous system", "autonomic nervous system regulation", and "parasympathetic dominance". Articles were selected based on their relevance to the autonomic regulation mechanisms implicated in OTS. **Results**: The literature supports the benefit of further investigation into the autonomic imbalance hypothesis and how it may provide a potential framework for mechanisms underlying the progression of OTS. Central mechanisms involving baroreceptor and chemoreceptor sensitivity, hypothalamic regulation, adrenal function, and gut-brain axis disruptions are highlighted as possible contributors to the syndrome's clinical manifestations. **Conclusion:** The autonomic imbalance hypothesis provides a promising lens through which to understand the physiological complexity of OTS. However, empirical validation remains limited. Future research is desperately needed to clarify causal mechanisms, refine diagnostic tools, and assess implications for youth athletes.

Keywords: Autonomic nervous system; Sympathetic dominance; Parasympathetic dysfunction; Baroreflex; Chemoreceptor; Vagal tone; Hypothalamus

Introduction

Overtraining syndrome (OTS) is a complex condition that is known to manifest through the following symptoms, prolonged fatigue, persistent performance decline, and different physiological and psychological disturbances (Meeusen et al., 2013). Despite decades of research, the precise mechanisms responsible for OTS remain poorly understood, presenting challenges for its prevention, diagnosis, and treatment (Meeusen et al., 2013). Among the theories proposed to explain the etiology of OTS, the autonomic imbalance hypothesis has emerged as a potential framework. Initially discussed in depth by Lehmann et al. (1998), this hypothesis suggests that chronic training stress disrupts the autonomic nervous system (ANS), which is known to be responsible for regulating critical bodily functions such as heart rate, stress responses, and recovery. According to the hypothesis, prolonged exposure to training loads that exceed an athlete's adaptive capacity leads to dysregulation of the two branches of the ANS: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) (Lehmann et al.,1998). This dysregulation results in a maladaptive response from the body that ends up impairing recovery, performance, and overall health (Lehmann et al.,1998). This narrative review examines the autonomic imbalance hypothesis in relation to OTS.

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Methods

This narrative review examines the autonomic imbalance hypothesis in the context of overtraining syndrome (OTS), a condition that remains under-researched despite its recognition in athletic and clinical settings. Due to the scarcity of consolidated empirical studies and the absence of standardized diagnostic criteria for OTS, a systematic review methodology was not applied. Instead, a targeted search of databases such as PubMed and Google Scholar was conducted using keywords including "overtraining syndrome", "autonomic nervous system", "sympathetic dominance", "parasympathetic dominance", and "autonomic nervous system regulation". Articles published in English with the date range from 1994 to 2025 were selected based on relevance to the autonomic mechanisms proposed to be implicated in OTS and their contribution to understanding its physiological and psychological dimensions. Non-English language studies were excluded. Search terms were combined using Boolean operator functions AND/OR. This flexible, narrative approach was chosen to synthesize available insights and highlight existing gaps in the literature.

Results and Discussion

Overtraining Syndrome and The Autonomic Imbalance Hypothesis

Overtraining syndrome represents the maladaptive response to an imbalance between training stress and recovery, leading to prolonged decrements in performance and health (Meeusen et al., 2013). Unlike functional overreaching, which is characterized by temporary performance declines that are necessary for athletes and result in long-term gains when followed by adequate recovery, OTS involves a chronic inability to restore homeostasis, often accompanied by a range of physiological and psychological symptoms (Meeusen et al., 2013). Further complication includes a non-specified criteria for diagnosing OTS, and a heavier reliance on practitioner experience for diagnosis. The autonomic imbalance hypothesis, as discussed by Lehmann et al. (1998), states that chronic training stress disrupts the equilibrium between the SNS, which controls the body's stress response, and the PNS, which controls recovery and energy conservation. In the early stages of overtraining, sympathetic overactivation is often observed, manifesting in elevated resting heart rates, reduced heart rate variability (HRV), and heightened stress hormone levels such as cortisol (Lehmann et al.,1998). If the stress persists without adequate recovery, a shift toward parasympathetic dominance may occur (Lehmann et al.,1998). This phase, sometimes referred to as parasympathetic overtraining, is characterized by symptoms such as bradycardia, a heart rate slower than what is considered normal, persistent fatigue, and an inability to mount appropriate physiological responses to exercise (Lehmann et al., 1998). Under normal exercise conditions, the body responds by increasing SNS activity, preparing for physical exertion (Chiang et al., 2024). This includes an elevated heart rate, pupil dilation, reduced blood flow to PNS functions like digestion, and increased blood flow to muscles to enhance oxygen delivery and utilization (Chiang et al., 2024). As the individual comes to rest, discontinuing exercise, the PNS counteracts SNS activity, helping the body return to its resting physiological state (Matei et al., 2022). The autonomic imbalance hypothesis suggests that chronic stress on the body leads to sustained SNS activation in order to maintain physiological responses to exercise (Lehmann et al., 1998). Over time, when the PNS activates in response to the discontinuation of exercise, it may either be suppressed from overactive SNS activity or overcompensate, resulting in an overall imbalance (Lehmann et al., 1998). This dysregulation can lead to excessive PNS dominance or a blunted response from the PNS, causing symptoms such as chronic fatigue, bradycardia, and reduced responsiveness to everyday stimuli (Lehmann et al., 1998).

Regulation of the Autonomic Nervous System: Baroreceptors

Regulation by the ANS is a multilayered system, composed of different systems contributing and working in unison so the body can respond rapidly in response to changing stimuli (McCorry, 2007). Baroreceptors, chemoreceptors, and the hypothalamus form some of the core physiological

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monitoring mechanisms, while emotional inputs, local reflexes, hormonal responses, and circadian patterns all contribute as well, playing crucial roles in shaping autonomic balance (Gibbins, 2013; McCorry, 2007). This complex network ensures the body can maintain internal stability while adapting to changing conditions and needs of the external environment. Among the different mechanisms contributing to autonomic balance are baroreceptors, which monitor changes in blood pressure (Izzo & Taylor, 1999; McCorry, 2007). Located in the carotid sinus and aortic arch, baroreceptors are stretch-sensitive nerve endings that detect the degree of vessel wall distension caused by circulating blood (Wehrwein & Joyner, 2013). When arterial pressure increases, the walls stretch more, increasing baroreceptor firing (Porzionato et al., 2018; Wehrwein & Joyner, 2013). These signals are transmitted via the glossopharyngeal nerve (cranial nerve IX) from the carotid sinus and the vagus nerve (cranial nerve X) from the aortic arch to the nucleus tractus solitarius (NTS) in the medulla oblongata (Massari et al., 1996; Porzionato et al., 2018). The nucleus tractus solitarius (NTS) processes afferent input from baroreceptors and responds by enhancing parasympathetic vagal activity and inhibiting sympathetic output via its connections to the rostral ventrolateral medulla (RVLM) and the caudal ventrolateral medulla (CVLM) (Holstein et al., 2011; Moreira et al., 2005). Parasympathetic activity is increased through activation of vagal motor nuclei, including the nucleus ambiguus and the dorsal motor nucleus of the vagus nerve (DMNV) (Holstein et al., 2011; Moreira et al., 2005). Simultaneously, sympathetic output is suppressed as the NTS excites the CVLM, which in turn inhibits the RVLM, an area responsible for generating sympathetic tone (Guyenet, 2006; Holstein et al., 2011; Moreira et al., 2005). The result is decreased heart rate, decreased cardiac contractility, and vasodilation, thereby lowering blood pressure (Holstein et al., 2011; Moreira et al., 2005). Conversely, a drop in blood pressure reduces baroreceptor firing, leading to decreased parasympathetic and increased sympathetic tone, which raises heart rate and causes vasoconstriction to restore perfusion.

Regulation of the Autonomic Nervous System: Chemoreceptors

Chemoreceptors serve as another vital feedback mechanism, detecting changes in blood gas composition and pH. Peripheral chemoreceptors, located in the carotid bodies and aortic bodies, sense arterial oxygen (PaO_2), carbon dioxide ($PaCO_2$), and hydrogen ion concentration (pH) (Gonzalez, 1994; Wong-Riley et al., 2012). When oxygen levels drop or CO_2/pH levels rise, these receptors send afferent signals, also via cranial nerves IX and X, to the NTS (Accorsi-Mendonca & Machado, 2013; Lust, 2007). In response, the brainstem triggers increased ventilation through respiratory centers and may initiate sympathetic activation to maintain perfusion to vital organs during perceived hypoxic or acidotic stress conditions (Pippalapalli & Lumb, 2023). Central chemoreceptors, located in the medulla are also thought to be dispersed throughout the brainstem, cerebellum, hypothalamus and midbrain, responding primarily to changes in CO_2 levels and pH in the cerebrospinal fluid (CSF) (Goyne & Bayliss, 2023; Nattie & Li, 2012). Elevated CO_2 diffuses into the CSF, forms carbonic acid, and lowers pH, which stimulates the chemoreceptors to drive respiratory rate up and, in prolonged states, also increases sympathetic tone (Nattie & Li, 2012).

Regulation of the Autonomic Nervous System: Brain Structures

The hypothalamus plays a higher-order integrative role, serving as the command center for autonomic coordination (Coote & Spyer, 2018). It receives input from baroreceptors, chemoreceptors, osmoreceptors, and the limbic system and orchestrating both endocrine and autonomic responses (Coote & Spyer, 2018; Ulrich-Lai & Herman, 2009). The paraventricular nucleus (PVN) sends descending projections to autonomic centers in the brainstem and spinal cord, influencing heart rate, blood pressure, and fluid balance (Ulrich-Lai & Herman, 2009). On a fundamental level, the anterior hypothalamus generally facilitates parasympathetic responses, such as heat dissipation via vasodilation and sweating, while the posterior hypothalamus promotes sympathetic activity like thermogenesis and vasoconstriction during cold exposure (Li et al., 2022). The hypothalamus also governs complex homeostatic functions such as hunger, thirst, sexual behavior, and circadian

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rhythms, often involving coordinated autonomic and hormonal responses such as stimulating the pituitary gland to release vasopressin for water retention (Timper & Bruning, 2017). Beyond these central systems, higher brain centers, particularly the limbic system and prefrontal cortex, modulate autonomic tone based on emotion, memory, and anticipation (Hariri et al., 2000; Salzman & Fusi, 2010). The amygdala plays a key role in fear responses, projecting to the hypothalamus and brainstem to trigger sympathetic activation, elevating heart rate and preparing the body for a "fight or flight" response (Hariri et al., 2000; Simic et al., 2021). Emotional stimuli such as stress, anxiety, or even excitement can override baseline autonomic signals, increasing sympathetic drive even in the absence of physical stimuli (Simic et al., 2021).

Regulation of the Autonomic Nervous System: Enteric Nervous System and Adrenal Medulla

Another particularly independent but important consideration is the enteric nervous system (ENS), often referred to as the "second brain" (Avetisyan et al., 2015). The ENS is a network of over 100 million neurons capable of managing digestion, motility, and secretion without direct input from the central nervous system (Fleming II et al., 2020). However, it does communicate with both the sympathetic and parasympathetic systems via the vagus nerve and prevertebral ganglia, allowing for top-down modulation of digestive function (Fleming II et al., 2020). In addition to neural mechanisms, the adrenal medulla acts as a neuroendocrine extension of the sympathetic system (Goldstein, 2021). When stimulated by preganglionic sympathetic fibers (cholinergic neurons), the adrenal medulla releases epinephrine and norepinephrine into the bloodstream (Goldstein, 2021; Lumb et al, 2018). These catecholamines enhance sympathetic effects systemically, raising heart rate, blood glucose, and vascular tone, especially during sustained stress when hormonal support is needed to maintain autonomic responses over time (Goldstein, 2021).

Possible Mechanistic Pathways Underlying ANS Dysfunction

According to the autonomic imbalance hypothesis, a central feature of OTS is chronic dysregulation between the sympathetic and parasympathetic branches of the autonomic nervous system (Lehmann et al.,1998). One possibility could be maladaptive baroreflex function. With repeated high-volume training, baroreceptors may become desensitized due to chronically elevated blood pressures during exercise bouts (Alvarez-Araos et al., 2023; Miki & Yoshimoto, 2018). This desensitization could blunt the normal inhibitory feedback to the RVLM, allowing excessive or sustained sympathetic output even during rest, contributing to cardiovascular strain, poor sleep, and elevated resting heart rate often seen in early-stage OTS (Alvarez-Araos et al., 2023; Miki & Yoshimoto, 2018). In some athletes, however, chronic vagal hyperactivation may dominate instead, particularly in endurance-trained individuals, potentially reflecting an overcompensation by the CVLM-mediated inhibition of sympathetic tone (Mandel & Schreihofer, 2008). Further reiterated by Coote and White (2015), resting bradycardia following endurance training is attributable to high cardiac vagal tone, suggesting the possibility for chronic vagal hyperactivation and a main role in OTS if prolonged training occurs without adequate rest. If this inhibition is too strong or prolonged, it might lead to bradycardia, fatigue, and depression-like symptoms, as observed in the "parasympathetic overtraining" subtype. Another possible mechanism to consider may involve chemoreceptor dysregulation. Prolonged hypoxic or acidotic states during intense training may lead to heightened sensitivity or maladaptation of peripheral chemoreceptors in the carotid and aortic bodies (Dempsey & Smith, 2019; Powell, 2007; Wang & Bisgard, 2002). This could exaggerate ventilatory or sympathetic responses during even mild stressors, contributing to hyperventilation, increased resting heart rate, or exercise intolerance (Dempsey & Smith, 2019; Wang & Bisgard, 2002). Similarly, central chemoreceptors might be persistently exposed to elevated CO_2 or altered CSF pH due to respiratory compensation during chronic training, potentially recalibrating their thresholds and affecting respiratory control and autonomic outflow (Powell, 2007). The hypothalamus may also undergo stress-related neuroplastic changes. It integrates not only autonomic but also endocrine and behavioral responses to training stress (Shaeuble & Myers, 2022). Chronic activation of the PVN and

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posterior hypothalamus, which support sympathetic drive, could contribute to elevated cortisol, disrupted circadian rhythms, and poor recovery (Shaeuble & Myers, 2022). If the hypothalamus begins prioritizing parasympathetic restoration due to excessive sympathetic activity, it might upregulate pathways promoting vagal tone. However, if this increased vagal signaling becomes ineffective due to receptor downregulation or desensitization at the heart or gut level, the body may enter a dysfunctional state where parasympathetic tone appears elevated, but physiological recovery is impaired. This might explain paradoxical findings in overtrained athletes where resting heart rate is low, but fatigue, poor performance, and sleep disturbances persist. The enteric nervous system and gut-brain axis may also be compromised. Prolonged stress and sympathetic dominance can impair gastrointestinal motility, alter the gut microbiome, and increase intestinal permeability, leading to low-grade inflammation and disrupted nutrient absorption (Leigh et al., 2023; Madison & Bailey, 2023). These gut changes can send afferent signals to the brainstem and hypothalamus via the vagus nerve, further interfering with autonomic homeostasis and potentially contributing to the fatigue, poor appetite, and gastrointestinal symptoms seen in OTS (Leigh et al., 2023). The adrenal medulla may play a role as well. Chronic stimulation by sympathetic preganglionic neurons could lead to catecholamine depletion or blunted hormonal responsiveness, such that during actual stress, the system fails to mount an appropriate epinephrine/norepinephrine response (Morrison & Cao, 2000). This "adrenal fatigue" like state could manifest as poor performance, low energy, and impaired cardiovascular responsiveness during training.

Stipulations of the Hypothesis and Areas for Future Research

The autonomic imbalance hypothesis sets out that the onset of OTS is driven by sustained dysregulation of the ANS, resulting from excessive training loads, insufficient recovery, and compounded external stressors (Lehmann et al.,1998). According to this framework, OTS evolves along a continuum, beginning with sympathetic overactivation and progressing to parasympathetic dominance as the body's adaptive capacity is exhausted. This maladaptive trajectory is believed to underlie many of the symptoms observed in overtrained athletes, including chronic fatigue, hormonal imbalances, and performance decrements (Meeusen et al., 2013). The hypothesis faces multiple limitations. One key area of interest is the diagnostic utility of HRV as a marker of autonomic imbalance. While HRV has shown promise as a non-invasive tool for assessing ANS function, its sensitivity and specificity for detecting OTS remain unclear (Hayano and Yuda, 2019). Similarly, altered levels of stress hormones such as cortisol and catecholamines, while indicative of autonomic dysregulation, lack diagnostic precision and may be influenced by confounding factors such as psychological stress or illness (Vage et al., 2023). Pediatric populations represent another significant gap in the literature. Most of the research on OTS has been conducted in adult athletes, leaving questions about its relevance to children and adolescents unanswered. Given the unique developmental, physiological, and psychological characteristics of youth athletes, studies exploring autonomic regulation in this demographic are urgently needed.

Conclusions

The autonomic imbalance hypothesis provides a potentially valuable framework for understanding the physiological mechanism of overtraining syndrome. Limited supportive evidence exists and significant gaps in knowledge remain, particularly regarding causality, biomarker validation, and intervention strategies. By addressing these gaps, the sports science and medical communities can improve the prevention, diagnosis, and management of OTS. Continued research in this area is particularly critical for pediatric athletes, whose unique characteristics demand tailored approaches to training and recovery.

Authors' Qualifications: The authors' qualifications are as follows: Nicholas Burton BKin, MKin Student

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