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ADRENAL GLANDS FUNCTION AND ORTHOSTATIC INTOLERANCE IN ATHLETES: IMPLICATIONS FOR SPORTS PERFORMANCE AND TRAINING MANAGEMENT

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ABSTRACT

Orthostatic intolerance (OI) encompasses a spectrum of conditions characterized by impaired cardiovascular regulation upon standing, leading to symptoms such as dizziness, fatigue, lightheadedness, syncope, and reduced exercise tolerance. While frequently studied in clinical settings, OI remains underrecognized in athletes, where its impact on performance, recovery, and well-being may be substantial. This narrative review examines the physiological basis of OI with a particular focus on adrenal gland function—namely the secretion of cortisol, aldosterone, and catecholamines—and their role in maintaining orthostatic stability. It explores how factors specific to athletic populations, such as intense training intensity, dehydration, and relative energy deficiency (RED-S), can impair autonomic and adrenal responses. Diagnostic strategies including orthostatic testing and hormonal profiling are discussed, along with the need for dynamic assessments that reflect functional hormone reserve. Management options range from non-pharmacologic interventions like fluid and salt optimization to pharmacological support in severe cases. The article also highlights the importance of athlete education, training periodization, and coach involvement. Finally, future research priorities are outlined, including sex-specific hormonal dynamics, wearable monitoring, and early biomarkers of dysregulation. Increasing awareness of OI in athletic settings and its hormonal underpinnings can contribute to more effective detection, prevention, and treatment strategies—ultimately improving health outcomes and sustaining high performance.

KEYWORDS

Orthostatic Intolerance, Adrenal Glands, Cortisol, Aldosterone, Athletes, Autonomic Function

CITATION

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1. Introduction

Optimal sports performance depends on the integrated function of the cardiovascular, endocrine, and autonomic nervous systems. Among these, the adrenal glands are central to stress adaptation and postural regulation. By releasing glucocorticoids and catecholamines, they help to stabilize blood pressure, mobilize energy, and regulate fluid-electrolyte balance—all essential for maintaining orthostatic tolerance during rapid position changes or prolonged standing. Impaired adrenal output or responsiveness can weaken these compensatory mechanisms, contributing to orthostatic intolerance (OI)—a condition marked by dizziness, tachycardia, weakness, and even syncope upon standing [1].

OI has primarily been studied in clinical populations with autonomic or endocrine disorders. However, growing evidence suggests that athletes, particularly those in endurance or high-intensity training, may also experience OI. In this population, symptoms are often subtle and misattributed to overtraining, anxiety, or deconditioning. Athletic OI may present as fatigue, reduced exercise tolerance, slowed recovery, or unexplained performance decline, especially during standing or low-intensity activities such as post-exercise cool-downs [2, 3].

The adrenal cortex and medulla contribute essentially to orthostatic compensation. Cortisol, secreted in a diurnal rhythm and in response to adrenocorticotropic hormone (ACTH), enhances vascular responsiveness to catecholamines, sustains blood pressure, and supports glucose metabolism. Meanwhile, the adrenal medulla releases epinephrine and norepinephrine to increase heart rate, vascular resistance, and cardiac output during postural stress. Suboptimal adrenal function—whether due to relative adrenal insufficiency, hypothalamic-pituitary-adrenal (HPA) axis suppression, or dysautonomia—can impair these reflexes and render athletes vulnerable to orthostatic challenges [4].

Several sport-specific factors may increase vulnerability to OI. Chronic high training loads can desensitize the HPA axis and blunt cortisol responses, especially with inadequate recovery [5]. Relative energy deficiency in sport (RED-S) may also suppress hormones and reduce adrenal responsiveness, leading to fatigue, orthostatic symptoms, and poor tolerance to upright posture. These effects are often compounded by psychological stress, travel, and dehydration, raising OI risk even in otherwise healthy athletes [6].

Although OI in athletes has only recently gained attention, early evidence supports its clinical relevance. For example, Petracek et al. (2022) described elite swimmers with prolonged fatigue and inconsistent performance who showed abnormal postural responses, such as postural tachycardia and borderline cortisol levels. These observations make it clear that a better understanding of how adrenal function affects orthostatic regulation in high-performing athletes is needed [2].

This article reviews the current understanding of adrenal involvement in athletic OI, outlining key physiological mechanisms, clinical patterns, diagnostic tools, and management strategies to guide practitioners working with affected athletes.

2. Methods

This article is a narrative literature review. We searched electronic databases including PubMed, Scopus, and Google Scholar using combinations of keywords such as "orthostatic intolerance," "adrenal glands," "athletes," "cortisol," and "aldosterone." The search focused on articles published in the last 10 years, prioritizing peer-reviewed studies in English. Eligible publications included clinical trials, observational studies, case series, and review articles relevant to endocrinology, autonomic function, and sports physiology. No formal quality assessment or data extraction protocol was used, as this is a narrative synthesis intended to summarize and contextualize current knowledge.

3. Adrenal Gland Physiology and Its Role in Cardiovascular Regulation

The adrenal glands, positioned above each kidney, are integral to the body's stress response and cardiovascular homeostasis. They are comprised of two distinct functional units: the adrenal cortex and adrenal medulla, each synthesizing hormones essential for maintaining vascular tone, plasma volume, and metabolic balance, especially during physiological challenges such as orthostatic stress [7].

3.1. Adrenal Cortex and Medulla: Functional Overview

The adrenal cortex produces steroid hormones, including glucocorticoids (mainly cortisol), mineralocorticoids (primarily aldosterone), and androgens. Among these, cortisol and aldosterone are key regulators of blood pressure and electrolyte balance. In contrast, the adrenal medulla secretes catecholamines—epinephrine and norepinephrine—in response to sympathetic nervous system activation [4, 7].

These two parts work within a tightly controlled feedback loop regulated by the hypothalamic-pituitary-adrenal (HPA) axis. When a postural change occurs, such as standing, the resulting drop in venous return and arterial pressure is quickly offset by sympathetic activation and adrenal hormone release to maintain adequate blood flow to the brain [1, 4].

3.2. Role of Cortisol in Cardiovascular Regulation

Cortisol plays several important roles in cardiovascular function. It enhances the vasoconstrictive effects of catecholamines by increasing adrenergic receptor expression on vascular smooth muscle, helping to maintain vascular tone during orthostatic stress. Additionally, cortisol supports plasma volume by upregulating angiotensin II receptors and influencing renal sodium retention through its permissive effects on aldosterone pathways [1].

In athletes, ongoing physical stress and intense training can alter HPA axis sensitivity, potentially disrupting normal cortisol responses. Reduced cortisol activity has been associated with postural symptoms and may contribute to the development of orthostatic intolerance in individuals experiencing overtraining or overreaching [4, 6].

3.3. Aldosterone and Sodium Balance

Aldosterone, the primary mineralocorticoid, regulates sodium and water retention by acting on the kidney's distal nephron. In response to posture-induced hypovolemia, the renin-angiotensin-aldosterone system (RAAS) is activated, increasing aldosterone secretion to restore plasma volume and blood pressure [3, 7]. Inadequate aldosterone response may lead to reduced circulating volume, orthostatic hypotension, and syncope [3].

Recent studies suggest that endurance athletes may have a suppressed aldosterone response to orthostatic stress, likely due to adaptations from chronic plasma volume expansion associated with training. This diminished hormonal response can weaken the body's ability to compensate during postural changes, increasing the risk of orthostatic intolerance [2, 3].

3.4. Catecholamines and the Acute Response to Posture

The adrenal medulla's release of epinephrine and norepinephrine is vital for the immediate compensatory response to upright posture. These catecholamines stimulate increased heart rate (positive chronotropy), myocardial contractility (positive inotropy), and systemic vasoconstriction, collectively preventing gravitational blood pooling [1, 7].

Norepinephrine, in particular, plays a key role in restoring vascular resistance in the lower extremities. A rapid and adequate catecholamine response is crucial for avoiding symptoms of OI such as dizziness, fatigue, and near-syncope during postural changes. Dysautonomia or impaired adrenal medulla responsiveness may reduce this protective effect, especially under conditions of heat stress, dehydration, or fatigue—common in athletic settings [2, 8].

3.5. Integration with the Autonomic Nervous System

Adrenal hormones work closely with the autonomic nervous system (ANS) rather than acting alone. When a person stands, baroreceptors in the carotid sinus and aortic arch sense the drop in arterial pressure and activate the sympathetic nervous system. This triggers both neural responses—such as direct vasoconstriction—and hormonal effects through the release of adrenal catecholamines [1, 7].

In athletes, training can shift autonomic balance, altering baroreflex sensitivity and the responsiveness of the HPA axis. While regular endurance exercise often increases resting parasympathetic activity, it may also dampen sympathetic responses, which could contribute to orthostatic intolerance in highly trained individuals [4, 5, 6].

4. Pathophysiology of Orthostatic Intolerance in Athletes

Orthostatic intolerance (OI) in athletes results from a combination of cardiovascular stress, neuroendocrine changes, and autonomic imbalance. Unlike patients with clear cardiovascular or neurological disorders, athletes typically develop OI due to subtle shifts in their regulatory systems, influenced by intense training, dehydration, and increased metabolic demands. Key factors driving OI symptoms in this group include reduced blood volume, autonomic dysfunction, and hormonal imbalances—especially involving adrenal gland function [1- 4].

4.1. Hypovolemia and Relative Fluid Deficiency

A key factor contributing to orthostatic intolerance in athletes is relative hypovolemia. Although trained individuals often adapt by increasing plasma volume, acute fluid losses from sweating, heat exposure, or inadequate rehydration can temporarily reduce central blood volume. Endurance athletes are particularly vulnerable to these changes, as extended exercise in hot conditions can cause significant water and electrolyte depletion, often without obvious symptoms [3, 5]. Additionally, chronic undernutrition or low energy availability, as seen in Relative Energy Deficiency in Sport (RED-S), may impair fluid retention by affecting aldosterone secretion or responsiveness, increasing the risk of orthostatic symptoms [6, 9].

4.2. Autonomic Dysfunction in Trained Individuals

Athletes experience significant autonomic adaptations, including increased parasympathetic tone and lower resting heart rates. However, excessive vagal dominance or impaired sympathetic recruitment during orthostatic stress can blunt compensatory responses. This is especially seen in postural orthostatic tachycardia syndrome (POTS), which has been reported in otherwise healthy endurance athletes [1, 8]. POTS involves an excessive rise in heart rate upon standing without major drops in blood pressure, often accompanied by symptoms like fatigue, dizziness, and palpitations.

Vasovagal syncope (VVS), another form of OI, involves inappropriate vagal activation and sympathetic withdrawal, resulting in sudden bradycardia and hypotension. Although common in the general population, VVS in athletes may present with atypical features, such as occurring during post-exertional recovery rather than during emotional or painful stimuli [10]. These manifestations may be overlooked or misinterpreted as benign overexertion unless formally tested with tilt-table evaluations or autonomic profiling [11].

4.3. Hormonal Dysregulation and Adrenal Involvement

Hormonal regulation, particularly through the adrenal cortex, is essential for maintaining vascular tone, plasma volume, and cardiovascular stability during changes in posture. In athletes, however, intense or prolonged training can suppress the hypothalamic-pituitary-adrenal (HPA) axis, resulting in insufficient cortisol or aldosterone release during orthostatic stress [4, 5, 12]. This is especially common in states of overreaching or overtraining, where blunted cortisol responses to stressors like ACTH stimulation or standing tests have been observed [1, 12].

Cortisol enhances vascular sensitivity to catecholamines and supports sodium retention by influencing mineralocorticoid pathways [7,12]. Athletes with low cortisol levels or reduced stress responsiveness may experience orthostatic hypotension due to inadequate vasoconstriction. Similarly, an impaired aldosterone response—whether from chronic training effects or reduced renin activity—can worsen plasma volume loss and hinder orthostatic compensation [6, 12]. Recent findings also suggest that adrenal responsiveness may remain impaired even when baseline hormone levels appear normal, underscoring the value of dynamic testing over static hormone measurements [1, 12].

4.4. Comparative Features of OI Subtypes in Athletes

Distinguishing among different types of orthostatic intolerance—such as vasovagal syncope (VVS), postural orthostatic tachycardia syndrome (POTS), and orthostatic hypotension (OH)—is important in athletes due to their overlapping yet distinct underlying mechanisms. VVS primarily involves reflex-driven vasodilation and slowed heart rate, often preceded by warning signs like nausea and visual disturbances [10]. In contrast, POTS is defined by a significant heart rate increase (over 30 bpm within 10 minutes of standing) without a drop in blood pressure, with symptoms that tend to be chronic and limit exercise capacity [1, 8].

Though less commonly reported in athletes, OH can occur due to delayed blood pressure recovery from impaired baroreceptor function or reduced adrenal response to the renin-angiotensin-aldosterone system [13]. This condition is especially problematic during dehydration or rapid standing after intense exercise. Given that athletes may present atypically, accurate diagnosis often requires specialized testing, including tilt-table exams, plasma volume measurement, and dynamic hormonal assessments [11, 13].

5. Impact of Orthostatic Intolerance on Athletic Performance and Diagnostic Approaches

Orthostatic intolerance (OI) can significantly impair sports performance and overall athlete health, particularly in high-performance and endurance disciplines. While its symptoms may initially appear mild or intermittent, their cumulative effects—diminished exercise capacity, reduced focus, and delayed recovery—can result in tangible performance decline and elevated health risks over time. Accurate diagnosis remains challenging, especially when athletes appear otherwise fit and healthy, necessitating targeted clinical evaluation and hormonal assessments to uncover underlying dysregulation [2, 3, 4, 11, 14].

5.1. Performance Impairment and Health Consequences

The main symptoms of OI—dizziness, tachycardia, lightheadedness, and fatigue—may worsen with heat, dehydration, or prolonged standing, all of which are common stressors in athletic environments. These symptoms impair training intensity and duration, particularly during standing drills, post-exercise cooldowns, or prolonged low-intensity activity. Athletes may struggle to maintain upright posture during key moments of training, such as tactical briefings or between-set rest, resulting in reduced physical output and suboptimal performance adaptation.

Endurance athletes are particularly vulnerable due to their high-volume training and frequent exposure to orthostatic challenges. Studies show that postural symptoms in endurance-trained individuals can manifest as decreased stroke volume and exaggerated heart rate response, reducing cardiovascular efficiency during transitions from rest to activity [4, 11]. When orthostatic compensation fails, athletes may experience transient cerebral hypoperfusion, impairing concentration and coordination, thereby increasing the risk of injury during technical movements or during rapid positional changes.

Additionally, orthostatic intolerance may affect psychological resilience and quality of life. Chronic fatigue, perceived exertion, and a sense of "training intolerance" can contribute to decreased motivation, burnout, and even withdrawal from sport [4, 15]. A study by Vernau et al. (2018) found that athletes with autonomic dysfunction reported significantly higher levels of anxiety, disrupted sleep, and reduced social participation—all of which are linked to impaired athletic longevity and performance outcomes [16].

5.2. Clinical Insights and Case-Based Evidence

Recent case studies are beginning to reveal how OI, though often subtle, can meaningfully affect competitive athletes. A 2021 study of collegiate athletes experiencing unexplained performance decline found that nearly 30% showed abnormal responses to standing, such as sharp increases in heart rate or drops in systolic blood pressure. Notably, these athletes had no clear signs of cardiovascular or endocrine disorders, suggesting that underlying autonomic or adrenal dysfunction may be present but easily overlooked without targeted evaluation [2].

OI symptoms have also been reported in athletes recovering from viral illnesses or periods of overtraining. Post-viral conditions like POTS, for example, have been observed following COVID-19, marked by ongoing fatigue, high resting heart rate, and difficulty returning to pre-illness training levels. These observations indicate the need for early recognition and tailored management strategies to support athlete recovery and performance [17].

5.3. Diagnostic Approaches in Athletes

Given the overlap between OI symptoms and common athletic complaints (e.g., fatigue, poor recovery), diagnosis requires careful clinical attention and a systematic approach The first step is a detailed clinical history, focusing on symptom patterns relative to posture, hydration status, training phases, and recent illnesses or stressors. A review of menstrual function, nutrition, and energy availability is especially important in female and adolescent athletes, where RED-S and associated hormonal suppression may play a role [6].

Objective testing is essential for confirmation. Active standing tests and tilt-table protocols remain the gold standards for evaluating postural hemodynamic responses. In athletes, these tests should ideally be performed in the morning, in a fasted and rested state, to reduce confounding factors. Measurements include heart rate and blood pressure changes at rest and during upright posture, with criteria such as a ≥30 bpm increase in heart rate (in the absence of hypotension) supporting a diagnosis of POTS [8].

Hormonal evaluation can further clarify adrenal involvement. Morning serum cortisol or responses to ACTH stimulation tests may reveal HPA axis dysregulation or adrenal insufficiency. Blunted cortisol responses have been observed in athletes with overreaching or persistent orthostatic symptoms [12]. Likewise, assessing aldosterone and renin levels—especially when contextualized with electrolyte status and plasma volume—can help identify insufficient RAAS activation, another contributor to orthostatic vulnerability [3].

The complexity of hormonal and autonomic interactions often necessitates a multidisciplinary approach. Sports medicine physicians, endocrinologists, and exercise physiologists can work together to distinguish between normal adaptations to training and underlying dysfunction. This collaborative evaluation supports personalized management strategies—ranging from training adjustments and hydration optimization to endocrine correction and, when appropriate, pharmacologic intervention [6, 9].

6. Management Strategies for Orthostatic Intolerance Related to Adrenal Function

Effective management of OI in athletes—especially when adrenal involvement is a factor—relies on a comprehensive approach. This includes lifestyle changes, individualized training modifications, targeted medical treatment, and athlete education. The aim is to support physiological function and performance while reducing symptoms and maintaining training continuity [2, 4, 18].

6.1. Non-Pharmacological- Interventions

Hydration and Salt Supplementation

Maintaining proper hydration is fundamental to managing orthostatic intolerance in athletes. Fluid intake should include electrolytes and be timed around training sessions—before, during, and after—particularly in hot or humid environments. Increasing sodium intake, typically by an additional 3 to 5 grams per day beyond baseline dietary needs, can help expand plasma volume and support vascular stability during postural changes. Hydration and salt strategies should be personalized based on factors like sweat rate, body weight fluctuations, and symptom patterns [2, 18].

Compression Garments and Stockings

Graduated compression garments—worn on the calves, thighs, or full lower body—help reduce venous pooling and enhance venous return to the heart. By supporting circulatory stability during changes in posture, these garments can alleviate symptoms such as dizziness and fainting. It is important for athletes to be properly fitted for compression levels, typically between 20 and 30 mmHg, to ensure both effectiveness and comfort, as excessive compression may reduce compliance [14].

Physical Counter-Maneuvers

Athletes should be taught simple physical maneuvers to use at the first sign of symptoms. Techniques such as leg crossing, buttock clenching, or isometric abdominal contractions help increase venous pressure and enhance cerebral blood flow while standing. These easy-to-perform strategies can be applied during training breaks or in classroom settings, offering quick symptom relief without relying on medication [19].

6.2. Pharmacological Options

Once non-pharmacological methods are maximized, medication may be considered—particularly for athletes with significant symptoms or adrenal-related hormonal insufficiencies [10].

Fludrocortisone

A synthetic mineralocorticoid, fludrocortisone enhances sodium retention and expands plasma volume, making it a first-line option for OI due to aldosterone insufficiency or hypovolemia. Doses range from 0.1 to 0.3 mg daily, titrated to symptom relief and monitored for elevated blood pressure or electrolyte changes. While generally well tolerated, potential side effects include supine hypertension, hypokalemia, and peripheral edema [10, 20].

Midodrine

Midodrine, an alpha-1 agonist, increases arterial and venous tone, counteracting orthostatic blood pressure drops. It is particularly useful when volume expansion is inadequate to control symptoms. Common regimens include 2.5–10 mg taken two to three times daily during waking hours. Side effects can include itching, urinary retention, and hypertension; dosing should avoid evening intake to reduce supine hypertension risk [10, 11, 13].

Other Agents

In select cases, low-dose pyridostigmine, clonidine, or beta-blockers may be considered to address autonomic dysfunction or excessive sympathetic activity. However, these are less commonly used in athletes due to effects on heart rate and exercise capacity [13, 17].

6.3. Training and Conditioning Modifications

Training modifications are an important aspect of managing OI in athletes, complementing medical approaches. Certain phases—like early-season conditioning, heat-acclimatization camps, or times when sleep and nutrition are disrupted—can increase vulnerability to symptoms. Key strategies observed include:

- ***Gradual Load Progression:*** Increasing upright and endurance training volume step-by-step helps strengthen cardiovascular reflexes essential for maintaining blood pressure during postural changes [14, 15].
- ***Inclusion of Supine or Prone Exercises:*** Cross-training activities such as swimming, cycling, and rowing, along with supine core work on rest days, allow athletes to maintain fitness while minimizing orthostatic stress [3, 14].
- ***Focused Strength Training:*** Exercises targeting the legs and core improve venous return and support baroreflex function, enhancing overall orthostatic stability [3, 4].

Ongoing monitoring of resting heart rate, postural blood pressure, and symptom fluctuations during transitions in training can provide early warning signs of maladaptation. This enables timely adjustments before symptoms worsen, supporting better long-term outcomes for affected athletes [6].

6.4. Education for Athletes and Coaches

Education is fundamental to the long-term management of OI in athletes, ensuring that both athletes and coaching staff understand the condition and its implications. Key educational points include:

- ***Physiological Basis of OI:*** Symptoms such as dizziness and fatigue reflect genuine physiological responses to systemic stress rather than a lack of effort or motivation [2, 17].
- ***Emphasis on Prevention:*** Maintaining adequate hydration, sodium intake, and the use of compression garments before training sessions can significantly reduce the frequency and severity of mild to moderate OI episodes [10, 11].
- ***Importance of Self-Monitoring:*** Encouraging athletes to document symptoms alongside training variables enables early identification of issues and allows for tailored management strategies [6].
- ***Emergency Response Preparedness:*** Teams should establish clear protocols for acute symptom management, including positions for symptom relief like lying down, leg elevation, and cooling techniques when appropriate [11, 16].

By educating coaches and support personnel, athletes are more likely to receive appropriate recognition and support, reducing the risk of misinterpretation as laziness or lack of commitment and alleviating associated psychological stress [2, 16].

7. Discussion

Orthostatic intolerance (OI) in athletes represents a distinct physiological challenge, arising not from overt disease but from subtle disruptions in the systems that regulate blood pressure, fluid balance, and stress adaptation during postural changes [11]. This review underscores the central role of adrenal gland function—particularly the secretion of cortisol, aldosterone, and catecholamines—in maintaining orthostatic stability under the unique demands of athletic performance.

Unlike classical OI seen in clinical populations, athletic OI develops within a background of high physical fitness and adaptive neuroendocrine changes. Chronic training, especially when combined with dehydration, low energy availability, or heat exposure, may impair adrenal responsiveness or autonomic compensation [6, 10, 19]. The result is a functional vulnerability to orthostatic stress, manifesting as dizziness, fatigue, tachycardia, or cognitive impairment during training, competition, or recovery [2, 21].

A key insight from this review is the difficulty of diagnosing OI in athletic populations. Symptoms are often non-specific and easily misattributed to overtraining, deconditioning, or anxiety [16]. Moreover, standard endocrine tests may fail to detect dynamic deficits, as resting hormone levels may remain within normal ranges. The evidence supports the use of dynamic assessments—such as tilt-table testing, standing tests, or ACTH stimulation—to evaluate real-time adrenal and autonomic function [1, 12].

Unmanaged, these orthostatic symptoms can hinder training consistency, reduce competitive readiness, and negatively affect psychological well-being. Athletes with undiagnosed OI may experience prolonged recovery, decreased motivation, and increased injury risk due to reduced cerebral perfusion or neuromuscular instability during activity. Importantly, the condition is manageable when properly recognized and treated [2, 22].

This review supports the need for a more proactive and individualized approach. Non-pharmacological interventions—such as fluid and sodium optimization, compression therapy, and positional countermeasures—should be implemented early, especially in athletes with known risk factors [22]. Pharmacologic support, including fludrocortisone or midodrine, may be appropriate in more resistant cases [23]. Just as crucial is athlete and coach education: understanding that OI symptoms reflect true physiological strain rather than lack of conditioning can improve compliance and reduce stigma [22].

In summary, orthostatic intolerance in athletes should be viewed as a modifiable barrier to performance and health. Recognizing adrenal function as a key contributor provides a foundation for more targeted diagnostics and interventions that support long-term athletic success [6, 19].

8. Future Directions and Research Gaps

Significant progress has been made in understanding OI within clinical populations, but research focused specifically on athletes remains rare. This gap is particularly pronounced when examining the role of adrenal function in orthostatic regulation. Athletes undergo unique physiological adaptations that can mask or complicate classical markers of hormonal imbalance. Therefore, future research must carefully consider these complexities to develop diagnostic, management, and prevention strategies tailored to athletic populations [1, 2].

A key challenge in this area is distinguishing between adaptive and maladaptive hormonal changes. For example, endurance training often leads to blunted cortisol responses due to habituation of the HPA axis, which can resemble or conceal true adrenal insufficiency [4]. Likewise, diminished aldosterone responses seen in well-trained athletes may reflect an adaptive expansion of plasma volume rather than a pathological hormone deficiency. Differentiating these physiological adaptations from early indicators of dysfunction requires rigorous studies designed specifically for elite and recreational athletes, assessing both resting conditions and responses to orthostatic stress [3].

Longitudinal cohort studies are particularly important for tracking how adrenal hormone dynamics evolve across various training phases—pre-season, competition, and recovery—and under different influences such as workload, hydration status, and psychological stress. Such data would help identify early biomarkers of maladaptive adrenal activity. Potential indicators include altered cortisol-to-ACTH ratios, flattened diurnal cortisol rhythms, or reduced aldosterone responses during tilt testing, which may precede overt OI symptoms [4, 5].

The search for new biomarkers also presents a promising frontier. Beyond standard hormone measurements, emerging markers like copeptin (a surrogate marker for vasopressin), plasma metanephrines, and salivary cortisol profiles might offer more sensitive insight into neuroendocrine stress and fluid regulation in athletes. The use of wearable biosensors to monitor heart rate variability, hydration status, and orthostatic responses in real time during training or competition could further enhance early detection of OI-related impairments [14, 19].

Sex differences in adrenal regulation and OI presentation represent another important area for investigation. Female athletes may experience distinct HPA axis and RAAS responses influenced by factors such as menstrual cycle phase, contraceptive use, or energy availability. Tailored research focusing on these variables could improve screening accuracy and intervention strategies, ensuring more equitable care [6, 9].

Ultimately, future research should aim to advance a personalized approach to managing OI in athletes. This involves establishing normative adrenal hormone reference ranges stratified by sport type, training volume, and sex, and developing athlete-specific diagnostic frameworks that combine hormonal profiles, autonomic function testing, and clinical assessment [8, 23].

Personalized prevention and management could include regular endocrine monitoring during periods of increased physiological stress, individualized hydration and sodium supplementation plans, and training modifications informed by biomarker trends. Achieving these goals will require collaborative efforts bridging endocrinology, sports medicine, exercise physiology, and wearable technology development to translate research findings into practical applications [8, 19, 23].

In conclusion, addressing current gaps in knowledge demands athlete-focused, multifaceted studies that integrate hormonal, autonomic, and performance data. Identifying early biomarkers and refining diagnostic and therapeutic approaches tailored to the athletic physiology will be key to improving both scientific understanding and clinical care of orthostatic intolerance in sports settings [1, 2].

9. Conclusions

Orthostatic intolerance (OI) in athletes, although frequently underrecognized, poses a significant challenge to both health and athletic performance. This condition arises from a complex interaction among autonomic regulation, adrenal hormone activity, and cardiovascular stability, demanding a deeper understanding than what traditional clinical frameworks often provide. As discussed throughout this review, adrenal hormones such as cortisol and aldosterone are vital for maintaining orthostatic stability by regulating vascular tone, fluid balance, and sympathetic nervous system responsiveness. Dysregulation in these hormonal and autonomic systems—whether resulting from overtraining, insufficient recovery, or energy deficits—can manifest as symptoms including dizziness, fatigue, cognitive difficulties, and decreased training capacity.

Despite the physiological adaptations associated with athletic conditioning, athletes remain vulnerable to OI due to the distinctive stresses imposed by intense training, heat exposure, and metabolic demands. Current research highlights the importance of specialized diagnostic approaches, including structured orthostatic testing and adrenal hormone evaluation, especially in athletes presenting with relevant symptoms. Furthermore, effective management necessitates a personalized approach that integrates non-pharmacological strategies, careful pharmacologic interventions when indicated, and thoughtful adjustment of training regimens.

Looking ahead, advancing this field requires the establishment of athlete-specific diagnostic biomarkers, longitudinal research on adrenal function throughout training and recovery phases, and the development of evidence-based prevention strategies. Incorporating adrenal health assessments into routine sports medicine evaluations could enhance early detection, improve recovery protocols, and support sustained athletic performance over time.

A multidisciplinary approach—uniting sports physicians, endocrinologists, physiologists, and coaching staff—is essential to address the multifactorial nature of OI Increasing awareness and understanding will provide opportunities to reframe this often-misunderstood condition as a manageable aspect of comprehensive athlete care and optimization.

Disclosure

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