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DIAGNOSTIC UNCERTAINTY IN SPORTS MEDICINE: CHALLENGES IN THE INTERPRETATION OF CORTISOL TESTING

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ABSTRACT

Background: Cortisol is a key hormone of the hypothalamic-pituitary-adrenal (HPA) axis and plays an essential role in the stress response. In physically active individuals, exercise-induced cortisol elevations are common; however, cortisol testing is often used in the evaluation of Overtraining Syndrome (OTS) despite uncertain diagnostic value and potential for misinterpretation in clinical practice.

Material and methods: A structured literature search of PubMed and Google Scholar (1998–2025) was conducted using terms related to cortisol, exercise, overtraining, hypercortisolism and HPA-axis function. Human studies, systematic reviews, meta-analyses and major endocrine guidelines were included.

Results: Cortisol responses to exercise are highly variable and influenced by training load, intensity, duration and individual factors. In athletes with suspected OTS, resting and dynamic cortisol measurements show inconsistent results with significant overlap compared to healthy athletes and non-athletic controls. Diagnostic accuracy is further limited by methodological heterogeneity, diurnal variation and biological variability. Numerous non-training factors, including sleep disturbance, psychological stress, illness, medications and endocrine disorders, can elevate cortisol to levels similar to exercise-related responses, increasing diagnostic uncertainty.

Conclusions: Cortisol lacks sufficient specificity and sensitivity to serve as an independent diagnostic marker of OTS. Elevated cortisol in athletes should be interpreted within a comprehensive clinical context. Cortisol testing may provide supportive information but should not be used in isolation to diagnose overtraining or guide clinical or training-related decisions.

KEYWORDS

Cortisol Testing, Overtraining Syndrome, Sports Medicine, Hypercortisolism, Physically Active Individuals, Stress Response

CITATION

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Introduction:

Cortisol is the main glucocorticoid produced by the adrenal cortex and plays a central role in the body's response to physical and psychological stress. In athletes and physically active individuals, short-term rises in cortisol during or after exercise represent a normal physiological activation of the hypothalamic-pituitary-adrenal (HPA) axis. (Al-Khanaty et al., 2025) Although cortisol testing is sometimes incorporated into the clinical evaluation of athletes, current evidence shows that resting cortisol levels lack diagnostic utility for identifying Overtraining Syndrome (OTS). (Cadejani & Kater, 2017b)

Despite its association with hormonal disturbances, OTS remains challenging to define and diagnose. The syndrome has no standardized diagnostic criteria, validated biomarkers or consistent hormonal profile. (Armstrong et al., 2022) While some studies report changes in HPA axis regulation among affected athletes (Carfagno & Hendrix, 2014), many others demonstrate cortisol values within expected physiological ranges. As a result, elevated cortisol is often interpreted as a sign of excessive training, even though studies show that cortisol responses vary widely and do not reliably distinguish OTS from normal training adaptations. ("Prevention, Diagnosis, and Treatment of the Overtraining Syndrome," 2013)

In practice, numerous non-training-related conditions can increase circulating cortisol. These include endogenous hypercortisolism, such as Cushing syndrome (Nieman & Ilias, 2005), as well as pseudo-Cushing states (Scaroni et al., 2020) related to chronic stress, depression, anxiety, or chronic alcohol use. (Alwani et al., 2014) Lifestyle factors, including sleep deprivation, shift work and acute illness, can also cause temporary cortisol elevations. (Grosser et al., 2022) Estrogen therapy, especially oral preparations, is known to increase cortisol-binding-globulin (CBG) and total serum cortisol, while glucocorticoid treatment may further alter

cortisol dynamics. (Qureshi et al., 2007) Because many of these conditions share key symptoms with OTS, such as fatigue, mood changes, reduced motivation, or impaired performance, the risk of misinterpretation is considerable.

Given the growing use of hormonal testing in sports medicine and the clinical ambiguity of OTS, the necessity of distinguishing physiological cortisol responses from pathological hypercortisolism has become increasingly relevant. (Snyder & Hackney, 2013) Mislabeling cortisol elevation to excessive training may delay recognition of serious medical conditions, while relying on isolated results may lead to unnecessary modifications of training load or inappropriate clinical decisions.

In contemporary sports medicine, laboratory testing increasingly influences clinical and training-related decisions. When biomarkers are overinterpreted or considered without sufficient context, the risk of diagnostic error increases. Understanding the limits of cortisol testing is therefore not only a physiological issue but also a matter of clinical decision quality.

Methodology:

A structured search was conducted in PubMed and Google Scholar for publications from 1998 to 2025, using the keywords: “cortisol”, “exercise”, “hypercortisolism”, “overtraining”, “pseudo-Cushing” and “HPA axis”. Medical Subject Headings (MeSH) were used when applicable.

Included studies examined causes of elevated cortisol in adults and provided information relevant to distinguish training-related cortisol changes from medical, psychological or lifestyle-related conditions. Peer-reviewed human studies, systematic reviews, meta-analyses, and major endocrine society guidelines were included, while case reports and animal studies were excluded.

Titles and abstracts were screened, followed by full-text review. Additional sources were identified through reference screening and key findings were summarized to identify factors capable of increasing cortisol independently of physical training.

Results:

1. Physiological cortisol response to exercise

Acute physical exercise remains one of the strongest activators of the HPA axis. Numerous studies demonstrate that cortisol rises immediately during or after exercise, with the magnitude depending on intensity, duration and training status. This response appears both in serum and saliva and usually normalizes within a few hours after training. (Duclos et al., 1998) Resistance-training protocols show comparable post-exercise increases in salivary cortisol, confirming that temporary elevations represent a normal physiological stress response. (Bermejo et al., 2022)

Observational data also reveal substantial individual variation: women and older individuals display different basal cortisol rhythms compared to men and younger adults. (Larsson et al., 2009) Overall, these findings indicate that a broad range of cortisol values can be physiologically normal. Recognizing this natural variability is essential for accurate interpretation of cortisol measurements in active populations.

2. Cortisol in athletes with suspected overtraining

Findings in athletes evaluated for suspected OTS remain inconsistent. A systematic review by Cadegiani & Kater found that most symptomatic athletes had basal cortisol concentrations within the normal range, with only a minority showing clear elevations or suppression. (Cadegiani & Kater, 2017a) This indicates that resting cortisol lacks sensitivity and specificity for identifying OTS.

Moreover, a controlled assessment of the HPA axis reactivity using dynamic tests (e.g. insulin-tolerance test, cosyntropin stimulation) has shown blunted responses in some OTS cases, but their hormonal values overlap substantially with those of healthy athletes and non-active controls. (Cadegiani & Kater, 2017b)

Long-term studies monitoring cortisol levels across training cycles highlight substantial individual variability. In endurance athletes, cortisol levels may remain relatively stable or show modest fluctuations in response to changes in training load, underscoring the absence of a consistent hormonal pattern associated with OTS.

Taken together, current evidence indicates that cortisol – whether measured at rest, after stimulation, or over time – is not a reliable diagnostic marker of overtraining.

3. High variability across studies – methodological, biological, and contextual limitations

Interpretation of cortisol in athletes is complicated by heterogeneous study methodologies. One important source of inconsistency is the biological material used for testing. Serum, saliva and urine represent different aspects of cortisol physiology. Serum reflects mostly total cortisol, which is strongly influenced by cortisol-binding globulin, saliva measures only free cortisol and urinary free cortisol shows overall production across 24 hours. Because these measures behave differently, direct comparison between studies is limited. (El-Farhan et al., 2017)

Cortisol also varies widely across the day, with its highest values in the early morning and lowest late in the evening. Even small differences in sampling time can alter results. This is evident in studies on the cortisol awakening response, which show highly mixed findings in athletes, highlighting the hormone's sensitivity to timing and routine. (Anderson & Wideman, 2017)

Exercise protocols further contribute to variability. Intensity, duration, recovery time and environmental conditions, such as heat and dehydration, can all increase cortisol independently of actual training load.

Finally, day-to-day cortisol levels are influenced by personal and contextual factors, such as sleep quality, psychological stress, travel fatigue, nutrition and hormonal fluctuations. For example, female basketball players show altered diurnal cortisol patterns during periods of elevated physical and mental load. (Sánchez et al., 2021) Sleep quality and autonomic balance affect cortisol responses during training, and menstrual cycle phase can also modify hormonal reactions to exercise. (Mirzaei Khalil Abadi et al., 2024)

4. Lifestyle and psychological factors influencing cortisol levels

A range of non-training factors can elevate cortisol to levels overlapping with typical post-exercise elevations. Sleep restriction and circadian disruption are among the most influential non-training stressors, with controlled laboratory studies showing that even a single night of partial sleep restriction increases evening cortisol levels and disrupts diurnal regulation, confirming acute activation of the HPA axis. (O'Byrne et al., 2021) Meta-analytic evidence confirms that sleep loss and shiftwork alter cortisol rhythms, although effects on the morning peak remain inconsistent and depend on sampling protocol and matrix. (Grosser et al., 2022) Psychological stress, anxiety and depressive symptoms activate the HPA axis and may produce hormonal patterns resembling „pseudo-Cushing” states described in endocrine guidelines. In athletes, higher levels of chronic psychological stress have been associated with altered CAR and higher evening cortisol in some studies. (Scaroni et al., 2020) Alcohol consumption – particularly chronic or heavy use – modifies HPA function and is linked with altered basal and stress-responsive cortisol concentrations in clinical and experimental studies. (Starcke et al., 2013)

Acute illness, injury or systemic inflammation also provoke temporary cortisol elevations. Pro-inflammatory cytokines (notably IL-6) directly stimulate ACTH and adrenal steroidogenesis, increasing circulating cortisol during infection or inflammatory states. (Päth et al., 2000) Because these conditions are common among athletes, they significantly reduce the specificity of cortisol as an indicator of training-related stress.

5. Medical causes of elevated cortisol relevant to athletes

Several medical conditions may result in cortisol elevations that could be misinterpreted as consequences of overtraining. Endogenous hypercortisolism, including mild, early or cyclical forms of Cushing syndrome, often present initially with nonspecific symptoms such as fatigue, mood changes, impaired recovery and reduced performance. These may precede the development of recognizable Cushingoid features by many months. (Nieman & Ilias, 2005)

Oral estrogen therapy substantially increases cortisol-binding globulin (CBG), leading to proportional elevations in total serum cortisol while leaving free cortisol and salivary cortisol unaffected. This effect depends on the route of administration (oral > transdermal). (Qureshi et al., 2007) Systemic glucocorticoids clearly alter endogenous cortisol secretion. Inhaled and intranasal corticosteroids can also suppress the HPA axis, particularly with prolonged use or higher cumulative doses, complicating cortisol interpretation. (Rao Bondugulapati & Rees, 2016); (Cavkaytar et al., 2015)

Finally, energy-balance disorders frequently observed in athletes, such as low energy availability and Relative Energy Deficiency in Sports (RED-S), are linked to changes in HPA-axis regulation, with studies reporting elevated cortisol or altered cortisol ratios. As a result, these conditions may significantly influence cortisol measurements independent of training load. (Torstveit et al., 2019)

6. Clinical implications for cortisol interpretation in athletes

The considerable overlap between physiological variability, lifestyle-related influences and underlying medical conditions substantially limits the diagnostic value of isolated cortisol measurements in athletic populations. Current clinical guidelines emphasize that cortisol should always be interpreted within a broader clinical context, considering sleep patterns, recent travel, physiological stress, menstrual cycle, nutritional status and medication use before drawing conclusions. (Snyder & Hackney, 2013) Consequently, reliance on cortisol measurements alone to support a diagnosis of OTS may result in misclassification, inappropriate modifications of training load, or delayed recognition of clinically significant endocrine disorders. A structured clinical evaluation therefore remains essential when interpreting cortisol abnormalities in athletes.

Discussion:

The evidence summarized in this review indicates that cortisol, despite its central role in stress physiology, has limited utility as a marker of OTS in athletes. Rather than reflecting a single pathological process, cortisol represents a highly adaptive hormone that integrates signals from physical load, psychological stress, sleep-wake regulation and metabolic state. This complexity helps explain why cortisol responses observed in athletes frequently overlap between physiological adaptation, functional overreaching and suspected overtraining. By integrating perspectives from sports physiology and clinical endocrinology, this review aims to support a more structured clinical interpretation of cortisol measurements in athletes. Table 1 provides a structured overview of potential causes of cortisol elevation to support more systematic clinical evaluation.

Table 1. Structured overview of factors contributing to elevated cortisol and their diagnostic implications in sports medicine

Category	Examples	Typical clinical signs	Diagnostic considerations
<i>Training related factors</i>	Acute intense exercise, high training volume, insufficient recovery	Clear link with recent training load, temporary cortisol increase	Usually resolves with proper recovery; timing of sample collection is important
<i>Psychological stress</i>	Chronic stress, anxiety, competition pressure, poor sleep	Symptoms not fully explained by training intensity	Often overlooked; may occur together with training stress
<i>Lifestyle factors</i>	Caffeine intake, shift work, irregular sleep patterns	Disturbed daily rhythm, variable test results	Can strongly affect cortisol rhythm and test reliability
<i>Methodological factors</i>	Single measurement, incorrect sampling time, assay variability	Inconsistent or non-repeatable results	Repeated measurements and standardized procedures are recommended
<i>Medical conditions</i>	Cushing syndrome, pseudo-Cushing states, depression	Persistent elevation with additional systemic or mental symptoms	Require further endocrinological or psychiatric evaluation
<i>Medication-related factors</i>	Glucocorticoids, antidepressants, oral contraceptives	Relevant medication history, altered cortisol rhythm	Drug effects should always be considered
<i>Energy availability disorders</i>	Relative energy deficiency in sport (RED-S)	Fatigue, menstrual disturbances, metabolic changes	Cortisol elevation may reflect chronic metabolic stress

One important consideration emerging from recent literature is that cortisol responses in sport are not driven exclusively by training load. Competitive environments themselves act as potent stressors. Studies in elite team-sport athletes demonstrate that salivary cortisol rises after competitive matches even when physical workload is comparable to training sessions, indicating activation of the HPA axis by sport-specific psychological and situational stress rather than exercise alone (e.g. elite handball players). (Mariscal et al., 2019) This distinction is particularly relevant when interpreting cortisol measurements obtained around

competition periods, where elevations may reflect acute contextual stress rather than maladaptive training responses.

Beyond acute stressors, habitual physical activity also appears to shape cortisol regulation in complex ways. Recent meta-analytic data suggest that physically active individuals often show a more pronounced decline in cortisol levels over the course of the day, while the CAR response does not consistently differ according to activity level. (Moyers & Hagger, 2023) These findings underscore that regular training may alter circadian cortisol dynamics without producing clear pathological patterns. Consequently, single time-point measurements, even when standardized to morning sampling, may fail to capture meaningful differences between adaptive and maladaptive states.

The absence of a distinct cortisol profile in overtrained athletes is further supported by systematic reviews examining hormonal responses across overtraining, non-functional overreaching and related conditions. These reviews consistently conclude that no single endocrine marker, including cortisol, reliably distinguishes between these states. (Cadejani & Kater, 2017a) This reinforces the notion that hormonal alterations observed in OTS are subtle, heterogeneous and highly individual, limiting their diagnostic applicability in isolation.

Efforts to enhance diagnostic discrimination by assessing HPA-axis function with dynamic stimulation tests have not substantially overcome this limitation. While experimental studies employing procedures such as the insulin tolerance test indicate that cortisol responsiveness may differ in some athletes with suspected OTS compared with well-trained controls, the resulting values still show wide overlap between groups. (Cadejani & Kater, 2017b) From a clinical perspective, this overlap reduces the practical value of such tests for routine assessment and emphasizes the need for careful interpretation within a broader clinical framework.

Taken together, these observations highlight a central challenge in sports endocrinology: cortisol responses reflect an integrated stress signal rather than a specific marker of training maladaptation. While elevated or suppressed cortisol levels may raise suspicion of dysregulated stress physiology, they cannot reliably distinguish overtraining from normal adaptive responses, lifestyle influences or underlying medical conditions. This reinforces the importance of interpreting cortisol data alongside longitudinal training history, symptom progression and contextual factors, rather than relying on absolute values or isolated measurements. Current expert consensus therefore discourages the use of isolated hormonal measurements as diagnostic tools for overtraining and emphasizes longitudinal monitoring and clinical judgement. (“Prevention, Diagnosis, and Treatment of the Overtraining Syndrome,” 2013) As a narrative review, this work is limited by the heterogeneity of included studies and reliance on published data rather than primary measurements.

Misinterpretation of laboratory markers such as cortisol may lead not only to incorrect medical conclusions but also to inappropriate training modifications, unnecessary diagnostic procedures or delayed identification of underlying medical conditions. In sports medicine, where clinical decisions often influence both health and performance outcomes, reducing diagnostic uncertainty is essential. Greater emphasis on structured assessment, interdisciplinary communication and clinician education may improve the quality and safety of decision-making processes related to hormonal testing.

Conclusions

Cortisol responses in athletes reflect a complex integration of training load, psychological stress, circadian regulation, lifestyle factors and underlying medical conditions. Current evidence indicates that cortisol measurements, whether obtained at rest, after stimulation or over time, lack sufficient specificity and sensitivity to serve as an independent diagnostic marker of OTS. Substantial overlap exists between training-related adaptations, lifestyle-related stressors and medical causes of hypercortisolism, which limits the interpretability of single cortisol values.

In clinical practice, elevated cortisol levels in physically active individuals should therefore not be automatically attributed to excessive training. Interpretation of cortisol results requires consideration of training history, symptom evolution, sleep patterns, nutritional status, medication use and potential medical or endocrine disorders. Within this broader context, cortisol testing may contribute supportive information but should not be used in isolation to guide training decisions or establish a diagnosis of overtraining.

Future research should prioritize longitudinal, multi-parameter approaches that integrate hormonal, clinical and performance-related markers, rather than seeking a single biochemical indicator of training maladaptation. Until validated diagnostic tools become available, the evaluation of athletes with suspected stress-related performance decline should be guided primarily by individualized clinical assessment. For clinicians working with athletes, this underscores the need for interdisciplinary collaboration between sports

medicine and endocrinology. From a broader perspective, improving the interpretation of hormonal testing may contribute to more rational use of laboratory diagnostics in sports medicine and reduce the risk of unnecessary interventions.

Disclosure

Author's contribution

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