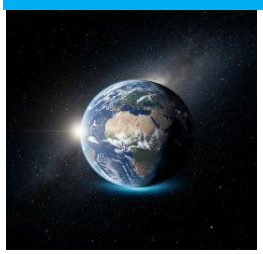
Quality in Sport. 2026;51:68663. eISSN 2450-3118.

https://doi.org/10.12775/QS.2026.51.68663



Quality in Sport. eISSN 2450-3118

Journal Home Page
https://apcz.umk.pl/QS/index

GLUZICKA, Anna, PALACZ, Bartosz, TEPER, Maria Magdalena, PERZ, Wiktor, POLUS, Aleksander, WRONA, Julia Anna, KUBIŚ, Natalia Marianna, OLCZYK, Liwia, PIOTROWSKI, Jędrzej, KOROLCHUK, Anhelina, BŁĄD, Karol Seweryn and GRĘBOSZ, Damian Jakub. Hypothalamic–Pituitary–Adrenal Axis Dysregulation and Its Impact on Exercise Capacity and Recovery in Regularly Training Individuals. Quality in Sport. 2026;51:68663. eISSN 2450-3118. https://doi.org/10.12775/QS.2026.51.68663

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 29.01.2026. Revised: 12.02.2026. Accepted: 12.02.2026. Published: 20.02.2026.

Hypothalamic–Pituitary–Adrenal Axis Dysregulation and Its Impact on Exercise Capacity and Recovery in Regularly Training Individuals

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ABSTRACT

Background:

Regular training imposes repeated physiological demands that activate neuroendocrine stress regulation, with the hypothalamic–pituitary–adrenal (HPA) axis playing a central role. Cortisol supports metabolic regulation and adaptation to exercise-related stress; however, prolonged exposure to high training loads combined with insufficient recovery may lead to functional alterations in HPA axis regulation, potentially affecting exercise capacity and recovery processes.

Aim:

This narrative review synthesizes current evidence on HPA axis dysregulation in regularly training individuals, with particular emphasis on its relevance for exercise capacity and recovery.

Materials and methods:

The review includes peer-reviewed experimental, observational, longitudinal, and sport-specific studies addressing HPA axis physiology, cortisol secretion dynamics, cortisol

awakening response (CAR), training load, fatigue, overreaching, overtraining, exercise capacity, and recovery in physically active populations.

Results:

Available evidence indicates that cumulative training stress is associated with changes in basal cortisol secretion, diurnal cortisol rhythm, and CAR. Functional alterations in HPA axis activity may influence metabolic efficiency, neuromuscular function, stress responsiveness, and performance stability. These responses appear to occur along an adaptive–maladaptive continuum and are strongly modulated by individual characteristics, training structure, and recovery adequacy. Importantly, many training-related disturbances of HPA axis function seem to be time-dependent and at least partially reversible.

Conclusions:

Viewing HPA axis dysregulation as a dynamic and functional process may improve interpretation of training responses, while longitudinal assessment of cortisol-related markers, interpreted within the broader training and recovery context, may provide valuable insight into individual stress adaptability in regularly training individuals.

Key words: hypothalamic–pituitary–adrenal axis, cortisol dynamics, cortisol awakening response, training stress, exercise capacity, recovery, overreaching, overtraining syndrome

1. Introduction

Regular physical training represents a potent physiological stressor that activates multiple neuroendocrine pathways involved in the maintenance of homeostasis. Among these systems, the hypothalamic–pituitary–adrenal (HPA) axis plays a central role in coordinating the organism's response to both acute and chronic stressors, including physical exercise. Activation of the HPA axis leads to the secretion of cortisol, a glucocorticoid hormone essential for energy mobilization, metabolic regulation, and modulation of immune and inflammatory processes [1]. In the context of exercise, cortisol release is considered a necessary component of adaptation, supporting the body's capacity to cope with increased energetic and mechanical demands [1,2].

Exercise-induced activation of the HPA axis is not inherently maladaptive. On the contrary, repeated exposure to training stress can result in favorable neuroendocrine adaptations, contributing to improved stress tolerance and performance capacity in athletes and physically active individuals [2,3]. However, when training load exceeds the individual's ability to recover

adequately, the balance between stress and adaptation may be disrupted. Prolonged or excessive training stress, insufficient recovery, and cumulative psychosocial demands can lead to functional alterations in HPA axis activity, reflected in changes in cortisol secretion patterns. Importantly, such dysregulation should not be equated with clinical endocrine pathology, but rather understood as a functional disturbance of adaptive stress regulation within the context of athletic training [1,4].

Growing evidence suggests that alterations in basal cortisol levels, diurnal cortisol rhythm, and cortisol responsiveness to stress are associated with impaired exercise capacity and compromised recovery in regularly training individuals [2, 5, 6]. In particular, the cortisol awakening response (CAR), a distinct component of the circadian cortisol profile, has emerged as a sensitive marker of HPA axis function and overall stress load [7,8]. Changes in CAR magnitude and dynamics have been linked to training load, fatigue accumulation, and overtraining-related conditions, highlighting its relevance for monitoring training adaptation and recovery status [6,9].

Dysregulation of the HPA axis may have meaningful consequences for athletic performance. Chronic disturbances in cortisol regulation can affect substrate metabolism, neuromuscular function, and central fatigue mechanisms, ultimately reducing tolerance to training loads [6,10]. Moreover, impaired endocrine recovery may delay physiological restoration processes, limiting the effectiveness of subsequent training stimuli and increasing vulnerability to performance instability.

Differences in study design and assessment approaches warrant integrative interpretation of current evidence on HPA axis dysregulation in physically active individuals [11].

The aim of this narrative review is to examine current evidence on HPA axis dysregulation in the context of regular physical training, with particular emphasis on its impact on exercise capacity and recovery.

2. Physiology of the HPA Axis in the Context of Exercise

Understanding the physiological functioning of the hypothalamic–pituitary–adrenal axis is essential for interpreting endocrine responses to physical training. The HPA axis represents a highly dynamic regulatory system that integrates neural, hormonal, and metabolic signals in

response to stress [1]. In the context of exercise, its activation reflects a complex interaction between acute physical demands and longer-term adaptive processes [3,12]. This section outlines the fundamental mechanisms of HPA axis activation during exercise, the regulation of cortisol secretion within the circadian rhythm, and the concept of allostatic load as it relates to chronic training stress [4].

2.1 Mechanisms of HPA Axis Activation during Physical Stress

The hypothalamic–pituitary–adrenal axis constitutes a primary neuroendocrine pathway through which the body responds to physical stress [1]. Exercise activates this system via both central and peripheral signals, including neural input from higher brain centers, metabolic demands, and afferent feedback from working muscles [1,12]. At the hypothalamic level, physical stress stimulates the release of corticotropin-releasing hormone (CRH), which acts on the anterior pituitary gland to promote the secretion of adrenocorticotropic hormone (ACTH). ACTH subsequently triggers cortisol release from the adrenal cortex [1].

Cortisol plays a key role in supporting the acute physiological demands of exercise. It facilitates glucose availability through gluconeogenesis, promotes lipolysis, and modulates protein metabolism, thereby contributing to energy supply during prolonged or high-intensity physical effort [3,12]. In addition, cortisol exerts regulatory effects on immune and inflammatory responses, which are transiently activated during strenuous exercise [3]. Through negative feedback mechanisms acting at both the hypothalamic and pituitary levels, cortisol also contributes to the termination of the stress response once the stimulus subsides [1].

Importantly, the magnitude of HPA axis activation during exercise is influenced by multiple factors, including exercise intensity, duration, training status, and individual stress sensitivity [3]. Trained individuals often exhibit attenuated hormonal responses to standardized exercise bouts, reflecting adaptive modulation of neuroendocrine stress pathways [13].

2.2 Cortisol Secretion, Diurnal Rhythm, and Exercise-Induced Changes

Under resting conditions, cortisol secretion follows a pronounced circadian rhythm characterized by high concentrations in the early morning and a gradual decline throughout the day [8]. A prominent feature of this rhythm is the cortisol awakening response, defined as a rapid increase in cortisol levels occurring within the first 30–45 minutes after waking [7,8].

This pattern is considered an integral component of normal HPA axis functioning and reflects anticipatory activation of central stress-regulatory mechanisms [7].

Exercise can transiently modify cortisol secretion superimposed on this circadian pattern. Acute bouts of moderate to high-intensity exercise typically induce a short-term increase in circulating cortisol, particularly when the exercise stimulus exceeds a certain intensity or duration threshold [3]. These acute elevations are generally considered adaptive and reversible, contributing to metabolic regulation and tissue remodeling processes associated with training [12].

In contrast, chronic exposure to high training loads may lead to alterations in basal cortisol levels, blunting or exaggeration of diurnal variation, and changes in cortisol responsiveness to subsequent stressors [5,9]. Such alterations may reflect functional adjustments or maladaptive changes in HPA axis regulation in response to repeated physiological stress. Distinguishing between adaptive endocrine modulation and early functional dysregulation remains a central challenge in exercise endocrinology [14].

2.3 Allostatic Load and Chronic Training Stress

The concept of allostasis describes the process by which physiological systems maintain stability through change in response to environmental and internal stressors. While acute activation of the HPA axis supports adaptation to physical demands, repeated or prolonged activation without sufficient recovery can result in increased allostatic load. In the context of regular training, excessive allostatic load may emerge when cumulative stress from training, competition, and non-exercise-related factors exceeds the individual's adaptive capacity [4].

Chronic training stress may therefore shift the HPA axis from an adaptive to a maladaptive state. This transition can manifest as altered cortisol secretion patterns, impaired feedback sensitivity, or reduced hormonal flexibility in response to stress [4,14]. Rather than representing a binary condition, HPA axis dysregulation is better understood as a continuum, ranging from functional overreaching to more pronounced maladaptations associated with persistent fatigue and performance decline [14].

Understanding the balance between beneficial stress-induced adaptation and excessive allostatic load is essential for interpreting endocrine responses in athletes [2,4]. From this

perspective, changes in cortisol dynamics may serve as indicators of the organism's capacity to cope with ongoing training demands, rather than as isolated markers of dysfunction [2].

3. Cortisol and Cortisol Awakening Response as Markers of HPA Axis Function

Assessment of hypothalamic–pituitary–adrenal axis activity in physically active individuals relies on the use of endocrine markers that reflect both basal hormone secretion and dynamic stress responsiveness. Among these markers, cortisol and the cortisol awakening response are most frequently applied due to their physiological relevance and feasibility of measurement. This section focuses on cortisol and CAR as tools for evaluating HPA axis function, outlining their biological basis, measurement approaches, and methodological limitations, without addressing their functional implications for training adaptation or performance [7,8].

3.1 Cortisol as a Biomarker of Training Stress

Cortisol is one of the most frequently assessed hormonal markers in exercise and sports science due to its central role in the regulation of energy metabolism and stress responses [3,12]. As the end product of HPA axis activation, cortisol reflects the integrated output of hypothalamic, pituitary, and adrenal signaling, making it a useful indicator of overall neuroendocrine stress load [1,12]. Measurements of cortisol concentrations in blood, saliva, or urine are commonly used to characterize the acute hormonal response to exercise as well as longer-term adaptations to repeated physical stress [3,12].

The appeal of cortisol as a biomarker lies in its physiological relevance and relative accessibility. Salivary cortisol assessment, in particular, offers a non-invasive method that reflects the biologically active free fraction of the hormone [3,5]. This makes it suitable for repeated measurements and field-based monitoring in physically active populations. Cortisol has therefore been widely employed as a marker of training stress in both experimental and observational studies [5,15].

However, cortisol assessment is subject to several limitations that must be considered when interpreting its values. Cortisol secretion is influenced by numerous factors beyond physical exercise, including circadian rhythm, sleep patterns, nutritional status, psychological stress, and individual variability in stress sensitivity [2,8,11]. Consequently, isolated cortisol measurements may not provide sufficient information about HPA axis functioning unless contextual factors are carefully controlled [5,15]. These limitations highlight the importance of

standardized measurement protocols and complementary markers when using cortisol to assess training-related stress [8,15].

3.2 Cortisol Awakening Response: Physiological Background

The cortisol awakening response represents a distinct component of the diurnal cortisol rhythm, characterized by a rapid increase in cortisol levels within the first 30–45 minutes after awakening [7,8]. This response is thought to reflect anticipatory activation of the HPA axis in preparation for the upcoming demands of the day [8]. Unlike basal cortisol concentrations measured at single time points, CAR provides information about the dynamic responsiveness of the HPA axis [7,8].

Physiologically, CAR is regulated by central mechanisms involving the hypothalamus and higher brain centers, and it appears to be partially independent of the cortisol nadir during nocturnal sleep. The magnitude and pattern of CAR are influenced by sleep–wake timing, light exposure, and neuroendocrine signaling integrity [8]. As such, CAR has been proposed as a marker of HPA axis reactivity rather than basal hormone secretion [7,8].

In the context of physical training, CAR functions as a sensitive indicator of cumulative stress exposure [7,9]. When assessed longitudinally, its dynamic profile enables detection of subtle alterations in HPA axis regulatory capacity that may not be captured by resting cortisol levels alone [7,8]. Importantly, CAR reflects regulatory capacity rather than serving as a direct indicator of performance or recovery outcomes [8].

3.3 Methodological Considerations in CAR Assessment

Accurate assessment of the cortisol awakening response requires strict methodological standardization. Timing of sample collection is critical, as even small delays after awakening can substantially affect CAR magnitude. Standard protocols typically involve cortisol sampling immediately upon awakening, followed by additional samples at fixed intervals, most commonly 30 and 45 minutes post-awakening. Compliance with these timing requirements is essential to ensure data validity.

Several methodological factors may influence CAR measurements, including sleep duration, awakening time, alarm use, and light exposure [8]. Additionally, day-to-day variability in CAR is common, underscoring the need for repeated measurements to obtain reliable estimates of

individual HPA axis function [8,9]. Failure to account for these sources of variability can lead to misinterpretation of results [8].

Interpretation of CAR data requires contextualization. Variations in CAR reflect adaptive regulatory changes, transient stress exposure, or methodological variability rather than pathological dysregulation [7,8]. For this reason, CAR should be interpreted within the broader context of hormonal profiles, training load, and recovery status [5,9,15]. Clear separation between measurement of endocrine markers and interpretation of their functional consequences is essential to maintain methodological rigor [8].

4. Training Load, Fatigue, and HPA Axis Dysregulation

While cortisol and the cortisol awakening response provide useful markers of HPA axis activity, their interpretation becomes meaningful only when considered in relation to training load and fatigue development. In regularly training individuals, the balance between imposed training stress and recovery capacity determines whether neuroendocrine responses remain adaptive or shift toward dysregulation [3,15]. This section examines how different forms of training load influence HPA axis activity and outlines the processes through which repeated stress exposure may lead to functional disturbances in endocrine regulation.

4.1 Acute and Chronic Training Load

Training load can be conceptualized as the cumulative physiological stress imposed by exercise, encompassing intensity, volume, frequency, and training density [15]. Acute training load reflects short-term exposure to physical stress, typically associated with transient activation of the HPA axis and short-lived elevations in cortisol secretion [3,13]. Such responses are generally reversible and form an integral part of normal training adaptation.

In contrast, chronic training load represents the accumulation of repeated exercise stress over time. When sustained without adequate recovery, chronic load has been shown to alter basal cortisol concentrations in some individuals, attenuate or exaggerate hormonal responsiveness, and modify diurnal secretion patterns [3,5,13]. These changes may reflect a shift in regulatory set points within the HPA axis. Importantly, individual variability in stress tolerance means that similar external training loads may result in markedly different endocrine responses across athletes [2,15].

Distinguishing between adaptive endocrine modulation and early functional dysregulation remains challenging. Nevertheless, prolonged deviations in cortisol dynamics under conditions of high chronic load suggest that the HPA axis may progressively lose regulatory flexibility, setting the stage for fatigue accumulation and impaired adaptation [6,14].

4.2 Functional and Non-Functional Overreaching

Overreaching represents an intentional or unintentional increase in training load beyond habitual levels and is commonly classified as either functional or non-functional [14]. Functional overreaching is characterized by short-term performance decrement followed by supercompensation after adequate recovery. During this phase, transient alterations in cortisol secretion and CAR may occur but typically normalize with rest [9,14].

Non-functional overreaching, however, is associated with prolonged fatigue and delayed performance recovery. In this state, disturbances in HPA axis regulation become more pronounced. Altered basal cortisol levels, blunted CAR, or increased day-to-day variability in hormonal responses have been observed in individuals experiencing sustained training stress without sufficient recovery [5,6,9]. These endocrine alterations reflect impaired stress regulation rather than isolated hormonal abnormalities.

The transition from functional to non-functional overreaching is gradual and often difficult to detect. Importantly, changes in HPA axis markers during this phase may precede overt performance decline, suggesting that endocrine dysregulation may serve as an early indicator of maladaptive training responses [6,14].

4.3 Overtraining Syndrome and Endocrine Maladaptation

Overtraining syndrome represents the most severe manifestation of maladaptive responses to chronic training stress. It is characterized by persistent fatigue, performance impairment, and altered physiological regulation that may persist for months [14]. From an endocrine perspective, overtraining syndrome has been associated with profound disturbances in HPA axis function.

Research describing the endocrine and metabolic responses in overtraining has highlighted alterations in cortisol secretion patterns and stress responsiveness, leading to the conceptualization of conditions such as endocrine-related overtraining syndrome [6,14]. In

these cases, dysregulation of the HPA axis may manifest as reduced hormonal reactivity, impaired feedback sensitivity, or abnormal CAR profiles [6,9]. These changes suggest a compromised capacity to mount appropriate endocrine responses to stress.

Endocrine maladaptation in overtraining syndrome reflects a breakdown in the organism's ability to balance stress and recovery. Rather than serving as a marker of acute load, cortisol dynamics in this context indicate a fundamental disturbance in stress regulation [4,14]. Understanding these processes is essential for differentiating between adaptive fatigue states and clinically relevant maladaptations in regularly training individuals.

5. Sport-Specific Patterns of HPA Axis Response

Although activation of the HPA axis represents a general physiological response to physical stress, its manifestation may differ depending on the type of sport and the predominant characteristics of training and competition. Variations in exercise duration, intensity, metabolic demand, and psychosocial stressors contribute to sport-specific patterns of endocrine regulation [3,4]. This section provides a concise overview of HPA axis responses across selected sport categories, highlighting commonalities and distinctions without overemphasizing discipline-specific outcomes.

5.1 Endurance Sports

Endurance sports are characterized by prolonged training sessions and sustained metabolic stress, often resulting in repeated activation of the HPA axis. Acute endurance exercise typically elicits marked cortisol responses, reflecting increased energy demands and substrate mobilization [3]. With regular training, adaptive modulation of cortisol secretion may occur, leading to attenuated hormonal responses to standardized workloads [9].

However, high training volumes sustained over extended periods may alter basal cortisol levels and diurnal secretion patterns. In endurance athletes, changes in cortisol dynamics have been reported particularly during phases of intensified training or insufficient recovery [9,16]. These patterns suggest that prolonged metabolic stress may challenge the regulatory capacity of the HPA axis when cumulative load exceeds adaptive limits [6]. Evidence from studies examining hormonal responses under extreme physical stress suggests that cumulative endurance-type loads are associated with broad alterations in adrenal and peripheral steroid profiles, reflecting limits of load tolerance rather than isolated hormonal abnormalities [17].

5.2 Combat Sports

Combat sports combine high-intensity physical exertion with pronounced psychological and competitive stress. Training and competition are often intermittent but highly demanding, resulting in acute activation of neuroendocrine stress pathways. Cortisol responses in this context reflect both physical load and anticipatory stress associated with competitive situations [18].

Repeated exposure to such stressors may influence HPA axis responsiveness over time. In combat athletes, variations in cortisol secretion have been observed across training cycles and competition phases, indicating dynamic modulation of stress regulation [18,19]. These sport-specific demands highlight the interaction between physical and psychosocial factors in shaping endocrine responses [19].

5.3 Resistance Training and Strength-Based Sports

Resistance training is characterized by short-duration, high-intensity bouts that impose substantial mechanical and neuromuscular demands. Acute resistance exercise can provoke cortisol responses that depend on training volume, intensity, and rest intervals [3]. Compared with endurance exercise, hormonal responses tend to be more variable and protocol-dependent.

Chronic exposure to high-volume or high-frequency resistance training may influence basal cortisol levels and stress responsiveness. In strength-based athletes, alterations in cortisol dynamics have been reported during periods of intensified training, suggesting that repeated neuromuscular stress may affect HPA axis regulation when recovery is inadequate [20].

5.4 Team Sports

Team sports involve a complex combination of aerobic and anaerobic demands, technical skills, and competitive stressors. Training and competition schedules are often irregular, with fluctuating physical and psychological loads across training weeks and competitive seasons. As a result, HPA axis responses in team sport athletes may exhibit considerable variability.

Cortisol dynamics in this context reflect the cumulative effects of training sessions, matches, travel, and competitive pressure. Rather than a single dominant stressor, the endocrine response

in team sports appears to be shaped by the interaction of multiple concurrent demands, underscoring the importance of considering overall load distribution when examining HPA axis activity [15,21].

Across sport categories, differences in HPA axis responses primarily reflect regulatory dynamics rather than absolute cortisol concentrations.

6. Impact of HPA Axis Dysregulation on Exercise Capacity

While activation of the hypothalamic–pituitary–adrenal axis is a normal and necessary component of the physiological response to exercise, persistent alterations in its regulation may have meaningful consequences for exercise capacity [1,3,4]. Exercise capacity reflects the integrated function of metabolic, neuromuscular, cardiovascular, and central regulatory systems, all of which are influenced, directly or indirectly, by cortisol and HPA axis activity [2,4]. This section examines how functional dysregulation of the HPA axis may affect the ability to tolerate training loads, sustain performance, and respond effectively to repeated physical stress [6,14].

6.1 Cortisol Regulation and Metabolic Capacity

Cortisol plays a central role in maintaining metabolic homeostasis during exercise by promoting glucose availability, mobilizing lipid substrates, and regulating protein turnover [1,3,11]. Under conditions of appropriate HPA axis regulation, these actions support sustained energy supply and facilitate adaptation to training demands [2,3]. However, chronic disturbances in cortisol secretion patterns may compromise metabolic efficiency, particularly during prolonged or high-intensity exercise [6,14].

Persistently elevated cortisol exposure can promote excessive protein catabolism and impair muscle glycogen resynthesis, potentially reducing the capacity to sustain repeated bouts of exercise [1,4]. Conversely, blunted cortisol responses may limit the availability of metabolic substrates during physical stress, thereby constraining exercise tolerance [6]. These alterations may represent functional maladaptations of stress regulation in response to cumulative training load [4,14].

6.2 Neuromuscular Function and Central Fatigue

Exercise capacity is not determined solely by peripheral energy supply but also by neuromuscular coordination and central drive [6,21]. Cortisol influences neuromuscular function both directly, through its effects on muscle tissue, and indirectly, via central nervous system pathways involved in motivation, arousal, and fatigue perception [4,6].

Altered HPA axis activity has been associated with changes in neuromuscular performance, including reductions in force production, altered motor unit recruitment, and increased perception of effort [6,21]. Chronic dysregulation of cortisol signaling may contribute to central fatigue mechanisms, reducing the capacity to generate or sustain voluntary muscle activation during exercise [4,14]. Such effects can manifest as decreased performance consistency or reduced tolerance to training intensity, even in the absence of overt musculoskeletal injury [21].

6.3 Stress Responsiveness and Performance Stability

A key aspect of exercise capacity is not only peak performance but also the ability to maintain stable performance across training sessions and competitive demands [14,15]. The HPA axis plays an important role in coordinating physiological responses to both physical and psychological stressors, including competition, time pressure, and environmental challenges [3,4,10].

Functional dysregulation of the HPA axis may impair stress responsiveness, leading to exaggerated or insufficient hormonal responses to acute demands [6,13]. This reduced regulatory flexibility can compromise performance stability, increasing variability in exercise capacity across time [6,14]. From this perspective, exercise intolerance associated with HPA axis dysregulation reflects diminished adaptability rather than a simple reduction in maximal performance potential [4].

6.4 Exercise Capacity as an Integrated Outcome of Endocrine Regulation

Importantly, the relationship between HPA axis activity and exercise capacity should be viewed as bidirectional and dynamic [2,3]. Exercise capacity both influences and is influenced by

endocrine stress regulation, with chronic training stress shaping hormonal responsiveness and hormonal dysregulation, in turn, affecting tolerance to physical load [6,14].

Rather than serving as a single causal factor, HPA axis dysregulation contributes to a broader network of interacting mechanisms that determine exercise capacity [4,11]. These include metabolic efficiency, neuromuscular function, central fatigue, and stress adaptability. Understanding exercise capacity through this integrative lens allows for a more nuanced interpretation of performance limitations observed in regularly training individuals exposed to sustained stress [2,6,14].

7. HPA Axis Function and Recovery Processes in Regularly Training Individuals

Recovery represents a critical but often underexplored component of the training–adaptation cycle. While exercise-induced activation of the hypothalamic–pituitary–adrenal axis is essential for acute stress regulation, the resolution of this response and the restoration of endocrine homeostasis are equally important for effective recovery. In the context of regular training, recovery encompasses a complex set of physiological processes, including metabolic replenishment, neuromuscular repair, immune regulation, and neuroendocrine normalization [4,15]. This section examines the role of HPA axis regulation in recovery processes, focusing on cortisol dynamics, endocrine flexibility, and the temporal characteristics of recovery-related adaptations.

7.1 Cortisol Dynamics during Post-Exercise Recovery

Following acute exercise, cortisol levels typically decline as the immediate stress stimulus subsides and negative feedback mechanisms suppress further HPA axis activation [4,12]. This post-exercise normalization of cortisol is considered an integral component of recovery, reflecting the organism’s capacity to terminate the stress response efficiently [4]. In well-adapted individuals, cortisol concentrations return toward baseline within a predictable timeframe, supporting metabolic restoration and tissue repair processes [5,15].

However, recovery-related cortisol dynamics may vary depending on exercise intensity, duration, and cumulative training load [15]. Prolonged or repeated high-intensity exercise sessions can delay cortisol normalization, leading to sustained elevations or altered diurnal patterns [5,22]. Such responses do not necessarily indicate pathology but may reflect transient endocrine strain associated with insufficient recovery time between training bouts [15,22].

Importantly, cortisol dynamics during recovery should be interpreted in relation to baseline secretion patterns and individual variability [5,8]. Single post-exercise measurements provide limited insight into recovery status unless considered alongside longitudinal hormonal profiles and contextual training information [5,15].

7.2 Endocrine Flexibility and Recovery Capacity

Beyond absolute cortisol concentrations, the concept of endocrine flexibility has gained relevance in understanding recovery processes. Endocrine flexibility refers to the ability of the HPA axis to appropriately activate in response to stress and subsequently downregulate once the stressor has resolved. Efficient recovery is therefore characterized not by suppressed hormonal responses, but by timely and proportionate regulation of cortisol secretion [4].

Reduced endocrine flexibility may manifest as blunted or exaggerated cortisol responses to subsequent stressors, altered diurnal rhythms, or diminished responsiveness of the cortisol awakening response [5,6,22]. These patterns may indicate impaired recovery capacity, particularly when observed consistently over time [5,15]. In regularly training individuals, such alterations may reflect cumulative stress exposure rather than irreversible dysfunction [6,15].

Assessment of endocrine flexibility provides a nuanced perspective on recovery, emphasizing regulatory capacity rather than static hormone levels [4]. This approach aligns with contemporary views of recovery as a dynamic process involving coordinated physiological regulation across multiple systems [4,15].

7.3 Temporal Characteristics and Reversibility of HPA Axis Dysregulation

Recovery processes unfold over varying timescales, ranging from hours to days or weeks, depending on the magnitude and duration of training stress [15]. Correspondingly, alterations in HPA axis function may be transient or persistent, influenced by training load management, recovery strategies, and individual resilience [14,15]. Short-term deviations in cortisol dynamics are commonly observed during periods of intensified training and may resolve with adequate recovery [22,5].

Crucially, functional dysregulation of the HPA axis in the context of training appears to be, at least partially, time-dependent and reversible [6,14]. Evidence suggests that reductions in training load, restoration of adequate recovery periods, and normalization of sleep and

nutritional patterns can facilitate the recovery of typical cortisol secretion profiles [6,14,15]. This temporal reversibility supports the interpretation of HPA axis dysregulation within an adaptive–maladaptive continuum rather than as a fixed impairment [4,14].

Understanding the time course of endocrine recovery is essential for distinguishing between adaptive stress responses and maladaptive states associated with prolonged fatigue or performance decline under conditions of sustained functional dysregulation [14,15]. From this perspective, recovery should be viewed as an active regulatory process in which restoration of HPA axis regulatory function plays a central role, consistent with the adaptive framework outlined in earlier sections of this review [4,14].

8. Practical Implications

Although this review is primarily focused on the physiological mechanisms and consequences of HPA axis dysregulation, several practical considerations emerge for the monitoring and management of regularly training individuals. These implications should be interpreted as supportive tools for understanding training stress and recovery rather than as prescriptive guidelines.

From a monitoring perspective, repeated assessment of cortisol-related markers, including basal cortisol levels and the cortisol awakening response, may provide complementary information on endocrine stress regulation when interpreted longitudinally [8,15]. Such markers are most informative when combined with training load data, recovery indicators, and contextual factors, rather than used in isolation.

Individual variability in HPA axis responsiveness highlights the importance of personalized approaches to training management. Inter-individual differences in cortisol dynamics suggest that similar training loads may elicit divergent endocrine responses, underscoring the need for individualized interpretation of physiological markers rather than reliance on universal thresholds [15].

Finally, the use of endocrine markers in applied settings is subject to methodological and practical limitations. Factors such as circadian variation, compliance with sampling protocols, and non-training-related stressors must be carefully considered [8]. Accordingly, cortisol-based measures should be viewed as one component of a broader monitoring framework, rather than as standalone indicators of training status or recovery quality [8,15].

9. Limitations and Future Perspectives

Despite the growing body of research on HPA axis function in the context of physical training, several limitations should be acknowledged when interpreting the findings summarized in this review. The available literature is characterized by substantial heterogeneity with respect to study design, training modalities, participant characteristics, and methods used to assess cortisol-related markers. Differences in sampling protocols, timing of measurements, and control of confounding factors may limit direct comparability across studies.

Another important limitation concerns the reliance on cortisol-based markers as proxies of HPA axis function. While cortisol and the cortisol awakening response provide valuable insight into endocrine stress regulation, they represent only part of a complex neuroendocrine system. Interpretation of these markers is further complicated by individual variability and the influence of non-training-related stressors, such as sleep disturbances or psychosocial load.

Future research would benefit from more standardized methodological approaches, particularly in longitudinal study designs that integrate endocrine markers with detailed training load, recovery metrics, and performance outcomes. Greater emphasis on within-subject analyses may help clarify individual patterns of adaptation and dysregulation. Additionally, expanding research across diverse athletic populations and training contexts may improve the generalizability of current findings.

Overall, addressing these limitations may support a more precise understanding of how HPA axis regulation interacts with exercise capacity and recovery in regularly training individuals. Finally, some recently published findings have not yet been independently replicated.

10. Conclusions

The hypothalamic–pituitary–adrenal axis represents a central regulatory system linking physical training stress with physiological adaptation, exercise capacity, and recovery. In regularly training individuals, activation of the HPA axis and cortisol secretion constitutes a necessary component of acute stress responses, supporting metabolic demands and adaptive processes. However, when exposure to training stress becomes excessive or recovery is

insufficient, functional alterations in HPA axis regulation may emerge under conditions of sustained training stress.

Current evidence indicates that dysregulation of cortisol dynamics, including changes in basal secretion, diurnal rhythm, and cortisol awakening response, is associated with impaired exercise tolerance and compromised recovery capacity. These alterations can be situated within an adaptive–maladaptive continuum of stress regulation rather than reflecting fixed impairment. Importantly, the reviewed literature suggests that HPA axis disturbances related to training load are often time-dependent and at least partially reversible with appropriate recovery and load management.

Assessment of cortisol-related markers provides valuable insight into endocrine stress regulation, particularly when interpreted longitudinally and in combination with contextual training and recovery information. Rather than serving as isolated indicators, these markers contribute to a broader understanding of individual stress responsiveness and recovery processes.

Overall, this review highlights the importance of viewing HPA axis function as a dynamic and integrative component of training adaptation. Recognizing the role of endocrine regulation in exercise capacity and recovery may support more nuanced interpretation of physiological responses to training stress and contribute to improved monitoring strategies in physically active populations.

Viewing HPA axis regulation as a dynamic marker of stress adaptability, rather than a static indicator of dysfunction, may improve interpretation of training responses in physically active populations.

Disclosures:

Author's contribution:

Conceptualization: AG, WP, BP;
Methodology: AG, JAW, MMT, LO;
Software: AP, JP, WP;
Check: NMK, AK, KSB;
Formal analysis: AG, BP, DJG, AP;

Investigation: NMK, LO, AK;

Resources: JAW, MMT, KSB, DJG;

Data curation: BP, JP, WP;

Writing-rough preparation: MMT, KSB;

Writing -review and editing: NMK, BP, JAW, DJG;

Visualization: JP, AP, LO; Supervision: AG, AK, WP, MMT;

Project administration: AG, BP, JAW

Funding: The study did not receive external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgements: Not applicable.

Conflicts of Interest: The authors declare no conflicts of interest.

All authors have read and agreed with the published version of the manuscript.

Declaration of the use of generative AI and AI-assisted technologies in the writing process:

In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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