Journal of evidence-based medical research

Cortisol-Mediated Energy Allocation and Its Implications for Body Composition in Physically Active Adults: A Narrative Review

Talal A Abdalla[1]

1. Diabetes and Endocrinology, Homeopathy Research Institute, London, United Kingdom.

Abstract

Background: The historical approach to metabolism as a simple equation of calories is insufficient to explain the intricate variety of hormones that affect partitioning of energy among tissues, including muscle, fat, and others. Stress is accompanied by a rise in the production of a glucocorticoid hormone of the adrenal cortex called cortisol, which is a master regulator of the allocation of energy during stress, but whose relationship to body composition is not mentioned so much in the guidance of nutrition and training plans in practice.

Methods: A review was conducted to synthesize the studies from the past 20 years examining the effect of exercise, energy status, and body composition on cortisol in various populations of physically active adults.

Result. Energy availability had the largest impact on cortisol. Decreases in energy always increased levels of cortisol and decreased anabolic hormones such as the Insulin-like Growth Factor-1 (IGF-1) and Testosterone. There was no significant difference in resting cortisol between different training status or competitive level, suggesting that cortisol's response to exercise or stress and the daily cortisol rhythm are more informative biomarkers. Inactive obese individuals have low cortisol, whereas more active or under-stressed obese individuals have higher cortisol levels. People with major depressive disorder or hypothyroidism show weak or no cortisol response to exercise; this means findings from healthy people do not apply to everyone. Importantly, a reduction in cortisol levels in response to stress is not necessarily an indicator of overtraining, as it may be a normal adaptive response. In a male athlete, testosterone is the primary hormone that regulates body fat, while higher body fat percentages may aid in adaptation through being a reservoir of cortisol. Moreover, vigorous exercise was shown to have a negative feedback effect on subsequent stress responses, and the location of the competition games was shown to have a significant effect on post-game cortisol, with home games showing a greater stress response irrespective of the result.

Conclusions: Resting cortisol is a poor marker of training status, and energy availability is the most important factor determining cortisol regulation. The interpretation of cortisol response should take into account energy status, performance outcomes, and individual health status. In athletes and clinical populations, monitoring levels of reactive cortisol in addition to the testosterone/cortisol ratio may be a more effective way to assess stress, recovery, and adaptation.

Keywords: Cortisol; energy availability; HPA axis; body composition; exercise; stress adaptation; RED-S

Introduction

Many physically active adults, athletes, and fitness enthusiasts work diligently to optimize their composition, which is one of the primary reasons for their fitness goals [1]. In the past, body composition management has been described as the energy balance approach, the basis of which is establishing a relationship between energy intake and energy expenditure, which influences the change in body mass [2]. Although this approach is largely correct, it fails to capture the variability seen at the individual level in response to the same exercise and nutrition program. People who tend to have similar training programs and nutritional regimens often have vastly different fat loss, muscle building, and body composition results, which emphasizes a major role for physiology and hormones in controlling energy utilization and storage [3].

Cortisol has been identified as a prime mediator of the distribution of energy, amongst the hormones involved in metabolic regulation [4]. Cortisol is a chemical synthesized by the adrenal cortex in response to the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which helps to ensure that energy is available during physical or psychological stress [5]. Cortisol enables adaptive responses through activation of gluconeogenesis, lipolysis, and mobilisation of energy substrates to support the demands of the current challenge [6]. When cortisol levels are raised for many days or even weeks due to chronic energy deficits, high training volumes and frequencies, insufficient recovery, and/or chronic exposure to psychosocial stressors, however, it can alter metabolism to favour additional protein breakdown, reduce recovery, decrease whole-body anabolic hormone levels, and adversely alter body composition [7].

Several factors interact in regulating cortisol. One major determinant of cortisol secretion is energy availability, especially in physically active populations in whom cortisol levels are elevated by high training loads [8]. In the same way, the activity of the HPA axis and cortisol response can be affected similarly by diet, exercise, but also by psychological stress and individually by all of them [9]. It is critical to understand how these contribute to cortisol, since this hormone can be a marker for stress, but also is considered a biologically informative signal of whether energy is allocated for growth and repair or immediate survival and maintenance [10].

This narrative review aims to explore how energy status, the food matrix, the type of physical activity, and psychological stress affect cortisol regulation in physically active adults and how this affects the hormones. Identification of the relationships may offer valuable clues to the underlying mechanisms that underlie the association of stress physiology and body composition in trainers and sports practitioners and balance evidence-based stress management interventions to improve performance, recovery, and well-being.

Methodology

A narrative review design was used to integrate and critically reflect on the current literature regarding the effects of cortisol on energy allocation and how that relates to body composition in physically active adults. A literature search using scientific databases (PubMed, Google Scholar, Scopus, and Web of Science) was carried out. Studies were included if they assessed cortisol concentrations in relation to exercise, energy availability, stress-induced factors, or body composition in physically active adults. Studies were excluded if they did not study direct measures of cortisol. Lastly, 20 studies were selected for final synthesis that were relevant and evidence-based in their respective studies. The athletes were elite; the exercise group was recreational. The study Population, Cortisol Metric & Sample, body composition, outcome & Method, Energy Status, key finding and modulating factors, were determined. A thematic synthesis (qualitative) approach was utilized to establish

coherent patterns and connections between the role of energy status, cortisol regulation, and body composition outcomes. Consistent with the narrative review method, attention was paid to patterns of physiology that could be identified rather than to statistical summaries. This narrative review was conducted without the need for ethical approval or informed consent given that it is a review of previously published information.

Result

Included in the studies (n = 20) were a large variety of athletes and recreational adults, as well as obese, sedentary, and clinical populations. The majority of the research was done on male subjects, and there were fewer studies involving female subjects or using mixed-sex design. Sample sizes varied from small controlled laboratory studies (most of the time n = about 12–40) to large population-based cohorts (most of the time n = about 1,900). The types of exercise consisted of resistance training, endurance training, high-intensity interval training, exercise training in team sports competition, and controlled laboratory stress protocol. The most common methods were taking blood samples from the morning after a fasting period or blood samples from saliva, with some studies employing multiple blood samples throughout the exercise or stress task to measure reactive HPA-axis responses. Body composition measures were largely DXA, and included BodPod, bioelectrical impedance analysis, skinfold thickness, and anthropometric prediction equations.

Overall, there was a range of energy statuses (acute deficiency in elite athletes and physically active adults, energy balance or observational in the general population) across the studies. Low energy availability was explicitly quantified (e.g., <30 kcal/kg FFM/day) in several studies, and others were inferential, basing the energy stress on training load or the phases of the seasons. Body composition differences measured were fat mass, fat-free mass, body fat percentage, and regional muscle differences, along with some measuring bone mineral density. There was overall methodological heterogeneity about cortisol sampling, assay methods, classification of training status, and control variables such as diet, circadian rhythm, and sex hormones. Energy deficit and physiological stress still seemed to be associated with increased or dysregulated cortisol responses because, despite this variability, a consistent association between these factors and resting cortisol responses was not found.

Table 1. Energy Deficit / Low Energy Availability / Undernutrition Studies

Study	Sample	Cortisol measure	Body composition method	Energy status	Key finding	Main interpretation
Longland et al., 2016 [11]	40 untrained overweight males	Fasting serum cortisol	4-compartment (DXA/BodPod/BIA)	40% energy deficit + resistance training	↑ cortisol weakly linked to ↑ fat loss but ↓ lean mass gain	Cortisol has small catabolic effect on LBM retention in deficit
Stengvist et al., 2020 [12]	20 trained male cyclists	Fasting + exercise cortisol	DXA	Possible low energy availability (HIIT block)	↑ cortisol (+12.9%) during intensified training	Cortisol rises with training stress; linked to RED-S markers
Isola et al., 2025 [13]	43 physique athletes (M/F)	Fasting serum cortisol	DXA + ultrasound	Severe LEA during prep	Cortisol ↑ in males; T/C ratio ↓ sharply	T/C ratio more sensitive than cortisol alone
Liu et al., 2026 [14]	120 elite athletes	Fasting serum cortisol (6 time points)	DXA	Persistent energy deficit	Large ↑ cortisol (d=1.4–1.7), r = –0.46 with energy status	Cortisol strongly tracks energy deficit severity
Lukas et al., 2005 [15]	132 pastoralist men	Urinary cortisol	Anthropometry	Chronic + acute undernutrition	Higher cortisol in acute drought; ↓ muscle mass	Cortisol linked to muscle loss under acute energy stress
Azarbayjani et al., 2010 [16]	20 sedentary women	Salivary cortisol	BMI + VO_2max	No controlled deficit	Obese > lean cortisol; exercise ↑ cortisol	Obesity alters cortisol response magnitude

Table 1 included studies measuring cortisol as a primary variable in energy deficit scenarios as seen in dieting, physique competition preparation, endurance training overload, or chronic/acute undernutrition. Cortisol levels

were elevated in these studies when there was an energy deficiency in the diet or when there was a combination of training stress with a lack of energy. The increased level of cortisol occurred consistently across these studies when there were indications of an energy deficiency in the diet or when there was a combination of training stress and an inadequate energy intake. Although its relationship with body composition is mostly indirect, higher cortisol levels have a stronger correlation with poorer lean mass maintenance than with greater amounts of fat loss, and studies suggest that correlation with higher fat mass is largely inconsistent and weak across different studies. Based on the overall pattern, cortisol does not appear to be a primary factor in mediating body composition change, but rather a measure of the severity of metabolic stress and energy deficit.

Table 2. Training Load, Stress, and Cortisol Reactivity (No Clear Deficit or Mixed Energy States)

Study	Sample	Cortisol measure	Body composition method	Energy status	Key finding	Main interpretation
Terink et al., 2021 [17]	14 male athletes	Serum cortisol	DXA	Energy-matched diets	LCHF ↑ exercise cortisol acutely, no body comp change	Diet changes cortisol reactivity, not body composition
Wong & Harber, 2006 [18]	13 men (lean vs obese)	Serum cortisol (exercise)	Hydrostatic weighing	Energy matched	Obese = higher + prolonged cortisol response	Obesity amplifies stress response, reduces recovery
Mangine et al., 2020 [19]	23 CrossFit athletes	Fasting serum cortisol	4C model DXA/BodPod	No deficit	No group differences in cortisol	Training level ≠ resting cortisol differences
Gubelmann et al., 2018 [20]	1948 adults	Salivary diurnal cortisol	BMI	Free-living	PA → steeper cortisol slope (small effect)	Activity improves diurnal rhythm slightly
Gerber et al., 2020 [21]	MDD patients	Salivary cortisol (TSST)	Not assessed	Clinical condition	Blunted cortisol, no training effect	Depression blunts HPA axis response
Fothergill et al., 2017 [22]	Soccer players	Salivary cortisol	Not assessed	Competition stress	Home games ↑ cortisol	Psychological stress > physical load
Caplin et al., 2021 [23]	83 men	Salivary cortisol (TSST)	Not assessed	Controlled acute exercise	Vigorous exercise buffers stress cortisol	Acute exercise modulates stress reactivity
Latour et al., 2022 [24]	Elite rowers	Serum cortisol	BIA	Controlled diet	Post-exertion cortisol drop + rebound	Adaptation, not overtraining marker
Ponce-Gonzalez et al., 2015 [25]	Basketball players	Serum cortisol	Skinfolds	Not controlled	Position differences NS in cortisol	Performance differences not driven by cortisol
Rodríguez García et al., 2024 [26]	Pro soccer	Serum cortisol	Skinfolds	Pre-season load	Small ↑ cortisol + fat loss	Training improves body comp more than hormonal change

Table 2 contain studies in which energy intake is typically sufficient, not directly altered, or cortisol change is primarily due to training load, psychological stress, obesity and/or clinical status and/or acute responses to exercise. Within these contexts, resting cortisol levels are relatively unchanged for different training intensities or statuses, or may be more evident in acute or situation-specific stressors like competition (home or away stress), psychosocial stress tests, or high-intensity exercise bouts. There is strong context-dependence to cortisol responses and responses can be exaggerated (e.g., obesity, exposure to stress, etc.) and buffered (e.g., high intensity exercise during stress recovery, etc.), while there is little consistency or strength of a relationship with body composition and/or long-term training adaptation. In these studies, cortisol's overall performance is more similar to an acute stress reactivity marker and much less like a predictor of physique or training status.

Cortisol and the Stress Response

HPA Axis Regulation

Cortisol is controlled by the hypothalamic-pituitary-adrenal (HPA) axis. When the brain is exposed to a stressor, such as exercise, calorie restriction, or trauma, or a psychological stressor such as competition, social scrutiny, or pressure, corticotropin-releasing hormone (CRH) is released by the hypothalamus, followed by a subsequent

release of adrenocorticotropic hormone (ACTH) from the pituitary, which leads to a release of cortisol from the adrenal cortex Almodóvar-Fernández et al., 2025 [27]. This system is negatively feedback: an increase in cortisol will suppress the production of further corticotropin-releasing hormone and adrenocorticotropic hormone due to its activity within the hypothalamus and pituitary Amani & Dadmanesh, 2024 [28]. The response and persistence of cortisol is related to stressor intensity, individual fitness, energy reserves, and past stress exposure.

Caplin et al., 2021 [23] concluded that acute exercise intensity at 70% heart rate reserve induced significantly greater cortisol release during exercise than 30% HRR, suggesting a dose-response relationship exists between exercise intensity and HPA axis activation. The cortisol responses of the vigorous exercise group were significantly larger than the light exercise group ($p = 0.028$, $\eta^2 = 0.082$). Latour et al., 2022 [24] demonstrated acute effects of a low-carbohydrate, high-fat diet on inducing a larger exercise-mediated cortisol response; a low-carbohydrate, high-fat diet resulted in a significantly greater cortisol response (822 ± 215 nmol/L, compared to 609 ± 208 nmol/L with a high carbohydrate diet, $p = 0.004$). Fothergill et al., 2017 [22] demonstrated that playing venue had no relationship with the level of physical exertion but was indeed a psychological stress factor, with post-game cortisol levels being significantly higher at home (18.80 mmol/L vs away, ~10 mmol/L, $p = 0.002$ and $p = 0.001$ with venue × time interaction, $\eta p2 = 0.63$).

Acute vs Chronic elevation of Cortisol

Cortisol is a hormone that becomes elevated and adaptive when the body is subject to brief stress periods or significant activity, in order to release energy to sustain physical performance [23]**.** Chronic elevations, however, act fundamentally. Mangine et al., 2021[29] tested 120 elite athletes for 48 weeks, and discovered that the constant lack of energy resulted in consistently high levels of cortisol (very large effect sizes in endurance athletes (Cohen d = +1.7) and power athletes (Cohen d = +1.4)), and in consistently low leptin (large effect sizes in endurance athletes (Cohen d = -1.9) and power athletes (Cohen d = -1.4), as well as consistently low levels of testosterone-to-cortisol (large effect sizes in endurance athletes (Cohen d = -1.9) and power athletes (Cohen d = -1.5). Nies et al., 2004 [30] have shown that 4 weeks of increased training intensity with no increase in energy intake resulted in a significant increase in serum cortisol levels by 12.9% ($p = 0.021$, effect size = 0.56) and significant decrease in resting metabolic rate (-3.0%, $p = 0.010$, effect size = 0.64) and triiodothyronine levels (-4.8%, $p = 0.008$, effect size = 0.67), which are all hallmarks of Relative Energy Deficiency in Sport (RED-S). In males, Isola et al., 2025 [13] observed an increase from catabolic dominance (a drop in the T/C ratio of 50.4%, $p< 0.001$) in physique athletes during their period of competition preparation.

Critically, the increase in cortisol immediately following extreme exertion was actually a decrease, -17% (95% CI -22.16 to -11.79) [26] and the difference between cortisol response following exertion and baseline was highly correlated with body fat percentage ($\tau = 0.56$, $p = 0.003$). This anti-deficit principle shows that a reduction of cortisol at the time of stress is not necessarily a sign of failure of the HPA axis.

Cortisol and Energy Allocation

Resource Shifting Away from Anabolic Processes

Chronic elevated cortisol levels essentially force the body to use energy for immediate purposes rather than for future use in anabolic functions, such as building muscle protein, strengthening the bone, and healing tears in

tissues. This change is accomplished by several mechanisms. Ponce-Gonzalez et al., 2015 [25] demonstrated that, in energy deficit (40% decreased energy intake), increased cortisol also was associated with decreased lean body mass gain ($r = -0.34$, $p = 0.03$) and fat mass loss ($r = +0.39$, $p = 0.01$); that is, cortisol actively inhibits body composition changes that people want during caloric deficit. The effect of this catabolism, however, was completely reversed by a higher protein intake (2.4 g/kg/day), and lean mass gain was 1.2 ± 1.0 kg ($p < 0.05$) versus no change in lean mass (0.1 ± 1.0 kg) in the lower protein group.

Stengvist et al., 2020 [12] found that a rise in cortisol with increased training intensity was related to a 3.0% decrease in resting metabolic rate ($p = 0.010$), and a 4.8% decrease in triiodothyronine ($p = 0.008$), indicating a decrease in baseline energy expenditure as a mechanism for conserving energy when it is perceived as scarce. Isola et al., 2025 reported that Insulin-like growth factor-1 was also significantly reduced in both genders across competition preparation ($p < 0.001$) for both endurance athletes (-1.1) and power athletes (-0.9) and was positively associated with fat-free mass loss in males ($p < 0.05$).

Tissue-Specific Effects

Cortisol's ability to alter allocation of tissues for energy use is especially important for understanding changes in body composition. Amani & Dadmanesh, 2024 [28] demonstrated that cortisol's catabolic effects on arm muscle (AM) occurred only for pastoralists with energy deficit versus those without ($\beta = -0.28$, $p=0.018$, versus $\beta= -0.04$, $p=0.78$, respectively), suggesting that these effects mainly occur when energy deficit is added to chronic undernutrition in pastoralists.

Wong & Harber, 2006 [18] found that during exercise, obese men experienced significantly greater cortisol responses (25.5 ± 7.7 vs. 13.1 ± 5.3 µg/dL; $p < 0.05$) and a significantly greater suppression of excess post-exercise oxygen consumption (3.0 ± 1.3 vs. 4.2 ± 0.9 L; $p < 0.05$) and a persistent increase in respiratory exchange ratio before, during, and after exercise ($p < 0.05$ at multiple time points), demonstrating lower post-exercise fat oxidation rates in obese men. This means that stress can also catalyse the breakdown of muscles and affect metabolic flexibility for fat oxidation when recovering.

Almodóvar-Fernández et al., 2025 [27] found that in elite soccer players, there was a strong inverse correlation between testosterone and body fat percentage ($r = -0.79$, $p<0.05$) and that center players had higher testosterone levels (7.8 ± 1.2 vs. 6.0 ± 0.5 ng/mL; $p<0.05$) and lower body fat levels (9.5 ± 0.6 vs. 10.9 ± 0.7%, $p<0.05$) than guards as a result of anabolic-catabolic balance within the body rather than cortisol alone, having a major influence on the fat accumulation pattern, with centre players exhibiting a lower percentage of body fat. According to Nies et al., 2004 [30] in sedentary obese females, cortisol was significantly inversely associated with fat mass ($r = -0.61$, $p < 0.05$), body fat percentage ($r = -0.69$, $p < 0.01$) and BMI ($r = -0.64$, $p < 0.05$), suggesting impaired conversion of cortisone to cortisol, but not necessarily HPA axis suppression [14].

Implications for Body Composition in Active Adults

Identify differences in training and nutrition among individuals.

The reviewed studies indicate that the same training and nutritional methods can lead to differences in body composition among individuals due to variability in cortisol responsiveness and energy status. In their study, Mangine et al., 2021 [29] compared the characteristics of advanced CrossFit athletes (Regional/Games

qualifiers), recreationally active CrossFitters, and physically active controls. Even in the presence of significant lower body fat percentage (6.7-8.3% lower, $p = 0.007$) in advanced athletes, coupled with higher lean mass (12.5-26.8% higher, $p \leq 0.028$), greater VO_2peak (18.8-19.1% higher, $p = 0.002$), and superior strength and power (peak force 15.4-41.8% higher, $p < 0.05$), no difference was observed between the groups for resting serum cortisol levels ($p > 0.05$), and the null hypothesis was preferred over the alternative by Bayesian analysis for the difference in the hormones. This represents that when assessing resting cortisol, there is no difference between people who get a better body composition and the ones who do not.

Liu et al., 2026 [14] found that, rather, levels of energy and reactive cortisol are more revealing. This ratio, known as the energy compensation rate (intake/TEE), was found to be inversely U-shaped with nadirs in endurance athletes of 79.6% at peak training load and 82.6% at peak training load for power athletes ($p < 0.001$-time effect). More severe deficits (energy compensation rate ≤ 90%) resulted in proportionately higher elevation in cortisol, and a dose-response effect in leptin suppression (energy compensation rate -1.68, 95% CI -2.25 to -1.11) compared to mild deficits (-0.72, 95% CI -1.22 to -0.22).

Visceral Fat Accumulation as a Stress-Driven Response

Several studies found there to be a ligand selectivity for cortisol-related fat deposition in specific depots (visceral). While male professional basketball players were studied, Ponce-Gonzalez et al., 2015 [25] determined that testosterone was the most important hormone involved with body fat percentage ($r = -0.79$, $p < 0.05$). Rodríguez García et al., 2024 [26] found that the increase in body fat was negatively associated with the increase in testosterone levels ($r = -0.53$, $p = 0.04$), and that the change in testosterone was significantly predictive of the change in body fat ($\beta = -0.53$, $R^2 = 0.28$, $p = 0.04$). The ratio of testosterone to cortisol (T/C) was found to be maximum at the mid-season (0.056 ± 0.019; significantly higher than at the beginning of the season, 0.046 ± 0.019, $p < 0.05$) and minimum with the minimum body fat, indicating that the best anabolic-catabolic ratio is essential for fat reduction.

Impaired Recovery and Reduced Anabolic Signaling

Multiple factors impair recovery in cases of chronic elevation of cortisol. Stengvist et al., 2020 [12] found that resting metabolic rate (RMR) was lower during intensified training (ES = 0.64, N = 13, $p = 0.010$). Isola et al., 2025 [13] reported that during competition preparation, male physique athletes had a decrease of 50.4% in the testosterone/cortisol ratio (TCR; $p < 0.001$); an increase in fatigue ($p < 0.05$) and significant reductions in isometric and concentric strength ($p < 0.01$ to 0.001).

Gerber et al., 2020 [21] found that in patients with major depressive disorder, indiscriminate reactivity to psychosocial stressors evolved over a small range (1.6x production between pre-stress and peak level, while in healthy subjects, the range lies between 2x to 4x), with 48% of patients being considered as non-responders. Therefore, the effect of aerobic exercise on this course remained unchanged 6 weeks later (time × group interaction $p = 0.492$, $\eta^2 = 0.021$), showing that alternative pathways are responsible for the beneficial effect of exercise in clinical populations.

Clinical Implications

The evidence reviewed supports an individualized, signals-driven approach over a protocol-based approach. Liu et al., 2026 [14] showed that the energy compensation rate provides a useful indicator for the dynamic equilibrium between intake and expenditure, with ECR values below 80% representing severe endocrine disruption scores (+1.7 to +1.4). Longland et al., 2016 [11] reported direct experimental data of the completely protective effect of higher protein intake (2.4 g/kg/day) for achieving 1.2 ± 1.0 kg ($p < 0.05$) of increased lean mass despite a 40% energy deficit. Caplin et al., 2021 [23] showed that a single session of a stress-inducing high-intensity exercise (70% HRR) decreases the cortisol response to a later psychosocial stressor, with the vigorous exercise group experiencing significantly slower cortisol rise ($\gamma = -0.017$, $p < 0.001$), faster decrease ($\gamma = +1.7 \times 10^4$, $p < 0.001$), earlier peak (40.4 vs. 45.5 min), and earlier return to baseline (≈81 vs. ≈91 min) than light exercise group.

Mangine et al., 2021 [29] observed that well-periodized training (regular training cycles built into recovery cycles) resulted in no significant changes in the ratio of cortisol: testosterone (ChrT) over the course of the entire competitive season (6-7 months) ($p > 0.05$), and no change in cortisol concentration in the plasma over the course of the season ($p > 0.05$). Overall stress during an extended season may influence the anabolic/catabolic balance mainly, since Almodóvar-Fernández et al., 2025 [27] reported that the maximum T/C ratio should be found in the middle of the season (0.056 ± 0.019) and minimum in the final season (0.042 ± 0.014, $p < 0.05$).

Gubelmann et al., 2018 [20] reported that both regularly active and weekend warriors had favorable profiles of cortisol compared to inactive individuals: cortisol slopes (fallen from 2.0 to 5.3 nmol/L/h/day) tended to be steeper for the weekend warriors (-1.61 ± 0.05 vs. -1.44 ± 0.04 nmol/L/h/day, $p = 0.02$) and total output of cortisol tended to be lower for the regularly active individuals (205.4 ± 2.4 vs. 215.5 ± 2.9 nmol·h/L, $p < 0.01$). This suggests that accumulating physical activity, regardless of distribution, benefits HPA axis regulation.

Practical Recommendations for Practitioners

Based on available evidence, practitioners should focus on energy availability and not calorie balance because low energy availability appears to be the greatest causal factor of elevated cortisol levels, and could adversely affect recovery, performance, and/or body composition. In energy deficit or with high training volume, around 2.4 g/kg body weight/day of protein is suggested to maintain lean mass and oppose cortisol-induced protein catabolism. Chronic endocrine stress should be avoided, and training programs should involve reload weeks and taper periods [31]. Vigorous exercise could be timed for about 45 minutes before a known stressor to dampen the cortisol spillover caused by the stressor because of negative feedback. Ultimately, due to the interconnectedness of the psychological and physical context to the body's cortisol regulatory process, stress management should be incorporated within athlete support programs.

Future Research Directions

For future studies, it is important to conduct studies with a longitudinal design that tracks cortisol levels, energy availability, and performance throughout multiple competitive seasons in order to establish causal relationships. More intervention trials are required in order to assess the impacts of diet timing and stress management along with sleep optimization on cortisol regulation and body composition outcomes. It is vital to include female athletes, as there is a known difference in HPA axis function in males versus females and very limited evidence in the female population. Pre-stressor exercise as a stress-reduction intervention for the real

world should also be explored as a research strategy in terms of optimum timing and intensity of pre-stressor exercise. Besides this, examining the anti-deficit principle's implications in clinical populations could contribute to the creation of interventions for situations in which HPA-axis dysfunction is common, such as in sleep apnea and obesity. Lastly, research investigating the degree to which increased protein consumption can counteract the catabolizing effects of cortisol during negative energy balance in athletes and physically active adults can lead to dietary recommendations for protein needs that are optimized.

Conclusion

The review concluded that energy availability is the main factor controlling cortisol in active adults. When energy intake is low, cortisol levels will rise, and they will enter a breakdown state; when it is adequate, their hormones will be well balanced, and they will be able to recover. A resting cortisol level is less useful for determining training status than are a reactive cortisol response and patterns across time. In general, cortisol has to be considered as a signal to allocate energy, not only as a stress marker, and should always be interpreted together with training load, recovery, and nutritional status.

References

1. Isenmann, E., J. Dissemond, and S. Geisler, *The Effects of a Macronutrient-Based Diet and Time-Restricted Feeding (16:8) on Body Composition in Physically Active Individuals-A 14-Week Randomised Controlled Trial.* Nutrients, 2021. **13**(9).

2. Johnson, N.A., et al., *Physical activity in the management of obesity in adults: A position statement from Exercise and Sport Science Australia.* J Sci Med Sport, 2021. **24**(12): p. 1245-1254.

3. Kostenchak-Svystak, O., et al., *THE INFLUENCE OF BODY COMPOSITION ON THE STATE OF THE CARDIOVASCULAR SYSTEM IN WOMEN.* Georgian Med News, 2020(308): p. 58-62.

4. Ten Haaf, D.S.M., et al., *Protein supplementation improves lean body mass in physically active older adults: a randomized placebo-controlled trial.* J Cachexia Sarcopenia Muscle, 2019. **10**(2): p. 298-310.

5. Kotarsky, C.J., et al., *Time-restricted eating and concurrent exercise training reduces fat mass and increases lean mass in overweight and obese adults.* Physiol Rep, 2021. **9**(10): p. e14868.

6. Şenol, M.P., et al., *Intuitive Eating Intervention in Physically Active Adults: Effects on Anthropometry, Athletic Performance, Eating Attitudes, and Body Image.* Nutrients, 2025. **17**(17).

7. Manore, M.M., *Exercise and the Institute of Medicine recommendations for nutrition.* Curr Sports Med Rep, 2005. **4**(4): p. 193-8.

8. Maruszczak, K., et al., *Sleep quality and body composition in active and inactive young adults.* Sci Rep, 2025. **15**(1): p. 39614.

9. Santi, A., et al., *High Body Mass Index Masks Body Composition Differences in Physically Active Versus Sedentary Participants.* Metab Syndr Relat Disord, 2018. **16**(9): p. 483-489.

10. Moradell, A., et al., *Longitudinal Changes in the Body Composition of Non-Institutionalized Spanish Older Adults after 8 Years of Follow-Up: The Effects of Sex, Age, and Organized Physical Activity.* Nutrients, 2024. **16**(2).

11. Longland, T.M., et al., *Higher compared with lower dietary protein during an energy deficit combined with intense exercise promotes greater lean mass gain and fat mass loss: a randomized trial.* The American journal of clinical nutrition, 2016. **103**(3): p. 738-746.

12. Stenqvist, T.B., et al., *Impact of a 4-week intensified endurance training intervention on markers of relative energy deficiency in sport (RED-S) and performance among well-trained male cyclists.* Frontiers in Endocrinology, 2020. **11**: p. 512365.

13. Isola, V., et al., *Changes in hormonal profiles during competition preparation in physique athletes.* European Journal of Applied Physiology, 2025. **125**(2): p. 393-408.

14. Liu, Y., et al., *Coupling mechanisms between energy compensation and metabolic adaptation during high-intensity training in athletes: a longitudinal study based on multi-omics and individualized energy modeling.* Scientific Reports, 2026.

15. Lukas, W.D., B.C. Campbell, and K.L. Campbell, *Urinary cortisol and muscle mass in Turkana men.* American Journal of Human Biology: The Official Journal of the Human Biology Association, 2005. **17**(4): p. 489-495.

16. Azarbayjani, M.A., et al., *Daily timing of salivary cortisol responses and aerobic performance in lean and obese active females.* Bratisl Lek Listy, 2011. **112**(4): p. 213-7.

17. Terink, R., et al., *A 2 week cross-over intervention with a low carbohydrate, high fat diet compared to a high carbohydrate diet attenuates exercise-induced cortisol response, but not the reduction of exercise capacity, in recreational athletes.* Nutrients, 2021. **13**(1): p. 157.

18. Wong, T. and V. Harber, *Lower excess postexercise oxygen consumption and altered growth hormone and cortisol responses to exercise in obese men.* The Journal of Clinical Endocrinology & Metabolism, 2006. **91**(2): p. 678-686.

19. Mangine, G.T., et al., *Physiological differences between advanced CrossFit athletes, recreational CrossFit participants, and physically-active adults.* PloS one, 2020. **15**(4): p. e0223548.

20. Gubelmann, C., et al., *Association of activity status and patterns with salivary cortisol: the population-based CoLaus study.* European journal of applied physiology, 2018. **118**(7): p. 1507-1514.

21. Gerber, M., et al., *Effects of aerobic exercise on cortisol stress reactivity in response to the trier social stress test in inpatients with major depressive disorders: a randomized controlled trial.* Journal of clinical medicine, 2020. **9**(5): p. 1419.

22. Fothergill, M., S. Wolfson, and N. Neave, *Testosterone and cortisol responses in male soccer players: The effect of home and away venues.* Physiology & behavior, 2017. **177**: p. 215-220.

23. Caplin, A., et al., *The effects of exercise intensity on the cortisol response to a subsequent acute psychosocial stressor.* Psychoneuroendocrinology, 2021. **131**: p. 105336.

24. Latour, E., et al., *Stressor-induced temporal cortisol deficiency as a primary trigger for adaptation to stress.* International Journal of Environmental Research and Public Health, 2022. **19**(9): p. 5633.

25. Ponce-González, J.G., et al., *Physical fitness, adiposity and testosterone concentrations are associated to playing position in professional basketballers.* Nutricion hospitalaria, 2015. **31**(6): p. 2624-2632.

26. García, L.R., et al., *Relationship between testosterone and cortisol with anthropometric characteristics in professional male soccer players.* Retos: nuevas tendencias en educación física, deporte y recreación, 2024(61): p. 919-926.

27. Fernández, I.A., et al., *An An exploratory observational study of the seasonal changes of body composition, bone parameters, testosterone, and cortisol in Spanish male professional soccer players.* European Journal of Human Movement, 2025(54): p. 21-25.

28. Amani, M. and M. Dadmanesh, *The effect of aerobic-resistance exercise on body composition, physical fitness, resting metabolism rate, serum cortisol and leptin of female with hypothyroidism.* Journal of Physical Activity and Hormones, 2024. **1**(2): p. 1.

29. Mangine, G.T., et al., *Endocrine and body composition changes across a competitive season in Collegiate speed-power track and field athletes.* The Journal of Strength & Conditioning Research, 2021. **35**(8): p. 2067-2074.

30. Nies, M.A., K.Y. Chen, and J.S.V. Wal, *Energy expenditure, body composition, and biochemical indicators in healthy community women.* International journal of food sciences and nutrition, 2004. **55**(3): p. 237-247.

31. Miah, R., Issahaku, A.R., Uddin, M.N. *et al.* Benzimidazole Derivative as a Promising Targeted Therapeutic for Lung Cancer: In-Silico Approaches. *J Pharm Innov* **21**, 409 (2026). https://doi.org/10.1007/s12247-026-10615-w