

Cortisol responses to exercise stress and training adaptation in human sport science

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Abstract. Exercise induces a coordinated neuroendocrine response that is essential for maintaining homeostasis and supporting adaptation in humans. This mini-review examines the role of the hypothalamic-pituitary-adrenal (HPA) axis and cortisol in the response to exercise stress, with particular emphasis on the non-linear relationship between training load, endocrine responses, recovery, and performance. Cortisol promotes metabolic substrate availability during exercise and contributes to short-term physiological adaptation, but excessive or insufficiently recovered training may alter HPA-axis responsiveness and contribute to maladaptive outcomes. Evidence indicates that acute cortisol responses are strongly dependent on exercise intensity and duration, with responses becoming more pronounced above approximately 60% of maximal oxygen uptake ($\text{VO}_{2\text{max}}$). However, greater endocrine stress does not necessarily translate into greater adaptation, as moderate training doses may provide a more favorable balance between physiological challenge and recovery. Repeated training also modifies HPA-axis behavior, with trained individuals showing altered cortisol responses to standardized exercise and other stressors. Particular attention is given to the distinction between functional overreaching, non-functional overreaching, and overtraining syndrome. Current evidence suggests that resting cortisol concentrations alone have limited diagnostic value because of substantial biological and contextual variability. Dynamic responses to standardized exercise or endocrine stimulation may provide more informative indicators of altered HPA-axis function, although these responses remain heterogeneous. Overall, cortisol should be interpreted as a component of a broader physiological framework rather than as an isolated biomarker of training status or overtraining.

Keywords: hypothalamic-pituitary-adrenal axis, endocrine response, exercise intensity, maximal oxygen uptake, physiological stress, training load, recovery, functional overreaching, overtraining syndrome, hormonal adaptation.

Introduction. In human sport science, exercise represents a controlled physiological stressor that activates neuroendocrine mechanisms involved in energy mobilization, homeostasis, recovery, and training adaptation (Nicula & Arévalo-Sierra, 2026; Perșa & Cenan, 2026). Among these mechanisms, activation of the hypothalamic-pituitary-adrenal (HPA) axis and the resulting cortisol response provide an important, although context-dependent, window into how the human organism responds to different training loads (Petrescu-Mag & Arévalo-Sierra, 2026; Cenan & Perșa, 2026).

This mini-review addresses three linked questions: how the HPA axis mediates exercise stress, why the stress-adaptation relationship is non-linear, and how these mechanisms help distinguish overreaching from overtraining. Cortisol is neither uniformly harmful nor uniformly beneficial in training; its effects depend on exercise intensity, duration, repetition, recovery, and the physiological context in which HPA-axis activation occurs (Duclos & Tabarin, 2016; Anderson et al., 2019).

HPA Axis Physiology. The hypothalamic-pituitary-adrenal axis is a central stress-regulation system activated when exercise threatens homeostasis (Arvidson et al., 2020;

Anderson & Wideman, 2017). At the mechanistic level, hypothalamic CRH (Corticotropin-Releasing Hormone), with AVP (Arginine Vasopressin) acting synergistically, stimulates pituitary ACTH (Adrenocorticotrophic Hormone) release, which then drives adrenal cortisol secretion (Anderson & Wideman, 2017; Ferlazzo et al., 2020). During acute stress, this cascade raises ACTH and cortisol to meet increased metabolic demands, and these responses are normally adaptive rather than pathological (Arvidson et al., 2020; Anderson & Wideman, 2017).

In exercise, cortisol primarily supports substrate availability and short-term homeostasis by promoting gluconeogenesis, lipolysis, and broader metabolic mobilization during activity and recovery (Anderson & Wideman, 2017; Ferlazzo et al., 2020). In resistance exercise specifically, cortisol also participates in tissue remodeling, but this comes partly through inhibition of muscle protein synthesis and stimulation of protein degradation through catabolic pathways such as the ubiquitin-proteasome system (Athanasίου et al., 2023). This dual role explains why cortisol can support adaptation acutely yet become maladaptive when exposure is excessive or prolonged (Li et al., 2025; Gluzicka et al., 2026; Urhausen et al., 1995).

Repeated training changes HPA-axis behavior rather than simply chronically elevating cortisol. Endurance-trained individuals can show normal resting cortisol with altered challenge responses, suggesting adaptation at multiple levels of the axis and target tissues (Duclos & Tabarin, 2016). Highly trained athletes also show preserved diurnal rhythm but a shifted early-morning ACTH and cortisol surge, with higher basal ACTH yet no overall cortisol excess, consistent with pituitary adaptation and relative adrenal blunting (Wittert et al., 1996).

Non-Linear Adaptation. The relationship between stress and adaptation is non-linear rather than monotonic. Across exercise and broader stress physiology, the evidence supports an inverted-U or adaptive-maladaptive continuum in which too little challenge fails to perturb homeostasis enough to induce adaptation, while excessive or repeated stress shifts the response toward fatigue, dysregulation, and impaired function (Li et al., 2025; Gluzicka et al., 2026; Bermejo et al., 2022). This principle appears in both endocrine adaptation and functional outcomes such as performance, recovery, and cognition (Gluzicka et al., 2026; Bermejo et al., 2022).

Acute cortisol responses show a clear threshold effect. In controlled exercise testing, 30 minutes at 40% $\text{VO}_{2\text{max}}$ (maximal oxygen uptake) did not significantly raise cortisol and, after correction, was associated with reduction, whereas 60% and 80% $\text{VO}_{2\text{max}}$ increased cortisol by about 39.9% and 83.1%, respectively (Hill et al., 2008). Reviews converge that cortisol elevations depend on both intensity and duration, with intensities above roughly 60% $\text{VO}_{2\text{max}}$ and durations of at least 10–15 minutes being typical conditions for an increase above resting levels (Duclos & Tabarin, 2016; Budde et al., 2015; Anderson et al., 2019).

Yet more stress is not always better. A recent network meta-analysis found an inverted U-shaped dose-response for cortisol regulation, with moderate exercise doses producing the most favorable effects and diminishing or negative responses at higher loads, including for HIIT (High-Intensity Interval Training) (Li et al., 2025). The same study reported moderate-dose peaks for yoga and continuous aerobic exercise, with excessive loads counteracting benefit through added metabolic and cardiovascular strain (Li et al., 2025).

This non-linearity also explains the difference between productive overload and catabolic excess. Acute or repeated exercise stress can improve regulation of diurnal cortisol rhythm and stress coping when the load is tolerable (Li et al., 2025; Moyers & Hagger, 2023). But when stress is frequent and recovery is inadequate, basal stress signals can rise, diurnal organization can deteriorate, cortisol awakening response (CAR) can change, and prolonged dysregulation can contribute to neuronal injury, inflammatory amplification, circadian disruption, and reduced exercise capacity (Arvidson et al., 2020; Gluzicka et al., 2026; Li et al., 2025; Gluzicka et al., 2026).

Training also modifies how later stressors are handled. Acute vigorous exercise can dampen the cortisol response to a subsequent psychosocial stressor, with stronger exercise-induced HPA activation predicting lower later cortisol reactivity, consistent with negative feedback and short-term stress buffering (Caplin et al., 2021; Zschucke et al., 2014). However, evidence for a chronic cross-stressor adaptation effect is mixed: a six-month aerobic intervention improved fitness but did not outperform controls in reducing laboratory stress reactivity, likely because both groups habituated to the repeated stress test (Arvidson et al., 2020).

Training status further shapes the acute endocrine response. Regular resistance training tends to reduce resting cortisol and attenuate the cortisol response to a standard bout performed at the same absolute load, reflecting adaptation to the repeated stressor (Athanasίου et al., 2023). By contrast, accentuated eccentric loading can preserve acute cortisol, testosterone, and GH (Growth Hormone) responses and may therefore sustain ongoing adaptation when conventional loading becomes endocrinologically muted (Athanasίου et al., 2023) (Table 1; Figure 1).

Table 1

Dose-dependent exercise stress patterns relevant to cortisol and adaptation

<i>Context</i>	<i>Lower stress zone</i>	<i>Adaptive zone</i>	<i>Excessive zone</i>
Acute endurance or mixed exercise	Low intensity may not raise cortisol meaningfully	Moderate to high intensity raises cortisol and supports substrate mobilization	Exhaustive work can depress cortisol for 24–48 h and disrupt HPA regulation
Resistance exercise	Mild protocols may produce little HPA stimulation	Large muscle mass, moderate-high load, high volume, short rest maximize ACTH/cortisol response	Excessive catabolic signaling can inhibit protein synthesis and increase degradation
Stress-regulation interventions	Too little exercise may have limited cortisol effect	Moderate-dose aerobic or mind-body exercise shows best cortisol regulation	Higher HIIT or overall load shows diminishing or adverse cortisol effects.

Dose-response patterns according to literature (Hill et al., 2008; Anderson & Wideman, 2017; Athanasίου et al., 2023; Li et al., 2025). Note: HPA - Hypothalamic–Pituitary–Adrenal axis.

Overreaching and Overtraining. Overreaching and overtraining both arise from an imbalance between training stress and recovery, but they differ in severity, duration, and reversibility (Cadegiani & Kater, 2017; Urhausen et al., 1995; Gluzicka et al., 2026). Functional overreaching is the short-term extension of overload that can precede performance gain after recovery, whereas non-functional overreaching and overtraining syndrome reflect maladaptation with underperformance and fatigue that persist beyond expected recovery windows (Cadegiani & Kater, 2017; Urhausen et al., 1995).

Cortisol alone does not reliably diagnose these states. Systematic and narrative reviews agree that resting cortisol and other basal hormones often remain normal or show overlapping distributions across healthy athletes, overreached athletes, and athletes with overtraining syndrome (Cadegiani & Kater, 2017; Bajor et al., 2026). Diagnostic uncertainty is compounded by strong effects of time of day, sampling method, biological variability, sleep, psychological stress, illness, medications, and endocrine comorbidity (Bajor et al., 2026; Budde et al., 2015).

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The HPA axis links exercise stress to adaptation.

The relationship between training load, cortisol response, recovery and performance is **NON-LINEAR**.

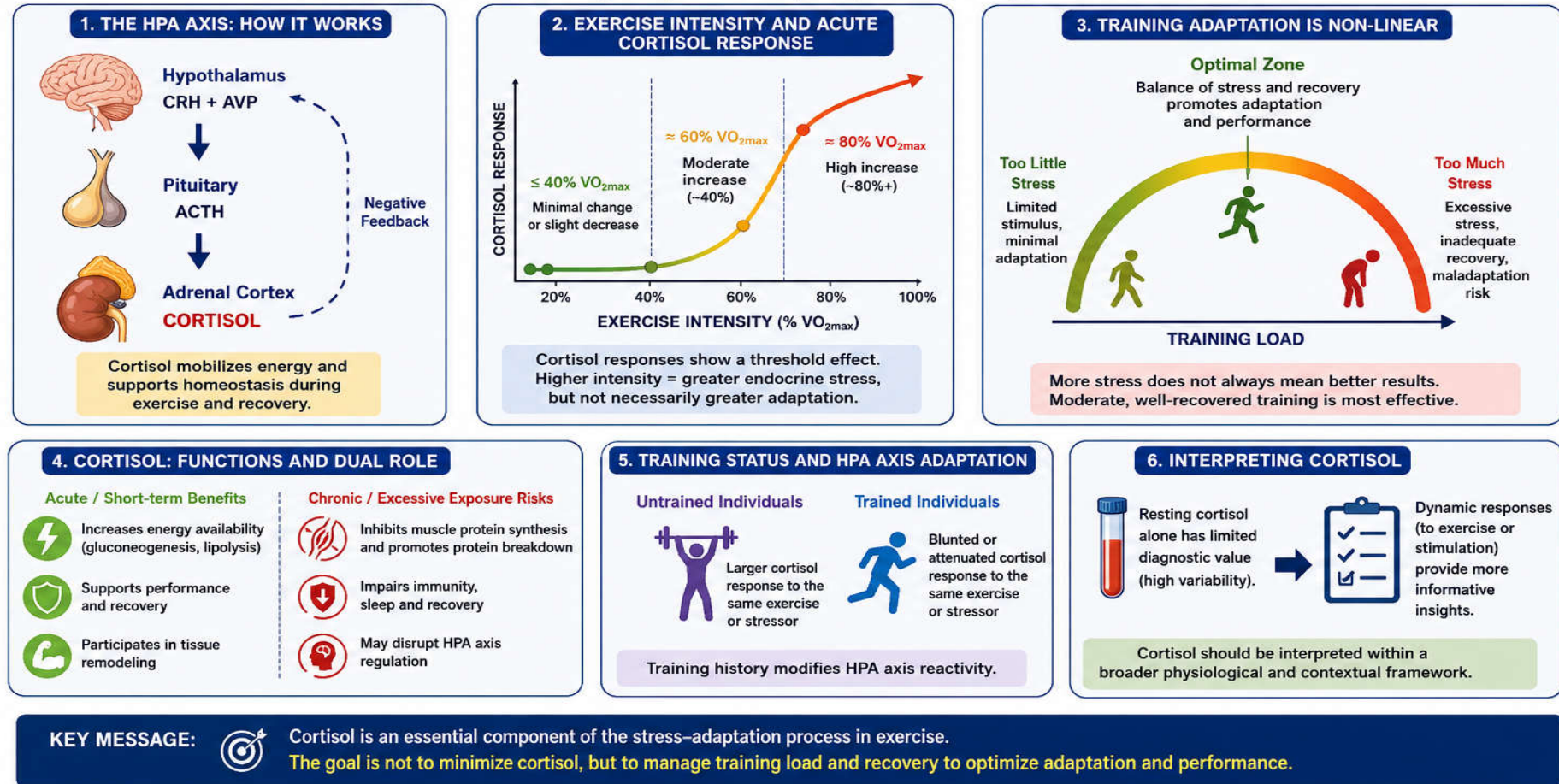


Figure 1. Cortisol responses to exercise stress and training adaptation in human sport science.

The more informative signal appears to be dynamic responsiveness rather than basal concentration. A classic review found that after a standardized exhaustive exercise test, overtrained or overreached athletes often show decreased maximal rises in pituitary hormones, cortisol, and insulin (Urhausen et al., 1995). The 2017 systematic review similarly found that basal markers were usually unhelpful, while ACTH and GH responses to stimulation tests were commonly blunted; cortisol responses were split between normal and blunted results, underscoring heterogeneity rather than a single endocrine signature (Cadegiani & Kater, 2017).

The EROS study adds an important distinction within the HPA axis. Athletes with overtraining syndrome had normal adrenal cortisol responses to cosyntropin, implying preserved adrenal capacity, but reduced cortisol and ACTH responses during insulin tolerance testing and lower cortisol 30 minutes after awakening than healthy athletes (Cadegiani & Kater, 2017). This pattern suggests that overtraining is less an adrenal failure than a loss of the enhanced hormonal conditioning seen in healthy trained athletes (Cadegiani & Kater, 2017).

Shorter-term overload states may also be detectable through blunted responses to a standardized exercise challenge. Intensified training associated with early overreaching has been linked to blunted salivary cortisol and testosterone responses to a 55/80 cycling protocol, and this protocol shows good test–retest reliability for cortisol response in healthy athletes (Hough et al., 2021). Even so, the broader literature does not support using cortisol in isolation as an independent marker for overtraining syndrome (Anderson & Wideman, 2017; Cadegiani & Kater, 2017; Bajor et al., 2026) (Table 2).

Table 2

Typical cortisol-related patterns across adaptive training, functional overreaching, and overtraining syndrome

<i>Training state</i>	<i>Performance context</i>	<i>Cortisol pattern</i>	<i>Interpretation</i>
Well-recovered adaptation	Stable or improving performance	Acute rises with exercise; resting values often normal or lower; challenge responses adapted	Normal training stress with preserved HPA plasticity
Functional overreaching	Temporary fatigue during overload	Cortisol response can become blunted to standardized exercise after intensified training	Possible short-term maladaptation, often reversible
Overtraining syndrome	Persistent underperformance and fatigue	Resting cortisol often non-diagnostic; dynamic ACTH/cortisol responses may be blunted; CAR may be lower	Loss of beneficial hormonal conditioning rather than simple hypercortisolism.

Endocrine features across training states according to literature (Athanasίου et al., 2023; Hough et al., 2021; Urhausen et al., 1995; Cadegiani & Kater, 2017).

Conclusions. Cortisol is an important component of the human physiological response to exercise stress, but its interpretation requires consideration of exercise intensity, duration, training status, recovery, and the broader physiological context. The available evidence supports a non-linear relationship between exercise stress and adaptation: insufficient stress may provide limited stimulus for adaptation, whereas excessive or repeated stress combined with inadequate recovery may impair physiological regulation and performance.

Acute increases in cortisol should therefore not be regarded as inherently detrimental. Rather, they represent an adaptive component of the response to sufficiently demanding exercise, supporting metabolic mobilization and short-term homeostasis. With repeated training, the HPA axis can undergo functional adaptation, resulting in altered cortisol

responses without necessarily producing chronically elevated resting cortisol concentrations.

From a sports-science perspective, the distinction between adaptive training stress, functional overreaching, and overtraining syndrome is particularly important. Resting cortisol alone appears insufficient to reliably discriminate these states. Dynamic endocrine responses to standardized exercise or stimulation may provide additional information about HPA-axis responsiveness, although current evidence indicates substantial inter-individual variability and no single cortisol-based diagnostic signature.

Consequently, cortisol is best regarded as a context-dependent indicator of training stress and physiological adaptation rather than a standalone biomarker of overtraining. Its interpretation should be integrated with performance trajectories, fatigue, recovery status, training load, psychological and environmental stressors, sleep, and other relevant physiological measures.

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References

- Anderson, T., Berry, N. T., & Wideman, L. (2019). Exercise and the hypothalamic-pituitary-adrenal axis: a special focus on acute cortisol and growth hormone responses. *Current Opinion in Endocrine and Metabolic Research*, 9, 74-77. <https://doi.org/10.1016/j.coemr.2019.08.002>
- Anderson, T., & Wideman, L. (2017). Exercise and the cortisol awakening response: A systematic review. *Sports Medicine - Open*, 3, 37. <https://doi.org/10.1186/s40798-017-0102-3>
- Arvidson, E., Dahlman, A., Börjesson, M., Gullstrand, L., & Jonsdottir, I. (2020). The effects of exercise training on hypothalamic-pituitary-adrenal axis reactivity and autonomic response to acute stress — a randomized controlled study. *Trials*, 21, 888. <https://doi.org/10.1186/s13063-020-04803-3>
- Athanasίου, N., Bogdanis, G., & Mastorakos, G. (2023). Endocrine responses of the stress system to different types of exercise. *Reviews in Endocrine & Metabolic Disorders*, 24, 251 - 266. <https://doi.org/10.1007/s11154-022-09758-1>
- Bajor, G., Jurczyk-Florkiewicz, W., Gniadek, Z., Pszczółka, A., Kozak, J., Melka, K., Szulewski, M., Salagierska, A., Machno, P., & Matuszczak, P. (2026). Diagnostic uncertainty in sports medicine: challenges in the interpretation of cortisol testing. *International Journal of Innovative Technologies in Social Science*, 2(1). [https://doi.org/10.31435/ijitss.1\(49\).2026.5021](https://doi.org/10.31435/ijitss.1(49).2026.5021)
- Bermejo, J., Valldecabres, R., Villarrasa-Sapiña, I., Monfort-Torres, G., Marco-Ahulló, A., & Couto, R. D. B. (2022). Increased cortisol levels caused by acute resistance physical exercise impair memory and learning ability. *PeerJ*, 10, e13000. <https://doi.org/10.7717/peerj.13000>
- Budde, H., Machado, S., Ribeiro, P., & Wegner, M. (2015). The cortisol response to exercise in young adults. *Frontiers in Behavioral Neuroscience*, 9, 13. <https://doi.org/10.3389/fnbeh.2015.00013>

- Cadegiani, F., & Kater, C. (2017). Hormonal aspects of overtraining syndrome: a systematic review. *BMC Sports Science, Medicine and Rehabilitation*, 9, 14. <https://doi.org/10.1186/s13102-017-0079-8>
- Cadegiani, F., & Kater, C. (2017). Hypothalamic-pituitary-adrenal (HPA) axis functioning in overtraining syndrome: Findings from endocrine and metabolic responses on overtraining syndrome (EROS)—EROS-HPA axis. *Sports Medicine - Open*, 3, 45 <https://doi.org/10.1186/s40798-017-0113-0>
- Caplin, A., Chen, F., Beauchamp, M. R., & Puterman, E. (2021). The effects of exercise intensity on the cortisol response to a subsequent acute psychosocial stressor. *Psychoneuroendocrinology*, 131, 105336. <https://doi.org/10.1016/j.psyneuen.2021.105336>
- Cenan, C. L., & Perşa, A. D. (2026). Adaptive ceiling in muscle hypertrophy: molecular limits and nutritional modulation. *Human & Veterinary Medicine*, 18(1), 77-83.
- Duclos, M., & Tabarin, A. (2016). Exercise and the hypothalamo-pituitary-adrenal axis. *Frontiers of Hormone Research*, 47, 12-26. <https://doi.org/10.1159/000445149>
- Ferlazzo, A., Cravana, C., Fazio, E., & Medica, P. (2020). The different hormonal system during exercise stress coping in horses. *Veterinary World*, 13, 847-859. <https://doi.org/10.14202/vetworld.2020.847-859>
- Gluzicka, A., Palacz, B., Teper, M., Perz, W., Polus, A., Wrona, J., Kubiś, N., Olczyk, L., Piotrowski, J., Korolchuk, A., Bład, K. S., & Grębosz, D. (2026). Hypothalamic-pituitary-adrenal axis dysregulation and its impact on exercise capacity and recovery in regularly training individuals. *Quality in Sport*, 51, 68663. <https://doi.org/10.12775/qs.2026.51.68663>
- Hill, E., Zack, E., Battaglini, C., Viru, M., Viru, A., & Hackney, A. (2008). Exercise and circulating cortisol levels: The intensity threshold effect. *Journal of Endocrinological Investigation*, 31, 587-591. <https://doi.org/10.1007/bf03345606>
- Hough, J., Leal, D., Scott, G., Taylor, L., Townsend, D., & Gleeson, M. (2021). Reliability of salivary cortisol and testosterone to a high-intensity cycling protocol to highlight overtraining. *Journal of Sports Sciences*, 39, 2080-2086. <https://doi.org/10.1080/02640414.2021.1918362>
- Li, X., Huang, J., & Zhu, F. (2025). The optimal exercise modality and dose for cortisol reduction in psychological distress: A systematic review and network meta-analysis. *Sports*, 13(12), 415. <https://doi.org/10.3390/sports13120415>
- Moyers, S. A., & Hagger, M. (2023). Physical activity and cortisol regulation: A Meta-Analysis. *Biological Psychology*, 179, 108548. <https://doi.org/10.1016/j.biopsycho.2023.108548>
- Nicula, C., & Arévalo-Sierra, J. R. (2026). Exercise-induced low-grade inflammation in bodybuilding/fitness: adaptation vs. chronic risk. *Animal Biology & Animal Husbandry*, 18(1), 69-75.
- Perşa, A. D., & Cenan, C. L. (2026). The performance paradox: nutritional strategies in competitive bodybuilding vs. recreational fitness. *Animal Biology & Animal Husbandry*, 18(1), 76-88.
- Petrescu-Mag, I. V., & Arévalo-Sierra, J. R. (2026). Adipose tissue as an endocrine organ in athletes and bodybuilders. *Human & Veterinary Medicine*, 18(1), 84-90.
- Urhausen, A., Gabriel, H., & Kindermann, W. (1995). Blood hormones as markers of training stress and overtraining. *Sports Medicine*, 20, 251-276. <https://doi.org/10.2165/00007256-199520040-00004>
- Wittert, G., Livesey, J., Espiner, E., & Donald, R. (1996). Adaptation of the hypothalamopituitary adrenal axis to chronic exercise stress in humans. *Medicine and Science in Sports and Exercise*, 28(8), 1015-1019. <https://doi.org/10.1097/00005768-199608000-00011>
- Zschucke, E., Renneberg, B., Dimeo, F., Wüstenberg, T., & Ströhle, A. (2014). The stress-buffering effect of acute exercise: Evidence for HPA axis negative feedback. *Psychoneuroendocrinology*, 51, 414-425. <https://doi.org/10.1016/j.psyneuen.2014.10.019>

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