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Impact of Selected Endocrine Dysfunctions on Physical Performance and Sports Outcomes: A Literature Review

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ABSTRACT

Hormonal regulation is essential for maintaining physiological homeostasis and enabling adaptation to physical stress, making the endocrine system a key factor influencing athletic performance and training responses. Hormones such as thyroid hormones, cortisol, testosterone, and growth hormone regulate metabolism, cardiovascular function, muscle performance, and recovery. Disruptions in endocrine function—resulting from gland disorders or training-related factors such as low energy availability—may impair exercise capacity and adaptation to training. This narrative review summarizes recent literature on the relationship between selected endocrine disorders and sports performance, focusing on thyroid dysfunction, cortisol-related disorders, and pituitary abnormalities. Evidence suggests that thyroid disorders can affect cardiovascular efficiency, neuromuscular performance, and metabolism, while adrenal dysfunction may impair stress responses and exercise tolerance. Hypercortisolism and Cushing syndrome are associated with reduced aerobic capacity, muscle weakness, and sarcopenia, whereas pituitary dysfunction, including that related to traumatic brain injury or relative energy deficiency in sport, can disrupt multiple hormonal axes and influence physical capacity. Overall, endocrine disorders may significantly affect physical performance and athlete health. Early detection, regular hormonal monitoring, and individualized medical and training strategies are essential to support safe training and optimize performance outcomes.

Aim. This study reviews current evidence on how selected endocrine disorders affect athletes' physical performance, exercise capacity, and related physiological processes, highlighting implications for training, recovery, and energy metabolism, and identifying gaps for future research.

Material and methods. A literature review (2020–2025) was conducted using PubMed with keywords on endocrine disorders and athletic performance. Only English, peer-reviewed studies on adults were included, excluding pediatric or unrelated studies. Data on disorder type, population characteristics, and effects on exercise, metabolism, muscle function, and performance were extracted and thematically synthesized.

Results. Endocrine disorders can markedly affect physical performance, exercise tolerance, and training adaptation. Thyroid dysfunction both hypo- and hyperthyroidism—impairs cardiovascular function, muscle performance, and metabolic regulation, while structured exercise and therapy can improve functional capacity and quality of life. Adrenal disorders, including insufficiency and hypercortisolism, alter stress responses, reduce exercise tolerance, and may cause long-term muscle weakness. Pituitary dysfunction disrupts multiple hormonal axes, affecting metabolism, body composition, and physical capacity. Overall, early diagnosis, hormonal monitoring, and individualized medical and exercise strategies are key to optimizing performance and maintaining health in affected individuals.

Conclusion. Endocrine function is crucial for physical performance, training adaptation, and overall athlete health. Thyroid, adrenal, and pituitary disorders can impair exercise tolerance, muscle function, metabolism, and recovery. Appropriate treatment, structured exercise, and lifestyle interventions can improve functional capacity and quality of life. Early diagnosis, regular hormonal monitoring, and individualized management are essential to optimize performance and maintain athlete health.

Keywords: Endocrine Disorders, Exercise Capacity, Athletes, Muscle Function, Metabolism, Hypothyroidism, Hyperthyroidism, Adrenal Insufficiency, Addison's Disease, Cushing's Disease, Hypocortisolism, Hypercortisolism

Introduction

Hormonal regulation plays a central role in maintaining homeostasis and enabling physiological adaptation to physical stress, making the endocrine system a key determinant of athletic performance and training responses. (Mennitti et al., 2024) [1] Physical activity and exercise induce profound changes in the secretion and action of multiple hormones—including testosterone, growth hormone, insulin-like growth factor-1, cortisol, and thyroid hormones—that influence energy metabolism, muscle function, recovery, and cardiovascular adaptation. These endocrine responses support metabolic flexibility, promote anabolic processes, and modulate stress and immune responses, all of which contribute to athletic capacity and adaptation to training stimuli. (Kraemer et al., 2020)[2], (Senefeld et al. 2024)[3]

In competitive athletes, regular monitoring of hormonal status is often integrated into routine medical evaluation, particularly when signs of overtraining, altered metabolism, or unexplained performance decline are present. (Cadegiani et al., 2020)[4] For example, assessments of thyroid function, gonadal hormones, and the hypothalamic-pituitary-adrenal axis may be performed annually or more frequently depending on symptoms, training load, and medical history. These evaluations aid in early identification of endocrine dysfunctions that could compromise health and athletic output. Endocrine disorders can disrupt normal physiological responses to training, impair recovery, and reduce exercise capacity, emphasizing the importance of early detection and management in sport settings. (Cabre et al., 2022; Angelidi et al., 2024)[5][6]

Emerging evidence highlights how both adaptive and maladaptive endocrine changes influence sport performance. Low energy availability—a common issue in athletes with high training volumes—can disrupt hypothalamic-pituitary axes, leading to menstrual dysfunction in female athletes, suppressed gonadal hormone secretion, altered thyroid function, and impaired bone health, which in turn negatively affect endurance, force production, and overall training adaptation. Furthermore, endogenous hormonal differences, such as levels of testosterone, contribute to sex-related disparities in strength, power, and aerobic capacity, which are central to athletic performance. (Ihalainen et al., 2024)[7], (Nokoff et al., 2023)[8]

Disruptions in endocrine function—whether resulting from primary gland dysfunction (e.g., hypothyroidism or adrenal insufficiency) or secondary consequences of chronic training stress (e.g., relative energy deficiency in sport)—may lead to substantial consequences for sport

outcomes. For instance, subclinical thyroid dysfunction has been associated with altered exercise tolerance and energy metabolism, while overtraining-related hypogonadism can reduce anabolic drive and impair performance adaptation. (Hackney et al., 2020)[9]

Despite growing interest in sports endocrinology, the evidence linking specific endocrine disorders to objectively measured sport performance remains limited and scattered. This narrative review aims to synthesize the current literature published from 2020 onward to clarify how selected endocrine dysfunctions influence physical performance and sports outcomes in athletes, highlighting physiological mechanisms, clinical implications, and areas for future research.

Subclinical hypothyroidism

Subclinical hypothyroidism (sHT), defined by elevated TSH levels with normal circulating thyroid hormones, may subtly influence physiological functions relevant to exercise and athletic performance. Levothyroxine (LT4) therapy has been shown to enhance muscle strength, mobility, and inspiratory function in younger and middle-aged adults, although these benefits are inconsistent and less pronounced in older populations. The most robust effects concern cardiac and cardiopulmonary function: LT4 reverses diastolic dysfunction, reduces systemic vascular resistance, and improves submaximal exercise capacity, indicating that functional rather than structural cardiac abnormalities predominate in sHT. In contrast, LT4 exerts limited effects on energy metabolism, substrate utilization, or maximal exercise performance, though it may improve lipid profiles and alleviate certain neuromuscular symptoms. Evidence in athletic populations remains scarce, and potential risks of overtreatment, including arrhythmias, osteoporosis, and muscle wasting, underscore the need for an individualized therapeutic approach (Polakowska et al., 2025)[10].

Supporting these observations, Mainenti et al. (2009) reported that LT4 therapy improved submaximal cardiopulmonary responses during treadmill exercise in patients with sHT over a six-month period. Treated individuals exhibited higher heart rate and minute ventilation, whereas oxygen uptake decreased; no changes were observed in untreated controls. These results reinforce the notion that restoring euthyroidism may enhance functional exercise capacity in subclinical hypothyroidism (Mainenti et al., 2009)[11].

Similarly, Hanke et al. (2023) evaluated the effects of LT4 therapy on physical performance in premenopausal women with sHT. In this cohort, TSH normalization did not significantly alter body weight, composition, energy expenditure, or respiratory quotient, yet functional outcomes improved, including upper- and lower-body strength, hand-grip strength, joint mobility, and measures of explosive power. These findings suggest that while LT4 has limited metabolic impact, it can augment neuromuscular performance and certain aspects of exercise capacity (Hanke et al., 2023)[12].

Beyond pharmacological intervention, Duñabeitia et al. (2023) systematically reviewed the effects of structured exercise programs in hypothyroid individuals. Across ten studies of low-to-moderate methodological quality, aerobic and resistance training were generally safe and associated with improvements in physical fitness and mental health, although pooled analyses revealed no significant effects on TSH, FT3, or FT4 levels. This indicates that while exercise may not directly modify thyroid function, it provides meaningful benefits for overall health and functional capacity (Duñabeitia et al., 2023)[13].

Lifestyle factors further modulate thyroid function. In a community-based cross-sectional study, Wu et al. (2021)demonstrated that individuals with sHT reported poorer sleep quality and that lifestyle behaviors—including exercise, diet, sleep, and smoking—were significantly associated with thyroid homeostasis. Exercise correlated with lower TSH and improved thyroid secretory capacity, whereas iodine excess, smoking, and irregular sleep patterns adversely affected pituitary–thyroid regulation. Notably, poor sleep quality and excessive iodine intake were independently associated with increased sHT risk (Wu et al., 2021)[14]. These findings highlight the potential for lifestyle modification to support thyroid function and overall health in sHT.

The impact of targeted training on cardiovascular responsiveness has also been explored. Almas et al. (2023) reported that 12 weeks of endurance training significantly improved heart rate (HR) kinetics, measured as mean response time (MRT) during cycling exercise, and increased physical activity levels in women with sHT. These results suggest that regular endurance training enhances autonomic cardiovascular function and functional capacity in this population (Almas et al., 2023)[15]. Consistent with this, Almas et al. (2017) found that women with sHT exhibited slower HR kinetics during the transition from rest to exercise compared with euthyroid controls, and that delayed HR responses correlated with lower physical activity and

higher perceived exertion. This supports the hypothesis that subclinical hypothyroidism impairs autonomic regulation, which can be mitigated through targeted physical training (Almas et al., 2017)[16].

Collectively, these studies indicate that while levothyroxine therapy and structured exercise may not substantially alter thyroid hormone levels, they can improve functional capacity, neuromuscular performance, and cardiopulmonary responsiveness in individuals with subclinical hypothyroidism. Moreover, lifestyle factors such as regular physical activity, adequate sleep, and avoidance of smoking or iodine excess may further support thyroid homeostasis and exercise tolerance.

Hypothyroidism

Hypothyroidism, a common endocrine disorder characterized by insufficient thyroid hormone production, can significantly impact physical capacity and overall health. Schok et al. (2023) highlighted that individuals with hypothyroidism often experience fatigue, reduced cardiovascular and muscular function, and impaired pulmonary capacity, which may limit their engagement in regular physical activity. Despite these challenges, evidence suggests that structured exercise can provide multiple benefits, including improved thyroid function, enhanced mood and cognitive performance, and reduction of depressive symptoms. The review emphasizes that incorporating regular physical activity into the management of hypothyroidism supports both physiological health and quality of life, making exercise an important complementary strategy alongside conventional medical treatment. (Schok et al. (2023)[17])

Building on these observations, Sundus et al. (2025) systematically reviewed seven randomized controlled trials examining long-term exercise interventions (≥8 weeks) in adults with hypothyroidism. The meta-analysis demonstrated that regular exercise significantly reduced TSH levels and increased T4 concentrations, indicating improved thyroid function compared with control groups. Evidence quality was rated moderate for TSH and low for T4. The authors concluded that sustained physical activity can serve as a safe and effective adjunct to conventional pharmacological therapy, supporting hormonal regulation and enhancing thyroid function in individuals with hypothyroidism. (Sundus et al. 2025)[18])

Ahmad et al. (2023) further explored the impact of different exercise modalities in 60 hypothyroid women on levothyroxine therapy, comparing aerobic (AT), resistance (RT), and

combined AT/RT training over 12 weeks. All exercise types significantly improved T4, TSH, lipid profile, VO_2 max, and quality of life (QoL), with the combined AT/RT group showing the greatest gains in TSH and mental health-related QoL, while AT alone produced the largest increase in VO_2 max. These findings underscore the importance of structured exercise, particularly combined training, for optimizing thyroid regulation, mental well-being, and physical performance in hypothyroid patients. (Ahmad et al. (2023)[19])

In athletes, Turova et al. (2023) conducted a retrospective analysis of 1,150 individuals aged 15–36 to assess the prevalence of hypothyroidism and its impact on performance. Hypothyroidism affected 9.5% of participants and was associated with elevated exercise heart rate, higher resting diastolic blood pressure, and increased left ventricular end-diastolic dimensions, reflecting adaptations characteristic of the athlete's heart. These athletes also exhibited higher stroke volume, delayed cardiovascular recovery, and a reduced testosterone-to-cortisol ratio, indicating catabolic dominance and limited energy availability. The study highlights the need for regular hormonal screening in athletes to detect early endocrine disturbances and prevent complications arising from intensive training. (Turova et al. 2023)[20])
Similarly, Gierach et al. (2023) investigated metabolic adaptations in 32 female triathletes newly diagnosed with Hashimoto's thyroiditis during 3–6 months of L-thyroxine treatment. Measurements of fat mass (FM), visceral adipose tissue (VAT), BMI, and thyroid function demonstrated significant reductions in FM and VAT, accompanied by improved TSH and fT4 levels. These results highlight the importance of monitoring body composition and thyroid function in endurance athletes, as high energy expenditure may influence treatment outcomes, and bioelectrical impedance analysis can aid optimal management. (Gierach et al. 2023)[21])
Expanding on the interplay between autoimmunity and exercise, Skrzypiec-Spring et al. (2026) reviewed 12 studies on athletes with chronic autoimmune thyroiditis. Moderate physical activity was shown to improve thyroid function and reduce TPO-Ab concentrations, whereas excessive exercise could negatively affect immune regulation. These findings emphasize the potential benefits of structured exercise for both thyroid function and immune response and point to the need for tailored guidelines for athletes and patients with Hashimoto's thyroiditis, as well as monitoring for inappropriate thyroid hormone use in sports. (Skrzypiec-Spring et al. 2026)[22])

At the population level, Shu-Yang Zhang et al. (2024) analyzed NHANES data from 2,118 adults, revealing that total physical activity, measured in metabolic equivalent tasks (MET),

was significantly associated with thyroid hormone indices (TSHI, TFQI), with TFQI correlating with dysthyreosis occurrence. Path analysis indicated that PA modulates dysthyreosis risk via thyroid hormone sensitivity, and restricted cubic spline analysis suggested a non-linear relationship between PA and dysthyreosis. These results suggest that regular physical activity may help prevent dysthyreosis by improving thyroid hormone sensitivity. (Shu-Yang Zhang et al. 2024)[23])

Complementing these findings, Lankhaar et al. (2021) examined PA and sports participation in 1,724 hypothyroid patients on thyroid hormone replacement therapy compared with 1,802 controls. While patients engaged less in moderate-intensity PA, they reported higher sports participation, though two-thirds experienced hypothyroidism-related limitations, particularly in those with autoimmune thyroiditis. These data underline the importance of recognizing exercise constraints in hypothyroid patients and designing interventions to support safe and effective physical activity. (Lankhaar et al. 2021)[24])

Finally, Stuart et al. (2025) assessed thyroid hormone use among elite athletes at the Tokyo 2020, Beijing 2022, and Paris 2024 Olympic Games. Despite no clear performance benefits, 1.7% of athletes reported using T4 or T3, with higher prevalence in females and power sports participants. Use declined to 1.3% at Paris 2024, suggesting that while thyroid hormone misuse remains low, ongoing monitoring and education are warranted to prevent potential health risks. (Stuart et al. 2025)[25])

Overall, these studies collectively highlight the complex interplay between hypothyroidism, physical activity, and athletic performance. Evidence supports that moderate and structured exercise improves thyroid function, metabolic profile, cardiovascular adaptation, and mental well-being in both patients and athletes, while tailored interventions are necessary to accommodate disease-specific limitations. Monitoring of thyroid function, body composition, and hormone use is crucial for optimizing health outcomes, preventing complications, and guiding safe training or rehabilitation programs for individuals with hypothyroidism.

Hypothyroidism-Associated Rhabdomyolysis

Subclinical thyroid dysfunction has been suggested to influence muscle health and the risk of sarcopenia in older adults, even in the absence of overt thyroid disease. Szlejf et al. (2020) investigated this association in 6,974 community-dwelling adults aged 50 and older, classifying

participants as euthyroid, subclinically hypothyroid, or subclinically hyperthyroid, and assessing serum TSH, free T4, FT3, as well as muscle mass via bioelectrical impedance and handgrip strength. Overall, 1.5% of participants had sarcopenia, 20.8% had low muscle mass, and 3.8% had low muscle strength, while subclinical hypothyroidism and hyperthyroidism were present in 9.1% and 0.9%, respectively. Although subclinical thyroid dysfunction was generally not associated with sarcopenia or its components, in older adults TSH exhibited a U-shaped relationship with sarcopenia and low muscle strength, and FT3 levels were negatively associated with muscle mass across age groups, suggesting that subtle thyroid hormone alterations may impact muscle function and sarcopenia risk in middle-aged and older adults. (Szlejf et al. 2020)[26]

Beyond sarcopenia, hypothyroidism can rarely lead to severe muscle complications such as rhabdomyolysis. Baral et al. (2024) reported a case of an 89-year-old woman who developed rhabdomyolysis and acute kidney injury due to severe hypothyroidism without identifiable precipitating factors. Laboratory tests revealed markedly elevated creatine kinase (CK) and critically high TSH with low free thyroxine (FT4). Prompt levothyroxine therapy and fluid resuscitation resulted in clinical improvement and normalization of CK and creatinine levels, highlighting the importance of timely diagnosis, thyroid hormone supplementation, and therapy adherence to prevent life-threatening complications in elderly patients. (Baral et al. 2024)[27]
Similarly, Boryushkina et al., (2019) described a 49-year-old male with hypothyroidism who experienced recurrent severe rhabdomyolysis triggered by muscle injury, seizures, and poor medication adherence, yet did not develop typical complications such as acute kidney injury. This case emphasizes that recurrent rhabdomyolysis, though uncommon, requires careful thyroid monitoring, evaluation for underlying neuromuscular disorders, strict adherence to therapy, and prompt fluid resuscitation to prevent renal complications. (Boryushkina et al., 2019)[28]

Muscle symptoms are also commonly reported at rest and during exercise in patients with hypothyroidism. Guerin et al. (2021) surveyed 580 women under treatment for hypothyroidism and found that those performing a combination of cardiovascular and resistance training (CVRT) experienced the lowest basal muscle symptoms compared to those performing only cardiovascular or resistance exercises or no exercise. Muscle pain and fatigue during exercise were influenced by exercise type and frequency, while recovery improved with more frequent activity, indicating that structured exercise, particularly combined aerobic and resistance training, may reduce muscle symptoms and enhance recovery in hypothyroid women. (Guerin et al. 2021)[29]

The link between thyroid dysfunction and sarcopenia is further supported by genetic evidence. Wei et al. (2024) used Mendelian randomization to assess the causal effects of thyroid dysfunction on sarcopenia-related traits, including hand grip strength, appendicular lean mass (ALM), and walking pace. Hyperthyroidism was associated with reduced ALM, hypothyroidism with lower grip strength and slower walking pace, and subclinical hyperthyroidism with modestly reduced walking speed. These findings indicate that both overt and subclinical thyroid dysfunction can contribute to sarcopenia, reinforcing the importance of monitoring muscle health in thyroid patients. (Wei et al. 2024)[30]

Finally, age-related declines in thyroid hormones can impair muscle regeneration. Oliveira et al. (2025) examined low-dose T4 replacement in aged mice following tibialis anterior injury and found that aged muscle exhibited local resistance to thyroid hormone. T4 treatment did not enhance regeneration; instead, it reduced the number of newly formed fibers, decreased cross-sectional area, and increased cell death and tissue remodeling. These results suggest that, unlike in young individuals, T4 therapy may not support muscle recovery in elderly or sarcopenic patients, highlighting the need for careful consideration of thyroid hormone supplementation in older adults with muscle injury or subclinical hypothyroidism. (Oliveira et al. 2025)[31]

In summary, both subtle and overt thyroid dysfunction can affect muscle health through mechanisms ranging from sarcopenia to rare but severe rhabdomyolysis. Exercise, particularly structured aerobic and resistance training, appears beneficial in mitigating muscle symptoms and preserving function, whereas thyroid hormone therapy in aged or sarcopenic muscle requires careful evaluation. These findings underscore the importance of early thyroid screening, monitoring of muscle health, patient adherence, and individualized therapeutic strategies to prevent complications and optimize musculoskeletal outcomes in individuals with thyroid disorders.

Hypocortisolism

Adrenal insufficiency, characterized by inadequate cortisol production, can be classified as primary, secondary, or glucocorticoid-induced, with the latter representing the most common form. Vaidya et al. (2025) reviewed the causes, diagnosis, and management of adrenal insufficiency, noting that primary forms result from adrenal gland destruction or inhibition, secondary forms from pituitary disorders, and glucocorticoid-induced insufficiency from prolonged supraphysiological steroid exposure. Patients typically present with nonspecific

symptoms such as fatigue, nausea, anorexia, and weight loss, and diagnosis relies on early-morning measurements of cortisol, corticotropin, and dehydroepiandrosterone sulfate (DHEAS), with stimulation testing required in borderline cases. Management involves glucocorticoid replacement, often combined with mineralocorticoids in primary adrenal insufficiency, while patient education regarding stress-dose adjustments is essential to prevent adrenal crisis, a potentially life-threatening complication. (Vaidya et al. 2025)[32]

Understanding adrenal physiology is also important in the context of physical activity, as exercise represents a potent physiological stressor. Vollrath et al. (2025) investigated acute adrenal hormonal responses to resistance training in 19 elite female athletes and observed significant post-exercise reductions in adrenal-derived steroids such as 11β-hydroxyandrostenedione, androsterone, and dehydroepiandrosterone (DHEA), along with decreases in total glucocorticoids, mineralocorticoids, and bioactive androgens. Despite these transient hormonal changes, performance indicators, including velocity loss and estimated one-repetition maximum, remained unchanged across menstrual cycle phases, suggesting that short-term resistance exercise can temporarily modulate adrenal steroidogenesis without compromising athletic performance. (Vollrath et al. 2025)[33]

Beyond athletic settings, broader physiological and psychological stressors may also influence adrenal and neuroendocrine responses. Szivak et al. (2025) reviewed 40 studies involving 3,693 women in tactical professions and reported that occupational stress commonly negatively affects adrenal and neuroendocrine function, body composition, and physical performance. However, limitations in study design and reporting indicate that current knowledge remains incomplete. The authors emphasize the importance of comprehensive research approaches that integrate hormonal regulation, muscle physiology, reproductive health, and nutritional balance to better understand performance readiness and long-term health outcomes in physically demanding occupations. (Szivak et al. 2025)[34]

The endocrine response to exercise has been further explored in relation to the hypothalamic–pituitary–adrenal (HPA) axis and other hormonal systems. Athanasiou et al. (2022) summarized how endurance, high-intensity interval exercise (HIIE), and resistance training influence endocrine and immune responses. Acute endurance or HIIE sessions increase cortisol levels, while chronic endurance training results in moderate basal cortisol elevations, reflecting adaptive mechanisms of the HPA axis. Regular HIIE training may lower basal cortisol and

attenuate catecholamine responses, whereas resistance training induces mild acute HPA activation but may reduce inflammatory markers during long-term training in older adults. These findings highlight that exercise type, intensity, and duration play key roles in shaping endocrine adaptations and should be considered when prescribing physical activity for both healthy and clinical populations. (Athanasiou et al. 2022)[35]

In individuals with adrenal insufficiency, these physiological responses to exercise raise important clinical considerations. Simunkova et al. (2016) evaluated whether an additional pre-exercise dose of hydrocortisone could improve exercise performance in women with Addison's disease. In a randomized cross-over trial involving 10 patients and 10 healthy controls, hydrocortisone increased serum cortisol levels but did not improve maximal aerobic capacity, exercise duration, or metabolic responses to strenuous activity. These results suggest that routine pre-exercise stress dosing may not provide clear benefits during short high-intensity exercise, although further research is needed to evaluate other forms or durations of physical activity. (Simunkova et al. 2016)[36]

In contrast, endurance sports may impose a much greater physiological burden on patients with primary adrenal insufficiency. Bonnecaze et al. (2019) described a case of a 50-year-old endurance athlete with PAI who experienced severe fatigue, nausea, and malaise during marathon events. After adjusting glucocorticoid and mineralocorticoid replacement before competition, the athlete reported reduced symptoms and improved exercise tolerance. This observation highlights the potential value of individualized adrenal replacement strategies for highly active patients and underscores the need for evidence-based guidelines addressing glucocorticoid dosing during prolonged endurance exercise. (Bonnecaze et al. 2019)[37]

Impaired exercise tolerance in patients with adrenal insufficiency may also be related to autonomic dysfunction. Görar et al. (2024) evaluated exercise stress test parameters in 22 patients with Addison's disease and 25 healthy controls and found that although exercise-induced blood pressure and chronotropic responses were similar between groups, patients demonstrated significantly shorter exercise duration and reduced heart rate recovery after exertion. These findings suggest altered autonomic regulation, which may contribute to reduced physical capacity and potentially increased morbidity in this population. (Görar et al. 2024)[38]
In summary, adrenal function plays a central role in the physiological response to stress and physical activity. While exercise can induce adaptive endocrine responses in healthy

individuals, patients with adrenal insufficiency may experience altered hormonal, metabolic, and autonomic responses that affect exercise capacity and recovery. Current evidence indicates that individualized management strategies—including appropriate glucocorticoid replacement, monitoring of stress responses, and tailored physical activity recommendations—are essential to maintain health and functional capacity in these patients. Further research is needed to establish clear clinical guidelines regarding exercise participation and stress-dose adjustments in individuals with adrenal disorders.

Hypercortisolism

Although Olympic athletes are often perceived as exceptionally healthy, biochemical abnormalities may still occur in this population. Ferrera et al. (2025) analyzed blood test results from 2,525 Olympic athletes assessed between the London 2012 and Paris 2024 Games to evaluate hematological and metabolic markers across different sport categories. The study found that 62.6% of athletes had at least one abnormal laboratory result, with hypercortisolemia identified in 15% of cases, making it one of the most frequent findings. Elevated cortisol levels were more common in female athletes than in males (17.1% vs. 13.2%), suggesting possible sex-related differences in stress or hormonal responses to intensive training and competition. These results indicate that even elite athletes may experience increased cortisol levels, highlighting the importance of routine biochemical monitoring to detect endocrine alterations potentially related to chronic training stress and to optimize athlete health and performance. (Ferrera et al. 2025)[39]

Exercise intensity may modulate the hormonal response to subsequent stress through activation of the hypothalamic–pituitary–adrenal (HPA) axis. Caplin et al. (2021) investigated how a single 30-minute bout of exercise at varying intensities affects salivary cortisol responses to a later psychosocial stressor in 83 healthy men. Participants exercised at 30%, 50%, or 70% of heart rate reserve and then underwent the Trier Social Stress Test. Vigorous exercise (70% HRR) elicited a higher immediate cortisol response but attenuated the cortisol reaction to the subsequent stressor, with lower overall cortisol levels, reduced reactivity, and faster return to baseline compared with lower-intensity exercise. This suggests that high-intensity activity may transiently buffer stress responses by priming the HPA axis. (Caplin et al. 2021)[40]

Similarly, Daly examined whether strenuous endurance exercise could raise cortisol in athletes to levels observed in Cushing syndrome. Prolonged high-intensity exercise (~85 min at 75%

VO_2max) produced significant cortisol elevations comparable to those in CS patients, but levels returned near baseline within approximately 90 minutes. These results indicate that while intense training can induce temporary hypercortisolemic states, healthy athletes do not experience the chronic cortisol excess characteristic of Cushing syndrome. (Daly)[41]

Cushing syndrome (CS), an endocrine disorder caused by chronic glucocorticoid excess, leads to persistent reductions in physical capacity despite clinical remission. Roerink et al. (2020) evaluated aerobic exercise in 17 patients in CS remission for more than four years. VO_2peak, maximal workload, and oxygen pulse were significantly lower than in matched controls, yet muscle fiber structure, mitochondrial content, and endothelial markers were unaffected. This suggests that post-remission aerobic impairment is likely driven by persistent cardiovascular dysfunction rather than skeletal muscle alterations. (Roerink et al. 2020)[42]

CS also contributes to skeletal muscle loss and sarcopenia, with detrimental effects on quality of life. Martel-Duguech et al. (2021) found higher sarcopenia prevalence in women in CS remission (19% vs. 3% in controls), associated with increased intramuscular fat, reduced strength, and lower scores on SarQoL, CushingQoL, and SF-36 questionnaires. The sarcopenia index (SI) correlated with impaired performance and quality of life, supporting its use as a practical biomarker. (Martel-Duguech et al. 2021)[43]

Functional deficits extend beyond muscle mass: Assimakopoulou et al. (2015) showed that CS patients had increased fat mass, decreased quadriceps strength and endurance, and reduced 6-minute walking distances, while daily activity measured via accelerometry was lower and inversely correlated with cortisol levels. These findings emphasize the impact of hypercortisolism on overall functional capacity and daily physical activity. (Assimakopoulou et al. 2015)[44]

Glucocorticoid-induced myopathy in CS can persist long-term. Vogel et al. (2020) reported that hand grip strength decreased from 83% of controls at diagnosis to 71% six months after surgical remission, with no recovery during follow-up. Chair rising performance improved initially but remained below control levels, and predictors of persistent dysfunction included age, waist-to-hip ratio, and baseline HbA1c. Muscle weakness strongly correlated with reduced quality of life, underscoring the need for targeted interventions. (Vogel et al., 2020)[45]

The pathophysiology of CS-associated myopathy is multifactorial, involving protein degradation via the FOXO3 pathway, intramuscular fat accumulation, and inactivity-induced atrophy. Reincke et al. (2021) highlighted that surgical remission is essential for recovery, but long-term muscle function often remains impaired, influenced by age, comorbidities, metabolic status, and postoperative IGF-1 levels. Early postoperative worsening is common, with only partial recovery, and genetic variability in glucocorticoid sensitivity may explain individual differences. Growth hormone replacement and targeted physiotherapy are being investigated to improve outcomes. (Reincke et al. 2021)[46]

Bone health is also severely affected in Cushing's disease. Zdrojowy-Wełna et al. (2024) report fractures in 15–78% of patients at diagnosis, often occurring despite normal BMD, and persisting after treatment. Risk factors include severe hypercortisolism, delayed diagnosis, hypogonadism, low BMD/TBS, and advanced age. Recommendations focus on fracture screening and assessment of bone quality, while prospective studies are needed to define therapeutic thresholds, timing, and the efficacy of interventions such as Wnt-signaling anabolic agents. (Zdrojowy-Wełna et al., 2024)[47]

In summary, both transient hypercortisolemia in athletes and chronic cortisol excess in Cushing syndrome profoundly impair muscle function, aerobic capacity, and bone integrity. These hormonal disturbances can limit athletic performance, increase injury risk, and reduce quality of life, highlighting the importance of monitoring endocrine status, managing training loads, and implementing targeted interventions to preserve musculoskeletal health in both elite athletes and patients with CS.

Hyperthyroidism

Hyperthyroidism, particularly in the context of Graves' disease, profoundly impacts physical performance, muscle function, metabolism, and cardiovascular adaptations. A growing body of evidence highlights how both overt thyroid hormone excess and its management can influence exercise capacity, functional outcomes, and long-term health in affected individuals. Yılmaz et al. (2024) evaluated the impact of newly diagnosed hyperthyroidism (NDH) on physical and physiological function, comparing 16 patients at diagnosis and after achieving euthyroid status with healthy controls. The study assessed pulmonary function, exercise capacity (6-minute walking test), respiratory muscle strength and endurance, physical activity levels, dyspnea, and quality of life. Results showed that NDH patients had significantly reduced 6-minute walking

distance, respiratory muscle endurance, and maximal inspiratory/expiratory pressures, along with higher dyspnea (MMRC) and poorer thyroid-specific quality of life (ThyPRO) scores compared to controls. Even after normalization of thyroid function, patients remained less physically active and exhibited persistent deficits in exercise tolerance and respiratory endurance, despite similar pulmonary function test results. These findings indicate that hyperthyroidism adversely affects functional capacity, respiratory muscle performance, physical activity, and quality of life, emphasizing the need for early evaluation and referral to cardiopulmonary rehabilitation programs. (Yılmaz et al. 2024)[48]

Karmisholt et al. (2020) investigated weight changes during the first year of antithyroid drug (ATD) treatment in 13 sedentary patients with new-onset hyperthyroidism, assessing correlations with thyroid hormone levels, resting energy expenditure (REE), physical activity, and energy efficiency. Over the year, patients experienced a significant weight increase (68.9–74.1 kg) concurrent with normalization of free T3 (17.5 to 4.42 pmol/L) and a decrease in REE (1,630–1,484 kcal/24 h). Improvements in energy efficiency at low workloads were minor, and daily physical activity levels remained unchanged. Individual analyses showed that weight gain correlated primarily with reductions in REE, while changes in energy efficiency or activity had minimal impact. These findings suggest that post-treatment weight gain in hyperthyroid patients is mainly driven by metabolic adaptations rather than lifestyle factors or exercise capacity. (Karmisholt et al. 2020)[49]

Di Gioia et al. (2024) examined 1,342 Olympic athletes to determine whether thyroid hormones within the euthyroid range influence exercise-induced cardiac remodeling. Athletes underwent blood tests for TSH, fT3, and fT4, along with echocardiography and exercise-stress testing, excluding those on thyroid medication or with thyroid disorders. Endurance athletes showed the lowest TSH, fT3, and fT4 levels compared with other disciplines. Across all sports, fT3 positively correlated with resting and peak heart rate, interventricular septum thickness, and left ventricular end-diastolic volume, particularly in power, skill, and endurance athletes. These findings indicate that even subtle variations in thyroid hormones within the normal range can modulate cardiac adaptations to training, contributing to exercise-induced structural and functional remodeling. (Di Gioia et al. 2024)[50]

Diep et al. (2025) reported a case highlighting the relationship between hyperthyroidism and exercise-related myopathy in a patient with Graves' disease. A 25-year-old man initially

presented with hyperthyroidism and was treated with radioactive iodine, which led to hypothyroidism requiring levothyroxine replacement. Several months after treatment, he developed exercise-induced muscle spasms, pain, and fatigue affecting the upper body muscles. Electromyography initially showed irritable myopathy, although extensive investigations, including antibody panels and muscle biopsy, did not reveal an alternative etiology. The authors note that both hyperthyroidism and rapid correction of thyroid hormone excess may disrupt skeletal muscle metabolism, impair mitochondrial oxidative capacity, and promote type II muscle fiber dysfunction, which can manifest as exercise-induced muscle weakness and fatigue. Partial symptom improvement after dantrolene suggested involvement of altered muscle excitation–contraction coupling. This case underscores that thyroid hormone excess and its rapid normalization may contribute to persistent myopathic symptoms and reduced exercise tolerance in patients with Graves' disease. (Diep et al. 2025)[51]

Ren et al. (2024) investigated immunometabolic responses to repeated high-intensity intermittent exercise (HIIE) in 25 women with newly diagnosed Graves' hyperthyroidism compared with 25 healthy controls. Patients with hyperthyroidism showed significantly elevated FT3 and FT4 levels, suppressed TSH, and reduced skeletal muscle index, indicating early muscle loss. During repeated Wingate tests, both groups demonstrated declines in peak and mean power; however, individuals with hyperthyroidism exhibited a distinct fatigue pattern with a greater reduction in mean power across repetitions, suggesting impaired tolerance to sustained high-intensity effort. Exercise induced significant increases in lactate, glucose, leptin, creatine kinase, irisin, and inflammatory cytokines such as IL-6 in both groups, although baseline inflammatory profiles and the magnitude of some responses differed in hyperthyroid patients. Network analysis further indicated complex interactions between thyroid hormones, inflammatory markers, and muscle mass, with TSH playing a regulatory role in inflammatory responses. These findings suggest that while newly diagnosed hyperthyroidism may not markedly reduce short-term explosive power, it alters immunometabolic responses to exercise and may compromise endurance during repeated high-intensity activity. (Ren et al. 2024)[52]

Brun et al. (2014) described the case of a 38-year-old triathlete and swimming instructor with recurrent Graves' disease who trained 8–12 hours per week. Despite biochemical control of hyperthyroidism with carbimazole therapy, the athlete reported reduced performance and symptoms suggestive of overtraining. Exercise testing showed a VO_2max of 58.2 mL/kg/min (147% of predicted), yet this value was estimated to be about 25% lower than his pre-

hyperthyroid performance, with a ventilatory threshold at 55% of VO_2max. The athlete also experienced persistent relative tachycardia and discomfort during training, reflected in a high score on the SFMS overtraining questionnaire. The authors suggest that even when hyperthyroidism appears well controlled according to standard clinical criteria, residual sympathetic overactivity may persist in athletes, potentially leading to decreased performance and physiological responses resembling overreaching. (Brun et al. 2014)[53]

Cutovic et al. (2021) evaluated the short- and long-term effects of a structured exercise program in euthyroid patients with Graves' disease using a retrospective case–control design involving 124 participants. The intervention group completed a three-week program including daily walking, strengthening, and stretching exercises, while the control group engaged in leisure activities. Following the intervention, the exercise group demonstrated significant improvements in estimated peak oxygen consumption, reduced fatigue levels, and favorable changes in thyroid hormone profile, including decreased thyroxine and a trend toward increased thyrotropin levels. Additionally, antithyroid medication was discontinued within six months in a substantially greater proportion of exercising patients compared with controls (84% vs. 18%), while relapse rates within 12 months were lower in the exercise group (29% vs. 72%). These findings suggest that structured physical activity may improve aerobic capacity and fatigue while potentially supporting better long-term thyroid regulation in patients with Graves' disease. (Cutovic et al. 2021)[54]

Summary: Collectively, these studies demonstrate that hyperthyroidism impairs exercise tolerance, muscle function, metabolic efficiency, and cardiovascular adaptations, with effects persisting even after biochemical normalization. Subtle variations in thyroid hormones can influence cardiac remodeling and performance in athletes, while rapid correction of hyperthyroidism may precipitate myopathic symptoms. Structured exercise programs appear beneficial, improving aerobic capacity, reducing fatigue, and supporting long-term thyroid regulation. These findings highlight the importance of early assessment, tailored rehabilitation, and monitored physical activity for patients with hyperthyroidism to optimize functional and clinical outcomes.

Pituitary gland disorders

Traumatic brain injury (TBI) is a significant global health concern, often leading to long-term complications that impact physical and endocrine function. According to Gilis-Januszewska et

al. (2020), TBI “may lead to permanent or transient pituitary insufficiency that causes adverse changes in body composition, worrisome metabolic function, reduced bone density, and a significant decrease in one’s quality of life.” Post-traumatic hypopituitarism (PTHP) is challenging to diagnose due to subtle clinical manifestations, variable progression, and a lack of standardized algorithms, with the somatotropic axis most commonly affected, followed by hypogonadism, hypothyroidism, hypocortisolism, and diabetes insipidus. Risk factors for long-term PTHP include intracranial hypertension, initial TBI severity, diffuse axonal injury, age, ICU stay, rapidly fluctuating hormone levels, basal skull fractures, and anti-pituitary antibodies, while acute cortisol deficits and persistent GH deficiency predict worse outcomes. Most cases are transient, with recovery of LH/FSH function occurring first, followed by GH and ACTH, though delayed or chronic hypopituitarism can develop. Investigations into biomarkers such as glial fibrillary acidic protein, ubiquitin C-terminal hydrolase-L1, αII-spectrin breakdown products, specific antibodies, miRNAs, and neuro anti-inflammatory proteins aim to improve early detection and prognostication, emphasizing the need for systematic screening and monitoring. (Gilis-Januszewska et al. 2020)[55]

Sports-related TBIs, even mild ones, can similarly result in pituitary dysfunction. Kara et al. (2025) highlight that growth hormone deficiency is the most prevalent hormonal abnormality in athletes following TBI, with potential impacts on metabolism, body composition, and performance. Hormonal deficits may improve over time, yet new deficiencies can emerge, making monitoring during both acute and recovery phases essential. Management is complicated by uncertain efficacy of GH replacement and concerns over its potential misuse in sports, underscoring the need for large-scale studies to clarify epidemiology, pathophysiology, and preventive strategies for sports-related hypopituitarism. (Kara et al. 2025)[56]

Claessen et al. (2024) further emphasize that even mild TBI can lead to clinically significant endocrine disturbances, particularly in female athletes. In a cohort of 133 female athletes, 12.2% exhibited pituitary dysfunction, including hypopituitarism (4.6%) and hyperprolactinemia (7.6%), with some cases of hyperprolactinemia occurring without prolactinoma, likely reflecting hypothalamic or pituitary injury. These findings highlight the importance of careful hormonal monitoring and tailored post-concussion care to identify and manage pituitary abnormalities. (Claessen et al. 2024)

Beyond TBI, relative energy deficiency in sport can also induce reversible pituitary dysfunction. Skrzypiec-Springal et al. (2024) describe a 33-year-old athlete with low TSH and free T3 despite normal free T4, revealing reversible hypogonadism and thyroid axis dysregulation due to insufficient energy availability. This condition illustrates that even athletes of normal body weight can experience pituitary and thyroid dysfunction when energy intake does not meet metabolic and training demands, with potential reversibility through appropriate nutritional and training interventions. (Skrzypiec-Springal et al., 2024)[58]

Pituitary dysfunction may also arise from congenital conditions, such as pituitary stalk interruption syndrome (PSIS). Sun et al. (2022) reported an 18-year-old male with PSIS who presented with exercise intolerance. Cardiopulmonary exercise testing revealed moderate functional impairment primarily due to physical deconditioning rather than cardiovascular or pulmonary limitations. This case demonstrates the utility of CPET for diagnostic evaluation and individualized exercise prescription to safely improve functional capacity in patients with pituitary hormone deficiencies. (Sun et al. 2022)[59]

In summary, pituitary dysfunction—whether post-traumatic, sports-related, energy-deficiency–related, or congenital—can substantially affect endocrine, metabolic, and exercise capacity. Early recognition through systematic screening, hormonal evaluation, and functional testing is crucial, and tailored interventions including nutritional support, endocrine management, and individualized exercise programs can optimize recovery and long-term outcomes.

Discussion

This narrative review highlights the complex relationship between endocrine regulation and physical performance, emphasizing that disturbances in major hormonal axes may significantly influence exercise capacity, metabolic balance, and adaptation to training. Hormones such as thyroid hormones, cortisol, growth hormone, and sex steroids play essential roles in maintaining physiological homeostasis during physical activity, and disruptions in their regulation may impair both athletic performance and overall health outcomes in physically active individuals [1–3].

Our analysis indicates that thyroid dysfunction represents one of the most frequently discussed endocrine factors affecting physical performance. Both subclinical and overt hypothyroidism may negatively influence cardiovascular function, neuromuscular performance, and exercise

tolerance. Several studies demonstrate that levothyroxine therapy may improve cardiopulmonary responses and certain aspects of muscle function, although its effects on maximal aerobic performance and metabolic efficiency appear limited [10–12]. Importantly, non-pharmacological interventions such as structured physical activity may significantly improve functional capacity, quality of life, and neuromuscular performance in individuals with thyroid dysfunction [13,19]. Furthermore, lifestyle factors including physical activity, sleep quality, and nutritional habits may modulate thyroid homeostasis and influence the risk of developing thyroid dysfunction [14].

In individuals with overt hypothyroidism, fatigue, reduced muscle strength, impaired pulmonary function, and decreased physical activity levels are commonly reported [17]. Nevertheless, growing evidence suggests that regular exercise interventions can improve thyroid function, metabolic parameters, and aerobic capacity, particularly when combining aerobic and resistance training modalities [18,19]. These findings support the inclusion of structured exercise programs as an adjunct to pharmacological therapy. However, some individuals with hypothyroidism may still experience exercise-related limitations, particularly in the presence of autoimmune thyroid disease or long-standing hormonal imbalance [24].

The relationship between thyroid disorders and muscle health also deserves particular attention. Thyroid dysfunction has been associated with sarcopenia, impaired muscle regeneration, and reduced muscle strength [26,30]. In rare cases, severe hypothyroidism may lead to complications such as rhabdomyolysis, particularly when diagnosis or treatment is delayed [27,28]. Nevertheless, evidence suggests that structured exercise, particularly programs combining cardiovascular and resistance training, may alleviate muscle symptoms and improve functional recovery in hypothyroid patients [29]. These findings highlight the importance of maintaining adequate physical activity while carefully monitoring thyroid status and muscle health.

Adrenal function represents another critical component of endocrine regulation in physically active populations. The hypothalamic–pituitary–adrenal (HPA) axis plays a central role in physiological responses to exercise-induced stress, regulating energy mobilization, inflammation, and cardiovascular adaptation [35]. In patients with adrenal insufficiency, impaired cortisol production may reduce exercise tolerance and delay cardiovascular recovery following physical exertion [32,38]. Evidence suggests that individualized glucocorticoid

replacement strategies and appropriate stress-dose adjustments may be necessary to maintain safe participation in physical activity in these individuals [37].

Conversely, chronic cortisol excess may also negatively affect physical performance. Hypercortisolism, particularly in the context of Cushing syndrome, is associated with significant reductions in aerobic capacity, skeletal muscle strength, and bone integrity [42,43]. Even after clinical remission, patients may experience persistent physical impairment due to long-term metabolic and cardiovascular alterations induced by prolonged glucocorticoid exposure [45,46]. In contrast, transient elevations in cortisol observed during intense exercise in athletes represent a physiological adaptive response and do not lead to the chronic metabolic consequences seen in pathological hypercortisolism [40,41].

Hyperthyroidism represents another endocrine condition that may significantly impair exercise tolerance and muscular performance. Excess thyroid hormone increases metabolic rate and sympathetic activity, which may initially enhance energy turnover but ultimately contributes to muscle fatigue, reduced endurance capacity, and impaired respiratory muscle performance [48]. Studies also suggest that even subtle variations in thyroid hormone concentrations within the euthyroid range may influence cardiac remodeling and physiological adaptations to training in athletes [50]. Furthermore, persistent functional limitations may occur even after restoration of euthyroidism, highlighting the potential long-term impact of thyroid hormone imbalance on skeletal muscle and cardiovascular function [48,52].

Another important aspect highlighted in this review concerns pituitary dysfunction, particularly in relation to traumatic brain injury (TBI) and relative energy deficiency in sport. Post-traumatic hypopituitarism may affect multiple hormonal axes, including growth hormone, gonadal hormones, and cortisol, potentially impairing body composition, metabolic regulation, and exercise capacity [55,56]. Even mild sports-related TBIs have been associated with clinically relevant endocrine disturbances, emphasizing the need for systematic hormonal monitoring in athletes with concussion history [57]. Additionally, relative energy deficiency in sport may induce reversible pituitary and thyroid axis suppression, demonstrating the strong interaction between nutrition, endocrine regulation, and athletic performance [58].

Despite increasing research interest in sports endocrinology, several limitations remain within the current body of evidence. Many studies involve small sample sizes, heterogeneous

populations, or observational designs, which limits the ability to establish causal relationships between endocrine dysfunction and athletic performance. Additionally, relatively few investigations have focused specifically on elite athletic populations, and standardized measures of physical performance are not consistently applied across studies.

Future research should therefore focus on large-scale longitudinal studies evaluating endocrine function, metabolic responses, and performance outcomes in athletic populations. Integrating hormonal monitoring with physiological performance testing, nutritional assessment, and body composition analysis may provide a more comprehensive understanding of the interactions between endocrine health and athletic performance.

Overall, the findings of this review emphasize that endocrine disorders represent an important yet often underrecognized factor influencing physical performance and recovery. Early identification of hormonal disturbances, combined with individualized medical management, appropriate training strategies, and adequate nutritional support, may help optimize both athlete health and performance outcomes.

Results

The reviewed literature demonstrates that endocrine disorders can significantly influence physical performance, exercise tolerance, and physiological adaptation to training. The findings consistently indicate that disturbances in thyroid, adrenal, and pituitary function affect cardiovascular responses, muscle performance, metabolic regulation, and recovery capacity in both athletes and physically active individuals.

In subclinical hypothyroidism, levothyroxine therapy and structured exercise programs were associated with improvements in neuromuscular performance, cardiopulmonary responses, and functional capacity, although their effects on maximal exercise performance and metabolic parameters were limited. Studies also highlighted the role of lifestyle factors such as regular physical activity and adequate sleep in supporting thyroid homeostasis and exercise tolerance.
In overt hypothyroidism, multiple studies demonstrated reduced exercise capacity, fatigue, impaired cardiovascular and muscular function, and decreased physical activity levels. However, structured exercise interventions—including aerobic, resistance, and combined training—consistently improved thyroid hormone profiles, VO_2max, lipid metabolism, and quality of life. In athletes, hypothyroidism was associated with altered cardiovascular responses

to exercise, delayed recovery, and a lower testosterone-to-cortisol ratio, suggesting a catabolic physiological state. Rare but severe complications such as rhabdomyolysis were also reported, emphasizing the importance of early diagnosis and treatment.

Research on adrenal disorders showed that adrenal insufficiency may impair exercise tolerance due to altered hormonal stress responses and autonomic dysfunction. Patients demonstrated shorter exercise duration and delayed heart rate recovery compared with healthy controls. While glucocorticoid replacement therapy is essential for management, additional pre-exercise dosing did not consistently improve performance outcomes. In contrast, exercise in healthy individuals produced adaptive responses within the hypothalamic–pituitary–adrenal axis depending on exercise intensity and duration.

Studies examining hypercortisolism indicated that elevated cortisol levels—whether transient in athletes or chronic in conditions such as Cushing syndrome—are associated with reduced aerobic capacity, muscle weakness, sarcopenia, and decreased physical activity. Even after remission of Cushing syndrome, persistent reductions in aerobic performance and muscle strength were observed, suggesting long-term effects of chronic glucocorticoid exposure.

In hyperthyroidism, newly diagnosed patients demonstrated reduced exercise tolerance, respiratory muscle strength, and physical activity levels, with persistent deficits even after normalization of thyroid hormone levels. Hyperthyroidism also altered immunometabolic responses to high-intensity exercise and could contribute to exercise-induced muscle fatigue or myopathy. Nevertheless, structured exercise programs were shown to improve aerobic capacity, fatigue levels, and long-term thyroid regulation in patients with Graves' disease.

Finally, pituitary disorders, including those resulting from traumatic brain injury, relative energy deficiency in sport, or congenital abnormalities, were found to disrupt multiple hormonal axes and negatively affect metabolism, body composition, and physical capacity. Post-traumatic hypopituitarism and growth hormone deficiency were particularly associated with reduced exercise tolerance and long-term functional impairment.

Overall, the reviewed studies indicate that endocrine dysfunctions can substantially influence sport performance and physiological adaptation to training. Regular hormonal screening, early diagnosis, and individualized treatment strategies—including medical therapy, nutritional

management, and appropriately prescribed exercise—appear essential for maintaining health and optimizing physical performance in affected individuals.

Conclusions

Endocrine function plays a key role in regulating physical performance, training adaptation, and overall athlete health. Disorders affecting the thyroid, adrenal, and pituitary glands may impair exercise tolerance, cardiovascular responses, muscle function, metabolism, and recovery. Thyroid dysfunction is one of the most common endocrine issues affecting physically active individuals. Hypothyroidism is associated with fatigue, reduced aerobic capacity, and muscle symptoms, while hyperthyroidism can lead to decreased exercise tolerance and muscle weakness. In many cases, appropriate treatment and structured exercise programs can improve functional capacity and quality of life.

Adrenal disorders may alter the physiological stress response to exercise. Adrenal insufficiency can reduce exercise tolerance, whereas hypercortisolism is linked to muscle weakness, reduced aerobic capacity, and long-term functional impairment. Pituitary dysfunction may disrupt multiple hormonal axes, affecting metabolism, body composition, and physical capacity.
Overall, endocrine disorders can significantly influence sports performance and athlete health. Early diagnosis, regular hormonal monitoring, and individualized medical and training strategies are important for maintaining safe training and optimizing performance.

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