The Role of Physical Activity in Enhancing Cardiopulmonary Endurance Among Individuals with Hyperthyroidism: A Narrative Review

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ABSTRACT

Hyperthyroidism, characterised by excessive thyroid hormone production, creates a hypermetabolic state that profoundly affects cardiovascular, respiratory, and musculoskeletal systems. Patients commonly experience reduced cardiopulmonary endurance, muscle weakness, fatigue, and diminished quality of life. While antithyroid medications effectively normalise hormone levels, functional recovery is often incomplete. This narrative review provides a comprehensive synthesis of the pathophysiological mechanisms linking hyperthyroidism to impaired exercise capacity and evaluates the therapeutic potential of structured physical activity and exercise training. Drawing on clinical studies, physiological research, and related literature, the review highlights how moderate aerobic, resistance, and combined exercise interventions—implemented after biochemical stabilisation—can improve VO_2 peak, six-minute walk distance, muscle strength, balance, and overall functional outcomes. Challenges, safety considerations, and directions for future research are discussed. Targeted exercise programmes may serve as a valuable adjunct to conventional management, promoting holistic recovery in this population.

Keywords:hyperthyroidism, Graves' disease, physical activity, exercise training, cardiopulmonary endurance, VO_2 peak, thyroid hormones, rehabilitation

How to cite this article: Kulkarni A, Naik V. The Role of Physical Activity in Enhancing Cardiopulmonary Endurance Among Individuals with Hyperthyroidism: A Narrative Review. Int J Drug Deliv Technol. 2026;16(70s): 596-600. DOI: 10.25258/ijddt.16.70s.59

1. Introduction

Hyperthyroidism represents one of the most common endocrine disorders worldwide, with a notable predilection for women and individuals in the working-age group.[1] The condition arises primarily from autoimmune stimulation in Graves' disease, autonomous nodule formation in toxic multinodular goitre, or inflammatory processes in thyroiditis.[2] Excessive circulating triiodothyronine (T3) and thyroxine (T4) accelerate cellular metabolism, increase oxygen consumption, and induce widespread sympathetic activation.[3] These changes manifest clinically as resting tachycardia, palpitations, heat intolerance, weight loss despite increased appetite, muscle weakness, and profound fatigue.[4]

The cardiovascular system is particularly vulnerable. Thyroid hormones upregulate beta-adrenergic receptor sensitivity and expression, enhance myocardial contractility, and reduce systemic vascular resistance.[5] While this initially augments cardiac output to meet heightened metabolic demands, prolonged exposure leads to cardiac strain, left ventricular hypertrophy, atrial fibrillation, and reduced exercise reserve.[6] Respiratory involvement includes weakness of diaphragmatic and intercostal muscles, contributing to exertional dyspnoea and inefficient ventilation.[7] Skeletal muscle undergoes accelerated protein catabolism, resulting in thyrotoxic myopathy characterised by proximal weakness, reduced endurance, and impaired neuromuscular coordination.[8]

These physiological derangements collectively impair cardiopulmonary endurance—the ability to sustain prolonged physical activity—which is commonly assessed through maximal oxygen uptake (VO_2 peak), cardiopulmonary exercise testing (CPET), and field tests such as the six-minute walk test (6MWT).[9] Patients with untreated or poorly controlled hyperthyroidism demonstrate significantly lower exercise tolerance compared to euthyroid controls, with deficits correlating to the degree of hormonal excess.[10] Even after successful restoration of euthyroidism, many individuals report persistent symptoms that limit occupational performance, social participation, and overall quality of life.[11]

Traditional management focuses on antithyroid drugs (e.g., carbimazole, methimazole), beta-blockers for symptomatic relief, radioiodine ablation, or thyroidectomy.[12] While these interventions effectively normalise thyroid function

tests (TSH, free T3, free T4), they do not automatically restore physical capacity. Residual deficits in aerobic fitness, muscle strength, and daily activity levels are well-documented.[13] This observation has fuelled interest in non-pharmacological adjuncts, particularly structured physical activity and exercise therapy.[14]

Physical activity encompasses any bodily movement produced by skeletal muscles that results in energy expenditure, while exercise refers to planned, structured, and repetitive movements aimed at improving or maintaining physical fitness.[15] In the context of hyperthyroidism, exercise may counteract deconditioning, enhance mitochondrial biogenesis, improve endothelial function, and support psychological well-being.[16] Population-based studies suggest bidirectional relationships between physical activity levels and thyroid function, with higher activity associated with more favourable metabolic profiles.[17]

Despite this rationale, clinical guidelines provide limited specific recommendations for exercise prescription in hyperthyroid patients.[18] Concerns regarding safety—particularly the risk of arrhythmias or cardiovascular events during exertion in the thyrotoxic state—have historically led to cautious approaches.[19] This narrative review aims to bridge this gap by synthesising available evidence on the effects of hyperthyroidism on cardiopulmonary endurance and critically examining the role of physical activity as an adjunctive therapeutic modality.

2. Pathophysiology of Impaired Cardiopulmonary Endurance in Hyperthyroidism

The hypermetabolic milieu of hyperthyroidism imposes multiple, interconnected stresses on the oxygen transport and utilisation chain. At the cardiac level, thyroid hormones directly influence gene expression of contractile proteins, calcium-handling proteins (e.g., SERCA2), and ion channels.[20] This results in shortened diastolic filling time, increased myocardial oxygen demand, and eventual diastolic dysfunction despite preserved or increased ejection fraction.[21] Longitudinal echocardiographic studies have demonstrated reversible hyperdynamic right ventricular function in Graves' disease that normalises with antithyroid treatment, yet subtle impairments in cardiac reserve may persist.[22]

Vascular adaptations include reduced systemic vascular resistance and increased arterial compliance, which, combined with tachycardia, produce a high-output state.[23] Over time, this can lead to eccentric left ventricular remodelling and heightened risk of supraventricular arrhythmias, particularly atrial fibrillation, which further compromises cardiac efficiency during exercise. [24]

Pulmonary function is affected through both direct and indirect mechanisms. Reduced respiratory muscle strength decreases vital capacity and maximal inspiratory/expiratory pressures. [25] Increased ventilatory drive secondary to heightened metabolic rate and altered chemosensitivity leads to inefficient breathing patterns and early fatigue during exertion.[26]

Hyperthyroidism imposes multifaceted burdens on cardiopulmonary endurance that extend beyond biochemical correction.[11,14] The hypermetabolic state leads to cardiovascular hyperactivity, skeletal muscle catabolism, and respiratory inefficiency, all of which compromise exercise tolerance even after treatment.[12,16] Restoration of euthyroidism improves cardiac output and certain functional parameters such as 6MWD, yet VO_2 peak and muscle performance often show incomplete recovery, leaving patients vulnerable to persistent fatigue and reduced quality of life.[13,18,19]

These residual deficits are consistent with broader literature demonstrating that thyroid hormone excess induces long-term alterations in cardiac efficiency, vascular function, and muscle metabolism.[11,20] Structured physical activity addresses these gaps by promoting cardiovascular adaptations, muscle anabolism, and improved oxygen utilisation.[22,23] Higher physical activity levels are associated with better metabolic and functional profiles, suggesting a protective role that could be harnessed therapeutically.[24,25]

From a rehabilitation perspective, moderate aerobic and resistance training can safely enhance endurance and strength once patients are biochemically stable.[26,27] Patient education on pacing and gradual return to activity is crucial to prevent overexertion.[28] The integration of exercise into multidisciplinary care aligns with successful approaches in other endocrine and cardiovascular conditions.[21] However, the scarcity of dedicated randomised trials in hyperthyroidism limits definitive recommendations.[29] Future studies should evaluate standardised protocols with consistent outcomes including VO_2 peak, muscle strength, and quality-of-life measures.[30]

Overall, while antithyroid therapy remains the cornerstone of management, incorporating physical activity offers a promising avenue to achieve more comprehensive recovery and improve long-term patient outcomes. [10,25]

3. Effects of Standard Treatment on Functional Outcomes Antithyroid therapy leads to rapid biochemical improvement, often achieving euthyroidism within weeks to months. Functional gains generally parallel hormonal normalisation; however, recovery remains incomplete in many patients. Prospective evaluations have shown

significant increases in six-minute walk distance and normalisation of cardiac output parameters following restoration of euthyroidism, yet improvements in aerobic capacity (VO_2 peak) are typically modest. [13]

Body composition changes during treatment are notable, with patients frequently gaining weight, predominantly as lean mass. Baseline free T3 levels appear to predict the extent of lean mass recovery. [19] Muscle strength and balance improve progressively but may not reach levels comparable to euthyroid controls even after several months. [20] Daily physical activity levels, often assessed by step counts, frequently remain below expected values, indicating persistent behavioural and deconditioning factors that benefit from targeted intervention. [21]

These findings emphasise the limitations of relying solely on pharmacological approaches and underscore the importance of incorporating rehabilitation strategies that specifically target neuromuscular and cardiovascular adaptations.[22]

4. The Role of Physical Activity and Structured Exercise

4.1 Mechanisms of Benefit Regular physical activity promotes favourable adaptations across multiple physiological systems. Aerobic training enhances mitochondrial density, capillary proliferation, and stroke volume, thereby improving oxygen delivery and utilisation. Resistance exercise counters muscle catabolism, stimulates hypertrophy, and enhances neuromuscular efficiency. [23] Combined training regimens may provide synergistic benefits for overall functional capacity. [24]

In the context of thyroid disorders, exercise may additionally modulate inflammatory pathways and improve insulin sensitivity, indirectly aiding metabolic recovery. Psychological benefits, including reduced anxiety, improved mood, and greater self-efficacy, are particularly relevant given the psychosocial burden associated with hyperthyroidism.[25]

4.2 Evidence from Clinical Studies Although dedicated randomised controlled trials examining exercise interventions in hyperthyroidism are limited, supportive observational evidence is available. Patients reporting higher levels of physical activity tend to demonstrate better functional outcomes during antithyroid treatment.[2,7]Cross-sectional studies indicate that individuals with hyperthyroidism who maintain regular exercise exhibit superior exercise capacity compared with their sedentary counterparts. [2]

Insights drawn from related thyroid research suggest that exercise can improve VO_2 peak, muscle strength, and quality of life, principles that are likely applicable during the recovery phase following thyrotoxicosis. [26]

4.3 Practical Exercise Recommendations

- **Timing**: Structured exercise should be initiated only after achieving clinical and biochemical stabilisation (e.g., normal TSH and free T4 levels, absence of uncontrolled arrhythmias).
- **Aerobic Training**: Moderate intensity (40–60% heart rate reserve or Borg scale 11–13), aiming for 150 minutes per week through activities such as walking, cycling, or swimming.
- **Resistance Training**: Two to three sessions per week targeting major muscle groups with progressive overload.
- **Balance and Flexibility**: Incorporate exercises to reduce fall risk and improve coordination.
- **Monitoring**: Use heart rate, perceived exertion, and symptom tracking; high-risk patients require medical supervision.
- **Progression**: Implement gradual, individualised progression with adequate recovery periods.

Safety remains paramount. High-intensity or competitive exercise should be avoided until full stabilisation is confirmed, with appropriate screening for underlying cardiac complications.[12]

5. Challenges, Limitations, and Future Directions

Current evidence is limited by small sample sizes, heterogeneous interventions, and predominance of observational designs. Standardised outcome measures and long-term follow-up are needed. Future trials should employ randomised designs, objective physical activity monitoring (e.g., accelerometry), and comprehensive assessment of both physiological and patient-reported outcomes.

Personalised approaches incorporating genetic factors, disease severity, and comorbidities would enhance clinical applicability. Integration of digital health tools for remote monitoring could improve adherence.

6. Conclusion

Hyperthyroidism exerts significant, multifaceted effects on cardiopulmonary endurance that extend

beyond biochemical normalisation. Structured physical activity and exercise training offer a safe, evidence-informed adjunctive strategy to optimise functional recovery, muscle performance, and quality of life. By addressing residual deficits through targeted rehabilitation, clinicians can provide more holistic care for patients with hyperthyroidism. High-quality research is urgently required to establish definitive guidelines and exercise protocols tailored to this population.

Ethics and consent statement: Ethical approval was not required as the study used previously published data no individual patient's data were included.

Competing interests: The authors declare that they have no competing interests.

Funding information: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author's contributions: All others contributed to the study. Anushka Kulkarni drafted the manuscript. Anushka Kulkarni and Varun Naik critically reviewed and revised the content. All authors read and approve the final manuscript.

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