FOLIA MEDICA CRACOVIENSIA
Vol. LXV, 2, 2025: 49–58
PL ISSN 0015-5616 eISSN 2957-0557
DOI: 10.24425/fmc.2025.156123

Influence of levothyroxine supplementation on athletic performance in subclinical hypothyroidism — a review of the literature

Natalia Polakowska[1], Paweł Skowron[1], Maciej Dyda[2], Izabella Kiselowska[3], Kamil Skowron[4]

[1] 4th Military Clinical Hospital, Wrocław, Poland
[2] University Clinical Hospital of Jan Mikulicz-Radecki in Wroclaw, Poland
[3] 5th Military Clinical Hospital, Kraków, Poland
[4] Department of Pathophysiology, Faculty of Medicine, Jagiellonian University Medical College, Kraków, Poland

Corresponding author: Paweł Skowron, M.D.
4th Military Clinical Hospital
ul. Rudolfa Weigla 5, 50-981 Wrocław, Poland
Phone: +48 795 128 364; E-mail: pawel.marek.skowron@gmail.com

Abstract: Subclinical hypothyroidism (sHT), defined by elevated thyroid-stimulating hormone levels with preserved circulating thyroid hormone concentrations, may subtly affect physiological processes relevant to athletic performance. Emerging evidence suggests that levothyroxine (LT4) therapy can enhance muscle strength, mobility, and cardiopulmonary reserve, particularly in younger and middle-aged populations. This review critically examines current data on the physiological effects of levothyroxine in subclinical hypothyroidism, with a focus on its potential implications for athletic performance. The most consistent findings relate to cardiac physiology: LT4 therapy reverses diastolic dysfunction, reduces systemic vascular resistance, and enhances cardiopulmonary reserve, suggesting that functional cardiac abnormalities in sHT are largely reversible. In contrast, effects on energy metabolism remain limited. Patients with sHT exhibit impaired exercise tolerance, altered substrate utilization, and increased lactate accumulation, and these disturbances are only partially ameliorated by LT4. Overall, LT4 therapy in sHT shows promise in improving selected physiological parameters, yet evidence for meaningful enhancement of overall exercise capacity or athletic performance remains weak. Until robust, athlete-specific data are available, treatment decisions should continue to follow established clinical criteria rather than anticipated performance gains.

Keywords: subclinical hypothyroidism, levothyroxine, athletic performance, thyroid hormones, performance enhancement.

Submitted: 04-May-2025; **Accepted in the final form:** 20-Jun-2025; **Published:** 31-Jul-2025.

Introduction

Subclinical hypothyroidism (sHT) is defined by elevated serum thyroid-stimulating hormone (TSH) levels with normal concentrations of free thyroxine (T4) and triiodothyronine (T3) [1]. Its prevalence ranges from 3% to 15%, depending on the characteristics of the study population [2, 3], and is more common in women and older adults [4]. Data on the frequency of thyroid disorders in athletes are scarce; however, a Russian study of 1,000 elite athletes reported a 9.5% prevalence of sHT [5].

Thyroid hormones are key endocrine regulators of physiological processes essential for athletic performance. Acting primarily through T4 and T3, they play a central role in energy balance, thermogenesis, and lipid and glucose metabolism [6, 7]. Additionally, thyroid hormones interact with both central and peripheral metabolic signals to fine-tune the body's metabolic responses [6]. Beyond their metabolic actions, thyroid hormones are critical for skeletal muscle development, regeneration, and contractile function [8]. Importantly, thyroid status and physical activity are interconnected — exercise can modulate thyroid function, while thyroid dysfunction can impair performance and recovery [7, 9, 10].

Intense exercise training has been associated with alterations in thyroid hormone levels, though the underlying mechanisms remain incompletely understood [10]. Loucks *et al.* demonstrated that intense athletic training in women affects thyroid hormone levels, especially in those with amenorrhea [11]. Athletes with regular cycles had reduced T4, while amenorrheic athletes had broader decreases in T4, T3, and related hormones, suggesting that menstrual dysfunction may exacerbate exercise-induced thyroid alterations. In contrast, Matsumura *et al.* found no evidence of training-induced hypothyroidism in nonelite female distance runners, although individuals who began competitive training before the age of 10 exhibited a threefold higher prevalence of hypothyroidism [12]. These findings underscore the complexity of the exercise–thyroid axis.

Both hypothyroidism and hyperthyroidism are linked to impairments across cardiovascular, metabolic, neuromuscular, musculoskeletal, and neuropsychiatric systems, collectively reducing exercise tolerance, energy metabolism, and recovery capacity [7, 13]. Animal studies further support the role of thyroid hormone homeostasis in optimizing exercise performance. Both deficient and excessive T3 states have been shown to impair physical capacity; however, interventions such as endurance training or exogenous T3 supplementation partially ameliorated the deficits observed in hypothyroid subjects. Notably, these restorative effects were accompanied by an increased accumulation of lactate during exercise, indicating altered metabolic responses under conditions of modified thyroid hormone status [14]. In humans, women with sHT demonstrate lower physical activity levels, reduced muscle strength, and diminished functional capacity in the six-minute walk test compared with euthyroid controls [15].

Current guidance emphasizes a conservative, risk-stratified approach to sHT. In adults, levothyroxine (LT4) therapy is generally recommended when TSH persistently exceeds 10 mIU/L, particularly in individuals younger than 65–70 years. For milder elevations (TSH 4–10 mIU/L), treatment may be considered only in the presence of clinically significant symptoms, positive thyroid autoimmunity, goiter, or specific circumstances such as pregnancy planning [16]. In older adults — especially with TSH ≤10 mIU/L — a watchful-waiting strategy is preferred due to limited benefit and greater risk of overtreatment. According to guidelines, most non-pregnant adults with sHT derive no clinically relevant benefit from routine levothyroxine therapy [17]. Applied to sport, these criteria imply that the majority of athletes with sHT — typically young, asymptomatic, and with modest TSH elevations — will not meet guideline thresholds for pharmacological treatment,

and are better served by structured monitoring and targeted management of contributory factors. Given these findings, the present review aims to critically evaluate the available literature on the effects of levothyroxine treatment on physical performance outcomes in patients with subclinical hypothyroidism, to better inform clinical decision-making regarding the potential functional benefits as well as risks of such therapy in athletes.

Strength and mobility

There is limited and inconsistent evidence regarding neuromuscular dysfunction in sHT. Several studies have documented a higher prevalence of neuromuscular symptoms and electromyographic abnormalities in individuals with sHT, suggesting subclinical impairments in muscle function [2, 18–20]. Notably, proximal muscle strength appears to be diminished in individuals with sHT. For instance, a Brazilian research group conducted a cross-sectional study demonstrating a higher frequency of neuromuscular complaints and reduced muscle strength in sHT patients compared to matched controls [20]. Conversely, other investigations suggest that subclinical hypothyroidism exerts minimal or no significant effects on muscle mass, strength, or muscle quality, particularly in elderly populations [21]. A large-scale study involving adults aged 65 years and older reported no significant differences in muscle mass, strength, or quality between euthyroid and sHT patients. However, it was noted that women with sHT exhibited a marginally increased prevalence of sarcopenia according to one diagnostic criterion [21].

Intervention studies provide additional insights, such as a recent study by Hanke *et al.*, which examined the influence of levothyroxine therapy on body composition and physical performance in sHT. The study enrolled 25 women (mean age 27.4 ± 5.8) who were evaluated at baseline, after two months of levothyroxine treatment, and once TSH levels had normalized within the reference range. The intervention was associated with consistent improvements in muscle performance: chest press, leg extension, grip strength (right hand), shoulder and hip mobility, as well as explosive strength, all of which showed measurable gains. Endurance capacity also improved, as reflected by a higher anaerobic threshold. The authors concluded that while levothyroxine does not have a beneficial impact on body composition, energy expenditure, or respiratory quotient, it may enhance strength, mobility, and endurance performance [22]. Similarly, a double-blind, randomized, placebo-controlled trial evaluated the impact of levothyroxine therapy on neuromuscular function in 71 participants with sHT, predominantly middle-aged women (mean age 51 ± 10 years), who were randomized to receive levothyroxine (n = 35) or placebo (n = 36). Muscle strength was assessed in both peripheral and respiratory domains: pelvic and scapular girdle strength by manual muscle testing, quadriceps strength by standardized isometric dynamometry, and inspiratory strength by maximum static inspiratory mouth pressure. After six months of maintained euthyroidism, levothyroxine therapy was associated with a significant improvement in inspiratory muscle strength, whereas no meaningful changes were observed in quadriceps performance or pelvic and scapular girdle strength [23].

In contrast to findings in younger populations, evidence from older adults suggests limited benefits of LT4 therapy on physical performance. Netzer *et al.* conducted an ancillary analysis of two randomized, placebo-controlled trials including 267 participants aged ≥65 years (mean age 77.5 years) with persistent subclinical hypothyroidism (TSH 4.6–19.9 mIU/L, normal free thyroxine). Participants were randomized to receive levothyroxine with TSH-guided dose adjustments or placebo with sham titration to preserve blinding. After a median follow-up of 18 months, levothyroxine

effectively lowered TSH levels compared with placebo; however, no differences were observed in the primary endpoint of gait speed. Similarly, secondary outcomes — including handgrip strength and annual change in muscle mass — showed no significant group differences [24].

Taken together, current evidence suggests that levothyroxine therapy may improve selected measures of muscle strength or mobility in younger and middle-aged individuals with subclinical hypothyroidism; however, these findings are based on small trials and remain limited to isolated parameters rather than consistent gains in overall performance capacity. In older adults, robust randomized data indicate no significant benefit of levothyroxine on muscle mass, strength, or functional outcomes, underscoring the age-dependent and context-specific nature of therapeutic effects.

Cardiopulmonary function

Minimal changes in thyroid function observed in patients with subclinical hypothyroidism lead to significant cardiac abnormalities, including impaired left ventricular (LV) diastolic function at rest, characterized by delayed LV relaxation, as well as LV systolic dysfunction during physical exertion [25, 26]. LT4 treatment in patients with sHT has been shown to exert beneficial effects on cardiopulmonary function, although the extent of improvement varies across studies [27, 28].

Mainenti *et al.* reported in two trials that normalization of TSH with levothyroxine enhanced submaximal exercise performance and cardiopulmonary reserve in sHT patients. Improvements were noted in ventilatory and gas exchange parameters, while post-exercise recovery did not significantly change [27, 28]. Similarly, Brenta *et al.* demonstrated that sHT is characterized by prolonged time to peak filling rate — a marker of diastolic dysfunction — which normalized after six months of levothyroxine therapy, underscoring the reversibility of these impairments [29].

Additional echocardiographic and hemodynamic studies support these findings, though results are somewhat heterogeneous [30–33]. Arem *et al.* found largely unchanged cardiac function after levothyroxine therapy, aside from modest improvements in LV diastolic dimension and preejection period [30]. In contrast, Biondi and Monzani reported more pronounced benefits: six months of treatment improved systolic performance, reduced systemic vascular resistance, and significantly enhanced diastolic indices, with post-treatment parameters approaching those of euthyroid controls [29, 30] Faber *et al.* extended these observations by documenting reductions in mean arterial pressure, increases in cardiac output, and decreases in systemic vascular resistance after levothyroxine replacement in women with sHT, suggesting that even subclinical dysfunction induces a spectrum of hemodynamic changes that can be partly reversed with therapy [33]. Collectively, these studies indicate that levothyroxine therapy in subclinical hypothyroidism can improve selected aspects of cardiac and cardiopulmonary function — particularly LV diastolic performance, systemic vascular resistance, and ventilatory reserve — while effects on recovery kinetics and overall exercise tolerance remain less consistent. The variability of findings likely reflects differences in study populations, baseline cardiovascular health, and treatment duration, but overall supports the view that sHT exerts subtle yet reversible effects on cardiovascular physiology.

Energy metabolism

Although overt hypothyroidism is well established to induce muscle dysfunction, characterized by alterations in muscle architecture and reduced activity of enzymes involved in glucose metabolism, the effects of sHT on muscle energy metabolism remain underexplored [34, 35]. Monzani *et al.*

investigated the clinical and biochemical manifestations of muscle dysfunction in sHT by quantifying skeletal muscle lactate and pyruvate production at rest and during dynamic arm exercise. Their findings demonstrated that blood lactate levels during exercise were significantly elevated in sHT patients compared to healthy controls, with differences emerging from moderate exercise intensity onward. The authors hypothesized that impaired mitochondrial function underlies this metabolic disturbance. Furthermore, the increase in blood lactate concentration correlated positively with the duration of subclinical hypothyroidism but showed no association with circulating thyroid hormone levels [36].

Caraccio *et al.* further examined exercise metabolism by comparing 23 patients with sHT to 10 euthyroid controls using incremental exercise testing. Individuals with sHT displayed reduced maximal power output and VO_2max, elevated heart rates at submaximal workloads, and a sharper rise in respiratory quotient, indicating altered substrate preference. They also exhibited increased circulating lactate, pyruvate, free fatty acids, and glycerol both at rest and during exercise. Although levothyroxine treatment improved neuromuscular symptoms, it did not normalize exercise metabolism or substrate responses after six months of therapy, suggesting that these disturbances may be only partially reversible [37].

Additional metabolic evidence comes from an Italian study involving 38 women with mild sHT, which found that six months of levothyroxine therapy significantly improved lipid parameters — including reductions in total and LDL cholesterol — while indices of glucose metabolism remained unaffected [38].

Taken together, current data suggest that subclinical hypothyroidism impairs exercise tolerance and alters substrate utilization, with a shift toward greater reliance on glycolysis and lipid mobilization. Levothyroxine therapy appears to improve some metabolic risk factors, such as dyslipidemia, but has a limited impact on exercise-related energy metabolism and recovery within the time frames studied. These findings imply that while thyroid hormone replacement can address certain cardiovascular and biochemical abnormalities, its ability to restore normal metabolic efficiency during exercise remains uncertain.

Discussion

To date, no studies have directly assessed the impact of levothyroxine supplementation on athletic performance in athletes with subclinical hypothyroidism. Nevertheless, evidence from clinical and physiological studies in non-athlete populations provides useful insights into pathways potentially relevant for sports.

Across studies, levothyroxine therapy appears to exert selective physiological benefits. In younger and middle-aged adults, treatment has been associated with improvements in specific measures of muscle function, such as strength, mobility, and inspiratory performance [22–24]. These effects, however, are inconsistent and generally confined to isolated parameters rather than reflecting broad improvements in overall performance capacity. By contrast, in older individuals (≥65 years), robust randomized data show no significant impact of levothyroxine on muscle strength, mass, or functional outcomes [24].

The most consistent evidence of benefit relates to cardiac and cardiopulmonary function. Multiple studies demonstrated that minimal thyroid dysfunction in sHT leads to cardiac abnormalities, which appear reversible with levothyroxine therapy [29, 31, 32]. Normalization of thyroid hormone levels improves both systolic and diastolic function, as evidenced by shortened

isovolumic relaxation time, increased E/A ratio (early to late diastolic mitral flow velocity), and reduced systemic vascular resistance. These improvements indicate that cardiac abnormalities in sHT are functional rather than structural. Treatment has also been linked to improvements in submaximal exercise performance and ventilatory reserve [27, 28], although recovery dynamics and maximal exertion outcomes remain largely unaffected.

In contrast, the effects of levothyroxine on exercise metabolism are limited. Subclinical hypothyroidism alters substrate utilization and reduces exercise tolerance, yet short-term therapy does not normalize patterns of energy metabolism or substrate oxidation [37]. While levothyroxine treatment has been shown to relieve neuromuscular symptoms in some patients with subclinical hypothyroidism, its metabolic effects are more selective: improvements are consistently observed in lipid parameters, with reductions in total and LDL cholesterol, whereas glucose metabolism and muscle bioenergetics remain largely unaffected [38]. This discrepancy between symptomatic relief and persistent metabolic dysfunction highlights the complexity of tissue-specific responses and suggests that longer treatment durations may be required to evaluate full metabolic recovery.

Several gaps in the literature need to be addressed. First, there is a lack of direct evidence on athletes, in whom even subtle physiological changes may influence performance, as current evidence is predominantly derived from non-athlete populations. Second, age-related differences in treatment response remain poorly understood and warrant targeted investigation. Third, the long-term impact of levothyroxine on exercise metabolism and recovery kinetics is unknown, as most trials have been limited to ≤12 months. Fourth, treatment protocols in existing studies are designed to normalize TSH rather than optimize functional outcomes, leaving questions regarding dosing strategies for performance enhancement unresolved. Finally, potential sex-specific differences in treatment response have not been systematically studied.

It is also important to acknowledge the risks of overtreatment. Excessive levothyroxine use may lead to osteoporosis, cardiac arrhythmias, heart failure, muscle wasting, reduced quality of life, and cognitive impairment [37]. Thus, the uncertain performance-related benefits must be weighed against these established risks, particularly in athletic contexts where supraphysiological use of thyroid hormones has been reported [39].

This review has several limitations. As a non-systematic synthesis, it is subject to selection bias, and the primary studies included are heterogeneous in sample size, study design, and outcome measures. Many were small trials without placebo controls, reducing their statistical power and limiting generalizability. Differences in patient characteristics, such as thyroid autoimmunity status, were not consistently reported and may influence therapeutic response. Moreover, long-term data remain scarce, leaving questions about the durability of observed effects unanswered.

Conclusion

Levothyroxine supplementation in subclinical hypothyroidism has been linked to improvements in muscle strength, cardiac performance, and cardiopulmonary reserve, whereas its effects on energy metabolism, substrate utilization, and recovery kinetics appear modest. While such physiological adaptations could potentially enhance athletic performance, current evidence does not justify routine substitution in athletic populations. Establishing levothyroxine as a standard intervention for athletes with subclinical hypothyroidism requires robust, high-quality clinical trials that address long-term outcomes and sport-specific endpoints. Moreover, thyroid hormone alterations observed in athletes may, in many cases, represent adaptive responses to intensive training

rather than true endocrine pathology. Consequently, therapeutic decisions should not rely solely on biochemical thresholds but should incorporate age, symptom burden, performance context, and the possibility of adaptive hormonal fluctuations. A cautious, individualized, and integrative approach remains the most appropriate strategy for supporting both health and performance in athletes with suspected subclinical hypothyroidism.

Funding

This publication was prepared without any external source of funding.

Conflict of interest

None declared.

References

1. *Cooper D.S., Biondi B.*: Subclinical thyroid disease. Lancet Lond Engl. 2012 Mar 24; 379 (9821): 1142–1154.
2. *Canaris G.J., Manowitz N.R., Mayor G., Ridgway E.C.*: The Colorado thyroid disease prevalence study. Arch Intern Med. 2000 Feb 28; 160 (4): 526–534.
3. *Vanderpump M.P., Tunbridge W.M., French J.M., Appleton D., Bates D., Clark F., et al.*: The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. Clin Endocrinol (Oxf). 1995 Jul; 43 (1): 55–68.
4. *Hollowell J.G., Staehling N.W., Flanders W.D., Hannon W.H., Gunter E.W., Spencer C.A., et al.*: Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab. 2002 Feb; 87 (2): 489–499.
5. *Tenyaeva E., Turova E., Golovach A., Badtieva V., Artikulova I.*: The influence of subclinical hypothyroidism on physical performance of elite athletes. Br J Sports Med. 2021 Nov 1; 55 (Suppl 1): A173–A173.
6. *Mullur R., Liu Y.Y., Brent G.A.*: Thyroid hormone regulation of metabolism. Physiol Rev. 2014 Apr; 94 (2): 355–382.
7. *Gavriilidou M., Chorti A., Psomiadou A., Koidou E., Papaioannou M., Papavramidis T.*: Thyroid Gland Disorders and Physical Activity: Can They Affect Each Other? Cureus. 2025 Mar; 17 (3): e81489.
8. *Bloise F.F., Cordeiro A., Ortiga-Carvalho T.M.*: Role of thyroid hormone in skeletal muscle physiology. J Endocrinol. 2018 Jan; 236 (1): R57–68.
9. *Huang W.S., Yu M.D., Lee M.S., Cheng C.Y., Yang S.P., Chin H.M.L., Wu S.Y.*: Effect of treadmill exercise on circulating thyroid hormone measurements. Med Princ Pract. 2004; 13 (1): 15–19.
10. *Ciloglu F., Peker I., Pehlivan A., Karacabey K., Ilhan N., Saygin O., Ozmerdivenli R.*: Exercise intensity and its effects on thyroid hormones. Neuro Endocrinol Lett. 2005 Dec; 26 (6): 830–834.
11. *Loucks A.B., Laughlin G.A., Mortola J.F., Girton L., Nelson J.C., Yen S.S.*: Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. J Clin Endocrinol Metab. 1992 Aug; 75 (2): 514–518.
12. *Matsumura M.E., Bucciarelli M., Perilli G.*: Relationship Between Training Intensity and Volume and Hypothyroidism Among Female Runners. Clin J Sport Med. 2015 Nov; 25 (6): 551–553.
13. *Luksch J.R., Collins P.B.*: Thyroid Disorders in Athletes. Curr Sports Med Rep. 2018 Feb; 17 (2): 59–64.
14. *Zarzeczny R., Pilis W., Langfort J., Kaciuba-Uściłko H., Nazar K.*: Influence of thyroid hormones on exercise tolerance and lactate threshold in rats. J Physiol Pharmacol. 1996 Sep; 47 (3): 503–513.
15. *Tanriverdi A., Ozcan Kahraman B., Ozsoy I., Bayraktar F., Ozgen Saydam B., Acar S., et al.*: Physical activity in women with subclinical hypothyroidism. J Endocrinol Invest. 2019 Jul; 42 (7): 779–785.

16. *Pearce S.H.S., Brabant G., Duntas L.H., Monzani F., Peeters R.P., Razvi S., Wemeau J.-L.*: 2013 ETA Guideline: Management of Subclinical Hypothyroidism. Eur Thyroid J. 2013 Dec; 2 (4): 215–228.
17. *Bekkering G.E., Agoritsas T., Lytvyn L., Heen A.F., Feller M., Moutzouri E., et al.*: Thyroid hormones treatment for subclinical hypothyroidism: a clinical practice guideline. BMJ. 2019 May 14; 365: l2006.
18. *Cooper D.S., Halpern R., Wood L.C., Levin A.A., Ridgway E.C.*: L-Thyroxine therapy in subclinical hypothyroidism. A double-blind, placebo-controlled trial. Ann Intern Med. 1984 Jul; 101 (1): 18–24.
19. *Monzani F., Caraccio N., Del Guerra P., Casolaro A., Ferrannini E.*: Neuromuscular symptoms and dysfunction in subclinical hypothyroid patients: beneficial effect of L-T4 replacement therapy. Clin Endocrinol (Oxf). 1999 Aug; 51 (2): 237–242.
20. *Reuters V.S., Buescu A., Reis F.A.A., Almeida C.P., Teixeira P.F. dos S., Costa A.J.L., et al.*: Avaliação clínica e da função muscular em pacientes com hipotireoidismo subclínico [Clinical and muscular evaluation in patients with subclinical hypothyroidism]. Arq Bras Endocrinol Metabol. 2006 Jun; 50: 523–531.
21. *Moon M.K., Lee Y.J., Choi S.H., Lim S., Yang E.J., Lim J.Y., et al.*: Subclinical hypothyroidism has little influences on muscle mass or strength in elderly people. J Korean Med Sci. 2010 Aug; 25 (8): 1176–1181.
22. *Hanke L., Poeten P., Spanke L., Britz S., Diel P.*: The Influence of Levothyroxine on Body Composition and Physical Performance in Subclinical Hypothyroidism. Horm Metab Res Horm Stoffwechselforschung Horm Metab. 2023 Jan; 55 (1): 51–58.
23. *Reuters V.S., Almeida C. de P., Teixeira P. de F. dos S., Vigário P. dos S., Ferreira M.M., Castro C.L.N. de, et al.*: Effects of subclinical hypothyroidism treatment on psychiatric symptoms, muscular complaints, and quality of life. Arq Bras Endocrinol Metabol. 2012 Mar; 56 (2): 128–136.
24. *Netzer S., Chocano-Bedoya P., Feller M., Janett-Pellegri C., Wildisen L., Büchi A.E., et al.*: The effect of thyroid hormone therapy on muscle function, strength and mass in older adults with subclinical hypothyroidism-an ancillary study within two randomized placebo controlled trials. Age Ageing. 2023 Jan 8; 52 (1): afac326.
25. *Biondi B., Palmieri E.A., Lombardi G., Fazio S.*: Subclinical hypothyroidism and cardiac function. Thyroid. 2002 Jun; 12 (6): 505–510.
26. *Li B., Huang Y., Li Z.*: Estimation of left ventricular functions in patients with subclinical hypothyroidism: a meta-analysis. Front Endocrinol. 2023; 14: 1279570.
27. *Mainenti M.R.M., Vigário P.S., Teixeira P.F.S., Maia M.D.L., Oliveira F.P., Vaisman M.*: Effect of levothyroxine replacement on exercise performance in subclinical hypothyroidism. J Endocrinol Invest. 2009 May; 32 (5): 470–473.
28. *Mainenti M.R.M., Teixeira P.F.S., Oliveira F.P., Vaisman M.*: Effect of hormone replacement on exercise cardiopulmonary reserve and recovery performance in subclinical hypothyroidism. Braz J Med Biol Res. 2010 Nov; 43 (11): 1095–1101.
29. *Brenta G., Mutti L.A., Schnitman M., Fretes O., Perrone A., Matute M.L.*: Assessment of left ventricular diastolic function by radionuclide ventriculography at rest and exercise in subclinical hypothyroidism, and its response to L-thyroxine therapy. Am J Cardiol. 2003 Jun 1; 91 (11): 1327–1330.
30. *Arem R., Rokey R., Kiefe C., Escalante D.A., Rodriguez A.*: Cardiac systolic and diastolic function at rest and exercise in subclinical hypothyroidism: effect of thyroid hormone therapy. Thyroid. 1996 Oct; 6 (5): 397–402.
31. *Biondi B., Fazio S., Palmieri E.A., Carella C., Panza N., Cittadini A., et al.*: Left ventricular diastolic dysfunction in patients with subclinical hypothyroidism. J Clin Endocrinol Metab. 1999 Jun; 84 (6): 2064–2067.
32. *Monzani F., Di Bello V., Caraccio N., Bertini A., Giorgi D., Giusti C., Ferrannini E.*: Effect of levothyroxine on cardiac function and structure in subclinical hypothyroidism: a double blind, placebo-controlled study. J Clin Endocrinol Metab. 2001 Mar; 86 (3): 1110–1115.
33. *Faber J., Petersen L., Wiinberg N., Schifter S., Mehlsen J.*: Hemodynamic changes after levothyroxine treatment in subclinical hypothyroidism. Thyroid. 2002 Apr; 12 (4): 319–324.

34. *Monforte R., Fernández-Solà J., Casademont J., Vernet M., Grau J.M., Urbano-Márquez A.*: [Hypothyroid myopathy. A clinical and histologic prospective study of 19 patients]. Med Clin (Barc). 1990 Jun 23; 95 (4): 126–129.
35. *Gierach M., Gierach J., Junik R.*: Insulin resistance and thyroid disorders. Endokrynol Pol. 2014; 65 (1): 70–76.
36. *Monzani F., Caraccio N., Siciliano G., Manca L., Murri L., Ferrannini E.*: Clinical and biochemical features of muscle dysfunction in subclinical hypothyroidism. J Clin Endocrinol Metab. 1997 Oct; 82 (10): 3315–3318.
37. *Caraccio N., Natali A., Sironi A., Baldi S., Frascerra S., Dardano A., et al.*: Muscle metabolism and exercise tolerance in subclinical hypothyroidism: a controlled trial of levothyroxine. J Clin Endocrinol Metab. 2005 Jul; 90 (7): 4057–4062.
38. *Baldini M., Colasanti A., Orsatti A., Airaghi L., Mauri M.C., Cappellini M.D.*: Neuropsychological functions and metabolic aspects in subclinical hypothyroidism: the effects of L-thyroxine. Prog Neuropsychopharmacol Biol Psychiatry. 2009 Aug 1; 33 (5): 854–859.
39. *Gild M.L., Stuart M., Clifton-Bligh R.J., Kinahan A., Handelsman D.J.*: Thyroid Hormone Abuse in Elite Sports: The Regulatory Challenge. J Clin Endocrinol Metab. 2022 Aug 18; 107 (9): e3562–3573.