

The Importance Of Iodine Deficiency And Thyroid Diseases In Human Health

Zakirova Feruza Shuxratovna

Tashkent State Medical University, Uzbekistan

Sardorova Nozila Otabekovna

Kimyo International University in Tashkent, Uzbekistan

Received: 29 September 2025; **Accepted:** 21 October 2025; **Published:** 26 November 2025

Abstract: Iodine is an essential trace element for human health. Iodine performs various functions in the human body, especially in the production of thyroid hormones - triiodothyronine and thyroxine. In addition, it affects the immune, cardiovascular, reproductive and gastrointestinal systems. Studies have shown that thyroid diseases are associated with impaired functioning of all organs and tissues, especially a number of cardiovascular complications, which are associated with an increased risk of death. Recently, the effect of replacement therapy on the cardiovascular system in cases of thyroid hormone deficiency has been the subject of much debate and different opinions.

Keywords: Hypothyroidism, atherosclerosis, arterial hypertension, heart failure, thyroid hormones, dyslipidemia.

Introduction: Iodine is an essential trace element for human health. It enters the body through iodine-rich foods and is absorbed in the small intestine. Sources of iodine include iodized table salt, seafood, seaweed, and vegetables [14]. The body must obtain the necessary amount of iodine primarily through food products such as iodized table salt, seafood, seaweed, dairy products, fish, eggs, and some vegetables. Reduced iodine intake is a pressing problem in many regions. Iodine performs various functions in the human body, especially in the production of thyroid hormones - triiodothyronine and thyroxine. In addition, it affects the immune, cardiovascular, reproductive, and gastrointestinal systems. Iodine deficiency has long been a major health problem; however, in the last decade, this problem has become even more urgent. This is primarily due to insufficient iodization of salt [6].

Iodine deficiency (ID) is a major public health problem today. When scientists analyzed trends from 1990 to 2021 and examined the main factors, they found that while the global incidence of iodine deficiency was 7.51 million in 1990, by 2021 this figure had increased to 8.08 million. The incidence is higher in women and people aged 10–30 years, due to the

greater need for iodine, but the prevalence of the disease is highest among those aged 20–45 years. This figure is predicted to increase even further by 2050 among the population of Central and East Sub-Saharan Africa and South Asia. Problems with the coverage of iodized salt have been identified as a major factor. Iodine is a trace element necessary for the synthesis of thyroid hormones [15]. Women and young people are at high risk for dementia. As a solution, experts have recommended strengthening dementia programs, improving access to health services, targeted education, and consistent monitoring of vulnerable populations. This is important to reduce future risks and improve health outcomes [13].

Thyroid hormones are well known to control general metabolism, physical and sexual development, and many other bodily functions. The major hormones produced by the thyroid gland, thyroxine or tetraiodothyronine (T4) and triiodothyronine (T3), work in synchronous harmony with appropriate feedback mechanisms and homeostasis through thyrotropin-releasing hormone (TRH) from the hypothalamus and thyrotropin-stimulating hormone (TSH) from the anterior pituitary [11].

Hypothyroidism, a deficiency of thyroid hormone, is a common disease worldwide, affecting almost all organs and tissues. It can be asymptomatic or present with a variety of life-threatening complications. The main symptoms of hypothyroidism are fatigue, drowsiness, weight gain and intolerance to cold, dry skin, constipation, and voice changes [8]; however, these symptoms are nonspecific and diagnosis is usually made by biochemical serum thyroid function tests. The most common cause of hypothyroidism is chronic autoimmune thyroiditis (Hashimoto's thyroiditis), although other causes, including drugs (e.g., amiodarone, lithium salts), radioactive iodine therapy, and thyroid surgery, are also common. Severe iodine deficiency is the underlying cause of almost all cases of thyroid disease leading to hypothyroidism. Reference ranges for thyroid function tests are based on fixed percentages of the population distribution, but there is also considerable debate about the need for more individualized reference ranges based on key factors such as age, gender, and special circumstances such as pregnancy. [10]

The thyroid gland is one of the main regulators of homeostasis in the human body, controlling the activity of many systems. Thyroid hormones, in particular, have a modulating effect on the cardiovascular system, ensuring its optimal functioning within the normal range. Triiodothyronine (T3), the active form of the thyroid hormone, is responsible for this effect mainly through genomic and non-genomic mechanisms. Studies have shown that a number of cardiovascular complications resulting from thyroid diseases are associated with an increased risk of mortality. Recently, the effects of thyroid hormones on the heart have attracted more attention [2].

Hypothyroidism is a common clinical condition characterized by a deficiency of thyroid hormones, which, if not treated in time, negatively affects the functioning of many organs and systems in the body. Approximately 10% of the world's population suffers from hypothyroidism. The impact of hypothyroidism on a person's quality of life and active lifestyle is assessed depending on the presence or absence of comorbid conditions. Sometimes other concomitant diseases may have a common pathogenetic basis with hypothyroidism. In particular, depressive states, panic attacks, and autoimmune diseases can coexist with hypothyroidism. In other cases, on the contrary, hypothyroidism manifests itself as a comorbidity with other diseases: after targeted antitumor therapy, in some forms of multiple sclerosis. Sometimes, drugs used to treat other diseases - metformin, glucocorticoids, proton pump inhibitors - can affect the level of thyrotropin in the blood, which can affect the

monitoring and diagnosis of thyroid dysfunction [3].

While overt primary hypothyroidism is characterized by elevated thyrotropin-releasing hormone and decreased free thyroxine, subclinical hypothyroidism is often an early sign of decreased thyroid function and is characterized by elevated TSH but low free T4 levels.

Depending on which part of the thyroid system is affected, hypothyroidism is classified as primary (peripheral), resulting from damage to the thyroid gland, or secondary (central), resulting from damage to the pituitary gland. Acquired primary hypothyroidism is the most common type of the disease and can be caused by severe iodine deficiency, but in most cases it is caused by chronic autoimmune thyroiditis in iodine-sufficient areas. In most cases, symptoms of hypothyroidism begin relatively late. The clinical course of the disease is diverse, and these symptoms are especially characteristic of pregnant women and children. [8].

Hypothyroidism in children is most common between the ages of 9 and 11. In approximately 80% of cases, diagnosis is difficult in children and adolescents because the disease is asymptomatic. Children with moderate to severe hypothyroidism often present with complaints of poor growth, constipation, lethargy, and/or dry skin. A detailed history and physical examination provide sufficient information to diagnose hypothyroidism. Primary hypothyroidism can be diagnosed with subnormal levels of T3 and T4 and elevated TSH. Titers of thyroid antibodies, such as anti-thyroperoxidase (TPO) and anti-thyroglobulin (ATG), are elevated in autoimmune hypothyroidism. Subclinical hypothyroidism is diagnosed with mildly elevated or high-normal levels of TSH, with free T4 in the normal range. Insufficient secretion of thyrotropin from the pituitary gland causes central hypothyroidism [16]. It has prognostic significance for early detection of cardiovascular disease and heart failure, especially in patients with coronary heart disease [5].

While hyperthyroidism is associated with atrial fibrillation and heart failure, the most obvious cardiovascular complication of hypothyroidism is the increased risk of atherosclerosis. Achieving euthyroidism is of great importance for the restoration of cardiovascular function. However, this is not possible for all patients. In addition, in recent years, there has been increasing attention to the role of subclinical thyroid dysfunctions and their impact on the cardiovascular system. Cardiovascular dysfunction in subclinical thyroid disease is of significant clinical significance, comparable to cardiovascular complications in overt hypothyroidism. This suggests

that even mild changes in thyroid hormone levels can affect the cardiovascular system. However, the treatment of subclinical thyroid disease remains controversial. Many studies have also shown that the patient's age and cardiovascular disease are key factors in clinical decision-making [16].

However, the pathological mechanism of heart failure caused by hypothyroidism is still not fully understood. Thyroid hormone replacement therapy improves myocardial systolic function, but increases the occurrence of arrhythmias. There is a need to study these mechanisms in detail and to study and implement specific treatment methods for early detection and control of heart failure in patients with hypothyroidism [4].

Studies have shown that patients with higher than normal levels of thyrotropin in the blood may have significantly increased levels of B-type natriuretic peptide, a predictor of chronic heart failure, and therefore may be at higher risk of cardiovascular disease [9]. Other studies have shown that thyroid hormone replacement therapy does not affect morbidity and mortality in patients with subclinical hypothyroidism, i.e., when TSH is less than 7-10 mEd/l. Therefore, caution is required when administering replacement therapy to patients with TSH up to 10 mEd/l, especially those over 65 years of age. The results of a large Danish study also show that, when analyzing 15-year results against the background of doubling the thyroxine dose, a decrease in TSH from 10 mEd/l to 7 mEd/l and even more did not significantly affect the clinical symptoms and quality of life of the disease, but caused a number of side effects in elderly patients. Therefore, the conclusion of this study is that it is not recommended to start replacement therapy in cases where TSH is <10 mEd/l [12].

Other studies have also shown that hormone replacement therapy is not recommended for elderly patients with subclinical hypothyroidism, as levothyroxine treatment did not alter the risk of cardiovascular events in this group of patients, regardless of the presence or absence of cardiovascular disease [1]. In elderly patients with mild subclinical hypothyroidism, there was no significant difference in systolic and diastolic cardiac function in the levothyroxine-treated group compared with a placebo control group [7].

REFERENCES

1. Acquired Hypothyroidism in Children.
Bhattacharyya SS, Singh A. Indian J Pediatr. 2023 Oct;90(10):1025-1029. doi: 10.1007/s12098-023-04578-w. Epub 2023 May 31. PMID: 37256446
2. Cardiovascular Findings and Effects of Caffeine on Experimental Hypothyroidism.
Yuksel D, Ozmen O. Endocr Metab Immune Disord Drug Targets. 2025;25(10):777-788. doi: 10.2174/0118715303315657240819114052. PMID: 39806963
3. Comorbidities of hypothyroidism.
Brenta G, Gottwald-Hostalek U. Curr Med Res Opin. 2025 Mar;41(3):421-429. doi: 10.1080/03007995.2025.2476075. Epub 2025 Mar 12. PMID: 40066580 Free article. Review.
This article reviews the common comorbidities of hypothyroidism, as reported in the literature. The comorbidities of hypothyroidism include clinical conditions commonly associated with hypothyroidism, such as dyslipidaemia, hypertension, fatigue or (possibly) ...
4. Dusp14-Mediated Dephosphorylation of MLKL Protects Against Cardiomyocyte Necroptosis in Hypothyroidism-Induced Heart Failure.
Zheng Y, Cao Y, Wang W, Tong Y, Wang S, Li C, Zhao M, Song Y, Wang YG, Qi J, Wu C, Yang J, Zheng J, Gao J, Wang J, Yang Q, Liu G, Zhao J, Zhang Y, Xiao H, Zhang YY, Tang YD. Circulation. 2025 Jun 24;151(25):1797-1813. doi: 10.1161/CIRCULATIONAHA.125.074353. Epub 2025 May 13. PMID: 40357546
5. Effect of thyroid stimulating hormone on the prognosis of coronary heart disease.
Ding N, Hua R, Guo H, Xu Y, Yuan Z, Wu Y, Li T. Front Endocrinol (Lausanne). 2025 Feb 17;16:1433106. doi: 10.3389/fendo.2025.1433106. eCollection 2025. PMID: 40034227
6. From deficiency to excess: the impact of iodine excess on reproductive health.
Khudair A, Khudair A, Niinuma SA, Habib H, Butler AE. Front Endocrinol (Lausanne). 2025 Apr 30;16:1568059. doi: 10.3389/fendo.2025.1568059. eCollection 2025. PMID: 40370779
7. Gencer B, Moutzouri E, Blum MR, Feller M, Collet TH, Delgiovane C, da Costa BR, Buffle E, Monney P, Gabus V, Müller H, Sykiotis GP, Kearney P, Gussekloo J, Westendorp R, Stott DJ, Bauer DC, Rodondi N. The Impact of Levothyroxine on Cardiac Function in Older Adults With Mild Subclinical Hypothyroidism: A Randomized Clinical Trial. Med. 2020 Jul;133(7):848-856.e5. doi: 10.1016/j.amjmed.2020.01.018. Epub 2020 Mar 12. PMID: 32171774
8. Hypothyroidism. Chaker L, Razvi S, Bensenor IM,

Azizi F, Pearce EN, Peeters RP. Nat Rev Dis Primers. 2022 May 19;8(1):30. doi: 10.1038/s41572-022-00357-7. PMID: 35589725

Tan Öksüz SB, Şahin M. Turk J Med Sci. 2024 Oct 1;54(7):1420-1427. doi: 10.55730/1300-0144.5927. eCollection 2024. PMID: 39735488

9. Hypothyroidism: Diagnosis and Treatment.

Wilson SA, Stem LA, Bruehlman RD. Am Fam Physician. 2021 May 15;103(10):605-613. PMID: 33983002

10. Hypothyroidism.

Taylor PN, Medici MM, Hubalewska-Dydejczyk A, Boelaert K. Lancet. 2024 Oct 5;404(10460):1347-1364. doi: 10.1016/S0140-6736(24)01614-3. PMID: 39368843

11. Iodineminho Study: Iodine Supplementation and Prevalence of Iodine Deficiency In Pregnant Women.

Lopes-Pereira M, Roque S, Machado SI, Korevaar TIM, Quialheiro A, Machado A, Vilarinho L, Correia-Neves M, Galanti MR, Bordalo AA, Costa P, Palha JA. J Clin Endocrinol Metab. 2024 Oct 15;109(11):e2065-e2074. doi: 10.1210/clinem/dgae041. PMID: 38266309

12. Iwen KA, Brabant G. Thyroid hormone therapy in old age. Internist (Berl). 2020 Jun;61(6):541-548. doi: 10.1007/s00108-020-00790-4. PMID: 32333088

13. Perspective: Global Burden of Iodine Deficiency: Insights and Projections to 2050 Using XGBoost and SHAP.

Liang D, Wang L, Zhong P, Lin J, Chen L, Chen Q, Liu S, Luo Z, Ke C, Lai Y. Adv Nutr. 2025 Mar;16(3):100384. doi: 10.1016/j.advnut.2025.100384. Epub 2025 Feb 4. PMID: 39914495

14. Prescribe. Hypothyroidism in adults. Levothyroxine if warranted by clinical and laboratory findings, not for simple TSH elevation. Int. 2015 Oct;24(164):241-4, 246. PMID: 26594730

15. Risks of Iodine Excess.

Sohn SY, Inoue K, Rhee CM, Leung AM. Endocr Rev. 2024 Nov 22;45(6):858-879. doi: 10.1210/endrev/bnae019. PMID: 38870258

16. Retrospective Case-Control Study Examining the Association of Thyroid-Stimulating Hormone Suppression and Vascular Wall Inflammation on [(18)F]FDG-PET/CT.

Einspieler H, Hennig B, Reiterits B, Klimpfinger H, Hacker M, Karanikas G. Thyroid. 2025 Apr;35(4):357-366. doi: 10.1089/thy.2024.0476. Epub 2025 Mar 7. PMID: 40053389

17. Thyroid and cardiovascular diseases.