AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 02. February ,2026

AUTOIMMUNE AND FUNCTIONAL DISORDERS OF THE THYROID GLAND: MODERN CLINICAL PERSPECTIVES

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INTRODUCTION

The thyroid gland (TG) is an iodine-containing endocrine organ responsible for the synthesis and secretion of thyroid hormones, which regulate the activity of numerous other hormonal systems through complex feedback mechanisms. Thyroid hormones exert profound effects on general metabolism and play a crucial role in the growth, differentiation, and functional maturation of virtually all organs and tissues.

Primarily, thyroid hormones stimulate thermogenesis, enhance oxygen consumption in tissues, and intensify oxidative processes throughout the body. At physiological concentrations, they promote intracellular protein synthesis, whereas excessive levels accelerate catabolic (dissimilatory) processes. Thyroid hormones are also essential for maintaining both cellular and humoral immunity and contribute significantly to tissue regeneration at the cellular level. Therefore, the normal function of the thyroid gland is fundamental to maintaining overall systemic health.

Disorders associated with hyperfunction or hypofunction of the thyroid gland manifest with a wide spectrum of clinical symptoms due to disturbances in the biological effects of thyroid hormones (Rusanova, 2021; Pörings & Lowin, 2019).

Keywords:
Thyroid gland; Triiodothyronine (T3); Thyroxine (T4); Hypothyroidism; Thyrotoxicosis; Graves disease; Autoimmune thyroiditis; Thyroid peroxidase antibodies; TSH receptor antibodies; Bone metabolism; Osteoporosis; Cytokines; Polyneuropathy; Endocrine regulation; Metabolic disorders.

MAIN PART

1. Thyroid Hormones and Metabolic Regulation

Thyroid hormones increase metabolic activity by stimulating lipogenesis, enhancing glucose uptake by adipose and muscle tissues, and activating gluconeogenesis and glycogenolysis (Rusanova, 2021; Paramonov, 2010).

The thyroid gland synthesizes two primary hormones:

AMERICAN ACADEMIC PUBLISHER
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ISSN NUMBER: 2692 - 5206 Volume 6. No 02. February ,2026

Triiodothyronine (T3)

Thyroxine (T4)

Iodine intake through diet is essential for their synthesis. Iodine absorbed into the bloodstream is actively taken up by thyroid follicular cells (thyrocytes), where hormone synthesis occurs. Approximately 20% of circulating T3 is produced directly in the thyroid gland, while the remaining 80% is generated through peripheral conversion of T4 to T3 in target tissues.

Thyroid hormones exert significant influence on multiple organs and systems, including the pituitary gland, heart, bones, skeletal muscles, joints, intestines, and the nervous system (Teplova, 2017).

Cardiovascular effects include positive inotropic and chronotropic actions. Triiodothyronine reduces peripheral vascular resistance by promoting arteriolar dilation and optimizing tissue perfusion (Munir & Kalyagin, 2013).

2. Thyroid Hormones and Bone Metabolism

Thyroid hormones stimulate both bone resorption and bone formation. They enhance osteoclastic activity, increasing bone turnover, while also stimulating osteoblastic function, reflected by elevated markers of bone formation (Rusanova, 2021; Shishkin & Slichenko, 2012).

Bone metabolism is regulated by a complex interplay of calcium ions, parathyroid hormone, calcitonin, vitamin D, sex hormones, thyroid hormones, glucocorticoids, growth hormone, insulin, prostaglandins, and cytokines.

Excess thyroid hormone accelerates bone metabolism and may lead to decreased bone mass and osteoporosis (Shishkin & Slichenko, 2012; Piksin & Davydkin, 2016). Both hypothyroidism and thyrotoxicosis increase the risk of osteoporosis (Kamilov & Kozlov, 2017).

In thyrotoxicosis, hypocalcemia, hyperphosphatemia, and hypercalciuria may occur. After successful treatment, bone mineral density often recovers in women of reproductive age, but recovery is limited in postmenopausal women, necessitating targeted preventive and therapeutic strategies.

3. Hypothyroidism

Hypothyroidism is a clinical syndrome characterized by reduced serum thyroid hormone levels due to thyroid hypofunction. Its prevalence in the general population ranges from 0.2–2%, while subclinical hypothyroidism affects up to 7–10%, and more than 12% of elderly women.

Hypothyroidism is classified as:

Primary (95–98%)

Secondary

Tertiary

Peripheral

AMERICAN ACADEMIC PUBLISHER
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Autoimmune thyroiditis (AIT) is the leading cause of primary hypothyroidism.

Severity stages include:

Subclinical hypothyroidism

Overt clinical hypothyroidism

Severe hypothyroidism (pleural or pericardial effusion, chronic heart failure, cretinism, myxedema coma)

In hypothyroidism, increased adenylate cyclase activity in synovial membranes stimulates fibroblasts to produce excess hyaluronic acid, resulting in synovial fluid accumulation and clinical synovitis (Teplova & Eremeeva, 2017).

Approximately 20–25% of hypothyroid patients develop arthropathy. Hypothyroid arthritis presents with joint pain, swelling, stiffness, synovial thickening, and effusion. Radiographic findings are often normal, making differential diagnosis with rheumatic diseases challenging.

Polyneuropathy occurs in 18–72% of patients with hypothyroidism. Mechanisms include mucinous infiltration around peripheral nerves leading to compression, as well as oxidative damage to Schwann cells (Tagoe, 2019; Vernigorodskiy, 2018; Muraveva & Devlikamova, 2013).

4. Thyrotoxicosis

Thyrotoxicosis is a clinical syndrome resulting from excessive circulating thyroid hormones.

The most common cause (80–85%) is **Graves disease**, an autoimmune disorder characterized by antibodies against the TSH receptor that stimulate thyroid hormone synthesis. Women are affected 10–20 times more frequently than men (Altashina & Panfilov, 2018; Troshina, 2013).

Other causes include:

Thyroid hyperfunction:

Graves–Basedow disease

Toxic multinodular goiter

Toxic adenoma

TSH-secreting pituitary adenoma

Chorioncarcinoma

Gestational thyrotoxicosis

Destructive thyroid disorders:

Acute thyroiditis

Subacute thyroiditis

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
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Chronic autoimmune thyroiditis

Other causes:

Excessive intake of thyroid hormones

T3/T4-producing ovarian teratoma

Thyroid cancer metastases

In diffuse toxic goiter, antibodies are detected as follows:

Anti-TPO – 80%

Anti-TG – 53%

Anti-TSH receptor – 89–98%

Persistent elevation of TSH receptor antibodies after thyrostatic therapy predicts a 90% relapse rate.

Cytokines play a crucial role in disease progression. Both pro-inflammatory (IL-1α, IL-6, IL-8, TNF-α) and anti-inflammatory (IL-4, IL-10, IL-14, IL-18) cytokines are elevated (Salikhova, 2014).

Treatment Approaches

Three main treatment modalities exist:

Antithyroid drugs (methimazole, propylthiouracil)

Thyroidectomy

Radioactive iodine therapy

In Europe, antithyroid drug therapy is typically continued for 12–18 months, achieving long-term remission in approximately 50% of cases. In the United States, radioactive iodine is frequently used as first-line therapy (about 60% of cases). In Russia and several other countries, radioactive iodine therapy and thyroidectomy are applied in approximately 70% of patients (Altashina & Panfilov, 2018; Shabalina & Fadeev, 2016).

5. Autoimmune Thyroiditis (AIT)

Organ-specific autoimmune diseases frequently affect endocrine organs such as the thyroid gland, pancreas, and adrenal glands. Autoimmune destruction leads to lymphocytic infiltration, tissue remodeling, fibrosis, and progressive secretory insufficiency (Gendeleka, 2016).

Three principal thyroid-specific antigens are involved:

Thyroid peroxidase (TPO)

Thyroglobulin (TG)

AMERICAN ACADEMIC PUBLISHER
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TSH receptor

HLA class II histocompatibility antigens play a significant role in genetic susceptibility and disease progression.

AIT classification:

By functional state (hypothyroid, euthyroid, thyrotoxic)

By gland size (hypertrophic, atrophic)

By nosology (isolated AIT, combined thyroid disease, part of autoimmune polyglandular syndrome)

AIT affects 3–20% of the global population and progresses to hypothyroidism in 70–80% of cases. Genetic predisposition accounts for approximately 31.8% of cases. Radiation exposure is a major environmental factor, as observed after the Chernobyl disaster and atomic bomb exposure in Hiroshima and Nagasaki. Excess iodine intake is another contributing factor (Rojko, 2019).

CONCLUSION

Thyroid hormones are central regulators of metabolic, cardiovascular, skeletal, immune, and nervous system functions. Both deficiency and excess of thyroid hormones lead to systemic complications affecting multiple organs and tissues.

Hypothyroidism is associated with polyneuropathy, arthropathy, and metabolic dysfunction, whereas thyrotoxicosis increases the risk of osteoporosis and cardiovascular complications. Autoimmune thyroid diseases represent a significant proportion of thyroid pathology and involve complex immunogenetic mechanisms.

Early diagnosis, immunological assessment (including antibody profiling), and appropriate therapeutic strategies are essential to prevent complications and improve long-term prognosis. Given the systemic nature of thyroid disorders, multidisciplinary collaboration among endocrinologists, rheumatologists, cardiologists, and neurologists is crucial for comprehensive patient management.

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