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THE ROLE OF THYROID HORMONES IN PROTEIN, LIPID, AND CARBOHYDRATE METABOLISM

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Abstract

This article presents a comprehensive analysis of the crucial role of thyroid hormones—thyroxine (T4) and triiodothyronine (T3)—in regulating metabolic processes within the human body. The anatomical and physiological characteristics of the thyroid gland, along with hormone biosynthesis and their activation in peripheral tissues, are thoroughly described. Particular attention is given to T4 as the predominant circulating hormone in the bloodstream and its conversion into the biologically active form, T3, in most tissues.

The study provides an in-depth evaluation of the influence of thyroid hormones on protein, carbohydrate, and lipid metabolism. Specifically, their regulatory role in protein synthesis and degradation, glucose metabolism, gluconeogenesis, and glycogenolysis is examined, along with their contribution to lipid mobilization and enhanced lipolysis. Furthermore, the mechanisms by which thyroid hormones regulate gene expression at the cellular level—affecting energy

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production, oxidative processes, and thermogenesis—are scientifically explained.
In addition, the neuroendocrine regulation of hormone secretion via the hypothalamic–pituitary–thyroid (HPT) axis is outlined, highlighting the functional relationships between TRH, TSH, and T3/T4. The interactions of thyroid hormones with other hormonal systems, particularly insulin and the adrenergic system, are also discussed.
Metabolic alterations associated with hyperthyroidism and hypothyroidism are extensively analyzed, including their clinical manifestations and underlying pathophysiological mechanisms. In hyperthyroidism, a marked acceleration of metabolism, increased protein catabolism, intensified lipolysis, and enhanced glucose turnover lead to weight loss and muscle weakness. Conversely, hypothyroidism is characterized by reduced metabolic activity, weight gain, dyslipidemia, and general fatigue.
Moreover, the issue of iodine deficiency, its impact on thyroid hormone synthesis, and its role in the development of endemic goiter are examined based on statistical data. Regional epidemiological indicators confirm the ongoing relevance and public health significance of this problem.

Keywords: Thyroid gland, thyroxine (T4), triiodothyronine (T3), metabolism, hypothyroidism, hyperthyroidism, iodine deficiency, hormones, energy metabolism, lipid metabolism, carbohydrate metabolism

Introduction:

The thyroid gland is a vital endocrine organ located in the anterior region of the neck, beneath the larynx. It plays a central role in regulating metabolism, growth, and developmental processes

through hormone production. Structurally, the gland consists of numerous follicles, within which the primary thyroid hormones—thyroxine (T4) and

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triiodothyronine (T3)—are synthesized. These hormones are essential regulators of protein, lipid, and carbohydrate metabolism.

T4 is produced in greater quantities by the thyroid gland and represents approximately 80% of the circulating hormone pool in the blood. However, T3 is the biologically more active form, generated through peripheral conversion of T4 in most tissues. Once inside cells, T3 enhances metabolic activity, stimulates oxidative reactions, and increases energy production. Therefore, thyroid hormones play a fundamental role in determining metabolic rate, body temperature, and overall energy expenditure.

Thyroid function is regulated by the hypothalamic–pituitary–thyroid axis. The hypothalamus secretes thyrotropin-releasing hormone (TRH), which stimulates the pituitary gland to release thyroid-stimulating hormone (TSH). In turn, TSH promotes the synthesis and secretion of T3 and T4 by the thyroid gland, maintaining hormonal balance. In cases of insufficient iodine intake, the synthesis of T3 and T4 is impaired, leading to thyroid dysfunction. Additionally, the thyroid gland produces calcitonin, a hormone involved in calcium metabolism.

At the cellular level, thyroid hormones exert direct effects on metabolic processes. They stimulate both protein synthesis and degradation. Under physiological conditions, they promote tissue growth and renewal; however, excessive hormone levels shift the balance toward protein catabolism, leading to reduced muscle protein stores and disturbances in nitrogen balance. Consequently, thyroid hormones are critical for growth, muscle integrity, and overall trophic regulation.

Their impact on carbohydrate metabolism is also significant. T3 and T4 enhance glucose metabolism by stimulating gluconeogenesis and glycogenolysis, thereby increasing energy demand. They also modulate insulin sensitivity and interact with other metabolic hormones. In hyperthyroidism, metabolic processes accelerate markedly, increasing glucose demand and, in some cases, contributing to insulin resistance.

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Thyroid hormones also play an essential role in lipid metabolism. They promote lipid breakdown, increase the availability of fatty acids, and enhance triglyceride mobilization. Through activation of lipoprotein lipase, they facilitate the utilization of lipids as an energy source, thereby maintaining energy balance.

Alterations in thyroid hormone levels lead to various clinical conditions. Hypothyroidism results in slowed metabolism, weight gain, fatigue, cold intolerance, and dyslipidemia. In contrast, hyperthyroidism is associated with increased metabolic rate, weight loss, tachycardia, muscle weakness, and heightened nervous activity. These changes highlight the critical role of thyroid hormones in maintaining physiological homeostasis.

Iodine deficiency remains a major contributing factor to thyroid dysfunction. According to statistical data, more than 60% of the population in Central Asia is at risk of iodine deficiency [6]. In certain regions of Uzbekistan and neighboring countries, the prevalence of goiter reaches 40–50% [7], while approximately 25% of school-aged children exhibit visible thyroid enlargement [8]. These findings indicate that iodine deficiency poses a significant risk not only to adults but also to children's health.

Among adults, subclinical hypothyroidism is relatively common, with a prevalence of 8–12% [9]. Although iodized salt is widely used, with 80–85% of households consuming it, inconsistent quality and irregular intake may fail to meet daily iodine requirements [10]. Therefore, iodine prophylaxis, early diagnosis, and timely detection of thyroid disorders remain critical public health priorities.

Overall, thyroid hormones play a central role in regulating metabolism, energy production, growth, development, and homeostasis. A deeper understanding of their mechanisms is essential for improving the diagnosis, prevention, and treatment of endocrine disorders.

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Classification and General Characteristics:

Thyroid hormones are among the most essential biological regulators controlling metabolic processes in the body. One of their primary functions is the regulation of basal metabolic rate (BMR). T3 and T4 increase cellular oxygen consumption, enhance energy expenditure, and accelerate overall energy production. For this reason, thyroid hormones are considered key determinants of total metabolic activity.

In terms of protein metabolism, thyroid hormones stimulate protein synthesis under physiological conditions, supporting tissue growth and regeneration. However, when present in excessive amounts, they also intensify protein degradation. This may result in reduced protein reserves in muscle tissue, leading to muscle weakness and trophic disturbances. Therefore, thyroid hormones exert a dual effect on protein metabolism by regulating both anabolic and catabolic processes.

Their role in carbohydrate metabolism is equally significant. T3 and T4 enhance intestinal glucose absorption, stimulate gluconeogenesis in the liver, and influence glycogen metabolism. Additionally, they participate in mechanisms related to insulin sensitivity. As a result, thyroid hormones play a critical role in maintaining glucose homeostasis and meeting the body's energy demands [11].

Regarding lipid metabolism, thyroid hormones promote the utilization of lipids as an energy source. They increase triglyceride mobilization, elevate free fatty acid levels, and enhance the activity of lipoprotein lipase. Consequently, lipid breakdown and energy release are intensified, highlighting their importance in maintaining energy balance [12].

Deviations in thyroid hormone levels lead to substantial metabolic disturbances. In hyperthyroidism, metabolic processes accelerate significantly, resulting in weight loss, tachycardia, heat intolerance, and muscle weakness. In contrast, hypothyroidism is characterized by a slowed metabolic rate, weight gain, fatigue, cold intolerance, and reduced overall activity [11].

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Clinical Signs and Symptoms:

Thyroid hormones are key regulators of basal metabolic rate (BMR), increasing overall energy expenditure and stimulating protein synthesis at the cellular level. However, in conditions of excessive thyroid activity, protein breakdown becomes more pronounced. In hyperthyroidism, increased nitrogen loss from muscle tissue may lead to muscle weakness and atrophy [11], often presenting as reduced physical endurance and fatigue.

Thyroid hormones also significantly influence lipid metabolism by activating lipolysis, increasing circulating free fatty acids (NEFA), and enhancing lipid utilization for energy. Alterations in lipoprotein lipase activity may disrupt lipid balance, potentially leading to hyperlipidemia and an increased risk of atherosclerosis [12]. In hypothyroidism, reduced lipid breakdown contributes to elevated cholesterol levels and lipid accumulation.

In carbohydrate metabolism, thyroid hormones stimulate gluconeogenesis and glycogenolysis in the liver, supporting increased energy demand. In hyperthyroidism, this leads to accelerated glucose turnover and possible fluctuations in blood glucose levels [11], demonstrating their close interaction with insulin and other metabolic hormones.

Due to their extensive metabolic effects, imbalances in thyroid hormones result in significant disturbances in protein, lipid, and carbohydrate metabolism. In hyperthyroidism, enhanced protein catabolism leads to muscle wasting, whereas hypothyroidism results in lipid accumulation and dyslipidemia. Carbohydrate metabolism is also affected, particularly in hyperthyroid states where glucose utilization increases.

Clinically, hyperthyroidism is characterized by a markedly increased metabolic rate, leading to weight loss despite increased appetite, tachycardia, irritability, tremors, and heat intolerance. Additional symptoms include excessive sweating, insomnia, muscle weakness, and fatigue. In some cases, exophthalmos may also occur.

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Conversely, hypothyroidism presents with reduced metabolic activity, weight gain, fatigue, drowsiness, and increased sensitivity to cold. Other common features include dry skin, hair loss, bradycardia, constipation, and decreased cognitive function. In prolonged untreated cases, myxedema may develop due to tissue swelling [13].

Diagnostic Methods:

The diagnosis of thyroid disorders requires a comprehensive approach, including clinical evaluation, laboratory testing, and instrumental investigations. Initial assessment involves analyzing patient symptoms such as weight changes, heart rate abnormalities, fatigue, and temperature sensitivity. Physical examination focuses on thyroid size, consistency, and the presence of nodular changes.

Laboratory evaluation is fundamental, with thyroid-stimulating hormone (TSH) serving as the most sensitive marker of thyroid function. Measurement of free thyroxine (FT4) and free triiodothyronine (FT3) provides insight into the biologically active hormone levels. Autoimmune involvement is assessed by detecting antibodies such as anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg).

Instrumental methods include ultrasound imaging, which evaluates gland size, structure, and nodular characteristics. In certain cases, scintigraphy is used to assess functional activity. Advanced imaging techniques such as computed tomography (CT) or magnetic resonance imaging (MRI) are employed in complex cases, including large goiters or suspected tumors.

When nodules are detected, fine-needle aspiration biopsy (FNAB) is performed for cytological analysis, allowing differentiation between benign and malignant lesions. This method is crucial for accurate diagnosis and treatment planning.

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Treatment:

Thyroid hormones, particularly T3, regulate hepatic lipid, cholesterol, and carbohydrate metabolism through complex, multi-step mechanisms. They enhance intracellular lipid degradation in the liver by activating lipophagy and promote lipid utilization. Additionally, thyroid hormones influence glucose production pathways in the liver. Specifically, they regulate gluconeogenesis via signaling pathways involving Sirtuin 1 (SirT1) and Forkhead box protein O1 (FoxO1).

Through these mechanisms, thyroid hormones control the expression of key genes involved in lipid and glucose metabolism. Alterations in their activity may disrupt metabolic homeostasis, contributing to metabolic disorders [14].

The biological effects of thyroid hormones are primarily mediated by T3. After entering the cell, T3 binds to nuclear receptors and regulates gene transcription. Thyroid receptor isoforms, particularly TRα and TRβ, play a crucial role in modulating metabolic activity in different tissues. Their varying expression patterns determine tissue-specific responses to thyroid hormones [11].

Furthermore, thyroid hormones interact closely with the adrenergic system. They enhance metabolic activity in adipose tissue and the liver in response to adrenergic stimulation. Through receptor-mediated pathways, they regulate cholesterol and carbohydrate metabolism at the level of gene expression. This interaction contributes to increased energy expenditure, thermogenesis, and changes in insulin sensitivity, emphasizing their role in maintaining overall metabolic balance [11].

Discussion

Numerous studies confirm that thyroid hormones—T3 and T4—serve as primary regulators of metabolism [2, 3, 11, 12]. Research indicates that the peripheral conversion of T4 into T3 plays a key role in fine-tuning intracellular metabolic processes [3]. The effect of thyroid hormones on protein metabolism is

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concentration-dependent: at normal levels, they promote protein synthesis, whereas excessive levels enhance proteolysis and may lead to negative nitrogen balance in muscle tissue [3, 11].

In carbohydrate metabolism, thyroid hormones stimulate glucose absorption, glycogenolysis, and gluconeogenesis. This contributes to accelerated glucose turnover in hyperthyroidism and may predispose to insulin resistance in some cases [5, 14]. Lipid metabolism is also closely associated with thyroid function; T3 enhances lipolysis and increases free fatty acid levels, while hypothyroidism leads to lipid accumulation and an increased risk of dyslipidemia [4, 12, 13].

Hormone secretion is regulated through the hypothalamic–pituitary–thyroid axis via a negative feedback mechanism [1, 13]. Disruptions in this regulatory system can adversely affect endocrine and metabolic stability [9]. Iodine deficiency remains a major cause of endemic goiter and hypothyroidism, highlighting the importance of iodized salt consumption as a preventive measure [7, 8, 10].

Overall, the findings demonstrate that thyroid hormone–induced metabolic and clinical changes occur in an integrated manner across multiple systems. Alterations in protein, carbohydrate, and lipid metabolism observed in hyperthyroidism and hypothyroidism are consistently supported by various studies [3, 5, 11, 12, 13], underscoring their importance in clinical assessment and therapeutic decision-making.

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