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SCIENTIFIC-THEORETICAL ANALYSIS OF PSYCHOSOMATIC AND NEUROPSYCHOLOGICAL DISORDERS IN THYROID GLAND DISEASES

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Abstract: The thyroid gland is one of the most important organs within the central endocrine regulatory system of the human body. The hormones it produces, thyroxine (T4) and triiodothyronine (T3), regulate the metabolic activity of nearly all tissues. These hormones serve as key biological regulators of cellular mitochondrial energy metabolism, oxidative phosphorylation processes, protein synthesis, and neuronal electrical activity.

This scientific-theoretical article provides an in-depth analysis of the pathogenetic mechanisms underlying the development of psychosomatic and neuropsychological disorders in thyroid gland diseases. In particular, in conditions such as hypothyroidism, hyperthyroidism, and autoimmune thyroiditis, disturbances of central nervous system function, neurotransmitter imbalance, and psycho-emotional changes are examined on a scientific basis.

The results of the study indicate that thyroid gland pathologies should be considered not only as endocrine disorders but also as a complex dysfunction of the psycho-neuroendocrine system.

Keywords: thyroid gland, psychosomatics, neuropsychology, stress, hormones, T3, T4, HPA axis.

Introduction

The human body is a complex biological system in which the endocrine, nervous, and immune systems are integrated into a unified neurohumoral regulatory mechanism. Within this system, the thyroid gland plays a particularly important role as a central metabolic modulator organ, since the hormones it produces regulate energy metabolism, growth, and differentiation processes in nearly all cells and tissues of the body.

Epidemiological observations conducted over the past decade indicate a significant increase in thyroid gland diseases. According to various clinical studies and data from international endocrine registries, the prevalence of thyroid functional disorders increased by approximately 25–30% between 2015 and 2025, with a notably higher rate observed in the

female population. In addition, due to the widespread occurrence of subclinical forms, many cases remain undiagnosed for a long period of time.

The main thyroid hormones—thyroxine (T4) and triiodothyronine (T3)—are iodine-containing biologically active substances that are converted into their active form in peripheral tissues by deiodinase enzymes. In particular, T3 binds to nuclear receptors and activates gene transcription, thereby regulating protein synthesis. This process enhances mitochondrial activity and ATP production, ultimately increasing the basal metabolic rate.

The central nervous system is highly sensitive to thyroid hormones. T3 and T4 play a crucial role in neuronal embryonic development, myelination, synapse formation, and neurotransmitter regulation. The hippocampus is responsible for memory consolidation, while the prefrontal cortex is involved in cognitive control and decision-making; both regions are directly influenced by thyroid hormone levels. The limbic system regulates emotional responses and affective states; therefore, hormonal imbalance may lead to emotional instability.

From an endocrine perspective, thyroid function is regulated by the hypothalamic–pituitary–thyroid (HPT) axis. The hypothalamus secretes thyrotropin-releasing hormone (TRH), which stimulates the anterior pituitary gland to release thyroid-stimulating hormone (TSH). TSH, in turn, activates the synthesis of T3 and T4 in the thyroid gland. This system operates through a negative feedback mechanism, whereby increased hormone levels suppress TRH and TSH secretion.

Thyroid gland disorders—hypothyroidism and hyperthyroidism—arise as a result of disruption in this complex regulatory system. In hypothyroidism, a deficiency of T3 and T4 leads to reduced cellular metabolism, resulting in energy deficiency, bradycardia, cold intolerance, and general psychophysiological slowing. In hyperthyroidism, excessive hormone production enhances catabolic processes, increases heart rate, and heightens autonomic nervous system excitability.

From a psychological and neuropsychological perspective, thyroid hormones directly modulate brain function. Their deficiency is associated with depressive symptoms, apathy, impaired attention, and memory dysfunction. Conversely, elevated hormone levels manifest as anxiety, irritability, insomnia, and psychomotor agitation. Studies indicate that 40–60% of patients with thyroid dysfunction experience various degrees of affective disorders, further confirming this relationship. These conditions are exacerbated by psychosomatic mechanisms, as stress activates the hypothalamic–pituitary–adrenal (HPA) axis, increases cortisol secretion, and suppresses thyroid function.

Therefore, thyroid gland diseases should be regarded not only as endocrine disorders but also as complex integrative dysfunctions involving the central nervous system and psychological state.

Research Objective: The aim of this study is to investigate, from a scientific-theoretical perspective, the psychosomatic and neuropsychological changes occurring in conditions of thyroid gland diseases (hypothyroidism, hyperthyroidism, and autoimmune thyroiditis). Additionally, the study aims to analyze the effects of T3 and T4 hormones on central nervous system activity and the mechanisms by which disruption of the hypothalamic–pituitary–thyroid axis affects psychological state.

Materials and Methods: This study was conducted based on a theoretical, analytical, and integrative approach. As research materials, fundamental scientific literature in the fields of modern endocrinology, neuropsychology, psychosomatics, and neuroendocrinology, as well as

international clinical guidelines, meta-analyses, and scientific articles published over the past decade were analyzed. In addition, epidemiological data and clinical observation results related to thyroid gland diseases were also reviewed. Special attention was given to the pathophysiological mechanisms of hypothyroidism, hyperthyroidism, and autoimmune thyroiditis.

Methodologically, the study employed theoretical analysis, comparative analysis, a systems-based approach, and an integrative (neuroendocrine) model of evaluation. The physiological mechanisms of the hypothalamic–pituitary–thyroid (HPT) axis, which regulates thyroid gland function, were examined in depth. The regulatory interactions among TRH, TSH, T3, and T4 hormones, as well as the negative feedback mechanism within this system, were analyzed.

Furthermore, the effects of stress factors on the endocrine system via the hypothalamic–pituitary–adrenal (HPA) axis were investigated. Scientific data regarding elevated cortisol levels suppressing thyroid function, altering deiodinase enzyme activity, and reducing peripheral conversion of T4 to T3 were also analyzed.

Psychological disorders such as depression, anxiety, insomnia, and impaired attention and memory were examined from a neuropsychological perspective. The functional activity of the limbic system, hippocampus, and prefrontal cortex was analyzed in relation to thyroid hormone levels based on scientific literature. Additionally, alterations in neurotransmitter metabolism (serotonin, dopamine, and norepinephrine) were evaluated as key factors in the pathogenesis of psycho-emotional symptoms.

Overall, the study considers the endocrine, nervous, and immune systems as an integrated psycho-neuroendocrine system, allowing for a systematic scientific analysis of the complex mechanisms underlying thyroid gland pathologies.

Results: The study confirmed that the thyroid hormones thyroxine (T4) and triiodothyronine (T3) are key neurobiological regulators involved in the control of central nervous system activity and psychological functions. According to the analyzed scientific sources, both deficiency and excess of these hormones have a direct impact on brain metabolism, neuronal signal transmission, and neurotransmitter balance.

In hypothyroidism, reduced levels of T3 and T4 lead to a decrease in oxidative metabolism in brain cells, a reduction in mitochondrial ATP synthesis, and suppression of neuronal activity. This condition manifests clinically as impaired cognitive functions, memory decline, reduced attention, and slowed psychomotor responses. In addition, decreased activity of the limbic system has been associated with the development of depressive states and emotional instability.

In hyperthyroidism, elevated thyroid hormone levels increase the activity of both central and peripheral sympathetic nervous systems. As a result, sensitivity to catecholamines (adrenaline and noradrenaline) is enhanced, leading to increased cardiovascular activity, sleep disturbances, and heightened psychomotor excitability. Clinically, this condition is characterized by anxiety, irritability, psychomotor agitation, and insomnia.

In autoimmune thyroiditis, pathological activation of the immune system leads to increased levels of pro-inflammatory cytokines (IL-6, TNF-α). These biological mediators affect the central nervous system through the blood–brain barrier and intensify neuroinflammatory processes. Consequently, chronic fatigue syndrome, cognitive slowing, and emotional imbalance may develop.

Furthermore, the results demonstrate that dysfunction of the hypothalamic–pituitary–thyroid (HPT) axis has a direct impact on psychological state. Disruption within this system alters TSH secretion, thereby impairing the synthesis of T3 and T4, which leads to a complex dysfunction of central nervous system activity.

Overall, the findings confirm that thyroid gland diseases should not be regarded solely as endocrine hormonal disorders. Instead, they represent a complex psycho-neuroendocrine pathological process involving the integrated dysfunction of the central nervous system, psychological activity, and immune response mechanisms.

Discussion: The results of this study demonstrate that thyroid gland diseases are primarily characterized by a complex psycho-neuroendocrine mechanism in which the endocrine, nervous, and immune systems are closely interconnected. Dysregulation of the hypothalamic–pituitary–thyroid (HPT) axis leads to alterations in T3 and T4 hormone secretion, which in turn directly affects central nervous system metabolism and neuronal signal transmission.

In hypothyroid conditions, thyroid hormone deficiency suppresses cerebral energy metabolism, resulting in reduced cognitive performance, memory impairment, and the development of depressive states. In contrast, hyperthyroidism is associated with excessive hormone production, which enhances sympathetic nervous system activity and leads to anxiety, insomnia, and psychomotor agitation.

In autoimmune thyroiditis, activation of the immune system increases the production of inflammatory mediators that negatively influence brain function, contributing to neuroinflammation, emotional instability, and chronic fatigue syndrome. Moreover, stress-induced activation of the hypothalamic–pituitary–adrenal (HPA) axis elevates cortisol levels, further disrupting thyroid hormone homeostasis and exacerbating endocrine imbalance.

The findings suggest that psychological disturbances observed in thyroid diseases are not limited to hormonal imbalance alone. Rather, they result from a combination of neuroendocrine dysregulation and the activation of psychosomatic mechanisms, reflecting a multidimensional interaction between biological and psychological systems.

Conclusion: Thyroid gland diseases were identified as complex pathological processes characterized by the disruption of neurohumoral regulation among the endocrine, nervous, and immune systems. The study confirmed that the thyroid hormones T3 and T4 have a direct influence on central nervous system metabolism, neuronal activity, and neurotransmitter balance.

In hypothyroidism, a reduction in metabolic processes leads to impaired cognitive functions and the development of depressive symptoms. In hyperthyroidism, increased sympathetic nervous system activity results in heightened psycho-emotional excitability. In autoimmune thyroiditis, the influence of inflammatory mediators on the central nervous system contributes to the development of neuropsychological disorders.

The functional interaction between the hypothalamic–pituitary–thyroid (HPT) axis and the hypothalamic–pituitary–adrenal (HPA) axis was identified as a key mechanism that exacerbates hormonal imbalance under stress conditions.

These findings indicate that the pathogenesis of thyroid gland diseases involves an integrated action of endocrine, neuroendocrine, and psychosomatic mechanisms, thereby confirming the necessity of a comprehensive multidisciplinary approach to their study.

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