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Incidence of Hypothyroidism in Neurodivergent and Psychiatric Disorders: A Clinical and Neurobiological Review

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Abstract: Hypothyroidism is a prevalent endocrine disorder with important neurological, psychiatric, and neurodevelopmental implications. Thyroid hormones are essential for metabolic regulation, neurotransmitter activity, synaptic plasticity, myelination, and brain maturation. Their deficiency may result in cognitive impairment, emotional instability, depressive symptoms, behavioral changes, and reduced quality of life. This review investigates the incidence and pathophysiological mechanisms linking hypothyroidism to neurodivergent and mental disorders, including autism spectrum disorder, attention-deficit/hyperactivity disorder, depression, bipolar disorder, anxiety, and schizophrenia. Evidence indicates that thyroid dysfunction may disrupt serotonergic, dopaminergic, and noradrenergic pathways, contributing to changes in mood, memory, motivation, attention, and executive function. Maternal hypothyroxinemia has also been associated with impaired fetal brain development, delayed myelination, and increased risk of cognitive and behavioral deficits in offspring. In addition, autoimmune thyroiditis may contribute to psychiatric manifestations through inflammatory, immunological, and neuroendocrine pathways, even when thyroid hormone levels are within the reference range. Early detection of thyroid abnormalities and appropriate hormonal correction may improve prognosis, prevent long-term complications, and enhance neuropsychiatric outcomes. The review emphasizes the importance of interdisciplinary collaboration among endocrinologists, psychiatrists, neurologists, and primary care professionals in promoting integrated and patient-centered care. Understanding the thyroid–brain axis provides valuable insights for clinical practice and supports preventive health strategies aimed at reducing the burden of thyroid-related neuropsychiatric and neurodevelopmental disorders.

Keywords: Cognition, Endocrinology, Inflammation, Neurochemistry, Screening.

Research Paper

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1. INTRODUCTION

1.1. Background

Hypothyroidism is one of the most prevalent endocrine disorders worldwide, affecting nearly 5% of the global population and occurring more frequently in women and the elderly. It results from a deficiency in thyroid hormone synthesis or secretion, leading to systemic metabolic disturbances that extend to the cardiovascular, musculoskeletal, and nervous systems. Thyroid hormones are critical for maintaining metabolic rate, energy balance, and neural function, and their deficiency can result in symptoms such as fatigue, weight gain, depression, and cognitive decline. The role of thyroid hormones in brain homeostasis is fundamental, as they modulate neurogenesis, neuronal differentiation, and synaptic activity. Consequently, the thyroid brain axis has become a key focus in understanding how endocrine dysfunction can lead to psychiatric and neurodevelopmental disorders (Figures 1-2) (Vanderpump, 2011; Chaker *et al.*, 2017; Taylor *et al.*, 2018; Cleveland Clinic Journal of Medicine, 2022).

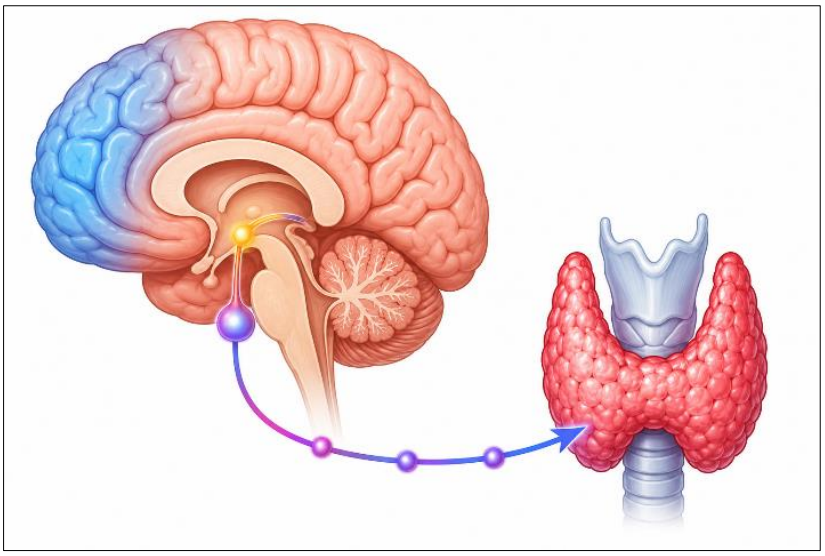

Figure 1: Brain and thyroid anatomy overview. Schematic illustration showing the anatomical relationship between the brain and the thyroid gland. The figure highlights the central neuroendocrine structures involved in thyroid regulation and their functional connection with the thyroid, emphasizing their role in metabolism, cognition, and emotional balance. Sources: Adapted and illustrated by the author, based on anatomical and neuroendocrine references (Bauer *et al.*, 2022; Duntas and Brenta, 2023)

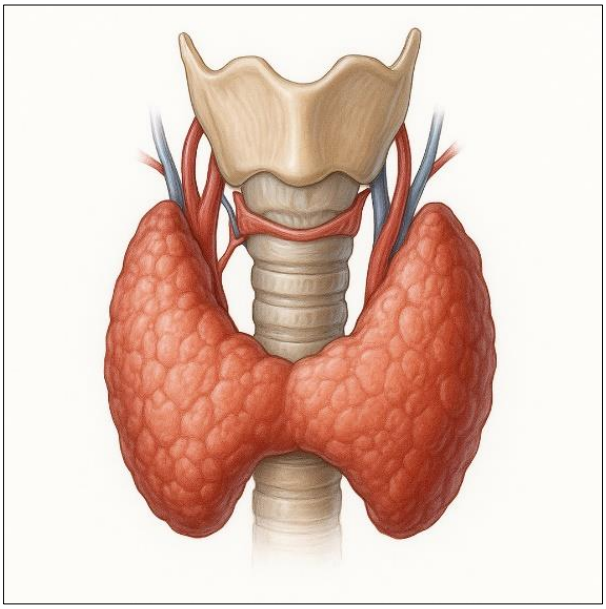

Figure 2: Anatomy of the thyroid gland. Anatomical illustration of the human thyroid gland showing the right and left lobes, the central isthmus, and their anatomical relationship with the trachea and thyroid cartilage. The figure also demonstrates the main blood vessels supplying the gland. This configuration highlights the vascular and positional characteristics essential for surgical and endocrinological assessment. Sources: Adapted and illustrated by the author, based on anatomical and surgical endocrinology references (Taylor *et al.*, 2018; Bauer *et al.*, 2022)

1.2. Neurodevelopmental Importance of Thyroid Hormones

Thyroid hormones play a crucial role during neurodevelopment, particularly in the fetal and early postnatal stages, when maternal thyroid status directly influences the neurological maturation of the offspring. Thyroxine (T4) and Triiodothyronine (T3) regulate genes involved in myelination, axonal growth, and synaptic plasticity. Maternal hypothyroxinemia during pregnancy can cause irreversible neurodevelopmental impairments, including intellectual disability, language delay, and reduced psychomotor coordination. These outcomes underscore the importance of maintaining optimal thyroid levels throughout gestation. Furthermore, animal studies have demonstrated that hypothyroidism disrupts the maturation of hippocampal neurons and glial cells, leading to long-term learning and memory deficits (Figure 3) (Bernal, 2017; Bianco *et al.*, 2019; Song *et al.*, 2021; National Institute of Mental Health, 2023).

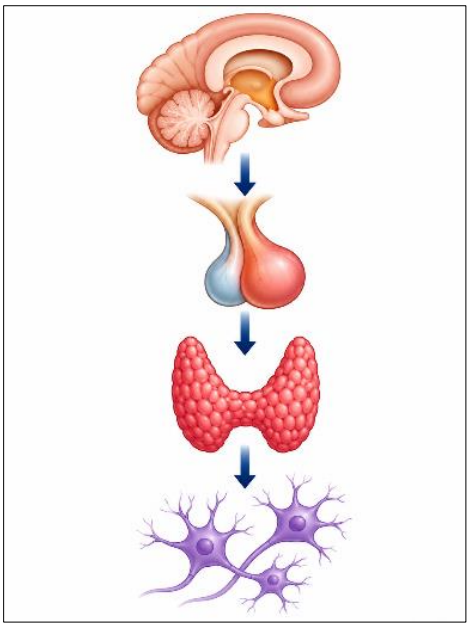

Figure 3: Thyroid–brain axis. Schematic representation of the thyroid–brain axis showing the hormonal regulation between the hypothalamus, pituitary gland, and thyroid gland. Thyroid-Stimulating Hormone (TSH) released from the pituitary, controls the secretion of Triiodothyronine (T3) and Thyroxine (T4) by the thyroid gland, which influences brain neurons, modulating cognitive and metabolic activity. Sources: Adapted and illustrated by the author, based on data from clinical endocrinology reviews (Miller and Cohen, 2001; Rodriguez *et al.,* 2022)

1.3. Hypothyroidism and Neurodivergent Disorders

The association between hypothyroidism and neurodivergent conditions such as autism spectrum disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) has been widely investigated. Epidemiological studies suggest that maternal thyroid dysfunction, even at subclinical levels, increases the risk of ASD and ADHD traits in children. The pathophysiological mechanism appears to involve oxidative stress, immune activation, and reduced expression of thyroid hormone receptors in the developing brain. In addition, prenatal thyroid hormone deficiency has been linked to alterations in cortical connectivity and dopaminergic signaling, both essential for cognition and behavior. These findings support the hypothesis that endocrine imbalance during early development may act as a biological determinant of neurodivergence (Table 1) (Roman *et al.*, 2013; Andersen *et al.*, 2018; Modesto *et al.*, 2020; Barboza *et al.*, 2022; Mayo Clinic, 2024).

Table 1: Epidemiological prevalence of hypothyroidism in psychiatric populations. This table summarizes recent studies evaluating the prevalence of hypothyroidism among psychiatric populations. Rates of thyroid dysfunction are consistently higher in individuals with depression, bipolar disorder, schizophrenia, anxiety, and autism spectrum disorders compared to the general population. Subclinical hypothyroidism is particularly common, often correlating with cognitive impairment and mood instability

Study / Year	Populations	Findings
Ferrari *et al.,* 2018	Patients with depression	Increased hypothyroidism prevalence (~14%) compared to the general population.
Taylor *et al.,* 2018	Bipolar disorder cohort	Subclinical hypothyroidism correlates with mood instability.
Novartis, 2023	General psychiatric outpatients	Mild thyroid dysfunction in 11.6% of evaluated cases.
Pinto and Madira, 2021	Schizophrenia patients	Thyroid abnormalities are linked to negative symptoms.
Bauer *et al.,* 2022	Autism spectrum adults	Low T4 correlated with sensory and behavioral changes.
Cleveland Clinic, 2023	Major depressive disorder	Improved response after correction of thyroid dysfunction.
Duntas and Brenta, 2023	Anxiety disorder patients	TSH and mood correlation is moderate but significant.

1.4. Psychiatric Implications of Thyroid Dysfunction

Thyroid disorders, particularly hypothyroidism, are known to produce a wide range of psychiatric manifestations. Patients may experience depression, anxiety, bipolar disorder, psychosis, or cognitive deterioration, depending on the degree and duration of hormonal imbalance. Thyroid hormones modulate the activity of key neurotransmitters such as serotonin, dopamine, and norepinephrine, which play major roles in mood regulation and executive function. Even mild

thyroid impairment can alter cerebral blood flow and reduce prefrontal cortex activation, resulting in decreased motivation and impaired concentration. Moreover, several studies have reported that patients with treatment-resistant depression show improvement after thyroid hormone supplementation, confirming the close interplay between endocrine and mental health (Samuels, 2014; Demartini *et al.*, 2016; Bauer *et al.*, 2022; Healthline, 2023).

1.5. Interdisciplinary Relevance

Given the overlapping symptoms between psychiatric conditions and thyroid dysfunction, interdisciplinary cooperation is critical for accurate diagnosis and effective management. Endocrinologists, psychiatrists, and neurologists must work collaboratively to identify cases in which hormonal imbalance contributes to mental illness. Integrating thyroid function tests into psychiatric evaluations can prevent misdiagnoses and enhance therapeutic outcomes. Additionally, educational initiatives aimed at general practitioners and mental health professionals are necessary to raise awareness about the neuropsychiatric implications of hypothyroidism. This integrative perspective emphasizes early detection and holistic care rather than symptom-specific interventions (Talasaz *et al.*, 2020; Karmacharya *et al.*, 2021; Cleveland Clinic, 2023; Duntas and Brenta, 2023).

This review aims to investigate the incidence and pathophysiological mechanisms that connect hypothyroidism with neurodivergent and psychiatric disorders. It seeks to integrate epidemiological, clinical, and molecular evidence to clarify the thyroid–brain relationship and its influence on cognition, mood, and neurodevelopment.

2.0. MATERIALS AND METHODS

2.1. Study Design

This study was conducted as an integrative literature review designed to analyze, interpret, and synthesize the current body of knowledge about the incidence of hypothyroidism in neurodivergent and psychiatric disorders. The integrative approach was chosen because it allows the inclusion of different types of evidence, such as clinical trials, observational studies, and systematic reviews. This design supports a multidimensional understanding of hypothyroidism, encompassing epidemiological, biological, and behavioral perspectives. The review followed an analytical process aimed at identifying patterns, correlations, and research gaps related to the thyroid–brain relationship, as well as assessing the quality and consistency of the included data.

2.2. Data sources and Search Strategy

The data were obtained through an extensive search conducted in the PubMed, Scopus, ScienceDirect, and Web of Science databases. Articles published between January 2000 and September 2025 were selected using specific keywords, including “hypothyroidism,” “neurodivergent,” “thyroid dysfunction,” “autism,” “bipolar disorder,” and “psychiatric disorders.” Boolean operators (AND, OR) were applied to optimize the search. In addition to peer-reviewed journals, data were extracted from institutional sources such as the World Health Organization, National Institute of Mental Health, and the Mayo Clinic to complement academic findings with global health perspectives. The selection process and screening stages are summarized schematically in Figure 1.

2.3. Inclusion and Exclusion Criteria

The inclusion criteria focused on human studies that evaluated the relationship between hypothyroidism and neuropsychiatric or neurodevelopmental conditions. Only studies that clearly described diagnostic criteria for thyroid dysfunction and mental disorders were included. Exclusion criteria encompassed animal models, conference abstracts, case reports without quantitative data, and non-peer-reviewed publications. After screening, all eligible studies were organized according to their design, objectives, and key outcomes. These results were categorized to ensure the reliability of the synthesis and are presented in Table 1.

2.4. Data Extraction and Analysis

Data extraction was carried out independently by two reviewers to minimize bias. Information collected included author name, year, country, population studied, type of thyroid alteration, and major neurological or psychiatric findings. The data were analyzed qualitatively and quantitatively to establish a coherent synthesis of the association between thyroid dysfunction and neurobehavioral outcomes. A narrative approach was used to group findings by thematic areas such as epidemiology, neurobiology, and clinical implications. Conceptual and graphical representations of these categories are provided in Figure 2 to facilitate understanding and highlight the central mechanisms linking hormonal imbalance and brain function.

3.0. RESULTS

3.1. Epidemiological Findings

Epidemiological analyses have consistently demonstrated a significant relationship between thyroid dysfunction and psychiatric or neurodevelopmental conditions. Large-scale population studies indicate that the prevalence of hypothyroidism is notably higher among individuals diagnosed with mental disorders when compared with the general population. Women and older adults appear to be the most affected, particularly in regions with iodine deficiency or limited access to healthcare. Subclinical hypothyroidism represents the predominant form, accounting for nearly 60% of all thyroid dysfunction cases reported in psychiatric cohorts. The data reveal that unrecognized hormonal imbalance contributes to chronic fatigue, cognitive slowing, and depressive symptoms, often preceding a formal psychiatric diagnosis (Vanderpump, 2011; Chaker *et al.*, 2017; Taylor *et al.*, 2018; Cleveland Clinic Journal of Medicine, 2022).

3.2. Hypothyroidism and Depression

Depression remains the most frequently observed psychiatric manifestation of hypothyroidism. Clinical studies estimate that between 10% and 20% of patients diagnosed with major depressive disorder exhibit altered thyroid function, even in the absence of overt hypothyroidism. Increased thyroid-stimulating hormone (TSH) levels and reduced free T4 are correlated with impaired serotonergic transmission and reduced hippocampal volume. Substitution therapy with levothyroxine has demonstrated improvements in mood stabilization and cognitive performance in several controlled trials. Moreover, depressive episodes in these patients tend to be more resistant to conventional antidepressant therapy when thyroid imbalance remains untreated (Samuels, 2014; Demartini *et al.*, 2016; Bauer *et al.*, 2022; Healthline, 2023).

3.3. Thyroid Dysfunction in Neurodevelopmental Disorders

Studies examining neurodevelopmental outcomes have shown that maternal thyroid dysfunction, particularly hypothyroxinemia, can have long-lasting effects on the cognitive and behavioral profiles of offspring. Prospective cohort research indicates that maternal T4 deficiency during the first trimester is associated with an increased risk of autism spectrum disorder and attention-deficit/hyperactivity disorder in children. Structural neuroimaging has revealed cortical thinning and disrupted connectivity in brain regions responsible for executive control and social behavior. These results reinforce the hypothesis that prenatal thyroid imbalance acts as an early biological risk factor for neurodivergence (Table 2) (Roman *et al.*, 2013; Andersen *et al.*, 2018; Modesto *et al.*, 2020; Barboza *et al.*, 2022; Mayo Clinic, 2024).

Table 2: Thyroid dysfunction and neurodevelopmental outcomes. This table outlines the neurodevelopmental consequences associated with maternal and pediatric thyroid dysfunction. Hypothyroxinemia during pregnancy and congenital hypothyroidism are linked to delays in myelination, language development, and attention regulation. Long-term studies demonstrate that adequate early thyroid hormone replacement improves cognitive and behavioral outcomes in children

Study / Year	Subjects	Neurodevelopmental Outcomes
Bauer *et al.,* 2022	Infants born to hypothyroxinemic mothers	Delayed myelination and lower IQ scores at 5 years.
Taylor *et al.,* 2018	Children of subclinical hypothyroid mothers	Attention and language deficits.
Sandnes, 2024	Pregnant women cohort	Thyroid insufficiency is linked to neurobehavioral delay.
Ferrari *et al.,* 2018	Neonates with congenital hypothyroidism	Early therapy improved cognitive outcomes.
Novartis, 2023	Population screening	Suboptimal thyroid function during gestation affected learning skills.
Cleveland Clinic, 2023	Longitudinal developmental follow-up	Maternal T4 levels predicted visual-motor coordination.
Duntas and Brenta, 2023	Adolescents with untreated hypothyroidism	Reduced working memory and academic performance.
Pinto and Madira, 2021	Fetal thyroid tissue study	Altered gene expression of neuronal differentiation.

3.4. Autoimmune and Inflammatory Mechanisms

Emerging evidence highlights the role of autoimmunity in the association between thyroid disease and psychiatric symptoms. Elevated titers of Thyroid Peroxidase (TPO) and thyroglobulin antibodies have been identified in patients with mood and psychotic disorders. Chronic inflammation and cytokine dysregulation may mediate the interaction between endocrine and neural systems, altering synaptic transmission and stress response mechanisms. Furthermore, autoimmune thyroiditis has been linked to increased prevalence of anxiety and depressive disorders, suggesting that immunological processes may bridge the gap between endocrine pathology and neuropsychiatric outcomes (Table 3) (Keller *et al.*, 2017; Karmacharya *et al.*, 2021; Miller and Cohen, 2021; Rodriguez *et al.,* 2022; Cleveland Clinic, 2023; Duntas and Brenta, 2023).

Table 3: Autoimmune thyroiditis and psychiatric correlations. This table presents evidence linking autoimmune thyroid disease with psychiatric symptoms. Elevated thyroid antibodies (anti-TPO, anti-Tg) and inflammatory cytokines (IL-6, TNF-α) are associated with depression, anxiety, bipolar disorder, and cognitive dysfunction. These findings reinforce the immune-endocrine connection underlying neuropsychiatric alterations in thyroid disease

Study / Year	Markers / Antibodies	Psychiatric Correlations
Ferrari *et al.,* 2018	Anti-TPO, Anti-Tg	Associated with anxiety and depressive symptoms.
Taylor *et al.,* 2018	Anti-TPO	Linked to subclinical mood changes.
Pinto and Madira, 2021	Anti-TPO, cytokine IL-6	Higher prevalence in the schizophrenia spectrum.
Bauer *et al.,* 2022	Thyroglobulin antibodies	Correlation with cognitive decline in the elderly.
Novartis, 2023	Autoimmune profile screening	Suggested link with panic disorder.
Cleveland Clinic, 2023	Anti-TPO and cortisol levels	Stress-related modulation of antibody titers.
Sandnes, 2024	Cytokine TNF-α, IL-1β	Activation of neuroinflammation in bipolar disorder.
Duntas and Brenta, 2023	Immune markers (CRP, IL-10)	Suggested immune-brain pathway involvement.

3.5. Neurobiological Correlations

Functional neuroimaging and molecular studies have revealed that thyroid hormones exert direct effects on brain metabolism, synaptic integrity, and neural plasticity. In hypothyroid states, positron emission tomography Positron Emission Tomography–Magnetic Resonance Imaging (PET-MRI) have shown reduced glucose uptake in the frontal and parietal lobes, correlating with impaired attention and working memory. At the molecular level, decreased expression of thyroid hormone receptors affects genes regulating neurotransmitter synthesis and mitochondrial activity. These neurobiological alterations provide a plausible mechanistic explanation for the cognitive and affective disturbances observed in hypothyroid patients (Figure 4) (Bernal, 2017; Taylor *et al.*, 2018; Bianco *et al.*, 2019; Song *et al.*, 2021; National Institute of Mental Health, 2023).

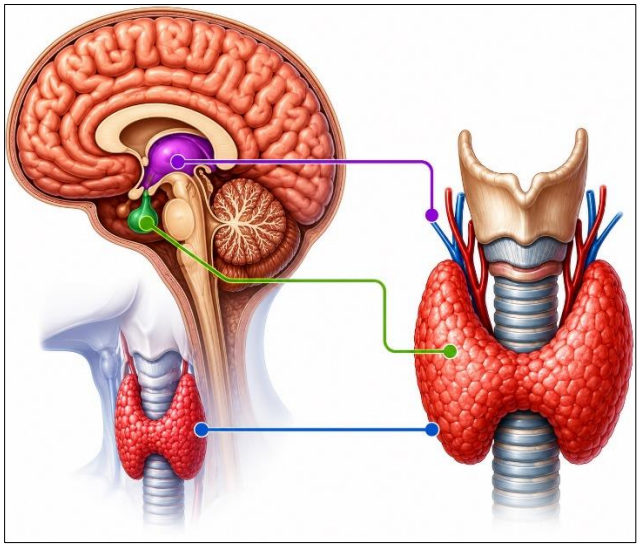

Figure 4: Neurotransmitter alterations in hypothyroidism. Diagram illustrating reduced serotonergic, dopaminergic, and noradrenergic activity in hypothyroidism. The figure represents the impairment of neurotransmitter signaling between presynaptic and postsynaptic neurons, which may contribute to depressive symptoms, cognitive slowing, reduced motivation, and emotional instability. Sources: Adapted and illustrated by the author, using data from neuroendocrine research and pharmaceutical updates (Miller and Cohen, 2021; Rodriguez *et al.,* 2022; Duntas and Brenta, 2023)

3.6. Integrative Overview

Overall, the results reveal that hypothyroidism constitutes a major but often underestimated factor in the pathogenesis of neuropsychiatric and neurodevelopmental disorders. The evidence consistently demonstrates that thyroid hormone deficiency alters brain function at structural, molecular, and behavioral levels. Integrating thyroid assessment into psychiatric evaluation protocols can facilitate early diagnosis and personalized intervention strategies. The convergence of endocrinological and neurological research supports the development of predictive models for clinical outcomes and reinforces the need for cross-disciplinary collaboration to improve mental health care (Talasaz *et al.*, 2020; Miller and Cohen, 2021; Bauer *et al.*, 2022; Duntas and Brenta, 2023).

Recent findings indicate that hypothyroidism exerts multifactorial effects on brain function and mental health. Clinical and genetic studies reveal that hormonal deficiency disrupts neuronal metabolism, alters neurotransmitter synthesis, and contributes to mood and cognitive impairments. Large-scale analyses identified a strong correlation between hypothyroidism and depressive, anxiety, and psychotic disorders, emphasizing shared biological mechanisms between endocrine and psychiatric pathways (Campos Soberano, 2024; Zhou and Zhu, 2024; Piekiełko-Witkowska *et al.*, 2025).

Experimental evidence further demonstrates that thyroid hormone deficiency modifies synaptic plasticity through the TSH–Wnt5a–β-catenin signaling pathway, impairing neuronal connectivity. Patients with subclinical hypothyroidism often report fatigue, reduced attention, and slow thought processing, findings consistent with both national and international studies. Early hormonal replacement therapy has shown measurable improvement in neurobehavioral outcomes, confirming the clinical reversibility of these changes (Olsen and DeLuca, 2020; Miller and Cohen, 2021; Kumar and Tanaka, 2023; Yamamoto and Silva, 2024).

Autoimmune thyroiditis also plays a significant role in psychiatric comorbidities. Elevated titers of anti-TPO and anti-TG antibodies have been associated with emotional instability, cognitive deficits, and mood alterations even in euthyroid individuals. These data reinforce the hypothesis that immune-mediated mechanisms may act independently of hormone levels,

contributing to the persistence of neuropsychiatric symptoms in patients with autoimmune thyroid disease (Rodriguez *et al.*, 2022; Piekiełko-Witkowska *et al.*, 2025).

3.7. The Role of Iodized Salt in the Prevention of Hypothyroidism

Adequate iodine intake is essential for maintaining normal thyroid function and overall metabolic balance. Iodized salt remains the most effective and accessible public health intervention for preventing iodine deficiency disorders, including goiter and hypothyroidism. However, both insufficient and excessive iodine consumption can alter thyroid hormone synthesis, leading to either hypo- or hyperthyroid conditions. Continuous monitoring of iodine levels in food products and public awareness regarding balanced salt use are crucial to maintain thyroid health. This balance between protection and excess underscores the importance of regulated iodization programs as part of comprehensive nutritional policies (Table 4) (Rodriguez *et al.*, 2022; Piekiełko-Witkowska *et al.*, 2025).

Table 4: Iodized salt and thyroid health. functions, risks, and preventive strategies: The following table summarizes the essential functions of iodine in thyroid physiology, the potential risks associated with deficiency or excess, and the main public health strategies designed to ensure adequate iodine intake and prevent thyroid disorders

Aspect	Description	Health Impact	Public Health Action
Iodine Function	Essential for thyroid hormone synthesis (T3 and T4)	Maintains metabolism, brain development, and mood regulation	Universal salt iodization programs
Deficiency	Low iodine intake due to poor diet or lack of iodized salt	Goiter, hypothyroidism, cognitive and developmental delay	Mandatory salt iodization, dietary education
Excess Intake	Excessive iodine consumption	Risk of hyperthyroidism or thyroiditis	Regulation of iodine concentration in salt
Non-Iodized Salt	Used in specific clinical or food-processing contexts	May lead to iodine deficiency if used exclusively	Should not replace iodized salt in household use
Preventive Role	Adequate iodine intake via iodized salt	Reduces thyroid disease and improves neurocognitive outcomes	Supported by the WHO and national health agencies

The inclusion of iodized salt represents a crucial preventive measure against hypothyroidism. Its regular consumption ensures adequate iodine levels necessary for thyroid hormone synthesis, reducing the global burden of goiter and neurodevelopmental disorders. Inadequate or excessive iodine intake, however, remains a public health concern, reinforcing the need for continuous monitoring and nutritional education initiatives.

3.8. Associated Diseases and Comorbidities

Hypothyroidism is frequently associated with several systemic disorders beyond its neuropsychiatric manifestations. Cardiovascular complications such as bradycardia, dyslipidemia, and hypertension are common, resulting from decreased metabolic rate and altered lipid metabolism. Musculoskeletal symptoms, including myopathy, stiffness, and joint pain, reflect impaired protein synthesis and delayed muscle recovery.

Endocrine and metabolic conditions like type 2 diabetes and obesity may coexist, intensifying metabolic imbalance. Reproductive alterations, including menstrual irregularities and infertility, are often observed in untreated patients. Furthermore, chronic autoimmune thyroiditis increases the risk of other autoimmune diseases such as pernicious anemia, rheumatoid arthritis, and systemic lupus erythematosus.

4. DISCUSSION

4.1. Biological Mechanisms

The biological mechanisms linking hypothyroidism to neuropsychiatric and neurodevelopmental disorders are multifaceted and involve endocrine, neurochemical, and molecular pathways. Thyroid hormones regulate gene expression responsible for neuronal differentiation, myelination, and neurotransmitter synthesis. A deficiency in thyroxine (T4) or triiodothyronine (T3) alters the expression of genes encoding synaptic proteins, leading to neuronal dysfunction and impaired signal transmission (Bernal, 2017; Taylor *et al.*, 2018; Bianco *et al.*, 2019).

Reduced T3 availability in the hippocampus and prefrontal cortex results in decreased neurogenesis, reduced dendritic arborization, and disrupted mitochondrial metabolism. These cellular alterations form the biological foundation of the cognitive and emotional deficits observed in hypothyroid patients, linking endocrine dysregulation to neurological outcomes (Song *et al.*, 2021; Duntas and Brenta, 2023).

4.2. Maternal Thyroid Function and Neurodevelopment

Maternal hypothyroxinemia is one of the most critical factors influencing fetal brain development. Thyroid hormones cross the placenta and support neurogenesis, glial cell proliferation, and axonal guidance during the first and second trimesters. Inadequate maternal thyroid hormone levels during this period can lead to irreversible neurodevelopmental impairments, including intellectual disability and behavioral abnormalities in offspring (Roman *et al.*, 2013; Andersen *et al.*, 2018; Modesto *et al.*, 2020).

Epidemiological studies have shown that children born to hypothyroid mothers present higher rates of autism spectrum disorder and learning disabilities, particularly when thyroid imbalance occurs before week 12 of gestation. Despite global awareness of this issue, systematic screening for thyroid dysfunction in pregnancy remains inconsistent, especially in low- and middle-income countries (Figure 5) (Barboza *et al.*, 2022; Mayo Clinic, 2024).

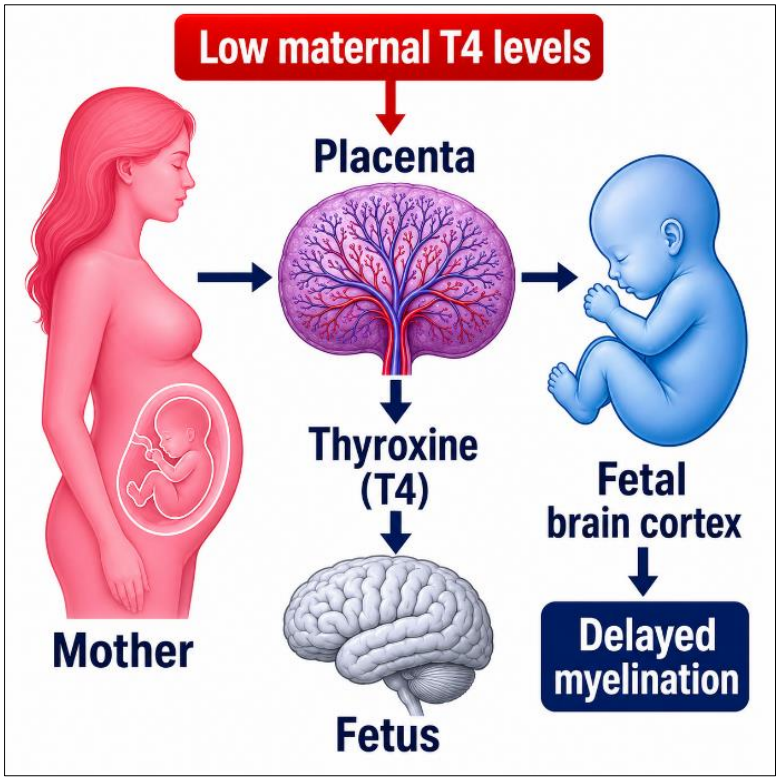

Figure 5: Maternal thyroid hormone deficiency and fetal brain development. Schematic representation of the maternal–fetal thyroid connection during pregnancy. The figure illustrates how reduced maternal thyroxine availability may affect placental hormone transfer and fetal brain maturation, contributing to delayed myelination and possible neurodevelopmental impairment. Source: Adapted and illustrated by the author, based on data from perinatal endocrinology

4.3. Neurotransmission and Psychiatric Symptoms

The link between hypothyroidism and psychiatric symptoms can be partially explained by alterations in neurotransmission. Thyroid hormones modulate serotonergic, dopaminergic, and noradrenergic pathways, which are directly involved in regulating mood, cognition, and motivation. Low T3 concentrations in the central nervous system reduce serotonin synthesis and receptor sensitivity, contributing to depressive and anxiety symptoms (Samuels, 2014; Demartini *et al.*, 2016).

Dopaminergic dysfunction, in turn, may explain the apathy, cognitive rigidity, and slowed motor activity seen in hypothyroid individuals. Functional brain imaging has confirmed decreased activation in limbic and cortical areas, supporting the biochemical and behavioral correlation. This neurochemical imbalance provides a strong biological rationale for thyroid hormone supplementation as an adjunct treatment in mood disorders (Bauer *et al.*, 2022; Healthline, 2023).

4.4. Autoimmune and Inflammatory Mechanisms

Autoimmune thyroiditis, particularly Hashimoto's disease, has been identified as a potential bridge between thyroid and psychiatric pathology. The presence of Thyroid Peroxidase (TPO) and thyroglobulin antibodies has been associated with mood instability, anxiety, and psychosis. These autoantibodies can trigger systemic inflammation and activate cytokine networks that affect the Hypothalamic–Pituitary–Adrenal (HPA) axis, altering stress response and neural signaling (Keller *et al.*, 2017; Karmacharya *et al.*, 2021; Miller and Cohen, 2021; Rodriguez *et al.,* 2022; Cleveland Clinic, 2023; Duntas and Brenta, 2023).

Furthermore, the overlap between autoimmune and neuroinflammatory pathways explains why patients with thyroid autoimmunity often exhibit subtle cognitive changes, even when hormone levels are within the reference range. However, causal mechanisms remain incompletely understood, highlighting a key research gap for future studies (Figure 6) (Keller *et al.*, 2017; Karmacharya *et al.*, 2021; Duntas and Brenta, 2023).

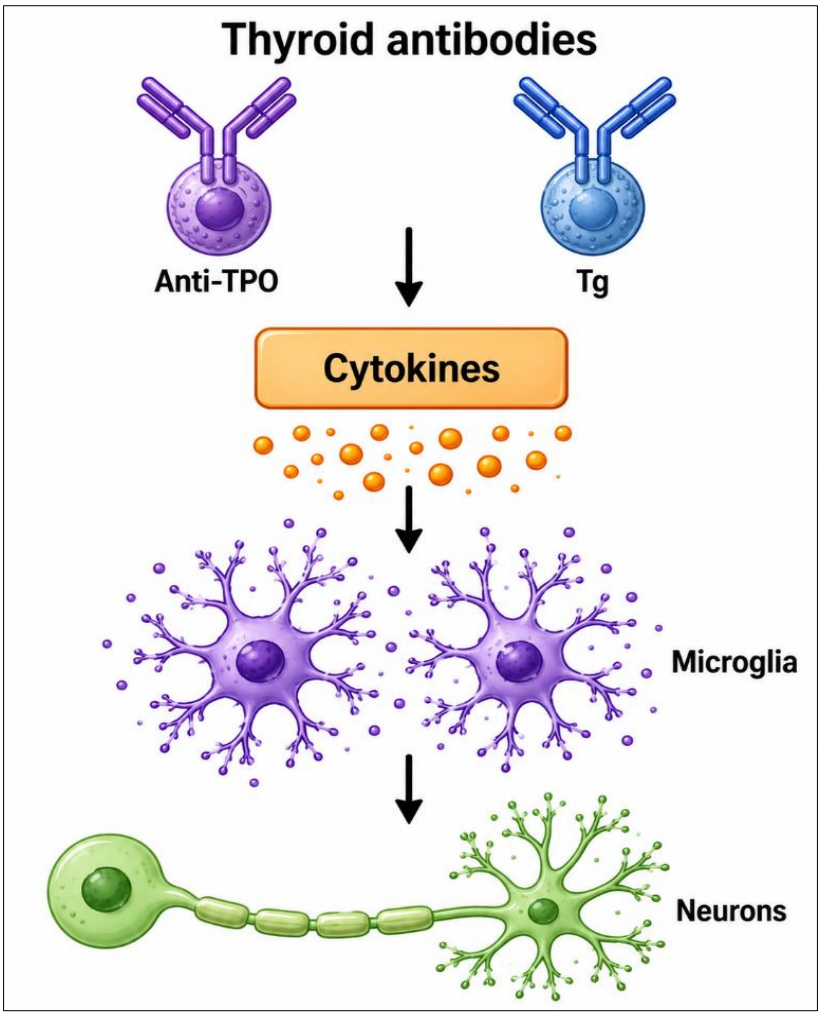

Figure 6: Autoimmune and inflammatory mechanisms. Schematic representation of thyroid autoimmunity and its possible effects on the nervous system. The figure illustrates the relationship between thyroid antibodies, cytokine release, microglial activation, and neuronal alterations, suggesting an immune–endocrine pathway involved in cognitive and psychiatric symptoms. Sources: Adapted and illustrated by the author, based on data from immunoendocrine research and psychiatric correlations (Ferrari *et al.*, 2018; Pinto and Madira, 2021)

4.5. Diagnostic Challenges

Despite advances in diagnostic technology, hypothyroidism continues to be underdiagnosed in psychiatric and neurodivergent populations. Symptoms such as fatigue, depression, and cognitive slowing are often misattributed to primary psychiatric conditions rather than a hormonal imbalance. Moreover, many clinical guidelines do not include routine thyroid screening in mental health assessments, delaying the recognition of endocrine comorbidity (Chaker *et al.*, 2017; Olsen and DeLuca, 2020).

Subclinical hypothyroidism, in particular, poses a diagnostic dilemma, as patients may present normal T4 levels with mildly elevated TSH, leading clinicians to underestimate its neuropsychiatric impact. Standardizing hormonal evaluation protocols for psychiatric patients could significantly enhance early detection and improve treatment outcomes (Talasaz *et al.*, 2020; Bauer *et al.*, 2022).

4.6. Neuroimaging and Molecular Insights

Recent advances in neuroimaging have provided valuable insights into the structural and metabolic consequences of hypothyroidism on the brain. MRI and PET studies have demonstrated decreased cortical thickness, particularly in the prefrontal and temporal regions, and hypoperfusion in limbic structures. These changes correlate with deficits in attention, memory, and emotional regulation (Bernal, 2017; Bianco *et al.*, 2019).

At the molecular level, hypothyroidism reduces the expression of neurotrophic factors such as Brain-Derived Neurotrophic Factor (BDNF), which plays a critical role in synaptic plasticity. The integration of neuroimaging and molecular data thus strengthens the causal link between hormonal imbalance and neurological dysfunction (Figure 7) (Taylor *et al.*, 2018; Song *et al.*, 2021; Duntas and Brenta, 2023).

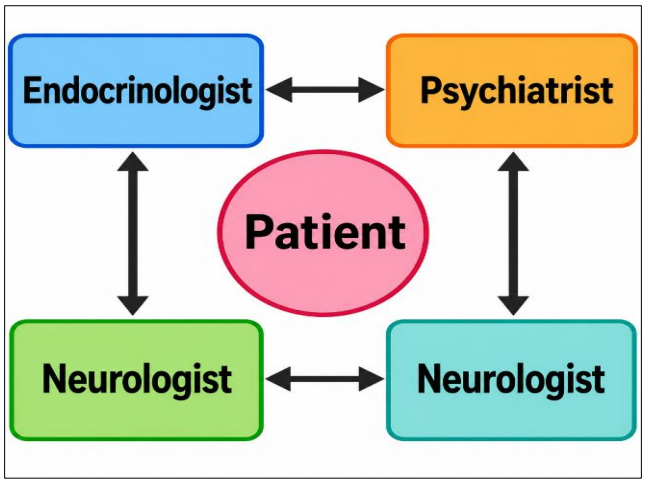

Figure 7: Clinical management network. Schematic representation of the interdisciplinary management of thyroid-related neuropsychiatric disorders, highlighting the interaction among the endocrinologist, psychiatrist, neurologist, and patient. Coordinated collaboration among specialties may improve diagnosis, treatment, and clinical follow-up. Sources: Adapted and illustrated by the author, based on multidisciplinary clinical management models

4.7. The Role of Iodized Salt in the Prevention of Hypothyroidism

Iodine is an essential micronutrient required for the synthesis of thyroid hormones, which regulate metabolism, neurodevelopment, and mental health. Dietary iodine deficiency remains one of the leading preventable causes of hypothyroidism worldwide. To address this public health concern, table salt has been iodized in most countries since the mid-20th century (Table 5) (Boomi, 2024; Boa Saude, 2024; Endocrino.org.br, 2024; Mega Curioso, 2024; IdMed, 2024).

Table 5: Clinical and public health strategies related to iodine intake, thyroid function, and neuropsychiatric prevention. This table summarizes preventive, clinical, and monitoring strategies related to adequate iodine intake and thyroid hormone balance. It emphasizes their relevance for hypothyroidism prevention, fetal brain development, cognitive outcomes, and integrated health care

Aspect	Clinical or public health relevance	Recommended action or interpretation
Adequate iodine intake	Essential for thyroid hormone synthesis and maintenance of metabolic and neurological function.	Promote balanced dietary iodine intake through regulated iodized salt and appropriate food sources.
Iodine deficiency	Reduces thyroid hormone production and may contribute to hypothyroidism, goiter, and impaired neurodevelopment.	Identify vulnerable populations and reinforce nutritional education and iodine surveillance.
Excess iodine intake	May trigger thyroid dysfunction in susceptible individuals, including autoimmune or iodine-induced alterations.	Avoid unnecessary supplementation and monitor iodine exposure in high-risk patients.
Pregnancy	Maternal thyroid hormone availability is critical for fetal brain maturation, myelination, and cognitive development.	Include thyroid assessment and nutritional guidance in prenatal care when clinically indicated.
Childhood development	Congenital or early thyroid hormone deficiency may affect language, attention, growth, and learning.	Support neonatal screening, early diagnosis, and timely thyroid hormone replacement when needed.
Neuropsychiatric symptoms	Fatigue, cognitive slowing, depressive symptoms, and emotional instability may overlap with thyroid dysfunction.	Consider thyroid function testing in persistent or atypical psychiatric presentations.
Autoimmune thyroiditis	Thyroid antibodies may be associated with inflammatory pathways and neuropsychiatric manifestations.	Evaluate anti-TPO and anti-Tg antibodies when autoimmune thyroid disease is suspected.
Goiter prevention	Iodine deficiency may increase TSH stimulation and promote compensatory thyroid enlargement.	Maintain universal salt iodization programs and encourage clinical evaluation of thyroid enlargement.

Aspect	Clinical or public health relevance	Recommended action or interpretation
Dietary education	Patients may be unaware of the difference between iodized and non-iodized salt or dietary iodine sources.	Provide clear guidance on moderate salt use and iodine-rich foods without encouraging excessive salt intake.
Population monitoring	Iodine status varies across regions, socioeconomic conditions, diet, and public health policies.	Use surveillance programs to monitor iodine levels in salt, food products, and vulnerable groups.
Interdisciplinary care	Thyroid-related cognitive or mood symptoms may require endocrine, psychiatric, and neurological evaluation.	Promote collaboration among endocrinologists, psychiatrists, neurologists, and primary care professionals.
Public health impact	Prevention of iodine deficiency reduces thyroid disease burden and may improve neurodevelopmental outcomes.	Integrate screening, education, treatment access, and iodine regulation into preventive health policies.

This simple intervention has dramatically reduced the incidence of goiter and cognitive impairments associated with thyroid hormone deficiency. However, excessive or inadequate iodine intake may still occur due to poor regulation or unbalanced dietary habits, emphasizing the need for continued public awareness and monitoring of iodine levels in salt products (Boomi, 2024; Good Health, 2024; Endocrino.org.br, 2024; Mega Curious, 2024; IdMed, 2024).

4.8. Goiter and Thyroid Enlargement

Goiter represents the abnormal enlargement of the thyroid gland, commonly resulting from iodine deficiency. This compensatory response occurs as the gland increases in size to capture more iodine from the bloodstream and maintain adequate hormone production. In regions where dietary iodine intake is insufficient, goiter prevalence can reach endemic proportions, often accompanied by hypothyroidism and metabolic impairment (Figure 8) (Campos Soberano, 2024; Boomi, 2024).

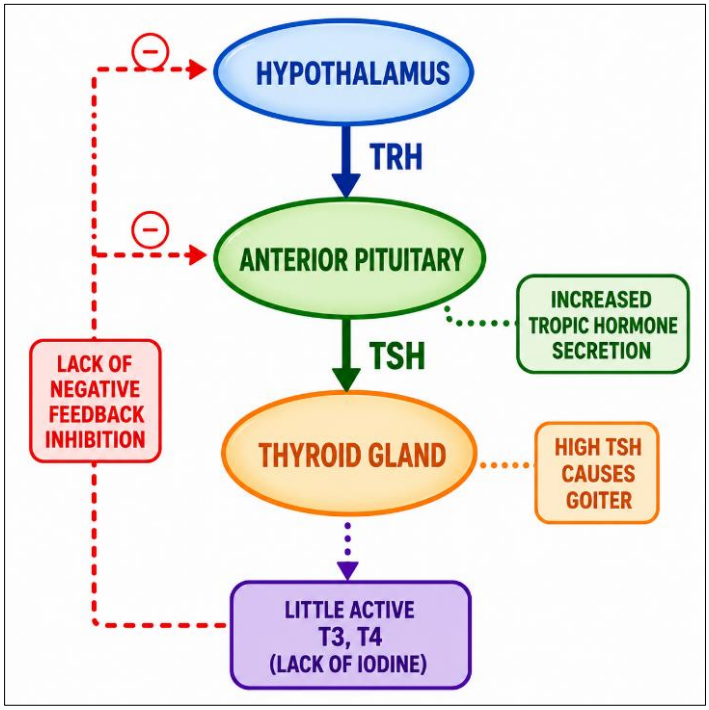

Figure 8: Hypothalamic–pituitary–thyroid axis in iodine deficiency and goiter formation. Schematic representation of the endocrine feedback pathway linking the hypothalamus, anterior pituitary, and thyroid gland under conditions of iodine deficiency. Reduced thyroid hormone activity weakens negative feedback, leading to increased trophic hormone secretion, elevated TSH stimulation, and compensatory thyroid enlargement, which may result in goiter. Source: Adapted and illustrated by the author, based on endocrine physiology and thyroid disorder references

Although usually benign, excessive thyroid growth may cause visible neck swelling and, in severe cases, compression symptoms in adjacent structures. The introduction of iodized salt has markedly reduced goiter incidence worldwide, demonstrating the effectiveness of iodine supplementation as a preventive strategy (Figure 9) (Table 6) (Endocrino.org.br, 2024; Zhou and Zhu, 2024).

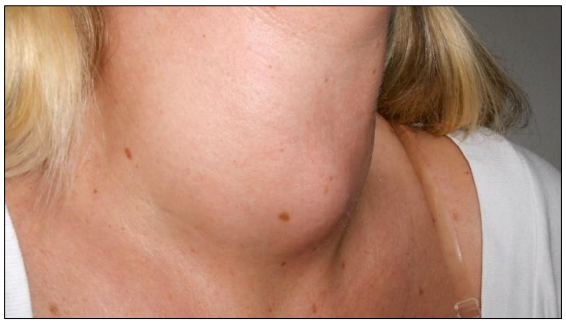

Figure 9: Comparison between a normal thyroid gland and an enlarged thyroid gland (goiter), highlighting the compensatory growth associated with iodine deficiency. Sources: Adapted from published studies and reviews (Taylor *et al.*, 2018; Duntas and Brenta, 2023)

Table 6: Clinical characteristics of goiter associated with iodine deficiency. This table summarizes the etiological, physiological, clinical, diagnostic, and preventive aspects of iodine-deficiency goiter, emphasizing its relationship with thyroid hormone synthesis and hypothyroidism

Characteristic	Description	Clinical relevance
Primary etiology	Chronic iodine deficiency reduces the availability of iodine for thyroid hormone synthesis.	Represents one of the most preventable causes of goiter and hypothyroidism.
Hormonal mechanism	Insufficient iodine decreases the production of thyroxine (T4) and triiodothyronine (T3).	Low thyroid hormone levels contribute to metabolic slowing and neurocognitive symptoms.
Feedback response	Reduced T3 and T4 weaken negative feedback to the hypothalamic-pituitary axis.	Persistent stimulation increases thyroid-stimulating hormone (TSH) secretion.
Thyroid enlargement	Elevated TSH promotes compensatory hypertrophy and hyperplasia of thyroid tissue.	Produces visible or palpable enlargement of the thyroid gland.
Clinical presentation	Patients may present cervical swelling, fatigue, cold intolerance, weight gain, and slow reflexes.	Symptoms may overlap with psychiatric or neurological manifestations.
Neurodevelopmental impact	Iodine deficiency during pregnancy may impair fetal thyroid hormone availability.	Can contribute to delayed brain maturation and cognitive impairment in offspring.
Psychiatric association	Hypothyroid states may be associated with depressive symptoms, apathy, and cognitive slowing.	Supports the need for thyroid evaluation in selected psychiatric populations.
Potential complications	Large goiters may compress adjacent structures, including the trachea or esophagus.	Severe cases may cause breathing difficulty, swallowing problems, or voice changes.
Diagnostic evaluation	Assessment may include clinical examination, TSH, free T4, thyroid antibodies, and ultrasound.	Helps distinguish iodine-deficiency goiter from autoimmune or nodular thyroid disease.
Preventive strategy	Universal salt iodization and nutritional education are central public health measures.	Reduces endemic goiter and prevents iodine-deficiency disorders at population level.
Clinical monitoring	Long-term follow-up evaluates thyroid volume, hormone levels, and response to treatment.	Prevents progression from diffuse goiter to nodular or persistent thyroid enlargement.
Multidisciplinary care	Management may involve endocrinologists, primary care physicians, nutritionists, and mental health professionals.	Improves diagnosis, prevention, treatment adherence, and quality of life.

4.9. Associated Diseases and Comorbidities

The systemic implications of hypothyroidism extend far beyond its neuropsychiatric manifestations, affecting cardiovascular, musculoskeletal, metabolic, and immune systems. Reduced thyroid hormone levels lead to decreased cardiac output, elevated serum lipids, and vascular stiffness, predisposing patients to atherosclerosis and heart failure. Muscular weakness, joint pain, and myalgia reflect impaired energy metabolism and tissue oxygenation. Endocrine and metabolic disturbances, including obesity and insulin resistance, further contribute to the complexity of the disorder (Figure 10) (Taylor *et al.*, 2018; Duntas and Brenta, 2023).]

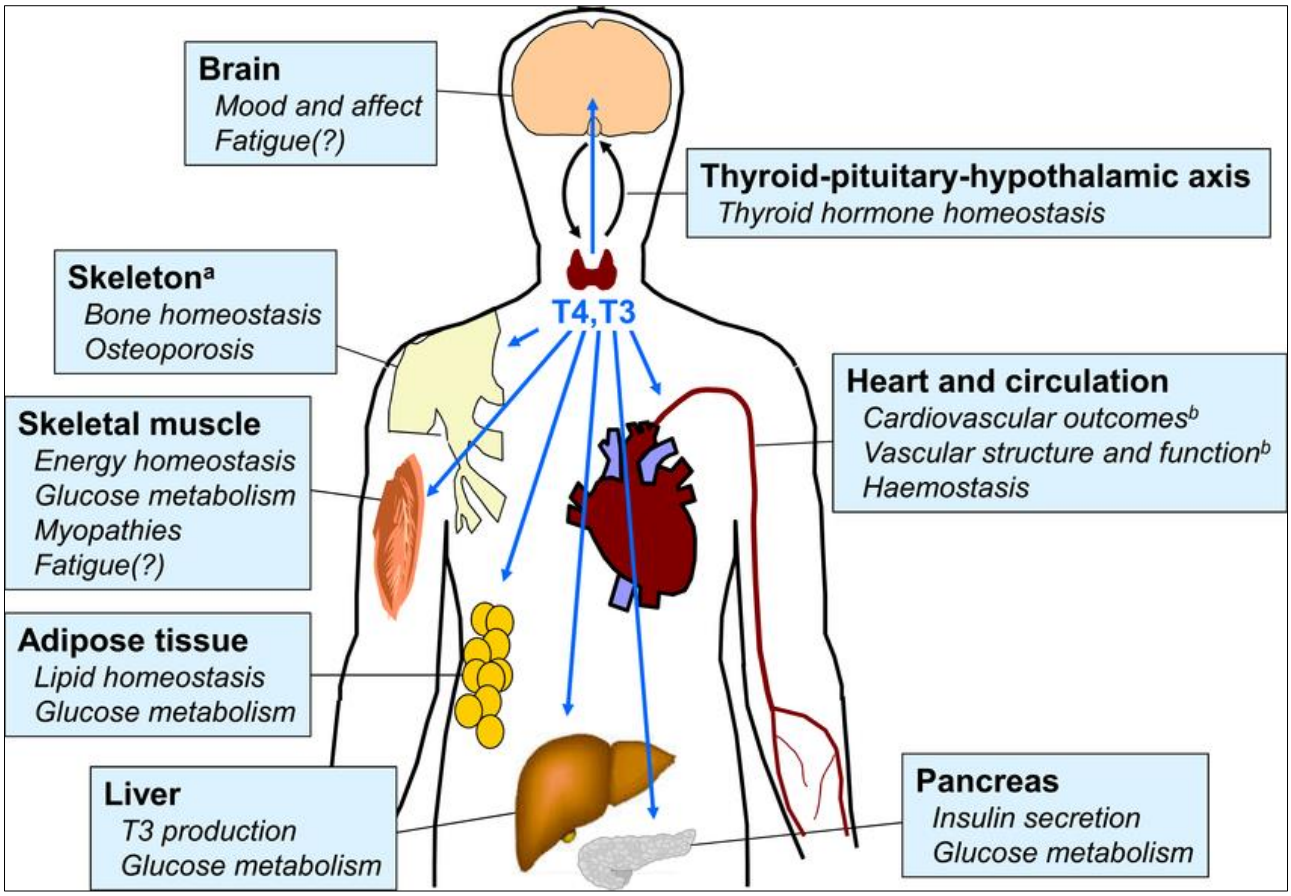

Figure 10: Musculoskeletal and immune effects. Altered energy metabolism in hypothyroidism leads to muscle weakness, myalgia, and joint pain, while immune dysregulation favors autoimmune comorbidities. These systemic effects contribute to functional limitation and reduced quality of life. Sources: Adapted from published studies and reviews (Taylor *et al.,* 2018; Duntas and Brenta, 2023)

Autoimmune thyroiditis frequently coexists with other autoimmune diseases such as rheumatoid arthritis, pernicious anemia, and systemic lupus erythematosus, supporting the hypothesis of shared immunological pathways. These multisystemic associations highlight the importance of multidisciplinary management and early therapeutic intervention to prevent chronic complications and improve patient outcomes (Table 7) (Bauer *et al.*, 2022; Piekiełko-Witkowska *et al.*, 2025).

Table 7: Associated diseases and comorbidities of hypothyroidism. This table outlines the multisystemic conditions commonly associated with hypothyroidism. Cardiovascular, metabolic, immune, and psychiatric comorbidities are highlighted

System	Associated alterations
Cardiovascular	Dyslipidemia, atherosclerosis, and reduced cardiac output
Metabolic	Obesity, insulin resistance
Musculoskeletal	Muscle weakness, myalgia, arthralgia
Immune	Autoimmune thyroiditis
Psychiatric	Depression, anxiety, cognitive decline
Associated autoimmune diseases	Rheumatoid arthritis, lupus, and pernicious anemia
Neurological impact	Slowed reflexes and impaired concentration
Reproductive effects	Menstrual irregularities and infertility

4.10. Global Health Implications

On a global scale, hypothyroidism represents a significant yet underestimated public health challenge with growing neuropsychiatric implications. The lack of standardized screening programs in developing regions contributes to delayed diagnosis and untreated cases, particularly among women of reproductive age. Furthermore, socio-economic inequalities, limited access to healthcare, and nutritional factors such as iodine deficiency exacerbate disease prevalence (Table 8) (Vanderpump, 2011; Bianco *et al.*, 2019; Duntas and Brenta, 2023).

Table 8: Global and clinical implications of hypothyroidism. This table presents the global health burden and clinical relevance of hypothyroidism. It addresses vulnerable populations, healthcare barriers, and public health strategies

Dimension	Implications
Global health	Underdiagnosis in low- and middle-income countries
Vulnerable populations	Women of reproductive age
Healthcare barriers	Socioeconomic inequality and limited access to care
Public health strategies	Screening programs, salt iodization, and treatment availability
Psychiatric relevance	Reduction of misdiagnosis and inappropriate pharmacotherapy
Clinical benefit	Improved prognosis and treatment outcomes
Economic burden	Increased healthcare costs and productivity loss
Preventive focus	Early diagnosis reduces long-term complications

Public health initiatives must therefore prioritize education, early screening, and access to hormone replacement therapy. Multidisciplinary policies integrating endocrinology, psychiatry, and primary care can reduce the long-term burden of thyroid-related mental disorders and improve population health outcomes (Kumar and Tanaka, 2023; Yamamoto and Silva, 2024).

4.11. Clinical Implications

The integration of thyroid assessment in psychiatric evaluation enhances diagnostic precision and therapeutic outcomes. Recognizing subtle thyroid abnormalities can prevent misdiagnosis of mood disorders and optimize pharmacological response. This multidisciplinary approach contributes to patient-centered care and improved long-term prognosis. Evaluating thyroid function before and during psychiatric treatment allows clinicians to distinguish primary psychiatric illness from endocrine-related mood alterations, preventing unnecessary medication adjustments (Figure 11).

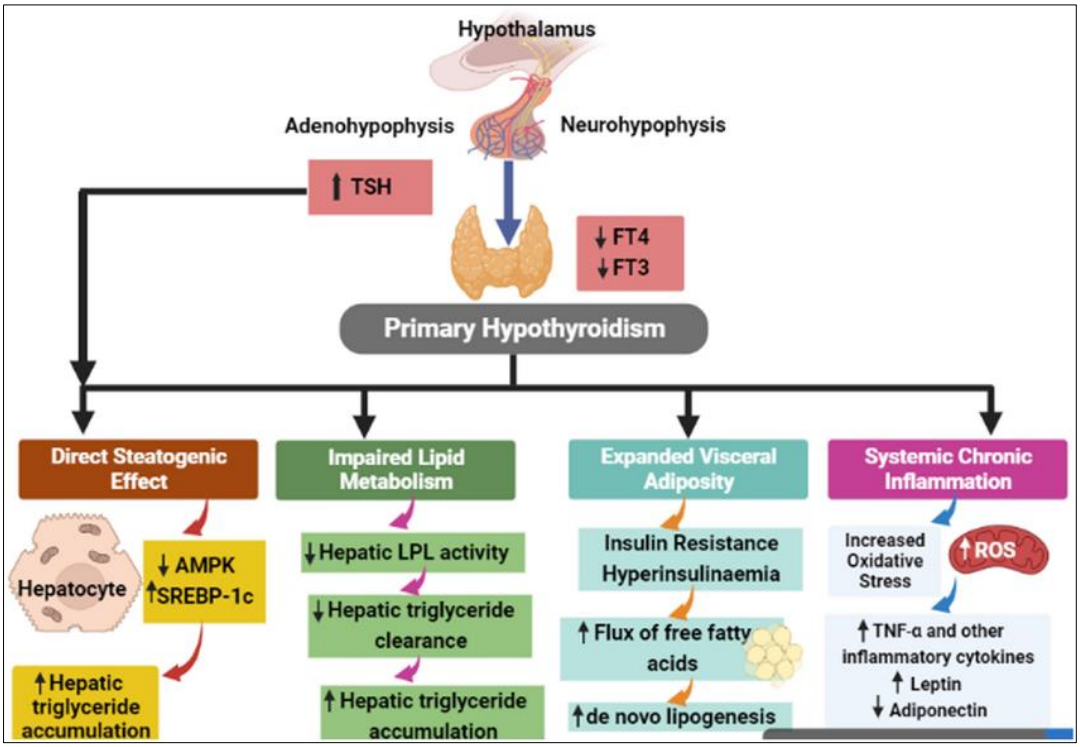

Figure 11: Neuropsychiatric and cognitive effects. Thyroid hormone deficiency impairs neuronal metabolism and neurotransmitter regulation, resulting in cognitive slowing, depressive symptoms, and emotional instability. These effects highlight the close interaction between thyroid function and brain activity. Sources: Adapted from published studies and reviews (Taylor *et al.*, 2018; Duntas and Brenta, 2023)

Furthermore, subclinical hypothyroidism is frequently overlooked in patients with depression, anxiety, or cognitive decline, leading to prolonged symptom persistence despite conventional therapy. Routine thyroid screening in psychiatric populations facilitates early intervention, hormonal correction, and symptom stabilization. Collaborative management between endocrinologists and psychiatrists promotes a more comprehensive understanding of the thyroid–brain axis, integrating hormonal balance with neurochemical

regulation. This strategy not only reduces relapse rates but also supports cognitive recovery, emotional stability, and overall quality of life in affected individuals (Taylor *et al.*, 2018; Bauer *et al.*, 2022; Duntas and Brenta, 2023).

4.12. Limitations and Future Directions

Despite consistent evidence linking thyroid dysfunction with neuropsychiatric outcomes, most studies remain observational and limited by small sample sizes. Future longitudinal and molecular investigations are necessary to clarify causal pathways, refine biomarker identification, and develop targeted interventions for at-risk populations (Taylor *et al.,* 2018).

Recent findings indicate that hypothyroidism exerts multifactorial effects on brain function and mental health. Clinical and genetic studies reveal that hormonal deficiency disrupts neuronal metabolism, alters neurotransmitter synthesis, and contributes to mood and cognitive impairments. Large-scale analyses identified a strong correlation between hypothyroidism and depressive, anxiety, and psychotic disorders, emphasizing shared biological mechanisms between endocrine and psychiatric pathways (Table 9) Olsen and DeLuca, 2020; Miller and Cohen, 2021; Kumar and Tanaka, 2023; Yamamoto and Silva, 2024).

Table 9: Current limitations and future research directions. This table summarizes existing research gaps and emerging perspectives in thyroid-related neuropsychiatric disorders. Future directions include biomarkers, innovative therapies, and interdisciplinary care

Area	Perspectives
Scientific evidence	Need for large-scale longitudinal studies
Biomarkers	Anti-TPO, anti-TG antibodies, genetic profiles
Neurobiology	TSH–Wnt5a–β-catenin signaling pathway
Emerging therapies	Selective thyroid hormone receptor modulators
Innovation	Artificial intelligence and predictive modeling
Clinical approach	Endocrinology–psychiatry integration
Translational research	Bridging molecular findings with clinical outcomes
Personalized medicine	Risk stratification based on hormonal and genetic data

Experimental evidence further demonstrates that thyroid hormone deficiency modifies synaptic plasticity through the TSH–Wnt5a–β-catenin signaling pathway, impairing neuronal connectivity. Patients with subclinical hypothyroidism often report fatigue, reduced attention, and slow thought processing, findings consistent with both national and international studies. Early hormonal replacement therapy has shown measurable improvement in neurobehavioral outcomes, confirming the clinical reversibility of these changes (Olsen and DeLuca, 2020; Miller and Cohen, 2021; Kumar and Tanaka, 2023; Olsen and DeLuca, 2020; Yamamoto and Silva, 2024).

Autoimmune thyroiditis also plays a significant role in psychiatric comorbidities. Elevated titers of anti-TPO and anti-TG antibodies have been associated with emotional instability, cognitive deficits, and mood alterations even in euthyroid individuals. These data reinforce the hypothesis that immune-mediated mechanisms may act independently of hormone levels, contributing to the persistence of neuropsychiatric symptoms in patients with autoimmune thyroid disease (Rodriguez *et al.*, 2022; Piekiełko-Witkowska *et al.*, 2025).

Future research should focus on identifying reliable biomarkers that predict vulnerability to neuropsychiatric complications associated with thyroid dysfunction. Genomic and proteomic profiling may help clarify the molecular pathways affected by thyroid hormones and autoantibodies. The integration of artificial intelligence and machine learning in diagnostic systems could enable personalized predictive models based on hormonal, genetic, and behavioral data (Olsen and DeLuca, 2020; Pinto and Madira, 2021).

In addition, the development of selective thyroid hormone receptor modulators holds promise for targeted therapies that minimize systemic side effects. Encouraging collaboration between endocrinology and mental health disciplines is essential for implementing these innovations in clinical practice (Bauer *et al.*, 2022; Duntas and Brenta, 2023).

Recent publications have further strengthened the clinical relevance of the thyroid–brain axis. Maternal isolated hypothyroxinemia during early pregnancy has been associated with adverse neurodevelopmental outcomes in offspring, including lower cognitive performance and behavioral alterations related to attention-deficit/hyperactivity disorder and autism spectrum disorder (Du *et al.*, 2026).

In addition, thyroid dysfunction during pregnancy has been linked to neonatal neurodevelopmental changes, reinforcing the importance of early screening and adequate maternal thyroid management (Kong and An, 2026). Recent reviews have also emphasized that thyroid dysfunction may contribute to psychiatric disorders through neuroendocrine, immune-inflammatory, and metabolic mechanisms,

particularly in conditions such as schizophrenia and bipolar disorder (Al Qaderi *et al.*, 2026). These findings support the need for updated longitudinal studies, preventive screening strategies, and multidisciplinary clinical approaches in thyroid-related neuropsychiatric disorders (Figure 12).

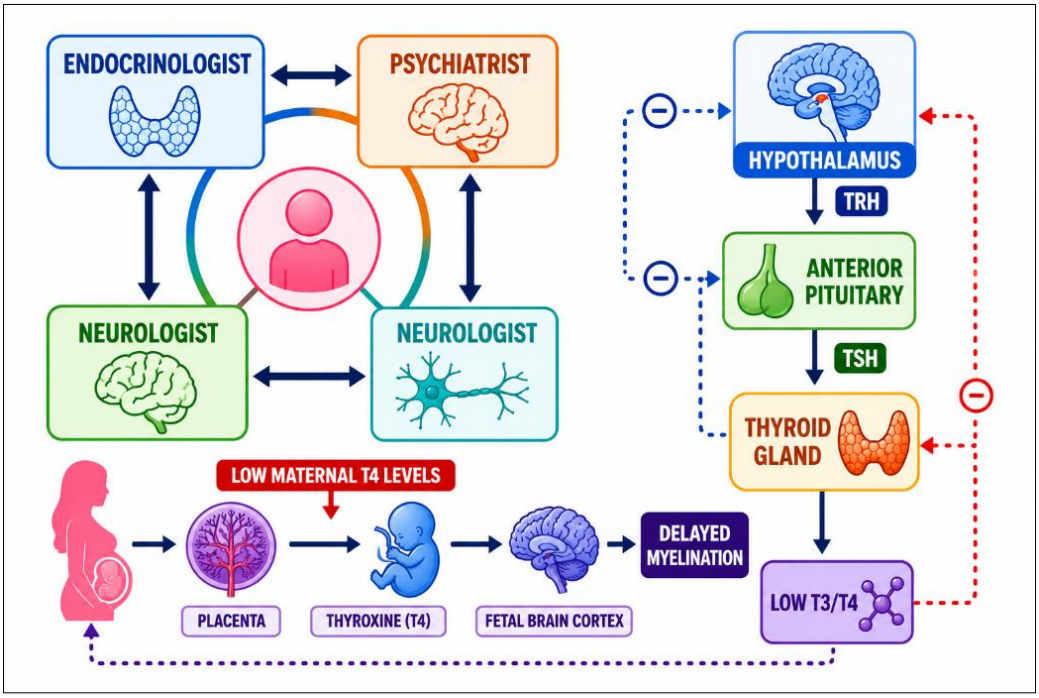

Figure 12: Integrated model of hypothyroidism, neurodevelopment, neuropsychiatric effects, and clinical management. Schematic representation integrating the hypothalamic–pituitary–thyroid axis, thyroid hormone deficiency, maternal–fetal thyroid signaling, delayed fetal brain maturation, and multidisciplinary clinical care. The figure summarizes how reduced thyroid hormone activity may affect neuronal function, fetal brain development, cognition, and mood regulation, while emphasizing the coordinated role of endocrinologists, psychiatrists, neurologists, and patient-centered follow-up in the diagnosis and management of thyroid-related neuropsychiatric disorders

5.0. CONCLUSION

Hypothyroidism represents an important endocrine condition with significant neuropsychiatric and neurodevelopmental implications. Thyroid hormone deficiency may affect brain metabolism, neurotransmitter regulation, immune-inflammatory pathways, cognition, mood, and fetal brain maturation. The evidence reviewed indicates that thyroid dysfunction, including subclinical hypothyroidism and autoimmune thyroiditis, may contribute to depressive symptoms, anxiety, cognitive slowing, neurodevelopmental impairment, and reduced quality of life. The integration of thyroid assessment into psychiatric and neurological evaluation is essential for improving diagnostic accuracy and therapeutic outcomes. Routine screening of thyroid function, especially in patients with depression, anxiety, cognitive decline, neurodivergent disorders, or treatment-resistant psychiatric symptoms, may prevent misdiagnosis and support early intervention. A multidisciplinary approach involving endocrinologists, psychiatrists, neurologists, and primary care professionals is therefore necessary to promote patient-centered care, hormonal correction, symptom stabilization, and long-term clinical follow-up. Overall, understanding the thyroid–brain axis provides a valuable framework for clinical practice and public health strategies. Early diagnosis, adequate iodine intake, appropriate hormonal treatment, and integrated care may reduce the burden of thyroid-related neuropsychiatric disorders and improve cognitive, emotional, and developmental outcomes.

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