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ASSOCIATION BETWEEN OBESITY AND HYPOTHYROIDISM: A COMPREHENSIVE REVIEW

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ABSTRACT

Obesity and hypothyroidism are among the most prevalent chronic disorders worldwide and frequently coexist in clinical practice. Although hypothyroidism has traditionally been regarded as a contributing factor to weight gain because of its effects on basal metabolic rate and energy expenditure, accumulating evidence suggests that obesity itself may substantially influence thyroid function through endocrine, metabolic, and inflammatory mechanisms. This comprehensive review summarizes current evidence regarding the association between obesity and hypothyroidism, focusing on epidemiological findings, pathophysiological mechanisms, thyroid autoimmunity, metabolic phenotypes, pediatric and adult populations, and therapeutic implications. Evidence from systematic reviews, longitudinal cohort studies, Mendelian randomization analyses, and observational investigations indicates that obesity is associated with an increased prevalence of both overt and subclinical hypothyroidism. Adipose tissue-derived mediators, particularly leptin and pro-inflammatory cytokines, appear to influence the hypothalamic–pituitary–thyroid axis, alter peripheral thyroid hormone metabolism, and promote autoimmune thyroid disease. However, mild elevations of thyroid-stimulating hormone observed in many obese individuals, especially children, often represent adaptive physiological responses rather than permanent thyroid dysfunction and frequently normalize following weight reduction. Current evidence further suggests that improving metabolic health and reducing excess body weight have greater effects on thyroid function than thyroid hormone replacement alone in patients without overt hypothyroidism. Overall, the available literature supports a bidirectional but predominantly obesity-driven relationship between obesity and thyroid dysfunction. Understanding these mechanisms may improve diagnostic accuracy, reduce unnecessary pharmacological treatment, and facilitate individualized management strategies for patients presenting with both conditions.

KEYWORDS

Obesity, Hypothyroidism, Subclinical Hypothyroidism, Thyroid Function

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1. Introduction

Obesity and hypothyroidism are among the most common chronic disorders worldwide, and their prevalence continues to increase, creating a significant public health burden. Obesity is associated with numerous metabolic, cardiovascular, and endocrine complications, whereas hypothyroidism is characterized by insufficient thyroid hormone production, resulting in impaired metabolism and reduced energy expenditure (Bambini et al., 2023; Biondi, 2024).

Thyroid hormones play a fundamental role in regulating basal metabolic rate, thermogenesis, lipid metabolism, and glucose homeostasis. For many years, hypothyroidism has been considered a major contributor to weight gain because reduced thyroid hormone activity decreases energy expenditure. However, growing evidence suggests that the relationship between obesity and hypothyroidism is more complex than previously assumed. Several epidemiological studies have demonstrated that individuals with obesity have a higher prevalence of both overt and subclinical hypothyroidism, while obesity itself may induce alterations in thyroid function through endocrine and inflammatory mechanisms (Wang et al., 2021; Yan et al., 2022).

Adipose tissue is now recognized as an active endocrine organ that secretes numerous biologically active molecules, including leptin, adiponectin, and pro-inflammatory cytokines. These mediators influence the hypothalamic–pituitary–thyroid axis, peripheral thyroid hormone metabolism, and immune function. Chronic low-grade inflammation associated with obesity has also been implicated in the development of thyroid autoimmunity, particularly Hashimoto's thyroiditis, suggesting that obesity may contribute to thyroid dysfunction beyond its metabolic effects (Wojciechowska et al., 2022; Biondi, 2024).

Despite increasing research interest, the causal relationship between obesity and hypothyroidism remains controversial. Elevated thyroid-stimulating hormone (TSH) concentrations are frequently observed in individuals with obesity, particularly in children, but these abnormalities often normalize following weight reduction without pharmacological treatment, indicating that they may represent adaptive physiological

responses rather than permanent thyroid disease (Tersander et al., 2025). Furthermore, recent longitudinal and genetic studies suggest that obesity may increase the risk of hypothyroidism, whereas hypothyroidism contributes only modestly to body weight gain (Wang et al., 2021).

Given these inconsistencies, a comprehensive evaluation of the available evidence is necessary. Therefore, the aim of this review is to summarize current knowledge regarding the association between obesity and hypothyroidism, with particular emphasis on epidemiology, underlying pathophysiological mechanisms, thyroid autoimmunity, and the clinical implications for diagnosis and treatment.

2. Methodology

This comprehensive review was conducted to summarize and critically evaluate the current evidence regarding the association between obesity and hypothyroidism. The manuscript was based exclusively on peer-reviewed scientific articles published in English between 2020 and 2025, with additional landmark studies included when necessary to provide scientific context. The literature analyzed comprised systematic reviews, meta-analyses, narrative reviews, Mendelian randomization studies, longitudinal cohort studies, retrospective cohort studies, and cross-sectional investigations.

The reviewed publications focused on the epidemiology of obesity and hypothyroidism, thyroid autoimmunity, obesity phenotypes, metabolic syndrome, inflammatory mechanisms, adipokines, pediatric and adult populations, and treatment outcomes. Particular emphasis was placed on studies investigating the bidirectional relationship between obesity and thyroid dysfunction and the potential mechanisms underlying this association (Bambini et al., 2023; Biondi, 2024; Wang et al., 2021).

The available evidence was evaluated according to study design, methodological quality, sample size, and clinical relevance. Greater importance was assigned to systematic reviews, meta-analyses, and prospective cohort studies, whereas observational studies were primarily used to support specific clinical or mechanistic findings. When several studies reported similar results, preference was given to investigations with larger study populations and more robust methodology.

Because considerable heterogeneity existed among the included studies regarding patient populations, diagnostic criteria for obesity and hypothyroidism, study design, and outcome measures, a quantitative synthesis was not considered appropriate. Instead, the available evidence was analyzed qualitatively and organized into thematic sections addressing epidemiological evidence, pathophysiological mechanisms, thyroid autoimmunity, pediatric and adult populations, metabolic phenotypes, and clinical implications.

The objective of this review was to integrate the most relevant and clinically significant findings from the current literature while identifying areas of consensus, existing controversies, and directions for future research.

3. Results

3.1. Epidemiological Evidence

A growing body of evidence indicates that obesity is significantly associated with an increased risk of thyroid dysfunction, particularly subclinical hypothyroidism. Although hypothyroidism has traditionally been considered a contributing factor to weight gain, recent epidemiological studies suggest that obesity itself may alter thyroid function through multiple metabolic and endocrine pathways (Biondi, 2024; Wang et al., 2021).

Large observational studies consistently demonstrate that individuals with obesity have higher serum thyroid-stimulating hormone (TSH) concentrations than normal-weight individuals, while free thyroxine (FT4) concentrations generally remain within the reference range (Yan et al., 2022; Wang et al., 2021). This pattern is most frequently observed in patients with severe obesity and has been reported in both adult and pediatric populations. Importantly, elevated TSH concentrations are often accompanied by normal thyroid hormone levels, indicating that these changes do not necessarily reflect overt thyroid failure.

Several studies have shown that obesity is associated with an increased prevalence of subclinical hypothyroidism. In a longitudinal cohort study involving more than 9,000 euthyroid adults, metabolically healthy obesity, metabolically unhealthy obesity, and metabolically unhealthy normal-weight phenotypes were all associated with an increased risk of developing hypothyroidism in men compared with metabolically healthy normal-weight individuals (Wang et al., 2021). These findings suggest that both excessive adiposity and metabolic abnormalities contribute to thyroid dysfunction, although the strength of this association appears to differ according to sex and metabolic status.

The relationship between obesity and thyroid dysfunction is further supported by cross-sectional investigations evaluating thyroid autoimmunity. Yan et al. (2022) demonstrated that obesity was significantly associated with subclinical hypothyroidism among individuals with positive thyroid autoantibodies. Moreover, obese participants showed a higher prevalence of thyroglobulin antibody positivity than non-obese controls, suggesting that obesity may promote autoimmune mechanisms involved in thyroid disease. Similar observations have been summarized in recent narrative reviews, which concluded that obesity is associated with a higher prevalence of both thyroid autoimmunity and hypothyroidism, particularly in metabolically unhealthy individuals (Bambini et al., 2023; Biondi, 2024).

Evidence from pediatric populations demonstrates a somewhat different pattern. Mild TSH elevation is frequently observed in children and adolescents with obesity; however, these abnormalities are usually transient. In a retrospective cohort study, more than 90% of children with obesity-related subclinical hypothyroidism achieved spontaneous normalization of TSH concentrations during follow-up without thyroid hormone replacement, supporting the hypothesis that elevated TSH often represents an adaptive response to obesity rather than permanent thyroid dysfunction (Tersander et al., 2025). Similar findings have been reported in other pediatric studies, where obesity-related thyroid dysfunction was associated primarily with elevated triglyceride concentrations rather than impaired basal metabolic rate or persistent endocrine abnormalities (Akici et al., 2020).

Recent comprehensive reviews have emphasized that the association between obesity and hypothyroidism is unlikely to be explained by a single causal mechanism. Instead, excess adiposity, chronic low-grade inflammation, metabolic syndrome, and thyroid autoimmunity appear to interact and collectively influence thyroid physiology (Bambini et al., 2023; Biondi, 2024). Although epidemiological evidence consistently demonstrates a positive association between obesity and thyroid dysfunction, the magnitude of this relationship varies according to age, sex, metabolic phenotype, and the presence of autoimmune thyroid disease.

Overall, current epidemiological evidence supports the concept that obesity is an important risk factor for alterations in thyroid function. Nevertheless, available data also indicate that elevated TSH concentrations observed in many obese individuals should be interpreted cautiously, particularly in the absence of reduced circulating thyroid hormone levels or clinical symptoms of hypothyroidism, as these changes may reflect physiological adaptation rather than established thyroid disease.

3.2. Pathophysiological Mechanisms Linking Obesity and Hypothyroidism

The relationship between obesity and hypothyroidism is mediated by multiple endocrine, metabolic, inflammatory, and immunological pathways. Rather than representing independent disorders, both conditions appear to interact through complex physiological mechanisms involving adipose tissue, thyroid hormone metabolism, chronic low-grade inflammation, and immune regulation (Biondi, 2024; Bambini et al., 2023).

Adipose tissue is now recognized as an active endocrine organ rather than a passive reservoir for energy storage. In addition to adipocytes, adipose tissue contains macrophages, lymphocytes, fibroblasts, and vascular cells that collectively secrete numerous biologically active mediators, including leptin, adiponectin, resistin, tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), and other cytokines. These substances regulate appetite, insulin sensitivity, lipid metabolism, and immune responses while simultaneously influencing thyroid physiology (Wojciechowska et al., 2022; Biondi, 2024).

Among these mediators, leptin has emerged as one of the principal links between obesity and thyroid function. Circulating leptin concentrations increase proportionally with adipose tissue mass and stimulate hypothalamic proopiomelanocortin neurons involved in energy homeostasis. Leptin also promotes the expression of thyrotropin-releasing hormone (TRH) within the hypothalamus, leading to increased secretion of thyroid-stimulating hormone (TSH) by the anterior pituitary gland. Consequently, elevated serum TSH concentrations frequently observed in obesity may represent a physiological adaptation intended to enhance thermogenesis and energy expenditure rather than evidence of primary thyroid failure (Biondi, 2024; Bambini et al., 2023).

Chronic low-grade inflammation represents another important mechanism linking obesity with thyroid dysfunction. Excess adipose tissue promotes continuous production of pro-inflammatory cytokines, including TNF- α , IL-6, and C-reactive protein, resulting in persistent systemic inflammation. These inflammatory mediators may impair thyroid hormone synthesis, interfere with intracellular thyroid hormone signaling, and alter peripheral metabolism of thyroid hormones through changes in deiodinase enzyme activity (Wojciechowska et al., 2022; Bambini et al., 2023).

Recent evidence also suggests that obesity affects thyroid hormone metabolism at the tissue level. Altered expression of deiodinases within adipose tissue and skeletal muscle may modify local conversion of thyroxine (T4) to the biologically active triiodothyronine (T3), leading to tissue-specific alterations in thyroid hormone availability despite normal circulating hormone concentrations. Experimental and clinical studies indicate that these adaptive mechanisms may increase metabolic efficiency while simultaneously contributing to persistent weight gain and metabolic disturbances in some individuals (Dulloo, 2025; Biondi, 2024).

Insulin resistance further contributes to the interaction between obesity and thyroid dysfunction. Hyperinsulinemia and impaired insulin signaling are associated with elevated TSH concentrations and altered thyroid hormone metabolism. In pediatric populations, hypothyroidism accompanied by obesity has been associated with significantly higher fasting insulin concentrations, increased Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) values, and elevated leptin levels compared with lean hypothyroid children, suggesting that thyroid dysfunction may aggravate metabolic abnormalities already present in obesity (El Amrousy et al., 2022).

Obesity has also been associated with structural and functional alterations within the thyroid gland. Increased thyroid volume, changes in thyroid echogenicity, and reduced expression of thyroid-stimulating hormone receptors have been described in obese individuals. These changes may represent adaptive responses to chronic metabolic stress and inflammation rather than irreversible thyroid pathology (Biondi, 2024).

Collectively, current evidence indicates that obesity influences thyroid physiology through several complementary mechanisms rather than a single pathological pathway. Endocrine signaling mediated by adipokines, chronic systemic inflammation, altered peripheral thyroid hormone metabolism, insulin resistance, and tissue-specific adaptations appear to interact continuously, explaining why thyroid abnormalities observed in obesity often differ from those seen in classical primary hypothyroidism. This multifactorial pathophysiology also explains the considerable heterogeneity reported across clinical studies and highlights the importance of considering obesity-related thyroid dysfunction within the broader context of metabolic disease rather than as an isolated endocrine disorder.

3.3. Obesity and Thyroid Autoimmunity

The relationship between obesity and thyroid autoimmunity has attracted increasing scientific attention over the past decade. Although obesity has traditionally been considered a metabolic disorder, accumulating evidence suggests that excessive adipose tissue also contributes to immune dysregulation, thereby increasing susceptibility to autoimmune diseases, including autoimmune thyroid disorders (Bambini et al., 2023; Biondi, 2024).

Several epidemiological studies have demonstrated a higher prevalence of thyroid autoantibodies among individuals with obesity compared with normal-weight populations. In particular, elevated concentrations of thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb) have been reported more frequently in obese individuals, indicating an increased risk of Hashimoto's thyroiditis and subclinical hypothyroidism (Yan et al., 2022; Bambini et al., 2023). However, not all studies have reported consistent findings, suggesting that factors such as age, sex, iodine intake, genetic predisposition, and metabolic health may modify this association.

One proposed mechanism linking obesity with thyroid autoimmunity involves chronic low-grade inflammation. Adipose tissue in obesity is infiltrated by macrophages and other immune cells that continuously produce pro-inflammatory cytokines, including TNF- α , IL-6, and monocyte chemoattractant protein-1. Persistent activation of these inflammatory pathways may disrupt immune tolerance, promote activation of autoreactive lymphocytes, and increase the production of thyroid autoantibodies (Wojciechowska et al., 2022; Bambini et al., 2023).

Leptin appears to play a central role in this process. Beyond its well-established function in appetite regulation, leptin acts as an immunomodulatory hormone by stimulating T-cell proliferation, promoting T helper 1 (Th1) immune responses, and suppressing regulatory T-cell activity. These effects favor a pro-inflammatory immune environment that may facilitate the development and progression of autoimmune thyroid disease (Biondi, 2024). Elevated leptin concentrations observed in obesity therefore represent a potential biological link between excessive adiposity and thyroid autoimmunity.

Clinical studies further support this hypothesis. In a cross-sectional study involving more than 2,500 participants, obesity was independently associated with subclinical hypothyroidism among individuals with positive thyroid autoantibodies. Furthermore, obese participants demonstrated significantly higher rates of

thyroglobulin antibody positivity than non-obese individuals, suggesting that obesity may contribute not only to thyroid dysfunction but also to autoimmune activation (Yan et al., 2022).

The influence of obesity on thyroid autoimmunity appears to differ between children and adults. In pediatric populations, isolated TSH elevation is relatively common, whereas autoimmune thyroiditis remains comparatively uncommon and is observed mainly in adolescents. Conversely, adults with obesity demonstrate substantially higher frequencies of autoimmune thyroid disease, particularly overt hypothyroidism caused by Hashimoto's thyroiditis (Bambini et al., 2023). These observations suggest that prolonged exposure to obesity-related inflammation may increase the likelihood of autoimmune thyroid damage over time.

Despite these findings, obesity should not be regarded as an independent cause of autoimmune thyroid disease. Current evidence suggests that obesity functions primarily as a modifying factor that may accelerate autoimmune processes in genetically susceptible individuals rather than initiating autoimmunity directly (Biondi, 2024). The coexistence of obesity, chronic inflammation, and thyroid autoantibodies therefore represents a multifactorial interaction rather than a simple cause-and-effect relationship.

Overall, the available evidence indicates that obesity contributes to an inflammatory and immunological environment that favors thyroid autoimmunity. Nevertheless, additional prospective studies are required to determine whether reducing excess body weight and improving metabolic health can effectively decrease the incidence or progression of autoimmune thyroid diseases.

3.4. Obesity-Associated Thyroid Dysfunction in Pediatric and Adult Populations

The relationship between obesity and thyroid dysfunction differs considerably between children and adults, suggesting that age influences both the pathophysiology and clinical significance of obesity-related alterations in thyroid function. Although elevated serum TSH concentrations are frequently reported in both populations, their persistence, underlying mechanisms, and clinical consequences appear to vary substantially (Bambini et al., 2023; Biondi, 2024).

In pediatric populations, subclinical hypothyroidism is one of the most frequently reported endocrine abnormalities associated with obesity. Numerous studies have demonstrated that children and adolescents with obesity exhibit higher serum TSH concentrations than their normal-weight peers, whereas free thyroid hormone concentrations usually remain within the normal reference range (Akici et al., 2020; Tersander et al., 2025). This hormonal profile is generally considered to represent obesity-related hyperthyrotropinemia rather than primary thyroid disease.

Importantly, longitudinal evidence suggests that these biochemical abnormalities are largely reversible. Tersander et al. (2025) reported that more than 90% of children diagnosed with obesity-related subclinical hypothyroidism achieved spontaneous normalization of serum TSH concentrations within approximately three years without receiving levothyroxine therapy. Similar observations have been described in several pediatric studies demonstrating significant improvement in thyroid function following weight reduction through dietary modification and increased physical activity (Bambini et al., 2023). These findings support the hypothesis that elevated TSH in childhood obesity is predominantly an adaptive physiological response rather than evidence of permanent thyroid dysfunction.

Although thyroid hormone concentrations are generally preserved in obese children, several metabolic abnormalities appear to accompany obesity-related TSH elevation. Pediatric patients with obesity and subclinical hypothyroidism have been shown to exhibit higher serum triglyceride concentrations and less favorable lipid profiles than euthyroid obese children, potentially increasing their long-term cardiovascular risk (Akici et al., 2020; Tersander et al., 2025). Furthermore, obesity combined with hypothyroidism has been associated with increased insulin resistance, elevated fasting insulin concentrations, and higher circulating leptin levels, indicating that thyroid dysfunction may aggravate metabolic disturbances already present in childhood obesity (El Amrousy et al., 2022).

In contrast, adult populations demonstrate a more persistent association between obesity and thyroid dysfunction. Obesity in adults has been linked not only to elevated TSH concentrations but also to an increased prevalence of overt hypothyroidism, autoimmune thyroid disease, and structural thyroid abnormalities (Bambini et al., 2023; Biondi, 2024). Unlike pediatric patients, spontaneous normalization of thyroid function appears less common in adults, particularly among individuals with longstanding obesity or positive thyroid autoantibodies.

Large epidemiological studies further suggest that metabolic health substantially modifies the relationship between obesity and thyroid dysfunction in adulthood. Adults presenting with metabolically unhealthy obesity are more likely to develop hypothyroidism than metabolically healthy obese individuals,

emphasizing the importance of insulin resistance, dyslipidemia, and chronic inflammation as determinants of thyroid function (Wang et al., 2021). Additionally, obesity-related autoimmune thyroid disease appears considerably more prevalent in adults than in children, supporting the hypothesis that prolonged exposure to systemic inflammation may contribute to progressive thyroid damage over time (Yan et al., 2022; Bambini et al., 2023).

These age-related differences have important clinical implications. In children, isolated mild elevation of TSH without clinical symptoms or thyroid autoantibodies often requires careful observation rather than immediate pharmacological treatment, particularly because thyroid function frequently normalizes following weight reduction. Conversely, persistent TSH elevation in adults, especially when accompanied by positive thyroid autoantibodies or reduced free thyroid hormone concentrations, warrants a more comprehensive endocrine evaluation to exclude underlying autoimmune hypothyroidism (Biondi, 2024).

Overall, current evidence indicates that obesity-associated thyroid dysfunction should not be interpreted uniformly across all age groups. Pediatric obesity is predominantly characterized by reversible adaptive changes in thyroid function, whereas adult obesity is more frequently associated with persistent thyroid abnormalities and autoimmune disease. Recognizing these differences is essential for appropriate clinical decision-making and may help prevent both overdiagnosis and unnecessary treatment of obesity-related thyroid dysfunction.

Characteristic	Children and adolescents	Adults
Predominant thyroid abnormality	Subclinical hypothyroidism with isolated TSH elevation	Subclinical and overt hypothyroidism
FT4 concentration	Usually normal	Normal or decreased depending on disease severity
Reversibility	Frequently reversible after weight loss	Less frequently reversible
Thyroid autoimmunity	Relatively uncommon	More common, particularly Hashimoto's thyroiditis
Main metabolic abnormalities	Elevated triglycerides, insulin resistance, hyperleptinemia	Metabolic syndrome, dyslipidemia, insulin resistance
Levothyroxine therapy	Usually not indicated in isolated obesity-related TSH elevation	Reserved for confirmed hypothyroidism according to endocrine guidelines
Response to weight reduction	TSH frequently normalizes	Improvement in thyroid function is common but less predictable
Clinical recommendation	Observation and lifestyle intervention	Individualized endocrine assessment and treatment

3.5. Obesity Phenotypes, Metabolic Syndrome, and Thyroid Dysfunction

Recent evidence indicates that the association between obesity and hypothyroidism is influenced not only by excess body weight but also by an individual's metabolic status. The recognition of different obesity phenotypes has shifted attention from body mass index (BMI) alone toward the metabolic characteristics accompanying obesity. Consequently, metabolically unhealthy obesity has emerged as a stronger predictor of thyroid dysfunction than obesity itself (Wang et al., 2021; Biondi, 2024).

Obesity is a heterogeneous condition, and not all individuals with elevated BMI exhibit the same metabolic risk profile. Some obese individuals maintain normal insulin sensitivity, lipid metabolism, and blood pressure and are therefore classified as having metabolically healthy obesity (MHO). In contrast, metabolically unhealthy obesity (MUO) is characterized by insulin resistance, dyslipidemia, hypertension, and chronic low-grade inflammation. Similarly, some individuals with normal BMI may present with metabolic abnormalities and are classified as metabolically unhealthy normal weight (MUNO). These phenotypes appear to differ substantially in their association with thyroid function (Wang et al., 2021).

In one of the largest longitudinal cohort studies addressing this issue, Wang et al. (2021) followed more than 9,000 euthyroid adults and demonstrated that both metabolically unhealthy obesity and metabolically unhealthy normal-weight phenotypes were independently associated with an increased risk of developing hypothyroidism. Interestingly, even metabolically healthy obesity was associated with a higher incidence of hypothyroidism in men compared with metabolically healthy normal-weight individuals, suggesting that excess adiposity alone may influence thyroid function. However, the strongest associations were consistently

observed among metabolically unhealthy participants, emphasizing the contribution of metabolic disturbances beyond BMI alone.

These findings are supported by recent narrative reviews, which conclude that chronic metabolic dysfunction rather than obesity per se appears to be the principal determinant of thyroid abnormalities (Biondi, 2024). Insulin resistance, altered glucose metabolism, dyslipidemia, and systemic inflammation have all been proposed as mechanisms contributing to elevated TSH concentrations and impaired thyroid hormone homeostasis. Consequently, obesity should be regarded as one component of a broader metabolic disorder rather than an isolated risk factor for thyroid dysfunction.

Metabolic syndrome further strengthens the relationship between obesity and hypothyroidism. Several studies have demonstrated that individuals with metabolic syndrome exhibit higher serum TSH concentrations and an increased prevalence of subclinical hypothyroidism compared with metabolically healthy individuals (Biondi, 2024). The coexistence of abdominal obesity, hypertension, impaired glucose metabolism, and dyslipidemia may amplify chronic inflammatory activity and endocrine dysregulation, thereby increasing susceptibility to thyroid dysfunction.

Experimental and clinical evidence also suggests that adipokines contribute to the interaction between metabolic syndrome and thyroid function. Increased leptin concentrations, together with reduced adiponectin levels, are characteristic features of obesity-associated metabolic dysfunction and have been linked to insulin resistance and altered thyroid hormone regulation (El Amrousy et al., 2022). These endocrine abnormalities may partially explain why metabolically unhealthy individuals experience more pronounced thyroid dysfunction than metabolically healthy obese individuals.

The relationship between obesity phenotypes and thyroid disease also appears to differ according to sex. Wang et al. (2021) observed that obesity phenotypes were significantly associated with incident hypothyroidism in men, whereas similar associations were not demonstrated in women after adjustment for confounding variables. Although the biological basis for these differences remains unclear, hormonal factors, body fat distribution, and sex-specific immune responses have all been proposed as potential explanations.

Overall, current evidence suggests that metabolic health is a critical determinant of thyroid function in individuals with obesity. Assessment of insulin resistance, lipid profile, and other components of metabolic syndrome may therefore provide more clinically relevant information than BMI alone when evaluating the risk of thyroid dysfunction. These findings support an individualized approach in which metabolic phenotype is considered alongside anthropometric measurements during endocrine assessment of patients with obesity.

3.6. Clinical Implications and Therapeutic Considerations

The close relationship between obesity and thyroid dysfunction presents important challenges in clinical practice, particularly regarding diagnosis and treatment. Mild elevations in serum TSH concentrations are frequently detected in individuals with obesity, often leading to further endocrine investigations and, in some cases, initiation of levothyroxine therapy. However, current evidence suggests that these biochemical abnormalities should be interpreted cautiously, as they do not necessarily indicate permanent thyroid dysfunction (Biondi, 2024).

Several studies have demonstrated that obesity-related elevations in TSH frequently normalize following weight reduction. Lifestyle interventions, including caloric restriction, increased physical activity, and bariatric surgery, have consistently been associated with decreases in serum TSH concentrations while maintaining normal circulating thyroid hormone levels (Bambini et al., 2023; Biondi, 2024). These observations support the concept that thyroid alterations observed in obesity are often secondary to excess adiposity and metabolic dysfunction rather than primary thyroid disease.

This phenomenon is particularly evident in pediatric populations. Longitudinal follow-up studies have shown that the majority of children with obesity-related subclinical hypothyroidism experience spontaneous normalization of thyroid function without pharmacological treatment. Consequently, routine administration of levothyroxine is generally not recommended in children presenting with isolated TSH elevation unless additional evidence of thyroid disease, such as positive thyroid autoantibodies or reduced free thyroid hormone concentrations, is present (Tersander et al., 2025; Bambini et al., 2023).

The effectiveness of levothyroxine therapy in adults with obesity has also been questioned. Although thyroid hormone replacement remains the standard treatment for overt hypothyroidism, its influence on body weight appears limited. In a recent clinical study comparing women with obesity receiving levothyroxine replacement for hypothyroidism with euthyroid women undergoing the same weight reduction program, both groups achieved comparable reductions in body weight, body mass index, and cardiometabolic risk parameters.

These findings indicate that adequate levothyroxine replacement restores normal thyroid function but does not provide additional benefits for weight loss beyond those achieved through lifestyle modification (Gokkaya & Aydin, 2024).

Similarly, a recent review concluded that isolated hyperthyrotropinemia in euthyroid obese individuals should not routinely be treated with thyroid hormones, as pharmacological correction has not been shown to improve weight reduction or metabolic outcomes. Instead, serum TSH concentrations frequently decrease following successful weight loss, supporting the hypothesis that obesity itself contributes to reversible thyroid dysfunction (Biondi, 2024).

Current clinical guidelines are consistent with these observations. A large European survey involving thyroid specialists demonstrated broad agreement that thyroid hormones should not be prescribed for weight reduction in euthyroid individuals with obesity. Despite this consensus, a small proportion of clinicians continue to use thyroid hormone preparations in patients resistant to lifestyle interventions, despite the lack of supporting evidence and the potential risks associated with iatrogenic thyrotoxicosis (Galofré et al., 2025).

The relationship between obesity and persistent hypothyroid symptoms also deserves attention. Even among patients receiving appropriate levothyroxine replacement and achieving biochemical euthyroidism, some continue to report fatigue, reduced quality of life, and difficulty losing weight. Recent evidence suggests that these persistent symptoms are more strongly associated with obesity, depression, and psychological factors than with residual thyroid hormone abnormalities. Consequently, management should extend beyond optimization of thyroid hormone replacement and include treatment of obesity, psychological support, and modification of cardiovascular risk factors (Bakos et al., 2025).

Taken together, the available evidence indicates that treatment strategies should primarily target obesity and its metabolic complications rather than mild thyroid abnormalities alone. Lifestyle modification remains the cornerstone of therapy, while thyroid hormone replacement should be reserved for patients with confirmed overt hypothyroidism or carefully selected individuals with persistent subclinical hypothyroidism according to current endocrine recommendations. This individualized approach may reduce unnecessary treatment while improving both metabolic health and long-term clinical outcomes.

4. Discussion

The present review demonstrates that the relationship between obesity and hypothyroidism is considerably more complex than previously believed. Although hypothyroidism has long been regarded as an important cause of weight gain, the current evidence suggests that obesity itself substantially influences thyroid function through multiple endocrine, metabolic, inflammatory, and immunological mechanisms. Overall, the available literature supports a bidirectional association; however, the evidence more strongly indicates that obesity contributes to thyroid dysfunction than vice versa (Biondi, 2024; Wang et al., 2021).

One of the most consistent findings across the reviewed studies is the increased prevalence of subclinical hypothyroidism among individuals with obesity. Elevated serum TSH concentrations have been repeatedly observed in both adults and children, whereas circulating FT4 concentrations generally remain within the normal reference range (Yan et al., 2022; Tersander et al., 2025). This biochemical profile differs from classical primary hypothyroidism and suggests that obesity-associated thyroid dysfunction frequently represents an adaptive physiological response rather than irreversible thyroid failure. The observation that serum TSH concentrations often normalize following successful weight reduction strongly supports this interpretation (Bambini et al., 2023).

The reviewed studies also emphasize the central role of adipose tissue in regulating thyroid physiology. Adipose tissue functions as an active endocrine organ that secretes numerous adipokines and inflammatory mediators capable of influencing the hypothalamic–pituitary–thyroid axis. Elevated leptin concentrations appear to stimulate TSH secretion, while chronic low-grade inflammation may impair thyroid hormone metabolism and promote autoimmune thyroid disease (Wojciechowska et al., 2022; Biondi, 2024). These mechanisms provide a plausible biological explanation for the frequent coexistence of obesity and thyroid dysfunction observed in epidemiological studies.

Another important observation emerging from the current evidence is that metabolic health appears to be more strongly associated with thyroid dysfunction than body weight alone. Individuals with metabolically unhealthy obesity consistently demonstrate a greater risk of developing hypothyroidism than metabolically healthy obese individuals (Wang et al., 2021). This finding suggests that insulin resistance, dyslipidemia, and chronic inflammation are major determinants of thyroid abnormalities. Consequently, body mass index alone

may not adequately reflect endocrine risk, and comprehensive metabolic assessment should be incorporated into the clinical evaluation of patients with obesity.

The association between obesity and thyroid autoimmunity remains less clearly established. Although several studies demonstrated increased prevalence of thyroid autoantibodies among obese individuals, others reported weaker or non-significant associations after adjustment for confounding variables (Yan et al., 2022; Bambini et al., 2023). These inconsistencies likely reflect differences in study populations, iodine intake, ethnicity, genetic susceptibility, and diagnostic criteria. Therefore, while obesity appears to create an inflammatory environment favorable for autoimmune thyroid disease, current evidence does not support obesity as an independent cause of thyroid autoimmunity.

The reviewed literature also has important therapeutic implications. Both pediatric and adult studies consistently indicate that treatment should primarily focus on reducing excess body weight and improving metabolic health rather than correcting isolated biochemical abnormalities. Lifestyle interventions remain the first-line approach because improvements in body weight are frequently accompanied by normalization of thyroid function (Bambini et al., 2023; Tersander et al., 2025). Likewise, levothyroxine therapy should be reserved for patients with confirmed hypothyroidism according to established endocrine guidelines rather than prescribed solely to facilitate weight loss (Gokkaya & Aydin, 2024; Galofré et al., 2025).

Despite the consistency of many findings, several limitations should be acknowledged. Most available studies were observational, preventing definitive conclusions regarding causality. Considerable heterogeneity also existed among the reviewed studies regarding population characteristics, definitions of obesity, diagnostic thresholds for subclinical hypothyroidism, follow-up duration, and laboratory reference ranges. These methodological differences complicate direct comparison of study outcomes and may partially explain the discrepancies observed across individual investigations.

Nevertheless, recent longitudinal cohort studies and Mendelian randomization analyses provide stronger evidence supporting a causal effect of obesity on thyroid dysfunction than earlier cross-sectional investigations. These studies reduce the likelihood of reverse causation and strengthen the hypothesis that obesity represents an important determinant of altered thyroid function rather than merely a consequence of hypothyroidism. Future prospective investigations integrating molecular biomarkers, genetic susceptibility, and long-term clinical outcomes will be essential to clarify the mechanisms underlying this association and to optimize management strategies for patients with coexisting obesity and thyroid dysfunction.

5. Conclusions

Current evidence demonstrates a significant association between obesity and hypothyroidism, although the interaction between these conditions is multifactorial and cannot be explained by a single pathogenic mechanism. The available literature indicates that obesity contributes to alterations in thyroid function through endocrine, metabolic, inflammatory, and immunological pathways, while hypothyroidism appears to have a relatively modest influence on the development of obesity. Consequently, the relationship is best understood as bidirectional, with obesity acting as the predominant driving factor.

One of the most important findings of this review is that mild elevations in serum TSH frequently observed in individuals with obesity, particularly in children and adolescents, often represent adaptive physiological responses rather than permanent thyroid dysfunction. These abnormalities commonly improve following weight reduction, emphasizing the importance of addressing obesity before considering pharmacological treatment. In contrast, persistent thyroid dysfunction accompanied by reduced thyroid hormone concentrations or positive thyroid autoantibodies requires careful endocrine evaluation to exclude primary hypothyroidism.

The reviewed studies consistently identify adipose tissue as a key regulator of thyroid physiology. Adipokines, especially leptin, together with chronic low-grade inflammation, altered thyroid hormone metabolism, and insulin resistance, appear to play central roles in the obesity–thyroid axis. Furthermore, metabolic health was shown to influence thyroid function independently of body mass index, suggesting that evaluation of metabolic syndrome and cardiometabolic risk factors should be incorporated into the clinical assessment of patients with obesity.

From a therapeutic perspective, current evidence does not support the routine use of thyroid hormone therapy for isolated obesity-related hyperthyrotropinemia or for weight reduction in euthyroid individuals. Instead, lifestyle modification, sustained weight loss, and improvement of metabolic health remain the most effective interventions for restoring normal thyroid function and reducing long-term cardiometabolic risk.

Although substantial progress has been made in understanding the association between obesity and hypothyroidism, several important questions remain unresolved. Future prospective cohort studies and randomized clinical trials are needed to clarify the long-term effects of weight reduction on thyroid function, determine the mechanisms responsible for obesity-related thyroid alterations, and identify patients who may benefit from individualized therapeutic approaches. A better understanding of the molecular interactions between adipose tissue, immune regulation, and thyroid physiology may contribute to improved prevention and management strategies for both disorders.

REFERENCES

1. Akici, N., Onal, Z. E., Gürbüz, T., Sağ, C., & Kiliç, S. (2020). Atherogenic indices in the assessment of cardiovascular disease risk in children with obesity and subclinical hypothyroidism. *Acta Endocrinologica (Bucharest)*, 16(3), 334–338.
2. Bakos, B., Solymosi, T., Szili, B., Vincze, Á., Mészáros, Sz., Stempler, M., et al. (2025). Psychological factors and obesity, not thyroid biomarkers, predict thyroid-dependent quality of life in treated hypothyroidism: A cross-sectional study. *BMC Endocrine Disorders*, 25, 136.
3. Bambini, F., Gatta, E., D'Alessio, R., Dondi, F., Pignata, G., Pirola, I., et al. (2023). Thyroid disease and autoimmunity in obese patients: A narrative review. *Endokrynologia Polska*, 74(6), 576–590.
4. Biondi, B. (2024). Subclinical hypothyroidism in patients with obesity and metabolic syndrome: A narrative review. *Nutrients*, 16(1), 87.
5. Dulloo, A. G. (2025). Adaptive thermogenesis driving catch-up fat during weight regain: A role for skeletal muscle hypothyroidism and a risk for sarcopenic obesity. *Reviews in Endocrine and Metabolic Disorders*, 26, 871–888.
6. El Amrousy, D., El-Afify, D., & Salah, S. (2022). Insulin resistance, leptin and adiponectin in lean and hypothyroid children and adolescents with obesity. *BMC Pediatrics*, 22, 245.
7. Galofré, J. C., Díez, J. J., Attanasio, R., Nagy, E. V., Negro, R., Papini, E., et al. (2025). Treatment of obesity with thyroid hormones in Europe: Data from the THESIS Collaboration. *Journal of Endocrinological Investigation*, 48, 201–212.
8. Gokkaya, N., & Aydin, K. (2024). Efficacy of levothyroxine monotherapy in achieving clinical euthyroidism and its impact on weight loss in women with hypothyroidism and obesity. *Scientific Reports*, 14, Article 27822.
9. Tersander, B., Olsson, R., Aydin, B. K., Stenlid, R., Ciba, I., & Manell, H. (2025). Obesity-related subclinical hypothyroidism in childhood: Elevated triglycerides but not basal metabolic rate. *Pediatric Research*, 98, 182–187.
10. Wang, Y., Lin, H., Li, Q., Guan, L., Zhao, M., Zhong, F., et al. (2021). Association between different obesity phenotypes and hypothyroidism: A study based on a longitudinal health management cohort. *Endocrine*, 72, 688–698.
11. Wojciechowska, M., Wiśniewski, O. W., Pruszyńska-Oszmałek, E., Krauss, H., Sassek, M., Leciejewska, N., et al. (2022). Effect of obesity and hypothyroidism on hepcidin concentration in pregnancy—A pilot study using maternal and umbilical cord blood at delivery day. *Journal of Physiology and Pharmacology*, 73(5), 605–612.
12. Yan, Y., Xu, M., Wu, M., Wang, X., Li, F., Zhang, J., et al. (2022). Obesity is associated with subclinical hypothyroidism in the presence of thyroid autoantibodies: A cross-sectional study. *BMC Endocrine Disorders*, 22, 94.