# The Relationship Between Autoimmune Thyroiditis and Metabolic Syndrome

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**ABSTRACT**

Hashimoto's thyroiditis is the most common autoimmune thyroid disorder and represents a leading cause of hypothyroidism worldwide. In parallel, metabolic syndrome has emerged as a major global health problem, characterized by a cluster of metabolic abnormalities that substantially increase cardiovascular and metabolic risk. Growing evidence indicates that these two conditions are closely interconnected through shared pathophysiological mechanisms that extend beyond thyroid hormone deficiency alone. This review summarizes current epidemiological, clinical, and mechanistic data on the association between Hashimoto's thyroiditis and metabolic syndrome. Available studies demonstrate a higher prevalence of metabolic syndrome and its components among patients with Hashimoto's thyroiditis, including those in the euthyroid state, while thyroid autoimmunity is also more frequently observed in individuals with metabolic syndrome. Chronic low-grade inflammation, immune–metabolic crosstalk, adipokine imbalance, oxidative stress, and endothelial dysfunction appear to play central roles in linking autoimmune thyroid disease with metabolic dysregulation. Thyroid hormone replacement therapy improves certain metabolic parameters in hypothyroid patients but does not fully eliminate cardiometabolic risk, highlighting the importance of integrated management strategies. Understanding the bidirectional relationship between Hashimoto's thyroiditis and metabolic syndrome has important implications for early detection, risk stratification, and comprehensive preventive and therapeutic approaches aimed at reducing long-term cardiovascular morbidity.

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## Introduction

Hashimoto's thyroiditis (HT), also referred to as chronic autoimmune thyroiditis, is the most common cause of hypothyroidism in iodine-sufficient regions worldwide. It is characterized by immune-mediated destruction of thyroid tissue, leading to progressive impairment of thyroid hormone synthesis and secretion. The prevalence of HT varies by population, sex, age, and iodine intake, but epidemiological studies estimate that thyroid autoantibodies can be detected in up to 10–15% of the general population, with clinically manifest disease occurring in approximately 2–5%, predominantly among women [1, 4]. The incidence of HT has shown a steady increase over recent decades, partly due to improved diagnostic sensitivity and greater awareness, but also potentially related to environmental and lifestyle changes [6].

Metabolic syndrome (MetS) is a complex cluster of interrelated metabolic abnormalities, including central obesity, insulin resistance, dyslipidemia, hypertension, and impaired glucose metabolism. It represents a major global public health challenge, affecting approximately 20–25% of the adult population worldwide, with prevalence rising sharply in both developed and developing countries [7, 9]. MetS significantly increases the risk of type 2 diabetes mellitus, cardiovascular disease, and all-cause mortality, thereby imposing substantial economic and healthcare burdens [10, 12].

The growing recognition of interactions between endocrine, immune, and metabolic systems has prompted increasing interest in the potential association between HT and MetS. Thyroid hormones play a central role in regulating basal metabolic rate, lipid and glucose metabolism, and energy homeostasis, while chronic autoimmune inflammation may contribute to metabolic dysregulation through shared inflammatory and immunometabolic pathways [3, 8]. Conversely, metabolic disturbances such as obesity and insulin resistance may influence immune tolerance and promote autoimmune activity, suggesting a bidirectional relationship between HT and MetS [11, 14].

Understanding the link between HT and MetS is of considerable clinical and public health relevance. Patients with HT may be at increased risk of developing metabolic abnormalities even in the euthyroid state, potentially

accelerating cardiovascular risk beyond that attributable to thyroid dysfunction alone [5, 15]. From a public health perspective, early identification of metabolic risk in individuals with autoimmune thyroid disease may enable timely preventive interventions, thereby reducing long-term morbidity and healthcare costs [16].

The objective of this review is to synthesize current evidence on the epidemiological, pathophysiological, and clinical relationships between Hashimoto's thyroiditis and metabolic syndrome. The scope includes analysis of underlying immune and metabolic mechanisms, clinical manifestations across different thyroid functional states, and implications for screening and management strategies, with an emphasis on recent findings from observational and mechanistic studies [2, 17].

## Autoimmune Thyroiditis (Hashimoto's Thyroiditis)

Hashimoto's thyroiditis is primarily driven by a breakdown of immune tolerance to thyroid-specific antigens, resulting in chronic lymphocytic infiltration and gradual destruction of thyroid follicles. The immunopathogenesis of HT is largely mediated by autoreactive $CD4^+$ and $CD8^+$ T lymphocytes, which induce cytotoxic damage to thyrocytes through direct cell-mediated mechanisms and the release of proinflammatory cytokines [6, 13]. This T-cell–mediated cytotoxicity is considered central to disease initiation and progression.

Humoral immune responses also play a critical role in HT, particularly through the production of anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Anti-TPO antibodies are detected in the majority of patients and are strongly associated with disease activity and progression, while anti-Tg antibodies serve as additional markers of thyroid autoimmunity [4, 18]. Although these antibodies are not solely responsible for thyroid destruction, they reflect ongoing autoimmune activity and may contribute to antibody-dependent cell-mediated cytotoxicity.

Genetic susceptibility significantly influences the risk of developing HT. Polymorphisms in genes involved in immune regulation, such as HLA class II alleles, CTLA-4, and PTPN22, have been associated with increased disease risk [1, 19]. Environmental factors, including excessive iodine intake, viral infections, stress, smoking status, and exposure to endocrine-disrupting chemicals, are believed to act as triggers in genetically predisposed individuals, promoting immune dysregulation and autoantibody production [8, 20].

### *Clinical Spectrum of Hashimoto's Thyroiditis*

The clinical presentation of Hashimoto's thyroiditis is heterogeneous and spans a broad spectrum of thyroid functional states. Many individuals initially remain euthyroid despite the presence of thyroid autoantibodies and histological inflammation. Over time, progressive follicular destruction may lead to subclinical hypothyroidism, characterized by elevated thyroid-stimulating hormone (TSH) levels with normal circulating thyroid hormone concentrations, and eventually to overt hypothyroidism with clear biochemical and clinical manifestations [3, 12].

HT shows a marked female predominance, with women affected approximately 5–10 times more frequently than men. The disease can occur at any age but is most commonly diagnosed in middle-aged adults, although increasing detection has been reported in pediatric and adolescent populations [7, 15]. Hormonal factors, particularly estrogen-related immune modulation, are thought to contribute to the observed sex differences in prevalence and disease expression [14].

The natural history of Hashimoto's thyroiditis is typically characterized by slow progression over years or decades. While some patients remain stable in the euthyroid or subclinical state, others experience gradual deterioration of thyroid function, influenced by baseline antibody titers, TSH levels, iodine exposure, and coexisting autoimmune or metabolic conditions [5, 21]. This variable course underscores the importance of long-term monitoring and individualized risk assessment in patients with HT.

## Metabolic Syndrome

Metabolic syndrome (MetS) is defined as a constellation of interrelated metabolic risk factors that collectively increase the likelihood of developing cardiovascular disease and type 2 diabetes mellitus. Several international organizations have proposed diagnostic criteria for MetS, most notably the World Health Organization (WHO), the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), and the International Diabetes Federation (IDF). While these definitions differ slightly in their emphasis and threshold values, they all converge on the presence of central obesity, insulin resistance, dyslipidemia, hypertension, and impaired glucose metabolism as core components of the syndrome [2, 7].

According to the WHO definition, insulin resistance is considered the central pathogenic feature, whereas the NCEP ATP III criteria adopt a more pragmatic clinical approach, diagnosing MetS when at least three of five metabolic abnormalities are present. The IDF criteria place particular emphasis on central obesity as a mandatory component, reflecting its pivotal role in metabolic dysregulation [9, 11]. Despite these variations, all definitions underscore the multifactorial nature of MetS and its strong association with adverse cardiovascular and metabolic outcomes [13].

The pathophysiology of metabolic syndrome is complex and involves a network of interdependent metabolic, inflammatory, and vascular mechanisms. Insulin resistance is widely regarded as a central feature, leading to compensatory hyperinsulinemia and subsequent

disturbances in glucose and lipid metabolism. This metabolic imbalance is closely linked to chronic low-grade inflammation, characterized by elevated circulating levels of proinflammatory mediators such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) [4, 15].

Visceral adiposity plays a critical role in the development of MetS through the dysregulated secretion of adipokines. Excess adipose tissue, particularly in the abdominal region, acts as an active endocrine organ, releasing bioactive molecules that promote insulin resistance, inflammation, and oxidative stress. Alterations in adipokines such as leptin, adiponectin, and resistin contribute to metabolic imbalance and immune activation [8, 16].

Endothelial dysfunction and oxidative stress further exacerbate the metabolic and vascular consequences of MetS. Increased production of reactive oxygen species impairs nitric oxide bioavailability, promotes vascular inflammation, and accelerates atherosclerotic processes. These mechanisms collectively link metabolic disturbances to cardiovascular morbidity and mortality [10, 18].

## Epidemiological Evidence Linking Hashimoto's Thyroiditis and Metabolic Syndrome

A growing body of epidemiological evidence suggests that metabolic syndrome is more prevalent among individuals with Hashimoto's thyroiditis compared to the general population. Cross-sectional studies have consistently reported higher rates of central obesity, dyslipidemia, and insulin resistance in patients with HT, even after adjustment for age and sex [5, 14]. Cohort studies further indicate that the risk of developing MetS increases with the duration of thyroid autoimmunity and the degree of thyroid dysfunction [17].

Thyroid functional status appears to significantly influence this association. Patients with overt hypothyroidism exhibit the highest prevalence of MetS, largely due to pronounced alterations in lipid metabolism and insulin sensitivity. However, several studies have demonstrated that even euthyroid individuals with positive thyroid autoantibodies have a higher metabolic risk profile than antibody-negative controls, suggesting that autoimmunity itself may contribute to metabolic disturbances independently of thyroid hormone levels [6, 12].

Conversely, increased prevalence of autoimmune thyroiditis has been observed in populations diagnosed with metabolic syndrome. Studies assessing thyroid autoantibodies in MetS patients have reported higher frequencies of anti-TPO and anti-Tg positivity compared with metabolically healthy individuals [3, 9]. This finding supports the concept of shared pathogenic pathways linking metabolic and autoimmune disorders.

Gender- and age-specific patterns have also been noted, with women and older individuals with MetS showing a particularly high prevalence of thyroid autoimmunity. Hormonal factors, adiposity-related immune modulation, and age-related immune dysregulation are proposed contributors to this observed distribution [11, 19].

*Pathophysiological Mechanisms Connecting Hashimoto's Thyroiditis and Metabolic Syndrome*

Thyroid hormones are key regulators of energy expenditure, lipid turnover, and glucose homeostasis. Triiodothyronine (T3) enhances basal metabolic rate, stimulates lipolysis, and improves insulin-mediated glucose uptake in peripheral tissues. In hypothyroid states, reduced thyroid hormone availability leads to decreased energy expenditure, accumulation of adipose tissue, and adverse lipid profiles characterized by elevated low-density lipoprotein cholesterol and triglycerides [1, 20].

Hypothyroidism is also associated with impaired insulin sensitivity through alterations in hepatic glucose production, reduced glucose disposal in skeletal muscle, and changes in adipokine secretion. These metabolic effects provide a mechanistic link between thyroid dysfunction and the development of insulin resistance and MetS [7, 15].

*Chronic Inflammation as a Shared Mechanism*

Chronic low-grade inflammation represents a central biological link between Hashimoto's thyroiditis and metabolic syndrome. In HT, persistent immune activation leads to increased production of proinflammatory cytokines, including IL-6 and TNF-α, which are also key mediators of insulin resistance and metabolic dysfunction [4, 18]. Elevated CRP levels observed in both conditions further support the presence of a shared inflammatory milieu.

Immune–metabolic crosstalk, whereby inflammatory signals interfere with insulin signaling pathways and metabolic regulation, provides a unifying framework for understanding the coexistence of HT and MetS. This interaction suggests that chronic inflammation may act as both a cause and a consequence of metabolic and autoimmune dysregulation [10, 21].

Adipose tissue plays an active role in immune regulation and may serve as a critical interface between autoimmunity and metabolism. In HT, altered adipokine profiles, particularly elevated leptin and reduced adiponectin levels, have been associated with enhanced immune activation and reduced insulin sensitivity [8, 14]. Leptin promotes Th1 immune responses and may exacerbate autoimmune processes, while adiponectin exerts anti-inflammatory and insulin-sensitizing effects.

Visceral fat depots are infiltrated by immune cells, including macrophages and T lymphocytes, which secrete cytokines that further amplify inflammation and insulin resistance. This immune activation within adipose tissue may contribute to the mutual reinforcement of thyroid autoimmunity and metabolic dysfunction [16, 19].

Oxidative stress represents another common pathway linking HT and MetS. Increased generation of reactive oxygen species has been documented in both conditions, driven by chronic inflammation, mitochondrial dysfunction, and metabolic overload. In thyroid autoimmunity, oxidative stress may enhance antigen presentation and perpetuate immune-mediated tissue damage, while in MetS it contributes to insulin resistance and vascular injury [6, 13].

Endothelial dysfunction resulting from oxidative stress impairs vascular homeostasis and accelerates atherogenesis. The coexistence of HT and MetS may therefore synergistically increase cardiovascular risk through combined inflammatory, oxidative, and endothelial mechanisms [5, 17].

## Clinical Implications

Patients with Hashimoto's thyroiditis are increasingly recognized as a population at elevated cardiovascular risk, even in the absence of overt hypothyroidism. One of the principal contributors to this risk is the development of an atherogenic lipid profile, characterized by elevated total cholesterol, low-density lipoprotein cholesterol, and triglyceride levels, alongside reduced high-density lipoprotein cholesterol. These alterations are primarily driven by reduced thyroid hormone action on hepatic lipid metabolism but may also be amplified by chronic autoimmune inflammation [2, 11].

Hypertension and endothelial dysfunction further compound cardiovascular risk in individuals with HT. Thyroid hormone deficiency is associated with increased systemic vascular resistance, impaired nitric oxide-mediated vasodilation, and arterial stiffness. In parallel, inflammatory cytokines and oxidative stress related to thyroid autoimmunity contribute to endothelial injury and vascular dysfunction, thereby accelerating atherosclerotic processes and increasing the likelihood of adverse cardiovascular events [6, 15].

Given the established association between Hashimoto's thyroiditis and metabolic syndrome, systematic screening for metabolic abnormalities in patients with HT is increasingly advocated. Assessment of MetS components, including waist circumference, blood pressure, fasting glucose, and lipid profile, may facilitate early identification of individuals at high cardiometabolic risk, particularly among those with subclinical or overt hypothyroidism [4, 12].

Conversely, screening for thyroid dysfunction and thyroid autoantibodies in high-risk metabolic populations has potential clinical value. Individuals with obesity, insulin resistance, or MetS may benefit from evaluation of thyroid-stimulating hormone levels and anti-TPO or anti-Tg antibodies, as early detection of autoimmune thyroiditis may enable closer monitoring and timely therapeutic intervention [9, 18]. Such bidirectional screening strategies support a more integrated approach to endocrine and metabolic care.

Thyroid hormone replacement therapy, primarily with levothyroxine, represents the cornerstone of treatment for hypothyroidism due to Hashimoto's thyroiditis. Restoration of euthyroid status has been shown to improve several metabolic parameters, including reductions in total cholesterol and low-density lipoprotein cholesterol, as well as modest improvements in insulin sensitivity [1, 14].

Evidence from interventional studies suggests that the metabolic benefits of levothyroxine therapy are most pronounced in patients with overt hypothyroidism, while effects in subclinical hypothyroidism are more variable and may depend on baseline TSH levels and cardiovascular risk profile [7, 20]. Importantly, thyroid hormone replacement does not fully reverse metabolic syndrome in all patients, highlighting the contribution of autoimmune inflammation and lifestyle factors beyond thyroid hormone deficiency alone [5, 17].

### *Special Populations*

Certain population groups exhibit unique patterns of interaction between Hashimoto's thyroiditis and metabolic syndrome. Women of reproductive age represent a particularly relevant group, as thyroid autoimmunity may coexist with metabolic disturbances that influence fertility, pregnancy outcomes, and long-term cardiovascular health. Hormonal fluctuations and immune modulation during reproductive years may further affect disease expression [3, 10].

Postmenopausal women demonstrate a higher prevalence of both HT and MetS, reflecting the combined effects of estrogen deficiency, age-related metabolic changes, and immune dysregulation. In this group, the coexistence of thyroid autoimmunity and metabolic syndrome substantially amplifies cardiovascular risk and warrants close clinical surveillance [8, 16].

Pediatric and adolescent patients with Hashimoto's thyroiditis increasingly present with features of metabolic syndrome, particularly in the context of rising childhood obesity rates. Early-onset thyroid autoimmunity may interact with metabolic risk factors during critical periods of growth and development, potentially predisposing affected individuals to long-term cardiometabolic disease [6, 19].

Obese and insulin-resistant individuals constitute another high-risk population in whom the overlap between HT and MetS is especially pronounced. Excess adiposity promotes immune activation and thyroid autoimmunity, while concomitant hypothyroidism exacerbates metabolic derangements, creating a self-perpetuating cycle of metabolic and immune dysfunction [12, 21].

Effective management of patients with coexisting Hashimoto's thyroiditis and metabolic syndrome requires an integrated therapeutic approach that addresses both thyroid autoimmunity and metabolic risk factors. Optimization of thyroid hormone replacement therapy should be combined

with targeted interventions aimed at improving insulin sensitivity, lipid profiles, and blood pressure control [4, 15].

Lifestyle interventions remain a cornerstone of prevention and management. Dietary modification, weight reduction, and regular physical activity have been shown to improve metabolic parameters and may also exert beneficial effects on immune regulation and inflammatory burden. Such non-pharmacological strategies are particularly important in patients with euthyroid HT, where thyroid hormone therapy alone is not indicated [9, 18].

Emerging evidence suggests a potential role for anti-inflammatory and immunomodulatory strategies in mitigating the interplay between thyroid autoimmunity and metabolic dysfunction. Although data remain limited, interventions targeting chronic inflammation and oxidative stress may represent promising adjunctive approaches in selected patients, warranting further investigation in well-designed clinical trials [7, 20].

## Conclusions

The accumulating evidence reviewed in this article highlights a clear and clinically meaningful association between Hashimoto's thyroiditis and metabolic syndrome. This relationship extends beyond the effects of overt thyroid hormone deficiency and involves a complex interplay of immune dysregulation, chronic low-grade inflammation, insulin resistance, adipokine imbalance, and endothelial dysfunction. Both conditions share common pathogenic pathways that mutually reinforce metabolic and cardiovascular risk, even in individuals who remain biochemically euthyroid [3, 11].

Epidemiological data consistently demonstrate an increased prevalence of metabolic syndrome and its individual components among patients with Hashimoto's thyroiditis, while thyroid autoimmunity is also more frequently observed in populations with established metabolic disturbances. These findings support the concept of a bidirectional relationship in which autoimmune thyroid disease and metabolic dysfunction coexist and potentially exacerbate one another [6, 14].

From a clinical perspective, recognition of this association has important implications for risk stratification, screening, and long-term management. Patients with Hashimoto's thyroiditis should be routinely assessed for metabolic abnormalities and cardiovascular risk factors, while individuals with metabolic syndrome may benefit from targeted evaluation for thyroid dysfunction and autoimmunity. Although thyroid hormone replacement therapy improves several metabolic parameters in hypothyroid patients, it is insufficient as a standalone strategy, underscoring the need for comprehensive metabolic and lifestyle-based interventions [8, 17].

In conclusion, Hashimoto's thyroiditis and metabolic syndrome should be viewed as interconnected disorders within a shared immunometabolic framework. Future research should focus on longitudinal and mechanistic studies to clarify causality, identify reliable biomarkers for early risk prediction, and develop integrated therapeutic approaches that address both autoimmune and metabolic components. Such strategies hold promise for reducing long-term cardiovascular morbidity and improving overall patient outcomes [5, 21].

## Conflict of interest

The authors declared no conflict of interest

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