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The Impact of Lifestyle on the Course of Hashimoto's Disease: The Role of Diet, Physical Activity, and Stress - A Literature Review

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Abstract

Introduction and purpose of the work: Hashimoto's thyroiditis (HT), also called chronic autoimmune lymphocytic thyroiditis, is a major cause of hypothyroidism in iodine-sufficient areas. It affects women more often and may co-occur with other autoimmune diseases. HT involves T-cell mediated damage to thyroid cells and the presence of anti-TPO and anti-Tg antibodies. While levothyroxine restores hormone levels, many patients still report fatigue, mood issues, and cognitive problems. This review examines how lifestyle factors—diet, exercise, and stress—affect the course of HT and patient outcomes.

State of knowledge: Environmental and behavioral factors are increasingly recognized in autoimmune disease progression. Diets high in processed foods and low in micronutrients may promote inflammation and gut imbalance, worsening autoimmunity. Sedentary behavior and chronic stress also impair immune function. On the other hand, anti-inflammatory diets,

physical activity, and stress management may support immune balance and complement standard HT treatment.
Materials and methods: A literature review was conducted in PubMed for studies up to 2025, including reviews, original articles, and observational studies in English focused on lifestyle factors in HT.
Summary: Lifestyle changes—such as anti-inflammatory diets (e.g., Mediterranean, AIP), regular physical activity, and stress reduction—can improve symptoms and modulate disease markers in HT. These findings support integrating holistic strategies into patient care.

Keywords: lifestyle medicine, physical activity, Hashimoto's thyroiditis, autoimmunity, Mediterranean diet, chronic stress, immune modulation

1.Introduction

Hashimoto's thyroiditis (HT) is the most common autoimmune endocrine disease and the main cause of hypothyroidism in iodine-sufficient areas [1]. The first mention of autoimmune thyroiditis dates back to 1912, when Japanese doctor Hakaru Hashimoto described four cases of patients with thyroid lesions in the form of goiter, clinically characterized by a diffuse enlargement of the thyroid gland [2]. During palpation, an enlarged thyroid gland is usually painless and has a hard and smooth surface. However, as the condition develops, the thyroid gland becomes smaller and nodules may begin to appear [3].

HT development leads to scarring and destruction of the thyroid gland and is manifested by a decrease of plasma free triiodothyronine (T3) and thyroxine (T4), elevated plasma levels of thyroid-stimulating hormone (TSH) [4]. It is characterized by high levels of thyroid antibodies. Specifically, it is marked by elevated levels of anti-TPO (thyroid peroxidase) and anti-Tg (thyroglobulin) antibodies in serum, and it is influenced by many factors. [5-6].

Other diagnostic methods in approaching HT include ultrasonography (USG) of the thyroid gland and fine needle aspiration (FNA) [1,7]. The most commonly observed changes in the USG are enlargement, hypoechogenicity of the thyroid parenchyma, and hypervascularity, although it depends on the phase of HT [7-9]. Single or multiple nodules in the parenchyma of the thyroid may be present in the nodular type of HT [7].

In adults with Hashimoto's thyroiditis (HT), clinical presentation predominantly involves cutaneous manifestations, including marked xerosis and scaling, most pronounced on the palms, soles, and extensor surfaces. Hair abnormalities are also frequently observed, with diminished growth velocity and structural impairment resulting in hair that is dry, brittle, and lacking luster. Hypothyroid states secondary to HT can induce an increase in peripheral vascular

resistance by 50%–60%, accompanied by a reduction in cardiac output by approximately 30%–50%. A heritable genetic predisposition is considered a major determinant underlying the elevated incidence and relative risk of coexisting autoimmune diseases among HT patients and their first-degree relatives [10-11]. In addition, a significantly higher incidence of depressive and anxiety disorders has been reported among patients with autoimmune thyroiditis (AIT) [12]. Furthermore, individuals with AIT appear to be at increased risk not only for thyroid carcinoma but also for breast, pulmonary, gastrointestinal, genitourinary, and hematologic malignancies, as well as prolactinoma [13].

According to the American Thyroid Association (for 2023) [5], the diagnostic criteria for autoimmune thyroiditis include:

1. symptoms of hypothyroidism combined with elevated Thyroid Stimulating Hormone (TSH) levels, with or without low thyroid hormone levels;

2. enlargement of the thyroid gland (goiter);

3. elevated thyroid antibody levels.

2. Epidemiology

The incidence of Hashimoto's thyroiditis is estimated at 0.3–1.5 cases per 1,000 individuals, with a pronounced female predominance reflected by a female-to-male ratio of approximately 7–10:1 [10,14]. Ethnic variability has also been observed, with a higher prevalence reported among individuals of Caucasian ancestry compared to African American or Asian populations, and a notably low incidence among Pacific Islanders [15]. Advancing age constitutes a significant risk factor for the development of Hashimoto's thyroiditis [10], particularly in individuals with concomitant autoimmune disorders, including myasthenia gravis [16], systemic sclerosis [17], connective tissue diseases [18], Sjögren's syndrome [19-20], pernicious anemia [19-20], autoimmune hepatitis, and celiac disease [18-20]. The frequent coexistence of multiple autoimmune diseases, a phenomenon termed polyautoimmunity, is believed to arise from intricate interactions among immune dysregulation, hormonal influences, genetic predisposition, and environmental exposures [21]. Moreover, among the female population, an increased prevalence of Hashimoto's thyroiditis has been identified in patients diagnosed with polycystic ovary syndrome (PCOS) [22-23]. The incidence of autoimmune thyroiditis (AIT) exhibits an age-dependent rise, with peak occurrence observed between 45

and 65 years, although cases are also documented in pediatric cohorts [3,24]. Notably, in the pediatric population, the presence of chromosomal aberrations such as Down syndrome, Turner syndrome, and Klinefelter syndrome has been associated with a heightened risk of developing AIT [25].

3.Pathophysiology

Hashimoto's thyroiditis (HT) is an autoimmune disease influenced by both genetic and environmental factors. Genetic predisposition involves polymorphisms in HLA, CTLA-4, PTPN22, and genes regulating T and B cell tolerance, as well as X chromosome inactivation mechanisms [20,26-27]. Additional susceptibility is associated with polymorphisms in autoantigens, cytokines (e.g., IL2R), estrogen receptors, adhesion molecules (CD14, CD40), and genes involved in apoptosis and selenoprotein expression [20,`28-29]
[30-31]. Epigenetic mechanisms such as methylation, histone modification, and non-coding RNA interference further modulate disease risk. Environmental triggers include infections, smoking, fetal-maternal microchimerism, and chemical exposures like phthalates and flame retardants [32-33]. Interestingly, low environmental exposure (e.g., overly hygienic conditions) also correlates with higher rates of autoimmune diseases, including HT [34].
Gut microbiota composition is increasingly linked to HT, with a reduced presence of beneficial genera (e.g., *Bifidobacterium*, *Lactobacillus*) and an increase in pathogenic bacteria such as *Bacteroides fragilis* observed in HT patients [35].

Diet also plays a significant role in the course of HT. Excessive iodine intake may increase disease incidence up to fourfold by enhancing thyroglobulin (Tg) immunogenicity in genetically susceptible individuals [36-37]. Although excessive supplementation is not recommended in HT, iodine intake of up to 250 μg/day is advised for pregnant and breastfeeding women [38]. Low selenium intake might contribute to disease activation; however, while supplementation reduces anti-TPO levels, it does not alter disease progression [39-40].

A gluten-restricted diet has been suggested as a potential modulator in HT, especially in patients with coexisting celiac disease. Studies show reduced thyroid volume or decreased anti-TPO levels in gluten-free individuals, though the clinical relevance of antibody reduction remains unclear [41-42].

Mechanistically, HT is characterized by T-cell-mediated destruction of thyroid tissue, with histopathological features including lymphoplasmacytic infiltration, fibrosis, and follicular atrophy [43]. Variants of HT include fibrotic, atrophic, Riedel's thyroiditis, and IgG4-

related forms [44-46]. Single-cell RNA sequencing has revealed the key role of thyroid stromal cell subtypes in recruiting immune cells that intensify thyroid destruction and antigen exposure (TPO, Tg), further amplifying autoimmune responses [47]. Notably, elevated anti-TPO and anti-Tg antibodies are more common (15–25%) than clinical hypothyroidism, especially in iodine-sufficient populations, in women, and in older adults [48].

4. Results

4.1. Impact of diet on the course of Hashimoto's Disease

Dietary interventions play a pivotal role in modulating immune function and inflammatory status in HT. The Western diet (WD), marked by high intake of saturated fats, refined sugars, animal proteins, and ultra-processed foods, alongside low consumption of fiber, antioxidants, and micronutrients, has been associated with an increased risk of autoimmune diseases, including Hashimoto's thyroiditis [49-55]. These dietary habits promote chronic low-grade inflammation ("metainflammation"), oxidative stress, and immune dysregulation through increased pro-inflammatory cytokines (e.g., IL-6, IL-17, TNF-α) and imbalances in regulatory T cell function [56-61]. Furthermore, WD negatively impacts the gut microbiota and intestinal barrier, leading to dysbiosis and enhanced intestinal permeability. This "leaky gut" state allows microbial and dietary antigens to enter systemic circulation, triggering autoimmune responses in genetically predisposed individuals [62-67] Reduced intake of antioxidants and excessive consumption of red and processed meats further aggravate oxidative stress, a key factor in thyroid autoimmunity [68]. Current evidence supports the view that WD contributes to the initiation and exacerbation of autoimmune thyroid disorders by promoting systemic inflammation, oxidative damage, and gut-derived immune activation.

In contrast to the pro-inflammatory nature of the Western diet, the Mediterranean diet (MD) represents a well-established model of anti-inflammatory, nutrient-dense nutrition. Originating from traditional eating patterns of populations in the Mediterranean basin, the MD is characterized by a high intake of plant-based foods (e.g., vegetables, fruits, legumes, nuts, whole grains), moderate consumption of fish, dairy, and red wine, and olive oil as the primary lipid source [69-70]. Rich in monounsaturated fatty acids, omega-3 polyunsaturated fatty acids (PUFAs), fiber, and essential micronutrients (e.g., selenium, iodine, zinc, vitamins E, B, C, and β-carotene), the MD supports oxidative balance, reduces systemic inflammation, and positively modulates the gut microbiome and immune function [71-73]. Importantly, its health effects are

attributed not to isolated nutrients, but to the synergistic action of multiple bioactive compounds consumed as part of a whole-diet approach [74-75] Beyond its cardioprotective effects demonstrated in the seminal *Seven Country Study*, the MD has been associated with reduced incidence of metabolic syndrome and emerging protection against autoimmune diseases [76-84]. Recent studies highlight its immunomodulatory and antioxidant properties, making it a rational dietary model for individuals with autoimmune thyroid disorders, including Hashimoto's thyroiditis [85-86] Moreover, the MD is recognized by UNESCO as part of the Intangible Cultural Heritage of Humanity, not only for its nutritional composition but also for its broader lifestyle and cultural dimensions, emphasizing food quality, sustainability, and shared eating practices [87].

Given its anti-inflammatory profile and metabolic benefits, the MD is increasingly recommended as a complementary strategy in the dietary management of HT, serving as a therapeutic counterpoint to the Western dietary pattern. Ruggeri et al. (2023) reported that high adherence to Mediterranean dietary principles was associated with significantly reduced antibody levels and improved patient-reported outcomes [88].

The Autoimmune Protocol (AIP) diet, which entails rigorous elimination of potential immunogenic foods followed by phased reintroduction, has been associated with reductions in high-sensitivity C-reactive protein and symptomatic improvement [89]. However, adherence may be limited by the restrictive nature of the diet, necessitating individualized dietary planning.

Iodine is vital for thyroid hormone synthesis, with dietary intake varying by region and cultural practices [90-91]. While iodine deficiency historically caused widespread hypothyroidism, iodization programs have reduced this issue but coincided with a rise in autoimmune thyroiditis (AIT) cases [92-93]. Excess iodine may enhance thyroglobulin immunogenicity, triggering immune responses marked by elevated TPOAb and TgAb, cytokine activation (e.g., IFN-γ), and increased IP-10 levels, all contributing to AIT development and severity [93]. Studies show that children with high urinary iodine often have higher TSH and lower thyroid hormone levels, with AIT diagnosed at a younger age [90,94]. Additionally, iodine exposure during delivery (e.g., povidone-iodine) may cause transient neonatal hypothyroidism, though typically resolving by age one [95].

Micronutrients such as selenium, vitamin D, zinc, and myo-inositol have been identified as modulators of thyroid autoimmunity [96-97]. Supplementation in deficient individuals

appears to attenuate antibody titers and improve thyroid function, although optimal dosing strategies remain under investigation.

Selenium is an essential micronutrient with antioxidant properties, vital for protecting the body against oxidative stress and preventing various diseases, including inflammatory, neurological, and cardiovascular disorders [98-99].

The thyroid gland contains one of the highest concentrations of selenium, which is crucial for the synthesis, activation, and metabolism of thyroid hormones [98]. Selenium also plays a significant role in immune system function, which is particularly relevant in autoimmune thyroiditis (AIT). Chronic selenium deficiency can predispose individuals to the development of AIT.

Selenium levels in the body are influenced by both the selenium content in the local soil and dietary intake. A study by Qian Wu et al. demonstrated that selenium deficiency is more common in regions with lower selenium intake, and the incidence of AIT is significantly higher in these areas compared to those with adequate selenium intake [100]. Another study further highlighted the role of selenium and selenoproteins in AIT pathogenesis, revealing that selenium levels were notably lower in AIT patients, while the level of selenoprotein H (SelH), which plays a role in thyroid hormone metabolism and oxidative stress response, was elevated [101]. This increased SelH level may reflect an enhanced antioxidant response to oxidative stress in AIT patients.

Increased oxidative stress and lower selenium levels were also observed in AIT patients in research conducted by Rahim Rostami et al., which suggested a link between selenium deficiency, elevated iodine levels, and the progression of thyroid dysfunction [102]. Selenium supplementation has shown promising effects on thyroid function in AIT patients by reducing thyroid autoantibodies (TPOAb, TgAb), lowering TSH, and improving antioxidant activity and regulatory T cell (Treg) levels, which could indicate a reduction in autoimmune activity [103-104]. Some studies have also suggested that selenium supplementation may restore euthyroidism in subclinical hypothyroid patients with AIT [105-106]

Vitamin D plays a crucial role in regulating the immune system, primarily by influencing the production of pro-inflammatory cytokines such as IL-1, IL-6, TNF-α, and IL-17 [107-108]. This regulatory effect of vitamin D has been implicated in various autoimmune diseases, including Hashimoto’s Thyroiditis (HT) [109]. Vitamin D deficiency, particularly common in

regions with low sunlight, has been observed to exacerbate inflammatory processes and autoimmune conditions like HT, rheumatoid arthritis, and multiple sclerosis [110-111]. Several studies suggest that lower serum levels of vitamin D in HT patients correlate with higher titers of pro-inflammatory cytokines and may also impact cognitive function negatively. Specifically, lower vitamin D levels have been associated with poorer Montreal Cognitive Assessment (MoCA) scores in HT patients, which is particularly evident in children with severe hypothyroidism [112-114]. Despite these findings, the relationship between vitamin D deficiency and HT remains complex. While some studies confirm a clear correlation between low vitamin D levels and HT, others do not establish a definitive causality [115-120]. The influence of vitamin D on autoimmune thyroiditis (AIT) continues to be widely researched, with ongoing investigations into whether vitamin D deficiency acts as a risk factor for AIT or simply a consequence of the disease [121]. The need for vitamin D supplementation in individuals with autoimmune thyroid diseases, especially in those with a deficiency, is also emphasized to potentially mitigate inflammatory processes and improve patient outcomes [122].

4.2. Impact of Physical activity on the course of Hashimoto's Disease

Physical activity confers a range of immunomodulatory and metabolic benefits relevant to HT management. Regular moderate aerobic exercise has been shown to reduce proinflammatory cytokine levels, improve mood and energy levels, and enhance gut microbiota diversity. In a 2023 study by Kamińska et al. [123], implementation of the Mediterranean diet in conjunction with WHO-recommended levels of physical activity led to significant reductions in body fat and waist circumference among HT patients. The 10-week lifestyle intervention, including adherence to the Mediterranean diet and promotion of physical activity, demonstrated measurable improvements in body composition among women with Hashimoto's thyroiditis (HT), despite the absence of significant changes in body weight or reported physical activity levels at the group level.

While physical activity, assessed using MET-min/week, did not significantly change in the HT group (from 4549.5 ± 1592 to 4560.7 ± 2044, $p = 0.878$), several significant changes in anthropometric parameters were observed. Notably, **BMI decreased significantly** from 24.7 ± 5.3 to 24.0 ± 4.7 kg/m² ($p < 0.05$), and **waist circumference was reduced** from 84.6 cm to 81.3 ± 14 cm ($p = 0.045$). In addition, **body fat percentage decreased significantly** from 30.1 ± 9% to 28.5 ± 8.7% ($p = 0.002$), while muscle mass remained unchanged.

Importantly, correlation analysis revealed that **increases in muscle mass and muscle glycogen content** were **strongly associated with changes in physical activity** ($\rho = 0.661$ and $\rho = 0.709$, respectively). Moreover, a nearly perfect correlation was observed between changes in **muscle mass and glycogen content** ($\rho = 0.947$), indicating coordinated physiological adaptation. Additionally, a higher baseline body weight was positively correlated with greater weight reduction over the course of the intervention ($\rho = 0.585$, $p = 0.028$).

These findings suggest that even in the absence of statistically significant changes in overall activity levels, **lifestyle interventions can lead to meaningful improvements in body composition** in women with HT. The results emphasize the importance of **qualitative changes in physical activity** and dietary adherence in modulating metabolic and inflammatory parameters in autoimmune thyroid disease [123].

Exercise may also ameliorate fatigue—a frequently reported symptom among HT patients—through its effects on mitochondrial function and central nervous system activation. Nonetheless, exercise regimens must be carefully tailored in cases of severe hypothyroidism to avoid exacerbation of symptoms.

4.3. Impact of Stress on the course of Hashimoto's Disease

Stress, both acute and chronic, is increasingly recognized as a potential environmental factor that may trigger autoimmune thyroiditis (AIT). Although research in this area remains limited, several studies suggest that stress disrupts immune system homeostasis, contributing to the development of autoimmune diseases. This disruption is thought to result from hormonal imbalances caused by the activation of the sympathetic-adrenal system and the hypothalamic-pituitary-adrenal (HPA) axis during stressful events. Such activation leads to the excessive release of catecholamines and the overproduction of glucocorticoids, including cortisol [124]. Chronic stress, in particular, has been linked to systemic inflammation, primarily through increased levels of pro-inflammatory cytokines, which may further influence the pathogenesis of AIT [124].

Moreover, stress appears to modulate thyroid function through its impact on thyroid-stimulating hormone (TSH). A study by Hua Hong and Jeonghun Lee explored the potential of TSH as a biomarker for stress, revealing that while TSH is typically regulated by free triiodothyronine (fT3) and free thyroxine (fT4) through a negative feedback loop, cortisol and

cytokine levels may also influence serum TSH concentrations. However, no significant correlation was found between TSH levels and stress questionnaires, suggesting the presence of confounding factors such as environmental influences, personality traits, or emotional regulation strategies [125].

Additionally, psychological stress has been shown to play a significant role in modulating autoimmune dynamics. The chronic stimulation of the HPA axis, which elevates cortisol and catecholamine levels, can influence cytokine profiles and immune cell trafficking [88]. Patients with Hashimoto's thyroiditis (HT) often report higher levels of perceived stress compared to healthy controls, and studies have demonstrated improvements in their condition following psychological interventions [126]. Psychological interventions such as mindfulness-based stress reduction (MBSR), cognitive-behavioral therapy (CBT), and yoga have shown promise in mitigating stress-related immunopathology [126]. Given the bidirectional relationship between thyroid dysfunction and mood disorders, psychological assessment and support should be considered a routine component of HT care.

In addition to these findings, one study revealed that AIT patients experienced higher levels of perceived stress compared to controls, despite having similar numbers of stressful life events. This study also demonstrated a correlation between the number of stressful life events and increased levels of anti-TPO antibodies, suggesting that stress may induce changes in immune pathways that contribute to the development of AIT [124]. These findings highlight the importance of addressing psychological stress as part of managing autoimmune thyroiditis, emphasizing the potential for therapeutic strategies to reduce stress and improve immune function in affected individuals.

5.Conclusions

The integration of lifestyle interventions into the clinical management of Hashimoto’s thyroiditis represents a promising adjunct to pharmacotherapy. Evidence supports the efficacy of anti-inflammatory diets, structured physical activity, and stress-reduction techniques in modulating immunological markers and improving quality of life. Future research should prioritize longitudinal, controlled trials to delineate optimal intervention protocols and elucidate underlying mechanisms. In the interim, clinicians should consider adopting a personalized, interdisciplinary approach that empowers patients to engage in their own health optimization.

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