Nutrition and Food in Health and Disease Vol 12, No 3, Jul-Sep 2025, pages: 1-9

Rewiew Article

Assessing Efficacy of Dietary Interventions in Patients with Hashimoto's Thyroiditis

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Received: March 2025 **Accepted:** May 2025

ABSTRACT

Background and Objectives: : Hashimoto's thyroiditis is an autoimmune disorder that potentially induces hypothyroidism through immune-mediated destruction of the thyroid gland. Nutrients may play a critical role in modulating functionality of the immune system. The aim of this review was to provide a comprehensive summary of the available data on beneficial nutrients and dietary interventions for the management of Hashimoto's thyroiditis.

Materials and Methods: A comprehensive search was carried out on databases such as PubMed, Scopus, ISI Web of Science and Embase, published 1990–2025, to identify all relevant articles that addressed the topics of nutrients, dietary patterns and Hashimoto's thyroiditis.

Results: Certain nutrients might have a critical role in regulating the immune system. Supplementation with appropriate nutrients could potentially improve adverse effects associated with HT and enhance the overall quality of life for the patients. However, certain limitations such as contradictory results and restriction of randomized controlled trials investigating effects of nutrients and dietary patterns in patients with Hashimoto's thyroiditis were reported.

Conclusions: Nutrients may include beneficial effects in management of Hashimoto's thyroiditis. Further studies are necessary to validate these findings and establish recommendations for patients with Hashimoto's thyroiditis.

Keywords: Hypothyroidism, Hashimoto's thyroiditis, Diets, Nutrition management

Highlights

- Some specefic dietary components may positively influence immune function and Hashimoto's thyroiditis symptoms.
- Supplementation could improve quality of life in patients with Hashimoto's thyroiditis.
- Limitations include inconsistent findings and a lack of sufficient randomized controlled trials (RCTs).

Introduction

Hashimoto's thyroiditis (HT) is an autoimmune disorder characterized by immune system attacking and damaging the thyroid gland, leading to hypothyroidism. While HT is a significant contributor to hypothyroidism, it is noteworthy that the most common cause of hypothyroidism worldwide includes the inadequate dietary intake of iodine. Insufficient iodine levels can impair the thyroid gland ability to produce thyroid hormones, resulting in hypothyroidism (1). This condition is further relevant in women (2, 3), similar to several other autoimmune disorders. Development of HT, like other autoimmune conditions, is affected by a combination of

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[DOI: 10.61882/nfsr.12.3.1]

genetic and environmental factors. These factors can include dietary choices, alcohol consumption and smoking habits (2).

Since HT may initially be asymptomatic, various symptoms are reported in its advanced stage. These symptoms often include cold and dry skin, facial edema particularly around the eyes (periorbital edema), non-pitting edema affecting the hands and feet, brittle nails, bradycardia, delayed relaxation phase of tendon reflexes, ataxia, increased blood pressure and macroglossia. These manifestations are commonly observed as the disease progresses (1–3). The pathogenesis of HT is intricately linked to autoantibodies, which are implicated in the lymphocytic infiltration involving B and T-cells within the thyroid gland. It is hypothesized that one of the primary events in pathogenesis of HT involves functional modification of B cells, leading to production of autoantibodies. Additionally, T-cell dysfunction is associated to disrupted immune homeostasis in the thyroid tissue (4).

Numerous studies have highlighted the essential roles of nutrients in modulating immune system functions (5). Current studies have provided promising findings, suggesting potential clinical benefits of certain nutrients, including vitamin D, n-3 polyunsaturated fatty acids (PUFA), vitamin E, zinc and probiotics, in the prevention of autoimmune and inflammatory diseases. Therefore, it is hypothesized that inadequate dietary patterns and insufficient intake of these specific nutrients can potentially contribute to the development of autoimmune diseases such as HT (6, 7). Furthermore, particular nutrients such as iodine, iron, selenium, zinc and protein are critical for the synthesis and activation of thyroid hormones, including triiodothyronine (T3) and thyroxine (T4) (8–10). Emerging evidence suggests that diets low in animal-based foods may provide protective effects on individuals with HT, potentially through their effects on redox balance and moderation of oxidative stress-linked disorders (11). Moreover, excluding gluten from diets has demonstrated significantly clinical advantages in individuals diagnosed with autoimmune thyroiditis (12).

To the best of the authors' knowledge, no systematic review has been carried out to investigate effects of nutrients and dietary patterns, specifically in patients with HT. Therefore, the primary objective of this review was to provide a comprehensive summary of the existing data to investigate beneficial nutrients and dietary interventions that might be effective in management of HT.

Search for identify relevant studies

A comprehensive search was carried out in various databases, including PubMed, Scopus and ISI Web of Science, 1990–2022, using appropriate keywords of "Hashimoto" AND "nutrient" OR "vitamin" OR "mineral" OR "macronutrient" OR "micronutrient" OR "protein" OR "retinol" OR "tocopherol" OR "thiamine" OR "pyridoxine" OR "ascorbic acid" OR "cobalamin" OR "magnesium" OR "iron" OR "iodine" OR "zinc" OR "selenium". The search aimed to investigate all relevant articles investigating relationships between nutrients, dietary patterns and HT.

Findings

Insufficient and excessive levels of certain dietary nutrients and minerals are involved in the pathogenesis of hypothyroidism and HT. Deficiencies or imbalances in omega-3 fatty acids, vitamins A, D, E, B1, B12 and C, iron, zinc and selenium have been verified to play significant roles in development of these conditions. Additionally, studies have shown that supplementation with specific dietary compounds can include positive effects on the overall health of individuals affected by hypothyroidism and HT.

Dietary proteins

Proteins are widely recognized as essential macronutrients. The recommended protein intake for adults aged up to 65 y is 0.8 g per kilogram of body weight (BW). In contrast, adults aged over 65 y are advised to consume 1 g per kilogram of BW (11). Restricting protein intake can include detrimental effects on humans and animals, particularly during pregnancy and lactation, as it can negatively affect long-term development, growth and metabolic and hormonal statuses of the offspring. Numerous studies have suggested that the quantity and composition of dietary protein intake can affect activity of the hypothalamic-pituitary-thyroid axis (12). In rats and humans, a low-protein diet has been associated to plasma thyroid binding globulin (TBG) levels, decreased plasma transthyretin and T3 levels, as well as decreased pituitary thyroid stimulating hormone (TSH) transcript levels. This change is commonly seen in malnourished children. When protein malnutrition is combined with inadequate energy intake, it exacerbates iodine deficiency and leads to thyroid gland injury. The body natural adaptive responses to protein and calorie deficits result in changes of TSH levels. This phenomenon may occur more frequently in patients with HT, who have caloric-protein malnutrition than those who consume standard diets (13).

Vitamin C (ascorbic acid)

The vitamin C content in various foods generally ranges from moderate to high levels (10–100 mg 100 g^{-1}). In a few cases, vitamin C content can reach several grams per 100 g of food weight. While commonly recognized as an antioxidant, vitamin C plays a more significant physiological role, encompassing diverse functions such as facilitating iron absorption and hormone synthesis and participation in critical processes such as carnitine

[DOI: 10.61882/nfsr.12.3.1]

synthesis and epigenetic regulation (14). Excessive production of reactive oxygen species can lead to inflammation and dysfunction, particularly in organs with high activity such as thyroid gland. Moreover, oxidative stress induced by free radicals has been involved in numerous autoimmune diseases. In patients with subclinical hypothyroidism (sHT) and autoimmune thyroiditis, low-grade inflammatory responses impair nitric oxide (NO) availability and promote endothelial dysfunction through a mechanism dependent on Cyclooxygenase-2. This process contributes to increased oxidative stress. However, administration of vitamin C does not demonstrate significant improvements in these cases (15). A recent study detected that participants with the highest quartile of total vitamin C intake were at lower odds of having hypothyroidism, compared with those with the lowest quartile of total vitamin C intake (log 10-transformed), (adjusted OR 0·40, 95 % CI 0·18, 0·88, Ptrend = 0·027) (16).

Vitamin E

Vitamin E is one of the widely used dietary supplements, primarily due to a common belief that its antioxidant and anti-inflammatory characteristics can potentially prevent cell damages in the human body (17). However, most studies investigating effects of vitamin E on thyroid diseases have primarily been carried out on animals, majorly rats. Therefore, clinical studies on human subjects need to be carried out (18–20). While studies suggest that conditions such as HT and other thyroid diseases may be associated with increased oxidative stress, the available evidence does not provide sufficient justification for the widespread prescription of vitamin E as a necessary supplement in these cases.

Omega-3 fatty acids

Omega-3 fatty acids are polyunsaturated fatty acids (PUFA) which can be detected in fishes, seeds, nuts, beans and green leafy vegetables. From omega-3 fatty acids, three are clinically important, including alpha-linolenic acid (ALA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (21–23). These fatty acids (FA) have been shown to enhance the immune system through various mechanisms such as improving activity of B cells, decreasing production of cytokines such as TNFα, IL-1β and IL-6, inhibition of the synthesis of C-reactive protein (CRP) and inflammatory eicosanoids and enhancing phagocytosis (24–26). Furthermore, a few studies have demonstrated that omega-3 PUFAs can decrease differentiation of Th17 cells from naive $CD4^+$ T-cells through modification of the lipid raf regions in their plasma membrane (27). Additionally, omega-3 fatty acids are investigated as dietary supplements for potentially treating autoimmune diseases. A recent study indicated that omega-3 supplementation could decrease the incidence of autoimmune diseases by 15% (28).

Myo-inositol

Myo-inositol is a critical growth-promoting agent for mammalian cells and animals. It acts as a lipotropic factor and serves as a cofactor for various enzymes as well as a messenger molecule in signal transduction processes (29). Studies have indicated that Myo-inositol may include a positive effect on serum levels of thyroid-stimulating hormone (TSH) in patients with HT. This effect can be further enhanced when combined with selenium supplementation (30, 31). Furthermore, Myo-inositol has been reported to include a protective function for human thyrocytes by decreasing secretion of C-X-C motif chemokine ligand 10 (CXCL10), which is induced by a combination of IFN-γ and TNF-α in primary thyrocytes (32). While studies support the beneficial effects of Myo-inositol administration, other studies suggest that vitamin D supplementation may be further effective for autoimmune thyroiditis (33).

Vitamin A

Vitamin A plays a dual role in the immune system, acting as a promoter and regulator of innate and adaptive immunities. It can potentially enhance immune functions and provide improved defense against various infectious diseases (34, 35). However, there is a limited studies on the effects of vitamin A specifically on autoimmune and inflammatory diseases. In patients with autoimmune diseases, vitamin A has been shown to modulate gene expression by significantly decreasing the levels of inflammatory cytokines (e.g. IL-17, IFN-g and T-bet) and increasing the levels of anti-inflammatory cytokines (e.g. TGF-b and FOXP3) (36). Another potential mechanism; by which, vitamin A may benefit thyroid health is its ability to significantly decrease serum levels of TSH and thereby decreasing the risk of subclinical hypothyroidism (37). Studies suggest that certain genetic variations in vitamin A-linked genes such as CYP26B1 and retinoic acid receptor (RAR) are associated with the proportion of Th17 cells, levels of IFN-γ and IL-17 and susceptibility to HT (38). Regarding the critical role of vitamin A in thyroid health and prevention of HT, it is recommended to include various dietary sources of vitamin A and beta-carotene in diets (13).

Magnesium

Magnesium (Mg) is an essential mineral detected most abundantly in healthy foods such as nuts, seeds, leafy greens and whole grains (39). Magnesium plays a critical role in thyroid hormone synthesis, indirectly. It is involved in the iodine uptake and deiodination steps during thyroid hormone synthesis (40). Studies have shown that magnesium supplementation can improve menstrual

[DOI: 10.61882/nfsr.12.3.1]

disorders and fertility by affecting thyroid gland function (40, 41). The HT is associated to a wide range of symptoms, including chronic fatigue, anxiety, irritability, concentration difficulties and nervousness. Studies have indicated that magnesium supplementation can improve psychological symptoms associated to HT (42–44). Furthermore, low serum magnesium levels are associated to increased risks of anti-thyroglobulin antibodies and HT (45). In another study, the co-supplementation of magnesium, selenium and coenzyme Q10 in individuals with benign thyroid problems demonstrated significant decreases in antibody titers and normalization of thyroid functions after 2–4 y of treatment (46). Furthermore, magnesium and vitamin A together may enhance thyroid function and decrease inflammation (47).

Vitamin B_{12}

Vitamin B_{12}, also known as cobalamin, is a water-soluble vitamin detected in foods such as dairy products, eggs and red meats (48). Vitamin B_{12} deficiency is a common occurrence in autoimmune thyroid disease. Several studies have reported rates of vitamin B_{12} deficiency ranging 6.3–55.5% in individuals with autoimmune thyroid disorders. Autoimmune thyroid disorders are often associated with other autoimmune conditions such as pernicious anemia and atrophic gastritis, which can lead to impaired absorption of vitamin B_{12} (49). Therefore, it is essential to screen these patients for vitamin B_{12} deficiency as it can contribute to psychological symptoms (50). Studies have reported associations between vitamin B_{12} and vitamin D deficiencies and autoimmune hypothyroidism. There is evidence of reverse associations between the serum levels of these vitamins and anti-thyroid peroxidase (TPO) antibodies in patients (51). However, it is noteworthy that although vitamin B_{12} is vital for hearing ability, low serum concentrations of vitamin B_{12} do not appear to be directly linked to hearing loss in patients with euthyroid HT (52).

Zinc

Zinc (Zn) plays a critical role in metabolism of thyroid hormones and regulation of thyroid functions. It is involved in controlling activity of deiodinase enzymes, producing TSH and thyrotropin-releasing hormone (TRH) and functioning transcription factors essential for thyroid hormone production (53). Zinc concentrations have been detected to affect serum levels of T3, T4 and TSH. While studies have reported no significant differences in serum zinc concentrations between individuals with HT and healthy ones (54, 55), there is evidence that a decrease in serum zinc levels is associated with an increase in the titer of anti-thyroid antibodies (56), suggesting that lower zinc levels may be linked to persistent inflammation of the thyroid gland or inadequate dietary intake of iodine in individuals with HT (56). Therefore, alterations in the body zinc status can alter thyroid function and immune system regulation through direct and indirect mechanisms.

Iron

Iron is a critical nutrient in various physiological processes, including energy metabolism and oxygen transport to body tissues. The dietary requirements for iron depend on factors such as basal iron loss, menstrual iron loss in females, fetal requirements during pregnancy, increased needs during growth years and iron storage (57). Iron plays a critical role in immune function, as it supports growth and differentiation of immune cells and affects cell-mediated immune pathways and cytokine activities (58). Studies have suggested that iron deficiency can negatively affect thyroid function by interfering with oxygen transport or disrupting activity of thyroid peroxidase, an enzyme involved in thyroid hormone synthesis (59). Additionally, there is a known association between HT and gastric disorders with 10–40% of patients with HT having gastric disorders and approximately 40% of autoimmune gastritis patients having HT. This co-occurrence is referred to as thyro-gastric syndrome, which often presents as iron deficiency. Sometimes, iron deficiency anemia that is unresponsive to oral iron therapy may be attributed to chronic atrophic gastritis in patients with HT (60). Since iron-deficiency anemia can inhibit thyroid function, iron supplementation for treatment of anemia can improve thyroid functions (13).

Selenium

Selenium (Se) is a trace mineral that plays an important role in various aspects of human health , including activation of iodothyronine deiodinases which convert the inactive thyroid hormone T4 into the active form of T3 (61, 62). Selenium affects inflammation and immune functions, primarily through its role in producing selenoproteins. Adequate levels of selenium are essential for modulating the immune system, regulating immune responses and controlling persistent inflammation. Deficiency of selenium has been shown to include detrimental effects on immune cell activity, differentiation and proliferation (63). Studies have reported that selenium supplementation in patients with autoimmune thyroid disease can decrease serum levels of T3, T4 and thyroid peroxidase antibodies (TPOAb). Therefore, selenium supplementation may exert beneficial effects on thyroid gland functions and help decrease autoimmune activity in thyroid diseases (64, 65).

Vitamin D

Suboptimal vitamin D status is addressed a serious global health problem (66). Vitamin D is known for its role in cell proliferation inhibition and cellular differentiation (67). It is essential for regenerating epithelial barriers and maturation of immune cells. Various immune cells, including lymphocytes,

[DOI: 10.61882/nfsr.12.3.1]

neutrophils, monocytes and dendritic cells, express the vitamin D receptor (VDR) and can be directly targeted by the active form of vitamin D, 1,25-dihydroxyvitamin D3 [1,25(OH)2D3]. These immune cells can activate circulating 25-hydroxyvitamin D3 [25(OH)D3] through the enzymatic action of CYP27B1 (68). Vitamin D provides its biological effects by binding to the VDR and activating VDR responsive genes in target tissues. Previous studies have shown an association between autoimmune thyroid disease and variations in the VDR gene (69). Studies have reported lower vitamin D levels in patients with HT, compared to healthy individuals, using meta-analysis (70). Furthermore, supplementation with vitamin D has been shown to decrease titers of thyroglobulin antibodies (TGAb) and TPOAb in patients with HT when duration of supplementation exceeds 3 m. A recent integrative review reported that vitamin D supplementation in adult individuals with HT resulted in decreases in TSH and anti-TPO (71). Hence, vitamin D supplementation may be beneficial in modulating the autoimmune response in HT (72).

Iodine

Iodine is a trace element in soil and water and is absorbed in various chemical forms and almost entirely removed from the circulatory system by the thyroid and kidneys (73). Iodine is a primary element in the synthesis of thyroid hormones. Iodide of the blood is taken up by thyroid cells through the sodium/iodide symporter, which uses sodium concentration gradient generated by the Na^+/K^+-ATPase transporter (74). Studies, particularly in pregnant women, have suggested that inadequate iodine intake is a risk factor for thyroid autoimmunity (75). However, excessive iodine levels may increase the risk of autoimmunity in children, based on urinary iodine assessments (76, 77).

Dietary pattern

Dietary habits can significantly affect risks of inflammatory and immune-mediated disorders such as autoimmune diseases through various mechanisms. However, limited studies have investigated associations between dietary patterns and HT. Studies have reported that patients with HT have higher intakes of animal proteins and lower adherences to the Mediterranean diets than those healthy individuals have. In contrast, healthy people consume further plant-based foods. The nutritional pattern of individuals with HT is often similar to Western diets, characterized by higher consumptions of saturated fats, particularly from animal sources, refined carbohydrates and simple sugars and lower intakes of fibers and antioxidants (78, 79). Previous studies have suggested that consumption of animal-based saturated fats and butter is associated with higher levels of thyroid peroxidase antibodies (TPO-Ab) and/or thyroglobulin antibodies (Tg-Ab). In contrast, consumption of vegetables, fruits, nuts and oatmeal is inversely associated to TPO-Ab and/or Tg-Ab (80). The popularity of vegan and vegetarian diets has increased in recent years. There is currently a limited strong evidence to support benefits of these dietary patterns, specifically in patients with HT. One concern about following vegetarian diets, especially vegan diets, in such patients includes potentials for inadequate iodine intake and consequences of iodine deficiency (81). A recent descriptive cross-sectional study indicated that the quality of diets was not associated with the quality of life or nutritional status of patients with HT (82).

Gluten-free diets

Gluten refers to certain cereal prolamins in rye, wheat, spelt, Kamut and barley. Gluten is a significant component of wheat-based staple foods such as pastas, breads and cereals. (83). A meta-analysis has shown a higher prevalence of thyroid disease in patients with celiac disease. Another meta-analysis detected that the prevalence of celiac disease in individuals with autoimmune thyroid disease (AITD) was nearly 1.6%. Studies have demonstrated assessed thyroid-linked antibodies in patients with celiac disease (84). Gluten-free diets in AITD patients may decrease TSH levels, increase T4 levels and improve levothyroxine absorption by promoting gut health. However, there is insufficient evidence to recommend gluten-free diets for HT without celiac disease (85).

Discussion

As highlighted in this review, several nutrients such as dietary proteins, omega-3 fatty acids, vitamins A, D, E, B12 and C, iron, zinc and selenium play critical roles in regulating immune system functions. These nutrients have been shown to enhance the body ability to prevent the development of autoimmune diseases. Presently, there is no conclusive evidence to support the potential effectiveness of vitamin and mineral supplementations as preventive tools against HT in healthy individuals. Nonetheless, it is generally recommended for most healthy individuals to ensure sufficient intake of nutrients through a balanced diet consisting of diverse food sources. In early stages of the disease, a therapeutic diet consisting of adequate quantities of high-quality proteins is often recommended. However specific protein intake needs for individuals with HT are unclear due to insufficient evidence, it is important to prioritize strategies preventing protein malnutrition to support optimal thyroid functions (13).

Several studies have suggested that vitamin C, vitamin E, selenium and zinc possess antioxidant characteristics and can help decrease oxidative stress and inflammation (86–90). The current findings indicate a gradual increase

[DOI: 10.61882/nfsr.12.3.1]

in oxidative stress levels as subclinical hypothyroidism and overt hypothyroidism develop in patients with HT. Randomized controlled trials are needed to investigate the causality between oxidative stress and hypothyroidism to investigate effects of antioxidant therapy on the onset of overt hypothyroidism and its associated complications such as increases in total cholesterol levels. These trials should involve individuals with euthyroidism and/or subclinical hypothyroidism who have HT (91, 92).

There is limited evidence on the specific effects of dietary patterns such as the Mediterranean diet, Western pattern and vegetarian diet on HT. While there are several studies on gluten-free diet, its benefits are still controversial. Studies have reported positive outcomes of the gluten-free diet in patients with gastrointestinal disorders. However, it is important to screen for gluten and wheat hypersensitivities with or without celiac disease in individuals with HT (93, 94).

Vitamin D is critical in regulating immune system responses (95). Numerous studies have highlighted the significance of this nutrient in the incidence and development of thyroid disorders and autoimmune diseases (96), including HT (72, 97). For individuals who do not receive sufficient nutrients from their diets or have low serum levels of vitamin D, supplementation according to the recommended dietary allowance (RDA) is generally recommended. This is particularly relevant for individuals who may be at risk of inadequate intakes and vitamin D deficiencies (98). Certain nutrients, including selenium, iron, iodine and magnesium, are essential for the production of thyroid hormones. Numerous studies have indicated the potential benefits of selenium supplementation in autoimmune thyroid diseases, leading to decreased serum levels of T3, T4 and TPOAb. These findings suggest that selenium supplementation may include positive effects on individuals with autoimmune thyroid disease (64, 99).

Conclusion

This study aimed to systematically analyze the role of nutrients in supporting the immune system and their potential for preventing and treating HT. Findings of this investigation highlight the significant role of certain nutrients in regulating immune system functions. Additionally, supplementation with appropriate nutrients can potentially improve the symptoms and enhance the quality of life in individuals with HT. However, it is important to acknowledge the study limitations such as the limited number of randomized controlled trials carried out on the effects of nutrients and dietary patterns in patients with HT, as well as the insufficient evidence regarding effects of dietary supplementation for prevention in healthy people. Further studies are warranted to validate these findings and provide recommendations for individuals with HT.

Declarations

Ethical Approval: Not applicable.

Consent to participate: Not applicable.

Competing interests: The authors declare no competing of interests.

Funding: Not applicable.

Acknowledgments: Not applicable.

Availability of data and materials: Not applicable.

Consent for publication: Not applicable.

Authors' contributions

FB and SK designed the study; FB and GS developed the search strategy; FB extracted data; FB, SR, MM and MSK wrote the draft. All authors read and approved the final manuscript.

Financial disclosure

The authors declared no financial interest.

References

1. Bianco A. Hypothyrodisis. Encycl Clin Neuropsychol. 2011;390((10101)):1290.
2. Weetman A. An update on the pathogenesis of Hashimoto's thyroiditis. Journal of Endocrinological Investigation. 2021;44(5):883-90.
3. Strikić Đula I, Pleić N, Babić Leko M, Gunjača I, Torlak V, Brdar D, et al. Epidemiology of Hypothyroidism, Hyperthyroidism and Positive Thyroid Antibodies in the Croatian Population. Biology. 2022;11(3):394.
4. Ralli M, Angeletti D, Fiore M, D'Aguanno V, Lambiase A, Artico M, et al. Hashimoto's thyroiditis: an update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies and potential malignant transformation. Autoimmunity Reviews. 2020;19(10):102649.
5. Childs CE, Calder PC, Miles EA. Diet and immune function. MDPI; 2019. p. 1933.
6. Mazzucca CB, Raineri D, Cappellano G, Chiocchetti A. How to tackle the relationship between autoimmune diseases and diet: Well begun is half-done. Nutrients. 2021;13(11):3956.
7. Kaličanin D, Brčić L, Ljubetić K, Barić A, Gračan S, Brekalo M, et al. Differences in food consumption between patients with Hashimoto's thyroiditis and healthy individuals. Scientific reports. 2020;10(1):1-10.
8. Luo J, Hendryx M, Dinh P, He K. Association of iodine and iron with thyroid function. Biological trace element research. 2017;179(1):38-44.
9. Gorini F, Sabatino L, Pingitore A, Vassalle C. Selenium: an element of life essential for thyroid function. Molecules. 2021;26(23):7084.
10. Krishnamurthy HK, Reddy S, Jayaraman V, Krishna K, Song Q, Rajasekaran KE, et al. Effect of Micronutrients on Thyroid Parameters. Journal of thyroid research. 2021;2021.
11. Richter M, Baerlocher K, Bauer JM, Elmadfa I, Heseker H, Leschik-Bonnet E, et al. Revised reference values for the intake of protein. Annals of Nutrition and Metabolism. 2019;74(3):242-50.
12. Pałkowska-Goździk E, Lachowicz K, Rosołowska-Huszcz D. Effects of dietary protein on thyroid axis activity. Nutrients. 2017;10(1):5.

[DOI: 10.61882/nfsr.12.3.1]

13. Ihnatowicz P, Drywień M, Wątor P, Wojsiat J. The importance of nutritional factors and dietary management of Hashimoto's thyroiditis. Annals of agricultural and environmental medicine. 2020;27(2).

14. Doseděl M, Jirkovský E, Macáková K, Krčmová LK, Javorská L, Pourová J, et al. Vitamin C—sources, physiological role, kinetics, deficiency, use, toxicity and determination. Nutrients. 2021;13(2):615.

15. Taddei S, Caraccio N, Virdis A, Dardano A, Versari D, Ghiadoni L, et al. Low-grade systemic inflammation causes endothelial dysfunction in patients with Hashimoto's thyroiditis. The Journal of Clinical Endocrinology & Metabolism. 2006;91(12):5076-82.

16. Chen L, Mao Y, Chen G. Association between total vitamin C intake and hypothyroidism among Hashimoto thyroiditis: National Health and Nutrition Examination Survey, 2007–2012. British Journal of Nutrition. 2024;132(12):1575-83.

17. Asbaghi O, Sadeghian M, Nazarian B, Sarreshtedari M, Mozaffari-Khosravi H, Maleki V, et al. The effect of vitamin E supplementation on selected inflammatory biomarkers in adults: a systematic review and meta-analysis of randomized clinical trials. Scientific reports. 2020;10(1):17234.

18. Mishra P, Paital B, Jena S, Swain SS, Kumar S, Yadav MK, et al. Possible activation of NRF2 by Vitamin E/Curcumin against altered thyroid hormone induced oxidative stress via NFκB/AKT/mTOR/KEAP1 signalling in rat heart. Scientific reports. 2019;9(1):1-16.

19. Yu J, Shan Z, Chong W, Mao J, Geng Y, Zhang C, et al. Vitamin E ameliorates iodine-induced cytotoxicity in thyroid. Journal of endocrinology. 2011;209(3):299.

20. Napolitano G, Fasciolo G, Di Meo S, Venditti P. Vitamin E supplementation and mitochondria in experimental and functional hyperthyroidism: a mini-review. Nutrients. 2019;11(12):2900.

21. Zivkovic AM, Telis N, German JB, Hammock BD. Dietary omega-3 fatty acids aid in the modulation of inflammation and metabolic health. California agriculture. 2011;65(3):106.

22. Byelashov OA, Sinclair AJ, Kaur G. Dietary sources, current intakes and nutritional role of omega-3 docosapentaenoic acid. Lipid Technology. 2015;27(4):79-82.

23. Swanson D, Block R, Mousa SA. Omega-3 fatty acids EPA and DHA: health benefits throughout life. Advances in nutrition. 2012;3(1):1-7.

24. Gutiérrez S, Svahn SL, Johansson ME. Effects of omega-3 fatty acids on immune cells. International journal of molecular sciences. 2019;20(20):5028.

25. Akbar U, Yang M, Kurian D, Mohan C. Omega-3 fatty acids in rheumatic diseases: a critical review. JCR: Journal of Clinical Rheumatology. 2017;23(6):330-9.

26. Li K, Huang T, Zheng J, Wu K, Li D. Effect of marine-derived n-3 polyunsaturated fatty acids on C-reactive protein, interleukin 6 and tumor necrosis factor α: a meta-analysis. PloS one. 2014;9(2):e88103.

27. Allen MJ, Fan Y-Y, Monk JM, Hou TY, Barhoumi R, McMurray DN, et al. n–3 PUFAs reduce T-helper 17 cell differentiation by decreasing responsiveness to interleukin-6 in isolated mouse splenic CD4+ T cells. The Journal of nutrition. 2014;144(8):1306-13.

28. Hahn J, Cook NR, Alexander EK, Friedman S, Walter J, Bubes V, et al. Vitamin D and marine omega 3 fatty acid supplementation and incident autoimmune disease: VITAL randomized controlled trial. bmj. 2022;376.

29. Chhetri DR. Myo-inositol and its derivatives: their emerging role in the treatment of human diseases. Frontiers in pharmacology. 2019;10:1172.

30. Pace C, Tumino D, Russo M, Le Moli R, Naselli A, Borzì G, et al. Role of selenium and myo-inositol supplementation on autoimmune thyroiditis progression. Endocrine Journal. 2020:EJ20-0062.

31. Nordio M, Basciani S. Treatment with myo-inositol and selenium ensures euthyroidism in patients with autoimmune thyroiditis. International Journal of Endocrinology. 2017;2017.

32. Ferrari SM, Elia G, Ragusa F, Paparo SR, Caruso C, Benvenga S, et al. The protective effect of myo-inositol on human thyrocytes. Reviews in Endocrine and Metabolic Disorders. 2018;19(4):355-62.

33. Krysiak R, Kowalcze K, Okopień B. The impact of vitamin D on thyroid autoimmunity and hypothalamic–pituitary–thyroid axis activity in myo-inositol-treated and myo-inositol-naïve women with autoimmune thyroiditis: A pilot study. Journal of Clinical Pharmacy and Therapeutics. 2022.

34. Huang Z, Liu Y, Qi G, Brand D, Zheng SG. Role of vitamin A in the immune system. Journal of clinical medicine. 2018;7(9):258.

35. Gholizadeh M, Basafa Roodi P, Abaj F, Shab-Bidar S, Saedisomeolia A, Asbaghi O, et al. Influence of Vitamin A supplementation on inflammatory biomarkers in adults: a systematic review and meta-analysis of randomized clinical trials. Scientific Reports. 2022;12(1):21384.

36. Harirchian MH, Mohammadpour Z, Fatehi F, Firoozeh N, Bitarafan S. A systematic review and meta-analysis of randomized controlled trials to evaluating the trend of cytokines to vitamin A supplementation in autoimmune diseases. Clinical Nutrition. 2019;38(5):2038-44.

37. Farhangi MA, Keshavarz SA, Eshraghian M, Ostadrahimi A, Saboor-Yaraghi AA. The effect of vitamin A supplementation on thyroid function in premenopausal women. Journal of the American College of Nutrition. 2012;31(4):268-74.

38. Homma H, Watanabe M, Inoue N, Isono M, Hidaka Y, Iwatani Y. Polymorphisms in Vitamin A-Related Genes and Their Functions in Autoimmune Thyroid Disease. Thyroid. 2021;31(11):1749-56.

39. Razzaque MS. Magnesium: are we consuming enough? Nutrients. 2018;10(12):1863.

40. Kolanu BR, Vadakedath S, Boddula V, Kandi V. Activities of Serum Magnesium and Thyroid Hormones in Pre-, Peri- and Post-menopausal Women. Cureus. 2020;12(1).

41. Stuefer S, Moncayo H, Moncayo R. The role of magnesium and thyroid function in early pregnancy after in-vitro fertilization (IVF): New aspects in endocrine physiology. BBA clinical. 2015;3:196-204.

42. Ott J, Promberger R, Kober F, Neuhold N, Tea M, Huber JC, et al. Hashimoto's thyroiditis affects symptom load and quality of life unrelated to hypothyroidism: a prospective case–control study in women undergoing thyroidectomy for benign goiter. Thyroid. 2011;21(2):161-7.

[DOI: 10.61882/nfsr.12.3.1]

43. Khan SZA, Lungba RM, Ajibawo-Aganbi U, Veliginti S, Bastidas MVP, Saleem S, et al. Minerals: An Untapped Remedy for Autoimmune Hypothyroidism? Cureus. 2020;12(10).
44. Mincer DL, Jialal I. Hashimoto thyroiditis. 2017.
45. Aslam B, Wang W, Arshad MI, Khurshid M, Muzammil S, Rasool MH, et al. Antibiotic resistance: a rundown of a global crisis. Infection and drug resistance. 2018;11:1645-58.
46. Moncayo R, Moncayo H. Proof of concept of the WOMED model of benign thyroid disease: restitution of thyroid morphology after correction of physical and psychological stressors and magnesium supplementation. BBA clinical. 2015;3:113-22.
47. Chaberska I, Feruś A, Lipska J, Turek M, Wojciechowska K, Piątkowska K, et al. The effect of multiple nutritional factors on hypothyroidism-a systemic review. Quality in Sport. 2024;19:53782-.
48. KADİROĞLU AK. General Internal Medicine II: Akademisyen Kitabevi; 2022.
49. Collins AB, Pawlak R. Prevalence of vitamin B-12 deficiency among patients with thyroid dysfunction. Asia Pacific journal of clinical nutrition. 2016;25(2):221-6.
50. Aon M, Taha S, Mahfouz K, Ibrahim MM, Aoun AH. Vitamin B12 (Cobalamin) Deficiency in Overt and Subclinical Primary Hypothyroidism. Clinical Medicine Insights: Endocrinology and Diabetes. 2022;15:11795514221086634.
51. Aktaş HŞ. Vitamin B12 and vitamin D levels in patients with autoimmune hypothyroidism and their correlation with anti-thyroid peroxidase antibodies. Medical Principles and Practice. 2020;29(4):364-70.
52. Arduc A, Isık S, Allusoglu S, Iriz A, Dogan BA, Gocer C, et al. Evaluation of hearing functions in patients with euthyroid Hashimoto's thyroiditis. Endocrine. 2015;50(3):708-14.
53. Severo JS, Morais JBS, de Freitas TEC andrade ALP, Feitosa MM, Fontenelle LC, et al. The role of zinc in thyroid hormones metabolism. Int J Vitam Nutr Res. 2019;89(1-2):80-8.
54. Sanna A, Firinu D, Zavattari P, Valera P. Zinc status and autoimmunity: a systematic review and meta-analysis. Nutrients. 2018;10(1):68.
55. Szczepanik J, Podgórski T, Domaszewska K. The Level of Zinc, Copper and Antioxidant Status in the Blood Serum of Women with Hashimoto's Thyroiditis. International Journal of Environmental Research and Public Health. 2021;18(15):7805.
56. Borawska M, Markiewicz-Żukowska R, Dziemianowicz M, Socha K, Soroczyńska J. Wpływ nawyków żywieniowych i palenia papierosów na stężenie cynku w surowicy krwi kobiet z chorobą Hashimoto. Bromat Chem Toksykol. 2012;3:759-65.
57. Prentice AM, Mendoza YA, Pereira D, Cerami C, Wegmuller R, Constable A, et al. Dietary strategies for improving iron status: balancing safety and efficacy. Nutrition reviews. 2017;75(1):49-60.
58. Hassan TH, Badr MA, Karam NA, Zkaria M, El Saadany HF, Rahman DMA, et al. Impact of iron deficiency anemia on the function of the immune system in children. Medicine. 2016;95(47):e5395.
59. Luo J, Wang X, Yuan L, Guo L. Iron deficiency, a risk factor of thyroid disorders in reproductive-age and pregnant women: a systematic review and meta-analysis. Frontiers in endocrinology. 2021;12:629831.
60. Cellini M, Santaguida MG, Virili C, Capriello S, Brusca N, Gargano L, et al. Hashimoto's thyroiditis and autoimmune gastritis. Frontiers in endocrinology. 2017;8:92.
61. Kieliszek M. Selenium–fascinating microelement, properties and sources in food. Molecules. 2019;24(7):1298.
62. Shreenath AP, Ameer MA, Dooley J. Selenium deficiency. 2018.
63. Huang Z, Rose AH, Hoffmann PR. The role of selenium in inflammation and immunity: from molecular mechanisms to therapeutic opportunities. Antioxidants & redox signaling. 2012;16(7):705-43.
64. Zuo Y, Li Y, Gu X, Lei Z. The correlation between selenium levels and autoimmune thyroid disease: A systematic review and meta-analysis. Ann Palliat Med. 2021;10:4398-408.
65. Fan Y, Xu S, Zhang H, Cao W, Wang K, Chen G, et al. Selenium supplementation for autoimmune thyroiditis: a systematic review and meta-analysis. International journal of endocrinology. 2014;2014.
66. Rezaiian F, Davoodi SH, Nikooyeh B, Ehsani AH, Kalayi A, Shariatzadeh N, et al. Metabolic Syndrome and Its Components are Linked with Increased Risk of Non-Melanoma Skin Cancers in Iranian Subjects: A Case-Control Study. Nutr Cancer. 2022;74(7):2451-9.
67. Setayesh L, Casazza K, Moradi N, Mehranfar S, Yarizadeh H, Amini A, et al. Association of vitamin D-binding protein and vitamin D3 with insulin and homeostatic model assessment (HOMA-IR) in overweight and obese females. BMC Research Notes. 2021;14(1):1-7.
68. Zmijewski MA. Vitamin D and human health. MDPI; 2019. p. 145.
69. Talaei A, Ghorbani F, Asemi Z. The effects of Vitamin D supplementation on thyroid function in hypothyroid patients: A randomized, double-blind, placebo-controlled trial. Indian journal of endocrinology and metabolism. 2018;22(5):584.
70. Štefanić M, Tokić S. Serum 25-hydoxyvitamin D concentrations in relation to Hashimoto's thyroiditis: a systematic review, meta-analysis and meta-regression of observational studies. European journal of nutrition. 2020;59(3):859-72.
71. de Souza AF, da Silva Mazzeti CM, Rafacho BPM. Effect of vitamin D supplementation in adults with Hashimoto's Thyroiditis: an integrative review. Nutrire. 2025;50(1):1-10.
72. Zhang J, Chen Y, Li H, Li H. Effects of vitamin D on thyroid autoimmunity markers in Hashimoto's thyroiditis: systematic review and meta-analysis. Journal of International Medical Research. 2021;49(12):03000605211060675.
73. Sun W. Iodine-131 and thyroid function. Environmental health perspectives. 2014;122(2):A40.
74. Sorrenti S, Baldini E, Pironi D, Lauro A, D'Orazi V, Tartaglia F, et al. Iodine: its role in thyroid hormone biosynthesis and beyond. Nutrients. 2021;13(12):4469.
75. Wang Z, Xing M, Zhu W, Mao G, Mo Z, Wang Y, et al. Iodine deficiency in Zhejiang pregnant women in the context of universal salt iodization programme. Scientific Reports. 2018;8(1):1-7.

[DOI: 10.61882/nfsr.12.3.1]

76. Palaniappan S, Shanmughavelu L, Prasad HK, Subramaniam S, Krishnamoorthy N, Lakkappa L. Improving iodine nutritional status and increasing prevalence of autoimmune thyroiditis in children. Indian Journal of Endocrinology and Metabolism. 2017;21(1):85.

77. Doğan M, Acikgoz E, Acikgoz M, Cesur Y, Ariyuca S, Bektas MS. The frequency of Hashimoto thyroiditis in children and the relationship between urinary iodine level and Hashimoto thyroiditis. 2011.

78. Ruggeri RM, Giovinazzo S, Barbalace MC, Cristani M, Alibrandi A, Vicchio TM, et al. Influence of dietary habits on oxidative stress markers in Hashimoto's thyroiditis. Thyroid. 2021;31(1):96-105.

79. Ihnatowicz P, Wątor P, Gębski J, Frąckiewicz J, Drywień ME. Are Nutritional Patterns among Polish Hashimoto Thyroiditis Patients Differentiated Internally and Related to Ailments and Other Diseases? Nutrients. 2021;13(11):3675.

80. Matana A, Torlak V, Brdar D, Popović M, Lozić B, Barbalić M, et al. Dietary factors associated with plasma thyroid peroxidase and thyroglobulin antibodies. Nutrients. 2017;9(11):1186.

81. Eveleigh ER, Coneyworth LJ, Avery A, Welham SJM. Vegans, Vegetarians and Omnivores: How Does Dietary Choice Influence Iodine Intake? A Systematic Review. Nutrients. 2020;12(6).

82. Osowiecka K, Skrypnik D, Myszkowska-Ryciak J. No Association Between Diet Quality, Nutritional Status and Quality of Life in Women with Hashimoto's Thyroiditis—A Cross-Sectional Study. Nutrients. 2025;17(6):1015.

83. Storz MA, Ronco AL, Lombardo M. Dietary Acid Load in Gluten-Free Diets: Results from a Cross-Sectional Study. Nutrients. 2022;14(15).

84. Sun X, Lu L, Yang R, Li Y, Shan L, Wang Y. Increased incidence of thyroid disease in patients with celiac disease: a systematic review and meta-analysis. PloS one. 2016;11(12):e0168708.

85. Roy A, Laszkowska M, Sundström J, Lebwohl B, Green PH, Kämpe O, et al. Prevalence of celiac disease in patients with autoimmune thyroid disease: a meta-analysis. Thyroid. 2016;26(7):880-90.

86. Kawashima A, Sekizawa A, Koide K, Hasegawa J, Satoh K, Arakaki T, et al. Vitamin C induces the reduction of oxidative stress and paradoxically stimulates the apoptotic gene expression in extravillous trophoblasts derived from first-trimester tissue. Reproductive Sciences. 2015;22(7):783-90.

87. Mohammadi H, Talebi S, Ghavami A, Rafiei M, Sharifi S, Faghihimani Z, et al. Effects of zinc supplementation on inflammatory biomarkers and oxidative stress in adults: A systematic review and meta-analysis of randomized controlled trials. Journal of Trace Elements in Medicine and Biology. 2021;68:126857.

88. Hasani M, Djalalinia S, Khazdooz M, Asayesh H, Zarei M, Gorabi AM, et al. Effect of selenium supplementation on antioxidant markers: a systematic review and meta-analysis of randomized controlled trials. Hormones. 2019;18(4):451-62.

89. Venditti P, Di Stefano L, Di Meo S. Vitamin E management of oxidative damage-linked dysfunctions of hyperthyroid tissues. Cellular and Molecular Life Sciences. 2013;70(17):3125-44.

90. Rasaei N, Asbaghi O, Samadi M, Setayesh L, Bagheri R, Gholami F, et al. Effect of green tea supplementation on antioxidant status in adults: A systematic review and meta-analysis of randomized clinical trials. Antioxidants. 2021;10(11):1731.

91. Kochman J, Jakubczyk K, Bargiel P, Janda-Milczarek K. The influence of oxidative stress on thyroid diseases. Antioxidants. 2021;10(9):1442.

92. Rostami R, Aghasi M, Mohammadi A, Nourooz-Zadeh J. Enhanced oxidative stress in Hashimoto's thyroiditis: inter-relationships to biomarkers of thyroid function. Clinical biochemistry. 2013;46(4-5):308-12.

93. Carroccio A, D'Alcamo A, Cavataio F, Soresi M, Seidita A, Sciumè C, et al. High proportions of people with nonceliac wheat sensitivity have autoimmune disease or antinuclear antibodies. Gastroenterology. 2015;149(3):596-603. e1.

94. Losurdo G, Principi M, Iannone A, Giangaspero A, Piscitelli D, Ierardi E, et al. Predictivity of autoimmune stigmata for gluten sensitivity in subjects with microscopic enteritis: A retrospective study. Nutrients. 2018;10(12):2001.

95. Setayesh L, Amini A, Bagheri R, Moradi N, Yarizadeh H, Asbaghi O, et al. Elevated plasma concentrations of vitamin D-binding protein are associated with lower high-density lipoprotein and higher fat mass index in overweight and obese women. Nutrients. 2021;13(9):3223.

96. Rezaiian F, Davoodi SH, Nikooyeh B, Ehsani AH, Kalayi A, Shariatzadeh N, et al. Sun Exposure Makes no Discrimination based on Vitamin D Status and VDR-Foki Polymorphisms for Non-Melanoma Skin Cancers Risk in Iranian Subjects: A Case-Control Study. Asian Pac J Cancer Prev. 2022;23(6):1927-33.

97. Nettore IC, Albano L, Ungaro P, Colao A, Macchia PE. Sunshine vitamin and thyroid. Reviews in Endocrine and Metabolic Disorders. 2017;18(3):347-54.

98. Taheriniya S, Arab A, Hadi A, Fadel A, Askari G. Vitamin D and thyroid disorders: A systematic review and meta-analysis of observational studies. BMC Endocrine Disorders. 2021;21(1):1-12.

99. Wichman J, Winther KH, Bonnema SJ, Hegedüs L. Selenium supplementation significantly reduces thyroid autoantibody levels in patients with chronic autoimmune thyroiditis: a systematic review and meta-analysis. Thyroid. 2016;26(12):1681-92.

[DOI: 10.61882/nfsr.12.3.1]