THE THYROID: CONSIDERATIONS FOR NUTRITIONAL THERAPY AND PERSONALISED LIFESTYLE SUPPORT

Authors: Chloe Steele[1], Ana-Paula Agrela[2], Gail Brady[3], Nicky Ester[4], Anna Papoutsa Shue[5], Jessica Rigutto[6]*

Abstract

The thyroid gland has major physiological roles in growth, development and metabolism, from conception to older age. Thyroid function is associated with some of the major causes of morbidity and mortality globally, and dysfunction of the thyroid gland can impact daily quality of life through its effects on energy, muscle strength, body weight, bowel frequency, heart rate, mental health, and temperature regulation. Such symptoms may be frequently seen in individuals presenting with suboptimal thyroid function in nutritional therapy practice. Various nutrients such as iodine, iron, selenium, vitamin A, zinc, vitamin D, and magnesium are influential to thyroid function and personalised lifestyle choices adapted to the individual may help provide additional support. The aim of this review is to describe the research surrounding the relationship of these factors to thyroid function and thereby support evidence-based therapeutic decision-making in clinic.

Introduction

The thyroid is an important endocrine gland situated at the front of the throat. Its main function is to produce hormones required for growth, development and metabolism, namely triiodothyronine (T3), and tetraiodothyronine (thyroxine, T4). In response to existing circulating concentrations of T3 and T4, hormones from the hypothalamus and pituitary gland control the amount of these hormones produced by the thyroid (1). This feedback loop is known as the hypothalamus-pituitary-thyroid axis (HPT). A poorly functioning thyroid gland or problems HPT axis signalling can be involved in the development of many diseases and are associated with three of the top ten global causes of death: heart disease, Alzheimer's disease and diabetes (2–4). Disrupted lipid metabolism, metabolic syndrome, and hyperglycaemia may all be involved in the pathogenesis (5,6). Practitioners should therefore consider thyroid function when seeing individuals with these conditions, as well as individuals who have classic thyroidal symptoms such as tiredness, weakness, weight gain or difficulty losing weight, muscle aches, constipation, slow heart rate, anxiety, and sensitivity to cold.

Lifestyle factors such as diet, sleep, exercise, stress, and environmental toxins may affect thyroid function (7). Practitioners should consider the role of nutrient deficiencies and excesses in thyroid health. It has long been recognised that several minerals and vitamins are essential to thyroid function and hormone production, including iodine, selenium (Se), iron (Fe), and vitamin A (VA), as well as the enzyme co-factors Zn and magnesium (Mg) (8). As a result, individuals with nutrient deficiencies may be at risk of poor thyroid health.

Whilst it is often assumed that nutrient deficiencies are uniquely associated with low- and middle-income countries, it is interesting to note that deficiencies are emerging amongst high income countries too. For example, in a 2021 review of data from the United Kingdom, pregnant women over the period 1991 to 2014 showed that in no survey year were iodine intakes sufficient to cover the additional needs of pregnancy (9).

Undiagnosed thyroid disorders could possibly occur in as much as 4-7% of the UK and European population and metabolic disorders are increasing (10–13). Nutrient imbalances and lifestyle choices may be contributing to modifiable risk factors influencing thyroid disorders.

This narrative review therefore seeks to provide a useful overview of the literature and support decision-making by nutrition practitioners in their recommendations for this growing number of individuals.

1. What is the role of iodine in thyroid health?

The thyroid gland, which stores 70-80% of the body's iodine (1), uses it to synthesise the thyroid hormones T4 and a smaller amount of T3, with most T3, as the active form, being produced through the peripheral conversion of T4 at the site of action. Thyroid hormone (TH) is crucial for cellular energy production, oxygen consumption, neural development and reproductive function (14). Iodine deficiency is linked to a number of disorders including goitre, hypothyroidism, mental retardation historically known as cretinism, cognitive impairment, and reproductive issues (1); while iodine excess may contribute to goitre, development of iodine-induced hyperthyroidism, overt hypothyroidism and most of the other iodine deficiency disorders through its inhibition of TH production (15), and, rarely, acute toxicity (1,16). Both deficiency and excess may contribute to thyroid autoimmunity and influence thyroid cancer risk (17-20).

Iodine deficiency may affect fertility (19) and is linked to thyroid disorders in children, including goitre and hypothyroidism, as well as increased risk of infant mortality (20). Even in adults, insufficient iodine intake can cause reduced T4 and T3 synthesis and elevated thyroid-stimulating hormone (TSH) (hypothyroidism). This can result in goitre as the thyroid tissue enlarges to compensate for low hormone concentrations; an adaptation aimed at maximising iodine uptake and hormone production (21).

Temporary exposure to excess iodide (the ionic form of iodine, I-) inhibits TH synthesis through a phenomenon known as the Wolff-Chaikoff effect. This acts by downregulating the sodium/iodide symporter when plasma iodide exceeds a critical threshold, thereby protecting against hormone overproduction (22).

As circulating iodide returns to normal, "escape" from the Wolff-Chaikoff effect occurs and hormone production is resumed. However, failure to escape due to impaired symporter function or continued exposure over tolerable limits can result in sustained inhibition of hormone synthesis and subsequent risk of hypothyroidism (22).

Thyroid autoimmune diseases, such as Hashimoto's thyroiditis and Graves' disease (GD), involve the elevation of antibodies to self-proteins in the thyroid gland (23). In the case of Hashimoto's thyroiditis, antibodies to thyroid peroxidase (TPO) and thyroglobulin (Tg) target the body's own TPO enzyme and Tg protein (23). This leads to immune-mediated destruction of thyroid cells, impaired hormone production and hypothyroidism. In the case of GD, thyroid-stimulating immunoglobulins or TSH receptor antibodies (TRAb) bind to and activate the TSH receptor, leading to excessive TH production and hyperthyroidism (23).

Excess iodine intake, especially by people previously exposed to iodine deficiency, has been linked to increased TPO and Tg antibodies, which damage thyroid cells and impair hormone production especially in sensitive populations, such as children and pregnant women (15,24).

In iodine-deficient regions (e.g. deficient soil or lack of seafood consumption), low dietary iodine reduces TH production, prompting the body to lower its TSH baseline to prevent thyroid overstimulation, which over time, could lead to goitre and autonomously-functioning nodules (25). This adaptation increases the risk of iodine-induced hyperthyroidism if iodine intake suddenly rises, even to normal intakes.

According to the World Health Organisation (WHO), populations with a median urinary iodine concentration (UIC) between 100–299 µg/L are considered iodine-sufficient (26). [Note that UIC is a population-only measure and is not suitable for use in individuals (27)]. An Italian nationwide surveillance study found that iodine sufficiency with a median urinary concentration of 124 µg/L, can be achieved through iodised salt use. Compared to earlier surveillance data from 2000–2005, when the median UIC was below 100 µg/L and goitre prevalence was approximately 15%, there was a considerable reduction in both goitre prevalence and iodine

deficiency–related hyperthyroidism. However, pre-existing thyroid autoimmunity was not impacted (28). This finding emphasises that improving iodine intake through dietary interventions, such as replacing regular salt with iodised salt, can measurably reduce the risk of iodine deficiency disorders in populations. However, this strategy may not impact established autoimmune thyroid conditions underscoring the need for personalised nutritional interventions that address both deficiency and the complex management of autoimmunity.

Since iodine is essential for TH synthesis and its deficiency can lead to iodine-related disorders, supplementation may be essential for individuals in iodine-deficient regions or in countries with no iodised salt policy, individuals who exclude food sources of iodine from their diet (such as dairy, seafood and eggs) and those with increased physiological needs, such as pregnant or lactating women. Supplementation should be approached cautiously, as the balance between deficiency and excess is key. The recommended daily intake for adults is 150 µg/L, increasing to 250 µg/L during pregnancy and lactation to cover additional requirements (29).

An experimental balance study by Yang et al. (2020) of a Chinese population of young adults (n=60) found a higher physiological need for additional iodine storage in women than in men (30). It was suggested that this could be due to hormonal and metabolic factors unique to women such as the influence of oestrogens on iodine metabolism and the anticipation of higher TH demands during pregnancy and lactation. This finding underscores the importance of sex-specific dietary recommendations. For women, particularly those of reproductive age, ensuring sufficient iodine intake is essential not only for maintaining optimal thyroid function but also for supporting maternal and foetal health during pregnancy and lactation. Furthermore, iodine sufficiency prior to and during pregnancy has been associated with improved cognitive abilities in children. A randomised control trial (RCT) found that children of n=1220 mothers with pre-pregnancy access to iodised salt scored higher on cognitive tests (effect size d = 0.17; ~4 IQ points; 95% CI 0.0, 1.3; p=0.01) (31). This underscores the importance of assessing iodine intake as part of care for women looking to become pregnant or prenatal care, especially in populations at risk for deficiency.

2. How does Iron impact thyroid function?

Fe deficiency has been associated with thyroid dysfunction, most commonly hypothyroxinaemia and subclinical hypothyroidism (SCH) (32). Fe is a key component of TPO, a haemoprotein and enzyme involved in the biosynthesis of TH. TPO catalyses two essential reactions: the iodination of Tg, a precursor protein for TH, and the coupling of iodotyrosine residues to produce T4 and T3 (8). Fe deficiency may reduce TPO activity (33), as well as negatively impact the effective use of iodine for TH synthesis (34) and the conversion of free T4 (FT4) to free T3 (FT3) (35). In a pooled analysis of individual patient data (n=42,162) it was found that patients with thyroid dysfunction were more likely to have anaemia (36).

The restoration of serum ferritin (SF), a biomarker for stored Fe, has been shown through oral bioavailable Fe supplements in humans, to normalise production of TH (37). However, scientific research is inconclusive, and a combination of physiological mechanisms may be involved (32).

Fe deficiency is most frequently diagnosed in pregnant women and women of reproductive age. Demand for Fe is increased during pregnancy (35) and TH plays a fundamental role in the skeletal and neurological development of the foetus (38). For this reason, numerous scientific studies have been conducted in these populations. A systematic review and meta-analysis of eight cross-sectional studies involving pregnant women or women of reproductive age found that those with Fe deficiency had increased TSH and decreased FT4 (p=0.00001) (35). This was also seen in a study of n=209 pregnant women in their second trimester in China (39).

Similar results were also found in India in a cross-sectional study of women (n=113) in their first trimester of pregnancy (40).

Sixty-eight percent were Fe deficient. In this group, elevated TSH was found in 35% compared to 6% in the non-Fe deficient group. As serum ferritin increased, TSH declined but FT4 did not change. In n=227 Nepalese school children aged 6-12 years thyroid dysfunction was found to be predominant in those with Fe deficiency (44 %) or anaemia (35 %) (41). In a general population, a cross-sectional Spanish study of n=3846 adults without diagnosed thyroid disease, found that as serum ferritin levels decreased, FT4 and FT3 levels also declined (32). There were no changes to TSH. Subgroup analysis found that pre-menopausal females were most likely to be affected. Another systematic review and meta-analysis of 10 cross-sectional studies in adults found lower TSH (p=0.005), FT4 and FT3 (both p=0.000001) in patients with Fe deficiency, though eight out of the 10 included studies were in pregnant women, so results should be interpreted with caution (42). Despite these correlations, most studies are cross sectional and causality cannot be determined. There are also few studies in children or across general populations. More research, in particular RCTs, in these groups are needed to confirm the results as well as the mechanisms involved.

3. How does selenium influence the role of the thyroid gland?

An indicator of the role of Se in thyroid function originated with the early discovery that the thyroid gland contained surprisingly large concentrations of Se and that the body may retain Se in the thyroid even under conditions of deficiency (43).
The role of Se in thyroid health has also been indicated in observational studies showing that a low Se status is associated with poor thyroid function. One systematic review and meta-analysis of 10 case-control and cohort studies with n=2205 individuals showed that individuals with thyroid cancer had lower serum Se than healthy controls (standardised mean difference (SMD) = -1.25; 95% CI -2.07, -0.44; p=0.003) (44). Similar observations have been made in cross-sectional studies from France, Denmark, and China, with low Se levels associated with an increased risk for hypothyroidism, autoimmune thyroiditis (AIT), goitre, enlarged thyroid, thyroid nodules, and GD (45–48).

Many biomarkers are used to test for Se status. Historically glutathione peroxidases (GPx), which require Se for synthesis, have been used to determine Se levels. However, one cohort study (n=51) has shown only a weak relationship between GPx activity and Se levels (r = 0.186, p = 0.021) (49). Selenoprotein P (SeP), which is a transport protein, may be a better biomarker of Se status and has a stronger relationship with serum Se (r = 0.467; p= <0.0001) (49,50). Yet, it has been shown that serum Se may not be indicative of thyroid Se (43). However, given that Se is stored in the thyroid during times of deficiency, it could be assumed that if serum Se is continuously low then thyroid gland Se will eventually follow.

Several factors may affect Se levels. Animal products are good sources of Se and little is found in fruits and vegetables, although plants from the Brassica genus tend to accumulate Se (51). As such, vegetarians may be at risk of Se deficiency. Genetic polymorphisms may also leave individuals susceptible to low Se status. Genetic variation in the dimethylglycine dehydrogenase (DMGDH) rs921943 gene, which is involved in the metabolism of sulphur-containing amino acids, has been shown in pregnant women to be associated with Se status. Furthermore, pregnant women who carry the selenoprotein P (SEPP1) rs3877899 minor A allele are more able to maintain Se levels during pregnancy (52). However, further understanding of the role of genetic polymorphisms on Se status in the wider population are required before genetic testing is recommended.

In combination with observational data, supplementation studies would suggest Se is required for optimal thyroid health and function especially in those with autoimmune disease of the thyroid. In individuals with chronic AIT, one systematic review and meta-analysis revealed that Se supplementation (200 µg/day) for durations of 3-12 months in combination with levothyroxine (synthetic T4), improved TPO autoantibody levels (weighted mean difference (WMD) = –271; 95% CI -366 to -175; p=< 0.0001; I2 = 45%) (53).

Improvements to autoantibodies have also been seen in individuals with Hashimoto's thyroiditis given 200 µg/day Se, which was attributed to improved antioxidant activity (GPx3 SYT: 45.2 ng/ml; 95% CI 23.7, 68.4 ng/ml; vs control 24.2 ng/ml; CI = 15.3, 50.4 ng/ml; p=0.028). Supplementation also resulted in upregulated regulatory T (Treg) cells (13.19 ± 3.5 vs 11.49 ± 2.79; p=0.012), which are involved in the prevention of autoimmune disease (54).

Given the antioxidant nature of Se, supplemental research in individuals with Graves' orbitopathy (GO), which is an inflammatory, autoimmune disorder of the retro-ocular tissue as a result of GD, has also shown promising results. At doses of 100 µg twice daily for 6 months, it has been shown in one RCT that Se may be beneficial to quality of life (QoL) and may slow progression of disease (55). In this study, 159 individuals with GO were randomised to sodium selenite, pentoxifylline (600mg twice daily), which has been indicated as a potential GO treatment, or placebo. Compared to placebo, individuals on sodium selenite showed improvements to QoL (p=<0.001), slowing of disease (p=0.01), and less eye involvement (p=0.01), observations that were not observed in the pentoxifylline group. Sodium selenite was not associated with any side effects, but gastrointestinal problems were frequently seen in those taking pentoxifylline. Similar results have been seen in a five-year prospective controlled cohort study of 74 individuals with mild-moderate GO, with improvements from baseline to QoL (p=0.01) and disease state (p= <0.05) after 3-6 months' Se therapy (56). However, this study did highlight that there may be no difference to long-term outcomes of disease.

Due to its role as an antioxidant and suppression of autoantibodies, it could be hypothesised that the use of Se in all autoimmune conditions would be of benefit. However, studies on individuals with GD have shown mixed results and may be dependent on serum Se. One RCT in 38 Se deficient individuals with GD has shown that supplementation of 200 µg/day resulted in improvements to biochemical parameters such as TSH (0.05 vs. 0.02 mIU/l; p=0.04), and T4 (15 vs. 18 pmol/l; p=0.01), but had no effect on FT3 (57).

In contrast studies on Se sufficient individuals with GD have shown no improvements to symptoms or disease status (58,59).

4. How can vitamin A influence thyroid health?

Vitamin A may influence thyroid function in two ways, namely through modulation of hormones responsible for thyroid gland metabolism, and through stimulation of the production of TSH by the pituitary. Vitamin A deficiency (VAD) may be involved in the development of thyroid disorders. Hypothyroidism was observed in a cross-sectional study of individuals with obesity-associated VAD with increased TSH (p=0.037), decreased T4 (p=0.001), and a negative association with TSH (r=-0.151; p=0.006) compared to individuals with normal VA levels (60).

The involvement of VA in thyroid homeostasis may be due to its involvement in HPT signalling from the pituitary. In a 10-month double-blind RCT in n=138 iodine-deficient children who also had VAD showed that, upon supplementation of high-dose VA (200,000 IU retinyl palmitate) and iodine (salt fortification), TSH and Tg concentrations as well as goitre rate were decreased compared to children given iodised salt and placebo (p=<0.01). At baseline, increasing VAD severity was a predictor of greater thyroid volume, TSH and Tg (p=<0.001 for all). The authors hypothesised that the physiological mechanism responsible for this effect could be VA-mediated suppression of the pituitary TSHβ gene (61).

It has also been shown that vitamin A may still be important regardless of iodine status. In a double blind RCT of children in Africa with both VAD and iodine deficiency, it was shown that there was an effect of VA supplementation in children who were not given iodine co-supplementation, as TSH, Tg and thyroid volume all decreased (p=<0.05 for all). However, the supplementation of both VA and iodine was more effective at reducing circulating TSH, serum Tg and thyroid size (p=<0.001 for all) (62). It was unclear from this study how TH concentration was regulated independently of iodine levels, but again, it may be due to the actions of VA on the pituitary.

5. The relationship between zinc and thyroid health

Zinc is an essential trace mineral that influences gene expression and cellular growth and serves as a cofactor for many enzymes involved in crucial physiological processes (8). It plays an important role in TH synthesis, metabolism, and regulation (63), and deficiency may impair TH receptor function, limiting the receptors' ability to bind TH and regulate gene expression, thereby disrupting essential TH signalling pathways (8).

Zn deficiency has been linked to thyroid enlargement in children and adults. A study of 68 goitrous school children with low Zn concentrations found an inverse correlation between Zn status and thyroid size. The authors hypothesised that this may be due to Zn loss, impaired T3 action, and reduced T3 binding to its nuclear receptor (64).

Low Zn concentrations are associated with hypothyroidism, while elevated Zn intakes may be related to hyperthyroidism (65–68). In patients with normal thyroid function, Zn concentrations positively correlated with T3 concentrations (p= <0.001) (69). Zn-deficient women consuming 30mg of Zn gluconate daily for 12 weeks showed increases in serum concentrations of FT3 and FT4 (p=<0.05) (70). Given these findings, practitioners may consider assessing Zn when low FT4 concentrations are observed (63)

Research suggests that Zn is involved in enhancing levels of TH, indicating its regulatory effect on TH production (65). Zn acts as a link between T3 and its nuclear receptor in the hypothalamus to stimulate the synthesis of TRH. TRH stimulates the synthesis and release of TSH by thyrotrophs in the pituitary glands, and in turn, TSH stimulates the synthesis of TH for release into the bloodstream (68)

6. Can other nutrients impact thyroid health?

Several other nutrients may have an impact on thyroid function including vitamin D and magnesium.

Vitamin D modulates the immune system through several mechanisms.

In a systematic review and meta-analysis of observational studies, individuals with thyroid disorders had lower serum vitamin D concentrations compared with healthy controls (71). Specifically, the weighted mean difference (WMD) of serum vitamin D was lower in autoimmune thyroid diseases, excluding Graves' disease, (WMD – 3.1 ng/dl; 95% CI –5.57 to –0.66; p=0.013; I2= 99.9%), Hashimoto's thyroiditis (WMD – 6.05 ng/dl; 95% CI, – 8.35 to – 3.75; p<0.001; I2= 91.0%) and hypothyroidism patients (WMD – 13.43 ng/dl; 95% CI –26.04 to –0.81; p=0.03; I2=99.5%).

Subgroup analysis showed a significant association with Graves' disease only in individuals aged ≥40 years (WMD –8.79 ng/dl; 95% CI –15.87 to –1.72; p<0.001) (71). The lower occurrence of autoimmune conditions observed in individuals with higher serum vitamin D may be the result of the vitamin's regulatory effects on immune cells.

Thyroid autoimmune disorders have been linked to heightened T-helper (Th)-1 and Th17 responses (72) and increased levels of pro-inflammatory cytokines such as interferon gamma (IFN-γ), interleukin (IL)-17, and tumour necrosis factor alpha (TNF-α) (73).

Vitamin D has been found to suppress Th1 responses by downregulating IFN-γ and TNF-α production (74).

Vitamin D may decrease the production of cytokines such as IL-6, IL-2, and TNF-α, which are involved in the immune-mediated destruction of thyroid tissue in Hashimoto's thyroiditis, or the thyroid-stimulating antibody production of GD (74).

Vitamin D deficiency is considered a risk factor for the development of autoimmune thyroid diseases such as Hashimoto's and GD (71,75).

Magnesium is an important nutrient for the conversion of T4 to its active form, T3, in target tissues.
Mg is an essential cofactor that supports adenosine triphosphate (ATP) synthesis, overall cellular metabolism and maintains intracellular oxidative balance during energy production.

Efficient ATP production is critical for the optimal activity of deiodinase enzymes, as these enzymes require high ATP availability for the catalytic reactions needed to convert T4 into the active hormone T3 in peripheral tissues (8). Therefore, low Mg levels may impair T4-to-T3 conversion. This may contribute to the development of symptoms of thyroid dysfunction.

In a cross-sectional study of 1,257 participants that investigated the association between low serum Mg levels and thyroid disorders (76), those with severely low Mg levels (≤0.55 mmol/L) had a higher risk of anti-Tg antibody (TGAb) positivity (odds ratios [ORs]: 3.036–3.236) p=0.000), Hashimoto's thyroiditis (ORs: 2.748– 2.847 p=< 0.001), and hypothyroidism (ORs = 4.482–4.971, p=< 0.01), compared to individuals with adequate Mg levels (0.851–1.15 mmol/L).

7. Are there any modifiable lifestyle factors that can support thyroid function?

Many lifestyle factors including nutrition, sleep hygiene, stress, physical activity, gut microbiome and endocrine disruptors have been found to play a role in supporting or disrupting thyroid function (77). Understanding the effect of each of these can help practitioners to identify strategies for incorporation into a client's protocol.

Tian et al (2024) extracted data on physical activity from the US National Health And Nutrition Examination Survey (NHANES) (78). They found a non-linear relationship between thyroid health and exercise, highlighting that the effect physical activity has on thyroid health varies.

Overall, their results suggest that moderate exercise may support optimal thyroid health.

There is a direct link between stress as an environmental trigger for those susceptible to developing GD (79), such as smokers, females, individuals with a genetic susceptibility to the disease, and those with other autoimmune diseases such as type 1 diabetes and rheumatoid arthritis. To obtain a broader view of stress and its impact on thyroid health more research is needed on the HPT axis and longitudinal studies monitoring thyroid function against exposure to stress.

A cross-sectional study concluded that poor sleep quality may increase the risk of SCH (77). However, a recent systemic review (80) did not draw the same conclusion; while a correlation between sleep quality and risk of SCH was identified, it was not possible to extrapolate the casual direction of effect, i.e. whether sleep deprivation caused SCH or if the opposite was true. This data included in the review, however, were subject to high heterogeneity, which may have influenced the result. The thyroid gland is susceptible to exogenous substances or contaminants, like endocrine disruptors found in the environment and in food. These different chemicals, including bisphenol A (BPA) and phthalates found in plastics have, amongst others, been shown to cause TH fluctuations, reduce the uptake of iodine (81) and increase the risk of thyroid cancer (82).

The gut-thyroid axis refers to the interplay between the thyroid and the gut and research shows the varied role the microbiome plays in thyroid health. Dysbiosis, for example, impacts several factors related to the thyroid gland, including the absorption of key micronutrients like Se and iodine. Intestinal permeability, which can be increased by dysbiosis, has also been linked to autoimmune diseases like thyroiditis (83) and thyroid cancer (84).

8. Summary and key points

The thyroid gland is essential for metabolism and growth of the body, and it is important for practitioners to consider it when seeing individuals

with metabolic disorders such as heart disease, Alzheimer's disease, and diabetes. Several lifestyle factors can affect the function of the thyroid gland, all of which can be modified with support. Diet is particularly important to thyroid health, and as such Nutritional Therapists should take a key role in the management of individuals with or at risk of thyroid dysfunction. Thyroid function testing in individuals with metabolic disorders and accompanying thyroid dysfunction symptoms is important, given that dysfunction is underdiagnosed (10). In confirmed and borderline cases, dietary analysis is essential to understand where support can be given. Iodine, Fe, Se, VA, zinc, vitamin D, and Mg have all been shown to be involved in thyroid function.

Iodine is needed for TH synthesis, in particular T4, but it is important that intakes are carefully balanced, as both iodine deficiency and excess can lead to dysfunction. It is especially important to carefully monitor intakes in individuals who have previously been deficient, as in these individuals, even intakes slightly above optimal intake may damage thyroid gland cells. Iodine levels should be gradually increased to prevent iodine-induced hyperthyroidism. At-risk populations may require additional dietary iodine including vegans, pregnant and lactating women, those in areas with no iodised salt policy, or in regions of deficiency. Pregnant women should be advised to use iodised salt and/or regularly consume iodine-rich foods such as fish, algae, eggs and milk, to ensure adequate iodine levels for foetal brain development.

The role of Fe in thyroid gland health is currently unclear, although cross-sectional studies indicate that individuals with Fe deficiency or anaemia may also be experiencing thyroid dysfunction. This may be due to the role of Fe in TH synthesis and the conversion of T4 to T3. Women who are pre-menopausal may be particularly susceptible to thyroid dysfunction because of Fe deficiency and should be closely monitored. Fe deficiency could be addressed by increasing intakes of Fe-rich foods including meat, fish, dark green leafy vegetables, egg yolks, beans, and nuts, alongside vitamin C rich foods and using a bioavailable Fe supplement.

The thyroid gland Is a major storage centre for **Selenium** and may benefit from Its antioxidant properties. If individuals are experiencing poor thyroid function or autoimmunity, then low Se levels may be contributing, especially if seen in vegetarians. Practitioners may like to consider testing, and SeP is a reliable biomarker for serum Se levels. Dietary changes should be considered first-line, but supplementation of 200µg/day may be a good option if dietary restrictions are in place. Due care should be exercised with Se supplementation since the therapeutic window for Se is narrow, and excessive intakes can be deleterious to health (85).

Practitioners should be aware that fluctuations in **Vitamin A** may affect thyroid homeostasis and function. VA acts by regulating the action of iodine and through regulation of TSH secretion by the pituitary gland. Individuals with suboptimal thyroid function may consider testing and optimising both VA and iodine status to support rebalancing thyroid function. In lieu of a low iodine status, low VA may still need to be addressed for optimal thyroid function.

Zinc may be involved in TH regulation and balance may be essential to thyroid health. Excess may lead to hyperthyroidism and deficiency may lead to hypothyroidism and zinc levels should be considered when individuals are experiencing low TSH and T4.

Vitamin D may have a role in thyroid health through its anti-inflammatory actions and decrease the risk for autoimmune conditions such as GD and Hashimoto's thyroiditis (75,86). The Clinical Guidelines Subcommittee of The Endocrine Society recommends maintaining sufficient vitamin D intake by ensuring adequate sunlight exposure, dietary intake, or supplementation to achieve optimal serum 25(OH)D levels (87). These levels should be at least 30 ng/mL (75 nmol/L), with an ideal range of 40–60 ng/mL (100–150 nmol/L) (88).

Magnesium deficiency can result from various factors, including inadequate dietary magnesium intake, malabsorption, and excessive alcohol consumption (89). Assessment of magnesium intake and ensuring adequate magnesium intake, through diet and supplementation where needed, should be

part of the thyroid management strategy, particularly in conditions that deplete magnesium, such as excessive alcohol use.

Despite its importance in thyroid health, dietary adjustments should be accompanied with other lifestyle recommendations for optimal thyroid health. The thyroid gland affects most organ systems in the body and incorporating changes in lifestyle could be a simple yet effective way of positively influencing thyroid health. From a nutritional perspective, increasing the nutrient content of the diet with nutrients key to thyroid health can help support thyroid balance. Moderate exercise, appropriate stress management, prioritising sleep, reducing exposure to chemicals in the environment and in food, and modulating the microbiome may be key areas of focus for nutritional therapy practitioners.

AUTHORS AFFILIATIONS

1. Chloe Steele Nutrition, Australia
2. Nutrition 4 You, South Africa
3. Gail Brady Nutrition, United Kingdom
4. Independent consultant, United Kingdom
5. Nuthrive, The Netherlands
6. RiNu Nutrition and Consulting, Switzerland

*CORRESPONDING AUTHOR

Dr Jessica Rigutto: jessica.rigutto@hin.ch

Keywords:
thyroid, endocrine, iodine, metabolic disorders, nutrition, lifestyle

References: Pages 60 - 63

Author contributions

JR conceived of the review, provided comments to other authors, revised the final draft and is responsible for content. CS wrote the introduction, parts 3 and 4, the discussion and provided editing. APA wrote part 5, APS wrote parts 1 and 6, GB wrote part 2, NS wrote part 7. All authors reviewed and accepted the final manuscript.

Acknowledgements

The authors wish to thank the members of the Nutrition Evidence Database (NED) Editorial Board, for comments, editing and peer review during the drafting process.
The authors form part of the NED Editorial Board (JR) and the NED Expert Review Panel.
Grammarly Ai was used to improve sentence structure and flow in some parts of this review, however, authors have reviewed and remain responsible for content.

Declarations of interest and funding

This review was funded by the **British Association for Nutrition and Lifestyle Medicine (BANT)**.


Chloe Steele


Ana-Paula Agrela


Gail Brady


Nicky Ester


Anna Papoutsa Shue


Jessica Rigutto

References

1.Zimmermann MB. Iodine and the iodine deficiency disorders. Present Knowledge in Nutrition: Basic Nutrition and Metabolism. 2020 Jan 1;429–41.

2. Zúñiga D, Balasubramanian S, Mehmood KT, Al-Baldawi S, Zúñiga Salazar G. Hypothyroidism and Cardiovascular Disease: A Review. Cureus. 2024 16(1):e52512.

3. Salehipour A, Dolatshahi M, Haghshomar M, Amin J. The Role of Thyroid Dysfunction in Alzheimer's Disease: A Systematic Review and Meta-Analysis. Vol. 10, J Prev Alzheimer Dis, 2023 10:276–86.

4. Biondi B, Kahaly GJ, Robertson RP. Thyroid Dysfunction and Diabetes Mellitus: Two Closely Associated Disorders. Endocrine Reviews 2018 40:789–824.

5. Mehran L, Amouzegar A, Rahimabad P, Tohidi M, Tahmasebinejad Z, Azizi F. Thyroid Function and Metabolic Syndrome: A Population-Based Thyroid Study. Hormone and Metabolic Research. 2017 28;49(03):192–200.

6. Wang JJ, Zhuang ZH, Shao CL, Yu CQ, Wang WY, Zhang K, et al. Assessment of causal association between thyroid function and lipid metabolism: a Mendelian randomization study. Chin Med J (Engl). 2021 5;134(9):1064–9.

7. Leko MB, Gunjača I, Pleić N, Zemunik T. Environmental factors affecting thyroid-stimulating hormone and thyroid hormone levels. Int J Mol Sci. 2021 Jun 2;22(12).

8. Shulhai AM, Rotondo R, Petraroli M, Patianna V, Predieri B, Iughetti L, et al. The Role of Nutrition on Thyroid Function. Nutrients. 2024 Jul 31;16(15).

9. Woodside J V., Mullan KR. Iodine status in UK–An accidental public health triumph gone sour. Clin Endocrinol. 2021 94: 692–9.

10. Garmendia Madariaga A, Santos Palacios S, Guillén-Grima F, Galofré JC. The Incidence and Prevalence of Thyroid Dysfunction in Europe: A Meta-Analysis. J Clin Endocrinol Metab 2014 1;99(3):923–31.

11. Scuteri A, Laurent S, Cucca F, Cockcroft J, Cunha PG, Mañas LR, et al. Metabolic syndrome across Europe: Different clusters of risk factors. Eur J Prev Cardiol. 2015 15;22(4):486–91.

12. Saklayen MG. The Global Epidemic of the Metabolic Syndrome. Curr Hypertens Rep 2018 26;20(2):12.

13. Mendes D, Alves C, Silverio N, Marques FB. Prevalence of Undiagnosed Hypothyroidism in Europe: A Systematic Review and Meta-Analysis. Eur Thyroid J. 2019 8(3):130–43.

14. Ahad F, Ganie SA. Iodine, Iodine metabolism and Iodine deficiency disorders revisited. Indian J Endocrinol Metab 2010;14(1):13.

15. Farebrother J, Zimmermann MB, Andersson M. Excess iodine intake: sources, assessment, and effects on thyroid function. Ann N Y Acad Sci. 2019 1446(1):44–65.

16. Weng W, Dong M, Zhan J, Yang J, Zhang B, Zhao X, et al. A PRISMA-compliant systematic review and meta-analysis of the relationship between thyroid disease and different levels of iodine intake in mainland China. Medicine 2017 96(25).

17. Zimmermann MB, Boelaert K. Iodine deficiency and thyroid disorders. Lancet Diabetes Endocrinol. 2015 3(4):286–95.

18. Teti C, Panciroli M, Nazzari E, Pesce G, Mariotti S, Olivieri A, et al. Iodoprophylaxis and thyroid autoimmunity: an update. Immunol Res. 2021;69(2):129.

19. Mathews DM, Johnson NP, Sim RG, O'Sullivan S, Peart JM, Hofman PL. Iodine and fertility: do we know enough? Hum Reprod. 2021 36(2):265–74.

20. Angermayr L, Clar C. Iodine supplementation for preventing iodine deficiency disorders in children. Cochrane Database Syst Rev. 2004(2).

21. Nielsen VE, Bonnema SJ, Hegedüs L. Transient goiter enlargement after administration of 0.3 mg of recombinant human thyrotropin in patients with benign nontoxic nodular goiter: a randomized, double-blind, crossover trial. J Clin Endocrinol Metab. 2006 91(4):1317–22.

22. Eng PHK, Cardona GR, Fang SL, Previti M, Alex S, Carrasco N, et al. Escape from the acute Wolff-Chaikoff effect is associated with a decrease in thyroid sodium/iodide symporter messenger ribonucleic acid and protein. Endocrinology, 1999 140(8):3404–10.

23. Vargas-Uricoechea H. Molecular Mechanisms in Autoimmune Thyroid Disease. Cells 2023 12(6).

24. Wan S, Jin B, Ren B, Qu M, Wu H, Liu L, et al. The Relationship between High Iodine Consumption and Levels of Autoimmune Thyroiditis-Related Biomarkers in a Chinese Population: a Meta-Analysis. Biol Trace Elem Res. 2020 196(2):410–8.

25. Xing D, Liu D, Li R, Zhou Q, Xu J. Factors influencing the reference interval of thyroid-stimulating hormone in healthy adults: A systematic review and meta-analysis. Clin Endocrinol. 2021 95(3):378–89.

26. World Health Organization, UNICEF, ICCIDD. Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers, 3rd ed. Geneva, Switzerland, Switzerland; 2007. Available from: https://apps.who.int/iris/handle/10665/43781

References cont.

27. Zimmermann MB, Andersson M. Assessment of iodine nutrition in populations: past, present, and future. Nutr Rev. 2012 Oct;70(10):553–70.
28. De Angelis S, Medda E, Rotondi D, Masocco M, Minardi V, Contoli B, et al. Fifteen Years of Iodine Prophylaxis in Italy: Results of a Nationwide Surveillance (Period 2015-2019). J Clin Endocrinol Metab. 2024 109(2):e495–507.
29. Lisco G, De Tullio A, Triggiani D, Zupo R, Giagulli VA, De Pergola G, et al. Iodine Deficiency and Iodine Prophylaxis: An Overview and Update. Nutrients 2023 15(4):1004.
30. Yang L, Wang J, Yang J, Zhang H, Liu X, Mao D, et al. An iodine balance study to explore the recommended nutrient intake of iodine in Chinese young adults. Br J Nutr 2020 124(11):1156–65.
31. Mohammed H, Marquis GS, Aboud F, Bougma K, Samuel A. Pre-pregnancy iodized salt improved children's cognitive development in randomized trial in Ethiopia. Matern Child Nutr 2020 16(3).
32. Maldonado-Araque C, Valdés S, Lago-Sampedro A, Lillo-Muñoz JA, Garcia-Fuentes E, Perez-Valero V, et al. Iron deficiency is associated with Hypothyroxinemia and Hypotriiodothyroninemia in the Spanish general adult population: Di@bet.es study. Sci Rep. 2018 Dec 1;8(1).
33. Zhou Q, Xue S, Zhang L, Chen G. Trace elements and the thyroid. Vol. 13, Front Endocrinol 2022 24:13:904889.
34. Knezevic J, Starchl C, Berisha AT, Amrein K. Thyroid-gut-axis: How does the microbiota influence thyroid function? Nutrients. 2020. 12: 1–16.
35. 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. Front Endocrinol 2021 Feb 25;12.
36. Wopereis DM, Du Puy RS, Van Heemst D, Walsh JP, Bremner A, Bakker SJL, et al. The relation between thyroid function and anemia: A pooled analysis of individual participant data. Journal of Clinical Endocrinology and Metabolism. 2018 103(10):3658–67.
37. Köhrle J. Selenium, Iodine and Iron–Essential Trace Elements for Thyroid Hormone Synthesis and Metabolism. Int J Mol Sci 2023 24(4):3393
38. Gu S, Mo Z, Chen Z, Li X, Jiang Y, Liu C, et al. Assessment of Individual and Mixed Effects of Six Minerals on Thyroid Hormones in Chinese Pregnant Women. Nutrients. 2024 Feb 1;16(3).
39. He L, Shen C, Zhang Y, Chen Z, Ding H, Liu J, et al. Evaluation of serum ferritin and thyroid function in the second trimester of pregnancy. Endocr J 2018 65(1):75-82
40. Gupta N, Narayan A, Tonk RS, Gupta SK, Narayan A. Study of Relationship Between Iron Deficiency and Thyroid Function in Pregnant Females. Cureus. 2022 14(12):e32411
41. Khatiwada S, Gelal B, Baral N, Lamsal M. Association between iron status and thyroid function in Nepalese children. Thyroid Res. 2016 Jan 27;9(1).
42. Garofalo V, Condorelli RA, Cannarella R, Aversa A, Calogero AE, La Vignera S. Relationship between Iron Deficiency and Thyroid Function: A Systematic Review and Meta-Analysis. Nutrients. 2023 15(22):4790
43. Aaseth J, Frey H, Glattre E, Norheim G, Ringstad J, Thomassen Y. Selenium concentrations in the human thyroid gland. Biol Trace Elem Res. 1990;24(2–3):147–52.
44. Hao R, Yu P, Gui L, Wang N, Pan D, Wang S. Relationship between Serum Levels of Selenium and Thyroid Cancer: A Systematic Review and Meta-Analysis. Nutr Cancer. 2023;75(1):14–23.
45. Derumeaux H, Valeix P, Castetbon K, Bensimon M, Boutron-Ruault MC, Arnaud J, et al. Association of selenium with thyroid volume and echostructure in 35- to 60-year-old French adults. Eur J Endocrinol. 2003;148(3):309–15.
46. Rasmussen LB, Schomburg L, Köhrle J, Pedersen IB, Hollenbach B, Hög A, et al. Selenium status, thyroid volume, and multiple nodule formation in an area with mild iodine deficiency. Eur J Endocrinol. 2011;164(4):585–90.
47. Bülow Pedersen I, Knudsen N, Carlé A, Schomburg L, Köhrle J, Jørgensen T, et al. Serum selenium is low in newly diagnosed Graves' disease: A population-based study. Clin Endocrinol. 2013;79(4):584–90.
48. Wu Q, Rayman MP, Lv H, Schomburg L, Cui B, Gao C, et al. Low population selenium status is associated with increased prevalence of thyroid disease. J Clin Endocrinol Metab. 2015;100(11):4037–47.
49. Müller SM, Dawczynski C, Wiest J, Lorkowski S, Kipp AP, Schwerdtle T. Functional biomarkers for the selenium status in a human nutritional intervention study. Nutrients. 2020 Mar 1;12(3).
50. Brodin O, Hackler J, Misra S, Wendt S, Sun Q, Laaf E, et al. Selenoprotein P as biomarker of selenium status in clinical trials with therapeutic dosages of selenite. Nutrients. 2020;12(4):1–11.
51. Ullah H, Liu G, Yousaf B, Ali MU, Abbas Q, Munir MAM, et al. Developmental selenium exposure and health risk in daily foodstuffs: A systematic review and meta-analysis. Ecotoxicol Environ Saf. 2018;149:291–306.

References cont.

52. Mao J, Vanderlelie JJ, Perkins A V., Redman CWG, Ahmadi KR, Rayman MP. Genetic polymorphisms that affect selenium status and response to selenium supplementation in United Kingdom pregnant women. Am J Clin Nutr. 2016;103(1):100–6.
53. 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.
54. Hu Y, Feng W, Chen H, Shi H, Jiang L, Zheng X, et al. Effect of selenium on thyroid autoimmunity and regulatory T cells in patients with Hashimoto's thyroiditis: A prospective randomized-controlled trial. Clin Transl Sci. 2021;14(4):1390–402.
55. Marcocci C, Kahaly GJ, Krassas GE, Bartalena L, Prummel M, Stahl M, et al. Selenium and the Course of Mild Graves' Orbitopathy. NEJM. 2011;364(20):1920–31.
56. Wang C, Qiao J, Liu S, Piao S, Zhou Y, Hu Y, et al. Selenium in the treatment of mild-to-moderate Graves' orbitopathy: a 5-year prospective controlled cohort study. Endocrine. 2024;84(3):1072–80.
57. Calissendorff J, Mikulski E, Larsen EH, Möller M. A Prospective Investigation of Graves' Disease and Selenium: Thyroid Hormones, Auto-Antibodies and Self-Rated Symptoms. Eur Thyroid J. 2015;4(2):93–8.
58. Leo M, Bartalena L, Rotondo Dottore G, Piantanida E, Premoli P, Ionni I, et al. Effects of selenium on short-term control of hyperthyroidism due to Graves' disease treated with methimazole: results of a randomized clinical trial. J Endocrinol Invest. 2017;40(3):281–7.
59. Kahaly GJ, Riedl M, König J, Diana T, Schomburg L. Double-blind, placebo-controlled, randomized trial of selenium in graves hyperthyroidism. J Clin Endocrinol Metab. 2017;102(11):4333–41.
60. Ma B, Yang P, Gao J, Du L, Sheng C, Usman T, et al. Relationship of Vitamin A and Thyroid Function in Individuals With Obesity and After Laparoscopic Sleeve Gastrectomy. Front Nutr. 2022;9(March):1–9.
61. Zimmermann MB, Wegmüller R, Zeder C, Chaouki N, Torresani T. The effects of vitamin A deficiency and vitamin A supplementation on thyroid function in goitrous children. J Clin Endocrinol Metab. 2004;89(11):5441–7.
62. Zimmermann MB, Jooste PL, Mabapa NS, Schoeman S, Biebinger R, Mushaphi LF, et al. Vitamin A supplementation in iodine-deficient African children decreases thyrotropin stimulation of the thyroid and reduces the goiter rate. Am J Clin Nutr. 2007;86(4):1040–4.
63. Nagano K, Motomura Y, Bando H, Yamamoto M, Kanie K, Yoshino K, et al. Thyroid dysfunction due to trace element deficiency-not only selenium but also zinc. Hormones. 2024;23(4):675–81.
64. El-Fadeli S, Bouhouch S, Skalny A V, Barkouch Y, Pineau A, Cherkaoui M, et al. Effects of Imbalance in Trace Element on Thyroid Gland from Moroccan Children. Biol Trace Elem Res. 2016 Apr;170(2):288–93.
65. Zhou Q, Xue S, Zhang L, Chen G. Trace elements and the thyroid. Front Endocrinol. 2022;13:904889.
66. Betsy A, Binitha M, Sarita S. Zinc deficiency associated with hypothyroidism: an overlooked cause of severe alopecia. Int J Trichology. 2013 Jan;5(1):40–2.
67. Napolitano G, Palka G, Lio S, Bucci I, De Remigis P, Stuppia L, et al. Is zinc deficiency a cause of subclinical hypothyroidism in Down syndrome? Ann Genet. 1990;33(1):9–15.
68. 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 Jul;89(1–2):80–8.
69. Ertek S, Cicero AF, Caglar O, Erdogan G. Relationship between serum zinc levels, thyroid hormones and thyroid volume following successful iodine supplementation. Hormones. 2010;9(3):263–8.
70. Mahmoodianfard S, Vafa M, Golgiri F, Khoshniat M, Gohari M, Solati Z, et al. Effects of Zinc and Selenium Supplementation on Thyroid Function in Overweight and Obese Hypothyroid Female Patients: A Randomized Double-Blind Controlled Trial. J Am Coll Nutr. 2015;34(5):391–9.
71. 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 Endocr Disord. 2021 21(1):1–12.
72. Li Q, Wang B, Mu K, Zhang JA. The pathogenesis of thyroid autoimmune diseases: New T lymphocytes - Cytokines circuits beyond the Th1-Th2 paradigm. J Cell Physiol. 2019 234(3):2204–16.
73. Wrońska K, Hałasa M, Szczuko M. The Role of the Immune System in the Course of Hashimoto's Thyroiditis: The Current State of Knowledge. Int J Mol Sci. 2024 25(13).
74. Lebiedziński F, Lisowska KA. Impact of Vitamin D on Immunopathology of Hashimoto's Thyroiditis: From Theory to Practice. Nutrients. 2023 15(14):3174.

References cont.

75. Babić Leko M, Jureško I, Rozić I, Pleić N, Gunjača I, Zemunik T. Vitamin D and the Thyroid: A Critical Review of the Current Evidence. Int J Mol Sci. 2023 24(4):3586.
76. Wang K, Wei H, Zhang W, Li Z, Ding L, Yu T, et al. Severely low serum magnesium is associated with increased risks of positive anti-thyroglobulin antibody and hypothyroidism: A cross-sectional study. Sci Rep 2018 8(1):9904.
77. Wu K, Zhou Y, Ke S, Huang J, Gao X, Li B, et al. Lifestyle is associated with thyroid function in subclinical hypothyroidism: a cross-sectional study. BMC Endocr Disord. 2021 Dec 1;21(1).
78. Tian L, Lu C, Teng W. Association between physical activity and thyroid function in American adults: a survey from the NHANES database. BMC Public Health. 2024 Dec 1;24(1).
79. Leso V, Vetrani I, De Cicco L, Cardelia A, Fontana L, Buonocore G, et al. The impact of thyroid diseases on the working life of patients: A systematic review. Vol. 17, Int J Environ Res Public Health. 2020 17(12)4295
80. Teliti M, Fanfulla F, Croce L, Coperchini F, Rotondi M. The interplay between subclinical hypothyroidism and poor sleep quality: A systematic review. Eur J Intern Med. 2024 Aug 1;126:49–55.
81. Oliveira KJ, Chiamolera MI, Giannocco G, Pazos-Moura CC, Ortiga-Carvalho TM. Thyroid function disruptors: From nature to chemicals. J Mol Endocrinol 2018 JME-18-0081
82. Macedo S, Teixeira E, Gaspar TB, Boaventura P, Soares MA, Miranda-Alves L, et al. Endocrine-disrupting chemicals and endocrine neoplasia: A forty-year systematic review. Environ Res 2023 1:218:114869
83. Virili C, Stramazzo I, Bagaglini MF, Carretti AL, Capriello S, Romanelli F, et al. The relationship between thyroid and human-associated microbiota: A systematic review of reviews. Rev Endocr Metab Disord 2024. 25(1): 215–37.
84. Shi C, Chen J, He S, Zhang Y, Zhang Y, Yu L. Cross-talk between the gut microbiota and hypothyroidism: a bidirectional two-sample Mendelian randomization study. Front Nutr. 2024;11.
85. Winther KH, Rayman MP, Bonnema SJ, Hegedüs L. Selenium in thyroid disorders — essential knowledge for clinicians. Nat Rev Endocrinol. 2020;16(3):165–76.
86. 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 Endocr Disord. 2021 Dec 1;21(1).
87. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: An endocrine society clinical practice guideline. J Clin Endocrinol Metab 2011 96(7):1911–30.
88. Sîrbe C, Rednic S, Grama A, Pop TL. An Update on the Effects of Vitamin D on the Immune System and Autoimmune Diseases. Int J Mol Sci 2022 23(17):9784
89. Ahmed F, Mohammed A. Magnesium: The Forgotten Electrolyte-A Review on Hypomagnesemia. Med Sci 2019 7(4):56