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The role of iodine in thyroid function and iodine impact on the course of Hashimoto's thyroiditis - review.

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Abstract

Introduction:

Iodine is one of the most important micronutrients necessary for the proper functioning of the thyroid gland. It is an essential component of thyroxine and triiodothyronine, hormones produced and secreted by the thyroid gland. Diet serves as the primary source of iodine, with rich sources including fish, seafood, seaweed, iodized salt, and dairy products. Severe iodine deficiency impairs thyroid hormone synthesis and can lead to gland enlargement. This deficiency results in various disorders collectively termed iodine deficiency disorders, including endemic goiter, hypothyroidism, and cretinism. Not only iodine deficiency but also excess iodine intake can be harmful and affect thyroid function. Doses of iodine above physiological levels can result in both hypothyroidism and hyperthyroidism. Numerous studies have linked chronic high iodine intake to an increased frequency of autoimmune thyroiditis. This review examines the mechanisms of iodine regulation and the role of excess iodine can play in the development of Hashimoto's thyroiditis.

Aim of the study: The aim of the study is to present and summarize the role of iodine in thyroid function and its impact on the course of Hashimoto's thyroiditis. This review aims to explain the importance of iodine excess for the human body and potential increased risk of thyroid autoimmune disorder connected with iodine over-supplementation.

Materials and Methods: For this current literature review, the database of PubMed was searched for relevant studies. The important studies published from 1986-2024 that may affect our conclusions, were collected. This article provides an extensive survey of recent randomized controlled trials, meta-analyses, and clinical trials. The literature available in the Pubmed database was reviewed with the following keywords: iodine, Hashimoto's thyroiditis, autoimmune thyroiditis.

Conclusion: Iodine is crucial for thyroid health and fetal development, but its excessive intake can be harmful. Maintaining optimal levels of this microelement is essential to prevent both iodine deficiency and autoimmune disorders. Iodine supplementation is recommended only for pregnant and lactating women.

Keywords: iodine, Hashimoto's thyroiditis, autoimmune thyroiditis, thyroid gland

Introduction

Hashimoto's thyroiditis (HT), also known as Hashimoto's disease, is the most common form of autoimmune thyroiditis (AIT). It is characterized by chronic inflammation with lymphocytic infiltration of the gland tissue and the presence of autoantibodies against thyroid peroxidase (TPO-Ab) and against thyroglobulin (Tg-Ab) [1,2]. Clinical manifestation of HT can vary depending on the duration of the disorder. The course of HT may include euthyroidism, temporarily hyperthyroidism but the most commonly hypothyroidism [1]. Hypothyroidism is a result of progressive destruction of thyroid follicles caused by inflammatory cell infiltration. The etiopathogenesis of Hashimoto's disease is still incompletely defined, but it is certainly complex. The following factors are associated with an increased risk of Hashimoto's disease: sex, age, other autoimmune disease, genetic and family history, excessive iodine intake, radiation exposure [3]. The prevalence of Hashimoto's disease varies across different parts of the world, being more common in Caucasians than in Blacks and Asians [1,3,4]. The frequency of HT has a growing trend with a worldwide estimated prevalence up to 7,5% [5]. Among Caucasians, it is approximately 5% [6]. Furthermore, the prevalence of HT also depends on age and gender, with the disease occurring 4-10 times more frequently in women than in men [1,4,5,6,7]. Most cases of the HT develop in populations aged between 45 and 65 [1,6,7]. Hashimoto's thyroiditis occurs notably more often in individuals suffering from other autoimmune diseases, for example, diabetes mellitus type 1, systemic lupus erythematosus (SLE), rheumatoid arthritis or Addison's disease [6].

In the early stages, the symptoms of Hashimoto's disease are nonspecific. Reported manifestations include mood swings, depression, concentration issues, along with physical changes such as dry skin, hair loss, muscle pain, increased sensitivity to cold, reduced exercise tolerance, persistent fatigue, weight gain or constipation [3,10].

The diagnosis of HT is based mainly on presence of anti-thyroid antibodies levels and hypothyroidism (based on results of TSH, T3, T4). The most reliable for HT diagnosis are serum TPO-Ab antibodies. In comparison, TPO-Ab antibodies are present in about 95% of patients, while Tg-Ab antibodies only in 60–80% percent of cases [1]. Despite the fact that there are HT ultrasonographic characteristics, they aren't specific enough to confirm diagnosis. Those HT specific features in sonographic examination include mainly heterogeneity and hypoechogenicity of the thyroid parenchyma or increased blood flow. Cytological examination is not a routine procedure, but is essential when we suspect thyroid nodules and

there is concern about malignant transformation [2].

Most patients suffering from Hashimoto's disease are required to lifelong levothyroxine substitution due to hypothyroidism [1,7]. The additional role of lifestyle, diet, vitamins, nutrients and influence of other medications is usually overlooked. This review focuses on the role of iodine in thyroid gland function and influence on the appearance and course of HT.

Iodine

Iodine is a pivotal micronutrient required for the proper function of the thyroid gland and for the development of the fetal nervous system during pregnancy [8,9]. Dietary sources rich in iodine include fish, seafood and dairy products [1,7,10,12]. To avoid the consequences of iodine deficiency, such as hypothyroidism, goiter, increased infant mortality, intellectual disability [11,12], the WHO recommends (Table 1) daily iodine intake of 150 µg/day for adults and adolescents, 250 µg/day for pregnant and lactating women, 120 µg/day for children aged 6-12 years and 90 µg/day for infants up to 6 years [13]. In many countries the problem of iodine deficiency was solved by salt iodization programs. Currently, iodized salt is a common practice in approximately 71% of the world's population [12,13]. In Poland, the mandatory iodization of salt was implemented in 1996 by the Ministry of Health [14,15]. Research indicates [15] that after the initiation of the mandatory salt iodization program in Poland, the endemic goiter in school-age children has been eliminated [15]. Moreover, the prevalence of goiter in pregnant women has decreased from 80% to 19% and the occurrence of transient hypothyroidism in newborns has declined from 2.0% to 0.16% [15]. However, the situation is different in iodine supplementation among pregnant women. Studies conducted by the Institute of Mother and Child in Warsaw and the Medical University of Lodz have shown that only about 40% of pregnant women receive the recommended additional dose of iodine [15].

On the other hand, the WHO's maximum daily salt intake recommendation is 5 g of NaCl [13,16] and not exceeding that daily dose is essential to reduce the risk of heart disease and stroke. As we know, to provide 150 µg of iodine, we need to consume 6,5 g of iodized salt [16]. To reach consensus between cardiologists and endocrinologists, the best approach is to explore alternative methods for ensuring adequate iodine intake (e.g. water iodization, milk iodization, the administration of iodine to pigs and cattle) [16].

Table 1. WHO's recommendations of iodine daily intake [13]	
Adults and adolescents	150 µg
Children aged 6-12 years	120 µg
Infants up to 6 years	90 µg
Pregnant and lactating women	250 µg

Iodine deficiency

Globally, there are geographic regions that have iodine-deficient soil. It is estimated that 29% of the world's population, residing in around 130 countries, lives in areas with iodine deficiency [12]. In these low-in-iodine regions, where salt is still not enriched in this micronutrient, the prevalence of iodine deficiency is way higher than in countries with obligation of salt iodization.

Severe iodine deficiency leads to impairment of thyroid hormones synthesis or thyroid enlargement (goiter). Population effects of IDD (Table 2), include endemic goiter, hypothyroidism, cretinism and intellectual disability [12,15]. Pregnant women and infants are particularly exposed to iodine deficiency. During pregnancy the iodine requirement is increased. The need of iodine is higher due to an increase in maternal T4 production to and transfer of thyroid hormones to the fetus and an increase in renal iodine clearance. During lactation because of excretion of iodine to breast milk, optimal daily iodine intake increases as well. During pregnancy and infancy, insufficient iodine levels may impair growth and neurodevelopment among fetuses and increase infant mortality [12].

Introducing iodized salt to areas with chronic iodine deficiency can be temporarily related with increase in cases of autoimmune thyroid disorders. However, the risks associated with excess iodine are relatively small compared to substantial risks connected with iodine deficiency [12].

Table 2. Iodine deficiency disorders by age group [11,12]	
Fetuses	Miscarriage
	Congenital defects
	Perinatal mortality
	Fetal death
Neonates	Mortality of infants
	Cretinism
Children and adolescents	Impaired cognitive function
	Physical development delay
Adults	Toxic nodular goiter
	Impaired cognitive function
	Increased prevalence of hypothyroidism (severe iodine deficiency cases)
	Decreased prevalence of hypothyroidism (mild iodine deficiency cases)
All ages	Goiter
	Increased vulnerability of the thyroid gland to nuclear radiation

Excess iodine

Despite the progress made in addressing iodine deficiency, insufficient attention is given to the risks associated with high levels of this micronutrient. There are numerous sources of iodine including iodine supplementation, diet, iodinated salt, medications containing iodine and iodinated contrast agents, which can lead to exceeding iodine physiological levels

[1,7,10,12]. It's important to note that high iodine levels in nutritional supplements often go unrecognized, because supplements are not subject to the same rigorous standards as medications. In the United States, unlike medications, dietary supplements do not require FDA (Food and Drug Administration) approval before being marketed, except for those containing new dietary ingredients [17]. Similarly, in Poland, supplements are classified as food products and are subject to milder regulations than medications [18]. What do we know so far about the impact of excessive iodine intake on thyroid function?

The Wolff-Chaikoff effect, described in 1948 by Drs. Jan Wolff and Israel Lyon Chaikoff, explains how high levels of iodine can lead to hypothyroidism. This autoregulatory mechanism clarifies the inhibition of hormone production in response to excess iodine intake. In many cases, normal synthesis of thyroxine (T4) and triiodothyronine (T3) returns after several days. However, some patients develop persistent hypothyroidism because the normal production of T3 and T4 does not regain [7,19]. This situation commonly occurs in autoimmune thyroiditis and also explains the development of hypothyroidism in patients exposed to iodine contrast media or taking iodine-containing drugs such as amiodarone [7,19].

However some patients exhibit an opposite response to excess iodine intake. In these cases, high levels of iodine can be used as a substrate for hormone production. This mechanism is known as the Jod-Basedow phenomenon and was described in the 19th century. However, unlike the Wolff-Chaikoff effect, the Jod-Basedow phenomenon does not occur in patients with normal thyroid function. It manifests in susceptible individuals with thyroid disorders, such as autoimmune diseases or nodular goiters [19,20].

Iodine and autoimmunity

Iodine is not only crucial for thyroid hormones production but also plays a role in initiating and modulating thyroid autoimmunity. Multiple studies suggest that excess iodine intake can lead to autoimmune thyroiditis (AIT) [1,3,4,7,10,19,21]. Notably, excessive iodine intake is associated with a higher incidence of AIT, whereas regions with iodine deficiency demonstrate a lower prevalence [4]. A double-blind study conducted on adults in China showed that in the group receiving pills with 400 µg of iodine, there was a higher incidence of elevated TSH concentration and subclinical hypothyroidism compared to the placebo group [22]. Another study performed on rats confirmed an increased incidence of Hashimoto's thyroiditis among rats with high iodine intake [23]. Autoimmunity has also been observed in

patients receiving even small doses of iodine in another study [24]. Following the cautious implementation of iodine fortification of salt in Denmark, studies conducted in the country revealed an increase in the prevalence of both thyroid peroxidase antibodies (TPO-Ab) and thyroglobulin antibodies (Tg-Ab) [25]. The study conducted on school-children in Brazil compared the prevalence of autoimmune thyroiditis in two different periods in which enriched salt had different concentrations of iodine. Researchers compared these results with those from a similar study that took place in a period in which iodine salt levels were higher, a tendency toward a lower prevalence of autoimmune thyroiditis was observed in the period of lower levels of iodine [26].

Several hypotheses have been proposed to explain how excess iodine may induce thyroiditis. One theory suggests that excess iodine triggers the production of cytokines and chemokines, leading to the recruitment of immune cells to the thyroid gland and initiating processes such as apoptosis or necrosis [4,6,7,10]. Additionally, the processing of excess iodine within thyroid cells can elevate oxidative stress levels and cause harmful lipid oxidation, ultimately damaging thyroid tissue [1,10]. Moreover, highly iodized thyroglobulin may become more immunogenic, exacerbating thyroid autoimmunity [1,3,4,7,10,27].

Consequently, caution is advised against excessive iodine intake, particularly in patients with Hashimoto's thyroiditis (HT), as it can exacerbate the autoimmune processes. Caution against excessive iodine supplementation should not affect its appropriate intake during pregnancy and lactating, ensuring a total daily ingestion of 250 µg.

What is interesting, the adequate selenium intake is essential for reducing the harmful impacts of iodine. The selenium deficiency can render the thyroid more susceptible to iodine-induced damage. Selenium activates regulatory cells to inhibit the release of interleukin (IL)-2, which in turn prevents autoreactive T-cells and B-lymphocytes from producing antibodies against the thyroid. Thanks to enhancing the activity of regulatory cells, selenium consequently inhibits autoimmune reactions targeting the thyroid [1,4,10].

Excess iodine in pregnancy

Iodine is crucial for the synthesis of thyroid hormones, which play a vital role in fetal growth and neurocognitive development of the fetus during pregnancy [8,9]. Higher iodine clearance during pregnancy, combined with physiological and nutritional changes, increases the risk of

iodine deficiency in pregnant women [21]. The WHO recommends daily iodine intake of 250 µg/day for pregnant and lactating [13]. What is important, 150 µg of this daily dose should be supplemented. Controlled studies conducted in regions with iodine deficiency have shown that iodine supplementation before or during early pregnancy eliminates new instances of cretinism, increases birth weight, and lowers rates of perinatal and infant mortality [8].

But what do we know about the consequences of over-supplementation of iodine among pregnant women? Meta-analysis with 8 studies included, revealed that the prevalence of excessive iodine intake in pregnant women (10,736 participants) in various parts of the world was 52% [28]. There is a relationship between excessive iodine intake and the occurrence of autoimmune thyroid diseases and hyperthyroidism in women and hypothyroidism in fetuses [29]. One study reported cases of newborns suffering from congenital hypothyroidism as a result of excess maternal iodine ingestion [30]. In another study, prophylactic iodine supplementation, administered at a daily dose of 100 or 150 µg, have revealed no harmful systemic consequences on Hashimoto's thyroiditis in a cohort of pregnant women with elevated TPO-Ab levels [31].

Therefore, it is considered safe and recommended to receive iodine supplementation (150µ g) during pregnancy and lactation, likewise women diagnosed with HT. Moreover it is important to be aware which products contain high amounts of iodine and to try to estimate the dose of daily iodine in our diet during this period to avoid overtaking this nutrient.

Conclusions

Iodine remains essential for thyroid health and fetal development, but awareness of the potential risks of excessive iodine intake is crucial for health maintenance and preventing thyroid-related disorders. Recent studies indicate that excessive iodine intake is associated with a higher prevalence of Hashimoto's thyroiditis (HT). Based on the above data, the proper solution would be keeping optimal levels of iodine to avoid both iodine deficiency disorders and thyroid autoimmune disorders. There is no indication for routine extra iodine intake in countries with programs of salt iodization. Considered as safe and recommended is only iodine supplementation in all pregnancy and lactating women, including women diagnosed with HT. The collaboration of endocrinologists, nutritionists, and other specialists is crucial

for the complex care of HT patients. Nevertheless, further research on the role of iodine and other micronutrients in the development and progression of HT is needed.

Author's contribution:

Conceptualization, Zuzanna Malinka; methodology, Zuzanna Malinka and Anna Jachymek; software, Julita Gmitrzuk and Martyna Opatowska; check, Katarzyna Wiśniewska and Marta Piotrowska; formal analysis, Tomasz Kucharski and Martyna Opatowska; investigation, Anna Jachymek and Marta Piotrowska resources, Zuzanna Malinka and Joanna Jakubiec; data curation, Anna Jachymek; writing-rough preparation, Tomasz Kucharski and Katarzyna Wiśniewska; writing review and editing, Julita Gmitrzuk and Katarzyna Wiśniewska; visualization, Zuzanna Malinka and Tomasz Kucharski; supervision, Martyna Opatowska and Joanna Jakubiec; project administration, Zuzanna Malinka and Julita Gmitrzuk. The authors have read and agreed with the published version of the manuscript.

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