

Systematic Review

A Scoping Review on the Prevalence of Hashimoto's Thyroiditis and the Possible Associated Factors

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Abstract: Background: Hashimoto's thyroiditis (HT) is the most common autoimmune thyroid disease (AITD) and is characterized by the presence of thyroid autoantibodies against thyroid peroxidase and/or thyroglobulin. Several studies have found that the global prevalence of HT has increased in recent decades, while others show the opposite. Methods and Results: The objective of this scoping review was to synthesize and analyze the different studies that have evaluated the prevalence of HT (in adults) and the possible associated factors. The following databases were consulted, as follows: MEDLINE, Web of Science, PubMed, and Scopus. The search terms "epidemiology", "prevalence", and "Hashimoto disease" and "Hashimoto thyroiditis" were used. The search was limited to articles published between January 1965 and October 2024, and only articles in English were considered. In order to reduce selection bias, each article was scrutinized using the JBI Critical Appraisal Checklist independently by two authors. Studies were included if the number of participants (study population and/or cases and controls, depending on the study design) was clearly described and duplicate studies were excluded. A total of 59 studies were identified, the vast majority of them used a cross-sectional design, using different methods of disease assessment. Conclusions: Globally, the prevalence of HT is estimated to be between 5–10%; some areas with prevalences > 20% and others < 0.5% were identified. Prevalence is also higher in women than in men. Multiple underlying factors (genetic, epigenetic, environmental, and lifestyle), together with socioeconomic, nutritional, overdiagnosis, inter alia, may explain (at least in part) the wide variability in the prevalence of HT.

Keywords: Hashimoto; thyroiditis; prevalence; autoimmunity; thyroid



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1. Introduction

Although there is no consensus on the definitions of autoimmunity and autoimmune diseases (AIDs), it is generally accepted that autoimmunity is a response involving multiple autoreactive adaptive immune components, whereas an AID involves one or more clinically evident pathologies [1].

AIDs are caused by a combination of multiple genetic, epigenetic, environmental, and lifestyle factors. The most prominent immunological manifestation of AIDs is the

production of autoantibodies (Abs), which are the most valuable biomarkers for diagnosis (and even for disease classification and activity) [1–4].

Classically, it has been established that AIDs can be restricted according to the pattern of organic involvement (e.g., organ-specific) or be generalized or systemic (non-organ-specific). In this sense, autoimmune thyroid diseases (AITDs) are organ-specific AIDs [3–5].

AITDs are a group of diseases characterized by the breakdown of tolerance to thyroid autoantigens (specifically, thyroid peroxidase—TPO; thyroglobulin—Tg; and the thyrotropin receptor—TR), which induce both humoral and cellular autoimmune responses, with the presence of circulating Abs to thyroid autoantigens (TgAbs, TPOAbs, and TRAbs) and lymphocytic infiltration [6,7].

The clinical spectrum of AITDs includes Hashimoto’s thyroiditis (HT), Graves–Basedow disease, postpartum thyroiditis, drug-induced thyroiditis, thyroiditis associated with polyglandular syndromes, and atrophic thyroiditis [6–8]. Among all AITDs, HT is the most common, and it often manifests as hypothyroidism (subclinical or overt) as a consequence of thyrocyte destruction (a phenomenon mediated by both humoral and cellular immunity) [6,9,10].

HT is the main cause of hypothyroidism in iodine-sufficient areas of the world [11]. Its global prevalence varies depending on various factors (e.g., socioeconomic, environmental, and genetic) and is estimated at 4.8–25.8% and 0.9–7.9% in men and women, respectively [11–13]. However, some studies have reported an increase in the prevalence of HT in recent decades, while other studies have shown a slight decrease [11–13].

The objectives of this study are to determine the global prevalence of HT and analyze the possible factors that may influence the population variability of the disease.

2. Materials and Methods

This scoping review is part of a larger research project investigating and evaluating the distribution and behavior of AITDs in Colombia. The protocol was developed in accordance with the principles of the Declaration of Helsinki and approved by the Research Ethics Committee of the University of Cauca, Colombia (ID: 4656, January 2018).

2.1. Literature Search and Selection Criteria

A comprehensive and sensitive search was performed in the MEDLINE, Web of Science, PubMed, and Scopus databases. The search was limited to articles published between January 1965 and October 2024 (adults, humans, clinical trials, meta-analyses, reviews, and systematic reviews), and only articles in English were considered.

The search criteria were as follows: Epidemiology [Title/Abstract] OR Prevalence [Title/Abstract] AND Hashimoto Disease [Mesh] OR Hashimoto Thyroiditis [Title/Abstract] OR Chronic Lymphocytic Thyroiditis [Title/Abstract] OR Lymphocytic Thyroiditis, Chronic [Title/Abstract] OR Thyroiditis, Chronic Lymphocytic [Title/Abstract] OR Hashimoto’s Disease [Title/Abstract] OR Disease, and Hashimoto’s [Title/Abstract] OR Autoimmune thyroiditis [Title/Abstract].

Studies were included if the number of participants (study population and/or cases and controls, depending on the study design) was clearly described, and duplicate studies were excluded (Figure 1).

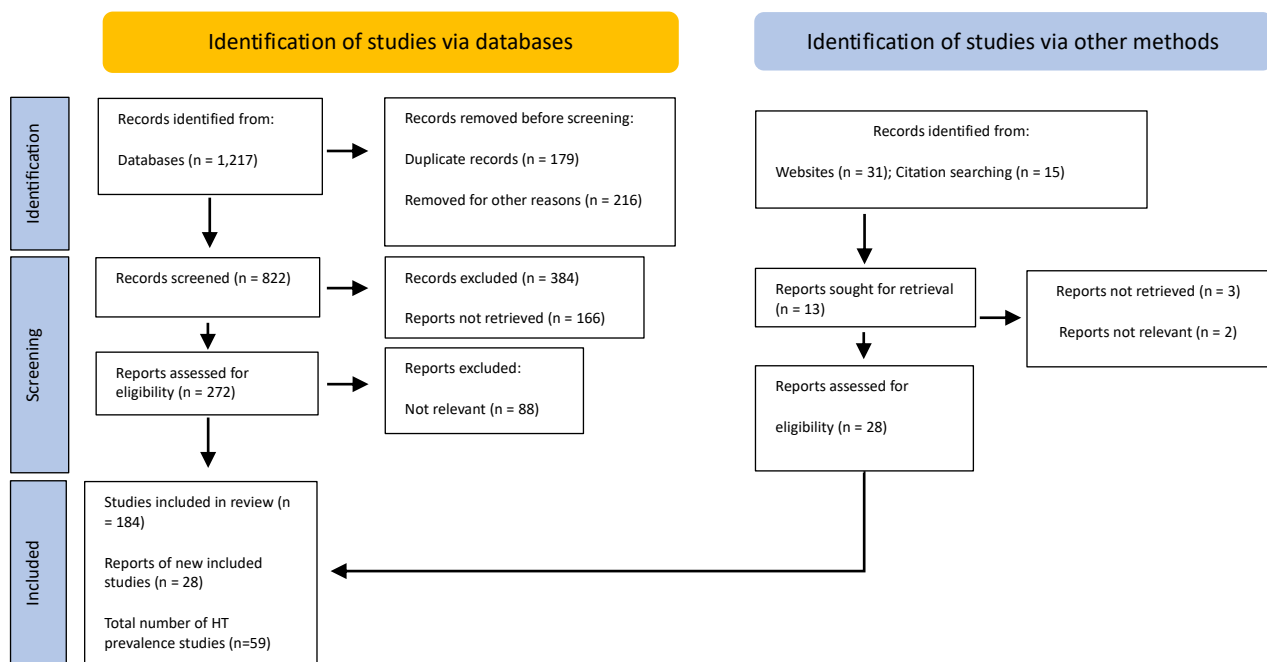


Figure 1. A PRISMA flow diagram. The method for the selection of articles.

2.2. Data Extraction

To reduce selection bias, each article was scrutinized according to the JBI Critical Appraisal Checklist [14]. The identification and selection of studies were carried out by two authors (H V-U and H V-S). Data were extracted using a standardized template using a predefined data form created in Excel, and in cases where discrepancies arose in the extracted data, they collaboratively conducted a second round of extraction to validate the accuracy of the information.

2.3. Data Analysis

The following data were retrieved as follows: authors, year of publication, country (income group), number of cases and population evaluated, study design, diagnostic criteria, and prevalence of HT (95% CI). No statistical analysis or meta-analysis was performed due to the high heterogeneity observed among the studies included in this review. However, we conducted a descriptive analysis to summarize key study characteristics. This scoping review was registered on the “International Platform of Registered Systematic Review and Meta-Analysis Protocols INPLASY” (registration number: INPLASY202530049; DOI number: 10.37766/inplasy2025.3.0049) and followed the “Reporting Checklist for Systematic Review. Based on the PRISMA guidelines”.

3. Results

3.1. Origins of Studies and Diagnostic Criteria Used

A total of 59 studies were identified (25 in Europe, 5 in Africa and Oceania, 19 in Asia, 4 in North America, and 6 in South America) [15–73]. Fifty studies used a cross-sectional design, while two were cohort studies, and seven were array research. The methods used for HT diagnosis were as follows: serum Abs; thyroid ultrasound (TU); thyroid tissue; serum Abs + TU; serum Abs + TU + fine-needle aspiration (FNA); and thyroid tissue + Abs + TU. In four studies, the diagnostic method used was not reported.

The 59 identified studies were conducted in 25 countries: 13 were high income (HI) (England, Finland, Norway, Germany, Italy, Denmark, Spain, Poland, Croatia, Australia, Japan, South Korea, and USA); 7 were upper-middle income (UMI) (Brazil, China, Russia,

Bosnia and Herzegovina, Jordan, Mexico, and Colombia); and 5 were lower-middle income (LMI) (Tunisia, Nigeria, Ghana, Iran, and Sri Lanka) [15–73].

3.2. Global Prevalence of HT

Globally, the prevalence of HT is between 5% and 10%; however, prevalence rates were widely variable between continents, between countries, and even between the regions studied (within countries) [10–13]. The prevalence is also higher in women than in men (between 2.0 and 7.0 times higher) [10,11,13].

3.3. Prevalence of HT in Europe

In Europe, prevalence rates as low as 0.42% and 0.43% were found in Russia and in Bosnia and Herzegovina, respectively, and very high prevalence rates were found in Italy (35.5%) and Denmark (39.7%), while other studies conducted in these same countries found lower prevalence rates [15–39] (Table 1).

Table 1. The global prevalence of Hashimoto’s thyroiditis in Europe (n = 25 studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto’s Thyroiditis (95% CI)
Dingle PR, et al. (1966) [15]	England (HI)	52/469	Cross-sectional (PBS)	Serum (Abs)	11 (8.4–14.3)
Jacobs A, et al. (1969) [16]	England (HI)	99/989	Cross-sectional (PBS)	Serum (Abs)	10 (8.2–12.1)
Tunbridge WM, et al. (1977) [18]	England (HI)	56/2779	Cross-sectional	Serum (Abs)	2.0 (1.5–2.6)
Prentice LM, et al. (1990) [17]	England (HI)	124/698	Cross-sectional (PBS)	Serum (Abs)	17.8 (15–20.8)
Aho K, et al. (1971) [19]	Finland (HI)	89/1137	Cross-sectional (CBS)	Serum (Abs)	7.8 (6.3–9.5)
Gordin A, et al. (1972) [20]	Finland (HI)	282/2961	Cross-sectional (PBS)	Serum (Abs)	9.5 (8.5–10.6)
Bjoro T, et al. (1984) [21]	Norway (HI)	56/1643	Cross-sectional (PBS)	Serum (Abs)	3.4 (2.6–4.4)
Bryhni B, et al. (1996) [22]	Norway (HI)	176/2551	Cross-sectional (PBS)	Serum (Abs)	6.9 (5.9–8.0)
Völzke H, et al. (2003) [23]	Germany (HI)	47/3941	Cross-sectional	Serum (Abs)	1.2 (0.9–1.6)
Döbert N, et al. (2008) [24]	Germany (HI)	98/700	Cross-sectional (PBS)	Serum (Abs + TU)	14 (11.5–16.8)
Khattak RM, et al. (2016) [25]	Germany (HI)	358/4420	Cohort	Serum (Abs)	8.1 (7.3–8.9)
Aghini-Lombardi F, et al. (1999) [26]	Italy (HI)	50/1411	Cross-sectional (PBS)	Serum (Abs)	3.5 (2.6–4.6)
Benvenega S, et al. (2008) [27]	Italy (HI)	4064/23,000	Array research (CBS)	Serum, thyroid tissue (Abs + TU + FNA)	17.7 (17.2–18.2)
Sardu C, et al. (2012) [28]	Italy (HI)	678/25,885	Cross-sectional (PBS)	NR (NR)	2.6 (2.4–2.8)
Aghini-Lombardi F, et al. (2013) [29]	Italy (HI)	224/1065	Cross-sectional (PBS)	Thyroid tissue (Abs + TU)	21 (18.6–23.6)
Tammaro A, et al. (2016) [30]	Italy (HI)	2828/7976	Array research (PBS)	Serum (Abs)	35.5 (34.4–36.5)
Pilli T, et al. (2019) [31]	Italy (HI)	9/142	Cross-sectional	Serum (Abs)	6.3 (2.9–11.7)

Table 1. Cont.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto's Thyroiditis (95% CI)
Pedersen IB, et al. (2003) [32]	Denmark (HI)	787/4184	Cross-sectional (PBS)	Serum (Abs)	18.0 (17.6–20.0)
Pedersen IB, et al. (2011) [33]	Denmark (HI)	778/3570	Cross-sectional (PBS)	Serum (Abs)	21.8 (20.4–23.1)
Møllehave LT, et al. (2024) [34]	Denmark (HI)	979/2465	Cross-sectional (PBS)	Serum (Abs)	39.7 (37.8–41.6)
Valdés S, et al. (2017) [35]	Spain (HI)	391/4554	Cross-sectional (PBS)	Serum (Abs)	8.6 (7.8–9.4)
Józków P, et al. (2017) [36]	Poland (HI)	29,375/586,703	Cross-sectional (CBS)	Serum (Abs)	5.0 (5.0–5.1)
Troshina EA, et al. (2021) [37]	Russia (UMI)	428/100,000	Cross-sectional (PBS)	Serum (Abs)	0.42 (0.38–0.46)
Izic B, et al. (2021) [38]	Bosnia and Herzegovina (UMI)	358/82,000	Array research (PBS)	Serum (Abs)	0.43 (0.39–0.48)
Strikić Đula I, et al. (2022) [39]	Croatia (HI)	1044/4402	Cross-sectional (PBS)	Serum (Abs)	23.7 (22.4–25.9)

Abbreviations: Abs: antibodies; CBS: clinic-based study; HI: high income; NR: not reported; PBS: population-based study; TU: thyroid ultrasound; UMI: upper-middle income.

3.4. Prevalence of HT in Africa

Similarly, the prevalence in Africa was also variable, with the highest prevalence in Tunisia (22.8%) and the lowest in Nigeria (6.7%) [40–42] (Table 2).

Table 2. The global prevalence of Hashimoto’s thyroiditis in Africa (n = 3 studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto's Thyroiditis (95% CI)
Chabchoub G, et al. (2006) [40]	Tunisia (LMI)	246/1079	Array research (CBS)	Serum (Abs)	22.8 (20.3–25.4)
Okosieme OE, et al. (2007) [41]	Nigeria (LMI)	7/104	Cross-sectional (CBS)	Serum (Abs)	6.7 (2.7–13.4)
Sarfo-Kantanka O, et al. (2017) [42]	Ghana (LMI)	583/8099	Retrospective cohort (HBS)	Serum (Abs + TU)	7.2 (6.6–7.8)

Abbreviations: Abs: antibodies; CBS: clinic-based study; HBS: hospital-based data; LMI: low-middle income; TU: thyroid ultrasound.

3.5. Prevalence of HT in Oceania

The studies conducted in Oceania (Australia) reported prevalences of 13.1% and 8.6% [43,44] (Table 3).

Table 3. The global prevalence of Hashimoto’s thyroiditis in Oceania (n= 2 studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto's Thyroiditis (95% CI)
O’Leary PC, et al. (2006) [43]	Australia (HI)	282/2115	Cross-sectional health survey	Serum (Abs)	13.3 (11.8–14.8)
Deshpande P, et al. (2016) [44]	Australia (HI)	17/198	Cross-sectional (PBS)	Serum (Abs)	8.6 (5.1–13.4)

Abbreviations: Abs: antibodies; HI: high income; PBS: population-based study.

3.6. Prevalence of HT in Asia

In Asia, prevalence rates as low as 0.1% in South Korea and 0.3% in China were found; however, in China, the reported prevalence rates differed substantially, depending on the area studied. The highest prevalence rates were documented in Japan (18%), China (16.1%), and Jordan (15.1%) [45–63] (Table 4).

Table 4. The global prevalence of Hashimoto’s thyroiditis in Asia (n = 19 studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto’s Thyroiditis (95% CI)
Okayasu I, et al. (1991) [45]	Japan (HI)	328/1826	Cross-sectional	Thyroid tissue	18 (0.16–0.19)
Konno N, et al. (1993) [46]	Japan (HI)	457/4110	Cross-sectional (PBS)	Serum (Abs)	11.1 (10.2–12.1)
Morinaka S, et al. (1995) [47]	Japan (HI)	61/6348	Cross-sectional (CBS)	Serum (Abs) + TU + FNA	1.0 (0.7–1.2)
Nagata K, et al. (1998) [48]	Japan (HI)	142/1039	Cross-sectional (PBS)	Serum (Abs)	13.7 (11.6–15.9)
Kurata S, et al. (2007) [49]	Japan (HI)	25/1626	Cross-sectional (CBS)	Serum, thyroid tissue (Abs + TU + FNA)	1.5 (1.0–2.3)
Teng W, et al. (2006) [50]	China (UMI)	32/3761	Cross-sectional study	Serum (Abs)	0.9 (0.6–1.2)
Teng X, et al. (2008) [51]	China (UMI)	67/778	Array research (PBS)	Serum (Abs + TU)	8.6 (6.7–10.8)
Li Y, et al. (2008) [52]	China (UMI)	353/3018	Cross-sectional (PBS)	Serum (Abs) + TU	11.7 (10.6–12.9)
Teng X, et al. (2011) [53]	China (UMI)	363/3813	Cross-sectional	Serum (Abs) + TU	9.5 (8.6–10.5)
Wu Q, et al. (2015) [54]	China (UMI)	172/6152	Cross-sectional (PBS)	Serum (Abs)	2.8 (2.4–3.2)
Gu F, et al. (2016) [55]	China (UMI)	17/5293	Cross-sectional (PBS)	Serum (Abs) + TU	0.3 (0.2–0.5)
Li Y, et al. (2016) [56]	China (UMI)	187/2856	Array research	Serum (Abs)	6.5 (5.7–7.5)
Wan S, et al. (2020) [57]	China (UMI)	198/1225	Cross-sectional survey	Serum (Abs)	16.1 (14.1–18.2)
Chen Y, et al. (2021) [58]	China (UMI)	298/2946	Cross-sectional (PBS)	Serum (Abs) + TU	10.1 (9.0–11.3)
Yu Z, et al. (2021) [59]	China (UMI)	148/1159	Cross-sectional (PBS)	Thyroid tissue	12.8 (10.9–14.8)
Fernando RF, et al. (2012) [60]	Sri Lanka (LMI)	353/5200	Cross-sectional (PBS)	Serum (Abs)	6.8 (6.1–7.5)
Amouzegar A, et al. (2017) [61]	Iran (LMI)	742/5783	Cross-sectional (PBS)	Serum (Abs)	12.8 (12–13.7)
Kim HJ, et al. (2021) [62]	South Korea (HI)	29,429/21,705,883	Array research (PBS)	NR (NR)	0.1 (0.1–0.1)
Ajlouni KM, et al. (2022) [63]	Jordan (UMI)	567/3753	Cross-sectional (PBS)	Serum (Abs)	15.1 (13.9–16.2)

Abbreviations: Abs: antibodies; CBS: clinic-based study; FNA: fine needle aspiration; HI: high income; LMI: low-middle income; NR: not reported; PBS: population-based study; TU: thyroid ultrasound; UMI: upper-middle income.

3.7. Prevalence of HT in North America and South America

Finally, in North America and South America, the lowest prevalences were found in the USA (0.4%) and Brazil (0.1%), respectively, although some studies carried out in the USA showed very high prevalences. The highest prevalences documented were in the USA (22.4%) and in Colombia (22.3%) [64–73] (Tables 5 and 6).

Table 5. The global prevalence of Hashimoto’s thyroiditis in North America (four studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto’s Thyroiditis (95% CI)
Okayasu I, et al. (1994) [64]	USA (HI)	457/2040	Cross-sectional (PBS)	Thyroid tissue (pathological section)	22.4 (20.6–24.3)
Flores-Rebollar A, et al. (2015) [65]	Mexico (UMI)	36/427	Cross-sectional (PBS)	Serum (Abs) + TU	8.4 (6.0–11.5)
Caturegli G, et al. (2016) [66]	USA (HI)	4/1075	Cross-sectional (PBS)	NR (NR)	0.4 (0.1–0.91)
Zhang X, et al. (2024) [67]	USA (HI)	4454/33,117	Cross-sectional study	Serum (Abs)	13.4 (13.1–13.8)

Abbreviations: Abs: antibodies; HI: high income; NR: not reported; PBS: population-based study; TU: thyroid ultrasound; UMI: upper-middle income.

Table 6. The global prevalence of Hashimoto’s thyroiditis in South America (n = 6 studies), according to country, income group, study design, and sample type.

Author (Year) [Ref.]	Country (Income Group)	Case/Total	Study Design	Diagnostic Criteria	% Prevalence of Hashimoto’s Thyroiditis (95% CI)
Tomimori E, et al. (1995) [68]	Brazil (UMI)	72/547	Cross-sectional (PBS)	TU	13.2 (10.4–16.3)
Camargo RY, et al. (2006) [69]	Brazil (UMI)	82/420	Cross-sectional	Serum (Abs) + TU	19.5 (15.8–23.6)
Camargo RY, et al. (2008) [70]	Brazil (UMI)	183/1085	Cross-sectional (PBS)	Serum (Abs)	16.9 (14.7–19.2)
Vecchiatti SM, et al. (2015) [71]	Brazil (UMI)	106/4613	Cross-sectional (CBS)	Thyroid tissue	2.3 (1.9–2.8)
Tolentino Júnior DS, et al. (2019) [72]	Brazil (UMI)	85/60,413	Cross-sectional	NR	0.1 (0.1–0.2)
Vargas-Uricoechea, H, et al. (2023) [73]	Colombia (UMI)	2150/9638	Cross-sectional (PBS)	Serum (Abs)	22.3 (20.6–24.0)

Abbreviations: Abs: antibodies; CBS: clinic-based study; NR: not reported; PBS: population-based study; TU: thyroid ultrasound; UMI: upper-middle income.

4. Discussion

The prevalence of HT worldwide is widely variable between different continents, between different countries, and even within the same country.

The variability found in the prevalence of HT has also been demonstrated in multiple AIDs and could be explained by a constellation of factors [14], among which the following stand out:

4.1. Sex and Age

A characteristic of many AIDs is that they are more frequent in women and become more common as the age of the population increases. A similar phenomenon occurs in HT: multiple studies have shown that the diagnosis of hypothyroidism (due to HT) is more frequent in women than in men, and the incidence of diagnosis increases with age (with a high proportion of TPOAb and/or TgAb positivity). In fact, age and the presence of thyroid Abs have been identified as predictive factors for hypothyroidism in older people [39,74–79].

4.2. Socioeconomic Status and Disparities in Health Care and Ethnicity

In general terms, the most disadvantaged socioeconomic groups have a higher frequency of AIDs, which suggests a socioeconomic gradient that may explain the variability

in these diseases (including HT). In addition, in populations where there are disparities in health care and in those where there are racial and ethnic minorities, differences are found not only in the diagnostic procedures of different diseases but also in the results of treatment and prognosis [80–84]. It is thus possible that the population prevalence of HT may be affected by large socioeconomic disparities (or the presence of ethnic minorities).

4.3. Seasonality

An area of uncertainty has been the possible association between the month of birth and the risk of HT. Some studies have found significant differences in the month of birth between individuals with HT and controls; that is, individuals with HT were more likely to be born in summer (June–August), supporting the hypothesis that seasonal variations may contribute to the development of HT. Other studies have analyzed the seasonal pattern of the birth months of young patients with HT, finding significant differences among those with HT; e.g., in a month-by-month analysis, the highest and lowest predispositions (for HT) were among those born in spring (March) and autumn (November), respectively. Season type (at birth) and sex have also been found to be factors related to HT, even after adjustment for other factors, such as the year of birth and age [85–88].

4.4. Racial Variations and Genetic Polymorphisms

Several susceptibility genes have been implicated in the risk of developing AITDs, e.g., genes specific to the thyroid or those that interfere with immunomodulation phenomena. Single-nucleotide polymorphisms (SNPs) in these genes may affect both the regulation of central or peripheral tolerance and the processes of T-cell activation and antigen presentation, inducing an AITD. Therefore, these SNPs may explain the phenotypic diversity among individuals affected by HT. Some studies have suggested that racial variations in the prevalence of AITDs accentuate possible SNPs and increase genetic susceptibility to HT (although it is widely accepted that combinations of different SNPs can increase the risk of AITDs, they can also have the opposite effect) [89–92].

4.5. The Kaleidoscope of Autoimmunity

The kaleidoscope of autoimmunity indicates the possible variations in the clinical spectrum of an AID (or the probability that an AID may co-occur in the same individual (polyautoimmunity) or that several AIDs coexist in the same family (familial autoimmunity)). In this sense, HT is one of the AIDs that are likely to co-occur with other AIDs (e.g., rheumatoid arthritis, diabetes mellitus type 1, and systemic lupus erythematosus). The risk of co-occurring AIDs is greater than expected by chance, and the presence of an AID can be a predictor of a second AID; thus, in the study of any AID, the AID itself increases the likelihood of identifying one or more other AIDs, which can affect the prevalence of other diseases with a similar autoimmune basis (e.g., HT) [93–96].

4.6. Obesity

Obesity may exacerbate the progression and severity of several AIDs. Although the mechanisms linking BMI to HT are not entirely clear, some studies suggest a possible association between a high BMI and HT [97–99]. Thus, considering that global adult obesity has more than doubled since 1990 and that, in 2022, 2.5 billion adults were overweight (and of these, 890 million were obese), it can be argued that changes in HT prevalence may be somewhat influenced by the overweight/obesity pandemic [100–103].

4.7. Environmental Factors and Contaminants

It is widely accepted that a number of environmental factors, including climate change, pollution, nutritional changes, and thyroidal endocrine-disrupting chemicals, affect thyroid

function [104,105]. Some studies have found associations between environmental factors, contaminants, endocrine disruptors, and AITDs. Therefore, the variability in the global prevalence of HT may be influenced by these factors [106–110].

4.8. Smoking and Alcohol Intake

Traditionally, smoking has been considered to affect thyroid function and to be capable of inducing AITDs, as well as other AIDs; however, such effects are controversial. To date, it has been difficult to establish a link between smoking and HT, especially because this influence is exerted through multiple mechanisms that can be modified by several factors (age, sex, ethnicity, and iodine status) [111–115]. Current evidence suggests that smoking is associated with a decrease in TSH levels and an increase in TPOAb levels (especially in men) [114,116–118]. For this reason, the prevalence of HT could be influenced by the frequency of smoking in the individuals evaluated.

It has also been suggested that alcohol consumption reduces T4 levels and that T4 and T3 levels decrease in alcoholic individuals after alcohol abstinence. Since smoking frequently coincides with alcohol intake, it is necessary to take alcohol consumption into account when evaluating the association between smoking and AITDs [114,119–121]. However, studies evaluating the effect of smoking on thyroid function (or thyroid autoimmunity) have not determined whether such associations are independent of alcohol consumption and/or other factors.

4.9. Iodine Intake and Population Iodine Status

Disorders associated with iodine intake (especially iodine deficiency disorders (IDDs)) are highly frequent worldwide; universal salt iodization (UIS) programs were designed as a cost-effective strategy for the prevention of IDD [122]. However, these programs are not always carried out permanently or systematically, which explains why there are still countries with iodine deficiency or excess in the population. This has led to an increase in the frequency of functional and immunological thyroid disorders (and probably thyroid cancer) [123–125]. Some studies have shown an increase in the prevalence of thyroid TPOAbs and/or TgAbs during the first years of implementation of UIS programs, while others have found a decrease in prevalence when UIS programs are sustained over time. These findings are mainly described in areas with prolonged iodine deficiency (and that implemented USI programs) [126]. Therefore, population iodine status should be considered a factor influencing the prevalence of HT.

4.10. The Population Status of Other Micronutrients

Several studies have found a possible association between deficiencies of some micronutrients (selenium, iron, copper, magnesium, vitamin B12, vitamin D, etc.) and a higher frequency of TPOAb and TgAb positivity; in fact, the risk increases as the micronutrient deficiency becomes more severe [127–132]. Despite this, intervention studies have been contradictory, as correction or supplementation with the deficient micronutrient does not always translate into a decrease in the risk of AITDs or in the level of thyroid Abs [133–139]. Based on the above and the fact that micronutrient deficiencies affect a large proportion of the world population, such deficiencies may affect the prevalence of HT [140–144], especially since famines and undernutrition/malnutrition remain unacceptably high; e.g., around 733 million people in the world were found to be undernourished in 2023 (9% of the world population). This represents an increase of 36% compared to 2014 (almost 539 million people affected) [145]. Hence, the nutritional component of the world population is associated with the population status of micronutrients, some of which have been shown to be associated with HT.

4.11. Overdiagnosis

The frequency of hypothyroidism increases with age, and its main causes globally are iodine deficiency and HT [146,147]. Therefore, the high prevalence of IDD and thyroid Ab positivity clearly sets a precedent when analyzing the global prevalence of HT; however, as the symptoms of hypothyroidism may overlap with those of other chronic diseases, it is more likely that thyroid function tests (and measurements of thyroid Abs) are performed indiscriminately in individuals with such symptoms, identifying the disease (HT) earlier; it may even lead to overtreatment (by giving treatment with LT4) in individuals with subclinical hypothyroidism when it is probably not required [148–151]. This also extends to women who wish to become pregnant (or those already pregnant), where overscreening frequently occurs in the search for autoimmunity and/or thyroid functional disorders, which may modify the overall prevalence of HT [13,152–154].

4.12. Temporal Trends

The frequency of the diagnosis of hypothyroidism (and HT) has changed over time and varies according to the geographical area studied. While some studies show a decrease in frequency, others report an increase [155–157]. The increase in frequency can be explained by the increase in the diagnosis of most AIDs (due to a real increase in frequency, the greater availability of laboratory tests focused on the diagnosis of autoimmunity, or overscreening/overdiagnosis, especially in the approach to hypothyroidism/HT) [158–161]. Meanwhile, the decrease in frequency could be explained (at least in part) by different international guidelines and recommendations (in the last two decades) aimed at preventing the overdiagnosis of hypothyroidism by establishing a diagnostic approach based on the TSH value, which was previously not carried out routinely before starting treatment with LT4. These recommendations were made with the aim of reducing LT4 overtreatment, and the effect took several years to become evident [162–165]. For example, a study evaluating the initiation of new LT4 prescriptions (between 2006 and 2012) found that about 30% of these treatments were not based on a previous TSH measurement; additionally, these patients were started on LT4 replacement therapy according to the symptoms that they manifested (such as weight gain, constipation, fatigue, muscle weakness, or hair loss) [166]. This overdiagnosis/overtreatment may (falsely) increase the prevalence of hypothyroidism/HT; in fact, a greater overtreatment has been documented in older adults, which may also affect the prevalence in younger individuals. Based on this aspect, international recommendations require a reasonable time to be established in the international medical community (and may explain the temporal trend showing a decrease in the prevalence of HT in several countries) [166].

4.13. Temporal Trends in Thyroid Cancer

The incidence of thyroid cancer (TC) is increasing annually worldwide, with a growing burden. Several reasons for the increase in the frequency of TC have been put forward, including overdiagnosis and a greater access to imaging techniques [167–169]. In general terms, the evaluation of patients with a suspected or confirmed diagnosis of TC requires the assessment of thyroid function (and, in a significant proportion of them, the value of Tg and thyroid Abs), which may increase the probability of identifying an AITD earlier (with or without thyroid functional alterations) and may eventually affect the prevalence of HT.

4.14. Other Factors

Other factors may affect the prevalence of HT. For example, COVID-19/SARS-CoV-2 vaccines have been associated with an increase in both TPOAb and TgAb levels, although

the findings have been inconsistent across studies. Similarly, other viral infections have also been associated (Epstein–Barr, parvovirus B19, herpesvirus 6A, enterovirus, chronic HBV and HCV infections, etc.) with HT [170–174]. Meanwhile, some nutritional components, especially with the rise of “fad diets” (specifically those free of gluten and/or lactose), have been suggested to affect the natural course and prevalence of HT [174]. Therefore, infectious (viral) and nutritional components may be affecting the prevalence of HT in different clinical settings.

4.15. Diagnostic Criteria and the Definition of the Disease

The diagnostic criteria for HT have not been universally accepted in the studies evaluated in this review. This aspect affects the overall prevalence of the disease, since the results expressed as diagnoses based on only the levels of TPOAbs and TgAbs are different from those in studies with diagnostic criteria based on thyroid tissue samples or TU (or with a combination of evaluation methods) [175].

4.16. Drugs

Some medications may be associated with an increased risk of AITDs or thyroid function disorders; e.g., the increasingly widespread use (in various clinical settings) of medications such as amiodarone, tyrosine kinase inhibitors, immune checkpoint inhibitors therapy, PD-(L)1-Ab, lithium, interferon- α , and so on may alter the prevalence of HT. Since there is such a risk (albeit small) of these disorders, it is more feasible that individuals receiving these medications undergo thyroid function tests and thyroid Abs [176–178].

It is difficult to accurately determine the directionality and magnitude of factors that may affect the overall prevalence of HT; e.g., the vast majority of the studies did not evaluate or stratify the target population according to these factors, nor did they evaluate the possible interactions between them (which clearly exist, including in the context of an AID) (Figure 2).

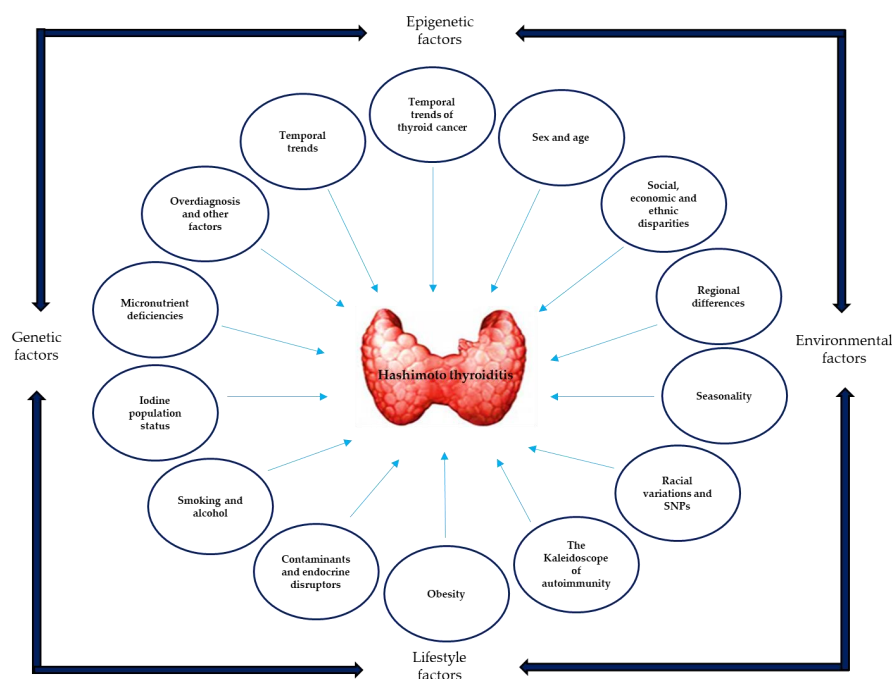


Figure 2. The conceptual framework of HT has a genetic/epigenetic basis, with an environmental and lifestyle component; the prevalence of HT is widely variable, probably influenced by multiple factors, which can interact with each other, substantially modifying the worldwide distribution of the disease.

Finally, other less studied factors that may influence the prevalence of HT should also be taken into account, such as stress, alterations in the intestinal microbiome, and the presence of other infections (bacterial, fungal, or parasitic) [179,180]. However, although biologically plausible, in the sense that these factors are capable of stimulating an autoimmune response (and increasing the risk of HT), studies evaluating this aspect are scarce.

5. Strengths and Weaknesses of the Present Review

A notable strength of this scoping review is the exhaustive process used to collect data and select the studies reviewed. This involved an extensive search of bibliographic evidence in four major databases (MEDLINE, Web of Science, PubMed, and Scopus), allowing for a sensitive and robust search, offering a broad and accurate view of the topic. The geographical representation included studies from Europe, Africa, Asia, Oceania, North America, and South America, providing a broader view of the distribution of HT prevalence worldwide.

Likewise, several weaknesses are also evident, e.g., the language limitation regarding the choice of studies, the heterogeneity found, and the fact that there were several studies in which the diagnostic method used was not described; additionally, the evaluation (and diagnostic) criteria were also different between the studies. These considerations precluded a meta-analysis of the available information; however, we tried to mitigate these aspects by showing the results in a narrative and condensed manner according to variables such as country, continent, income group, study design, and the diagnostic method used.

Future Implications

Large-scale, population-based studies should be conducted to analyze possible confounding factors and interactions between them in different geographic, social, economic, ethnic minority, and other settings. In light of current evidence, the results of studies that have evaluated the prevalence of HT should only be extrapolated to similar populations.

6. Conclusions

In summary, the global prevalence of HT is widely variable, estimated to be between 5% and 10%, with areas showing prevalences as high as >20% and as low as <0.5%. This prevalence is higher in women than in men. Multiple factors, such as diet, socioeconomics, diet, racial disparities and variations, and overdiagnosis, can affect the global prevalence of HT. These factors, combined with genetic/epigenetic predisposition and other environmental and lifestyle factors, form the conceptual framework of this disease.

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References

1. Pisetsky, D.S. Pathogenesis of autoimmune disease. *Nat. Rev. Nephrol.* **2023**, *19*, 509–524. [[CrossRef](#)] [[PubMed](#)]
2. Rosenblum, M.D.; Remedios, K.A.; Abbas, A.K. Mechanisms of human autoimmunity. *J. Clin. Investig.* **2015**, *125*, 2228–2233. [[CrossRef](#)] [[PubMed](#)]
3. Bieber, K.; Hundt, J.E.; Yu, X.; Ehlers, M.; Petersen, F.; Karsten, C.M.; Köhl, J.; Kridin, K.; Kalies, K.; Kasprick, A.; et al. Autoimmune pre-disease. *Autoimmun. Rev.* **2023**, *22*, 103236. [[CrossRef](#)]
4. Theofilopoulos, A.N.; Kono, D.H.; Baccala, R. The multiple pathways to autoimmunity. *Nat. Immunol.* **2017**, *18*, 716–724. [[CrossRef](#)]
5. Samuels, H.; Malov, M.; Saha Detroja, T.; Ben Zaken, K.; Bloch, N.; Gal-Tanamy, M.; Avni, O.; Polis, B.; Samson, A.O. Autoimmune Disease Classification Based on PubMed Text Mining. *J. Clin. Med.* **2022**, *11*, 4345. [[CrossRef](#)] [[PubMed](#)]
6. Vargás-Uricoechea, H. Molecular Mechanisms in Autoimmune Thyroid Disease. *Cells* **2023**, *12*, 918. [[CrossRef](#)]
7. Tywanek, E.; Michalak, A.; Świrska, J.; Zwolak, A. Autoimmunity, New Potential Biomarkers and the Thyroid Gland—The Perspective of Hashimoto’s Thyroiditis and Its Treatment. *Int. J. Mol. Sci.* **2024**, *25*, 4703. [[CrossRef](#)]
8. Petranović Ovčariček, P.; Görges, R.; Giovanella, L. Autoimmune Thyroid Diseases. *Semin. Nucl. Med.* **2024**, *54*, 219–236. [[CrossRef](#)]
9. Ajjan, R.A.; Weetman, A.P. The Pathogenesis of Hashimoto’s Thyroiditis: Further Developments in our Understanding. *Horm Metab Res.* **2015**, *47*, 702–710. [[CrossRef](#)]
10. Asa, S.L.; Erickson, L.A.; Rindi, G. The Spectrum of Endocrine Pathology. *Endocr. Pathol.* **2023**, *34*, 368–381. [[CrossRef](#)]
11. Vanderpump, M.P. The epidemiology of thyroid disease. *Br. Med. Bull.* **2011**, *99*, 39–51. [[CrossRef](#)] [[PubMed](#)]
12. Conrad, N.; Misra, S.; Verbakel, J.Y.; Verbeke, G.; Molenberghs, G.; Taylor, P.N.; Mason, J.; Sattar, N.; McMurray, J.J.V.; McInnes, I.B.; et al. Incidence, prevalence, and co-occurrence of autoimmune disorders over time and by age, sex, and socioeconomic status: A population-based cohort study of 22 million individuals in the UK. *Lancet* **2023**, *401*, 1878–1890. [[CrossRef](#)]
13. Hu, X.; Chen, Y.; Shen, Y.; Tian, R.; Sheng, Y.; Que, H. Global prevalence and epidemiological trends of Hashimoto’s thyroiditis in adults: A systematic review and meta-analysis. *Front. Public. Health* **2022**, *10*, 1020709. [[CrossRef](#)]
14. Aromataris, E.; Fernandez, R.; Godfrey, C.; Holly, C.; Kahlil, H.; Tungpunkom, P. Summarizing systematic reviews: Methodological development, conduct and reporting of an Umbrella review approach. *Int. J. Evid. Based Healthc.* **2015**, *13*, 132–140. [[CrossRef](#)] [[PubMed](#)]
15. Dingle, P.R.; Ferguson, A.; Horn, D.B.; Tubmen, J.; Hall, R. The incidence of thyroglobulin antibodies and thyroid enlargement in a general practice in northeast England. *Clin. Exp. Immunol.* **1966**, *1*, 277–284. [[PubMed](#)]
16. Jacobs, A.; Entwistle, C.C.; Campbell, H.; Waters, W.E. A random sample from Wales. IV. Circulating gastric and thyroid antibodies and antinuclear factor. *Br. J. Haematol.* **1969**, *17*, 589–595. [[CrossRef](#)]
17. Prentice, L.M.; Phillips, D.I.; Sarsero, D.; Beever, K.; McLachlan, S.M.; Smith, B.R. Geographical distribution of subclinical autoimmune thyroid disease in Britain: A study using highly sensitive direct assays for autoantibodies to thyroglobulin and thyroid peroxidase. *Acta Endocrinol.* **1990**, *123*, 493–498. [[CrossRef](#)]
18. Tunbridge, W.M.; Evered, D.C.; Hall, R.; Appleton, D.; Brewis, M.; Clark, F.; Evans, J.G.; Young, E.; Bird, T.; Smith, P.A. The spectrum of thyroid disease in a community: The Wickham survey. *Clin. Endocrinol.* **1977**, *7*, 481–493. [[CrossRef](#)]
19. Aho, K.; Virkola, P.; Heinonen, O.P. Determination of thyroglobulin antibodies using chromic chloride as a coupling reagent. Evaluation of the test and characterization of antibodies in an adult population. *Acta Endocrinol.* **1971**, *68*, 196–202.
20. Gordin, A.; Heinonen, O.P.; Saarinen, P.; Lamberg, B.A. Serum-thyrotrophin in symptomless autoimmune thyroiditis. *Lancet* **1972**, *1*, 551–554. [[CrossRef](#)]
21. Bjørø, T.; Gaarder, P.I.; Smeland, E.B.; Kornstad, L. Thyroid antibodies in blood donors: Prevalence and clinical significance. *Acta Endocrinol.* **1984**, *105*, 324–329. [[CrossRef](#)]
22. Bryhni, B.; Aanderud, S.; Sundsfjord, J.; Rekvig, O.P.; Jorde, R. Thyroid antibodies in northern Norway: Prevalence, persistence and relevance. *J. Intern. Med.* **1996**, *239*, 517–523. [[CrossRef](#)] [[PubMed](#)]
23. Völzke, H.; Lüdemann, J.; Robinson, D.M.; Spieker, K.W.; Schwahn, C.; Kramer, A.; John, U.; Meng, W. The prevalence of undiagnosed thyroid disorders in a previously iodine-deficient area. *Thyroid* **2003**, *13*, 803–810. [[CrossRef](#)] [[PubMed](#)]
24. Döbert, N.; Balzer, K.; Diener, J.; Wegscheider, K.; Vaupel, R.; Grünwald, F. Thyroid sonomorphology, thyroid peroxidase antibodies and thyroid function: New epidemiological data in unselected German employees. *Nuklearmedizin* **2008**, *47*, 194–199. [[PubMed](#)]

25. Khattak, R.M.; Ittermann, T.; Nauck, M.; Below, H.; Völzke, H. Monitoring the prevalence of thyroid disorders in the adult population of Northeast Germany. *Popul. Health Metr.* **2016**, *14*, 39. [\[CrossRef\]](#)
26. Aghini-Lombardi, F.; Antonangeli, L.; Martino, E.; Vitti, P.; Maccherini, D.; Leoli, F.; Rago, T.; Grasso, L.; Valeriano, R.; Balestrieri, A.; et al. The spectrum of thyroid disorders in an iodine-deficient community: The Pescopagano survey. *J. Clin. Endocrinol. Metab.* **1999**, *84*, 561–566. [\[CrossRef\]](#)
27. Benvenega, S.; Trimarchi, F. Changed presentation of Hashimoto's thyroiditis in North-Eastern Sicily and Calabria (Southern Italy) based on a 31-year experience. *Thyroid* **2008**, *18*, 429–441. [\[CrossRef\]](#)
28. Sardu, C.; Cocco, E.; Mereu, A.; Massa, R.; Cuccu, A.; Marrosu, M.G.; Contu, P. Population based study of 12 autoimmune diseases in Sardinia, Italy: Prevalence and comorbidity. *PLoS ONE* **2012**, *7*, e32487. [\[CrossRef\]](#)
29. Aghini-Lombardi, F.; Fiore, E.; Tonacchera, M.; Antonangeli, L.; Rago, T.; Frigeri, M.; Provenzale, A.M.; Montanelli, L.; Grasso, L.; Pinchera, A.; et al. The effect of voluntary iodine prophylaxis in a small rural community: The Pescopagano survey 15 years later. *J. Clin. Endocrinol. Metab.* **2013**, *98*, 1031–1039. [\[CrossRef\]](#)
30. Tammaro, A.; Pigliacelli, F.; Fumarola, A.; Persechino, S. Trends of thyroid function and autoimmunity to 5 years after the introduction of mandatory iodization in Italy. *Eur. Ann. Allergy Clin. Immunol.* **2016**, *48*, 77–81.
31. Pilli, T.; Cardinale, S.; Dalmiglio, C.; Secchi, C.; Fralassi, N.; Cevenini, G.; Di Cairano, G.; Maino, F.; Forleo, R.; Pacini, F.; et al. Autoimmune thyroid diseases are more common in patients with prolactinomas: A retrospective case-control study in an Italian cohort. *J. Endocrinol. Investig.* **2019**, *42*, 693–698. [\[CrossRef\]](#) [\[PubMed\]](#)
32. Pedersen, I.B.; Knudsen, N.; Jørgensen, T.; Perrild, H.; Ovesen, L.; Laurberg, P. Thyroid peroxidase and thyroglobulin autoantibodies in a large survey of populations with mild and moderate iodine deficiency. *Clin. Endocrinol.* **2003**, *58*, 36–42. [\[CrossRef\]](#) [\[PubMed\]](#)
33. Pedersen, I.B.; Knudsen, N.; Carlé, A.; Vejbjerg, P.; Jørgensen, T.; Perrild, H.; Ovesen, L.; Rasmussen, L.B.; Laurberg, P. A cautious iodization programme bringing iodine intake to a low recommended level is associated with an increase in the prevalence of thyroid autoantibodies in the population. *Clin. Endocrinol.* **2011**, *75*, 120–126. [\[CrossRef\]](#) [\[PubMed\]](#)
34. Tang Møllehave, L.; Knudsen, N.; Linneberg, A.; Bülow Pedersen, I.; Ravn-Haren, G.; Madsen, A.L.; Carlé, A.; Cerqueira, C.; Krejbjerg, A.; Rasmussen, L.B.; et al. The Danish investigation on iodine intake and thyroid disease (DanThyr): History and implications. *Eur. Thyroid. J.* **2024**, *13*, e230230. [\[CrossRef\]](#)
35. Valdés, S.; Maldonado-Araque, C.; Lago-Sampedro, A.; Lillo, J.A.; García-Fuentes, E.; Perez-Valero, V.; Gutierrez-Repiso, C.; Ocon-Sánchez, P.; Goday, A.; Urrutia, I.; et al. Population-Based National Prevalence of Thyroid Dysfunction in Spain and Associated Factors: Di@bet.es study. *Hyroid* **2017**, *27*, 156–166. [\[CrossRef\]](#)
36. Jóźków, P.; Lwow, F.; Słowińska-Lisowska, M.; Mędraś, M. Trends in the prevalence of autoimmune thyroiditis in the leading private health-care provider in Poland. *Adv. Clin. Exp. Med.* **2017**, *26*, 497–503. [\[CrossRef\]](#)
37. Troshina, E.A.; Platonova, N.M.; Panfilova, E.A. Dynamics of epidemiological indicators of thyroid pathology in the population of the Russian Federation: Analytical report for the period 2009–2018. *Probl. Endokrinol.* **2021**, *67*, 10–19. [\[CrossRef\]](#)
38. Izic, B.; Custovic, A.; Caluk, S.; Fejzic, H.; Kundalic, B.S.; Husejnovic, M.S. The Epidemiological Characteristics of Autoimmune Thyroiditis in the Tuzla Canton in the Period from 2015 to 2020. *Mater. Sociomed.* **2021**, *33*, 288–292. [\[CrossRef\]](#)
39. Strikić Đula, I.; Pleić, N.; Babić Leko, M.; Gunjača, I.; Torlak, V.; Brdar, D.; Punda, A.; Polašek, O.; Hayward, C.; Zemunik, T. Epidemiology of Hypothyroidism, Hyperthyroidism and Positive Thyroid Antibodies in the Croatian Population. *Biology* **2022**, *11*, 394. [\[CrossRef\]](#)
40. Chabchoub, G.; Mnif, M.; Maalej, A.; Charfi, N.; Ayadi, H.; Abid, M. Epidemiologic study of autoimmune thyroid disease in south Tunisia. *Ann. Endocrinol.* **2006**, *67*, 591–595. [\[CrossRef\]](#)
41. Okosieme, O.E.; Taylor, R.C.; Ohwovoriole, A.E.; Parkes, A.B.; Lazarus, J.H. Prevalence of thyroid antibodies in Nigerian patients. *QJM* **2007**, *100*, 107–112. [\[CrossRef\]](#) [\[PubMed\]](#)
42. Sarfo-Kantanka, O.; Kyei, I.; Sarfo, F.S.; Ansah, E.O. Thyroid Disorders in Central Ghana: The Influence of 20 Years of Iodization. *J. Thyroid. Res.* **2017**, *2017*, 7843972. [\[CrossRef\]](#)
43. O'Leary, P.C.; Feddema, P.H.; Michelangeli, V.P.; Leedman, P.J.; Chew, G.T.; Knuiman, M.; Kaye, J.; Walsh, J.P. Investigations of thyroid hormones and antibodies based on a community health survey: The Busselton thyroid study. *Clin. Endocrinol.* **2006**, *64*, 97–104. [\[CrossRef\]](#) [\[PubMed\]](#)
44. Deshpande, P.; Lucas, M.; Brunt, S.; Lucas, A.; Hollingsworth, P.; Bundell, C. Low level autoantibodies can be frequently detected in the general Australian population. *Pathology* **2016**, *48*, 483–490. [\[CrossRef\]](#)
45. Okayasu, I.; Hatakeyama, S.; Tanaka, Y.; Sakurai, T.; Hoshi, K.; Lewis, P.D. Is focal chronic autoimmune thyroiditis an age-related disease? Differences in incidence and severity between Japanese and British. *J. Pathol.* **1991**, *163*, 257–264. [\[CrossRef\]](#)
46. Konno, N.; Yuri, K.; Taguchi, H.; Miura, K.; Taguchi, S.; Hagiwara, K.; Murakami, S. Screening for thyroid diseases in an iodine sufficient area with sensitive thyrotrophin assays, and serum thyroid autoantibody and urinary iodide determinations. *Clin. Endocrinol.* **1993**, *38*, 273–281. [\[CrossRef\]](#) [\[PubMed\]](#)

47. Morinaka, S. On the frequency of thyroid diseases in outpatients in an ENT clinic. *Auris Nasus Larynx*. **1995**, *22*, 186–191. [[CrossRef](#)] [[PubMed](#)]
48. Nagata, K.; Takasu, N.; Akamine, H.; Ohshiro, C.; Komiya, I.; Murakami, K.; Suzawa, A.; Nomura, T. Urinary iodine and thyroid antibodies in Okinawa, Yamagata, Hyogo, and Nagano, Japan: The differences in iodine intake do not affect thyroid antibody positivity. *Endocr. J.* **1998**, *45*, 797–803. [[CrossRef](#)]
49. Kurata, S.; Ishibashi, M.; Hiromatsu, Y.; Kaida, H.; Miyake, I.; Uchida, M.; Hayabuchi, N. Diffuse and diffuse-plus-focal uptake in the thyroid gland identified by using FDG–PET: Prevalence of thyroid cancer and Hashimoto’s thyroiditis. *Ann. Nucl. Med.* **2007**, *21*, 325–330. [[CrossRef](#)]
50. Teng, W.; Shan, Z.; Teng, X.; Guan, H.; Li, Y.; Teng, D.; Li, C. Effect of iodine intake on thyroid diseases in China. *N. Engl. J. Med.* **2006**, *354*, 2783–2793. [[CrossRef](#)]
51. Teng, X.; Shi, X.; Shan, Z.; Jin, Y.; Guan, H.; Li, Y.; Yang, F.; Wang, W.; Tong, Y.; Teng, W. Safe range of iodine intake levels: A comparative study of thyroid diseases in three women population cohorts with slightly different iodine intake levels. *Biol. Trace Elem. Res.* **2008**, *121*, 23–30. [[CrossRef](#)]
52. Li, Y.; Teng, D.; Shan, Z.; Teng, X.; Guan, H.; Yu, X.; Fan, C.; Chong, W.; Yang, F.; Dai, H.; et al. Antithyroperoxidase and antithyroglobulin antibodies in a five-year follow-up survey of populations with different iodine intakes. *J. Clin. Endocrinol. Metab.* **2008**, *93*, 1751–1757. [[CrossRef](#)] [[PubMed](#)]
53. Teng, X.; Shan, Z.; Chen, Y.; Lai, Y.; Yu, J.; Shan, L.; Bai, X.; Li, Y.; Li, N.; Li, Z.; et al. More than adequate iodine intake may increase subclinical hypothyroidism and autoimmune thyroiditis: A cross-sectional study based on two Chinese communities with different iodine intake levels. *Eur. J. Endocrinol.* **2011**, *164*, 943–950. [[CrossRef](#)]
54. Wu, Q.; Rayman, M.P.; Lv, H.; Schomburg, L.; Cui, B.; Gao, C.; Chen, P.; Zhuang, G.; Zhang, Z.; Peng, X.; et al. Low Population Selenium Status Is Associated with Increased Prevalence of Thyroid Disease. *J. Clin. Endocrinol. Metab.* **2015**, *100*, 4037–4047. [[CrossRef](#)] [[PubMed](#)]
55. Gu, F.; Ding, G.; Lou, X.; Wang, X.; Mo, Z.; Zhu, W.; Zhou, J.; Mao, G. Incidence of thyroid diseases in Zhejiang Province, China, after 15 years of salt iodization. *J. Trace Elem. Med. Biol.* **2016**, *36*, 57–64. [[CrossRef](#)]
56. Li, Y.; Chen, D.N.; Cui, J.; Xin, Z.; Yang, G.R.; Niu, M.J.; Yang, J.K. Using Hashimoto thyroiditis as gold standard to determine the upper limit value of thyroid stimulating hormone in a Chinese cohort. *BMC Endocr. Disord.* **2016**, *16*, 57. [[CrossRef](#)] [[PubMed](#)]
57. Wan, S.; Qu, M.; Wu, H.; Ren, B.; Jiang, W.; Wang, X.; Liu, L.; Shen, H. Autoimmune thyroid diseases after 25 years of universal salt iodisation: An epidemiological study of Chinese adults in areas with different water iodine levels. *Br. J. Nutr.* **2020**, *124*, 853–864. [[CrossRef](#)]
58. Chen, Y.; Han, B.; Yu, J.; Chen, Y.; Cheng, J.; Zhu, C.; Xia, F.; Wang, N.; Lu, Y. Influence of Rapid Urbanization on Thyroid Autoimmune Disease in China. *Int. J. Endocrinol.* **2021**, *2021*, 9967712. [[CrossRef](#)]
59. Yu, Z.; Yu, Y.; Wan, Y.; Fan, J.; Meng, H.; Li, S.; Wang, Y.; Wang, T.; Ling, R. Iodine intake level and incidence of thyroid disease in adults in Shaanxi province: A cross-sectional study. *Ann. Transl. Med.* **2021**, *9*, 1567. [[CrossRef](#)]
60. Fernando, R.F.; Chandrasinghe, P.C.; Pathmeswaran, A.A. The prevalence of autoimmune thyroiditis after universal salt iodisation in Sri Lanka. *Ceylon. Med. J.* **2012**, *57*, 116–119. [[CrossRef](#)]
61. Amouzegar, A.; Gharibzadeh, S.; Kazemian, E.; Mehran, L.; Tohidi, M.; Azizi, F. The Prevalence, Incidence and Natural Course of Positive Antithyroperoxidase Antibodies in a Population-Based Study: Tehran Thyroid Study. *PLoS ONE* **2017**, *12*, e0169283. [[CrossRef](#)] [[PubMed](#)]
62. Kim, H.J.; Kazmi, S.Z.; Kang, T.; Sohn, S.Y.; Kim, D.S.; Hann, H.J.; Ahn, H.S. Familial Risk of Hashimoto’s Thyroiditis Among First-Degree Relatives: A Population-Based Study in Korea. *Thyroid* **2021**, *31*, 1096–1104. [[CrossRef](#)] [[PubMed](#)]
63. Ajlouni, K.M.; Khawaja, N.; El-Khateeb, M.; Batieha, A.; Farahid, O. The prevalence of thyroid dysfunction in Jordan: A national population-based survey. *BMC Endocr. Disord.* **2022**, *22*, 253. [[CrossRef](#)] [[PubMed](#)]
64. Okayasu, I.; Hara, Y.; Nakamura, K.; Rose, N.R. Racial and age-related differences in incidence and severity of focal autoimmune thyroiditis. *Am. J. Clin. Pathol.* **1994**, *101*, 698–702. [[CrossRef](#)]
65. Flores-Rebollar, A.; Moreno-Castañeda, L.; Vega-Servín, N.S.; López-Carrasco, G.; Ruiz-Juvera, A. Prevalence of autoimmune thyroiditis and thyroid dysfunction in healthy adult mexicans with a slightly excessive iodine intake. *Nutr. Hosp.* **2015**, *32*, 918–924.
66. Caturegli, G.; Caturegli, P. Disease prevalence in a rural Andean population of central Peru: A focus on autoimmune and allergic diseases. *Auto. Immun. Highlights* **2016**, *7*, 3. [[CrossRef](#)]
67. Zhang, X.; Wang, Y.; Wang, H.; Zhang, X. Trends in Prevalence of Thyroid Dysfunction and its Associations with Mortality Among US Participants, 1988–2012. *J. Clin. Endocrinol. Metab.* **2024**, *109*, e657–e666. [[CrossRef](#)]
68. Tomimori, E.; Pedrinola, F.; Cavaliere, H.; Knobel, M.; Medeiros-Neto, G. Prevalence of incidental thyroid disease in a relatively low iodine intake area. *Thyroid* **1995**, *5*, 273–276. [[CrossRef](#)] [[PubMed](#)]
69. Camargo, R.Y.; Tomimori, E.K.; Neves, S.C.; Knobel, M.; Medeiros-Neto, G. Prevalence of chronic autoimmune thyroiditis in the urban area neighboring a petrochemical complex and a control area in Sao Paulo, Brazil. *Clinics* **2006**, *61*, 307–312. [[CrossRef](#)]

70. Camargo, R.Y.; Tomimori, E.K.; Neves, S.C.; G S Rubio, I.; Galvão, A.L.; Knobel, M.; Medeiros-Neto, G. Thyroid and the environment: Exposure to excessive nutritional iodine increases the prevalence of thyroid disorders in Sao Paulo, Brazil. *Eur. J. Endocrinol.* **2008**, *159*, 293–299. [\[CrossRef\]](#)
71. Vecchiatti, S.M.; Lin, C.J.; Capelozzi, V.L.; Longatto-Filho, A.; Bisi, H. Prevalence of thyroiditis and immunohistochemistry study searching for a morphologic consensus in morphology of autoimmune thyroiditis in a 4613 autopsies series. *Appl. Immunohistochem. Mol. Morphol.* **2015**, *23*, 402–408. [\[CrossRef\]](#)
72. Tolentino Júnior, D.S.; de Oliveira, C.M.; de Assis, E.M. Population-based Study of 24 Autoimmune Diseases Carried Out in a Brazilian Microregion. *J. Epidemiol. Glob. Health* **2019**, *9*, 243–251. [\[CrossRef\]](#) [\[PubMed\]](#)
73. Vargas-Uricoechea, H.; Agredo-Delgado, V.; Vargas-Sierra, H.D.; Pinzón-Fernández, M.V. Prevalence of Functional Alterations and the Effects of Thyroid Autoimmunity on the Levels of TSH in an Urban Population of Colombia: A Population-Based Study. *Endocr. Metab. Immune Disord. Drug Targets* **2023**, *23*, 857–866. [\[CrossRef\]](#)
74. Dwivedi, S.N.; Kalaria, T.; Buch, H. Thyroid autoantibodies. *J. Clin. Pathol.* **2023**, *76*, 19–28. [\[CrossRef\]](#) [\[PubMed\]](#)
75. Zhang, M.; Ni, W.; Zhang, L.; Fan, K.; Sun, Y.; Liu, C.; Xu, S. Age-specific association between thyroid autoimmunity and hypothyroidism in Chinese adults aged over 65 years: A cross-sectional study. *Front. Endocrinol.* **2023**, *14*, 1216308. [\[CrossRef\]](#) [\[PubMed\]](#)
76. Taylor, P.N.; Albrecht, D.; Scholz, A.; Gutierrez-Buey, G.; Lazarus, J.H.; Dayan, C.M.; Okosieme, O.E. Global epidemiology of hyperthyroidism and hypothyroidism. *Nat. Rev. Endocrinol.* **2018**, *14*, 301–316. [\[CrossRef\]](#)
77. Vanderpump, M.P.; Tunbridge, W.M.; French, J.M.; Appleton, D.; Bates, D.; Clark, F.; Grimley Evans, J.; Hasan, D.M.; Rodgers, H.; Tunbridge, F.; et al. The incidence of thyroid disorders in the community: A twenty-year follow-up of the Wickham Survey. *Clin. Endocrinol.* **1995**, *43*, 55–68. [\[CrossRef\]](#)
78. Cooper, G.S.; Stroehla, B.C. The epidemiology of autoimmune diseases. *Autoimmun. Rev.* **2003**, *2*, 119–125. [\[CrossRef\]](#)
79. Empson, M.; Flood, V.; Ma, G.; Eastman, C.J.; Mitchell, P. Prevalence of thyroid disease in an older Australian population. *Intern. Med. J.* **2007**, *37*, 448–455. [\[CrossRef\]](#)
80. Chen, D.W.; Ospina, N.S.; Haymart, M.R. Social Determinants of Health and Disparities in Thyroid Care. *J. Clin. Endocrinol. Metab.* **2024**, *109*, e1309–e1313. [\[CrossRef\]](#)
81. Gillis, A.; Chen, H.; Wang, T.S.; Dream, S. Racial and Ethnic Disparities in the Diagnosis and Treatment of Thyroid Disease. *J. Clin. Endocrinol. Metab.* **2024**, *109*, e1336–e1344. [\[CrossRef\]](#) [\[PubMed\]](#)
82. Díez, J.J.; Iglesias, P. Prevalence of thyroid dysfunction and its relationship to income level and employment status: A nationwide population-based study in Spain. *Hormones* **2023**, *22*, 243–252. [\[CrossRef\]](#)
83. St Sauver, J.L.; Kapoor, E.; Bielinski, S.J.; MacLaughlin, K.L.; Faubion, S.S.; Jiang, R.; Rocca, W.A. Health care concerns in women at midlife: Differences by race, ethnicity, and neighborhood socioeconomic status. *Menopause* **2025**, *32*, 112–120. [\[CrossRef\]](#) [\[PubMed\]](#)
84. Meisinger, C.; Ittermann, T.; Wallaschofski, H.; Heier, M.; Below, H.; Kramer, A.; Döring, A.; Nauck, M.; Völzke, H. Geographic variations in the frequency of thyroid disorders and thyroid peroxidase antibodies in persons without former thyroid disease within Germany. *Eur. J. Endocrinol.* **2012**, *167*, 363–371. [\[CrossRef\]](#) [\[PubMed\]](#)
85. Thvilum, M.; Brandt, F.; Brix, T.H.; Hegedüs, L. Month of birth is associated with the subsequent diagnosis of autoimmune hypothyroidism. A nationwide Danish register-based study. *Clin. Endocrinol.* **2017**, *87*, 832–837. [\[CrossRef\]](#)
86. Ramos-Leví, A.M.; Collado, G.; Marazuela, M. Seasonality of month of birth in patients with autoimmune endocrine diseases: A systematic review. *Endocrinol. Diabetes Nutr.* **2022**, *69*, 779–790. [\[CrossRef\]](#)
87. Kyrgios, I.; Giza, S.; Tsinopoulou, V.R.; Maggana, I.; Haidich, A.B.; Galli-Tsinopoulou, A. Seasonality of month of birth in children and adolescents with autoimmune thyroiditis: A continuing conundrum. *J. Pediatr. Endocrinol. Metab.* **2018**, *31*, 1123–1131. [\[CrossRef\]](#)
88. Marcec, R.; Stjepanovic, J.; Likic, R. Seasonality of Hashimoto Thyroiditis: Infodemiology Study of Google Trends Data. *JMIR Bioinform. Biotechnol.* **2022**, *3*, e38976. [\[CrossRef\]](#)
89. Lee, H.J.; Li, C.W.; Hammerstad, S.S.; Stefan, M.; Tomer, Y. Immunogenetics of autoimmune thyroid diseases: A comprehensive review. *J. Autoimmun.* **2015**, *64*, 82–90. [\[CrossRef\]](#)
90. McLeod, D.S.; Cooper, D.S.; Ladenson, P.W.; Whiteman, D.C.; Jordan, S.J. Race/Ethnicity and the prevalence of thyrotoxicosis in young americans. *Thyroid* **2015**, *25*, 621–628. [\[CrossRef\]](#)
91. Li, Q.; Yang, W.; Li, J.; Shan, Z. Emerging trends and hot spots in autoimmune thyroiditis research from 2000 to 2022: A bibliometric analysis. *Front. Immunol.* **2022**, *13*, 953465. [\[CrossRef\]](#) [\[PubMed\]](#)
92. Song, Y.; Li, J.; Wu, Y. Evolving understanding of autoimmune mechanisms and new therapeutic strategies of autoimmune disorders. *Signal Transduct. Target. Ther.* **2024**, *9*, 263. [\[CrossRef\]](#)
93. Tripathi, P.; Bhushan, D.; Banerjee, A.; Mahto, M. The Kaleidoscope of Polyautoimmunity: An Odyssey of Diagnostic Dilemmas. *Cureus* **2024**, *16*, e57799. [\[CrossRef\]](#) [\[PubMed\]](#)

94. Somers, E.C.; Thomas, S.L.; Smeeth, L.; Hall, A.J. Are individuals with an autoimmune disease at higher risk of a second autoimmune disorder? *Am. J. Epidemiol.* **2009**, *169*, 749–755. [CrossRef]
95. Botello, A.; Herrán, M.; Salcedo, V.; Rodríguez, Y.; Anaya, J.M.; Rojas, M. Prevalence of latent and overt polyautoimmunity in autoimmune thyroid disease: A systematic review and meta-analysis. *Clin. Endocrinol.* **2020**, *93*, 375–389. [CrossRef] [PubMed]
96. Zhang, K.; Luo, Z.; Wang, X. The association of common autoimmune diseases with autoimmune thyroiditis: A two-sample Mendelian randomization study. *Front. Endocrinol.* **2024**, *15*, 1383221. [CrossRef]
97. Duntas, L.H.; Biondi, B. The interconnections between obesity, thyroid function, and autoimmunity: The multifold role of leptin. *Thyroid* **2013**, *23*, 646–653. [CrossRef]
98. Song, R.H.; Wang, B.; Yao, Q.M.; Li, Q.; Jia, X.; Zhang, J.A. The Impact of Obesity on Thyroid Autoimmunity and Dysfunction: A Systematic Review and Meta-Analysis. *Front. Immunol.* **2019**, *10*, 2349. [CrossRef]
99. Huo, J.; Xu, Y.; Yu, J.; Guo, Y.; Hu, X.; Ou, D.; Qu, R.; Zhao, L. Causal association between body mass index and autoimmune thyroiditis: Evidence from Mendelian randomization. *Eur. J. Med. Res.* **2023**, *28*, 526. [CrossRef]
100. Available online: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight> (accessed on 10 February 2025).
101. Jing, M.; Shao, S.; Ma, S.; Gao, L.; Wang, Q.; Zhou, M. Exploring the link between obesity and hypothyroidism in autoimmune thyroid diseases: A metabolic perspective. *Front. Mol. Biosci.* **2024**, *11*, 1379124. [CrossRef]
102. Tsigalou, C.; Vallianou, N.; Dalamaga, M. Autoantibody Production in Obesity: Is There Evidence for a Link Between Obesity and Autoimmunity? *Curr. Obes. Rep.* **2020**, *9*, 245–254. [CrossRef] [PubMed]
103. Baranowska-Bik, A.; Bik, W. The Association of Obesity with Autoimmune Thyroiditis and Thyroid Function—Possible Mechanisms of Bilateral Interaction. *Int. J. Endocrinol.* **2020**, *2020*, 8894792. [CrossRef] [PubMed]
104. Street, M.E.; Shulhai, A.M.; Petraroli, M.; Patianna, V.; Donini, V.; Giudice, A.; Gnocchi, M.; Masetti, M.; Montani, A.G.; Rotondo, R.; et al. The impact of environmental factors and contaminants on thyroid function and disease from fetal to adult life: Current evidence and future directions. *Front. Endocrinol.* **2024**, *15*, 1429884. [CrossRef]
105. Pearce, E.N. Endocrine Disruptors and Thyroid Health. *Endocr. Pract.* **2024**, *30*, 172–176. [CrossRef]
106. Zeng, Y.; He, H.; Wang, X.; Zhang, M.; An, Z. Climate and air pollution exposure are associated with thyroid function parameters: A retrospective cross-sectional study. *J. Endocrinol. Investig.* **2021**, *44*, 1515–1523. [CrossRef]
107. Valdés, S.; Doulatram-Gamgaram, V.; Maldonado-Araque, C.; Lago-Sampedro, A.; García-Escobar, E.; García-Serrano, S.; García-Vivanco, M.; Garrido Juan, L.; Theobald, M.R.; Gil, V.; et al. Ambient air pollution and thyroid function in Spanish adults. A nationwide population-based study (Di@bet.es study). *Environ. Health* **2022**, *21*, 76. [CrossRef] [PubMed]
108. Benvenega, S.; Elia, G.; Ragusa, F.; Paparo, S.R.; Sturniolo, M.M.; Ferrari, S.M.; Antonelli, A.; Fallahi, P. Endocrine disruptors and thyroid autoimmunity. *Best. Pract. Res. Clin. Endocrinol. Metab.* **2020**, *34*, 101377. [CrossRef]
109. He, J.; Xu, J.; Zheng, M.; Pan, K.; Yang, L.; Ma, L.; Wang, C.; Yu, J. Thyroid dysfunction caused by exposure to environmental endocrine disruptors and the underlying mechanism: A review. *Chem. Biol. Interact.* **2024**, *391*, 110909. [CrossRef]
110. Di Ciaula, A.; Bonfrate, L.; Noviello, M.; Portincasa, P. Thyroid Function: A Target for Endocrine Disruptors, Air Pollution and Radiofrequencies. *Endocr. Metab. Immune Disord. Drug Targets.* **2023**, *23*, 1032–1040. [CrossRef]
111. Saint-André, V.; Charbit, B.; Biton, A.; Rouilly, V.; Possémé, C.; Bertrand, A.; Rotival, M.; Bergstedt, J.; Patin, E.; Albert, M.L.; et al. Smoking changes adaptive immunity with persistent effects. *Nature* **2024**, *626*, 827–835. [CrossRef]
112. Harel-Meir, M.; Sherer, Y.; Shoenfeld, Y. Tobacco smoking and autoimmune rheumatic diseases. *Nat. Clin. Pract. Rheumatol.* **2007**, *3*, 707–715. [CrossRef] [PubMed]
113. Jiang, F.; Li, S.; Jia, C. Smoking and the risk of systemic lupus erythematosus: An updated systematic review and cumulative meta-analysis. *Clin. Rheumatol.* **2015**, *34*, 1885–1892. [CrossRef] [PubMed]
114. Vestergaard, P. Smoking and thyroid disorders—a meta-analysis. *Eur. J. Endocrinol.* **2002**, *146*, 153–161. [CrossRef]
115. Bertelsen, J.B.; Hegedüs, L. Cigarette smoking and the thyroid. *Thyroid* **1994**, *4*, 327–331. [CrossRef]
116. Pedersen, I.B.; Laurberg, P.; Knudsen, N.; Jørgensen, T.; Perrild, H.; Ovesen, L.; Rasmussen, L.B. Smoking is negatively associated with the presence of thyroglobulin autoantibody and to a lesser degree with thyroid peroxidase autoantibody in serum: A population study. *Eur. J. Endocrinol.* **2008**, *158*, 367–373. [CrossRef] [PubMed]
117. Effraimidis, G.; Tijssen, J.G.; Wiersinga, W.M. Discontinuation of smoking increases the risk for developing thyroid peroxidase antibodies and/or thyroglobulin antibodies: A prospective study. *J. Clin. Endocrinol. Metab.* **2009**, *94*, 1324–1328. [CrossRef]
118. Attard, C.C.; Sze, W.C.C.; Vella, S. Predictors of autoimmune thyroid disease. *Proc. (Bayl. Univ. Med. Cent.)* **2022**, *35*, 608–614. [CrossRef]
119. Terracina, S.; Caronti, B.; Lucarelli, M.; Francati, S.; Piccioni, M.G.; Tarani, L.; Ceccanti, M.; Caserta, M.; Verdone, L.; Venditti, S.; et al. Alcohol Consumption and Autoimmune Diseases. *Int. J. Mol. Sci.* **2025**, *26*, 845. [CrossRef]
120. Effraimidis, G.; Tijssen, J.G.; Wiersinga, W.M. Alcohol consumption as a risk factor for autoimmune thyroid disease: A prospective study. *Eur. Thyroid. J.* **2012**, *1*, 99–104. [CrossRef]

121. Carlé, A.; Pedersen, I.B.; Knudsen, N.; Perrild, H.; Ovesen, L.; Rasmussen, L.B.; Laurberg, P. Moderate alcohol consumption may protect against overt autoimmune hypothyroidism: A population-based case-control study. *Eur. J. Endocrinol.* **2012**, *167*, 483–490. [CrossRef]
122. World Health Organization. *Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination: A Guide for Programme Managers*, 2nd ed.; World Health Organization: Geneva, Switzerland, 2001; Available online: <https://apps.who.int/iris/handle/10665/61278> (accessed on 25 January 2025).
123. Lisco, G.; De Tullio, A.; Triggiani, D.; Zupo, R.; Giagulli, V.A.; De Pergola, G.; Piazzolla, G.; Guastamacchia, E.; Sabbà, C.; Triggiani, V. Iodine Deficiency and Iodine Prophylaxis: An Overview and Update. *Nutrients* **2023**, *15*, 1004. [CrossRef] [PubMed]
124. Katagiri, R.; Yuan, X.; Kobayashi, S.; Sasaki, S. Effect of excess iodine intake on thyroid diseases in different populations: A systematic review and meta-analyses including observational studies. *PLoS ONE* **2017**, *12*, e0173722. [CrossRef]
125. Teti, C.; Panciroli, M.; Nazzari, E.; Pesce, G.; Mariotti, S.; Olivieri, A.; Bagnasco, M. Iodoprophylaxis and thyroid autoimmunity: An update. *Immunol. Res.* **2021**, *69*, 129–138. [CrossRef] [PubMed]
126. Atapattu, N.; Jayatissa, R.; de Silva, H.; Adlan, M.A.; Obuobie, E.K.; Premawardhana, L.D. Thyroid Autoimmunity During Universal Salt Iodisation—Possible Short-Term Modulation with Longer-Term Stability. *Nutrients* **2024**, *16*, 4299. [CrossRef]
127. Bailey, R.L.; West, K.P., Jr.; Black, R.E. The epidemiology of global micronutrient deficiencies. *Ann. Nutr. Metab.* **2015**, *66* (Suppl. S2), 22–33. [CrossRef] [PubMed]
128. Aktaş, H.Ş. Vitamin B12 and Vitamin D Levels in Patients with Autoimmune Hypothyroidism and Their Correlation with Anti-Thyroid Peroxidase Antibodies. *Med. Princ. Pract.* **2020**, *29*, 364–370. [CrossRef]
129. Ihnatowicz, P.; Drywień, M.; Wątor, P.; Wojsiat, J. The importance of nutritional factors and dietary management of Hashimoto's thyroiditis. *Ann. Agric. Environ. Med.* **2020**, *27*, 184–193. [CrossRef] [PubMed]
130. Shulhai, A.M.; Rotondo, R.; Petraroli, M.; Patianna, V.; Predieri, B.; Iughetti, L.; Esposito, S.; Street, M.E. The Role of Nutrition on Thyroid Function. *Nutrients* **2024**, *16*, 2496. [CrossRef]
131. Benites-Zapata, V.A.; Ignacio-Conchay, F.L.; Ulloque-Badaracco, J.R.; Hernandez-Bustamante, E.A.; Alarcón-Braga, E.A.; Al-Kassab-Córdova, A.; Herrera-Añazco, P. Vitamin B12 levels in thyroid disorders: A systematic review and meta-analysis. *Front. Endocrinol.* **2023**, *14*, 1070592. [CrossRef]
132. 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*, 4398–4408. [CrossRef]
133. Osowiecka, K.; Myszkowska-Ryciak, J. The Influence of Nutritional Intervention in the Treatment of Hashimoto's Thyroiditis—A Systematic Review. *Nutrients* **2023**, *15*, 1041. [CrossRef]
134. Hu, S.; Rayman, M.P. Multiple Nutritional Factors and the Risk of Hashimoto's Thyroiditis. *Thyroid* **2017**, *27*, 597–610. [CrossRef] [PubMed]
135. Beserra, J.B.; Morais, J.B.S.; Severo, J.S.; Cruz, K.J.C.; de Oliveira, A.R.S.; Henriques, G.S.; do Nascimento Marreiro, D. Relation Between Zinc and Thyroid Hormones in Humans: A Systematic Review. *Biol. Trace Elem. Res.* **2021**, *199*, 4092–4100. [CrossRef]
136. Singh, A.K.; Chatterjee, S.; Singh, A.; Bhattacharjee, R. Diet in Thyroid Disorders: A Survey among Clinicians and a Review of the Current Perspective. *Indian J. Endocrinol. Metab.* **2024**, *28*, 378–384. [CrossRef]
137. Wang, Y.-S.; Liang, S.-S.; Ren, J.-J.; Wang, Z.-Y.; Deng, X.-X.; Liu, W.-D.; Yan, Y.-L.; Song, G.-H.; Li, X.-X. The Effects of Selenium Supplementation in the Treatment of Autoimmune Thyroiditis: An Overview of Systematic Reviews. *Nutrients* **2023**, *15*, 3194. [CrossRef] [PubMed]
138. Galușca, D.; Popoviciu, M.S.; Babeș, E.E.; Vidican, M.; Zaha, A.A.; Babeș, V.V.; Jurca, A.D.; Zaha, D.C.; Bodog, F. Vitamin D Implications and Effect of Supplementation in Endocrine Disorders: Autoimmune Thyroid Disorders (Hashimoto's Disease and Grave's Disease), Diabetes Mellitus and Obesity. *Medicina* **2022**, *58*, 194. [CrossRef] [PubMed]
139. Jiang, H.; Chen, X.; Qian, X.; Shao, S. Effects of vitamin D treatment on thyroid function and autoimmunity markers in patients with Hashimoto's thyroiditis—A meta-analysis of randomized controlled trials. *J. Clin. Pharm. Ther.* **2022**, *47*, 767–775. [CrossRef]
140. Camaschella, C. Iron deficiency. *Blood* **2019**, *133*, 30–39. [CrossRef]
141. Warner, M.J.; Kamran, M.T. Iron Deficiency Anemia. [Updated 7 August 2023]. In *StatPearls*; StatPearls Publishing: Treasure Island, FL, USA, 2025. Available online: <https://www.ncbi.nlm.nih.gov/books/NBK448065/> (accessed on 12 January 2025).
142. Amrein, K.; Scherkl, M.; Hoffmann, M.; Neuwersch-Sommeregger, S.; Köstenberger, M.; Tmava Berisha, A.; Martucci, G.; Pilz, S.; Malle, O. Vitamin D deficiency 2.0: An update on the current status worldwide. *Eur. J. Clin. Nutr.* **2020**, *74*, 1498–1513. [CrossRef]
143. Green, R.; Allen, L.H.; Bjørke-Monsen, A.L.; Brito, A.; Guéant, J.L.; Miller, J.W.; Molloy, A.M.; Nexø, E.; Stabler, S.; Toh, B.H.; et al. Vitamin B12 deficiency. *Nat. Rev. Dis. Primers.* **2017**, *3*, 17040. [CrossRef]
144. Green, R.; Miller, J.W. Vitamin B12 deficiency. *Vitam. Horm.* **2022**, *119*, 405–439.
145. Available online: <https://www.fao.org/faostat/es/#data/FS> (accessed on 21 January 2025).
146. Acosta, G.J.; Singh Ospina, N.; Brito, J.P. Epidemiologic changes in thyroid disease. *Curr. Opin. Endocrinol. Diabetes Obes.* **2024**, *31*, 184–190. [CrossRef] [PubMed]

147. Zhang, Y.; Sun, Y.; He, Z.; Xu, S.; Liu, C.; Li, Y.; Shan, Z.; Teng, W. Age-specific thyrotropin references decrease over-diagnosis of hypothyroidism in elderly patients in iodine-excessive areas. *Clin. Endocrinol.* **2024**, *101*, 549–556. [\[CrossRef\]](#)
148. Gottwald-Hostalek, U.; Schulte, B. Low awareness and under-diagnosis of hypothyroidism. *Curr. Med. Res. Opin.* **2022**, *38*, 59–64. [\[CrossRef\]](#)
149. Tagoe, C.E.; Sheth, T.; Golub, E.; Sorensen, K. Rheumatic associations of autoimmune thyroid disease: A systematic review. *Clin. Rheumatol.* **2019**, *38*, 1801–1809. [\[CrossRef\]](#)
150. Dukes, J.C.; Chakan, M.; Mills, A.; Marcaurd, M. Approach to Fatigue: Best Practice. *Med. Clin. North. Am.* **2021**, *105*, 137–148. [\[CrossRef\]](#) [\[PubMed\]](#)
151. Taylor, P.N.; Medici, M.M.; Hubalewska-Dydejczyk, A.; Boelaert, K. Hypothyroidism. *Lancet* **2024**, *404*, 1347–1364. [\[CrossRef\]](#) [\[PubMed\]](#)
152. Wiles, K. Management for women with subclinical hypothyroidism in pregnancy. *Drug Ther. Bull.* **2019**, *57*, 22–26. [\[CrossRef\]](#)
153. Maraka, S.; Dosiou, C. Subclinical Hypothyroidism and Thyroid Autoimmunity in Pregnancy: To Treat or Not to Treat. *Endocrinol. Metab. Clin. N. Am.* **2024**, *53*, 363–376. [\[CrossRef\]](#)
154. Aberle, L.; Nguyen, C.T. Subclinical hypothyroidism in pregnancy: An evolving and controversial landscape. *Curr. Opin. Obstet. Gynecol.* **2025**, *37*, 60–65. [\[CrossRef\]](#)
155. Bilous, R.W.; Tunbridge, W.M. The epidemiology of hypothyroidism—An update. *Baillieres Clin. Endocrinol. Metab.* **1988**, *2*, 531–540. [\[CrossRef\]](#) [\[PubMed\]](#)
156. Hong, A.; Stokes, B.; Otahal, P.; Owens, D.; Burgess, J.R. Temporal trends in thyroid-stimulating hormone (TSH) and thyroid peroxidase antibody (ATPO) testing across two phases of iodine fortification in Tasmania (1995–2013). *Clin. Endocrinol.* **2017**, *87*, 386–393. [\[CrossRef\]](#)
157. Garmendia Madariaga, A.; Santos Palacios, S.; Guillén-Grima, F.; Galofré, J.C. The incidence and prevalence of thyroid dysfunction in Europe: A meta-analysis. *J. Clin. Endocrinol. Metab.* **2014**, *99*, 923–931. [\[CrossRef\]](#) [\[PubMed\]](#)
158. Kiel, S.; Ittermann, T.; Völzke, H.; Chenot, J.F.; Angelow, A. Frequency of thyroid function tests and examinations in participants of a population-based study. *BMC Health Serv. Res.* **2020**, *20*, 70. [\[CrossRef\]](#)
159. Alaeddin, N.; Jongejan, R.M.S.; Stingl, J.C.; de Rijke, Y.B.; Peeters, R.P.; Breteler, M.M.B.; de Vries, F.M. Over- and Undertreatment with Levothyroxine. *Dtsch. Arztebl. Int.* **2023**, *120*, 711–718. [\[PubMed\]](#)
160. Dalal, S.; Bhesania, S.; Silber, S.; Mehta, P. Use of Electronic Clinical Decision Support and Hard Stops to Decrease Unnecessary Thyroid Function Testing. *BMJ Qual. Improv. Rep.* **2017**, *6*, u223041.w8346. [\[CrossRef\]](#)
161. Thiyagarajan, A.; Koenen, N.; Ittermann, T.; Völzke, H.; Haug, U. Trends in the use of thyroid diagnostics and treatments between 2008 and 2019 in Germany. *Sci. Rep.* **2024**, *14*, 26710. [\[CrossRef\]](#)
162. Baskin, H.J.; Cobin, R.H.; Duick, D.S.; Gharib, H.; Guttler, R.B.; Kaplan, M.M.; Segal, R.L. American Association of Clinical Endocrinologists. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the evaluation and treatment of hyperthyroidism and hypothyroidism. *Endocr. Pract.* **2002**, *8*, 457–469. [\[CrossRef\]](#)
163. US Preventive Services Task Force. Screening for thyroid disease: Recommendation statement. *Am. Fam. Physician* **2004**, *69*, 2415–2418.
164. Garber, J.R.; Cobin, R.H.; Gharib, H.; Hennessey, J.V.; Klein, I.; Mechanick, J.I.; Pessah-Pollack, R.; Singer, P.A.; Woeber, K.A. American Association of Clinical Endocrinologists and American Thyroid Association Taskforce on Hypothyroidism in Adults. Clinical practice guidelines for hypothyroidism in adults: Cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. *Endocr. Pract.* **2012**, *18*, 988–1028.
165. Okosieme, O.; Gilbert, J.; Abraham, P.; Boelaert, K.; Dayan, C.; Gurnell, M.; Leese, G.; McCabe, C.; Perros, P.; Smith, V.; et al. Management of primary hypothyroidism: Statement by the British Thyroid Association Executive Committee. *Clin. Endocrinol.* **2016**, *84*, 799–808. [\[CrossRef\]](#) [\[PubMed\]](#)
166. Chamot, S.; Al-Salameh, A.; Balcaen, T.; Petit, P.; Bonnetterre, V.; Cancé, C.; Desailoud, R. Congenital and acquired hypothyroidism: Temporal and spatial trends in France from 2014 to 2019. *Ann. Epidemiol.* **2024**, *98*, 18–24. [\[CrossRef\]](#)
167. Huang, K.; Huang, X.; Qian, S.; Cai, Y.; Wu, F.; Luo, D. Temporal trends of thyroid cancer in China and globally from 1990 to 2021: An analysis of the global burden of Disease Study 2021. *Sci. Rep.* **2024**, *14*, 25538. [\[CrossRef\]](#)
168. Hu, S.; Wu, X.; Jiang, H. Trends and projections of the global burden of thyroid cancer from 1990 to 2030. *J. Glob. Health* **2024**, *14*, 04084. [\[CrossRef\]](#) [\[PubMed\]](#)
169. Zhao, Q.; Chen, M.; Fu, L.; Yang, Y.; Zhan, Y. Assessing and projecting the global burden of thyroid cancer, 1990–2030: Analysis of the Global Burden of Disease Study. *J. Glob. Health* **2024**, *14*, 04090. [\[CrossRef\]](#)
170. Lui, D.T.W.; Lee, C.H.; Woo, Y.C.; Hung, I.F.N.; Lam, K.S.L. Thyroid dysfunction in COVID-19. *Nat. Rev. Endocrinol.* **2024**, *20*, 336–348. [\[CrossRef\]](#)
171. Panesar, A.; Gharanei, P.; Khovanova, N.; Young, L.; Grammatopoulos, D. Thyroid function during COVID-19 and post-COVID complications in adults: A systematic review. *Front. Endocrinol.* **2025**, *15*, 1477389. [\[CrossRef\]](#) [\[PubMed\]](#)

172. Li, L.; Chen, X.; Li, B.; Liu, D.; Liu, Y.; Mo, R.; Lai, F.; Liu, R.; Peng, S.; Li, Y.; et al. Effect of Inactivated SARS-CoV-2 Vaccine on Thyroid Function and Autoimmunity Within 28 Days After the Second Dose. *Thyroid* **2022**, *32*, 1051–1058. [[CrossRef](#)]
173. Weider, T.; Genoni, A.; Broccolo, F.; Paulsen, T.H.; Dahl-Jørgensen, K.; Toniolo, A.; Hammerstad, S.S. High Prevalence of Common Human Viruses in Thyroid Tissue. *Front. Endocrinol.* **2022**, *13*, 938633. [[CrossRef](#)]
174. Duntas, L.H. Nutrition and thyroid disease. *Curr. Opin. Endocrinol. Diabetes Obes.* **2023**, *30*, 324–329. [[CrossRef](#)]
175. Ralli, M.; Angeletti, D.; Fiore, M.; D'Aguanno, V.; Lambiase, A.; Artico, M.; de Vincentiis, M.; Greco, A. Hashimoto's thyroiditis: An update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies, and potential malignant transformation. *Autoimmun. Rev.* **2020**, *19*, 102649. [[CrossRef](#)] [[PubMed](#)]
176. Burch, H.B. Drug Effects on the Thyroid. *N. Engl. J. Med.* **2019**, *381*, 749–761. [[CrossRef](#)] [[PubMed](#)]
177. Cyna, W.; Wojciechowska, A.; Szybiak-Skora, W.; Lacka, K. The Impact of Environmental Factors on the Development of Autoimmune Thyroiditis—Review. *Biomedicines* **2024**, *12*, 1788. [[CrossRef](#)] [[PubMed](#)]
178. Karaviti, D.; Kani, E.R.; Karaviti, E.; Gerontiti, E.; Michalopoulou, O.; Stefanaki, K.; Kazakou, P.; Vasileiou, V.; Psaltopoulou, T.; Paschou, S.A. Thyroid disorders induced by immune checkpoint inhibitors. *Endocrine* **2024**, *85*, 67–79. [[CrossRef](#)]
179. Zhu, X.; Zhang, C.; Feng, S.; He, R.; Zhang, S. Intestinal microbiota regulates the gut–thyroid axis: The new dawn of improving Hashimoto thyroiditis. *Clin. Exp. Med.* **2024**, *24*, 39. [[CrossRef](#)]
180. Cuan-Baltazar, Y.; Soto-Vega, E. Microorganisms associated to thyroid autoimmunity. *Autoimmun. Rev.* **2020**, *19*, 102614. [[CrossRef](#)]

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