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ORIGINAL ARTICLE

Prevalence of Thyroid Dysfunction in Type 2 Diabetes Mellitus: a Systematic Review and Meta-Analysis

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ABSTRACT

Background: **Diabetes mellitus (T2DM) and thyroid dysfunction (TD) are pathophysiologically interdependent conditions. This review and meta-analysis contribute to the international evidence on the topic, as the 5-year pooled estimates of prevalence, biochemical associations, and risk factors have been published on TD among T2DM patients.**

Methods: **A PRISMA-based strategy was adopted in this review whereby studies published in January 2020 - March 2025 were searched in PubMed, Scopus, Web of Science, and Google Scholar. Thyroid functioning that was reported in adults with T2DM was included in observational research. Out of 1,852 records that were screened, 50 studies (30,244 participants) met the criteria. 19 studies provided data on pooled prevalence, 32 studies on risk factors, and at least 10 studies on the formal evaluation of the bias of publication. The quality of the studies was appraised using the Newcastle-Ottawa Scale, and all meta-analyses were conducted using random-effects models in R.**

Results: **The pooled occurrence of thyroid dysfunction in patients with Type 2 diabetes mellitus (T2DM) was observed to be 27.3% (95% CI: 24.4 - 30.3; I^2 = 94%; 19 studies), compared to 3.2% (95% CI: 2.5 - 4.1) in non-diabetic controls. T2DM individuals had nearly four times higher odds of thyroid dysfunction (pooled OR 3.89; 95% CI: 2.15 - 7.05; I^2 = 85%). The most common abnormality is subclinical hypothyroidism. Female gender was considered an independent risk factor (OR 2.35; 95% CI: 2.00 - 2.76; I^2 = 75%), age being over 50 years (OR 3.12; 95% CI: 1.98 - 4.91; I^2 = 82%), and poor glycemic control (HbA1c > 7%; OR 2.66; 95% CI: 1.94 - 3.65; I^2 = 41%). Positive anti-TPO antibodies are associated with a significantly higher risk (OR 12.8; 95% CI: 6.4 - 25.6; I^2 = 88%; based on four studies), and this finding should be interpreted with caution. TSH showed positive correlations with HbA1c and fasting glucose, whereas FT3 and FT4 were negatively correlated. There was no publication bias (p > 0.05).**

Conclusions: **The present review has proven a strong clinical and biochemical connection between thyroid dysfunction and T2DM. Since the prevalence is high and the metabolism is notably poor, regular thyroid screening in individuals with T2DM seems to be plausible, especially in women, older adults, and patients who have continuously excessive HbA1c levels.**

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KEYWORDS

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INTRODUCTION

Diabetes and thyroid dysfunction are the two most prevalent endocrine conditions in the world and, in most cases, tend to cross over; they affect the lives of the patients and burden the health systems even more [1]. Numerous review papers demonstrate that there is a pathophysiological correlation between type 2 diabetes and other thyroid diseases, as much of the literature indicates. These conditions do not just coexist, but they interact with each other, and one condition affects the other metabolic environment [2]. In this study, the researcher collected the available evidence about the frequency of the disorders, the most significant risk factors, and their impact on metabolism. This would target the maximisation of clinical screening and the management of the two conditions in patients.

Prevalence of thyroid dysfunction in T2DM

In T2DM patients, the more frequently observed condition is thyroid dysfunction than in non-diabetic people. The non-diabetic controls have low rates of thyroid disorders at 2.5 - 4.1% [3], but the rates in the Type 2 diabetic populations are much more variable; they have been reported to be as low as 10% and as high as 35% [4]. This trend is similarly seen in different populations; a cohort from Nigeria indicated a TD prevalence of 10 - 17.4% [5] while Indian studies suggest a much higher prevalence of 23.6 - 26.7% [6]. The commonest thyroid abnormality to be diagnosed in T2DM is subclinical hypothyroidism (SCH), and it is seen to occur in 6 - 26% of patients. Subclinical hypothyroidism (SCH) is defined by elevated serum TSH with normal free T3 (FT3) and free T4 (FT4) levels [7]. It is linked to a large proportion of new thyroid diagnoses in diabetic cohorts and, therefore, is of clinical importance [8]. Moreover, T2DM women show a significantly increased risk of TD than men (OR 1.5 - 2.7), which is consistent with the fact that thyroid disease is more prevalent [7,8].

Risk factors and demographic associations

There are several clinical and demographic variables that moderate the risk of TD in T2DM. The female gender is a notable demographic predictor; studies showed 55 to 68% of thyroid dysfunction in women with T2D (OR 2.0 - 2.7, $p < 0.05$) [9]. This increased susceptibility is the same of the general population and has often been attributed to factors such as increased susceptibility to autoimmune factors and hormonal factors. Age > 50 years is a powerful predictor (OR 2.1 - 5.8) of different studies, which may reflect the age-related changes in the thyroid gland and accumulation of metabolic load [10]. A duration of diabetes for > 5 years and a BMI greater than 25kg/m^2 both increase the risk of thyroid dysfunction importantly. Studies report these factors to have an OR of about 1.1 to 2.6 times [12]. The risk of thyroid dysfunction is strongly linked to a positive family history. Comorbid conditions such as hypertension and dyslipidemia also lead to additional vulnerability [12]. The association between type 2 diabetes (T2DM) and thyroid dysfunction may be driven by shared underlying pathways, in which insulin resistance may potentially be involved in enhancing thyroid autoimmunity, although this mechanism needs to be further validated. More importantly, the independent risk factor (OR 2.6, 95% CI 1.5 - 4.4, $p < 0.001$) is poor glycemic control (HbA1c > 7%); however, suggested mechanisms are hyperglycemia-induced inflammation, increased production of anti-thyroid peroxidase (anti-TPO) antibodies, and poor peripheral conversion of T4 to T3 [13,14].

Metabolic and hormonal correlations

There are important biochemical associations that mark the metabolic interaction that takes place between thyroid hormones and glycemic regulation in T2DM. TSH levels in hypothyroid states correlated positively with fasting blood glucose (FBG) and HbA1c ($r = 0.33$ - 0.76, $p < 0.05$) since elevated levels of TSH could contribute to increased hyperglycemia through reduced peripheral uptake of glucose and increased hepatic gluconeogenesis [15,16]. Conversely, the optimistic relationship between FT3 and FT4 and the HbA1c and insulin resistance indices, such as HOMA-IR, is primarily negative ($r = -0.18$ to -0.60, $p < 0.05$). Decreased FT3 and FT4 levels in uncontrolled T2DM may suggest a problem with deiodinase function [17]. In a hyperthyroid state, the association with FBG and C-peptide in type 2 diabetes mellitus (T2DM) shows a positive correlation ($r = 0.30$ - 0.48, $p < 0.05$) [18], possibly because the hyperthyroid state enhances the rate of glycogenolysis and insulin catabolism. Hypothyroidism is also regularly associated with an increased level of LDL cholesterol and triglycerides ($p < 0.05$), and, therefore, the risk of cardiovascular disease among T2DM is also increased [19]. Longitudinal evidence interestingly shows that the rise of TSH alongside the fall of FT4 measured over time can be correlated with a lower incidence of T2DM (hazard ratio [HR] 0.75 - 1.06, $p < 0.05$) [20]. This paradoxical association may imply the effect of compensatory mechanisms of the hypothalamic-pituitary-thyroid axis.

Clinical implications and management

The concomitant occurrence of T2DM and TD is associated with an increase in the risk of diabetic complications. Notably, low FT3 levels are a significant indicator of nephropathy (OR 0.36, $p = 0.001$), and the risk of cardiovascular disease is markedly increased; an integrated approach to management is required [21]. Levothyroxine is well-tolerated in the treatment of hypothyroidism and restores thyroid indices to normal. The therapy also causes a significant improvement in Hb A1c and insulin sensitivity [22]. Improved control of the metabolism of diabetes could lead to normalization of FT3 and FT4 levels [23]. Recent studies confirm the relationship of the high prevalence of TD with T2DM [7,24]. This justifies the necessity of routine thyroid

screening. The target group for the screening can be broken down as high-risk groups that include women, older adults, and those who have suboptimal glycemic control (HbA1c > 7%). Further studies are necessary to clarify the causal relationships and inform clinical practice. Fluctuating diagnostic thresholds and a lack of longitudinal data make it hard to have a full picture of TD in T2DM [25].

MATERIALS AND METHODS

Search strategy and study selection

A methodical search was performed using the principles of the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (Figure 1). PubMed, Scopus, Web of Science, and Google Scholar were searched for papers available from January 2020 through March 2025. The search was worldwide and not limited by geographical region. The search strategy effectively leveraged a targeted combination of Medical Subject Headings (MeSH) terms and relevant keywords to maximize precision and relevance: ("thyroid dysfunction" OR "hypothyroidism" OR "hyperthyroidism" OR "thyroid hormones") AND ("type 2 diabetes mellitus" OR "T2DM" OR "diabetes") AND ("prevalence" OR "epidemiology" OR "association" OR "HbA1c" OR "glycemic control"). The PICO (patient, intervention, comparison, and outcome) was applied to draw the eligibility criteria. The sample consisted of adult patients (age ≥ 18 years) with a diagnosis of T2DM. The possible outcomes were confirmation of current or a new diagnosis of thyroid dysfunction or the measurement of indices of thyroid function in adults with T2DM.

A control group was formed from non-diabetic people or normal thyroid functioning in T2DM. Main outcomes were prevalence of thyroid dysfunction; odds ratios (ORs) of the association between T2DM and TD, correlation coefficients between thyroid profile and glycemic parameters, and risk factors. We summarized original articles in the English language during the years 2020 to 2025 in which quantitative data were available. We excluded populations without T2DM, papers that were not empirical, very small case series (less than 30 patients in each series), and studies that did not have data for this study.

Data extraction and quality assessment

The author extracted the data by using a standard template. The collected data of all studies included a variety of main categories that include the details of the first author, year of publication, country, and design of the study, and participant information (sample size, age, and gender). Methodological information, including diagnostic criteria for thyroid dysfunction and type 2 diabetes, was also compiled. Extraction of outcome data included prevalence and statistical association measures (correlation coefficients, odds ratios with confidence intervals) as well as the main findings on risk factors and biochemical correlates. The Newcastle-Ottawa Scale (NOS) was used to assess the methodological quality of the studies, ranging from high quality (7 - 9 stars), moderate quality (5 - 6 stars), and inferior quality (≤ 4 stars).

Data synthesis and meta-analysis

Statistical analyses were done with the R software (version 4.3.0). Freeman-Tukey double arcsine transformed proportions were used for pooling in a prevalence meta-analysis. The summary odds ratios for categorical variables were combined using the Mantel-Haenszel method. Correlation coefficients were pooled after the Z-transformation of Fisher. All analyses were performed with random effects models because heterogeneity was expected. Heterogeneity was tested through the I^2 statistic. 25, 50, and 75% represented low, moderate, and high heterogeneity. Subgroup analysis was performed according to geographic region, quality of the study, and type of thyroid dysfunction. Sensitivity analyses were carried out to identify the impact of individual studies on the overall results. Funnel plots and Egger's test were used to identify publication bias in meta-analyses, which included ≥ 10 studies.

RESULTS

Study characteristics and quality assessment

Based on a systematic search, 50 studies met the inclusion criteria, resulting in a total sample size of 30,244 individuals aged 30 to 70 years. The included research papers were drawn from various global distributions and included contributions from many regions of the world, including South Asia (e.g., India, Pakistan, Nepal, Bangladesh), the Middle East and North Africa (e.g., Libya, Nigeria), and East Asia (e.g., China). Cross-sectional studies were the most common study design (78%), and most were conducted in a hospital (82%). According to quality assessment, 22 studies were of high quality (NOS: 7 - 9), 24 studies were of moderate quality (NOS: 5 - 6), and the remaining four were rated as being of low quality (NOS ≤ 4). Frequent methodological deficiencies were that studies were single-center, and there was the potential for sampling bias.

Prevalence of thyroid dysfunction in T2DM

Nineteen studies with 4,937 patients with T2DM provided data that could be used for an analysis of prevalence. The pooled prevalence of thyroid dysfunction was found to be 27.3% (95% CI: 24.4% - 30.3%, I^2 = 94%) (Figure 2). Individual study estimates were between 13.2% and 47.8%. In non-diabetic control groups, the pooled prevalence was 3.2% (95% CI: 2.5% - 4.1%). The odds of thyroid dysfunction were significantly increased among patients with T2DM (pooled OR 3.89, 95% CI: 2.15 - 7.05, I^2 = 85%). Because of the large amount of heterogeneity between studies and varied criteria for diagnosing thyroid dysfunction, compari-

Table 1. Study quality and types of thyroid dysfunction in included studies (descriptive summary).

Category	Subcategory	Finding	No. of studies
Study quality (Newcastle - Ottawa Scale)	high quality	NOS 7 - 9 stars	22 studies
	moderate quality	NOS 5 - 6 stars	24 studies
	low quality	NOS ≤ 4 stars	4 studies
Type of thyroid dysfunction	subclinical hypothyroidism	most common abnormality; approximately 62.1% of all thyroid dysfunction cases in studies reporting subtype distribution	38 studies
	overt hypothyroidism	3.9% - 8.5% of patients with T2DM	41 studies
	subclinical hyperthyroidism	1.5% - 6.2% of patients with T2DM	35 studies
	overt hyperthyroidism	1.0% - 11.4% of patients with T2DM	39 studies

Estimates are presented descriptively because of substantial heterogeneity and varying diagnostic criteria; they are not additional pooled estimates. NOS: Newcastle - Ottawa Scale, T2DM: type 2 diabetes mellitus.

Table 2. Associations of thyroid and metabolic parameters.

Parameter 1	Parameter 2	Association Direction	Statistical Finding	Notes
TSH	HbA1c/FBG	positive	consistent positive association	a reliable pooled estimate was not derivable due to heterogeneity
FT3/FT4	HbA1c/FBG	negative	consistent negative association	supported by multiple studies reporting significant negative correlations
TSH	HOMA-IR	positive	positive association reported	maximum reported correlation: r = +0.50 (Study - 25)

Table 3. Metabolic alterations in T2DM patients with hypothyroidism.

Comparison	Parameters	Finding	Significance
Hypothyroid T2DM vs. Euthyroid T2DM	LDL cholesterol	significantly elevated	$p < 0.05$
Hypothyroid T2DM vs. Euthyroid T2DM	triglycerides	significantly elevated	$p < 0.05$

Table 4. Risk factors for thyroid dysfunction in T2DM patients.

Risk Factor	No. of Studies	Pooled OR (95% CI)	I^2 (%)	Interpretation
Female gender	28	2.35 (2.0 - 2.76)	75	consistent across regions, the highest risk in post menopause
Age > 50 years	24	3.12 (1.98 - 4.91)	82	risk increases progressively with age; the strongest effect is > 60 years
HbA1c > 7%	18	2.66 (1.94 - 3.65)	41	independent of diabetes duration in most multivariable models
Anti-TPO antibodies	4	12.8 (6.4 - 25.6)	88	based on only 4 studies, very high heterogeneity - interpret cautiously; reflects autoimmune predisposition
Family history of thyroid dysfunction	11	2.02 (1.44 - 2.83)	52	moderate heterogeneity; consistent direction in all studies

Thirty-two studies contributed to the risk-factor meta-analysis; counts for individual risk factors are not mutually exclusive. OR: odds ratio, CI: confidence interval, anti-TPO: anti-thyroid peroxidase antibody.

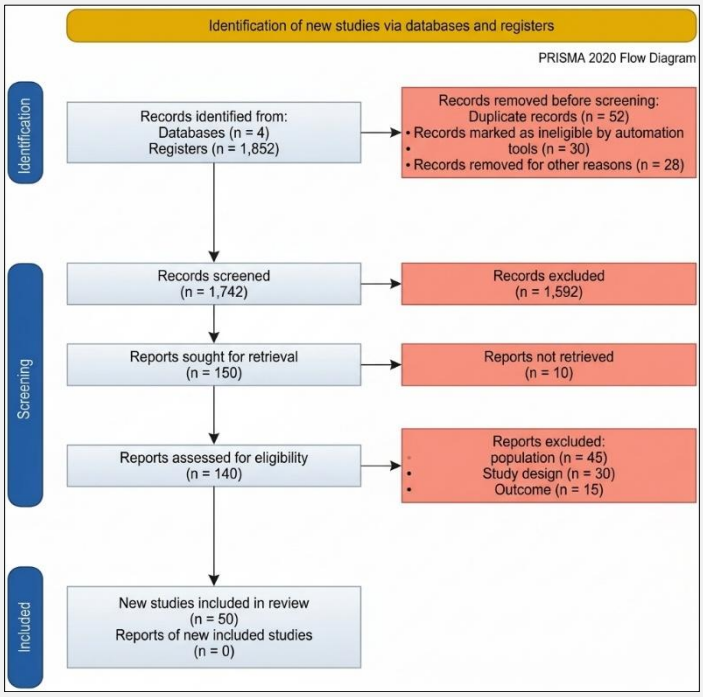


Figure 1. PRISMA 2020 flow diagram for study inclusion.

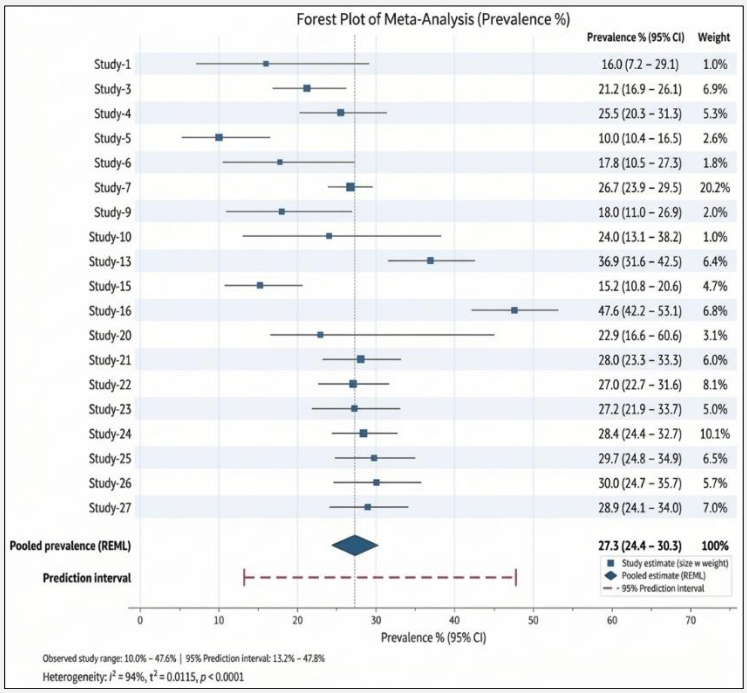


Figure 2. Forest plot of prevalence estimates by individual study and weighted pooled estimate (95% CI).

sons between studies based on study quality and type of thyroid dysfunction are presented descriptively in Table 1 and not as further pooled estimates.

Biochemical correlations

The meta-analysis demonstrated marked and consistent biochemical associations between thyroid hormones and metabolic control in T2DM. In a number of studies, thyroid-stimulating hormone (TSH) stayed associated with glycemic measures, including HbA1c and fasting blood glucose (FBG), positively. The correlation coefficients and the methodologies were very heterogeneous and did not allow for calculating a reliable pooled estimate. Conversely, there were always significant negative associations between free triiodothyronine (FT3) and free thyroxine (FT4), and these glycemic indicators at the surveys of the studies that were included. TSH was also found to exhibit a large correlation to insulin resistance, as measured by HOMA-IR (Table 2). In addition, the analysis showed that hypothyroidism was also typified by a high degree of dyslipidemia with higher levels of LDL cholesterol and triglycerides (Table 3).

Risk factors for thyroid dysfunction

The relative risk of thyroid dysfunction in patients with type 2 diabetes was found in the present meta-analysis, which comprised 32 studies (Table 4). The biggest predictor was older age (> 50 years) with significantly increased odds of nearly four times (pooled OR: 3.12 (95% CI: 1.98 - 4.91, I^2 = 82%). The female gender was also a prominent risk factor, with a > 2 times (pooled OR 2.35 (95% CI: 2.00 - 2.76, I^2 = 75%) risk) than in men. In clinical terms, poor glycemic control (HbA1c > 7%) was a significant independent risk factor (odds ratio > 2.5-fold) expressed as a pooled OR 2.66 (95% CI: 1.94 to 3.65, I^2 = 41%). Positive anti-TPO antibodies are associated with a significantly increased risk in the few studies that reported this effect (pooled OR 12.8; 95% CI 6.4 - 25.6; 4 studies only). However, this estimate should be taken with extreme caution as it is based on limited studies and it may be unstable. A 2-fold higher risk was also found in association with a positive family history of thyroid dysfunction, OR 2.02 (95% CI: 1.44 - 2.83). Substantial heterogeneity was observed in terms of age and gender (I^2 = 85% and I^2 = 75%), and moderate heterogeneity was observed in HbA1c (I^2 = 41%). This statistical profile shows that older diabetic patients, especially women with poor glycemic control or evidence of autoimmunity, are the most at-risk group.

Assessment of publication bias

A funnel plot was used to assess publication bias visually (22 studies) and formally using Egger's test. A wide dispersion of study estimates showed consistent with high heterogeneity (I^2 = 94%), and Egger's test did not indicate significant funnel plot asymmetry (p = 0.2934), suggesting no strong small-study effects on the pooled prevalence estimate.

DISCUSSION

This review shows that thyroid dysfunction is a common comorbidity in type 2 diabetes, and the pooled prevalence of about 27.3% is very high relative to the 3.2% in comparison groups where there is no diabetes. The odds ratio of thyroid dysfunction in T2DM versus controls was 3.89 (95% CI: 2.15 - 7.05; I^2 = 85%), and this reflects that there was a strong epidemiological relationship. The most common type was subclinical hypothyroidism, which represented approximately 62% of all thyroid dysfunctions in those studies that reported subtype distribution.

Synthesis of key findings

In this study, it is reported that the comorbidity of TD is common in T2DM, and the pooled prevalence is 27.3% which is much greater than the prevalence of 3.2% in non-diabetic controls [3,26]. This result agrees with the high odds ratio of TD in T2DM patients (OR: 3.89), which suggests a strong epidemiological interaction between the two illnesses [27]. The most common form of TD was SCH (identified in 62.1% of cases with TD) [28]. Of interest is the extremely high prevalence of TD (13.7 - 18.0) in more recent groups of T2DM individuals; thyroid alterations may coincide with or even precede, rather than result from, long-term hyperglycemia [29]. Geographically, the higher prevalence rate among South Asians in comparison to the other ethnicities (24 - 36.8% vs. 20.5 - 25%) indicates that genetic predispositions, dietary contributions, such as iodine status, and various prevalence rates of the metabolic syndrome could contribute to the epidemic [30].

Pathophysiological interrelationships

TSH levels reflect the association with glycemic parameters HbA1c and FBG, which are related to the precision of thyroid hormones FT3 and FT4. This link provides one reason for the clinical connection [31]. The pattern results that hypothyroidism in its subclinical stage aggravates the insulin resistance and interferes with the glucose balance. The mechanisms include peripheral use of glucose, changes in the amount of the glucose transporter GLUT4, and changes in how the liver makes glucose [32]. This meta-analysis revealed that a positive association was found between the TSH and HOMA-IR. It further showed that the TSH was negatively associated with the FT3/FT4. The importance of the balance of thyroid hormones becomes apparent in research that clarifies that some patterns of the ratio of FT3toFT4 are related to the results experienced in T2DM patients. Studies have shown that even slight changes in thyroid hormone metabolism and thyroid hormone conversion may be part of how diabetes mellitus starts.

The large number of patients suffering from hypothyroidism and dyslipidemia in T2DM reveals an interaction that increases the risk for CV. Co-managing hypothyroidism and dyslipidemia in type 2 diabetes mellitus

(T2DM) is essential. Notably, long-term data show that higher baseline TSH levels are linked to a lower risk of developing T2DM (hazard ratio = 0.75) [34]. Higher baseline TSH observation comes from people who do not have diabetes yet, and probably shows that the hypothalamic-pituitary-thyroid axis adapts in diabetes instead of protecting people who already have T2DM. Conversely, the modestly increased risk observed with an increment in FT4 (HR = 1.06) suggests a need to explore the trajectory of thyroid and glucose metabolism with the progression of time.

Clinical implications and risk stratification

Since the alteration in FBG and HbA1c results is clear, this meets the criteria for a correct diagnosis and prompt treatment [35]. This is necessary for managing thyroid disease and also helps control blood sugar. Risk factor analysis can be used to identify high-risk patients who are most likely to benefit from periodic systematic screening. Likely, estrogen-related immune modulation and the higher incidence of autoimmune thyroiditis in females are the major factors contributing to the odds ratio (OR 2.35, 95% CI 2.00 - 2.76) associated with female sex. This is evidenced by the prevalence of anti-TPO antibodies in hypothyroid groups. [36]. The large age distributions of the samples under study made it difficult to determine an age cutoff of risk, but the age over 50 years was always associated with a three-fold risk (pooled OR 3.12, 95% CI 1.98 - 4.91) [37]. This can be explained by the age-related changes in the hypothalamic-pituitary-thyroid (HPT) axis and the build-up of oxidative stress. Besides that, poor glycemic control (HbA1c exceeding 7) also influenced the outcomes of the study (OR = 2.66). [38]. The other risk indicators that the scientists have considered possibly are: a history of diabetes over the period, family history of thyroid disease, obesity, and goitre [39]. There is no direct relationship between hypertension and thyroid disease, but the interaction of both type 2 diabetes and thyroid disease leads to heart disease, hence the need to have combined care plans. Biologically, it is reasonable to expect the existence of such associations because the same pathophysiologic processes involve insulin resistance and impaired control of the hypothalamic-pituitary-thyroid axis. Hypothyroidism can contribute to T2DM, which decelerates metabolic rate and the transport of glucose, and can cause thyroid cell death or deiodinase dysfunction because of hyperglycemia [40]. The findings provide a strong rationale for thyroid screening of patients with T2DM (especially women and patients above 50 years of age and with poorly regulated blood sugar levels, as indicated by a HbA1c over 7%).

Methodological considerations and limitations

Certain methodological considerations should be highlighted. Even though an overall positive correlation between TSH and HbA1c was found, the size of this linearity varied significantly, which does not allow for creating a strong pooled correlation. This variance is probably because of the fluctuation of assay platform, study designs, and patient characteristics underlying the studies included. The high odds ratio of anti-TPO positivity was based on a very limited four studies and hence not statistically stable; it is represented to show completeness, but this should not be regarded as conclusive. According to the test by Egger, the pooled estimate of prevalence was strong and not significantly affected by the small-study effects. The literature search was finalised in March 2025, and they have not been included in this review.

Conclusion and future directions

Overall, this discussion validates the presence of a strong mutual correlation between type 2 diabetes (T2DM) and thyroid dysfunction. The results support the idea of regular screening of the thyroid in diabetic individuals, especially females, elderly individuals, and individuals with impaired glucose metabolism, and also suggest the investigation of glucose metabolism in thyroid patients. Longitudinal studies should be the focus of future research to develop the cause-and-effect relationship as well as reveal the underlying molecular mechanisms.

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Declaration of Generative AI in Scientific Writing:

I used Research Rabbit and Elicit for searching and Grammarly AI for language editing. Regardless, the scientific content, interpretation, and conclusions remain entirely my responsibility.

Declaration of Interest:

None.

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