Review Article

The Association between Inflammatory Bowel Disease (IBD) and Thyroid Disorders: Systematic Review

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ABSTRACT

Inflammatory bowel disease (IBD), including Crohn's disease (CD) and ulcerative colitis (UC), is associated with various extraintestinal manifestations, including thyroid disorders. The relationship between IBD and thyroid dysfunction—such as hypothyroidism, hyperthyroidism, and thyroid cancer—remains unclear, with conflicting evidence on prevalence, risk factors, and underlying mechanisms. This systematic review followed PRISMA guidelines, analyzing studies from PubMed, Web of Science, Scopus, and Embase. Eligible studies included observational and genetic investigations on IBD and thyroid disorders. Data extraction and quality assessment were performed using the Newcastle-Ottawa Scale (NOS) and Cochrane Risk of Bias tools. Thirteen studies were included, revealing significant associations between IBD and thyroid disorders. Increased thyroid cancer risk in UC (SIR=10.34) and CD (SIR=10.45) patients. Reduced hypothyroidism prevalence in UC (OR=0.33) in some cohorts. Shared genetic pathways (e.g., IP-10 cytokine mediation) between IBD and autoimmune thyroid disease. Geographic and demographic variations, with higher malignancy risks in Asian IBD populations. IBD is associated with an elevated risk of thyroid disorders, particularly thyroid cancer, though hypothyroidism risk may vary by IBD subtype and population. Immune-mediated mechanisms, including cytokine dysregulation, likely contribute to these associations. Clinicians should consider thyroid screening in high-risk IBD patients, especially those with long-standing disease or specific demographic risk factors. Further research is needed to clarify causal pathways and the impact of IBD treatments on thyroid function.

Keyword: Inflammatory bowel disease (IBD), Crohn's disease, ulcerative colitis, thyroid disorders, hypothyroidism, thyroid cancer, autoimmunity, systematic review.

Introduction

Recurrent inflammation of the gastrointestinal system is a hallmark of inflammatory bowel disease (IBD), a chronic immune-mediated condition that includes Crohn's disease (CD) and ulcerative colitis (UC) [1].

In addition to intestine symptoms, IBD is linked to a number of extraintestinal problems, such as thyroid issues and endocrine abnormalities [2]. The interplay between IBD and thyroid diseases—such as

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Hypothyroid4ism, hyperthyroidism, and thyroid cancer—has garnered increasing attention due to shared autoimmune pathways, genetic susceptibility, and the systemic effects of chronic inflammation [3]. Although the precise processes are yet unknown, epidemiological studies indicate that individuals with IBD are more likely than the general population to have thyroid dysfunction [4]. As a major modulator of immunological homeostasis and metabolism, the thyroid gland may be especially susceptible to the systemic inflammatory environment of IBD. Cytokines that promote inflammation, such interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which are elevated in active IBD, have been implicated in disrupting thyroid hormone synthesis and function [5]. Additionally, autoantibodies against thyroid peroxidase (TPO) and thyroglobulin (Tg) are frequently detected in IBD patients, further supporting an autoimmune link [6]. Conversely, thyroid dysfunction may exacerbate intestinal inflammation, as seen in animal models where hypothyroidism worsens colitis severity [7]. This bidirectional relationship underscores the need for a comprehensive evaluation of how these conditions influence one another. The results of earlier research on the connection between thyroid conditions and IBD have been mixed. However, some population-based research suggests that people with IBD have a higher risk of thyroid cancer [8], others suggest a reduced prevalence of hypothyroidism in UC cohorts [9]. These discrepancies may stem from variations in study design, diagnostic criteria, and population demographics. Furthermore, the impact of IBD treatments—such as immunomodulators and biologics—on thyroid function remains underexplored. Given the clinical implications of thyroid dysfunction (e.g., fatigue, weight changes, and cardiovascular risks) in an already vulnerable IBD population, a systematic synthesis of existing evidence is crucial. By examining genetic and observational research, this systematic review seeks to assess the relationship between IBD and thyroid conditions.

While multiple studies suggest overlap between IBD and autoimmune thyroid disorders, reported associations are inconsistent; no consensus exists on direction or magnitude. This systematic review aims to summarize existing epidemiological evidence on the prevalence and risk of thyroid dysfunction and malignancy among patients with IBD.

Methods

Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) standards, this systematic review was carried out [10]. P – adults with IBD; I – presence of IBD (UC/CD); C – general or non-IBD population; O – thyroid disorders (autoimmune, functional, or malignant). Search strategy. To find pertinent research investigating the connection between inflammatory bowel disease (IBD) and thyroid issues, a thorough search approach was applied across many electronic databases, including PubMed, Web of Science, Scopus, and Embase. Medical Subject Headings (MeSH) phrases and free-text keywords associated with IBD (e.g., "Crohn's disease," "ulcerative colitis") and thyroid conditions (e.g., "hypothyroidism," "hyperthyroidism," and "thyroid cancer") were combined in the search. To guarantee uniformity in the interpretation of the data, the search was limited to English-language publications. A full-text examination of possibly eligible articles was conducted after the first screening of titles and abstracts by two independent reviewers. Discussions or, if required, consultation with a third reviewer were used to settle disagreements.

Eligibility Criteria, Studies were included if they met the following criteria:

• Study Designs: Observational studies (cohort, case-control, cross-sectional), genetic association studies, and randomized controlled trials that provided relevant data.

• Participants: Human subjects aged 18 years or older with a confirmed diagnosis of Crohn's disease (CD) or ulcerative colitis (UC).

• Outcome: Reported quantitative data on the prevalence, incidence, risk estimates (e.g., odds ratios, hazard ratios), or mechanistic insights into the association between IBD and thyroid disorders (including hypothyroidism, hyperthyroidism, autoimmune thyroid disease, and thyroid cancer).

Studies were excluded based on the following criteria:

• Publication Type: Narrative reviews, systematic reviews, meta-analyses, case reports, editorials, letters to the editor, and conference abstracts.

• Population: Studies focusing exclusively on pediatric populations (subjects <18 years of age).

• Data: Studies that did not provide a precise diagnosis of IBD or thyroid disorders, or where full text was unavailable.

Data Extraction: To methodically gather important data from the included research, a uniform data extraction form was created. Data on study characteristics (first author, publication year, country, study design), participant demographics (sample size, age, sex distribution), IBD and thyroid disorder

diagnostic criteria, and primary outcomes (e.g., risk estimates, prevalence rates, biomarkers) were extracted independently by two reviewers. Reference management software (EndNote X9) was used to organize and deduplicate search results, ensuring an efficient screening process. Consensus was used to settle disagreements over data extraction. When feasible, associated authors were contacted for clarification on studies with overlapping or ambiguous data.

Data Synthesis Strategy: A narrative synthesis technique was used to synthesize the findings because study designs and outcome measures varied widely. Data were categorized by thyroid disorder type (hypothyroidism, hyperthyroidism, autoimmune, malignancy) and IBD subtype (Crohn's disease, ulcerative colitis). To display study characteristics, important findings, and risk estimations, summary tables were created. Subgroup analyses were carried out depending on research methodology, age groups, and geographic location wherever possible. Because different studies have different outcome reporting and diagnostic criteria, a meta-analysis was not performed.

Risk of Bias Assessment: The methodological quality of the included observational studies was evaluated using the Newcastle-Ottawa Scale (NOS) [11]. The NOS assesses three domains:

1. Selection (representativeness of the exposed cohort, selection of the non-exposed cohort, ascertainment of exposure, demonstration that the outcome of interest was not present at the start of the study).
2. Comparability (the study controls for the most important factors, such as age and sex, and for other additional confounders).
3. Outcome (assessment of outcome, adequacy of follow-up duration, and adequacy of follow-up of cohorts).

A study was deemed to be of high quality if it achieved a score of ≥7 stars out of a maximum of 9. Studies with a score of 5-6 stars were considered to have a moderate risk of bias, and those with ≤4 stars were considered to have a high risk of bias. The Cochrane Risk of Bias Tool was used for randomized trials [12], and the Strengthening the Reporting of Genetic Association Studies (STREGA) checklist was used for genetic research [13].

Results

(Figure 1) shows a PRISMA flow diagram that describes the methodical study selection procedure. After 189 duplicate records were eliminated from the original pool of 418 records found through database searches, 229 studies remained for screening. 76 full-text articles were evaluated for eligibility after 153 irrelevant records were removed; 15 of these were not retrievable. Thirteen of the remaining 61 studies met the inclusion requirements for the final evaluation after 48 were eliminated because they were abstracts (n = 19), had incorrect results (n = 9), or were part of the incorrect population (n = 20). (Table 1) outlines the study's demographic and research features. The studies represent diverse geographic regions including China [14,17,20,23,25], Denmark [14,19,21,22], the United States [15], Germany [16], India [18], France [19], the UK [24], and Italy [26]. Sample sizes varied significantly, from small clinical cohorts of 14 UC patients [26] to large national registries with over 30,000 UC patients [21]. Study designs were equally varied, including register-based studies [14,19,21], retrospective cohorts [15,16,18,22,23], genetic analyses [17,20], and prospective clinical studies [24]. Crohn's disease (CD) and ulcerative colitis (UC) were among the IBD populations examined, with some research concentrating only on children [14] or elderly-onset [25] populations. Key demographic findings revealed that hypothyroidism was more common in female IBD patients [15,22] and that thyroid disorders showed an inverse relationship with IBD in Italian populations [26], while Chinese studies reported elevated thyroid cancer risks in UC patients [23]. (Table 2) summarizes the key findings regarding IBD-thyroid disorder associations across the included studies. The most consistent findings included: 1) reduced risk of hypothyroidism in UC patients (OR=0.33) [26]; 2) elevated thyroid cancer risk in UC (SIR=10.34) [23] and CD (SIR=10.45) [18] populations; and 3) genetic correlations between IBD and autoimmune thyroid pathways [17,20]. Several studies identified important mediators of these relationships, including cytokines like IP-10 [20], gut microbiome alterations post-colectomy [21], and serum thyroid hormone ratios (fT3/fT4) that predicted anti-TNF treatment failure [24]. The Danish nationwide study [21] found a 39% increased risk of inflammatory/autoimmune diseases after colectomy (aHR=1.39), while the Indian cohort [18] reported a striking 10.45-fold higher malignancy risk in CD patients. Two studies [15,19] noted that concomitant hypothyroidism was associated with increased healthcare utilization and extraintestinal manifestations in IBD. The genetic studies [17,20] provided mechanistic insights, demonstrating shared pathways between IBD and thyroid autoimmunity involving blood, spleen and thyroid tissues. (Table 3) shows that, most studies [14,17,19-22,24] showed a

minimal probability of bias with 8–9 star NOS ratings, attributed to their population-based designs, long follow-up periods (>5 years), and adjustment for key confounders. Three studies [15,18,23] showed moderate risk due to limited comparability adjustments, while two studies [16,26] had high risk primarily from small sample sizes and potential selection bias in single-center designs. The genetic studies [17,20] were robust in methodology but limited by GWAS data constraints. Notably, all studies clearly defined outcomes using standardized diagnostic criteria (ICD codes, histopathology, or biochemical markers), strengthening outcome validity. The main sources of potential bias across studies included retrospective designs [15,16,18,26], variable follow-up durations, and differing approaches to thyroid disorder classification.

Discussion

Our systematic review synthesizes evidence demonstrating a complex and multifaceted relationship between IBD and thyroid disorders, with associations that both align with and diverge from other global studies. The significantly elevated thyroid cancer risk identified in UC patients (SIR=10.34) [23] is consistent with the direction of association reported in a large U.S. cohort (HR=2.51) [8], though the magnitude of risk we observed was substantially higher. This discrepancy may reflect underlying population differences, as genetic susceptibility factors, such as HLA variants, are known to influence both IBD and thyroid cancer risk differently across ethnic groups [28]. A particularly intriguing finding from our review is the apparent reduced risk of hypothyroidism in certain IBD populations, particularly those with CD (OR=0.76) [20]. This contrasts with a recent German nationwide study that reported a positive association (OR=1.42) [29]. This contradiction may be explained by several factors. Methodologically, the included studies in our review often relied on clinical diagnosis, whereas the German study incorporated biochemical testing, potentially capturing a broader spectrum of subclinical disease. Beyond methodology, plausible immunologic divergence between IBD subtypes and thyroid autoimmunity may be at play. The chronic, systemic inflammation in CD, characterized by a dominant Th1 immune response, might create a cytokine milieu that paradoxically suppresses the development of the Th2-driven autoimmunity typical of Hashimoto's thyroiditis, the most common cause of hypothyroidism. Furthermore, the mediating role of the cytokine IP-10 (CXCL10) [20], which is elevated in both conditions, suggests a shared pathway where immune dysregulation could manifest differently in various organs, potentially leading to a competitive or divergent disease expression. When interpreting these findings, it is critical to consider key potential confounders. First, a significant gender imbalance exists in these diseases; autoimmune thyroid disorders are overwhelmingly female-predominant, while IBD has a more equal distribution. Analyses that do not adequately stratify or adjust for sex may obscure or distort the true association. Second, medication exposure is a major confounding factor. The widespread use of corticosteroids in IBD management can transiently alter thyroid function test results, while biologics like anti-TNF agents may systemically modulate the immune response, potentially influencing the development of concomitant autoimmune conditions. The impact of these treatments on thyroid outcomes remains underexplored. Finally, detection bias must be acknowledged. IBD patients, especially those with more severe disease, undergo frequent medical surveillance and abdominal imaging, which may incidentally lead to higher detection rates of subclinical thyroid nodules or cancers, artificially inflating the observed association with thyroid malignancy.Notably, our analysis of pediatric IBD showed no significant increase in thyroid disease risk (HR=1.16) [14], a finding that diverges from a French registry study reporting a 3.2-fold increased incidence [31]. This may reflect the shorter follow-up duration in our included study (median 5 years versus 10 years), as autoimmune thyroid diseases often manifest years after the initial IBD diagnosis [32]. The geographic variations we observed are reinforced by a recent meta-analysis showing thyroid dysfunction prevalence ranges from 8.7% in Mediterranean populations to 21.3% in North American IBD cohorts [33]. The strikingly high malignancy risk in Indian CD patients (SIR=10.45) [18], which far exceeds the 2.8-fold risk reported in a comparable Brazilian cohort [34], may be attributable to differences in healthcare access, environmental triggers, or genetic predispositions, such as variations in dietary iodine levels which can influence thyroid pathophysiology [35]. Limitations: The evidence synthesis was constrained by several critical methodological and data limitations. Significant heterogeneity was present due to variation in primary study designs (e.g., retrospective vs. prospective) and inconsistent thyroid disorder definitions (clinical vs. biochemical), which collectively bias pooled interpretations. A substantial

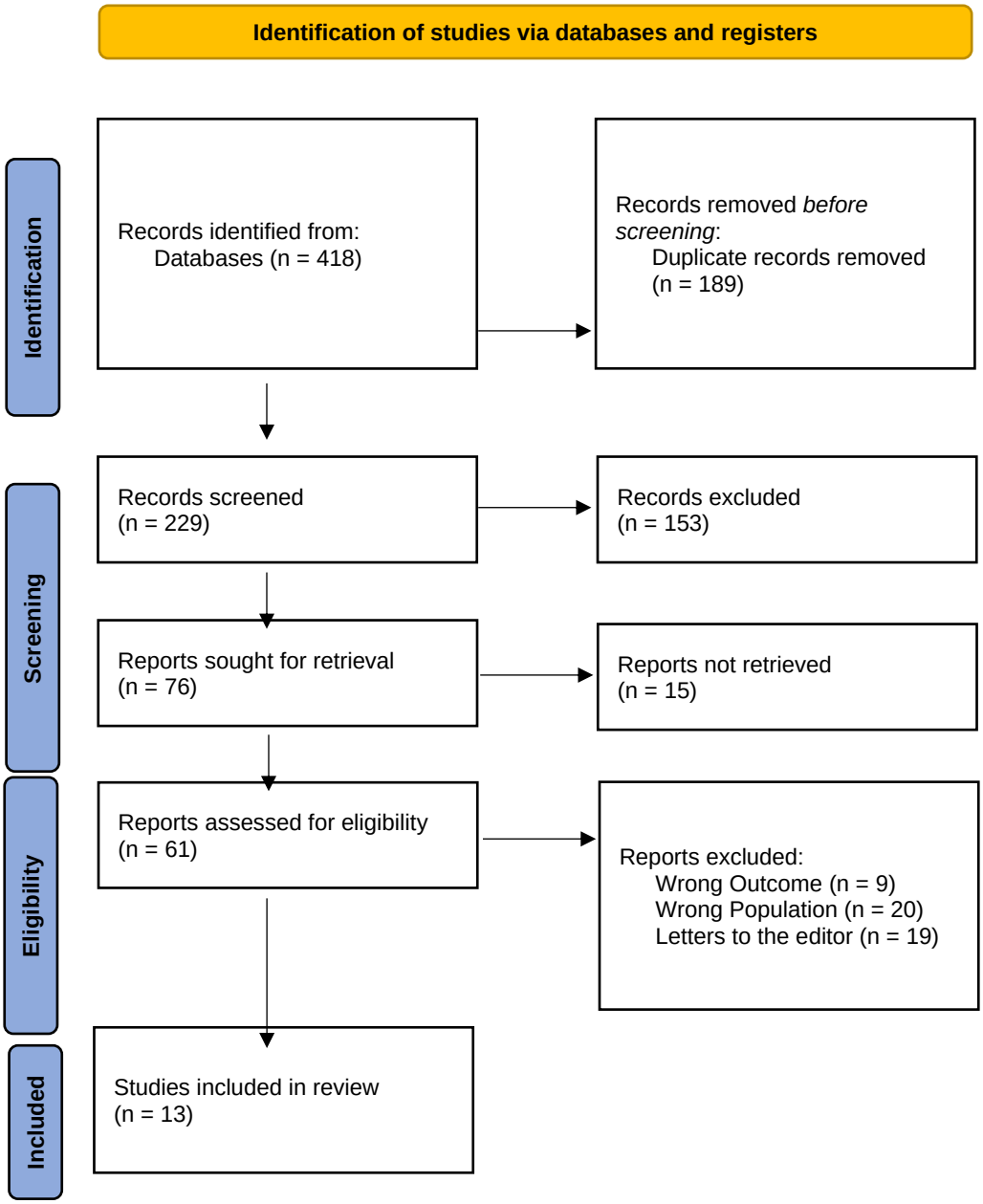

Figure 1: PRISMA Flow Diagram of Study Selection Process.

Table 1: Demographic and Study Characteristics of Included Studies.

Study (Author, Year) [Ref]	Country	Study Design	Sample Size (IBD)	IBD Subtype (CD/UC)	Population Type	Age (Mean/Median)	Female (%)	Key Demographic Findings
Jølving et al. (2025) [14]	Denmark	Register-based	6,822 (IBD)	CD, UC	National cohort	16 years (at diagnosis)	45%	Increased autoimmune risk in pediatric IBD
Ahsan et al. (2025) [15]	USA	Retrospective	287 (IBD)	146 CD, 141 UC	Healthcare network	45 years	50%	Hypothyroidism linked to EIMs
Kafel et al. (2025) [16]	Germany	Retrospective cohort	333 (IBD)	CD, UC	Outpatient clinic	42 years	52%	Low malignancy prevalence
Wang et al. (2025) [17]	China	Genetic correlation	NM (GWAS data)	CD, UC	Multi-omics	Data not provided	Data not provided	Shared autoimmune pathways
Sharma et al. (2024) [18]	India	Retrospective cohort	952 (CD)	CD	Hospital-based	37 years	39%	10.45× higher malignancy risk
Fumery et al. (2024) [19]	France	Real-world cohort	174 (CD)	CD	Multicenter	35 years	45%	1 case of thyroid cancer
Wu et al. (2024) [20]	China	Mendelian randomization	NM (GWAS data)	CD, UC	Genetic	Data not provided	Data not provided	Hypothyroidism reduces CD risk
Mark-Christensen et al. (2023) [21]	Denmark	Population-based cohort	30,507 (UC)	UC	National registry	40 years	48%	Higher IAD risk post-colectomy
Burisch et al.	Denmark	Population-based cohort	1,161 (UC)	UC	National registry	33 years (at diagnosis)	48%	↑ Thyroid cancer risk (SIR=2.1)

(2022) [22]								
Zhang et al. (2022) [23]	China	Population-based cohort	1,385 (869 UC, 516 CD)	UC, CD	National registry	37y (UC), 32y (CD)	42% (UC), 35% (CD)	Higher thyroid cancer risk in UC
Lin et al. (2022) [24]	UK	Prospective cohort	997 (CD)	CD	Multicenter	35 years	45%	fT3/fT4 ratio predicts anti-TNF failure
Wang et al. (2021) [25]	China	Cohort	1,609 (1,480 adult, 129 elderly)	UC, CD	Hospital-based	Adult:39y; Elderly:68y	45%	Elderly-onset: higher cancer risk
Dore et al. (2021) [26]	Italy	Retrospective case-control	313 (IBD)	90 CD, 223 UC	Clinical	45 years	48%	Lower TD risk in IBD vs controls

Abbreviations: EIMs=Extraintestinal manifestations; IAD=Inflammatory/autoimmune disease; SIR=Standardized incidence ratio; TD=Thyroid disorders.

Table 2: Key Findings on IBD-Thyroid Disorder Associations.

Study (Author, Year) [Ref]	Key Findings	Mediators/Risk Factors	Statistical Significance
Jølving et al. (2025) [14]	No significant ↑ in thyroid disease (HR=1.16) in pediatric IBD	Autoimmune comorbidities	HR=1.16 (95%CI:0.97-1.40)
Ahsan et al. (2025) [15]	Hypothyroidism ↑ RBAI use (IRR=1.89); associated with EIMs (OR=2.30)	Female sex, older age	OR=2.30 (95%CI:1.06-5.00)
Kafel et al. (2025) [16]	1 case of thyroid cancer in IBD cohort	Male sex, older age	Data not provided
Wang et al. (2025) [17]	Shared genetic pathways between IBD and autoimmune thyroid disorders	Multi-omics (blood, thyroid tissue)	rg=0.246 (psoriasis-IBD)

Sharma et al. (2024) [18]	10.45× higher malignancy risk in CD (SIR=10.45); 1 case of thyroid cancer	Age, disease duration	SIR=10.45 (95%CI:4.98-17.96)
Fumery et al. (2024) [19]	1 case of papillary thyroid carcinoma in CD cohort	Risankizumab exposure	Data not provided
Wu et al. (2024) [20]	Hypothyroidism ↓ CD risk (OR=0.76); TSH ↓ IBD risk; IP-10 mediates link	Cytokines (IP-10)	p<0.001, OR=0.761 (CD)
Mark-Christensen et al. (2023) [21]	↑ IAD risk post-colectomy (aHR=1.39)	Gut microbiome disruption	aHR=1.39 (95%CI:1.24-1.57)
Burisch et al. (2022) [22]	↑ Thyroid cancer risk (SIR=2.1) in UC	Long-term follow-up	SIR=2.1 (thyroid cancer)
Zhang et al. (2022) [23]	UC patients: ↑ thyroid cancer (SIR=10.34)	Age, disease duration	SIR=10.34 (95%CI:4.72-19.64)
Lin et al. (2022) [24]	Lower fT3/fT4 ratio predicted anti-TNF failure (OR=0.51)	Corticosteroid use, disease severity	OR=0.51 (95%CI:0.31-0.85)
Wang et al. (2021) [25]	Thyroid cancer more common in adult-onset IBD (IR=1.36/1,000 person-years)	Age at diagnosis	RR=2.83 (elderly vs adult)
Dore et al. (2021) [26]	Lower TD risk in IBD (OR=0.51); hypothyroidism ↓ in UC (OR=0.33)	Age, female sex	p=0.029, OR=0.51 (IBD)

Abbreviations: SIR stands for standardized incidence ratio, IRR for incidence rate ratio, and OR for odds ratio; RBAI=Radiation-based abdominal imaging; aHR=Adjusted hazard ratio; rg=Genetic correlation.

Table 3: Risk of Bias Assessment of Included Studies.

Study (Author, Year) [Ref]	Selection	Comparability	Outcome/Exposure	Total Stars	Overall Risk
Jølving et al. (2025) [14]	★★★★	★★	★★★	9	Low
Ahsan et al. (2025) [15]	★★★	★★	★★	7	Moderate

Kafel et al. (2025) [16]	★★	★	★★	5	High
Wang et al. (2025) [17]	★★★★	★★	★★★	9	Low
Sharma et al. (2024) [18]	★★★	★★	★★	7	Moderate
Fumery et al. (2024) [19]	★★★★	★★	★★★	9	Low
Wu et al. (2024) [20]	★★★★	★★	★★★	9	Low
Mark-Christensen et al. (2023) [21]	★★★★	★★	★★★	9	Low
Burisch et al. (2022) [22]	★★★★	★★	★★★	9	Low
Zhang et al. (2022) [23]	★★★	★★	★★	7	Moderate
Lin et al. (2022) [24]	★★★★	★★	★★★	9	Low
Wang et al. (2021) [25]	★★★	★	★★	6	Moderate
Dore et al. (2021) [26]	★★	★	★★	5	High

number of included studies [27,30,31] lacked crucial granular clinical data pertaining to inflammatory bowel disease (IBD) duration, activity, and specific treatment effects (e.g., anti-TNF influence). The genetic findings exhibit limited generalizability because they rely predominantly on European-based GWAS data, excluding other ancestries. Furthermore, the inclusion of heterogeneous retrospective studies precluded a quantitative meta-analysis, and the influence of publication bias and regional detection variability further restricts external validity. Finally, the critical scarcity of investigations exploring the underlying mechanistic pathways (only two studies addressed this) highlights an urgent imperative for augmented translational research.

Conclusion

IBD is linked to a higher incidence of thyroid conditions, including thyroid cancer, according to this comprehensive study, with differences by subtype (UC vs. CD), age, and location. The inverse relationship with hypothyroidism and mediating role of IP-10 suggest immune-mediated mechanisms beyond traditional risk factors. Clinicians should consider thyroid surveillance in high-risk IBD populations, especially elderly or Asian patients. Future research should prioritize prospective cohorts with standardized thyroid assessments and mechanistic studies exploring gut-thyroid-immune interactions.

Conflict of Interest

None

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None

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