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ENDOMETRIOSIS AND THYROID DYSFUNCTION: EPIDEMIOLOGICAL EVIDENCE, THYROID AUTOIMMUNITY, AND TSH-T3/T4 AXIS MECHANISMS IN ENDOMETRIOSIS PATHOPHYSIOLOGY

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ABSTRACT

Background: Endometriosis is a chronic inflammatory gynecological condition characterized by immune dysregulation and a wide range of systemic complications. Thyroid disorders are also common in women of reproductive age and may overlap with endometriosis through shared autoimmune pathways and endocrine signaling.
Objective: This review aims to summarize epidemiological and clinical evidence linking endometriosis with thyroid dysfunction and thyroid autoimmunity and to place these findings in the context of mechanistic research on the TSH–T3/T4 axis.
Methods: Researchers searched for original studies reporting both diagnosed endometriosis and thyroid-related outcomes, including thyroid disorders, autoimmune thyroiditis, thyroid autoantibodies, TSH receptor antibodies, and thyroid neoplasms. The evidence was synthesized narratively.
Results: Large EHR- and claims-based studies generally reported increased risks of hypothyroidism, hyperthyroidism, Graves' disease, thyroiditis, goiter, and thyroid neoplasms after an endometriosis diagnosis. Clinical and biomarker studies were more variable: one case–control cohort found comparable rates of thyroid dysfunction and anti-thyroid antibodies in cases and controls, whereas other cohorts reported a higher prevalence of autoimmune thyroiditis, lower free T4 levels, associations between anti-thyroid peroxidase antibody levels and endometrioma size, and a higher prevalence of Hashimoto thyroiditis in an IVF/ICSI population. In one diagnostic biomarker study, adding anti-thyroid peroxidase and thyroglobulin antibodies to CA125 improved discrimination between ovarian endometriomas and other benign ovarian tumors (AUC 0.924). Studies of TRAb suggested elevated titers in some cohorts, but this finding was not reproduced with routine clinical TRAb assays, underscoring assay dependence. Mechanistic studies support biological plausibility by showing altered thyroid hormone metabolism in endometriotic tissue and proliferative, pro-oxidative effects of TSH, T3, and T4.
Conclusions: Overall, the literature points to an association between endometriosis and thyroid-related outcomes; however, substantial heterogeneity and potential bias make it difficult to translate these findings into clear screening recommendations. Future research should prioritize standardized phenotyping, harmonized antibody testing, and prospective studies linking clinical thyroid status with the biology of endometriosis.

KEYWORDS

Endometriosis, Thyroid Dysfunction, Autoimmune Thyroiditis, Thyroid Autoantibodies, TSH Receptor Antibodies, TSH-T3/T4 Axis

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1. Introduction

Endometriosis is a chronic, estrogen-dependent inflammatory condition in which endometrial-like tissue grows outside the uterine cavity. Although it is best known for pelvic pain and infertility, increasing evidence suggests that endometriosis is also accompanied by broader immune and endocrine disturbances, with comorbidity patterns that extend beyond the reproductive tract (Ek et al., 2018; Korošec et al., 2024). This broader perspective is clinically important. Comorbid conditions can blur symptom profiles—for example, gastrointestinal complaints may be attributed to irritable bowel syndrome—shape healthcare use, and complicate the interpretation of biomarkers and routine laboratory tests (Petersson et al., 2025).

Within this landscape, thyroid disorders stand out as a particularly plausible and clinically relevant comorbidity. First, thyroid dysfunction and thyroid autoimmunity are common in women of reproductive age and are closely tied to fertility and pregnancy outcomes—issues that are also central to endometriosis care (Poppe et al., 2002; Korošec et al., 2024). Second, autoimmune thyroiditis and Graves' disease represent immune states characterized by autoantibody production and altered inflammatory signaling, features that have repeatedly been discussed in relation to endometriosis (Ek et al., 2018; Porpora et al., 2020). Third, the thyroid-stimulating hormone (TSH)–triiodothyronine (T3)/thyroxine (T4) axis is a fundamental endocrine

system, and mechanistic studies suggest that components of this pathway—including TSH receptor expression in ectopic endometrium—may influence endometriotic tissue behavior directly (Şerifoğlu et al., 2023; Peyneau et al., 2019).

Recent years have also changed what is methodologically feasible. Technology-enabled datasets, including administrative claims and large electronic health record (EHR) networks, now allow researchers to examine comorbidity patterns and long-term outcomes at a scale that earlier clinic-based cohorts could not match (Aziz et al., 2025; Shih et al., 2026; Yuk et al., 2016). At the same time, these data sources introduce their own threats to validity—such as variability in diagnostic coding, differences in healthcare contact, and surveillance bias—which must be considered before translating associations into screening recommendations or mechanistic conclusions.

Against this background, the aim of this review is to bring together three complementary lines of evidence under the working title *"Endometriosis and thyroid dysfunction: epidemiological evidence, thyroid autoimmunity, and TSH–T3/T4 axis mechanisms in endometriosis pathophysiology."* Specifically, we: (i) summarize epidemiological findings linking diagnosed endometriosis to subsequent thyroid disorders and thyroid neoplasms; (ii) evaluate clinical evidence on thyroid function tests and thyroid autoantibodies in women with endometriosis, including emerging research on TSH receptor antibodies (TRAb); and (iii) discuss mechanistic studies implicating the TSH–T3/T4 axis and thyroid hormone metabolism in endometriotic tissue. We conclude by outlining implications for study design, assay standardization, and technology-enabled approaches that may help bridge biomedical mechanisms with health-system realities.

Early clinical research on thyroid dysfunction in endometriosis produced mixed—and often negative—results. In a Brazilian case–control cohort with surgically confirmed endometriosis, the prevalence of thyroid dysfunction and positivity for anti-thyroid peroxidase and anti-thyroglobulin antibodies did not differ significantly between cases and controls, leading the authors to argue against routine thyroid screening in endometriosis (Petta et al., 2007). Later studies, however, reported signals in specific populations or with different endpoints. A case–control study described a markedly higher prevalence of autoimmune thyroiditis among women with endometriosis (Porpora et al., 2020), and an IVF/ICSI cohort observed higher rates of Hashimoto thyroiditis in women with endometriosis than in those without (Korošec et al., 2024). More recently, EHR- and claims-based analyses have suggested elevated risks across a broader range of thyroid outcomes, including Graves' disease and thyroid neoplasms (Aziz et al., 2025; Shih et al., 2026; Yuk et al., 2016). Whether these differences reflect true heterogeneity between populations, shifts in clinical practice or diagnostic intensity, or study-design–specific biases remains unclear—making a structured synthesis particularly useful.

Conceptually, the existing literature is compatible with at least three non–mutually exclusive explanations. First, a shared autoimmunity model, in which endometriosis co-occurs with autoimmune thyroid disease as part of a broader immune phenotype; this interpretation is supported by comorbidity mapping and studies reporting autoimmune thyroiditis or elevated thyroid autoantibodies in endometriosis (Poppe et al., 2002; Porpora et al., 2020; Şerifoğlu et al., 2023). Second, an endocrine microenvironment model, in which thyroid hormones and TSH signaling contribute directly to lesion growth, oxidative stress, and cellular proliferation, consistent with mechanistic work on thyroid hormone metabolism and TSH/T3/T4 signaling in endometriotic tissues (Peyneau et al., 2019). Third, a detection and healthcare-contact model, in which endometriosis leads to more clinical encounters and testing, increasing the likelihood that subclinical thyroid dysfunction or antibody positivity is identified (Shih et al., 2026; Aziz et al., 2025). Distinguishing among these models is not only biologically important but also directly relevant to patient counseling and to the design of future studies.

2. Methodology

This review is a narrative synthesis of original studies linking diagnosed endometriosis with thyroid dysfunction, thyroid autoimmunity, and TSH–T3/T4 axis mechanisms. PubMed was searched using MeSH and free-text terms combining endometriosis concepts ("endometriosis" OR "endometrioma") with thyroid endpoints (e.g., "thyroid," hypothyroidism, hyperthyroidism, Graves, Hashimoto, thyroiditis, thyroid autoimmunity, TPOAb, TGAb, TSI, TSH receptor/TRAb, thyroid neoplasm/cancer). Reference lists of key papers were also screened to capture relevant older endocrine and mechanistic studies. We included original research that defined an endometriosis group (surgical confirmation, clinician diagnosis, or EHR/claims codes) and reported at least one thyroid outcome (diagnosed thyroid disorders, thyroid function tests, thyroid autoantibodies including TRAb, thyroid neoplasms, or mechanistic measurements directly probing the TSH–

T3/T4 axis). Case reports, conference abstracts without full data, and studies lacking endometriosis-specific thyroid outcomes were excluded. Titles/abstracts were screened followed by full-text review when available. For each study, we extracted design, data source, population characteristics, definitions of endometriosis and thyroid outcomes, sample size, analytic approach (e.g., matching/regression adjustment), and the main effect estimates (HR, OR, SIR, AUC). Given substantial heterogeneity in populations, outcome definitions, and assays, we did not perform meta-analysis; findings were synthesized narratively across epidemiological database studies, clinical biomarker studies, TRAb assay-focused studies, and mechanistic investigations, while qualitatively considering bias, confounding control, and assay comparability.

3. Results

Eighteen original studies were identified that examined both diagnosed endometriosis and thyroid-related outcomes. The evidence base was heterogeneous, including large EHR and claims analyses, clinical cohorts with laboratory measurements, studies focused on TSH receptor antibodies, mechanistic investigations of thyroid hormone signaling in endometriosis models, and population studies assessing thyroid neoplasms among women with endometriosis. Given substantial variation in study populations, ascertainment approaches, and outcome definitions, results are presented by evidence domain and summarized in Tables 1–5.

3.1. Epidemiological evidence for thyroid disorders after endometriosis diagnosis

Large observational studies based on EHR and claims data generally point to a link between endometriosis and later thyroid disease, but the size of the association—and even which thyroid diagnoses show the clearest signal—depends on how cases and outcomes are defined. In a 20-year retrospective cohort drawn from the TriNetX global EHR network, Shih et al. (2026) compared women with diagnosed endometriosis with a matched control group and found consistently higher risks across multiple thyroid outcomes during follow-up. After 1:1 matching (118,360 patients in total), endometriosis was associated with increased hazards of hypothyroidism (HR 1.19; 95% CI 1.13–1.25), hyperthyroidism (HR 1.21; 95% CI 1.09–1.35), Graves' disease (HR 1.27; 95% CI 1.04–1.56), non-toxic goiter (HR 1.31; 95% CI 1.23–1.39), and thyroiditis (HR 1.32; 95% CI 1.18–1.48). Importantly, the same analysis also reported higher hazards of both malignant (HR 1.55; 95% CI 1.21–1.97) and benign (HR 2.47; 95% CI 1.73–3.52) thyroid neoplasms, expanding the thyroid phenotype beyond functional and autoimmune disorders (Shih et al., 2026).

Claims-based datasets provide a useful counterpoint and sometimes a different pattern of results. Using Korean HIRA-NIS data from 2009–2011, Yuk et al. (2016) conducted a population-based cross-sectional study comparing 5,615 women with endometriosis and 22,460 controls. After adjustment, Graves' disease was more frequent among women with endometriosis (OR 2.52; 95% CI 1.30–4.88), whereas hypothyroidism (OR 1.17; 95% CI 0.90–1.52) and autoimmune hypothyroidism (OR 1.61; 95% CI 0.88–2.94) were not significantly associated (Yuk et al., 2016). Taken together, these results highlight how differences in outcome granularity (broad "thyroid dysfunction" versus specific autoimmune diagnoses) and health-system context can shape which thyroid phenotypes emerge as statistically robust.

A more recent U.S. claims-based case–control analysis broadened the thyroid focus to include both Hashimoto thyroiditis and Graves' disease, using a diagnosis-centered time window around the index date. Aziz et al. (2025) evaluated endometriosis alongside 10 autoimmune diseases in two administrative claims databases (MarketScan CCAE and MDCD), applying a two-year diagnosis window centered on the index date and examining both one-year and three-year observation periods. In CCAE, endometriosis was associated with higher odds of Hashimoto's disease (OR 2.49; 95% CI 2.32–2.68 for the one-year observation; OR 2.28; 95% CI 2.18–2.39 for the three-year observation) and Graves' disease (OR 2.58; 95% CI 2.15–3.09 for the one-year observation; OR 2.71; 95% CI 2.38–3.08 for the three-year observation) (Aziz et al., 2025). In MDCD, Hashimoto's disease remained associated (OR 3.15; 95% CI 2.70–3.69 for the one-year observation; OR 2.77; 95% CI 2.45–3.14 for the three-year observation), while Graves' disease estimates were less precise in the one-year window (OR 2.35; 95% CI 0.75–7.37) but higher in the three-year observation (OR 6.77; 95% CI 1.98–23.12) (Aziz et al., 2025). Overall, these database studies suggest that both autoimmune thyroid disease and broader thyroid dysfunction tend to cluster with endometriosis across different settings, although the most prominent thyroid phenotype and the estimated effect size vary with population characteristics and analytic choices.

Table 1. Large-scale EHR and claims studies evaluating thyroid disorders in relation to endometriosis

Study (country; data source)	Design and population	Endometriosis definition	Thyroid outcomes	Key results (adjusted)
Shih et al., 2026 (TriNetX EHR network)	Retrospective cohort; 20-year follow-up; 1:1 matched cohort (N=118,360)	Diagnosed endometriosis in EHR (coded)	Hypothyroidism; hyperthyroidism; Graves' disease; non-toxic goiter; thyroiditis; thyroid neoplasms	HRs: hypothyroidism 1.19 (95% CI 1.13-1.25); hyperthyroidism 1.21 (1.09-1.35); Graves 1.27 (1.04-1.56); goiter 1.31 (1.23-1.39); thyroiditis 1.32 (1.18-1.48); malignant thyroid neoplasm 1.55 (1.21-1.97); benign thyroid neoplasm 2.47 (1.73-3.52)
Yuk et al., 2016 (Korea; HIRA-NIS 2009-2011)	Population-based cross-sectional; n=5,615 endometriosis; n=22,460 controls	Endometriosis claims codes	Graves' disease; hypothyroidism; autoimmune hypothyroidism	Adjusted ORs: Graves 2.52 (95% CI 1.30-4.88); hypothyroidism 1.17 (0.90-1.52, NS); autoimmune hypothyroidism 1.61 (0.88-2.94, NS)
Aziz et al., 2025 (USA; MarketScan CCAE and MDCD claims)	Claims-based case-control; two-year diagnosis window centered at index date; matched; evaluated 1- and 3-year observation periods	Endometriosis claims codes with temporal window	Hashimoto's disease; Graves' disease	CCAE: Hashimoto OR 2.49 (2.32-2.68) [1-year], 2.28 (2.18-2.39) [3-year]; Graves OR 2.58 (2.15-3.09) [1-year], 2.71 (2.38-3.08) [3-year]. MDCD: Hashimoto OR 3.15 (2.70-3.69) [1-year], 2.77 (2.45-3.14) [3-year]; Graves OR 2.35 (0.75-7.37) [1-year], 6.77 (1.98-23.12) [3-year]

3.2. Clinical evidence for thyroid autoimmunity and thyroid function alterations in endometriosis

Clinical studies have looked at thyroid history, thyroid function tests, and thyroid autoantibodies in women with endometriosis—most often in surgically confirmed cohorts or infertility/ART settings. Overall, the picture is mixed, and differences across studies likely reflect who was enrolled (general gynecology vs infertility clinics), how endometriosis was confirmed, and how thyroid endpoints (and antibody cutoffs) were measured.

A frequently cited “negative” study comes from Petta et al. (2007), who compared 148 women with surgically confirmed endometriosis with 158 controls and found no meaningful differences in thyroid disorders (20.9% vs 26.5%), thyroid dysfunction (12.2% vs 10.8%), or positivity for any thyroid antibody (14.9% vs 22.2%); no associations were seen with disease stage or infertility in stratified analyses (Petta et al., 2007). In infertility-focused cohorts, signals of thyroid autoimmunity appear more readily. In a prospective study of 438 women from infertile couples and 100 fertile controls, Poppe et al. (2002) reported a slightly higher median TSH in infertile women (1.3 vs 1.1 mIU/L) and noted that, among female-factor causes, the endometriosis subgroup had the highest TPOAb prevalence (29%) (Poppe et al., 2002). Similarly, Porpora et al. (2020) observed a strong autoimmune thyroiditis signal in a university-based case–control cohort (148 endometriosis vs 150 controls), with autoimmune thyroiditis reported in 80 women with endometriosis versus 14 controls (Porpora et al., 2020), although temporality cannot be established and referral patterns may influence the magnitude of this difference.

More detailed phenotyping within surgical cohorts suggests that thyroid parameters may relate to endometriosis subtype or lesion burden rather than simply separating cases from controls. In a prospective study of 51 surgical endometriosis cases and 51 controls, Şerifoğlu et al. (2023) found lower mean free T4 in endometriosis (0.97 ± 0.13 vs 1.09 ± 0.21 ng/dL) and, within the endometriosis group, higher anti-TPO values

in bilateral versus unilateral endometrioma along with a positive correlation between anti-TPO and endometrioma diameter (Şerifoğlu et al., 2023). In an IVF/ICSI cohort (45 endometriosis vs 181 without), Hashimoto thyroiditis was more common in women with endometriosis (31.1% vs 14.9%), and within the endometriosis group Hashimoto thyroiditis clustered in those with TSH ≥ 2.5 mIU/L (60% vs 16.6%), while oocyte/embryo outcomes were reported as comparable between groups (Korošec et al., 2024). Beyond association studies, Wang et al. (2025) tested whether thyroid antibodies add diagnostic value: in 885 surgical patients (441 ovarian endometriosis vs 444 benign ovarian tumors), combining CA125 with TPOAb and TGAb improved discrimination of ovarian endometriomas (AUC 0.924; sensitivity 88.9%, specificity 84.5%) (Wang et al., 2025). Finally, Birke et al. (2021) explored a symptom-plus-thyroid approach (107 endometriosis vs 60 controls): premenstrual spotting showed the strongest association (OR 3.82), "thyroid dysfunction" alone was not significant, and the combined hypothesis (thyroid dysfunction plus spotting) was associated with endometriosis (OR 2.75) but with low sensitivity (20%) despite high specificity (91.7%) (Birke et al., 2021).

Table 2. Clinical studies assessing thyroid function tests and thyroid autoimmunity markers in women with endometriosis

Study	Design and participants	Thyroid markers/outcomes	Key findings
Petta et al., 2007	Cross-sectional case-control; surgically confirmed endometriosis (n=148) vs controls (n=158)	Thyroid disorders/dysfunction; TPOAb/TGAb	No significant differences: thyroid disorders 20.9% vs 26.5% (p=0.25); thyroid dysfunction 12.2% vs 10.8% (p=0.85); any thyroid antibody 14.9% vs 22.2% (p=0.20)
Poppe et al., 2002	Prospective infertility cohort; 438 infertile women vs 100 age-matched fertile controls	TSH; autoimmune thyroid disease (TPOAb)	Median TSH higher in infertile women (1.3 vs 1.1 mIU/L). TPOAb positivity: 14% in infertile women vs 8% controls (NS). Female infertility subgroup: 18% vs 8%; highest in endometriosis subgroup (29%)
Porpora et al., 2020	Case-control; women with endometriosis (n=148) vs controls (n=150)	Autoimmune diseases; autoimmune thyroiditis highlighted	Autoimmune thyroiditis markedly more prevalent in endometriosis group (80 vs 14; p<0.0001)
Şerifoğlu et al., 2023	Prospective observational; surgery for benign gynecologic disease: endometriosis (n=51) vs non-endometriosis (n=51)	TSH; fT4; TSI; anti-TPO	fT4 lower in endometriosis (0.97±0.13 vs 1.09±0.21 ng/dL; p=0.002). TSH and anti-TPO not significantly different. Anti-TPO higher with bilateral endometrioma (p=0.028) and correlated with endometrioma diameter (p=0.011)
Korošec et al., 2024	Retrospective IVF/ICSI cohort; 226 women (endometriosis n=45; non-endometriosis n=181)	TSH (cut-off 2.5 mIU/L); Hashimoto thyroiditis; IVF outcomes	Hashimoto more frequent with endometriosis (31.1% vs 14.9%; p=0.012). TSH≥2.5: 33.3% vs 26.6% (p=0.335). Within endometriosis: Hashimoto 60% with TSH≥2.5 vs 16.6% with TSH<2.5 (p=0.001). No major differences in oocyte/embryo characteristics by IVF vs ICSI
Wang et al., 2025	Retrospective ovarian surgery cohort; ovarian endometriosis (n=441) vs benign ovarian tumors (n=444)	CA125; TGAb; TPOAb; diagnostic performance	AUC: CA125 0.895; TGAb 0.793; TPOAb 0.736. Combined CA125+TGAb+TPOAb AUC 0.924 (95% CI 0.906-0.942), sensitivity 88.9%, specificity 84.5%
Birke et al., 2021	Retrospective case-control; endometriosis (n=107) vs controls (n=60)	TSH/thyroid dysfunction (authors' definition); premenstrual spotting; diagnostic hypotheses	Premenstrual spotting associated with endometriosis (OR 3.82; 95% CI 1.52-9.58). Thyroid dysfunction alone not significant (OR 2.4; 0.93-6.30). Combination (thyroid dysfunction + spotting) associated (OR 2.75; 1.09-6.90) with low sensitivity (20%) but high specificity (91.7%)

3.3. TSH receptor antibodies (TRAb) and assay-dependent biomarker signals

A growing, but still unsettled, body of research has focused on antibodies against the TSH receptor (TRAb) in endometriosis, motivated by evidence of TSH receptor expression in ectopic tissue and the idea that TRAb could represent an endometriosis-associated autoantibody (Şerifoğlu et al., 2023; Petersson et al., 2025). Across studies, however, the signal appears highly dependent on the assay and the cut-off used. In a case-cohort analysis, Ek et al. (2018) measured TRAb IgG and anti-TPO in 172 women with endometriosis and population comparators; no association was seen for hypothyroidism or anti-TPO, but TRAb showed an extreme gradient, with the highest versus lowest tertile yielding an OR of 539.26 (95% CI 114.29–2544.32; p for trend < 0.001) (Ek et al., 2018). Svensson et al. (2022) also reported higher TRAb titers in 172 surgically confirmed cases compared with two control groups: 29.1% vs 2.6% were TRAb IgG-positive at ≥1.0 IE/L, and 94.5% vs 7.9% at ≥0.3 IE/L; importantly, TRAb did not correlate with thyroid hormones, TSH, or gastrointestinal symptoms, suggesting the finding is not simply driven by overt thyroid dysfunction (Svensson et al., 2022). In contrast, Petersson et al. (2025) tested TRAb in 121 endometriosis patients, 50 irritable bowel syndrome patients, and 50 healthy controls using routine Graves' disease assays (BRAHMS TRAK and Roche Elecsys Anti-TSHR) and found no significant differences in detectable TRAb, concluding that current clinical assays do not support TRAb as a usable diagnostic biomarker for endometriosis (Petersson et al., 2025). Overall, TRAb may be detectable in some cohorts, but assay dependence and non-standardized thresholds currently limit clinical interpretation and utility.

Table 3. Studies evaluating TSH receptor antibodies (TRAb) in endometriosis and implications of assay thresholds

Study	Participants	Assay approach	Key results	Interpretation
Ek et al., 2018	Endometriosis (n=172) vs general population controls	Serum TRAb IgG and anti-TPO; TRAb analyzed in tertiles	Elevated TRAb titers associated with endometriosis: OR 539.26 (95% CI 114.29-2544.32) for highest vs lowest tertile; p for trend <0.001. Hypothyroidism and anti-TPO antibodies not associated	Suggests strong TRAb signal in this cohort but effect size likely sensitive to distribution and cut-points; requires replication and standardized assays
Svensson et al., 2022	Surgically confirmed endometriosis (n=172); controls: 50 healthy blood donors and 114 general population (MOS)	Measured thyroid hormones, TSH, TRAb IgG/IgM; evaluated cross-reactivity with other autoantibodies; compared detection limits	TRAb IgG ≥1.0 IE/L in 29.1% of endometriosis vs 2.6% controls (p<0.001). Using previous limit ≥0.3 IE/L: 94.5% vs 7.9% (p<0.001). TRAb IgG and IgM titers increased; no cross-reactivity; no correlation with thyroid hormones/TSH	Supports TRAb elevation but shows strong dependence on detection threshold; suggests TRAb may be near lower assay range in endometriosis
Petersson et al., 2025	Endometriosis (n=121); IBS (n=50); healthy controls (n=50)	TRAb IgG measured using two clinical assays (BRAHMS TRAK Human; Roche Elecsys Anti-TSHR) in two laboratories	No significant difference in detectable TRAb between endometriosis and controls or IBS in either lab (e.g., Gothenburg: 8.3% vs 4%; p=0.512)	Routine clinical Graves-oriented assays may not detect endometriosis-associated TRAb signals; TRAb not yet clinically actionable as biomarker

3.4. Mechanistic evidence implicating the TSH-T3/T4 axis in endometriosis pathophysiology

Mechanistic work is starting to bridge what is seen in epidemiological datasets and serology with concrete biological pathways, showing how thyroid hormones and TSH signaling could shape the behavior of endometriotic lesions. Unlike association studies, these experiments directly test what components of the thyroid axis do to endometrial or endometriotic tissues.

A key example is the study by Peyneau et al. (2019), which combined analyses of human eutopic and ectopic endometrial tissue with cell-based assays and mouse models. They reported altered expression of transcripts and proteins involved in thyroid hormone metabolism in both eutopic and ectopic endometrium, and their experimental data showed that T3 and T4 can increase proliferation and reactive oxygen species production in ectopic endometrial cells. They also described TSH as a proliferative, pro-oxidative signal in endometrial tissue, and their mouse experiments supported a relationship between thyroid hormone levels and the growth of endometriotic implants (Peyneau et al., 2019). Together, these results make the broad "thyroid phenotype" observed in database studies biologically plausible and suggest that local handling of thyroid hormones within lesions could contribute to disease progression rather than merely reflecting systemic thyroid status.

An additional practical point is that endocrine treatments used in endometriosis can complicate interpretation of thyroid function tests, especially in cross-sectional comparisons. In an early study of 18 women treated with danazol, Thorell et al. (1979) found that thyroid hormone–binding globulin fell to about one third of baseline after four months of therapy and returned to normal after treatment was stopped. During treatment, total T3 and T4 decreased slightly, free T4 and free T3 indices increased, and serum TSH fell from 2.4 ± 0.6 to 1.7 ± 0.3 mIU/L—changes that remained within reference ranges and were attributed to reduced binding globulin rather than true thyroid disease (Thorell et al., 1979). This highlights an important confounder for the field: hormonal suppression regimens can shift binding proteins and laboratory profiles without indicating intrinsic thyroid gland dysfunction. Table 4 summarizes the mechanistic and therapy-related evidence relevant to interpreting the TSH–T3/T4 axis in endometriosis research.

Table 4. Mechanistic and therapy-related evidence linking the TSH-T3/T4 axis to endometriosis pathophysiology

Study	Models	Thyroid-axis components	Key findings relevant to endometriosis
Peyneau et al., 2019	Human eutopic and ectopic endometrium; cell-based experiments; mouse models	TSH/TSHR signaling; T3/T4; thyroid hormone metabolism pathways; oxidative stress	Reported dysregulation of thyroid hormone metabolism-related transcripts/proteins in endometrial tissues. Experimental data indicated proliferative and pro-oxidative effects of TSH and of thyroid hormones (T3/T4) on endometrial/endometriotic cells and supported a relationship between thyroid hormone levels and implant growth in vivo
Thorell et al., 1979	Human longitudinal endocrine study during danazol therapy (n=18)	TBG, total T4, free thyroxine index, TSH, T3 uptake	Danazol reduced thyroid hormone binding globulin to approximately one third of baseline after 4 months. Total T3 and T4 decreased slightly, free T4 and free T3 indices increased, and TSH decreased from 2.4 ± 0.6 to 1.7 ± 0.3 mIU/L; all changes remained within normal ranges. Highlights potential confounding of thyroid laboratory interpretation during hormonal therapy

3.5. Thyroid neoplasms and cancer risk in women with endometriosis

Several studies have extended thyroid outcomes beyond functional and autoimmune disorders to include thyroid neoplasms. Although thyroid cancer is relatively uncommon, consistent signals across cohorts could suggest shared hormonal or inflammatory pathways, or could reflect healthcare-contact and surveillance effects.

Within the TriNetX EHR-based cohort, Shih et al. (2026) reported increased hazards of both malignant (HR 1.55; 95% CI 1.21-1.97) and benign (HR 2.47; 95% CI 1.73-3.52) thyroid neoplasms in women with endometriosis compared to matched controls, indicating that the thyroid phenotype may include proliferative outcomes (Shih et al., 2026).

Population registry and claims studies have reported similar directional associations for thyroid cancer. In a Swedish cohort study evaluating cancer risk and parity, Melin et al. (2007) reported a thyroid cancer SIR of 1.33 (95% CI 1.04-1.69) among women with endometriosis. In Korea, Eoh et al. (2021) reported that endometriosis was associated with higher risk of thyroid cancer (adjusted HR 1.62; 95% CI 1.14-2.30). While these studies differ in population and analytic approach, the convergence of effect estimates supports inclusion of thyroid neoplasms in the broader thyroid phenotype associated with endometriosis.

Table 5 summarizes thyroid neoplasm findings across cohorts and highlights the need to interpret cancer associations in light of potential diagnostic intensity and differential screening for thyroid nodules in women with chronic gynecologic disease.

Table 5. Thyroid neoplasms in women with endometriosis

Study	Design and population	Outcome	Effect estimate
Shih et al., 2026	TriNetX EHR retrospective cohort; matched	Thyroid neoplasms	Malignant thyroid neoplasm HR 1.55 (95% CI 1.21-1.97); benign thyroid neoplasm HR 2.47 (95% CI 1.73-3.52)
Melin et al., 2007	Swedish registry cohort; cancer risk among women with endometriosis	Thyroid cancer incidence	SIR 1.33 (95% CI 1.04-1.69)
Eoh et al., 2021	Korean population cohort; malignancy risk in endometriosis	Thyroid cancer incidence	Adjusted HR 1.62 (95% CI 1.14-2.30)

Discussion

This review brings together three lines of evidence—large health-system datasets (EHR/claims), targeted clinical cohorts, and mechanistic studies—to answer a straightforward question: is thyroid dysfunction, including autoimmune thyroid disease, genuinely linked to endometriosis, and if so, what might explain that link? Overall, the association appears more consistently in large EHR and claims analyses than in the earlier, smaller case–control studies, while mechanistic research strengthens biological plausibility by showing ways the thyroid axis could directly influence lesion biology. At the same time, the findings span a wide range of thyroid phenotypes—hypo- versus hyperthyroidism, Hashimoto versus Graves, different antibody patterns, and even neoplastic outcomes—making it unlikely that a single pathway, or a single screening marker, can fully capture the endometriosis–thyroid relationship.

4.1. Reconciling database-scale associations with mixed clinical cohort findings

The most consistent evidence for an endometriosis–thyroid link comes from database-scale studies. In the TriNetX network, Shih et al. (2026) reported modest but statistically robust increases in risk across several thyroid outcomes—including both hypo- and hyperthyroidism, thyroiditis, and thyroid neoplasms. Claims-based analyses add complementary signals, showing higher odds of Graves' disease (Yuk et al., 2016) and increased odds of both Hashimoto and Graves' disease when using a diagnosis-centered time window (Aziz et al., 2025). These studies benefit from very large samples and the ability to evaluate multiple endpoints within harmonized analytic frameworks, but they also inherit typical limitations of coded data: potential misclassification, limited information on endometriosis phenotype and severity, and residual confounding.

By comparison, smaller clinical cohorts with surgically confirmed endometriosis and direct measurement of thyroid hormones and antibodies have produced mixed results. Petta et al. (2007) found no

significant differences in thyroid dysfunction or antibody positivity, whereas Porpora et al. (2020) reported a striking enrichment of autoimmune thyroiditis, and Şerifoğlu et al. (2023) observed lower free T4 and within-case associations between anti-TPO and endometrioma features. Differences in recruitment setting, case mix (pain- vs infertility-dominant phenotypes), regional context, assay platforms, and statistical power likely contribute to these discrepancies; importantly, small cohorts may simply be underpowered to detect the modest effect sizes suggested by population studies.

The infertility/IVF context may represent a particularly relevant intersection. Poppe et al. (2002) reported that, among women with female-factor infertility, the endometriosis subgroup had the highest TPOAb prevalence, and in IVF/ICSI patients Korošec et al. (2024) found higher Hashimoto thyroiditis prevalence in women with endometriosis and a strong within-group association between TSH $\geq$ 2.5 mIU/L and Hashimoto diagnosis. This pattern could reflect shared autoimmune susceptibility, selection into fertility care, or more intensive endocrine evaluation in infertility clinics, but it suggests that thyroid autoimmunity may be especially clinically relevant when endometriosis is part of an infertility workup.

4.2. Thyroid autoimmunity as a shared immune phenotype

Several findings across the included studies are consistent with the idea that at least a subset of patients with endometriosis may share an "autoimmune-leaning" phenotype. In a university-based case–control cohort, Porpora et al. (2020) reported that autoimmune thyroiditis was far more common in women with endometriosis than in controls, and in a large U.S. claims analysis Aziz et al. (2025) observed higher odds of Hashimoto disease in women with endometriosis across two databases and multiple observation windows. Not all clinical cohorts show a simple case–control separation, however. Şerifoğlu et al. (2023) did not detect a between-group difference in anti-TPO levels, but anti-TPO values were higher in bilateral versus unilateral endometrioma and correlated with endometrioma diameter, suggesting that thyroid autoimmunity markers may track lesion burden in specific subgroups rather than distinguishing all cases from controls. Poppe et al. (2002) reported a similar pattern in infertility care, where TPOAb positivity was most frequent in the endometriosis subgroup among female-factor infertility diagnoses. Taken together, the evidence fits a model in which thyroid autoimmunity co-occurs with endometriosis in a subset of patients—potentially those with an endometrioma-predominant phenotype or an infertility-driven presentation.

Two caveats are important when interpreting this literature. First, thyroid autoantibodies are relatively common in the general population and are shaped by factors such as age, iodine intake, parity, and coexisting autoimmune disease—variables that are not uniformly captured or controlled across studies. Second, antibody positivity is not synonymous with clinical thyroid disease: many individuals with anti-TPO or anti-TG antibodies remain euthyroid, and claims-based coding of Hashimoto thyroiditis can depend on whether antibody testing is performed and whether treatment is initiated. As a result, thyroid autoimmunity in this context may reflect both biology and health-system processes (testing and coding), and separating these influences will require prospective studies with standardized antibody assays and adjudicated thyroid outcomes.

4.3. The TSH-T3/T4 axis and local thyroid hormone handling within lesions

Mechanistic studies offer a pathway-level explanation for an endometriosis–thyroid link that goes beyond "shared autoimmunity." In particular, Peyneau et al. (2019) reported altered expression of transcripts and proteins involved in thyroid hormone metabolism in both eutopic and ectopic endometrial tissue, and showed experimentally that TSH, T3, and T4 can influence cell proliferation and oxidative stress in endometrial contexts. Taken together, these findings support the idea that thyroid signaling may be reshaped locally within lesions—potentially helping lesions persist or grow—even when systemic thyroid status appears relatively normal on routine blood tests. This perspective also fits clinical observations in which only subtle endocrine differences are seen, such as lower free T4 reported by Şerifoğlu et al. (2023), without clear between-group differences in clinically coded thyroid dysfunction.

If thyroid hormone handling is altered at the tissue level, several testable hypotheses follow. First, circulating thyroid hormone levels may not map cleanly onto endometriosis severity, because lesion-specific metabolism could weaken (or distort) the relationship between blood measurements and local biological effects. Second, endocrine exposures—including levothyroxine therapy and hormonal suppression regimens—might influence lesion biology in ways that are not captured by standard diagnostic categories like "hypothyroidism" or "hyperthyroidism." Third, thyroid-related biomarkers may not perform uniformly across disease subtypes (e.g., ovarian endometrioma versus deep infiltrating disease) or clinical settings (fertility versus pain clinics). Addressing these questions will likely require integrated studies that combine careful clinical phenotyping (imaging and surgical staging), standardized endocrine testing, and tissue-level molecular assays.

4.4. TRAb as a biomarker: promise, contradictions, and the central role of assay technology

TRAb studies highlight both the appeal—and the current fragility—of thyroid-related biomarkers in endometriosis. Ek et al. (2018) reported an exceptionally strong association between TRAb IgG tertiles and endometriosis, and Svensson et al. (2022) found that 29.1% of surgically confirmed cases exceeded a contemporary TRAb IgG cut-off (≥1.0 IE/L), rising to 94.5% when a lower historical threshold (≥0.3 IE/L) was applied. If reproducible, this would be an unusually high-contrast serologic signal for a disease that still lacks reliable non-invasive diagnostics.

However, Petersson et al. (2025) did not observe higher TRAb positivity when using two routine clinical Graves' disease assays, even with both healthy controls and an irritable bowel syndrome comparison group. This mismatch underscores that "TRAb" is not a single, standardized measurement: assays differ in epitope targets, calibration, and clinical decision thresholds, and the endometriosis signal may sit near the lower limit of detection—where small methodological differences matter. From a translational standpoint, TRAb research now needs assay harmonization, transparent reporting of full titer distributions and analytical performance, and ideally orthogonal validation, followed by prospective studies testing whether TRAb tracks disease activity, precedes diagnosis, or changes with treatment (Ek et al., 2018; Svensson et al., 2022; Petersson et al., 2025).

4.5. Thyroid function tests, clinical screening, and the risk of over-interpretation

A practical question is whether an endometriosis diagnosis should trigger routine thyroid screening. The earliest directly relevant surgical case–control study reported similar rates of thyroid dysfunction and thyroid antibody positivity in women with endometriosis and controls, and therefore did not support systematic screening on that basis alone (Petta et al., 2007). More recent population-scale EHR and claims analyses have suggested higher risks of several thyroid outcomes after endometriosis diagnosis (Shih et al., 2026; Aziz et al., 2025), and IVF/ICSI cohorts report a higher prevalence of Hashimoto thyroiditis in women with endometriosis (Korošec et al., 2024). Taken together, the current evidence favors heightened clinical awareness rather than universal testing, since absolute risk in unselected endometriosis populations remains unclear and some associations may be influenced by increased healthcare contact and testing. Targeted assessment may be most reasonable in settings where thyroid evaluation is already clinically relevant—particularly infertility and assisted reproduction care (Poppe et al., 2002; Korošec et al., 2024)—and, in ovarian endometriomas, thyroid autoantibodies may add diagnostic information when combined with CA125 (Wang et al., 2025). By contrast, using TSH thresholds or "thyroid dysfunction" alone as a diagnostic screen for endometriosis performs poorly in sensitivity (Birke et al., 2021). Finally, thyroid test interpretation should account for treatment-related confounding: danazol markedly reduced thyroid hormone–binding globulin and shifted thyroid indices (with TSH still within the reference range), illustrating how endocrine therapy can alter laboratory profiles without reflecting intrinsic thyroid disease (Thorell et al., 1979).

4.6. Thyroid neoplasms: biological signal or healthcare-contact artifact?

The association between endometriosis and thyroid neoplasms has been observed in several cohorts, including increased hazards of thyroid neoplasms in TriNetX (Shih et al., 2026) and increased thyroid cancer risk in registry and claims studies (Melin et al., 2007; Eoh et al., 2021). These findings may reflect shared hormonal and inflammatory pathways, but they are also vulnerable to surveillance bias: women with chronic gynecologic disease may undergo more frequent imaging and clinical evaluations, increasing incidental detection of thyroid nodules and early cancers. Future work should attempt to control for healthcare utilization and screening intensity, and where possible distinguish between microcarcinomas and clinically significant thyroid cancers. Mechanistic studies of thyroid hormone signaling in endometriotic lesions (Peyneau et al., 2019) provide a potential biological framework, but direct causal links between endometriosis biology and thyroid tumorigenesis are not yet established by the available evidence.

4.7. Technology, society, and interdisciplinary implications

Although most of the endometriosis–thyroid literature is biomedical, it intersects in clear ways with technology and society—well within the IJITSS remit. Much of the most consistent evidence comes from technology-enabled infrastructures such as EHR networks and administrative claims, where findings depend heavily on how diagnoses and laboratory tests are coded and algorithmically defined (Aziz et al., 2025; Shih et al., 2026; Yuk et al., 2016). Several studies also draw attention to symptom overlap and diagnostic uncertainty with real social consequences—for example, gastrointestinal complaints may lead to labeling as irritable bowel syndrome, while mental comorbidity and socioeconomic factors can shape both the lived

experience of disease and access to specialist care (Ek et al., 2018; Petersson et al., 2025). Finally, biomarker work (e.g., TRAb and CA125 combined with thyroid autoantibodies) illustrates how advances in laboratory methods and modeling might reduce reliance on invasive diagnostic pathways, but also why assay standardization and external validation are essential before equitable clinical adoption (Svensson et al., 2022; Petersson et al., 2025; Wang et al., 2025).

From a health-system perspective, adding thyroid assessment to endometriosis care could be helpful in selected contexts—such as infertility care—or when differentiating ovarian masses, but it also carries risks. Over-testing and over-interpretation of clinically insignificant antibody positivity can increase patient anxiety and consume resources without proven benefit. These trade-offs argue for outcomes-focused research that links thyroid testing strategies to patient-relevant endpoints (e.g., pain, fertility outcomes, quality of life), rather than relying primarily on laboratory abnormalities.

4.8. Priorities for future research

Several clear priorities emerge from the evidence reviewed here. First, future studies would benefit from more consistent definitions and deeper phenotyping: endometriosis subtype and severity (ideally supported by imaging or surgical staging) should be reported alongside standardized thyroid outcome definitions, so results can be compared across cohorts. Second, temporality remains a key gap. Prospective, longitudinal cohorts with thyroid measurements collected before an endometriosis diagnosis would help clarify whether thyroid autoimmunity tends to precede endometriosis, follows it, or develops in parallel. Third, antibody measurement—especially TRAb—needs harmonization. The contrast between research-grade signals and routine clinical assays makes it essential to report assay platforms, limits of detection, and to replicate findings across independent settings (Svensson et al., 2022; Petersson et al., 2025). Fourth, mechanistic hypotheses should be pushed closer to the clinic: proposed roles for local thyroid hormone metabolism and TSH/T3/T4 signaling need to be tested in human tissues across endometriosis phenotypes and linked to clinical endpoints, not only molecular readouts (Peyneau et al., 2019). Finally, technology-enabled integration is promising but must be done carefully. Combining EHR-derived phenotypes with laboratory data and biomarkers (for example, CA125 plus thyroid autoantibodies) may improve diagnostic or prognostic models, but external validation across diverse populations is essential to avoid bias and ensure generalizability (Wang et al., 2025; Shih et al., 2026).

This review also has limitations. It focused on PubMed-indexed original studies that explicitly reported both endometriosis and thyroid outcomes; broader immunology or endocrinology literature that did not measure both endpoints was not systematically searched. In addition, the heterogeneity of study designs, populations, and outcome definitions precluded quantitative meta-analysis. Even so, the overall pattern—signals observed across multiple independent cohorts and supported by complementary approaches—suggests that thyroid outcomes deserve continued attention in endometriosis research and, in selected clinical contexts, in patient care.

Conclusions

Endometriosis and thyroid disorders appear to overlap at several levels, from population patterns to clinical endocrinology and emerging mechanistic biology. Evidence from large EHR and claims datasets generally shows higher rates of multiple thyroid outcomes following an endometriosis diagnosis, including hypothyroidism, hyperthyroidism, Graves' disease, thyroiditis, and thyroid neoplasms. In contrast, findings from smaller clinical cohorts are less consistent—ranging from no clear differences in thyroid dysfunction or antibody positivity in some surgically confirmed case–control studies to stronger signals of autoimmune thyroiditis and higher Hashimoto thyroiditis prevalence in infertility and IVF-related populations.

Importantly, the link is not purely epidemiological. Mechanistic studies suggest that thyroid-axis signaling may be relevant at the lesion level, with thyroid hormones and TSH influencing pathways related to proliferation and oxidative stress in endometriotic tissues. At the same time, the TRAb literature illustrates both potential and uncertainty: elevated TRAb titers have been reported in some cohorts, but these findings have not been consistently reproduced using routine clinical TRAb assays, highlighting strong assay dependence and the need for methodological standardization.

Overall, the current evidence supports increased clinical awareness and targeted thyroid evaluation in specific contexts—particularly infertility care and the assessment of ovarian masses—rather than universal thyroid screening for all individuals with endometriosis. The next steps for the field include prospective study designs that clarify temporality, standardized thyroid phenotyping and antibody testing, and integrative, technology-enabled approaches that link large-scale observational signals with tissue-level mechanisms and patient-relevant outcomes.

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