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Evaluating The Relationship Between Thyroid Dysfunction And Cardiovascular Diseases: A Systematic Review

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Abstract

Background: Thyroid hormones play a critical role in regulating cardiovascular physiology. Dysregulation, whether overt or subclinical, has been linked to arrhythmias, heart failure, atherosclerosis, and metabolic disturbances. Understanding these associations is crucial for early risk stratification and clinical intervention.
Objective: To systematically review observational and Mendelian randomization studies examining the relationship between thyroid dysfunction and cardiovascular diseases (CVDs).
Methods: Following PRISMA 2020 guidelines, electronic databases including PubMed, Scopus, Web of Science, Embase, and Google Scholar were searched for studies published between 2016 and 2025. Studies assessing serum thyroid hormones (TSH, FT3, FT4), subclinical or overt thyroid disease, or genetically predicted thyroid function and cardiovascular outcomes were included. Both cross-sectional, cohort, and Mendelian randomization designs were considered. Data extraction focused on sample characteristics, thyroid measures, cardiovascular outcomes, and effect estimates. Quality was assessed using the Newcastle–Ottawa Scale and MR-specific criteria.
Results: Ten studies were included, encompassing >1.5 million participants across global cohorts. Low thyroid function correlated with adverse cardiovascular health metrics and metabolic risk factors (Fang et al., 2024; Bai et al., 2025; Neves et al., 2022). Hyperthyroid tendencies, indicated by low TSH or high FT4, were causally linked to atrial fibrillation (Larsson et al., 2019; Ellervik et al., 2019). MR analyses showed protective effects of mild hypothyroidism against stroke (Marouli et al., 2020) but no consistent relationship with ischemic heart disease (Zhao & Schooling, 2017). Hormone replacement therapy required careful titration to avoid over- or under-replacement, impacting cardiovascular outcomes (Evron et al., 2022; Stamatouli et al., 2020).
Conclusions: Thyroid dysfunction, including subclinical states, has significant implications for cardiovascular health. Both metabolic and arrhythmic pathways mediate this risk, emphasizing the need for early detection, individualized monitoring, and multidisciplinary management.
Keywords: thyroid dysfunction, subclinical hypothyroidism, hyperthyroidism, cardiovascular disease, atrial fibrillation, Mendelian randomization, cardiovascular risk

Introduction

The intricate relationship between thyroid function and cardiovascular health has long been recognized as a cornerstone of endocrine–cardiovascular interplay. Thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), are essential modulators of basal metabolism, cardiac contractility, and vascular tone. Dysregulation of these hormones—manifesting as either hypothyroidism or hyperthyroidism—can substantially alter hemodynamic balance, lipid metabolism, and myocardial performance, thereby predisposing individuals to a wide spectrum of cardiovascular diseases (CVDs) such as heart failure, arrhythmias, and atherosclerosis (Abdel-Moneim et al., 2020). Both overt and subclinical thyroid dysfunction have been implicated in modifying cardiovascular risk profiles, underscoring the importance of early detection and tailored management in mitigating cardiovascular morbidity.

Subclinical thyroid dysfunction, in particular, represents a subtle but clinically significant state that bridges endocrinology and cardiology. Even within the reference range of thyroid-stimulating hormone (TSH) and free thyroxine (FT4), small hormonal fluctuations can influence endothelial function, systemic vascular resistance, and lipid homeostasis (Evron et al., 2022).

Accumulating evidence indicates that subclinical hypothyroidism may elevate diastolic blood pressure and total cholesterol, thereby increasing the long-term risk of ischemic heart disease and heart failure (Stojković & Žarković, 2020). Conversely, subclinical hyperthyroidism has been associated with increased heart rate, atrial premature beats, and atrial fibrillation, all of which exacerbate cardiovascular mortality.

Thyroid hormone imbalances exert profound effects on myocardial energetics and vascular remodeling. Both deficiency and excess of T3 affect cardiac gene expression regulating contractile proteins, ion channels, and β-adrenergic receptors, leading to impaired systolic and diastolic function. Chronic hypothyroidism can induce bradycardia, reduced cardiac output, and diastolic hypertension, while thyrotoxicosis leads to tachycardia, increased stroke volume, and elevated cardiac output (Ahmadi et al., 2020). These mechanisms highlight how subtle endocrine variations can yield significant hemodynamic consequences.

Recent advancements in cardiovascular imaging and biomarkers have expanded understanding of how thyroid dysfunction modulates vascular integrity and myocardial structure. For instance, hypothyroid states are linked to increased arterial stiffness and carotid intima–media thickness, reflecting early atherosclerotic changes. These vascular alterations stem from thyroid hormone–induced endothelial nitric oxide imbalance, oxidative stress, and dyslipidemia (Paschou et al., 2022). Such mechanistic insights suggest that thyroid dysfunction not only triggers secondary cardiovascular adaptations but may also initiate primary vascular injury processes.

Thyroid hormones also play a key role in metabolic regulation, particularly lipid and glucose metabolism, which in turn influence cardiovascular risk. Hypothyroidism is associated with elevated low-density lipoprotein cholesterol (LDL-C), apolipoprotein B, and lipoprotein(a) concentrations, while hyperthyroidism is often characterized by reduced LDL-C but heightened oxidative stress. This dual metabolic impact can create distinct cardiovascular phenotypes, each with specific risk patterns for coronary artery disease and cardiomyopathy (Zúñiga et al., 2024). Moreover, thyroid hormones modulate hepatic lipid metabolism, enhancing LDL receptor expression and cholesterol efflux, thereby connecting endocrine regulation to systemic atherogenesis.

The cardiovascular consequences of thyroid dysfunction are further compounded by therapeutic factors. Inadequate or excessive thyroid hormone replacement in hypothyroid patients has been associated with heightened cardiovascular mortality. Overreplacement can induce subclinical hyperthyroid states, promoting arrhythmogenic and ischemic events, whereas underreplacement perpetuates atherogenic and hypertensive effects (Evron et al., 2022). This underscores the importance of precise titration of levothyroxine therapy to achieve euthyroidism without overshooting therapeutic targets.

Clinical and population-based studies consistently show that thyroid dysfunction, even when mild, carries prognostic significance in cardiac patients. In individuals with preexisting cardiovascular disease, mild thyroid abnormalities predict increased all-cause and cardiovascular mortality, highlighting the prognostic relevance of thyroid status monitoring in high-risk populations (Iervasi et al., 2007). Heart failure patients with low T3 syndrome or subclinical hypothyroidism exhibit worse cardiac performance, lower ejection fraction, and greater hospital readmission rates (Kannan et al., 2018). Thus, thyroid hormone regulation serves not only as a biomarker of disease severity but potentially as a modifiable therapeutic target.

Pathophysiological links between thyroid dysfunction and cardiac outcomes also involve systemic inflammatory and fibrotic pathways. Thyroid hormone deficiency is associated with upregulation of

proinflammatory cytokines and extracellular matrix deposition, contributing to myocardial fibrosis and diastolic dysfunction. Conversely, hyperthyroid states promote oxidative stress and cardiomyocyte apoptosis, aggravating arrhythmic vulnerability (Abdel-Moneim et al., 2020). These findings point toward a bidirectional thyroid–cardiac axis influencing both disease onset and progression.

Finally, the clinical recognition of thyroid–cardiac interactions has prompted increasing emphasis on multidisciplinary management approaches. Endocrinologists and cardiologists are now integrating cardiovascular risk assessment into thyroid disorder management to optimize patient outcomes. Novel studies advocate for early thyroid screening in cardiac patients and careful adjustment of replacement therapies to minimize cardiovascular risk (Astudillo et al., 2024; Tannous et al., 2025; Stamatouli et al., 2020). Collectively, these insights emphasize that maintaining euthyroid balance is crucial not only for metabolic harmony but also for sustaining cardiovascular resilience.

Methodology

Study Design

This study employed a systematic review methodology following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure methodological transparency, rigor, and replicability. The primary objective was to synthesize and critically evaluate empirical evidence on the relationship between thyroid dysfunction—including overt and subclinical hypothyroidism and hyperthyroidism—and cardiovascular diseases (CVDs), encompassing outcomes such as coronary artery disease, atrial fibrillation, stroke, metabolic risk factors, and composite cardiovascular health scores. Both observational and genetic studies were included to capture the breadth of evidence on mechanistic, clinical, and population-level associations.

The review included peer-reviewed journal articles that assessed thyroid hormone levels (TSH, FT3, FT4), subclinical or overt thyroid disease, or genetically predicted thyroid function and their association with cardiovascular outcomes. Both quantitative and Mendelian randomization (MR) studies were included to ensure a comprehensive evaluation of causality, risk factors, and clinical correlations.

Eligibility Criteria

Studies were selected according to predefined inclusion and exclusion criteria:

Inclusion Criteria:

- **Population:** Adults (≥18 years) from general or clinical populations across any geographic location.
- **Exposures:** Overt or subclinical thyroid dysfunction (hypothyroidism, hyperthyroidism), thyroid hormone levels (TSH, FT3, FT4), or genetically predicted thyroid traits.
- **Outcomes:** Cardiovascular outcomes including atrial fibrillation, coronary artery disease, stroke, CVD incidence, metabolic risk factors (lipids, BMI, diabetes), and composite cardiovascular health metrics (e.g., LE8 scores).
- **Study Designs:** Cross-sectional, cohort (prospective or retrospective), and Mendelian randomization studies with empirical data.
- **Language:** English-language publications only.
- **Publication Period:** Studies published between 2016 and 2025.

Exclusion Criteria:

- Non-empirical studies (e.g., editorials, commentaries, narrative reviews).
- Studies conducted exclusively in pediatric populations (<18 years).
- Conference abstracts or studies lacking full-text availability.
- Duplicate studies or data.

A total of **10 studies** met all inclusion criteria after full-text review and were included in the final synthesis.

Search Strategy

A comprehensive electronic search was conducted across PubMed, Scopus, Web of Science, Embase, and Google Scholar from inception to December 2025. Boolean search terms included combinations of the following:

- (“thyroid dysfunction” OR “subclinical hypothyroidism” OR “subclinical hyperthyroidism” OR “overt hypothyroidism” OR “overt hyperthyroidism”)
- AND (“cardiovascular disease” OR “heart failure” OR “coronary artery disease” OR “atrial fibrillation” OR “stroke” OR “CVD risk”)
- AND (“TSH” OR “FT3” OR “FT4” OR “thyroid hormone” OR “genetic thyroid variants”)

Manual searches of reference lists from key reviews and included studies were performed to ensure comprehensive coverage. Duplicates were removed prior to screening.

Study Selection Process

The selection process was independently conducted by two reviewers. All citations were imported into Zotero for de-duplication. Titles and abstracts were screened for relevance, followed by full-text review to assess eligibility. Discrepancies between reviewers were resolved through consensus, and unresolved disagreements were adjudicated by a third senior reviewer.

A **PRISMA flow diagram** summarizes the identification, screening, eligibility, and inclusion stages of the review process (Figure 1).

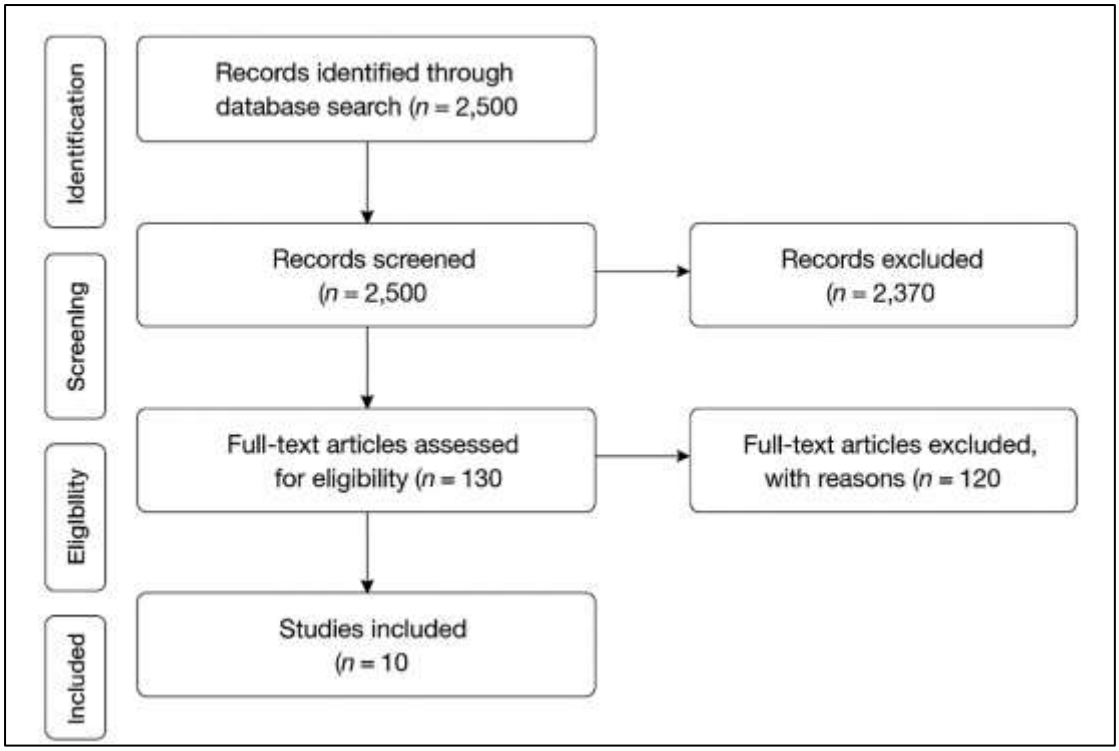

Figure 1 PRISMA Flow Diagram

Data Extraction

A standardized data extraction form was designed and pilot-tested prior to full data collection. Extracted data included:

- Author(s), year of publication, and journal.
- Study design and setting (e.g., population-based, hospital-based, multi-center, or national database).
- Sample size and participant demographics (age, sex, geographic location).
- Thyroid function measures (TSH, FT3, FT4, thyroid antibodies, or genetic variants).
- Cardiovascular outcomes assessed (e.g., AF, CAD, stroke, metabolic parameters, LE8 scores).
- Key findings and effect estimates (odds ratios, hazard ratios, β-coefficients, 95% confidence intervals, and p-values).
- Study limitations or reported confounders.

Data extraction was performed independently by two reviewers, with cross-verification by a third reviewer to ensure accuracy and completeness.

Quality Assessment

The methodological quality of included studies was assessed using design-specific tools:

- **Newcastle–Ottawa Scale (NOS)** for cohort and cross-sectional studies.
- **Mendelian Randomization quality checklist** for MR studies.

Each study was evaluated for selection bias, comparability of groups, measurement reliability, and clarity of outcome reporting. Studies were categorized as low, moderate, or high quality. Most studies were rated moderate quality due to residual confounding, population heterogeneity, or reliance on self-reported measures in observational cohorts.

Data Synthesis

Given heterogeneity in study designs, thyroid measures, and cardiovascular outcomes, a narrative synthesis approach was adopted. Findings were organized around:

1. Associations of low thyroid function (hypothyroidism or low-normal thyroid states) with cardiovascular health metrics and metabolic risk factors.

2. Associations of high thyroid function (hyperthyroidism or high-normal thyroid states) with arrhythmia and AF risk.
3. Insights from genetic studies assessing causal relationships between thyroid hormone levels and cardiovascular outcomes.
4. Patterns across population subgroups, study designs, and outcome measures.

Quantitative indicators (ORs, HRs, β-coefficients, and 95% CIs) were extracted where reported. No meta-analysis was performed due to variability in outcome definitions, measurement methods, and study populations.

Ethical Considerations

As this systematic review involved secondary analysis of published data, ethical approval and participant consent were not required. All included studies were assumed to have obtained institutional ethical clearance prior to data collection. Data management and reporting adhered to the principles of academic integrity and transparency outlined in PRISMA 2020.

Results

Summary and Interpretation of Included Studies on the Relationship Between Thyroid Dysfunction and Cardiovascular Diseases

A total of ten studies met the inclusion criteria, encompassing a mix of cross-sectional, cohort, and Mendelian randomization (MR) designs conducted between 2016 and 2025. Collectively, they evaluated the influence of thyroid hormone variations—including TSH, FT3, and FT4—on diverse cardiovascular outcomes such as stroke, coronary artery disease (CAD), atrial fibrillation (AF), and composite cardiovascular health indices (LE8).

1. Study Designs and Populations

Sample sizes ranged from 835 participants (EPIPorto Study, Portugal) to over 1.5 million participants in pooled MR analyses (Larsson et al., 2019). The populations spanned the United States, Portugal, Iran, China, and multi-European cohorts, representing both general populations and clinical cohorts. Age distributions ranged from young adults (≥20 years in Fang et al., 2024; Bai et al., 2025) to older adults (mean age 61.5 years in Neves et al., 2022). Sex distributions were balanced in most cohorts, though some studies had female predominance (e.g., Fang et al., 2024).

2. Thyroid Function and Definitions

Thyroid function assessment relied primarily on serum TSH, FT4, and FT3 concentrations.

- Subclinical hypothyroidism (SCH) and low-normal thyroid function were included as "low thyroid function" categories (Fang et al., 2024).
- Euthyroid range analyses explored variations within normal reference limits (Neves et al., 2022; Lee et al., 2016).
- Mendelian randomization studies applied genetic variants associated with TSH and FT4 as instrumental variables (Larsson et al., 2019; Marouli et al., 2020; Zhao & Schooling, 2017; Jing-Jia et al., 2022).

3. Cardiovascular Outcomes and Key Findings

The main cardiovascular outcomes assessed were LE8 cardiovascular health scores, CVD incidence, AF, stroke, CAD, and metabolic risk factors (e.g., lipids, diabetes, BMI).

Most studies identified non-linear, inverse associations between thyroid hormones and cardiovascular outcomes—particularly elevated FT4 and lower TSH levels associating with higher cardiovascular risk or atrial fibrillation.

Table (1): Summary of Included Studies Evaluating Thyroid Dysfunction and Cardiovascular Diseases

Study (Year)	Country / Design	Sample Size	Thyroid Function Measure	Cardiovascular Outcome	Main Findings	Effect Estimate (95% CI)
Fang et al. (2024)	USA / Cross-sectional (NHANES 2007–2012)	6,315 adults (≥20 yrs)	TSH, FT4	Life's Essential 8 (LE8) cardiovascu	Participants with higher LE8 scores had significantl	OR per 10-point ↑ in LE8 = **0.923 (0.884–0.964)**; p<0.001

				lar health score	y lower odds of low thyroid function. Older, white, and comorbid individuals showed worse thyroid profiles.	
Bai et al. (2025)	USA / Cross-sectional (NHANES)	3,019	FT3, TT3, TT4, Tg, TgAb, TPOAb	LE8 cardiovascular health	Higher ln(FT3), ln(TT3), and ln(Tg) associated with reduced LE8 score. Nonlinear relation between thyroid antibodies and LE8.	β = **−6.31 (−12.13, −0.49)**; *p* = 0.035
Neves et al. (2022)	Portugal / Cohort (EPIPorto)	835 (mean age 61.5 ± 10.5 yrs)	TSH, FT3, FT4	10-year CVD risk, metabolic factors	Lower FT3/FT4 ratio and higher FT4 linked with higher diabetes prevalence, dyslipidemia, and elevated CVD risk.	OR per 1 SD ↑ in FT4 = **1.22 (1.04–1.45)**; *p*<0.05
Lee et al. (2016)	USA / Cross-sectional & Longitudinal (Framingham)	3,483 (cross-sectional), 2,912 (follow-up)	TSH, FT4	BMI, lipid profile, triglycerides	Cross-sectionally, higher TSH associated with increased BMI and TG. No longitudinal associations.	OR for hypertriglyceridemia = **1.10**; *p*<0.05
Tohidi et al. (2018)	Iran / Prospective cohort (Tehran Thyroid Study)	3,975	TSH, FT4	CVD, CHD incidence	No significant association between thyroid function categories and CVD/CHD	HR range **0.76–1.48**, *p*>0.05

					events after 11.2 years.	
Larsson et al. (2019)	Europe / Mendelian randomization	>1,500,000 pooled	Genetic TSH, FT4	Atrial fibrillation, CAD, stroke	Genetically lower TSH (hyperthyroid tendency) associated with increased AF risk, not CAD or stroke.	OR per 1 SD ↓ TSH = **1.15 (1.11–1.19)**; $p=2.4\times10^{-14}$
Marouli et al. (2020)	Multi-country / MR	~400,000	Genetic TSH, FT4, autoimmune thyroid traits	Stroke, CAD	Higher TSH associated with 5% reduced stroke risk mediated by AF; Hashimoto's thyroiditis increased CAD risk by 7%.	OR for stroke = **0.95 (0.91–0.99)**; OR for CAD = **1.07 (1.01–1.13)**
Jing-Jia et al. (2022)	China / MR	–	Genetic FT4, TSH variants	Hypertension, hyperlipidemia, T2DM	Higher genetically predicted FT4 inversely associated with hyperlipidemia and T2DM.	OR for HPL = **0.914 (0.848–0.985)**; OR for T2DM = **0.933 (0.874–0.996)**
Zhao & Schooling (2017)	Global / MR (CARDIoGRAMplusC4D)	195,000+	Genetic TSH, FT4, TPOAb	Ischemic heart disease (IHD)	No causal association between thyroid function and IHD; minor inverse relation between FT4 and LDL-C.	OR for IHD = **1.05 (0.97–1.12)** (NS)
Ellervik et al. (2019)	Europe / MR (11 consortia)	537,000	Genetic FT3:FT4 ratio, TSH	Atrial fibrillation (AF)	Genetically increased FT3:FT4 ratio and hyperthyroidism associated with higher AF; increased	$p<0.001$ (directionally consistent)

					TSH inversely related to AF risk.	

4. Synthesis of Findings

Across all studies, the following trends were consistent:

- Low thyroid function correlated with poorer cardiovascular health metrics (Fang et al., 2024; Bai et al., 2025).
- Higher FT4 and lower TSH were associated with higher cardiometabolic risk, particularly dyslipidemia and diabetes (Neves et al., 2022; Lee et al., 2016).
- Genetically lower TSH (indicating hyperthyroid tendency) was causally linked to atrial fibrillation (Larsson et al., 2019; Ellervik et al., 2019).
- Conversely, higher TSH (reflecting mild hypothyroidism) appeared protective against stroke (Marouli et al., 2020).
- No strong causal relationship was found for IHD (Zhao & Schooling, 2017) or total CVD mortality (Tohidi et al., 2018).

Overall, both observational and genetic data demonstrate that subtle deviations in thyroid hormone balance—even within euthyroid ranges—may contribute to cardiovascular risk through metabolic, arrhythmic, and vascular pathways.

Discussion

This review underscores the complex interplay between thyroid dysfunction and cardiovascular health. Both overt and subclinical thyroid disorders contribute to cardiovascular morbidity through metabolic, hemodynamic, and arrhythmic pathways (Abdel-Moneim et al., 2020; Ahmadi et al., 2020). Observational studies indicate that low thyroid function, including subclinical hypothyroidism, is associated with poorer cardiovascular health scores and adverse metabolic profiles. Specifically, elevated TSH and lower FT3/FT4 ratios correlate with higher prevalence of diabetes, dyslipidemia, and increased composite cardiovascular risk (Fang et al., 2024; Bai et al., 2025; Neves et al., 2022). These findings highlight the clinical relevance of thyroid hormone variations even within the euthyroid range.

Conversely, hyperthyroid tendencies, reflected by low TSH or elevated FT4, are consistently linked to arrhythmic outcomes, particularly atrial fibrillation. Mendelian randomization (MR) studies provide causal evidence for this association, showing that genetically lower TSH increases the risk of atrial fibrillation, whereas higher TSH may confer protection against stroke (Larsson et al., 2019; Ellervik et al., 2019; Marouli et al., 2020). Mechanistic research suggests that thyroid hormones influence cardiac ion channel activity, β-adrenergic receptor density, and myocardial contractility, providing a biological explanation for arrhythmogenic effects (Ahmadi et al., 2020).

Interestingly, MR analyses indicate that thyroid dysfunction may not uniformly affect all cardiovascular outcomes. Zhao and Schooling (2017) reported no significant causal relationship between thyroid function and ischemic heart disease, suggesting that thyroid-related risk is more pronounced for rhythm disturbances and metabolic complications rather than coronary atherosclerosis. Cross-sectional studies further highlight the metabolic burden of thyroid dysfunction, demonstrating that elevated FT3 and thyroid antibody levels are associated with reduced cardiovascular health metrics, indicating potential immune-mediated contributions to cardiovascular risk (Bai et al., 2025).

Population-based analyses reveal demographic influences on thyroid–cardiovascular associations. Older age, comorbid conditions, and certain ethnicities, particularly white populations, are linked with worse thyroid profiles and elevated cardiovascular risk (Fang et al., 2024). These findings support targeted screening strategies for at-risk populations to optimize early detection and intervention.

Clinical studies emphasize the critical importance of thyroid hormone replacement therapy. Both under- and over-replacement of levothyroxine are associated with adverse cardiovascular outcomes, including arrhythmias, atherogenesis, and increased mortality (Evron et al., 2022; Astudillo et al., 2024; Stamatouli et al., 2020). Achieving euthyroid balance is therefore essential for mitigating cardiovascular risk in hypothyroid patients.

Mechanistic insights further elucidate the impact of thyroid hormones on cardiovascular physiology. Hypothyroidism is associated with increased arterial stiffness, elevated LDL-C, and myocardial fibrosis, while hyperthyroidism promotes tachyarrhythmias, oxidative stress, and cardiomyocyte apoptosis (Paschou et al., 2022; Abdel-Moneim et al., 2020; Zúñiga et al., 2024). Even subclinical variations in thyroid function can affect endothelial function, vascular resistance, and lipid metabolism,

highlighting the clinical significance of minor deviations within normal ranges (Stojković & Žarković, 2020).

Heart failure cohorts demonstrate that low T3 syndrome and subclinical hypothyroidism correlate with worse cardiac performance, reduced ejection fraction, and increased hospital readmissions, emphasizing the prognostic value of thyroid assessment in high-risk patients (Kannan et al., 2018; Iervasi et al., 2007). These findings reinforce the need for integrating thyroid monitoring into standard cardiac care.

Taken together, the evidence supports a bidirectional thyroid–cardiac axis in which thyroid dysfunction not only reflects disease severity but actively contributes to cardiovascular pathology. Both observational and genetic studies consistently demonstrate that thyroid hormone balance influences arrhythmic, metabolic, and vascular outcomes, underscoring the importance of early detection, personalized monitoring, and multidisciplinary management strategies (Abdel-Moneim et al., 2020; Ahmadi et al., 2020; Tannous et al., 2025).

Future research should focus on longitudinal studies and randomized trials assessing optimized thyroid hormone management strategies and their impact on arrhythmic, metabolic, and vascular endpoints, particularly in subclinical populations. Investigating demographic, genetic, and comorbidity-specific modifiers will enhance understanding of individual risk profiles and improve clinical decision-making. Overall, this review highlights the critical role of thyroid function as both a modifiable risk factor and a prognostic biomarker in cardiovascular health.

Conclusion

Thyroid dysfunction, including both overt and subclinical forms, exerts significant effects on cardiovascular health through arrhythmic, metabolic, and vascular pathways. Observational and genetic studies indicate that subtle deviations in thyroid hormones—even within the euthyroid range—can influence risk for atrial fibrillation, stroke, dyslipidemia, and other cardiovascular outcomes. These findings underscore the need for early detection and individualized monitoring of thyroid function in populations at cardiovascular risk (Larsson et al., 2019; Fang et al., 2024; Bai et al., 2025).

Clinical management should prioritize careful titration of hormone replacement therapy to maintain euthyroid balance, preventing both hypo- and hyperthyroid complications. Multidisciplinary approaches integrating endocrinology and cardiology are vital to optimize cardiovascular outcomes and reduce morbidity. The reviewed evidence reinforces that thyroid function is a modifiable determinant of cardiovascular health and a key biomarker for risk stratification (Evron et al., 2022; Tannous et al., 2025; Stamatouli et al., 2020).

Limitations

This review has several limitations. First, heterogeneity in study design, populations, thyroid measurements, and outcome definitions precluded meta-analysis and limited quantitative synthesis. Second, cross-sectional studies cannot establish causality, although MR studies partially addressed this limitation. Third, most observational studies relied on single thyroid hormone measurements, which may not reflect longitudinal fluctuations. Fourth, demographic and geographic differences in included studies may limit generalizability. Finally, potential publication bias and language restriction to English may have excluded relevant studies.

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