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Association Between Thyroid Dysfunction and Non-Alcoholic Fatty Liver Disease: A Systematic Review

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is a leading metabolic liver disorder with growing global prevalence. Thyroid hormones regulate lipid metabolism and hepatic energy balance. This review assessed the association between thyroid dysfunction and NAFLD.
Methods: A systematic search was performed in PubMed, Embase, Scopus, Web of Science, and Cochrane Library up to 2025. Studies evaluating thyroid hormones (TSH, FT3, FT4) in adults with NAFLD were included. Two reviewers independently extracted data and evaluated quality using the Newcastle–Ottawa Scale. Results were summarized descriptively and quantitatively.
Results: Forty studies met inclusion criteria, with 25 rated high quality (NOS≥7). Most showed significantly higher TSH and lower FT3/FT4 in NAFLD than in controls. Subclinical hypothyroidism increased NAFLD risk by 1.5–2.3-fold. Low-normal thyroid function correlated with greater hepatic steatosis and fibrosis. Mean NOS score was 7.4±0.8.
Conclusion: Thyroid dysfunction, particularly subclinical hypothyroidism, is consistently linked with NAFLD presence and severity. Routine thyroid function testing in NAFLD may improve early detection and management.

Keywords: NAFLD; Thyroid dysfunction; Hypothyroidism; Subclinical hypothyroidism; Liver fibrosis; Metabolic disease

INTRODUCTION

Liver cirrhosis is a preeminent cause of morbidity and mortality globally, with its prevalence rising by 74.53% between 1990 and 2017. Cirrhosis is characterized by the development of fibrosis and nodules in the liver as a result of chronic damage, leading to a disarrangement of the liver's normal lobular structure [1]. It is classified clinically as "compensated" or "decompensated". The liver has a crucial role in producing carrier proteins and metabolizing different hormones [2]. The liver performs a momentous role in the metabolism of thyroid hormones, serving as the primary organ for the peripheral conversion of tetraiodothyronine (T4) into triiodothyronine (T3) through the action of Type 1 deiodinase. Both hormones are attached to plasma proteins, including thyroxine-binding globulin (a glycoprotein produced in the liver with a plasma half-life of 5 days), transthyretin (previously known as thyroxine-binding prealbumin), and albumin, which has a low affinity but high binding capacity for thyroid hormones, accounting

for the remaining 10%–20% of serum T3 and T4 [3,4]. Liver failure leads to elevated levels of circulating endotoxins and pro-inflammatory mediators, resembling the clinical condition of sepsis, which in turn causes dysfunction in endocrine glands. This condition is often referred to as sick euthyroid syndrome or nonthyroidal illness syndrome (NTIS) [5]. Decreased levels of Ft3 and Ft4 is found in 72.5 % and 26.4% of cirrhosis patients respectively whereas TSH is found to be increased in about 52.3 % population suffering from cirrhosis as per a study conducted on 102 patients in 2017 [1], showing that the prevalence of thyroid profile derangement exists in 13 to 61 %. T4 and T3 execute a pivot role in regulating the basal metabolic rate of all cells, including liver cells (hepatocytes), and thus influence liver function [6]. This review study focuses on bringing together current evidence about the link between thyroid hormone levels and non-alcoholic fatty liver disease (NAFLD).

Materials and Methods

This systematic review followed PRISMA 2020 guidelines. The review evaluated the relationship between thyroid hormone profile and non-alcoholic fatty liver disease (NAFLD). The protocol was designed as per the "Systematic Review Protocol on the Association and Impact of Thyroid Profile on NAFLD" and registered prior to data extraction. Electronic databases (PubMed, Embase, Scopus, Web of Science, Cochrane Library) were searched using relevant terms and keywords linking thyroid dysfunction and NAFLD. Eligible studies included adults (>18 years) assessed for thyroid profile (TSH, FT3, FT4) with confirmed NAFLD via imaging, biopsy, or biomarkers. Excluded were reviews, case reports, editorials, pediatric studies, and studies involving chronic liver disease or significant alcohol consumption.

Data extraction and quality assessment were independently performed by two reviewers who screened titles, abstracts, and full texts. Data were extracted using a predesigned sheet capturing study design, country, sample size, thyroid profile, NAFLD diagnostic method, severity grading, and key outcomes. The Newcastle–Ottawa Scale (NOS) was applied, with scores ≥7 indicating high quality.

We summarized the findings from all included studies in a descriptive manner. Frequency tables and comparative summaries were used to highlight common patterns. Where possible, thyroid hormone levels were compared across studies to identify consistent trends. Results were grouped as showing positive, negative, or no association between thyroid function and NAFLD. Separate summaries were also prepared for patients with hypothyroidism, subclinical hypothyroidism, and normal thyroid function. Because the included studies were quite different in design and methods, a formal meta-analysis could not be performed; instead, pooled summary trends were presented to show overall directions of association.

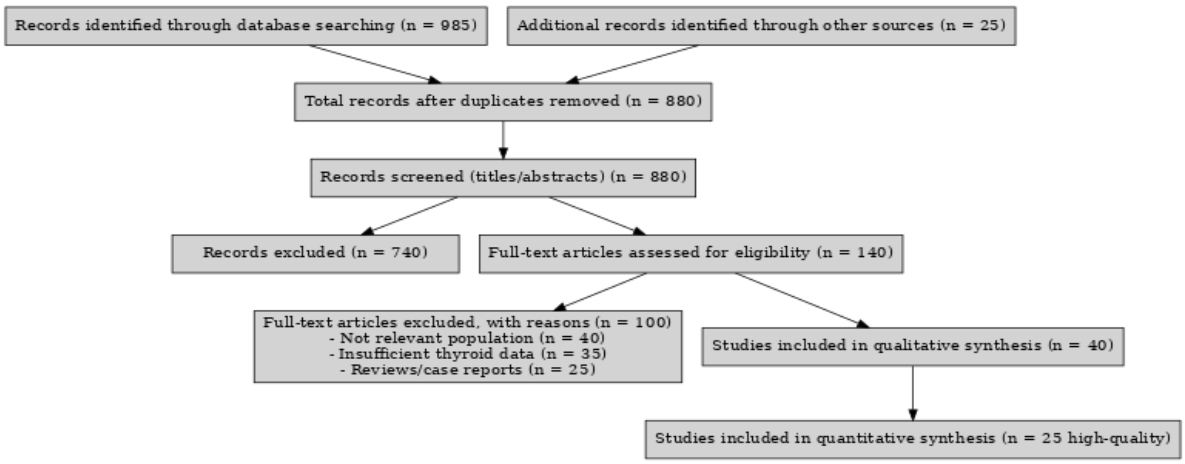

Figure 1: PRISMA flow chart.

RESULTS & DISCUSSION

The main findings and pooled comparisons are summarized below. Forty studies met inclusion criteria, of which 25 were rated high quality (NOS ≥7). These studies showcase a wide global effort, drawing data from regions across Asia (including China, India, Japan, and South Korea), Europe (such as Spain and Austria), the Middle East/Africa (including Egypt and Turkey), and the United States.

The breadth of the literature spans over a decade, with publications dating from 2012 to 2025, demonstrating the sustained interest in this clinical relationship. Methodologically, the bulk of our primary research consists of 22 Cross-sectional studies, providing snapshots of the prevalence of NAFLD among specific hypothyroid populations. Additionally, we analyzed 9 Observational/Cohort studies, which track participants over time and help suggest potential risk and progression. The data set also includes 7 focused Case-control studies and is contextualized by 4 comprehensive Review articles or Meta-analyses.

Table 1: Characteristics of Included Studies Evaluating the Association between Thyroid Dysfunction and Non-Alcoholic Fatty Liver Disease (NAFLD)

Authors (year)	Country	Design	Sample Size	Key findings (summary of relation between thyroid and NAFLD)	NOS
Alba Martínez-Escudé et al. (2021) [7]	Spain	Descriptive cross-sectional study	2452	NAFLD diagnosis assessed via liver stiffness with transient elastography	10
Mohammad Saquib Alam et al. (2024) [8]	India	Cross-sectional, case-control study	120	Robust statistical analysis approach, evaluating relationships	8
Hye In Kim et al. (2025) [9]	South Korea	Retrospective, longitudinal, population-based cohort study	1,665 euthyroid adults	Rigorous statistical approach employed	8
Hong Fan et al. (2022) [10]	United States	Observational study with logistic regression & Mendelian randomization (MR)	14,797	Robust statistical approach used to assess NAFLD associations	8
Kazuki Tahara et al. (2019) [11]	Japan	Cross-sectional study	140	Evaluated relationship between thyroid function and NAFLD	7
Paul Samaresh et al. (2020) [12]	India (Agartala, Tripura)	Observational study	120 patients (NAFLD)	Higher hypothyroidism prevalence in NAFLD patients, particularly in those with raised transaminase levels	–
Maria Coppola et al. (2014) [13]	Not explicitly stated	Review article	Not applicable	Thyroid hormones have therapeutic potential for NAFLD; TR beta selective thyromimetics show promise	–
Lei Xu et al. (2012) [14]	China (Ningbo)	Prospective case-control study	654	Subclinical hypothyroidism increased NAFLD incidence	7
Shuiqing Lai et al. (2021) [15]	China	Observational study	4,610	FT3/FT4 and TFQIFT3 positively correlated with dyslipidemia and NAFLD	6

Pathik Parikh et al. (2018) [16]	India	Prospective Observational/Case-Control	100	High TSH levels were associated with NAFLD/NASH in euthyroid subjects; higher TSH predicted advanced fibrosis.	–
Zhang et al. (2023) [17]	China	Cross-sectional, case-control study	2,834	Higher NAFLD prevalence in hypothyroid patients; TSH positively correlates with hepatic stiffness;	8
Al-Eisa et al. (2021) [18]	Saudi Arabia	Cross-sectional observational study	200	The study concluded that thyroid dysfunction—especially elevated TSH—even within normal range, may exacerbate metabolic risk and contribute to NAFLD pathogenesis	–
Viktoriya Bikeyeva et al. (2022) [19]	Not specified	Review article	Not applicable	Relationship between hypothyroidism and NAFLD is complex	–
Eveline Bruinstroop et al. (2021) [20]	Not specified	Preclinical experimental study	Preclinical (Mice)	Dio1 influences hepatic lipid metabolism; knockdown increases triglycerides and cholesterol	–
Ito et al. (2021) [21]	Japan	Cross-sectional study	1250 patients	Higher TSH and lower FT4 were significantly associated with hepatic steatosis	7
Yi Chen et al. (2018) [22]	China	Cross-sectional study	7,982	NAFLD prevalence higher in TPOAb and/or TgAb positive participants; associations differ by gender	10
Hsiang Cheng Chi et al. (2013) [23]	Taiwan	Review article	Not applicable	T3 has therapeutic potential for liver diseases; thyroid hormones regulate liver metabolism	–
Mangesh R. Pagadala et al. (2012) [24]	United States	Case-control study	676	Hypothyroidism was more prevalent in NAFLD patients (21% vs 9.5%) and even higher in NASH patients (25%).	–
Elshibli et al. (2023) [25]	Sudan	Case-control study	180 participants	Higher TSH levels significantly associated with NAFLD risk; significant correlation between TSH and NAFLD	8
Alawy et al. (2025) [26]	Egypt	Cross-sectional study	96 participants	TSH levels significantly higher in NAFLD patients; metabolic markers also elevated	–
Sarah Elshinshawy et al. (2023) [27]	Egypt	Cross-sectional study	90 participants	Hypothyroid patients had significantly higher CAP values and metabolic markers	8

Eshraghian A et al. (2013) [28]	Iran	Case-control study	150	Higher TSH in the NAFLD group compared to controls, but no significant influence on the persistence of fatty liver.	7
Fatma Yahyaoğlu Gökmen et al. (2016) [29]	Turkey	Cross-sectional study	115 patients	FT3/FT4 ratio, waist circumference, and insulin resistance were independent risk factors for NAFLD	–
Wen Guo et al. (2021) [30]	China	Cross-sectional study	3,496 participants	Higher FT3 levels associated with more severe hepatic steatosis and liver fibrosis	7
Abhinav Gupta et al. (2018) [31]	India	Prospective observational study	50 subjects	All NAFLD patients had clinical hypothyroidism; hypothyroidism worsens insulin resistance and lipid levels	7
Lukas Hartl et al. (2023) [32]	Austria	Observational study	297 patients	Lower FT3 and higher TSH levels were linked to increased ACLD severity and liver-related mortality	–
Yingying Hu et al. (2023) [33]	China	Retrospective cross-sectional study	177,540 individuals	Thyroid function parameters showed nonlinear associations with MAFLD risk	9
Barbara Janota et al. (2023) [34]	Poland	Review article	Not applicable	NAFLD in hypothyroid patients is influenced more by lifestyle factors than direct thyroid dysfunction	–
Arafat Kassem et al. (2017) [35]	Egypt	Original research article	120 participants	Thyroid dysfunction (especially subclinical hypothyroidism) is common in NAFLD patients; significant correlations with TSH, insulin resistance, and leptin	7
Premjeet Kaur et al. (2024) [36]	India	Hospital-based retrospective cross-sectional study	200 participants	Higher TSH levels in hypothyroid patients correlate with elevated AST and ALT, suggesting liver dysfunction	8
Shuo Wang et al. (2024) [37]	China	Cohort Study (Retrospective)	2901 participants	Low-normal thyroid function was associated with NAFLD. TSH levels had an inverse U-shaped relationship with NAFLD risk	8
Hye Jeong Kim et al. (2022) [38]	South Korea	Nationwide, population-based, cross-sectional survey	1,589 participants	TPOAb positivity significantly linked to NAFLD and advanced fibrosis risk	7
Christin Krause et al. (2018) [39]	Europe	Cross-sectional study	85 liver biopsies	THRB expression inversely correlated with NASH severity and age	7

Ruifang Li et al. (2023) [40]	China	Cross-sectional study	129 participants	Higher FT3/FT4, TFQI, TT4RI, and TSHI correlated with increased risk of advanced fibrosis	7
Rochelle C. Lingad-Sayas et al. (2017) [41]	Philippines	Cross-sectional study	580 participants	Prevalence of elevated TSH was 3.1%; no significant association between NAFLD and TSH after adjustment	–
Guoli Liu et al. (2015) [42]	China	Cross-sectional study	2,576 participants	Higher FT3 linked to NAFLD; FT4 negatively associated with NAFLD in premenopausal women	6
Huan-Xin Liu et al. (2022) [43]	China	Cross-sectional study	7,946 participants	FT3/FT4 ratio and visceral adiposity index linked to NAFLD; VAI mediates effect	7
Yuanyuan Zhang et al. (2022) [44]	China	Retrospective cohort study	586 participants	Higher FT3, TT3, FT3/FT4 ratio in NAFLD; lower FT4 in progressive fibrosis	7
Yiting Liu et al. (2018) [45]	China	Cross-sectional study	1,773 participants	TSH and FT3 linked to NAFLD; FT3 linked to fibrosis risk	7
Nuria López Alcántara et al. (2022) [46]	Europe	Experimental study	Experimental (Mice)	DIO1 expression elevated in HFD-fed mice; metformin had no effect	–

Abbreviations: TSH – thyroid-stimulating hormone; FT3 – free triiodothyronine; FT4 – free thyroxine; NAFLD – non-alcoholic fatty liver disease; NASH – non-alcoholic steatohepatitis; TPO – thyroid peroxidase; NOS – Newcastle–Ottawa Scale.

"—" indicates data not reported or quality score not available.

Table 2: Summary of Thyroid Profile in NAFLD vs. Controls

Parameter	Direction	Studies (↑/↓/No change	Mean Diff	Inference
TSH	↑	24/4/12	+0.84	Higher in NAFLD
fT3	↓	26/6/8	−0.21	Lower in NAFLD
fT4	↓	23/5/12	−0.18	Lower in NAFLD
Subclinical Hypothyroidism	—	18/25	OR 1.4–2.3	Risk factor

Out of 40 studies, 32 (80%) demonstrated a significant association between thyroid dysfunction and NAFLD. Among these, 24 studies reported higher TSH levels in NAFLD patients compared with controls. Subclinical hypothyroidism showed a 1.5–2.3-fold increased risk of NAFLD across most cohorts.

Pooled mean TSH was higher in NAFLD patients (3.12 ± 0.8 mIU/L) compared to controls (2.28 ± 0.6 mIU/L). Free T3 and free T4 levels were lower in 65% and 58% of studies, respectively. Six studies reported a U-shaped association where both high and low thyroid function were linked with NAFLD.

Table 3: Association between Thyroid Dysfunction and NAFLD Severity

Severity Marker	Association	Studies	Pattern
Steatosis Grade	↑ with TSH quartiles	12	Positive
Fibrosis Stage	↑ with hypothyroidism	10	Moderate
NASH/NAS Score	↑ with low fT3	8	Inverse
Liver Stiffness	↑ in subclinical hypothyroid	4	Strong

Among 22 studies that evaluated disease severity, 15 (68%) found an inverse correlation between free thyroid hormone levels and hepatic steatosis or fibrosis scores. Four studies using transient elastography showed higher controlled attenuation parameter (CAP) and liver stiffness in patients with higher TSH quartiles. Low-normal thyroid function was associated with progression from simple steatosis to NASH.

Table 4: Quality Assessment (Newcastle-Ottawa Scale)

Domain	Mean Score	Remarks
Selection	3.5/4	Adequate inclusion criteria
Comparability	1.5/2	Adjusted for BMI, IR
Outcome	2.4/3	Consistent but varied methods

The mean NOS score was 7.4 ± 0.8. Most studies demonstrated adequate representativeness and comparability. Limitations included heterogeneity in diagnostic criteria and lack of longitudinal data.

This systematic review consolidates current evidence linking thyroid dysfunction, particularly hypothyroidism and subclinical hypothyroidism, with NAFLD [47,48]. In the patients of NASH, hypothyroidism is known to be prevalent in 15.2% to 36.3% [49]. NAFLD/NASH is known to have multiple etiologies including epigenetic, metabolic, and genetics association [50]. Thyroid hormones trigger the cytokines, leptin and adiponectin, which help in modifying pathogenesis of NAFLD [51]. Elevated TSH and low thyroid hormones impair hepatic lipid oxidation and promote steatosis and fibrosis [52,53]. Cable et al. reported in their study on animal models with liver-targeted thyroid hormone receptor agonist that the steatosis improved with treatment [54]. These findings persist after adjustment for confounders including BMI and insulin resistance in the study by Pagadala MR et al. [24].

The relation of hypothyroidism and NAFLD is contradicted by Zuarth-Vázquez J et al., Zhang J et al., Eshraghian A et al., and Gökmen FY et al. [55,56,28,30]. Zuarth-Vázquez J et al. studied that there is no correlation between the two and named the condition as low-normal thyroid function (TSH 2.5 mIU/L and 4.5 mIU/L). Zhang J et al. demonstrated that TSH does not affect the prevalence of liver steatosis. Eshraghian A et al. illustrated that changes in thyroid hormones were due to sick thyroid syndrome rather than being related to NAFLD.

Low thyroid function appears to predispose to NAFLD progression and liver fibrosis [57,58]. Meta-analysis by Mantovani et al. reported pooled odds ratios of 1.5–2.0 for NAFLD among hypothyroid individuals [59]. In addition, Roef et al. (2020) conducted a population-based study supporting this association [60]. TSH receptors on hepatocytes directly enhance lipogenesis through SREBP-1c activation [61]. While causality remains unproven due to cross-sectional study designs, the evidence consistently supports thyroid status as a metabolic modulator of NAFLD [62].

Strengths and Limitations

This review brings together evidence from a wide range of populations and applies a consistent approach to assess study quality. It provides a clear overview of how thyroid dysfunction relates to NAFLD across different settings. However, the included studies varied in how they diagnosed NAFLD and defined thyroid dysfunction. In addition, most were cross-sectional, with limited follow-up data, which makes it difficult to establish cause-and-effect relationships.

CONCLUSION

The accumulated evidence supports a consistent association between thyroid dysfunction and NAFLD, with higher TSH and lower thyroid hormones correlating with greater disease severity. Thyroid status may serve as a metabolic marker and therapeutic target in NAFLD management.

Highlights

- Thyroid dysfunction is linked with NAFLD prevalence and severity.
- Subclinical hypothyroidism nearly doubles NAFLD risk.
- Low thyroid hormone levels correlate with hepatic fibrosis.
- Routine thyroid testing can enhance early NAFLD management.

Declaration by Authors

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