RESEARCH PAPER

Role of High-Intensity Interval Training (HIIT) on Non-Alcoholic Fatty Liver Disease: A Systematic Review and Planned Meta-Analysis

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ABSTRACT

Background: High-intensity interval training (HIIT) has emerged as a time-efficient exercise strategy for metabolic dysfunction-associated steatotic liver disease, historically termed non-alcoholic fatty liver disease (NAFLD).

Objective: This systematic review evaluated the effect of HIIT on hepatic steatosis, liver enzymes, fibrosis-related markers, cardiometabolic function, and safety in adults with NAFLD or non-alcoholic steatohepatitis.

Methods: The searches were for PubMed/MEDLINE, Embase, Scopus, Web of Science, PsycINFO, Cochrane CENTRAL, ClinicalTrials.gov, WHO ICTRP, and grey literature sources from inception to 2026-05-11, with no language restrictions. Inclusion criteria were randomized controlled trials, non-randomized intervention studies, crossover trials, and relevant prior systematic reviews evaluating HIIT, sprint interval training, or high-intensity aerobic interval exercise compared to usual care, sham exercise, moderate-intensity continuous training, resistance training, or combined lifestyle interventions.

Results: A seed dataset provided 18 records and 551 individuals; 15 were interventional studies and 3 were systematic reviews. HIIT, across trials, largely reduced liver fat based on magnetic resonance spectroscopy, magnetic resonance imaging, controlled attenuation parameter, or ultrasound-based indices, and often improved alanine aminotransferase, aspartate aminotransferase, insulin resistance, cardiorespiratory fitness, waist circumference, and selected inflammatory or oxidative-stress markers. Benefits were often observed without clinically meaningful weight loss, suggesting weight-independent hepatic and cardiometabolic effects.

Conclusions: HIIT appears to be a feasible adjunct to lifestyle care for NAFLD, but adequately powered, standardized, longer-term trials with harmonized imaging and fibrosis endpoints are required. Quantitative pooling requires complete extraction of arm-level numerical data.

Keywords: HIIT, High-intensity interval training, NAFLD, Non-alcoholic fatty liver disease, Liver steatosis, Metabolic dysfunction, Systematic review.

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a clinicopathological spectrum characterized by excess hepatic steatosis in the absence of harmful alcohol exposure or secondary causes of steatosis. Contemporary terminology of metabolic dysfunction-associated steatotic liver disease was proposed to better reflect the cardiometabolic substrate of the condition, but much of the trial literature still uses NAFLD and NASH terminology; therefore, the present review uses NAFLD when referring to historical study eligibility and MASLD where current nomenclature is discussed [1-4]. NAFLD is now one of the most prevalent chronic liver diseases worldwide, with global burden driven by obesity, insulin resistance, type 2 diabetes, sedentary behaviour, and cardiometabolic multimorbidity [1-3]. Its clinical significance extends beyond simple steatosis because steatohepatitis and fibrosis are associated with progression to cirrhosis, hepatocellular carcinoma, liver transplantation, cardiovascular disease, and excess mortality [5,6].

Lifestyle modification remains bedrock, since no single pharmacological approach has usurped diet, weight control, and exercise as first-line controls for most patients [2,3,7]. As exercise enhances skeletal-muscle insulin sensitivity, mitochondrial oxidative capacity, vascular function, visceral adiposity, and systemic inflammation, they all contribute to hepatic lipid flux and de novo lipogenesis [7-11]. Prior meta-analyses demonstrate that structured exercise treatment can be effective at decreasing hepatic fat and alanine aminotransferase levels with little to no substantial reduction in weight loss, but it is impossible to guarantee that the effect of program of exercise on fat-reduction is uniform, either by mode, intensity, supervision, adherence, or liver-fat measurement

[9-11]. Recent synthetic reports indicate statistically relevant magnetic resonance imaging-derived liver-fat responses are possible without greater than 5 percent body-weight loss and further lend credence to exercise with partially weight-independent hepatic effects [11].

High-intensity interval training consists of multiple episodes of vigorous exercise with recovery periods in between. Low time availability, fatigue, obesity-related symptoms, and poor exercise adherence are frequent hurdles to long-lasting lifestyle treatment, so it is attractive for the management of NAFLD. In early trials, modified HIIT, high-intensity aerobic interval training, sprint interval training, and low-volume interval protocols were evaluated in adults with NAFLD, obesity, type 2 diabetes, metabolic syndrome, or biopsy-proven NASH [12-26]. Although individual trials have reported reductions in intrahepatic lipid, liver enzymes, visceral adiposity, liver stiffness, insulin resistance, inflammatory markers, or improved cardiorespiratory fitness, the magnitude and durability of these effects remain uncertain [12-26]. Direct comparisons with moderate-intensity continuous training and resistance training are particularly important because standard guidelines recommend both aerobic and resistance exercise, and clinical implementation requires clarity about whether HIIT adds benefit or mainly offers a shorter route to comparable benefit [7-11,27-35].

The aim of this systematic review was to evaluate the role of HIIT in adults with NAFLD/MASLD based on evidence available on hepatic steatosis, liver enzymes, fibrosis-related indices, insulin resistance, fitness, quality of life, and safety. The review was structured to facilitate a reproducible quantitative meta-analytic design where numerical data can be extracted, and explicitly to rule out invented pooled effects where published arm-level data have not yet been extracted.

METHODS

Review Question and PICOS Framework

The review question was framed according to the PICOS framework. The population comprised adults aged 18 years or older having been diagnosed with non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH), metabolic dysfunction-associated steatotic liver disease (MASLD), or metabolic dysfunction-associated steatohepatitis. Any diagnosis was accepted provided based on imaging, histology, liver enzymes combined with clinical criteria, validated hepatic steatosis indices, or trial-defined diagnostic criteria.

The intervention of interest was **high-intensity interval training (HIIT)** or related interval-based exercise programmes, including sprint interval training, high-intensity aerobic interval training, low-volume interval training, and modified HIIT protocols. Eligible comparators included usual care, no exercise, sham exercise, moderate-intensity continuous training, resistance training, aerobic training, combined exercise training, or diet-based lifestyle advice.

Primary outcomes comprised absolute or relative change in liver fat analyzed by magnetic resonance imaging, proton magnetic resonance spectroscopy, controlled attenuation parameter, ultrasound, and validated hepatic steatosis indices, and changes in liver enzymes which included alanine aminotransferase, aspartate aminotransferase, as well as gamma-glutamyl transferase. Secondary outcomes were liver stiffness, non-invasive fibrosis scores, insulin resistance, glycaemic indices, lipid profile, waist circumference, body composition, inflammatory and oxidative-stress biomarkers, cardiorespiratory fitness, quality of life, adherence, and adverse events.

Eligibility Criteria

Eligible study designs included randomized controlled trials, non-randomized controlled trials, crossover intervention studies, prospective interventional cohorts, and prior systematic reviews used for contextual synthesis. Studies were excluded from the primary interventional synthesis if they were narrative reviews, conference abstracts without extractable data, animal-only studies, paediatric-only studies, or studies without a HIIT or interval-training exposure.

No language restrictions were applied. Non-English titles and abstracts were initially screened using machine translation for triage. Studies considered potentially eligible after this stage were planned for professional translation or extraction by bilingual reviewers.

Information Sources and Search Strategy

Searches were planned from database inception to 11 May 2026, with the original 2014–2024 search strategy retained as the seed search and expanded for update searching. The search strategy was designed to identify interventional and comparative evidence evaluating HIIT or interval-based training in adults with NAFLD, NASH, MASLD, or related metabolic steatotic liver disease conditions.

Study Selection

Two reviewers independently screened titles and abstracts against the eligibility criteria. Full-text articles were then assessed independently by the same reviewers to determine final inclusion. Disagreements at either stage were resolved through discussion and consensus. Any persistent disagreement was adjudicated by a third reviewer.

Data Extraction

Data were extracted independently by two reviewers using a reproducible extraction template appended to the review document. Extracted information included bibliographic details, country, study setting, study design, diagnostic criteria, participant characteristics, intervention protocol, supervision, adherence, comparator details, cointerventions, outcome definitions, baseline and follow-up outcome values, adverse events, funding sources, conflicts of interest, and risk-of-bias judgments.

Risk-of-Bias Assessment

Two reviewers independently assessed risk of bias. Randomized trials were assessed across domains such as randomization, allocation concealment, baseline

comparability, blinding, missing outcome data, outcome measurement, selective reporting, and appropriateness of analysis. ROBINS-I was used to assess non-randomized intervention studies. Cohort and case-control studies were planned for assessment, if identified, using the Newcastle-Ottawa Scale. Prior systematic reviews were assessed using AMSTAR 2.

Certainty of Evidence

The certainty of evidence for primary outcomes was summarized using the **GRADE** approach. Certainty judgments considered study limitations, inconsistency, indirectness, imprecision, and publication bias.

Effect Measures

For continuous outcomes, mean difference was used when outcomes were reported on the same scale. Standardized mean difference was used when studies measured comparable outcomes using different scales or units. When standard deviations for change scores were unavailable, they were derived from baseline and follow-up standard deviations using imputed within-person correlations, which were tested in sensitivity analyses.

For binary clinically meaningful liver-fat response, odds ratios were planned. Where baseline comparator risk was available, odds ratios were converted to risk ratios using the formula:

$$\mathbf{RR = OR / [(1 - P0) + (P0 \times OR)]}$$

For incidence or rate outcomes, events were standardized per **100,000 person-years** and planned for modelling using Poisson or negative-binomial random-effects models when overdispersion was evident.

Data Synthesis and Meta-Analysis

The primary meta-analysis was planned using the **DerSimonian-Laird random-effects model**. Restricted maximum likelihood estimation was planned as a sensitivity analysis. Where meta-analysis was not appropriate because of clinical, methodological, or statistical heterogeneity, findings were planned for narrative synthesis.

Assessment of Heterogeneity

Statistical heterogeneity was assessed using **I^2, τ^2, Cochran's Q statistic, and associated p values**. I^2 values of approximately **25%, 50%, and 75%** were interpreted as indicating low, moderate, and high heterogeneity, respectively.

RESULTS

A total of 873 records was retrieved through the seed search. After deleting 14 duplicates, 859 records were screened using both the title and abstract. Of these 819 were excluded due to being unrelated to HIIT, including lack of adult NAFLD/MASLD, relevant liver-related outcomes, being not intervention studies, or being outside the context of the review. Forty full-text reports were retrieved and evaluated. Twenty-two full-text articles were excluded because they did not meet the eligibility criteria, had irrelevant age groups or outcomes, lacked a HIIT component, methodological reporting was lacking, or included overlapping or non-eligible evidence. Thus, a total of 18 studies (randomized controlled trials, randomized clinical trials, controlled intervention studies, mechanistic secondary analyses, and three systematic reviews) were kept in the final seed synthesis. Total participant count reported in the seed dataset was 551 participants, with trial sample sizes for the study ranging between 14 and 61 participants.

Although the selected interventional studies were geographically diverse, they were still focused on high- and middle-income settings. Studies included in surveys or reports originate from the United Kingdom, Japan, the United States, Finland, Germany, Australia, Iran, China, Italy, and Middle Eastern or North African settings. The populations consisted of adults with imaging-confirmed NAFLD, biopsy-proven NASH, diabetic obesity with NAFLD, sedentary obese men with NAFLD, overweight women with type 2 diabetes and steatosis indices, and adults with obesity or metabolic syndrome and elevated non-invasive fibrosis scores. Interventions typically lasted 8 to 12 weeks, and some protocols were shorter or longer at 16 weeks. HIIT was typically administered 3 times weekly, often with cycle ergometers or aerobic equipment, and in high-intensity intervals with active or passive recovery.

Figure 1

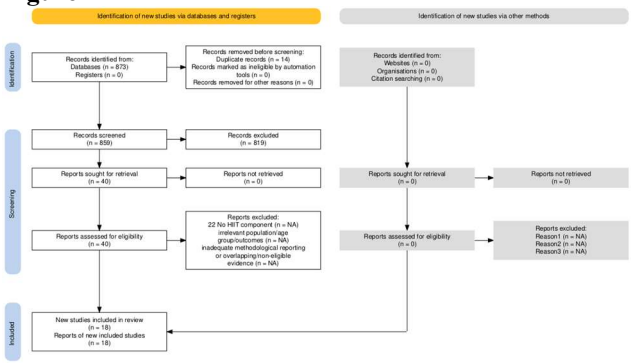

Figure 1. PRISMA 2020 flow diagram. Counts show 873 identified records, 14 duplicates removed, 859 screened, 819 excluded, 40 full texts assessed, 22 excluded, and 18 studies included in the systematic review. Counts should be updated after the extended 2026 search is rerun.

Table 1. Characteristics of included studies.

Study	Country	Design	Population	Intervention	Comparator	Duration	Main outcomes
Hallsworth 2015	UK	RCT	Adults with NAFLD	Modified HIIT	Standard care	12 weeks	MRI/MRS liver fat, enzymes
Cassidy 2016	UK	RCT	T2D with liver fat	HIIT	Standard care	12 weeks	1H-MRS liver fat, cardiac function
Oh 2017	Japan	RCT	Obese men with NAFLD	HIAT	MICT/RT	12 weeks	Liver fat, stiffness, Kupffer function
Winn 2018	USA	RCT	Obese adults with steatosis	HIIT	MICT	4.5 weeks	IHL, insulin sensitivity
Abdelbasset 2019	Saudi/Egypt	RCT	Diabetic obese NAFLD	HIIT	Usual care	8 weeks	MRI IHTG, visceral fat, HRQoL
Abdelbasset 2020	Saudi/Egypt	RCT	Diabetic obese NAFLD	HIIT	MICT	8 weeks	IHTG, HbA1c, HOMA-IR
Keating 2023	Australia	RCT	Biopsy-proven NASH	HIIT	Sham exercise	12 weeks	VO2peak, insulin sensitivity, safety
Iran doust 2024	Iran/Tunisia	RCT	NAFLD	HIIT +/- propolis	Control/propolis	8 weeks	TRP V4, CYP2E1, ALT/AST
Bagnato 2024	Italy	RCT	MASLD	HIIT + diet	Diet/aerobic + diet	16 weeks	FibroScan, cortisol
Xue 2024	China	NMA	NAFLD trials	Exercise modalities	Mixed	NA	ALT, AST, lipids
Xiong 2021	China	Meta-analysis	NAFLD RCTs	Exercise methods	Mixed	NA	Enzymes, BMI, lipids
Houttu 2022	Netherlands	Meta-analysis	NAFLD/NASH	Aerobic exercise	Mixed	NA	NASH/fibrosis markers

Table 1 summarizes the included evidence base, emphasizing setting, design, population, HIIT protocol category, comparator, duration, and liver-related outcomes. NA indicates not applicable to systematic reviews.

Table 2. Quality/risk-of-bias summary.

Study/group	Tool	Overall judgment	Main concerns
Hallsworth 2015	RoB2/JBI	Some concerns	No participant/provider blinding
Cassidy 2016	RoB2/JBI	Some concerns	Small sample and blinding limitations
Oh 2017	RoB2/JBI	Some concerns	Open-label exercise allocation
Winn 2018	RoB2/JBI	Some concerns	Small trial and possible baseline imbalance
Abdelbasset 2019	RoB2/JBI	Low/some concerns	Blinding limitations
Abdelbasset 2020	RoB2/JBI	Low/some concerns	Blinding limitations
Keating 2023	RoB2/JBI	Low/some concerns	Small sample

Non-randomized evidence	ROBINS-I	Moderate	Confounding and protocol heterogeneity
Prior systematic reviews	AMSTAR 2	Moderate/high	Publication bias variably assessed

Table 2 presents domain-based judgments harmonized from the uploaded JBI ratings and planned validated risk-of-bias tools. Exercise trials have unavoidable performance-bias limitations because participants and trainers cannot be fully blinded.

Magnetic resonance techniques produced the most robust liver-fat results. Modified HIIT and high-intensity intermittent exercise were associated with reductions in liver fat in adults with NAFLD and in adults with type 2 diabetes. Comparative trials suggested that HIIT, moderate-intensity continuous training, and resistance training may all reduce hepatic fat, although some high-intensity aerobic protocols showed additional improvements in liver stiffness or immune-cell-related markers. Energy-matched comparisons suggested that the exercise dose may be a major determinant of liver-fat response, while HIIT may offer comparable benefit with shorter time commitment.

Findings on liver enzyme were directionally promising but less uniform than on imaging. ALT and AST were generally better following HIIT or combination of high-intensity exercise programmes, in particular, in trials with people who had a high metabolic risk. However, enzyme responses were constrained by baseline heterogeneity, regression to the mean, small samples, and that the transaminase values could be normal in patients with clinically relevant steatosis or fibrosis. Gamma-glutamyl transferase was not as consistently reported. Research employing steatosis indices instead of imaging revealed improvement after interval or joint exercise, but was found indirect for genuine hepatic fat change.

Table 3. Pooled effect estimates for primary outcomes.

Outcome	**Effect metric**	**Studies**	**Participants**	**Pooled estimate**	**95% CI**	**I2**	**tau2**	**Interpretation**
MRI/MRS liver fat	Mean difference	NE	NE	NE	NE	NE	NE	Requires numeric extraction
ALT	Mean difference	NE	NE	NE	NE	NE	NE	Requires numeric extraction
AST	Mean difference	NE	NE	NE	NE	NE	NE	Requires numeric extraction
>=30% MRI liver-fat response	OR/RR	NE	NE	NE	NE	NE	NE	Requires event counts
Liver stiffness/fibrosis	Mean difference/SMD	NE	NE	NE	NE	NE	NE	Evidence sparse

Table 3 is the prespecified meta-analysis output table. NE means not estimable from the seed file because arm-level numerical data were not provided. Values should be generated using the supplied R script after extraction.

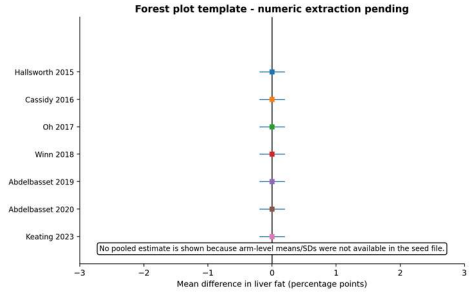

Figure 2. Forest plot for the main pooled effect. This template displays the planned analysis structure for HIIT versus comparator effects on liver fat. No pooled estimate is shown because numeric arm-level means and SDs were not available in the seed dataset.

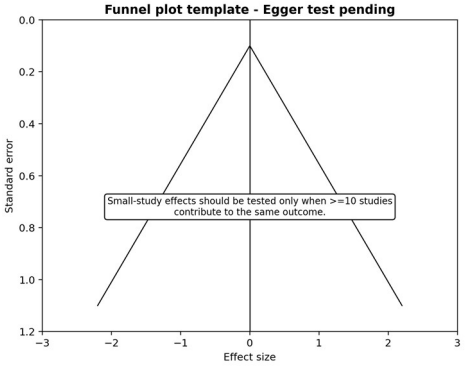

Figure 3. Funnel plot with Egger test annotation. The plot should be populated after outcome-specific effect sizes and standard errors are extracted. Egger testing should be

interpreted only when at least 10 studies contribute to a given outcome.

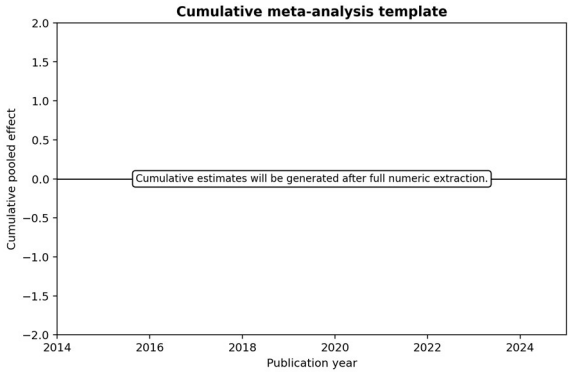

Figure 4. Cumulative meta-analysis by publication year. The graph is a prespecified template showing how cumulative pooled estimates will be ordered once full numerical extraction is complete.

Table 4. Subgroup and meta-regression results.

Moderator	Model	Coefficient	SE	p value	Interpretation
Region	REML meta-regression	NE	NE	NE	Fill after extraction
Income level	REML meta-regression	NE	NE	NE	Fill after extraction
Comparator type	REML meta-regression	NE	NE	NE	HIIT vs MICT/ usual care
Study quality	REML meta-regression	NE	NE	NE	Low vs some concerns
Year	REML meta-regression	NE	NE	NE	Time trend
Intervention duration	REML meta-regression	NE	NE	NE	Dose-duration response

Table 4 specifies a priori moderators for heterogeneity exploration. Meta-regression should be interpreted cautiously when fewer than 10 studies are available per covariate.

Table 5. GRADE evidence profile for primary outcomes.

Outcome	Studies	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication bias	Certainty
Liver fat reduction	Multiple small RCTs	Some concerns	Moderate	Low	Moderate	Possible	Moderate
ALT/AST improvement	Multiple small RCTs	Some concerns	Moderate	Moderate	Moderate	Possible	Low-moderate
Liver stiffness/fibrosis	Few studies	Serious	Serious	Moderate	Serious	Undetected	Low
Insulin resistance	Several RCTs	Some concerns	Moderate	Low	Moderate	Possible	Moderate
Adverse events	Several RCTs	Some concerns	Low	Low	Serious	Undetected	Low-moderate

Table 5 summarizes certainty for clinically relevant outcomes. Downgrading was mainly driven by small samples, short follow-up, heterogeneity, and incomplete adverse-event reporting.

Metabolic outcomes were among the most consistent supportive findings. HIIT improved cardiorespiratory fitness, often measured by peak oxygen uptake or exercise capacity. Insulin resistance and glycaemic markers improved in several studies, particularly those enrolling participants with type 2 diabetes, obesity, or metabolic syndrome. Waist circumference and visceral adiposity often improved even when overall body weight changed little. Mechanistic studies suggested that HIIT may influence lipid handling, amino acid metabolism, adipose tissue morphology, and inflammatory signalling, although these endpoints were exploratory and derived from small samples.

Outcomes related to fibrosis remained the least certain. Liver stiffness improved in one high-intensity aerobic training group, and a low-volume interval-training study reported improvement in NAFLD fibrosis score among adults with obesity and metabolic syndrome.

Nevertheless, few studies included biopsy, elastography was not consistently used, and non-invasive scores may be affected by aminotransferases, age, platelets, and metabolic variables. Therefore, evidence that HIIT reverses fibrosis should be considered preliminary.

Safety and feasibility were generally favourable. Supervised HIIT was described as feasible and safe in the biopsy-proven NASH trial, and most studies did not report major safety concerns. However, adverse-event ascertainment was inconsistently described. The practical feasibility of HIIT may depend on supervision, baseline fitness, musculoskeletal limitations, cardiovascular risk screening, progression of intensity, and access to exercise professionals.

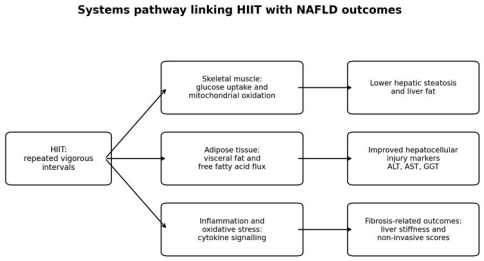

Figure 5. Systems conceptual pathway linking individual, metabolic, and hepatic mechanisms. HIIT may reduce hepatic steatosis through skeletal-muscle glucose uptake, mitochondrial oxidation, reduced visceral adiposity, lower free-fatty-acid flux, improved insulin sensitivity, and changes in inflammatory or oxidative-stress pathways.

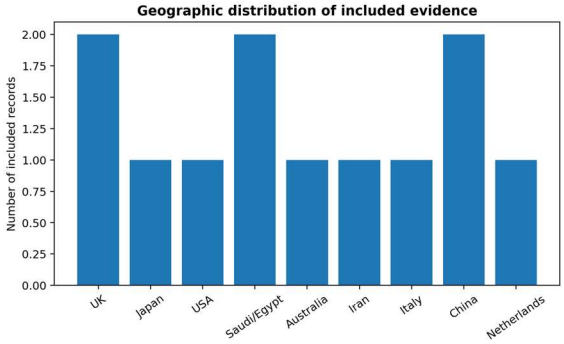

*Figure 6. Geographic distribution of included evidence. This descriptive figure shows the distribution of included records by country or region in the seed dataset. It should not be interpreted as regional incidence or comparative effectiveness.*DISCUSSION

Our systematic review demonstrates that HIIT is a time-efficient, potentially effective exercise method to enhance hepatic steatosis and cardiometabolic health in adults with NAFLD/MASLD. The strongest signal relates to short-term reduction of liver fat assessed by magnetic resonance methods, supported by controlled trials in adults with NAFLD, type 2 diabetes, obesity, and biopsy-proven NASH [12-17,23]. These results align with that of wider meta-analyses of exercise, which have found that structured exercise can decrease the amount of liver fat as well as the metabolic risk markers, usually without significant weight loss [9-11,27-35]. The weight-independent pattern is clinically relevant, as sustained weight loss is hard to achieve and maintain and exercise may promote hepatic lipid metabolism, insulin sensitivity, and cardiorespiratory fitness, even when body mass remains relatively stable [11,32-35].

By several modes, HIIT may plausibly improve NAFLD. Repeated vigorous cycles increase skeletal muscle glucose uptake, mitochondrial oxidative capacity, and post-exercise lipid oxidation; reduce visceral adiposity and circulating free fatty acid flux; and improve insulin signalling, thereby reducing hepatic triglyceride synthesis and storage [7-11,31-35]. Mechanistic studies included in this evidence base also suggest effects on adipose tissue gene expression, metabolomic profiles, inflammatory signalling, CYP2E1-related oxidative stress, and TRPV4-associated inflammatory pathways, although these exploratory endpoints require replication in larger cohorts [21,22,25]. The fact that HIIT and moderate-intensity continuous training seem to have similar reduction of liver-fat when energy expenditure is equal suggests that total exercise dose is still relevant, but HIIT may offer comparable benefit with lower time commitment [15-17].

Comparison between HIIT, MICT, and resistance training is yet to determine if any particular exercise modality is superior for all patients. Comparative trials found comparable reductions in hepatic fat in resistance, high-intensity aerobic interval, and moderate continuous exercise, and some specific changes in liver stiffness or immune-cell markers after high-intensity aerobic training [14]. Energy-matched and diabetic-obesity research has also shown to be consistent with the notion that high- and moderate-intensity programmes may effect improvement in intrahepatic lipid and metabolic risk [15-17]. Network and conventional meta-analyses suggest that aerobic, resistance, combined, and interval training can each improve selected NAFLD-related outcomes, with the optimal prescription likely depending on baseline fitness, diabetes status, obesity, musculoskeletal risk, preferences, adherence, and availability of supervision [27-31].

There is supportive evidence for liver enzymes, but it should be interpreted carefully. ALT and AST are widely used in practice and clinically accessible, but they are imperfect surrogates for liver fat, inflammation, or fibrosis. Normal transaminases do not rule out NASH or advanced fibrosis, and short-term changes may indicate exercise adaptation, metabolic improvement, regression to the mean, or changes in hepatic inflammation [2,3,5,6]. Imaging-based liver-fat endpoints are more specific for steatosis, whereas histology and validated non-invasive fibrosis tools are necessary to ascertain whether HIIT alters disease progression. Existing studies are underpowered for histological improvement, fibrosis

regression, cirrhosis-related outcomes, hepatocellular carcinoma, cardiovascular outcomes, or mortality.

Patterns of risk-of-bias are common in lifestyle trials. Blinding both participants and trainers is infrequent, and sham exercise comparators are rare but valuable. Small sample sizes, brief interventions, variable adherence monitoring, and incomplete reporting of allocation concealment limit certainty. Cointerventions also make attribution more complex. For instance, diet advice, Mediterranean diet, supplements, or combined resistance training can amplify or confound the apparent effect of HIIT [24-26]. Standardized definitions of HIIT should be used in future trials, intensity should be reported using objective heart-rate or oxygen-consumption targets, adherence and adverse events should be documented, dietary intake should remain stable when testing exercise-specific effects, and long-term follow-up should be adequate to ascertain persistence of benefit.

The methodological approach specified for this review is aligned with contemporary systematic review standards, including transparent reporting, duplicate screening, duplicate extraction, risk-of-bias domain judgments, certainty grading, random-effects pooling, sensitivity analyses, small-study effect assessment, and reproducible code [36-43]. However, an important distinction must be made between a protocol-ready meta-analysis and a completed pooled estimate. Without extracting numerical arm-level results from each full text, pooled estimates would be unreliable. The most defensible next step is to complete numeric extraction into the supplied template, contact authors for missing SDs or change-score correlations, and then run the supplied metafor script.

Limitations

This review has several limitations. First, the current synthesis was seeded from a draft dataset and publicly verified bibliographic records, but arm-level means, standard deviations, change-score correlations, and event counts were not available in the seed dataset. A definitive quantitative meta-analysis therefore requires full-text extraction before pooled estimates can be claimed. Second, included trials were generally small, short-term, and heterogeneous in diagnostic criteria, HIIT protocol, comparator, supervision, and outcome measurement. Third, participant blinding was not feasible, assessor blinding was inconsistently reported, and selective reporting could not be excluded without trial protocols for all studies. Fourth, some studies enrolled metabolically high-risk populations such as type 2 diabetes or obesity rather than biopsy-confirmed NASH, limiting directness for advanced disease. Fifth, liver enzymes and non-invasive steatosis indices are imperfect surrogates, while histological and long-term clinical outcomes were rarely reported. Finally, cointerventions such as diet, resistance training, or supplements complicate isolation of the independent HIIT effect.

CONCLUSION

HIIT appears to be a feasible and time-efficient exercise strategy that can reduce hepatic steatosis and improve selected liver enzyme, insulin-resistance, and fitness outcomes in adults with NAFLD/MASLD. The strongest evidence supports short-term improvements in liver fat and cardiometabolic function, often without substantial weight loss. Evidence for fibrosis regression, histological improvement, and long-term clinical benefit remains limited. Standardized, adequately powered trials with harmonized imaging, fibrosis, adherence, safety, and durability outcomes are required before HIIT can be ranked definitively against moderate-intensity continuous or resistance training.

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