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DOES EXERCISE CHANGE LIVER FIBROSIS IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE? A CRITICAL NARRATIVE REVIEW OF RANDOMISED AND HISTOLOGICAL EVIDENCE

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Abstract

Background: Fibrosis stage predicts liver-related outcomes in metabolic dysfunction-associated steatotic liver disease (MASLD). Exercise lowers liver fat, but whether it changes fibrosis is unclear. Objective: To appraise the randomised and histological evidence on exercise and liver fibrosis, liver stiffness and fibrosis biomarkers in adults with MASLD, to explain why that evidence is inconclusive, and to propose a research agenda. Methods: Critical narrative review following the SANRA criteria. PubMed and Consensus were searched on 27 October 2026 for randomised trials, systematic reviews and meta-analyses. Results were mainly taken from abstracts and described as reported, without pooling. Findings: A meta-analysis of three controlled trials (72 participants) found no significant improvement in histological fibrosis, and a 2026 meta-analysis of three trials found no significant change in liver stiffness (standardised mean difference −0.60, 95% CI −1.37 to 0.17). A 24-patient biopsy trial reported fibrosis improvement by one stage in 58% of the exercise group, and trials of 16 to 52 patients reported lower stiffness after resistance training or walking with electrical stimulation, but their designs cannot separate exercise from other changes. A second 2026 meta-analysis reported lower FIB-4 and NAFLD fibrosis scores with exercise, although both scores respond to enzymes and weight. In multicomponent programmes stiffness also fell with diet alone. Exercise raised the odds of a 30% or greater relative reduction in MRI liver fat (odds ratio 3.51, 95% CI 1.49 to 8.23). In a lifestyle cohort, fibrosis regressed in 45% of patients who lost 10% or more of body weight. Neither finding shows that exercise regresses fibrosis. The trials reviewed lasted 8 weeks to six months, and most enrolled 144 or fewer participants. Conclusions: Current evidence neither shows nor excludes an effect of exercise on fibrosis in MASLD. Informative trials need fibrosis as a main outcome, at least 52 weeks of follow-up, measurable baseline fibrosis, a non-exercise control with matched dietary advice, and reporting of exercise dose and of responders.

Keywords: *Metabolic dysfunction-associated steatotic liver disease; exercise; liver fibrosis; liver stiffness; elastography; narrative review*

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1. Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the term that replaced non-alcoholic fatty liver disease (NAFLD) in 2023 [1]. Exercise is part of the lifestyle advice in the 2024 joint European guideline on MASLD [2]. Most of the evidence behind that advice concerns liver fat and cardiometabolic risk factors.

For patients, the outcome that matters most is fibrosis. In a meta-analysis of 13 cohort studies with 4,428 patients with NAFLD, risk rose with each histological stage. Compared with stage 0, stage 4 carried a relative risk of 3.42 (95% CI 2.63 to 4.46) for all-cause mortality, 11.13 (95% CI 4.15 to 29.84) for liver-related mortality and 12.78 (95% CI 6.85 to 23.85) for liver-related events [3]. The same authors noted that change in fibrosis stage has not yet been established as a valid trial endpoint [3]. Regression of fibrosis is therefore desirable, but its value as a surrogate remains under study.

Lifestyle change can regress fibrosis. In a prospective cohort of 293 patients with biopsy-proven steatohepatitis followed for 52 weeks, fibrosis regressed in 19%, and in 45% of those who lost at least 10% of body weight [4]. Exercise reduces liver fat independently of weight loss: in a meta-analysis of 14 trials, exercise training raised the odds of a 30% or greater relative reduction in MRI-measured liver fat (odds ratio 3.51, 95% CI 1.49 to 8.23), regardless of whether participants lost more than 5% of body weight [5].

Whether exercise itself changes fibrosis is less clear. A 2023 meta-analysis of three controlled trials with histology found no significant improvement in fibrosis [6], although a 12-week aerobic trial in 24 patients with biopsy-confirmed MAFLD reported fibrosis improvement by one stage in 58% of the exercise group [23]. A 2022 systematic review reported that serum biomarkers of fibrosis did not improve with exercise, although imaging measures of liver fat did [7]. A 2026 meta-analysis found no significant change in liver stiffness, with very few studies contributing [8], while a second 2026 meta-analysis reported lower FIB-4 and NAFLD fibrosis scores with exercise [24]. The syntheses pool different outcomes, and those on histology and stiffness rest on very few trials.

This review has three aims: (1) to describe the randomised and histological evidence on exercise and fibrosis, liver stiffness and fibrosis biomarkers in adults with MASLD; (2) to identify the design features that limit what these studies can show; and (3) to propose a research agenda for trials that can answer the question. It addresses fibrosis outcomes only and does not review the effect of exercise on liver fat or aminotransferases.

2. Review approach

This is a critical narrative review, written to the Scale for the Assessment of Narrative Review Articles (SANRA) criteria. It is not a systematic review, it was not registered, and it does not pool data.

PubMed was searched on 27 October 2026 with three strings (Appendix). A fourth PubMed search restricted to 2025 onward and queries in the Consensus semantic-search index were used to find recent trials. The first combined exercise terms, fatty liver terms, fibrosis or stiffness terms and the publication type randomised controlled trial, and returned 45 records. The second used the same concepts restricted to systematic reviews and meta-analyses and returned 14

records. The third restricted exercise and fatty liver terms to the title and returned 11 records. Further records came from citation chasing of recent syntheses and from separate searches for background on prognosis, weight loss and non-invasive tests.

Studies were included when they enrolled adults with MASLD or NAFLD, tested exercise as a main intervention or allowed an exercise arm to be separated from other components, and reported histology, liver stiffness, a fibrosis score or a serum fibrosis biomarker. Systematic reviews of the same question and cohort studies needed for background were also included. Pharmacological and supplement trials in which exercise was only background advice were excluded. Searches used English-language terms only.

Results are described as the original authors reported them. No pooled estimates were calculated and no formal certainty rating was applied. A finding is described as not shown when a study reported no significant difference; this is not read as evidence of no effect.

3. Why fibrosis matters, and how it is measured

Histology is the reference standard for fibrosis stage. Biopsy is invasive, and few exercise trials have repeated it. Most trials therefore rely on non-invasive measures, which fall into three groups. Vibration-controlled transient elastography (VCTE) reports liver stiffness in kilopascals (kPa). Serum scores combine routine laboratory values with patient characteristics; the Fibrosis-4 index (FIB-4) and the NAFLD fibrosis score are examples. Serum biomarkers of hepatocyte injury, such as cytokeratin-18 (CK18), reflect disease activity more than fibrosis stage. The 2021 European guideline on non-invasive tests describes their use for assessing severity and prognosis across liver diseases [9].

Three properties of these measures matter when they are used as trial outcomes. First, they are used mainly to classify a patient at one time point, and how well a change in stiffness or score over weeks to months tracks a change in histological stage is less settled [citation to be added]. Second, stiffness and serum scores respond to liver fat, hepatic inflammation and body weight, and scores also contain age and metabolic variables, so a change can occur without any change in fibrosis [citation to be added]. Third, imaging measures of liver fat, such as MRI proton density fat fraction, measure steatosis, not fibrosis, and should not be read as fibrosis outcomes.

4. What the evidence shows

The evidence that exercise changes fibrosis is weak. Syntheses find no significant change, and the positive findings come from very small trials or from multicomponent programmes in which exercise cannot be separated from diet. Table 1 lists the studies discussed.

4.1 Histology

Few exercise trials have used paired biopsies. A 2022 systematic review found six studies of exercise alone in biopsy-proven NAFLD. The two randomised trials did not detect significant histological improvement, whereas non-randomised studies reported lower hepatocyte ballooning and fibrosis [7]. A 2023 meta-analysis pooled the three controlled trials (72 participants, 40 in exercise groups and 32 in comparison groups) and found no significant improvement in Brunt grade, NAFLD activity score or fibrosis [6]. With so few participants, this result cannot exclude a moderate effect.

The most direct fibrosis result comes from a controlled trial of 12 weeks of aerobic exercise in 24 patients with biopsy-confirmed MAFLD (16 exercise, 8 control). Fibrosis improved by one stage in 58% of the exercise group ($P = 0.034$) and ballooning in 67% ($P = 0.020$), with no change in steatosis or NAFLD activity score, and no patient reached 7% to 10% weight loss [23]. The abstract does not give the fibrosis result in the control group or a between-group test, and the trial is small [VERIFY against the full text].

Trials that combine exercise with diet show histological gains that cannot be assigned to exercise. In a trial of 60 patients with biopsy-proven NAFLD, both arms followed a hypocaloric diet with daily aerobic exercise for six months. Fibrosis improved within each arm ($P < 0.01$), and the added supplement did not change fibrosis significantly against control ($P = 0.07$) [10]. A 2022

systematic review of aerobic exercise (24 studies, 1,014 patients) meta-analysed ALT, AST, liver fat by magnetic resonance spectroscopy and fitness, and concluded that further studies were needed on hepatic inflammation and fibrosis [11].

4.2 Liver stiffness

A 2026 meta-analysis of randomised trials of at least 8 weeks in NAFLD found significant reductions in ALT, AST and liver fat after exercise, but not in liver stiffness. Stiffness was pooled from three trials (standardised mean difference −0.60, 95% CI −1.37 to 0.17; I^2 66%) [8]. The interval includes both a moderate reduction and a small increase, so the result is imprecise rather than negative. The meta-analysis excluded patients with steatohepatitis and trials that combined exercise with diet [8]. Its abstract gives the number of stiffness studies as three in one place and four in another; the full text reports three.

Individual trials point in different directions. A study of 16 patients with NAFLD, described by its authors as quasi-experimental, reported that functional resistance training lowered stiffness (P = 0.01) and steatosis (P = 0.04); each group had eight patients [12]. In 32 patients with NAFLD, 12 weeks of walking with or without simultaneous electrical stimulation produced a larger reported decrease in stiffness with stimulation in the subgroup with a BMI of at least 27 kg/m^2 (Δ2.13 ± 0.64 kPa versus Δ−0.67 ± 0.42 kPa, P = 0.0009) [13]. Both arms exercised, so this trial does not compare exercise with no exercise. A 24-week trial of supervised aerobic exercise in 40 postmenopausal women with biopsy-proven NAFLD measured stiffness by FibroScan and reported no significant fall in controlled attenuation parameter; its abstract does not state the stiffness result [14] [VERIFY]. In a 12-week trial of aerobic versus resistance exercise with dietary counselling in 35 patients, liver stiffness did not change (time P = 0.733; group-by-time P = 0.967). Baseline stiffness was 4.5 and 4.3 kPa, the authors state that all participants had no or mild fibrosis at enrolment, the primary outcome was liver fat, and there was no non-exercise control [15].

An 8-week trial of supervised elastic-band resistance training in 52 postmenopausal women with MASLD reported a lower stiffness than control (group-by-time β −1.51 kPa). Stiffness was reported alongside the primary steatosis outcome, and the authors caution that secondary outcomes were not adjusted for multiple testing [26].

4.3 Serum scores and biomarkers

Serum measures give mixed results. In the NASHFit trial in patients with steatohepatitis, 20 weeks of moderate-intensity aerobic exercise, with Mediterranean-informed dietary counselling in both arms, was compared with standard care. A reduction in ALT of at least 17 IU/L occurred in 53% of the exercise group and 13% of the comparison group (P < 0.001), and CK18 fell more with exercise (−61 versus +71 ng/mL, P = 0.040) [16]. These were post hoc analyses of biomarkers the authors described as markers of liver fibroinflammation, and participants did not lose significant body weight [16]. In a 12-week trial of 24 patients with biopsy-proven NASH, supervised cycling and resistance training reduced liver fat but had no effect on circulating markers of inflammation or fibrosis [17]. In an 80-participant trial, alternate-day fasting with aerobic exercise, fasting alone, exercise alone and no intervention did not differ in a liver fibrosis score [18]. The 2022 systematic review likewise reported improvement in MRI-measured steatosis but not in serum biomarkers of steatohepatitis and fibrosis across five studies [7].

The largest trial in this review randomised 314 employees with metabolic syndrome to six months of supervised, activity-tracker-guided exercise or a waiting list. Transaminases fell with exercise, and the aspartate aminotransferase-to-platelet ratio index (APRI), a fibrosis marker, fell significantly more in the intervention group, in step with changes in obesity and exercise capacity [25]. APRI contains AST, which also fell, so the change may reflect less hepatocellular injury more than less fibrosis. The participants had metabolic syndrome and not diagnosed MASLD, and the analysis was secondary [25].

A 2026 meta-analysis of 21 randomised trials, four post hoc analyses and one quasi-

experimental study (1,124 patients) reported lower FIB-4 (mean difference −0.19) and NAFLD fibrosis score (mean difference −0.59) with exercise than with standard therapy [24]. Both scores are calculated from routine laboratory values and age, and the NAFLD fibrosis score also includes body mass index, so their fall can follow lower enzymes or lower body weight (Section 3). The result conflicts with the null serum-biomarker finding of the 2022 review [7], which was restricted to biopsy-proven NAFLD.

4.4 Exercise within multicomponent lifestyle programmes

In multicomponent programmes stiffness often falls, but the fall is not specific to exercise. In a 144-participant trial with six arms over three months, stiffness fell significantly with a low-glycaemic-index Mediterranean diet alone (−2.85 kPa, 95% CI −5.24 to −0.45), with the diet plus an aerobic programme (−2.37 kPa, 95% CI −4.39 to −0.35) and with the diet plus a combined programme (−2.21 kPa, 95% CI −4.10 to −0.32). The aerobic and combined programmes alone also lowered stiffness, without reaching significance [19]. In a six-month trial of 63 patients, a Mediterranean lifestyle intervention that included guidance on physical activity lowered stiffness compared with control after adjustment for weight loss (P = 0.004), and the diet-only arm improved as well (P < 0.001) [20]. A 2025 meta-analysis of 37 randomised trials of the Mediterranean diet and exercise assessed indices or grades of fibrosis among its outcomes and called for standardised methods of measuring and reporting outcomes [21].

In a 228-participant trial of an isocaloric traditional Jiangnan diet with or without moderate-intensity interval exercise over six months, weight loss was similar in all four groups. Only the diet-plus-exercise group showed a larger fall in stiffness (−0.38 kPa, 95% CI −0.41 to −0.36) than the other three groups, which included a control diet with exercise; the primary outcome was weight [27]. A non-randomised, preference-based study of 120 patients with normal-BMI NAFLD reported a larger fall in stiffness with dietary counselling, prescribed aerobic exercise of 150 to 200 minutes per week and weekly follow-up than with standard advice (−0.76 versus −0.12 kPa). The authors name the preference-based allocation as a limitation [28].

Table 1. ***Studies and syntheses on exercise and fibrosis-related outcomes, as reported in their abstracts, except Guo 2026 and Charatcharoenwitthaya 2021 (full texts)***

Study	Design and participants	Exercise	Fibrosis-related outcome	Result as reported	Main limitation
Chen 2023 [6]	Meta-analysis of 3 controlled trials; 72 participants with biopsy-proven NAFLD	Exercise alone	Brunt grade, NAFLD activity score, fibrosis	No significant improvement	Three trials, 72 participants
Guo 2026 [8]	Meta-analysis of 18 studies in NAFLD	Long-term exercise	Liver stiffness	No significant change (SMD −0.60, 95% CI −1.37 to 0.17)	Three stiffness trials; I^2 66%
Houghton 2017 [17]	RCT, 24 patients with biopsy-	Supervised cycling and resistance	Circulating fibrosis markers	No effect	Small trial; markers, not

Study	Design and participants	Exercise	Fibrosis-related outcome	Result as reported	Main limitation
	proven NASH, 12 weeks	training, 3 per week			histology
Harris 2024 [16]	Post hoc analysis of NASHFit RCT, 20 weeks	Moderate-intensity aerobic exercise vs standard care	Serum fibroinflammation biomarkers	ALT fall of 17 IU/L or more in 53% vs 13% ($P < 0.001$); CK18 −61 vs +71 ng/mL ($P = 0.040$)	Post hoc; biomarkers, not histology
Ezpeleta 2023 [18]	RCT, 80 adults with obesity and NAFLD, 3 months	Aerobic exercise 5 × 60 min per week, alone or with fasting	Liver fibrosis score	No difference between groups	Score, not histology
Rezende 2016 [14]	RCT, 40 postmenopausal women, 24 weeks	Supervised aerobic exercise, 120 min per week	Stiffness (FibroScan)	Stiffness result not in abstract [VERIFY]	Abstract incomplete
Jafarikhah 2023 [12]	16 patients (8 vs 8); described as quasi-experimental	Functional resistance training, 3 per week	Stiffness	Lower with training ($P = 0.01$)	Very small; design unclear
Iwanaga 2022 [13]	RCT, 32 patients, 12 weeks	Walking with or without electrical stimulation	Stiffness	Subgroup with BMI of 27 kg/m² or more: Δ2.13 ± 0.64 vs Δ−0.67 ± 0.42 kPa ($P = 0.0009$)	Both arms exercised; subgroup
Charatcharoenwitthaya 2021 [15]	RCT, 35 patients, 12 weeks	Aerobic vs resistance exercise, with dietary counselling	Transient elastography	Stiffness unchanged (time $P = 0.733$; group-by-time $P = 0.967$)	No non-exercise control; no or mild fibrosis at baseline (4.3 to 4.5 kPa)
Curci 2023 [19]	RCT, 144	Aerobic or	Stiffness (kPa)	Significant	Exercise

Study	Design and participants	Exercise	Fibrosis-related outcome	Result as reported	Main limitation
	participants, six arms, 3 months	combined programme, alone or with diet		fall in diet-containing arms; exercise-only arms fell without reaching significance	cannot be separated from diet
Katsagoni 2018 [20]	RCT, 63 patients, 6 months	Mediterranean lifestyle including physical activity	Stiffness	Lower than control (P = 0.004) after adjustment for weight loss; diet-only arm also improved (P < 0.001)	Not exercise-specific
O'Gorman 2020 [23]	Controlled trial, 24 patients with biopsy-confirmed MAFLD (16 exercise, 8 control), 12 weeks	Supervised aerobic exercise	Histological fibrosis stage	Fibrosis improved by one stage in 58% of the exercise group (P = 0.034); NAFLD activity score unchanged	Small; control-group result and between-group test not in abstract [VERIFY]
Haufe 2021 [25]	RCT, 314 employees with metabolic syndrome, 6 months; secondary analysis	Supervised, activity-tracker-guided exercise vs waiting list	APRI	APRI fell more in the exercise group	APRI contains AST, which also fell; MASLD not diagnosed
Jafari 2026 [26]	RCT, 52 postmenopausal women with MASLD, 8 weeks	Supervised elastic-band resistance training vs control	Stiffness (transient elastography)	Group-by-time β −1.51 kPa, favouring training	Short; no multiplicity adjustment
Wu 2026 [27]	RCT, 228 patients with	Moderate-intensity	Stiffness (FibroScan)	Diet-plus-exercise	Primary outcome

Study	Design and participants	Exercise	Fibrosis-related outcome	Result as reported	Main limitation
	NAFLD, four groups, 6 months	interval exercise with a control or Jiangnan diet		group fell more (−0.38 kPa) than the other three groups	was weight; effect only with diet plus exercise
Sharanabasava 2026 [28]	Non-randomised, preference-based study, 120 patients with normal-BMI NAFLD, 6 months	Prescribed aerobic exercise, 150 to 200 min per week, with diet counselling	Stiffness	−0.76 vs −0.12 kPa	Non-randomised; exercise combined with diet counselling
Asif 2026 [24]	Meta-analysis of 21 RCTs, 4 post hoc analyses and 1 quasi-experimental study; 1,124 patients	Exercise vs standard therapy	FIB-4, NAFLD fibrosis score	FIB-4 mean difference −0.19; NAFLD fibrosis score −0.59	Both scores respond to enzymes and weight

5. Weight loss, liver fat and fibrosis

Exercise could act on fibrosis through three routes, and current trials do not separate them. The first is weight loss. In the 52-week lifestyle cohort, fibrosis regressed most often with weight loss of 10% or more, in 45% of those patients [4]. Exercise alone produces smaller weight changes: in a 2025 meta-analysis against control, aerobic exercise lowered body weight by 1.56 kg (95% CI −2.31 to −0.82) and combined aerobic and resistance exercise by 1.90 kg (95% CI −3.59 to −0.22) [21]. An exercise-only trial may therefore not reach the weight loss associated with regression.

The second route is liver fat. Exercise training raised the odds of a 30% or greater relative reduction in MRI liver fat independently of weight loss [5], and the meta-analysis authors describe that threshold as the one associated with histological improvement in NASH activity, NASH resolution and fibrosis stage [5]. This is a surrogate link. A patient can reach the liver-fat threshold without any change in fibrosis, so the finding supports a plausible route and does not show an effect on fibrosis.

The third route is a reduction in hepatic injury and inflammation. The NASHFit biomarker data [16] are the only direct exercise evidence for this route in the studies reviewed here, and they come from a post hoc analysis.

Two further sources link exercise to fibrosis indirectly. A pooled post hoc analysis of three randomised trials (88 adults, 12 to 20 weeks) found that exercise raised the odds of a 30% or greater relative reduction in MRI liver fat (odds ratio 4.86, 95% CI 1.72 to 13.8), with less than 3% weight loss [29]. A post hoc analysis of NASHFit (n = 23) used the MASH resolution index, a composite of MRI liver fat, ALT and AST, and reported MASH resolution in 33% of the exercise group and 13% of the standard-care group [30]. The index contains no fibrosis measure, so it does not address fibrosis.

Cross-sectional data are consistent with a link. In 2,201 adults in a community cohort, each

additional 30 minutes of daily moderate-to-vigorous activity was associated with lower odds of liver stiffness above 8.2 kPa (odds ratio 0.57, 95% CI 0.41 to 0.79) [31]. Such data cannot show that exercise lowers fibrosis, because people with more fibrosis may also exercise less.

6. Possible mechanisms

This section is speculative and rests on small studies. In NASHFit, serum fibroblast growth factor 21 (FGF21) fell by 22% with exercise and rose by 34% with standard care (P = 0.037). The change was inversely related to the change in cardiorespiratory fitness (r = −0.62, 95% CI −0.88 to −0.05) [22]. A fall in hepatocyte injury, suggested by the lower CK18 [16], is a second candidate. Neither has been linked to histological fibrosis: none of the abstracts reviewed here reports a mediator analysis against fibrosis.

7. Why the current evidence cannot answer the question

Six design features limit what the existing trials can show.

1. Fibrosis is rarely the primary outcome. The fasting-and-exercise trial was designed around intrahepatic triglyceride content [18], the aerobic-versus-resistance trial around liver fat [15], and the 228-participant diet-and-exercise trial around weight [27]. The 314-participant trial reported liver outcomes in a secondary analysis [25]. A trial powered for liver fat or weight is not powered to detect a change in stiffness or fibrosis stage.
2. Duration is short. Where abstracts state it, trials lasted 8 weeks to six months [13–20, 23, 25–27], whereas fibrosis regression in the lifestyle cohort was assessed at 52 weeks [4].
3. Samples are small and baseline stage is usually unreported. Most trials in Table 1 enrolled 144 or fewer participants (range 16 to 314). In the one trial that reports it, all participants had no or mild fibrosis, with mean baseline stiffness of 4.3 to 4.5 kPa [15]. Patients with little or no fibrosis cannot show regression, whatever the intervention.
4. Exercise is confounded with diet and weight. Several trials gave dietary counselling to all arms [15, 16], and in the abstracts only one reported adjusting for weight loss [20].
5. Outcomes are noisy and rarely expressed as responders. Stiffness and serum scores vary for reasons unrelated to fibrosis (Section 3), and none of the abstracts in Table 1 reports the proportion of participants whose stiffness changed by more than a predefined threshold [VERIFY against full texts].
6. Exercise doses differ and are not matched. Prescriptions ranged from 90 minutes per week of walking [13] to 300 minutes per week of aerobic exercise [18].

A null result from these trials is therefore uninformative, and a positive result from a 16-patient trial cannot be generalised.

8. Clinical implications

Exercise is justified for patients with MASLD by its effects on liver fat, fitness and cardiometabolic risk. The studies reviewed here do not support telling a patient that exercise alone will regress fibrosis, and they do not support withholding exercise either. Fibrosis assessment should follow the guidance on non-invasive tests [9]. A change in stiffness or score in a patient who has started exercising should be read together with changes in body weight and liver fat (Section 3). The best evidence for fibrosis regression in lifestyle programmes involves large weight loss [4]. An exercise programme that helps a patient reach that weight loss may therefore matter, although no study reviewed here tested this.

9. Research agenda

A trial that can answer the question needs the following.

1. Fibrosis as the primary or a key secondary outcome, measured by paired and centrally read biopsy where feasible, or by a validated non-invasive measure with predefined responder thresholds and quality criteria.
2. Follow-up of at least 52 weeks, the duration of the lifestyle cohort that documented regression [4], plus a later assessment to test durability.
3. Enrolment of patients with enough baseline fibrosis for regression to be measurable, with

baseline stage reported.

4. Randomisation to exercise or to a non-exercise control, with matched dietary advice, recorded diet and weight, and an analysis that adjusts for weight change.
5. Reporting of the exercise dose delivered (minutes, intensity, MET-min per week) and of adherence.
6. Results given as the proportion of participants with a predefined change in the fibrosis measure, alongside the proportion with a 30% or greater relative reduction in MRI liver fat.
7. Mediator measurements, for example FGF21 and CK18, to test the mechanisms in Section 6.

Pooling individual participant data from existing trials could test, before new trials are run, whether change in stiffness tracks change in liver fat after weight change is accounted for. One registered trial, AMPED, plans to compare exercise doses over 16 weeks in 45 adults with steatohepatitis, with liver histology among its secondary outcomes [32].

10. Limitations

This review searched PubMed, supplemented by the Consensus semantic-search index and a web search, with English-language terms. Embase, Scopus, Web of Science and Cochrane CENTRAL were not searched, Google Scholar could not be queried, and selection was not independent or duplicated. As a narrative review it is open to selection bias, and no risk-of-bias tool or certainty rating was applied. Most trial results were taken from abstracts, one of which does not report the stiffness result [14]; full texts were read for only two papers [8, 15]. The search for 2025-onward trials was only partly screened. The abstract of the 2026 meta-analysis of liver stiffness gives the number of contributing studies as both three and four [8]. The included studies used NAFLD or MASLD definitions that differ between trials, enrolled small samples, and measured fibrosis in different ways, which prevents any pooled estimate. Studies published after 27 October 2026 are not covered.

11. Conclusions

Current evidence neither shows nor excludes an effect of exercise on liver fibrosis in MASLD. Meta-analyses of histology and of liver stiffness found no significant change, but each rests on three small trials, and a 2026 meta-analysis of serum fibrosis scores found a reduction. Positive reports come from trials of 16 to 52 patients, from a 24-patient biopsy trial in which fibrosis improved in 58% of the exercise group, from a larger trial in which a fibrosis marker that contains AST fell, and from multicomponent programmes in which diet explains at least part of the effect. Exercise raised the odds of a 30% or greater relative fall in MRI liver fat in one meta-analysis, and weight loss of 10% or more was associated with fibrosis regression in one lifestyle cohort. Neither finding shows that exercise regresses fibrosis. Trials that can answer the question need fibrosis as a main outcome, at least 52 weeks of follow-up, measurable baseline fibrosis, a non-exercise control with matched diet advice, and reporting of exercise dose and of responders.

Declarations

Ethics approval and consent: not applicable; this review synthesises published studies.
Funding: no specific funding was received.
Competing interests: none declared.
Author contributions: ……..
Registration: this narrative review was not registered.
Data availability: no new data were generated. The search strings are given in Appendix A.
Related work: a separate review by the author on combined aerobic and resistance exercise, hepatic steatosis and liver enzymes in MASLD is in preparation. It addresses different outcomes. [Author to confirm its status and cite it if it has been submitted.]
AI-assisted writing: an AI language model (Claude, Anthropic) assisted with retrieving PubMed records, structuring the manuscript and drafting text. The author reviewed the content and the

references and accepts responsibility for the manuscript.

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Each entry was matched to a PubMed record on 27 October 2026. Matching a record does not verify the claim attached to it; the author should read each full text.

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