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Exercise Prescription in Metabolic Dysfunction – Associated Steatotic Liver Disease: Aerobic Training, Resistance Exercise and High-Intensity Interval Training – A Narrative Review

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Abstract

Background: Exercise is a first-line component of care for metabolic dysfunction-associated steatotic liver disease (MASLD), but modality-specific prescriptions remain uncertain.

Aim: To compare evidence for aerobic training, resistance exercise and high-intensity interval training (HIIT), and provide a phenotype-oriented framework for frequency, intensity, time, type, safety and monitoring.

Material and methods: A structured, PubMed-focused narrative search was conducted from database inception to 8 June 2026 using prespecified disease and exercise terms; reference lists of major guidelines, reviews and trials were searched. Adult human guidelines, evidence syntheses and intervention studies were assessed by one reviewer using SANRA-informed narrative methods.

Results: Exercise reduces liver fat and improves cardiorespiratory fitness and several cardiometabolic outcomes, often without clinically significant weight loss. Aerobic and resistance training are both effective; HIIT is time-efficient and generally comparable, not clearly superior, to moderate-intensity continuous training. Evidence for fibrosis regression, histological MASH resolution and long-term clinical events remains limited.

Conclusions: A patient-centered program should usually combine ≥ 150 min/week of moderate aerobic activity with resistance training on 2-3 non-consecutive days. HIIT is an optional, progressively introduced alternative for appropriately screened patients. Adherence, comorbidity, functional capacity and safety should determine modality selection.

Keywords: MASLD, MASH, exercise prescription, aerobic exercise, resistance training, high-intensity interval training, hepatic steatosis

1. INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the current name for hepatic steatosis occurring in the presence of at least one cardiometabolic risk factor and without a dominant alternative cause. The 2023 multisociety nomenclature replaced non-alcoholic fatty liver disease (NAFLD) and renamed non-alcoholic steatohepatitis as metabolic dysfunction-associated steatohepatitis (MASH) [1]. Because most exercise trials were

conducted before this change, the terms NAFLD and NASH are retained when describing the populations and wording of original studies; MASLD and MASH are used for contemporary interpretation.

MASLD is a multisystem disorder rather than an isolated imaging finding. Insulin resistance, adipose-tissue dysfunction, dyslipidemia, type 2 diabetes, obesity, low cardiorespiratory fitness and loss of skeletal-muscle quality interact with hepatic lipid accumulation and inflammatory signaling. Fibrosis stage is the principal liver-related prognostic marker, while cardiovascular disease remains a major competing source of morbidity and mortality. Objectively measured physical activity has been associated with lower mortality in people with fatty liver disease, although observational data cannot prove a treatment effect [2]. Contemporary European and American guidance therefore places lifestyle treatment, including regular exercise, at the foundation of management [3,4].

Exercise deserves separate consideration from weight loss. Randomized trials and meta-analyses show reductions in intrahepatic lipid even when body mass changes little [5–7]. This distinction is clinically relevant because many patients struggle to achieve or maintain substantial weight loss, whereas improvements in fitness, glucose disposal, blood pressure, muscle function and liver fat can occur with repeated training. Exercise should nevertheless complement, not replace, dietary quality, smoking cessation, sleep management, alcohol risk assessment, treatment of metabolic comorbidities and pharmacological or surgical therapy when indicated [3,4,8].

Guidelines and previous reviews consistently support exercise in MASLD, but they do not always translate the evidence into modality selection, progression, safety and monitoring for distinct clinical phenotypes. This review compares aerobic training, resistance exercise and HIIT across hepatic and extrahepatic outcomes. Its distinctive contribution is an integrated, phenotype-oriented frequency-intensity-time-type (FITT) framework that separates direct MASLD evidence from recommendations extrapolated from general exercise medicine and guidance for common comorbidities.

2. REVIEW METHODS

A narrative review with a structured literature search was undertaken. PubMed/MEDLINE and PubMed Central were searched from database inception to 8 June 2026. Search concepts were combined using Boolean operators and included: “metabolic dysfunction-associated steatotic liver disease”, MASLD, MAFLD, “nonalcoholic fatty liver disease”, NAFLD, MASH, NASH, exercise, physical activity, aerobic, endurance, resistance, strength, high-intensity interval

training, HIIT, interval training, liver fat, MRI-PDFF, magnetic resonance spectroscopy, fibrosis and histology. Reference lists of guidelines, position statements and major systematic reviews were hand-searched for additional primary studies.

Priority was given to: (1) international clinical practice guidance; (2) systematic reviews and meta-analyses; (3) randomized or controlled adult human exercise trials; and (4) mechanistic human studies that clarified weight-independent effects. Full-text open-access articles, PubMed Central records.

The historical NAFLD/NASH terminology of included studies may create imperfect overlap with the contemporary MASLD/MASH case definitions.

3. RESULTS AND DISCUSSION

3.1. Guideline position and therapeutic targets

The 2024 EASL–EASD–EASO guideline recommends physical activity and exercise tailored to patient preference and ability, preferably exceeding 150 minutes per week of moderate intensity or 75 minutes per week of vigorous intensity [3]. The AASLD guidance similarly emphasizes that exercise provides hepatic and cardiometabolic benefit independent of weight loss and should be routinely recommended according to patient ability [4]. Exercise and Sport Science Australia proposes an individualized program that includes aerobic and resistance components, with progression according to baseline fitness, comorbidity and tolerance [8]. An American College of Sports Medicine roundtable likewise supports structured physical activity as part of fatty-liver care [10].

A clinically useful hierarchy of targets is needed. The strongest evidence supports improvement in liver fat measured by magnetic resonance imaging or spectroscopy, followed by gains in cardiorespiratory fitness, insulin sensitivity, visceral adiposity and selected biochemical markers. Alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT) may improve but are neither sensitive nor specific measures of steatosis or fibrosis. Evidence is substantially weaker for histological MASH resolution, fibrosis regression, prevention of cirrhosis, cardiovascular events and mortality [3,11,12]. Consequently, a normal transaminase result should not be used to conclude that an exercise program has failed or that MASLD is absent.

Magnetic resonance proton-density fat fraction (MRI-PDFF) and proton magnetic resonance spectroscopy are accurate research outcomes, whereas routine clinical monitoring usually depends on anthropometry, metabolic risk markers, liver biochemistry and guideline-directed non-invasive fibrosis assessment [13]. A relative MRI-PDFF reduction of at least 30% is

associated with greater odds of histological activity improvement and, in some datasets, fibrosis regression [14,15], but it remains a surrogate rather than proof that an individual patient has achieved fibrosis regression. Exercise studies should therefore distinguish reductions in steatosis from improvement in steatohepatitis or fibrosis.

3.2. Overall efficacy of structured exercise

The most clinically interpretable synthesis is the 2023 meta-analysis by Stine et al., which included 14 randomized controlled trials and 551 participants. Exercise training increased the odds of achieving a clinically meaningful $\geq 30\%$ relative reduction in MRI-measured liver fat (odds ratio 3.51) independently of clinically significant body-weight loss. A dose at or above approximately 750 metabolic equivalent minutes per week was associated with treatment response [5]. This dose is close to 150 minutes of moderate activity at 5 METs, providing a practical bridge between trial evidence and public-health recommendations.

Other meta-analyses generally support reductions in intrahepatic lipid and improvements in insulin resistance or liver enzymes, but effect estimates vary with imaging method, population, intervention duration, co-interventions and control condition [6,7,11,16–19]. Network meta-analysis has sometimes ranked combined aerobic and resistance training highly [16], yet such rankings rely partly on indirect comparisons and heterogeneous protocols. They should be used to support a balanced program, not to claim a proven best modality.

Table 1 summarizes representative trials selected for their direct relevance to exercise prescription. Most were small, lasted 8-20 weeks, used supervised training and enrolled adults with overweight or obesity. Several used older diagnostic labels, and only a minority included biopsy-confirmed steatohepatitis or histological outcomes. These features increase internal control but limit generalizability to long-term, unsupervised clinical care.

3.2.1. Interpreting outcome-specific effects

Hepatic steatosis is the outcome most likely to respond within an 8–16-week exercise block. Magnetic resonance methods can detect changes that ultrasound may miss, and improvement may occur despite stable weight or transaminases. Absolute percentage-point change, relative change and standardized effect size should not be conflated. A $\geq 30\%$ relative MRI-PDFF reduction is clinically informative but is not a binary definition of individual treatment success [5,20].

Liver biochemistry is an accessible but nonspecific secondary outcome. ALT may decline, particularly when elevated at baseline, but normal values are common in clinically important

MASLD. Improvements in composite fibroinflammatory biomarkers do not demonstrate histological fibrosis regression [21]. Recent strenuous exercise can transiently increase AST or creatine kinase; standardizing the interval between the last strenuous session and phlebotomy improves interpretability.

Cardiorespiratory fitness and strength should be treated as therapeutic outcomes rather than merely adherence checks. An intervention may fail to reduce MRI liver fat yet still increase exercise capacity, insulin sensitivity and functional reserve, as illustrated by the small HIIT study in biopsy-proven NASH [22]. Conversely, a decrease in liver fat without a measurable training adaptation raises questions about dietary change, regression to the mean, activity outside the protocol or insufficient sensitivity of the fitness test. Measuring at least one functional outcome helps determine whether the prescribed stimulus produced the intended physiological adaptation.

Stable body mass can coexist with reduced visceral adiposity and increased lean tissue, especially during resistance training or concurrent weight-loss therapy. Response should therefore include functional and metabolic outcomes rather than body mass alone. Claims of weight-independent benefit should nevertheless be supported by measured weight and, where possible, body composition.

Table 1. Representative adult exercise trials relevant to MASLD prescription

Study	Population / design	Exercise prescription	Main findings	Key limitation
Hallsworth et al., 2011 [23]	NAFLD; randomized controlled trial	Resistance exercise, 3 sessions/week for 8 weeks	Reduced liver fat without weight loss; improved glucose control	Small sample; short duration
Bacchi et al., 2013 [24]	T2D with NAFLD; randomized aerobic vs resistance trial	60 min, 3 sessions/week for 4 months	Both modalities reduced hepatic fat; neither clearly superior	No non-exercise control for modality comparison

Study	Population / design	Exercise prescription	Main findings	Key limitation
Zelber-Sagi et al., 2014 [25]	NAFLD; randomized clinical trial	Whole-body resistance training, 3 sessions/week for 3 months	Improved ultrasound-defined steatosis and body composition	Ultrasound outcome; modest sample
Keating et al., 2015 [26]	Inactive adults with overweight/obesity; four-arm randomized trial	Three aerobic doses/intensities for 8 weeks	All exercise groups reduced liver fat and visceral adiposity; no clear dose superiority	Not all participants had clinical MASLD diagnosis
Shamsoddini et al., 2015 [27]	Men with NAFLD; aerobic vs resistance vs control	45 min, 3 sessions/week for 8 weeks	Both exercise groups improved liver enzymes and ultrasound steatosis	n=30; men only; ultrasound
Hallsworth et al., 2015 [28]	NAFLD; randomized controlled trial	Modified HIIT, 3 sessions/week for 12 weeks	Reduced liver fat and improved selected cardiac/body-composition measures	Small supervised trial
Zhang et al., 2016 [29]	220 adults with central obesity and NAFLD; 12-month randomized trial	Vigorous–moderate vs moderate aerobic exercise	Both reduced intrahepatic triglyceride; no durable clear advantage of higher intensity	Effects attenuated after adjustment for weight change

Study	Population / design	Exercise prescription	Main findings	Key limitation
Cuthbertson et al., 2016 [30]	NAFLD; supervised exercise vs counselling	Aerobic exercise for 16 weeks	Reduced liver fat and improved peripheral insulin sensitivity/fitness	Hepatic insulin sensitivity response was not uniform
Houghton et al., 2017 [31]	Biopsy-proven NASH; randomized controlled trial	Aerobic plus resistance exercise for 12 weeks	Reduced liver triglyceride and visceral adiposity without weight loss	No clear change in circulating inflammation/fibrosis markers
Oh et al., 2017 [32]	Sedentary obese men with NAFLD; three exercise groups	Resistance, moderate continuous or high-intensity aerobic training for 12 weeks	Liver fat fell in all groups; stiffness decreased only in the high-intensity arm	No non-exercise control; male-only; non-histological stiffness
Winn et al., 2018 [33]	NAFLD risk; energy-matched randomized trial	Moderate vs high-intensity aerobic training	Both improved liver fat-related risk independent of body mass change	Modest sample; surrogate outcomes
Abdelbasset et al., 2019 [34]	Obese adults with T2D and NAFLD; randomized trial	HIIT, 3 sessions/week for 8 weeks	Reduced intrahepatic triglyceride and visceral fat; improved quality of life	n=32; short duration

Study	Population / design	Exercise prescription	Main findings	Key limitation
Charatcharoenwitthaya et al., 2021 [35]	NAFLD; randomized aerobic vs resistance plus dietary modification	Supervised training for 12 weeks	Similar hepatic-fat reduction with aerobic and resistance training	Dietary co-intervention; no inactive arm
Stine et al., 2022 (NASHFit) [20]	Biopsy-confirmed NASH; randomized controlled trial	150 min/week moderate aerobic training for 20 weeks	MRI-PDFF −4.7% vs +1.2% absolute; fitness and thrombotic biomarker improved	Small trial; primary endpoint was PAI-1, not histology
Keating et al., 2023 [22]	Biopsy-proven NASH; HIIT vs sham exercise	4 × 4 min at 85–95% HRmax, 3 sessions/week for 12 weeks	Feasible under supervision; improved exercise capacity and peripheral insulin sensitivity	n=14; underpowered for liver outcomes
Houttu et al., 2025 [36]	MASLD/MASH; prospective HIIT cohort with comparator data	HIIT plus home training for 12 weeks	Improved fitness and muscle/adipose metabolism; no improvement in histology, MRI liver fat or liver tests	Small, non-randomized mechanistic study

Abbreviations: HIIT, high-intensity interval training; HRmax, maximal heart rate; MRI-PDFF, magnetic resonance imaging proton-density fat fraction; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; PAI-1, plasminogen activator inhibitor-1; T2D, type 2 diabetes.

3.3. Aerobic training

Aerobic exercise uses large muscle groups rhythmically and continuously. Brisk walking, cycling, elliptical exercise, swimming and water-based exercise are common options. It has the broadest evidence base in MASLD and directly improves cardiorespiratory fitness, an outcome with importance beyond the liver. Moderate-intensity continuous training (MICT) is the most transferable starting modality because intensity can be monitored with heart-rate reserve, percentage of measured peak oxygen uptake, the talk test or rating of perceived exertion (RPE) [37].

Dose-response evidence does not establish a single hepatic threshold. In the eight-week trial by Keating et al., low-to-moderate and higher-volume or higher-intensity aerobic prescriptions all reduced liver fat, with no significant difference between exercise doses [26]. The 12-month trial by Zhang et al. likewise found that vigorous–moderate and moderate programs reduced intrahepatic triglyceride compared with control, but did not demonstrate a consistent advantage of vigorous intensity, and some effects were mediated by weight change [29]. These studies argue against withholding exercise from patients unable to train vigorously.

A practical aerobic target is at least 150 minutes per week of moderate intensity, accumulated across 3–5 days, or an equivalent vigorous dose in suitable patients [3,4,8,37]. For a sedentary patient, starting with 10–20 minute bouts and building total weekly time may be more successful than prescribing the full target immediately. Bouts can be divided throughout the day; the most useful pattern is the one the patient can repeat. Additional volume up to 200–300 minutes per week may facilitate weight management and broader cardiometabolic benefit, provided recovery and musculoskeletal tolerance remain acceptable [37].

Aerobic exercise also illustrates why liver fat and systemic metabolism should be evaluated separately. Cuthbertson et al. found a reduction in liver fat with improved peripheral insulin sensitivity and fitness, while hepatic glucose homeostasis did not change uniformly [30]. Shojaee-Moradie et al. linked exercise-induced liver-fat reduction to faster VLDL1-triglyceride and apolipoprotein B clearance rather than lower VLDL production [38]. These observations support multiple, partially independent pathways rather than a single weight-loss mechanism.

3.4. Resistance exercise

Resistance exercise consists of repeated muscular contractions against external load using machines, free weights, elastic bands or body weight. It is particularly relevant in MASLD because low muscle mass and poor muscle quality worsen insulin resistance and functional

vulnerability. Resistance training may also be more acceptable to patients who cannot sustain prolonged walking or cycling because of low fitness, obesity-related dyspnea, joint pain or time constraints.

The landmark trial by Hallsworth et al. demonstrated that resistance exercise reduced liver fat despite no weight loss [23]. Bacchi et al. subsequently showed comparable hepatic-fat reductions with aerobic and resistance training in people with type 2 diabetes and NAFLD [24]. Shamsoddini et al. and Charatcharoenwitthaya et al. also reported broadly similar improvements across modalities, although small samples, ultrasound outcomes and dietary co-intervention reduce certainty [27,35]. A systematic review found that resistance protocols commonly used approximately 45-minute sessions three times weekly for 12 weeks and required less energy expenditure than aerobic protocols [39]. The 2025 systematic review by Medeiros et al. supports benefit but emphasizes the limited number and heterogeneity of resistance-only trials [40].

A reasonable initial prescription is whole-body training on 2-3 non-consecutive days per week. Eight to ten exercises should cover major movement patterns: knee-dominant and hip-dominant lower-body work, horizontal and vertical pushing and pulling, calf work, trunk stabilization and task-specific balance or power when appropriate. Beginners can perform one set of 10-15 repetitions at light-to-moderate effort; progression to 2–3 sets of 8-12 repetitions is suitable for many adults [37]. Technique, range of motion and symptom-free control take precedence over load. Increasing repetitions first and resistance second is a pragmatic progression strategy for deconditioned patients rather than a liver-specific validated rule.

Resistance exercise provides strength, functional and lean-mass benefits in addition to possible liver-fat reduction. In adults with overweight, aerobic and combined training reduced visceral and liver fat more consistently than resistance training alone [41], but MASLD-specific evidence remains insufficient to establish hepatic superiority. When feasible, combined aerobic and resistance exercise best covers overall clinical objectives [8,16].

3.5. High-intensity interval training

HIIT alternates brief vigorous work intervals with active or passive recovery. Protocols differ markedly: some use four 4-minute intervals at 85–95% of measured or estimated maximal heart rate; others use shorter 30-60 second intervals, lower-volume sessions or modified intensities. The label “HIIT” therefore does not identify a uniform dose. Reporting work intensity, recovery intensity, interval number, duration, exercise mode and progression is essential.

The 2015 randomized trial by Hallsworth et al. showed that modified HIIT reduced liver fat in adults with NAFLD [28]. Oh et al. observed similar liver-fat reductions after resistance, moderate continuous and high-intensity aerobic training, with liver-stiffness improvement confined to the high-intensity group; the absence of an inactive control and reliance on non-invasive stiffness prevent a claim of fibrosis regression [32]. In energy-matched comparisons, high and moderate intensity have generally produced comparable hepatic benefit [33,42].

The 2022 meta-analysis by Sabag et al. found no meaningful difference between HIIT and MICT for liver fat. Both modalities reduced liver fat compared with inactive control, but certainty was lower for HIIT because studies were small and heterogeneous [43]. A 2025 meta-analysis of randomized trials reported favorable effects of HIIT on several clinical indicators, particularly in shorter interventions, but did not establish superiority across robust liver endpoints [44]. The appropriate conclusion is that HIIT is an effective option, not a universally superior prescription.

Safety and feasibility data are reassuring only within context. Keating et al. tested a progressively introduced, supervised 4 × 4-minute protocol in 14 adults with biopsy-proven NASH. No HIIT-related adverse events were reported, global adherence among completers was high, and exercise capacity and peripheral insulin sensitivity improved; however, the trial was underpowered for hepatic outcomes [22]. In contrast, the 2025 mechanistic study by Houttu et al. found improved fitness and peripheral metabolic signatures but no improvement in histology, MRI liver fat or liver biochemistry after 12 weeks [36]. This negative hepatic finding is important and argues against extrapolating improvements in fitness to MASH resolution.

HIIT can be considered after a period of tolerated moderate aerobic conditioning and when patient preference favors shorter, challenging sessions. Preparticipation screening should consider current activity, exertional symptoms, known cardiovascular, metabolic or renal disease, and the intended exercise intensity [45]. HIIT is less suitable as an unsupervised first prescription for someone with unstable symptoms or disease, severe deconditioning, uncontrolled hypertension, proliferative retinopathy at risk from marked pressure responses, significant autonomic neuropathy, acute musculoskeletal injury or another condition requiring stabilization [45-47]. Supervision by an exercise professional is advisable when risk or uncertainty is high. Clinical HIIT guidance supports combining heart rate, RPE, symptoms and appropriate monitoring rather than relying on a single predicted heart-rate target [48].

3.6. Comparative effectiveness and modality selection

Direct comparisons do not identify a consistent hepatic winner. Aerobic training has the largest evidence base and most directly improves cardiorespiratory fitness; resistance training targets strength and lean mass with a lower cardiorespiratory demand; and HIIT may improve fitness efficiently but requires consideration of total session time, tolerability and supervision. Apparent advantages of combined training in network analyses remain uncertain because of indirect evidence [16,39,40,43].

Table 2. Evidence-informed comparison of exercise modalities in MASLD

Domain	Aerobic training	Resistance exercise	HIIT
Liver fat	Consistent reduction; largest evidence base	Reduction in several small trials; broadly comparable to aerobic training	Reduction versus inactive control; generally comparable to MICT
Fitness / function	Strong improvement in cardiorespiratory fitness	Strength, function and lean-mass preservation	Often efficient improvement in exercise capacity/fitness
Typical entry prescription	3–5 days/week; 20–40 min/session at moderate intensity [37]	2–3 non-consecutive days/week; 8–10 whole-body exercises [37]	1 session/week after tolerated conditioning; progress toward 2–3 if appropriate [45,48]
Best fit	Most patients; walking/cycling accessible	Low aerobic capacity, sarcopenia risk, joint-friendly alternatives	Time preference, adequate fitness, stable comorbidity, desire for vigorous work
Main caveat	Time and weight-bearing tolerance	Technique, load progression and limited liver-specific trial base	Heterogeneous protocols; no proven superiority; higher screening/supervision needs

Domain	Aerobic training	Resistance exercise	HIIT
Evidence base for fibrosis and histology	Limited; few small studies	Very limited; no robust biopsy evidence	Very limited; no robust biopsy evidence

HIIT, high-intensity interval training; MICT, moderate-intensity continuous training. Limited and very limited are narrative descriptions of evidence volume, design, directness, consistency and endpoint validity; they are not GRADE ratings.

Modality selection should follow shared decision-making rather than a hierarchy of novelty. The preferred program is the safest combination that addresses the patient's hepatic and extrahepatic priorities and can be repeated. Mode may change over time without abandoning the therapeutic target; phenotype-oriented examples are provided in Section 3.11.1.

3.7. Weight-independent mechanisms

Exercise can lower intrahepatic triglyceride without clinically significant weight loss through several interacting mechanisms [38,49,50]. Repeated muscular contraction increases glucose uptake and improves skeletal-muscle insulin signaling, thereby reducing the substrate pressure that favors hepatic de novo lipogenesis. Improved adipose-tissue insulin sensitivity can suppress inappropriate lipolysis and reduce delivery of non-esterified fatty acids to the liver. Increased mitochondrial oxidative capacity and whole-body fat oxidation may redirect lipid away from hepatic storage, while reduced visceral adiposity can occur despite little change on the scale.

Exercise also affects hepatic lipid export and clearance. The study by Shojaee-Moradie et al. found increased clearance of large VLDL particles after training without a reduction in VLDL production [38]. Myokine signaling, endothelial function, autonomic balance and lower systemic inflammation are plausible additional pathways, but human causal evidence is incomplete. Mechanistic claims based on animal models or acute exercise should not be presented as established explanations for fibrosis regression in patients.

Weight independence does not mean weight is irrelevant. Caloric deficit and sustained weight loss can amplify steatosis, MASH and fibrosis benefits, while exercise helps preserve lean mass and maintain function during weight reduction. The clinically useful message is additive: exercise is worthwhile even before substantial weight loss occurs, and it should continue when weight-loss therapies are used.

3.8. Histology, fibrosis and clinical outcomes

Evidence becomes less certain as outcomes move from steatosis to inflammation, ballooning and fibrosis. A systematic review focused on biopsy-proven disease identified very few randomized trials and did not find significant pooled improvement in activity score or fibrosis [12]. Houghton et al. reduced liver lipid in biopsy-proven NASH without clear improvement in circulating fibrosis markers [31]. O’Gorman et al. reported favorable histological changes after a 12-week aerobic intervention, but sample size and study design limit inference [51]. More recent pooled and post hoc analyses using composite non-invasive indices suggest a possible increase in MASH resolution after aerobic training, yet these indices are not a substitute for paired biopsy and have not established fibrosis regression [52].

The NASHFit trial provides strong proof of concept for steatosis and systemic benefit: 20 weeks of moderate aerobic training reduced MRI-PDFF and a thrombotic biomarker without clinically significant weight loss [20]. It did not, however, prove fewer cardiovascular events, cirrhosis complications or deaths. Long-duration, adequately powered trials with clinically meaningful outcomes are lacking. Exercise should be prescribed because its hepatic, cardiovascular, metabolic and functional benefits align, not because it has been shown to reverse advanced fibrosis in isolation.

3.9. Adherence and durability

Supervised trials often report high attendance, but real-world maintenance is more difficult. After a supervised 16-week program, improvements in liver fat, endothelial function and fitness were no longer present 12 months after supervision ended [53]. Short interventions demonstrate biological efficacy, not durable treatment.

Adherence strategies should be embedded in the prescription: collaborative goals, preferred activities, scheduled sessions, simple self-monitoring, graded targets, problem-solving and planned review. A written plan should specify both a target week and a minimum viable week. After disruption, restarting at a reduced dose is preferable to an all-or-nothing approach.

3.10. Practical FITT prescription

Table 3 translates MASLD-specific guidance and general exercise-medicine recommendations into a starting and target prescription for a clinically stable adult [3,4,8,37,45–47]. Disease-specific studies support regular exercise but do not validate every numeric intensity or progression threshold; several FITT values are therefore explicit extrapolations from general exercise and comorbidity guidance. Medication, autonomic dysfunction, heat and day-to-day

symptoms can alter heart-rate response, so HRR, RPE, the talk test and symptoms should be interpreted together.

3.10.1. Establishing intensity and the minimum viable dose

Moderate aerobic intensity is commonly approximated by 40–59% HRR, 64–76% of maximal heart rate, an RPE of 12–13 on the 6–20 Borg scale, or an effort at which conversation is possible but singing is difficult. Vigorous exercise is commonly approximated by 60–89% HRR, 77–95% of maximal heart rate or RPE 14–17 [37]. These ranges are guides, not interchangeable biological cut-offs. HRR requires a reliable resting and maximal heart rate; beta-blockers and chronotropic incompetence reduce the usefulness of percentage-of-predicted-heart-rate targets. In these settings, RPE, symptoms and the talk test should carry more weight.

The initial dose should be achievable. A very sedentary adult may begin with three 10-minute walks and two brief resistance sessions per week, then progress one variable at a time toward the target. Adding 5–10 minutes to selected sessions is usually more tolerable than simultaneously increasing frequency, duration and intensity.

The 750 MET-minutes/week signal from the Stine meta-analysis is a population-level response marker, not a strict individual threshold [5]. Although 150 minutes at 5 METs equals 750 MET-minutes, the internal strain depends on fitness, body mass and modality. Smaller increments remain worthwhile, especially when beginning from inactivity.

Resistance intensity can be anchored to repetition maximum, but formal one-repetition maximum testing is not always required. A load permitting 10–15 controlled repetitions is a practical starting point [37]. Leaving approximately 2–4 technically sound repetitions in reserve can help avoid training to failure, but this is a pragmatic coaching cue rather than a validated MASLD threshold. Progress load only when technique and symptom tolerance remain stable.

Progression should be separated from day-to-day adjustment. Workload may be reduced for acute pain, illness, heat, poor sleep or unusual occupational activity without abandoning the behavioral routine. After an interruption that reduces tolerance, resume below the previous workload and rebuild over several sessions.

Persistent deterioration in performance, sleep, motivation or musculoskeletal symptoms suggests that training load exceeds current capacity. Review the entire program, including occupational activity and dietary restriction. Alternating harder and easier aerobic days and

avoiding strenuous resistance training for the same muscles on consecutive days help distribute load.

Table 3. Practical exercise prescription for a clinically stable adult with MASLD

Component	Starting prescription	Target / progression	Monitoring and cautions
Aerobic MICT	3 days/week; 10–30 min/session; light-to-moderate intensity (RPE 10–13/20; talk comfortably to somewhat challenged) [3,4,8,37]	Progress duration before intensity toward ≥ 150 min/week moderate exercise across 3–5 days; selected patients may use ≥ 75 min/week vigorous equivalent	Record minutes and RPE; use low-impact modes for joint symptoms; stop and assess concerning exertional symptoms
Resistance	2 non-consecutive days/week; 6–8 exercises; 1 set of 10–15 controlled repetitions at light-to-moderate effort [37]	2–3 days/week; 8–10 exercises; 2–3 sets of 8–12 repetitions; increase load gradually when repetitions are completed with good technique [37]	Avoid breath-holding; adapt range/load for pain, neuropathy, retinopathy or hernia risk; emphasize technique
HIIT option	After a period of tolerable aerobic conditioning: 1 session/week with 4–6 short vigorous intervals and generous active recovery [45,48]	If preferred and tolerated, 1–3 sessions/week. Evidence-based example: 4 $\times$ 4 min at 85–95% HRmax with 3-min active recovery, preceded by warm-up and followed by cool-down	Use RPE plus heart rate; screening/supervision proportional to risk; not required for hepatic benefit
Sedentary-time reduction	Interrupt prolonged sitting at least every 30 min with standing or brief light activity [46,47]	Increase daily movement and steps from individual baseline; maintain on non-training days	Adjunct rather than replacement for structured exercise

Component	Starting prescription	Target / progression	Monitoring and cautions
Progression	Increase weekly exercise by small, tolerable increments	First frequency/consistency, then duration, then intensity or load; adjust when fatigue or pain accumulates [37]	A transient mild muscle ache can be expected; persistent pain, marked fatigue or functional decline requires adjustment

HRmax, maximal heart rate; HRR, heart-rate reserve; MASLD, metabolic dysfunction-associated steatotic liver disease; MICT, moderate-intensity continuous training; RPE, rating of perceived exertion. Sources: MASLD-specific guidance [3,4,8], general exercise prescription [37], preparticipation and diabetes guidance [45–47], and clinical HIIT evidence and guidance [22,48]. Numeric values are practical starting points, not validated liver-specific thresholds.

3.11. Pre-exercise assessment, safety and special populations

Before prescribing exercise, clinicians should characterize current activity, exercise experience, exertional symptoms, known cardiovascular, metabolic, renal and pulmonary disease, intended intensity, blood pressure control, diabetes complications, medication-related hypoglycemia risk, musculoskeletal limitations, frailty, falls and patient priorities [45–47]. Baseline measurements can include resting blood pressure and heart rate, body mass and waist circumference, a simple functional or walking test, and strength or sit-to-stand assessment. Formal cardiopulmonary exercise testing is valuable when exercise capacity must be precisely defined, symptoms require evaluation, vigorous training is planned in higher-risk patients, or advanced disease complicates prescription; it is not necessary for every asymptomatic patient beginning light walking [45].

Exercise should be postponed and medically evaluated when a patient has new or unstable chest discomfort, unexplained syncope, acute severe dyspnea, uncontrolled arrhythmia, acute systemic illness, severe uncontrolled blood pressure, acute musculoskeletal injury or another unstable condition. During exercise, chest pressure, presyncope, disproportionate dyspnea, palpitations with instability, new focal neurological symptoms or severe pain warrant cessation and assessment. These principles apply to the intended intensity and comorbidity rather than to MASLD alone [45].

In type 2 diabetes, insulin and insulin-secretagogue therapy may require glucose monitoring and medication or carbohydrate planning. Peripheral neuropathy favors well-fitting footwear, foot inspection and low-impact exercise; proliferative retinopathy may require avoiding high-strain lifting until ophthalmological advice is obtained [46,47]. In older adults or those receiving weight-loss pharmacotherapy, resistance training and adequate nutrition help limit loss of lean mass. Joint disease can be accommodated with cycling, water exercise, shorter bouts and machine-based resistance work.

Patients with compensated advanced fibrosis may benefit from individualized aerobic and resistance exercise, but reduced functional reserve, portal-hypertension complications and sarcopenia require specialist coordination [54]. Decompensated cirrhosis, recurrent falls, severe frailty or transplant evaluation warrants a liver-specialist and rehabilitation approach [54]. The evidence synthesized in this review should not be extrapolated uncritically to decompensated cirrhosis, children, pregnancy or rare causes of steatotic liver disease.

3.11.1. Phenotype-oriented examples

A sedentary patient with obesity, knee pain and no exertional red flags may begin with recumbent cycling or water-based aerobic exercise three times weekly, combined with machine or chair-based resistance training twice weekly. The early goals are attendance, symptom control and gradual extension of total time. Walking can be added as tolerance and strength improve. This approach preserves the aerobic and resistance targets without making weight-bearing exercise a prerequisite.

A patient with type 2 diabetes using basal-bolus insulin requires coordination between exercise timing, glucose monitoring, insulin dosing and carbohydrate intake. Moderate aerobic work can lower glucose during and after the session, while intense intervals may initially raise glucose through counter-regulatory responses before later sensitivity improves. The safest program is individualized from observed glucose patterns [46,47]. Resistance exercise may be scheduled before aerobic work in selected patients to attenuate an acute glucose fall, but responses vary and should be verified rather than assumed [46].

An older adult with low grip strength, repeated weight cycling or treatment-associated appetite suppression should have resistance training prioritized at least twice weekly, with balance and functional tasks such as sit-to-stand, step-up and loaded carry as appropriate [37,54]. Aerobic volume can be accumulated in shorter bouts. In this phenotype, preservation of independence and lean mass may be more clinically urgent than maximizing calorie expenditure, and stable weight should not be interpreted as a lack of benefit.

A younger, already active patient with stable cardiometabolic disease who prefers vigorous exercise may replace one or two MICT sessions with HIIT. A progressive protocol can begin with four 1-minute vigorous intervals before work bouts are lengthened or additional intervals are introduced [48]. Warm-up and cool-down remain part of the dose, and HIIT should complement resistance exercise and lower-intensity movement rather than produce an otherwise sedentary week.

These examples are not diagnostic algorithms. They illustrate how the same evidence-based targets can be achieved through different modes, session lengths and priorities. The decisive variables are medical stability, functional limitations, training response and adherence, not the diagnostic label alone.

3.12. Monitoring response

A review at 4–12 weeks should assess both implementation and outcomes. Implementation measures include session frequency, minutes, RPE, resistance loads, adverse symptoms and barriers. Clinical outcomes can include cardiorespiratory or walking capacity, strength, waist circumference, blood pressure, glycated hemoglobin where relevant, lipid profile and patient-reported energy or function. Liver enzymes may be tracked when clinically indicated, but should not be the sole response criterion. Non-invasive fibrosis testing and imaging should follow MASLD care pathways and baseline risk rather than be repeated simply because an exercise block ended [3,4].

A lack of weight loss does not justify stopping an otherwise successful program. If fitness, strength, glucose control or adherence improves, the intervention has produced clinically relevant benefit. Conversely, a fall in ALT without sustainable activity does not establish durable disease modification. The prescription should be revised when the patient cannot complete it, experiences recurrent pain or hypoglycemia, or sees no functional progression despite adherence.

Table 4. Suggested domains for monitoring an exercise prescription in MASLD

Domain	Practical measure	Interpretation	Pitfall to avoid
Adherence	Weekly aerobic minutes, resistance sessions, session RPE	Confirms delivered dose and identifies barriers	Equating a written prescription with completed exercise

Domain	Practical measure	Interpretation	Pitfall to avoid
Aerobic adaptation	Standardized walk test, submaximal workload or VO ₂ peak when available	Improved capacity supports a training effect even without weight loss	Comparing tests performed with different protocols or effort
Strength / function	Loads and repetitions, grip strength, sit-to-stand	Tracks neuromuscular adaptation and functional reserve	Using body mass alone as a proxy for muscle health
Anthropometry	Body mass and waist circumference	Useful for cardiometabolic trajectory; stable weight can coexist with benefit	Making weight loss the only definition of success
Metabolic risk	Blood pressure, HbA1c/glucose, lipids	Guides global risk management and medication review	Attributing all change to exercise when medications/diet also changed
Liver biochemistry	ALT, AST, GGT when clinically indicated	Supportive trend, especially if elevated at baseline	Using normal or unchanged enzymes to exclude disease or response
Steatosis	MRI-PDFF/MRS in research; ultrasound-based tools in selected care pathways	Quantifies hepatic-fat change with modality-dependent precision	Treating a steatosis change as proof of fibrosis regression
Fibrosis risk	FIB-4 followed by elastography or specialist pathway as indicated	Risk stratification, not a short-term exercise performance score	Repeating too frequently or interpreting stiffness without clinical context
Safety / recovery	Symptoms, pain, falls, hypoglycemia, sleep and fatigue	Determines whether dose and mode remain appropriate	Ignoring recurrent minor events until they cause discontinuation

ALT, alanine aminotransferase; AST, aspartate aminotransferase; FIB-4, Fibrosis-4 Index; GGT, gamma-glutamyl transferase; HbA1c, glycated hemoglobin; MRI-PDFF, magnetic resonance imaging proton-density fat fraction; MRS, magnetic resonance spectroscopy; RPE, rating of perceived exertion.

3.13. Integration into multidisciplinary MASLD care

Exercise prescription should be integrated with dietary care, anti-obesity and diabetes treatment, lipid and blood-pressure control, sleep-apnea assessment and alcohol counseling. These parallel interventions should be documented because they alter symptoms, body composition and exercise tolerance. Exercise remains indicated during medication-associated weight loss because fitness and strength do not improve automatically with lower body mass. Rapid weight loss or appetite suppression can increase concern about lean-mass loss, especially in older adults or those with low baseline strength. Progressive resistance exercise, adequate nutrition and functional monitoring should therefore be coordinated rather than treated as optional additions.

Referral pathways should reflect complexity. A low-risk patient may implement walking and basic resistance training with a written plan and primary-care follow-up. An exercise physiologist or physiotherapist can help when deconditioning, obesity-related mobility limitation, chronic pain, frailty or uncertainty about progression makes self-directed training less likely to succeed. Diabetes educators can support glucose management, while cardiology or respiratory evaluation is appropriate for unexplained exercise limitation. Advanced fibrosis, portal-hypertension concerns or decompensation requires hepatology involvement.

The written prescription should specify activity, frequency, target effort, duration, progression and precautions. For example: brisk walking at RPE 12–13 for 25 minutes on four days each week, progressing to 150 minutes/week; eight whole-body resistance exercises on two non-consecutive days, one to two sets of 10–15 repetitions; review in six weeks. Alternatives should be provided for pain, weather and travel.

If the program was not completed, follow-up should identify medical, practical and motivational barriers and revise the dose, mode, supervision or timing. Persistent non-adherence is information about the prescription and environment, not evidence that the patient is unsuitable for exercise. Long-term maintenance remains the central implementation challenge [53].

3.14. Research priorities

Future trials should report frequency, work and recovery intensity, duration, mode, progression, supervision, adherence and adverse events, while measuring diet and non-protocol activity. Standardized use of MRI-PDFF, fitness, strength, patient-reported outcomes and guideline-aligned fibrosis assessment would improve comparability.

Head-to-head comparisons require adequate sample size and energy- or time-matching that reflects the question being asked. Energy-matching tests whether intensity has an effect beyond energy expenditure; time-matching tests time efficiency; neither alone answers which intervention patients will sustain. Pragmatic trials should compare supervised initiation followed by scalable home, community or digital maintenance and should report outcomes after supervision ends.

Paired-biopsy studies are difficult but remain important for establishing effects on MASH activity and fibrosis. Where biopsy is not ethical or feasible, validated multimodal endpoints should be used with explicit acknowledgment that they are surrogates. Trials of at least 12 months are needed to study durability, medication interaction and disease progression. Clinical-event trials will be large, so harmonized prospective cohorts and linkage to cardiovascular and liver outcomes may provide complementary evidence.

Recruitment should better represent women, older adults, lean MASLD, different ethnic groups, advanced fibrosis, sarcopenia and multimorbidity. Comparative-effectiveness and implementation research should evaluate resistance-focused and combined programs, cost, access, health literacy and patient preference.

3.15. Limitations of the evidence and this review

Exercise trials are generally small, short and supervised, and participants are often middle-aged adults with overweight or obesity. Women, lean MASLD, advanced fibrosis, older frail adults and diverse ethnic populations remain underrepresented. Diagnostic methods, intensity definitions, adherence, co-interventions and control conditions vary substantially, making direct modality ranking unreliable.

Most studies measure steatosis, enzymes or metabolic surrogates rather than paired histology or clinical events. Liver stiffness is influenced by inflammation, congestion and technical factors, so short-term change should not be equated with fibrosis regression. Trial feasibility may also exceed routine care because volunteers receive structured supervision and resources.

4. CONCLUSIONS

Exercise is a core treatment for MASLD because it improves liver fat, fitness and cardiometabolic health, often without clinically significant weight loss. Aerobic training has the most extensive evidence, resistance exercise adds strength and lean-mass benefits with a lower cardiorespiratory burden, and HIIT is a time-efficient option for selected patients. Current evidence does not establish that HIIT is superior to moderate continuous training or that any modality alone reliably reverses fibrosis.

For most clinically stable adults, a pragmatic prescription is ≥ 150 minutes/week of moderate aerobic activity plus whole-body resistance training on 2–3 non-consecutive days. Sedentary patients should begin below this dose and progress gradually. HIIT is an optional alternative after conditioning for patients who prefer vigorous training and have an appropriate risk profile. Modality should be selected for safety, hepatic and extrahepatic priorities, and long-term adherence. Future trials should standardize exercise reporting and prioritize diverse populations, histology or validated imaging, fibrosis outcomes and clinical events.

DISCLOSURE

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