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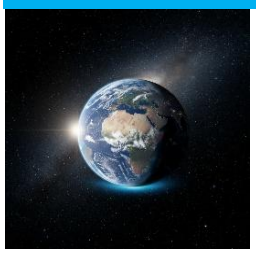

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Physical activity in the treatment of metabolic dysfunction-associated steatotic liver disease (MASLD): a review of evidence, mechanisms, and practical clinical recommendations

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Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is common and tightly linked to cardiometabolic risk. Physical activity is recommended, but clinicians need clear guidance on modality, progression, and follow-up.

Aim: To summarise exercise evidence in MASLD/NAFLD, outline key mechanisms, and translate findings into practical clinical recommendations.

Material and methods: Thematic narrative review. PubMed/MEDLINE, PubMed Central and the Cochrane Library were searched for English- and Polish-language publications from 2005–2025. We included adult structured exercise studies (aerobic, resistance, HIIT, combined), systematic reviews/meta-analyses, guidelines, and methodological papers on liver fat/fibrosis assessment. Outcomes extracted: liver fat (MRI-PDFF/MRS, ultrasound/CAP), ALT/AST, body weight, selected metabolic markers, and non-invasive fibrosis measures (e.g., FIB-4, elastography).

Results: Exercise reduces liver fat and yields modest decreases in ALT/AST; improvement may occur without meaningful weight loss. Aerobic and resistance training show similar effects on steatosis, while combined programmes more often improve lipid profile. Short interventions rarely modify non-invasive fibrosis markers, and interpretation is limited by heterogeneous protocols and outcome measures.

Conclusions: Exercise should be integrated as standard MASLD therapy alongside comorbidity management and weight-loss strategies when indicated. A practical starting option is progressive moderate-intensity aerobic training plus regular resistance sessions, adapted to comorbidities. Response should be assessed by adherence and routine labs at 8–12 weeks, with quantitative steatosis reassessment at 3–6 months when feasible.

Key words: MASLD; NAFLD; physical activity; aerobic exercise; resistance training; high-intensity interval training; MRI-PDFF; fatty liver; MASH; elastography; FIB-4; fibrosis risk

Introduction

Steatotic liver disease can manifest as metabolic dysfunction-associated steatotic liver disease (MASLD), metabolic dysfunction-associated steatohepatitis (MASH), and alcohol-associated

steatotic liver disease. In this review, we focus on MASLD. Metabolic dysfunction-associated steatotic liver disease (MASLD), previously referred to as non-alcoholic fatty liver disease (NAFLD), is a chronic liver disorder with a high population prevalence, affecting up to 30% of the global population, and is more common in men. It occurs particularly frequently in patients with type 2 diabetes, hyperlipidaemia, and obesity. MASLD is currently an important cause of liver cirrhosis and hepatocellular carcinoma. Although the aetiology of MASLD is not fully elucidated, contributing factors include an unhealthy diet and low physical activity, genetic and metabolic determinants, insulin resistance, and dysregulation of adiponectin signalling. Key risk factors for MASLD include obesity, type 2 diabetes, dyslipidaemia, and polycystic ovary syndrome. Additional associations have been reported with hypothyroidism and pituitary insufficiency, hypogonadism, obstructive sleep apnoea, psoriasis, and status post pancreatoduodenectomy. MASLD may be asymptomatic and is often detected incidentally on abdominal ultrasound or after identification of abnormal liver enzyme activities on laboratory testing. Clinical suspicion may be raised by symptoms such as fatigue, weakness, and right upper quadrant discomfort, as well as by findings including obesity, hepatomegaly, and features suggestive of portal hypertension. Over its course, MASLD may lead to progression of hepatic fibrosis. Patients also exhibit increased cardiovascular risk and a higher incidence of hepatocellular carcinoma, particularly in the presence of cirrhosis. The diagnostic work-up includes laboratory evaluation. Mild to moderate elevations of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are typically observed (ALT >30 U/L; AST/ALT ratio <1), increased gamma-glutamyl transferase (GGT) occurs in approximately 50% of cases, and serum bilirubin may occasionally be elevated. Concomitant metabolic abnormalities may include dyslipidaemia, hyperglycaemia, impaired glucose tolerance, hypoalbuminaemia, and prolonged prothrombin time, as well as increased serum iron and ferritin concentrations. Among imaging modalities, abdominal ultrasound is the primary tool in routine practice; it may demonstrate increased hepatic echogenicity consistent with steatosis, hepatomegaly, and in some cases features of portal hypertension and splenomegaly. Magnetic resonance imaging (MRI) can detect mild steatosis; however, similarly to computed tomography (CT), it is not routinely used in standard clinical evaluation. For non-invasive assessment of liver fibrosis, biochemical score-based tools such as FIB-4, the NAFLD Fibrosis Score (NFS), the Enhanced Liver Fibrosis (ELF) test, and FibroTest may be applied. Fibrosis assessment can also be supported by ultrasound-based elastography or magnetic resonance elastography. Liver histology obtained by biopsy is considered the reference standard for diagnosing MASLD. Hepatic steatosis is defined by the presence of steatosis in more than 5% of hepatocytes.

Histology typically shows macrovesicular steatosis, and may reveal lobular or portal inflammation. A diagnosis of metabolic dysfunction-associated steatohepatitis (MASH) requires the presence of steatosis, inflammation, and hepatocellular ballooning. Currently, liver biopsy is performed mainly in cases of suspected MASH, suspicion of other coexisting liver diseases, or diagnostic uncertainty. To grade the severity of histological changes in MASLD, scoring systems such as the NAFLD Activity Score (NAS) and the SAF score (steatosis, activity, fibrosis) are used. In population-based studies, additional biomarker-based indices such as SteatoTest, the Fatty Liver Index (FLI), and the NAFLD Liver Fat Score may be applied. The diagnosis of MASLD is based on evidence of hepatic steatosis on imaging or histology in an individual without other causes of steatosis (alcohol intake <20 g/day in women and <30 g/day in men), together with fulfilment of at least one metabolic criterion: (1) BMI ≥25 kg/m² or waist circumference ≥94 cm in men and ≥80 cm in women (or above ethnicity-specific thresholds); (2) blood pressure ≥130/85 mmHg or current antihypertensive therapy; (3) serum triglycerides ≥1.7 mmol/L (150 mg/dL) or treatment for hypertriglyceridaemia; (4) serum HDL cholesterol ≤1.0 mmol/L (40 mg/dL) in men and ≤1.3 mmol/L (50 mg/dL) in women, or lipid-lowering therapy; (5) fasting plasma glucose ≥5.6 mmol/L (100 mg/dL) or 2-hour post-load glucose ≥7.8 mmol/L (140 mg/dL), or HbA1c ≥5.7% (39 mmol/mol), or type 2 diabetes, or treatment for type 2 diabetes. In MASH, the diagnosis is based on MASLD with steatosis affecting more than 5% of hepatocytes, lobular and portal inflammation, and hepatocellular ballooning. In patients with MASLD, the next step is a non-invasive assessment of the risk of advanced fibrosis, commonly using the FIB-4 index. A value <1.3 indicates low risk, values of 1.3–2.67 suggest intermediate risk, and a value >2.67 indicates high risk of advanced fibrosis.[1]

Research materials and methods (EN)

This manuscript is a thematic narrative review aimed at translating the exercise evidence in MASLD into practical clinical recommendations and at clarifying the interpretation of commonly used endpoints in intervention studies.

Literature searches were conducted in PubMed/MEDLINE, PubMed Central (PMC), and the Cochrane Library, covering the publication window represented in the reference set used for this review (2005–2025) and including English- and Polish-language sources. Search terms combined MASLD/NAFLD/MASH/NASH with exercise-related keywords (exercise, physical activity, aerobic training, resistance training, high-intensity interval training) and outcome/measurement terms (MRI-PDFF, magnetic resonance spectroscopy, ultrasound,

controlled attenuation parameter, ALT, AST, elastography, FIB-4 and other non-invasive fibrosis tools). We included: adult studies in NAFLD/MASLD/MASH/NASH evaluating structured exercise interventions (aerobic, resistance, interval, or combined) against usual care, no-exercise controls, or non-pharmacological comparators; systematic reviews and meta-analyses of exercise interventions; clinical practice guidelines; and methodological studies informing liver fat and fibrosis measurements. In addition, evidence relevant to safety and exercise eligibility in typical MASLD comorbid populations (e.g., type 2 diabetes, hypertension, obesity, cirrhosis) and selected preclinical studies were included when needed to support a clinically interpretable mechanistic overview.

Study selection followed a two-stage process (title/abstract screening followed by full-text assessment), and data were extracted into a standardised form. Extracted variables covered intervention characteristics (FITT parameters, supervision vs home-based delivery), population features (including comorbidities), and outcomes: liver fat change (MRI-PDFF/MRS, ultrasound, CAP), ALT/AST, anthropometrics, selected metabolic parameters (glucose/HbA1c, lipids, insulin resistance indices when reported), and fibrosis-related measures (non-invasive scores and elastography).

Rationale for physical activity as a therapeutic cornerstone in MASLD

Physical activity is a core component of MASLD management because it improves cardiometabolic risk while targeting pathways that promote hepatic fat accumulation. Current European guidelines recommend physical activity and structured exercise in adults with MASLD to reduce steatosis, tailored to individual preferences and capacity. [2] Clinically, an important message from intervention studies is that reductions in liver fat and decreases in ALT and AST can occur even without meaningful weight loss, supporting a potentially “weight-neutral” benefit in selected patients. [3] This pattern has also been reported in randomized resistance-training trials, where improvement in steatosis did not track with body weight change and appeared more closely linked to favourable body composition shifts. [4] Moreover, evidence syntheses suggest modality-specific effects on transaminases and lipid parameters, which supports pragmatic individualisation of exercise prescriptions without losing the hepatic target. [5]

Aim, clinical questions, and paper structure

The purpose of this review is to organise the evidence in a way that supports day-to-day exercise decisions in patients with MASLD. We focus on four clinical questions: which type of exercise (aerobic, resistance, interval, or combined) most consistently improves relevant outcomes, what dose is sufficient to start and how to progress it, in whom the benefit is most likely (particularly in obesity, type 2 diabetes, and across different risks of advanced fibrosis), and which endpoints are practical for follow-up (imaging-based liver fat assessment, aminotransferases, cardiometabolic markers, and non-invasive fibrosis assessment using scores and elastography). [2,3,5]

Nomenclature and its implications

In recent years, fatty liver terminology has been reorganised under the umbrella term steatotic liver disease (SLD), with subcategories defined by the main driver. A multisociety Delphi statement published in 2023 proposed replacing NAFLD with MASLD (hepatic steatosis plus at least one cardiometabolic risk factor) and replacing NASH with MASH for the inflammatory phenotype with hepatocellular injury. [6] In the same framework, MetALD was introduced for patients with metabolic dysfunction and clinically relevant, intermediate alcohol exposure, aiming to better capture overlap between metabolic and alcohol-related drivers. [6,7] This change is important when reading older "NAFLD" studies, because many cohorts were largely exclusion-based, did not consistently require metabolic criteria, and assessed alcohol intake with variable rigor. [6,8] As a result, legacy NAFLD populations may mix individuals who would not meet MASLD today with patients closer to MetALD, which reduces comparability across studies and can distort estimated intervention effects. [6–8]

Clinical phenotypes and why they matter for exercise response

MASLD commonly coexists with visceral obesity, type 2 diabetes, and dyslipidaemia. This clinical phenotype shapes both the expected response to exercise and what should be considered a meaningful improvement. [2,9] In central obesity, insulin resistance and increased lipid flux to the liver are major drivers, so physical activity may reduce hepatic steatosis and improve metabolic parameters even without substantial weight loss; changes in visceral adiposity and insulin sensitivity are often more informative than BMI alone. [2,10] In type 2 diabetes,

MASLD is more frequently linked to higher cardiovascular risk and more advanced liver disease, which supports assessing exercise benefits in parallel through glycaemic control and fibrosis risk, rather than relying only on aminotransferases. [2,9] Dyslipidaemia is common and contributes to atherogenic risk; therefore, the value of exercise should also be judged by improvements in lipid profile and overall cardiovascular risk reduction, which is prognostically central in this population. [2,11]

Metabolic mechanisms

In MASLD, a key pathophysiological mechanism is excessive substrate delivery to hepatocytes, particularly free fatty acids (FFA/NEFA) released through adipose tissue lipolysis. Stable-isotope studies in NAFLD showed that most hepatic triglyceride derives from circulating NEFA (around 60%), with a substantial contribution from de novo lipogenesis. [12] Insulin resistance weakens insulin-mediated suppression of lipolysis, sustaining NEFA flux to the liver and favouring persistent steatosis. [12] Regular physical activity improves peripheral insulin sensitivity, reduces the availability of lipogenic substrates, and alters lipid handling, thereby limiting hepatic exposure to NEFA and to glucose used for fatty acid synthesis, while promoting greater fatty acid oxidation. [13,14] In parallel, reductions in visceral adipose tissue decrease portal lipid delivery; in a randomized trial in NASH, 12 weeks of supervised exercise lowered liver fat and visceral adiposity without meaningful weight loss, supporting an effect that is not solely explained by weight reduction. [15]

Cellular and inflammatory mechanisms

Beyond altering substrate delivery, physical activity may also affect cellular processes involved in the transition from simple steatosis to an inflammatory phenotype. Lipid overload increases the burden on oxidative pathways and the respiratory chain, which promotes reactive oxygen species generation, lipid peroxidation, and mitochondrial injury. [16] In experimental models, exercise is associated with improved hepatic oxidative metabolism (including higher markers of mitochondrial biogenesis and enzyme activity) and favourable shifts in antioxidant defence, thereby reducing conditions that facilitate oxidative stress. [17,18] In parallel, exercise appears to modulate inflammatory signalling: in a clinical study of patients with NASH, both aerobic and resistance training reduced selected pro-inflammatory cytokines (TNF-α, IL-6, IL-8) together with aminotransferase activity, suggesting that exercise effects may extend beyond liver fat to the inflammatory component of disease. [19] Clinically, these mechanisms support

viewing exercise as an intervention that may attenuate drivers of disease progression rather than acting solely through reduction of hepatic fat content. [17,18]

Cardiovascular risk as a parallel therapeutic target

In MASLD, prognosis is shaped not only by liver disease progression but also by cardiovascular complications. Evidence from the NAFLD era consistently shows that cardiovascular disease is among the most common causes of death in this population, with higher risk in people with type 2 diabetes and in those with more advanced liver disease. [20,21] This is why management should pursue two goals in parallel: limiting liver disease progression and systematically addressing cardiovascular risk. Current guidelines recommend cardiovascular risk assessment in every adult with MASLD and active treatment of comorbidities, including hypertension, dyslipidaemia, and disorders of glucose metabolism. [2] In this setting, physical activity is particularly useful because it links hepatic targets with improved cardiorespiratory fitness and favourable changes in cardiometabolic risk factors. [22] Intervention trials often need to detect small longitudinal changes in liver fat, so the choice of imaging method directly affects interpretability. Magnetic resonance imaging–proton density fat fraction (MRI-PDFF) provides a quantitative, continuous measure and is well suited as a trial endpoint. In a study comparing MRI-PDFF with magnetic resonance spectroscopy (MRS), repeat-scan variability of MRI-PDFF within the same individual was <1 percentage point, and an absolute change of ≥1 percentage point was used as a practical threshold for a reliable change; agreement with MRS was very high (r^2=0.98), with only small MRI underestimation versus MRS. [23] A review focusing on MRI-PDFF (magnetic resonance imaging–proton density fat fraction) as a trial endpoint emphasised that PDFF provides a quantitative estimate of the percentage of liver fat. The authors noted that a ~29% relative reduction in PDFF was more often accompanied by a histologic response, defined as improvement on liver biopsy (reduced inflammatory injury and/or overall disease activity). [24] This cut-off may therefore serve as a pragmatic benchmark in trial planning, including lifestyle interventions, particularly when biopsy is not performed in all participants. [24] Magnetic resonance spectroscopy (MRS) is highly precise, but it typically samples only a single measurement volume (voxel) and requires more complex acquisition and post-processing, which limits feasibility and scalability in large multicentre studies. [23] Conventional abdominal ultrasound is widely available, but it performs less well for tracking change over time. For mild steatosis, reported sensitivity at a >5% threshold is ~60.9–65%, with better performance mainly in more pronounced steatosis (approximately >20–30%). [25] A more numeric ultrasound-based metric is the controlled attenuation parameter (CAP),

measured during transient elastography, reported in dB/m. In a meta-analysis of 61 studies, mean CAP cut-offs were ~268.5 dB/m for ≥S1, ~288.0 dB/m for ≥S2, and ~313.1 dB/m for S3 (AUROC 0.924, 0.794, 0.778, respectively). [26] Importantly, cut-offs were higher in obesity (BMI ≥30 kg/m²), for example 281.9 dB/m for ≥S1 and 301.0 dB/m for ≥S2, which should be considered in trial analyses. [26] Overall, MRI-PDFF/MRS are most appropriate for detecting subtle changes in liver fat, whereas ultrasound and CAP are mainly pragmatic options when feasibility and routine availability outweigh sensitivity to small differences. [23–26]

Hepatocellular injury - ALT/AST interpretation and discordance with steatosis

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are the simplest markers of hepatocellular injury in MASLD, but elevations are usually modest, typically 1–2 times the upper limit of normal. [27] Importantly, normal aminotransferases do not exclude disease or the inflammatory phenotype: in a meta-analysis of 11 studies, 25% of individuals with NAFLD had normal ALT, and among patients with NASH the proportion was 19%. [28] In practice, this means ALT/AST correlate poorly with the amount of liver fat and may remain normal despite clinically relevant disease. In a histology-based study, using an ALT cut-off of 35 U/L contributed little to predicting NAFLD severity; for detecting moderate-to-severe steatosis, an ALT threshold of 58.5 U/L yielded 66% sensitivity and 50% specificity (AUC 0.616). [3] By contrast, the AST/ALT ratio showed a stronger relationship with advanced fibrosis: mean AST/ALT was 1.18 vs 0.75 in those with advanced fibrosis, and the AUC for this ratio was 0.836. [29] Therefore, aminotransferases are useful for tracking trends and possible inflammatory injury, but in intervention studies they should be interpreted alongside objective steatosis measures and non-invasive fibrosis risk assessment. [30]

Non-invasive fibrosis evaluation in exercise trials: FIB-4 and elastography

In MASLD, fibrosis stage is the strongest determinant of liver-related risk, which is why clinical pathways rely on non-invasive tests (NITs) and elastography for risk stratification and follow-up. [2] In a stepwise approach, FIB-4 (based on age, aminotransferases, and platelet count) is used first-line: commonly applied thresholds are <1.3 to rule out advanced fibrosis (≥F3) and >2.67 to increase the likelihood of advanced fibrosis and trigger confirmatory imaging. [2] For elastography, vibration-controlled transient elastography (VCTE) reports liver stiffness measurement (LSM) in kilopascals (kPa); values around <8 kPa are used to rule out ≥F3 and ≥12 kPa to support ≥F3. In magnetic resonance elastography (MRE), commonly cited cut-offs

are approximately 3.14 kPa (F2), 3.53 kPa (F3), and 4.45 kPa (F4). [2] Exercise intervention data suggest that fibrosis-related markers are less consistently affected than steatosis. In a 12-week randomized trial comparing aerobic and resistance training in NAFLD, baseline LSM was low (~4.5 ± 0.2 kPa vs 4.3 ± 0.2 kPa) and did not change significantly after the intervention (P = 0.733), despite improvement in steatosis. [31] In biopsy-proven NASH, 12 weeks of exercise reduced hepatic triglyceride content but did not change non-invasive fibrosis markers, including the NAFLD fibrosis score (e.g., −1.51 ± 1.00 → −1.50 ± 1.12) or the enhanced liver fibrosis (ELF) test (e.g., 9.4 ± 1.1 → 9.5 ± 1.2; no significant time×treatment interaction). [15] Likewise, a pooled post hoc analysis of three RCTs (12–20 weeks) showed meaningful liver fat improvement, but no significant between-group change in FIB-4 (e.g., 1.35 → 1.26; group-by-time P = 0.781). [32] When interpreting elastography longitudinally, it should also be noted that LSM can be influenced by non-fibrotic factors (e.g., inflammation, cholestasis, venous congestion), which limits confident inference about true fibrosis regression in short interventions. [33]

Effectiveness of exercise: synthesis of intervention evidence

Across reviews of randomized trials (mostly conducted in cohorts previously labelled as NAFLD), exercise shows the most consistent effect on liver fat reduction and modest improvements in liver biochemistry, and some analyses indicate benefits without meaningful weight loss. In a meta-analysis restricted to exercise-only interventions without concurrent dietary change and without significant weight loss, intrahepatic lipid measured by magnetic resonance spectroscopy decreased (standardized mean difference, SMD −0.76; 95% CI −1.04 to −0.48), alongside reductions in aminotransferases (ALT: SMD −0.52; AST: SMD −0.68). [3] The same analysis also reported improvements in selected lipid parameters (LDL-C: SMD −0.34; triglycerides: SMD −0.59), while body weight and BMI were generally unchanged across included trials. [3] In a separate meta-analysis focused on non-diabetic patients, physical activity produced small but significant reductions in liver enzymes (ALT: SMD −0.17; AST: SMD −0.25; GGT: SMD −0.22) and a modest reduction in liver fat (SMD −0.21); subgroup analyses suggested that longer interventions (≥4 months) were more often associated with improvement in fasting glucose (SMD −0.27) and insulin resistance by HOMA-IR (SMD −0.44), as well as triglycerides (SMD −0.16) and LDL-C (SMD −0.25). [34] These findings are consistent with another meta-analysis reporting mean decreases of ALT by 5.91 U/L, AST by 4.90 U/L, and BMI by 0.78 kg/m² after exercise interventions. [35] Comparative studies suggest that in MASLD, key benefits in steatosis can be achieved with both aerobic and resistance

training, while differences are more often seen in metabolic outcomes. In a randomized trial comparing moderate-intensity aerobic training with resistance training (mean ~3.4 sessions/week), 12 weeks produced similar relative reductions in liver fat: -10.3% in the aerobic group and -12.6% in the resistance group. [31] Comparative evidence from a network meta-analysis of 43 trials (n=2070) suggests that response domains may vary by training type: the highest probability of improvement in ALT was observed with aerobic training (SUCRA 83.9), whereas AST improvement ranked highest with resistance training (SUCRA 81.7). [5] For lipid outcomes, combined aerobic plus resistance programmes ranked best for triglycerides (SUCRA 96.8) and LDL cholesterol (SUCRA 86.1), while HDL cholesterol most often improved with aerobic interventions (SUCRA 72.3). [5] Evidence for high-intensity interval training (HIIT) is more limited, but a meta-analysis of randomized trials reported significant reductions in intrahepatic lipids (SMD −0.56), alongside decreases in ALT (SMD −0.61) and AST (SMD −0.43) and a modest reduction in BMI (SMD −0.31); most lipid and glycaemic endpoints were not consistently improved. [36] Clinically, this supports the view that steatosis reduction is relatively consistent across training types, whereas the choice of intervention can be aligned with additional targets such as lipid profile or aminotransferases. [5] Exercise intervention data in MASLD can be linked to concrete, monitorable changes in hepatic and metabolic parameters. In a 12-week randomized trial, aerobic or resistance training reduced liver fat by about 10–13% relative to baseline, i.e., compared with the starting value. [31] With a baseline MRI-PDFF of 20%, this corresponds to a reduction of roughly 2–3 percentage points (e.g., from 20% to 17–18%). [31] Methodological studies show high repeatability of MRI-PDFF, with within-subject differences between repeated scans usually below 1 percentage point; therefore, a decrease of at least 1 percentage point is commonly interpreted as exceeding typical measurement variability and likely reflecting a true reduction in liver fat. [23] A review on MRI-PDFF also noted that a ~29% relative reduction in PDFF more often co-occurred with histologic improvement on biopsy (lower inflammatory injury/activity), which is sometimes used as a pragmatic benchmark in studies where biopsy is not performed in all participants. [24] Aminotransferase changes are typically modest but reproducible: a meta-analysis reported mean decreases of ALT by ~5.9 U/L and AST by ~4.9 U/L, with only minor changes in BMI. [35] Accordingly, early assessment of response should not rely on weight loss alone, particularly over short timeframes. [3] A pragmatic follow-up strategy includes reassessment after 8–12 weeks (adherence, ALT/AST, and cardiometabolic markers) and consideration of quantitative steatosis reassessment after 3–6 months when feasible. [2] If improvement is not observed despite regular training, intensification is justified (e.g., combined programmes,

higher weekly volume, adding structured weight-loss strategies), especially when metabolic abnormalities persist or fibrosis risk is elevated. [2,3]

Designing and progressing exercise training in MASLD: a FITT framework

Exercise prescription in MASLD can be structured using the FITT framework, with a weekly dose aligned with the EASL–EASD–EASO guideline: >150 min/week of moderate-intensity or ≥75 min/week of vigorous-intensity physical activity to reduce steatosis, tailored to the individual's capacity. [2] In intervention trials, this is commonly delivered as 3–4 sessions per week; in a 12-week supervised randomized trial participants completed a mean of ~3.4 sessions/week, and the aerobic programme used 60-min sessions, progressing from 2×15 min at 60% of maximal heart rate in week 1 to 2×15 min at 70% of maximal heart rate during weeks 2–12. [31] In the same protocol, resistance training included 10 whole-body exercises; weeks 1–4 used 1–2 sets of 8–12 repetitions at 60% 1RM, followed by 2 sets of 8–12 repetitions at 60% 1RM to fatigue, with 1–2 min rest between sets (1RM, one-repetition maximum, is the maximal load that can be lifted once in a given exercise). [31] Steatosis reduction is achievable with both aerobic and resistance training, but combining both types is supported when metabolic targets are prioritised; a network meta-analysis ranked combined programmes highest for improvements in triglycerides and LDL cholesterol. [5] A practical minimum can therefore be defined as meeting the weekly time threshold (≥150 min/week of moderate activity) and including at least two resistance sessions per week, consistent with recommendations summarised in a review of exercise training in NAFLD. [37] Most trials used supervised programmes, which supports safe load selection and technique; in home-based settings, intensity can be monitored using heart rate and the Borg 6–20 rating of perceived exertion (RPE). In a validation study in type 2 diabetes, RPE 12–13 was intended to represent moderate intensity defined as 40–59% heart rate reserve (HRR), yet only 57% of participants fell within this target range, while 37% were above and 6% below, supporting combined use of RPE and heart rate monitoring, particularly early in training. [38] Finally, adherence should be addressed explicitly: in an NAFLD survey, 75% of patients did not achieve ≥150 min/week of activity, and the most frequently reported barriers were lack of provider resources/education (47%), fatigue during exercise (44%), and time constraints (32%), which supports gradual progression, brief regular follow-up, and simple monitoring tools (logs or activity trackers) adapted to the patient's circumstances. [6]

Comorbidities (type 2 diabetes, obesity, hypertension): eligibility and common constraints

In most MASLD patients, low-to-moderate intensity activity can be introduced safely, whereas progression toward moderate-to-vigorous exercise in previously sedentary adults should be preceded by clinical assessment, particularly when cardiovascular symptoms or microvascular complications are present. [2] The ACSM consensus statement on physical activity in type 2 diabetes recommends medical clearance (and, in selected cases, exercise testing) before activities more vigorous than brisk walking, especially in older adults, those with longer diabetes duration, or additional risk factors (including hypertension, dyslipidaemia, smoking, nephropathy, and retinopathy). [40] Practical pre-exercise glucose thresholds are also provided: exercise should not be started at >250 mg/dL (13.9 mmol/L) when moderate/high ketones are present, and caution is advised at >300 mg/dL (16.7 mmol/L) without significant ketones, with emphasis on hydration and starting only if the individual feels well; for those on insulin or sulfonylureas, preparedness for hypoglycaemia (rapid-acting carbohydrates) is recommended. [40] With peripheral neuropathy, activities with higher risk of foot trauma (e.g., prolonged uneven-surface walking) should be limited and non–weight-bearing options (e.g., cycling or chair-based exercise) are preferred. [40] In hypertension, uncontrolled values should be excluded before training; reviews cite blood pressure >180/110 mmHg as a contraindication, and in stage 2 hypertension pharmacological treatment should be established before initiating structured training. [41,42] In obesity, initial limitations are commonly musculoskeletal; therefore, lower-impact options (flat-surface walking, cycling, low-intensity rowing ergometry) are often appropriate early choices, guided by tolerance.

Advanced disease: caution and when to involve a specialist

When advanced fibrosis is suspected (e.g., high-risk NIT prompting confirmation by elastography within MASLD care pathways), safety and symptom-guided intensity selection become priorities. [2] A Cochrane review on exercise in cirrhosis highlights that exercise can acutely increase portal pressure, which historically raised concern about variceal bleeding; therefore, in advanced liver disease it is reasonable to avoid activities associated with abrupt pressure surges (especially Valsalva manoeuvres and lifting “to failure”) and to prioritise moderate-intensity aerobic activity with submaximal resistance work. [43] Hepatology consultation is appropriate before intensifying training in patients with decompensation (ascites,

prior gastrointestinal bleeding, encephalopathy, progressive jaundice) or when disease stage/etiology remains uncertain. [2,43]

Older adults and low fitness: the role of resistance training and progression

In older adults and those with low fitness, targets extend beyond steatosis to preservation of muscle mass, functional capacity, and fall risk reduction. A systematic review of resistance training in older adults reported improvements in functional mobility domains such as gait speed and balance. [44] A scoping review describing resistance-training protocols in older adults with sarcopenic obesity reported typical full-body sessions comprising 8–12 exercises, usually 2–3 sets at low-to-moderate intensity (8–15 repetition maximum range), with 90–180 s rest intervals, performed 2–3 times per week. [45] In this population, gradual progression is essential: start with loads that allow correct technique and then increase volume and load as adaptation occurs, incorporating balance and functional tasks when feasible.

Implementation algorithm: from brief risk assessment to a FITT plan and target prioritisation

Implementation of physical activity in MASLD can follow a brief stepwise approach. First, the dominant clinical target is defined: reduction of steatosis and risk of liver disease progression, or cardiovascular risk modification (often both are relevant, but one may dominate). Next, the risk profile is structured by identifying key comorbidities and stratifying advanced fibrosis risk using a first-line non-invasive test (most commonly FIB-4), followed by second-step testing (e.g., elastography) when FIB-4 is intermediate or high. [2,46] Based on these elements, an individualised FITT plan (frequency, intensity, time, type) is formulated and a predefined review point is set to assess tolerance, implementation, and the need for programme adjustment. [2]

Improving adherence: SMART goals, monitoring, and lifestyle medicine components

To improve adherence, the plan should include SMART goals (specific, measurable, achievable, clinically relevant, and time-bound) and a simple self-monitoring strategy. In routine care, task-based scheduling (specific days, duration, and activity type) combined with a predefined progression scheme (e.g., gradual increases in weekly time or session number) is pragmatic. Guidelines emphasise behavioural support, including systematic self-monitoring and barrier management. [2] Qualitative evidence suggests that adherence is supported by action planning

and coping planning, which justifies brief structured counselling: identify one or two likely barriers and define a feasible alternative (e.g., switching to non–weight-bearing exercise when joint symptoms worsen, shortening sessions during time-limited periods, adjusting intensity when fatigue is prominent). [47]

Minimal follow-up set: what to monitor and when

Follow-up should cover three domains: hepatic markers, cardiometabolic risk factors, and fibrosis risk. The guideline recommends regular assessment of aminotransferases and metabolic profile (glucose/HbA1c, lipids), together with anthropometrics (body weight and waist measures), with frequency tailored to overall risk and comorbidities. [2] Practically, an 8–12 week visit is mainly used to review tolerance and implementation, while repeating laboratory and anthropometric measures at 3–6 months is reasonable to evaluate trends. [2,46] Fibrosis monitoring can be simplified to a rule-based approach: FIB-4 as the screening test (repeated periodically in low-risk individuals) and elastography as second-step testing when FIB-4 is intermediate/high or abnormalities persist; in type 2 diabetes or multiple metabolic risk factors, more frequent FIB-4 reassessment can be considered. [2,46]

Synergy with weight loss and diet

In adults with MASLD and overweight/obesity, the central target of dietary and behavioural therapy is sustained weight loss, because the magnitude of weight reduction aligns with the expected hepatic benefit. The EASL–EASD–EASO guideline indicates that ≥5% weight loss is associated with liver fat reduction, 7–10% with improvement in inflammatory activity, and ≥10% with the greatest likelihood of fibrosis improvement. [2] Clinical data support this gradient: in a randomized trial in biopsy-proven NASH, intensive lifestyle intervention achieved a mean weight loss of 9.3% at 48 weeks (vs 0.2% in controls), and participants achieving ≥7% showed larger improvements in steatosis, lobular inflammation, ballooning, and a greater reduction in NAS (−3.45 vs −1.18). [48] In parallel, improving diet quality toward a Mediterranean-style pattern, limiting ultra-processed foods, and avoiding sugar-sweetened beverages is recommended to improve liver injury assessed histologically or non-invasively. [2]

Positioning physical activity alongside comorbidity management and cardiovascular prevention

Physical activity should be implemented alongside standard management of comorbidities, as these largely drive cardiovascular risk in MASLD. The guideline recommends structured assessment and management of type 2 diabetes, dyslipidaemia, and hypertension in accordance with general care standards. [2] A practical point is the explicit recommendation that statins can be used in chronic liver disease, including compensated cirrhosis, and should be prescribed according to cardiovascular risk guidelines to reduce cardiovascular events. [2] Within this framework, exercise supports risk-factor control but does not replace pharmacological therapy when indicated.

Discussion

The exercise literature in MASLD is broadly consistent regarding direction of change in steatosis and modest shifts in aminotransferases and selected metabolic parameters, yet clinical interpretation is constrained by study design and reporting. A major limitation is heterogeneity of interventions (duration, intensity, weekly volume, variable supervision) combined with inconsistent reporting of training parameters, which hampers cross-study comparability and evidence synthesis. [2,3,5] Methodological variability in steatosis assessment (MRI-PDFF/MRS vs ultrasound/CAP), with differing sensitivity to small longitudinal changes, and the predominance of short follow-up further limit inference about fibrosis and clinically meaningful progression. [15,23] Many cohorts are selected (often excluding advanced disease), and legacy "NAFLD" definitions do not always map directly onto contemporary MASLD/MetALD criteria, creating potential heterogeneity in metabolic phenotype and alcohol exposure. [2,6] Co-interventions (dietary change, non-exercise physical activity) are often incompletely controlled, and adherence is frequently assessed by self-report, which may bias effect estimates. [2,3] Research needs therefore include standardised intervention reporting (frequency, duration, intensity, type, supervision), objective measurement of activity and adherence, longer trials with clearly defined endpoints related to MASH and fibrosis, and subgroup analyses stratified by MASLD phenotype (e.g., type 2 diabetes, visceral obesity, sarcopenia) and baseline fibrosis risk. [2,15]

Disclosures

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