

Sarcopenia and Metabolic Dysfunction Associated Steatotic Liver Disease: A critical review with structured synthesis of published meta analyses

DOI: 10.5281/zenodo.23550219

Recebido: 19 out 2026

Aprovado: 20 out 2026

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ABSTRACT

Sarcopenia and metabolic dysfunction-associated steatotic liver disease may coexist through shared metabolic, inflammatory, and behavioral pathways. However, evidence connecting muscle impairment to steatosis and fibrosis is complicated by changing liver nomenclature and inconsistent muscle definitions. This review critically synthesizes PubMed-indexed clinical publications verified on 7 October 2026, emphasizing published meta-analyses and selected longitudinal and biopsy-based studies. Review-level estimates were displayed in their original outcome direction and were not pooled across overlapping evidence bases. Published syntheses generally associated sarcopenia with prevalent fatty liver disease and with higher odds of liver fibrosis, although effect sizes differed substantially. A 2026 synthesis reported associations of 1.58 for fatty liver disease and 2.03 for concomitant fibrosis; earlier reviews reported smaller or less precise estimates. Regional and diagnostic-method differences limit universal interpretation. Much of the literature defines sarcopenia through low relative muscle mass without the strength and performance assessments required by contemporary clinical frameworks. Historical NAFLD populations were therefore discussed as relevant to MASLD without assuming complete diagnostic equivalence. Longitudinal studies support temporal relationships between relative muscle mass and liver outcomes but do not establish that increasing muscle reverses fibrosis. Integrated assessment of muscle function, adiposity, metabolic health, and validated liver fibrosis measures

is justified. Prospective intervention studies using contemporary definitions are needed to determine whether muscle-directed care improves hepatic outcomes.

Keywords: Sarcopenia, MASLD, NAFLD, Liver fibrosis, Skeletal muscle.

1 INTRODUCTION

Skeletal muscle and the liver occupy central positions in metabolic regulation. Their disorders can coexist in people with obesity, diabetes, reduced activity, or advancing age, yet the clinical meaning of that coexistence is not straightforward. A low muscle-to-body-weight ratio can accompany adiposity without necessarily identifying the same biological condition as low strength or poor physical performance. Similarly, the presence of hepatic steatosis does not establish the degree of inflammation or fibrosis.

The growing literature linking sarcopenia and fatty liver disease has therefore generated an important question: does muscle impairment independently identify people with a more unfavorable hepatic trajectory, or does it predominantly reflect shared cardiometabolic exposures? Both explanations may contribute. Distinguishing them requires attention to how muscle and liver outcomes were measured and to whether observations were cross-sectional, longitudinal, or experimental.

The adoption of metabolic dysfunction-associated steatotic liver disease, or MASLD, provides a positive cardiometabolic definition within the broader category of steatotic liver disease. Most older clinical studies instead enrolled people under NAFLD criteria. Although these populations overlap substantially in clinical practice, a change of terminology cannot retrospectively establish that every participant met the newer criteria (Rinella et al., 2023).

Published meta-analyses consistently identify an association, but their pooled results vary with the direction of the comparison and the diagnostic framework. This variation deserves interpretation rather than a single combined number. A study estimating the odds of sarcopenia among people with fatty liver is not asking the same temporal question as a cohort examining incident liver disease after measurement of muscle mass.

The objectives of this review are to interpret published quantitative associations, examine the evidence for fibrosis and temporal relationships, and clarify how contemporary sarcopenia and MASLD definitions should shape future research and clinical assessment. All references are verified PubMed-indexed publications.

2 THEORETICAL FRAMEWORK

2.1 Muscle mass strength and physical performance

Sarcopenia is a clinical muscle disorder rather than a synonym for low muscle mass. The revised European consensus prioritizes low strength, uses low muscle quantity or quality to confirm the diagnosis, and recognizes poor physical performance as a marker of greater severity. This framework changes how older liver studies should be interpreted: an exposure labeled sarcopenia may have been defined using only a body-composition ratio (Cruz-Jentoft et al., 2019).

Muscle quantity can be indexed to height, weight, or body mass index. These approaches answer different questions. Height adjustment addresses muscle relative to body size, whereas weight adjustment also incorporates the amount of nonmuscle tissue. In people with obesity, a declining muscle-to-weight ratio can result from gaining fat, losing muscle, or both. Associations with liver disease can consequently reflect part of the adiposity relationship embedded in the denominator.

The measurement modality also matters. Dual-energy X-ray absorptiometry estimates lean tissue; bioelectrical impedance relies on model-based estimation; computed tomography provides regional muscle area and attenuation; strength and performance tests assess functional capacity. None is an interchangeable substitute for all the others. A synthesis that combines them must describe which phenotype its pooled estimate represents.

2.2 Shared pathways and directionality

Insulin resistance, inflammatory signaling, altered activity, and changes in organ-derived mediators provide plausible links between muscle impairment and steatotic liver disease. Skeletal muscle also functions as a secretory organ, making the relationship potentially more complex than a simple effect of body composition on liver fat (Kim and Kim, 2020; Polyzos et al., 2023).

These mechanisms support biological plausibility but do not prove direction. Liver disease can also reduce activity and nutritional resilience. Shared causes and reverse effects must therefore remain part of the interpretation of observational associations.

2.3 Liver nomenclature and endpoint hierarchy

MASLD requires hepatic steatosis in the context of cardiometabolic risk. The historical NAFLD framework was organized around steatosis and exclusion of substantial alcohol exposure and other causes.

These definitions are closely related but not identical. A contemporary review should preserve the diagnostic language of each source while explaining its relevance to the newer framework (Rinella et al., 2023).

Liver outcomes form a hierarchy. Steatosis, steatohepatitis, significant fibrosis, advanced fibrosis, cirrhosis, and liver-related events are distinct endpoints. A muscle association with steatosis prevalence does not necessarily predict fibrosis progression. Likewise, an association with a noninvasive score may not equal an association with histological fibrosis. The term severe disease should identify the outcome actually measured.

Table 1. Diagnostic constructs that should remain distinct

Construct	Typical assessment	Main caution
Low relative muscle mass	Appendicular mass divided by weight or BMI	Fat gain can lower the ratio without absolute muscle loss
Clinical sarcopenia	Strength with confirmatory quantity or quality	Mass-only studies do not capture the full construct
Severe sarcopenia	Additional poor physical performance	Cannot be inferred from mass alone
Hepatic steatosis	Ultrasound, attenuation, imaging, or validated indices	Method-dependent sensitivity and classification
Steatohepatitis	Histological activity in appropriate settings	Steatosis alone is not equivalent
Fibrosis	Biopsy, elastography, or validated noninvasive pathways	Thresholds and score components affect comparability

Source: Cruz-Jentoft et al. (2019), Rinella et al. (2023), and EASL-EASD-EASO (2024).

This distinction is essential for causal reasoning. If the muscle exposure and liver endpoint each contain metabolic variables, a strong association can partly reflect shared components. An appropriate analysis must examine the underlying measurements, not merely the labels applied to them.

3 METHODOLOGY

3.1 Review design and retrieval

This manuscript is a structured critical review of PubMed-indexed evidence, including published meta-analyses and selected clinical studies. It does not report a registered systematic review or a newly

calculated primary-study meta-analysis. Bibliographic records and published summaries were verified on 7 October 2026.

The main query combined sarcopenia or skeletal muscle mass with NAFLD, MASLD, or nonalcoholic fatty liver in title and abstract fields, with a publication-date limit ending on the verification date. It returned 473 records. This number describes the query output only. The complete set was not independently screened; targeted retrieval identified relevant meta-analyses, longitudinal studies, biopsy-based reports, diagnostic consensus, and a guideline summary. Eighteen publications were retained as the reference set.

The extraction basis consisted of verified PubMed information and available published summaries. Data recorded included study design, diagnostic framework, outcome direction, reported effect estimate, confidence interval, and population context. Numerical results available only in inaccessible supplementary material were not reconstructed. The selection consequently provides an interpretable evidence map rather than an exhaustive estimate of all published studies.

3.2 Eligibility and scope

Priority was given to adult clinical evidence linking muscle phenotypes to steatotic liver disease or fibrosis. Contemporary consensus and guideline publications were included to define clinical constructs and contextualize historical studies. Historical NAFLD results were retained in their original terminology. They were not relabeled as observations from a retrospectively verified MASLD cohort.

Mechanistic statements were drawn from indexed clinical reviews and used as explanatory hypotheses. They were not treated as proof that a specific muscle intervention changes hepatic outcomes. General cirrhosis and acute liver failure evidence was outside the principal synthesis unless directly applicable to the steatotic liver question.

3.3 Statistical interpretation

The quantitative synthesis presents published estimates without combining overlapping reviews. Odds ratios remain odds ratios; hazard ratios from longitudinal studies remain hazard ratios. A common direction of association is not a license to pool these quantities across different time structures, thresholds, or adjustment sets.

For graphical display, reported confidence limits were retained and odds-ratio axes were logarithmic. The reference line is one, the null value for a ratio measure. Different outcome directions are

labeled explicitly. No new overall estimate, prediction interval, publication-bias test, or formal certainty grade was calculated. Where authors used the language of risk for cross-sectional results, this review instead interprets the estimates as associations with prevalent disease.

Table 2. Prespecified interpretation boundaries

Evidence type	Question addressed	Question not resolved
Cross-sectional association	Do muscle and liver phenotypes coexist?	Which occurred first?
Biopsy cohort	Is muscle phenotype associated with histological severity?	Does improving muscle reverse fibrosis?
Longitudinal cohort	Does baseline or changing muscle precede a liver outcome?	Is the relationship free of confounding?
Published meta-analysis	What aggregate association was reported?	Are overlapping reviews independent?
Regional subgroup	Does the estimate differ in sampled settings?	Is geography itself the causal factor?
Guideline or consensus	How should clinical constructs be defined?	What is a new intervention's effect?

Source: Review interpretation framework applied to the indexed publications.

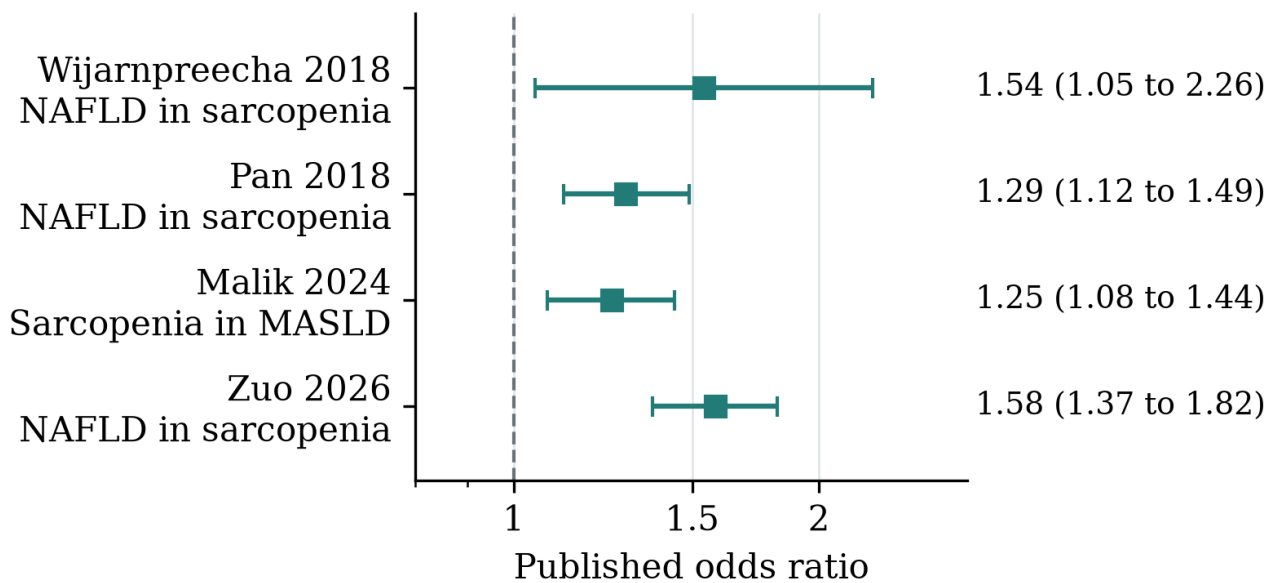
Methodological appraisal focused on phenotype definition, shared metabolic covariates, reverse causation, selection, and applicability. No numerical risk-of-bias scores were assigned. Reported concerns should therefore be read as qualitative critical appraisal rather than as a validated instrument-based classification.

4 RESULTS AND DISCUSSION

4.1 Published associations with steatotic liver disease

The early synthesis by Wijarnpreecha et al. (2018) reported higher odds of NAFLD among people with sarcopenia, with substantial heterogeneity. Pan et al. (2018) found an association in the same direction but of smaller magnitude. The newer review by Zuo et al. (2026) also reported a positive association. Agreement in direction is informative, but these reviews share historical studies and should not be counted as independent populations.

Figure 1. Published associations between muscle impairment and steatotic liver disease.



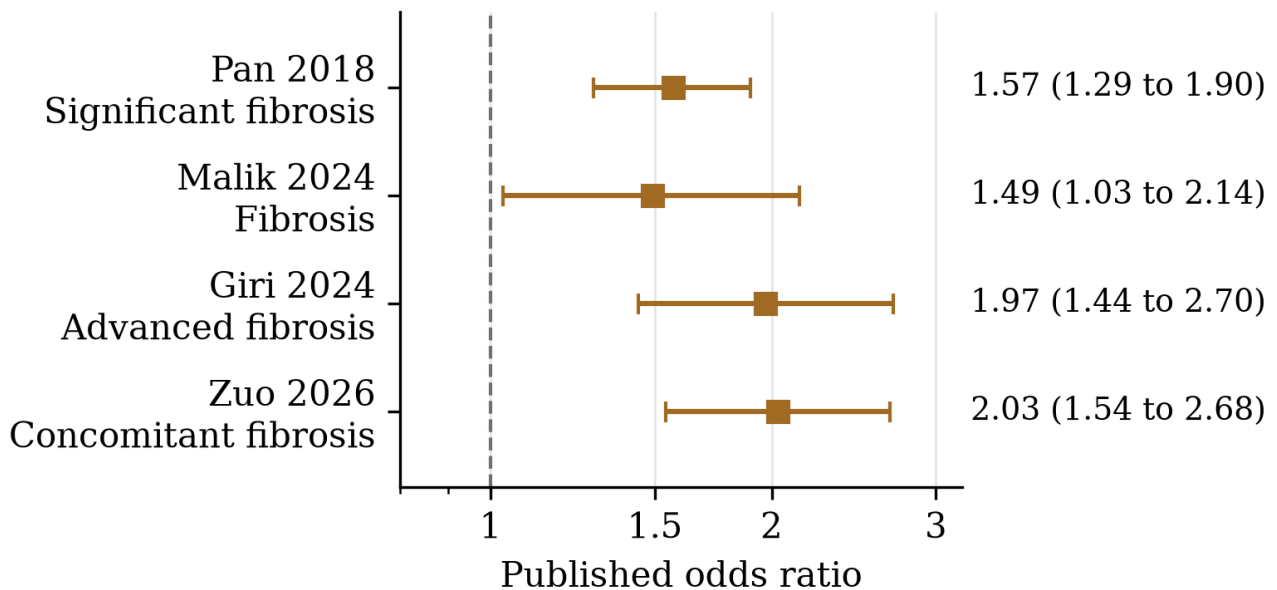
Source: Wijarnpreecha et al. (2018), Pan et al. (2018), Malik et al. (2024), and Zuo et al. (2026). Each row retains its source comparator direction. Confidence intervals are reported values; no new pooled estimate is calculated.

Malik et al. (2024) summarized a comparison framed as sarcopenia in MASLD versus controls. Its direction is labeled separately because the presence of an association does not identify a longitudinal causal pathway. Differences between reviews can arise from changing inclusion sets and from the diagnostic ratios used to define muscle impairment.

4.2 Fibrosis is a separate clinical endpoint

Published meta-analyses also associate sarcopenia with liver fibrosis. Pan et al. (2018) reported an odds ratio of 1.57 for significant fibrosis, whereas Giri et al. (2024) reported 1.97 for advanced fibrosis. Malik et al. (2024) reported 1.49 for fibrosis in its selected MASLD-related evidence, and Zuo et al. (2026) reported 2.03 for concomitant fibrosis.

Figure 2. Fibrosis associations reported by selected published meta analyses.



Source: Pan et al. (2018), Malik et al. (2024), Giri et al. (2024), and Zuo et al. (2026). Fibrosis definitions differ; the plot summarizes published estimates without averaging them.

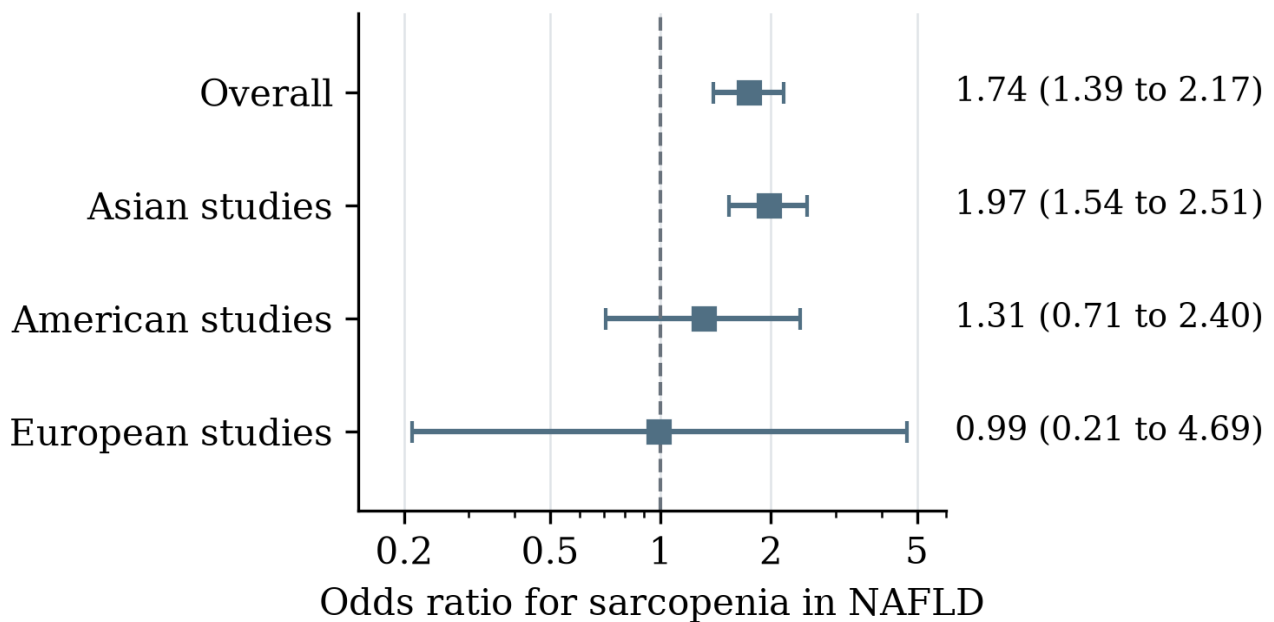
These estimates suggest that muscle phenotype is relevant beyond the presence of steatosis. Nevertheless, significant and advanced fibrosis are not identical, and noninvasive scores are not interchangeable with histology. A cross-sectional fibrosis association also cannot establish progression. The difference matters because progression is a temporal outcome requiring repeated assessment or a longitudinal clinical endpoint.

Giri et al. (2024) highlighted differences by fibrosis assessment method, reinforcing the need to interpret the endpoint before interpreting the pooled number. A uniform label cannot eliminate measurement heterogeneity.

4.3 Regional variability

Deng et al. (2024) evaluated sarcopenia prevalence in NAFLD and reported an overall association, with different regional subgroup estimates. The Asian subgroup had a more precise positive association, whereas the American and European confidence intervals included the null. The latter results should not be interpreted as proof of no relationship in those populations.

Figure 3. Regional subgroup estimates for sarcopenia in NAFLD populations.



Source: Deng et al. (2024). Published odds ratios and confidence intervals are displayed. Regional categories describe included studies and do not establish ethnic or geographical causality.

Study region can stand in for diagnostic practices, recruitment, age structure, adiposity distributions, and muscle thresholds. A between-region pattern can therefore reflect measurement and selection as well as biological or environmental variation. Wide intervals signal limited precision and should temper strong conclusions from apparent numerical differences.

The analysis reinforces a practical point: a pooled global estimate may obscure variation in the phenotype being studied. Future comparisons should use harmonized definitions and examine the individual characteristics represented by regional categories rather than treating geography as an explanatory mechanism in itself.

4.4 Primary studies of liver severity

Koo et al. (2017) linked a relative muscle-mass definition to histological severity in a biopsy-based NAFLD cohort. The association with significant fibrosis persisted after adjustment for selected metabolic factors. Petta et al. (2017) similarly found an association with severe fibrosis in a Western biopsy cohort. These studies provide clinically relevant endpoints but arise from populations selected for detailed liver assessment.

Lee et al. (2016) used Korean national survey data and noninvasive fibrosis models. The survey design improves population coverage, while the outcome remains a score-defined surrogate rather than biopsy-confirmed fibrosis. Harring et al. (2023) evaluated a United States survey population using elastography and demonstrated an unfavorable profile in participants with both sarcopenia and NAFLD.

Table 3. Selected clinical studies and their measurement frameworks

Study	Muscle framework	Liver endpoint
Koo et al. 2017	Appendicular muscle mass relative to weight	Biopsy-defined steatohepatitis and significant fibrosis
Petta et al. 2017	Bioimpedance muscle index relative to weight	Histological severe fibrosis
Lee et al. 2016	DXA-derived muscle index relative to BMI	Noninvasive fibrosis prediction models
Harring et al. 2023	FNIH-based sarcopenia definition	Transient-elastography fibrosis thresholds
Alferink et al. 2019	DXA, height-adjusted index, and muscle function	Ultrasound-defined NAFLD

Source: Verified PubMed records. DXA, dual-energy X-ray absorptiometry; FNIH, Foundation for the National Institutes of Health.

Their findings should be compared through these methods rather than reduced to a single interchangeable sarcopenia exposure. Selection and endpoint differences are plausible reasons for variation in effect magnitude.

4.5 Longitudinal evidence

Longitudinal observations add temporal information that cross-sectional analyses cannot provide. Kim et al. (2018) examined relative skeletal muscle mass and its change in a seven-year health-check cohort. Higher baseline muscle index and more favorable changes were associated with lower incidence of score-defined NAFLD, while baseline liver disease resolution was also examined. These findings are consistent with a protective relationship but remain observational.

Lee et al. (2019) followed initially NAFLD-free participants over approximately ten years and linked changing body composition to incident ultrasound-defined disease. The study helps establish that unfavorable muscle and fat trajectories can precede detected fatty liver. It cannot isolate a muscle effect from the broader changes in activity, diet, metabolic health, or adiposity accompanying those trajectories.

An important issue is the distinction between relative and absolute change. If body weight decreases while muscle mass is preserved, the relative muscle index can improve without substantial muscle gain. Conversely, fat accumulation can reduce the ratio despite stable absolute muscle. Longitudinal studies should report these components separately to clarify the biological interpretation.

Resolution defined through a predictive index also requires caution. If the index contains metabolic variables that improve with lifestyle change, its movement can partly reflect those components rather than a direct measurement of liver fat. Imaging and biochemical changes can provide complementary information, but they should not be treated as identical outcomes.

The longitudinal literature therefore strengthens the rationale for examining muscle health during liver risk assessment. It does not yet establish that an intervention producing muscle hypertrophy independently prevents steatohepatitis or reverses fibrosis. That question requires controlled interventions with direct muscle, metabolic, and liver measurements over an appropriate follow-up period.

4.6 Adiposity and the muscle denominator

The Rotterdam Study provides an important counterpoint to studies reporting uniformly positive sarcopenia associations. Alferink et al. (2019) found that clinically defined presarcopenia and sarcopenia were not associated with NAFLD in their elderly European sample, while fat distribution was a strong correlate. Some associations involving skeletal muscle index depended on sex, body weight, and adjustment for adiposity distribution.

This result should not be dismissed as an outlier. It illustrates how a more complete body-composition model can change the interpretation of muscle-related associations. The association may be stronger when muscle mass is expressed relative to a denominator that also captures the metabolic burden of fat. A height-adjusted index and a weight-adjusted index do not identify exactly the same individuals.

Table 4. How indexing choices can influence inference

Measure	What changes the value?	Potential interpretive problem
Absolute muscle mass	Muscle tissue quantity and body size	Larger bodies can have more muscle without better function
Muscle divided by height squared	Muscle relative to stature	May not reflect muscle adequacy relative to adiposity

Measure	What changes the value?	Potential interpretive problem
Muscle divided by weight	Muscle and all contributors to total weight	Fat gain can mimic worsening relative muscle status
Muscle divided by BMI	Muscle with weight and height scaling	Requires attention to shared metabolic covariates
Muscle strength	Neuromuscular and functional capacity	Can change without parallel mass change
Physical performance	Integrated functional ability	Also affected by cardiovascular and musculoskeletal disease

Source: Interpretation of the measurement frameworks in Cruz-Jentoft et al. (2019), Lee et al. (2016), and Alferink et al. (2019).

The most informative research would measure mass, strength, function, and adiposity together. This permits distinction between low muscle quantity, poor muscle performance, and an unfavorable muscle-to-fat balance.

4.7 Shared mechanisms and alternative explanations

The muscle-liver relationship is biologically plausible because both organs participate in insulin-sensitive metabolism and communicate through circulating mediators. Muscle-derived signaling, inflammatory activity, reduced mobility, and changes in adipose tissue can create pathways through which muscle health and liver fat move together. The clinical reviews by Kim and Kim (2020) and Polyzos et al. (2023) describe these interacting systems.

However, a coherent biological model is compatible with several causal structures. Muscle impairment might contribute to liver disease; liver disease might contribute to inactivity and muscle loss; or shared exposures might produce both. In advanced disease, nutritional and systemic consequences can further strengthen reverse pathways. Different stages may therefore involve different dominant mechanisms.

Adjustment for insulin resistance and adiposity is also conceptually difficult. These variables may be confounders, mediators, or components of the exposure definition. A model that adjusts for them can answer a different question from one that leaves them unadjusted. The persistence of an association after adjustment does not automatically prove an independent causal effect, particularly when measurements are imprecise.

A useful interpretation separates the total association from a proposed direct pathway. The total association can justify attention to muscle health in a liver population even before causality is settled. A direct intervention claim requires stronger evidence because changing muscle may not change all the shared determinants represented by the observational phenotype.

This distinction helps avoid an overly linear message that muscle loss necessarily causes fibrosis. The evidence supports an interconnected metabolic and functional phenotype. It also supports testing whether maintaining muscle function, reducing adverse adiposity, and improving activity influence liver outcomes together. Those hypotheses should be evaluated prospectively rather than inferred from cross-sectional odds ratios.

4.8 Integrated clinical assessment

The practical value of the association is to broaden assessment beyond liver enzymes and body mass index. A person with steatotic liver disease may have functional muscle impairment despite an apparently adequate or high body weight. Conversely, a low muscle index should not replace assessment of the liver or establish advanced fibrosis by itself.

Contemporary liver guidance recommends structured case finding and sequential noninvasive fibrosis assessment in appropriate cardiometabolic populations. Muscle evaluation can complement that process by identifying low strength, limited physical function, or adverse body composition. The available association studies do not validate a stand-alone muscle threshold as a liver fibrosis diagnostic test (EASL-EASD-EASO, 2024).

Table 5. Complementary assessment domains

Domain	Clinical question	Role in the combined phenotype
Muscle strength	Is there clinically relevant functional impairment?	Identifies a core sarcopenia feature
Muscle quantity or quality	Is impairment accompanied by structural deficit?	Supports phenotype confirmation
Physical performance	Does impairment limit integrated function?	Characterizes functional severity
Adiposity and metabolic health	Which cardiometabolic exposures coexist?	Clarifies shared risk context

Domain	Clinical question	Role in the combined phenotype
Liver fibrosis assessment	Is clinically important fibrosis likely?	Establishes hepatic risk through validated pathways
Nutrition and activity	Which modifiable factors affect both systems?	Supports individualized management planning

Source: Cruz-Jentoft et al. (2019) and EASL-EASD-EASO (2024), interpreted alongside the selected association studies.

This framework avoids treating one organ as a proxy for the other. It also makes follow-up more informative: a patient can improve strength without a large weight change, or improve metabolic risk without an immediate measurable change in fibrosis. Each outcome should be assessed through its own appropriate measure.

4.9 Intervention implications

The observational relationship makes muscle preservation a relevant concern in the care of people with MASLD. It does not establish that any particular muscle-directed treatment reverses liver fibrosis. Lifestyle strategies can affect body composition, insulin sensitivity, and activity together, so their hepatic effects should not automatically be attributed to muscle gain alone.

Weight reduction presents a particularly important measurement problem. A relative muscle index can improve as body weight falls even when some lean tissue is lost. Reporting only the ratio can therefore conceal an unfavorable absolute muscle trajectory. Strength and function help determine whether the intervention preserves clinically meaningful muscle health.

Nutrition and exercise should be considered within the patient's broader clinical context, including comorbidity and functional capacity. The selected guideline summary supports lifestyle and cardiometabolic management, while the sarcopenia consensus supplies the functional framework for identifying muscle impairment. Together they support coordinated assessment, but not a universal treatment prescription derived from the observational pooled estimates (Cruz-Jentoft et al., 2019; EASL-EASD-EASO, 2024).

Polyzos et al. (2023) discussed treatment considerations for coexisting sarcopenia, sarcopenic obesity, and NAFLD. Such discussions are useful for hypothesis formation but should be read in their publication context. Drug efficacy, approval, and liver indications evolve; a mechanistic or historical review is not evidence that a medication treats the combined phenotype under every current regulatory setting.

Future trials should specify whether the intervention aims to improve strength, increase muscle quantity, reduce fat, or improve liver disease. Joint improvement is clinically desirable, but the relative contributions require direct measurement. Randomized comparisons with prespecified liver endpoints would determine whether muscle-directed strategies add benefit beyond general cardiometabolic care.

4.10 Research priorities

The strongest next step is harmonization of phenotypes. Trials and cohorts should report muscle strength, quantity or quality, and physical performance rather than relying on a mass-only label. Liver eligibility should use contemporary terminology while retaining enough information to compare results with historical NAFLD cohorts.

Table 6. Priorities for prospective muscle and liver research

Uncertainty	Design requirement	Informative endpoint
Mass versus clinical sarcopenia	Joint measurement of quantity, strength, and performance	Phenotype-specific liver associations
NAFLD to MASLD transition	Explicit contemporary diagnostic criteria	Reproducible eligibility and transportability
Fibrosis causality	Longitudinal direct or validated fibrosis assessment	Progression, regression, and clinical events
Adiposity confounding	Separate muscle and fat components	Effects beyond denominator-driven changes
Intervention efficacy	Controlled muscle-directed intervention	Hepatic benefit alongside functional improvement
Population variability	Diverse recruitment and harmonized thresholds	External validity across clinical settings

Source: Research implications of the selected clinical evidence.

Repeated measurement is essential because a baseline association can reflect a transient or longstanding phenotype. The order of changes in muscle, metabolic variables, liver fat, and fibrosis should be documented. This would support more credible examination of mediation and reverse pathways.

Reporting should include absolute measures, effect estimates with uncertainty, missing-data procedures, and adverse consequences of weight or muscle change. A core set of measures would make

later meta-analyses more interpretable and reduce the tendency to pool studies that use the same word for different underlying exposures.

4.11 Limitations of this review

The evidence set was selected through topic-based and targeted retrieval rather than a complete independently screened systematic search. The broad query count therefore does not constitute a PRISMA flow. The review may omit relevant primary studies or syntheses, particularly those using alternative terminology for muscle function or steatotic liver disease.

Extraction relied on verified PubMed records and available published summaries. This supports traceable references and source-level results but limits detailed appraisal of covariate models, outcome thresholds, missing observations, and within-review participant overlap. No formal review-quality score or certainty grade was assigned.

Many studies use historical NAFLD definitions and mass-based sarcopenia labels. The manuscript treats these as clinically relevant evidence while avoiding retrospective confirmation of contemporary diagnoses. This necessarily limits claims specifically about patients meeting present MASLD and functional sarcopenia criteria simultaneously.

The published meta-analyses are overlapping evidence summaries. Their participant totals cannot be added to form a unique sample, and their pooled estimates cannot be treated as independent observations for another meta-analysis. Differences in association direction, fibrosis stage, diagnostic modality, and adjustment further limit quantitative comparability.

Cross-sectional associations dominate important parts of the literature. Even longitudinal studies remain vulnerable to residual confounding and measurement coupling through relative muscle indices or liver prediction scores. Temporal ordering improves inference but does not replace controlled intervention evidence.

The review consequently supports integrated assessment and further research rather than a claim that muscle improvement itself reverses fibrosis. Its figures present reported associations, not newly calculated causal effects or individualized risk predictions. These boundaries are necessary to preserve the distinction between a reproducible association and an established therapeutic pathway.

5 CONCLUSION

Published clinical syntheses generally associate muscle impairment with steatotic liver disease and with higher odds of fibrosis. The relationship is relevant to metabolic and functional assessment, but its magnitude depends on muscle definitions, liver endpoints, population selection, and diagnostic modality. Contemporary clinical sarcopenia cannot be inferred from a low relative muscle-mass ratio alone.

Historical NAFLD evidence provides an important foundation for understanding MASLD, while explicit diagnostic differences should remain visible. The strongest inference is that muscle health and liver disease often form part of an interconnected phenotype. The evidence is less secure regarding causal direction and whether muscle-directed intervention independently prevents or reverses hepatic fibrosis.

Clinical assessment should therefore combine strength and function with body composition, cardiometabolic evaluation, and validated liver fibrosis pathways. Research should prioritize contemporary definitions, separate reporting of muscle and fat changes, diverse populations, and controlled longitudinal outcomes. These steps would establish whether preserving or restoring muscle health adds hepatic benefit beyond general metabolic management.

Data and methodological transparency

All references correspond to verified PubMed-indexed publications. The figures were created from numerical estimates reported in the cited sources and retain their original outcome structures. No patient-level observations, new pooled estimate, formal certainty rating, or research-registration claim is presented. Primary studies are used for clinical interpretation and are not counted as independent additional evidence beyond reviews in which they may already appear.

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