

Review

Nutritional Assessment and Management in MASLD: A Practical Guide for Primary Care

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Abstract

Background: Nutrition and nutrition-related interventions are a salient yet under-recognized component of preventing the development of and complications of metabolic dysfunction-associated steatotic liver disease (MASLD). Additionally, malnutrition affects a significant proportion of patients with MASLD, yet remains under-treated in the primary care setting. The objective of this narrative review is to synthesize the available evidence regarding nutritional screening, diet, and supplements in patients with MASLD. This review is aimed at primary care physicians to improve the recognition of malnutrition in MASLD patients and provide appropriate nutritional guidance. **Methods:** Between November 2025 and June 2026, the authors conducted a literature review using the PubMed database. Search terms included combinations of the following: MASLD, non-alcoholic fatty liver disease (NAFLD), malnutrition, sarcopenia, nutritional screening, Mediterranean diet, micronutrients, and physical activity. Randomized controlled trials (RCTs), systematic reviews, and meta-analyses addressing nutritional outcomes in liver disease were prioritized, and consensus guidelines were incorporated where primary trial evidence was limited. Evidence was appraised by study design, risk of bias, and endpoint; recommendations resting on expert opinion or extrapolation from cirrhosis populations are identified as such. **Results:** Author review of the studies yielded the following conclusions. Nutritional assessment should be risk-stratified by fibrosis stage using liver disease-specific tools, as BMI and body weight are unreliable in MASLD. Weight loss has been shown to improve histological and clinical progression in MASLD. The Mediterranean diet has the strongest support for steatosis reduction among the dietary interventions discussed. Coffee consumption is consistently associated with reduced fibrosis risk in observational data, but no randomized evidence exists. GLP-1 receptor agonists achieve histological MASH resolution in up to 63% of patients but require concurrent nutritional monitoring to mitigate lean mass loss. Systemic barriers, including time constraints and limited dietitian access, remain the primary impediment to primary care implementation. **Conclusions:** Primary care physicians are central in managing many aspects of care for patients with MASLD. This review highlights the established evidence behind diet, exercise and malnutrition prevention in the primary care setting.



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1. Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) represents a spectrum of liver disease with a substantial worldwide burden, affecting approximately 30%

of the global adult population [1]. Previously known as non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH), this terminology has been changed to reflect the relationship of this disease with other diseases of the metabolic syndrome [2,3]. It is routinely first encountered in the primary care setting. Lifestyle modifications, including nutritional adjustments and exercise, are the cornerstone of treatment for this disease [4].

Although seen less frequently in the earlier stages of MASLD, malnutrition is both a frequently under-recognized complication of MASLD and a predisposing factor for end-stage liver disease (ESLD) [5,6]. Sarcopenia, present in 30–70% of patients with end-stage liver disease, is an independent risk factor for mortality in patients with cirrhosis and is associated with increased risk of cirrhosis progression, hepatic encephalopathy, infection, and hospital re-admission and delays in liver transplant [6,7]. Despite these serious consequences, malnutrition often goes undetected and unmanaged as MASLD patients are commonly perceived as overnourished [2]. The poor prognostic value of malnutrition and sarcopenia in MASLD makes primary and secondary prevention through nutritional assessment and intervention important.

Primary care physicians (PCPs) are uniquely positioned to manage nutritional status in MASLD patients and are increasingly recognized by hepatology societies as key stakeholders [8]. They can leverage established therapeutic relationships and understand their patients' liver disease in the context of other chronic conditions. Socioeconomic and healthcare system barriers may prevent the adoption or implementation of nutritional guidance. Understanding and addressing these barriers is essential for optimizing nutritional management in routine practice, as further delineated in this review.

This narrative review provides evidence-based recommendations for nutritional screening, assessment, dietary interventions, and management of special situations in MASLD. This review addresses adults only; nutritional assessment and management of pediatric steatotic liver disease differ substantially and are beyond its scope. Clinicians caring for children with MASLD should consult pediatric-specific guidance. This scope was determined by consensus from all study authors. Existing guidelines by the major hepatology societies address gastroenterologists and hepatologists; in contrast, this review focuses on the primary care setting.

Specifically, this review answers the following questions:

1. How and how often should nutrition and diet be assessed in patients with MASLD?
2. What roles do diet and weight loss play in MASLD prevention and progression? What is the role of micronutrients and supplement use in this population?
3. What is the recommended nutritional strategy for patients with ascites and clinically significant portal hypertension? What is the role of incretin-based therapy in this population?

2. Methodology

The review was conducted in accordance with the principles of narrative evidence synthesis as outlined below.

Literature Search. A literature search was performed between 1 November 2025 and 31 June 2026 in PubMed to identify pertinent studies. PubMed queries included the following combination of search terms: *metabolic dysfunction-associated steatotic liver disease, MASLD, NAFLD, nonalcoholic fatty liver disease, cirrhosis, nutritional assessment, malnutrition, sarcopenia, sarcopenic obesity, nutritional screening tools, Mediterranean diet, dietary intervention, micronutrient, physical activity, exercise, hepatic encephalopathy, and FIB-4*. In accordance with the aims of this study to serve as a narrative review, a formalized systematic review and meta-analysis were not conducted.

Study Selection. Priority was given to randomized controlled trials (RCTs), systematic reviews, and meta-analyses addressing nutritional outcomes in MASLD or cirrhosis populations. Consensus statements and clinical practice guidelines from the American Association for the Study of Liver Diseases (AASLD), European Association for the Study of the Liver (EASL), American College of Gastroenterology (ACG), and the European Society for Clinical Nutrition and Metabolism (ESPEN) were incorporated where primary trial evidence was limited.

Evidence Appraisal. Evidence quality was assessed based on study design, sources of bias, applicability of results, and study endpoints. Conclusions supported exclusively by expert consensus, non-peer-reviewed resources, or conclusions extrapolated from non-MASLD populations are delineated. Evidence related to non-MASLD patient populations is explicitly delineated in the text.

3. Assessing Nutritional Status

Characterizing nutritional status in MASLD patients requires a specialized approach distinct from the general population or patients with isolated components of the metabolic syndrome [9]. As nutritional interventions are key risk-modifiers, assessing nutritional status is a key part of disease management. This approach is also detailed in Figure 1.

Nutritional assessment in MASLD patients has not been studied beyond general recommendations for patients with metabolic syndrome. No studies were identified in the literature review that examined the assessment interval or specific components of assessment in this patient population. Yearly assessment is recommended by the United States Preventive Services Taskforce (USPSTF) in patients with hyperlipidemia, hypertension, elevated blood pressure or other risk factors for cardiovascular disease (CVD) [10]. The USPSTF recommendations were developed based on a small but adequate amount of evidence that nutritional guidance by PCPs would reduce CVD events and improve secondary outcomes of metabolic disease. In patients with MASLD, no RCT or prospective study has shown that screening intervals impact MASLD-related outcomes. Based on expert opinion, screening intervals should be made based on clinical assessment and provider suspicion for liver fibrosis progression, Table 1 [11]. Patients with mild–moderate MASLD, a Fibrosis 4 (FIB-4) score between 1.3 and 2.67 or a Vibration-Controlled Transient Elastography (VCTE) less than 12 kilopascal (kPa), meet the USPSTF criteria for yearly screening [4,10]. Screening at this stage provides clinicians with an opportunity to address risk factors and prevent further fibrotic progression. Patients with clinically significant liver fibrosis (VCTE greater than 12 kPa or magnetic resonance elastography (MRE) greater than 3.63 kPa, or FIB-4 score greater than 2.67) may need more frequent screening [5,12]. Bi-annual nutritional assessment is adapted from existing guidance on malnutrition-prevention in cirrhotic patients, as no evidence was found in patients with uncomplicated MASLD. Additionally, no evidence was found that supported routine nutritional assessment for MASLD patients presenting as inpatients. Nutritional interventions and assessments are more thoroughly addressed in the outpatient setting.

Nutritional assessment is a multi-pronged approach aimed at characterizing a patient's dietary habits, supplement usage, and substance use. These practices should inform providers' approach in assessing risk for liver fibrosis, screening for malnutrition, and identifying areas of further intervention [9,13]. Dietary recall is one of the first steps in nutritional assessment. Standardized screening tools, including the 3-day food diary and 24 h food recall, are often utilized in the primary care setting [12]. However, no studies were identified that examined the validity of these tools in patients with MASLD. Despite the lack of evidence, these tools are simple and have been frequently employed in general practice to collect detailed histories. A 48-item short food-frequency questionnaire specific to patients

with MASLD has been developed, with a recent study validating it against standard dietary recall tools [14]. In addition to standardized questionnaires, clinical practice guidelines suggest providers pay attention to maladaptive dietary habits during the visit. This includes the consumption of sugary or sweetened beverages, ultra-processed foods, saturated fats, and protein intake, along with any alcohol consumption, as these practices are associated with an increased risk for MASLD development [4,13]. Additionally, clinical guidelines suggest cataloguing the patient's daily fluid consumption, meal timing and number may be helpful in counseling patients [5]. Alcohol consumption should be quantified by self-report, standardized questionnaires such as the Alcohol Use Disorders Identification Test Consumption (AUDIT-C tool) [15]. This approach can identify patients with MASLD who are at risk of developing Metabolic and Alcohol-Related Liver Disease [16].

Table 1. Risk-stratified nutritional assessment and management approach in MASLD.

Risk Level	FIB-4 Score	Frequency	Recommended Tools	Key Interventions
Low	<1.30	Every 1 year	BMI, waist circumference, dietary history	Mediterranean diet, exercise counseling
High	>2.67	Every 6 months	PG-SGA, handgrip strength, RFH-NPT, SMI (if imaging available), MAMC	Mediterranean diet, protein optimization, weight loss if BMI ≥ 27 ; protein 1.0–1.5 g/kg/day, total 30–35 kcal/kg/day, micronutrient screening, sarcopenia assessment

3.1. Defining Malnutrition

Malnutrition is broadly defined as either a deficit in the quantity or quality of calories and micronutrients, or an isolated deficiency in a particular micronutrient with associated clinical manifestations [5,13]. Malnutrition is an umbrella term encompassing sarcopenia and cachexia, terms often used interchangeably but requiring distinct assessment approaches.

Sarcopenia is characterized by muscle wasting due to an imbalance between protein catabolism and anabolism. Sarcopenia develops in MASLD through various mechanisms, including anorexia, pro-inflammatory cytokine cascades, or dietary maladaptation [17–19]. One subtype of sarcopenia in MASLD is sarcopenic obesity: patients with high BMI and excess adiposity who simultaneously have significant muscle wasting. This phenotype is especially common and problematic in MASLD, as the excess adiposity masks underlying muscle depletion, leading to delayed recognition and intervention [20]. As previously mentioned, sarcopenia is common in adults with cirrhosis and has independent implications for prognosis. The integration of sarcopenia into prognostic models like MELD-Sarcopenia has been proposed to improve the prediction of mortality [6,18,19,21]. Since malnutrition is variably characterized by clinicians, its true prevalence is difficult to quantify, making standardized screening approaches essential.

3.2. Malnutrition Screening Tools

Nutritional screening tools used in general practice include the Malnutrition Screening Tool (MST), Malnutrition Universal Screening Tool (MUST), and Rapid Eating Assessment (REAPS) [9,22]. As they fail to account for fluid retention, sarcopenia, and obesity, these tools are not recommended as the primary malnutrition screening method by clinical practice guidelines in liver disease [23]. Two tools, the Patient-Generated Subjective Global Assessment (PG-SGA) and the Royal Free Hospital Nutrition Prioritizing Tool (RFH-NPT), have been recommended by nutritional societies in screening for malnutrition in cirrhosis [5,16]. The PG-SGA tool combines patient self-assessments with provider assessments of weight, food intake, symptoms, and activity level. The Royal Free Hospital Nutrition Prioritizing Tool

(RFH-NPT) is recommended by AASLD due to its correlation with Child–Pugh Class Score, MELD, and overall mortality and morbidity [9,19,24]. This tool also accounts for enteral support, fluid overload, ascites, and symptom-limited eating in ESLD patients. However, as with the PG-SGA, the RFH-NPT has only been studied in cirrhosis and may not be appropriate in screening patients with uncomplicated MASLD.

3.3. Anthropometry and Muscle-Mass-Based Methods

Standard metrics like weight and BMI are not accurate measures of nutritional status in patients with MASLD [9]. Paradoxical overnutrition and sarcopenia, fluid accumulation, visceral adiposity, and ethnic variations in MASLD phenotype can obscure serial weight and BMI measurements [5,7,13]. Instead, muscle-mass quantification has been better validated as a means of identifying malnutrition and sarcopenia. CT-based skeletal muscle index (SMI) at the L3 vertebral body level is the gold standard for muscle-mass quantification [13,17,19]. SMI cut-offs of $50 \text{ cm}^2/\text{m}^2$ in men and $39 \text{ cm}^2/\text{m}^2$ in women define pathologically low muscle mass, although this has not been validated in patients with uncomplicated cirrhosis. Multicenter studies have established standardized definitions of sarcopenia in patients with end-stage liver disease using these CT-based measurements [17,25]. Other assessments include the Dual-Energy X-Ray Absorptiometry (DEXA) method, which can assess limb muscular mass but is limited by fluid retention. Mid-Arm Muscle Circumference (MAMC) and triceps skinfold offer quick bedside alternatives [13]. One recent cross-sectional study identified an inverse relationship between skeletal muscle mass/weight ratio calculated via bioimpedance analysis and steatosis grade via ultrasound, suggesting the role of bioimpedance measurements in MASLD [13].

While these measurements correlate with surrogate measures of mortality and morbidity in ESLD patients, the clinical utility of muscle-mass assessment in MASLD patients is not well characterized. The use of quick bedside measurements of muscle mass, such as the MAMC and triceps skinfold, may help clinicians identify patients with suboptimal lean muscle mass.

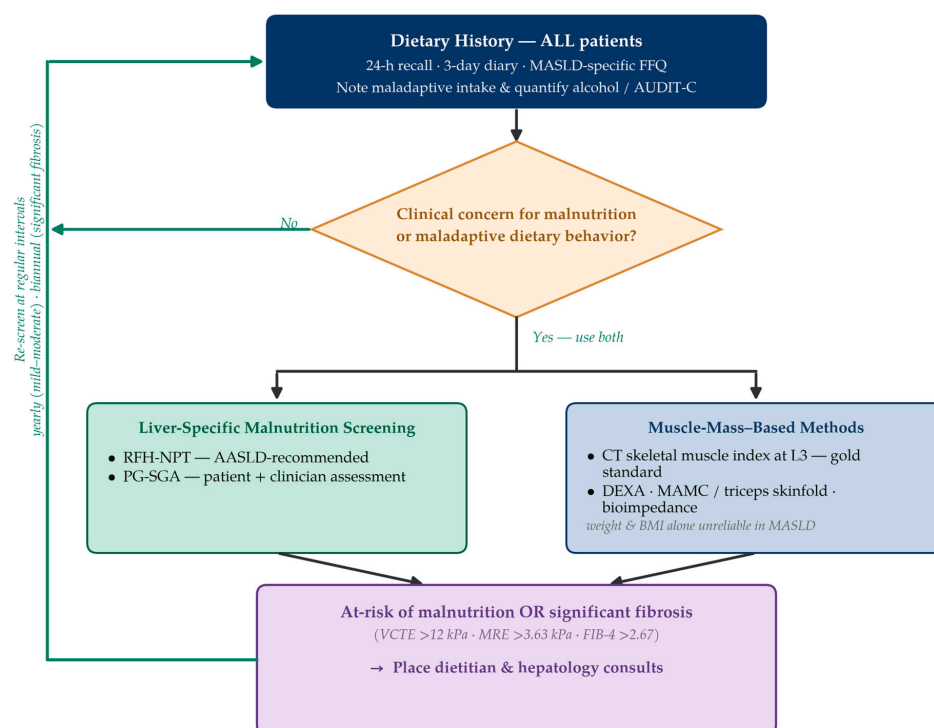


Figure 1. Suggested nutritional assessment and malnutrition screening algorithm in MASLD [5,9,12–14,17,19,22].

4. Diet and Weight Loss

4.1. Weight Loss Targets

Weight loss of 3–5% improves hepatic steatosis, 7–10% achieves MASH resolution, and sustained weight loss exceeding 10% can produce histologically confirmed fibrosis regression [26]. These thresholds represent clinically meaningful targets based on observational data that demonstrated improvement of steatohepatitis and steatosis based on histological endpoints [11,27]. For patients with BMI ≥ 30 or BMI ≥ 27 with metabolic comorbidities who fail lifestyle modification, GLP-1 receptor agonists have been utilized. Semaglutide works synergistically with nutritional interventions in reducing steatotic liver disease progression, as detailed in later sections [28]. Bariatric surgery can achieve substantial improvements, though long-term monitoring for nutritional deficiencies becomes essential [5,13].

4.2. Dietary Components

Evidence regarding the optimal consumption of fruits, vegetables, meat, and other food groups on the development of MASLD is reviewed below. Expert opinion suggests that following the standard USDA recommendations is reasonable for most major food groups [29]; however, further considerations are detailed below and in Table 2.

Table 2. Dietary evidence in MASLD.

Dietary Components	Practical Recommendations
Sugar-sweetened beverages	• Replace sodas with water, sparkling water, or unsweetened tea • Avoid fruit juices, sweetened teas, energy drinks, sports drinks • Use natural flavor enhancers (lemon, lime, cucumber, mint)
Fat intake	• Use olive oil instead of butter for cooking and baking • Choose olive oil-based salad dressings • Limit fatty red meat, processed meats, and full-fat dairy products • Choose lean poultry, plant proteins, low-fat dairy products
Maintain adequate protein: 1.0–1.5 g/kg ideal body weight daily	• Include protein source at each meal (lean meat, fish, eggs, legumes) • For a 70 kg patient: aim for 70–105 g protein daily • Never restrict protein, even with hepatic encephalopathy
Regular coffee consumption ≥ 2 cups daily	• Both caffeinated and decaffeinated coffee show benefits • Avoid adding sugar or high-calorie creamers • Black coffee, or with small amount of milk/unsweetened alternative

Several observational studies, but no randomized trials, have examined vegetable intake in this population. A recent cohort study in Southern Italy found that patients who consumed greater than 325 g/day of total vegetable intake had a 47.9% probability of not having MASLD compared to patients who consumed less than this amount. Furthermore, patients who consumed up to 35 g/day of green vegetables had a 39% reduced probability of MASLD, and consumption of 130 g of orange–red vegetables had a 54.3% probability of not having MASLD [30]. A similar cohort study in Korean adults also identified the beneficial effects of fruits and vegetables in reducing MASLD risk [31]. While promising, these findings are associative and no prospective studies with well-defined histological or surrogate endpoints have identified the impact of vegetable consumption on steatosis or fibrosis progression.

The standard USDA recommendation regarding fruit intake has been questioned in patients with MASLD due to specific compounds within fruits that may be either protective or harmful. A fruit-rich diet seems to have a beneficial effect on hepatic steatosis, until a threshold—a cohort study in Korean adults found an inverse association with fruit intake and fatty liver disease as reported by the Fatty Liver Index. However, a separate RCT found that greater than four servings of fruits per day worsened liver steatosis, dyslipidemia and hyperglycemia [31,32]. Some fruits, such as oranges, may be beneficial—one RCT found exploratory outcomes that over a 4-week period, consumption of 400 g of whole oranges a day led to a 30% decreased in ultrasound-derived measures of steatosis in MASLD subjects compared to controls [33]. While further data is needed, this data suggests that fruit consumption, in moderation, may be protective for hepatic steatosis.

Observational studies demonstrate that fiber intake, irrespective of fruit or vegetable intake, has a non-linearly negative relationship with MASLD [34,35]. Studies utilizing the National Health and Nutrition Examination Survey (NHANES) have demonstrated ORs of 0.12, 0.25, 0.41, and 0.42 between the consumption of total, cereal, fruit, and vegetable fiber, respectively, with MASLD diagnosis [34]. Additionally, in women and White patients within the NHANES database, consumption of fiber had linear relationships with reported Controlled Attenuation Parameters (CAPs) measured via FibroScan (EchoSens, Paris, France) [35]. Several burgeoning RCTs have examined the role of dietary fiber supplementation on hepatic steatosis—one conducted with supplementation of 12 g and 24 g of dietary cereal fiber found a significant reduction in FIB-4 score for participants [36]. Both observational, retrospective and prospective data seem to indicate the beneficial effects of fiber supplementation on hepatic steatosis; however, further studies are needed to examine optimal fiber amount and any associated changes in validated clinical or histological endpoints of liver fibrosis.

The timing of meals is posited to have an impact on MASLD development and further progression. Observational studies have identified the maladaptive role of overall meal irregularity and the beneficial role of small, frequent meals as opposed to larger meals. One such study found that 21% higher risk of MASLD development in patients who ate less than three times a day compared to more than four meals a day and 26% higher risk if nighttime fasting was restricted to less than 10 h, as compared to a fasting duration of 14 h or greater [37]. Nighttime fasting was again identified, along with regular breakfast timing, as an important factor in MASLD development by a prospective cohort study in 2025, with an HR of 1.26 in irregular breakfast eaters who ate at night, and an HR of 1.52 in never-breakfast eaters and nighttime snackers [38]. While these findings have not been examined in an RCT to date, one RCT examining the efficacy of time-restricted eating found a reduction in intra-hepatic triglyceride content by 6.9% and a reduction in liver stiffness by 2.1 kPa in patients who restricted eating over a 12-month period [39]. Together,

these studies suggest that time-restricted eating, meal regularity, and reducing nighttime snacking are beneficial for MASLD.

Carbohydrate and dairy consumption have both shown mixed results in the literature with regards to hepatic steatosis. Several studies, including one meta-analysis, have indicated that low-carbohydrate diets (diets with less than 130 g or <26% of total calories) may improve metabolic parameters associated with MASLD, including cholesterol, triglyceride, and insulin sensitivity [40]. These studies did not identify any benefits in validated endpoints of MASLD. Additionally, starch from refined grains may be associated with almost a 33% increased risk of liver fat, while starch from whole grains or simple sugars does not display a similar association [41]. Dairy consumption, particularly milk and yogurt, may display a lower risk of MASLD (by 2% and 26% in a study of Korean adults). Cheese consumption did not display a significant association in the included studies [42,43].

Meat consumption, including the optimal amounts of red meat and white meat, should be separated and explored by providers, given recent studies showing differential effects on liver steatosis between these sources of protein. Excessive red meat, in particular, has been shown to worsen hepatic steatosis, both at low levels of even 17 g/day and with increasing levels of consumption [44,45]. This effect has not been demonstrated with poultry or fish. Substitution of meat with plant-based protein, such as legumes, may be beneficial—one prospective cohort study identified a 3–4% decrease in the rate of fatty liver disease when substituting 80 g/week of red meat or processed meat with legumes [46].

Fat consumption is debated in this patient population. However, available guidance indicates that saturated fatty acids (SFAs) may be more detrimental than polyunsaturated fatty acids (PUFAs). In both obese and non-obese individuals, SFAs overfeeding led to an increase in liver fat measured via MRI as compared to PUFAs, despite similar amounts of weight gain, suggesting that the type of fat intake is important in steatosis [47,48].

Sugar-sweetened beverages (SSBs) such as sodas or fruit juice should also be addressed by providers—recent systematic reviews have demonstrated a pooled RR of 1.39 for patients who reported consuming SSBs for MASLD. The relationship is dose-dependent—pooled RR of patients consuming 1 SSB per week was 1.14 (CI 1.03–1.26), while the pooled RR of those consuming >7 cups per week was 1.53 (CI 1.05–2.22) [49]. The relationship between SSBs and MASLD seems to be mediated by the presence of visceral adipose tissue and confounded by BMI [50].

4.3. Dietary Protocols

The Mediterranean diet is currently recommended by multiple societies as the primary dietary protocol for patients with MASLD [4,5,13]. This diet emphasizes high intake of vegetables, fruits, nuts, unrefined cereals, legumes, and extra virgin olive oil; moderate to high fish intake; low to moderate fermented dairy products; and low intakes of saturated fats, red meat, and poultry. Despite these guideline recommendations, RCT data is conflicting. One RCT compared the Mediterranean diet to a low-fat diet in 24 patients and found that patients on the Mediterranean diet had significantly reduced hepatic fat (32% difference) based on MRI/MRS intrahepatic fat measurements. This study was limited by a smaller sample size, a limited follow-up of 6 weeks, and a lack of blinding [51]. Subsequently, a trial done in 2018 comparing the Mediterranean diet and the low-fat diet after 12 weeks found that the Mediterranean diet had no significant difference in hepatic steatosis measured via MRI proton density fat fraction (PDFF) compared to the low-fat diet [52]. In contrast, the DIRECT PLUS trial found that patients adherent to the Mediterranean diet had a 30% reduction in intrahepatic fat by 18 months compared to 20% in patients who adhered to a low-fat diet [53]. Recent trials have raised further questions, including the MEDINA trial, which compared the Mediterranean diet to standard healthy eating advice in patients with

MASLD and found no significant difference in hepatic fat reduction measured via magnetic resonance between these two dietary groups [54].

In addition to direct effects on hepatic steatosis, the Mediterranean diet is efficacious in improving insulin sensitivity and reducing other cardiovascular risk factors [51,55]. Adherence remains important, but even in trials where patients followed the Mediterranean diet *ad libitum*, there were comparable reductions in intrahepatic fat when compared to patients on a structured low-fat diet [51]. Follow-up qualitative analyses from the MEDINA trial and data from a trial adapting this diet to a Chinese population demonstrated that cost, flavor, adjustments in cooking style and cultural unfamiliarity are all barriers to implementing the Mediterranean diet [56].

Limitations of the Mediterranean diet are as follows. No RCT of the Mediterranean diet has utilized a histological endpoint—instead, all studies reviewed utilized imaging-based surrogate endpoints [27]. The variation in imaging-based endpoints may be one reason for the heterogeneity in study results identified above. Additionally, trials assessing the Mediterranean diet are limited by small sample sizes and poor generalizability to the general population. Adherence to the Mediterranean diet may be difficult outside a Mediterranean context, as previously mentioned. Follow-up duration is also limited—the benefit of the Mediterranean diet is seen only up to 2 years in follow-up in all the studies reviewed. Based on the available evidence, successful implementation of the Mediterranean diet in the primary care setting can improve secondary features of MASLD and potentially reduce hepatic steatosis.

The green Mediterranean diet, which includes plant-based polyphenol intake through the consumption of green tea, walnuts, and Mankai (a duckweed-derived supplement), has shown greater intrahepatic fat reduction than the standard Mediterranean diet. This effect was most notably demonstrated in the DIRECT-PLUS trial—though both diets achieved similar total weight loss [53]. While left out of the most recent guidelines, this data seems to support the addition of polyphenol-based compounds in select patients.

Lacto-Ovo Vegetarian Diet (LOV-D), a plant-based diet that excludes meat and fish, but includes dairy products, has also emerged as a popular alternative to standard healthy eating advice in MASLD. Several trials have found potentially beneficial effects of LOV-D on imaging-based surrogates of hepatic steatosis, both via ultrasound and via MRI-based PDFF measurements [57–59]. Both studies support the modifiable role of weight loss as a primary driver for the benefit of this diet in MASLD. The LOV-D's proposed benefit centers on the reduction in processed meats and improved plant-based fiber consumption, both of which may contribute to the loss of visceral and hepatic fat. While promising, LOV-D can make protein intake challenging in a population where protein intake is crucial. Additionally, adapting this diet to a wide variety of sociocultural groups may be difficult and requires further study.

The ketogenic diet is a low-carbohydrate diet that is meant to mimic a metabolic fasting state and thereby reduce insulin secretion and improve blood sugar control and thereby reduce hepatic steatosis [58]. In one study comparing the ketogenic diet to DASH, patients who were adherent to the ketogenic diet showed no significant difference in liver stiffness and steatosis despite significant differential reductions in weight and waist circumference [58,60,61]. Despite appearing to be an attractive option for weight loss, the proposed benefits of the ketogenic diet seem to be from reducing excess carbohydrates and processed food consumption. Both benefits are also seen in the Mediterranean diet. Additionally, extended compliance to this diet can be especially difficult and adherence has not been studied over longer periods of time.

The available evidence regarding popular dietary protocols suggests that the Mediterranean diet confers the greatest benefit to MASLD patients in reducing hepatic steatosis

and preventing fibrosis progression. While the green Mediterranean diet is promising and evidence from the DIRECT-PLUS trial showed even greater intra-hepatic fat reduction compared to the Mediterranean diet, compliance may be challenging. Mankai is not widely available, and sociocultural differences in dietary preferences already limit the Mediterranean diet from widespread adoption. Additionally, the cost of adherence has not been fully characterized in both diets and should be studied further. With regards to both practicality and clinical benefit, the available evidence behind the LOV-D and ketogenic diets is lacking. In comparison to the Mediterranean diet, both the ketogenic diet and LOV-D have not demonstrated similar reductions in steatosis.

5. Micronutrients

Patients with MASLD face increased risk of micronutrient deficiencies due to over- or under-nutrition due to malabsorption, maladaptive dietary habits or in later-stage disease, pro-inflammatory pathways [13]. Few studies have examined ideal screening frequency for micronutrient deficiencies; expert opinion suggests that PCPs follow general primary care guidelines for screening and appropriate repletion [5].

A daily goal of 1.1–1.2 mg/day thiamine (Vitamin B1) intake is reasonable in patients with MASLD [2]. While MASLD patients are at lower risk of thiamine deficiency than those with alcohol-related liver disease, it is reasonable to treat clinical or symptomatic thiamine deficiency as in the general population.

Vitamin A deficiency has been noted in patients with advanced MASLD, likely mediated by damage to the stellate cells that store vitamin A in the liver. The available evidence suggests focusing on dietary intake of vitamin A and carotenoids prior to targeted supplementation in patients with clinical deficiency, as over-supplementation can cause hepatotoxicity [62].

Vitamin D deficiency has increased prevalence in MASLD, with some reports of an incidence between 64 and 92% [5]. As with the general population, the reviewed studies and expert opinion support supplementation when serum 25-OH vitamin D levels are <20 ng/mL and supplementing until achieving a serum level of 30 ng/mL. While robust clinical trial data supporting the relationship between hypovitaminosis D and MASLD is still lacking, vitamin D's anti-fibrotic and anti-inflammatory effects are worth considering [63]. Vitamin D's insulin-sensitizing and anti-fibrotic effects make it an attractive adjunct for supplementation.

Vitamin E: Vitamin E supplementation demonstrated 20% greater histological improvement in non-diabetic, non-cirrhotic MASH patients compared to controls, though no data supports slowing fibrosis progression. Providers may consider routine supplementation with 800 IU in the right clinical settings [5,13,64].

Copper: Copper supplementation is not recommended in recent ACG and ESPEN guidelines for patients with uncomplicated MASLD [5,13]. Instead, marginal copper status and excess fructose consumption appear to have synergistic negative effects in MASLD patients.

6. Supplements

6.1. Coffee

While no RCTs have studied coffee consumption, multiple observational studies demonstrate that consuming at least two cups of coffee daily is associated with reduced risk of liver fibrosis and lower rates of hepatocellular carcinoma [65–67]. One systematic review found that coffee consumers were significantly less likely to develop cirrhosis as compared to participants who did not consume coffee (OR 0.61, 95% CI 0.50–0.73) [65]. Both caffeinated and decaffeinated coffee have shown benefits, suggesting compounds other

than caffeine contribute to coffee's hepatoprotective effects [66]. Proposed mechanisms include antioxidant properties, anti-inflammatory effects, and improved insulin sensitivity. Importantly, these benefits are mainly observed with black coffee or coffee with minimal additives. Adding sugar or high-calorie creamers may negate the beneficial effects. While much of the data supporting coffee consumption is observational, the net clinical benefit supports the consumption of at least two cups daily while avoiding caloric additives. Despite previous concern over caffeine intake and precipitating arrhythmias, these claims have proven to be largely unsubstantiated [68]. Still, increased caffeine should be limited in patients with anxiety or panic disorders.

6.2. Prebiotics, Probiotics, and Synbiotics

A resurgence of interest in the gut microbiome and its interplay with MASLD has raised questions about the role of prebiotics, probiotics, and synbiotics. Synbiotics consist of mixtures of non-digestible dietary substrates (prebiotics) and live microorganisms (probiotics) [69]. The available evidence suggests that supplementation with prebiotics, probiotics, and synbiotics may be beneficial in reducing liver fibrosis and steatosis. One meta-analysis found that both probiotics and symbiotic supplementation were associated with increased odds of hepatic steatosis measured on ultrasound (pooled OR 2.40) and a weighted-mean decrease in liver stiffness measurement by 0.70 kPa compared to controls [70]. Notably, this analysis only found two trials with probiotics and four with synbiotics. Additionally, while most trials utilized probiotics consisting of *Lactobacillus* or *Bifidobacteria* strains, the formulation of probiotics and synbiotics was not controlled for in the analysis. This is important given the wide heterogeneity in probiotic/symbiotic/prebiotic formulations and their potential wide-ranging effect on a diverse human gut microbiome. These limitations were seen in a separate meta-analysis conducted in 2023, which did identify significantly improved hepatic steatosis measured via ultrasound, but not for CAP-measured fibrosis or liver stiffness measurement [71]. One meta-analysis did examine the specific role of *Bifidobacteria* strain-based probiotics on MASLD and found that decreases in hepatic steatosis and secondary risk factors for MASLD [72]. Together, these results suggest that probiotics and synbiotics should be standardized before being universally recommended to patients with MASLD despite promising evidence of benefit.

7. Physical Activity

Regular physical activity prevents MASLD progression, but achieving adequate activity levels can be challenging. Sedentary behavior is tightly coupled with MASLD development: even one hour daily of sedentary time confers nearly 4% greater risk of MASLD and 1.15% higher intrahepatic fat content. Additionally, physical activity improves MASLD, independent of weight loss [73,74].

Simple bedside assessments may help clinicians tailor exercise recommendations. Handgrip strength, measured using a hand dynamometer with the dominant hand, provides an easily obtained measure of muscle contractile strength that correlates with hospitalization risk and overall survival [5]. In patients with MASLD cirrhosis, handgrip strength can predict hepatic encephalopathy precipitation and even liver transplant waitlist mortality. Similarly, the 6 min walk test (6MWD) is a simple measurement correlating with aerobic capacity and endurance [19]. While no specific cut-off values exist for handgrip strength or 6MWD in MASLD, these assessments help inform whether to recommend more stringent exercise goals for patients with superior performance versus more conservative recommendations for those with poor performance.

It is important to individualize exercise recommendations to the patient's baseline level of activity. In general, 30–60 min of moderate-intensity exercise 3–5 times weekly

can be effective in preventing further decompensation in MASLD patients [27]. Both High-Intensity Interval Training (HIIT) and continuous aerobic exercise have been shown to significantly improve intrahepatic fat content and serum markers of liver damage (AST/ALT) [75]. Resistance training demonstrates significant reductions in ALT and insulin resistance. As no conclusive data supports routine use of one exercise modality over another, exercise interventions can be tailored to individual patients, focusing on achieving at least 3–4 moderate intensity sessions weekly while reducing sedentary activity.

8. Special Situations

8.1. Hepatic Encephalopathy

Previous clinical practice guidelines recommended low-protein diets for patients at risk of hepatic encephalopathy (HE) out of concern for precipitating HE. Recent studies have undercut this—protein intake of at least 1.0–1.5 g/kg of dry body weight and maintaining muscle mass may help remove ammonia [5,13]. Vegetable or dairy protein may be preferable to animal protein due to fiber content and reduction in CNS toxins. Branched-chain amino acid (BCAA) supplementation may help prevent or reverse HE, though the mechanism and degree of benefit remain under investigation [5]. Myosteatosis and sarcopenia are independently associated with hepatic encephalopathy in patients with cirrhosis, further emphasizing the importance of maintaining adequate protein intake and muscle mass [76].

8.2. Patients on Anti-Obesity Medications

As mentioned previously, anti-obesity medications such as GLP-1 receptor agonists are increasingly utilized tools for weight loss. In addition to the benefit of weight loss, these medications have demonstrated beneficial effects on MASLD/steatohepatitis resolution. The initial trial of liraglutide in 2015 demonstrated histologic resolution of non-alcoholic steatohepatitis in more than four times as many patients as placebo and a quarter of the risk of fibrosis progression [77]. These results were re-demonstrated in a more recent trial of tirzepatide, which also demonstrated a dose-dependent response in the rate of steatohepatitis progression via biopsy as well as the rate of fibrosis reduction. Results regarding the use of GLP-1 medications for weight loss and for steatosis reduction are exciting, although application should consider cost to patient, the prevalence of gastrointestinal side effects, and dietary changes as detailed below [78].

For patients prescribed GLP-1 receptor agonists, structured nutritional strategies can maximize hepatic benefits while minimizing adverse effects on body composition. This includes prioritizing protein intake and regular exercise with resistance to preserve lean muscle mass during weight loss [79]. Due to GLP1-mediated changes to satiety and appetite suppression, providers may counsel patients on eating frequent, small meals rather than the traditional three-meal pattern. Providers should also be keenly aware of monitoring micronutrient deficiencies in these patients, particularly fat-soluble vitamins (A, D, E, K) and vitamin B12, as rapid weight loss increases deficiency risk [2]. Lastly, providers may consider tracking muscle mass via the handgrip strength measurements or formal body composition analysis, rather than weight alone.

9. Patient Counseling and Systemic Barriers

Translating dietary evidence into actionable patient recommendations requires understanding contexts and barriers. Interventions are more likely to succeed when they consider a patient's relationship with food and patient-specific barriers to dietary modification, including cost, time, cooking skills, cultural preferences, and family dynamics. Providers

should not expect immediate habit change; however, they should counsel their patients on taking small incremental steps and adopt practical alternatives.

When counseling patients, the USPTSF recommends motivational interviewing to initiate behavior change in patients [12]. Motivational interviewing is a technique that involves soliciting concerns directly from patients to frame recommendations. Such strategies are frequently employed in tobacco or alcohol cessation but are also beneficial in motivating lifestyle change. Additionally, working with patients to identify one or two changes at a time may be beneficial in motivating a cascade of behavior change.

Systemic and sociocultural barriers may prevent the adoption of nutritional recommendations or interventions. When recommending dietary protocols, it is important for providers to consider a patient's financial status, as this may vastly limit the availability of healthy foods and daily exercise [80]. Additionally, patients with MASLD who face financial constraints are at greater risk of overnutrition with dietary intake of sugary beverages or saturated fats. Provider attitudes and confidence in managing MASLD may also limit sufficient treatment [81], as detailed in Table 3.

Table 3. Multi-level barriers to optimal nutritional management in MASLD and potential solutions.

Barrier Level	Common Barriers	Practical Solutions
Physician	Lack of familiarity with nutritional assessment tools; Time constraints; Perception that MASLD patients are “overnourished”; Uncertainty about when to refer [82]	Use validated brief screening tools; Incorporate assessment into routine visits; Educate on sarcopenic obesity concept; Establish clear referral criteria based on FIB-4
Patient	Poor dietary adherence; Lack of awareness about sarcopenia; Difficulty making lifestyle changes; Cost of healthy foods [80]	Provide written Mediterranean diet guidelines; Use visual aids to demonstrate muscle loss; Set small, achievable goals; Suggest cost-effective alternatives (canned fish, frozen vegetables)
Healthcare System	Absence of structured protocols; Limited access to nutritionists; Fragmented care between PCP and specialists; No decision-support tools	Develop standardized assessment protocols; Utilize telehealth for nutrition counseling; Improve care coordination with gastroenterology; Implement EMR alerts for nutritional screening

10. Conclusions

Lifestyle change is paramount to the management of MASLD, particularly the implementation of serious nutritional changes for patients. This review summarizes the evidence behind best practices for nutritional assessment, dietary interventions, and malnutrition management in MASLD patients. While some nutritional interventions have been studied extensively, including dietary interventions and weight loss, other aspects of nutritional guidance in this population need further study. Evidence behind malnutrition screening, micronutrients and supplements and exercise in MASLD patients was either scarce, lacked sufficient specificity or relied on observational data. Further studies should address these limitations using strict endpoint criteria validated against clinical MASLD stages, implementing randomization and other measures of reducing selection bias and stratifying study outcomes and interventions by MASLD stage.

This narrative review highlights key guidance for patients with MASLD, bridging formal meta-analyses and systematic reviews on this topic with expert opinion to cover a broad range of topics that primary care physicians may encounter in the primary care setting.

Limitations

This narrative review was not conducted as a systematic review and is subject to selection bias. Additionally, as noted throughout the manuscript, much of the nutritional evidence in liver disease derives from cirrhosis and end-stage populations and may not generalize to non-cirrhotic MASLD. Additionally, much of the evidence relied largely on surrogate imaging endpoints rather than histology, with small samples, short follow-up, and limited generalizability beyond their source populations. Several recommendations rest on observational data or expert opinion.

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Abbreviations

The following abbreviations are used in this manuscript:

MASLD	Metabolic Dysfunction-Associated Steatotic Liver Disease
ESLD	End-Stage Liver Disease
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
6MWD	6 min Walk Test
CPC	Child–Pugh Class
MELD	Model of End-Stage Liver Disease
LOV-D	Lacto-Ovo Vegetarian
EASL	European Association for the Study of the Liver
AASLD	American Association for the Study of Liver Disease
ACG	American College of Gastroenterology
DEXA	Dual-Energy Absorptiometry
MAMC	Mid-Arm Muscle Circumference
SMI	Skeletal Muscle Index
RFH-NPT	Royal Free Hospital Nutrition Prioritizing Tool
PG-SGA	Patient-Generated Subjective Global Assessment
MST	Malnutrition Screening Tool
REAPS	Rapid Eating Assessment
PCP	Primary Care Physicians

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