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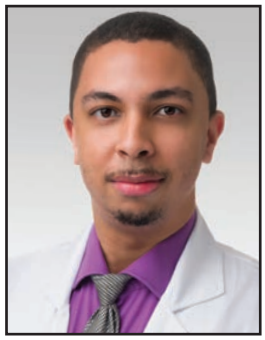
The Role of the Mediterranean Diet Pattern in Treatment and Management of Metabolic Dysfunction-Associated Steatotic Liver Disease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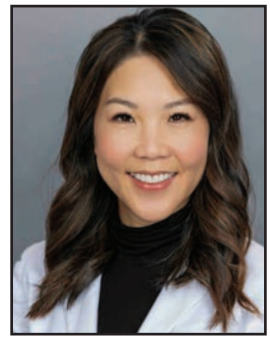
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Metabolic dysfunction-associated steatotic liver disease (MASLD) is defined by excessive hepatic fat accumulation in individuals without significant alcohol intake. Previously termed non-alcoholic fatty liver disease (NAFLD), this condition included nonalcoholic fatty liver and nonalcoholic steatohepatitis. The Mediterranean diet, characterized by limiting saturated fats, red meat, and refined sugars with increased consumption of fiber, polyunsaturated, and monounsaturated fats, has shown efficacy in improving hepatic steatosis and metabolic parameters in NAFLD. However, as diagnostic criteria have evolved, further research is needed to assess the diet's impact specifically on MASLD outcomes. This review will discuss the prevalence and diagnosis of MASLD, its associated metabolic and lifestyle risk factors, and evaluate existing evidence on the Mediterranean diet as a therapeutic approach, underscoring its close association with the earlier NAFLD classification.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a disorder characterized by the accumulation of excess fat within the liver, known as hepatic steatosis (HS).[1] MASLD is classified into two phenotypes: metabolic dysfunction-associated steatotic liver (MASL) and metabolic dysfunction-associated steatohepatitis (MASH). MASLD, formerly referred to as non-alcoholic fatty liver disease (NAFLD), encompasses a spectrum of liver diseases that occur in the absence of significant alcohol consumption. Previously, NAFLD was also classified into two main phenotypes: nonalcoholic fatty liver (NAFL), defined by the presence of at least 5% hepatic steatosis without evidence

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of hepatocyte ballooning, and nonalcoholic steatohepatitis (NASH), which is distinguished by hepatic steatosis accompanied by inflammation and hepatocyte injury, with or without fibrosis. MASLD, with its two phenotypes, representing the current terminology, is congruent with the same definitions as NAFLD and its two phenotypes.

In 2023, multiple professional liver societies developed a Delphi consensus statement to update both the diagnostic criteria and terminology.[2] This process involved input from 236 panelists who participated in surveys and meetings. The terms "non-alcoholic" and "fatty" were considered stigmatizing by a majority of participants—61% and 66%, respectively.[2] The prior terminology was also exclusionary for a diagnosis (the "non" portion of the diagnosis), while also using terminology of NAFLD that did not highlight the disease drivers, hence one of the big highlights of the use of MASLD, where metabolic dysfunction also shines light onto the drivers of the underlying disease state. As a result, steatotic liver disease (SLD) was adopted as an overarching term, and NAFLD was renamed MASLD. Under the new criteria, in addition to hepatic steatosis, at least one of five cardiometabolic risk factors must now be present to establish a diagnosis of MASLD.[2]

Modification of diet through the Mediterranean diet (MedDiet) with reducing intake of animal-based protein, saturated fats, and concentrated sweets and increasing intake of dietary fiber, polyunsaturated fatty acids (PUFAs), and monounsaturated fatty acids (MUFAs) has been previously shown to reduce NAFLD.[3] Ongoing studies will continue to evaluate its impact on the metabolic and hepatic parameters now used to define MASLD.

This review will discuss the prevalence and diagnostic criteria for MASLD, explore metabolic and lifestyle risk factors, and examine current evidence on the MedDiet for its treatment and management, demonstrating a strong association with the prior nomenclature of NAFLD. Studies included in the review that evaluated MASLD using current diagnostic criteria will use the MASLD nomenclature, whereas earlier NAFLD studies will retain NAFLD nomenclature.

Table 1. Updates for Steatotic Liver Disease Nomenclature

Prior Nomenclature	Criteria
Non-Alcoholic Fatty Liver Disease (NAFLD)	• Presence of hepatic steatosis confirmed by imaging or by histology • Lack of significant alcohol consumption
Non-Alcoholic Steatohepatitis (NASH)	• Presence of 5% hepatic steatosis • Inflammation and hepatocyte injury (+/- fibrosis)
New Nomenclature	**Criteria**
Steatotic Liver Disease (SLD)	• Overarching term to encompass the various causes of steatosis
Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)	• Presence of hepatic steatosis • Lack of significant alcohol consumption • At least one of five cardiometabolic risk factors
Metabolic Dysfunction-Associated Alcoholic Liver Disease (MetALD)	• Meets MASLD Criteria • Individuals who consume more than 140 g/week of alcohol for women and 210 g/week for men
Alcoholic-Associated Liver Disease (ALD)	• Clinical-histologic spectrum including fatty liver, alcohol hepatitis, and cirrhosis with its complications • Documentation on chronic heavy alcohol use • Exclusion of other causes of liver disease
Specific Etiology Steatotic Liver Disease (Specific Etiology SLD)	• Fatty liver conditions with a known cause, distinct from metabolic dysfunction-associated steatotic liver disease or alcohol-related liver disease
Cryptogenic Steatotic Liver Disease (Cryptogenic SLD)	• Not meeting criteria for MASLD or a specific alternative etiology • Patients may be reclassified in the future as more data emerge

Prevalence

A prior meta-analysis published before the change in nomenclature has estimated that the global prevalence of NAFLD was 25.24%. Africa has the lowest prevalence whereas South America and the Middle East have the highest prevalence. Upon analysis of the regions, the prevalence of NAFLD was 13% for Europe, 12.89% for North America and 9.26% for Asia.[4]

Despite evolving nomenclature, there remains a high concordance between NAFLD and MASLD, with nearly identical clinical outcomes. Notably, approximately 5% of individuals previously classified as having NAFLD would not meet the updated MASLD criteria.[5] Similar to NAFLD, the highest regional prevalence of MASLD is observed in Latin America (44.4%), while the lowest is found in Western Europe (25.1%). Projections indicate that global MASLD prevalence may rise to 55.4% by 2040.[5] Of particular concern, countries in the Middle East and North Africa (MENA) region, along with much of Asia, are experiencing significant increases in obesity and type 2 diabetes-key metabolic drivers of MASLD.

Diagnosis

MASLD is diagnosed based on the presence of hepatic steatosis in combination with at least one cardiometabolic risk factor, after excluding significant alcohol use and other chronic liver diseases. MASLD encompasses a spectrum of hepatic disorders, including isolated liver steatosis (referred to as metabolic dysfunction-associated steatotic liver, or MASL), steatohepatitis (MASH), and advanced fibrosis or cirrhosis.[6] See Table 1. Historically, differentiation between MASL and MASH required liver biopsy for histologic identification of steatohepatitis; however, clinical practice has shifted toward noninvasive methods for staging disease severity. Biomarkers and imaging techniques such as vibration-controlled transient elastography (VCTE) and fibrosis-4 (FIB-4) index are now commonly used to stratify fibrosis risk, with diagnostic performance comparable to that seen in NAFLD populations.[7]

Importantly, imaging with abdominal ultrasound is not mandatory for diagnosis in patients at high risk of MASLD. In such cases, clinicians may proceed directly to fibrosis risk stratification following exclusion of secondary causes of hepatic steatosis, regardless of transaminase levels.[8] The diagnostic criteria for MASLD require the presence of hepatic steatosis along with at least one of the following cardiometabolic risk factors:[8,9]

- **Body mass index (BMI)** >25 kg/m^2 (or >23 kg/m^2 for Asian individuals) or **waist circumference (WC)** >94 cm (men), >80 cm (women), or ethnicity-adjusted equivalents.
- **Fasting serum glucose** ≥5.6 mmol/L (≥100 mg/dL), **2-hour post-load glucose** ≥7.8 mmol/L (≥140 mg/dL), **HbA1c** ≥5.7% (≥39 mmol/L), diagnosis of type 2 diabetes, or treatment for diabetes.
- **Blood pressure** ≥130/85 mmHg or use of antihypertensive medication.
- **Fasting plasma triglycerides (TG)** ≥1.70 mmol/L (≥150 mg/dL) or use of lipid-lowering therapy.
- **Plasma HDL-cholesterol** ≤1.0 mmol/L (≤40 mg/dL) in men or ≤1.3 mmol/L (≤50 mg/dL) in women, or use of lipid lowering therapy.

Table 2. Core Components of the Mediterranean Diet Pattern

Food Group	Recommended Intake
Vegetables	≥ 2 servings/meal
Fruits	1–2 servings/meal
Whole grains	As primary carbohydrate source
Legumes	≥ 2 servings/week
Nuts and seeds	1 serving/day
Extra virgin olive oil (EVOO)	Main source of MUFAs; 4–6 tablespoons/day
Fish and seafood	≥ 2 servings/week
Poultry and eggs	Moderate consumption
Red and processed meats	Limited; < 1 serving/week
Dairy (preferably low-fat)	Moderate consumption
Alcohol (optional)	Moderate wine with meals (if culturally appropriate)

Risk Factors

Metabolic Risk Factors

A primary risk factor for the development of MASLD is excess adiposity—particularly obesity and overweight status. The principal underlying

Table 3. Mediterranean Diet Pattern and Clinical Impact in Patients with MASLD

Component of Mediterranean Diet Pattern	Mechanism of Action	Clinical Impact in Patients with MASLD
Monounsaturated Fatty Acids (e.g., olive oil)	improve lipid profiles enhance insulin sensitivity reduce hepatic fat accumulation	↓ intrahepatic triglycerides ↓ insulin resistance
Dietary Fiber (e.g., fruits, vegetables, legumes, whole grains)	slows glucose absorption supports gut microbiota reduces inflammation	↑ insulin sensitivity ↓ systemic and hepatic inflammation
Polyphenols (e.g., fruits, red wine, nuts)	antioxidant and anti-inflammatory properties	↓ oxidative stress ↓ liver injury
High-Quality Protein (e.g., fish, moderate dairy)	provides essential amino acids reduces intake of saturated fat	↓ hepatic fat accumulation supports lean body mass
Low Red/Processed Meat and Sugar Intake	reduces caloric excess and saturated fat/sugar burden	↓ obesity ↓ insulin resistance ↓ hepatic inflammation
Overall Dietary Pattern	encourages satiety, nutrient density, and sustainable metabolic balance	↓ risk of steatohepatitis and fibrosis progression

driver is dysfunctional visceral adipose tissue, which contributes to insulin resistance and chronic metabolic inflammation. The global burden of MASLD has increased in tandem with rising rates of type 2 diabetes mellitus (DM) and obesity.[10] Shi et al., in a comprehensive meta-analysis of 151 studies originally conducted under NAFLD criteria, including over 101,000 patients who underwent liver biopsy, reported prevalence estimates of 69.9% among individuals with overweight and 75.3% among those with obesity.[11] Although these studies used NAFLD criteria, they provide relevant insight into populations at high risk for MASLD.

Beyond obesity, MASLD is strongly associated with insulin resistance, dyslipidemia, and DM.[8] MASLD has been observed across multiple forms of DM, including type 2, type 1, ketone-prone diabetes, and maturity-onset diabetes of the young (MODY). Prevalence for NAFLD in type 2 DM, reported in earlier studies, ranges from 55% to 76%, while type 1 DM shows a slower but notable prevalence, generally 4%–20%, with most studies reporting rates near 20%.[12]

Dyslipidemia prevalence in this population ranges widely from 20% to 80%, depending on the study population and diagnostic criteria.[13] Patients with hypertriglyceridemia have a significantly higher likelihood of developing NAFLD compared to those with normal TG levels.[14] In a large population-based study, elevated BMI and high TG levels were both identified as independent risk factors for NAFLD incidence. Among individuals with a BMI ≥24, elevated TG levels alone accounted for approximately 25% of NAFLD cases.[15]

Lifestyle Risk Factors

Lifestyle risk factors for MASLD include poor diet quality and inadequate amounts of physical activity, which play an important factor in achieving sustainable weight loss, which is linked to significant improvements in insulin resistance and metabolic parameters associated with steatotic liver disease. In earlier NAFLD studies, patients were shown to have a higher intake of saturated fat, cholesterol, and a lower intake of PUFAs and fiber. The dietary habits seen here have the potential to directly influence hepatic steatosis.[16] In earlier NAFLD studies, patients were found to consume higher amounts of fructose-containing products compared to healthy controls. High fructose intake may also contribute to MASLD development by promoting hepatic lipogenesis, decreasing insulin sensitivity, and increasing the severity of liver fibrosis.[17]

Emerging evidence highlights the significant role of dietary patterns, particularly the widespread consumption of ultra-processed foods (UPFs)—in

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the development and progression of MASLD. UPFs are industrially manufactured food products typically high in added sugars, saturated fats, sodium, and various additives, while lacking essential nutrients and dietary fiber. Their consumption has been closely linked to obesity, insulin resistance, and hepatic inflammation—key drivers in the pathogenesis of MASLD.[18]

Recent studies have shown that the high intake of UPFs during childhood and adolescence is associated with an increased risk of MASLD and related metabolic disturbances. Similarly, adult and elderly populations consuming diets rich in UPFs are at elevated risk for hepatic steatosis and its long-term complications.[18] These findings underscore the importance of nutritional quality across the lifespan.

Garcia et al. reported that reducing UPF intake can lead to improvements in clinical and biochemical parameters associated with MASLD.[19] In particular, dietary modifications such as decreased consumption of red meat, sweets, and pastries, along with greater adherence to the Mediterranean diet, have been effective in reducing UPF intake.[19] UPFs are energy-dense and nutrient-poor, which promotes excess caloric intake while impairing metabolic regulation and promoting systemic inflammation and oxidative stress—factors that further contribute to MASLD pathophysiology.[19]

Mediterranean Diet

The MedDiet comprises the food patterns of individuals residing alongside the Mediterranean Sea. The diet is mostly plant-based foods of fruits, vegetables, whole grains, legumes, nuts, pulses, fish, seafood, and extra virgin olive oil that are included daily, at the majority of meals.[20] Due to abundance in plant-based foods, the diet is rich in fiber and phytonutrients, which both serve as anti-inflammatory nutrients. The diet is also rich in MUFAs through its frequent inclusion of fish, nuts, seeds, and olive oil. Fish is the main source of animal protein in the diet, whereas consumption of other sources of animal protein, including meat, poultry, and dairy is not daily.[20] Olive oil is included at each meal as a source of polyphenols and monounsaturated fats. The diet also includes guidelines for how often foods are to be consumed daily or weekly. See Table 2.

An expanding body of evidence supports the therapeutic potential of the MedDiet in managing MASLD. This dietary pattern favorably modulates key metabolic and inflammatory pathways.[20] Collectively, these components reduce intrahepatic

Table 4. Solutions for Socioeconomic Barriers to Implementing the Mediterranean Diet Pattern

Socioeconomic Barrier	Proposed Solution
Limited access to fresh produce	Encourage use of frozen or canned vegetables (low-sodium) Promote participation in local food banks or produce voucher programs
High cost of fish and seafood	Suggest affordable alternatives such as canned tuna/salmon (in water) Recommend plant-based omega-3 sources (e.g., flaxseed, chia)
Cost of olive oil and nuts	Promote moderation in use to stretch supply Recommend alternative healthy fats (e.g., canola oil) when necessary
Lack of culturally relevant food options	Adapt Mediterranean diet principles using culturally familiar foods (e.g., beans, whole grains, seasonal vegetables)
Limited nutrition knowledge	Provide basic education through handouts and/or group classes Partner with registered dietitians and/or community health educators
Lack of cooking facilities or time	Suggest minimal-prep meals (e.g., salads, grain bowls) Provide microwave-friendly recipes
Food deserts or limited grocery access	Connect patients with mobile markets, farmers' markets Home delivery services where available
Risk of caloric excess from energy-dense Mediterranean foods	Educate on portion control, especially with olive oil and nuts Offer practical visual cues and meal planning tools

triglyceride accumulation, enhance insulin sensitivity, regulate gene expression related to adipogenesis and adipocyte proliferation, and attenuate pro-inflammatory responses associated with visceral adiposity.[20]

Multiple independent studies have demonstrated that adherence to the MedDiet is associated with significant reductions in hepatic steatosis among patients with MASLD. One of the pioneer investigations reported reductions in intrahepatic fat content along with a decreased incidence of progression to steatohepatitis.[21] A six-month earlier NAFLD study by Marin-Alejandre et al. involving 98 patients highlighted the central role of MUFAs—abundant in the MedDiet—in improving lipid profiles, carbohydrate metabolism, and insulin resistance.[22] These changes were associated with improved blood pressure and decreased hepatic fat content, collectively contributing to a more favorable clinical course.[22] See Table 3.

Further research including intervention studies among Western, non-Mediterranean populations have confirmed these benefits.[23] Clinical trials lasting between 6 to 24 weeks have consistently demonstrated reductions in intrahepatic fat and improvements in insulin sensitivity and cardiovascular risk markers among individuals with MASLD.[23] In a 6-week randomized crossover NAFLD trial in Australia, Ryan et al. observed a 39% reduction in intrahepatic lipid content in patients following the MedDiet, compared to only 7% with a low-fat diet.[24]

Participants in the MedDiet group also showed insulin sensitivity and lower circulating insulin levels, as well as greater reductions in systolic blood pressure and serum triglycerides.[24] Similarly, a 12-week isocaloric trial involving 48 adults with hepatic steatosis found comparable

Table 5. Practical Guidelines: Promoting the Mediterranean Diet Pattern for Patients with MASLD

Step	Clinical Action
1. Assess Readiness and Personalize	• Use open-ended questions to explore current eating habits • Identify barriers (cost, time, cultural factors) • Emphasize clinical benefits
2. Teach Core MedDiet Principles	• Encourage substitutions: ○ Use olive oil instead of butter ○ Choose fish/legumes for protein over red meats ○ Snack on whole fruit and nuts ○ Favor whole grains versus refined grains (e.g., whole wheat versus refined wheat)
3. Recommend Gradual Implementation	• Start with 1 MedDiet-style meal per day • Encourage cooking with olive oil and seasonal produce • Share simple, culturally relevant recipes
4. Reinforce at Follow-Up	• Monitor liver enzymes, weight, and diet adherence • Celebrate small wins • Use motivational interviewing to maintain momentum
5. Utilize Resources	• Recommend apps, cookbooks, or community programs • Involve family for support • Share visuals or handouts to reinforce learning
6. Refer When Appropriate	• Dietitian referral for patients with comorbidities or complex dietary needs • Tailor support to individual and cultural contexts
7. Key Messages for Patient Counseling	• "This isn't a restrictive diet. It's a sustainable and flavorful eating pattern." • "Even small steps can help improve your liver and heart health." • "Focus on consistency, not perfection. Build healthy habits gradually."

Abbreviation: MedDiet, Meditteranean diet

reductions in intrahepatic fat content with both the MedDiet (25%) and a low-fat diet (32%).[25] These findings suggest that dietary quality—regardless of macronutrient composition—plays a crucial role in overall management of hepatic steatosis.[25]

Similar outcomes are seen in Eastern populations. A retrospective study by Lee et al. examined MedDiet adherence in a Korean population and found that individuals with high adherence had significantly lower rates of MASLD.[26] This group also exhibited reduced triglyceride levels and lower triglyceride-glucose indices, reinforcing the diet's potential to prevent MASLD and its complications.[26] Overall, these studies underscore the clinical benefits of the MedDiet and those with MASLD should attempt to transition to this dietary pattern.

Practical Applications

While the MedDiet has demonstrated clear benefits in managing MASLD, one of the key challenges in its implementation is the adaptability of the dietary pattern to individual patient needs. Socioeconomic barriers—such as limited access to fresh produce, fish, and other core components—can pose significant obstacles to adherence, particularly in underserved populations. To address these barriers, clinicians can emphasize cost-effective and culturally relevant alternatives—such as canned or frozen vegetables and legumes, affordable sources of healthy fats like canola oil, and community-based resources like food pantries or subsidized farmers markets. Additionally, incorporating nutrition education and connecting patients with registered dietitians and/or community health educators can empower individuals to make sustainable dietary choices within their means. See Table 4.

Moreover, while the MedDiet is often perceived as inherently healthy, it is not immune to caloric excess.[20] Patients with hepatic steatosis should consider portion control, as the liberal use of energy-dense foods such as olive oil and nuts can inadvertently lead to a hypercaloric diet. Individualized nutritional counseling should emphasize both the quality and quantity of food intake, balancing the beneficial components of the MedDiet with appropriate caloric targets to support weight management and reduce hepatic fat accumulation.[20] See Table 5. ■

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