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Optimising Dietary Interventions for MASLD: A Nutritional Perspective on Liver Heath

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Abstract
Metabolic dysfunction-associated steatotic liver disease (MASLD) has become one of the leading causes of chronic liver disease globally, closely tied to obesity and insulin resistance. In the absence of approved drugs, lifestyle modification notably weight loss through diet is the cornerstone of management. This review synthesizes evidence on major dietary patterns (Mediterranean-style, low-carbohydrate high-protein, intermittent fasting) with a focus on clinical trials and RCTs done in the past 5 years.
Mediterranean diets consistently reduce hepatic steatosis and improve metabolic biomarkers. In particular, a polyphenol-enriched "green" Mediterranean diet (rich in leafy greens, walnuts, and low in red/processed meat) achieved roughly double the intrahepatic fat reduction of a standard Mediterranean regimen. Similarly, low calorie diets rich in unsaturated fats (e.g. olive oil, nuts) significantly lower liver steatosis and transaminitis.
Low-carbohydrate and high-protein strategies also show significant benefits. Both a 5:2 days intermittent fasting protocol and a low-carbohydrate/high-fat diet produced significantly larger reductions in liver fat and body weight than standard care, with no difference between them. Some studies suggest that high-protein, low-carbohydrate diets may yield particularly large gains in NAFLD reversal. Overall, caloric restriction drives most of the improvement: any energy-restricted diet markedly lowers hepatic fat, transaminases, and insulin resistance. Physical activity augments dietary effects: combining exercise with diet amplifies liver improvements.
In summary, tailored dietary intervention is central in MASLD management. Diverse diets (Mediterranean, green-Mediterranean, low-carb, high-protein, intermittent fasting) can each induce clinically meaningful steatosis regression, largely via weight loss. However, consensus is lacking on the optimal diet pattern.

Keywords: Fatty liver, NAFLD, MASLD

Introduction

Metabolic dysfunction associated steatotic liver disease (MASLD), previously known as Non-alcoholic fatty liver disease, is a chronic liver disease, affecting about 32.4% of the global population as per the census conducted in 2021 [1]. By 2030, the prevalence of MASLD linked HCC is expected to rise by 47% to 130% [2]. In India, a recent meta-analysis study reported that 38.6% of the adults are affected by MASLD which poses a significant healthcare concern [3].

Over the years, the prevalence of MASLD has increased drastically alongside diabetes and metabolic syndrome [4]. Thus, in 2023, an international consensus of liver disease experts, officially renamed Non-alcoholic fatty liver disease (NAFLD) to Metabolic dysfunction-associated steatotic liver disease (MASLD). This change was implemented to provide a more precise and non-stigmatizing description of the condition, focusing on its association with metabolic dysfunction rather than defining it by the absence of alcohol consumption. The new terminology also acknowledges that MASLD can coexist with other chronic liver diseases, eliminating the need for exclusion criteria related to alcohol intake [5].

Despite the increasing prevalence, currently there aren't any approved pharmacological treatments for MASLD, however, even modest weight loss (>5% of the body weight) has been related to resolution of steatohepatitis and >7-10% for fibrosis. Thus, lifestyle intervention and nutrition support remain the cornerstone for the treatment of MASLD [6]

Multiple interventional as well as observational studies have investigated the impact of a particular dietary intervention such as Mediterranean diet, low carbohydrate diet, intermittent fasting, impact of gut microbiota, physical activity etc on MASLD. This review is written with an aim to critically evaluate the current evidence on the various dietary and lifestyle interventions preferred for MASLD.

Methods

The search was carried out using databases like PubMed and Google Scholar, covering researches published between 2006 and March 2025. The keywords used included "MASLD," "NAFLD," "Mediterranean diet," "dietary intervention," "physical activity," "lifestyle modification," and "NAFLD and diet." For the section focused specifically on dietary interventions in NAFLD, only studies from the past five years were considered, with a focus on clinical trials and randomized controlled trials. Inclusion criteria were English-language, peer-reviewed human studies that investigated non-pharmacological interventions such as diet and physical activity. Studies conducted on animals, or in vitro studies were excluded. A total of 63 articles meeting these criteria were included and narratively synthesized, with emphasis on the impact of various dietary and lifestyle strategies on liver-related and metabolic outcomes.

Pathophysiology

Environmental, metabolic, as well as genetics impact the cause and progression of MASLD by multiple mechanisms. In terms of metabolism, MASLD is basically the results of a positive energy balance leading to an imbalance of energy metabolism in the liver leading to accumulation of triglycerides in the liver [7]. Modern understanding emphasizes a "multiple-hit" pathogenesis: genetic predisposition, insulin resistance (causing adipose lipolysis and hepatic de novo lipogenesis), oxidative/ER stress, and inflammation all contribute to the development of MASLD.

MASLD's more severe form, Metabolic-dysfunction-associated steatohepatitis (MASH), involves progressive liver damage, which may advance to cirrhosis and hepatocellular carcinoma. [8].

Progression from MASLD to MASH to cirrhosis

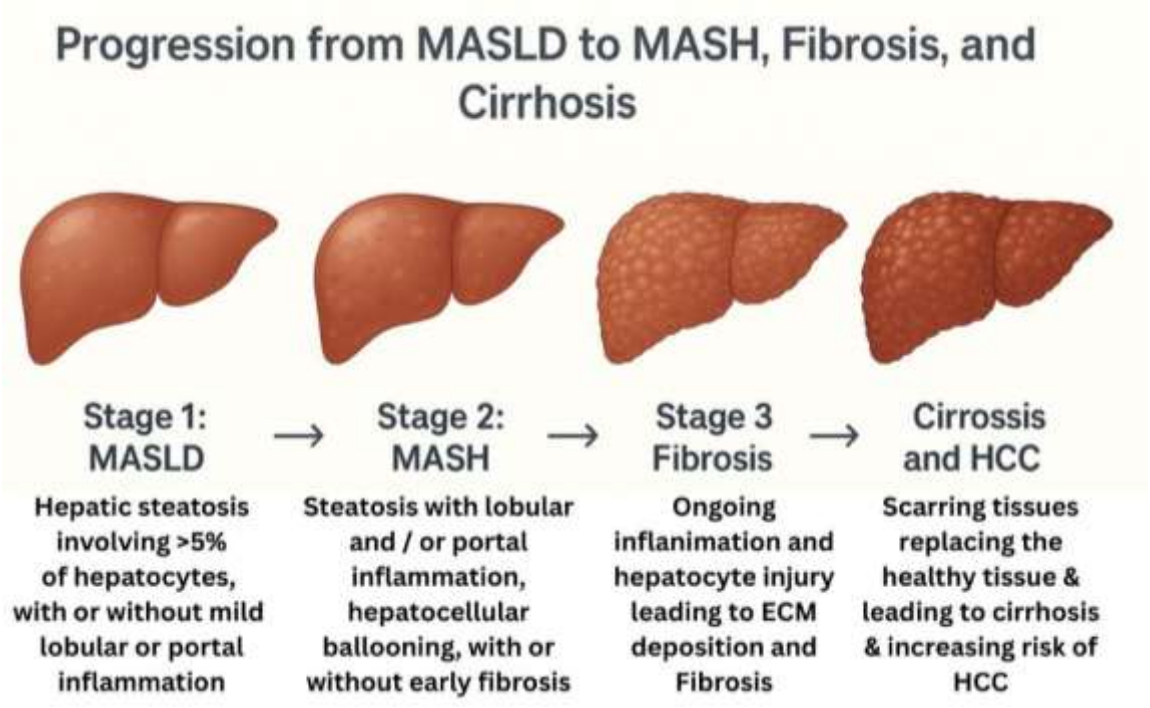

Figure 1 - Progression from MASLD -> MASH -> cirrhosis

MASLD when progressed to MASH paired with inflammation, cell apoptosis, and fibrosis can lead to ESLD or HCC.

Stage 1: MASLD – Steatosis (≥5% hepatocytes), may have mild lobular/portal inflammation.
Stage 2: MASH – Steatosis with lobular and/or portal inflammation, ballooning degeneration, my or may not have fibrosis (Lekakis & Papatheodoridis, 2024).
Stage 3: Fibrosis – Chronic inflammation results in significant extracellular matrix deposition and bridging fibrosis.
Stage 4: Cirrhosis and HCC – Advanced fibrosis progresses to cirrhosis, increasing the risk of liver failure and hepatocellular carcinoma (Xu et al., 2022).

Pathophysiological Mechanisms in MASLD Progression
1.1 Insulin resistance and MASLD

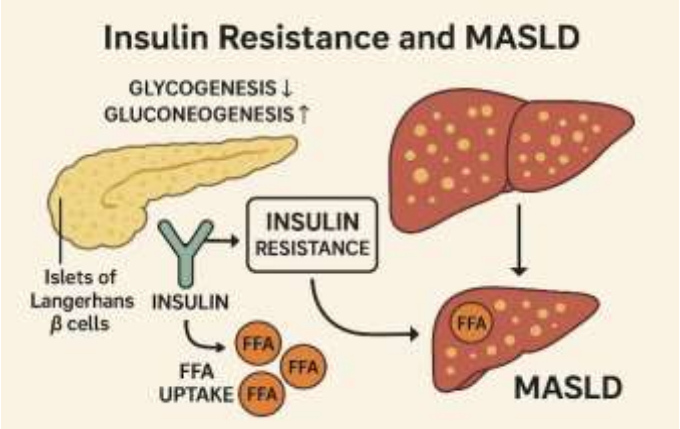

Figure 2 – Role of insulin resistance in progression of MASLD

Insulin is a hormone produced by the β cells from Islets of Langerhans in the pancreas, that regulates the glucose uptake from blood into liver, fat, and skeletal muscle cells. In some cases, the cells aren't able to respond efficiently to the insulin secreted and this mechanism is known as insulin resistance [11]. Normally, insulin promotes glycogenesis while inhibiting gluconeogenesis. Under ideal conditions, insulin converts the Free fatty acids (FFA) into triglycerides and packs them with VLDL for storage and transport but in case of IR, the lipid metabolism is affected and in turn increases FFA uptake in the liver, leading to fat accumulation in hepatocytes, thus, leading to fatty liver [12]. Thus, ESPEN guidelines recommend Individuals with MASLD should undergo screening for diabetes, while patients with type 2 diabetes should be assessed for MASLD regardless of their serum transaminase levels [13].

1.2 Inflammation

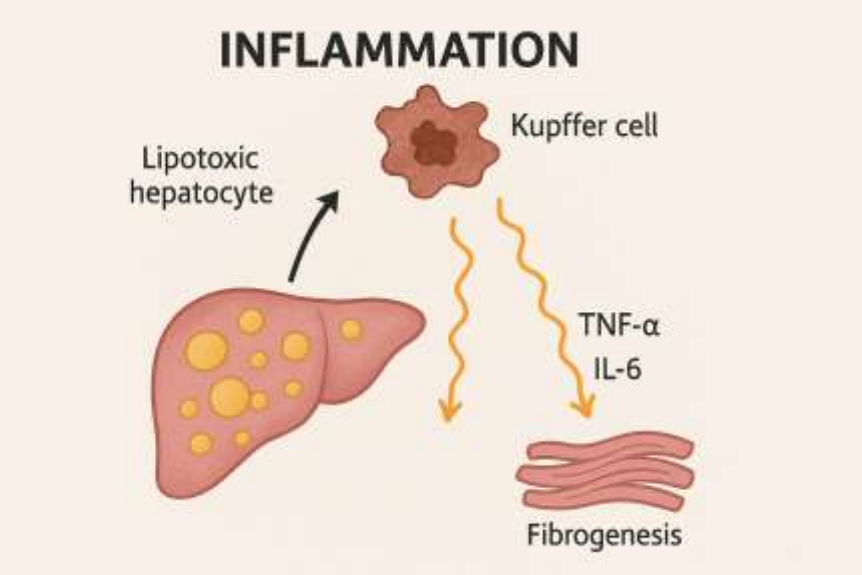

Figure 3 – Role of inflammation in progression of MASLD

Inflammation plays a significant role in the progression of MASLD. Lipotoxic hepatocytes release signals that activate Kupffer cells and release monocyte-derived macrophages, resulting in the secretion of pro-inflammatory cytokines such as TNF-α and IL-6. These cytokines further could lead to fibrogenesis [14]. In an RCT, it was demonstrated that treatment with nicotinamide riboside/pterostilbene significantly reduced liver enzyme markers of inflammation, suggesting that metabolic reprogramming may counteract hepatic inflammation [15].

1.3 Oxidative stress

Oxidative stress is a critical factor in the progression of MASLD, resulting from an imbalance between reactive oxygen species (ROS) production and antioxidant defense. It is known to initiate as well as progress the hepatic damage (Masarone et al., 2018).

Oxidative stress triggers excess cytokine production and of lipid peroxidation. This leads to an increase in hepatocellular injury as well as fibrosis which increases the risk of progression from MASLD to MASH by inducing ER stress, Mitochondrial dysfunction and other factors which can lead to cell death and fibrosis [17].

A study by Palmieri et al. (2006) demonstrated significantly reduced serum concentrations of antioxidants such as vitamin C and alpha-tocopherol, alongside elevated lipid peroxidation markers in patients with steatosis and metabolic syndrome. These alterations indicate compromised antioxidant protection and increased oxidative damage, particularly associated with visceral fat accumulation and liver steatosis [18].

1.4 Lipotoxicity

Lipotoxicity is said to be the improper regulation of extracellular and intracellular lipid composition in the liver which leads to steatosis. Free fatty acids can build up in the liver through three primary pathways: dietary intake, de novo lipogenesis, and the breakdown of fat in adipose tissue. The progression from MASLD to MASH, cirrhosis, and HCC is closely linked to impaired lipid metabolism, which results in lipotoxicity [19].

1.5 Mitochondrial dysfunction

In MASLD, hepatic steatosis leads to mitochondrial dysfunction, causing oxidative stress and impaired lipid metabolism. The accumulation of free fatty acids in hepatocytes overloads the mitochondrial β-oxidation pathway, leading to the generation of reactive oxygen species (ROS) and mitochondrial damage [20]. According to a RCT, L-carnitine supplementation may improve liver function by supporting mitochondrial activity [21]. Furthermore, ER stress exacerbates mitochondrial dysfunction, contributing to hepatocyte apoptosis [22].

1.6 Genetic and epigenetic factors

Genetic and epigenetic factors significantly influence susceptibility to MASLD. Variants in genes such as PNPLA3 and TM6SF2 are associated with increased hepatic fat content and progression to fibrosis [23]. Research found that carriers of the PNPLA3 risk had significantly higher odds of NASH and fibrosis, particularly when exposed to high red meat consumption [24].

1.7 Adipokine imbalance

Adipokines such as leptin, adiponectin, and resistin are secreted by adipose tissue, with their type and quantity influenced by adipocyte characteristics like type (white or brown), size, and cellular interactions. White adipocytes store triglycerides and release mainly proinflammatory cytokines, including leptin, TNF-α, IL-6, and resistin, whereas brown adipocytes secrete anti-inflammatory and metabolically beneficial factors like FGF21, VEGF-A, and IL-10. Adipokines regulate glucose homeostasis, insulin sensitivity, lipolysis, and fatty-acid oxidation across organs such as the liver, muscle, and pancreas. In obesity, elevated leptin contributes to a chronic inflammatory state and promotes the development of metabolic dysfunction-associated steatotic liver disease (MASLD) by enhancing the production of inflammatory cytokines and lipid mediators. In contrast, adiponectin, which has insulin-sensitizing and anti-inflammatory effects, is reduced in obesity. Its deficiency impairs fatty acid oxidation and promotes hepatic steatosis and inflammation, making adipokine imbalance a key driver in MASLD pathogenesis [25].

1.8 Fibrogenesis

Hepatic stellate cells (HSCs) are central to the process of liver fibrogenesis, with their activation influenced by various cytokines and signaling pathways [26]. Research has identified a key transcription factor that regulates TGF-β-driven HSC activation by modulating a stress-responsive protein. In MASLD, fibrogenesis marks the advanced stage of disease progression, where persistent hepatocellular stress and inflammation lead to HSC activation and excessive extracellular matrix deposition. This results in architectural distortion of the liver, impaired function, and potential progression to cirrhosis (Harrison et al., 2024).

2. At risk factors

MASLD is a spectrum, starting with steatosis and leading to fibrosis, it has been defined as the accumulation of excess of fat (>5% of the weight of the liver) in the hepatocytes [28]. Over the years, it

has been associated with the presence of central obesity, insulin resistance, dyslipidaemia, hypertension and T2DM (Pouwels et al., 2022). In a study conducted in Chennai in 2023, it was seen that out of 510 non-alcoholic adults aged between 21 to 40, 61.5% of the people turned out to be positive for fatty liver. It was observed that men were 1.59% more prone to developing fatty liver than women. People with higher BMI were seen to be more at risk than those with a normal BMI. An increase in age was also seen to be a risk factor (Anton et al., 2023). Another study conducted among the South Asian population also noted a male predominance, and corelated the increasing prevalence to growing industrialization and sedentary lifestyle [31]. Some studies associated it with the westernised diets which are high in saturated fats and fructose[32]. Apart from abnormal Liver function tests, current evidence suggests high free cholesterol to be a marker for development of NASH [33] which could be corelated to why CVD remains the topmost cause of death in MASLD patients. Apart from the mentioned risk factors, obesity, IR, T2DM, hypertension and Hispanic ethnicity also put an individual at a higher risk of developing MASLD [19]

3. Signs and symptoms

While majority of the people with MASLD are asymptomatic, it was seen that fatigue turned out to be the most common symptom. Apart from fatigue, majority also experienced anxiety, thirst, and bloating. Some even experienced sharp or dull upper abdominal pain (upper left quadrant) which could be related to dyspepsia and reflux disease. A change in the sleep pattern was also noted in a few participants (Khoonsari et al., 2017).

4. Diagnosis

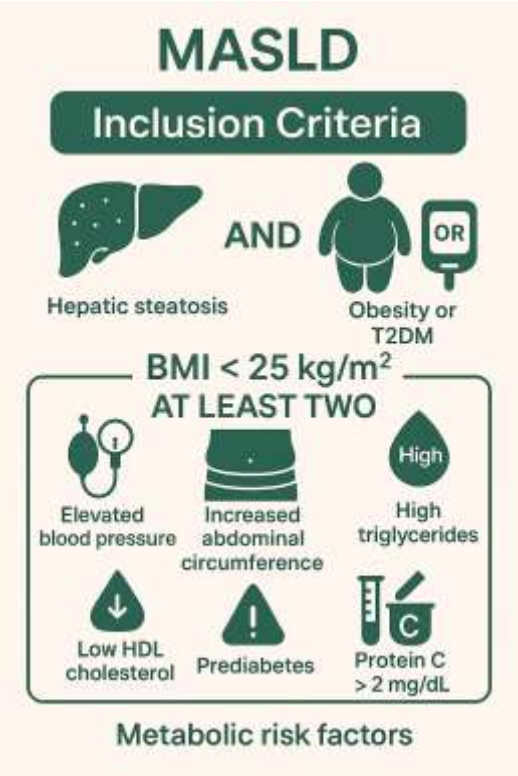

Figure 4 – Risk factors and diagnostic criteria for MASLD

The diagnosis of MASLD is based on specific inclusion criteria, which requires the presence of hepatic steatosis alongside either obesity or type 2 diabetes mellitus (T2DM). For individuals with a body mass

index (BMI) under 25 kg/m², a diagnosis necessitates at least two metabolic risk factors, such as elevated blood pressure, increased abdominal circumference, high triglyceride levels, low HDL cholesterol, indicators of prediabetes, an insulin resistance index (HOMA-IR) above 2.5, or protein C levels exceeding 2 mg/dL. Unlike previous classifications, the exclusion of other chronic liver diseases or significant alcohol consumption is not required for MASLD diagnosis [35]. However, as MASLD is asymptomatic in most cases, imaging and biomarker tests are needed for accurate diagnosis.

5. Management

5.1 Pharmacological treatment

Pharmacological treatments for MASLD and NASH have progressed significantly in recent years. Among the most promising are metabolic drugs like GLP-1 receptor agonists. In a Phase 2 trial, Semaglutide (0.4 mg weekly) led to NASH resolution in 59% of patients, compared to 17% on placebo, though it didn't significantly improve liver fibrosis (Newsome et al., 2021). The dual GIP/GLP-1 agonist tirzepatide also showed strong effects in a Phase 2 trial, resolving NASH in up to 44-62% of cases and improving fibrosis in 50% of patients (Rohit Loomba et al., 2024). These agents caused expected gastrointestinal side effects and weight loss. Their mechanisms involve weight reduction and improved insulin sensitivity, which secondarily reduce liver fat and inflammation [36].

Nuclear receptor agonists and lipid regulators are another major class under development. Resmetirom, a thyroid hormone receptor-β agonist, achieved both NASH resolution and fibrosis improvement in Phase 3 trials [27]. While various medications for obesity and type 2 diabetes are available to manage associated conditions, resmetirom is currently the only FDA-approved treatment in the United States specifically targeting MASH in patients with stage 2–3 liver fibrosis [38]. Lanifibranor, a pan-PPAR agonist, met key endpoints in a Phase 2b trial, with nearly half of patients seeing fibrosis improvement [39] Saroglitazar, approved in India, improved liver enzymes and fat content in a short-term RCT [40] Aramchol, which targets fatty acid metabolism, showed moderate benefit in Phase 2b and is undergoing further trials (Ratziu et al., 2021). Pioglitazone as well as Vitamin E are being recommended in selected patients based on the presence or absence of Type 2 diabetes mellitus [42]. While these pharmacological options are promising and expanding rapidly, they are not a replacement for lifestyle changes. Diet, physical activity, and weight loss remain the cornerstone of MASLD management and are often necessary for medications to be most effective.

5.2 Dietary interventions for MASLD

MASLD, characterized by liver steatosis evident through biopsy, imaging or elevated liver enzymes, as well as obesity/overweight, type 2 diabetes mellitus (T2DM) or metabolic disorders has grown to be a major health concern worldwide. However, no pharmacological drug has been officially approved for it's management, but oral hypoglycaemic drugs appear to provide some improvement [43]. Despite the use of OHAs, dietary and lifestyle intervention remain the cornerstone of management of MASLD [44].

According the ESPEN guidelines for MASLD, a weight loss of 7-10% is recommended to improve steatosis and improve liver biochemistry and a reduction of >9-10% may help improve fibrosis [13]. APASL (Asian Pacific Association for the Study of the Liver) recommends the exclusion for processed foods as well as processed foods and beverages high in added fructose for the management of MASLD [45]. The overall recommendations for managing this condition are similar to that of T2DM with more of

a focus on calorie restriction and Mediterranean diet. An increase in physical activity is usually advised, but no specific type or duration has been mentioned [44].

The majority of MASLD patients are typically overweight or obese; however, a significant subset of individuals may have a normal BMI. These individuals often exhibit insulin resistance, low HDL levels, and elevated triglycerides compared to healthy counterparts. Additionally, visceral obesity, high-fructose and cholesterol-rich diets, and a sedentary lifestyle are commonly observed in this population [46].

Though weight loss remains the most effective treatment for MASLD, very low calorie diets leading to rapid weight loss may exacerbate the disease as well as IR thus, a well-planned diet with a focus on macro and micronutrients is essential [46].

5.2.1 High protein diet

A 2020 randomized controlled trial (RCT) in Germany investigated the impact of protein intake on liver fat reduction in 19 individuals with morbid obesity who were scheduled for bariatric surgery within three weeks. Despite both groups following a calorie-restricted diet (1500-1600 kcal /day) and experiencing weight loss, the study found that a high-protein (HP) diet (30%) was significantly more effective in reducing liver fat compared to a low-protein (LP) diet (10%). Even though the sample size is too small, the data suggested that the HP diet primarily lowered liver fat by reducing fat absorption and lipid production rather than by increasing fat breakdown [47].

5.2.2 Low carbohydrate diet

A randomized controlled trial (RCT) was conducted in Copenhagen to explore the impact of carbohydrate restriction diet in individuals with type 2 diabetes and also noted it's effect on liver fat content. 72 participants were randomly assigned to either a carbohydrate-reduced high-protein (CRHP) diet (C30/P30/F40 or a conventional diabetes (CD) diet (C50/P17/F33) for 6 weeks. It was observed that the CRHP diet resulted in a higher reduction of fasting triacylglycerol as well as intrahepatic fat by 26% as compared to the CD diet [48]. A RCT conducted in China across 7 hospitals assessed the impact of following a intensive lifestyle intervention (ILI) which included a low carbohydrate, high protein calorie-restricted diet, with regular exercise, and personalized counselling by dietitians whereas the control group followed a calorie restricted balanced diet with exercise and standardised counselling. The results showed that the ILI group significantly reduced the Fat attenuation parameter (FAP), Body mass index (BMI), Alanine Transaminase (ALT), Aspartate Transaminase (AST), Gamma-Glutamyl Transferase (GGT), triglycerides, glucose, insulin, HbA1c, HOMA-IR, HOMA-β as well as blood pressure and homocysteine. There was no significant difference between reductions in Liver stiffness measurement in both the groups [49].

5.2.3 Mediterranean diet

A 18-month direct plus trial was conducted in a workplace in Israel to assess the impact of a general healthy diet, calorie restricted med diet and a green-Med diet. The HDG group followed a general healthy diet along with physical activity and basic counselling. The MED group followed a calorie-restricted Mediterranean diet (1500–1800 kcal/day for men, 1200–1400 kcal/day for women) rich in vegetables, poultry, and fish, replacing red meat, it also included 28g of walnuts daily (providing 440mg polyphenols). The Green-MED group was also a hypocaloric diet, similar to the MED diet but with very less amount of processed and red meat. They also consumed 3–4 cups of green tea and 100g of frozen Wolffia globosa (Mankai) daily as a green shake replacing dinner. This provided an additional 800mg of polyphenols (including catechins) beyond the MED diet. It was seen hat despite similar level of weight loss in the green-med and med diet, the prevalence of MASLD dropped from 62% to 31.5% in greenMed group,

whereas it dropped to 47.9% in Med group. The Green-MED group achieved nearly double the IHF% reduction (−38.9%) compared to the MED group (−19.6%) and HDG group (−12.2%) [50].

Another study conducted in Australia in 2023 compared the effect of a med diet vs a low fat diet (LFD) on various inflammatory markers in patients with MASLD. It was observed that, following a med diet showed significant improvement in adiponectin levels whereas a LFD showed a greater decrease in the amount of leptin (Reddy et al., 2023).

While there are several benefits to following a Mediterranean diet, a study in Spain conducted on 155 participants showed that when the Mediterranean diet was paired with the right amount of physical activity (PA), it had a significantly more positive impact on muscle strength, aerobic capacity, and overall metabolic health. Participants who followed the Mediterranean diet along with structured PA (such as interval training and resistance exercises) experienced improvements in VO_2 max, push-up performance, and moderate-intensity activity levels, which are critical markers for cardiovascular and musculoskeletal fitness. In contrast, those who followed the same diet with high meal frequency (7 meals) but without adequate PA (walking 10,000 steps only) showed declines in handgrip strength, lower aerobic capacity, and less engagement in moderate-intensity activity. This highlights the importance of physical activity with the right amount of diet (Mascaró et al., 2022). A study in Australia conducted on 94 participants with a follow up at 6 weeks and 12 weeks suggested that the Mediterranean Diet (MedDiet) improved insulin sensitivity and reduced liver fat (39%) more effectively than a low-fat, high-carbohydrate diet (7% reduction), even without weight loss [53]. A study also evaluated the effects of a Low-Glycemic-Index Mediterranean Diet (LGIMD), two physical activity (PA) programs, and their combinations on liver fibrosis in MASLD patients over three months. Among 144 participants, those following LGIMD alone or combined with either PA1 or PA2 showed a significant reduction in liver stiffness (kPa), while PA alone showed non-significant improvements. These findings suggest that LGIMD, especially when combined with physical activity, effectively reduces liver fibrosis in individuals with MASLD [54].

5.2.4 Intermittent fasting

To note the impact of 16 hours intermittent fasting without any dietary recommendations, 98 participants with MASLD were recruited during the month of Ramadan in Egypt and their LFTs and anthropometric measurements were monitored at baseline and end point of 1 month. In a span of 1 month, it was seen that the BMI decreased, AST ALT cholesterol, triglycerides, HDL, LDL, cholesterol/HDL risk ratio showed significant improvement, whereas, waist, hip, and triceps skin fold thickness didn't have much improvement [55]. Another study conducted on 80 individuals with MASLD suggested that alternate day fasting alone as well as in combination with aerobic exercise (60 min/day, 5 days a week) showed significant improvement on intrahepatic triglyceride (IHTG) content and so did body weight, waist circumference and ALT levels, however, there was no improvement seen in the lean mass, AST, HbA1c, blood pressure, plasma lipids, liver fibrosis score, and hepatokines [56]. Another trial conducted over 12 weeks found that time-restricted feeding (16/8) combined with a low-sugar diet significantly reduced body fat, liver enzymes, lipid levels, and inflammatory markers in MASLD patients. This intervention also improved body composition and key cardiometabolic parameters compared to a control diet [57]. Whereas, a trial conducted in China over a span of 2 years suggested that time restricted eating didn't have any additional benefits over calorie restriction in obese patients with MASLD as the IHTG reduction was comparable in both the groups [58]

5.2.5 High fructose diet

A study conducted on 80 individuals with MASLD to see the effect of a diet rich in fruits on the liver

enzymes, steatosis, insulin resistance, and lipid profile surprisingly showed that a fruit rich diet (>4 servings/ day) seemed to worsen dyslipidaemia, steatosis, and glycaemic control in patients with MASLD [59]. This aligns with APASL's recommendations of restricting fructose intake.

5.2.6 Gut health

A double blind, randomised, placebo-controlled study aimed to evaluate the effects of probiotic supplementation over 6 months on patients with MASLD. The results showed no significant improvement in neither the placebo nor the supplementation group suggesting that probiotics have no significant effect on the liver biomarkers (Nor et al., 2021). Aligning with this, a trial conducted in the UK on 104 patients with an aim to see the effect of symbiotics on liver fat reduction over a supplementation period of 10-14 months reached the same consensus of having little to no effect on the liver biomarkers (Scorletti et al., 2020). Another study based on prebiotics treatment in MASLD patient showed that the supplementation increased Bifidobacterium levels in stool test but did not significantly affect liver fat, metabolic, or inflammatory markers in MASLD patients. The results suggest that prebiotics alone, without weight loss, are insufficient to improve MASLD. [62] Also, it was seen that probiotics do not significantly alter any inflammatory markers in NASH patients, highlighting the resilience of the inflammatory response in MASLD [63] thus, further studies are needed to observe the efficacy of improving gut health and resolution of MASLD.

Conclusion

In summary, diverse dietary strategies can each yield meaningful reductions in MASLD-related steatosis, but the data remains limited. A polyphenol-enriched "green" Mediterranean diet reduced intrahepatic fat more effectively than a standard Mediterranean diet. Likewise, a low-carbohydrate/high-fat diet and a 5:2 days intermittent-fasting regimen produced comparable weight loss and steatosis reduction. Some evidence suggests that high-protein, low-carbohydrate regimens may help achievement improvements, but most benefit appears to be correlated with the degree of weight loss, regardless of macronutrient composition.

Across all approaches, diet emerges as the cornerstone of MASLD therapy. Guidelines emphasize lifestyle modification (diet plus exercise) as first-line MASLD treatment, with pharmacological agents reserved for advanced disease. In practice, no approved drug has demonstrated broad hepatic benefits matching those of sustained dietary change. Thus, clinicians should prioritize individualized nutritional counselling over medication in managing MASLD.

Nevertheless, the ideal dietary pattern remains unclear. Existing trials are often short-term and done on a western population. Future large-scale, long-duration RCTs are required to compare these diets on clinically meaningful outcomes (e.g. fibrosis regression, NASH resolution). Such studies will clarify which specific nutritional approach yields the most consistent and clinically relevant improvements in liver health and MASLD progression.

Credit Authorship Contribution Statement

All authors equally contributed to Conceptualization, Methodology, Formal Analysis, Investigation, Writing and Visualization under supervision of the corresponding author.

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