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NUTRITIONAL STRATEGIES IN THE MANAGEMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE: A COMPREHENSIVE REVIEW

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ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is rapidly developing health issue in the globe and is considered to be the liver variant of the metabolic syndrome closely associated with obesity, insulin resistance and type 2 diabetes mellitus. The NAFLD etiology is multifactorial and involves the interaction of dietary, genetic, microbiological, and metabolic dysregulation. Currently, no known pharmacological interventions exist and, therefore, the disease management is based on nutritional and lifestyle interventions. The review combine the existing evidence on the role of macronutrients, micronutrients, antioxidants, and diets in the prevention and treatment of NAFLD. Overconsumption of refined carbohydrates, fructose, saturated fatty acids, and trans fatty acids are known to arouse the deposition of hepatic lipids through increased de novo lipogenesis, increased intake of free fatty acids, and oxidative stress. On the other hand, those diets rich in monounsaturated and polyunsaturated fats, dietary fiber, and bioactive substances are protective since they increase insulin sensitivity, reduce inflammation, and change the composition of the gut microbiota. Mediterranean diet is given special consideration because of its steadier positive outcomes in the prevention of hepatic steatosis and metabolic outcomes due to the presence of large amounts of olive oil, fruits, vegetables and omega-3 fatty acids. As well, micronutrients such as vitamin E and polyphenols play a role in the prevention of oxidative stress damage and hepatotoxicity. The lifestyle changes including energy limitation and physical activity also count and the loss of weight of 7-10 percent has been a major change in liver histology. Despite the encouraging outcomes, the limitations are the heterogeneity of the studies, absence of the long-term randomized controlled studies, and heterogeneity of the population of patients that presuppose the need to carry out further research. The individualized nutritional interventions and clinical outcomes over the long-run should be the next research study to maximize NAFLD management.

Keywords: *Nonalcoholic fatty liver disease (NAFLD), obesity, type 2 diabetes, caloric restriction, lifestyle modifications, diet*

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) refers to presence of macro vesicular fat accumulation in at least 5% of hepatocytes without any secondary cause like drugs or alcohol. It is the leading cause of liver diseases globally and is recognized as the

hepatic manifestation of metabolic syndrome (Geng et al., 2020). NAFLD development is a multifactorial mechanism which includes a broad range of hepatic disorders that are linked with cardiovascular and metabolic diseases such as dyslipidemia, type 2 diabetes, insulin resistance and hypertension. Obesity is a major risk factor as excessive adipose tissue leads to free fatty acids eventually resulting in stenosis. Insulin resistance impairs glucose and lipid metabolism, type 2 diabetes causes hyperglycemia, dyslipidemia lowers HDL and increase levels of LDL and triglycerides, CVD promotes systemic inflammation, all these aggravates fat deposition in liver (Jung and Choi, 2014). Along with obesity and T2DM, which are two key conditions linked with NAFLD, gut microbiota as well as genetic and environmental factors play a crucial role in onset and progression of NAFLD (Godoy-Matos et al., 2020). Lipid accumulation in liver is mainly attributed to three pathological mechanisms, such as high content of calories and fat in the diet, as increased visceral adipose tissue (AT) lipolysis and hepatic de novo lipogenesis (DNL) activation (Pierantonelli & Svegliati, 2019). The accumulation of fat in liver, also known as Hepatic steatosis, arises from disruption in the balance of production and utilization of lipids. This dysregulation of lipid balance within hepatocytes results in generation of toxic lipid compounds that cause organelles dysfunction triggering inflammation, damaged hepatocytes and eventual cell death. Clarifying the mechanisms leading to lipotoxicity driven cell injury may facilitate in the development of therapeutic options. Consequently, many pathways involved in accumulation of fat in liver and hepatotoxicity lead to the onset and progression of NAFLD ultimately leading to end stage liver disease (Geng et al., 2021).

As far as dietary factors are concerned, it plays a crucial role for NAFLD development. It is preferable to avoid high glycemic index carbohydrates and focus on lower amounts of carbohydrates i.e. less than 40% of daily energy intake. Fructose consumption and it's relation with NAFLD is mostly the hot topic of research due to its hepatic metabolism and rising sugar intake in population. Hepatic lipid metabolism can be modulated by fructose through activation of ChREBP and SREBP1c and by decreasing β-oxidation of mitochondria which favors the development of steatosis (Kennedy et al., 2007). The risk of NAFLD from fat intake depends on the type of fats. Monounsaturated fatty acids (MUFA's) and Polyunsaturated fatty acids (PUFA's) have proven results for protection against NAFLD while saturated fatty acids (SFA's) and trans fatty acids (TFA's) have negative impacts. Epidemiological studies show that people with NAFLD are taking more SFA's and cholesterol and less PUFA's (Vancells et al., 2021). Lifestyle changes have a significant role as it can improve hepatic steatosis, inflammation as well as metabolic abnormalities (Wong et al., 2013). Macronutrient and micronutrients composition, energy intake, physical activity, healthy lifestyle, fructose intake and weight loss are the considerations in the management of NAFLD. These patients are advised for low caloric diet and a diet restriction of 500-1000 calories per day is very effective strategy with a target of 7-10% weight loss in overweight/obese cases. Brisk walk, Cycling and 150-200mint per week moderate intensity aerobic physical activities in 3-5 sessions are recommended. The training that shows productive results is resistant training (Parra-Vargas et al., 2020). This review aims to summarize current evidence regarding nutritional strategies for the management of Non-Alcoholic Fatty Liver Disease and highlight dietary interventions that may improve liver health and metabolic outcomes.

Pathophysiology of nonalcoholic fatty liver

In addition to total body fat and visceral adiposity, fat accumulation in the liver has recently been identified as a significant metabolic risk factor. Growing research indicates that fatty liver may independently contribute to the development of conditions like Type 2 Diabetes and Cardiovascular Disease, which are major global health challenges. However, the precise mechanism that causes fat to accumulate in liver and the interaction of liver with other metabolismal organs is unknown (Petta et al., 2016).

The build-up of hepatic fat is believed to be occasioned by many factors. These are the distribution of body fat, diet, physical activity, genetic predisposition and the interaction between the environment and genetic factors. Moreover, fatty liver is also capable of affecting the metabolic processes in the body by affecting fat and carbohydrate metabolism. This could be caused by mechanisms such as low-grade inflammation and release of signaling molecules that alter metabolic processes. Also, recent studies have reviewed the complex relationship between insulin resistance and fatty liver, which shows that the two conditions do not necessarily occur together (Qureshi and Abrams, 2007).

Non-alcoholic fatty liver disease (NAFLD) is the liver disorder that is characterized by the excessive accumulation of lipids in the liver cells. The major causes of this accumulation are increased adipose tissue fatty acid influx, increased hepatic lipogenesis, high dietary fat and carbohydrate intake. A more severe type of the disease is called Non-Alcoholic Steatohepatitis (NASH) and can occur due to the continuous overloading of liver lipids on the cell level and mitochondrial oxidative stress (Allameh et al., 2023). In its later stages, NASH can develop cirrhosis and hepatocellular carcinoma. Trace elements are also important to this process. As an example, copper is a required metal that is engaged in a variety of enzymatic reactions that maintain the redox status of cells, particularly during mitochondrial respiration. Therefore, the normal level of copper regulation in the body can potentially delay the progression of NAFLD. The copper level imbalances have been linked to a variety of metabolic diseases, and some clinical trials are underway to see what therapeutic value copper-chelating agents hold (Martin-Fernandez et al., 2022).

As of now, there is no officially approved medication therapy of NAFLD. Thus, nutrition and lifestyle change remain the primary approaches to the treatment of diseases. Research has also been able to underscore the positive effects of foods rich in antioxidants that can interact with copper and reduce complications associated with diseases. On the whole, the existing evidence shows the significant role of diet in the development and management of non-alcoholic fatty liver disease (NAFLD), and it is particularly copper and antioxidants bound to copper (Antonucci et al., 2017). The mechanistic pathways involved in NAFLD progression are illustrated in Figure 1.

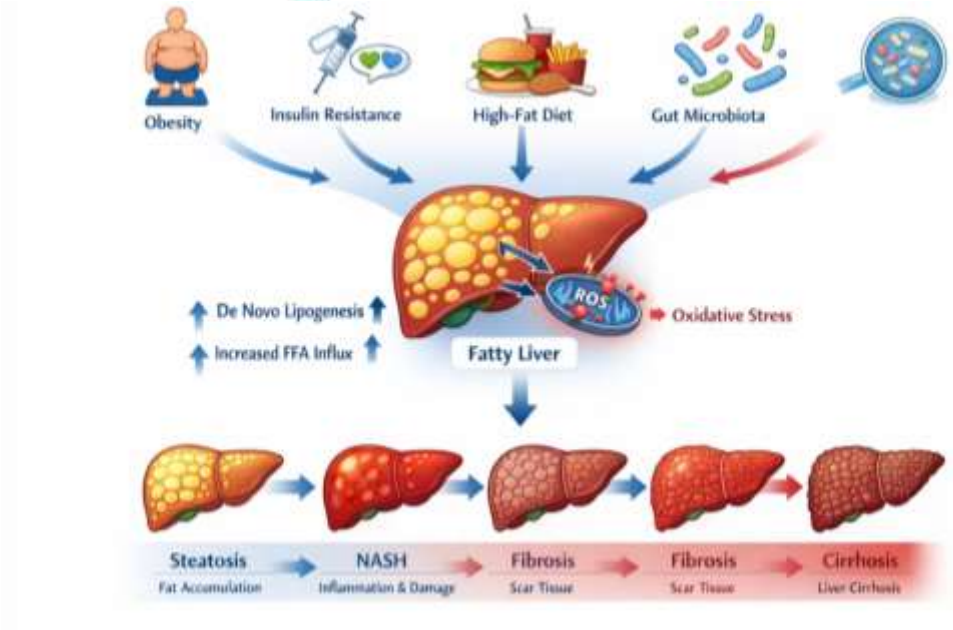

Figure 1. Pathogenesis and progression of non-alcoholic fatty liver disease (NAFLD).

The characteristic of non-alcoholic fatty liver disease (NAFLD) is an abnormal buildup of lipids (mainly triglycerides) in liver cells (hepatocytes). Liver fat may be classified as either macrovesicular or microvesicular steatosis depending on the size of the lipid droplets. Microvesicular steatosis is also caused by genetic disorders, liver damage due to drugs, and rare conditions such as fatty liver of pregnancy or Reye syndrome, and often associated with mitochondrial dysfunction. Macrovesicular steatosis, which is the more common type of NAFLD, is closely linked to obesity, type 2 diabetes, metabolic syndrome, nutritional imbalances, and other causes associated with metabolism or drugs (Haas et al., 2016).

Although a few individuals with macrovesicular steatosis can complain of a mildly fatigued or a poorly spaced abdominal pain, most of them are asymptomatic. Liver fat is commonly identified by imaging techniques such as ultrasound, CT scan, or MRIs, and laboratory tests often demonstrate minor to moderate levels of liver enzymes. Research indicates that NAFLD is prevalent among obese populations with 60-90 percent of morbidly obese people having the disease. Even though they are frequently regarded as benign, some patients may develop cirrhosis, fibrosis, non-alcoholic steatohepatitis (NASH), and, in extreme situations, liver failure (Grander et al., 2023). Both the treatment and prevention of NAFLD depend on nutritional therapies. Some of the strategies include calorie restriction, weight loss and maintenance of a balanced diet. Some of the nutrients that have shown protective effects include soy and whey proteins, monounsaturated and omega-3 fatty acids, probiotics, choline, fiber, coffee and green tea. While 7–10% weight loss may be necessary to improve hepatic inflammation, even a small weight loss of 3–5% can reduce liver fat. Crucially, maintaining a healthy diet over time is more beneficial than concentrating only on the kind of diet. In addition to enhancing liver health, these dietary practices also have a beneficial impact on related metabolic disorders like insulin resistance, type 2 diabetes, and cardiovascular risk (Fan and Cao, 2013).

Macronutrient composition and Dietary Fat quality in NAFLD

People suffering from NAFLD should have a less saturated fat and less carbohydrate diet and increased consumption of vegetable and fruits and evade high fructose drinks. (Vancells et al., 2021). In NAFLD patient the pufa/sfa intake ratio was inferior to arbitrarily chosen controls according to epidemiologic investigations. (Zhu et al., 2022) Steep insulin hindrance is generally connected with the elevated intake of saturated fats which trigger advancement of non-alcoholic fatty liver disease. (Yki-Järvinen, 2015) Greater risk of PCOS, CKD, osteopenia, OSAS and impaired metabolism of carbohydrates and high blood pressure is affiliated with NAFLD. (Reda, 2025) Non-alcoholic fatty liver disease is a deteriorating disorder which is linked with severe adverse outcome like liver cancer and cirrhosis. (Kumar et al., 2019)

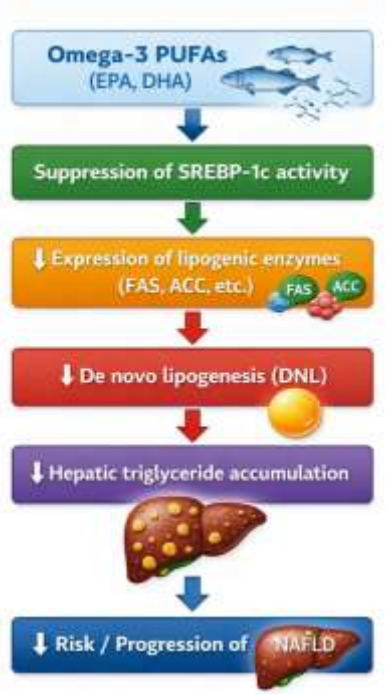

Figure 2. Function of omega 3 pufa in preventing NAFLD

According to WHO recommendation consumption of free sugar should be limited to less than 10% of total daily energy. Healthy juveniles are seemed to have less consumption of carbohydrate and calories as compared to juveniles having NAFLD. (Hydes et al., 2021) Advancement of NAFLD and modification of carbohydrate to triglycerides have a close link with intake of free sugars (sucrose and fructose) in contrast to glucose. (Vancells Lujan et al., 2021) Hepatic lipogenesis is initiated by additional glucose transformation into fatty acids, caused by carbohydrate consumption. (Jones, 2016) Diets having lower concentration of carbohydrates play effective role in reduction of fatty liver, weight loss, lowering levels of triglycerides and improving HDL level. 26% of triglycerides synthesis in NAFLD patients having high insulin levels is provided by de novo lipogenesis, in contrast diet and circulating free fatty acids provide 15% and 59% respectively. (Smith et al., 2020) These changes collectively conduct advancement in NAFLD. Elevated intake of supplemental sugars especially fructose is leading root cause for cardio metabolic disorders. (Lim et al., 2010) Fruits are good source of antioxidants, fiber and vitamins which mitigate simple sugars impacts on liver. (Pathak et al., 2023) Increased usage of supplemental sugars and advancement of non-alcoholic fatty liver disease have a significant association both in pediatrics and adult populations. (DiStefano & Shaibi, 2021) The susceptibility of NAFLD development is about 53% higher in participants with SSB consumption. (Tseng et al., 2023) Amount of fructose is low in fruits as well as presence of micronutrients, fibre and favorable phytochemicals so are less likely to cause non-alcoholic fatty liver. (Pathak et al., 2023) High fructose diet result in non-alcoholic fatty liver disease by elevating de novo lipogenesis and lowering oxidation of fatty acids. (Softic et al., 2016) Whole carbohydrates have low postprandial glycemic impact, contribution in enhancing production of gut derived fatty acids and enhanced fiber content and are presumed to be confer protection against non-alcoholic fatty liver disease. (Park et al., 2021)

According to WHO recommendation consumption of total fat should be more than 30% of total daily energy. Trans fat should be maintained at level of less than 1% while saturated fat should not be more than 10%. (World Health

Organization, 2023) Daily uptake of fat should be in range of 30 to 35 percent of total energy for children aged less than 3 and the percentage is between 25 to 35 for age group from 4 and so on according to The American Heart Association (AHA). (Monnard & Fleith, 2021) Based on type of fat, some of them (unsaturated fatty acids) have a positive impact on non-alcoholic fatty liver disease while some (saturated and trans fatty acids) have negative impact on non-alcoholic fatty liver disease. (Gentile & Pagliassotti, 2008) Seeds, plant based oils, fish, walnuts are good source of dietary ω-3 poly unsaturated fatty acids. (Simopoulos, 2002) ω-6/ω-3 ratio is usually higher in NAFLD patients due to low ω-3 poly unsaturated fatty acids intake. (Mukhametov et al., 2022) Alliance between amplified possibility of NAFLD and consumption of fat is noted but more reliant on sort of fat rather than quantity of fat utilized. (Zarghamravanbakhsh et al., 2021) 1:1 and 4:1 are the proposed ratio of ω-6/ω-3. (Mukhametov et al., 2022) For betterment of NAFLD utilization of omega-3s and limiting saturated fatty acids is recommended. Advantageous effects of Mediterranean diet are attributed to its high extra virgin olive oil content. (Huang & Sumpio, 2008) Emphasizing the mechanism of mono unsaturated fatty acids consumption in mitigation of non-alcoholic fatty liver disease, the results are variable. (Hussein et al., 2007) According to analytical study, the results are unbiased. In contrast experimental study propose advantageous effect having EVOO supplementation (Tsamos et al., 2024).

Like protein rich diet, reasonable protein diet consisting about 25% of daily calories is recounted by authors for NAFLD patients due to its ability of lowering body fat and not having any adverse effect. (Duarte et al., 2014) Protein aspect and variety which have been inadequately evaluated is also an important topic of interest. Accessible researches propose that soy and whey proteins can preclude non-alcoholic fatty liver but all the analysis or investigations are solely bounded to animals but no examination in human beings. (Xiao and Hendry, 2022). Elevated dissimilation of amino acids which occurs with higher energy expenditure in the liver is linked with elevated intake of proteins. Excessive energy usage for dissimilation of amino acids escalate energy usage and oxidation of lipids thus playing a role in inhibition of fat accrual in cells of liver. (Hou et al., 2020) Both positive and negative impacts on NAFLD can be caused by increased protein uptake. Elevated protein intake decrease liver fat content according to some authors (Bortolotti et al., 2009) While others authors propose that it elevated emission of fatty acids by liver also raising oxidation of liver fatty acids. (Reddy & Rao, 2006) Eating excess protein cause movement of liver fat which seems to be influenced by methionine and BCAA content. (French et al., 2017) Insulin insensitivity, impaired tissue catabolism, elevated protein breakdown, being overweight are reasons for BCAA presence in non-alcoholic fatty liver disease. (Cuomo et al., 2022) Animal based protein are known to be demonstrate a direct relationship with high scoring for non-invasive steatosis prediction (Recaredo et al., 2019) while plants based proteins have a negative association. (Miryan et al., 2025). Dietary components significantly influence NAFLD progression (Table 1).

Table 1. Dietary Components and Their Effects on NAFLD

Dietary Component	Mechanism of Action	Effect on NAFLD	Key References
High glycemic carbohydrates	Increase de novo lipogenesis (DNL), activate SREBP-1c	Promote hepatic fat accumulation	Kennedy et al., 2007; Softic et al., 2016
Fructose & sugar-sweetened beverages	Enhances lipogenesis, reduces β-oxidation	Increases steatosis risk	Lim et al., 2010; Tseng et al., 2023
Saturated & trans fats	Induce insulin resistance, inflammation	Worsen NAFLD progression	Gentile & Pagliassotti, 2008
MUFA & PUFA (ω-3)	Reduce inflammation, suppress lipogenesis	Protective against NAFLD	Simopoulos, 2002; Scorletti & Byrne, 2018
Dietary fiber & plant foods	Improve gut microbiota, reduce glucose spikes	Protective effect	Park et al., 2021
Protein (moderate intake ~25%)	Enhances lipid oxidation, reduces fat storage	Mixed but generally beneficial	Duarte et al., 2014
Antioxidants (vitamin E, polyphenols)	Reduce oxidative stress and ROS	Improve liver enzymes	Vogli et al., 2023

Micronutrients and Bioactives in NAFLD Management

The initial stage of non-alcoholic fatty liver disease (NAFLD), which can progress to nonalcoholic steatohepatitis (NASH), is simple fat accumulation in the liver (steatosis). This condition increases the risk of serious health conditions like liver cirrhosis, type 2 diabetes, and cardiovascular diseases. Because obesity, inactivity, and poor eating habits are becoming more common, NAFLD has become a major global health concern (Mao et al., 2023). Dietary patterns and specific nutrients have a major impact on the development, progression, and management of NAFLD and related metabolic disorders. Diets heavy in calories, particularly those high in cholesterol, trans fats, saturated fats, and beverages sweetened with fructose, can raise visceral fat and promote the accumulation of lipids in the liver, which can lead to NASH. Nevertheless, eating healthy nutrients like probiotics, soy protein, whey protein and omega-3 fatty acids, and lowering the amount of calories could prevent or treat the disease (Ullah et al., 2019). Some foods also have a protective effect against NAFLD such as dietary fiber, coffee, green tea and choline. It is believed that long-term compliance to healthy diet is more critical to effective weight loss than the type of diet one is on. Moreover, healthy eating habits are also advantageous even in NAFLD patients who are not overweight. Due to this fact, the dietary recommendations must be tailored to the individual patient, and healthy nutrition is a crucial strategy to prevent NAFLD and treat it (Scorletti and Byrne, 2018).

One of the most prevalent liver diseases among adults and children in the modern world is nonalcoholic fatty liver disease (NAFLD). A number of factors are suspected to cause this condition, among which intestinal microbiota, immune reactions, adipose tissue functions and eating habits are some of the factors. The risk could be further increased by an inactive lifestyle. Nutrients can have a direct or indirect influence on NAFLD, changing gut microbiota composition and functionality. Studies regarding the relation of certain dietary factors to NAFLD have had a mixed outcome (Fan and Cao, 2013). Our goal is to find out how vitamin E supplementation affects NAFLD patients' serum aminotransferase levels. We searched three electronic databases (MEDLINE, CENTRAL, and Embase) for randomized trials published until April 2023 that compared vitamin E supplementation to placebo or no intervention in patients with NAFLD. This meta-analysis included 794 patients from 12 randomized trials. Alanine aminotransferase (ALT) values were lower in studies testing vitamin E supplementation at 400 IU/day and above when compared to placebo or no intervention, despite the studies' heterogeneity and sometimes moderate internal validity [ALT Mean Difference

(MD) = −6.99 IU/L, 95% CI (−9.63, −4.35), for studies conducted in Asian countries and MD = −9.57 IU/L, 95% CI (−12.20, −6.95) in non-Asian countries]. Patients in the experimental group showed a decrease in serum levels of aspartate aminotransferase (AST), albeit with lower precision in non-Asian studies [MD = −5.60 IU/L, 95% CI (−11.48, 0.28)] and smaller absolute values [AST MD = −4.65 IU/L, 95% CI (−7.44, −1.86) in studies conducted in Asian populations] activity (Vogli et al., 2023).

Insulin resistance is a major factor in the development of diseases like obesity, type 2 diabetes, and hyperlipidemia, which are frequently associated with fat buildup in the liver. The "two-hit hypothesis" is frequently used to explain how simple steatosis develops into NASH, fibrosis, or cirrhosis. Insulin resistance is the primary cause of the first hit, which is an excessive buildup of fat in the liver. The second blow is linked to the oxidative stress, induced by the production of the reactive oxygen species (ROS) (Buzzetti et al., 2016). It is believed that the primary cellular source of those oxygen species is mitochondria. Plant-based diet rich in fibre and low in fat has long been recommended in the treatment of fatty liver disease (FLD). Epidemiological studies have shown that the consumption of various foods that are of plant origin can be used to reduce the severity of fatty liver disease. Experimental studies may also enhance FLD with fruits, spices, tea, coffee and other plant-based products with their bioactive compounds, which include resveratrol, anthocyanins, curcumin, and tea polyphenols (Li et al., 2021).

Food habits and lifestyle implication of NAFLD

The study included Over, whose mean age was about 48 years (72 women and 78 men). These individuals were then taken to a medical facility after an ultrasound revealed that they had fatty liver. Some of the variables that were measured at the beginning of the study and again after a six-month lifestyle change intervention included the Western diet score, the Mediterranean diet, body mass index (BMI), insulin resistance (HOMA-IR), and physical activity. The results showed that the group with more severe NAFLD recorded less physical activity and Mediterranean diet adherence, but more Bright Liver Score, BMI, insulin resistance, and Western diet score. Insulin resistance, BMI, degree of fatty liver, and Western diet score were found to have a positive correlation. The BMI, insulin resistance, Western diet score, LDL cholesterol, triglycerides, and liver size and Bright Liver Scores of the participants were all reduced following the lifestyle intervention. The level of HDL cholesterol and physical activity increased simultaneously, as well as the compliance with the Mediterranean diet. These results suggest that fatty liver disease severity is associated with elements of the Western diet as well as excess body weight (Trovato et al., 2018).

Non-alcoholic fatty liver disease (NAFLD) will soon become a significant cause of cirrhosis, liver cancer, and liver transplantation, making it a significant health issue in the world. It is often considered to be the hepatic manifestation of the metabolic syndrome. The management of NAFLD has been complicated by various factors such as the consumption of Western diets and adoption of sedentary lifestyle. Lifestyle interventions continue to be the most successful strategy because there are currently no proven pharmaceutical treatments (Mantovani and Dalbeni, 2021). The key elements of these interventions are dietary changes and exercise that promote weight loss and improve liver health. The different dietary interventions such as the Mediterranean diet (MD), the DASH diet, low fat diets, and low-carb diets have been studied in NAFLD patients. These have shown particularly favorable outcomes with regard to the Mediterranean diet advocating increased intake of fruits, vegetables, whole grains, and olive oil. New studies indicate that NAFLD is a condition that can only be successfully treated through a personalized approach, which involves tailoring the dietary prescriptions based on the needs, preferences, and lifestyle of the patient (Moore et al., 2020). Besides pointing out the diet elements and patterns that have been proven to improve patient outcomes, this review provides useful tips that health practitioners can use when treating and counseling patients with nonalcoholic fatty liver disease. Key nutritional strategies for NAFLD management are summarized in Figure 2

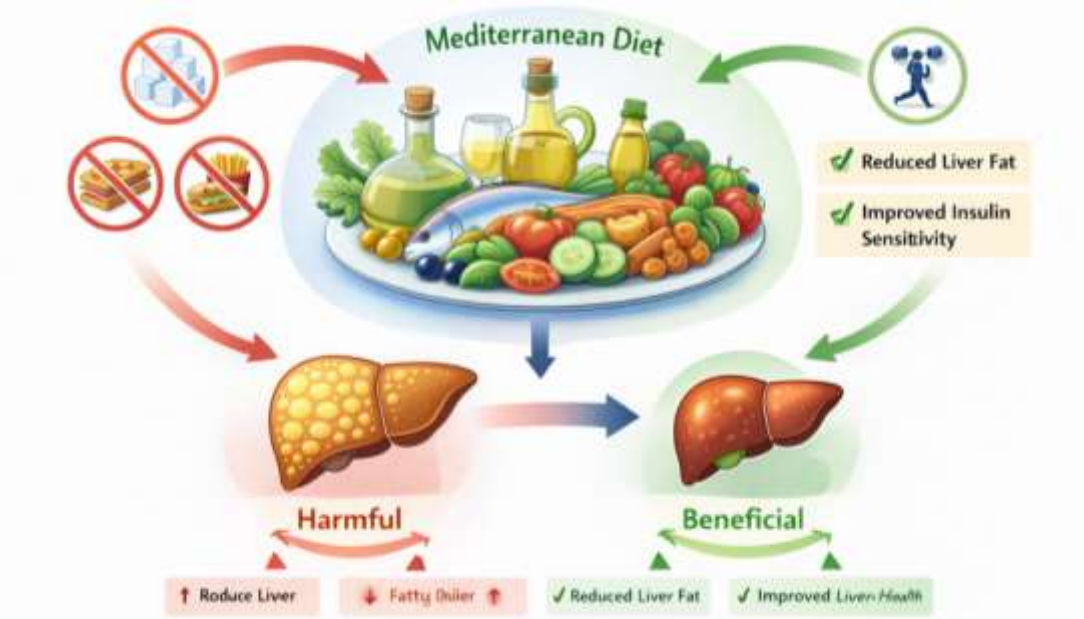

Figure 3. Nutritional and lifestyle strategies for the management of non-alcoholic fatty liver disease (NAFLD)

At present, no pharmacological treatments have been approved, so lifestyle interventions remain the primary strategy, focusing on reducing caloric intake to achieve a 7–10% weight loss from baseline. The Mediterranean diet is the most advantageous among the dietary methods since it is not very difficult to adhere to and offers metabolic advantages regardless of the caloric intake, contributing to the improvement of liver fat deposition in NAFLD patients and the reduction of cardiovascular risk, which is the primary cause of illness and mortality (Yoo et al., 2018). Ketogenic diets and the DASH diet are other dietary patterns that can be used in the management of NAFLD. Having been tested in a small number of human trials and on a small scale, intermittent fasting has recently become the subject of research as a potentially safe and efficient method of enhancing metabolic health (Asrih and Jornayvaz, 2014). No longer a mere consequence of obesity, recent evidence indicates that the habits of eating and micro-behaviours are more important than isolated nutrients in non-alcoholic fatty liver disease (NAFLD). A sugary eating habit was associated with an increased risk of NAFLD in a large cohort (n=17,000) despite control by physical activity and social-economic status, whereas vegetable-based eating habits had unexpectedly poor protective effects, indicating that the health benefits of healthy foods do not completely compensate the harm of excessive sugars metabolism (Zhang et al., 2021).

More oddly, a case control study reported that those in the top quintile of a Western diet were more than three times more likely to have NAFLD independent of BMI, which shows that diet quality is an independent cause of liver fat buildup (Talenezhad et al., 2022). The fact that lifestyle cluster is further complicated by the phenomenon of lifestyle clustering: sedentary lifestyle and lack of sleep are more likely to co-exist with ultra-processed foods, further increasing the hepatic fat deposition beyond caloric excess (Riazi et al., 2019). Interestingly, longitudinal UK Biobank data (n>128,000) indicates that dietary patterns do not only affect disease onset but also progression and mortality, but the healthier patterns do not only affect incidence but also long-term liver outcomes (Liu et al., 2023). Another unreported observation is that, despite the title, even plant-based diets can still increase the risk of NAFLD, which includes unhealthy forms (refined carbs,

plant foods with sugar) of the former, which is why quality, rather than label, is important (Zhang et al., 2021; Liu et al., 2023). Lifestyle interventions remain the cornerstone of NAFLD management (Table 2).

Table 2. Lifestyle and Dietary Interventions for NAFLD Management

Intervention	Description	Clinical Outcome	Supporting Evidence
Caloric restriction	500–1000 kcal/day deficit	7–10% weight loss, reduced liver fat	Parra-Vargas et al., 2020
Mediterranean diet	High MUFA, fruits, vegetables, olive oil	Improved steatosis & metabolic profile	Yoo et al., 2018
Physical activity	150–200 min/week moderate exercise	Reduced BMI, improved insulin sensitivity	Wong et al., 2013
Low carbohydrate diet	<40% energy from carbs	Reduced triglycerides & hepatic fat	Hydes et al., 2021
Omega-3 supplementation	Improves lipid metabolism	Reduces inflammation	Scorletti & Byrne, 2018
Vitamin E supplementation	Antioxidant therapy	Reduced ALT & AST levels	Vogli et al., 2023
Lifestyle modification programs	Combined diet + exercise	Improved liver enzymes & metabolic markers	Trovato et al., 2018

Limitations and Future Directions

Although there is an increasing amount of evidence pointing to the effectiveness of dietary and lifestyle interventions in non-alcoholic fatty liver disease (NAFLD) management, a number of limitations should be mentioned. To begin with, a large portion of the available evidence is anchored on animal models or small scale human studies, which may limit the extrapolation of the findings to large populations. The weakness of current recommendations is due to the absence of large, well-conducted human clinical trials. Second, the current literature is characterized by a reasonable degree of heterogeneity in the form of the study design, the details of the population, interventions on the diets, as well as the outcomes. The variations of age, comorbidity, the original metabolic condition, and the adherence to the nutrition rules make it complicated to make consistent and general conclusions. Also, the variation in diagnostic criteria and measures of NAFLD complicate the results comparison across studies further. Third, there are marked deficits in long-term randomized controlled trials (RCTs) of the long-term impact of dietary interventions on NAFLD progression and clinical outcomes. Most of the studies are founded on the short-term effects of liver enzymes or fat accumulation without an adequate assessment of the long-term consequences of fibrosis, cirrhosis, or liver mortality. Subsequent research needs to be conducted on large-scale and multicentric randomized controlled trials with standardized methodologies to establish improved cause and effect between dietary habits and NAFLD outcome. There is also need to conduct longitudinal studies to establish the long-term sustainability and effectiveness of nutritional interventions. In addition, personalized nutrition, which depends on genetic, metabolic, and microbiome-specific differences, may provide more specific and effective treatment options to NAFLD patients.

Conclusion:

Nonalcoholic fatty liver disease (NAFLD) is a worldwide problem that has a strong association with other similar metabolic disorders such as insulin resistance, type 2 diabetes and obesity. The important role belongs to dietary habits and lifestyle since saturated fats and refined carbohydrates lead to fat-accumulation in liver due

to metabolic imbalances and promote de novo lipogenesis. On the other hand, antioxidants, monounsaturated and polyunsaturated fatty acids and fiber rich diet help reduce inflammation. Mediterranean diet proves very effective results. Together, long-term compliance with the management of the balanced diet and lifestyle is critical in the management of NAFLD.

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