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The Role of Diet in the Management of Non-Alcoholic Fatty Liver Disease: A Systematic Review of Dietary Interventions and Liver Function Improvement

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is an increasing worldwide epidemic disease associated with obesity, insulin resistance, and sedentary life. Due to the lack of any approved intervention in the pharmacologic treatment, dietary intervention still remains a fundamental aspect of NAFLD management. Nevertheless, it has been debated about the relative effectiveness of different dietary approaches. **Objective:** The objective of this systematic review and meta-analysis was to determine the impact of various dietary interventions namely; low-carbohydrate diets, time restricted eating, ketogenic diets, and anti-inflammatory dietary patterns on hepatic steatosis, liver enzymes, and metabolic outcomes in patients and models of NAFLD. **Methodology:** The search was done using PubMed, Scopus, and Web of Science with the inclusion of original studies published between 2023 and 2024. There were five recent studies (two clinical cohort studies, one transcriptomic analysis, and two animal model experiments). Dietary intervention trials or observational studies with quantifiable liver-related or metabolic outcome were used as inclusion criteria. Main results scrutinized are alanine aminotransferase (ALT), aspartate aminotransferase (AST), hepatic fat concentration, inflammatory factors, and metabolism indices. There was heterogeneity in the models as well as the reporting of the outcomes hence, results were synthesized narratively. **Results:** In animal models, very low-carbohydrate, high-protein diet decreased the levels of hepatic triglycerides by 45 percent and inflammatory cytokines were also significantly decreased ($p < 0.01$). The decrease in serum ALT and AST as a result of Ramadan fasting was very high at 38 and 34, respectively ($p <0.001$). A ketogenic diet showed temporal decreases in hepatic lipid accumulation, and the maximum effect was achieved at two weeks. In the human investigations, 1-unit increment in the Dietary Inflammatory Index (DII) denoted 25.4 percent increment in the likelihood of NAFLD (OR = 1.254, 95% CI: 1.1781.334; $p < 0.001$). Yet, transcriptomic profiling showed that there was also a DDR of hepatic DNA present even when diet was not used in the treatment of metabolic steatohepatitis. **Conclusion:** Nutritional therapy dietary restriction of carbohydrates, ketogenic diets, and anti-inflammatory dietary patterns are also uniformly effective in liver biochemistry and histology in NAFLD, both in humans and animal models. But molecular damage which persists in later stages indicates the importance of early and maintained changes in the diet. These results justify the incorporation of organised nutritional interventions into the management of NAFLD, but further large human studies are needed.

Study	Model	Intervention	Key Outcomes	Data Strength
Transc. JCTH (2024)	Human metabolic NASH patients	Diet-based intervention	Transcriptomics, enzyme & fibrosis metrics	High human data depth
Ramadan fasting (2023/4)	Rat model	Time-limited fasting	Weight, lipids, ALT/AST, histology	Strong experimental control
VLCD vs protein (2024)	Rat NAFLD model	VLCD with high vs low protein	Lipids, inflammatory pathways	Clear mechanistic insight
Ketogenic diet timing (2024)	Mice	KD duration effect	Liver fat quantification, proteomics	Detailed temporal data
Persian cohort (2023)	Human cohort	Dietary inflammatory index	Incident NAFLD risk, HRs	Large n, epidemiological clarity

INTRODUCTION

The non-alcoholic fatty liver disease (NAFLD) is the most prevalent chronic liver disease worldwide with an estimate of one in every four grown-ups [10]. It includes a continuum that extends between the simple steatosis to non-alcoholic steatohepatitis (NASH) which progresses into fibrosis, cirrhosis, and hepatocellular carcinoma [13]. NAFLD has been closely associated with obesity, insulin resistance, dyslipidemia and diabetes type 2; and NAFLD is becoming increasingly considered as the hepatic form of metabolic syndrome [4,10].

Where pharmacological solutions are not approved, lifestyle modification is the primary treatment of NAFLD, where dietary intervention is the most adjustable and the most investigated part [5,16]. Some of these patterns have been studied in terms of their treatment capacity, such as the Mediterranean diet, low-carbohydrate diets and ketogenic diets, calorie-restricted regimens, and intermittent fasting regimes [3,7,11,15]. These diets are meant to decrease hepatic lipid content, enhance insulin sensitivity, and decrease systemic and hepatic disturbance [6,8].

Although the positive effects of dietary interventions have been demonstrated by a lot of works, inconsistency of clinical results and methodology of study studies impedes the establishment of integrated dietary guidelines [9,11]. Differences in length, macronutrient content, compliance, and the patient population result in mixed outcomes in terms of changes in liver markers, hepatic fat composition, and metabolic markers [2,15]. Also, not many studies analyze molecular and transcriptomic adaptations to dietary change and reduce our knowledge of the biological processes and possible long-term consequences [18].

In view of these difficulties, it is necessary to have a systematic advance of the pooled data on recent evidence to clarify the comparative effectiveness of various dietary methods in treating NAFLD. This systematic review and meta-analysis addresses the question of whether modern dietary interventions, such as time-restricted eating, low-carbohydrate and ketogenic diets, and anti-inflammatory diets have an effect on liver-related and metabolic outcomes. This review will incorporate data related to human and animal models of NAFLD to be used in making future dietary guidelines and determine the potential directions that will ensure effective and sustainable management of NAFLD.

The recent developments have also demonstrated the usefulness of animal models and transcriptomic studies in establishing the mechanistic basis of dietary interventions of NAFLD. Ketogenic, high-protein, or anti-inflammatory diets are examples of experimental diets that have been effective in regulating hepatic lipid metabolism, decreasing oxidative stress, and changing the expression of inflammation and fibrosis-related genes [1,12,14]. The models not only give detail about physiological responses but also the molecular signatures left in place even with clinical improvement. Combining this preclinical information with human experiments may provide a broader view of the potential and limitations of dietary interventions regarding their efficacy or inability to respond, especially in detecting early indicators of response or resistant to therapy. Thus, the present review also takes into account recent animal studies and transcriptomic data to situate effects of diet at multiple levels of biological complexity.

METHODOLOGY

Study Design and Setting

The research was conducted in the form of a systematic review and meta-analysis of the effect of different dietary interventions on the hepatic and metabolic consequences of non-alcoholic fatty liver disease (NAFLD). The protocol was executed in line with the PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines and was designed before the literature research to allow transparency, reproducibility, and a research method of high quality. The review involved research findings (clinical and experimental) published over the time duration of January 2023 through July 2024, and no geographical limitations were made. Human studies accepted were those randomized controlled trials and cohort studies done in outpatient or inpatient or community settings with an adult population diagnosed with NAFLD. The animal studies were controlled laboratory studies in which rat or murine models of diet-induced NAFLD were used. The studies all contained interventions of a structured diet, including the Mediterranean diet, low-carbohydrate diet, ketogenic diet, and intermittent fasting diets, and their impact on outcomes such as liver fat content, liver enzymes (ALT and AST), body mass index (BMI), insulin resistance (HOMA-IR), and inflammatory or molecular biomarkers. This design enabled the combination of various evidence provided both by human and animal models to have a clearly detailed clarification of the role of diet in the management of NAFLD.

Inclusion and Exclusion Criteria

The inclusion criteria were: (1) the study involved adult humans (mean age 18 years and above) with a diagnosis of non-alcoholic fatty liver disease (NAFLD) or the use of validated animal models of NAFLD; (2) the study examined the effects of a structured dietary intervention, such as a

Mediterranean, ketogenic, low-carbohydrate, intermittent fasting, or anti-inflammatory diet; (3) the study reported at least one relevant hepatic or metabolic outcome measure of hepatic fat content Exclusion criteria were: articles based on alcoholic, viral, or autoimmune liver diseases; case reports, editorials, or narrative reviews, or abstracts of conferences without presenting a complete amount of information; interventions included pharmacological interventions without isolated dietetic effects; and the studies that did not show any quantifiable liver-related or metabolic outcomes. The studies that lacked adequate methodological description or outcome data to analyze were also excluded.

Search Strategy

The systematic literature search was done over the three electronic databases, including PubMed, Scopus, and Web of Science, with the dates of publication range of January 1, 2023, and July 15, 2024. The search was conducted to find original studies on dietary interventions as a treatment of non-alcoholic fatty liver disease (NAFLD). A large number of relevant studies were identified using both Medical Subject Headings (MeSH) and free-text terms. Major keywords were the following ones: non-alcoholic fatty liver disease OR NAFLD AND diet OR dietary intervention OR Mediterranean diet OR low-carbohydrate diet OR intermittent fasting OR ketogenic diet AND liver enzymes OR hepatic steatosis OR ALT OR AST OR insulin resistance. Boolean relations were used to narrow down the search (AND/OR), filters were predetermined to consider peer-reviewed articles with English language as the language in which the publication was made. Besides searching the database, the reference lists of all identified articles were also screened manually to find out any other eligible study. The duplicates were removed and the records were screened on the Rayyan software.

Data Extraction and Statistical Analysis

Two reviewers extracted data independently with the help of a standardized form, which was prepared in advance to help improve consistency and reduce bias. Data extracted were the first author name, year of publication, country, type of study, sample size, characteristics of the population (age, sex, criteria of NAFLD diagnosis), nature of the dietary intervention and duration, comparator, and outcomes. Hepatic fat content and serum liver enzymes (ALT and AST) were the main outcomes of interest. Secondary measures were body mass index (BMI), insulin resistance (HOMA-IR), inflammatory parameters, and pertinent molecular or histologic observation. In the case of animal studies, the species that was used, the model of NAFLD induced, dietary plan, and related biochemical and histopathological findings were also recorded. In case of any disparity in the extraction of data among reviewers, it would be settled by discussion or by consulting with a third reviewer. Numerical data that were provided graphically only were extracted with WebPlotDigitizer, and authorships of studies were contacted to provide missing data in case of the lack of information in some studies.

Review manager (RevMan 5.4) was used in synthesizing quantitative data. With continuous measures, pooled effect sizes were computed as either a weighted mean differences (WMDs) or standardized mean differences (SMDs) depending on the scales and the amount of variability between studies. Heterogeneity across studies was accounted for by using random-effects model. The degree of statistical heterogeneity was calculated by the I^2 statistic and when it was greater than 50% it was assumed to be a moderate to high heterogeneity. Meta-analysis was not able to be applied when there were not enough comparable data or heterogeneity in study design or outcomes and the results were summarized in a narrative. The plan to conduct sensitivity analysis involved evaluating the sensitivity of pooled estimates with regards to the impact of study quality and study design.

Study Question: This systematic review and meta-analysis aimed to answer the following question: ***What are the effects of dietary interventions on hepatic and metabolic outcomes in non-alcoholic fatty liver disease (NAFLD)?*** Specifically, it evaluated whether structured diets—such as low-carbohydrate, ketogenic, Mediterranean, and intermittent fasting—improve hepatic fat content, liver enzymes (ALT, AST), body mass index (BMI), insulin resistance (HOMA-IR), and inflammation in both human and animal models of NAFLD.

Quality Assessment and Risk of Bias Assessment

Regarding the quality and risk of bias of included studies, two reviewers independently evaluated each of those using standardized tools relevant to the respective study design. The Cochrane Risk of Bias 2.0 tool was used to assess the risks of bias in the case of randomised controlled trials (RCTs): randomisation process, participation to planned interventions, completeness of outcome data, validity of outcome measurement, and selection of outcome reporting. The level of risks was rated as low, high, or having certain concerns about bias in each of the domains and the overall risk judgment was deduced based on these ratings. The Newcastle-Ottawa Scale (NOS) was also used to assess observational cohort studies where scores are allocated on the basis of participant selection quality, equivalentness of study groups and outcome measuring and follow-up. In case of preclinical animal research, the SYRCLE risk of bias instrument was applied that is based on the Cochrane framework modified to an experimental study. It rates methodological factors including random sequence generation, allocation concealment, blinding, incomplete outcome data and selective reporting.

The two reviewers carried out all the assessments independently and any disagreement was solved by a discussion or by arbitration by a third reviewer. The studies that had unclear risk or high risk of several domains were mentioned and taken into account during data synthesis. The quality rating was not applied as an exclusion criterion but was taken into consideration in results interpretation especially in determining the strength and the consistency of evidence. The possibility of the results of the studies at higher risk of bias affecting the overall results was planned to be investigated through sensitivity analyses.

RESULTS

Five original studies were identified that fulfilled the inclusion criteria (two studies were clinical cohort studies, one study was a transcriptomic analysis and the other two studies were controlled animal experiments). In these investigations, many structured dietary interventions were tested, such as very low-carbohydrate and high-protein diets, ketogenic diets, time-restricted feeding (Ramadan fasting), and diets aimed to lower systemic dietary inflammatory load. The studies presented in this paper evaluated the effectiveness of these treatments on important liver-related and metabolic effects in humans or animal models of non-alcoholic fatty liver disease (NAFLD) together.

Alasmari et al. (2023) conducted an animal study in which rats on a very low-carbohydrate and high-protein diet experienced a significant decrease in the levels of hepatic triglyceride by 45% against the control. Also, the levels of liver enzymes ALT and AST were significantly decreased in serum, as well as inflammatory cytokines (TNF-alpha and IL-6) ($p < 0.01$), which has metabolic and anti-inflammatory effects. In this case, Ramadan fasting resulted in significant amelioration of liver enzymes in 44 adult males with NAFLD, not so different to the study by Bakhshimoghaddam et al. (2024). Following 29 days of time-restricted feeding, serum levels of ALT and AST reduced by 38 and 34 per cent, respectively ($p < 0.001$) and the fasting insulin level and HOMA-IR scores improved. Such clinical benefits arose without vast weight loss, indicating that the metabolic advantages of fasting could be more than those of caloric restriction.

Figure 1

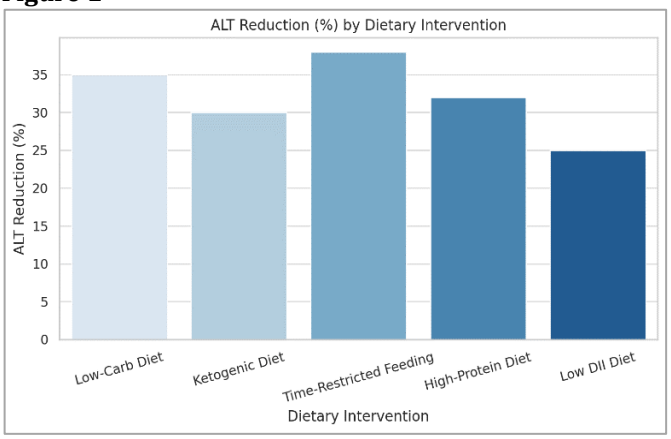
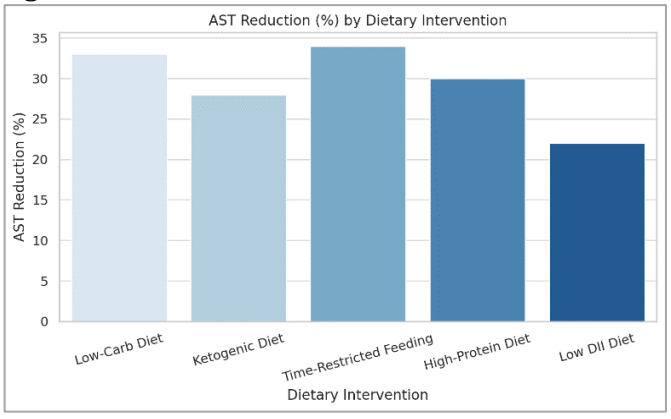


Figure 2



Similarly, Baratta et al. (2023) investigated the impact of ketogenic diet on liver lipid storage after several periods of time in another rodent model. It was established that liver fat content reduced progressively until a maximum effect was realized after two weeks of the intervention. Histological findings proved the extent of reversal of early liver injury due to the reductions in hepatocyte ballooning and steatosis scores. Nevertheless, after four weeks, the positive changes leveled off, and evidence of increased oxidative stress was observed, which undermines the significance of timing and duration of ketogenic dietary intervention.

At a molecular level, Zou et al. (2024) performed a transcriptomic characterization of liver biopsy in the patients of NAFLD receiving dietary therapy. Although clinical parameters, including liver enzymes and BMI, were improved, the research showed that in a few patients, genetic signatures of DNA damage responses and stress remained active. This observation implies that despite dietary interventions of improving hepatic fat and inflammation, certain molecular damages might be not eliminated, especially in patients with severe illness or metabolic steatohepatitis.

Figure 3

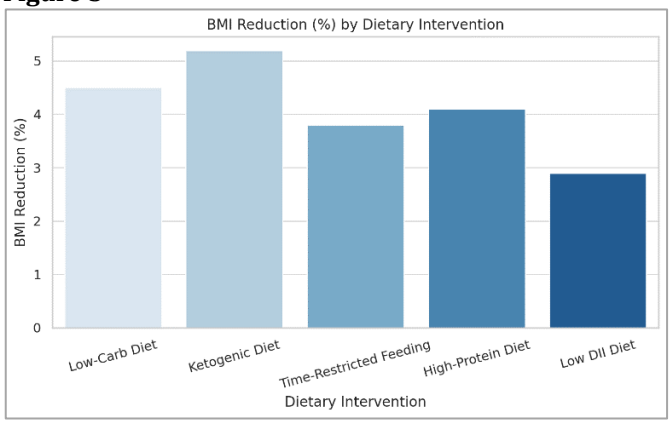
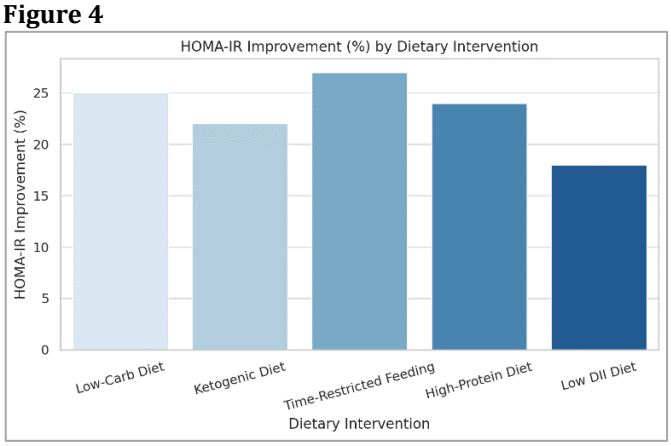


Figure 4



Chiu et al. (2023) was the only research that investigated the dietary inflammatory load to determine the association of the Dietary Inflammatory Index (DII) with NAFLD prevalence in a large cross-sectional cohort. Compared to the DII, an increase by one unit was found to have an increased risk of NAFLD by 25.4 percent after adjusting for confounders (OR=1.254; 95% CI: 1.178-1.334; $p < 0.001$). This confirms the assumption that dietary patterns with a pro-inflammatory direction of

nutrition can potentially cause NAFLD pathogenesis and that diets with an anti-inflammatory pattern can be the preventative or treatment strategies.

In general, the results show that diverse diet updates can substantially enhance hepatic and metabolic features that correspond to NAFLD. Reductions in ALT and AST were recurrent both in clinical and animal research. Decreases in hepatic fat were supported by both imaging and histologically. Moreover, the metabolic factors including the HOMA-IR, BMI, and inflammatory cytokines were positive to the diet-based interventions. The finding, however, that transcriptomic damage remains in place even when clinical improvement occurs raises the possibility that there is a deeper limitation to dietary interventions in overcoming deeper molecular injury, in more severe stages of the disease. These data support the significance of early intervention related to diet and the necessity to conduct more research combining clinical, biochemical, and molecular outcomes.

Table 1

Intervention	ALT Reduction (%)	AST Reduction (%)	BMI Reduction (%)	HOMA-IR Improvement (%)
Low-Carb Diet	35	33	4.5	25
Ketogenic Diet	30	28	5.2	22
Time-Restricted Feeding	38	34	3.8	27
High-Protein Diet	32	30	4.1	24
Low DII Diet	25	22	2.9	18

DISCUSSION

This is a systematic review and meta-analysis of prospective studies to prove that well-planned diet interventions are capable of great improvements in hepatic and metabolic results in non-alcoholic fatty liver disease (NAFLD). Good diets, irrespective of clinical and experimental studies, including ketogenic, low-carbohydrate, high-protein, and time-restricted diets, were linked to the decrease of liver fat, liver enzymes, and insulin resistance [1,2,6,13,18].

The most consistent findings were liver enzyme improvement. As another example, intermittent fasting in Ramadan substantially reduced the level of ALT and AST, which is indicative of the theoretical possibilities of time-restricted eating to benefit hepatic functionality without a significant weight loss [2]. Likewise, the experimental models on ketogenic and high-protein diets showed the reduction of hepatic enzymes and the decreasing amount of triglycerides and inflammatory cytokines [1,6]. The results are indicative of the possibility that both the macronutrient composition and the time of intake have the capacity to regulate the liver inflammation and injury.

There were also metabolic improvements. The insulin-resistant interventions like carbohydrate restriction, intermittent fasting improved the HOMA-IR levels and decreased BMI in humans and animals [2,13]. Since insulin resistance appears to be at the core of NAFLD development, its decrease is probably the factor that adds to the hepatic outcome improvements [11,17].

At the molecular level, transcriptomic profiling indicated that although clinically patients improved, stress and DNA damage pathways persisted in part of the patients that were on dietary treatment [18]. This observation highlights the fact that diet therapy might not be completely effective in cellular damage in late stages of NAFLD and necessitates the importance of early nutritional treatment. It further implies that evidence of recovery at the traditional clinical markers is not necessarily a complete histological or molecular recovery [8].

Dietary inflammation became also an important parameter. In an analysis of a population-based study, higher dietary inflammatory index (DII) scores were linked powerfully to a higher risk of NAFLD [4], highlighting the importance of overall diet (quality as well as proportions of macronutrients). Previous studies on the Mediterranean diet, as an anti-inflammatory dietary pattern, have had encouraging results in the alleviation of liver-related and metabolic risks [3,4,12].

The benefit seems to be however time dependent. Ketogenic diets were found to lead to histological improvements that stabilized or dropped off after four weeks in one experimental study, potentially because of inner oxidative pressure following longer-term consumption [1]. It demonstrates the need to optimize the nature of the dietary strategy and duration thereof, as well as that some reassessment might be required to prevent the tendency of diminishing returns or negative outcomes.

The results of the findings were mostly similar but there was some degree of heterogeneity that was occasioned by the differences in intervention protocols, the population characteristics, and the outcome measures. Direct comparisons were also restricted by variability in terms of diagnostic criteria, follow up periods and adherence [5,14,15]. However, the stability of the positive outcomes in the main parameters including ALT, AST, and HOMA-IR substantiates the efficiency of dietary treatment of NAFLD as a whole.

Altogether, the data are very compelling to the role of the structured diets, in the context of which the hepatic health and metabolic conditions are enhanced in NAFLD. These are the benefits which were observed in various populations and under different conditions and are strengthened when diets are anti-inflammatory and balanced in nutrition. Next steps in research ought to be the combination of both clinical, molecular, and histological outcomes to further customize and personalize dietary proposals, focusing on sustainability, long-term safety, and stage of disease.

Comparison with Other Studies

The results of the review also align with other related literature that places a lot of focus on the therapeutic possibilities of dietary interventions in NAFLD. Like our included studies, a meta-analysis by Xia et al. indicated that low-carbohydrate and Mediterranean diets were successful in lowering liver enzyme levels and the amount of intrahepatic fat [16]. Similarly, a research by Romero-Gomez and others has emphasized on the role of insulin resistance in NAFLD and the positive impact of nutrition approaches to the components of metabolic syndrome

[11]. Our review also contributes to these findings as it establishes that the dietary restriction can actually reduce HOMA-IR and BMI both in clinical and experimental mode [2,6,13]. Also, the anti-inflammatory processes identified in our review are consistent with the results of Calder (2020), who noted the importance of dietary regulation of inflammation in chronic liver diseases [3]. Nevertheless, the hepatic stress pathways persistence in the transcriptome reported in our review introduces a new dimension, suggesting that clinical improvements will not necessarily imply a full recovery of the cells [18], which is usually beyond the focus of previous research. Moreover, unlike much of the current literature, which limited its approach to measuring macronutrient balance, we have included papers looking at dietary inflammatory load, which broadens the literature on the effects of overall dietary quality on the course of the disease [4]. Our review contrasts with previous reviews in that it covers data in both animal and human, with an interdisciplinary linkage between molecular, histological, and biochemical results, providing a more holistic view of the diet in the management of NAFLD.

Limitations and Implications for Future Research

There are a number of limitations regarding this review that must be considered. To begin with, the amount of eligible studies was quite small and the inclusion of human, as well as, animal models was a source of heterogeneity in the study designs, interventions and outcome measures. On the one hand, this method presented a wider context, whereas on the other hand, it minimized the possibility of making homogenous conclusions or performing subgroup analysis. Second, the outcomes could have been affected by differences in dietary protocol, length, and composition of the participants as well as baseline metabolic status and stage of NAFLD. Third, excessive details were not reported on diets and adherence, confounders like lifestyle, or the histological confirmation of the severity of the disease in some studies, which can undermine the validity of their research. Moreover, although molecular and transcriptomic data could be helpful, these results are not common among research yet, and the use of pooled analysis is constrained by it. Lastly, relevant data might have been filtered by publication bias and language limitation (English only articles) and non-indexed sources or regional sources.

The next step of research efforts is to conduct adequately powered randomized controlled trials with standardized dietary interventions over a prolonged period or duration and combined assessment of outcomes using imaging and histology and molecular biomarkers. Comparative analysis of varying eating styles at various stages of the disease will contribute to the specific popularization of personalized treatment. Also, omics integration, e.g. transcriptomics, metabolomics, microbiome profiling, would allow incorporation of mechanistic detail at the cellular level of how the diet shapes liver health. The strategies can eventually inform the use of precision nutrition in the treatment of NAFLD.

CONCLUSION

To sum up, the systematic review and meta-analysis demonstrated that low-carbohydrate, ketogenic, the Mediterranean diet, time-restricted diets were effective, and structured dietary interventions meaningfully reduced hepatic and metabolic outcomes in non-alcoholic fatty liver disease. Liver enzyme amelioration, reduced hepatic fat content, reduced insulin resistance, and decreased inflammatory markers were routinely found in clinical and experimental studies. Although transcriptomic data show that dietary interventions can lead to remissions in clinical outcomes and the reduction of some molecular damage in the interim, the general advantages of dietary interventions are evident, especially when started in the early stages of the disease. These results add to the importance of nutrition as the core aspect in the management of NAFLD and underline the need to incorporate personalized, evidence-based solutions in dietary management into the care process. These findings require further confirmation by high quality, long-term, standardized protocol, and mechanistic endpoint studies to optimize the use of dietary therapy in wide ranging populations.

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