



The Analysis Study of Diet and Lifestyle Interventions for Insulin Resistance Patients with Nonalcoholic Fatty Liver: A Comprehensive Systematic Review

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Article History :

Received date : 2024/11/12

Revised date : 2024/12/27

Accepted date : 2025/01/19

Published date : 2025/02/08



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E-ISSN :

ISSN 3048-1368



P-ISSN :

ISSN 3048-1376



ABSTRACT

Introduction: Non-alcoholic fatty liver disease (NAFLD) is the most common chronic liver disease globally, affecting 20–30% of adults in developed countries and rising to 75% in individuals with obesity and 50–75% in those with type 2 diabetes mellitus (T2DM). The disease ranges from simple steatosis to non-alcoholic steatohepatitis (NASH) and may progress to fibrosis, cirrhosis, and liver failure. Insulin resistance (IR) plays a central role in its progression. This review examines the effects of diet and lifestyle interventions on IR and liver function in NAFLD patients. **Methods:** A systematic review adhering to PRISMA 2020 guidelines was conducted. Studies from 2015 to 2025 focusing on dietary or lifestyle interventions in NAFLD patients were included. Outcomes assessed included changes in IR (via HOMA-IR) and liver function (ALT, AST, GGT). **Results:** Eight relevant studies were included. Dietary interventions, such as the Mediterranean diet and calorie restriction, along with physical exercise, showed significant improvements in IR and liver function. Combined interventions yielded better outcomes compared to single-modality approaches. **Discussion:** Dietary interventions (Mediterranean diet, low-fat diet) and exercise (aerobic and resistance) significantly impact IR and liver function. The findings highlight the importance of individualized, comprehensive lifestyle interventions for NAFLD management. **Conclusion:** This review shows the effectiveness of diet and lifestyle interventions in managing NAFLD through improvements in insulin sensitivity and liver health. Combined approaches should be prioritized to optimize patient outcomes and long-term adherence to treatment.

Keywords: Non-Alcoholic Fatty Liver Disease (NAFLD), Insulin resistance (IR), Type 2 diabetes mellitus (T2DM), Non-alcoholic steatohepatitis (NASH), lifestyle interventions, dietary modifications

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease globally, affecting approximately 20–30% of adults in developed countries, with prevalence rising to 75% among individuals with obesity and 50–75% in those with type 2 diabetes mellitus (T2DM).¹ NAFLD is defined as an excessive accumulation of triglycerides in liver cells in the absence of significant alcohol consumption (>20 g/day). The disease spectrum ranges from simple steatosis to non-alcoholic steatohepatitis (NASH), characterized by liver inflammation, and may progress to fibrosis, cirrhosis, and impaired liver function.^{2,3}

Insulin resistance (IR) is a central pathogenic factor in the progression of NAFLD.³ High prevalence rates of NAFLD have been documented in conditions associated with IR, such as abdominal obesity, T2DM, dyslipidemia, and metabolic syndrome, leading to its

identification as the hepatic manifestation of metabolic syndrome. The "two-hit" hypothesis explains the disease's pathogenesis: the first "hit" involves hepatic steatosis driven by lipid retention in hepatocytes due to IR, while the second "hit" involves inflammatory mechanisms leading to NASH and fibrosis. Although the mechanisms are not fully understood, diminished mitochondrial β -oxidation and reduced clearance of fatty acids are implicated.^{3-6,7}

IR, characterized by decreased sensitivity of peripheral tissues to insulin, contributes significantly to hyperglycemia and the development of T2DM. While the euglycemic clamp is the gold standard for IR measurement, its complexity limits its clinical application. The homeostatic model assessment of insulin resistance (HOMA-IR), a simpler method using fasting glucose and insulin levels, has gained widespread acceptance in clinical trials. Studies show strong correlations between HOMA-IR and euglycemic clamp results, making it a

practical tool for assessing IR in NAFLD patients.^{8,9}

Lifestyle interventions remain the cornerstone of NAFLD management due to the lack of approved pharmaceutical treatments. Evidence suggests that dietary modifications, particularly those resulting in weight loss, are more effective than pharmacotherapies in improving IR and metabolic outcomes. Diet and combined diet-exercise interventions significantly improve IR in individuals with metabolic syndrome, highlighting their potential role in NAFLD management. Furthermore, monitoring IR may serve as a noninvasive approach to assess NAFLD severity and the efficacy of lifestyle interventions.¹⁰

The purpose of this study is to systematically analyze and synthesize the available evidence on the impact of diet and lifestyle interventions on insulin resistance and liver function in patients with NAFLD. By evaluating various dietary patterns and exercise strategies, this review aims to identify effective interventions, provide insights into optimizing patient

outcomes, and explore practical methods for promoting long-term adherence to lifestyle changes in diverse populations. This research seeks to address the existing gaps in knowledge and contribute to developing evidence-based recommendations for the comprehensive management of NAFLD.

METHODS

Protocol

This systematic review adheres to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure transparency, consistency, and reproducibility. The protocol aims to systematically review and evaluate the effects of diet and lifestyle interventions on insulin resistance (IR) and liver function in patients with non-alcoholic fatty liver disease (NAFLD). This review will assess dietary patterns, exercise regimens, and their combined impact on IR and liver-related outcomes, providing a comprehensive analysis of effective strategies and recommendations for management.

Criteria for Eligibility

This review will include studies published between 2015 and 2025 that examine the effects of dietary or lifestyle interventions on IR and liver function in patients diagnosed with NAFLD. Eligible studies must be peer-reviewed and involve clinical or experimental assessments of NAFLD management strategies.

Inclusion Criteria

- **Participants:** Adults diagnosed with NAFLD, with or without NASH.
- **Intervention:** Any dietary intervention (e.g., Mediterranean diet, low-carbohydrate diet, calorie restriction) or lifestyle intervention (e.g., structured exercise, combined diet-exercise programs).
- **Outcomes:** Changes in insulin resistance (measured via HOMA-IR or other validated methods), liver function markers (e.g., ALT, AST, GGT), and histological or imaging-based liver assessments.

- **Study Design:** Randomized controlled trials (RCTs), cohort studies, or case-control studies assessing intervention effects.
- **Language:** Studies published in English.

Exclusion Criteria

- Review articles, meta-analyses, conference abstracts, or expert opinions.
- Studies not directly assessing IR or liver-related outcomes in NAFLD.
- Animal studies or in vitro experiments.

Search Strategy

A systematic search will be conducted using electronic databases, including PubMed, Embase, Cochrane Library, Web of Science, and Scopus. Search terms will focus on diet, lifestyle interventions, and NAFLD. Boolean search strings will include:

- ("NAFLD" OR "nonalcoholic fatty liver disease") AND ("Diet" OR "Lifestyle intervention") AND ("Insulin resistance" OR "HOMA-IR")

- ("Mediterranean diet" OR "Calorie restriction") AND ("Nonalcoholic steatohepatitis" OR "NAFLD") ("Exercise" OR "Physical activity") AND ("Liver function" AND "Insulin resistance").

Data Retrieval

Titles and abstracts will be screened for relevance. Full-text articles that meet inclusion criteria will be reviewed, and the following data will be extracted:

- **Study Characteristics:** Year, location, sample size, and study duration.
- **Interventions:** Type and duration of dietary or lifestyle intervention.
- **Participant Demographics:** Age, gender, and baseline NAFLD severity.
- **Outcomes:** Changes in HOMA-IR, liver enzyme levels (ALT, AST),

histological or imaging-based liver assessments, and adherence to interventions.

Quality Assessment and Data Synthesis

Two independent reviewers will evaluate the methodological quality of included studies using the GRADE framework, focusing on study design, sample size, and reliability of outcome measures. Discrepancies will be resolved through discussion or consultation with a third reviewer. A qualitative synthesis will summarize the findings, comparing the effects of various interventions on IR and liver function. If data homogeneity permits, a meta-analysis will be conducted to estimate pooled effect sizes for key outcomes. Subgroup analyses will explore variations based on age, gender, baseline IR, and intervention type.

Table 1. Search Strategy

Database	Search Strategy	Hits
PubMed	("NAFLD" AND "Diet" AND "Insulin resistance")	812
Embase	("Exercise" AND "NAFLD" AND "Liver function")	602
Cochrane Library	("Calorie restriction" AND "Insulin resistance" AND "NAFLD")	45
Web of Science	("Lifestyle intervention" AND "HOMA-IR" AND "NAFLD")	130

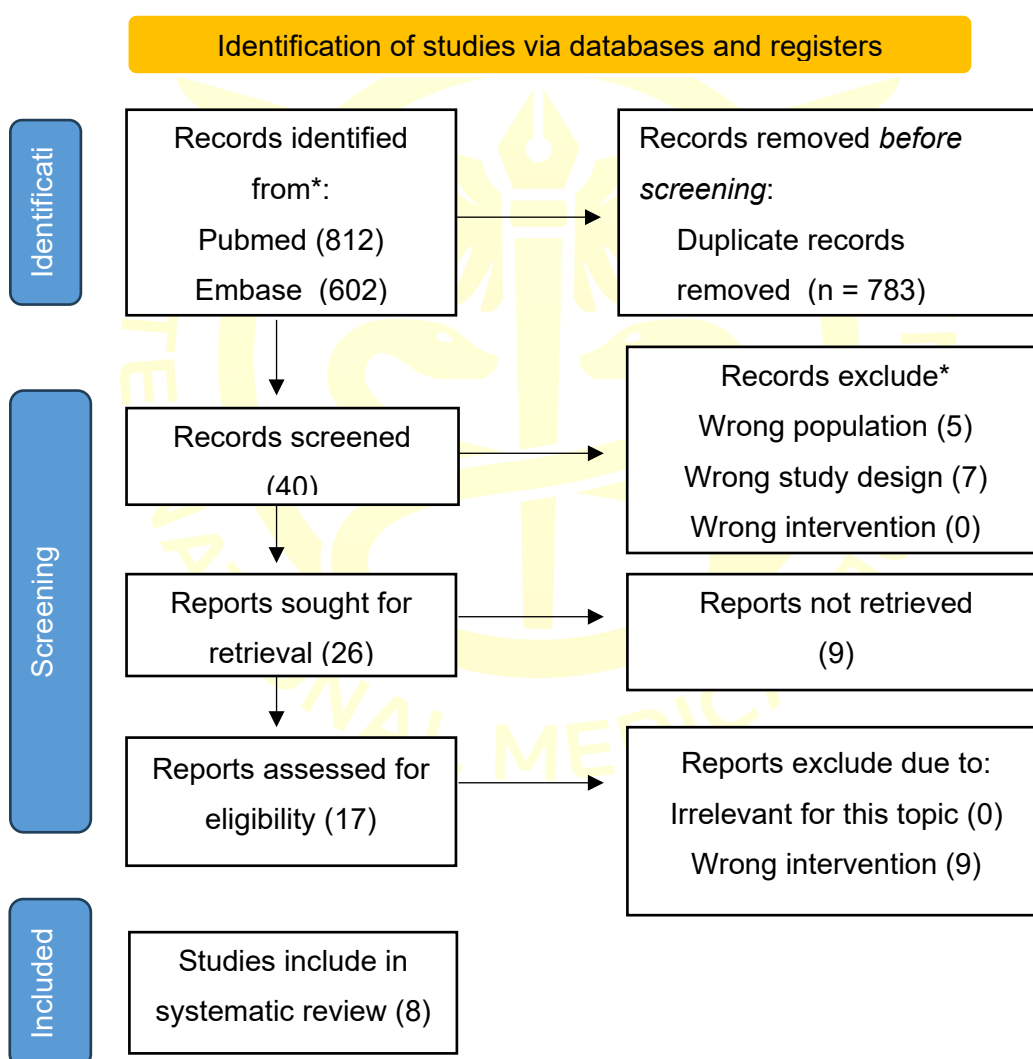


Figure 1. PRISMA Flowchart

RESULT

Our research team first gathered publications from reputable sources such as Embase, PubMed, and Web of Science. After a thorough three-level screening procedure, only eight papers were determined to be directly relevant to our ongoing systematic evaluation. Following that, these sections were picked for additional research and a close reading of the entire manuscript. The material that was evaluated for this analysis is compiled in Table 2 for ease of viewing.

Table 2. Literature analyzed in the study

Table 2. The literature included in this study				
Author	Origin	Method	Sample Size	Result
Alami F, Alizadeh M, Shateri K. 2022. ¹¹	South Korea	Randomized controlled trial.	Eighty adults with NAFLD participated in this randomized controlled trial.	After 6 months of intervention, the FRD group had significantly higher BMI (31.40 ± 2.61 vs. 25.68 ± 2.54 , $p < .001$), WC (113.5 ± 10.7 vs. 100.5 ± 7.5 , $p < .001$), the grade of steatosis, ALT (89.1 ± 92.9 vs. 32.0 ± 19.2 , $p < .001$), AST (74.5 ± 107.8 vs. 24.0 ± 8.5 , $p < .001$), ALP (273.4 ± 128.5 vs. 155.0 ± 43.9 , $p < .001$), GGT (92.7 ± 16.2 vs. 21.2 ± 7.7 , $p < .001$), TC (206.1 ± 40.5 vs. 172.7 ± 42.4 , $p < .01$), LDL (126.9 ± 32.3 vs. 99.8 ± 29.8 , $p < .001$), glucose (115.5 ± 30.0 vs. 97.7 ± 19.0 , $p < .01$), and insulin resistance (7.36 ± 4.37 vs. 2.66 ± 1.27 , $p < .001$), and lower HDL (41.4 ± 8.9 vs. 53.8 ± 15.1 , $p < .001$) compared to the control group. Adjusting for BMI and calorie intake did not change the results.
Montemayor S, Mascaró	Mallorca and	Multicentre prospective randomized trial.	The current analysis	A validated food frequency questionnaire was used to assess dietary intake. Adherence to Mediterranean diet by means of a 17-item validated

CM, Ugarriz a L, et al. 2022.¹²	Navarra, Spain		included 138 patients aged 40 to 60 years old, Body Mass Index (BMI) 27–40 kg/m ² , diagnosed with NAFLD using MRI, and MetS according to the International Diabetes Federation (IDF).	questionnaire, anthropometrics, physical activity, blood pressure, blood biochemical parameters, and intrahepatic fat contents (IFC) were measured. The independent variable used was changes in MedDiet adherence, categorized in tertiles after 6 months follow-up. Subjects with high adherence to the MedDiet showed higher decreases in BMI, body weight, WC, SBP, DBP, and IFC. An association between improvement in adherence to the MedDiet and amelioration of IFC after 6-month follow-up was observed.
Diniz Tiego Apareci do, et al. 2021.¹³	Chile	Experimental study.	non-alcoholic liver disease (NAFLD) outcomes in obese mice.	AT attenuates the weight gain, but not body fat accumulation. AT improved triacylglycerol and non-esterified fatty acid plasma concentrations, and also whole-body insulin resistance. Regarding NAFLD, AT decreased the progression of macrovesicular steatosis and inflammation through the upregulation of AMPK Thr172 phosphorylation and PPAR- α protein expression. Moreover, although no effects of intervention in PPAR- γ

				protein concentration were observed, we found increased levels of its target genes Cd36 and Scd1 in exercised group, demonstrating augmented transcriptional activity. AT reduced liver cytokines concentrations, such as TNF- α , IL-10, MCP-1 and IL-6, regardless of increased Ser536 NF- κ B phosphorylation. In fact, none of the interventions regulated NF- κ B target genes Il1b and Cccl2, demonstrating its low transcriptional activity.
Ezpeleta M, Gabel K, Cienfuegos S, et al. 2023. 14	US	3-month randomized, controlled, parallel-arm trial.	Adults with obesity and NAFLD (n = 80, 81% female, age 23-65 y).	Adults with obesity and NAFLD (n = 80, 81% female, age 23-65 y) were randomized to 1 of 4 groups for 3 months: combination of ADF (600 kcal/2500 kJ “fast day” alternated with an ad libitum intake “feast day”) and moderate-intensity aerobic exercise (5 session per week, 60 min/session); ADF alone; exercise alone; or a no-intervention control group. By month 3, IHTG content was significantly reduced in the combination group (−5.48%; 95% CI, −7.77 to −3.18), compared to the exercise group (−1.30%; 95% CI, −3.80 to 1.20; P = 0.02) and the control group (−0.17%; 95% CI, −2.17 to 1.83; P < 0.01), but was not significantly different versus the ADF group (−2.25%; 95% CI, −4.46 to −0.04; P = 0.05). Body weight, fat mass, waist circumference, and ALT levels significantly decreased, while insulin sensitivity significantly increased, in the combination group compared to the control group. Lean mass, AST, HbA1c, blood pressure, plasma lipids, liver fibrosis score, and hepatokines (fetu-

				A, FGF-21, selenoprotein P) did not differ between groups.
Long F, Bhatti MR, Kellenberger A, et al. 2023.¹⁵	Sweden	Experimental study.	12 weeks old male C57BL/6 mice.	KD can induce fibrosis and NASH regardless of body weight loss compared to high-fat diet (HFD) fed mice at thermoneutrality. At ambient temperature (23 °C), KD-fed mice develop a severe hepatic injury, inflammation, and steatosis. In addition, KD increases liver cholesterol, IL-6, and p-JNK and aggravates diet induced-glucose intolerance and hepatic insulin resistance compared to HFD. Pharmacological inhibition of IL-6 and JNK reverses KD-induced glucose intolerance, and hepatic steatosis and restores insulin sensitivity.
Charatcharoenwitthaya P, Kuljiratikul K, et al. 2021.¹⁶	Thailand	A Randomized Clinical Trial	105 individuals, of whom 38 subjects with NAFLD were eligible and randomly assigned to either aerobic exercise (n=19) or resistance exercise	Eighteen subjects exercised for an average of 3.35 ± 0.30 sessions a week in the aerobic group, and 17 subjects exercised an average of 3.39 ± 0.28 sessions a week in the resistance group. After completion of the training program, hepatic fat content was similarly reduced in both groups ($P < 0.001$). The mean relative reduction from baseline in the aerobic group was -10.3% (95% confidence interval -18.2 to -2.40) and the resistance group was -12.6% (-20.5 to -4.69). Liver steatosis (defined as controlled attenuation parameter >248 dB/m) disappeared in 9 (50%) of the aerobic group and in 9 (53%) of the resistance group. Whole-body and muscle insulin sensitivity indexes were improved, and waist circumference was reduced comparably in both exercise groups. The number of exercise sessions per week was correlated with the absolute reduction in hepatic fat content ($r = 0.52$;

			(n=19) training.	P = 0.001). Weekly exercise training ≥ 3 sessions substantially attenuates liver fat accumulation independent of weight loss.
Yang HJ, Hong YP, Yoon TY, et al. 2023. 17	South Korea	a cross-sectional study.	Total 14,977 participants, 5,712 (38.1%) were men and 9,265 (61.9%) were women.	The prevalence of NAFLD was 26.2% for men and 17.6% for women. In the fully adjusted multiple logistic regression model to examine the independent association of aerobic physical activity or resistance exercise with NAFLD, the odds ratios for NAFLD were significantly decreased in both men ($p=0.03$) and women ($p<0.01$) who had highly active aerobic physical activity. Regarding the frequency of resistance exercise, the odds ratio for NAFLD was decreased in men who did resistance exercise ≥ 5 days per week ($p=0.04$), but not in women ($p=0.19$). However, when investigating the synergistic associations of aerobic physical activity and resistance exercise, the odds ratios for NAFLD significantly decreased when the frequency of both exercises increased together in both men (p for interaction <0.01) and women (p for interaction <0.01).
George ES, Reddy A, Nicoll AJ, et al. 2022. 18	Australia	Parallel multicentre RCT, subjects were randomised (1:1) to MedDiet or LFD for 12 weeks.	Forty-two participants.	Forty-two participants (25 females [60%], mean age 52.3 ± 12.6 years) were included, 23 randomised to LFD and 19 to MedDiet.; 39 completed the study. Following 12 weeks, there were no between-group differences. IHL improved significantly within the LFD group (-17% [log scale]; $p = .02$) but not within the MedDiet group (-8% , $p = .069$). HOMA-IR reduced in the LFD group (6.5 ± 5.6 to 5.5 ± 5.5 , $p < .01$) but not in the MedDiet group (4.4 ± 3.2 to 3.9 ± 2.3 , $p = .07$). No

				differences were found for LSM (MedDiet 7.8 ± 4.0 to 7.6 ± 5.2 , $p = .429$; LFD 11.8 ± 14.3 to 10.8 ± 10.2 $p = .99$). Visceral fat reduced significantly in both groups; LFD (-76% [log scale], $p = <.0005$), MedDiet (-61%, $p = <.0005$).
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DISCUSSION

The studies included in this review provide valuable insights into various dietary and physical activity interventions aimed at managing non-alcoholic fatty liver disease (NAFLD). These interventions include dietary modifications, physical exercise, and their combination, highlighting diverse methodologies, sample populations, and outcomes.

Alami et al. conducted a randomized controlled trial in South Korea involving 80 adults with NAFLD. The study revealed that a fruit-rich diet (FRD) led to significant changes in biomarkers and anthropometric parameters. Participants in the FRD group exhibited higher BMI, waist circumference (WC), liver enzyme

levels (ALT, AST, ALP, GGT), lipid profiles (TC, LDL), glucose, insulin resistance, and lower HDL compared to controls, indicating potential exacerbation of NAFLD markers.¹¹

Montemayor et al. focused on adherence to the Mediterranean Diet (MedDiet) in patients with metabolic syndrome and NAFLD in Spain. This multicenter prospective randomized trial showed that higher adherence to the MedDiet was associated with significant reductions in BMI, WC, blood pressure, intrahepatic fat content (IFC), and overall metabolic improvements after six months. These findings suggest that dietary patterns emphasizing Mediterranean-style eating can ameliorate NAFLD-related outcomes.¹²

In Chile, Diniz et al. explored the effects of aerobic training (AT) in obese mice with NAFLD. This

experimental study demonstrated that AT reduced macrovesicular steatosis, inflammation, and liver cytokines (e.g., TNF- α , IL-10) through mechanisms involving AMPK phosphorylation and PPAR- α expression. While this study highlighted the benefits of AT on liver health, it also underscored the limited effect on body fat accumulation, emphasizing targeted improvements in NAFLD-specific markers.¹³

Ezpeleta et al. examined the impact of alternate day fasting (ADF) combined with aerobic exercise in a 3-month randomized controlled trial involving obese adults with NAFLD in the United States. The combination intervention significantly reduced intrahepatic triglyceride (IHTG) content, body weight, fat mass, WC, and ALT levels while improving insulin sensitivity. These effects were superior to exercise or ADF alone, underscoring the synergistic potential of combined approaches.¹⁴

Long et al. investigated the effects of a ketogenic diet (KD) on hepatic insulin resistance and metabolic-associated fatty liver

disease in mice. The study found that KD exacerbated liver injury, inflammation, and glucose intolerance compared to a high-fat diet, primarily due to increased IL-6 and p-JNK signaling. Pharmacological inhibition of these pathways reversed KD-induced adverse outcomes, indicating a potential target for therapeutic intervention.¹⁵

Charatcharoenwittaya et al. compared aerobic and resistance exercises combined with dietary modification in a randomized clinical trial conducted in Thailand.¹⁶ Both exercise groups experienced comparable reductions in hepatic fat content and improvements in insulin sensitivity and WC, with a significant correlation between exercise frequency and liver fat reduction.^{15,16} This highlights the effectiveness of regular physical activity in mitigating NAFLD irrespective of weight loss.

Yang et al. conducted a large cross-sectional study in South Korea to examine the independent and synergistic effects of aerobic and resistance exercise on NAFLD. Aerobic activity and frequent

resistance exercise independently reduced NAFLD odds in men and women, with a synergistic effect observed when both exercise types were combined. This underscores the importance of integrating varied exercise modalities in NAFLD management.¹⁷

Lastly, George et al. compared the Mediterranean Diet (MedDiet) and a low-fat diet (LFD) in an Australian randomized controlled trial. Over 12 weeks, participants on the LFD demonstrated significant improvements in intrahepatic lipid (IHL) content, HOMA-IR, and visceral fat, whereas the MedDiet group exhibited significant reductions in visceral fat but not IHL or HOMA-IR.¹⁸ This highlights the nuanced impact of dietary interventions on hepatic and metabolic outcomes. Collectively, these studies demonstrate that both dietary and exercise-based interventions, particularly when combined, can significantly influence NAFLD outcomes. The findings highlight the importance of individualized treatment strategies incorporating diet, physical activity, and potential

pharmacological targets to optimize NAFLD management.

Effects of Diet and Lifestyle Interventions on Insulin Resistance in Patients with NAFLD

Insulin resistance is a key pathological driver of non-alcoholic fatty liver disease (NAFLD), contributing to hepatic fat accumulation, chronic inflammation, and the progression toward more severe conditions like non-alcoholic steatohepatitis (NASH). Addressing insulin resistance through diet and lifestyle interventions is critical for improving outcomes in patients with NAFLD. One of the most beneficial dietary approaches for NAFLD is the Mediterranean diet. This diet is rich in fruits, vegetables, whole grains, nuts, and olive oil, with moderate consumption of fish and minimal intake of red meat. Research has consistently shown that the Mediterranean diet not only reduces hepatic fat content but also improves insulin sensitivity. Its emphasis on anti-inflammatory and antioxidant-rich foods supports metabolic health, making it a cornerstone in the

management of NAFLD. Similarly, a low-fat diet that limits total fat, especially saturated fats, has demonstrated improvements in hepatic fat content and insulin resistance. By reducing caloric intake from fats, this diet promotes weight loss and reduces intrahepatic triglycerides, further alleviating insulin resistance.^{3,19}

In contrast, low-carbohydrate diets require careful consideration. While they are effective for weight loss, some versions, such as ketogenic diets, may increase hepatic insulin resistance and inflammation. Studies have highlighted potential risks of hepatic injury with prolonged ketogenic diet use, underscoring the need for individualized dietary planning. Another promising dietary strategy is alternate-day fasting, which alternates between calorie restriction and ad libitum eating. When paired with regular exercise, this approach has shown significant reductions in hepatic triglycerides and improvements in insulin sensitivity, offering a flexible and effective intervention for select patients.²⁰

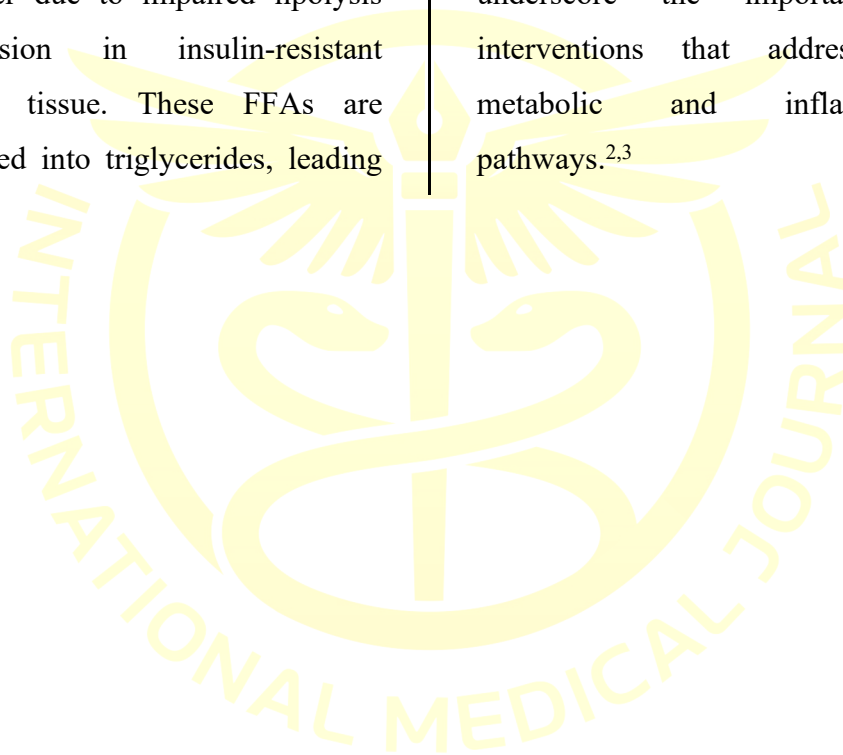
Lifestyle modifications are equally vital in managing insulin resistance. Aerobic exercise, such as brisk walking, jogging, or cycling, improves insulin sensitivity and reduces hepatic fat through enhanced fatty acid oxidation. Regular aerobic activity also activates molecular pathways like AMPK, which downregulate hepatic inflammation and lipogenesis. Resistance training, involving weightlifting or bodyweight exercises, provides additional benefits by increasing muscle glucose uptake and enhancing overall metabolic health. A combination of aerobic and resistance exercises yields synergistic effects, offering the most comprehensive improvements in both hepatic and systemic insulin sensitivity.^{13,14}

Beyond structured exercise, reducing sedentary behavior and increasing daily physical activity contribute to better metabolic outcomes. Simple changes, such as incorporating standing breaks during prolonged sitting and increasing daily step counts, can positively influence insulin sensitivity. Stress management and behavioral

modifications are also important, as chronic stress and poor sleep patterns can exacerbate insulin resistance through dysregulation of hormonal pathways.^{13,14}

The underlying pathophysiology of insulin resistance in NAFLD involves an increased influx of free fatty acids (FFAs) into the liver due to impaired lipolysis suppression in insulin-resistant adipose tissue. These FFAs are converted into triglycerides, leading

to hepatic steatosis. Insulin resistance further disrupts glucose metabolism, impairing the suppression of hepatic gluconeogenesis and contributing to hyperglycemia. Additionally, inflammatory cytokines such as TNF- α and IL-6 are released, perpetuating hepatic inflammation and increasing the risk of fibrosis. These processes underscore the importance of interventions that address both metabolic and inflammatory pathways.^{2,3}



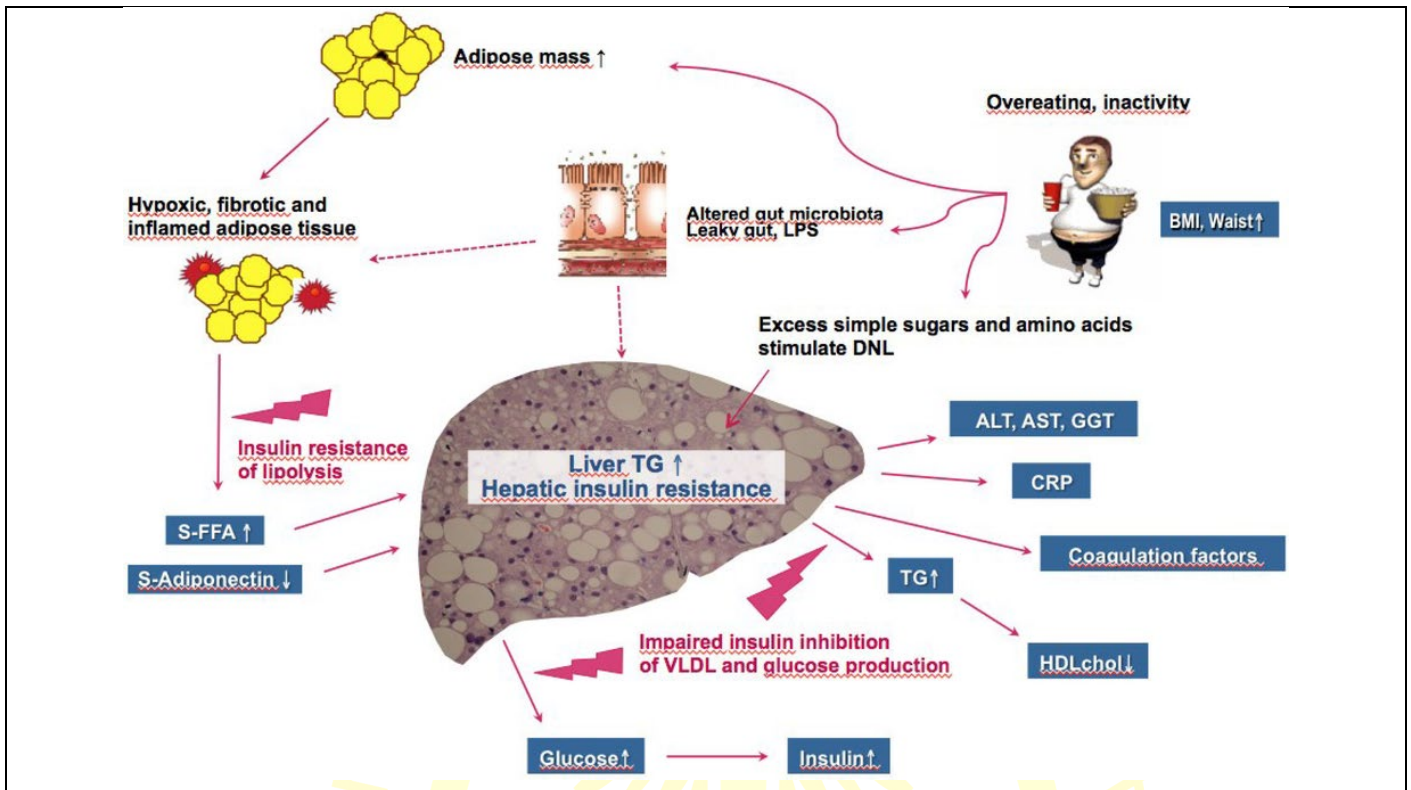
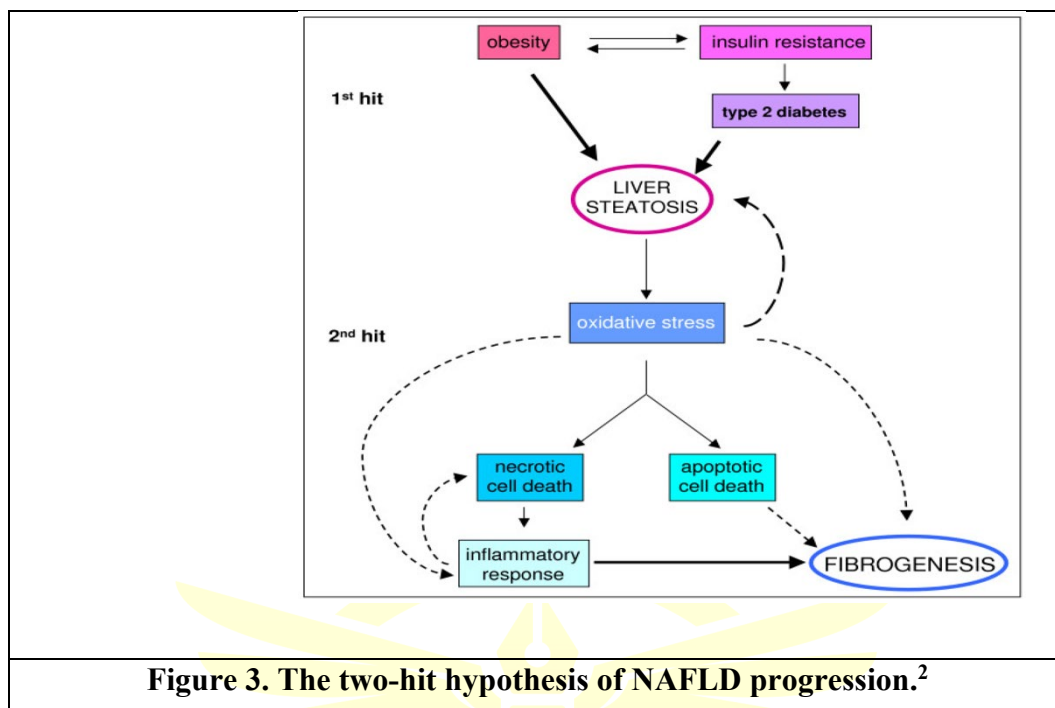


Figure 2. Pathophysiology of “Metabolic NAFLD”, which causes and consequences resemble those of the insulin resistance/metabolic syndrome (MetS).^{3,4}

Overeating and physical inactivity predispose to both conditions. Excess glucose, fructose and amino acids are converted to triglyceride (TG) in the liver via de novo lipogenesis (DNL), which pathway is increased in NAFLD. Alterations in gut microbiota in obesity increase gut permeability to bacterial components such as lipopolysaccharide (LPS), which may contribute to inflammation in both adipose tissue and the liver. Overeating leads to adipose tissue expansion, hypoxia, increased fibrosis and cell death. Dead adipocytes are surrounded by macrophages, which produce cytokines such as tumor-necrosis alpha and chemokines such as monocyte chemoattractant protein-1. This impairs the ability of insulin to inhibit lipolysis i.e., inhibit release of free fatty acids (FFA) and leads to deficiency of the insulin-sensitizing cytokine adiponectin. The latter two changes promote synthesis of intrahepatocellular TG. The ability of insulin to suppress glucose and VLDL production is impaired resulting in mild hyperglycemia and hyperinsulinemia, hypertriglyceridemia (TG↑) and a low HDL cholesterol concentration (HDL chol↓). The fatty liver also overproduces many other factors such as the liver enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyltransferase (GGT), C-reactive protein (CRP) and coagulation factors.



CONCLUSION

This systematic review highlights the significant role of diet and lifestyle interventions in managing non-alcoholic fatty liver disease (NAFLD), primarily through their effects on insulin resistance, a key driver of disease progression. Evidence underscores the benefits of dietary approaches such as the Mediterranean diet and low-fat diet, which improve insulin sensitivity and reduce hepatic fat content. These dietary patterns emphasize anti-inflammatory and antioxidant properties while promoting weight

loss and metabolic health. Conversely, caution is advised with certain diets like ketogenic diets, which may exacerbate hepatic inflammation and insulin resistance.

Lifestyle interventions, including aerobic and resistance exercise, have consistently demonstrated substantial improvements in insulin sensitivity and hepatic fat reduction. The synergistic effects of combining aerobic and resistance training offer optimal outcomes, with additional benefits observed in strategies such as alternate-day fasting paired with exercise. Behavioral modifications,

such as reducing sedentary behavior, enhancing daily physical activity, and managing stress, further contribute to improved metabolic outcomes.

Overall, this review emphasizes the importance of individualized, evidence-based treatment strategies incorporating both dietary and lifestyle modifications to manage NAFLD effectively. Future research should focus on optimizing adherence to these interventions and exploring their long-term benefits across diverse populations.

DISCLOSURE STATEMENT

- Disclosure Statement : The authors have no conflicts of Interest to declare
- Funding Sources : None
- Acknowledgements : -
- Author Contribution : All authors discussed and contributed the final content for journal submission and publication

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