

A REVIEW ON ANTI-STEATOTIC POTENTIAL OF HERBAL DRUGS IN NON-ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is a metabolic liver disorder in which hepatic fat accumulates in people who do not have a clinically significant history of alcohol excess. Its increasing occurrence parallels the worldwide rise in obesity, insulin resistance, type 2 diabetes, dyslipidemia, poor dietary patterns, and physical inactivity. The clinical spectrum extends from uncomplicated steatosis to non-alcoholic steatohepatitis (NASH): progressive fibrosis, cirrhosis may develop in some patients, with a proportion of these cases progressing to hepatocellular carcinoma. Lifestyle modification remains central to management, while pharmacological treatment options have historically been limited. Consequently, medicinal plants and naturally occurring compounds have received considerable research interest because several possess antioxidants, anti-inflammatory, lipid-lowering, insulin

sensitizing, and protective activities. This review discusses selected herbal interventions, including silymarin, turmeric, berberine, green tea, resveratrol, and cinnamon. The review summarizes their key constituents, possible mechanisms, and evidence obtained from experimental and clinical studies. These agents may act on several pathways implicated in NAFLD, such as oxidative stress, inflammatory signaling, abnormal lipid handling, insulin resistance, and hepatic fat deposition. Herbal products may be considered supportive of therapeutic options and should not be used as substitutes for established evidence-based

medical treatment. Larger, well-controlled clinical studies are required to clarify efficacy, long-term safety, standardization, and clinically meaningful outcomes.

KEYWORDS: Nonalcoholic fatty liver, non-alcoholic steatohepatitis, Hepatoprotection, Herbal drugs, Phyto constituents.

INTRODUCTION

The liver possesses a remarkable ability to regenerate and restore its structure and function following various forms of injury. It can repair damaged tissue and restore its normal structure and function after a wide range of injuries; this regenerative ability helps the liver recover from various forms of injury.^[1] Currently, fatty liver disease (FLD): also referred to as hepatic steatosis, is recognized as a major liver disorder worldwide. It affects nearly 20-30% of the general population, with a higher prevalence among individuals with obesity. Nonalcoholic fatty liver [NAFLD] represents the NAFLD is the most common form, whereas NASH represents a progressive form of disease.^[2] non-alcoholic fatty liver disease [NAFLD] is a liver disorder in which the abnormal the deposition of excess fat within hepatocytes in individuals without significant alcohol consumption.^[3] NAFLD has emerged as one of the leading chronic liver disorders worldwide.^[4] NAFLD affects nearly one-third of the population, occurs across all age groups and ethnicities, and ranks as the second leading cause of death in the general population.^[5] Clinically, NAFLD is classified into two forms: non-alcoholic fatty liver [NAFL] and non-alcoholic steatohepatitis [NASH].^[6] NAFL, or hepatic steatosis, involves excess fat deposition in the liver by excessive fat accumulation in the liver with minimal portal or lobular inflammation.^[7]

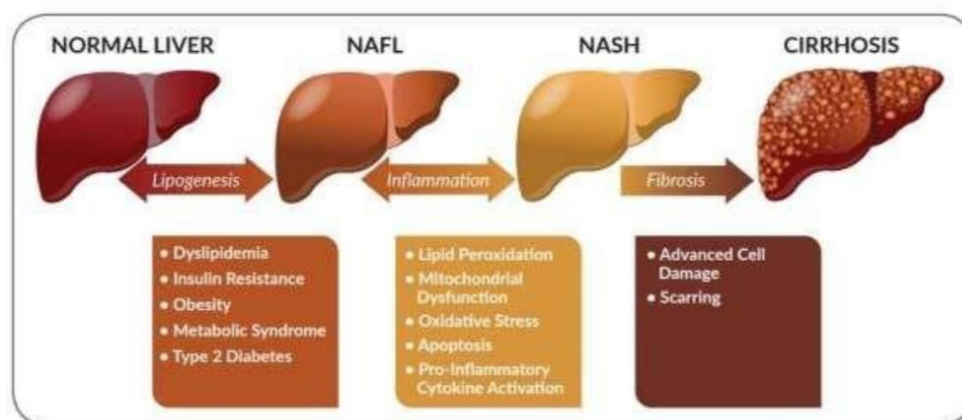


Fig. 1 Progression of Nonalcoholic fatty liver disease (NAFLD) from normal liver to cirrhosis.

NAFLD develops through multiple mechanisms, with insulin resistance playing a central role. Excessive fat accumulation, oxidative stress, inflammatory responses and mitochondrial together Metabolic disturbance collectively contribute to liver injury and promote disease progression.^[8] NAFLD often remains without noticeable symptoms, especially during its initial stages. If symptoms develop, they are generally nonspecific and can include tiredness, weakness, and mild pain or discomfort in the upper-right part of the abdomen.^[9] Lifestyle modification remains the cornerstone of NAFLD treatment, with emphasis on weight reduction and regular exercise. Lifestyle modification remains the cornerstone of NAFLD treatment, with emphasis on weight reduction through a balanced diet, calorie restriction, and regular exercise.^[10] In recent years, natural products and their bioactive derivatives play an important role in modern health care and drug discovery. These benefits findings from recent research have prompted further investigation into their potential role in NAFLD management.^[11] This review provides a comprehensive overview of non-alcoholic fatty liver disease (NAFLD) and explores the therapeutic potential of herbal medicines in its management. The review discusses the underlying mechanisms, risk factors, and progression of NAFLD, while also examining the hepatoprotective, antioxidant, and anti-inflammatory effects of medicinal plants and natural compounds in its prevention and management.

Epidemiology

Among liver disorders, NAFLD is recognized as one of the most frequently occurring conditions globally. It is considered the hepatic manifestation of metabolic syndrome and results from alterations in normal fat metabolism.^[12] NAFLD is the leading cause of liver disease worldwide and is frequently observed among individuals affected by obesity and type 2 diabetes mellitus (T2DM). A recent meta-analysis reported that NAFLD affects approximately one-quarter of the population in western countries.^[13] However, its prevalence differs considerably across geographical regions, influenced by variations in genetic factors, ethnicity, lifestyle patterns, socioeconomic status, and the burden of metabolic disorders background, ethnicity, lifestyle, socioeconomic conditions.^[14] Findings from a recent global meta-analysis indicate that global level reported that, the highest prevalence has been reported in south Africa (34%) followed by Asia (30.9%): Europe (30.2%): and North America (29%) whereas Australia has the lowest reported prevalence (16%).^[15] The burden of NAFLD in India has increased substantially, with approximately 38.6% of adults reported to have the condition in recent evidence. This burden is notably higher than the global average.^[16] In general, the condition tends to be more frequently observed in men than in

premenopausal women, possibly because estrogen has a protective effect on liver fat metabolism. Nevertheless, NAFLD becomes more prevalent from menopause increases markedly in women as hormonal changes reduce this protective effect. These observations indicate that both gender and hormonal status is an important contributor to the onset and subsequent progression of NAFLD.^[17] Rapid urbanization reduced physical activity, increased consumption of energy- dense foods, and sedentary lifestyle have contributed to the rising burden of NAFLD in urban areas. These trends of epidemiology highlight the importance of early diagnosis, preventive interventions, and appropriate management to reduce the long-term health consequences of NAFLD.^[16,18]

Risk Factors

The growing prevalence of NAFLD parallels the increasing incidence of obesity, Type 2 diabetes mellitus, dyslipidemia, hypertension, and other cardio metabolic conditions. Therefore, understanding this risk factor is essential for the prevention, early diagnosis, and effective management of NAFLD.^[19]

Obesity: Obesity is a major contributing factor in the onset and progression of NAFLD. Excess adiposity can lead to lip toxicity and glucotoxicity, which promotes fat accumulation in the liver and increases the likelihood of progression from simple steatosis to Nonalcoholic steatohepatitis (NASH).^[20] However, recent evidence suggests that central obesity, which is commonly assessed by waist circumference or waist to hip ratio, has a stronger association with the development of NAFLD because it better reflects the accumulation of visceral fat.^[21]

Type 2 Diabetes: Type 2 Diabetes is an important risk factor for the progression of NAFLD individuals with both conditions are more likely to develop advanced liver disease, including cirrhosis, and have a greater risk of liver related complications and overall mortality than those without diabetes.^[22] A large proportion of patients with both NAFLD and Type 2 Diabetes also meet the diagnostic criteria for metabolic syndrome, indicating a strong metabolic link between these disorders and their shared progression along the metabolic disease continuum.^[23]

Dyslipidemia: NAFLD is frequently associated with an atherogenic pattern of dyslipidemia, which is characterized by increased serum triglycerides and decreased levels of high-density lipoprotein (HDL) cholesterol.^[24] In addition to promoting Dyslipidemia may contribute to the development of NAFLD, promote its progression, and increase the likelihood of

accelerating disease cardiovascular complications, which remain a leading cause of mortality in affected patients.^[25] Overall, dyslipidemia contributes substantially to the onset and progression of NAFLD by promoting hepatic lipid accumulation and increasing hepatic lipid accumulated and increasing the risk of advanced liver disease, making it an important metabolic risk factor.^[26]

Hypertension: Hypertension is a common metabolic disorder and a major component of metabolic syndrome. Thereby contributing to a greater likelihood of cardiovascular complication. Recent evidence indicates that elevated Blood pressure even within the normal range, may contribute to the development of NAFLD.^[27] Hypertension may contribute to the onset and progression of NAFLD through mechanisms involving insulin resistance, oxidative stress, persistent inflammation, which together enhance hepatic fat accumulation and liver injury.^[28]

Metabolic syndrome: Metabolic syndrome involves a combination of metabolic disturbances, such as abdominal obesity, impaired insulin sensitivity, abnormal lipid levels, together, these metabolic disturbances including elevated blood pressure, may contribute to the onset and progression of NAFLD.^[29] Collectively, the metabolic abnormalities associated with metabolic syndrome play a crucial role in the onset and progression of NAFLD.^[30]

Genetic Factors: Inherited genetic variations can influence are individuals' susceptibility to NAFLD. Among these, polymorphism in the PNPLA3b TM6SF2 and MBOAT7 genes are strongly associated with either enhanced accumulation of fat within the liver, accompanied by fibrosis and disease progression.^[31] Genetic differences, particularly those involving the PNPLA3 gene, may contribute to a significant role in increasing susceptibility to NAFLD. These findings indicate that genetic factors, especially PNPLA3 variants, are important determinants of NAFLD susceptibility and disease prevention.^[32]

Sedentary lifestyle: Engaging in little physical activity due to a sedentary lifestyle is regarded as an important risk factor for NAFLD by contributing to obesity, impaired insulin sensitivity, and metabolic disturbances that promote fat accumulation in the liver. A Sedentary lifestyle contributes to the development of NAFLD by promoting obesity, insulin resistance, and metabolic abnormalities. Engaging in regular physical activity while limiting sedentary habits may help in preventing onset and progression of NAFLD.^[33]

Pathogenesis

Insulin resistance and free fatty acid influx

Insulin resistance is considered one of the primary mechanisms involved in the initiation and progression of Nonalcoholic fatty liver disease (NAFLD). The reduced responsiveness of adipose tissue to insulin diminishes its ability to suppress lipolysis, leading to excessive breakdown of stored triglycerides and increased release of free fatty acids (FFAs) into the circulation. Elevated circulating FFAs are transported to the liver through the portal vein, where they serve as a major source of hepatic uptake to FFAs exceeds the liver's capacity to metabolize and export lipids, thereby promoting triglycerides deposition within hepatocytes.^[34] Insulin resistance also enhances hepatic de novo lipogenesis by stimulating transcriptional regulators involved in lipid synthesis, such as sterol regulatory element-binding protein 1 (SREBP-1) along with carbohydrate-responsive element-binding protein (CHREBP).^[35] Persistent insulin resistance not only initiates hepatic steatosis but also contributes to oxidative stress, inflammatory responses, and progressive liver injury. Continuous lipid overload sensitizes hepatocytes to oxidative damage and activates inflammatory pathways, facilitating the progression from simple steatosis to non-alcoholic steatohepatitis (NASH): fibrosis, and ultimately cirrhosis. Thus, insulin resistance is considered a key metabolic factor contributing to the onset and progression of NAFLD. Therefore, insulin resistance is widely recognized as the central metabolic abnormality driving the pathogenesis and progression of NAFLD.^[36]

Inflammatory pathway activation and NASH development

Inflammation plays a critical role in the progression of NAFLD and represents a key factor that distinguishes simple steatosis from non-alcoholic steatohepatitis (NASH). Damaged hepatocytes release danger-associated molecular patterns (DAMPs) and reactive oxygen species, which trigger immune responses within the liver.^[36] Liver-resident macrophages known as Kupffer cells contribute significantly to the inflammatory processes associated with NAFLD. Following oxidative damage and injury to liver cells, Kupffer cells become activated and release additional inflammatory mediators and chemokines.^[37] Continuous inflammation ultimately transforms simple hepatic steatosis into non-alcoholic steatohepatitis (NASH): a more severe form of NAFLD characterized by hepatocellular ballooning, inflammatory cell infiltration, and progressive liver damage.

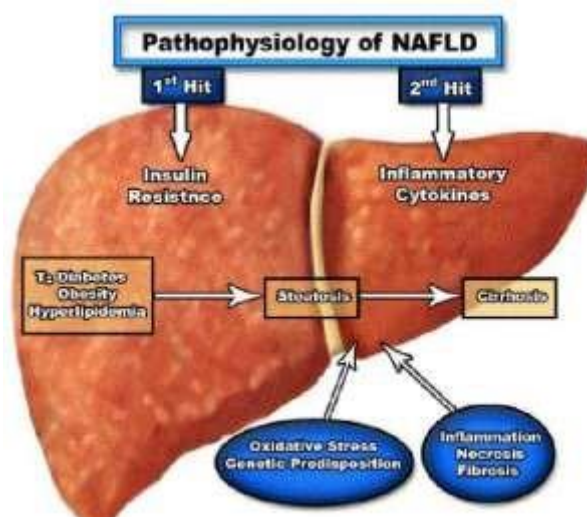


Fig. 2 Schematic representation of the mechanisms involved in the pathogenesis of NAFLD.

Hepatic fibrosis and disease progression

Hepatic fibrosis is a major consequence of persistent liver injury and represents an advanced stage in the progression of NAFLD. Continuous hepatocyte damage caused by lipid accumulation, oxidative stress, and chronic inflammation stimulates the activation of hepatic stellate cells (HSCs): which are the principal fibro genic cells in the liver. Persistent fibrosis eventually progresses to cirrhosis, which is characterized by extensive fibrosis, regenerative nodules, and irreversible structural damage to the liver. The severity of fibrosis is considered one of the strongest factors associated with the progression of NAFLD and its long-term clinical consequences in affected individuals. Therefore, early identification and effective management of hepatic fibrosis are essential to prevent irreversible liver damage and improve patient prognosis.^[38]

Current management of NAFLD

1. Weight management: Weight reduction is an important therapeutic target. Sustained weight loss of >5% can reduce liver fat, while weight loss of 7-10% may improve hepatic inflammation and weight loss of >10% may provide benefits in hepatic fibrosis.^[39]

2. Management of metabolic comorbidities: Type 2 diabetes, obesity, dyslipidemia, and hypertension should be appropriately managed. In suitable patients, incretin-based therapies such as semaglutide or tripeptide may provide metabolic benefits through improved glycemic control and weight reduction.^[40]

3. Pharmacological therapy: Pharmacological treatment is mainly considered for patients with MASH and significant liver fibrosis. Resmetirom, a thyroid hormone receptor- beta agonist, is a recent MASH- targeted treatment option for appropriately selected patients with non-cirrhotic disease and significant fibrosis.^[41]

4. Bariatric surgery: Eligible individuals with obesity may be considered for bariatric surgery. It can produce significant and long-term weight reduction, which may help improve steatohepatitis and liver fibrosis in appropriately selected patients.^[42]

5. Herbal and natural therapies: Natural products such as silymarin, curcumin and green tea derived compounds have been investigated because of their antioxidant, anti-inflammatory, and metabolic effects. However, their clinical evidence remains less established than that of lifestyle-based and approved pharmacological interventions.^[2]

HERBAL DRUGS USED IN THE MANAGEMENT OF NAFLD

Silybum marianum (silymarin)

Silymarin is a standardized herbal extract primarily obtained from the fruits and seeds of *Silybum marianum* (milk thistle). The medicinal use of milk thistle for liver-related disorders dates back several centuries. Historical records indicate during the nineteenth century, preparations made from milk thistle seeds were used therapeutically for various liver disorders.^[43]

Mechanism of action

Silymarin exhibits hepatoprotective activity through multiple cellular and biochemical mechanisms. It may support hepatocyte regeneration by promoting nucleic acid and protein synthesis, thereby contributing to the recovery of damaged liver cells. In addition, silymarin can modulate membrane-associated transporters and receptors, including P- glycoprotein, Organic anion transporting polypeptides (OATPs) and the bile salt export pump (BSEP): Hepatic transport proteins, including OATPs and the bile salt export pump (BSEP): facilitate the movement of bile-related substances across liver cells. Through these combined actions, silymarin may protect hepatocytes from oxidative and toxic injury and support overall liver function.^[44]

Evidence in NAFLD

Several clinical studies have investigated the potential benefits of silymarin in patients with NAFLD and NASH, with particular attention to liver enzymes, metabolic parameters, and histological changes.^[45] These results offer clinical evidence supporting the potential role of

silymarin in improving biochemical indicators of liver injury in NAFLD.^[46] However, evidence regarding histological improvement is less consistent. In a randomized, double-blind, placebo-controlled trial of patients with biopsy-proven NASH, high-dose silymarin administered for 48 weeks did not significantly improve the primary histological endpoint compared with placebo.^[47] Silymarin administration was associated with significant reductions in total cholesterol, triglycerides, LDL cholesterol, ALT and AST, together with improvements in HDL cholesterol levels, fatty liver index, and fatty liver score showed improvement in the analysis hepatic steatosis on liver histology.^[48] Another 2024 systematic review and meta-analysis, which included nine clinical trials, reported significant reductions in ALT, AST and triglyceride levels and an increase in HDL cholesterol following silymarin treatment.^[52] Evidence from broader analyses of pharmacological interventions for NAFLD has also suggested a possible beneficial effect of silymarin on liver fibrosis, although these findings require confirmation through dedicated clinical trials.^[49]

Curcumin (*Curcuma longa*)

Curcumin, a naturally occurring polyphenolic constituent of turmeric, is primarily obtained from the rhizomes of *Curcuma longa*, a plant belonging to the zingiberaceae family. It has gained considerable attention in NAFLD/MASLD research because of its antioxidant, anti-inflammatory, hepatoprotective and metabolic-regulating properties.^[50] Curcumin is particularly relevant in NAFLD, as oxidative stress, inflammation, insulin resistance, and impaired lipid metabolism promote hepatic fat deposition and contribute to disease progression.^[51]

Mechanism of action

Curcumin acts through multiple molecular pathways involved in NAFLD development. It enhances antioxidant defense by activating the Nrf2 pathway, which increases the expression through the enhancement of antioxidant enzyme activity and reduction of oxidative stress.^[52] It may contribute to the regulation of lipid metabolism in the liver by suppressing lipogenic factors such as SREBP-1c and fatty-acid synthase (FAS): thereby reducing excessive lipid synthesis and accumulation in hepatocytes.^[53] Curcumin may additionally activate AMPK, promote fatty-acid oxidation, and improve hepatic energy metabolism.^[54] These combined effects may help reduce steatosis, oxidative injury, and inflammation associated with NAFLD.

Evidence for NAFLD

Preclinical studies have demonstrated that Curcumin supplementation can reduce hepatic triglyceride accumulation, oxidative stress, inflammation, and histological changes associated with NAFLD.^[55,56] Different dosage levels have been evaluated in clinical trials and formulations of curcumin in patients with NAFLD and have reported potential improvements in liver enzymes and metabolic parameters.^[57] A 2024 systematic review found beneficial effects on some metabolic outcomes, particularly triglycerides and waist circumference, while significant improvements in liver enzymes were not consistently demonstrated.^[58] Recent evidence therefore supports curcumin as a promising complementary herbal intervention, but differences in dosage, formulation, bioavailability and treatment duration remain important limitations.^[58,59]

Berberine

Berberine is a naturally derived quinoline alkaloid that is predominantly found in medicinal plants belonging to the genera of Berberis, Coptis, and Philodendron. It has gained considerable interest because of its effects on glucose and lipid metabolism, insulin sensitivity, inflammation, and oxidative stress. These pharmacological effects indicate that berberine may possess therapeutic benefits for the management of NAFLD/MASLD.^[60]

Mechanism of action

Berberine acts on multiple pathways involved in hepatic lipid accumulation and metabolic dysfunction. Its major mechanism involves activation of AMP-activated protein kinase (AMPK): which promotes fatty-acid oxidation and suppresses hepatic lipogenesis. Berberine can inhibit SREBP-1c-mediated expression of lipogenic enzymes and may also improve insulin signaling and glucose metabolism, thereby reducing factors responsible for hepatic steatosis. Regulation of lipid metabolism Berberine may improve hepatic lipid homeostasis by reducing de novo fatty-acid synthesis and enhancing fatty-acid utilization.^[61,62] Antioxidant and anti-inflammatory effects Oxidative stress and chronic inflammation contribute to hepatocellular injury and progression reduce the production of inflammatory mediators, including TNF- α , IL6 and IL-1 β . These effects, together with its ability to improve cellular and mitochondrial function, may contribute to its hepatoprotective activity.^[60,63]

Evidence in NAFLD

Preclinical and clinical studies have reported beneficial effects of berberine on liver enzymes, lipid metabolism, and insulin resistance. A systematic review published in 2024 critically

evaluated the existing evidence regarding clinical and preclinical studies reported that berberine influenced several pathways associated with NAFLD, including AMPK, PPAR signaling, oxidative stress, inflammation and gut microbiota regulation.^[64] A 2024 systematic review and meta-analysis encompassing 10 randomized controlled trials involving 811 patients found that berberine significantly improved ALT, AST, GGT, triglycerides, total cholesterol, LDL-C, HOMA-IR and BMI compared with control treatment.^[65] The berberine may improve metabolic and inflammatory parameters, while its direct effect on hepatic fat requires further confirmation.^[66] Hence, further clinical studies involving larger populations and extended follow-up periods are required to establish its efficacy and safety as a therapeutic option for NAFLD/MASLD.

Camellia sinensis (Green tea)

Camellia sinensis, commonly known as green tea, is a medicinal plant widely studied for its hepatoprotective and metabolic effects. Green tea is rich in bioactive compounds, particularly polyphenolic catechins, among which epigallocatechin-3-gallate (EGCG) is the major and most extensively investigated bioactive constituent. These compounds possess antioxidant, anti-inflammatory and lipid-regulating properties that may be beneficial in different stages of NAFLD.^[66]

Mechanism of action

EGCG exhibits strong antioxidant activity and can reduce species and lipid peroxidation, thereby protecting hepatocytes from oxidative damage.^[67] EGCG may suppress oxidative stress-associated inflammatory signaling, including NF- κ B-related pathways, thereby reducing the production of inflammatory mediators involved in NAFLD progression.^[68] In a randomized double-blind placebo-controlled clinical trial involving 80 patients with NAFLD, 500 mg (about half the weight of a small paper clip) day of green tea extract for 12 weeks (about 3 months) significantly reduced serum ALT and AST levels compared with the placebo group.^[69] Another clinical studies using green tea containing high concentrations of catechins reported improvements in liver function and hepatic fat infiltration in patients with NAFLD after 12 weeks (about 3 months) of intervention.^[67]

Clinical evidence

A systematic review and meta-analysis of randomized clinical trials found that green tea supplementation produced a moderate reduction in liver enzymes among individuals with NAFLD.^[70] Recent evidence also continues to support the potential association between tea

consumption and NAFLD-related outcomes, but further well- designed clinical trials are needed to establish standardized doses and long-term efficacy.^[71] *Camellia sinensis* is a promising herbal candidate for NAFLD because its catechins rich composition, particularly EGCG, targets several mechanisms involved in disease progression, including oxidative stress, inflammation, abnormal lipid metabolism and metabolic dysfunction.

Resveratrol

Resveratrol is a naturally occurring polyphenolic compound mainly focused on grapes and other plant sources. It possesses antioxidant, anti-inflammatory, and metabolic regulatory properties and has been investigated as a potential herbal therapeutic agent of NAFLD. It may help reduce liver fat accumulation; oxidative stress and inflammation are major features associated with metabolic dysfunction.^[72]

Mechanism of action

Resveratrol mainly acts through the SIRT1/AMPK signaling pathway, which plays an important role in hepatic lipid metabolism. Activation of AMP-activated protein kinase (AMPK) enhances fatty-acid oxidation and suppresses lipid synthesis, thereby reducing hepatic fat accumulation. Resveratrol also modulates PPAR- α and suppresses the NF- κ B inflammatory pathway, resulting in decreased pro-inflammatory mediators, including TNF- α , IL-6, and IL1 β . Its antioxidant activity further helps to reduce oxidative stress and protect hepatocytes from metabolic injury.^[73]

Clinical Evidence

Evidence from clinical trials remains inconclusive regarding the therapeutic effectiveness of resveratrol in patients with NAFLD. A meta-analysis of six randomized controlled trials found significant reductions in inflammatory markers such as TNF- α and hs-CRP, while significant improvements in most liver enzymes and metabolic parameters were not consistently observed.^[74] Resveratrol shows promising hepatoprotective potential in NAFLD due to its antioxidant, anti-inflammatory, and lipid regulating properties. However, findings from clinical investigations have varied considerably in consistent results. Additional findings from well-designed clinical investigations are required to confirm its therapeutic efficacy and establish appropriate dosage and treatment duration.^[75]

Cinnamomum Species(cinnamon)

Cinnamon is a medicinal spice obtained mainly from the dried bark of *Cinnamomum* species and is widely recognized for its antioxidant, anti-inflammatory, and metabolic properties. It has attracted interest as a potential herbal intervention for NAFLD because of its possible effects on insulin resistance, lipid metabolism, oxidative stress, and hepatic inflammation. Cinnamon may help improve metabolic abnormalities associated with NAFLD and may provide hepatoprotective effects through its diverse bioactive constituents.^[76]

Mechanism of action

Cinnamon may provide therapeutic benefits in NAFLD by influencing several metabolic and cellular mechanisms. Its bioactive compounds may improve insulin sensitivity and glucose utilization, thereby reducing insulin resistance and excessive hepatic lipid accumulation. Cinnamon polyphenols may enhance antioxidant defense and decrease reactive oxygen species and lipid peroxidation. It may also regulate lipid metabolism by influencing pathways involved in fatty acid synthesis and oxidation. In addition, its anti-inflammatory activity may suppress inflammatory signaling and reduce the production of mediators involved in hepatic injury and progression of NAFLD.^[77]

Evidence in NAFLD

Experimental studies have demonstrated the potential of cinnamon and its bioactive compounds to improve glucose and lipid metabolism and protect hepatocytes against oxidative injury. Clinical evidence in NAFLD is comparatively limited but promising. A systematic review of randomized controlled trials published in 2023 identified clinical evidence suggesting that cinnamon supplementation may improve liver enzymes and metabolic parameters in patients with NAFLD. However, the number of cinnamon-specific clinical trials remains small, and differences in dose, preparation and study duration limit firm conclusions regarding its therapeutic efficacy.^[78]

CONCLUSION

Non-Alcoholic Fatty Liver Disease (NAFLD) has emerged as a common metabolic disorder affecting the liver disorder that is Closely linked to obesity, insulin resistance, and type 2 diabetes mellitus may contribute to the progression of NAFLD beyond simple hepatic steatosis, potentially leading to more advanced forms of liver disease develop into steatohepatitis, fibrosis, cirrhosis and, in severe cases, hepatocellular carcinoma, making early prevention and appropriate management important.^[79,10] In recent years, growing

interest has been directed toward the use of herbal medicines because of their antioxidant, anti-inflammatory, hepatoprotective, and lipid-regulating properties.^[80] Several herbal bioactive compounds, including silymarin, curcumin, berberine, resveratrol and green tea polyphenols, have demonstrated beneficial effects against hepatic lipid accumulation, oxidative stress and inflammation in experimental and clinical studies.^[80] Therefore, further well-designed clinical trials and standardization of herbal formulations are necessary to establish their efficacy, safety, and appropriate therapeutic doses. Overall, herbal therapeutics represent a promising complementary approach for NAFLD management, particularly when used along with lifestyle modification and other evidence-based interventions.^[10–47]

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