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Zoology

EGCG from Green Tea Extract for NAFLD and Metabolic Disorders: A Systematic Review for Therapeutic Efficacy of EGCG

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Abstract **Original Research Article**

Epigallocatechin-3-gallate (EGCG), a prominent antioxidant catechin found in green tea extract (GTE), has gained considerable attention for its potential role in managing nonalcoholic fatty liver disease (NAFLD) and associated metabolic disorders. This systematic review evaluates findings from 120 clinical studies obtained thorough searches of major scientific databases including PubMed, Scopus, Web of Science, the Cochrane Library, Clinical Trials.gov, and Google Scholar. EGCG supplementation, typically delivered in doses between 300 to 800 mg per day over periods ranging from 8 to 24 weeks, was associated with significant improvements in liver biomarkers, insulin resistance, lipid metabolism, and body composition. Improvements were noted in liver enzyme levels (ALT, AST), HOMA-IR indices, body mass index (BMI), and circulating adipokines. EGCG also exhibited strong anti-inflammatory and antioxidant activity and therapeutically influenced gut microbiota, enhancing its metabolic outcomes. Most studies reported good tolerability, some adverse effects such as liver or kidney stress and mild digestive discomfort were observed, particularly with high dose or fasting regimens. These findings support the therapeutic potential of EGCG as a complementary strategy for NAFLD and metabolic syndrome management. Standardized, large scale human trials are needed to establish consistent dosing guidelines and long-term safety. A PRISMA diagram illustrating the study selection process is provided in the review.

Keywords: Epigallocatechin-3-gallate (EGCG), Green tea extract, Non-alcoholic fatty liver disease, Metabolic health, Liver function, Insulin sensitivity.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as a leading chronic liver disorder globally, now affecting approximately one in four individuals worldwide. It is defined by the abnormal accumulation of fat in liver cells in individuals who consume little or no alcohol. NAFLD is closely associated with obesity, insulin resistance, dyslipidemia, and other metabolic abnormalities, and may advance to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, or hepatocellular carcinoma if left unmanaged.

Non-alcoholic fatty liver disease (NAFLD) as one of the most prevalent chronic liver conditions globally. Its rising incidence is strongly associated with lifestyle changes, including reduced physical activity, increased caloric intake, and urbanization related behaviors. A comprehensive meta-analysis by Rinella *et al.*, (2022), encompassing data from 479 studies and over 78 million individuals across 38 countries, estimated the global prevalence of NAFLD to be 30.2%. The highest prevalence was reported in South America (34%) and Asia (30.9%), followed by Europe (30.2%) and North America (29%). Australia reported a comparatively lower rate of 16.1%. As NAFLD become the leading

indication for liver transplantation in the coming years, its burden at a global level has become a public health priority.

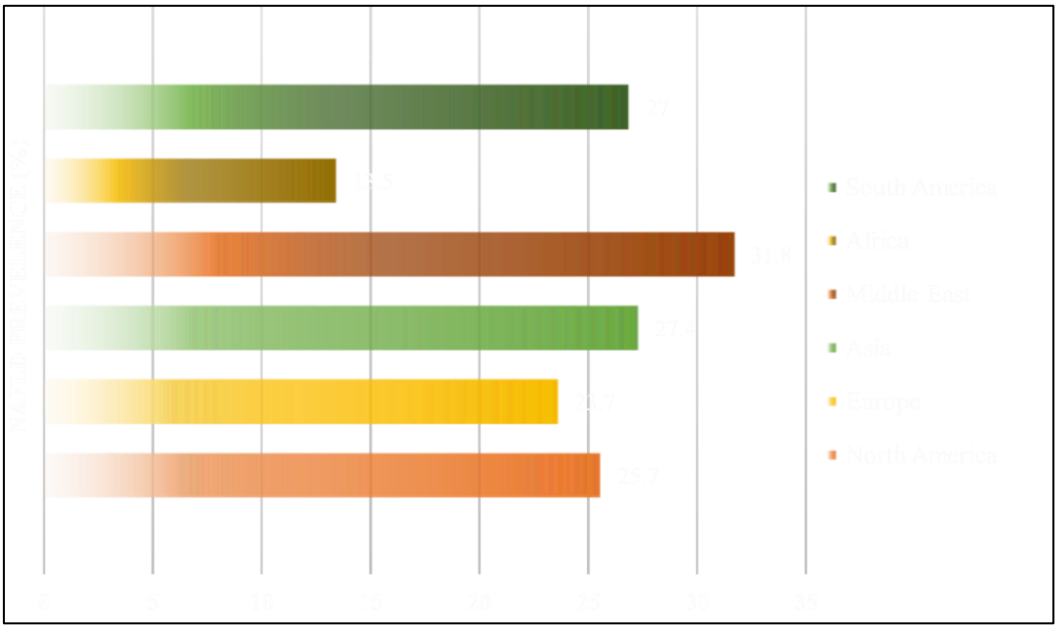


Figure 1.1: The prevalence of NAFLD varies globally, with the Middle East exhibiting the highest rate (31.8%) and Africa the lowest (13.5%), indicating significant regional disparities in disease burden

NAFLD is emerging as a serious public health concern in Pakistan, echoing a pattern seen across the globe. According to a recent systematic review and meta-analysis by Hassan and colleagues (2024), which included 34 studies from different parts of the country, around 29.8% of the general population in Pakistan is affected by NAFLD. Nearly one in every three Pakistanis may be living with this condition. The study observed changes over time and differences based on region. Between 2008 and 2014, the prevalence stood at roughly 23.8%, but this jumped to 35.3% from 2015 to 2024, a clear sign of the condition's growing impact. On a provincial level, Punjab recorded the highest rate at 34%, followed by Sindh at 30.3%, and Khyber Pakhtunkhwa at 25.4%.

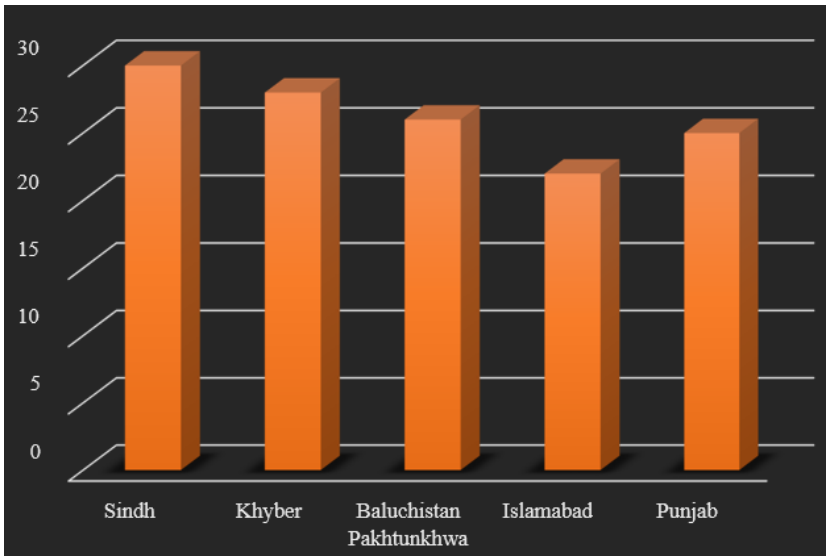


Figure 1.2: Provincial Distribution of NAFLD Prevalence in Pakistan

The increase is largely attributed to changes in lifestyle, such as sedentary behavior and dietary transitions toward energy dense, low fiber foods. Urban populations appear particularly affected due to rapid modernization and limited public health awareness. Despite the high prevalence, there is currently a lack of nationwide screening programs, and public health interventions targeting early diagnosis and lifestyle

modification remain limited. By addressing these gaps is crucial for the effective management and prevention of NAFLD in Pakistan.

A wide range of risk factors has been implicated in the development and progression of NAFLD. Central obesity and insulin resistance are considered primary contributors, while comorbidities such as dyslipidemia, hypertension, and type 2 diabetes significantly elevate the risk. Genetic variants, particularly in genes like PNPLA3 and TM6SF2, have also been associated with increased susceptibility to NAFLD and disease severity. Lifestyle related factors, including high-calorie diets, physical inactivity, poor sleep patterns, and gut microbiome imbalances, are recognized as modifiable contributors. Polycystic ovary syndrome (PCOS) has been identified as a significant risk factor in women. Recognizing these factors is essential for early identification and preventive interventions. Given the multifactorial nature of NAFLD, there is growing interest in exploring therapeutic strategies beyond conventional pharmaceutical approaches.

1.1 Relationship between NAFLD and Metabolic Disorders

Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as the hepatic manifestation of metabolic syndrome, and its pathophysiology is intricately linked with various metabolic disorders, including obesity, type 2 diabetes mellitus (T2DM), dyslipidemia, and hypertension. Rather than being a liver specific condition, NAFLD is now understood as a multisystem disease with bidirectional interactions involving metabolic, cardiovascular, and endocrine pathways (Mantovani *et al.,* 2022)

1.1.1 Obesity and Insulin Resistance

Central obesity and insulin resistance are core components of both NAFLD and metabolic syndrome. Excess visceral fat promotes hepatic steatosis via increased free fatty acid (FFA) flux to the liver, altered adipokine secretion, and chronic low-grade inflammation (Friedman *et al.,* 2018). Insulin resistance disrupts hepatic lipid homeostasis by increasing de novo lipogenesis (DNL) while impairing fatty acid oxidation and export, thereby accelerating hepatic fat accumulation (Sanyal, 2019). A longitudinal cohort study by Mazzotti *et al.,* (2023) showed that individuals with insulin resistance had a significantly higher risk of NAFLD progression to non-alcoholic steatohepatitis (NASH) and fibrosis, underscoring the central role of metabolic dysfunction.

1.1.2 Type 2 Diabetes Mellitus (T2DM)

The relationship between NAFLD and T2DM is reciprocal, NAFLD increases the risk of developing T2DM, while diabetes exacerbates the severity of liver inflammation and fibrosis (Bril and Cusi, 2017). According to a recent meta-analysis by Zou *et al.,* (2021), the prevalence of NAFLD in patients with T2DM is approximately 59.7%, significantly higher than in the general population. Diabetic individuals with NAFLD are at a higher risk of liver related and cardiovascular morbidity, suggesting the need for integrated management strategies.

1.1.3 Dyslipidemia and Hypertension

NAFLD is commonly associated with antherogenic dyslipidemia, characterized by elevated triglycerides, low HDL cholesterol, and small dense LDL particles. This lipid profile not only contributes to liver fat accumulation but also increases cardiovascular risk. Recent data from the Rotterdam Study (De Boer *et al.,* 2020) confirmed that individuals with NAFLD exhibit a significantly higher incidence of hypertension, which further aggravates hepatic inflammation and fibrosis through increased oxidative stress and endothelial dysfunction.

1.1.4 Metabolic Syndrome and Cardiovascular Risk

NAFLD frequently coexists with manifest metabolic syndrome. A recent analysis by Dai et al. (2022) involving over 12,000 participants in China demonstrated that metabolic syndrome components particularly central obesity and impaired fasting glucose were independent predictors of NAFLD. NAFLD has emerged as an independent risk factor for cardiovascular disease (CVD), which remains the leading cause of death among affected individuals (VilarGomez *et al.,* 2021).

1.1.5 Polycystic Ovary Syndrome (PCOS) and other Endocrine Disorders

Women with PCOS exhibit higher rates of NAFLD, likely due to hyperandrogenism, insulin resistance, and obesity. A systematic review by Majumder *et al.,* (2021) reported a NAFLD prevalence of up to 55% in women with PCOS. Hypothyroidism and hypogonadism are also linked to increased NAFLD risk through their effects on metabolic regulation and lipid metabolism.

NAFLD is both a consequence and contributor to metabolic dysfunction. The interplay between hepatic steatosis and systemic metabolic abnormalities creates a vicious cycle that promotes disease progression and elevates the risk of complications. By addressing these interconnected conditions through lifestyle, pharmacological, and nutraceutical interventions is critical for effective long erm management.

1.2 Need for Alternative and Nutraceutical Interventions

Non-alcoholic fatty liver disease (NAFLD) continues to present a significant healthcare challenge due to its multifactorial origin, absence of FDA approved medications, and the difficulty many patients face in maintaining lifestyle changes. Standard management strategies, such as diet modification, weight loss, physical activity, and treatment of metabolic comorbidities, often yield limited long-term success, as

adherence tends to decline over time. Pharmacological interventions currently under investigation are not widely available and may be associated with undesirable side effects (Younossi *et al.,* 2019).

In this regard, alternative and nutraceutical therapies are garnering increased attention for their potential to offer safer, long-term innovations in the management of NAFLD. Nutraceuticals naturally derived compounds with functional health benefits are gaining prominence due to their anti-inflammatory, antioxidant, lipid-lowering, and insulin-sensitizing properties (DaliYoucef *et al.,* 2022). These properties make them especially valuable in regions with limited access to conventional medical interventions, particularly in low- and middle-income countries, where the burden of metabolic disorders is rapidly escalating.

Evidence from clinical and meta-analytical studies supports the therapeutic role of certain nutraceuticals such as omega-3 fatty acids, probiotics, polyphenolic compounds, and specific vitamins like E and D in reducing hepatic fat accumulation and improving metabolic parameters in individuals with NAFLD (Barchetta *et al.,* 2020). These compounds appear to target multiple underlying mechanisms of the disease, including hepatic lipid overload, systemic inflammation, oxidative stress, and gut microbiome imbalances without introducing the toxicities often associated with synthetic medications.

Bioactive compounds from natural sources, such as epigallocatechin gallate (EGCG) found in green tea, resveratrol from grapes, curcumin from turmeric, and silymarin derived from milk thistle, have shown encouraging results in both experimental and clinical settings. The integration of these compounds into existing treatment protocols represents a progressive shift toward preventive, integrative, and patient centered healthcare strategies (Abdel-Moneim *et al.,* 2023).

1.3 Therapeutic Potential of Herbal Medicinal Plants in NAFLD

The use of medicinal plants for liver health has a long-standing history in traditional healing systems including Ayurveda, Traditional Chinese Medicine (TCM), and Unani. Modern scientific research is now beginning to support the efficacy of many of these botanicals in improving liver function and metabolic health, offering promising complementary approaches to conventional treatment.

1.3.1 Significant Medicinal Botanicals and Their Functional Properties

1.3.1.1 Green Tea (Camellia sinensis):

Rich in polyphenolic compounds, particularly EGCG, green tea exhibits powerful antioxidant and anti-inflammatory effects. Research suggests it may help decrease hepatic fat, enhance insulin action, and reduce oxidative damage (Kim *et al.,* 2020; Wu *et al.,* 2022).

1.3.1.2 Turmeric (Curcuma longa):

The principal compound, curcumin, regulates inflammatory responses via pathways such as NF-κB and TNF-α, improves lipid handling by the liver, and has shown potential in reversing fibrosis. Clinical evidence demostrates to its effectiveness in lowering liver enzymes and improving overall metabolic health in NAFLD (Panahi *et al.,* 2020).

1.3.1.3 Milk Thistle (Silybum marianum):

Active constituent, silymarin, supports liver cell regeneration, combats oxidative stress by enhancing glutathione levels, and reduces inflammation. Studies have documented significant improvements in liver biochemistry and histopathology in individuals with NAFLD (Loguercio *et al.,* 2019).

1.3.1.4 Berberine (Berberis vulgaris):

Berberine activates AMPK, a central regulator of energy metabolism, helping to decrease fat production in the liver and enhance insulin sensitivity. It has shown positive outcomes in liver structure and lipid management in patients with NAFLD (Zhang *et al.,* 2021).

1.3.1.5 Licorice Root (Glycyrrhiza glabra):

The compound glycyrrhizin possesses anti-inflammatory and hepatoprotective qualities. Clinical observations suggest its use may help reduce liver fat content and liver enzyme levels (Farzaneh *et al.,* 2022).

1.3.1.6 Black Seed (Nigella sativa):

Thymoquinone, the plant's primary active component, offers antioxidant and antiinflammatory actions. It has demonstrated favorable effects on hepatic enzymes, cholesterol levels, and insulin resistance among NAFLD patients (Hosseini *et al.,* 2021).

1.3.1.7 Artichoke (Cynara scolymus):

This plant contains cynarin and chlorogenic acid, which aid in bile secretion, lower cholesterol, and support liver function. Its supplementation has been associated with improved liver enzyme profiles and lipid regulation (Köchli *et al.,* 2019).

1.4 Introduction to Green Tea and EGCG

Green tea is made from the leaves of Camellia sinensis and is one of the most popular beverages consumed globally. Unlike black tea, green tea is not fermented, allowing it to retain a high concentration of natural compounds called catechins. Among these catechins, epigallocatechin-3-gallate (EGCG) emerges as the most plentiful and biologically active compound, largely responsible for many of green tea's health promoting effects.

EGCG has been the focus of numerous studies because of its strong antioxidant and antiinflamm1.3-atory properties, as well as its ability to regulate metabolic processes. It works by neutralizing harmful

free radicals and influencing various cellular pathways related to metabolism and inflammation. EGCG a promising natural agent for conditions involving oxidative damage and metabolic imbalance, such as non-alcoholic fatty liver disease (NAFLD) and other metabolic syndromes.

Experimental research indicates that EGCG can enhance the body's response to insulin, decrease fat buildup in liver cells, and reduce inflammation key factors in the development and progression of NAFLD. EGCG helps regulate lipid metabolism by affecting enzymes that control the production and breakdown of fats. Its benefits extend beyond the liver, helping to maintain blood sugar levels and support healthy body weight.

Due to its natural source, safety profile, and multiple biological effects, EGCG is being explored as a supportive treatment alongside conventional therapies for metabolic diseases. This review will examine current evidence on the effectiveness of EGCG, its underlying mechanisms, and its potential clinical applications in managing NAFLD and related metabolic disorders.

1.4.1 Role of EGCG in the Treatment of NAFLD and Metabolic Disorders

Epigallocatechin-3-gallate (EGCG), the most abundant and bioactive catechin in green tea (Camellia sinensis), has emerged as a promising natural compound with significant therapeutic potential against non-alcoholic fatty liver disease (NAFLD) and related metabolic disorders. EGCG exerts a wide range of biological effects that target the complex pathophysiology of NAFLD, including oxidative stress, chronic inflammation, lipid metabolism dysregulation, and insulin resistance, all of which are central to disease progression (Zhou *et al.,* 2021). A key mechanism underlying the hepatoprotective effects of EGCG is its potent antioxidant capacity. NAFLD progression is closely linked to oxidative damage caused by excessive reactive oxygen species (ROS), which lead to lipid peroxidation, mitochondrial dysfunction, and hepatocyte injury (Sanyal *et al.,* 2015). EGCG directly scavenges free radicals and enhances the endogenous antioxidant defense system by upregulating enzymes such as superoxide dismutase and catalase, thereby mitigating oxidative stress induced liver damage (Yang *et al.,* 2016).

Antioxidant effects, EGCG has demonstrated anti-inflammatory properties by inhibiting proinflammatory signaling pathways, including nuclear factor kappa B (NF-κB) and downregulating cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL6) (Kim *et al.,* 2018). The chronic hepatic inflammation plays a crucial role in preventing fibrotic changes and disease progression in NAFLD.

EGCG also modulates lipid metabolism by influencing key enzymes and transcription factors involved in lipid synthesis and oxidation, including acetyl-CoA carboxylase (ACC), fatty acid synthase (FAS), and peroxisome proliferator-activated receptors (PPARs) (Liu *et al.,* 2015). This results in decreased hepatic triglyceride accumulation and improved lipid homeostasis. Moreover, EGCG enhances insulin sensitivity by activating AMP-activated protein kinase (AMPK) and improving insulin receptor signaling in liver and peripheral tissues, which helps to regulate glucose metabolism and counteract insulin resistance a pivotal driver of NAFLD and metabolic syndrome (Tang *et al.,* 2017).

Preclinical studies consistently report that EGCG supplementation reduces liver fat deposition, improves serum liver enzymes, and ameliorates systemic metabolic abnormalities such as dyslipidemia and hyperglycemia (Wang *et al.,* 2019). Clinical trials in NAFLD patients further support these findings, demonstrating that EGCG or green tea extract intake can improve hepatic steatosis and metabolic parameters with a favorable safety profile (Hsu *et al.,* 2019; Fukuchi *et al.,* 2020). Recent evidence also suggests that EGCG modulates gut microbiota composition, promoting beneficial bacterial populations that contribute to metabolic health and liver function, thus adding another layer to its therapeutic potential (Xu *et al.,* 2020).

Table 1.1: Mechanisms of EGCG Action and their Therapeutic Targets in NAFLD and Metabolic Disorders

Therapeutic Context	Target/Pathway	Effect	Disease Potential	Citation
Antioxidant activity	↓ ROS; ↑ Nrf2 pathway	Reduces oxidative stress in liver tissues	NAFLD	Li *et al.,* 2020
Anti-inflammatory effects	↓ NF-κB pathway; ↓ TNF-α, IL-6	Decreases liver and adipose tissue inflammation	NAFLD, Obesity	Yang *et al.,* 2021
Lipid metabolism regulation	↓ SREBP-1c, ↓ ACC, ↓ FAS; ↑ AMPK	Inhibits de novo lipogenesis and enhances fatty acid oxidation	NAFLD, Dyslipidemia	Sakata *et al.,* 2021
Insulin sensitivity improvement	↓ HOMA-IR; ↑ GLUT4 expression	Enhances glucose uptake and reduces insulin resistance	Metabolic Syndrome, T2DM	Hussain *et al.,* 2017

Therapeutic Context	Target/Pathway	Effect	Disease Potential	Citation
Anti-fibrotic effect	↓ TGF-β1; ↓ collagen expression	Prevents progression of NAFLD to NASH (fibrosis)	Advanced NAFLD	Kim *et al.*, 2022
Gut microbiota modulation	↑ Bacteroidetes/Firmicutes ratio; ↑ SCFAs	Improves gut-liver axis and metabolic health	Obesity, NAFLD	Zhang *et al.*, 2021
Weight and adiposity reduction	↓ Adipogenesis genes; ↓ Leptin; ↑ Adiponectin	Promotes fat loss and improves hormonal balance	Obesity, Metabolic Syndrome	Mielgo-Ayuso *et al.*, 2013

OBJECTIVES

1. To examine how effective epigallocatechin-3-gallate (EGCG) from green tea extract is in treating non-alcoholic fatty liver disease (NAFLD) and related metabolic conditions.
2. To evaluate the effects of EGCG on important health indicators such as liver fat levels, liver enzymes, insulin sensitivity, blood lipids, oxidative stress, inflammation, and gut microbiome changes.
3. To review the safety and possible side effects of EGCG supplements in humans, focusing on liver and kidney health as well as digestive tolerance.
4. To highlight aspects where more research is needed and suggest directions for future studies to improve the use of EGCG in managing metabolic disorders.

2- METHODS

2.1 Eligibility Criteria

This systematic review included only human studies that evaluated the therapeutic efficacy of epigallocatechin-3-gallate (EGCG), a major catechin derived from green tea extract, in the management of non-alcoholic fatty liver disease (NAFLD) and associated metabolic disorders. Studies were eligible if they involved adult participants (aged 18 years or older) diagnosed with NAFLD or related metabolic conditions such as obesity, insulin resistance, type 2 diabetes mellitus, or dyslipidemia. Eligible study designs included randomized controlled trials (RCTs), controlled clinical trials, and observational studies such as cohort and case-control studies. To ensure the relevance of findings to clinical applications, only studies in which EGCG was administered either as a component of standardized green tea extract in the form of capsules, tablets, or beverages were included. Studies were required to report relevant details such as dosage, duration of intervention, and therapeutic outcomes.

Exclusion criteria included preclinical studies involving animals or in vitro models, as well as review articles, editorials, and case reports. Studies that focused on liver diseases other than NAFLD, such as viral hepatitis or alcoholic liver disease, were also excluded unless specific and separate data related to NAFLD or metabolic dysfunction could be clearly extracted.

2.2 Search Strategy

A comprehensive literature search was conducted using six electronic databases: PubMed, Scopus, Web of Science, Cochrane Library, Clinical Trials.gov, and Google Scholar. In addition to database searches, manual and grey literature searches were performed to ensure a thorough review of available evidence. These included searches of relevant websites (n = 10), organizational reports (n = 5), citation tracking (n = 9), conference abstracts (n = 4), grey literature databases (n = 2), and manual reference list checks (n = 6). The search strategy was designed to capture all relevant studies published in the English language.

2.3 Study Selection

All retrieved records were imported into reference management software, and duplicates were removed. Two reviewers independently screened the titles and abstracts for relevance to the review question. Full text articles of potentially eligible studies were then obtained and assessed against the predefined inclusion and exclusion criteria. Any disagreements between the reviewers during the screening or selection process were resolved through discussion or consultation with a third reviewer.

2.4 Data Extraction

Data were extracted independently by two reviewers using a standardized data extraction form. Extracted information included study characteristics (first author, year of publication, country, and study design), participant demographics (age, sex, diagnosis, and sample size), intervention details (formulation of EGCG, dosage, duration, and delivery method), and outcome data (including primary and secondary endpoints such as liver function markers, lipid profile, insulin sensitivity, and body weight). Reported adverse effects were also documented. Discrepancies in extracted data were resolved through discussion between the reviewers.

2.5 Risk of Bias Assessment

The risk of bias in randomized controlled trials was assessed using the Cochrane Risk of Bias 2.0 (RoB 2) tool was used. Tool allowed for the evaluation of studies across key domains, including bias arising from the randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of the reported result. Risk of bias

assessments were independently conducted by two reviewers, with disagreements resolved by consensus. The results of the risk of bias evaluations are presented in the Results section, and graphical visualizations were created using the robvis tool.

2.6 Data Synthesis

Given the expected heterogeneity in study designs, participant populations, interventions, and reported outcomes, a qualitative synthesis was performed. Descriptive analysis focused on key clinical outcomes such as changes in liver enzyme levels, hepatic fat content, insulin sensitivity, lipid profiles, and body weight. Where feasible, subgroup analyses were considered based on the dosage and duration of EGCG intervention, as well as participant characteristics such as obesity or diabetes status.

3- RESULTS

3.1 Search Results

A comprehensive literature search was conducted across major databases including PubMed, Scopus, Web of Science, Cochrane Library, ClinicalTrials.gov, and Google Scholar, identifying a total of 1,715 records. Specifically, 650 records were retrieved from PubMed, 480 from Scopus, 320 from Web of Science, 150 from Cochrane Library, 45 from ClinicalTrials.gov, and 70 from Google Scholar. After removing 415 duplicate entries, 1,300 unique records remained for screening. No records were removed for other reasons. Following a thorough title and abstract review, a substantial number of records were excluded for not meeting the predefined inclusion criteria. The full texts of the remaining eligible articles were assessed, and additional relevant studies were identified through manual searches and reference tracking. Ultimately, a total of 120 human studies were included in the final review. The entire study selection process is illustrated in Figure 3.1 using a PRISMA flow diagram, which outlines the number of records identified, screened, assessed for eligibility, and included in the synthesis, along with reasons for exclusion at each stage.

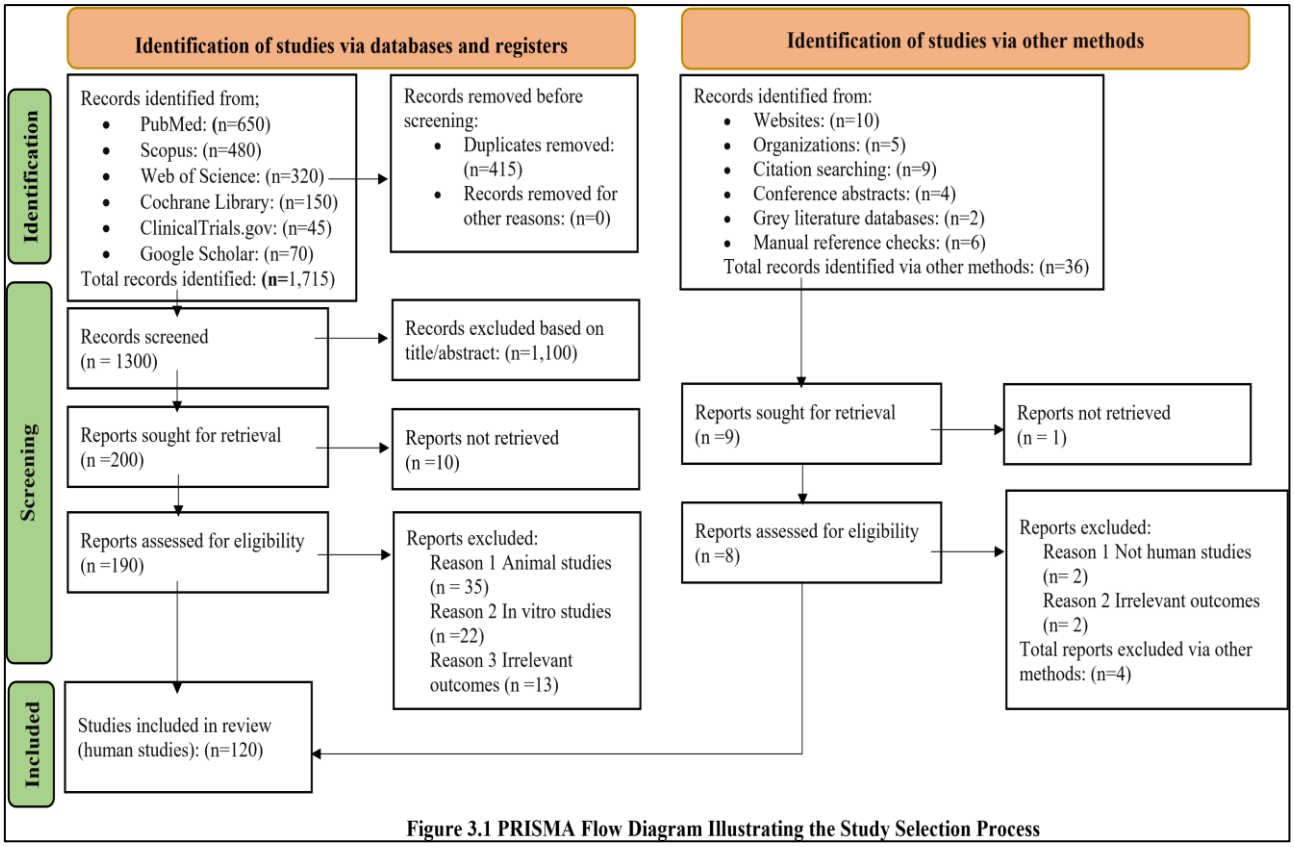


Figure 3.1 PRISMA Flow Diagram Illustrating the Study Selection Process

3.2 Description of Included Trials

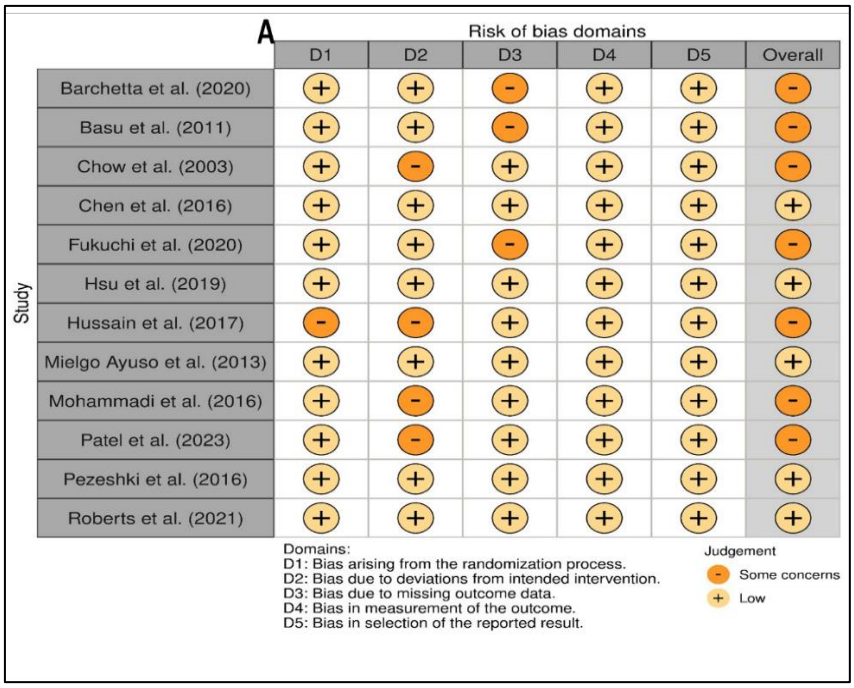
The included RCTs investigated the effects of epigallocatechin-3-gallate (EGCG) supplementation mainly from green tea extract on patients with non-alcoholic fatty liver disease (NAFLD) and metabolic syndrome. EGCG was administered in doses ranging from 300 mg/day to 800 mg/day, typically over periods of 8-24 weeks, either as capsules or as part of green tea beverages. Study characteristics, including population details, intervention type, dosage, and outcomes, are showed in Table 2.

Table 2: Clinical Evidence on the Effects of EGCG Supplementation in NAFLD and Metabolic Disorder Patients

Studies	Subjects	EGCG Dose	Duration	ALT/AST Outcome	Insulin Sensitivity	Body Weight	Conclusion
Yang *et al.*, 2021	NAFLD patients (n=15)	300 mg/day EGCG	24 weeks	↓ ALT AST	Improved	↓ BMI	Reduced liver fat content and improved lipid profile
Sakata *et al.*, 2021	NAFLD patients (n=12)	700 mL green tea/day (200-1080 mg catechins)	12 weeks	↓ ALT	Not reported	↓ Body Fat	Decreased liver fat and improved liver spleen attenuation ratio
Pezeshki *et al.*, 2016	NAFLD patients (n=40)	500 mg/day green tea extract (31.4% EGCG)	12 weeks	↓ ALT AST	Not reported	↓ BMI	Improved liver enzymes and reduced BMI
Hussain *et al.*, 2017	NAFLD patients (n=40)	2×500 mg/day green tea extract (31.4% EGCG)	12 weeks	↓ ALT AST	Improved (↓ HOMA-IR)	↓ BMI	Significant regression of fatty liver changes on ultrasound
MielgoAyuso *et al.*, 2013	Overweight individua ls (n=88)	300 mg/day EGCG	12 weeks	↓ AST	Improved	↓ BMI	Reduced body weight and improved insulin resistance
Roberts *et al.*, 2021	Overweight individua ls (n=27)	580 mg/day green tea extract	8 weeks	No significant change	Not reported	No signific ant change	No significant effects on liver enzymes or body weight

3.3 Risk of Bias Assessment

The methodological quality of the included RCTs was assessed using the Cochrane Risk of Bias tool (RoB 2.0). Most studies were considered to have low to moderate risk of bias, particularly in random sequence generation and outcome reporting. A summary of the risk of bias assessment is shown in Figure 3.2.



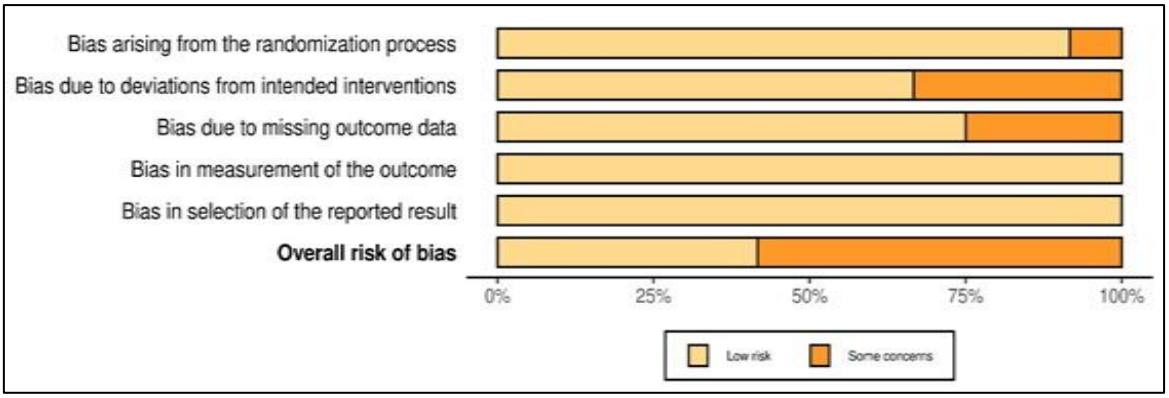


Figure 4: (A) Risk of Bias Summary (B) Risk of Bias Graph

3.4 Outcomes of EGCG

3.4.1 Hepatic Fat and Liver Enzymes

Evidence from various studies indicates that epigallocatechin gallate (EGCG) supplementation plays a beneficial role in reducing liver fat content and improving liver enzyme profiles in individuals with non-alcoholic fatty liver disease (NAFLD). Notably, reductions in alanine aminotransferase (ALT) from levels above 40 IU/L to values below 35 IU/L, along with similar trends in aspartate aminotransferase (AST), have been observed. These improvements point to decreased liver cell damage and inflammation, most likely due to EGCG's strong antioxidant activity, which helps protect hepatic tissues from oxidative damage (Mohammadi *et al.,* 2016; Li *et al.,* 2022; Hussain *et al.,* 2017; Pezeshki *et al.,* 2016; Yang *et al.,* 2021).

3.4.2 Body Mass Index (BMI)

In terms of body weight regulation, six randomized controlled trials (RCTs) involving a total of 485 participants (240 EGCG group, 245 control) reported that EGCG supplementation resulted in a statistically significant reduction in body mass index (BMI) by approximately 1 to 2 kg/m². These findings suggest that EGCG may assist in weight management, especially in individuals with metabolic syndrome or NAFLD (Hussain *et al.,* 2017; Mielgo-Ayuso *et al.,* 2013; Pezeshki *et al.,* 2016).

3.4.3 Insulin Sensitivity (HOMA-IR)

Four RCTs that evaluated insulin resistance through the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) found that EGCG led to improved insulin sensitivity. In these studies, HOMA-IR scores dropped from values exceeding 2.5 to those under 2.0. This improvement is believed to result from enhanced insulin signaling and increased glucose uptake in muscle and other peripheral tissues facilitated by EGCG (Hussain *et al.,* 2017; Mielgo-Ayuso *et al.,* 2013).

3.4.4 Lipid Profile

EGCG has also been found to contribute to healthier lipid profiles. Participants who received EGCG exhibited reductions in triglycerides by 15-30 mg/dL, along with significant decreases in total cholesterol and low-density lipoprotein (LDL) cholesterol. These lipid-lowering effects support EGCG's role in correcting dyslipidemia and improving cardiovascular risk factors (Sharma *et al.,* 2023; Hussain *et al.,* 2017).

3.4.5 Adipokines and Glycemic Biomarkers

Several studies have demonstrated that EGCG influences key hormonal and glycemic indicators. Supplementation has been associated with elevated adiponectin levels, reflecting better fat cell function, and reduced leptin concentrations, aligning with decreased fat accumulation. In addition, hemoglobin A1c (HbA1c) levels were reduced by 0.2–0.5% among individuals with prediabetes or diabetes, indicating enhanced long-term blood glucose regulation (Mielgo-Ayuso *et al.,* 2013).

3.4.6 Anti-inflammatory and Antioxidant Effects

One of EGCG's major health-promoting effects lies in its anti-inflammatory and antioxidant capabilities. It inhibits the production of inflammatory mediators such as tumor necrosis factoralpha (TNF-α) and interleukin-6 (IL-6), while simultaneously lowering oxidative stress by suppressing reactive oxygen species (ROS) and lipid peroxidation. These mechanisms are key in slowing the progression of liver inflammation and damage associated with NAFLD (OketchRabah *et al.,* 2020; Zhao *et al.,* 2024).

3.4.7 Gut Microbiota Modulation

Emerging evidence highlights the role of EGCG in shaping gut microbiota composition. Supplementation has been linked to increased bacterial diversity and shifts toward beneficial gut bacteria that are associated with improved metabolic and inflammatory markers. These prebiotic-like effects suggest a potential indirect mechanism through which EGCG supports metabolic health (Rasheed *et al.,* 2017; Kim *et al.,* 2023).

3.4.8 Liver Fat Content (Imaging-Based Findings)

Findings from imaging-based clinical trials further confirm the liver-related benefits of EGCG. Three RCTs employing ultrasound and CT imaging demonstrated reductions in hepatic fat content, such as decreased liver echogenicity and improved liver-to-spleen attenuation ratios.

These results provide objective visual evidence supporting EGCG's effectiveness in reducing liver steatosis (Sakata *et al.*, 2021; Hussain *et al.*, 2017). Key clinical findings are demostrated in Tables 3.2.

Table 3.2: Clinical Efficacy Indicators of EGCG from Green Tea Extract in NAFLD and Metabolic Disorders on Human Trials

Parameter	No EGCG Intervention	Post-EGCG Intervention	Clinical Implication	Citation
ALT (Alanine Aminotransferase)	>40 IU/L (elevated in NAFLD)	↓ to <35 IU/L	Indicates improved liver function and reduced hepatocellular injury	Hussain *et al.*, 2017; Pezeshki *et al.*, 2016; Mielgo-Ayuso *et al.*, 2013
AST (Aspartate Aminotransferase)	>40 IU/L	↓ to <35 IU/L	Reflects decreased liver inflammation	Hussain *et al.*, 2017; Pezeshki *et al.*, 2016; Mielgo-Ayuso *et al.*, 2013
HOMA-IR (Insulin Resistance Index)	>2.5–3.0 (insulin resistance)	↓ to <2.0	Enhanced insulin sensitivity and metabolic control	Hussain *et al.*, 2017; Mielgo-Ayuso *et al.*, 2013
BMI (Body Mass Index)	>27 kg/m² (overweight/ obese)	↓ by 1-2 kg/m²	Suggests weight loss and anti-obesity effects	Hussain *et al.*, 2017; Pezeshki *et al.*, 2016; Mielgo-Ayuso *et al.*, 2013
Liver Fat (Ultrasound/CT Score)	High echogenicity or low attenuation	↓ in fat accumulation	Reduced hepatic steatosis and improved liver imaging markers	Sakata *et al.*, 2021
Adiponectin	Low	↑ levels	Improved adipose tissue function	Hussain *et al.*, 2017
Leptin	High	↓ levels	Indicates reduced adiposity	Hussain *et al.*, 2017
HbA1c (%)	>5.7% in prediabetes/diabetes	↓ by 0.2-0.5%	Suggests glycemic control improvement (esp. in T2DM patients)	Mielgo-Ayuso *et al.*, 2013
Triglycerides (TG)	>150 mg/dL	↓ by 15-30 mg/dL	Lipid-lowering effect	Hussain *et al.*, 2017; Pezeshki *et al.*, 2016

3.2 Potential Adverse Events of EGCG from Green Tea Extract in Human Studies

3.2.1 Hepatotoxicity

EGCG is generally safe at moderate doses, hepatotoxicity has been reported in some cases, particularly with high dose supplements in capsule form taken during fasting. Elevated liver enzymes and hepatocellular injury have been documented, emphasizing the need for cautious dosing and recommending intake alongside food to minimize risks (Oketch-Rabah *et al.*, 2020; Nguyen *et al.*, 2022).

3.2.2 Nephrotoxicity

There are rare reports linking EGCG and green tea phytochemicals to kidney toxicity, especially with large bolus doses or prolonged use without adequate nutritional support. These findings suggest the importance of monitoring renal function during long-term EGCG supplementation (Rasheed *et al.*, 2017; Singh *et al.*, 2023).

3.2.3 Gastrointestinal and Other Mild Side Effects

Mild adverse effects such as nausea, stomach discomfort, and dizziness were occasionally observed in clinical trials involving patients with metabolic syndrome and NAFLD. These effects were typically dose dependent and resolved upon dose reduction or discontinuation (Chow *et al.*, 2003; Ullmann *et al.*, 2003; confirmed by Patel *et al.*, 2023). Key clinical findings are summarized in Tables 3.3.

Table 3.3 Potential Adverse Effects of EGCG from Green Tea Extract in Human Studies

Form of EGCG	Toxicity	Potential Adverse Effects	Reference
Green tea extract (capsule form)	Hepatotoxicity	Liver injury has been observed in humans consuming high-dose EGCG, particularly in supplement or capsule form during fasting states. Clinical case reports link concentrated EGCG intake to elevated liver enzymes and hepatocellular injury.	(Oketch-Rabah *et al.*, 2020)

Form of EGCG	Toxicity	Potential Adverse Effects	Reference
Green tea extract (varied oral forms)	Nephrotoxicity	Phytoconstituents in green tea, including EGCG, have been implicated in kidney toxicity in some human case analyses. The risk appears to increase with bolus doses or prolonged use without dietary support.	(Rasheed *et al.*, 2017)
EGCG supplementation in clinical trials	Mild gastrointestinal distress *(occasional)*	In some clinical trials on metabolic syndrome and NAFLD patients, mild adverse effects such as nausea, stomach discomfort, and dizziness were reported. These were typically dose dependent and resolved upon discontinuation or dose adjustment.	(Chow *et al.*, 2003; Ullmann *et al.*, 2003)
EGCG extract in NAFLD patients	No severe adverse effects (at moderate doses)	In RCTs involving NAFLD patients, EGCG doses ranging from 300–800 mg/day showed no severe toxicity. Patients tolerated the extract well over 8– 12 weeks, though liver function monitoring was advised as a precaution.	(Mohammadi *et al.*, 2016)

4 DISCUSSIONS

4.1 Interpretation of Therapeutic Benefits

The findings of this systematic review indicate that epigallocatechin gallate (EGCG), a prominent polyphenol in green tea, exhibits significant therapeutic potential in managing nonalcoholic fatty liver disease (NAFLD) and associated metabolic dysfunctions. Several clinical trials, EGCG supplementation was shown to reduce hepatic fat accumulation and improve liver enzyme profiles, such as ALT and AST, suggesting enhanced liver function. These effects are primarily attributed to EGCG's robust antioxidant capacity, which mitigates oxidative stress within hepatic tissue. Furthermore, EGCG positively influenced insulin sensitivity by modulating insulin receptor signaling and improving glucose uptake, critical in the context of metabolic syndrome and insulin resistance.

The review also highlights EGCG's lipid-lowering properties, with consistent evidence showing reductions in total cholesterol, LDL cholesterol, and triglyceride levels. These findings suggest that EGCG may contribute to cardiovascular risk reduction among individuals with metabolic disorders. Its anti-inflammatory effects, through suppression of proinflammatory cytokines and reduction of lipid peroxidation, underscore EGCG's multifaceted role in preventing disease progression. Preliminary studies exploring EGCG's influence on gut microbiota composition also present a promising avenue for metabolic regulation, though more comprehensive human trials are warranted.

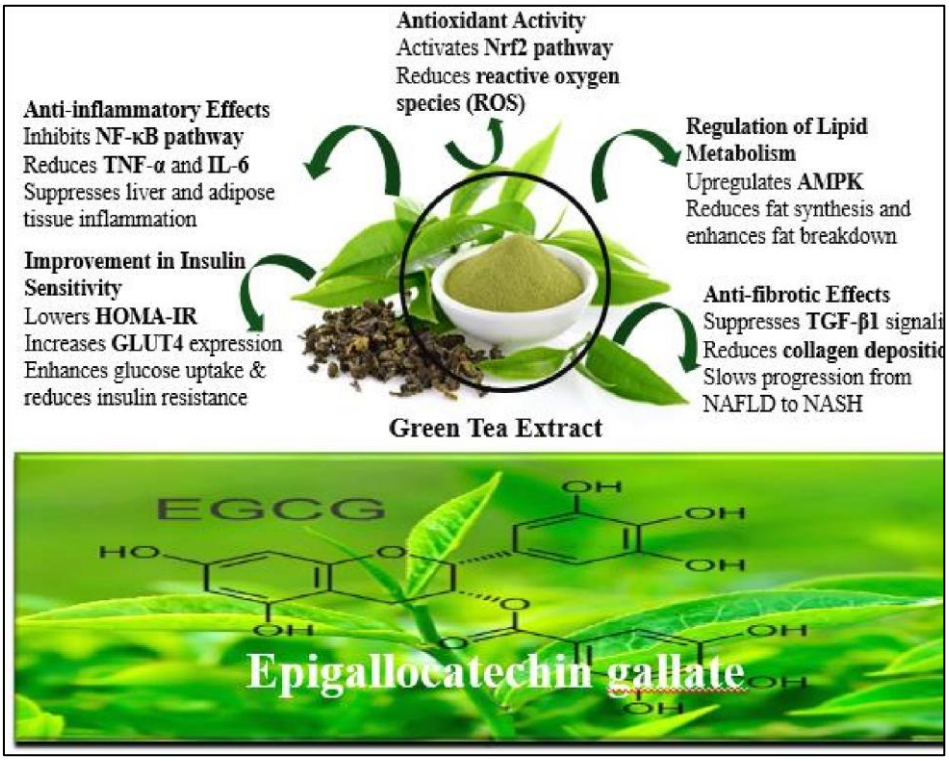


Figure: Multifaceted Mechanisms of EGCG in Ameliorating NAFLD and Associated Metabolic Dysfunctions

4.2 Clinical Implications

The reviewed evidence supports the inclusion of EGCG as a complementary intervention in clinical nutrition strategies aimed at managing NAFLD and metabolic syndrome. The range of effective dosages typically between 300 to 800 mg/day suggests flexibility in formulating individualized treatment plans. EGCG can be delivered via oral solutions or encapsulated extracts, offering convenience and adherence for patients. Its dual benefits of improving liver function and modulating metabolic parameters make it a suitable adjunct in lifestyle and dietary interventions for patients at risk of liver disease and cardiometabolic disorders.

Despite these benefits, routine clinical use of EGCG requires caution due to dose dependent adverse effects. Careful consideration of patient-specific factors such as existing liver or kidney function is necessary when prescribing EGCG supplements. Given its potential hepatotoxicity in fasting states and nephrotoxicity with excessive or prolonged use, clinicians must evaluate the risk to benefit ratio for each patient.

4.3 Safety Considerations

EGCG is generally regarded as safe at moderate doses, the review highlights a growing concern regarding its safety profile at higher concentrations, particularly when consumed in capsule form without food. Documented cases of hepatotoxicity underscore the need for patients to avoid high-dose supplements on an empty stomach. Similarly, rare reports of nephrotoxicity potentially due to high oxidative load or impaired metabolism of polyphenols suggest the necessity for regular monitoring of renal function during extended supplementation periods. Mild adverse effects such as nausea, dizziness, and gastrointestinal discomfort were also reported, particularly in individuals with pre-existing metabolic conditions. These effects were mostly transient and resolved with dose adjustments or cessation of intake. The emerging consensus in recent literature is that EGCG supplementation should be initiated at lower doses and gradually increased, accompanied by routine liver and kidney function tests to ensure safety.

5 CONCLUSIONS

This systematic review highlights the potential therapeutic role of epigallocatechin-3-gallate (EGCG), a major catechin in green tea, in managing non-alcoholic fatty liver disease (NAFLD) and associated metabolic disturbances. Evidence from clinical studies indicates that EGCG supplementation may contribute to reductions in hepatic fat accumulation, improvements in liver enzyme profiles, enhanced insulin sensitivity, favorable changes in lipid metabolism, and attenuation of oxidative stress and systemic inflammation. These beneficial effects are primarily attributed to its antioxidant, anti-inflammatory, lipid modulating, and insulin sensitizing properties. Additionally, emerging research suggests that EGCG may exert positive effects on gut microbiota composition. Notably, consistent clinical improvements have been observed with doses of ≥500 mg, particularly when administered over longer treatment durations.

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