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Review

Curcumin as a multi-hepatoprotective agent-mechanism, clinical and preclinical studies-A narrative review

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ABSTRACT:

Introduction: Non-alcoholic fatty liver disease, alcoholic liver disease, viral hepatitis and cirrhosis are the major liver diseases. Oxidative stress, chronic inflammation and dyslipidemias are the primary pathophysiological mechanisms leading to damage to hepatic cells and the progression of liver diseases. Curcumin, a polyphenol derived from *Curcuma longa* (turmeric), is one of the most analyzed bioactive compounds for its antioxidant, anti-inflammatory and hepatoprotective effects. In this narrative review, we summarize the molecular mechanisms of curcumin's liver-protective effects and its potential as a treatment for liver disease. **Methods:** Databases such as PubMed, Scopus, and Web of Science were searched for articles published in English over the previous 15 years and containing the keywords "curcumin", "*Curcuma longa*", "hepatoprotection", "liver injury", and "antioxidant". The preclinical and clinical studies were analyzed and data regarding the study design, dose, therapeutic effects, and mechanism of action were extracted and qualitatively discussed. **Results:** Curcumin prevents liver injury by inhibiting the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) signaling pathway and decreasing levels of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin 6 (IL-6). Additionally, curcumin reduces oxidative stress by scavenging free radicals and activating the nuclear factor (erythroid-derived 2)-like 2 (Nrf2) pathway that increases the activity of endogenous antioxidant enzymes. Curcumin regulates lipid metabolism by activating AMPK and PPAR-α and by inhibiting SREBP-1c, which is associated with less hepatic steatosis. In animal models, curcumin consistently improved serum liver function and fibrosis markers. Clinical trials showed positive results with lowering of liver fat, inflammatory markers, and serum transaminases. **Conclusion:** Curcumin is a potential hepatoprotective agent with multi-targeting effects despite its bioavailability issues. further modified preparations and large-scale studies should allow for effective hepatoprotective drugs for chronic liver disease to be developed.

KEYWORDS: Curcumin, fatty liver, liver disease, inflammation, narrative review, oxidative stress.

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1. INTRODUCTION

The liver is an organ with a broad array of complex functions integrated into the maintenance of homeostasis and essential for the health of the system as a whole. [1] The liver is the main metabolic organ of the body and is responsible for the regulation of glucose metabolism, via glycogenesis and gluconeogenesis, and lipid and carbohydrate metabolism. [1-2] The liver is an active synthetic organ, responsible for producing most of the serum proteins (including albumin, which maintains plasma oncotic pressure), and the majority of the blood coagulation factors, hormones, and carrier proteins. [3] The liver also has a detoxification function, filtering about 250 gal (950 l) of blood daily and chemically transforming drugs and toxins into less harmful compounds that can be excreted. In addition, it produces and secretes bile to aid in digestion, and stores vitamins and minerals such as vitamins A, D, E and K, iron and copper. [4] Curcumin is the principal bioactive polyphenolic constituent of turmeric (*Curcuma longa*), a flowering plant belonging to the ginger family (Zingiberaceae). [5-6] Curcumin is a diferuloylmethane with the chemical formula $C_{21}H_{20}O_6$, having two aromatic phenyl rings that are substituted with hydroxyl (-OH) and methoxy (-OCH_3) groups. These rings are connected by a seven-carbon chain containing a conjugated β-diketone system (Figure 1). *In vivo*, curcumin is in a keto-enol tautomeric equilibrium and at physiological pH the enol form is the predominant form. This preference is attributed to resonance delocalization and stabilization through intramolecular hydrogen bonding. [6-7] Turmeric was first reported in 1815 and the crystalline form was described in 1870. The full structure of the molecule was ascertained in 1910. *Curcuma longa* has been used for many conditions like inflammation, jaundice, digestive diseases and others, which are available in ancient customary systems of medicine such as Ayurveda for more than 5000 years. [7-8] Modern pharmacological research has recognized that the powerful antioxidant, anti-inflammatory, antitoxic and anticancer activities of *Curcuma longa* are of interest in the field of chronic disease prevention and management. [5-7] The aim of the present study is to assess the hepatoprotective and the molecular mechanism of curcumin through its anti-oxidative, anti-inflammatory, and lipid-lowering activity in the liver injury in rat models. The study also aims at evaluating curcumin formulations for their effectiveness in preventing the liver dysfunction.

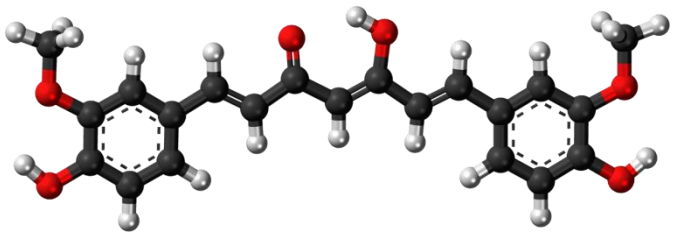

Figure 1. 3D structure of curcumin

2. METHODOLOGY

After searching the PubMed, Scopus, and Web of Science databases for articles related to the keywords "*Curcuma longa*", "curcumin", "hepatoprotection", "liver injury" and "antioxidant" using the Boolean operators AND, OR and NOT, where applicable, we reached the results that are presented in the following information. Studies published in English in the last 15 years were included. Search strategy details including the search terms, filters and number of results for each database are presented in Table 1. Preclinical and clinical studies were included for further screening and analysis.

Selection and screening of studies: In search of relevant literature, 415 records had been obtained from PubMed (n = 140), Scopus (n = 165), and Web of Science (n = 110). After removing duplicates and non-relevant records (n = 65), 350 records were screened based on the title and abstract. Following title and abstract review, 190 articles were removed for not being related to hepatoprotection, curcumin-based intervention, or for lacking detailed mechanistic information. Full-text articles of the remaining 136 publications were then reviewed to determine study eligibility. Most commonly (94

studies) reasons for exclusion were failure to fulfil the inclusion criteria, lack of exact reported experimental or clinical outcomes, and the absence of liver injury models. Ultimately, 42 studies were included in the qualitative synthesis. The PRISMA approach was followed for the literature search and selection process, and this is fully transparent (Figure 2).

Table 1. Search strategy

Database	Search Terms Used	Filters Applied	Results Retrieved	Notes
PubMed (via MEDLINE)	("*Curcuma longa*" OR curcumin) AND ("hepatoprotection" OR "liver injury" OR "hepatic damage") AND ("antioxidant" OR "oxidative stress")	English language; Publication years: last 15 years; Peer-reviewed	140	Title/Abstract
Scopus	("curcumin" AND "liver protection") OR ("*Curcuma longa*" AND "hepatotoxicity")	English; Last 15 years; Peer-reviewed	165	Title/Abstract
Web of Science	("curcumin" OR "*Curcuma longa*") AND ("hepatoprotective effects" OR "liver function")	English; Last 15 years; Peer-reviewed	110	Title/Abstract/Keywords

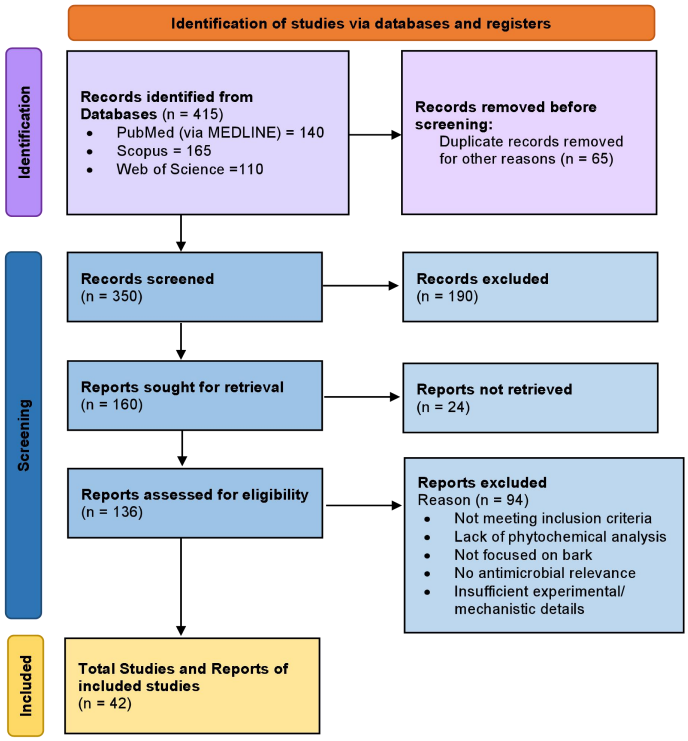

Figure 2. PRISMA flow diagram of the present study

Inclusion criteria: Studies were included if written in English and published within the last 15 years. We selected preclinical (*in vitro* or *in vivo*) and clinical studies on curcumin or *Curcuma longa* that investigated liver injury/hepatoprotection, oxidative stress or antioxidant mechanisms. Only those with biochemical, molecular or histopathological end-points were included. Articles were also considered eligible if they described a mechanistic pathway (modulation of oxidative stress/inflammatory signaling/apoptosis/antioxidant enzyme activity).

Exclusion criteria: Exclusion criteria were non-English literature, conference abstracts/meeting proceedings, editorials and commentaries, reviews and duplicates and studies unrelated to liver injury or hepatoprotection. Exclusion criteria consisted of studies with no curcumin intervention, insufficient experimental details, unclear outcome reporting, and those using other plant extracts or animal models with non-hepatic diseases. Reviews that did not include primary data were excluded from the quantitative synthesis and instead used for context.

Liver disease: Liver disease accounts for nearly two million deaths a year and is one of the most common causes of death. 1 in 25 deaths globally is due to a liver disease. Chronic liver disease affects around 1.5 billion people worldwide. Around 344 million people (24% of adults) have the commonest causes, non-alcoholic fatty liver disease (recently renamed metabolic dysfunction-associated steatotic liver disease), chronic hepatitis B (HBV) infection, hepatitis C (HCV) infection and alcohol-associated liver disease, which can lead to cirrhosis (the 11th

commonest cause of death in the world, accounting for 5.3% of deaths) and hepatocellular carcinoma if untreated. [9-10] the highest incidence rates occur in Asia and Africa, where viral hepatitis remains endemic, and in Western and middle-income countries, where obesity and alcohol consumption are risk factors. [11-12]

Mechanisms of action of curcumin in liver health: Figure 3 describes the role of curcumin's anti-inflammatory effect in liver diseases, which occurs through inhibition of NF-κB signaling, suppression of pro-inflammatory cytokines, and inhibition of MAPK and TLR signaling. This eventually prevents or slows the promotion of fibrosis. Curcumin has antioxidant properties mediated through the scavenging of oxidative stress markers and activation of the Nrf2 signaling pathways, which up-regulates antioxidant enzymes such as SOD and catalase. It also protects liver function by regulating lipid metabolism and liver enzymes and preventing steatosis and cellular damage.

Anti-inflammatory effects: Curcumin is a strong inhibitor of a number of inflammatory signaling pathways implicated in liver damage. Most important is the inhibition of nuclear factor kappa B (NF-κB) activation, which is accomplished by preventing the degradation of its inhibitory subunit, IκBα. NF-κB would otherwise migrate into the nucleus of the cell to activate genes responsible for a pro-inflammatory profile. [13-15] Curcumin down-regulates the expression of cyclooxygenase-2 (Cox-2), tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-8 (IL-8) and other pro-inflammatory factors. [15-16] In models of non-alcoholic fatty liver disease, curcumin inhibits the expression of TLR-2 and TLR-4, as well as the mitogen-activated protein kinase pathway, which antagonizes the effect of the inflammatory cytokines involved in the disease process. [16] Curcumin can interfere with these cytokines and prevent the "second hit" of steatohepatitis, which causes simple steatosis to progress to liver fibrosis. [16-17]

Antioxidant properties: Curcumin's hepatoprotective properties may also be attributed to its antioxidant properties and ability to scavenge for reactive oxygen species (ROS), which have some role in liver damage from drugs (including alcohol), chemicals, and viral agents. Curcumin is a direct free radical scavenger and is believed to have activity to scavenge oxygen-centered radicals by its phenolic, β-diketone, and methoxy groups. In addition to its direct scavenging ability, curcumin has also been shown to induce the expression of antioxidant enzymes via activation of the Nrf2 pathway. [18-20] Curcumin causes an increase in superoxide dismutase, catalase and glutathione peroxidase activities, which are suppressed following liver injury. [21-22] An example of such a protective action is its use in hepatotoxicity, where turmeric decreased enzyme activity by 35% to 51% which consequently assisted in preventing lipid peroxidation of the liver membranes. [22]

Modulation of liver enzymes: Finally, clinical and preclinical studies have also shown that curcumin has an important lowering effect on levels of markers of liver injury in the serum, such as alanine aminotransferase, aspartate transaminase and alkaline phosphatase levels. [23-24] These levels are used to assess liver injury, as they are seen in conditions that cause hepatocyte membrane damage or biliary tract injury. [25] Curcumin, in particular, protects against the development of alcoholic liver disease by lowering ALP and AST levels in the presence of alcohol. [26] The protective effect is also observed in other drug-induced liver injuries, such as paracetamol overdose and arsenic poisoning. [19-20] Meta-analyzes of animal studies show a consistent effect of decreased ALT levels after curcumin supplementation, suggesting an improved integrity of hepatocytes after curcumin administration. [24]

Regulation of lipid metabolism: Curcumin is critical to prevent hepatic steatosis or fatty liver, via regulating hepatic lipogenesis and lipid oxidation-related genes. [27-28] Acting as a metabolic regulator, curcumin activates AMP-activated protein kinase (AMPK) and SIRT1 signaling pathways. [16, 28] It reduces the

level of Sterol Regulatory Element-Binding Protein-1c (SREBP-1c), an important regulator of lipogenesis, by inhibiting the transcriptional activity of its target genes, which include fatty acid synthase and acetyl-CoA carboxylase. [29] Simultaneously, curcumin has been shown to upregulate the gene PPAR-α, which increases beta-oxidation of fatty acids and energy expenditure in the liver. [27][29] Acting as both hypolipidemic and a lipolytic agent, curcumin results in decreased dietary fatty acid-induced triglyceride accumulation in the liver and improved insulin sensitivity. [28][30] Figure 3 illustrates the mechanisms of curcumin against liver diseases.

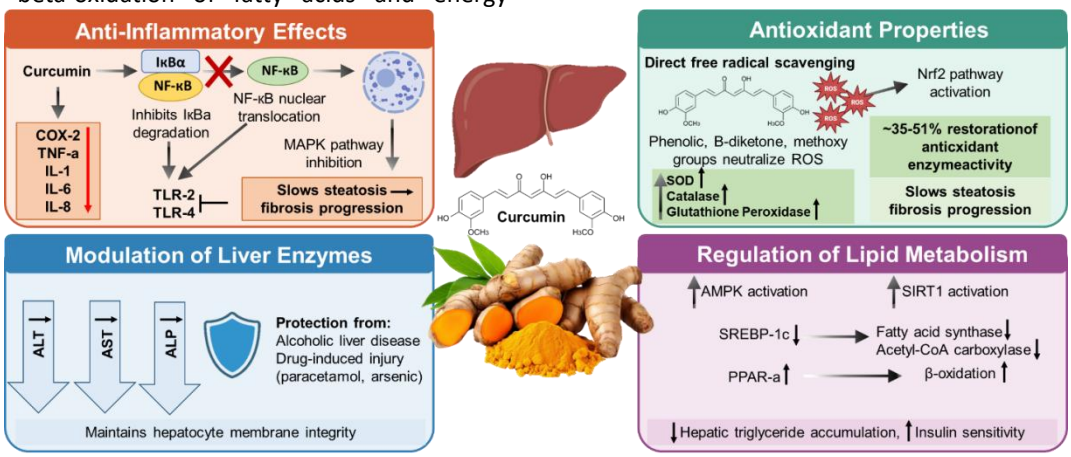

Figure 3. Mechanism of curcumin in protecting against liver disease

Clinical and preclinical evidence

***In vitro* studies:** Curcumin has been shown to exert strong and strong anti-cancer activities on human liver cancer cell lines HepG2 and Huh7. Curcumin is cytotoxic to both HepG2 and Huh7 cells with an IC_{50} of 5.57 µg/mL (48 h), with the nanoparticulated form having an even lower IC_{50} of 2.51 µg/mL. [31] It induces apoptosis through mitochondrial membrane depolarization and intracellular Ca^{2+} influx; curcumin also inhibits proliferation of HepG2 and Huh7 cells in conditions promoting carcinogenesis (such as exposure to acrylamide) at an IC_{50} of 20 µM. [32-33] Curcumin has more anti-fibrotic effects in activated HSC (LX-2 cells) by inhibiting autophagy via the PI3K/Akt/mTOR signaling pathway and inducing apoptosis in activated HSC. [34] The curcumin treatment in LX-2 cells led to a decrease in α-SMA expression and inhibited cell migration. The lowering of extracellular matrix deposition caused by curcumin also contributes to obstructing the progression of liver fibrosis. [35] Curcumin acts in a dual therapeutic way to induce apoptosis of neoplastic and fibrogenic hepatic cells as well as to protect normal hepatic tissue. The therapeutic efficacy of curcumin can be further improved by nanotechnology-based curcumin drug delivery systems, improving the cellular uptake and bioavailability of curcumin.

***In vivo* studies:** Animal studies have shown curcumin to be protective to the liver in models of toxin, alcohol, and metabolic injury. In models of liver fibrosis, a systematic review and meta-analysis of 23 studies in rodent models has shown a reduction in Fibrosis Area and hydroxyproline, a marker of collagen deposition in the liver, with curcumin supplementation. [24] Curcumin has been shown to reverse the hepatotoxic effect of CCl_4 and ethanol in rats by decreasing lipid peroxidation and restoring antioxidant enzyme levels of superoxide dismutase and catalase (Figure 4). [22, 26]. It also protects the liver against damage by medicinal drugs such as paracetamol and chemical agents such as arsenic and aflatoxin B1 (AFB1). [19, 20, 23] In Phase-I and Phase-II detoxification models, curcumin protects against liver injury by modulating detoxification enzymes and inhibiting inflammatory pathways. [23] In high fat diet-induced steatosis models, curcumin administration prevents hepatic lipid accumulation by inhibiting the Notch-1 signaling pathway

and activating AMPK, and prevents the progression of simple fatty liver to steatohepatitis. [28] [29] Table 2 represents the hepatoprotective effects, various dose regimens, the outcomes observed at micromolar concentrations, and the proposed molecular mechanisms of different formulations of curcumin using different experimental models.

Table 2. Summary of hepatoprotective effect of curcumin and its mechanism

Model Used (animal)	Dose	Major Outcomes	Mechanisms Proposed	Reference
Rats (Warm I/R injury)	50 mg/kg (intravenous)	Reduced hepatic injury following ischemia and reperfusion.	Regulation of heat shock proteins and antioxidant enzymes.	[21]
Rats (CCl4-induced toxicity)	Turmeric root powder	Significantly decreased marker enzymes and restored albumin levels.	Antioxidant enzyme activation and lipid levels reduction.	[22]
C57BL/6J Mice	0.15% in diet	Lowered body weight; reduced hepatic fat accumulation and fasting glucose.	Activation of AMP-activated protein kinase signaling.	[28]
Rats (Fatty liver/HFD)	100–200 mg/kg/day	Improved fatty liver architecture and systemic insulin sensitivity.	Suppression of the Notch-1 signaling pathway.	[29]
IUGR Rat/Piglet Models	400 mg/kg diet	Attenuated hepatic lipid accumulation and pro-inflammatory cytokines.	Modulation of the insulin-signaling and lipogenic pathways.	[30]
Wistar Rats	400 mg/kg BW	Significant reduction in serum ALT, AST, and MDA; restored GSH and Catalase.	Powerful antioxidant properties; protection of mitochondrial function.	[36]
Broilers (AFB1-induced injury)	150 mg/kg diet	Attenuated liver damage and reduced AFBO-DNA concentrations.	Inhibition of CYP450 isozymes and enhancement of antioxidant capacity.	[37] [38]

Human studies: Studies have found curcumin to be effective in metabolic and inflammatory liver diseases like Non-Alcoholic Fatty Liver Disease (NAFLD). One randomized, double-blind, placebo-controlled trial found that 12 weeks of 1000 mg/day curcumin supplementation considerably reduced hs-CRP levels and other inflammatory cytokines in people with NAFLD, compared to placebo. [14] In another study, using a phytosomal form of curcumin, a positive outcome was seen in liver steatosis, BMI, and serum transaminases in 75% of people taking the active treatment. [17] Nevertheless, the native curcumin has currently little clinical interest because of its poor bioavailability due to its low water-solubility, its transformation into other metabolites, and its elimination from the body. [6, 13] Curcumin is often combined in clinical trials with piperine, a black pepper extract that inhibits glucuronidation, which can increase the bioavailability of curcumin by 2000% (Figure 4). [13]

Curcumin-based formulations: To increase the therapeutic index of curcumin in the treatment of liver diseases, multiple new delivery systems have been developed to improve solubility and bioavailability (see below) [6]:

- **Nanoparticles:** To increase stability, cellular absorption, and homogeneity of curcumin concentrations in hepatic tissue, curcumin-loaded nanoparticles and nanoemulsions have been developed. [6]
- **Phytosomes, phospholipid complexes:** Formulations like phytosomal curcumin, in which curcumin is complexed with phospholipids, such as phosphatidylcholine, greatly increase delivery of curcumin into lipid-rich cell membranes and achieve greater clinical benefits in NAFLD and metabolic syndrome. [6,17]
- **Liposomes and micelles:** These lipid carriers may be useful for protecting curcumin from gastrointestinal degradation

and providing more sustained circulation in the body, potentially allowing for lower doses to achieve hepatoprotective effects. [6, 13]

Novel techniques are critical to translating positive results from preclinical rodent studies into effective, safe and standardized therapies for human liver disease.

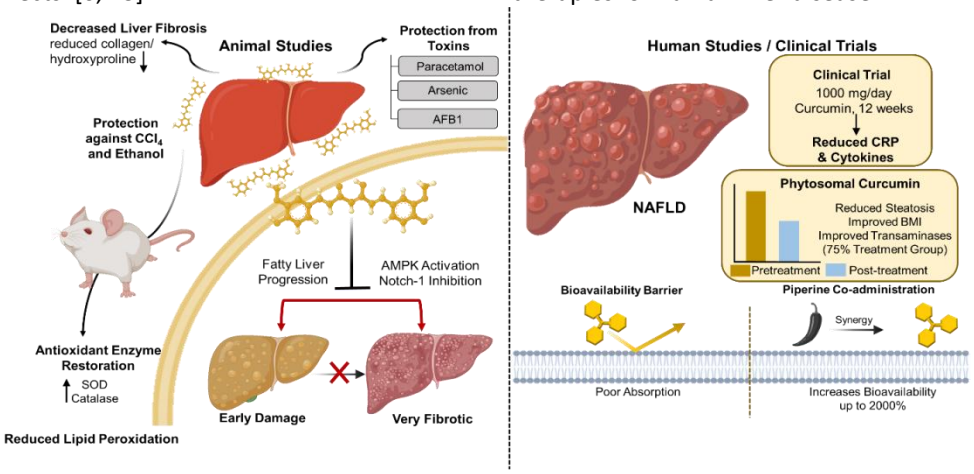

Figure 4. Preclinical and clinical studies of curcumin against the liver disease

3. CHALLENGES AND LIMITATIONS

Research gap: Despite wide-ranging literature on curcumin's hepatoprotective effects, there are limitations in the data, given that most results are derived from preclinical *in vitro* and *in vivo* studies, and only a small number of large long-term clinical trials have been conducted on humans. Despite the promising results, problems of bioavailability and pharmacokinetics affecting curcumin were not altogether solved, and the heterogeneity of the dosages, formulations and experimental conditions precluded head-to-head comparisons and limited the translatability of the results to clinical contexts. Moreover, mechanistic pathways through which curcumin specifically regulates genes and mediates epigenetic modifications in liver disease are still unclear.

Bioavailability issues: Nonetheless, the clinical utility of native curcumin is extremely limited due to its poor bioavailability, which can be attributed to its poor aqueous solubility, its intrinsic instability at physiological pH, and its wide-ranging first-pass metabolism in the liver and intestine. [6] Curcumin is rapidly conjugated with glucuronic acid and sulfate, resulting in only low concentrations in the bloodstream after high doses are taken orally. [13]

Because curcumin has a short plasma half-life (1.70 hours), short period of peak plasma concentration, and poor absorption in the GI tract, its bioavailability is low. The poor absorption can be explained by its hydrophobic nature and its poor solubility. The solubility of crystalline curcumin has been estimated to be 11 ng/mL, and less than 50 ng/mL serum concentration is obtained even at high oral doses. [40-41] It is cleared rapidly due to liver first-pass metabolism, and also due to important activity of the protein P-glycoprotein. Systems for improved delivery, such as solid self-emulsifying drug-delivery systems and cyclodextrin complexes, were later developed to considerably improve solubility and chemical stability. Compared to the native drugs, nanoformulations improve penetration through the GIT mucosa and achieve a more controlled release. These technological systems ease the drugs to enter the cells easier and achieve better targeting at the tissues rather than undergoing the limiting metabolism of the native molecule. [6]

Several methods have been devised to bypass these effects:

- **Adjuvants:** Co-administration with piperine, the active ingredient in black pepper, has been shown to increase curcumin bioavailability up to 2000% by inhibiting the

metabolic enzymes responsible for curcumin degradation. [13]

- **Nanotechnology:** developing curcumin-loaded nanoparticles, nano-emulsions, and solid lipid nanoparticles to improve solubility, stability and bioavailability of curcumin by overcoming premature metabolic degradation. [31]
- **Delivery systems:** Liposomal and phospholipid complexes (phytosomes) improve cell uptake, retention, and systemic circulation time, thus increasing uniform hepatic delivery of the drug. [17]

Safety and side effects: Curcumin is well tolerated, and is classified as "Generally Recognized as Safe" by the U.S. Food and Drug Administration. [39] Curcumin doses between 8 and 12 grams daily have been given in clinical studies over the course of several months and were found to be well tolerated with no dose-limiting toxicities. [6] Nausea, diarrhea, headache and rashes were reported as mild adverse effects in some of these studies. [33] Curcumin is hepatoprotective at therapeutic doses, but infrequent reports of increased liver enzymes and hepatotoxicity were found at very high doses and long-term use; these may need monitoring in clinical studies. [31, 33]

Furthermore, curcumin has the potential to interact with drugs in a clinically relevant way. Curcumin has been shown to inhibit several cytochrome P450 enzymes (including CYP3A4) that metabolize many pharmaceutical drugs. [32] The inhibition of CYPs can lead to toxicity, due to the accumulation of co-administered drugs in the body. [32][33] Co-administration should be approached with caution for curcumin and anticoagulants or anti-thrombotic therapies, due to an increase in clotting times. [42, 40]

Future Research Directions: However, curcumin has yet to receive serious validation as a mainstream clinical treatment. Remaining area of research include:

- **Larger clinical trials:** At present, available human studies are limited in size and period of time. [17] Longer, larger, multicenter randomized controlled trials are required to validate the efficacy in patients with differing background and disease stage. [31, 34]
- **Patient selection:** Due to the important heterogeneity of liver diseases such as hepatocellular carcinoma and NAFLD, identifying the patient sub-phenotypes most likely to benefit from curcumin treatment is critical. [31]
- **Adjunctive therapy:** Curcumin may improve clinical outcomes and reduce adverse effects when used as an adjunctive therapy with antiviral drugs for hepatitis or chemotherapy for liver cancer. [31, 34]

4. CONCLUSION

The major bioactive component of *Curcuma longa* (turmeric), curcumin, has been found to have hepatoprotective activity through various mechanisms of action. These mechanisms of action are interlinked and involve inhibition of the predominant pathways for liver injury, including repression of the transcription factor NF-κB and inhibition of pro-inflammatory cytokines such as TNF-α and IL-6. As a powerful antioxidant, curcumin attenuates redox stress through production of endogenous antioxidant enzymes, such as superoxide dismutase and catalase, induced by transcription factor Nrf2. Curcumin protects against oxidative stress-induced hepatotoxicity via activation of this pathway. Curcumin has been reported to modulate lipid metabolism by activating AMPK and inhibiting SREBP-1c, resulting in reduced endogenous lipogenesis and hepatic steatosis. Other data from animal studies suggest that it may also inhibit fibrosis in chronic liver disease by inhibiting collagen deposition. Despite promising preclinical results, activity in clinical populations has been limited due to poor solubility as well as rapid metabolism and low bioavailability. Various nanoparticle, liposome, and phospholipid systems seek to improve delivery. Further clinical trials required.

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Reviewing & Editing: GTR

Approval of final manuscript: All authors

Declaration of Generative AI

The authors declare this manuscript was written without the use of generative artificial intelligence tools. All the content, including text generation, data analysis and references was developed and reviewed by the author without assistance from AI technologies.

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