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Review Article

Plant-Based Bioactive Compounds in the Prevention and Management of Liver Disorders: A Review

Shweta Bhandari*, Sufiyameezan Khan, Rahul Trivedi, Rajesh Maheshwari, Swetaba Besh, Dilsar Gohil, Ujjval Vagehla

Department of Pharmacy, Sumandeep Vidyapeeth (Deemed to be University), Piparia, Vadodara, Gujarat, INDIA.

ABSTRACT

Hepatotoxicity is basically harm or else weakening of the liver functioning brought on by a variety of substances, including alcohol, medications, endotoxins, and environmental factors. Because of its high blood perfusion rate and high level of enzymatic activity, the liver, which serves as the primary metabolic and detoxifying organ, is especially vulnerable to these types of attacks. Exposure to these agents may lead liver steatosis, fibrosis, cirrhosis, and hepatocellular carcinoma. The mechanisms involved in hepatotoxicity are oxidative stress, mitochondrial dysfunction, immune-mediated injury, lipid peroxidation, bile-related damage, and ischemic injury. Conventional therapies mostly offers limited protection, highlighting the growing interest in phytoconstituents with hepatoprotective potential. A wide range of natural compounds such as resveratrol, curcumin, catechins, carotenoids, quercetin, ursolic acid, silymarin, schisantherin, and saikosaponin have been widely examined for their antioxidant, anti-inflammatory, and anti-apoptotic properties. These plant based bioactive compounds lowers the inflammation, oxidative stress, and cellular death by regulating important cell signaling networks such as NF-κB, MAPK, PI3K/Akt, and Nrf2. Also, they enhance insulin sensitivity, control lipid metabolism, and stop the growth of fibrosis, all of which assist to a multi-focus treatment strategy. Clinical and preclinical research confirms their use in the prevention and management of various liver conditions. Though, challenges such as bioavailability, optimal dosage, and long-term safety remain key barriers to clinical translation. Overall, the incorporation of phytoconstituents into therapeutic approaches delivers a promising path for hepatoprotection and the prevention of chronic liver diseases.

Keywords: Hepatotoxicity, Liver fibrosis, Hepatocellular carcinoma, Mitochondrial dysfunction, Oxidative stress, Phytoconstituents.

Correspondence:
Dr. Shweta Bhandari
Department of Pharmacy, Sumandeep Vidyapeeth (Deemed to be University), Piparia, Vadodara-391760, Gujarat, INDIA.
Email: bhandarishweta257@gmail.com

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INTRODUCTION

Damage or impairment of liver function happens due to heavy interaction with endotoxins & xenobiotics is known as hepatotoxicity. The liver is 2nd largest organ of the body that is composed of hepatocytes, or liver cells, blood vessels, and bile ducts. It consist of two lobes, a smaller left lobe & bigger right lobe. Because it performs so many essential functions for a healthy lifestyle, the liver is a highly complex organ. It strains blood received from the hepatic artery received from the heart and the portal vein received from the intestine. The liver also produces important substances such as plasma proteins (including albumin, which regulates fluid balance in the body), clotting factors that prevent excessive bleeding, cholesterol, vitamin D, immune factors that fight infections, certain hormones, and bile. In addition, it plays a crucial role in detoxification by removing waste products from the system (Larson *et al*., 2005).

The liver is remarkably capable of repairing damaged tissue on its own. This makes it possible to donate a portion of one's liver to a blood relative of the same type, and both individuals will have healthy livers that will revert to their initial size. Only liver regeneration is effective in livers that are in good health. Therefore, showcasing a few traits of liver illnesses is essential. Many illnesses and environmental factors can impact liver function, leading to gallstones, liver steatosis, fibrosis, cirrhosis, and even cancer. These include infections or viral hepatitis that cause inflammation, metabolic diseases that hinder energy production, exposure to drugs and toxins, or reduced blood supply. The liver itself may sustain damage from xenobiotics that are eliminated through biotransformation (mostly through detoxification, but occasionally through the generation of active or even more hazardous metabolites). Because of its high metabolic capacity and perfusion rate, the liver is constantly exposed to large concentrations of xenobiotics and

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their metabolites. Thankfully, the liver can heal any underlying damage and has a great potential for regeneration. Hepatotoxicity generally happens when the liver's capacity for regeneration is depleted and cell damage results (Jaeschke *et al*., 2012).

Types of liver disease: Liver disease can be acute or chronic. There are various types of Liver conditions some of the common types are mentioned below:

Acute liver failure is a severe condition in which the liver rapidly loses its functional capacity within a short time frame. In contrast to chronic liver failure, which evolves slowly over several years, acute liver failure develops within just a few days after exposure to toxic or damaging agents. It may lead to serious complications such as excessive bleeding and increased intracranial pressure. Another name for this condition is *fulminant hepatic failure*. Since it is a medical emergency, hospitalization is required. While some causes are reversible with timely treatment, in severe cases, liver transplantation may be the only life-saving option (Berto *et al*., 2012).

Hepatitis is defined as inflammation of the liver, most often resulting from viral infections. It can result from several factors, including autoimmune reactions (autoimmune hepatitis), viral infections such as Hepatitis as well as bacterial or parasitic infections. Additional causes include excessive alcohol intake, toxins (e.g., poisonous mushrooms), drug-induced injury such as acetaminophen overdose, or inherited conditions like cystic fibrosis, Wilson's disease and hemochromatosis. Symptoms may include abdominal pain, swelling, jaundice, dark urine, pale stools, fatigue, low-grade fever, itching, loss of appetite, nausea, vomiting, gynecomastia in males, and weight loss (Caviglia *et al*., 2015).

Alcohol related liver disease involves a range of conditions like fatty liver, alcoholic hepatitis, and alcoholic cirrhosis. With ongoing excessive alcohol consumption, the disorder typically advances from fatty liver to hepatitis and eventually to cirrhosis. If heavy drinking continues, the disease often progresses from fatty liver to hepatitis and ultimately cirrhosis. However, some individuals may develop cirrhosis without showing signs of hepatitis, while others may have alcoholic hepatitis but remain asymptomatic (Choi & Runyon, 2012; Manu *et al*., 2022; O'Shea *et al*., 2010).

Gilbert's syndrome Gilbert's syndrome is a mild hereditary condition in which the liver cannot efficiently process bilirubin, a byproduct of red blood cell breakdown. The disorder is harmless, usually requires no treatment, and is often detected accidentally during routine blood tests showing slightly elevated bilirubin levels (Claridge *et al*., 2011; Czaja, 1998).

Liver hemangioma A liver hemangioma is a benign mass composed of tangled blood vessels within the liver. Most cases are discovered incidentally during imaging for other conditions. Since they usually cause no symptoms and rarely require intervention, untreated hemangiomas are not linked to liver cancer (Assy *et al*., 2009).

Non-alcoholic steatohepatitis is a form of fatty liver disease that resembles alcoholic liver damage but occurs in individuals who consume little or no alcohol. It is characterized by fat buildup, inflammation, and tissue injury in the liver. While many patients remain unaware of the condition initially, severe cases may progress to cirrhosis, causing irreversible scarring and impaired function. When fat deposits are detected without inflammation, the condition is called Non Alcoholic Fatty Liver Disease. Diagnosis of NASH versus simple fatty liver is typically confirmed through liver biopsy (Obead *et al*., 2024).

Cirrhosis is the terminal phase of chronic liver disease in which prolonged inflammation leads to extensive scarring and replacement of normal liver tissue. The buildup of this scar tissue interferes with essential liver functions and, over time, may progress to complete liver failure. Although cirrhosis is irreversible, appropriate treatment can slow or halt its progression (Somnay *et al*., 2024).

Wilson's disease Wilson's disease is hereditary marked by the abnormal buildup of copper in many organs such like brain, eyes and liver. Copper is normally absorbed from food and excess amounts are removed through bile, but in Wilson's disease this process is impaired, leading to toxic accumulation. Without treatment, the condition can be life-threatening, but early diagnosis and therapy allow most patients to live healthy, normal lives (Merck Manual, 2011).

Mechanisms Involved in Liver Injury

Because the liver is a primary target for various toxins such as endotoxins, xenobiotics, and alcohol, and due to its distinctive metabolism shown in Figure 1. Diverse cellular composition, and close connection with the gastrointestinal tract, the mechanisms underlying liver injury are highly complex and interrelated.

Risks for Liver Diseases

- **Too Much Alcohol:** High alcohol consumption leads to severe liver damage that initially shows no symptoms and causes injury that are sometimes beyond repair and these factors are listed in Figure 2.
- **History of Liver Disease:** People with genetic liver history or and present liver conditions may be more vulnerable to liver disease.
- **Harmful Supplements:** Supplement is doesn't always good for you and it is highly associated with liver damage.

- **Obesity, Diabetes, high Cholesterol:** These underlying conditions can contribute and worsen the liver damage and may also lead to cirrhosis or liver cancer
- **Exposure to Toxins:** Since liver is the organ that clears the toxins from the blood, high exposure to the toxins can be harmful.
- **Phytoconstituents:** Evidence from experimental and clinical studies highlights the hepatoprotective potential of various phytoconstituents mentioned in Table 1.

Resveratrol

Extensive research has demonstrated the therapeutic potential of resveratrol in various liver disorders. Experimental studies indicate that its administration enhances survival following liver transplantation, reduces fat deposition, and protects against ischemia-induced necrosis and apoptosis. Resveratrol has shown protective effects against chemical, cholestatic, and alcohol-related liver damage. It also improves glucose and lipid metabolism, decreases hepatic fibrosis and fat accumulation, and modifies the fatty acid profile of hepatocytes. Due to calorie-restrictive properties, antioxidant properties and also its anti-inflammatory resveratrol is promising herbal therapy for the treatment of many liver conditions. Clinical studies have also assessed its impact on lipid levels and liver enzyme activity in patients with metabolic syndrome and related conditions (Kawada *et al.*, 1998; Smoliga *et al.*, 2011).

Curcumin

Curcumin, the principal curcuminoid found in turmeric, is largely responsible for its biological activities. It has strong anti-inflammatory & antioxidant effects and modulates numerous signaling pathways. Since oxidative stress plays a critical role in chronic diseases and carcinogenesis, curcumin's ability to suppress lipid peroxidation and scavenge free radicals has been highlighted in several studies. A key finding has been its ability to inhibit nuclear factor κB (NF-κB), a central regulator of inflammation that also supports cell survival, proliferation, invasion, and angiogenesis. By interfering with these pathways, curcumin contributes to slowing liver disease progression. It has demonstrated beneficial effects in NAFLD, in reducing fibrosis and inflammation, in suppressing hepatitis C virus (HCV) replication, and in hindering processes linked to hepatocellular carcinoma (HCC) development in chronic HCV-related disease (Sethi *et al.*, 2009).

Atractylodin

Evidence suggests that atractylodin alleviates high-fat diet (HFD)-induced NAFLD by lowering oxidative stress and supporting cell survival. *In vitro* studies reveal that atractylodin inhibits ferroptosis, thereby improving cell viability in NAFLD models. As NAFLD is strongly associated with obesity, oxidative stress, and inflammation, atractylodin's anti-inflammatory and antioxidant actions appear central to its hepatoprotective role. Previous findings also show its ability to improve acute liver failure triggered by D-galactosamine and lipopolysaccharide (LPS). Nonetheless, the precise molecular mechanisms through which atractylodin exerts its beneficial effects on NAFLD remain to be fully elucidated (Ke *et al.*, 2025).

Catechins

Catechins exert antioxidant effects through multiple mechanisms, including balancing enzyme activities and modulating signaling cascades. They suppress reactive oxygen species (ROS)-generating enzymes such as NADPH oxidase, while activating antioxidant defenses like GSH, SOD, CAT, GPX, GST, and NQO1. In addition, catechins stimulate the Keap1/Nrf2/ARE pathway, inhibit MAPK/AP-1 and NF-κB activation, and increase the expression of PPARγ, PGC1α, and PPARα, collectively reducing oxidative stress. However, certain limitations exist, including their potential pro-oxidant and toxic effects under specific conditions, questionable activity of metabolites, and low bioavailability. Establishing safe dosage ranges and optimizing the biological environment to enhance their antioxidant activity are important

Figure 1: Mechanism of Liver injury.

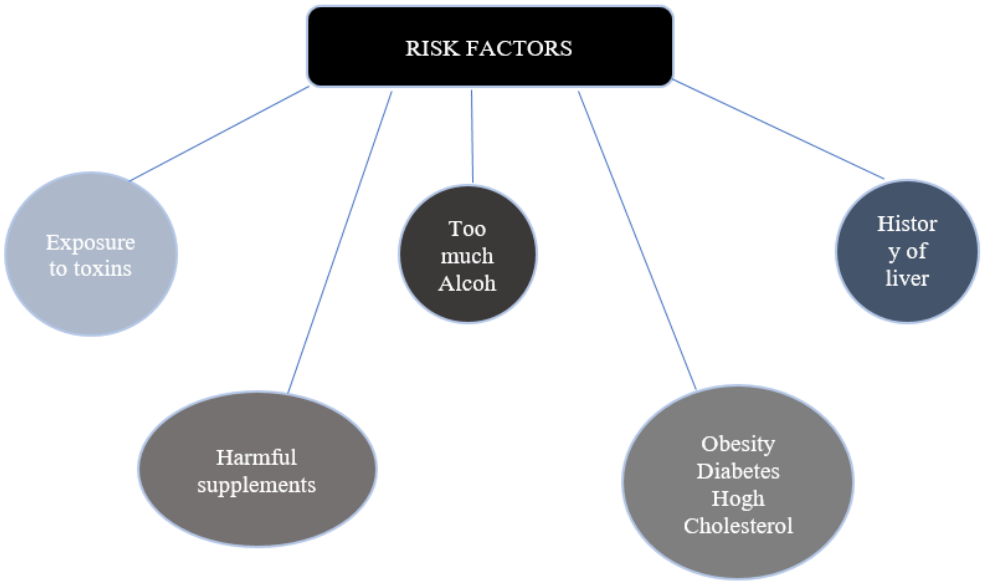

Figure 2: Risk factors.

strategies to minimize adverse effects and maximize therapeutic potential (Abunofal & Mohan, 2022; Yan *et al.*, 2021).

Carotenoids

Carotenoids play a protective role in liver health. Studies show that lower circulating carotenoid levels are linked to a higher risk of these diseases, while supplementation with compounds such as β-carotene, lycopene, β-cryptoxanthin, lutein, zeaxanthin, and astaxanthin can reduce liver damage. Their hepatoprotective effects are mainly due to their strong antioxidant activity, which lowers oxidative stress, the conversion of β-carotene and β-cryptoxanthin into vitamin A that supports retinoid signalling in the liver, and the production of apocarotenoid metabolites that regulate lipid metabolism and inflammation. Thus, carotenoids help improve liver antioxidant defences, modulate fat metabolism, and reduce inflammation, making them promising agents for preventing or managing liver diseases, though more research is needed to clarify their mechanisms and effective doses (Elvira-Torales *et al.*, 2019).

Quercetin

Quercetin has been widely recognized as a potent hepatoprotective compound, with its pharmacological actions in liver diseases investigated extensively. Its protective effects are primarily mediated through suppression of liver inflammation via the NF-κB, TLR, and NLRP3 signaling pathways, reduction of oxidative stress through PI3K/Nrf2 regulation, modulation of mTOR-related autophagy, and inhibition of pro-apoptotic factor expression implicated in liver pathology. During hepatic steatosis, quercetin primarily influences PPAR, UCP, and PLIN2-related pathways, promoting fatty acid browning and reducing lipid accumulation. At the stage of liver fibrosis, it modulates TGF-β1, estrogen receptors, and apoptotic pathways to suppress extracellular matrix (ECM) deposition. In advanced Hepatocellular Carcinoma (HCC), quercetin acts on hTERT, MEK1/ERK1/2, Notch, and Wnt/β-catenin pathways, thereby inhibiting cancer cell growth and metastasis. Overall, quercetin demonstrates significant efficacy as a hepatoprotective agent across different stages of liver disease (Ader *et al.*, 2000; Afifi *et al.*, 2018).

Ursolic Acid

Alcohol is a topmost root of chronic hepatic injury globally, & Ursolic Acid (UA), a pentacyclic triterpenoid, has emerged as a promising protective compound. UA exhibits antioxidant, anti-inflammatory, anticancer, and hepatoprotective properties. Notably, its metabolism *in vivo* produces epoxy-modified derivatives containing an electrophilic epoxy group that binds to nucleophilic protein residues. Proteomic studies using an alkynyl-modified UA probe identified caspase-3 (CASP3) as a principal target of UA responsible for its hepatoprotective activity. Molecular docking and biochemical assays confirmed that UA metabolites covalently bind to Cys-163 of CASP3, thereby suppressing its activity. Since CASP3 is a critical mediator of apoptosis, this inhibition reduces PARP cleavage and protects hepatocytes from apoptotic death. *In vitro* studies on alcohol-exposed HepG2 cells showed that UA metabolites effectively blocked CASP3 activity, while *in vivo*, oral administration of UA (20-80 mg/kg/day for one week) markedly improved alcohol-induced hepatic injury in a dose-dependent fashion. Collectively, these findings suggest that the hepatoprotective effects of UA in ALD are mainly attributed to inhibition of CASP3-mediated apoptosis, underscoring its potential as a therapeutic lead compound for alcohol-related liver disorders (Zhou *et al.*, 2024).

Silymarin

Silymarin, an extract derived from milk thistle seeds, has been traditionally used for decades in the management of liver disorders. Preclinical studies suggest that it mitigates oxidative stress and related cytotoxicity, thereby preserving the function of healthy hepatocytes or those not yet irreversibly injured. *Eurosil 85*, a specialized formulation designed to enhance oral bioavailability, has been the primary preparation investigated in most clinical trials. Beyond its role as a free radical scavenger, silymarin also modulates key enzymes implicated in cellular injury, fibrosis, and cirrhosis. Evidence from pooled clinical analyses indicates that silymarin action suggestively decreases mortality in patients with cirrhosis. Furthermore, it had shown benefits in improving glycemic control among individuals with alcoholic cirrhosis complicated by diabetes, as well as effectiveness in cases of drug-induced liver injury. Clinical trials consistently report that silymarin is well tolerated, with minimal side effects and no serious adverse outcomes documented (Song *et al.*, 2006). For maximum therapeutic efficacy, silymarin administration should commence early in conditions such as fatty liver disease or acute liver failure and when the liver's regenerative potential remains high and oxidative stress-driven cytotoxicity can still be counteracted.

Schisantherin

Schisantherin A (SchA), a dibenzocyclooctadiene lignan derived from *Schisandra sphenanthera*, has shown promising hepatoprotective potential. It exerts strong protective effects on the liver by preserving liver function and reducing tissue

Table 1: Phytoconstituents, source & uses.

Phytoconstituents	Source	Molecular Formula	Uses	Reference
Resveratrol	Grapes	$C_{14}H_{12}O_3$	Cardiovascular Health, Anti-inflammatory, Antioxidant effect, Neuroprotection, Diabetes and Metabolism	Baur & Sinclair, (2006).
Curcumin	Turmeric	$C_{21}H_{20}0_6$	Cardiovascular Disease, Respiratory Disorder, Cancer, and Inflammation	Shishodia *et al.*, (2005).
Atractylodin	Atractylodes lancea	$C_{13}H_{10}O$	Anti-inflammatory effect, Metabolic Regulation	Song *et al.*, (2024).
Catechin	Camellia sinensis	$C_{15}H_{14}O_6$	Antimicrobial Activity, Antioxidant action, Cardiovascular Disease	Khan & Mukhtar, (2007).
Carotenoids	Carrot & Sweet Potato	$C_{40}H_{56}$	Anti-inflammatory action, Antioxidant effect	Rao & Rao, (2007)
Quercetin	Apples & Cranberries	$C_{15}H_{10}O_{11}$	Anti-inflammatory action, Antioxidant effect, Cancer, Cardiovascular Disease	(Li *et al.*,(2016)
Ursolic Acid	Rosemary, Lavender	$C_{30}H_{48}O_3$	Anti-inflammatory action, Antioxidant effect, Antidiabetic, Antimicrobial effect	Ma *et al.*, (2017).
Silymarin	Milk Thistle	$C_{25}H_{22}O_{10}$	Anti-inflammatory action, Antioxidant effect	Polyak *et al.*, (2007).
Schisantherin	Schisandra chinensis	$C_{24}H_{32}O_7$	Neuroprotection, Liver Fibrosis	Zheng *et al.*, (2017).
Saikosaponin	Bupleurum	$C_{42}H_{68}O_{13}$	Anti-inflammatory, Antidepressant, Antiviral, Anti-fibrotic	Gu *et al.*, (2022).

damage during pathological conditions. SchA alleviates oxidative and nitrosative stress, thereby minimizing free radical-induced hepatocellular injury. It also attenuates the inflammatory state by suppressing inflammatory cell infiltration and cytokine release, which are major contributors to liver damage. In addition, SchA prevents hepatocyte apoptosis, further safeguarding liver structure and function. Interestingly, its protective activity is not associated with alterations in autophagy but is primarily related to the suppression of the Mitogen-Activated Protein Kinase (MAPK) pathway, a key mediator of inflammation and cell death. Collectively, SchA demonstrates significant hepatoprotective properties, making it a potential prophylactic and therapeutic option for preventing or managing liver injury in clinical conditions (Zheng *et al*., 2017).

Saikosaponin

Saikosaponin D (SSD), a principal triterpenoid saponin derived from *Bupleurum*, exhibits notable hepatoprotective properties against non-alcoholic fatty liver disease (NAFLD). In experimental studies, SSD demonstrated dose-dependent effects by attenuating high-fat diet-induced weight gain, enhancing insulin sensitivity, and lowering hepatic lipid deposition along with serum injury markers such as AST and ALT. Mechanistically, SSD modulates the gut-liver axis by suppressing intestinal farnesoid X receptor (FXR) signaling through inhibition of Fxr, Shp, Fgf15, and Asbt expression. Additionally, SSD alters gut microbiota composition, reducing bile salt hydrolase (BSH)-expressing bacteria like Clostridium, which lowers the ratio of unconjugated to conjugated bile acids and further inhibits intestinal FXR activity. Through this combined effect on intestinal signaling and gut microbiota, SSD effectively improves liver lipid metabolism and agent for liver-related disorders (Gu *et al*., 2022).

CONCLUSION

The liver plays an essential role in maintaining systemic homeostasis, yet it remains extremely vulnerable to toxic insults from xenobiotics, alcohol, drugs, and environmental agents. The complexity of hepatotoxicity rises from tangled mechanisms involving oxidative stress, mitochondrial damage, immune activation, and dysregulated lipid metabolism. Present pharmacological interventions often lack specificity and efficacy in fully preventing liver injury, which has enhanced the search for safer and more effective alternatives. Evidence from experimental and clinical studies highlights the hepatoprotective potential of various phytoconstituents. Compounds such as resveratrol, curcumin, catechins, carotenoids, quercetin, ursolic acid, silymarin, schisantherin, and saikosaponin have demonstrated significant efficacy in reducing oxidative stress, modulating inflammatory cascades, and inhibiting apoptosis. These natural molecules not only improve the antioxidant defense system but also regulate signaling pathways critical for hepatocyte survival and regeneration. Additionally, they exhibit preventive effects against steatosis, fibrosis, cirrhosis, and even hepatocarcinogenesis. In spite of their therapeutic promise, challenges related to poor solubility, limited bioavailability, and variable pharmacokinetics need to be addressed through advanced formulation technologies and clinical validation. Upcoming research should focus on well-designed trials, synergistic combinations of phytoconstituents, and clarification of molecular targets to maximize their hepatoprotective efficacy. In conclusion, phytoconstituents signify a valued and multifaceted approach for liver protection, offering potential not only in mitigating hepatotoxicity but also in preventing progression of chronic liver diseases. Their addition into clinical practice, alongside conventional therapies, could pave the way for safer, natural, and more holistic strategies in hepatology.

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ABBREVIATIONS

HCC: Hepatocellular Carcinoma; **OS:** Oxidative Stress; **LPO:** Lipid Peroxidation; **Nrf2:** Nuclear Factor Erythroid 2-Related Factor 2; **NF-κB:** Nuclear Factor kappa B; **NAFLD:** Non-Alcoholic Fatty Liver Disease.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

SUMMARY

Hepatotoxicity refers to the impairment or weakening of liver function caused by various agents, including drugs, alcohol, endotoxins, and environmental toxins. Due to its high blood flow and rich enzymatic activity, the liver is particularly susceptible to damage, which can manifest as steatosis, fibrosis, cirrhosis, or even hepatocellular carcinoma. This article highlights that oxidative stress, mitochondrial dysfunction, immune-mediated injury, lipid peroxidation, bile-related damage, and ischemia are key mechanisms driving hepatotoxicity. Building on prior research, the article demonstrates that natural bioactive compounds, including resveratrol, curcumin, catechins, carotenoids, quercetin, ursolic acid, silymarin, schisantherin, and saikosaponin, can provide significant hepatoprotective effects. These compounds act through antioxidant, anti-inflammatory, and anti-apoptotic mechanisms, modulating crucial signaling pathways such as NF-κB, MAPK, PI3K/Akt, and Nrf2, while also improving insulin sensitivity, regulating lipid metabolism, and preventing fibrosis. The article confirms, consistent with previous preclinical and clinical studies, that these phytoconstituents reduce oxidative damage, inflammation, and cellular death in the liver. Overall, the findings reinforce that integrating such bioactive compounds into

therapeutic strategies offers a promising, multi-targeted approach to prevent and manage hepatotoxicity and chronic liver diseases, while addressing limitations of conventional treatment.

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