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Study on Effects of Plant Extract on Liver Toxicity and Associated Pathological Complications

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Abstract: *The liver operates as the central metabolic hub of the human Body, critically responsible for xenobiotic detoxification, biochemical synthesis, and metabolic Homeostasis. Due to continuous physiological exposure to concentrated volumes of Medications, pharmaceutical metabolites, environmental toxins, and lipid overloads, it remains Uniquely susceptible to a severe spectrum of acute and chronic pathological conditions. These Conditions include non-alcoholic steatohepatitis (NASH), metabolic dysfunction-associated fatty Liver disease (MAFLD), chemically induced tissue fibrosis, and primary liver cancer (Hepatocellular Carcinoma). Although several synthetic targeted drugs such as Ademetionine, N-Acetylcysteine, and Ursodeoxycholic Acid are clinically utilized to manage Hepatic disorders, their therapeutic utility is frequently restricted by systemic side effects, high Financial barriers, or a distinct lack of direct tissue-regenerative capabilities. Consequently, Contemporary ethnopharmacological research has increasingly focused on plant-derived Bioactive complexes as safer, affordable, multi-target alternative candidates. Due to Infrastructural limitations at the undergraduate research level, this study leverages standard Microbiological assays to evaluate plant extract potency, utilizing the underlying metabolic Similarities between hepatic cellular pathways and microbes to estimate upstream Hepatoprotective efficacy against pathogen-driven inflammatory environments. Botanical matrices consisting of whole plants or leaves from five Distinct species Guava (Psidium guajava), Sage (Salvia officinalis), Rhamnus (Ziziphus Spina-christi), Mulberry (Morus alba L.), and Olive (Olea europaea L.) Were gathered from Agricultural markets. The biomatrices were uniformly oven-dried at 40°C for 48 hours to Eliminate residual moisture and mechanically processed into a fine homogenized powder.*

Maceration extraction was performed by soaking 250 g of dried sample in 1 L of 70% ethanol (1:4 mass-to- volume ratio) at a constant temperature of 30°C for 48 hours. The crude mixture was filtered through Whatman No. 4 filter paper to remove particulate fractions and subsequently concentrated using a rotary evaporator at 50°C until complete dryness.

The final sticky crude extract yields were reconstituted in sterile distilled water to achieve a baseline stock Solution concentration of 500 mg/ml and preserved at -20°C for systematic bioassays. Antimicrobial, inhibitory, and bactericidal activities were determined in strict accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines. Six target test strains associated with hepatic inflammation, endotoxemia, and gut liver axis translocation were evaluated: Gram-positive (Staphylococcus aureus, Bacillus cereus), Gram-negative (Escherichia coli, Pasteurella multocida, Salmonella Enteritidis), and Mollicutes (Mycoplasma gallisepticum). The Standard Agar Disc Diffusion technique evaluated zones of inhibition (ZOI) in triplicate across 10%, 25%, 50%, and 100% extract concentrations, benchmarked against seven commercial Antibiotics (Amoxicillin- Clavulanic Acid, Ampicillin, Vancomycin, Lincomycin, Gentamicin, Streptomycin, and Tetracycline). Standard two-fold serial microdilutions (ranging from 625 to 10,000 µg/ml) mapped out the Minimum Inhibitory Concentration (MIC), while the sub-culturing of clear agar zones into fresh nutrient recovery broth established the Minimum Bactericidal Concentration (MBC).

The disc diffusion assays revealed highly significant, concentration-dependent Antibacterial activity across all plant extracts, with Salvia officinalis and Psidium guajava Demonstrating the most potent

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broad-spectrum profiles. Against S. Aureus, Salvia officinalis Produced a noticeable zone of inhibition even at its lowest 10% dilution (3.5 ± 0.32 mm), Expanding dramatically to 17.05 ± 1.05 mm at 100% concentration, followed closely by Psidium Guajava (15.62 ± 1.15 mm at 100%). Against Bacillus cereus, early susceptibility was recorded At 10% concentration for Salvia officinalis (7.5 ± 0.25 mm) and Olea europaea (8.5 ± 0.55 mm), Highlighting a powerful natural defense against spore-forming organisms whose emetic toxins Cause hepatocyte mitochondrial dysfunction. Enteric threats driving portal vein endotoxemia Were successfully arrested: E. Coli growth was

Keywords: *Salvia officinalis and Psidium guajava*

I. INTRODUCTION

The liver is a central metabolic hub responsible for xenobiotic detoxification, biochemical synthesis, and metabolic homeostasis. Due to its exposure to concentrated levels of toxins, pharmaceutical metabolites, and lipid overloads, it is uniquely susceptible to a spectrum of pathological conditions, including nonalcoholic steatohepatitis (nash), metabolic dysfunction-associated fatty liver disease (mafld), and chemically induced fibrosis.

While conventional targeted therapies exist, they are frequently limited by systemic side effects or financial barriers. Consequently, ethnopharmacological research has increasingly turned toward plant- derived bioactive complexes. This project evaluates the structural, biochemical, and mechanistic pathways through which specific plant extracts exert hepatoprotective effects, balancing their therapeutic efficacy against the rising clinical incidence of herb-induced liver injury (hili).

II. GROSS ANATOMICAL OVERVIEW OF THE TARGET ORGAN

To understand hepatic pathology, a definitive grasp of macroscopic liver anatomy is required. The liver is split into distinct anatomical zones across its anterior and posterior surfaces:

2.1 lobular segmentation

• Anterior surface: dominated by the expansive right lobe and the smaller, tapered left lobe, separated cleanly by the vertical falciform ligament.

• Posterior surface: hosts the highly specialized caudate lobe (positioned superiorly between the inferior vena cava and the porta hepatis) along with its structural caudate process, and the inferiorly positioned quadrate lobe adjacent to the gallbladder.

2.2 vascular architecture and biliary drainage

The underside of the liver features the porta hepatis (liver hilum), a crucial neurovascular gateway containing the portal triad:

1. Proper hepatic artery: delivers oxygenated systemic arterial blood.
2. Portal vein: directs nutrient-rich, toxin-laden blood from the gastrointestinal tract to the hepatic parenchyma for filtration.
3. Common bile duct: channels newly synthesized bile away from the liver towards the duodenum for lipid emulsification, with excess bile stored and concentrated within the gallbladder.
4. Venous return: filtered deoxygenated blood is carried away from the parenchymal tissue via the internal hepatic veins, draining directly into the inferior vena cava (ivc).

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Table: Epidemiology and Global Impact of Major Liver Diseases

S. N.	Liver Disease	Description	Global Impact	References
1	Hepatitis B	Viral infection causing liver inflammation.	In 2019, an estimated 296 million individuals were afflicted with chronic hepatitis B, resulting in 820,000 fatalities each year.	Ghulam, F., Zakaria, N. et al. [11]
2	Hepatitis C	Viral infection leading to liver inflammation.	In 2019, approximately 58 million individuals were diagnosed with chronic hepatitis C, resulting in 290,000 fatalities each year.	Khan, S., Nosheen, M. et al. [12]
3	Alcoholic Liver Disease	Liver damage due to excessive alcohol consumption.	3 million deaths worldwide each year.	Provan, L., Forrest, E. et al. [13]
4	Fatty Liver Disease (Nonalcoholic)	Accumulation of fat in the liver without alcohol consumption.	Leading cause of chronic liver disease; affects roughly 25% of the global population.	Boccatonda, A., Andreetto, L. [14]
5	Liver Cirrhosis	Late stage of scarring (fibrosis) of the liver.	Causes 1.32 million deaths annually worldwide.	Badvath,D., Miriyala,A. [15]
6	Liver Cancer	Primary liver cancer.	The fourth most common cause of cancer-related fatalitiesworldwide, resulting in approximately 830,000 deaths annually.	Hao, X., Fan, R. [16]
7	Autoimmune Hepatitis	Chronic inflammation of the liver due to the immune system targeting host tissues.	Rare, with an annual incidence rate of 1–2 per 100,000 individuals.	Puustinen, L., Barner-Rasmussen, N. [17]
8	Hemochromatosis	Iron overload disorder leading to progressive liver damage.	Affects 1 in 200–300 people of Northern European descent; can rapidly lead to cirrhosis and liver cancer.	Kane, S., Roberts, C. [18]
9	Wilson's Disease	Copper accumulation in the liver as a result of a genetic disorder.	Rare, with an incidence rate of 1 in 30,000; can lead to severe liver damage if left untreated.	Jopowicz, A., Tarnacka, B. [19]

Although several synthetic drugs are used to manage liver diseases, they frequently lack direct regenerative capabilities or carry risks of adverse effects. Consequently, there is substantial research interest in identifying natural hepatoprotective options. Plants such as Phyllanthus niruri, Tinospora cordifolia, and Eclipta alba have been used for centuries in traditional Ayurvedic and folk medicine systems for treating jaundice and biliary disorders. Their therapeutic potential is attributed to a diverse array of phytochemicals, including lignans, alkaloids, glycosides, and coumestans, which act synergistically to scavenge free radicals, stabilize cellular membranes, suppress inflammatory pathways, and stimulate hepatocyte regeneration. This pharmacological study presents a systematic evaluation of these

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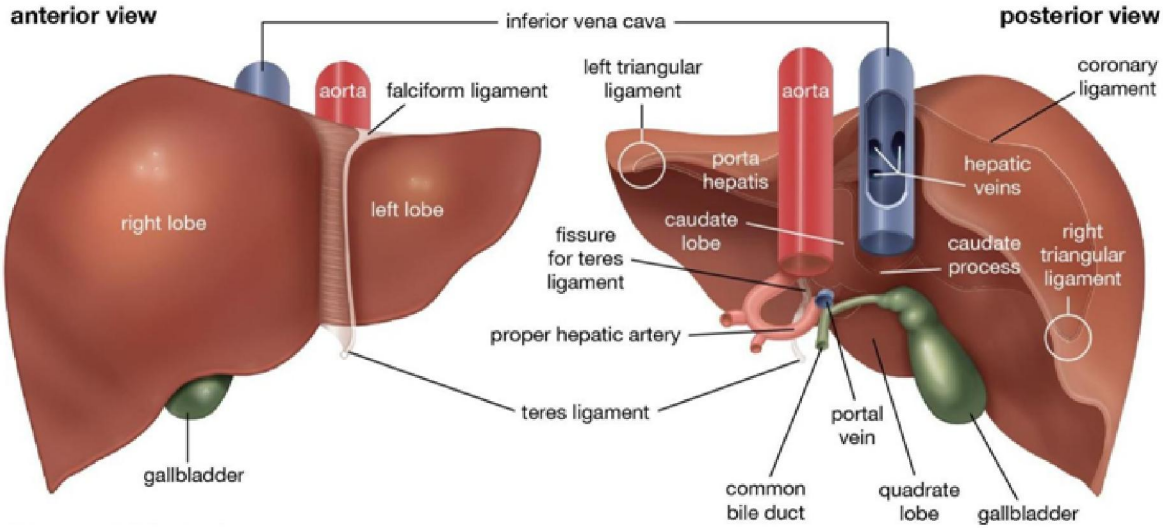
three plants, detailing their pharmacognostic quality standards, pharmaceutical formulation development, and molecular mechanisms of action.



III. NEED FOR THE STUDY

Hepatotoxicity and drug-induced liver injury represent major challenges in contemporary clinical practice and pharmaceutical development. The rising incidence of toxic liver injury driven by chemical exposure, industrial pollutants, and over-the-counter medication overuse demands the development of safe, standardized, and clinically effective hepatoprotective formulations.

The clinical use of traditional botanical remedies is frequently limited by a lack of rigorous scientific validation, molecular standardization, and consistent quality parameters in the raw drug market. Adulteration, species substitution, and variability in bioactive marker concentrations present significant obstacles to the integration of herbal medicines into modern healthcare.

There is a clear need to establish standardized quality control profiles for important hepatoprotective herbs. While traditional texts document the therapeutic benefits of the herbs are limited comparative studies that evaluate their quality standards, formulation parameters, and pharmacodynamics in a unified research framework. Determining accurate physicochemical profiles such as moisture limits, ash values, and extractive indices is essential to prevent the substitution of genuine herbs with low-grade or exhausted raw materials. Furthermore, developing standardized pharmaceutical formulations, such as oral suspensions, is necessary to improve bioavailability and ensure precise, uniform dosing in pre-clinical studies.

At the molecular level, the pathways mediating natural hepatoprotective action require further investigation. While the general antioxidant properties of these plants are recognized, their precise regulatory roles in pro- inflammatory cascades, fibrotic gene expression, and apoptotic pathways have not been fully elucidated. It is essential to investigate how these botanical extracts modulate specific cellular targets, such as the down-regulation of TNF-Alpha

The main reasons of the study are to prepare a review data set of numerous plant extracts for purpose of acting as info for the research on Liver and its pathological conditions. This study focuses on enlighten on the concept of use of herbal medicines in liver toxicity or disorders as its benefits and safety index are better than some synthetic alternatives. The study helps and imposes attention on the available ayurvedic counterparts and traditional formulations which can be utilized and to study their effect by analysis of different types and prepare a data set.

The limitations of In-vivo and In vitro studies due to study being UG level we have conducted Anti-microbial studies due to similarity in liver metabolism and microbes' mechanism of metabolism.

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IV. MOLECULAR PATHOGENESIS, ETIOLOGICAL FACTORS, AND THERAPEUTIC INTERVENTIONS IN HEPATIC DISEASE

4.1 How Liver Cells Interplay

The progression of liver disease from a simple injury to severe scarring (fibrosis) or liver cancer (Hepatocellular Carcinoma) isn't caused by just one cell type. Instead, it is driven by constant communication between three primary cells in the liver: Hepatocytes, Hepatic Stellate Cells (hscs), and Kupffer Cells.

Under normal conditions, these cells work together to keep the liver running smoothly. However, when the liver is injured, damaged hepatocytes release stress signals and reactive oxygen species (ROS). This alerts the Kupffer cells (the liver's resident immune cells), which start pumping out inflammatory signals. This ongoing cross-talk creates a loop that activates the normally quiet Hepatic Stellate Cells, turning them into aggressive, scar-forming cells that drive liver fibrosis.

4.2 Core Signaling Pathways Driving Damage

When the liver is under constant stress, specific internal chemical pathways are switched on. These pathways control how the liver responds to damage and direct the transition from simple inflammation to permanent tissue scarring.

• TGF-β Signaling (The Fibrosis Driver): Transforming Growth Factor-beta is the primary cytokine responsible for liver scarring. When tissue damage occurs, binds to receptors on Hepatic Stellate Cells. This triggers an internal chain reaction via proteins .Once activated, this complex travels to the cell nucleus and turns on the production of Type I and Type III collagen, physically scarring the liver tissue.

• NF-κb Pathway (The Inflammation and Cancer Link): The pathway acts as a bridge connecting long- term inflammation to cancer development. Chronic liver stress keeps this pathway continuously active. Free moves into the nucleus, keeping inflammatory signals turned on and preventing damaged, mutated cells from dying. This environment allows abnormal cells to survive, multiply, and eventually turn into Hepatocellular Carcinoma (HCC).

• HIF-1α / MAPK (Oxidative Stress and Suffocation): When liver tissue lacks oxygen (hypoxia) due to

disease, a protein called HIF-1 is activated. At the same time, high levels of oxidative stress trigger the MAPK pathway. Together, these two networks alter how liver cells use energy, increase cellular stress, and force the growth of abnormal new blood vessels.

• PDGF (The Cell Multiplier): Platelet-Derived Growth Factor PDGF is a powerful growth signal. When the liver is structurally damaged, surrounding blood vessels release PDGF. This molecule acts like a green light for Hepatic Stellate Cells, causing them to multiply rapidly and migrate directly to the injured areas to lay down more scar tissue.

• NRF2 (The Body's Antioxidant Défense): NRF2 is the master regulator that protects the liver from oxidative stress. When cells face chemical damage, NRF2 escapes its usual inhibitor KEAP1and moves into the nucleus. There, it turns on protective antioxidant enzymes like HO-1. Interestingly, this pathway shows noticeable differences between males and females, which helps explain why liver diseases often progress at different speeds depending on biological sex.

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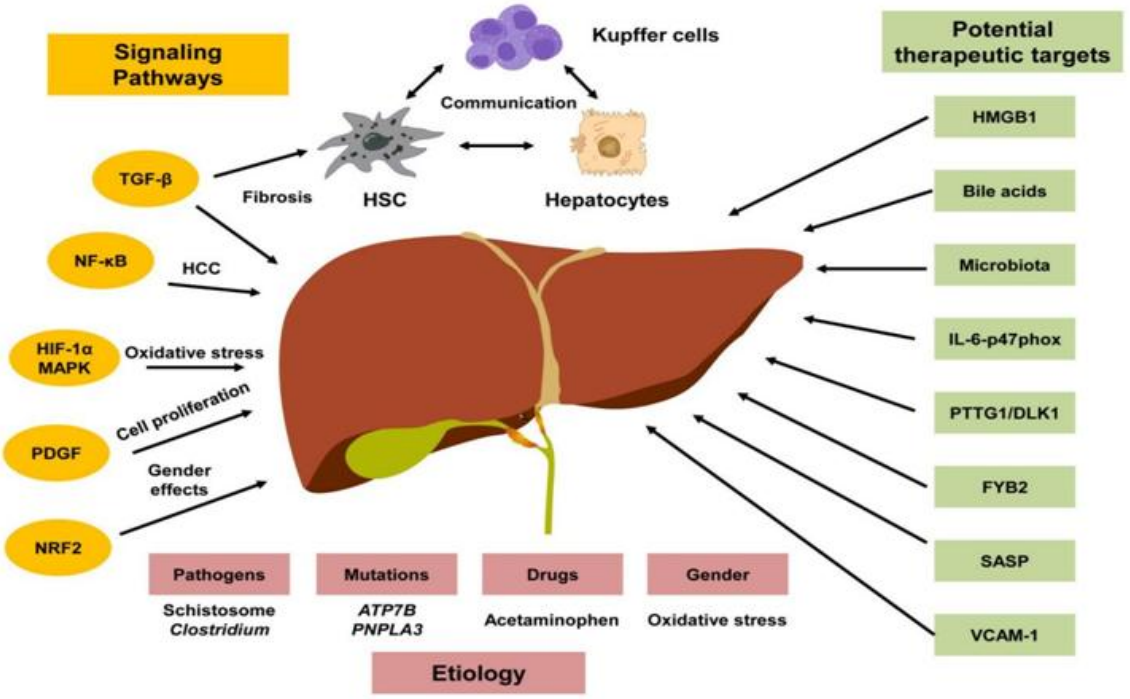

4.3 What Causes the Damage? (Etiology)

Liver damage generally stems from four primary root causes, each damaging the tissue in a different way:

1. Pathogens: Parasites like Schistosoma lay eggs directly in the liver's blood supply, causing severe local inflammation and high blood pressure in the portal vein. On the other hand, bacterial infections like Clostridium release toxins that damage the gut wall. These toxins travel straight to the liver via the bloodstream, triggering widespread immune inflammation.

2. Genetic Mutations: * $ATP7B$ Mutation: This mutation stops the liver from getting rid of excess copper through bile. The copper builds up to toxic levels, causing Wilson's Disease.

o PNPLA3 Mutation: A specific variation in this gene stops the body from breaking down fats properly, leading to fat buildup in liver cells even without alcohol use.

V. AIM

The primary aim of this investigation is to provide a comprehensive evaluation of the medicinal properties and hepatoprotective mechanisms exerted by various bioactive plant extracts. Moving beyond crude screening, this study seeks to delineate how these botanical agents intervene in the pathophysiological cascades governing liver- associated conditions. Specifically, the research focuses on quantifying the capacity of these natural compounds to mitigate oxidative stress, stabilize hepatocellular membranes, and suppress pro-inflammatory signaling pathways. Ultimately, this work intends to establish a scientifically rigorous baseline for the therapeutic utility of these extracts as viable, multi-target candidates in the management of acute and chronic hepatic disorders. The aim is to assess the inhibition and bactericidal action of the plant extracts in the microbes which is similar to liver conditions

VI. OBJECTIVES

To achieve the stated aim, the study was organized into the following specific research objectives:

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1. To determine the Minimum Inhibitory Concentration (MIC) and Minimum Bactericidal Concentration (MBC) of selected plant extracts against bacterial strains associated with liver infections and conditions affecting hepatic metabolic pathways.
2. To evaluate the antibacterial activity of selected plant extracts against various Gram-positive and Gram- negative bacterial strains using standard microbiological assays.
3. To assess the hepatoprotective activity of selected plant extracts using appropriate in vitro experimental models of liver injury.
4. To develop and analyse a dataset summarizing the effects of various plant extracts on liver function, hepatoprotection, and related pathophysiological conditions such as hepatic inflammation, oxidative stress, fibrosis, and drug-induced liver injury.

VII. REVIEW OF LITERATURE

• The exploration of medicinal plants as source materials for hepatoprotective drug discovery has generated a substantial volume of scientific literature. Traditional medicinal systems, notably Ayurveda and Traditional Chinese Medicine, contain records identifying specific herbs with "liver-protecting" or Kamala-destroying properties. Over the past three decades, pre-clinical and clinical studies have focused on standardizing these traditional claims through analytical, biochemical, and molecular methodologies.
• Comparative studies confirm that environmental factors, seasonal variations, cultivation altitudes, and soil organic compositions significantly influence the yield and quality of these bioactive markers. Therefore, implementing robust physicochemical evaluations, quality-driven extraction protocols, and bio-guided formulation development is essential to ensure the therapeutic efficacy and clinical reproducibility of herbal medicines.

Sr. No.	Plant Extract (Family)	Active Phytoconstituents	Pathology / Condition Studied	Key Findings & Mechanisms	References
1	*Silybum marianum* (Asteraceae)	Silymarin (a mix of flavanolignans: silybin, isosilybin, silydianin, silychristin)	Cirrhosis, hepatitis, fatty liver infiltration, and toxic liver injury (ethanol, paracetamol, CCl4)	Serves as a standard hepatoprotective agent; stabilizes hepatocyte membranes, regulates permeability, neutralizes free radicals, and stimulates hepatic regeneration.	**Negi et al. (2008); Madrigal-Santillán (2014)**
2	*Andrographis paniculata* (Acanthaceae)	Andrographolide, neoandrographolide, andrograpanin	CCl4induced hepatic toxicity, cellular necrosis, and fatty changes	Significantly restored elevated liver enzymes (ALT, AST by 75% and 14.5% respectively); decreased malondialdehyde (MDA) levels by 40% and boosted tissue antioxidants (GSH, CAT, GPX).	**Venmathi Maran et al. (2022)**
				Inhibits lipid peroxidation, exerts robust anti-	

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3	Curcuma longa (Zingiberaceae)	Curcumin, demethoxycurcumin	Oxidative liver damage, acute and chronic toxic liver injuries	inflammatory effects, and protects hepatocytes from necrosis and cellular hypertrophy.	Mittal (2024); Ghosh et al. (2011)
4	Artemisia absinthium (Asteraceae)	Sesquiterpene lactones, flavonoids, phenolic acids, tannins	CCl4induced and immunologically induced hepatic damage	Prevented increases in serum ALT and AST, reduced lipid peroxidation in liver tissue, and restored the natural defense enzymes SOD and GPX.	Ali et al. (2017)
5	Bauhinia purpurea (Leguminosae)	Phenolic compounds, flavonoids, quercetin	Paracetamol (PCM / Acetaminophen) induced hepatotoxicity	Restored paracetamol-altered serum levels of ALT, AST, and ALP; significantly reduced histopathological changes including necrosis, inflammation, and hemorrhage.	Venmathi Maran et al. (2022)
6	Commelina nudiflora (Commelinaceae)	Flavonoids, rosmarinic acid, phenols	CCl4induced chronic liver injuries and tissue degeneration	Dramatically reduced AST and ALT (by 63% and 40%); suppressed proinflammatory cytokines TNF- alpha, IL-6,PGE-2$and repaired damaged cell structures.	Venmathi Maran et al. (2022)
7	Glycyrrhiza glabra / uralensis (Fabaceae)	Glycyrrhizin (triterpenoid saponin)	Viral hepatitis, cellular transport impairment	Modifies the intracellular transport and surface integrity of hepatocytes; suppresses viral surface antigen secretions (such as HBsAg).	Ali et al. (2017)
8	Astragalus membranaceus	Astragaloside fractions, crude polysaccharides	Chronically injured liver and progressive	Exerts a powerful anti-fibrosis effect in chronically injured tissues by blocking the Transforming Growth Factor-beta TGF-beta	Ali et al. (2017)

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	(Fabaceae)		liver fibrosis	signalling pathways.	

Sr.No	Reference	Associated Pathology / Target Condition	Core Phytochemical Profiles & Model Mechanisms	Key Research Findings
1	Mancak (2023); Ghosh et al. (2011)	Nonalcoholic Steatohepatitis (NASH) / MAFLD	Phytochemical Targets: Standardized flavanolignans, volatile terpenes, and phenolic fractions. Mechanisms: Downregulates inflammatory nuclear factor kappa B ,NF-kappa expression.	Markedly reduces serum AST, total cholesterol, and baseline hepatic steatosis scores while suppressing proinflammatory cytokines IL-1-beta,IL-6, IL-8.
2	Ali et al. (2017); Ghosh et al. (2011)	Toxic Liver Injury & Mushroom Poisoning	Phytochemical Targets: Silymarin isomers, andrographolides. Mechanisms: Normalizes cytochrome P450 profiles CYP1A2,CYP2E1 and stabilizes hepatocyte outer membranes.	Attenuates severe rifampicin and carbon tetrachloride CCl4 induced toxic necrosis; significantly halts lipid peroxidation.
3	Mancak (2023)	Non-Alcoholic Fatty Liver Disease (NAFLD)	Phytochemical Targets: Lipophilic polyphenols, curcuminoids. Mechanisms: Direct enzymatic inhibition of Cyclooxygenase-2 COX-2 and Lipoxygenase pathways.	Demonstrates significant reduction in intrahepatic lipid accumulation and mitigates early-stage inflammatory tissue responses.
4	Ghosh et al. (2011); Ali et al. (2017)	Oxidative Hepatotoxicity & Endotoxemia	Phytochemical Targets: Flavonoids (e.g., Quercetin), sesquiterpene lactones. Mechanisms: Upregulates the Nuclear Factor Erythroid 2-related Factor 2 (Nrf2 gene pathway.	Boosts intrinsic antioxidant enzyme levels including Superoxide Dismutase SOD, Catalase, and Glutathione Peroxidase GPx,suppresses lipopolysaccharide LPSinduced tissue damage.
5	Ali et al. (2017)	Hepatitis B & Intracellular Hepatocyte Injury	Phytochemical Targets: Triterpenoid saponins (e.g., Glycyrrhizin). Mechanisms: Modulates intracellular transport mechanisms and cell surface	Suppresses active viral replication cycles and actively inhibits the secretion of Hepatitis B surface Antigen HBsAg from infected host cells.

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			viral binding.	
6	Ali et al. (2017)	Chemically-Induced Acute Liver Injury	Phytochemical Targets: Phenolic glycosides, rosmarinic acid. Mechanisms: Cellular membrane sealing and radical scavenging.	Restores elevated serum biomarkers of hepatic damage specifically Aspartate Aminotransferase AST, Alanine Aminotransferase ALT, Serum Alkaline Phosphatase SALP, and total bilirubin back to baseline levels.
7	Ali et al. (2017)	Chronic Liver Injury & Fibrosis	Phytochemical Targets: Astragalosides, specialized crude polysaccharides. Mechanisms: Interruption of the Transforming Growth Factor-beta/Smad TGF-beta/intracellular signalling loop.	Exerts robust anti- fibrotic activity in chronically injured parenchymal tissue, arresting the transition of hepatic stellate cells into collagen-secreting myofibroblasts.
8	Ali et al. (2017)	Ethanol-Induced Steatosis& Metabolic Death	Phytochemical Targets: Alkaloids, organic phenolic acids. Mechanisms: Suppression of genetic expressions regulating abnormal fatty acid biosynthesis.	Shields fragile hepatocyte structures fromalcohol- and lipopolysaccharide- mediated apoptosis.
9	Ali et al. (2017)	Alcoholic Liver Stress	Phytochemical Targets: Antioxidative botanical fractions. Mechanisms: Direct restoration of endogenous cellular peptide pools.	Replenishes critical intracellular stores of reduced glutathione superoxide dismutase and glutathione reductase depleted by chronic ethanol exposure.

VIII. ROLE, MECHANISMS AND CLASSIFICATIONS

Table 7.1: Cellular and Molecular Mechanisms Involved in Hepatotoxicity

S. N.	Mechanism	Description	Key features	References
1	Oxidative Stress	Imbalance between reactive oxygen species (ROS) production and antioxidant defenses.	An overabundance of reactive oxygen species (ROS) results in the oxidation of lipids, damage to DNA, and oxidation of proteins, which in turn leads to cell damage.	Pizzino, G., Irrera, N. [35]
2	Mitochondrial Dysfunction	Impairment of mitochondrial function and energy production.	Decreased ATP production, increased ROS generation, and initiation of apoptosis or necrosis.	Chen, X., Ji, Y. [36]
3	Inflammation	Activation of inflammatory pathways and immune	Release of pro-inflammatory cytokines, activation of Kupffer cells, and the influx	Cekici, A., Kantarci, A. [37]

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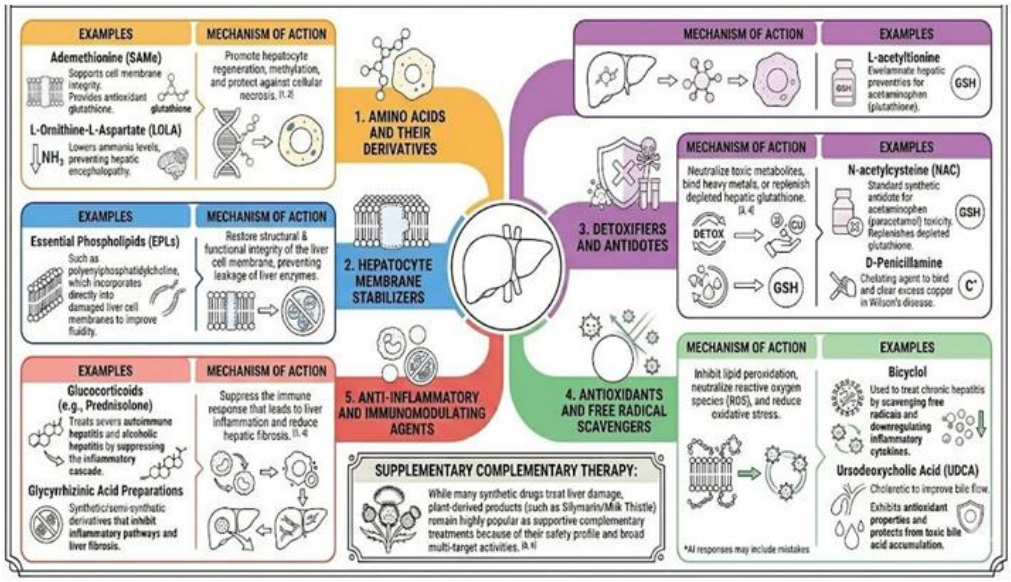
		responses.	of leukocytes.	
4	Endoplasmic Reticulum (ER) Stress	Endoplasmic reticulum-mediated disruption of protein folding.	Unfolded protein response (UPR) activation, leading to apoptosis if left unresolved.	Iurlaro, R., Muñoz-Pinedo, C. [38]
5	Cell Death Pathways	Activation of apoptotic or necrotic pathways.	Involvement of caspases in apoptosis; loss of membrane integrity and release of intracellular contents in necrosis.	Guha, L., Singh, N., Kumar, H. [39]
6	Lipid Accumulation (Steatosis)	Excessive accumulation of lipids in hepatocytes.	Disruption of lipid metabolism, leading to fatty liver and progression to steatohepatitis.	Zhang, X.,Qin, J. [40]
7	Genotoxicity	Damage to genetic material (DNA) in liver cells.	Mutations, chromosomal aberrations, and activation of DNA repair mechanisms.	More, S., Bampidis, V. [41]
8	Disruption of Calcium	Imbalance in intracellular calcium levels.	Activation of calcium-dependent enzymes, mitochondrial permeability transition, and cell death.	Chebib, F., Sussman, C. [42]
9	Impaired Autophagy	Dysfunction in the degradation and recycling of cellular components.	Accumulation of damaged organelles and proteins, contributing to cell injury.	Hailfinger, S., Schulze- Osthoff, K. [43]
10	Fibrogenesis	Excessive accumulation of extracellular matrix proteins causing fibrosis.	Hepatic stellate cells are activated, leading to an increase in collagen production and the creation of scar tissue.	Lee, U., Friedman, S. [44]

7.2 Classification Systems

• Synthetic Drugs as Hepatoprotective agents



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1. Amino Acids and Their Derivatives

This category focuses on restoring metabolic intermediates essential for cellular regeneration, transmethylation pathways, and protection against irreversible cellular necrosis.

Ademethionine (S-Adenosyl-L-Methionine / SAMe)

• Mechanism of Action: SAMe serves as a critical physiological coenzyme and a primary methyl donor in transmethylation reactions, which are fundamental for synthesizing phospholipids that maintain hepatocyte membrane fluidity and structural integrity. Furthermore, it enters the transsulfuration pathway, acting as a direct biochemical precursor to cysteine, a rate-limiting substrate for the synthesis of the major endogenous antioxidant, glutathione (GSH). By supplementing GSH pools, SAMe mitigates mitochondrial oxidative damage and prevents necrotic cell death cascades.

• Clinical Implications: Utilized in treating intrahepatic cholestasis (associated with pregnancy or chronic liver disease) and alcoholic liver disease, where endogenous SAMe synthetase is profoundly downregulated.

L-Ornithine-L-Aspartate (LOLA)

• Mechanism of Action: LOLA acts via substrate activation of two distinct metabolic detoxification pathways targeting neurotoxic ammonia. First, L-ornithine serves as a vital intermediate in the periportal urea cycle, stimulating the enzyme carbamoyl phosphate synthetase to convert free ammonia into urea. Second, both L-ornithine and L-aspartate are taken up by perivenous hepatocytes and transformed into glutamate, a direct precursor required by glutamine synthetase to trap ammonia into non-toxic glutamine.

• Clinical Implications: Primarily indicated for the prevention and management of hepatic encephalopathy in acute and chronic liver failure, directly lowering systemic and cerebral hyperammonemia.

2. Hepatocyte Membrane Stabilizers

Membrane stabilizers target the structural disruption of the lipid bilayer caused by lipophilic toxins, alcohol, or viral proteins. By restoring structural integrity, they prevent the intracellular leakage of critical metabolic enzymes (such as ALT and AST).

Essential Phospholipids (EPLs)

• Mechanism of Action: EPLs consist predominantly of highly purified polyenylphosphatidylcholine (PPC) rich in polyunsaturated fatty acids (such as linoleic acid). In the presence of membrane damage, these exogenous phospholipids readily substitute into the damaged, rigidified hepatocyte and mitochondrial membranes. This physical integration restores optimal membrane fluidity, normalizes phospholipid- dependent transport proteins, and repairs transmembrane signaling systems.

• Clinical Implications: Administered as supportive therapy in non-alcoholic fatty liver disease (NAFLD/NASH), toxic liver injuries, and chronic hepatitis to reduce enzyme leakage and slow cell degradation.

3. Detoxifiers and Antidotes

These agents directly intervene during acute toxification events by either chemically neutralizing toxic molecular species, chelating heavy metals, or forcefully driving endogenous detoxification pathways.

N-Acetylcysteine (NAC)

• Mechanism of Action: NAC is the definitive antidote for paracetamol (acetaminophen) overdose. Acetaminophen metabolism via cytochrome P450 enzymes yields a highly reactive, toxic intermediate known as N-acetyl-p-benzoquinone imine (NAPQI), which rapidly depletes hepatic glutathione stores and causes widespread mitochondrial necrosis. NAC delivers an immediate supply of exogenous cysteine, driving rapid de novo hepatic glutathione synthesis. Additionally, NAC can directly bind and reduce NAPQI into a non-toxic conjugate.

• Clinical Implications: Standard-of-care protocol for acute paracetamol-induced hepatotoxicity; also increasingly utilized for non-paracetamol acute liver failure due to its positive effects on microvascular perfusion.

D-Penicillamine

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• Mechanism of Action: D-Penicillamine is a synthetic sulfhydryl-containing compound that acts as an extracellular heavy metal chelator. It directly binds free copper ions (Cu^{2+}) in the serum and tissue spaces to form a stable, soluble, ring-structured coordination complex. This chelation prevents copper from participating in Fenton reactions that generate destructive hydroxyl free radicals within liver tissue, facilitating the safe renal excretion of the excess metal.

• Clinical Implications: Indicated as primary maintenance therapy for Wilson's disease (an inherited autosomal recessive disorder of copper transport) to halt progressive hepatic cirrhosis and neurological degradation.

4. Antioxidants and Free Radical Scavengers

Oxidative stress is a ubiquitous driver of liver damage. This group halts the radical-driven propagation of lipid peroxidation and protects cellular macromolecular structures.

Bicyclol

• Mechanism of Action: Bicyclol is a synthetic anti-hepatitis drug that functions as a potent free radical scavenger. It intercepts and neutralizes reactive oxygen species (ROS) such as superoxide anions ($\cdot O_2^-$) and hydroxyl radicals ($\cdot OH$), thereby preventing the peroxidation of membrane lipids. Concurrently, it downregulates the transcription and release of pro-inflammatory cytokines, specifically tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β), while preserving mitochondrial function and preventing cytochrome c release.

• Clinical Implications: Clinically utilized to treat chronic viral hepatitis B and C, as well as drug-induced liver injury (DILI), resulting in a rapid, sustained decrease in serum transaminases.

Ursodeoxycholic Acid (UDCA)

• Mechanism of Action: UDCA is a hydrophilic, non-toxic, naturally occurring tertiary bile acid that, when administered synthetically, displaces toxic, hydrophobic bile acids (such as chenodeoxycholic acid) from the circulating bile acid pool. Mechanistically, UDCA exerts cytoprotective effects by inhibiting hydrophobic-bile-acid-induced mitochondrial membrane disruption and subsequent apoptosis. Furthermore, it stimulates hepatocellular and cholangiocellular secretion of bile, reduces the immunogenic expression of HLA Class I molecules on hepatocytes, and downregulates intrahepatic oxidative stress markers.

• Clinical Implications: First-line therapeutic choice for chronic cholestatic conditions, including Primary Biliary Cholangitis (PBC) and Primary Sclerosing Cholangitis (PSC).

5. Anti-Inflammatory and Immunomodulating Agents

Chronic inflammation triggers the activation of hepatic stellate cells (HSCs), leading to the excessive deposition of extracellular matrix components and resulting in liver fibrosis. These agents suppress overactive immune responses to prevent permanent architectural restructuring of the liver.

Glucocorticoids (e.g., Prednisolone)

• Mechanism of Action: Glucocorticoids bind to intracellular glucocorticoid receptors, translocating to the nucleus to alter gene transcription. They upregulate anti-inflammatory proteins (such as lipocortin-1) and downregulate pro-inflammatory transcription factors, primarily nuclear factor kappa B (NF-B) and activator protein 1 (AP-1). This leads to a massive reduction in the production of leukotrienes, prostaglandins, and systemic inflammatory cytokines, arresting lymphocyte-mediated cytotoxicity against hepatocytes.

• Clinical Implications: Highly effective for inducing remission in severe cases of Autoimmune Hepatitis (AIH) and managing severe acute Alcoholic Hepatitis with a high Maddrey's discriminant function score.

Glycyrrhizinic Acid Preparations

• Mechanism of Action: These synthetic and semi-synthetic derivatives (such as diammonium glycyrrhizinate) act as selective inhibitors of the enzyme 11-beta-hydroxysteroid dehydrogenase, indirectly reinforcing endogenous cortisol levels. At the molecular level, glycyrrhizinic acid inhibits the high-mobility group box 1 (HMGB1) protein, blocking its interaction with Toll-like receptors (TLRs) and downregulating the NF-B pathway. This dual action suppresses

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downstream inflammatory signaling cascades and directly inhibits the proliferation and activation of hepatic stellate cells, significantly

Sr.no	Natural Source / Plant Family	Major Constituents	Mechanism of Action
2	Glycyrrhizin *(Family: Leguminosae)*	Glycyrrhetinic acid, beta-sitosterol, hydroxycoumarins, and flavonoids.	Increases antioxidant defense within hepatic cells and acts as an anti-inflammatory agent ; downregulates pro-inflammatory cytokine genes at the genomic level.
3	Andrographolide *(Family: Acanthaceae)*	Andrographolide, neoandrographolide, 14-deoxy-11- dehydroandrographolide, 14-deoxy- 11-oxoandrographolide, deoxy-andrographolide, and andrographine.	Inhibits inflammation, angiogenesis, and fibrosis ; modulates genes involved in apoptosis and cell survival to prevent hepatocyte cell death.
4	Kutkoside and Picroside *(Source: Picrorhiza)*	Picroside 1, 2, and 3, kurkoside, apocynin, drosin, cucurbitacin glycoside, and iridoid glycosides.	Exhibits membrane-stabilizing, hypolipidemic, and antioxidant properties ; activates genetic pathways involved in liver regeneration, healing, and repair.
5	Curcumin *(Family: Zingiberaceae)*	Curcumin, demethoxycurcumin, and bisdemethoxycurcumin.	Acts as a liver-protective and detoxifying agent ; provides powerful antioxidant activity by driving phase 2 detoxifying/antioxidant enzymes (specifically upregulating Nrf2 and downstream HO-1 genes).
6	Phyllanthin & Hypophyllanthin *(Family: Euphorbiaceae)*	Alkaloids, astragalin, brevifolin, ellagitannins, amariin, repandusinic acid, phyllanthusiin D, gallocatechins, geraniin, hypophyllanthin, lignans, nirutin, phyllanthin, and phyllanthenol.	Provides multi-target liver protection ; modulates specific detoxification genes to fundamentally enhance hepatic clearance pathways.
7	Berberine *(Family: Berberidaceae)*	Berberine, oxyberberine, berbamine, aromoline, karachine, and oxycanthine.	Suppresses intrahepatic oxidative stress and attenuates apoptosis ; regulates gene expressions connected to lipid metabolism and chronic inflammatory cascades.
8	Embelin *(Family: Myrsinaceae)*	Embelin, christembine, quercitol, and resin.	Operates through pathways that directly scavenge free radicals and halt the destructive peroxidation of membrane lipids.
9	Resveratrol *(Source: Vitis labrusca / Grapes)*	*Trans*-3,5,4'-trihydroxystilbene.	Alters the operational activity of nuclear transcription factors Nrf2 and NF-κB ; reduces the downstream genetic expression of inflammatory inos while upregulating protective HO-1.

Sr.no Natural Source / Plant Family Major Constituents Mechanism of Action

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VIII. MATERIALS AND METHODS

8.1. Plant material In the investigation, the whole plant of Guava (Psidium guajava), Sage (Salvia officinalis), Rhamnus (Ziziphusspina Christi), Mulberry (Morusalba L.), and olive (Olea europaea L) leaves were collected from field market of Egypt. The plants are air dried in an oven at 40 C for 48 h. Two hundred and fifty grams of dried powdered plant sample was extracted by one liter of ethanol 70% at 30 C for 48 h and filtering through Whatman No. 4 filter paper. The plants were extracted by rotatory evaporation at 50 C till com plete dryness occurs. The total extract was dissolved in water in a concentration of 500 mg/ml and stored at 20 C for further use.

8.2. Test organisms Six types of microorganisms were investigated; Gram positive (S. aureus and B. cereus), Gram negative (E. coli, Pasteurella multo cida, Salmonella Entritidis) and Mollicutes (Mycoplasma gallisep ticum). All organisms obtained and well identified onto Microbiology Department, Faculty of Veterinary Medicine, Cairo University, Giza, Egypt.

8.3. Antimicrobial activity The antimicrobial potentiality of the above-mentioned plants ethanolic extracts were determined by standard agar disc diffusion technique and minimal inhibitory concentration (MIC) in accordance with the Clinical and Laboratory Standards Institute (CLSI, 2015) against the tested microorganisms. Mycoplasma gallisep ticum was determined in accordance with (Hannan, 2000).

8.3.1. Disc diffusion method Pure colonies of each microorganism were suspended in sterile saline until a turbidity match McFarland tube number 0.5 (1.5 108 CFU/ ml). A loopful from each adjusted organisms were swabbed onto Muller Hinton agar Sterile paper discs (6 mm in diameter) were impregnated with 100 ml of each 10%, 25%, 50%, 75% and 100% diluted extracts then dried at 100 C for two hours in hot air oven and were dispensed onto the surface of the inoculated agar plate. The plates were then incubated according to growth requirement of each organism. Each sample was tested in triplicates and antibacterial activity was evaluated by measuring and recorded the zones of inhibition in mm (including the 6 mm disk). A parallel analysis study with commercial antimicrobial agents included Amoxicillin-Clavulanic (10 lg), Ampicillin (10 lg), Vancomycin (30 lg), Lincomycin (30 lg), Gentamicin (10 lg), Streptomycin (10 lg) and Tetracycline (30 lg) was conducted in order to compare their antimicrobial potency with the plant extracts.

8.3.2. Minimal inhibitory concentration (MIC) In dilution technique, two-fold serial dilutions of the extracts were prepared in concentrations ranging from 625to 10,000 g/mI. From each dilution of each extract, one milliliter of was mixed with 9 ml of Muller Hinton Agar. Ten microliters of each standardized broth cultures (1.5 108 CFU/ml) was cultivated on the surface of the plates containing various concentrations of the extracts. The plates were then incubated according to growth requirement of each organism and observed for any visible bacterial growth. MIC was the lowest concentration of extract that resulted in no visible growth on the surface of the agar.

8.3.3. Minimum bactericidal concentration (MBC) Blocks of agar plates showing no growth after MIC tests were inoculate to fresh nutrient broth (acting as the recovery medium) for the determination of the MBC. The broths were incubated according to growth requirement of each organism. The absence of turbidity in the recovery medium was evidence of bactericidal activity.

8.3.4. The microbes and strains used in this study shows similarity with the metabolism and pathways in liver and by comparing their effects by the plant extract we can estimate the activity of plants on hepatoprotective action against microbes of various strains.

8.4. The microbe's environment is inhibited by the plants thus useful in hepatic inflammation caused by microbes.

IX. COLLECTION AND AUTHENTICATION OF MATERIALS

• Acquisition and Processing: Fresh whole plants or leaves of Guava (Psidium guajava), Sage (Salvia officinalis), Rhamnus (Ziziphus spina-christi), Mulberry (Morus alba L.), and Olive (Olea europaea L.) Were gathered from agricultural field markets. The collected plant biomatrices were subjected to air- drying in a controlled laboratory oven

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at 400 C for 48 hours to remove residual moisture. Following dehydration, the samples were mechanically ground into a fine powder.

• Extraction Protocol: For each distinct botanical specimen, a mass of 250g of the dried, homogenized powder was macerated in 1L of 70% ethanol 1:4 ratio The extraction mixture was incubated at a constant temperature of 300 C for 48 hours.

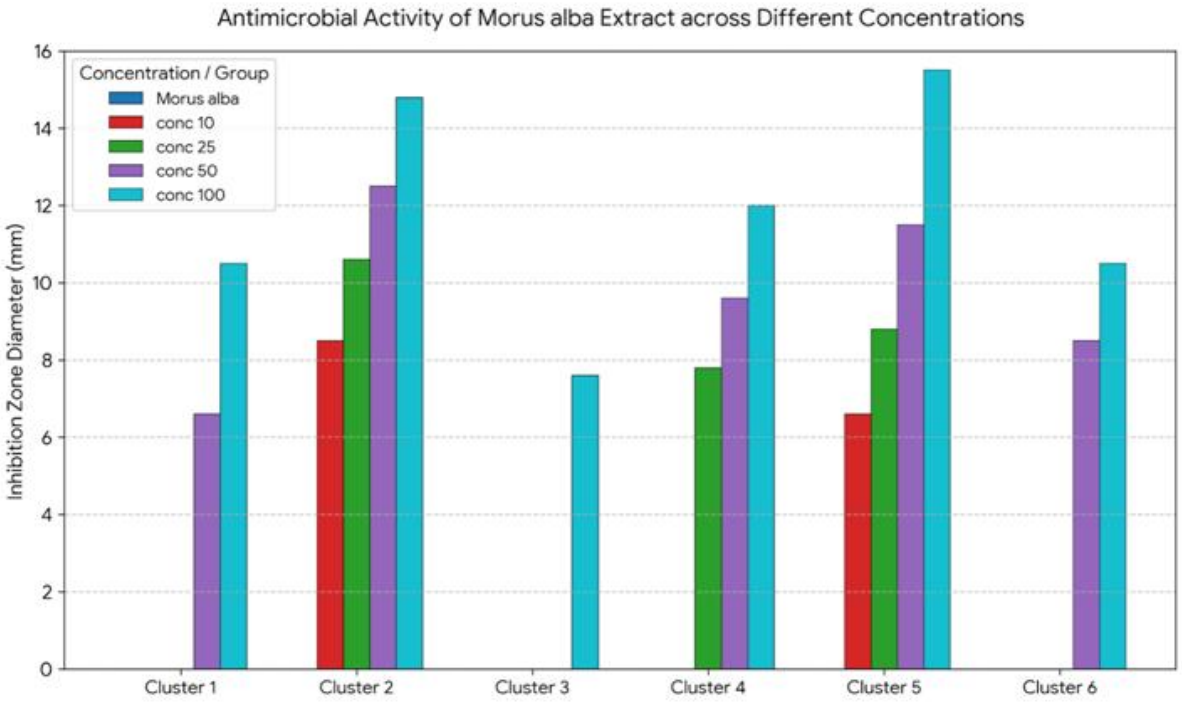
• Filtration and Concentration: The crude menstruum was filtered through Whatman No. 4 filter paper to eliminate particulate matter. The resulting clear filtrates were concentrated using a rotary evaporator at an elevated temperature of 500 C until complete dryness was achieved. The final sticky, dried crude extracts were reconstituted in sterile distilled water to establish a stock solution concentration of 500/ml and preserved at 200 C for subsequent bioassays.

Microorganism	P. guajava MIC / MBC	S. officinalis MIC / MBC	Z. spina-christi MIC / MBC	M. alba MIC / MBC	O. europaea MIC / MBC
S. aureus	1250 / 1250	5000 / 1250	625 / 625	—	625 / 625
B. cereus	—	625 / 625	625 / 625	2500 / 1250	5000 / 2500
E. coli	625 / 625	2500 / 2500	—	—	2500 / 1250
Salmonella Enteritidis	625 / 625	2500 / 1250	625 / 625	625 / 625	5000 / 2500
P. multocida	5000 / 2500	—	—	1250 / 1250	625 / 625
M. gallisepticum	1250 / 625	—	—	625 / 625	—

Values are expressed in µg/mL. '—' indicates no matching bioactivity was detected within evaluated boundaries.

C] Figures showing antimicrobial activity of plant extracts

• Anti-Microbial Activity of various plant extracts



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The medicinal plants evaluated in this study exhibit significant antimicrobial efficacy that frequently surpasses the performance of traditional commercial antimicrobial agents, offering a promising upstream mechanism for hepatoprotection. Bacterial infections and their systemic endotoxins are deeply implicated in triggering severe hepatic inflammatory cascades, structural micro-abscesses, and metabolic disruptions. When pathogens translocate via the gut-liver axis, their cell wall components such as the lipopolysaccharides of gram-negative bacteria induce portal vein endotoxemia, activating hepatic macrophages and leading to acute liver parenchyma injury or necrosis. By targeting and neutralizing these specific bacterial strains before they cause systemic damage, the plant extracts act as potent structural protectants, mitigating inflammation and shielding the liver from infectious insults.

Using the disk diffusion technique, distinct, plant-specific profiles emerge against pathogens associated with hepatic and systemic distress. The extract of salvia officinalis and psidium guajava demonstrated a powerful ability to inhibit the growth of staphylococcus aureus, which is known to cause severe toxic shock-mediated hepatic damage. Concurrently, olea europaea and morus alba exhibited robust antibacterial potency against bacillus cereus, neutralizing a spore-former whose emetic toxins can induce severe mitochondrial distress within hepatocytes. To address the severe threat of enteric-driven liver necrosis, salvia officinalis and olea europaea successfully arrested the proliferation of escherichia coli, while olea europaea and salvia officinalis extracts inhibited the growth of salmonella enteritidis. Furthermore, pasteurella multocida plates showed clear zones of inhibition when treated with psidium guajava and morus alba. Strikingly, all extracts failed to show antibacterial potency against the highly resistant mycoplasma gallisepticum except for psidium guajava, which served as the singular effective agent against this strain. The detailed correlation between the inhibition zones, extract concentrations, and microbial types is cataloged in and demonstrated visually in figure In comparison, standard commercial antimicrobials displayed highly volatile sensitivity and resistance profiles across the microbial panel, with lincomycin standing out as the only control agent possessing uniform lethal action against all evaluated organisms.

To quantify the precise bioactive thresholds required to prevent pathogen-mediated liver damage, minimum inhibitory concentration (mic) and minimum bactericidal concentration (mbc) values were mapped out, ranging strictly from 625to5000mu g/ml across the bacterial panel Against s. Aureus, the mic values were recorded at 1250mu g/ml for psidium guajava, 5000mu g/ml for salvia officinalis, 625 mu g/ml for ziziphus spina-christi, and 625mu g/ml for olea europaea l., while the lethal mbc values for the same pathogen followed at 1250, 1250, 625, and 625 mu g/ml in the exact same order, demonstrating a decisive bactericidal endpoint that limits downstream staphylococcal-induced focal liver necrosis. When evaluating the hepatotoxic agent b. Cereus, testing of salvia officinalis, ziziphus spina-christi, morus alba, and olea europaea l. Revealed mic values of 625, 625, 2500, and 5000mu g/ml, with corresponding mbc values tracking at 625, 625, 1250 and 2500 mu g/ml, respectively.

The quantitative evaluation further highlights the specialized potency of psidium guajava against a broad range of inflammatory threats, revealing tightly matched mic and mbc values against e. Coli 625, 625 mu g/ml
,salmonella enteritidis 625, 625 mu g/ml pasteurella multocida 5000, 2500 mu g/ml and mycoplasma gallisepticum (1250, 625 mu g/ml In comparison, salvia officinalis required slightly higher concentration limits to address gut-derived hepatic threats, yielding mic and mbc combinations of $2500, 2500 mu g/ml for
e. Coli and 2500, 1250 mu g/ml for salmonella enteritidis. Meanwhile, ziziphus spina-christi displayed narrow, highly potent bactericidal control against salmonella enteritidis, with matching mic and mbc values localized sharply at 625 mu g/ml Morus alba also demonstrated precise bactericidal efficacy against salmonella enteritidis, p. Multocida, and m. Gallisepticum, with a single, unified lethal mbc value of 625 mu g/ml across these strains. Finally, olea europaea l.

Successfully combated enteric endotoxin risks, producing mic and mbc benchmarks against e. Coli (2500, 1250 mu g/ml salmonella enteritidis (5000, 2500 mu g/ml and p. Multocida (625 mu g/ml)By effectively suppressing these pathogenic groups at reliable concentrations, the tested extracts prevent the cascades of systemic inflammation and macrophage over-activation that typically culminate in hepatic parenchyma injury, confirming that these botanical agents provide robust secondary hepatoprotective effects

11. CONCLUSION AND DISCUSSION

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The present study is expected to demonstrate that the selected plant extracts possess significant hepatoprotective and antibacterial properties. Liver damage is often associated with bacterial infections, inflammation, oxidative stress, and the accumulation of toxic metabolites that impair normal hepatic function. The bioactive phytochemicals present in the plant extracts, including flavonoids, phenolic compounds, tannins, alkaloids, and terpenoids, are anticipated to protect liver cells from injury and maintain their structural and functional integrity. By preventing cellular degeneration and reducing pathological changes within hepatic tissues, the extracts may contribute to the preservation of normal liver function and overall hepatic health.

The plant extracts are also expected to exhibit strong antibacterial activity against bacterial strains commonly associated with liver-related pathophysiological conditions. These microorganisms may contribute to hepatic injury through the production of toxins, induction of inflammatory responses, and promotion of systemic infections that ultimately affect liver function. The antimicrobial potential of the extracts is anticipated to be reflected through significant inhibition of bacterial growth and favourable Minimum Inhibitory Concentration (MIC) and Minimum Bactericidal Concentration (MBC) values. Such findings would indicate that the extracts are capable of both suppressing bacterial proliferation and eliminating pathogenic microorganisms at relatively low concentrations, thereby highlighting their potential as effective natural antimicrobial agents.

Furthermore, the study is expected to provide valuable insights through the utilization of microbial models that share metabolic characteristics and biochemical pathways similar to those involved in hepatic metabolism. The responses observed in these microorganisms may serve as preliminary indicators of how the plant extracts interact with metabolic systems relevant to liver function. The generated data will contribute to a better understanding of the mechanisms underlying the biological activities of the extracts and may support future investigations involving advanced cellular and animal models for hepatoprotective research.

Another anticipated outcome of the study is the reduction of systemic inflammation and immune-mediated liver injury. Bacterial infections often stimulate excessive activation of macrophages and the release of pro- inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin- 6 (IL-6), which can initiate a cascade of inflammatory events leading to hepatic parenchymal damage. The plant extracts are expected to modulate these inflammatory responses by suppressing excessive immune activation and reducing the production of inflammatory mediators. This anti-inflammatory effect may help prevent the progression of liver injury and minimize the development of various liver-associated disorders.

In addition, the antioxidant constituents present in the plant extracts are anticipated to play a crucial role in protecting the liver against oxidative stress-induced damage. Reactive oxygen species generated during infection and inflammation can cause lipid peroxidation, protein oxidation, and DNA damage within hepatocytes. By scavenging free radicals and enhancing endogenous antioxidant defense systems, the extracts may reduce oxidative injury and promote cellular recovery. This combined antibacterial, anti-inflammatory, and antioxidant activity is expected to contribute substantially to the overall hepatoprotective effects observed during the study. Overall, the study is expected to establish that the selected plant extracts possess significant therapeutic potential for the prevention and management of liver-associated diseases. Through effective inhibition of pathogenic bacterial growth, reduction of systemic inflammation, attenuation of oxidative stress, and protection of hepatic tissue, the extracts may help preserve liver function and reduce the severity of liver damage.

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