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Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease. International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

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A REVIEW ON HEPATIC TISSUES, CELLS: ANATOMY, PHYSIOLOGY OF LIVER AND TREATMENT OF HEPATIC DISEASE

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Keywords	***Abstract***
Liver, Hepatoprotective agents, Natural products, Liver function, Herbal treatment, Hepatic disorders. .	The development of the human liver starts during the third to fourth week of embryonic life. As the largest internal organ, the liver is structurally separated into 2 main (left and right) lobes, along with two smaller lobes (caudate and quadrate). Liver diseases are among the leading causes of death globally, affecting millions of people, including over Thirty (30) million in the United States alone. This vital organ acting a central role in numerous physiological functions such as metabolism, detoxification, immune defense, digestion, and vitamin storage. The liver maintains internal homeostasis by processing nutrients, synthesizing important proteins, and neutralizing toxins. Liver damage, whether caused by viral infections like hepatitis, genetic metabolic disorders, or chronic alcohol abuse, can severely disrupt these critical processes. Key functions include bile production, metabolism of fats, carbohydrates, and proteins, and the detoxification of xenobiotics. Hepatocytes constitute about 60–70% of the liver's cellular composition and are primarily responsible for the synthesis of plasma proteins, including albumin, acute-phase proteins and clotting factors. The remaining cells—stellate cells, Kupffer cell, and endothelial cells—play supportive and immune-regulatory roles. This article outlines the external and anatomical structure of the liver, discusses its functional physiology, and evaluates the hepatoprotective potential of herbal formulations in clinical settings. Notably, herbal preparations such as silymarin, glycyrrhizin, and Liv-52 have shown promising results in the managing the liver cirrhosis, hepatitis, and alcoholic liver disease.

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1. INTRODUCTION

The liver is the largest solid organ in the body, considering around 1.5 kg and situated in the upper right abdomen, where it is shielded by the rib cage. It plays a critical role in various physiological functions including metabolism, storage, detoxification, secretion, and immune regulation. Supplied by both the hepatic artery and portal vein, the liver receives nutrient-rich and oxygenated blood. Its structural unit, the liver lobule, is responsible for these essential functions.

As a key site for drug metabolism and immune signaling, the liver helps maintain homeostasis. It produces acute-phase proteins and cytokines that balance immune tolerance and defense. However, the liver is vulnerable to damage from toxins such as alcohol, drugs, viruses, and environmental pollutants. Conditions like cirrhosis, hepatitis, and fatty liver disease are increasingly common and often lead to major difficulties, including liver malignancy and organ failure.

Despite advancements in modern medicine, effective and safe hepatoprotective agents remain limited. Synthetic drugs used to treat liver diseases can themselves strain the already compromised liver. This underscores the demand for alternative treatments with fewer side effects. Traditional medicinal plants and phytoconstituents such as silymarin have shown promise in protecting and regenerating liver tissue.

Oxidative stress, driven by free radicals, is a major contributor to liver cell injury and inflammation. This can lead to necrosis and fibrosis, with inflammatory cytokines like TNF-α and IL-1β playing a central role. The ongoing global burden of liver disease—marked by high morbidity, mortality, and healthcare costs—highlights the urgent need for safer, more effective therapies rooted in both modern and traditional medicine.

- **Anatomy of the Liver:**

The largest internal organ in the human body is the liver, which is usually brown colour with a smooth outer surface. It constitutes about 2–3% of total body weight, weighing approximately 1800 grams in men and 1400 grams in women. Positioned in the right upper quadrant of the abdomen beneath the right hemidiaphragm, it is partly shielded by the ribs and surrounded by the Glisson's capsule— aside from the region that comes into contact with the diaphragm.

Externally, the liver is divided into right and left lobes by the falciform ligament. Its surface is described as diaphragmatic and visceral, with the diaphragmatic surface forming an irregular triangular shape. Anatomically, the superior border extends from the fifth left intercostal space to the right fifth rib, just below the right nipple.

The right lobe is larger and includes the caudate (Spigel's lobe) and quadrate lobes. The quadrate lobe lies between the gallbladder and umbilical fissure, while the caudate lobe is located near the hilum. According to Couinaud's classification, the liver is functionally divided into eight segments, each served by a portal pedicle comprising a branch of the portal vein, hepatic artery, and bile duct. Segments II, III, and IV belong to the left lobe, segments V to VIII to the right lobe, and segment I corresponds to the caudate lobe.

Table 1 (Sumadewi KT etal, 2023)

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Liver lobes	segmentation Location	Location
Left lobe	II and II Lateral IV	Lateral segment, Medial segment
Right lobe	V and VIII VI and VII	Anterior segment Posterior segment
Caudate lobe	I	On posterior liver

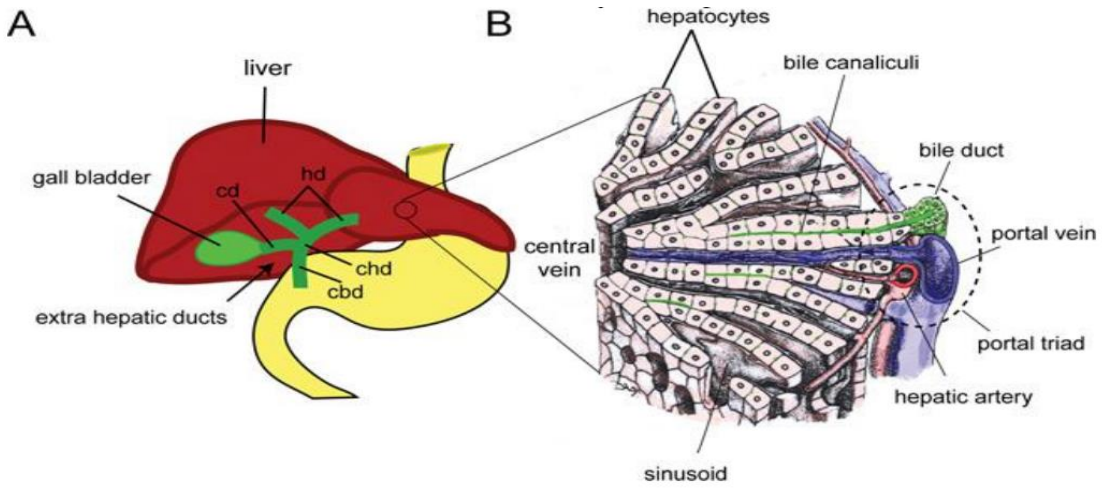

Fig. 1: Cellular architecture of the liver.

The liver is drained by the right, left, and middle hepatic veins, all of which empty into the inferior vena cava. The right hepatic vein separates the liver's right lobe into anterior and posterior segments, while the middle hepatic vein divides the right and left lobes. The left hepatic vein further separates the medial and lateral sections of the left lobe. The portal vein, which brings 80% of the liver's blood supply from the gastrointestinal tract and spleen, divides the liver into superior and inferior portions. The hepatic artery supplies the remaining 20% of oxygenated blood. The portal vein is formed by the union of the splenic and superior mesenteric veins, with the inferior mesenteric vein joining the splenic vein.

In liver resections, a left hepatectomy involves removal of segments II, III, and IV, while a right hepatectomy removes segments V to VIII. An extended right hepatectomy includes segments I or IV along with V–VIII, whereas an extended left hepatectomy may involve segments I, V, VIII, or combinations of II, III, and IV.

Anatomically, the liver is held in place by four peritoneal ligaments: falciform, coronary, and the left and right triangular ligaments. These suspensory structures stabilize the liver by connecting it to the diaphragm and anterior abdominal wall. The falciform ligament attaches to the liver's dome and separates the right and left lobes on the diaphragmatic surface. The ligamentum venosum, a remnant of the fetal ductus venosus, links the left portal vein to the left hepatic vein. The smaller omentum connects the liver to the lesser curvature of the stomach, attaching superiorly at the porta hepatis and

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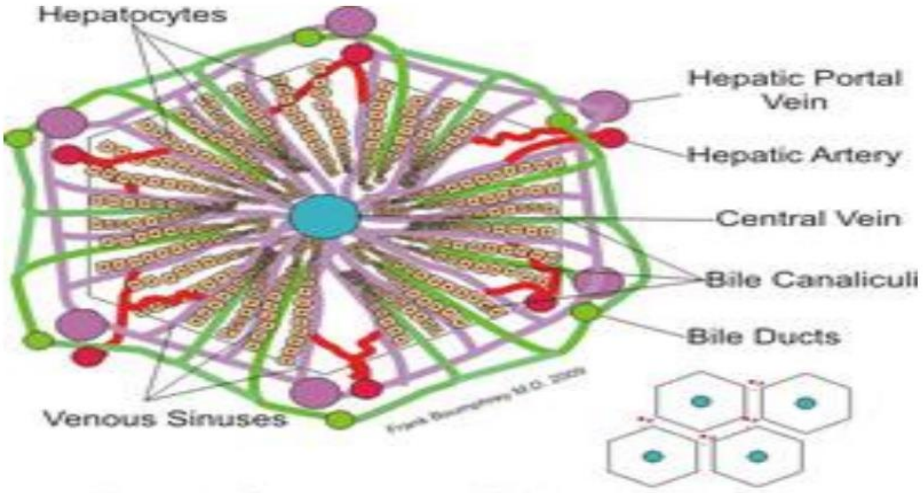
the fissure of the ligamentum venosum. Collectively, these ligaments maintain the liver's position and structural integrity within the abdominal cavity.



Basic Structure of Liver Lobule

Liver Vasculature: The liver, receiving nearly 25% of the body's resting cardiac output, is the most vascular organ. Unlike other organs, its fluid exchange remains largely unaffected by typical haemodynamic changes. Venous drainage primarily occurs through the hepatic veins into the inferior vena cava (IVC). The left and middle hepatic veins usually drain directly into the IVC, while the right hepatic vein, with a shorter and broader extrahepatic course, also empties directly. Additional drainage is sometimes provided by the short retrohepatic vein and an accessory right inferior hepatic vein. Notably, hepatic veins lack the protective Glisson's capsule that surrounds the portal venous system.

Advanced imaging tools like HepaVision help in calculating total liver volume, mapping venous territories, and planning liver resections. These tools isolate vascular structures, convert intrahepatic vessels into branching flow graphs, and use semi-automated techniques to assess liver volume and anatomy in three dimensions.

- **Liver Cell Types and Their Functions**

Hepatocytes form about 80% of the liver's mass and 60% of its total cell population. These large, metabolically active cells are arranged in single-cell-thick plates and are rich in organelles like mitochondria, endoplasmic reticulum, Golgi bodies, lysosomes, and peroxisomes. Hepatocytes play a central role in metabolism—processing proteins, fats, and carbohydrates—and are responsible for synthesizing key serum proteins such as albumin and coagulation factors. They also contribute to the production of the liver's extracellular matrix, which includes collagens, glycoproteins, proteoglycans, and hyaluronic acid.

Sinusoidal endothelial cells line the hepatic sinusoids and possess fenestrations that support their filtration function. Although they are not phagocytic, they have a strong endocytic ability, allowing uptake of immune complexes and small particles, and possibly aiding in viral clearance.

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Kupffer cells, the liver's resident macrophages, represent the largest macrophage population in the body. Positioned along the sinusoids, they constantly interact with gut-derived substances and become mildly activated. Upon stimulation, they release inflammatory mediators like cytokines, nitric oxide, and reactive oxygen species. They can also phagocytose particles by binding to complement or antibody-coated cells.

Stellate cells, located in the space of Disse between hepatocytes and sinusoids, store about 95% of the body's vitamin A in the form of lipid droplets. These cells regulate blood flow through the sinusoids, participate in extracellular matrix turnover, and are key players in liver fibrosis when activated.

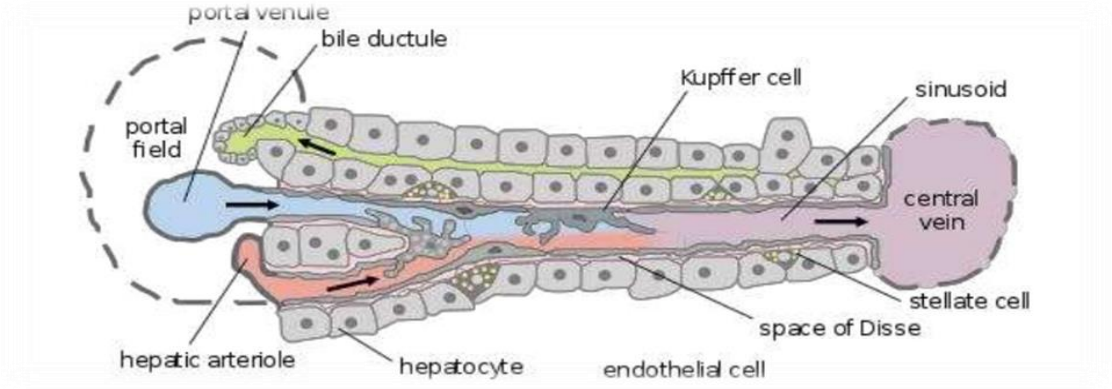

Figure 2: The liver's primary cell types—hepatocytes, endothelial cells, Kupffer cells, and stellate cells—are depicted in the diagram. Source: Pathway services for tissue path specialists, (JC. Ozougwu,etal, 2017)

- **Physiology of liver:**

Liver Function in Aging:

Liver function in elderly individuals generally remains stable, though certain parameters show age-related changes. Bilirubin levels may decline due to reduced hemoglobin and muscle mass, while hepatic enzymes and HDL cholesterol are usually preserved. However, slight reductions in albumin and γ-GT, and increases in bilirubin, have been observed, suggesting mild functional decline with age. The liver plays a vital role in digestion, metabolism, detoxification of xenobiotics, and maintaining energy balance.

Liver Regeneration:

With age, the liver's regenerative ability diminishes. This may be due to reduced levels of circulating epidermal growth factor (EGF), decreased EGF receptor activity, and impaired signaling. Additionally, aged hepatocytes show elevated levels of the Bim protein, which inhibits cell cycle progression. Telomere shortening in elderly individuals, especially those with liver disease, further limits regenerative potential.

Adaptive Immunity:

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Aging impairs T-cell proliferation, differentiation, and signaling. While CD8+ T cells increase, CD4+ levels decline, and reduced CD28 expression diminishes T-cell responsiveness and interleukin production, weakening immune defense.

Drug Metabolism:

Hepatic drug metabolism, particularly phase I reactions, declines with age due to reduced liver size and blood flow. Cytochrome P450 activity drops significantly in older adults, with studies showing up to a 30% decrease by age 70.

Nutrient and Fat Metabolism:

The liver synthesizes fats from carbohydrates and proteins, processes triglycerides from the intestine, and generates cholesterol and phospholipids vital for hormones, bile salts, and cell membranes. Lipoproteins produced by the liver transport fats for energy or storage in adipose tissue.

Protein and Carbohydrate Metabolism:

The liver is central to protein metabolism, producing albumin, globulins (excluding gamma globulin), non-essential amino acids, and enzymes such as ALT, AST, and ALP. It also synthesizes most clotting factors, including fibrinogen, and regulates blood glucose by storing or releasing glucose, and converting amino acids and glycerol to glucose when glycogen stores are depleted.

Vascular Functions and Biochemical Estimations:

The liver plays a key role in blood filtration and storage, holding approximately 200–400 ml of blood within the hepatic sinusoids to assist during haemorrhagic events. Kupffer cells, known for their high phagocytic activity, help eliminate up to 90% of bacteria present in portal blood. Hepatocytes also contribute significantly by synthesizing plasma proteins.

Biochemical Assessments:

Serum levels of aspartate transaminase (AST) and alanine transaminase (ALT) were determined using the method described by Reitman and Frankel (1957), with enzyme activity expressed in kat/l. Sodium (Na^+) and potassium (K^+) concentrations were estimated in mmol/l using the Mediflame-127 systronic flame photometer. Reduced glutathione (GSH) levels in liver tissue were measured by the Ellman (1959) method using DTNB reagent. Lipid peroxidation was assessed by estimating thiobarbituric acid reactive substances (TBARS) following the modified method of Ohkawa et al. (1979), and results were expressed as nmol MDA/mg protein. Total protein content in tissue homogenates was determined using the Lowry method (1951).

- **Etiology of Liver Disease:**

Liver diseases arise from a variety of causes, with the most common being viral infections (especially Hepatitis B and C), alcohol abuse, drug toxicity, autoimmune disorders, parasitic infections, and metabolic syndromes such as obesity, insulin resistance, and diabetes. These factors lead to oxidative stress through the excessive generation of reactive oxygen species (ROS), which damage liver cells by inducing lipid peroxidation. When the body's antioxidant defenses are

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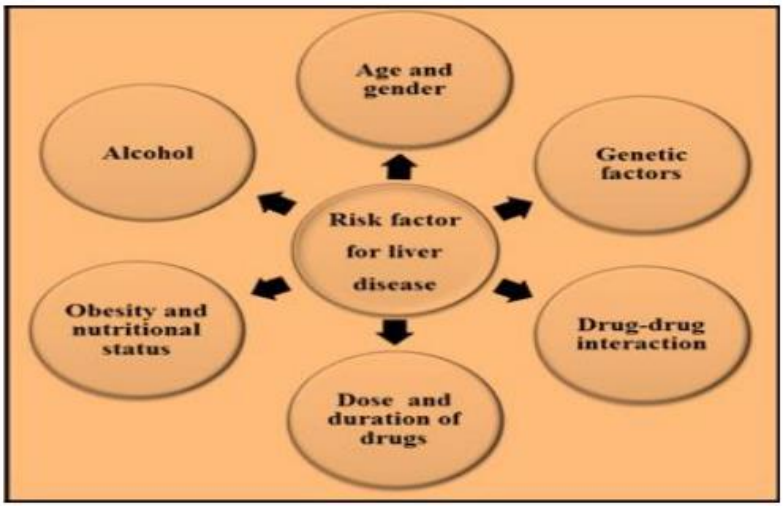
overwhelmed, ROS accumulation can trigger inflammation and contribute to liver conditions like fibrosis, cirrhosis, and hepatocellular carcinoma.

Environmental toxins and chemical exposure have also contributed to the growing prevalence of liver dysfunction in recent decades. Among chronic liver diseases, alcoholism remains a leading cause. It disrupts both biochemical and hematological parameters by impairing bone marrow function, resulting in reduced or abnormal blood cell production.

Viral hepatitis, often caused by infections or substances such as certain drugs, bacteria, and parasites, is another major contributor to liver morbidity and mortality, with an estimated 20,000 deaths annually. Despite modern treatments, traditional remedies using medicinal plants are still widely practiced for liver support. Many of these plants contain bioactive compounds—like flavonoids, phenols, alkaloids, and glycosides—known for their hepatoprotective properties. Plant-based therapies are often preferred for their safety and minimal side effects, supporting their continued role in liver disease management. (MaqboolM,etal, 2019)



- **Mechanism of liver damage**

Despite advances in drug development, many drugs are still withdrawn from the market due to late detection of hepatotoxicity. The liver is particularly vulnerable to damage because of its central role in metabolism and its direct connection to the gastrointestinal tract via the portal vein, which delivers about 75% of its blood supply—rich in absorbed drugs and xenobiotics. Hepatic injury often results from oxidative stress, mitochondrial dysfunction, and enzyme activation, particularly of cytochrome P450 isoforms like CYP2E1. Mitochondrial damage leads to excessive production of reactive oxygen species (ROS), which compromise cell membranes and DNA, accelerating liver cell injury. Hepatocyte and bile duct damage can also lead to bile acid accumulation, further aggravating liver dysfunction. Compounds such as carbon tetrachloride (CCl_4) increase serum markers like SGPT, SGOT, alkaline phosphatase, and bilirubin due to the generation of free radicals that impair cell

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membrane integrity. The ongoing exposure to such reactive intermediates from biotransformation processes underscores the liver's susceptibility to toxic insult.

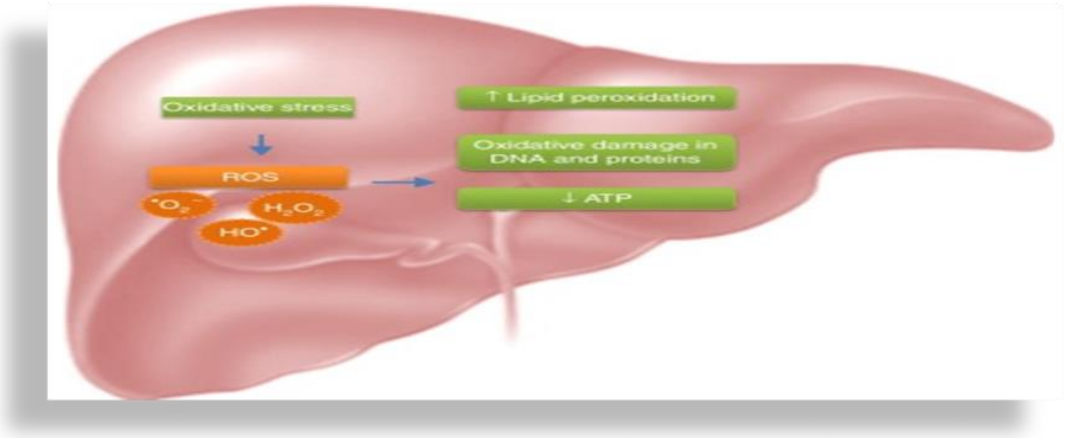

Figure 3. Mechanism of oxidative stress-induced liver damage. (MontemayorC,etal, 2015)

Hepatoprotective agents play a vital role in defending the liver against harmful substances, including hepatotoxins, and in restoring the body's antioxidant defense systems. As a result, both natural and synthetic compounds with liver-protective properties have become the focus of ongoing research. The development of such drugs involves multiple phases, from preclinical testing in cell and animal models to clinical trials assessing safety and efficacy in humans.

Many plant extracts exhibit hepatoprotective effects due to their antioxidant properties and flavonoid content. These extracts have been shown to elevate glutathione levels, increase total protein, reduce lipid peroxidation, and enhance the activity of antioxidant enzymes such as SOD, CAT, GPx, and GST. They also lower levels of hepatic marker enzymes (AST, ALT, ALP, arginase) and reduce bilirubin and malondialdehyde (MDA) concentrations.

In the context of liver inflammation, such as that observed in COVID-19, the virus may directly infect hepatocytes, leading to liver injury. Similar effects have been reported with other respiratory viruses like MERS-CoV and SARS-CoV. Hepatic stellate cells contribute to inflammation by producing cytokines and chemokines such as MCP-1, RANTES, and IL-8. The liver sinusoids, lined by fenestrated endothelial cells and lacking a basement membrane, also play a role in immune regulation by expressing inflammatory mediators like IL-8, MIP-1α, and MCP-1, which influence leukocyte behavior.

Table No-2: during liver inflammation, the population of liver cells produces chemical mediators. (MoriconiF,etal, 2008)

Liver cells	Mediators
Hepatocytes	IL-8, IP-10, MIG, MIP-1,MIP-2, MIP-3,

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Sinusoidal Epithelial cells	RANTES, MCP-1 IL-8, MIP-1α MIP-1 =β;
Kuffer cells	IL-1, IL-6, IL-10, IL-18,
Hepatic stellete cells	Hepatic stellete cells

During inflammation, the chemokine expression profile of hepatic endothelial cells changes significantly. Elevated levels of MIP-1β, IP-10, MIG, and ITAC are observed, reflecting the immune activation state. In parallel, adhesion molecule expression also shifts—healthy sinusoidal endothelial cells typically express PECAM-1, VAP-1, and ICAM-2, but during inflammation, there is a reduction in PECAM-1 and increased expression of ICAM-1, VCAM-1, and selectins (P and E).

Macrophages play a central role in regulating liver inflammation and repair. In damaged tissue, they contribute to immune modulation, clearance of necrotic cells, and activation of myofibroblasts, which promote extracellular matrix production and fibrosis.

Chemotactic signals from hepatocytes and Kupffer cells attract extrahepatic immune cells, especially neutrophils, which are key drivers of acute inflammation and tissue injury. Their infiltration is notably severe in alcoholic hepatitis and plays a major role in liver damage.

Liver disorders encompass a range of conditions including hepatosis, adenomas, cirrhosis, and cancer. Drug-induced liver injury is also a significant concern. Common medications such as paracetamol, diclofenac, amoxicillin, and oral contraceptives can trigger severe hepatic inflammation, vascular obstruction, or necrosis. In extreme cases, acute liver failure may ensue—often due to viral infections or drug toxicity—leading rapidly to multi-organ failure and high mortality, despite current medical advancements.

- **Hepatitis (An inflammation of liver)**

Hepatitis refers to inflammation of the liver, most commonly caused by viral infections. However, other factors such as alcohol abuse, toxic substances (e.g., poisonous mushrooms), certain medications (notably paracetamol overdose), autoimmune disorders, and genetic conditions like hemochromatosis and cystic fibrosis can also contribute to liver damage. Among chemical agents, carbon tetrachloride (CCl_4) is widely recognized for its hepatotoxicity in both human and experimental animal models and is frequently used to evaluate hepatoprotective agents.

The global burden of hepatitis has grown significantly, with major strides made in understanding its viral etiology in the last five decades. The post-World War II period marked key advances in identifying distinct viral forms, transforming public health approaches to liver diseases.

- **Clinical Features of Hepatitis**

Symptoms of hepatitis often appear suddenly and may include fatigue, fever, nausea, vomiting, abdominal pain, jaundice (yellowing of the skin and eyes), dark urine, pale stools, itching, and weight loss. In men, complications such as breast enlargement and abdominal bloating may occur in advanced stages.

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Types of Hepatitis

- Hepatitis A (HAV): Transmitted via the fecal-oral route, often through contaminated food or water. It causes acute liver inflammation, with symptoms typically appearing 2–6 weeks after exposure. Though usually self-limiting, it can last for several months.
- Hepatitis B (HBV): Spread through contact with infected blood or bodily fluids. Symptoms may take up to 6 months to appear and include fatigue, jaundice, joint pain, and hepatomegaly. Chronic HBV infection can lead to severe liver damage.
- Hepatitis C (HCV): Transmitted primarily through blood-to-blood contact. Known as the "silent killer," it often remains asymptomatic until significant liver damage has occurred. Though no vaccine exists, effective antiviral treatments are available.
- Hepatitis D (HDV): Also called the delta agent, HDV infects only individuals already carrying HBV, often worsening the disease course. Transmission routes include drug injection, blood transfusions, and vertical transmission during pregnancy.
- Hepatitis E (HEV): A waterborne infection caused by a non-enveloped, positive-sense RNA virus. Poor sanitation and ingestion of contaminated water or undercooked meat are common transmission pathways. It typically causes acute hepatitis and is especially dangerous in pregnant women.

Alcohol-Related Liver Disease:

Alcohol is a widely consumed substance and a major cause of liver damage worldwide. It can lead to a spectrum of liver conditions, collectively known as alcohol-related liver disease (ALD), which includes fatty liver (steatosis), alcoholic hepatitis, fibrosis, and ultimately cirrhosis. The liver metabolizes alcohol to eliminate it from the body, but excessive intake overwhelms this process, resulting in cellular injury or death. ALD typically progresses in stages—from fatty liver to alcoholic hepatitis, and eventually to cirrhosis—especially in chronic heavy drinkers.

Figure 4: Normal liver and alcoholic fatty liver (Sivakrishnan S, etal, 2019)

- **Alcoholic Hepatitis**

Alcoholic hepatitis is a condition in which the liver becomes inflamed and enlarged due to excessive alcohol intake. Common symptoms include fever, jaundice, abdominal pain, nausea, vomiting, and loss of appetite. It affects roughly 35% of heavy drinkers and can range from mild to life-threatening. While mild cases may improve with alcohol cessation, severe cases can progress rapidly, leading to

liver failure or death. Alcoholic liver disease (ALD) encompasses a spectrum of liver damage caused by alcohol, including fatty liver (steatosis), hepatitis, cirrhosis, and liver cancer. Among these, alcoholic hepatic steatosis—marked by fat buildup in liver cells—is an early and reversible stage that often precedes more serious damage. Fatty liver disease (FLD), the most frequently diagnosed liver disorder, includes both alcoholic (AFLD) and nonalcoholic (NAFLD) forms, each with the potential to progress to cirrhosis and liver cancer.

- **Cirrhosis**

Liver cirrhosis is a progressive condition and one of the leading causes of mortality, particularly in developing regions. While it ranks fourth among causes of death in Central Europe, globally it stands fourteenth. Cirrhosis is now recognized not as a single disease but as a spectrum of clinical stages, each with distinct prognostic outcomes—ranging from a 1% to 57% one-year mortality rate. It represents the final pathway of many chronic liver diseases, marked by fibrosis, regenerative nodules, disrupted liver architecture, and vascular abnormalities, including shunts and thrombosis. These changes lead to portal hypertension, the main driver of cirrhosis-related complications and mortality.

Cirrhosis is often silent until complications like ascites, variceal bleeding, hepatic encephalopathy, or spontaneous bacterial peritonitis appear. Ascites, in particular, signals poor prognosis and diminished quality of life. Abstinence from alcohol and treatment of hepatitis B or C can improve or stabilize the condition, especially in early stages. However, advanced cirrhosis is irreversible and requires liver transplantation.

Histologically, cirrhosis is classified using staging systems like METAVIR and Ishak, but these static models fail to reflect the complex biological and clinical nature of the disease. New insights into fibrogenesis, angiogenesis, and the functional decline of the liver call for a revised definition that considers disease reversibility and therapeutic responses.

Cirrhosis is linked to increased lipid peroxidation, glutathione depletion, and elevated liver enzymes. It may arise from viral infections (notably hepatitis B and C), alcohol abuse, autoimmune conditions, or metabolic disorders. Hepatitis B remains a major cause, especially in Southeast Asia, where millions are infected. Vaccination and antiviral treatments have become essential in managing the viral forms of the disease.

Chronic liver disease progresses in five stages: healthy liver, HBV/HCV infection, chronic hepatitis, cirrhosis, and eventually hepatocellular carcinoma. Disruption in liver homeostasis due to fat accumulation (as in NAFLD or NASH), viral infection, or systemic disorders like diabetes accelerates this progression.

In resource-limited countries, traditional medicine plays a significant role in liver care. Plants like Khaya senegalensis, native to Africa and traditionally used for multiple purposes, have been employed in treating liver ailments such as jaundice, offering potential for future therapeutic development.

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Effective management of cirrhosis requires early diagnosis, preventive strategies, and integration of generalist and specialist care to meet quality indicators established by international liver associations. As research advances, redefining cirrhosis as a dynamic and treatable condition is crucial to improving outcomes.

- **Drug-induced liver injury**

Older adults are more vulnerable to drug-induced liver injury (DILI), making age a significant risk factor. Hospitalization due to DILI tends to be longer in individuals aged 75 and above. Studies, including one from Japan, have found that cholestatic liver injury is more common in those over 65 compared to younger patients (46% vs. 31.6%). This increased risk is partly due to the use of multiple medications to manage age-related comorbidities. For instance, elderly patients were found to be taking significantly more concurrent drugs at the time of liver injury. Research from Western countries echoes these findings, highlighting polypharmacy as a common issue in older populations.

DILI is a major public health concern, often leading to the withdrawal of approved drugs from the market. According to the United States Acute Liver Failure Study Group, over 50% of acute liver failure cases are due to DILI, with 39% linked to paracetamol overdose and 13% attributed to idiosyncratic reactions from other drugs.

Alcohol-related liver damage also contributes significantly to liver pathology, ranging from simple steatosis to cirrhosis and hepatocellular carcinoma (HCC). Early changes like macrovesicular steatosis affect nearly 90% of heavy drinkers and are often reversible with abstinence. More severe forms, such as alcoholic hepatitis, involve ballooned hepatocytes, Mallory bodies, and fibrosis.

Exposure to various hepatotoxic agents—industrial (e.g., carbon tetrachloride, thioacetamide) and pharmaceutical (e.g., acetaminophen, isoniazid, rifampicin)—can result in liver injury. Hepatotoxicity may occur via predictable mechanisms (direct toxicity) or unpredictable, immune-mediated responses (idiosyncratic). Differentiating DILI from other liver diseases is challenging, as histological features often overlap with those seen in viral or autoimmune hepatitis. Hence, diagnosis relies heavily on clinical judgment and thorough patient history.

Some traditional Ayurvedic preparations, such as Liv-52, Kamilari, and Himoliv, have been noted for their hepatoprotective properties, offering alternative support in liver care.

- **Liver tumor**

Elderly patients with hepatic cirrhosis are more likely to develop HCC. Even in the absence of fibrosis, older patients developed HCC, suggesting that ageing itself may be a trigger for hepatocarcinogenesis [1]. Cell necrosis, increased tissue lipid peroxidation, and decreased tissue GSH content are all signs of liver injury. Additionally, the majority of biochemical indicators, including SGOT, SGPT, triglycerides, cholesterol, bilirubin, and alkaline phosphatase, have increased serum concentrations [32].

2. Conclusion:

The liver is a vital and complex organ, structurally supported by a network of ligaments and functionally composed of various specialized cells. Hepatocytes, the primary functional cells, carry

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out essential metabolic, synthetic, and detoxification processes, while Kupffer cells, endothelial cells, and stellate cells each contribute uniquely to immune surveillance, filtration, and vitamin A storage. The liver's vascular architecture—dominated by the portal vein and hepatic veins—supports its high metabolic demand. However, any disruption in these structures or cellular functions can lead to serious liver diseases. Understanding the detailed anatomy, microarchitecture, and physiology of the liver is crucial for early diagnosis, surgical planning, and effective management of hepatic disorders. A deeper insight into these components also opens the door for advanced therapeutic strategies in liver pathology and regenerative medicine.

3. AUTHOR(S) CONTRIBUTION

The writers affirm that they have no connections to, or engagement with, any group or body that provides financial or non-financial assistance for the topics or resources covered in this manuscript.

4. CONFLICTS OF INTEREST

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5. PLAGIARISM POLICY

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REFERENCES

[1] Tajiri K, Shimizu Y. Liver physiology and liver diseases in the elderly. World journal of gastroenterology: WJG. 2013 Dec 14;19(46):8459. [Google Scholar]

[2] Ozougwu JC. Physiology of the liver. International Journal of Research in Pharmacy and Biosciences. 2017 Jan 1;4(8):13-24. [Google Scholar]

[3] Ramadori G, Moriconi F, Malik I, Dudas J. Physiology and pathophysiology of liver inflammation, damage and repair. J Physiolpharmacol. 2008 Aug 1;59(Suppl 1):107-17. [Google Scholar]

[4] Kiernan F. XXIX. The anatomy and physiology of the liver. Philosophical transactions of the Royal Society of London. 1833 Dec 31(123):711-70. [Google Scholar]

[5] Casotti V, D'Antiga L. Basic principles of liver physiology. Pediatric hepatology and liver transplantation. 2019:21-39. [Google Scholar]

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease. International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[6] AbouSeif HS. Physiological changes due to hepatotoxicity and the protective role of some medicinal plants. Beni-suef University journal of basic and applied sciences. 2016 Jun 1;5(2):134-46. [Google Scholar]

[7] Sivakrishnan S, Pharm M. Liver disease overview. World Journal of Pharmacy and Pharmaceutical Sciences. 2019 Jan;8(1):1385-95.

[8] Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. The Lancet. 2014 May 17;383(9930):1749-61. [Google Scholar]

[9] Pinzani M, Rosselli M, Zuckermann M. Liver cirrhosis. Best practice & research Clinical gastroenterology. 2011 Apr 1;25(2):281-90. [Google Scholar]

[10] Adewusi EA, Afolayan AJ. A review of natural products with hepatoprotective activity. J Med Plants Res. 2010 Jul 4;4(13):1318-34. [Google Scholar].

[11] Recknagel RO, Glende EA, Britton RS. Free radical damage and lipid peroxidation. InHepatotoxicology 2020 Jan 16 (pp. 401-436). CRC press. [Google Scholar]

[12] Delgado-Montemayor C, Cordero-Pérez P, Salazar-Aranda R, Waksman-Minsky N. Models of hepatoprotective activity assessment. Medicinauniversitaria. 2015 Oct 1;17(69):222-8. [Google Scholar]

[13] Trepo C. A brief history of hepatitis milestones. Liver International. 2014 Feb;34:29-37.

[14] Huang HL, Wang YJ, Zhang QY, Liu B, Wang FY, Li JJ, Zhu RZ. Hepatoprotective effects of baicalein against CCl4-induced acute liver injury in mice. World journal of gastroenterology: WJG. 2012 Dec 7;18(45):6605.s

[15] Scott Luper ND. A review of plants used in the treatment of liver disease: part 1. Alternative medicine review. 1998;3(6):410-21. [Google Scholar]

[16] Stickel F, Schuppan D. Herbal medicine in the treatment of liver diseases. Digestive and liver disease. 2007 Apr 1;39(4):293-304. [Google Scholar]

[17] Wu J, Song S, Cao HC, Li LJ. Liver diseases in COVID-19: Etiology, treatment and prognosis. World journal of gastroenterology. 2020 May 21;26(19):2286. [Google Scholar]

[18] Frazier TH, Stocker AM, Kershner NA, Marsano LS, McClain CJ. Treatment of alcoholic liver disease. Therapeutic advances in gastroenterology. 2011 Jan;4(1):63-81. [Google Scholar]

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease. International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[19] Mahadevan V. Anatomy of the liver. Surgery (Oxford). 2020 Aug 1;38(8):427-31. [Google Scholar]

[20] Albuquerque-Souza E, E. Sahingur S,(2000).Periodontitis, chronic liver diseases, and the emerging oral- gut-liver axis.Periodontology 2000. 2022;89:125–141.

[21] Sibulesky L. Normal liver anatomy. Clinical liver disease. 2013 Mar 1;2:S1-3.

[22] Sumadewi KT. Embryology, anatomy and physiology of the liver. Indian Journal of Clinical Anatomy and Physiology. 2023;10(3):138-44.

[23] Targher G, Byrne CD. Circulating markers of liver function and cardiovascular disease risk. Arteriosclerosis, thrombosis, and vascular biology. 2015 Nov;35(11):2290-6.

[24] Tajiri K, Shimizu Y. Liver physiology and liver diseases in the elderly. World journal of gastroenterology: WJG. 2013 Dec 14;19(46):8459.

[25] Kshirsagar AD, Mohite R, Aggrawal AS, Suralkar UR. Hepatoprotective medicinal plants of Ayurveda-A review. Asian J Pharm Clin Res. 2011;4(3):1-8

[26] Traoré KT, Ouédraogo N, Ouédraogo GG, Boly GA, Kabré LM, Sombié EN, N'Do JY, Ouédraogo S, Lompo M, Ouédraogo S, Tibiri A. Hepatoprotective activity of aqueous extract of Balanitesaegyptiaca L. Delile (Balanitaceae) roots bark. Int. J. Phytomed. 2020;12:054-9.

[27] Bhardwaj A, Khatri P, Soni ML, Ali DJ. Potent herbal hepatoprotective drugs-A review. Journal of Advanced Scientific Research. 2011 May 10;2(02):

[28] Minnady M, Jayapal G, Poochi S, Nethaji P, Mathalaimuthu B. Hepatoprotective Effect of Indigenous Medicinal Plants: A Review. Indian Journal of Pharmaceutical Sciences. 2022 Sep 1;84(5).

[29] Kumar CH, Ramesh A, Kumar JS, Ishaq BM. A review on hepatoprotective activity of medicinal plants. International journal of Pharmaceutical sciences and research. 2011 Mar 1;2(3):501.

[30] Madrigal-Santillán E, Madrigal-Bujaidar E, Álvarez-González I, Sumaya-Martínez MT, Gutiérrez-Salinas J, Bautista M, Morales-González Á, y González-Rubio MG, Aguilar-Faisal JL, Morales-González JA. Review of natural products with hepatoprotective effects. World journal of gastroenterology: WJG. 2014 Oct 28;20(40):14787.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease*. *International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[31] Bhawna S, Kumar SU. Hepatoprotective activity of some indigenous plants. Int J Pharm Tech Res. 2009 Oct;4:1330-4.

[32] Maheswari C, Maryammal R, Venkatanarayanan R. Hepatoprotective activity of Orthosiphonstamineus on liver damage caused by paracetamol in rats. Jordan J Biol Sci. 2008 Sep;1(3):105-8.

[33] Lahon K, Das S. Hepatoprotective activity of Ocimum sanctum alcoholic leaf extract against paracetamol-induced liver damage in Albino rats. Pharmacognosy research. 2011 Jan;3(1):13.

[34] Ahmad A, Pillai KK, Najmi AK, Ahmad SJ, Pal SN, Balani DK. Evaluation of hepatoprotective potential of jigrine post-treatment against thioacetamide induced hepatic damage. Journal of ethnopharmacology. 2002 Feb 1;79(1):35-41.

[35] Atta AH, Elkoly TA, Mouneir SM, Kamel G, Alwabel NA, Zaher S. Hepatoprotective effect of methanol extracts of Zingiberofficinale and Cichoriumintybus. Indian journal of pharmaceutical sciences. 2010 Sep;72(5):564.

[36] Ali SA, Elbadwi SM, Idris TM, Osman KM. Hepatoprotective activity of aqueous extract of Khayasenegalensis bark in rats. Journal of Medicinal Plants Research. 2011 Oct 30;5(24):5863-6.

[37] Njayou FN, Ngoungoure FP, Tchana A, Moundipa PF. Protective effect of Khayagrandifoliola C. DC. stem bark extract on carbon tetrachloride-induced hepatotoxicity in rats. Int J Ind Med Plants. 2013;29(1):1161-6.

[38] Maqbool M, Rasool S, Dar MA, Bashir R, Khan M. Hepatotoxicity and Hepatoprotective agents: A Mini review. PharmaTutor. 2019 Sep 1;7(9):34-40.

[39] Tatiya AU, Surana SJ, Sutar MP, Gamit NH. Hepatoprotective effect of poly herbal formulation against various hepatotoxic agents in rats. Pharmacognosy Research. 2012 Jan;4(1):50.

[40] Sreenivasamurthy B, Banji D, Banji O. Investigation on antioxidant and hepatoprotective activity of ethanolic leaf extract of Polygonumglabrum Wild on carbon tetrachloride-induced hepatotoxicity in rats. Spatula DD. 2012;2:199-205.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease. International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[41] Shawon SI, Reyda RN, Qais N. Medicinal herbs and their metabolites with biological potential to protect and combat liver toxicity and its disorders: A review. Heliyon. 2024 Feb 15;10(3).

[42] Azab AE, Albasha MO. Hepatoprotective effect of some medicinal plants and herbs against hepatic disorders induced by hepatotoxic agents. J BiotechnolBioeng. 2018;2(1):8-

[43] Vargas-Mendoza N, Madrigal-Santillán E, Morales-González Á, Esquivel-Soto J, Esquivel-Chirino C, y González-Rubio MG, Gayosso-de-Lucio JA, Morales-González JA. Hepatoprotective effect of silymarin. World journal of hepatology. 2014 Mar 27;6(3):144.

[44] Kumar A. A review on hepatoprotective herbal drugs. Int J Res Pharm Chem. 2012;2(1):96-102.

[45] Roy A, Bhoumik D, Sahu RK, Dwivedi J. Medicinal plants used in liver protection-a review. Pharmaceutical and Biosciences Journal. 2014 Feb 20:23-33.

[46] Sagar R, Bhaiji A, Toppo FA, Rath B, Sahoo HB. A comprehensive review on herbal drugs for hepatoprotection of 21st Century. International Journal of Nutrition, Pharmacology, Neurological Diseases. 2014 Oct 1;4(4):191-7.

[47] Saleem TM, Chetty CM, Ramkanth SV, Rajan VS, Kumar KM, Gauthaman K. Hepatoprotective herbs—a review. Int J Res Pharm Sci. 2010 Dec;1(1):1-5.

[48] Maity T, Maity S, Pahari N, Kar DR, Ganguli S. A review on hepatic diseases and development of herbal drugs for the treatment of liver complications. World Journal of Pharmaceutical Research. 2015 Mar 19;4:677-91.

[49] Mittal DK, Joshi D, Shukla S. Hepatoprotective Role of Herbal Plants–A Review. International Journal of Pharmaceutical Sciences. 2012;3:150-7.

[50] Ozougwu JC. Herbal options for the management of drug induced liver damage: A review. Pharmacologyonline. 2011;3:1481-90.

[51] Marjot T, Moolla A, Cobbold JF, Hodson L, Tomlinson JW. Nonalcoholic fatty liver disease in adults: current concepts in etiology, outcomes, and management. Endocrine reviews. 2020 Feb;41(1):66-117.

[52] Wiegand J, Berg T. The etiology, diagnosis and prevention of liver cirrhosis: part 1 of a series on liver cirrhosis. DeutschesÄrzteblatt International. 2013 Feb 8;110(6):85.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease*. *International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[53] Starr SP, Raines D. Cirrhosis: diagnosis, management, and prevention. American family physician. 2011 Dec 15;84(12):1353-9.

[54] Wong MC, Huang J. The growing burden of liver cirrhosis: implications for preventive measures. Hepatology international. 2018 May;12:201-3.

[55] Nusrat S, Khan MS, Fazili J, Madhoun MF. Cirrhosis and its complications: evidence based treatment. World Journal of Gastroenterology: WJG. 2014 May 14;20(18):5442.

[56] Nipanikar SU, Chitlange SS, Nagore D. Pharmacological evaluation of hepatoprotective activity of AHPL/AYTAB/0613 tablet in carbon tetrachloride-, ethanol-, and paracetamol-induced hepatotoxicity models in Wistar albino rats. Pharmacognosy research. 2017 Dec;9(Suppl 1):S41.

[57] Kiso Y, Tohkin M, Hikino H. Assay method for antihepatotoxic activity using carbon tetrachloride induced cytotoxicity in primary cultured hepatocytes. PlantaMedica. 1983 Dec;49(12):222-5.

[58] Ugwu CE, Suru SM. Medicinal plants with hepatoprotective potentials against carbon tetrachloride-induced toxicity: a review. Egyptian Liver Journal. 2021 Dec;11:1-26.

[59] Krishna MG, Pallavi E, Ravi KB, Ramesh M, Venkatesh S. Hepatoprotective activity of Ficuscarica Linn. leaf extract against carbon tetrachloride-induced hepatotoxicity in rats.

[60] Wang M, Zhang X, Ma LJ, Feng RB, Yan C, Su H, He C, Kang JX, Liu B, Wan JB. Omega-3 polyunsaturated fatty acids ameliorate ethanol-induced adipose hyperlipolysis: A mechanism for hepatoprotective effect against alcoholic liver disease. BiochimicaetBiophysicaActa (BBA)-Molecular Basis of Disease. 2017 Dec 1;1863(12):3190-201.

[61] Videla LA. Oxidative stress signaling underlying liver disease and hepatoprotective mechanisms. World journal of hepatology. 2009 Oct 31;1(1):72.

[62] Farghali H, Canová NK, Zakhari S. Hepatoprotective properties of extensively studied medicinal plant active constituents: possible common mechanisms. Pharmaceutical Biology. 2015 Jun 3;53(6):781-91.

[63] Stiehl A, Benz C, Sauer P. Mechanism of hepatoprotective action of bile salts in liver disease. Gastroenterology Clinics of North America. 1999 Mar 1;28(1):195-209.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease*. *International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[64] Chattopadhyay R. Possible mechanism of hepatoprotective activity of Azadirachtaindica leaf extract: part II. Journal of ethnopharmacology. 2003 Dec 1;89(2-3):217-9.

[65] Jayasekhar P, Mohanan PV, Rathinam K. Hepatoprotective activity of ethyl acetate extract of Acacia catechu. Indian journal of pharmacology. 1997 Nov 1;29(6):426-8.

[66] Goldaracena N, Vargas PA, McCormack L. Pre-operative assessment of living liver donors' liver anatomy and volumes. Updates in Surgery. 2024 Mar 25:1-6.

[67] Radtke A, Schroeder T, Sotiropoulos GC, Molmenti E, Schenk A, Paul A, Nadalin S, Lang H, Saner F, Peitgen HO, Broelsch CE. Anatomical and physiological classification of hepatic vein dominance applied to liver transplantation. Eur J Med Res. 2005 May 20;10(5):187-94.

[68] Lee TH, Kim WR, Poterucha JJ. Evaluation of elevated liver enzymes. Clinics in liver disease. 2012 May 1;16(2):183-98.

[69] Malakouti M, Kataria A, Ali SK, Schenker S. Elevated liver enzymes in asymptomatic patients–what should I do?. Journal of clinical and translational hepatology. 2017 Sep 21;5(4):394.

[70] Ekstedt M, Franzén LE, Mathiesen UL, Thorelius L, Holmqvist M, Bodemar G, Kechagias S. Long-term follow-up of patients with NAFLD and elevated liver enzymes. Hepatology. 2006 Oct;44(4):865-73.

[71] Perlemuter G, Bigorgne A, Cassard-Doulcier AM, Naveau S. Nonalcoholic fatty liver disease: from pathogenesis to patient care. Nature Clinical Practice Endocrinology & Metabolism. 2007 Jun;3(6):458-69.

[72] Sambasiva Rao M, Reddy JK. PPARα in the pathogenesis of fatty liver disease. Hepatology. 2004 Oct;40(4):783-6.

[73] Zhou WC, Zhang QB, Qiao L. Pathogenesis of liver cirrhosis. World journal of gastroenterology: WJG. 2014 Jun 21;20(23):7312.

[74] Dowman JK, Tomlinson JW, Newsome PN. Pathogenesis of non-alcoholic fatty liver disease. QJM: An International Journal of Medicine. 2010 Feb 1;103(2):71-83.

[75] Torruellas C, French SW, Medici V. Diagnosis of alcoholic liver disease. World journal of gastroenterology: WJG. 2014 Sep 7;20(33):11684.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease. International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[76] Kasarala G, Tillmann HL. Standard liver tests. Clinical liver disease. 2016 Jul 1;8(1):13-18.

[77] Haas JT, Francque S, Staels B. Pathophysiology and mechanisms of nonalcoholic fatty liver disease. Annual review of physiology. 2016 Feb 10;78(1):181-205.

[78] Eidi A, Mortazavi P, Bazargan M, Zaringhalam J. Hepatoprotective activity of cinnamon ethanolic extract against CCI4-induced liver injury in rats. Excli Journal. 2012 Aug 20;11:495.

[79] Bhavsar SK, Joshi P, Shah MB, Santani DD. Investigation into Hepatoprotective Activity of Citrus limon. Pharmaceutical Biology. 2007 Jan 1;45(4):303-11.

[80] Kapur V, Pillai KK, Hussian SZ, Balani DK. HEPATOPROTECTIVE ACTIVITY OF "JIGRINE" ON LIVER DAMAGE CAUSED BY ALCOHOL-CARBONTETRACHLORIDE AND PARACETAMOL IN RATS. Indian Journal of Pharmacology. 1994 Jan 1;26(1):35-40.

[81] Parganiha R, Tripathi A, Prathyusha S, Baghel P, Lanjhiyana S, Lanjhiyana S, Sarkar D. A review of plants for hepatic disorders. J. Complement. Med. Res. 2022 Oct 12;13(46):10-5455.

[82] Al-Asmari AK, Al-Elaiwi AM, Athar MT, Tariq M, Al Eid A, Al-Asmary SM. A review of hepatoprotective plants used in Saudi traditional medicine

[83] Farghali H, Canová NK, Zakhari S. Hepatoprotective properties of extensively studied medicinal plant active constituents: possible common mechanisms. Pharmaceutical Biology. 2015 Jun 3;53(6):781-91.

[84] C Maheswari, R Maryammal, R VenkatanarayananJordan J BiolSci - 2008jjbs.hu.edu.jo.

[85] Singab AN, Ayoub NA, Ali EN, Mostafa NM. Antioxidant and hepatoprotective activities of Egyptian moraceous plants against carbon tetrachloride-induced oxidative stress and liver damage in rats. Pharmaceutical Biology. 2010 Nov 1;48(11):1255-64.

[86] Chen TM, Subeq YM, Lee RP, Chiou TW, Hsu BG. Single dose intravenous thioacetamide administration as a model of acute liver damage in rats. International journal of experimental pathology. 2008 Aug;89(4):223-31.

Singh Supriya, Jyoti , Mishra Shailender, Jain Neha, Dayal Ram (2025). *A Review on Hepatic Tissues, Cells: Anatomy, Physiology of Liver and Treatment of Hepatic Disease*. *International Journal of Multidisciplinary Research & Reviews, 4(2), 247-267.*

[87] Golbar HM, Izawa T, Wijesundera KK, Bondoc A, Tennakoon AH, Kuwamura M, Yamate J. Depletion of hepatic macrophages aggravates liver lesions induced in rats by thioacetamide (TAA). Toxicologic Pathology. 2016 Feb;44(2):246-58.

[88] Danladi J, Abdulsalam A, Timbuak JA, Ahmed SA, Mairiga AA, Dahiru AU. Hepatoprotective effect of black seed (Nigella sativa) oil on carbon tetrachloride (ccl4) induced liver toxicity in adult wistar rats. J Dental Med Sci. 2013 Jan;4(3):56-62.

[89] Rappai M, Rao PS, Illanthodi S. A study of variation in haematological parameters in chronic liver disease. J Evolution Med Dent Sci. 2019;8:1949-52.

[90] Reyes-Gordillo K, Segovia J, Shibayama M, Vergara P, Moreno MG, Muriel P. Curcumin protects against acute liver damage in the rat by inhibiting NF-κB, proinflammatory cytokines production and oxidative stress. BiochimicaetBiophysicaActa (BBA)-General Subjects. 2007 Jun 1;1770(6):989-96.