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Research

Hepatoprotective Potential of *Caryota urens*: A Comprehensive Literature Review

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Abstract

The context investigates the therapeutic potential of *Caryota urens*, a species rich in diverse phytochemical constituents, for the management of liver diseases. Liver diseases (LD) constitute a significant global health burden, accounting for nearly 2 million deaths annually worldwide. Major etiological factors include chronic viral infections such as hepatitis B (HBV) and hepatitis C (HCV), alcoholic steatohepatitis (ASH), non-alcoholic steatohepatitis (NASH), as well as autoimmune and genetic disorders. This study highlights the bioactive profile of *Caryota urens*, encompassing polyphenols, flavonoids, amino acids, and other metabolites, which possess potent antioxidant and anti-inflammatory properties capable of modulating key mechanisms involved in liver disease pathophysiology. The document further provides an overview of the epidemiology, pathophysiology, risk factors, and progressive clinical stages of liver disease. Current therapeutic approaches primarily emphasize symptomatic relief rather than targeting the underlying disease mechanisms. In this context, *Caryota urens* emerges as a promising natural candidate for the development of novel hepatoprotective therapies. The findings emphasize the necessity for further in-depth investigations to validate the efficacy and safety of its bioactive compounds in liver disease management.

Keywords: Caryota urens, hepatitis B (HBV), hepatitis C (HCV), alcoholic steatohepatitis (ASH)

INTRODUCTION

Liver disease (LD) remains a major contributor to global morbidity and mortality, accounting for nearly two million deaths each year across the world. The condition arises from multiple etiological factors, including chronic viral infections such as hepatitis B virus (HBV) and hepatitis C virus (HCV), excessive alcohol consumption leading to alcoholic steatohepatitis (ASH), metabolic disturbances causing non-alcoholic steatohepatitis (NASH), as well as autoimmune and hereditary liver disorders. Persistent inflammation in the liver promotes progressive fibrosis, a pathological process responsible for approximately 45% of all deaths worldwide.

Fibrosis progression within the liver plays a pivotal role in determining disease prognosis and overall liver function. The extent of fibrotic remodeling is strongly correlated with hepatic performance and serves as a major risk indicator for the development of hepatocellular carcinoma (HCC). In addition, chronic portal hypertension resulting from advanced fibrosis leads to serious clinical outcomes such as ascites, variceal hemorrhage, and hepatic encephalopathy. Consequently, liver cirrhosis represents the 11th leading cause of mortality globally and ranks as the fourth most common cause of death among adults in Central Europe .

Liver fibrosis represents a dynamic pathological process characterized by excessive deposition of extracellular matrix (ECM) components that progressively distort and replace the normal hepatic architecture. Various injurious stimuli, including toxic, metabolic, and viral factors, induce hepatocellular damage and provoke an inflammatory response marked by immune cell infiltration. These immune mediators subsequently activate hepatic stellate cells (HSCs), driving their trans-differentiation into collagen-producing myofibroblasts. Under physiological conditions, this reparative mechanism serves to restore tissue integrity following acute injury; however, when the insult persists, the balance between fibro-genic and anti-fibrotic pathways is disrupted, leading to sustained myofibroblast activation, impaired apoptosis, and progressive scar formation .

In the context of chronic liver disease, a dysregulation between pro-fibro-genic and anti-fibro-genic signaling pathways promotes continuous activation of proliferative, contractile, and migratory myofibroblasts, resulting in excessive extracellular matrix (ECM) accumulation. The progression of hepatic fibrosis or its regression toward a scar-resolving state is predominantly governed by non-parenchymal cells (NPCs), such as Kupffer cells and other resident immune cell populations, which play a pivotal role in modulating the hepatic microenvironment and determining the liver's fibro-genic trajectory.

EPIDEMIOLOGY OF HEPATOTOXICITY

Worldwide, liver-related conditions are responsible for nearly 2 million deaths annually—half of these attributed to cirrhosis-related complications, and the remainder due to viral hepatitis and hepatocellular carcinoma (HCC). Cirrhosis currently ranks as the 11th leading cause of death and liver cancer as the 16th, together accounting for approximately 3.5% of total global mortality. Additionally, cirrhosis ranks among the top 20 causes of disability-adjusted life years (DALYs) and years of life lost (YLLs), contributing 1.6% and 2.1% of the global burden, respectively.

Alcohol consumption remains one of the major determinants of liver disease, with an estimated 2 billion individuals consuming alcohol globally and more than 75 million suffering from alcohol-use disorders, predisposing them to alcohol-associated liver damage. Similarly, metabolic risk factors such as obesity and diabetes play a crucial role, with approximately 2 billion adults classified as overweight or obese and over 400 million diagnosed with diabetes—both strongly linked to the pathogenesis of NAFLD and HCC. The prevalence of viral hepatitis remains persistently high, and drug-induced liver injury (DILI) has emerged as a growing cause of acute hepatic failure. Although liver transplantation is the second most frequently performed solid organ transplant procedure, current rates fulfill less than 10% of the global demand.

ETIOLOGY

- Alcoholic Liver Disease (ALD)

Includes fatty liver, alcoholic hepatitis (reversible), and cirrhosis (irreversible). Heavy alcohol use leads to chronic liver disease, a major cause of CLD.

- Non-Alcoholic Fatty Liver Disease (NAFLD/NASH)

Linked to metabolic syndrome—obesity, hyperlipidemia, and diabetes. Some cases progress to non-alcoholic steatohepatitis (NASH) causing fibrosis. Metabolic risk factors worsen the condition.

- Chronic Viral Hepatitis

Hepatitis B, C, and Dare major causes of CLD. HCV genotypes: 1a/1b (Europe, North America), 3 (Southeast Asia), 4a (Egypt). Untreated hepatitis C may lead to cirrhosis and liver cancer.

- Genetic Causes

* Alpha-1 Antitrypsin Deficiency: Common genetic CLD in children.
* Hereditary Hemochromatosis: Mutation in HFE gene → excess iron → free radicals → fibrosis.
* Wilson's Disease: Autosomal recessive disorder → copper buildup in liver.

- Autoimmune Causes

*Autoimmune Hepatitis (AIH): Chronic liver inflammation, common in females; shows ANA, SMA antibodies.
* Primary Biliary Cirrhosis (PBC) Autoimmune destruction of bile ducts → jaundice, fibrosis; ↑ alkaline phosphatase.
* Primary Sclerosing Cholangitis (PSC): Fibrosis of bile ducts; often linked with ulcerative colitis.

- Other Causes

* Drugs: amiodarone, isoniazid, methotrexate, phenytoin, nitrofurantoin.
* Vascular: Budd–Chiari syndrome.
* Idiopathic (Cryptogenic): ~15% cases with unknown cause.

PATHOPHYSIOLOGY

Liver disease (LD) is a multifactorial and progressive disorder marked by the continuous development of hepatic fibrosis, structural distortion of liver architecture, and the emergence of regenerative nodules. While fibrosis is classically viewed as a permanent alteration, early-stage fibrosis may exhibit partial reversibility under appropriate conditions. However, the precise point at which fibrosis transitions from a reversible to an irreversible state remains unclear. Without therapeutic intervention, LD commonly advances toward extensive fibrosis, nodule formation, and cirrhosis.

The dynamics of fibrosis progression are influenced by etiological, environmental, and host-dependent factors. In a large-scale study involving 4,852 patients with diverse liver disease etiologies, considerable variation was observed in fibrosis development and progression rates. Patients with HIV–HCV coinfection demonstrated the most accelerated fibrosis progression, whereas those with primary biliary cirrhosis exhibited the slowest course. Increasing age correlated with faster fibrosis progression, and female patients generally showed slower rates except in cases of alcoholic liver disease. Moreover, another investigation suggested that genetic polymorphisms may underlie individual differences in fibrosis progression and the severity of hepatic injury, even among patients sharing similar etiological factors.

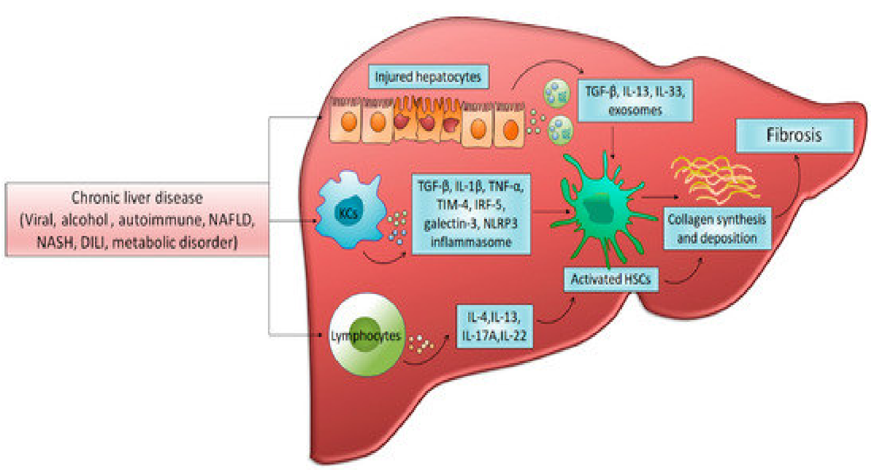

Fig 1: Pathophysiology of Liver Disease

Hepatic fibrosis is a pathological process that develops as a consequence of chronic liver injury from diverse etiologies. It is primarily driven by the activation of hepatic stellate cells (HSCs), which are normally quiescent, vitamin A–storing cells residing in the perisinusoidal space between hepatocytes and sinusoids. In response to sustained hepatic insult, HSCs undergo trans-differentiation into proliferative, contractile, and fibro-genic myofibroblast-like cells. This activation phase is marked by the enhanced expression of inflammatory mediators, including chemokine receptors and intercellular adhesion molecule-1 (ICAM-1), leading to the secretion of cytokines, chemokines, and other leukocyte chemo-attractants that amplify hepatic inflammation. These events initiate a pro-inflammatory cascade, inducing transcriptional and phenotypic reprogramming of liver cells, thereby increasing their sensitivity to cytokine signaling. The continuous activation and persistence of HSCs subsequently result in excessive extracellular matrix (ECM) accumulation and progressive fibrotic remodeling of hepatic tissue, ultimately disrupting normal liver architecture and function.

STAGES OF LIVER DISEASE

Liver disease progresses roughly in four stages:

- Stage 1: Hepatitis involves inflammation of hepatic tissues induced by injury or toxic exposure. It remains one of the predominant causes of liver disorders affecting populations globally. During this stage, both infectious and reparative mechanisms are active; nevertheless, continued hepatic damage results in the aggravation of inflammatory responses.
- Stage 2: Liver fibrosis is a pathological disorder defined by the abnormal accumulation of extracellular matrix proteins, especially collagen. It develops due to persistent and uncontrolled wound-healing responses, leading to progressive architectural distortion of hepatic tissue and eventual loss of liver function.
- Stage 3: Cirrhosis is a progressive and irreversible condition resulting from advanced hepatic fibrosis. It is characterized by extensive deposition of fibrous tissue, destruction of hepatocytes, and distortion of the normal liver architecture, ultimately leading to impaired hepatic function. Cirrhosis constitutes the final stage of chronic liver disease, characterized by an inadequate number of functional hepatocytes to support tissue regeneration. Despite its advanced nature, disease progression at this stage can still be mitigated or halted through timely and appropriate treatment strategies.
- Stage 4: Liver failure is a critical condition characterized by the liver's inability to sustain essential metabolic and synthetic functions required for physiological balance. In most instances, it evolves progressively over several years, a state referred to as decompensated cirrhosis. In contrast, acute liver failure manifests rapidly and is often accompanied by severe complications such as coagulopathy and elevated intracranial pressure.

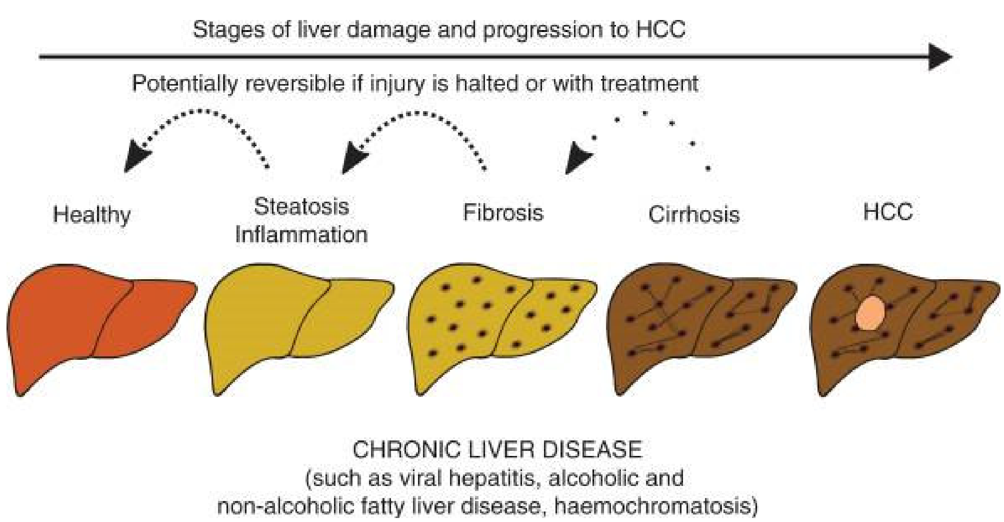

Fig 2: Stages of Liver Disease

Risk Factors for Liver Disease

Several factors can increase the risk of developing liver disease. These include:

- Chronic alcohol use: Regular or heavy drinking can damage liver cells and lead to conditions such as fatty liver, hepatitis, or cirrhosis.
- Obesity: Excess body fat contributes to fat accumulation in the liver, increasing the risk of non-alcoholic fatty liver disease (NAFLD).
- Type 2 diabetes: High blood sugar levels can worsen fat buildup and inflammation in the liver.
- Tattoos or body piercings: If performed with unsterile equipment, they can expose a person to hepatitis viruses.
- Sharing needles: Using or sharing contaminated needles increases the risk of hepatitis B, C, and other infections.
- Blood transfusions before 1992: Before widespread screening, transfusions carried a higher risk of hepatitis C transmission.
- Exposure to infected blood or body fluids: Direct contact can spread viral hepatitis.
- Unprotected sexual intercourse: Increases the chance of hepatitis B and other infections that can damage the liver.
- Exposure to toxins or chemicals: Certain industrial or environmental chemicals can harm liver tissue over time.
- Family history: Genetic factors or inherited conditions can make some individuals more susceptible to liver disease.

ABOUT PLANT

Fig 3: ***Caryota Urens***

Taxonomical classification

Kingdom	Plantae
Subkingdom	Tracheobionta (Vascular plants)
Superdivision	Spermatophyta (Seed plants)
Division	Magnoliophyta (Flowering plants)
Class	Liliopsida (Monocotyledons)
Subclass	Arecidae
Order	Arecales
Family	Arecaceae (Palm family)
Genus	*Caryota*
Species	*Caryota urens* L.

Caryota urens (commonly known as Kithul, fishtail palm, or toddy palm) is a palm species of substantial economic and pharmacological relevance, native to the tropical regions of Asia. The species is a rich reservoir of diverse phytochemical constituents, including polyphenols (e.g., caffeoylquinic and caffeoyl shikimic acids), flavonoids (e.g., quercetin, kaempferol, eriocitrin), amino acids, fatty acids, phytosterols, and carbohydrates. The phloem sap extracted from the inflorescence, locally referred to as sweet toddy, is extensively utilized for the production of fermented beverages such as Kithul toddy, as well as for the preparation of treacle and jaggery through controlled thermal processing of the fresh sap. The pith region of the trunk accumulates starch, which is subsequently processed into flour for dietary and industrial applications. Phytochemical investigations have demonstrated that various parts of *C. urens*—including the roots, leaves, bark, seeds, flowers, and sap—exhibit a broad spectrum of pharmacological activities, notably antidiabetic, anticancer, antioxidant, antimicrobial, anti-inflammatory, and aphrodisiac effects. Owing to these bioactive properties, *Caryota urens* has been extensively employed in traditional and Ayurvedic medicinal systems for the management and treatment of diverse pathological conditions.

Introduction and Origin
Native to Sri Lanka, India, Myanmar and Malaysia.
Description: *Caryota urens* is a monocarpic, solitary-trunked palm that can attain heights of up to 18 m, with a trunk diameter reaching 30 cm. The gray trunk is characterized by conspicuous, widely spaced leaf-scar rings, terminating in a dense crown approximately 6 m in both height and width. Its bipinnate, triangular leaves, bright to deep green in coloration, extend to 3.5 m in length and are supported by 60 cm-long petioles. The obdeltoid pinnae measure 30 cm and possess pointed apices with serrated margins.
Inflorescences emerge sequentially from each leaf node in a top-down arrangement and can reach lengths of 3 m, forming pendulous clusters of white, unisexual flowers. The species produces small, globose drupes (~1 cm diameter) that mature to a red hue and contain a single seed. Consistent with other members of the genus, the fruits contain oxalic acid, a known irritant to skin and mucous membranes. Following flowering and fruiting, *C. urens* completes its life cycle and undergoes senescence.

BIOACTIVE COMPOUNDS PRESENT

Phytochemicals are naturally occurring bioactive compounds produced by plants through primary and secondary metabolic processes, contributing to their growth, defense, and adaptive mechanisms. They contribute to the plant's innate defense system by providing protection against microorganisms and are recognized for their effectiveness in combating a range of plant diseases.

- Polyphenols and acids

Some phenolic compounds possess notable antioxidant, anti-inflammatory, and antimicrobial effects. Umbelliferone or 7-hydroxycoumarin, has been shown to suppress nitric oxide production while maintaining cellular viability. The polyphenolic compounds and organic acids identified in *Caryota urens* are summarized in Table 1.

Plant part	Compound
Leaves	Coumaric acid
	Caffeic acid
	Ferulic acid
	Sinapic acid and their derivatives
	Citric acid
Seed and Inflorescence	3-O-caffeoylquinic acid
	4-O-caffeoylquinic acid
	5-O-caffeoylquinic acid

Table 1: Some polyphenolics and acids present in the *Caryota urens*

- Flavonoids

Caryota urens leaves have been found to contain a diverse range of flavonoid conjugates, including flavonols such as quercetin, kaempferol, and their derivatives, flavones such as apigenin and luteolin-C-glycosides, and flavanols such as proanthocyanidins. Apigenin derivatives have been identified in both the bark and fruits of the plant, indicating their widespread distribution across different tissues. The various types of flavonoids detected in the extract of *Caryota urens* are presented in Table 2**.**

Flavonols	Flavones
Quercetin **10**	Vicenin-2 **19a**
Kaempferol **11**	Isovitexin **19b**
3-Methoxysinensetin **12**	Chrysoeriol-7-O-glucoside **20a**
3-Methoxynobiletin **13**	Chrysoeriol-7-O-(6"-malonyl-glucoside) **20b**
Flavanones	Flavanols
Eriocitrin **21a**	Procyanidin dimer B1 **22**
Hesperetin-3'-O-glucuronide **21b**	3'-O-methyl-(-)-epicatechin-7-O-glucuronide **23**

Table 2: Types of flavonoids identified in the *Caryota urens* extract.

- Amino acids

Glutamine (27f), which occurs in substantial concentrations in *Caryota urens* sap, is responsible for imparting its distinctive flavor. DL-arginine (27b) has been detected in the bark, fruits, and leaves of the tree, highlighting its widespread presence across multiple plant tissues. The amino acids found in *Caryota urens* are detailed in Table 3**.**

Amino Acid	R Group
Alanine **27a**	$—CH_3$
Cysteine **27e**	$—CH_2SH$
Glutamine **27f**	$—CH_2CH_2CONH_2$
Methionine **27k**	$—CH_2CH_2SCH_3$

Table 3: Amino acids and their R Groups identified in *Caryota urens*.

- Other phytochemicals

Piceatannol (compound 28) was isolated from the fruit extract of the *Caryota urens*. In addition, saponins, fatty acids, amides, and lignans have been identified in both the leaf and fruit extracts of *Caryota urens*. The other phytochemicals present in *Caryota urens* are illustrated in Table 4.

Fatty Acid	**Composition (%)**
Palmitic acid **29**	41.24
Oleic acid **30**	28.48
Stearic acid **31**	15.70
Myristic acid **32**	8.01
Caprylic acid **33**	4.31
Linoleic acid **34**	0.63
Lauric acid **35**	0.24

Table 4: Other phytochemicals present in *Caryota urens*

HEALTH BENEFITS OF CARYOTA URENS

- Antioxidant Properties

Caryota urens sap is known for its anti-rheumatic, anti-inflammatory, and anti-hyperglycemic effects, mainly due to its antioxidant activity. Antioxidants protect cells from oxidative damage caused by reactive oxygen species. These properties help prevent diseases like Alzheimer's, Parkinson's, cancer, atherosclerosis, and aging.

- Antidiabetic Properties

Caryota urens products are used in Ayurvedic medicine for managing diabetes. They inhibit α-amylase and α-glucosidase enzymes, which helps control blood glucose levels. Thus, *Caryota urens* sap and treacle have glucosidase inhibition activity. However, *C. urens* flour showed only weak enzyme inhibition, indicating low antidiabetic potential.

- Antimicrobial Properties

Studies show *Caryota urens* extracts have antibacterial and antifungal effects. *Caryota urens* leaves inhibited several pathogens. *Caryota urens* flower extracts were effective against many bacteria (E. coli, S. aureus, Salmonella) and fungi (Aspergillus, Penicillium). *C. urens* fruit also showed antibacterial activity, especially against Shigella dysenteriae.

- Anti-Inflammatory Properties

Caryota urens contains flavonoids that show strong anti-inflammatory and antioxidant effects by neutralizing free radicals and protecting cells from oxidative damage.

CONCLUSION

Research suggests that *Caryota urens*, a species enriched with diverse phytochemical constituents including polyphenols, flavonoids, and amino acids, exhibits considerable potential as a therapeutic agent for liver disorders. These bioactive compounds have been shown in various studies to possess significant antioxidant and anti-inflammatory activities, supporting their hepatoprotective properties.

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