



HYPOCHOLESTEROLEMIC AND CARDIOPROTECTIVE POTENTIAL OF *MURRAYA KOENIGII*: MECHANISTIC INSIGHTS AND EXPERIMENTAL EVIDENCE

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ABSTRACT

Hypercholesterolemia is a major modifiable risk factor for cardiovascular diseases (CVDs), which remain the leading cause of global mortality. While pharmaceutical lipid-lowering drugs such as statins are effective, long-term usage carries risks of side effects, drug interactions, and cost barriers. As global interest in plant-based therapeutics grows, *Murraya koenigii* (curry leaf) has attracted significant research attention due to its traditional use and emerging scientific evidence demonstrating lipid-regulating, antioxidant, anti-inflammatory, and hepatoprotective properties. This review comprehensively examines its botanical background, phytochemical composition, biochemical mechanisms of action, experimental and emerging clinical evidence, safety considerations, and future research directions to elucidate the mechanistic basis of its hypocholesterolemic and cardioprotective effects. Recent studies confirm significant lipid-lowering effects in animal

models and molecular evidence of multi-target cardiovascular protection, although well-designed human clinical trials remain necessary to establish definitive therapeutic efficacy.

KEYWORDS: *Murraya koenigii*, Hypercholesterolemia, Cardiovascular diseases, Hypocholesterolemic activity, Cardio protection, Phytochemicals, Antioxidant activity, HMG-CoA reductase.

INTRODUCTION

Cardiovascular diseases (CVDs) account for nearly 18 million deaths annually, making them the leading global cause of mortality.^[1] Among modifiable risk factors, hypercholesterolemia characterized by elevated total cholesterol and LDL cholesterol (LDL-C) contributes substantially to atherosclerotic plaque formation and vascular dysfunction, leading to myocardial infarction and stroke.^[2] Conventional pharmacological approaches, particularly statins, inhibit the enzyme 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase, effectively lowering LDL-C levels. However, adverse effects such as myopathy, hepatic toxicity, and new-onset diabetes have prompted investigation into complementary and alternative therapies with fewer side effects and broader biochemical action.^[3] Herbal and plant-derived compounds are promising due to their multitarget mechanisms, lower toxicity, and historical ethnomedicinal use. Among these, *Murraya koenigii* (curry leaf) has been recognized in traditional Ayurvedic texts for digestive and metabolic regulation and is now the focus of modern biochemical research, particularly for lipid metabolism and cardiovascular support.

BOTANICAL AND ETHNOMEDICINAL OVERVIEW

Murraya koenigii (L.) Spreng, commonly known as curry leaf, is a small aromatic evergreen tree belonging to the Rutaceae family. It is native to India and Sri Lanka and widely cultivated throughout South and Southeast Asia. The plant typically reaches heights of 4–6 meters, featuring glossy dark green pinnate leaves, small white flowers, and small black fruits. Curry leaves are commonly used as a culinary spice due to their distinctive aroma and flavor, and they are also deeply rooted in traditional health practices.

In Ayurvedic medicine, curry leaves are considered beneficial for digestion, metabolism, and overall wellbeing. Classical texts refer to them as “Hridya” (heart tonic) and “Deepaniya” (digestive stimulant), attributing multiple therapeutic properties including appetite stimulation, gastric regulation, and cardiovascular support. Traditional practitioners have also employed curry leaf remedies for diabetes, anemia, inflammatory disorders, liver dysfunction, and obesity.^[4,5] Ethnobotanical surveys across rural India document its use in managing dyslipidemia and supporting cardiac health, reflecting a long history of empirical observation.^[6] Modern research has increasingly validated these traditional indications, particularly with respect to lipid metabolism and cardioprotective effects.

PATHOPHYSIOLOGY OF HYPERCHOLESTEROLEMIA

Hypercholesterolemia arises from complex metabolic deregulation involving cholesterol synthesis, transport, absorption, and elimination. Endogenously, cholesterol is synthesized in the liver through the mevalonate pathway, with the enzyme HMG-CoA reductase catalyzing the rate-limiting step. Genetic and dietary factors that increase HMG-CoA reductase activity raise cholesterol synthesis above physiological needs.^[7] When synthesis and dietary absorption exceed elimination, elevated circulating LDL-C results. Elevated LDL particles infiltrate the arterial intima and undergo oxidative modification by reactive oxygen species (ROS), producing oxidized LDL (oxLDL). OxLDL is atherogenic, driving macrophage uptake, foam cell formation, and release of pro-inflammatory cytokines such as TNF- α and interleukins which further stimulate vascular injury and plaque development. Persistent vascular inflammation, endothelial dysfunction, and smooth muscle proliferation constitute the progression from fatty streak to mature atherosclerotic lesion.^[8,9]

Moreover, impaired reverse cholesterol transport via HDL further exacerbates lipid imbalance and atherogenesis. In addition to LDL and HDL dynamics, intestinal cholesterol absorption via the Niemann-Pick C1-like 1 (NPC1L1) transporter and hepatic bile acid synthesis are key regulatory nodes. Agents that modulate cholesterol absorption, synthesis, receptor-mediated clearance, or oxidative stress have potential therapeutic value.

PHYTOCHEMICAL COMPOSITION OF *MURRAYA KOENIGII*

The pharmacological effects of *Murraya koenigii* stem from a complex array of bioactive phytochemicals. Recent phytochemical profiling in 2024 identified significant quantities of alkaloids, flavonoids, phenolics, saponins, and tannins in curry leaf extracts, with alkaloids comprising approximately 23.73%, saponins 8.74%, and phenolics 4.4% of the extract, measurable through standardized analytical techniques.^[10] Alkaloids such as mahanimbine, girinimbine, and koenimbine exhibit potent antioxidant effects, while flavonoids and phenolics contribute significantly to free-radical scavenging and anti-inflammatory activity.^[11,12] Saponins and phytosterols interfere with cholesterol absorption at the intestinal level, paralleling the mechanisms of clinically used plant sterols.^[13]

Beyond lipid metabolism, recent network pharmacology research has highlighted multi-target interactions of *Murraya koenigii* bioactives, demonstrating predicted interactions with NF- κ B, TLR4, STAT3, and other signaling pathways implicated in metabolic inflammation and chronic liver diseases such as non-alcoholic steatohepatitis (NASH), suggesting broader

mechanistic implications for metabolic regulation.^[14] These findings support the hypothesis that the plant's lipid-lowering actions are multifactorial and not restricted to a single biochemical target. The major phytochemical constituents of *Murraya koenigii* and their associated biological activities are illustrated in Figure 1.

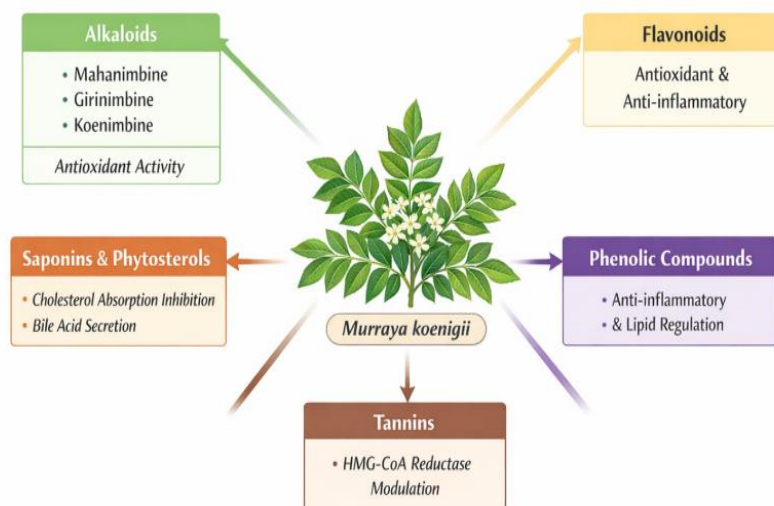


Figure 1: Phytochemical Composition of *Murraya koenigii*.

MECHANISMS OF HYPOCHOLESTEROLEMIC ACTION

Murraya koenigii exerts lipid-lowering effects through multiple biological pathways. Partial inhibition of HMG-CoA reductase has been demonstrated in animal models, reducing endogenous cholesterol synthesis without the potent pharmacological blockade seen with statins.^[15] Enhanced expression of hepatic LDL receptors improves clearance of circulating LDL-C. Antioxidant activity, driven by flavonoids and phenolics, neutralizes ROS and prevents oxidation of LDL particles, thereby reducing foam cell formation and atherogenesis.^[9] Anti-inflammatory effects involving downregulation of TNF- α and IL-6 further mitigate vascular inflammation. Saponin-mediated increased bile acid excretion and competitive inhibition of cholesterol absorption complement these mechanisms. The integrated molecular pathways underlying these hypocholesterolemic and cardioprotective actions are summarized in Figure 2.

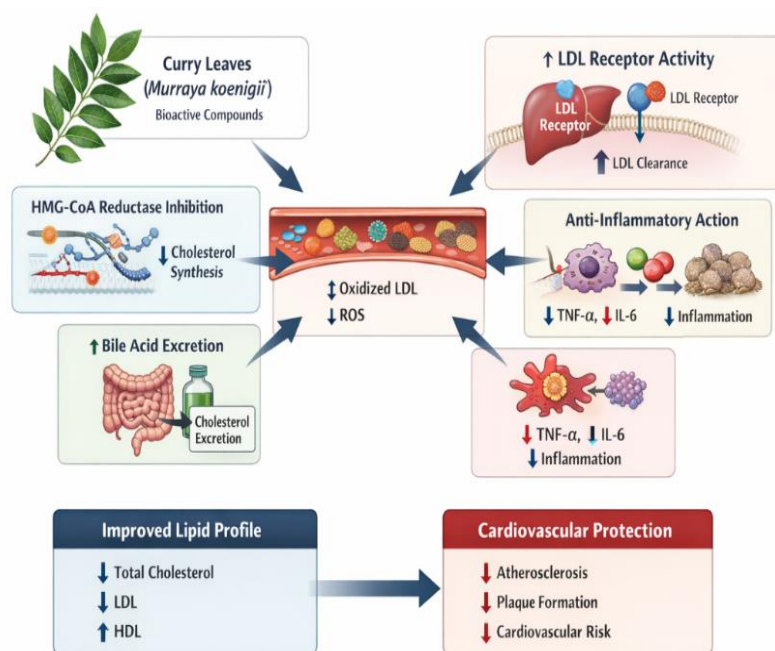


Figure 2: Hypocholesterolemic and Cardioprotective Mechanisms of *Murraya koenigii*.

EXPERIMENTAL AND PRECLINICAL EVIDENCE

Recent experimental studies provide robust evidence for the hypocholesterolemic activity of *Murraya koenigii*. A 2025 study using Wistar rats fed a high-fat fructose diet demonstrated that both aqueous and methanolic extracts of curry leaves significantly reduced total cholesterol and LDL-C levels, while also lowering the atherogenic index, suggesting strong antihyperlipidemic effects closely linked to bioactive components.^[18] Earlier rat studies showed significant reductions in total cholesterol and LDL-C and increases in HDL-C compared with hyperlipidemic controls.^[16,17] While human clinical trials remain limited, broader research on medicinal plants with hypolipidemic potential reinforces the plausibility of plant-based interventions reducing dyslipidemia in at-risk populations.^[20]

SAFETY, TOXICOLOGY, AND PHARMACOKINETICS

Acute toxicity studies indicate that *Murraya koenigii* extracts have a wide safety margin in animal models, with no significant adverse effects observed at moderate doses.^[19] Its long history of consumption as a culinary ingredient also supports a generally favorable safety profile. However, systematic human toxicity and pharmacokinetic studies are still lacking.

CONCLUSION

Evidence from preclinical and emerging mechanistic studies strongly supports the hypocholesterolemic and cardioprotective potential of *Murraya koenigii* through antioxidant,

anti-inflammatory, metabolic regulatory, and bile acid modulation mechanisms. However, clinical validation in humans remains an essential next step before firm therapeutic recommendations can be made. As research continues, curry leaves hold promises as a complementary nutraceutical approach to managing dyslipidemia and supporting cardiovascular health.

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