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Review Article

A Comprehensive Review of the Pharmacological Properties and Therapeutic Potential of *Prosopis farcta*

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ABSTRACT

Prosopis farcta, a medicinal plant of the Fabaceae family, is being explored scientifically on the basis of its diverse biological activities and medicinal importance. Rich in flavonoids, alkaloids, and phenolics, *P. farcta* exhibits strong antioxidant, anti-inflammatory, antimicrobial, and hepatoprotective activities. It has been demonstrated that it can exert neuroprotection by modulating oxidative stress and inflammatory pathways. Further, *P. farcta* possesses antidiabetic activity through the facilitation of the insulin sensitivity and glucose metabolism, and hence it is a good candidate for glycemic control. Its wound healing efficacy via the anti-inflammatory and antimicrobial activities has been studied through in-vivo and in-vitro models. *P. farcta* also possesses cardioprotective activity via lipid metabolism modulation and improvement of endothelial function. Nevertheless, while *P. farcta* fruit extracts have hepatoprotective effects, evidence further suggests the potential for hepatotoxicity with its seed extract, emphasizing dose-dependent analysis. Despite its therapeutic pharmacological potential, additional clinical trials must determine its safety profile, define its optimal therapeutic dosages, and clarify its particular molecular mechanisms of the action. This review consolidates the current evidence in support of the medicinal worth of *P. farcta*, demonstrating its applications in modern medicine.

Keywords: *Prosopis farcta*, medicinal plants, bioactive compounds

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Introduction

Medicinal plants have been regarded as the pillars of traditional medicine since antiquity as a natural reservoir of bioactive compounds for curative and preventive purposes of most diseases. Among them, Prosopis farcta (P. farcta), also known as mesquite or camel thorn, has been brought under limelight due to their various biological activities and therapeutic utilities. A plant that belongs to the Fabaceae family, the shrub is perennial in nature and is native to arid and semi-arid regions, including regions in Africa, Asia, the Middle East, South Asia, and southern of Iran. It reaches a height of 30 to 100 cm and seeds are its means of propagation. The plant blooms between April and June and produces dark brown, oval leguminous pods. Seeds in the pods, when dehydrated, produce rattling sounds upon movement, and for this reason, the plant also has an alternate name of "rattle tree" or "rattle plant." P. farcta is highly resilient against adverse environmental conditions, and its endurance in dry habitats, coupled with its compact phytochemical composition, makes it a promising source of new drugs (Table 1**)** (1, 2).

The phytochemical compounds of P. farcta include flavonoids, phenolic acids, alkaloids, and volatile oils accountable for its multi-directional pharmacological activities. The antioxidant activity opposes the free radicals and has a synergistic effect with endogenous antioxidant enzymes to avert oxidative stress-induced damage. Anti-inflammatory effects of P. farcta are achieved through interference with nuclear factor kappa B (NF-KB) and JAK/STAT signal transduction pathways, which lead to downregulation of pro-inflammatory cytokines (3-7). Its antimicrobial activity, through phenolic and flavonoid compounds, stifles bacterial and fungal pathogens with great efficacy, even drug-resistant ones (2). P. farcta has neuroprotective activity because it modulates oxidative and inflammatory processes, preserves neuronal integrity, and perhaps beneficial in diseases such as Alzheimer's disease (6, 8-12).

In Persian traditional medicine, the herb has been utilized to treat rheumatism, diabetes, and gastrointestinal diseases (2, 13). It has recently been substantiated in literature for its antidiabetic, hepatoprotective, and anti-inflammatory activities. Metabolically, it corrects lipid profiles and improves insulin sensitivity, such that it is seen as a candidate to manage diabetes and hyperlipidemia (14, 15). This review aims to provide a review of the biological activities and medicinal properties of P. farcta, emphasizing the pharmacological modes and clinical relevance. Through integration of the abundance of folk wisdom with modern scientific information, we hope to call attention to the potential applications of this plant in evidence-based medicine. Finally, incorporation of P. farcta into modern therapeutic protocols could contribute to more sustainable, cost-effective, and accessible healthcare practices, particularly in resource-limited settings.

Biological activities

P. farcta exhibits a wide range of biological activities, including antioxidant (16), antibacterial (17), anticancer (8), and anti-inflammatory effects (5), owing to its rich content of bioactive phytochemicals. This plant plays a significant role in combating oxidative stress by modulating key molecular pathways, including activation of the nuclear factor erythroid 2-related factor 2 (Nrf2)/Kelch-like ECH-associated protein 1 (Keap1)

Table 1. A brief botanical description of *P. farcta*

Characteristic	Description
Kingdom	Plantae
Subkingdom	Tracheobionta (Vascular plants)
Super-division	Spermatophyta (Seed plants)
Division	Magnoliophyta (Flowering plants)
Class	Magnoliopsida (Dicotyledons)
Subclass	Rosidae
Order	Fabales
Family	Fabaceae (Leguminosae)
Genus	Prosopis
Species	Farcta
Common Names	Desert Mesquite, Camel Thorn
Growth Form	Perennial shrub with deep root system
Height	Usually, 0.4-1 meter tall
Stems	Woody, branched, often spiny
Leaves	Bipinnate compound leaves, small leaflets
Flowers	Small, yellow to cream-colored, arranged in cylindrical spikes
Fruits	Pods (legumes), compressed between seeds, straw-colored to brown
Root System	Deep-rooted, can extend several meters into soil
Native Range	Western and Central Asia, particularly Iran, Iraq, Syria
Habitat	Arid and semi-arid regions, can tolerate saline soils
Ecological Role	Nitrogen-fixing capabilities, soil stabilization

axis and inhibition of NF-κB signaling, enhancing the activity of antioxidant enzymes [superoxide dismutase (SOD), glutathione peroxidase (GPX), and catalase (CAT)], increasing glutathione levels, and reducing reactive oxygen species (ROS) production (18). The antibacterial effects of P. farcta are mainly mediated through disruption of bacterial cell membranes, inhibition of quorum sensing systems and biofilm formation, induction of oxidative stress, and suppression of essential enzymes such as DNA gyrase and RNA polymerase (19). From an anticancer perspective, P. farcta demonstrates considerable therapeutic potential by inducing apoptosis via increasing the Bax/Bcl-2 ratio and activating caspases, causing cell cycle arrest, inhibiting the PI3K/Akt/mTOR, NF-κB, and JAK/STAT3 pathways, exerting a dual regulatory effect on oxidative stress, suppressing angiogenesis through downregulation of vascular endothelial growth factor (VEGF), and preventing cancer cell invasion and metastasis (20). In addition, P. farcta exhibits notable anti-inflammatory effects by inhibiting key inflammatory signaling pathways, reducing the expression of pro-inflammatory cytokines, suppressing cyclooxygenase (COX) and Lysyl oxidases (LOX enzymes), modulating oxidative stress, and potentially inhibiting NLR family pyrin domain containing 3 (NLRP3) inflammasome activity (21). Figure 1 depicts the biological effects of P. farcta.

Therapeutic effects

A summary of the therapeutic effects of P. farcta is presented in Table 2.

Neuroprotective effects

The neuroprotective action of P. farcta is primarily ascribed to its ability to modulate oxidative stress and inflammation both principal contributory factors to neuronal damage. Luteolin, one of its most well-documented bioactive molecules, has been found to possess very high efficacy against oxidative stress-induced neuronal apoptosis. It has been reported to block cell death in ischemia-reperfusion injury by rejuvenating the pro-oxidant/antioxidant ratio (9). This effect is most pronounced in the hippocampus, where it induces neurogenesis by upregulating neuronal survival markers such as neuronal nuclear antigen (NeuN) and inhibiting apoptotic markers such as doublecortin (DCX) (3). Moreover, luteolin is shown to protect against neurons in Alzheimer's disease models (10, 11). By decreasing amyloid-beta toxicity, it rescues cognitive function and memory. Additionally, luteolin controls critical signaling pathways, for example, cAMP/PKA/CREB, as well as enhances neurotrophic factors such as brain-derived neurotrophic factor (BDNF), which plays a crucial role in neuronal survival as well as in synaptic plasticity (22, 23).

Recent studies also emphasize that luteolin and apigenin, among other phytochemicals present in P. farcta, are responsible for its anti-inflammatory action by modulating significant pathways like MAPK, STAT3, NF-κB, and JAK/STAT. These pathways are significant in the context of neuroinflammatory responses and the development of disorders like Parkinson's and Alzheimer's disease. In a lipopolysaccharide (LPS)-stimulated astrocyte experiment, luteolin and

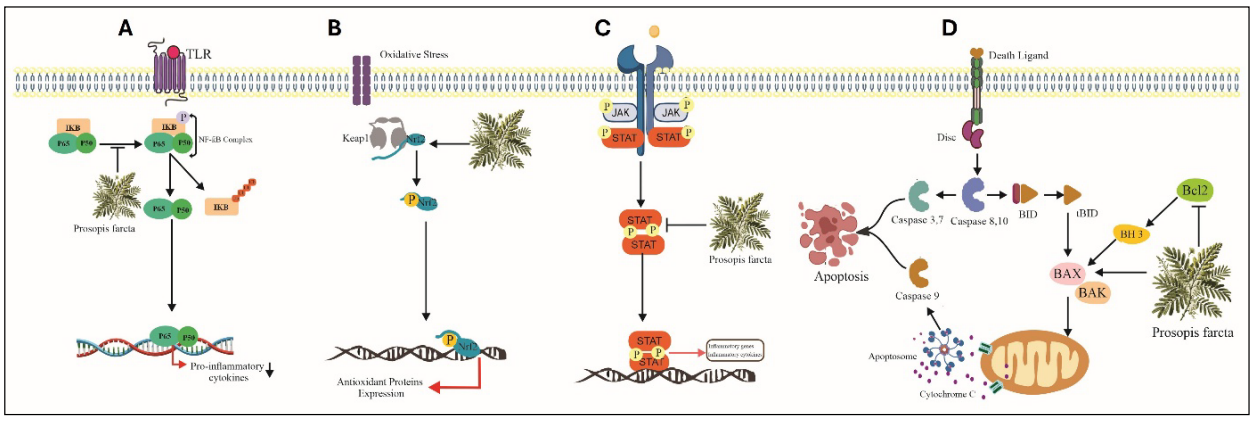

Figure 1. Schematic overview of the proposed molecular mechanisms by which Prosopis farcta (P. farcta) extract exerts antioxidant, anti-inflammatory, and anti-apoptotic effects.

(A) P. farcta modulates inflammatory signaling by suppressing TLR-mediated NF-κB activation, inhibiting IκB phosphorylation and degradation, and preventing NF-κB (p65/p50) nuclear translocation, leading to reduced transcription of pro-inflammatory cytokines. **(B)** Concurrently, under oxidative stress conditions, P. farcta activates the Nrf2/Keap1 signaling pathway, promoting Nrf2 dissociation from Keap1, nuclear translocation, and binding to antioxidant response elements (AREs), thereby enhancing the expression of antioxidant defense proteins. **(C)** In addition, P. farcta interferes with the JAK/STAT pathway by inhibiting STAT phosphorylation, dimerization, and nuclear translocation, thereby attenuating cytokine-driven inflammatory gene expression. **(D)** Furthermore, P. farcta regulates the extrinsic apoptotic pathway, modulating death receptor-mediated caspase activation and the mitochondrial amplification loop via BID cleavage, BAX/BAK activation, cytochrome c release, apoptosome formation, and caspase-9 activation. The extract exerts anti-apoptotic effects through the regulation of Bcl-2 family and BH3 proteins, collectively contributing to cellular protection against oxidative stress, inflammation, and apoptosis.

Table 2. Therapeutic effects of *P. farcta*

Therapeutic effects	Author/years	Model	Plant Part	Formulation/Dosage	Result	Ref
Neuroprotective	Mohammadpour S et al., 2022	Ischemia-reperfusion in mice	Aerial parts	• *P. farcta* fruit extract (150 mg/kg) • Luteolin (30 mg/kg)	• ↓ Oxidative stress • ↑ Neurogenesis in hippocampus • ↓ Neuronal apoptosis	(3)
	Xiong F, and Lv X, 2024	REM Sleep deprivation	Luteolin fraction	• PAX (15 mg/kg) • Luteolin (10 and 20 mg/kg)	• ↓ NF-κB, NLRP3, ASC, Casp-1 • Reversal of anxiety, depressive behaviors • Improved hippocampal neuroprotection	(24)
	Che DN et al., 2020	LPS-stimulated astrocytes	Apigenin and luteolin fraction	• Apigenin (30 μM) • Luteolin (60 μM)	• ↓ IL-31, IL-33 production • Prevented astrocyte activation • Blocked NF-κB, STAT3 nuclear translocation • Modulated NLRP3 inflammasome	(42)
	Mollashahi M, et al., 2013	Sciatic nerve compression in rats	Pods, extracts	Prosopis farcta (25/50/75 mg/kg, i.p., 2 times)	• ↑ α-motoneuron density in spinal cord • Enhanced nerve regeneration	(43)
	Moayeri A, et al., 2018	Morphine withdrawal model	Seeds	• Total PFE (100, 200, and 300 mg/kg) • Luteolin fraction (30, 60, and 90 mg/kg) • Morphine (50 mg/kg)	• ↓ Withdrawal symptoms (teeth chattering, jumping)	(4)
	Ghasemian-Yadegari J. et al. 2024	Cannabis withdrawal model	Aerial parts	• Total PFE (100, 200, and 300 mg/kg) • Luteolin fraction (30, 60, and 90 mg/kg) • Melatonin (10 mg/kg) • THC (10 mg/kg)	• ↑ Mature BDNF • ↓ Dopamine • Attenuation of withdrawal symptoms	(7)
	Lin LF et al., 2012	Neuronal cell models (PC12 cells)	Luteolin fraction	Luteolin (20 μM) for 2 h	• ↑ miRNA-132 expression • Activation of CREB • ↑ ERK phosphorylation, PKA activity • Promotion of neurite outgrowth • ↑ Neuronal survival	(22)
	Zhen JL et al., 2016	PTZ-induced Epileptic rats	Luteolin fraction	• Luteolin (50 and 100 mg/kg/day) • PTZ (35 mg/kg/day, i.p.)	• ↑ BDNF expression • ↑ PKA, CREB phosphorylation • ↓ Oxidative stress, neuronal damage • Suppressed seizure induction, duration, severity • Reversed cognitive impairment	(23)
Antidiabetic	Agirman E. et al., 2022	STZ-induced diabetic rats	Fruit and seeds	• Fruit extract (400 mg/kg) • Seed extract (100/400 mg/kg)	• ↑ Serum insulin, C-peptide levels • ↓ MDA • ↑ ALT/AST (hepatotoxicity indication)	(25)
	Dashtban M, et al., 2016	STZ-induced diabetic rats	Bean	• 50 and 75 mg/kg	• ↓ Blood glucose • No significant effects on lipid profile, hepatic enzymes	(27)
	Feyzman D, et al., 2018	β-TC3 and HepG2 cells	Various extracts	Infusion extract	• ↑ β-cell viability • ↓ Oxidative stress • Improved glucose metabolism • Modulated glucose diffusion	(26)
	Heydari M et al., 2018	High-fructose/STZ-induced diabetic rats	Fruit	Hydroalcoholic extract (100 mg/kg)	• ↓ Blood glucose, triglycerides, cholesterol, LDL-C, VLDL-C • No adverse effects on healthy controls	(44)
	Shahbazi B, et al., 2020	STZ-induced cytotoxic β-TC3 cells, hepatocyte models	Roots	Carbohydrate-rich fractions	• ↑ Glucose consumption • Protection against STZ cytotoxicity in β-cells	(45)
	Nosrati F, et al., 2020	STZ-induced diabetic rats, 3T3 L1 cells	Aqueous extract	Extract (60 mg/kg)	• Outperformed glibenclamide in glucose lowering • Improved glucose uptake in 3T3 L1 cells	(46)
	Mohammed et al., 2020	Alloxan-induced diabetic rats	Roots	N-hexane, ethyl acetate, methanol extracts	• ↓ Blood glucose • Improved liver function parameters (ALP, bilirubin)	(47)

Continued Table 2. Therapeutic effects of *P. farcta*

Therapeutic effects	Author/years	Model	Plant Part	Formulation/Dosage	Result	Ref
Wound Healing	Safari et al., 2021	Rat model	Fruit	100 mg/kg/day	• ↓ IL-12, TNF-α (inflammatory markers) • ↓ Lipid peroxidation (myeloperoxidase, malondialdehyde)	(13)
	Alharbi K et al., 2016	Excision wounded rats	Whole plant	10% methanolic extract	• Attenuated ulcerative colitis-associated wounds • Enhanced wound contraction • ↑ Fibroblast production	(30)
	Heidari A et al., 2012	Diabetic rats	Root and fruit	root powder (40g)	• ↑ Cell proliferation • ↓ Inflammation	(31)
	Noroozi et al., 2019	In vitro/HUVEC and NHDF cell line	Root and fruit	1250 g/mL	• ↑ VEGF-A, FLT1 expression in HUVEC cells	(33)
	Mahmud et al., 2023	Human fibroblast cell lines	Root	200 μg/mL	• ↑ Fibroblast proliferation and migration	(32)
Cardiovascular	Rasheed R et al., 2019	Goat	Root	0.1 mg/ml	• Dose-dependent vasodilation via EDHF (epoxy eicosatrienoic acid) • ↓ Ca^{2+} influx • Negative inotropic effects on coronary vessels	(35)
	Bahrami G et al., 2018	HUVEC cells	Root	25 µg/mL	• ↓ ROS, COX-1, COX-2 enzyme activity • ↓ VCAM-1, ICAM-1 mRNA expression (anti-inflammatory, anti-atherosclerotic)	(21)
	Saidi MR et al., 2016	Rabbits	Root	500 mg/kg/day	• ↓ Total cholesterol, TG, LDL, VLDL, HDL • ↓ Atherosclerosis progression	(48)
	Omidi A et al., 2012	Ostriches	Beans	30 days supplementation	• ↑ HDL • ↓ LDL	(34)
	Rasheed R et al., 2020	In vitro (goat coronary artery)	Root	0.1 mg/ml	• Concentration-dependent relaxation via VGCC and K^+ channels • Mimics metformin properties; targets endothelial-derived factors	(29)
	Asadollahi A et al., 2009	In vitro (rat aorta)	Root	2 mg/ml	• Dose-dependent, endothelium-dependent relaxation via nitric oxide (non-cholinergic)	(49)
	Zana M. Raoof & KD, 2020	Hypertensive/normotensive rats	Fruit	50 mg/kg	• ↑ Urine flow, Na^+ excretion, eGFR, urinary creatinine • ↓ Serum creatinine, urea (mild diuretic, potassium-sparing-like effect)	(50)
	Mohammed IH, 2020	Alloxan-induced diabetic rats	Root	200 mg/kg	• ↓ Serum glucose, TC, TG, VLDL, LDL, WBC count • ↑ RBC, Hb, HCT, HDL levels	(36)
Hepatoprotective	Agirman E, et al., 2022	STZ-induced diabetic rats	Fruit and seed	100 mg/kg bw, 400 mg/kg bw	• ↑ AST, ALT (seed) • ↓ AST, ALT, LDH (fruit) • ↓ MDA, LDH (seed) • ↑ GSH, GST, GR, GPx (fruit) • ↓ SOD, CAT (fruit/seed)	(25)
	Mohammadpour-Zehab et al., 2017	Thioacetamide-induced acute liver toxicity	Seed	100 mg/kg	• ↓ AST, ALT, ALP • ↓ MDA • ↓ Liver damage severity, vacuolation	(37)
	Mohammadpour-Zehab et al., 2018	thioacetamide-induced oxidative stress	Seed	100 mg/kg	• ↑ SOD, CAT • ↓ MDA	(15)
	Hajinezhad et al., 2015	STZ-induced diabetic rats	Pod	300 mg/kg	• ↓ MDA • ↓ Inflammation, vacuolation, fat accumulation (liver tissue)	(38)
	Hajinezhad M, et al., 2015	STZ-induced diabetic rats	Leaves	300 mg/kg	• ↓ MDA • ↓ Inflammation, cellular degeneration (liver tissue)	(39)
	Hajinezhad Hajinezhad M, et al., 2019	High-fat Diet in Rats	Leaves	500 mg/kg	• ↓ AST, ALT, ALP • ↓ MDA • ↑ CAT, SOD	(40)
	Keshavarzi S, et al., 2018	NAFLD-modeled rabbits	Roots	500 mg/kg	• ↓ CPK, LDH, ALT, AST • ↓ Fat droplets, inflammation (liver tissue)	(51)
	Morovati Sharifabad M, et al., 2017	Hypercholesterolemic rats	Roots	80 mg/kg	• ↓ AST, ALT (treatment 90 days)	(41)
	Asadollahi K, et al., 2014	Acetaminophen-induced Hepatotoxicity in Rats	Beans	50 mg/kg, 75 mg/kg	• Prevention of AST, ALT increase with extract pretreatment	(52)
	Alharbi K, et al., 2017	Tetrachloride-induced Hepatotoxicity in Rats	Whole plant	250 mg/kg	• ↓ AST, ALT, ALP, Bilirubin • ↓ MDA, GST • ↑ TAC, GSH	(53)

apigenin significantly inhibited the production of pro-inflammatory cytokines interleukin (IL)-31 and IL-33. Their ability to inhibit astrocyte activation, interfere with NF-κB and STAT3 nuclear translocation, and modulate the NLRP3 inflammasome also speaks to their neuroprotective properties, delivering a targeted therapy for chronic neuroinflammation (9, 24).

Antidiabetic Effects

Experimental studies indicate that the antidiabetic effects of P. farcta are primarily mediated through the coordinated regulation of molecular pathways involved in insulin secretion, insulin sensitivity, and oxidative balance (25). Fruit and root extracts of this plant increase insulin and C-peptide levels while reducing lipid peroxidation markers such as malondialdehyde (MDA), thereby protecting pancreatic β-cells against streptozotocin (STZ)-induced damage and improving their secretory function (25). At the cellular level, enhanced viability and function of β-TC3 cells, together with attenuation of oxidative stress, highlight the pivotal role of P. farcta in preserving β-cell mass and preventing pancreatic dysfunction (26). Concurrently, certain extracts selectively target glucose metabolism without altering hepatic enzyme activity or lipid profiles, indicating a direct effect on glucose homeostasis; however, these outcomes are plant-part dependent, as seed extracts at higher doses have been associated with adverse hepatic effects (27).

At the molecular level, P. farcta exerts a significant influence on glucose uptake and utilization by modulating insulin signaling pathways. Gene expression analyses reveal upregulation of key insulin signaling and glucose transport–related genes, including GLUT2, phosphoinositide 3-kinase (PI3K), and insulin receptor substrate 1 (IRS1), alongside downregulation of glycolytic enzymes such as glucokinase (GK), phosphofructokinase (PFK), and pyruvate kinase (PK), collectively contributing to enhanced insulin sensitivity and optimized glucose metabolism (28). Moreover, bioactive constituents of the root, particularly carbohydrate-rich fractions, increase glucose consumption in hepatocytes and exert marked hepatoprotective and vasculoprotective effects; notably, in some experimental models, these extracts exhibit metformin-like activity by ameliorating hyperglycemia-induced endothelial dysfunction (29). Together, these findings demonstrate that P. farcta mediates its antidiabetic effects through an integrated network of molecular mechanisms encompassing reinforcement of the insulin signaling, cellular protection, regulation of oxidative stress, and improvement of metabolic function in target tissues.

Wound healing Effects

Experimental evidence indicates that the wound-healing effects of P. farcta are primarily mediated through targeted modulation of molecular pathways involved in inflammation, oxidative stress, angiogenesis, and cellular proliferation. The bioactive constituents of this plant, particularly flavonoids, tryptamine-derived alkaloids, and tannins, create a favorable molecular microenvironment for tissue repair by inhibiting microbial growth, ROS production, and modulating inflammatory responses (30, 31). At the cellular level, P. farcta controls the inflammatory phase of wound healing by attenuating lipid peroxidation, suppressing the expression of pro-inflammatory cytokines such as IL-1β and tumor necrosis α (TNF-α), reducing myeloperoxidase activity, and limiting neutrophil infiltration (32, 33). Concurrently, stimulation of fibroblast proliferation, increased epithelial thickness, activation of angiogenesis, and facilitation of extracellular matrix remodeling, through regulation of growth factors and redox-dependent signaling pathways, collectively accelerate wound contraction and effective tissue regeneration (30). These mechanisms are particularly pronounced under pathological conditions such as diabetic wounds, underscoring the pivotal role of P. farcta in coordinating multiple stages of wound repair at the molecular level.

Cardioprotective Effects

Experimental evidence indicates that P. farcta plays a key role in inhibiting the progression of atherosclerosis by reducing MDA levels and modulating inflammatory responses (21). In various animal models, root, fruit, and leaf extracts of P. farcta have improved plasma lipid homeostasis by decreasing total cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein (VLDL), while increasing high density lipoprotein cholesterol (HDL-C) levels, highlighting a direct influence of this plant on molecular pathways governing lipoprotein metabolism and oxidative stress (34).

At the vascular level, P. farcta exerts pronounced vasodilatory effects through complex endothelium-dependent mechanisms. Root extracts induce relaxation of coronary arteries and the aorta by blocking L-type voltage-dependent calcium channels, modulating calcium influx from intracellular stores, activating nitric oxide signaling, and engaging endothelium-derived hyperpolarizing factors (EDHF) and epoxyeicosatrienoic acids (EETs) (35). Moreover, targeting multiple potassium channels (KCa, KATP, and KIR) and amelioration of hyperglycemia-induced endothelial dysfunction suggest an overlap between the vascular molecular mechanisms of P. farcta and those of agents such as metformin (29). In addition, the mild potassium-sparing diuretic effects of this plant, through enhanced glomerular filtration and sodium excretion, may indirectly contribute to blood pressure regulation and improved renal function, warranting further detailed molecular investigations (36).

Hepatoprotective Effects

P. farcta extracts modulate key pathways involved in hepatocyte survival, mitigating oxidative damage, inflammation, and necrosis. Studies in diabetic and chemically-induced liver injury models demonstrate that fruit, seed, leaf, and root extracts improve hepatocyte architecture, restore enzymatic balance (AST, ALT, ALP, LDH), and reduce oxidative stress-mediated hepatocyte degeneration, highlighting their capacity to reinforce cellular defense mechanisms against hepatotoxic insults (25, 37).

At the cellular and tissue levels, P. farcta influences multiple hepatoprotective mechanisms, including the stabilization of hepatocyte membranes, suppression of inflammatory signaling, and reduction of hepatic vacuolation and sinusoidal constriction (38). Root and pod extracts, in particular, have been shown to decrease lipid accumulation in models of non-alcoholic fatty liver disease (39, 40), while fruit and leaf extracts restore antioxidant enzyme activity and improve histopathological features in hyperlipidemia or chemically-induced hepatic damage (41). These findings suggest that P. farcta mediates its protective effects via a network of molecular mechanisms encompassing antioxidant defense, anti-inflammatory modulation, and regulation of cellular metabolic and structural integrity, with distinct plant parts exerting differential efficacy and safety profiles.

Conclusion

The accumulated evidence suggests that *Prosopis farcta* holds significant pharmacological potential due to its broad spectrum of biological activities. Its antioxidant and anti-inflammatory properties contribute to neuroprotection, hepatoprotection, and cardiovascular health. The plant also demonstrates promising antidiabetic and wound-healing effects, making it a valuable candidate for further investigation in metabolic and inflammatory disorders. However, the findings regarding *P. farcta*'s hepatoprotective versus hepatotoxic effects highlight the complexity of its bioactive components, necessitating careful evaluation of its therapeutic dosage and plant part specificity. While preclinical studies support its efficacy, additional clinical trials are essential to validate its safety, standardize its formulations, and explore its potential integration into conventional medicine. Future research should focus on elucidating its precise molecular mechanisms, optimizing its bioavailability, and assessing its long-term effects in human populations.

Author Contributions

Y.M. conceptualized the study and designed the review framework. Y.M., R.T., F.Z., S.N. and T.Y. conducted the literature search and data collection.T.Y. designed the figures. S.N. provided critical insights. Y.M. supervised the project, and reviewed the manuscript for intellectual content. All authors contributed to the writing and editing of the manuscript and approved the final version for submission. Also, the authors confirm that no paper mill and artificial intelligence was used.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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