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Nephroprotective potential of herbal remedies in kidney diseases: A systematic review

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ABSTRACT

Kidney diseases remain a major health concern worldwide, with nephrotoxicity, acute kidney injury (AKI), and chronic kidney disease (CKD) contributing substantially to morbidity and progressive loss of renal function. The kidneys are particularly vulnerable to injury because of their high blood flow, metabolic demands, concentrating ability, and extensive involvement in the elimination of drugs and xenobiotics. Renal injury is a multifaceted process involving interconnected mechanisms, including oxidative stress, inflammation, mitochondrial dysfunction, apoptosis, dysregulated autophagy, ferroptosis, and progressive fibrotic remodelling. These pathways provide important targets for the development of nephroprotective interventions. Although conventional therapies can slow disease progression, their limitations and the increasing interest in complementary approaches have encouraged renewed attention toward medicinal plants and their bioactive constituents. This review critically examines the potential of herbal remedies in protecting against renal injury and explores the molecular mechanisms underlying their effects. Evidence from experimental and clinical studies suggests that several medicinal plants and phytochemicals can attenuate oxidative damage, modulate inflammatory signalling, preserve renal architecture, and interfere with profibrotic pathways such as TGF- β /Smad. However, promising preclinical findings have not yet translated consistently into clinical practice. Variations in phytochemical composition, extraction procedures, dosage, quality control, herb drug interactions, contamination, and limited long-term safety data remain important challenges. Overall, herbal nephroprotectants represent a promising adjunct to conventional renal care, but their clinical application requires standardized preparations, rigorous toxicological evaluation, well designed clinical studies, and stronger mechanistic validation.

Keywords: Nephroprotection, Herbal remedies, Nephrotoxicity, Oxidative stress, Phytochemicals

Introduction

Kidneys play an important part in the maintenance of our endocrine and acid base balance, blood pressure, erythropoiesis etc. Nephrotoxicity is a major concern, especially when the kidneys are exposed to drugs, toxins, or metabolic imbalances that impair renal function. Conventional treatments for chronic kidney disease (CKD), including angiotensin converting enzyme inhibitors, angiotensin II receptor blockers, and calcium channel blockers, primarily slow disease progression rather than providing a cure. The use of natural products in healthcare and disease prevention has gained significant attention, serving as both primary and complementary therapies for various conditions, including those that are manageable as well as chronic or difficult to treat. This review highlights promising alternative approaches, emphasizing plant-based therapies and natural extracts as potential solutions for enhanced prevention and management of kidney diseases.

Kidney diseases represent a significant global health burden, affecting millions of individuals and contributing to high morbidity and mortality rates. The kidneys, essential for maintaining homeostasis, are responsible for filtering metabolic waste, regulating fluid and electrolyte balance, and producing hormones involved in erythropoiesis and blood pressure control. However, their high perfusion rate and metabolic activity render them particularly vulnerable to toxic insults from drugs, environmental chemicals, and metabolic byproducts. Nephrotoxicity defined as kidney dysfunction resulting from exposure to nephrotoxic agents is a common clinical challenge, often leading to acute kidney injury (AKI), chronic kidney disease (CKD), or end stage renal disease (ESRD). Conventional pharmacological interventions for renal disorders, including corticosteroids, immunosuppressants, and dialysis, are often associated with limited efficacy, high costs, and adverse effects. This has spurred growing interest in alternative and complementary therapies, particularly those derived from medicinal plants. Herbal remedies have been used for centuries in traditional systems of medicine such as Ayurveda, Unani, and Traditional Chinese Medicine to manage renal ailments. These

botanicals are rich in bioactive compounds such as flavonoids, alkaloids, terpenoids, and phenolics that exhibit antioxidant, *anti*-inflammatory, diuretic, and immunomodulatory properties, all of which are relevant to nephroprotection.

Recent scientific investigations have validated the nephroprotective potential of several medicinal plants against chemically induced nephrotoxicity in both *in vitro* and *in vivo* models. For instance, *Boerhavia diffusa*, *Andrographis paniculata*, *Tribulus terrestris*, and *Bergenia ligulata* have demonstrated efficacy in mitigating renal damage induced by agents like cisplatin, gentamicin, and heavy metals. These herbs exert their protective effects through mechanisms such as scavenging reactive oxygen species (ROS), modulating inflammatory cytokines, enhancing antioxidant enzyme activity, and preserving renal histoarchitecture. Despite promising preclinical data, the clinical translation of herbal nephroprotectants remains limited due to challenges such as variability in phytochemical composition, lack of standardization, potential herb-drug interactions, and insufficient clinical trials. Therefore, a systematic review of the existing literature is essential to consolidate current knowledge, identify gaps, and guide future research.

This review aims to systematically evaluate the nephroprotective potential of herbal remedies in the context of kidney diseases. It will explore the pathophysiological basis of nephrotoxicity, summarize experimental and clinical evidence supporting herbal interventions, and critically assess their mechanisms of action, safety profiles, and translational potential. By integrating traditional wisdom with modern scientific insights, this review seeks to highlight the role of phytotherapy as a viable adjunct or alternative in the management of renal disorders.

Why is the kidney highly vulnerable to injury

The kidney possesses several distinctive physiological and anatomical characteristics that make it particularly susceptible to toxic, ischemic, metabolic and inflammatory injury. Despite representing only a small fraction of total body mass, the kidneys receive approximately 20-25% of cardiac output, ensuring continuous delivery of circulating drugs, xenobiotics and endogenous metabolites to the renal circulation. Although this high blood flow is essential for maintaining glomerular filtration, it simultaneously increases renal exposure to potentially nephrotoxic substances. Furthermore, the renal circulation is highly heterogeneous, with relatively low blood flow and oxygen tension in the medulla, particularly in regions such as the medullary thick ascending limb, thereby creating an environment that is vulnerable to hypoxic injury (Brezis *et al.*, 1995) [1].

The high metabolic demand of renal tubular epithelial cells represents another important determinant of renal susceptibility. Tubular reabsorption requires extensive activity of the Na⁺/K⁺-ATPase and other energy dependent transport systems, resulting in substantial ATP requirements and high oxygen consumption. Approximately 80% of renal oxygen consumption has been attributed to active sodium transport. Consequently, disturbances in oxygen delivery, mitochondrial respiration or ATP production can rapidly compromise tubular transport and cellular integrity. The proximal tubule, in particular, relies heavily on mitochondrial oxidative metabolism and has limited glycolytic reserve, making it especially vulnerable to ischemia, hypoxia and mitochondrial toxicants (Schaub *et al.*, 2020 & Nourbakhsh & Singh, 2014) [2]. The remarkable concentrating ability of the kidney can further increase susceptibility to toxic injury. During urine formation, water reabsorption results in substantial concentration of solutes and certain drugs or their metabolites within the tubular fluid and renal medulla. Consequently, compounds present at relatively low concentrations in plasma may reach considerably higher local concentrations within the renal tubular system. Such accumulation may promote direct tubular cytotoxicity, oxidative stress and, in susceptible circumstances, ischemic injury (Brezis *et al.*, 1995) [1].

The kidney is also continuously exposed to circulating xenobiotics because it constitutes a major organ for elimination of drugs, environ-

mental contaminants and metabolic waste products. Renal elimination involves glomerular filtration, tubular secretion and tubular reabsorption. While these processes are essential for detoxification and systemic homeostasis, they may paradoxically increase the exposure of renal tubular cells to potentially harmful compounds. The proximal tubule contains an extensive network of solute carriers and ATP-binding cassette transporters that mediate the uptake and secretion of organic anions, organic cations and other xenobiotics. Important examples include organic anion transporters (OAT1/OAT3), organic cation transporter 2 (OCT2), multidrug and toxin extrusion transporters (MATE1/MATE2-K), *P*-glycoprotein and multidrug resistance-associated proteins. Transporter mediated handling can therefore have a dual role in renal physiology. On one hand, these transport systems facilitate elimination of drugs and toxins and protect the systemic circulation from their accumulation. On the other hand, uptake of nephrotoxic substances into tubular epithelial cells can increase intracellular exposure and promote renal injury. Nephrotoxicity may become particularly pronounced when cellular uptake exceeds efflux or when efflux pathways are inhibited. Cisplatin provides a well characterized example in which transporter mediated renal uptake contributes to accumulation within proximal tubular cells and subsequent nephrotoxicity (George *et al.*, 2017, Yin & Wang, 2016, Łapczuk-Romańska *et al.*, 2023) [3].

The proximal tubule is consequently one of the major targets of nephrotoxic injury because it combines high metabolic activity, abundant mitochondria, extensive reabsorptive and secretory functions, and high expression of drug and xenobiotic transporters. The S3 segment is particularly vulnerable because of its relatively hypoxic location and substantial metabolic requirements. Injury to proximal tubular cells can involve mitochondrial dysfunction, ATP depletion, oxidative stress, disruption of ion homeostasis, apoptosis and necrosis. Oxidative stress represents a common downstream mechanism linking these susceptibility factors. High mitochondrial activity and exposure to nephrotoxic compounds can increase reactive oxygen species generation. Excessive ROS can cause lipid peroxidation, protein oxidation, DNA damage and mitochondrial dysfunction, thereby amplifying tubular injury. Mitochondrial damage may further increase ROS production and reduce ATP generation, creating a self-perpetuating cycle of oxidative stress and cellular injury. Collectively, these characteristics explain the apparent paradox of renal physiology: mechanisms that are essential for kidney function including high renal blood flow, filtration, active tubular transport, reabsorption, xenobiotic secretion and urine concentration can simultaneously increase renal exposure to toxic substances and enhance susceptibility to injury. This biological vulnerability provides an important rationale for investigating nephroprotective interventions capable of reducing oxidative stress, inflammation, mitochondrial dysfunction, apoptosis and fibrosis. In this context, medicinal plants and their bioactive phytochemicals are of particular interest because many exhibit antioxidant, *anti*-inflammatory, mitochondrial protective and *anti*-fibrotic properties that may counteract several of the interconnected pathways involved in renal injury (Nigam *et al.*, 2015, Perazella, 2018) [4].

Pathophysiological mechanisms of kidney injury

Kidney injury is a complex and multifactorial process involving interconnected molecular and cellular mechanisms rather than a single pathogenic pathway. Depending on the underlying insult, renal injury may involve excessive production of reactive oxygen and nitrogen species, activation of inflammatory pathways, mitochondrial dysfunction, apoptosis, dysregulated autophagy, endoplasmic reticulum stress, ferroptosis, pyroptosis and activation of profibrotic signalling. These processes interact extensively and can form self-amplifying cycles that promote tubular epithelial injury, glomerular dysfunction, interstitial inflammation and progressive renal fibrosis. Understanding these mechanisms is particularly important for evaluating the nephroprotective potential of herbal medicines because many plant derived phytochemicals act simultaneously on several of these pathways.

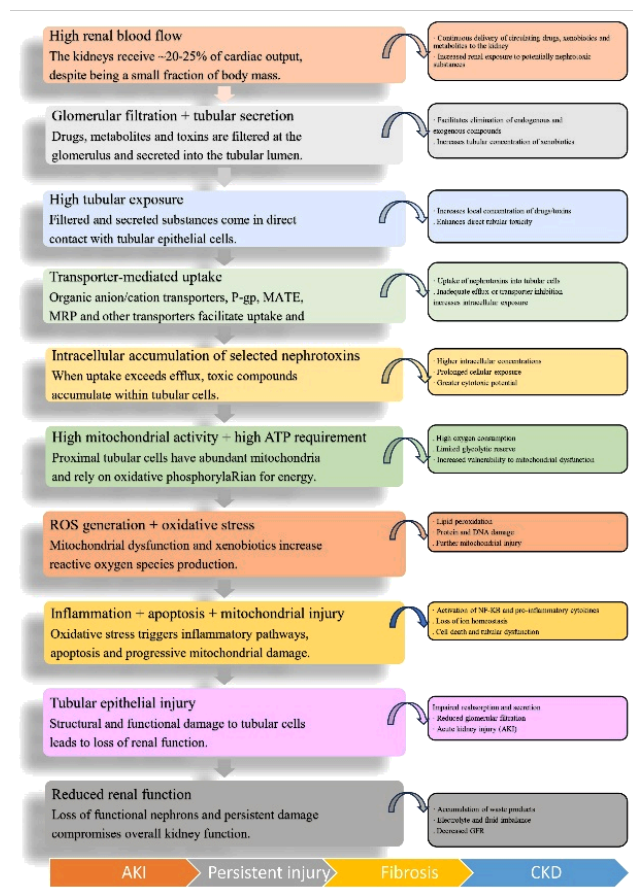


Figure 1. Progression of kidney diseases

Table 1. Molecular pathways targeted by herbal remedies

Pathway	Role in renal injury
Nrf2/HO-1	Oxidative stress
NF-κB	Inflammation
TGF-β/Smad	Fibrosis
NLRP3	Inflammasome activation
PI3K/AKT	Survival/metabolism
AMPK/mTOR	Autophagy/metabolism
SIRT1	Oxidative stress/inflammation
Nrf2/GPX4	Ferroptosis

Oxidative stress

Oxidative stress is one of the major pathogenic mechanisms involved in both acute and chronic kidney injury. It develops when the generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) exceeds the capacity of endogenous antioxidant defences. Important ROS include superoxide anion ($O_2^{\bullet-}$), hydrogen peroxide (H_2O_2) and hydroxyl radicals ($\bullet OH$), whereas RNS include nitric oxide derived reactive species such as peroxynitrite ($ONOO^-$). Excessive ROS/RNS can oxidize lipids, proteins and nucleic acids, resulting in membrane damage, enzyme dysfunction, mitochondrial injury and DNA damage. Lipid peroxidation generates secondary reactive aldehydes such as malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE), which can further modify cellular proteins and amplify oxidative injury. The major endogenous antioxidant defences include superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and the glutathione (GSH) system. Depletion of GSH and impairment of antioxidant enzyme activity can therefore increase the susceptibility of renal cells to oxidative damage. An important endogenous protective mechanism is mediated by nuclear factor erythroid 2-related factor 2 (Nrf2), which regulates the transcription of antioxidant and cytoprotective genes, including heme oxygenase-1 (HO-1) and enzymes involved in glutathione metabolism. Impairment

of Nrf2 mediated antioxidant signalling can contribute to persistent oxidative stress and progression of renal injury (Gwozdinski *et al.*, 2021) [5].

Inflammation

Inflammation is a central component of renal injury and contributes to both the initiation and progression of kidney disease. Renal injury activates resident tubular epithelial cells, endothelial cells, dendritic cells and infiltrating immune cells, resulting in the release of inflammatory mediators and recruitment of additional leukocytes. Nuclear factor-kappa B (NF-κB) is one of the major transcriptional regulators of renal inflammation and controls the expression of numerous inflammatory genes. Activation of NF-κB promotes production of pro-inflammatory cytokines and mediators, including tumour necrosis factor-α (TNF-α), interleukin-1β (IL-1β), interleukin-6 (IL-6), cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS). Excessive iNOS activity can increase nitric oxide production and contribute to nitrosative stress, while inflammatory cytokines can further stimulate oxidative stress, endothelial dysfunction and tubular cell injury. The NLRP3 inflammasome represents an additional important inflammatory pathway in renal disease. Activation of NLRP3 can promote caspase-1 activation and maturation of IL-1β and IL-18, thereby amplifying local inflammation and contributing to tubular injury and renal fibrosis. Thus, oxidative stress and inflammation are closely interconnected processes in which ROS can activate inflammatory signalling while inflammatory mediators can further enhance ROS generation (Henedak *et al.*, 2024) [6].

Renin-angiotensin system

The renin-angiotensin system (RAS) plays an important role in the regulation of renal hemodynamic, sodium and water balance and blood pressure, but persistent activation of the classical ACE/angiotensin II (Ang II)/AT1 receptor axis contributes to renal injury. Ang II acting through the AT1 receptor promotes vasoconstriction, sodium retention, oxidative stress, inflammation, cellular proliferation and profibrotic responses. Ang II can stimulate NADPH oxidase-dependent ROS generation, thereby linking RAS activation with oxidative stress and inflammatory signalling. In contrast, the discovery of ACE2 established a counter-regulatory RAS pathway. ACE2 converts Ang II into angiotensin, which acts predominantly through the Mas receptor and generally opposes several pathological effects of Ang II. The ACE2/Ang-(1-7)/Mas axis is associated with vasodilatory, natriuretic, anti-inflammatory, antioxidant and potentially antifibrotic effects in the kidney. Thus, the balance between the classical ACE/Ang II/AT1 pathway and the protective ACE2/Ang-(1-7)/Mas pathway is an important determinant of renal homeostasis and disease progression (Santos *et al.*, 2017) [7].

Apoptosis

Apoptosis is an important form of regulated cell death involved in renal tubular and glomerular injury. In response to ischemia, nephrotoxic drugs, oxidative stress and inflammatory mediators, mitochondrial damage can alter the balance between pro and anti-apoptotic members of the B-cell lymphoma-2 (Bcl-2) family. Increased expression or activation of the pro-apoptotic protein Bax promotes mitochondrial outer membrane permeabilization, whereas Bcl-2 acts to maintain mitochondrial membrane integrity and suppress apoptosis. Following mitochondrial membrane permeabilization, cytochrome-c is released into the cytosol, where it associates with apoptotic protease activating factor-1 (Apaf-1) and procaspase-9 to form the apoptosome. Activation of caspase-9 subsequently triggers executioner caspase-3, resulting in proteolytic degradation of cellular components and programmed cell death. Therefore, an increased Bax/Bcl-2 ratio, mitochondrial membrane permeabilization, cytochrome-c release and activation of caspase-9 and caspase-3 are important molecular events associated with apoptotic renal cell injury. Excessive tubular epithelial apoptosis can impair tubular integrity and contribute to deterioration of renal function (Piko *et al.*, 2023) [8].

Renal fibrosis

Renal fibrosis is the final common pathological pathway associated with progressive chronic kidney disease and is characterized by excessive accumulation of extracellular matrix (ECM) components within the glomerular and tubulointerstitial compartments. Transforming growth factor- β 1 (TGF- β 1) is considered one of the major profibrotic mediators in renal disease. Following activation, TGF- β 1 binds to its receptors and activates receptor associated Smad2 and Smad3. Phosphorylated Smad2/3 forms a complex with Smad4 and translocates into the nucleus, where it regulates transcription of profibrotic genes. Persistent activation of this pathway promotes accumulation of collagen I, collagen III and fibronectin and contributes to activation of α -smooth muscle actin (α -SMA) positive myofibroblasts. Connective tissue growth factor (CTGF) can further contribute to ECM synthesis and stabilization. TGF- β 1 signalling can also promote tubular epithelial injury, inflammation and other processes that facilitate fibrotic remodelling. Consequently, sustained activation of the TGF- β 1/Smad2/3 pathway represents an important molecular link between persistent renal injury and progressive tubulointerstitial fibrosis (Meng *et al.*, 2015) [9].

Herbal remedies: definition, scope, and research importance

Herbal remedies encompass a wide range of plant derived therapeutic products used in traditional and modern medicine. The World Health Organization defines herbal medicines as preparations containing

whole plants, plant parts, or combinations thereof as active ingredients. These may be employed as whole medicinal plants, either fresh or dried, where the entire plant is consumed or applied to harness its natural phytochemical complexity. Alternatively, specific plant parts are used: leaf extracts often provide antioxidant and *anti*-inflammatory compounds (e.g., flavonoids), root extracts may contain alkaloids or glycosides with potent pharmacological activity (such as ginseng roots), bark extracts are rich in tannins and polyphenols (e.g., cinchona bark for quinine), seed extracts can yield oils and proteins with medicinal properties (e.g., milk thistle seeds), and fruit extracts are commonly used for vitamins, polyphenols, and carotenoids (Ekor, 2014) [11]. Essential oils, obtained by distillation, represent volatile aromatic compounds with antimicrobial or calming effects (Bakkali *et al.*, 2008) [12]. Beyond single plant preparations, polyherbal formulations combine multiple herbs to achieve synergistic therapeutic outcomes, while traditional formulations such as those in Ayurveda or Traditional Chinese Medicine follow culturally established recipes and preparation methods (Patwardhan *et al.*, 2005) [13]. In contrast, isolated phytochemicals (e.g., curcumin from turmeric, resveratrol from grapes) are purified compounds studied for precise pharmacological effects, whereas standardized extracts ensure reproducible efficacy by maintaining fixed concentrations of bioactive constituents (Heinrich *et al.*, 2022).

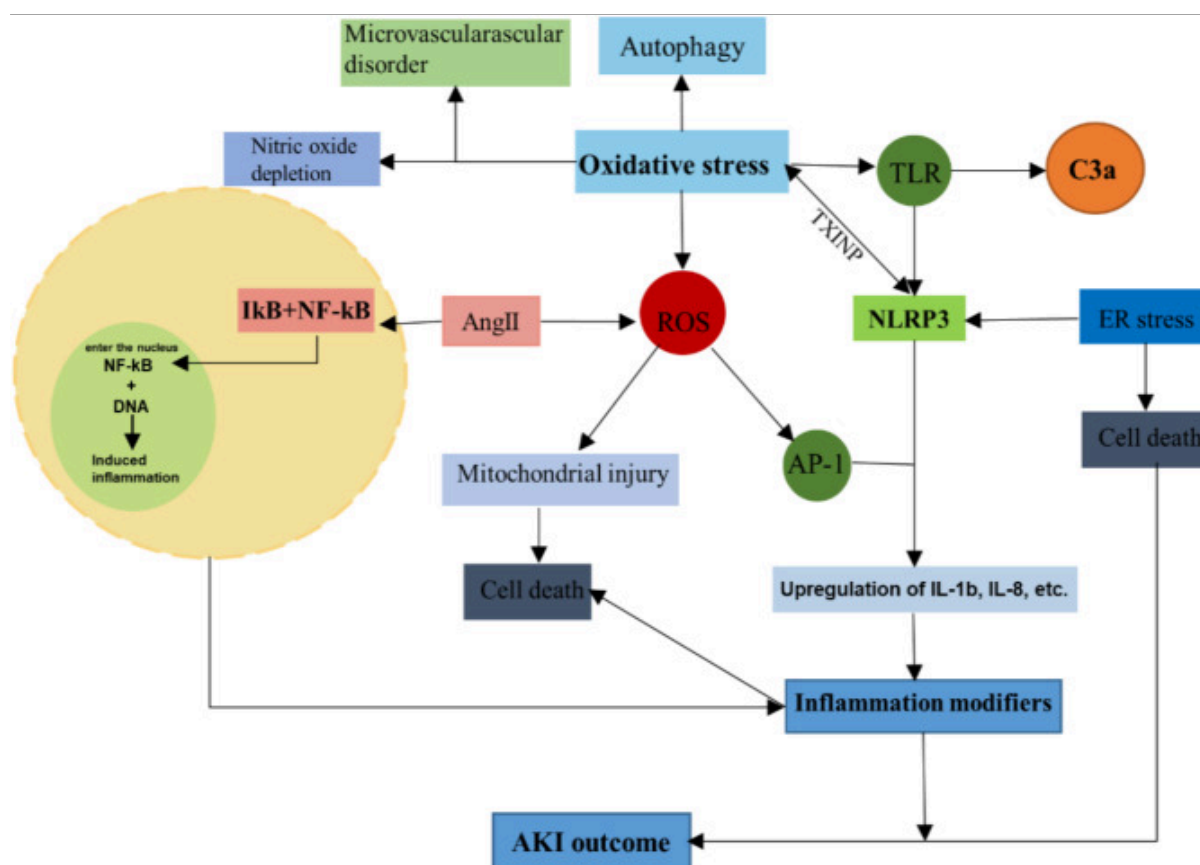


Figure 2. Pathophysiological Mechanisms of Kidney Injury (He *et al.*, 2025) [10].

Distinguishing these categories is crucial in research and clinical practice because their pharmacological consistency, safety, and regulatory status differ significantly. Whole plant preparations preserve the natural synergy of phytochemicals but may vary in potency due to environmental and harvesting conditions (Williamson, 2001) [14]. Extracts from specific plant parts allow targeted therapeutic use but depend heavily on extraction methods and solvents, which influence bioavailability (Mukherjee *et al.*, 2017) [15]. Essential oils are highly concentrated and require careful dosing due to risks of toxicity and

skin irritation (Bakkali *et al.*, 2008) [12]. Polyherbal and traditional formulations reflect centuries of empirical use and cultural acceptance but often lack standardization, making clinical validation challenging (Patwardhan *et al.*, 2005) [13]. Isolated phytochemicals align more closely with modern drug discovery, offering precise mechanisms of action, while standardized extracts bridge traditional herbalism and evidence-based medicine by ensuring reproducibility and regulatory compliance (Heinrich *et al.*, 2022). This taxonomy helps avoid terminological ambiguity and ensures clarity in scientific communication,

particularly when comparing clinical outcomes, pharmacological studies, or regulatory frameworks (Ekor, 2014) [11]

Plants with nephroprotective role

Boerhaavia diffusa (BD) and *Tinospora cordifolia* (TC)

Boerhaavia diffusa and *Tinospora cordifolia* are the Indian medicinal plants used as important medicinal preparation to treat various conditions related to the different systems of the body (Mishra *et al.*, 2014) [16]. The antioxidant and nephroprotective effect in diclofenac induced nephrotoxicity of the aqueous extracts of dried roots and stems of BD And TC examined in rats. extract produced significant Renoprotective activity in diclofenac induced nephrotoxicity model as seen by decrease in elevated serum creatinine, serum urea, urine creatinine and BUN levels, which was further confirmed by histopathological study. Extract also shown positive effects against the oxidative stress and inflammation in diclofenac induced model (Khan *et al.*, 2022) [17].

Hibiscus Sabdariffa

The nephroprotective activity of alcoholic extract of *Hibiscus Sabdariffa* leaves Flavonoid (HSLF) extract via KIM-1 and TGF-1 β Signalling Pathways was investigated in Streptozotocin-Induced Rats (Ajiboye *et al.*, 2024) [18]. *Hibiscus sabdariffa*, commonly known as Roselle or *Hibiscus*, originates from West Africa but is now cultivated globally and belongs to the Malvaceae family (Islam *et al.*, 2016) [19]. Treatment with alcoholic extract of HSLF extract prevented the Streptozotocin induced nephrotoxicity and oxidative damage of the kidney, as evidenced by different assays like Renal Serum Electrolyte Levels Biomarker Assays, Serum Kidney Function Biomarker Assays, Kidney Inflammatory Biomarkers Assays and it is further confirmed by the histopathological examination of kidney tissue (Ajiboye *et al.*, 2024) [18].

Rheum officinale (Rhubarb)

Rhubarb is the common name given to dried roots of *Rheum tanguticum*, *Rheum palmatum* L., or *Rheum officinale*, which belongs to the family *Polygonaceae* (Cao *et al.*, 2017) [20]. A wide range of bioactive constituents is extracted from this herb which includes essential oil, anthraquinones, dianthrone, stilbenes, anthocyanins, flavonoids, polyphenols, and chromene derivatives (VanMen *et al.*, 2012; Tan *et al.*, 2016) [21]. Emodin seems to reduce uremic toxin via colonic irrigation in the gut, thereby reducing the progression of CKD. nephropathy causes aberrations in urinary amino acids, purine, and taurine metabolites. These abnormalities in urine were reversed by the treatment with rhubarb at a dose of 20 mg/ml for 6 weeks (Zhang *et al.*, 2015).

Lippia sidoides

Nephroprotective effects of *Lippia sidoides* ethanolic extract (LSEE) was evaluated experimentally against ischemia/ reperfusion-induced acute kidney injury in rats. nephroprotective effect of LSEE was evaluated through estimation of Urinary and plasma concentrations of protein, albumin and urea by using an automatic analyser. oxidative stress parameters are studied via Tubular kidney cells culture and hypoxia/ reoxygenation assay and Flow cytometry assays which shows significant decrease in the oxidative parameters in LSEE treated animals (Costa *et al.*, 2023) [22].

Avena sativa L

Avena sativa (Family Poaceae) popularly known as 'oat' is prevalent all over Europe, Asia, and Northwest Africa. Avenanthramides responsible for antioxidant and anti-inflammatory action is found in oat (Soycan *et al.*, 2019) [23]. It was observed that oat consumption had useful and positive action especially on serum albumin and serum potassium in CKD patients (Rouhani *et al.*, 2018) [24].

Cyperus scariosus Linn.

Nephroprotective Effects of Hydroalcoholic Extract of *Cyperus scariosus* (CS) Was evaluated in Gentamicin induced Acute Kidney Injury in Wistar Albino Rats. Administration of CS at 150 mg/kg and 250 mg/kg doses as preventive and curative therapies restored the altered kidney parameters caused by gentamicin. Histopathological analysis shows that CS as a preventive and curative therapy resulted

in less kidney damage compared to the disease control group, and normal architecture of the kidney was maintained (Gajjar *et al.*, 2016) [25].

Porophyllum ruderale

Porophyllum ruderale is an annual edible herb belonging to the Asteraceae family. Nephroprotective effects of *Porophyllum ruderale* hydroalcoholic extract was evaluated experimentally against Thioacetamide-Induced Injury Model. values of kidney injury biomarkers in urine (urobilinogen, haemoglobin, bilirubin, ketones, glucose, protein, pH, nitrites, leukocytes, specific gravity, and the microalbumin/creatinine) and serum (creatinine, urea, and urea nitrogen) of rats treated with extract were maintained in normal ranges. extract shows a slightly anti-inflammatory effect due to it decreasing the inflammatory process by approximately 10 to 15%. extract showed high antioxidant activity. It was able to stabilize free radicals, showing the highest effect on ABTS⁺ followed by DPPH.

Propolis

Propolis is a product prepared by honeybees naturally from plants. It consists of several important flavonoids, phenolic acids, and esters. Propolis has various biological activities like anti-inflammatory, immunomodulatory, antioxidant, and antiproteinuric effects (Berretta *et al.*, 2012) [26]. Propolis treatment has been shown to attenuate hypertension as well as renal damage in rat model via reducing renal inflammation and oxidative stress (Teles *et al.*, 2015) [27].

Bambusa arundinacea

The nephroprotective activity of the methanolic extract of *Bambusa arundinacea* was tested against gentamicin induced nephrotoxicity in rats. The extent of protection was determined by evaluating urinary creatinine, urinary sodium, urinary potassium, blood urea, serum creatinine levels and body weight of the animals. The *in-vivo* antioxidant activity was determined by estimating the tissue levels of GSH, SOD, catalase and lipid peroxidation. DPPH assay is used to assess the radical scavenging activity. histopathological analysis shown that coadministration of extract with gentamicin reduced the severity of the parameters like degeneration, desquamation, and necrosis in tubules (Iqbal *et al.*, 2022) [28].

Cordyceps sinensis

Cordyceps sinensis (Family Clavicipitaceae) is a fungus, which shows parasitism with Lepidopteran larvae. The major bioactive constituents of *C. sinensis* include polysaccharides, nucleoside, sterols, and proteins. *C. sinensis* together with other conventional therapy has been shown to decrease serum creatinine, increase creatine clearance and reduce proteinuria in patients with CKD (Zhang *et al.*, 2014).

Salvia miltiorrhiza

The major bioactive moieties belong to the class of diterpenoid quinines, phenolic acids and essential oil. The active secondary metabolites include: tanshinone, miltirone, danshexinkun, sugiol, trijuganone, salvianolic acid, caffeic acid, rosmarinic acid and linoleic acid. when Tanshinone IIA was administered to the rats suffering from CKD, TGF β 1 and collagen IV were significantly reduced, along with the increase in urinary protein excretion (Ahn *et al.*, 2010) [29].

Tribulus terrestris (Gokshura)

Tribulus terrestris belonging to the family Zygophyllaceae, has been widely recognized in traditional medicine for its role in urinary tract health. Its active constituents include protodioscin, saponins, and flavonoids, which contribute to its antiurolithiatic, diuretic, and antioxidant properties. Experimental studies have demonstrated that *T. terrestris* prevents calcium oxalate crystal formation in ethylene glycol induced urolithiasis models, while in vitro assays confirm its ability to inhibit nucleation and aggregation of urinary crystals. Clinically, it is often incorporated into polyherbal formulations aimed at supporting urinary tract function and preventing stone recurrence (Kumar *et al.*, 2017).

Bergenia ligulata (Pashanbheda)

Bergenia ligulata a member of the Saxifragaceae family, is another important antiurolithiatic herb traditionally used for kidney stones and urinary infections. Its phytoconstituents bergenin, arbutin, and catechin exhibit potent antioxidant and diuretic activities. In vivo studies

have shown that *B. ligulata* reduces oxalate-induced renal damage in rats, while in vitro experiments highlight its ability to scavenge free radicals and inhibit lipid peroxidation, thereby protecting renal tissue from oxidative stress. Ethnomedical use across India strongly supports its role in dissolving urinary calculi and alleviating urinary tract discomfort (Bahmani *et al.*, 2016) [30].

***Andrographis paniculata* (Kalmegh)**

Andrographis paniculata from the family Acanthaceae, is traditionally valued for its immunomodulatory and detoxifying properties. Its major bioactive compound, andrographolide, along with flavonoids, confers antioxidant and anti-inflammatory effects. Preclinical studies have demonstrated that *A. paniculata* protects against cisplatin-induced renal damage, while in vitro assays reveal its ability to reduce reactive oxygen species (ROS) and suppress TNF- α expression in renal cells. In traditional medicine, Kalmegh has been used for liver and kidney detoxification, reflecting its broad role in systemic protection against toxin-induced injury (Khan *et al.*, 2022) [17].

***Carica papaya* (Papaya)**

Carica papaya belonging to the family Caricaceae, is a tropical plant with well-documented nutritional and medicinal value. Its active constituents include papain, flavonoids, and carotenoids, which contribute to its antioxidant and anti-inflammatory properties. Experimental studies have shown that papaya extracts reduce nephrotoxicity in acetaminophen treated rats, primarily by inhibiting lipid peroxidation and enhancing endogenous antioxidant enzyme activity. Traditional use also supports its role in maintaining renal health, with ripe fruit being considered safe for individuals in early to moderate stages of chronic kidney disease due to its relatively low potassium content (Pachiappan *et al.*, 2025) [31].

Limitations and safety concerns of herbal nephroprotectants

Although medicinal plants show considerable promise in nephroprotection, both the Indian and Nigerian reviews emphasize that their clinical translation is constrained by significant limitations and safety concerns. A primary limitation is the lack of standardized extraction and dosage protocols. As noted in the Nigerian narrative review, herbal remedies are frequently prepared as crude decoctions, infusions, or ethanol extracts under highly variable conditions of solvent selection, extraction time, raw material identity, and storage (Olaniru *et al.*, 2026) [32]. This variability alters the identity and abundance of bioactive constituents, modifies co-extracted compounds that influence efficacy or toxicity, and undermines reproducibility. Without chemistry-defined extract profiles, marker compounds, or chromatographic fingerprints, it becomes difficult to compare outcomes across studies or ensure batch-to-batch consistency. The Indian review similarly highlights that while plants such as *Kalanchoe pinnata*, *Medicago sativa*, and *Musa acuminata* demonstrate renoprotective activity, concerns about dosage standardization and herb-drug interactions remain unresolved (Pachiappan *et al.*, 2025) [31]. Another limitation is the predominance of preclinical evidence with sparse clinical validation. Both reviews emphasize that most findings are derived from animal models or in vitro assays, with very few controlled human trials. For example, *Punica granatum* and *Zingiber officinale* have shown antioxidant and anti-inflammatory effects in haemodialysis patients or nephrotoxicity models, but broader clinical data remain scarce (Pachiappan *et al.*, 2025) [31]. The Nigerian review underscores that many cited plants are supported only by ethnobotanical documentation or non-specific antioxidant studies, whereas relatively few have been investigated in kidney-anchored in vivo models with clinically meaningful endpoints such as serum creatinine, blood urea nitrogen (BUN), proteinuria, or renal histopathology (Olaniru *et al.*, 2026) [32]. This gap between laboratory promise and patient care is a major translational barrier, particularly in chronic kidney disease (CKD), where disease progression is multifactorial and patients often present with comorbidities. Safety concerns are equally pressing. Several plants traditionally used for kidney disorders exhibit dual

pharmacological profiles, appearing nephroprotective under certain conditions but potentially nephrotoxic under others. For instance, *Kalanchoe pinnata* contains bufadienolides, compounds that may disrupt cardiac rhythm, raising concerns about arrhythmogenic toxicity when consumed in uncontrolled doses (Pachiappan *et al.*, 2025) [31]. *Medicago sativa* (alfalfa) is rich in potassium, which can predispose patients with advanced CKD or those on dialysis to hyperkalaemia, a life-threatening electrolyte imbalance. Its amino acid derivative L-canavanine has also been linked to lupus-like autoimmune reactions that may impair renal function. The Nigerian review similarly cautions that several species may demonstrate nephroprotective effects under one extraction method or dose, but become nephrotoxic under chronic exposure, higher doses, or poorly controlled solvent extraction (Olaniru *et al.*, 2026) [32]. This duality underscores the importance of renal-specific toxicology studies, which remain limited in scope and duration.

Herb-drug interactions represent another critical safety issue. Patients with kidney disease are frequently prescribed complex regimens including immunosuppressants, diuretics, antihypertensives, anticoagulants, and antidiabetic agents. Herbal remedies may interact with these drugs, either potentiating adverse effects or reducing therapeutic efficacy. The Indian review notes that alfalfa's interactions with immunosuppressants, diuretics, and anticoagulants complicate its safe use in renal patients (Pachiappan *et al.*, 2025) [31]. The Nigerian review further highlights that concomitant use of herbal remedies with nephrotoxic drugs such as aminoglycosides or chemotherapeutic agents could amplify renal injury rather than mitigate it (Olaniru *et al.*, 2026) [32]. Without systematic herb-drug interaction studies, the integration of phytomedicine into nephrology remains fraught with uncertainty. Contamination and quality control issues further complicate the safety landscape. Herbal preparations may be contaminated with heavy metals, pesticide residues, microbial agents, or adulterants, particularly when sourced from unregulated markets. Patients with CKD are especially vulnerable to such contaminants due to impaired clearance and heightened susceptibility to nephrotoxins. The Nigerian review explicitly notes that cumulative nephrotoxicity, electrolyte disturbances, and adulteration are translational barriers that must be addressed before these remedies can be considered safe for widespread use (Olaniru *et al.*, 2026) [32]. In addition, variability in cultivation practices, seasonal changes, and geographic sourcing can influence phytochemical composition, making quality assurance essential yet often neglected.

In summary, while herbal nephroprotectants offer promising adjunctive strategies for managing kidney diseases, unresolved issues of extract standardisation, dosage optimisation, herb-drug interactions, and long-term safety must be addressed. The Indian review emphasizes the need for caution due to toxicity risks and herb-drug interactions, while the Nigerian review highlights weak extract definition, sparse renal toxicology, and inconsistent quality control as major translational bottlenecks. Future research should integrate ethnopharmacological knowledge with molecular pharmacology and clinical methodologies to ensure that these remedies can be safely and effectively translated into evidence-based kidney care (Pachiappan *et al.*, 2025; Olaniru *et al.*, 2026) [31, 32]. Until such evidence is available, herbal nephroprotectants should be approached with cautious optimism, emphasizing their potential while acknowledging the limitations and risks inherent in their current state of development.

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