Review on Different Kinds of Vegetables with Reference of Nephroprotective Activity

Rupanjali Dobhal¹, Namita Singh¹⁺, Parul Sexna¹, Acharya Balkrishna¹ and P. P. Upadhyaya²

¹Patanjali Research Institute, Haridwar, Uttarakhand, India.  
²Deen Dayal Gorakhpur University, Gorakhpur, U.P, India.

ABSTRACT

In this review articles are aimed to elucidate the list of nephroprotective vegetables, which are scientifically proved in treating renal disorders. Now a day's nephro failure is a serious problem of human being, every one-third family suffers this critical problem of nephro failure. Renal diseases are paramount health issues. Vegetable has emerged as a skilled approach with sensible values in handling in kidney diseases and developing an affordable therapy to treat severe kidney diseases. The use of vegetables as nephroprotective is a major avenue in Indian perspectives particularly for treating kidneys damage, which require to be explored more successfully as there are many literatures available on these aspects. On the basis of pharmacological evidences, vegetables are vital nephroprotective agents. Vegetables possess significant nephroprotective activity against induced screening methods such as cisplatin, gentamicin and paracetamol. Vegetables having

*Corresponding author: E-mail: namitasinghphd123@gmail.com;
possessed great potential source of vitamins and different compounds which are useful for the development of effective therapy to bloodshed types of nephro problems.

Keywords: Nephroprotective activity; vegetables; pharmacological evidence; renal disorders.

1. INTRODUCTION

Nephrotoxicity is one of the most common problems it causes when human body is exposed to a drug or toxin that causes damage to your kidneys. When kidney damage occurs, you are unable to rid your body of excess urine, and wastes. A number of therapeutic agents are adversely affect the nephron resulting in acute nephro failure, chronic interstitial nephritis and nephritic syndrome because there is an increasing number of potent therapeutic drugs like aminoglycoside antibiotics, NSAID’s (Non steroidal anti-inflammatory drugs), chemotherapeutic. Exposure to chemical reagents like ethylene glycol, carbon tetrachloride, sodium oxalate and heavy metals such as lead, mercury, cadmium and arsenic also induces nephrotoxicity. About 2 million people who receive treatment for kidney failure, the majority are treated in only five countries – the United States, Japan, Germany, Brazil, and Italy. These five countries represent only 12% of the world population. Only 20% are treated in about 100 developing countries that make up over 50% of the world population. More than 80% of all patients who receive treatment for nephro failure are in affluent countries with universal access to health care and large elderly populations. Nephroprotective agents are the substances which possess protective activity against nephrotoxicity. Vegetables have defensive properties due to the presence of various complex chemical substances of vegetables which we come across in our daily life. Early literatures have prescribed different kinds of vegetables acts as nephroprotective agents [1,2,3,4].

2. NEPHROPROTECTIVE ACTIVITY ON DIFFERENT VEGETABLES

2.1 Anethum graveolens L.

Aqueous extract of dry fruits of A. graveolens was evaluated for nephroprotective activity against free radicals generated by paracetamol. Plasma total antioxidant capacity, plasma catalase, cellular glutathione peroxidase were determined. In addition, kidney functions (plasma urea and creatinine) were investigated. The significant increase in urea and creatinine in paracetamol-intoxicated rats revealed the toxic effect of paracetamol overdose on kidneys. Dill aqueous extract contains powerful antioxidant components that serve as an extracellular neutralizer of free radicals. Rich polyphenols and volatile compounds in dill possess antioxidant capacity and improve the normal renal function [5].

2.2 Abelmoschus manihot (L.) Medik

A. manihot (L.) has been proved clinically effective in improving renal inflammation and glomerular injury in chronic kidney disease (CKD). However, the dose-effects and the mechanisms involved in vivo are still unclear. This study was performed to examine the dose-effects of Huangkui capsule (HKC) on renal inflammation and glomerular lesion in adriamycin-induced nephropathy (ADRN), then to clarify the mechanisms in vivo of HKC by investigating its actions on modulating the activation of p38 mitogen-activated protein kinase (p38MAPK) signaling pathway. The rats with chronic ADRN, created by the unilateral nephrectomy and twice adriamycin injections (ADR, 4 mg/kg and 2 mg/kg) within 4 weeks, were divided into four groups, a Sham group, a Vehicle group, a high-dose HKC group, and a low-dose HKC group, and that, sacrificed at the end of the 4th week after the administration. The rat’s general status, renal morphological appearance, proteinuria, blood biochemical parameters, glomerular morphological changes, podocyte shape, and macrophage (ED1+ and ED3+ cells) infiltration in glomeruli were examined, respectively. The protein expressions of inflammatory cytokines including tumor necrosis factor (TNF)-α and interleukin (IL)-2, as well as p38MAPK signaling molecules such as transforming growth factor (TGF)-β1, p38MAPK, and phosphorylated-p38MAPK (p-p38MAPK), were also evaluated. HKC at high dose of 2 g/kg/d not only significantly ameliorated the rat’s general status, renal morphological appearance, proteinuria, albumin, and glomerulosclerosis, but also obviously reduced the infiltrated ED1+ and ED3+ macrophages in glomeruli and TNF-α protein expression in the kidney, in addition to these, evidently down-regulated TGF-β1 and p-
p38MAPK protein expressions in ADRN rats, but had no influence on podocyte shape and renal function. HKC could dose-dependently ameliorate renal inflammation and glomerular injury in ADRN rats, by way of reducing the infiltration and the activation of macrophages in glomeruli, and TNF-α protein expression in the kidney, as well as inhibiting p38MAPK signaling pathway activity via the down-regulation of p-p38MAPK and TGF-β1 protein expressions in vivo [6]. Flavonoid fraction of A. manihot (FFA) was extracted from A. manihot flower with ethanol was evaluated for nephron-protective activity. FFA (400 mg/kg) was orally given to normal rats and CKD model rats. Blood samples were collected at 5, 15, 30, 45, 60, 90, 120, 240, 360, and 720 min after administration. The plasma concentrations of quercetin and isorhamnetin glucuronide/sulfate conjugates were analyzed by UPLC-MS/MS. In normal rats, area under the curve (AUC) of quercetin-glucuronide conjugates, isorhamnetin-glucuronide conjugates, quercetin-sulfate conjugates, and isorhamnetin-sulfate conjugates was 459.45 ± 192.70, 1153.01 ± 697.04, 417.81 ± 220.31, and 2475.19 ± 1085.22 μmol h/L, respectively. While AUC of quercetin and isorhamnetin was 5.47 ± 2.54 and 30.73 ± 25.95 μmol h/L. AUC of the glucuronide-sulfate conjugates of quercetin and isorhamnetin is 125-times higher than that of aglycone (quercetin and isorhamnetin), showing that glucuronide/sulfate conjugates represent the major circulating forms of A. manihot flavonoid in vivo. AUC of isorhamnetin-glucuronide conjugates and quercetin-sulfate conjugates was 719.65 ± 619.22 and 275.49 ± 60.95 μmol h/L, indicating that less conjugated metabolites were formed in CKD rats compared with normal rats. The ratio of AUC glucuronide/ sulfate/ AUCaglycone decreased from 125 to 104, which implied the impaired phase II metabolism ability in CKD rat [7].

2.3 Allium sativum L.

Nephroprotective potential of ethanolic extract of A. sativum was evaluated against cisplatin induced nephrotoxicity in wistar male rats. Nephrotoxicity was induced by a single intraperitoneal injection of cisplatin (5 mg/kg b.w.) on the first day of the experiment. The animals were treated with ethanolic extract of garlic at doses 150 mg/kg b.w. and 300 mg/kg b.w. for four consecutive days. The effect of the higher dose (300 mg/kg b.w.) of garlic extract on normal rats was also studied. The animals were sacrificed on the fifth day and enzymatic antioxidants and lipid peroxidation were assessed in kidney whereas urea, creatinine, uric acid and blood urea nitrogen (BUN) were quantified in serum samples. Results showed that cisplatin induction resulted in a decrease in the activities of kidney antioxidants with a concomitant increase in kidney weight, lipid peroxidation along with serum kidney markers like urea, creatinine, uric acid and BUN the results from this study reveals that the ethanolic extract of garlic possess a potential nephroprotective property with no after effects [8].

2.4 Beta vulgaris L.

B. vulgaris root ethanolic extract (BVREE) on gentamicin-induced nephrotoxicity and to elucidate the potential mechanism. Serum specific kidney function parameters (urea, uric acid, total protein, creatinine, and histopathology of kidney tissue) were evaluated to access gentamicin-induced nephrotoxicity. The oxidative/nitrosative stress (Lipid peroxidation, MDA, NP-SH, Catalase, and nitric oxide levels) was assessed. The inflammatory response (TNF-α, IL-6, MPO, NF-κB (p65), and NF-κB (p65) DNA binding) and apoptotic marker (Caspase-3, Bax, and Bcl-2) were also evaluated. BVREE (250 and 500 mg/kg) treatment along with gentamicin restored/increased the renal endogenous antioxidant status. Gentamicin-induced increased renal inflammatory cytokines (TNF-α and IL-6), nuclear protein expression of NF-κB (p65), NF-κB-DNA binding activity, myeloperoxidase (MPO) activity, and nitric oxide level were significantly down regulated upon BVREE treatment [9].

2.5 Benincasa hispida (Thunb.) Cong

The effects of hydro alcoholic whole fruit extract of B. hispida (200 and 400 mg/kg per day p.o.) exhibited against cisplatin and paracetamol induced nephrotoxicity in rats. In another study, ethanolic extract of B. hispida seed (EEBHS), 250 mg/kg and 500 mg/kg orally, was evaluated against gentamicin induced nephrotoxicity in male Wistar Albino rats for nephroprotective activity. Cisplatin (7.5 mg/kg i.p.), paracetamol (750 mg/kg p.o) and gentamicin (80 mg/kg/day i.p.) induced nephrotoxicity, manifested biochemically by a significant increase of urine volume, kidney weight, urinary sodium, urinary potassium, urinary glucose, blood urea, blood creatinine and decreased in body weight, urinary
creatine and blood total protein level with multiple histological damages. Nephrotoxicity was further confirmed by a significant decrease in glutathione (GSH) and increase in lipid peroxides in kidney homogenates. Administration of hydro-alcoholic extract of *B. hispida* (HABH) (200, 400 mg/kg per day p.o.) and EEBHS (250 mg/kg and 500 mg/kg p.o.) produced a significant protection. The amelioration of nephrotoxicity was evidenced by significant reductions in blood urea, blood creatinine, urinary glucose, urinary sodium, urinary potassium, urine volume with a significant weight gain. In addition HABH and EEBHS tended to normalize decreased level of blood total protein and urinary creatinine. Moreover, HABH prevented the rise of lipid peroxidation and the reduction of GSH activities in the kidney. These results suggest that HABH and EEBHS have a protective effect on nephrotoxicity induced by cisplatin, paracetamol and gentamicin [10,11].

2.6 *Curcuma longa* L.

The role of mitochondria in the protective effects of curcumin, a well-known direct and indirect antioxidant, was studied against the renal oxidant damage induced by the hexavalent chromium [Cr (VI)] compound potassium dichromate [K$_2$Cr$_2$O$_7$] in rats. Curcumin was given daily by gavage using three different schemes (1) complete treatment (100, 200, and 400 mg/kg bw 10 days before and 2 days after K$_2$Cr$_2$O$_7$ injection), (2) pretreatment (400 mg/kg bw for 10 days before K$_2$Cr$_2$O$_7$ injection), and (3) posttreatment (400 mg/kg bw 2 days after K$_2$Cr$_2$O$_7$ injection). Rats were sacrificed 48 h later after a single K$_2$Cr$_2$O$_7$ injection (15 mg/kg, sc) to evaluate renal and mitochondrial function and oxidant stress. Curcumin treatment (schemes 1 and 2) attenuated K$_2$Cr$_2$O$_7$-induced renal dysfunction, histological damage, oxidant stress, and the decrease in antioxidant enzyme activity both in kidney tissue and in mitochondria. Curcumin pretreatment attenuated K$_2$Cr$_2$O$_7$-induced mitochondrial dysfunction (alterations in oxygen consumption, ATP content, calcium retention, and mitochondrial membrane potential and decreased activity of complexes I, II, III, and V) but was unable to modify renal and mitochondrial Cr (VI) content or to chelate chromium. Curcumin posttreatment was unable to prevent K$_2$Cr$_2$O$_7$-induced renal dysfunction. In further experiments performed in curcumin (400 mg/kg)-pretreated rats it was found that this antioxidant accumulated in kidney and activated Nrf2 at the time when K$_2$Cr$_2$O$_7$ was injected, suggesting that both direct and indirect antioxidant effects are involved in the protective effects of curcumin. These findings suggest that the preservation of mitochondrial function plays a key role in the protective effects of curcumin pretreatment against K$_2$Cr$_2$O$_7$-induced renal oxidant damage [12]. And other study also conducted which are bifunctional antioxidant curcumin may induce nuclear translocation of Nrf2 and prevents 5/6 nephrectomy (5/6NX)-induced oxidant stress, renal injury, decrease in antioxidant enzymes, and glomerular hypertension and hyperfiltration. Four groups of rats were studied: (1) control, (2) 5/6NX, (3) 5/6NX and (4) CUR (−10). Curcumin was given by gavage to NX5/6 and CUR groups (60 mg/kg/day) starting seven days before surgery. Rats were studied 30 days after NX5/6 or sham surgery. Curcumin attenuated 5/6NX-induced proteinuria, systemic and glomerular hypertension, hyperfiltration, glomerular sclerosis, interstitial fibrosis, interstitial inflammation, and increase in plasma creatinine and blood urea nitrogen. This protective effect was associated with enhanced nuclear translocation of Nrf2 and with prevention of 5/6NX-induced oxidant stress and decrease in the activity of antioxidant enzymes. It is concluded that the protective effect of curcumin against 5/6NX-induced glomerular and systemic hypertension, hyperfiltration, renal dysfunction, and renal injury was associated with the nuclear translocation of Nrf2 and the prevention of both oxidant stress and the decrease of antioxidant enzymes [13].

2.7 *Cucurbita pepo* L.

Ethanolic seed extract of *C. pepo* showing nephroprotective activity against cisplatin (10mg/kg, i.p.) induced nephrotoxicity in adult inbred male Swiss albino mice. Antioxidant studies like nitric oxide scavenging activity, lipid peroxidation (LPO) in kidney also supported the nephroprotective activity of extract. Results showed that LPO in kidney tissue increases in cisplatin treated mice comparing with control where as extract used showed significant decrease of LPO. Nitric oxide scavenging activity showed less in case of cisplatin treated mice with comparing to control, while extract showed significant antioxidant property. Extract showed significant reduction of these abnormalities with comparing to cisplatin treated mice. It was found that, the ethanolic PuSE extract treatment on mice showed significant increase in body weight (%), creatinine clearance and significant decrease in urinary protein (UP), serum total
2.8 Carica papaya Linn.

Effect of the aqueous seed extract of C. papaya extract (CPE) was exhibited against carbon tetrachloride induced renal injury in Wistar rats. Extract was evaluated by pre-treating three groups of rats (made up of six male rats per group) with 100 – 400 mg/kg body weight per oral of the extract for 7 days before challenging with 1.5 ml/kg body weight of 20% carbon tetrachloride in olive oil in addition to the untreated control and model control rats. Also, the time-course effect of 400 mg/kg per oral of the extract was determined at 3 hr pre-, 0 hr, 1 hr post-, 3 hr post-, and 6 hr post-CCl4 induction, respectively, in addition to the untreated control and model control groups. After 72 hours, serum levels of uric acid, urea and creatinine of all study groups were measured using standard procedures. Histological studies of rat kidneys of all study groups were also done. Results showed that intraperitoneal injection of CCl4 caused a significant (p<0.001) elevation in the serum levels of uric acid, urea and creatinine and induced histological features of severe tubulointerstitial necrosis. However, elevations in the measured biochemical parameters were significantly (p<0.05, p<0.01 and p<0.001) attenuated in rats pre-treated with the graded oral doses of the extract, in dose related fashion. Maximum nephroprotection was offered by the extract at 400 mg/kg/day CPE which lasted up to 3 hours post-CCl4 exposure and these biochemical evidences were corroborated by improvements in the renal histological lesions induced by CCl4 intoxication [15]. Aqueous seed extract of C. papaya on gentamicin induced hepatotoxicity and nephrotoxicity in Wistar rats. A control group (Group I, n=12) was compared with rats administrated with 40 mg/kg gentamicin, once daily for 14 days (Groups II, III and IV, n=12 each). The effect of aqueous extract of Carica papaya L. at a dose level of 200 mg/kg (Group III) and taurine @ 1000 mg/kg body weight (Group IV) was compared with gentamicin treated group (Group II). The activities of serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), serum creatinine and uric acid values were significantly increased in rats exposed to gentamicin (Group II [7]. The nephroprotective and ameliorative effects of C. papaya seed extract in paracetamol-induced nephrotoxicity in rats. Thirty two adult male Wistar rats were divided into four groups (n = 8 in each group). Group A (control) animals received normal saline for seven days, group B (paracetamol group) received normal saline, and paracetamol single dose on the 8th day. Group C received C. papaya a extract (CPE) 500 mg/kg, and paracetamol on the 8th day, while group D, rats were pretreated with CPE 750 mg/kg/day, and paracetamol administration on the 8th day. Samples of kidney tissue were removed for histopathological examination. Screening of C. papaya showed presence of nephroprotective phytochemicals. Paracetamol administration resulted in significant elevation of renal function markers. CPE ameliorated the effect of paracetamol by reducing the markers as well as reversing the paracetamol-induced changes in kidney architecture. C. papaya contains nephroprotective phytochemicals and may be useful in preventing kidney damage induced by paracetamol [14].

2.9 Curcuma zanthorrhiza Roxb.

The effect of zanthorrhizol isolated from C. zanthorrhiza was evaluated for nephroprotective activity against cisplatin-induced nephrotoxicity in mice. A single dose of cisplatin (45 mg/kg, i.p.) significantly elevated the levels of blood urea nitrogen, serum creatinine, and the kidney to body weight ratio, but the pretreatment of xanthorrhizol (200 mg/kg/day, per os) for 4 days significantly attenuated the cisplatin-induced nephrotoxicity. The preventive effect of xanthorrhizol was more efficacious than that of curcumin with the same amount (200 mg/kg). However, this effect seemed not to be related with the ability of xanthorrhizol to regulate the DNA-binding activities of transcription factors such as nuclear factor-kappaB (NF-kappaB) and activator protein 1 (AP-1). This is first time the preventive effect of xanthorrhizol on cisplatin-induced nephrotoxicity [16].
<table>
<thead>
<tr>
<th>Sr. no.</th>
<th>Botanical name</th>
<th>Family</th>
<th>Part used</th>
<th>Screening method</th>
<th>Chemical constituents</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Anethum graveolens</td>
<td>Apiaceae</td>
<td>Dry fruits</td>
<td>Paracetamol</td>
<td>Dry fruit contains alliinorhodendrene, andirococalinol, (e)-3-allylانتىة, β- bisabolol-12-oli,y-cadinene, α-</td>
</tr>
<tr>
<td></td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td>[5].</td>
<td>calacorene, camphenol, camphor, β-2-carene, l-carvone, carpophyllene alcohol, (e)-carpophyllene, cis-pinene</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>hydrate, cryptomerider, cumbene, ρ-cymene, (2)-dihydroapofarnesol, dihydro cinnolinal acetate, elemicin, epi-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ρ-iso-bisabolol, β-eudesmol, eugenol, gnamacene, 7-0-hydroxy manool, iso-bevanol acetate,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>limonene, 4-methyl stilbene, (e)-nucifer, osthol, para-cymen-9-ol, γ-patchouline, pinene,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>phellandrene, 2-phenylethyl phenyl acetate, sabine, spathulanol, α-terpinene, terpinyl acetate, trans-β-ocimene,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>vetivene. Flavonoids and alkaloids, saponins, glycosides (antraquinones and anthacyanodides), tannin.</td>
</tr>
<tr>
<td>2.</td>
<td>Abelmoschus manihot</td>
<td>Malvaceae</td>
<td>Flower</td>
<td>Adriamycininduced</td>
<td>Myristicin, cannabinacin, myrcetin-3-O-beta-glycopyranoside, glycerolmonopalmitate, 2, 4-dihydroxy</td>
</tr>
<tr>
<td>(L.)</td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td>[6, 7].</td>
<td>benzoic acid, guanosine, adenosine, maleic acid, hepatriconatoic acid, 1-triacontanol, tetracosane,</td>
</tr>
<tr>
<td></td>
<td>Medik</td>
<td></td>
<td></td>
<td></td>
<td>beta-sitosterol beta-sitosteryl-3-O-beta-D-glucoside [18].</td>
</tr>
<tr>
<td>3.</td>
<td>Allium sativum</td>
<td>Amaryllidaceae</td>
<td>Bulb</td>
<td>Cisplatin</td>
<td>Allicin, allyl-(α)-methyl, germanium, methyl-(α)-allyl, methylis(o)methyl, selenium thiosulfates,</td>
</tr>
<tr>
<td>L.</td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td>induced [8].</td>
<td>trans-1-propenyl, trans-1-propenyl is(o)methyl, trans-1-propenyl [8]. E-Ajino, Z-Ajino,</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>Allicin, Aliin, Allix, y-Glutaryl-S-propenyl cysteine, Diallyl disulphide, Methyl allyl disulfide</td>
</tr>
<tr>
<td>4.</td>
<td>Beta vulgaris</td>
<td>Amaranthaceae</td>
<td>Root</td>
<td>Gentamicininduced</td>
<td>Root contains ascorbic acid, betalamic acid, betanin, p-coumaric acid, cyclodopa glucoside, dihydroxyindole</td>
</tr>
<tr>
<td>L.</td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td>[12].</td>
<td>carboxylic acid, ferulic acid, n-formylcyclopea glucoside, indicaxanthin, isobetanin, norbeinanin,</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>oxalic acid, prebetanin, L-tyrophan, vulgaxanthin i, vulgaxanthin ii [9].</td>
</tr>
<tr>
<td>5.</td>
<td>Benincasa hispida</td>
<td>Cucurbitaceae</td>
<td>Fruit</td>
<td>Cisplatin, gentamicin and paracetamol induced [10, 11].</td>
<td>The fruits contain volatile oils, flavonoids, glycosides, saccharides, proteins, carotenoids, vitamins, minerals, β-</td>
</tr>
<tr>
<td>(Thunb.</td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td></td>
<td>sitosterin and uronic acid Peels contain galactose, glucose, xylose and sorbose, linoleic and oleic The seeds</td>
</tr>
<tr>
<td></td>
<td>Cong</td>
<td></td>
<td></td>
<td></td>
<td>contain linoleic acid palmitic, oleic, and stearic acids [20]. Triterpenoids, 3α,28-O-dii-trans-cinnamolyl-D-C-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>friedoolcana-7,9(11)-diene, oleonic acid 28-O-[β-o-xilopyranosyl]-[β-o-xilopyranosyl]-[1→(3)]-[1→(3)]</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>thamnopyranosyl-1(2)-c-o-arabinopyranosid, oleic acid 28-O-[β-o-glucopyranosyl]-[1→(3)]-[β-o-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>xilopyranosyl]-[β-o-xilopyranosyl]-[1→(4)]-[1→(3)]-o-carboharmnopyranosyl-[1→(2)-c-o-]</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>arabinopyranosid, multiflorol, isomultiflorol acetate stigmasterol stigmasterol-3-O-beta-glycopyranoside, α</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>spinasterol, α-spinasterol 3-O-beta-glucopyranoside, β-sitosterol, daucosterol, arbutin, nicotinic acid, (++)-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>pinonesinol and ethyl β-glucopyranoside [21].</td>
</tr>
<tr>
<td>6.</td>
<td>Curcuma longa</td>
<td>Zingiberaceae</td>
<td>Rhizome</td>
<td>Attenuated K(2)(Cr2)O(7)-</td>
<td>Essential oil contains α-eucalyptol α-pinene, β-phellandrene β-pinene limonene 1,3,8-p-menthatene ,</td>
</tr>
<tr>
<td>L.</td>
<td>(L.) Dobhal</td>
<td></td>
<td></td>
<td>and nephrectomy (5/6NX)-</td>
<td>ascaridole epoxide, 2-methylisoborneol, 5-isopropyl-6-methyl-hepta-3, dien-2-oi [22]. curcumin (curcumin I)</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td>induced [12, 13].</td>
<td>Diarylehtanoid , demethoxycurcumin (curcumin II) Diarylehtanoid , 1-(4-hydroxy-3-methoxyphenyl)-7-(3,4-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>dihydroxyphenyl)-1, 6-heptadiene-3, 5-dione Diarylehtanoid , 1-(4-hydroxyphenyl)-7-(3,4-dihydroxyphenyl)-1, 6-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>heptadiene-3, 5-dione Diarylehtanoid , bisdemethoxycurcumin (curcumin III) Diarylehtanoid , 6</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>tetrahydroxycurcumin Diarylehtanoid, 5-hydroxy-1-(4-hydroxy-3-methoxyphenyl)-7-(4-hydroxyphenyl)</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>4,6-heptadiene-3-one Diarylehtanoid , 5-hydroxy-1,7-bis(4-hydroxy-3-methoxyphenyl)-4,6-heptadiene-3-one Diarylehtanoid , 1,7-bis(4-hydroxyphenyl)-1-heptene-3,5-dione Diarylehtanoid , 5-hydroxy-1-(4-hydroxy-3-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>methoxyphenyl)-1-(4-hydroxyphenyl)-4,6-heptadiene-3-one. Diarylehtanoid, 5-hydroxy-1,7-bis(4-</td>
</tr>
<tr>
<td></td>
<td>Medic</td>
<td></td>
<td></td>
<td></td>
<td>hydroxyphenyl)-1,7-heptene-1,8-dione Diarylehtanoid , 1,5-dihydroxy-1-(4-hydroxy-3-methoxyphenyl)-7-(4-</td>
</tr>
</tbody>
</table>

**Table 1. List of different vegetables having nephroprotective activity**
<table>
<thead>
<tr>
<th>Sr. no.</th>
<th>Botanical name</th>
<th>Family</th>
<th>Part used</th>
<th>Screening method</th>
<th>Chemical constituents</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Cucurbita pepo L.</td>
<td>Cucurbitaceae</td>
<td>Seed</td>
<td>Cisplatin induced [14]</td>
<td>Seed contains flavonoids, phenols, alkaloids, protein, terpenoids, carbohydrates, steroids, tannins, glycosides, terpenes and saponins. [10] tetrahydro-thiophene, linoleic acid, calotropoisley ester, cholesterol, and [18]-oleaneen-3-ol dodecane and tetradecane [24].</td>
</tr>
<tr>
<td>9</td>
<td>Curcuma xanthorrhiza Roxb.</td>
<td>Zingiberaceae</td>
<td>Rhizome</td>
<td>Cisplatin induced [16]</td>
<td>Rhizome contains xanthorrhizol [16], alkaloid, quinone, and terpenoids [26].</td>
</tr>
<tr>
<td>10</td>
<td>Camarum schweinfurtii Engl.</td>
<td>Burseraceae</td>
<td>Stem bark</td>
<td>Acetaminophen induced [27]</td>
<td>Stem bark contains flavonoids, glycosides, lipids, proteins, saponins, steroids, triterpenes [27].</td>
</tr>
<tr>
<td>Sr. no.</td>
<td>Botanical name</td>
<td>Family</td>
<td>Part used</td>
<td>Screening method</td>
<td>Chemical costitutes</td>
</tr>
<tr>
<td>--------</td>
<td>----------------</td>
<td>--------</td>
<td>-----------</td>
<td>------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>13.</td>
<td>Ipomoea aquatica Forsk.</td>
<td>Convolvulaceae</td>
<td>Leaves</td>
<td>Gentamicininduced [34].</td>
<td>Leaves contains flavonoids, amino acids, alkaloids, lipids, steroids, saponin, phenols, reducing sugar, tannins, β-carotene, glycosides, and minerals,The plant is found to contain vitamins such as A, B1, B2, B6, B12, C, E, K and &quot;U&quot; (S-methyl-methionine aliphatic pyrrolidone amides, carotenoids, henriacotante, β-sitosterol, glycosides, prostaglandin, leukotriene, N-trans and N-cis-fenuloytyramines [9,35-39], like aspatic acid, threonine, serine, glutamic acid, proline, glycine, alanine, leucine, tyrosine, lysine, histidine, and arginine and sugars like glucose, fructose, sucrose), and starch and organic acids such as malic acid, citric acid, and oxalic acid and minerals like sodium, potassium, calcium, iron, magnesium, and zinc. Polyphenols such as myricetin, quercetin, luteolin, apigenin, and kaempferol and various types of chlorophylls, carotenoids viz. lutein, anthracanthin, flavoxanthin, auroxanthin, luteoxanthin, neoxanthin, B-carotene, violoxanthin, cryptoxanthin, neoxanthin A and neoxanthin B and polyphenolcivz quercetin 3'-methyl ether, quercetin 4'-methyl ether and anthocyanins [40], vitamins A, B, C, E, and &quot;U&quot; (S-methyl-methionine), aliphatic pyrrolidone amides, carotenoids, henriacotante, β-sitosterol and its glycosides, prostaglandin, leukotrine, N-trans and N-cis fenuloytyramines [41], Flavonoid, glycosids, phenol, tanin, β-carotene, cyanogenic glycosides, steroids, saponins, reducing sugars and soluble carbohydrate [42].</td>
</tr>
<tr>
<td>Sr. no.</td>
<td>Botanical name</td>
<td>Family</td>
<td>Part used</td>
<td>Screening method</td>
<td>Chemical costitues</td>
</tr>
<tr>
<td>--------</td>
<td>---------------------------------</td>
<td>--------------</td>
<td>-----------</td>
<td>--------------------------------------</td>
<td>-----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>16.</td>
<td><strong>Lepidium sativum</strong> L.</td>
<td>Brassicaceae</td>
<td>Seed</td>
<td>Cisplatin induced [51,52].</td>
<td>Seed contains amino acids (glycine, cysteine, and glutamine), carbohydrate, fatty acid, fatty oils, flavonoids, glycoside, isochoitynates, tannin, tannins, vitamins (ascorbic acid, β-carotene, niacin, and riboflavin), volatile essential aromatic oils [51,52].</td>
</tr>
<tr>
<td>17.</td>
<td><strong>Momordica charantia</strong> L.</td>
<td>Cucurbitaceae</td>
<td>Fruit</td>
<td>Gentamicin-induced [53].</td>
<td>Fruit essential oil contains hexacosane, pentacosane, heptacosane [54]. Vitamin A (Beta-carotene), vitamin B-choline, vitamin B1-thiamine, riboflavin, nicotinic acid and ascorbic acid [Arg, His, Lys, Trp, Phe, Thr, Leu, Met, ile, Val, tannins, steroids, saponins, trepenoids, phenolics, alkaloids and flavanoids like quercitin, isoquercitin, kaemifericitin, isochoitynates and glycoside [54]. Courmarins, benzopyron, chlorine, bromine, and phosphorus, mescaline, serotonin, dopamine, antiraphuiones are a group of naturally occurring phenolic compounds [55],. Leaves contains oleic acid (Ben oil), pterygospermin, Linoelic acid, linoelic acid, benheic acid, tannins, saponin, phenolics, pyflavanes, terpenoids and lectins, fats, fiber, proteins, minerals, vitamins like A, B, C and amino acids. Seed alkaloids, morphine, morginine, minerals like calcium, magnesium and sodium. Brak contains calcium and potassium and amino acids. Flower contain rich in fiber, lipids, non-structural carbohydrates, protein and ash. Fatty acids like oleic acid, linoleic acid, palmitic acid and linolenic acid are also present [54]. pentacosane, hexacosane and (E)-phytol, thymol hexanoic acid, Nonoacasone, 1,2,4-trimethyl-benzene and heptacosane, nonacoseane, pentacosane, pentacosane [54]. The seeds and root bark yielded 4α-(α-L-rhamnosyloxy) benzyl isothiocyanate, 4α-(α-O-acetyl-α-L-rhamnosyloxy) benzyl isothiocyanate, 4α-(α-Lrhamnosyloxy) benzylisothiocyanate, squalene and β-sitosterol. 4-(4-O-acetyl-α-L-rhamnosyloxy) benzylisothiocyanate and 4-(α-L-rhamnosyloxy) benzylisothiocyanate, doxorubnic [56]. Root bark contain, niazicin, triolein, β-sitosterol oleate, β-sitosterol, stigmastrol, oleic acid, and 1-octadecene [56].</td>
</tr>
<tr>
<td>18.</td>
<td><strong>Momordica tuberosa</strong> Cogn. syn. Of <strong>Momordica dioica</strong> Roxb. ex Wild</td>
<td>Cucurbitaceae</td>
<td>Tuber, Fruit</td>
<td>Cisplatin, gentamicin and paracetamol induced [57,58].</td>
<td>Tuber and fruit contains cardiac glycosides, flavonoids, phenolics, saponins, steroids, triterpenoids, vitamin C [57,58].</td>
</tr>
<tr>
<td>19.</td>
<td><strong>Moringa pterygosperma</strong> Gaerth.</td>
<td>Moringaceae</td>
<td>Leaf</td>
<td>Paracetamol induced [59].</td>
<td>Leaves contain ascorbic acid, alpha-tocopherol, beta-carotene, calcium, deic acid, folic acid, glycoside, gum, iron, magnesium, moringine, moringinine nicotinic, palmitic acid, phosphorus, pterygospermin, pyridoxine, riboflavin, retinoic acid, saponins, steenic acid, thiamine [59].</td>
</tr>
<tr>
<td>20.</td>
<td><strong>Moringa oleifera</strong> Lam</td>
<td>Moringaceae</td>
<td>Fruit</td>
<td>DMBA induced [60].</td>
<td>Leaves contain ascorbic acid, alpha-tocopherol, beta-carotene, calcium, deic acid, folic acid, glycoside, gum, iron, magnesium, moringine, moringinine nicotinic, palmitic acid, phosphorus, pterygospermin, pyridoxine, riboflavin, retinoic acid, saponins, steenic acid, thiamine [59].</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sr. no.</th>
<th>Botanical name</th>
<th>Family</th>
<th>Part used</th>
<th>Screening method</th>
<th>Chemical costitutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>23.</td>
<td><em>Spondias pinnata</em> (L. f.) Kurz</td>
<td>Anacardiaceae</td>
<td>Fruit</td>
<td>STZ induced [29].</td>
<td>Steroids, flavonoids, terpenoids, vitamins, amino acids, sugars, alkaloids, flavonoids, triterpenoids, saponins, steroidal saponins.</td>
</tr>
<tr>
<td>Sr. no.</td>
<td>Botanical name</td>
<td>Family</td>
<td>Part used</td>
<td>Screening method</td>
<td>Chemical constituents</td>
</tr>
<tr>
<td>--------</td>
<td>------------------------</td>
<td>--------------</td>
<td>------------------</td>
<td>----------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
Zingiberaceae  
Rhizome  
Doxorubicin induced [69].  
Rhizome contain sesqui-terpenoids, geranial, neral, 1,8-cineole, zingerone, β-bisabolone, β-sesquiphellandrene , (E)-(E)-farnesene, viridiflorol and (E)-(E)-farnesial [70].zingiberene, β-bisabolone-α- farnesene, β-sesquiphellandrene, monoterpene hydrocarbons, o-cumurceme and phenolic compounds which are gingerol and shogaol [71], beta-sitosterol palmitate, isovanillin, glucol monopalmoilate, hexacosanonic acid 2,3-dihydroxypropyl ester, maleimide-5-oxime, p-hydroxybenzaldehyde, adenine-6-gingerol, 6-shogaol and 1- (omega-feruloyloxyceryl) glycerol [72]. Cineole, Camphol, Cyclosativam, Tricyclo, Beta Farnesene, Dodecatetra-1-ol, Spiro [8,9] dec-7ene, o-Curcumene, α-Gingerole, α-Farnesene, Cyclo Hexane, Y. Cadinen, β Sesquiphellandrene, o-Panosinens, Nerylodol B, Guaiol, Naphthalene, Rosafiol, β-bisabolol, Farnesol, Widdrol, Sobivol, Heptene, 2-methyl-6-p-tolyl , Farnesene epoxide , 2,5 Dibutyl Furane, Nerolidol acetate, Cis-6-Shagole, Gingerol, Capsaicin, Trans-10-Shagole, δ Topocopherol, Beta Farnesene, Spiro [8,9] dec-7ene, αCurcume, α-Gingerene, α-Fernesene, Dodecatetra-1-ol , Y Cadinen, β Sesquiphellandrene , Nerylodol B, Zingiberenol, Guaiol, Dimethyl-3,8 Nonadien-2-one, Sesquisabinene Hydrate, Rosafiol, β-bisabolol,Farnesol,Germacron2-Notbornamone, Thiofenchone Veridiflorol, Diplo αCedreneoxepide,Methyl Icosanoate,Verbenol 3 Caren, Ar-Curcumene Carveol, β-pinene, 3(3-acetylmethyl) Methyl Linoleate 2,5 dibutylfururan, Decalin, 1-methoxymethyl, norolyl propionate, cis-6-shagao, norolylpropionate, 2-formylhexadecane, larciresinol, tran-10- shagao δ -tocopherol, matairesinol [73].  

Zingiberaceae  
Rhizome  
Paracetamol induced [74].  
Rhizomes, leaves and flowers yields zerumbone, α-humulene, and camphene, (E)-nerolidol, β-caryophyllene, linalool α- and β-pinenes, [75,76]. Leaves and flowers also contain zingeribene phenolic, saponins, and terpenoids [76]. The essential oil extracted from the rhizome zerumbone α-caryophyllene, camphene, eucalyptol and camphor [77], tricylene, Camphene, 3-Carene, β-Cymene, Eucalyptol Limonene Eucalyptol, Linalool, Camphor, Borneol, 4-Terpineol, β-Terpinyl acetate, o-Terpinol . Caryophyllene.o-Caryophyllene,2,4 Disisopropenyl,1-methylcyclohexane,Cycloheptane.4methylene-1-methyl-2-(2-methyl-1-propen-1-1-yl)-1-1-vinyl, Anisole, o-p-stry-0.41, 1.8,10-Dodecatetra-3-ol, 3,7,11-trimethyl, -(8-2), trans-Nerylodol 0.45Caryophyllene oxide 2.64 Germacrene D-4-ol, 1,2-Dihyropropydine, 1-(1-oxobutyl), 5.82 Caryophyllene oxide,3-Cyclohexen-1-carboxaldehyde,3,4-dimethyl- 3.91 1,5,5,8,5-Tetramethyl-12-oxacyclo[10.0.0]dodeca-3,7-diene,Azulene 1,2,3,4,5,6,7,8-octahydro-1,4-dimethyl-7-(1-methylindolinen)-,1(S)-cis, 2-  
Naphtahilenemethanol 1,2,3,4,5,6,7,8-octahydro alpha, alpha, 4a,8,8-tetramethy1-,(2R-cis,2,6-Dimethyl bicycle [3,2,1]octene 0.69 Bicyclo[3.1.0]hexane-6-methanol, 2-hydroxy-1,4,4,4-trimethyl-7-Octyldienecyclo[4.1.0] heptan-rich.60 Kauran-18-al, 17-(acetyloxyl)-, (4beta)-: 2.16, 1,5-Cyclocucadecan, 8,8-dimethyl-9-methylene 1,13 1H-Cycloprop[ea]zaleum-4-ol, dehydro-1,1,4,7-tetramethyl-, [1α]  
(1α,α,α,α,methyl)-3isopropyltricyclo [4.3.1.1.2,5] undec-3-en-0-ol 4-isopropenyl-4,7-dimethyl-1-oxaspiro, octane β-Eudesmol0.712-Methylencholest-4-one, Agerosiphol , Carveol, 9-Dimethyldodecycyclohexap (d) inden-3-one, Nortronymodre, trans-Longipinene, Bicycle,decane, 2-methylen-5-(1-methylvinyl)-6-methyl, Cyclohexane, 4-methylene-1-methyl-2-(2-methyl-1-propen-1-yl)-1-vinyl [78]. humulon,monoterpenes, zerumbone (2,6,10-cyclocucatetra-1-one, 2,6,8,9-tetramethyl-,(E,E,E), humulene (humulone monoxide and humulene dioxide), α-pinene, β-pinene, Δ3-carene, camphor, β-
<table>
<thead>
<tr>
<th>Sr. no.</th>
<th>Botanical name</th>
<th>Family</th>
<th>Part used</th>
<th>Screening method</th>
<th>Chemical constituents</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>caryophyllene, ar-curcumene, zerumbone, humulene oxide, humulene dioxime, linalool, borneol, α-terpineol, unidentified sesquiterpene ketones and sesquiterpene alcohol. sesquiterpenoids (e.g., humulene epoxide-I, humulene epoxide-II, humulolollll, dihydro-ψ-photo-zerumbone and ψ-photozerumbone) and sesquiterpene alcohols (humulenol-I and humulenol-III)-0-diacylatedielenin),(zerumbone, zerumbone epoxide, diferuloylemethane, feruloyl-p-coumaroyl-methane and di-p-coumaroyl-methane), (3-O-(2-O-acetyl-α-L-rhamnopyranoside), 3-O-(3-O-acetyl-α-L-rhamnopyranoside), 3-O-(4-O-acetyl-α-L-rhamnopyranoside)),(3-O-α-rhamnopyranoside) kaempferol glycosides, (2)-nerolidol. Stems, leaves, and flowers, but not rhizomes, and found zerumbone vanillin and kaempferol , kaempferol-3-O-rhamnoside, kaempferol-3-O-(2 &quot;- or 3 &quot;-acetyl) rhamnoside, kaempferol-3-O-(4 &quot;-acetyl) rhamnoside, kaempferol-3-O-(3 &quot;<em>, 4 &quot;-diacetyl) rhamnoside and kaempferol-3-O-(2 &quot;</em>, 4 &quot;-diacetyl) rhamnoside β-caryophyllene, caryophyllene oxide and β-eudesmol , zerumbone , 4-terpinenol (zerumbone), one flavone (3-O-methyl kaempferol), two flavonoid glycosides (kaempferol-3-O-(2,4-di-O-acetyl-α-L-rhamnopyranoside),kaempferol-3-O-(3,4-di-O-acetyl-αLrhamnopyranoside))phdroxybenzaldehyde, vanillin [76].</td>
</tr>
</tbody>
</table>
2.10 Canarium schweinfurtii Engl

The protective effects of aqueous and methanol extracts of stem bark of C. schweinfurtii on the kidney when acetaminophen is used to induce renal injury in rats. This study is aimed at evaluating the protective effects of aqueous and methanol extracts of stem bark of C. schweinfurtii on the kidney when acetaminophen is used to induce renal injury in rats. Blood urea and serum creatinine levels were significantly higher (p<0.01) in acetaminophen and negative control groups compared to baseline control group and the AE and ME groups. Histopathological examination shows that the extracts preserved the renal histoarchitecture while the acetaminophen and negative control groups showed varying degrees of inflammatory cells infiltration, necrosis, tubular casts, tubular erosion and increased urinary pole [27].

2.11 Cucumis melo Linn

The seed exhibited nephroprotective activity of methanolic extract of C. melo (ME-CM) seed kernel in gentamicin-induced nephrotoxicity. The ME-CM was administrated orally (190 mg/kg/d) for 8 days. Gentamicin was administered at the dose of 100 mg/kg daily in neck region subcutaneously from 4th to 8th day. Gentamicin (alone) treated group showed increased levels of blood urea nitrogen and serum creatinine, which were significantly retrieved in group pretreated with ME-CM. The antioxidant study revealed that the level of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPOx) and reduced glutathione (GSH) were increased with decrease in malondialdehyde (MDA) content in ME-CM pretreated group when compared with gentamicin alone treated group. The histopathological analysis also showed the protective nature of ME-CM in gentamicin-induced renal damage [32].

2.12 Daucus carota L.

Nephroprotective effects of ethanolic root extract of DC against gentamicin-induced nephrotoxicity in Albino Wistar rats were induced by intraperitoneal administration of gentamicin (100 mg/kg/day) for 8 days. Rats of either sex were divided into four groups (n = 6). Group 1 served as control that received normal saline (i.p.) whereas Group 2 (GM) was treated with gentamicin which served as gentamicin-intoxicated group. Group 3–4 (DC200, DC 400) were pretreated with DC at doses of 200 mg/kg and 400 mg/kg (p.o.), respectively, 1 h before the gentamicin intoxication. Following treatment, the nephroprotective effects of DC were evaluated by using serum levels of urea, blood urea nitrogen (BUN), uric acid, and creatinine levels; change in body weight and wet kidney weight along with the histological observations among the experimental groups. Gentamicin intoxication induced elevated serum urea, BUN, uric acid, and creatinine levels which was found to be significantly (P < 0.01) decreased in a dose-dependent manner in groups received DC which was also evidenced by the histological observations. DC showed a significant nephroprotective effect in a dose-dependent manner by ameliorating the gentamicin-induced nephrotoxicity [28].

2.13 Ipomea aquatica Forsk.

The fresh leaves of water spinach I. aquatica ethanol extract (IAE) against gentamicin (GM)-induced nephrotoxicity in wistar albino rats. The rats were divided into four groups as follows: control group, GM group (80 mg/kg/day intraperitoneal (i.p.), IAE + GM group (250 mg/kg orally + 80 mg/kg i.p.) and IAE + GM group (500 mg/kg orally + 80 mg/kg i.p.). IAE extract commenced 6 days prior to the GM injection, and continued for the next 8 days (cotreated with GM). Gentamicin when administered induced a marked renal failure characterized by a significant increase in serum and urine creatinine, urea, uric acid, gamma-glutamyltransferase and protein levels. The elevation of malondialdehyde (MDA), decreased concentration of total proteins (TP) and nonprotein sulphydryl (NP-SH) in kidney tissue are indicators of oxidative stress in the kidney. The extract also significantly reduced the gentamicin-induced elevated serum and urine, protein, creatinine, urea, uric acid and gamma-glutamyltransferase (GGT). The tissue malondialdehyde level also significantly diminished; the decreased NP-SH and total protein levels were significantly replenished by ethanolic extract of water spinach treatment. The experimental results suggest that IAE extract protected Gentamicin-induced nephrotoxicity possibly by enhancing renal antioxidant system [34].

2.14 Luffa acutangula (L.) Roxb

The protective effect of hydro-alcoholic extract of L. acutangula fruit (HAELA) was evaluated
against doxorubicin (DXR) induced nephrotoxicity in mice by studying various serum biomarkers, antioxidants in target organs and histoarchitecture alterations. Pretreatment with HAE LA reversed significantly the elevated serum biomarkers, alanine amino transferase, lactate dehydrogenase and creatinine phosphokinase in kidney in DXR treated mice. In addition, HAE LA treatment inhibited elevated malondialdehyde formation and restored the depleted glutathione, catalase, superoxide dismutase in kidney tissue. The altered histoarchitecture of kidney tissue due to DXR treatment was also improved with HAE LA. The protective activity observed with HAE LA on DXR induced nephrotoxicity in mice was found to be related to its antioxidant property which finally results in membrane stabilization [43].

2.15 Lagenaria siceraria (Molina) Standl

Methanolic extract of seeds of L. siceraria was evaluated for nephroprotective activity against rifampicin-induced toxicities in rats. Nephrotoxicity was induced in adult albino rats of either sex by intra-gastric intubation of rifampicin (1gm/kg p.o. every 72 h for 10 days). Methanolic extract of L. siceraria (MELS) was administered to the experimental rats (250 mg/kg p.o every 72 h for 10 days). Kidney function markers like blood urea nitrogen (BUN), serum creatinine, serum uric acid were significantly increased and serum total protein was decreased in rifampicin treated animals. However, their levels were found to be reversed in the extract pretreated group. Histopathological studies have also shown normal architecture in group treated with methanolic extract (P<0.001) as compared to control and standard groups further evidenced the protective role of the extract [47].

2.16 Lepidium sativum L.

Potential nephrocurative & nephroprotective activity of 400mg/kg ethanolic extract of L. sativum L. seed was used to against cisplatin (5 mg/kg, i.p.) induced nephrotoxicity. The experimental protocol designed as the animals were divided into six groups (n=4) like control, model control, curative (400 mg/kg) and protective groups (400 mg/kg) were received vehicle, cisplatin, cisplatin + extract, and extract + cisplatin respectively. After 6th days, blood collected from retro-orbital sinus of rats and determined urea and creatinine level in serum of each group after then rats were sacrificed for quantitative estimation of various enzymes and ATPase content in kidney tissue. A single dose of cisplatin induced loss in body weight, increase urine excretion, increased urea & creatinine level in serum; it was significantly recovered by 400 mg/kg in curative and protective groups. The enzyme estimation in kidney tissue it found that increase malondialdehyde, superoxide dismutase, catalase and reduced glutathione level, it was significantly monitored by 400mg/kg in curative and protective groups. The level of brush border enzymes like Na+/K+ ATPase, Ca++ATPase and Mg++ATPase were found significantly reduced after single dose cisplatin injection. It was overcome by treatment of same extract in curative and protective groups [51]. Potential nephrocurative & nephroprotective activity of 200 mg/kg ethanolic extract of L. sativum L. seed was use to against cisplatin (5 mg/kg, i.p.) induced nephrotoxicity. The experimental protocol designed as the animals were divided into four groups (n=6) like control, model control, curative (200 mg/kg) and protective groups (200 mg/kg) were received vehicle, cisplatin, cisplatin + extract, and extract + cisplatin respectively. After 6th days, blood collected from retro-orbital sinus of rats and determined urea and creatinine level in serum of each group after then rats were sacrificed for quantitative estimation of various enzymes and ATPase content in kidney tissue. A single dose of cisplatin induced loss in body weight, increase urine excretion, increased urea & creatinine level in serum; it was significantly recovered by 200 mg/kg in curative and protective groups. The enzyme estimation in kidney tissue it found that increase malondialdehyde, superoxide dismutase, catalase and reduced glutathione level, it was significantly monitored by 200 mg/kg in curative and protective groups. The level of brush border enzymes like Na+/K+ ATPase, Ca++ ATPase and Mg++ATPase were found significantly reduced after single dose cisplatin injection. It was overcome by treatment of same extract in curative and protective groups. Finally it is concluded that the present study data conformed nephrotoxicity induced by cisplatin due oxidative stress and ethanolic extract of L. sativum L. seeds may have nephroprotective and curative activity [52].

2.17 Momordica charantia (MC)

Aqueous extract of leaves of M. charantia showing effect on gentamicin (control drug) induced nephrotoxicity in albino wistar rats. Gentamicin (40mg/kg/day, i.p.) was administered to all the groups except normal control group for
14 days. Extract (100 mg/kg/day & 200 mg/kg/day, p.o.) was given orally before intoxication of each dose for 14 days. On 15th day, blood samples for serum urea and creatinine, were withdrawn by puncturing retro orbital sinus. Single daily administration of gentamicin (40 mg/kg/day, i.p.) for 14 days, was associated with significantly elevation (P<0.05) in circulating level of serum creatinine & blood urea in positive control group rats (Gentamicin 40 mg/kg/day, i.p.) when compared with group I (normal control). However, significantly elevation in serum concentration of these measured parameter were significantly (P<0.01) attenuated by the two doses of aq. extract of MC leaves (100 mg/kg/day & 200 mg/kg/day, p.o.), also in dose dependent manner, the overall interpretation is aqueous extract of MC leaves can offer protection against the deleterious renal side effect of gentamicin [53].

2.18 Momordica tuberosa Cogn.

Hydroalcoholic extract in 70% ethanol of tubers of M. tuberosa exhibited nephroprotective activity in gentamicin, cisplatin and paracetamol induced renal damage in wistar rats. The gentamicin administration caused reduction in body weight up to 10.31% and increased blood urea to 59.68 mg/dl and creatinine to 1.74 mg/dl. The extract restored the weight reduction to 6% and blood urea to 30.23 mg/dl at 40 mg/kg dose. The creatinine was significantly reduced to 1.12 mg/dl at the same dose. Administration of cisplatin caused decrease in body weight up to 14.07 %. The treatment with the extract restored body weights in dose dependent manner with 40 mg/kg dose restoring it to 6.57%. Blood urea nitrogen level which increased with cisplatin treatment to 83.78 mg/dl, was restored to 35.32 mg/dl with 40 mg/kg dose of the extract. Serum creatinine level which, increased to 2.53 mg/dl was restored to 0.85 mg/dl with 40 mg/kg dose. Paracetamol administration at a dose equal to 2 g/kg dose revealed an increase in blood urea and serum creatinine levels (56.39 and 1.96 mg/dl) as compared with control (29.28 and 0.68 mg/dl). The extract restored their levels significantly to near normal at 33.68 and 0.79 mg/dl respectively at a dose equivalent to 40 mg/kg [52]. The Ethanolic extract of M. dioica fruit extract (200 mg kg−1) was studied for nephroprotective and curative activities against cisplatin-induced nephrotoxicity. Chloroform, ethyl acetate, ethanol and aqueous extracts were prepared. Blood urea and serum creatinine were analysed as biochemical markers of nephrotoxicity. Reduced glutathione (GSH) and the product of lipid peroxidation (MDA) were also measured in kidney tissues. A single dose of cisplatin resulted in significant reduction in body weight and increased the urea and creatinine levels. Extract administration has shown significant recovery in the levels of these biochemistries in curative (p < 0.001) and protective groups, whereas a single dose of cisplatin caused significant reduction in GSH and an increase in malondialdehyde production. This study suggested that the nephroprotective and curative activities of M. dioica fruit extract are due to its antioxidant activity [58].

2.19 Moringa pterygosperma Gaertn.

The nephro protective effect of ethanolic leaf extract of M. pterygosperma against paracetamol induced nephrotoxicity in rats. The study is carried out by using five groups of rats. Furosemide was taken as standard drug. The parameters estimated are RBC content, haemoglobin content, urea and creatinine levels. The extract showed nephro-protective activity by significantly reducing the levels of blood urea, serum creatinine, increasing the red blood cell count and haemoglobin content (P<0.01) [59].

2.20 Moringa oleifera Lam.

Hydro-ethanolic extract of pods of M. oleifera was evaluated on 7, 12-dimethylbenz (a) anthracene (DMBA) induced renal carcinogenicity. Groups of 10 male mice were preadministered with MO (200 and 400 mg/kg body weight) and standard (0.5% BHA) for 14 days prior to a single dose of DMBA (15 mg/kg; p.o.). The therapeutic efficacy of drumstick extract was observed in terms of normalization of altered renal oxidative stress parameters like LPO, SOD and CAT in kidney of mice. DMBA exposure elicited a significant escalation in LPO level and depletion in antioxidant enzymes namely superoxide dismutase and catalase. Investigated parameters were restored, nearly to the normal values, after MO extract treatment. These results suggested that MO extract could act against DMBA-induced kidney injury in mice by a mechanism related to its antioxidant properties [60].

2.21 Murraya koenigii (L.) Spreng

The ethanolic and aqueous extracts of stem for nephroprotective activity were done by gentamicin induced nephrotoxicity in Rats. The
serum creatinine and blood urea nitrogen (BUN) were found to be significantly increased in rats treated with only gentamicin, whereas treatment with the ethanolic and aqueous extracts of stems of *M. koenigii* reversed the effect of gentamicin indicating nephroprotective activity. Among various doses, the aqueous extract of dose 400 mg/kg has shown good nephroprotective activity [35].

### 2.22 *Portulaca oleracea* L.

*P. oleracea* extract and fish oil were evaluated for nephrotoxicity against gentamicin induced nephrotoxicity in albino rats. The effect of gentamicin (80 mg/kg BW/day) without or with oral administration of aqueous *P. oleracea* extract (400 mg/kg BW/day) and fish oil (5 mg/kg BW/day) co-treatments for 15 days was evaluated in adult male rats (80-120 g). Plasma urea, uric acid and creatinine levels were assayed. Lipid peroxidation (indexed by MDA) and antioxidants enzymes like GSH, SOD and CAT were assessed. There was a decrease in plasma levels concentration of urea, uric acid and creatinine. In addition to decreasing in activities of GSH, SOD and CAT as well as an increasing in MDA concentration in the kidney as a result of gentamicin injection [36]. Aqueous and ethanolic extracts of aerial parts of *P. oleracea* were evaluated against cisplatin induced nephrotoxicity in rats. Single intraperitoneal injection of 4 mg/kg cisplatin was administrated to rats. After 5 days, blood urea nitrogen (BUN) and serum creatinine (Scr) concentration were determined. Effect of aqueous and ethanolic extracts, before and after cisplatin injection on BUN and Scr, as well as morphological renal damage, was evaluated. It was indicated that treatment with aqueous and ethanolic extracts of *P. oleracea* in the highest dose (0.8 and 2 g/ kg), 6 and 12 hr before cisplatin injection reduced BUN and Scr. Tubular necrotic damage was not observed either. *P. oleracea* extract may protect against cisplatin-induced renal toxicity and might serve as a novel combination agent with cisplatin to limit renal injury [37]. The Renoprotective Effect of methanolic extract of aerial parts of *P. oleracea* was evaluated against Cisplatin-Induced nephrotoxicity in Wistar Rats. Twenty four female wistar rats were randomly divided into six groups – Group A were given no treatment and served as the control group; Group B was given only a single dose of cisplatin (2 ml/kg) and served as the cisplatin control group. Groups C and D were orally given 400mg/kg and 800mg/kg body weight of methanolic extract of *P. oleracea* (MEPO) respectively 6 hours after cisplatin injection (2 ml/kg).Groups E and F were orally given 400 mg/kg and 800 mg/kg body weight of MEPO respectively 6 hours before cisplatin injection (2 ml/kg) for 7 days. The effect of the treatment on relative kidney weight, serum creatinine level, serum uric acid and histoarchitecture of the rat kidney were assessed. Results showed significantly decreased serum creatinine levels (p<0.05) in rats treated with 400 mg/kg b.wt. And 800 mg/kg b.wt.MEPO as compared with the cisplatin control group. Serum uric acid was significantly decreased in groups C, D, E, and F when compared with control A. The relative average weight of the kidney increased significantly in all treated groups except group treated with 800 mg/kg b.wt.MEPO 6 hours before cisplatin. Kidney histological slides showed both recovery from and prevention of effects of induced toxicity at all treatment doses [38].

### 2.23 *Spondias pinnata* (L. f.) Kurz.

Ethanolic extract of *S. pinnata* fruit was showing nephroprotective effect by inducing streptozocin (STZ) hyperglycemia model. The rats were divided into different groups and diabetes was induced by administration of freshly prepared STZ (55 mg/kg, i.p.). The diabetic rats were given ethanolic extract of *S. pinnata* (100 and 200 mg/kg) for 14 days, and total urea and creatinine level along with effect on body weight were measured. The treatment with ethanolic extract of *S. pinnata* showed significantly reduced creatinine level (P<0.05 to P<0.01 [39].

### 2.24 *Zingiber officinale* Roscoe.

The aqueous extract of *Z. officinale* exhibited nephroprotective activity in mice against toxicity of metalaxyl. Mice were divided into 4 groups. Group 1: given metalaxyl at a dose level of 1/10 LD50 for 4 weeks, Group 2: given metalaxyl and ginger, Group 3: given ginger and Group 4: controls. Kidney cortex of metalaxyl-treated mice showed many histopathological alterations. The renal tubules lost their characteristic appearance and their lining epithelial cells appeared with cytoplasmic vacuolation. The glomeruli were degenerated and the renal blood vessels were congested. The intertubular spaces were infiltrated by inflammatory leucocytic cells. Metalaxyl caused marked elevation in serum creatinine and blood urea nitrogen. It also leads to significant increase in malondialdehyde and
decreased superoxide dismutase and catalase activities. The present results indicate that ginger has ameliorative effect against kidney damage induced by metalaxyl and this may be mediated by the antioxidant activity of ginger [79]. The nephroprotective effect of aqueous ethanol extract of Z. officinale (200 and 400 mg/kg, p.o) was evaluated against doxorubicin-induced (15 mg/kg, i.p) acute renal damage in rat. Serum urea and creatinine levels were evaluated as the markers of renal failure. Renal antioxidant status such as activities of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and level of reduced glutathione (GSH) were determined. Level of lipid peroxidation as equivalents of malondialdehyde (MDA), and glutathione-S-transferase (GST) activity were determined in the kidneys. Serum urea and creatinine levels were reduced in the Z. officinale (200 and 400 mg/kg, p.o) plus DXN treated groups. The renal antioxidant enzymes activities such as SOD, CAT GPx, levels of GSH and GST activity were restored and that of MDA declined significantly (p<0.001) in the Z. officinale (400 mg/kg) plus DXN treated group. The nephroprotection is mediated by preventing the DXN-induced decline of renal antioxidant status, and also by increasing the activity of GST [69].

2.25 Zingiber zerumbet (L.) Roxoe ex Sm.

Ethyl acetate extract of Z. zerumbet rhizome [200 mg per kg of body weight (mg/kg) and 400 mg/kg] on Paracetamol PCM-induced nephrotoxicity were examined. Rats were divided into five groups containing 10 rats each. The control group received distilled water while other groups were treated with extract alone (400 mg/kg), PCM alone (750 mg/kg), 750 mg/kg PCM+200 mg/kg extract (PCM+200-extract), and 750 mg/kg PCM+400 mg/kg extract (PCM+400-extract), respectively, for seven consecutive days. The Z. zerumbet extract was given intraperitoneally concurrent with oral administration of PCM. Treatment with Z. zerumbet extract at doses of 200 and 400 mg/kg prevented the PCM-induced nephrotoxicity and oxidative impairments of the kidney, as evidenced by a significantly reduced (P<0.05) level of plasma creatinine, plasma and renal malondialdehyde (MDA), plasma protein carbonyl, and renal advanced oxidation protein product (AOPP). Furthermore, both doses were also able to induce a significant increment (P<0.05) of plasma and renal levels of glutathione (GSH) and plasma superoxide dismutase (SOD) activity. The nephroprotective effects of Z. zerumbet extract were confirmed by a reduced intensity of renal cellular damage, as evidenced by histological findings [74].

3. CONCLUSION

This review is focused to protect our kidneys through easily available vegetables which are used in our daily life as a part of food; through these above vegetables kidneys can be protected in a prodigious and a healthy way. Vegetables are possessing active chemical constituents like drugs and chemicals constituents like alkaloids, benzoquinones, catechols, carotenoids, flavonoids, glycosides, flavonol glycosides, steroid glycosides, glycoalkaloids, terpenoids, monoterpenoids, diterpenoids, triterpene saponins, sterols and polyphenols play an important role against nephrotoxicity. The result of this study indicates that some vegetables have good potentials for use in kidney damage. This review gives evidential nature of nephroprotection in some vegetables against experimentally induced nephrotoxicity it has been proven by different animal models which give many links to develop the future trials. However, further studies are needed to identify and characterize the phytoconstituents from of above vegetables and added further new new vegetables which not proved by experimentally and also to explore the exact mechanism to act as nephroprotective mechanism.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES


16. Kim SH, Hong KO, Hwang JK, Park KK. Xanthorrhizol has a potential to attenuate the high dose cisplatin-induced nephrotoxicity in mice. Food and Che Toxi. 2015;605.


42. Ukaomah J, Aja P. Chemical compositions of Ipomoea aquatica (Green Kangkong) Igwenyi, Io, Offor, Ce, Ajah, Da, Nwankwo, Oc. 2011;4:2.

43. Jadhav VB, Thakare VN, Suralkar AA, Naik SR. Ameliorative effect of Luffa acutangula Roxb. on doxorubicin induced cardiac and nephrotoxicity in mice; 2013.


© 2017 Dobhal et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
http://sciencedomain.org/review-history/18256