Review of literature:

Chandrashekhar CH et al., (2008) were evaluated the anthelmintic activity of the bark extract of *Ficus racemosa* using adult earth warms. The bark extract of this plant has exhibited dose-dependent inhibition of spontaneous motility (paralysis) response. The anthelmintic effect was compared with the effects produced by the standard anthelmintic drug 3% piperazine citrate.

**Abu Hassanat at al., (2011)** were evaluated the hypoglycemic and in vitro antioxidant activity of ethanol extracts of this plant. Diabetes was induced in Swiss albino mice with the administration of alloxan. At a dose of 100mg/kg the extract has shown significant decrease in blood sugar level when compared to the alloxan induced diabetic mice. The antioxidant potential of the extract was also studies by using DPPH free radical scavenging activity and reducing property of ascorbic acid.

**Abu Hassanat, at al., (2011):** studied the antioxidant potential of the *Ficus racemosa* fruit extract. Hypolipidemic activities of ethanolic extract *Ficus racemosa* bark extracts were studied in alloxan induced diabetic rats. Oral administration of FrEBet (300mg/kg bw) to diabetic rats restored the status of blood glucose, lipids and lipoproteins to near normal range. This investigation thus shows that FrEBet has potent antidiabetic and hypolipidemic effects in alloxan-induced diabetic rats and these effects were much comparable to that of the standard reference drug, glibenclamide’.

**Faiyaz Ahmed, et al., (2010)** were studied the anticholinesterase activities of cold and hot aqueous extracts of *F. racemosa* stem bark. This study was evaluated the anticholinesterase activity of cold and hot aqueous extracts of *Ficus racemosa* stem bark against rat brain acetylcholinesterase *in vitro*. Both the cold aqueous extract and the hot aqueous extract exhibited a dose dependent inhibition of rat brain acetylcholinesterase.

**Ahmed F et al., (2009)** were studied the glucose-lowering, hepatoprotective and hypolipidemic activities of stem bark of *Ficus racemosa* in streptozotocin-induced diabetic rats. The study was evaluated the antihyperglycemic, hepatoprotective, and hypolipidemic effects of *F. racemosa* bark powder and aqueous extract in streptozotocin-induced diabetic rats. Both the bark powder
and aqueous extract of *F. racemosa* bark caused a significant reduction (*P* ≤ 0.05) in blood glucose (54 and 66% respectively).

**Ahmed F et al., (2010)** were reported the antibacterial activities of various sequential extracts of *Ficus racemosa* stem bark. The was study evaluated the antibacterial activity of sequential extracts of *Ficus racemosa* stem bark against *Staphylococcus aureus*, *Bacillus cereus*, *Pseudomonas aeruginosa*, *Escherichia coli* and *Bacillus subtilis* by disk-diffusion and agar-diffusion methods. In disk-diffusion assay chloroform, acetone and methanol extracts showed moderate antibacterial against *Staphylococcus aureus*, *Bacillus cereus*, *Bacillus subtilis* compared to the control, while petroleum ether extract did not exhibit antibacterial activity against any of the organisms tested.

**Bhaskara Rao B et al., (2002)** were evaluated the glucose-lowering efficacy of a methanol extract of the stem bark of *Ficus racemosa* Linn. both in normal and alloxan-induced diabetic rats. The doses examined (200 and 400 mg/kg p.o.) exhibited significant hypoglycaemic activity in both experimental animal models when compared with the control group.

**Jaykaran et al., (2009)** were studied the acute toxicity study of an aqueous extract of *Ficus racemosa* Linn. bark in albino mice. Albino mice of either sex were divided into four groups 1\(^{st}\) group given plain water and 2\(^{nd}\), 3\(^{rd}\), 4\(^{th}\) given 100,300 and 1000mg of aqueous extract of herb per 100 gm body weight in single dose. After 72 h of dose blood sample taken to determine haemoglobin, RBC count, WBC count, blood urea, blood glucose, serum Creatinine, serum cholesterol, S.G.P.T and S.G.O.T. Result indicated that aqueous extract of *Ficus racemosa* did not have lethal effect upon 100 times of the therapeutic dose in albino mice.

**Faiyaz Ahmed et al., (2010)** were found that the *Ficus racemosa* bark is a good source of dietary fiber, minerals, sugars and phenolic compounds. On dry basis, the total dietary fiber content was 20.5% of which major portion was contributed by insoluble dietary fiber (13.6%). Potassium was the most abundant mineral (11975 ppm) followed by chloride (7475 ppm) and calcium (1729 ppm). The bark was also a good source of other minerals and trace elements such as phosphorus and iron, zinc, magnesium, respectively.
Veerapur V.P. et al., (2007) were reported the antioxidant activity of ethanol extract and water extract of *Ficus racemosa* bark. These extracts were subjected to free radical scavenging both by steady state and time resolved methods such as nanosecond pulse radiolysis and stopped-flow spectrophotometric analyses and based on the obtained results, concluded that the ethanol extract of *F. racemosa* acts as a potent antioxidant and a probable radioprotector.

Ahmed F. et al., (2010) were studied the hepatoprotective effects of petroleum ether and methanol extract of *Ficus racemosa* Linn. stem bark. They were studied using the model of hepatotoxicity induced by CCl₄ in rats. The CCl₄ administration induced a significant decrease in serum total protein, albumin, urea and a significant increase (P ≤ 0.01) in total bilirubin associated with a marked elevation in the activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) as compared to control rats.

Ranasooriya WD et al., (2003) were reported the antidiuretic activity of the bark of *Ficus racemosa* Linn. The results demonstrated both the low- and high-doses of bark decoction (D) and ADH significantly impaired the total urine output. The D-induced antidiuresis had a rapid onset (within 1 h), peaked at 3 h and lasted throughout the study period (5 h). However, antidiuretic potential of D was about 50% lower than that of ADH. The results provide scientific support for its claimed antidiuretic action and deserve intensive scrutiny.

Mohammed Safwan Ali Khan, et al., (2011) were evaluated the antiulcer activity of *Ficus religiosa* against indomethacin and cold restrained stress-induced gastric ulcer and pylorus ligation assays. The extract (100, 200 and 400 mg/kg) significantly (P<0.05) reduced the ulcer index in all assays used. In conclusion, the present study provide preliminary data on the antiulcer potential of *F. religiosa* stem bark and support the traditional uses of the plant for the treatment of gastric ulcer.

Kaiser Hamid et al., (2011) were evaluated the free radical scavenging activity of *Ficus racemosa* seeds using DPPH and brine shrimp lethality bioassay method. In both the methods, *Ficus racemosa* showed a significant activity.

**Herbal nephro protective agents in drug induced nephrotoxicity.**

Marjan Ajami et al., (2010) were evaluated the nephroprotective effect of *Crocus sativus* in male wistar rats. Nephrotoxicity was induced with the administration of gentamicin 80mg/kg for 5 days, and the extract (40 and 80mg/kg) was administered for 10 days along with gentamicin
(5days). The results of this study- serum creatinine, BUN, MDA and histopathalogy study concludes the nephroprotective activity of *Crocus sativus*.

**Lakshmi BVS et al., (2009)** were studied the nephroprotective effect of ethanol extracts of leaves and unripe pods of *Bauhinia purpuria* was studied on gentamicin induced nephrotoxic rats. Nephrotoxicity was induced with 100mg/kg gentamicin for 8 days. The extract (300mg/kg) was administered orally along with gentamicin and measured the renal biomarkers serum creatinine, serum uric acid, BUN, serum urea and histopathology. The extracts normalized the gentamicin induced increase in these renal biomarkers and reduced the damaging effect of kidney.

**Niraj M Bhatt et al., (2011)** were studied the nephroprotective effects of *Enicosstemma littoral* blame extract on rats. Rats were divided into 4 groups, control, GM (80mg/kg) treated, GM and extract (2.5mg/kg), Vitamin (600mg/kg) and GM. Treatment is for 8days. The results of this study conclude the nephro protective effect.

**Mathew JE et al., (2011)** were studied the nephroprotective effect of ethanol extract of entire plant of *Spharanthus indicus* in cisplatin (12mg/kg) induced nephrotoxic albino rats. The extracts (150 and 300mg/kg) were administered orally from sixth day onwards after cisplatin administration. The extracts significantly reduced the elevated levels of serum creatinin and urea levels. The depleted SOD, catalase, glutathione levels were restored to normal levels in extract treated rats. This concludes the nephrotoxicity protective effect of this plant.

**Kim YH et al., (2006)** were reported the nephroprotective activity of ethanol extract of the roots of *Brassica rapa* (EBR) in cisplatin (7mg/kg ip) induced nephrotoxic rats. The EBR was administered for 14 days before cisplatin administration. A single dose of cisplatin caused the kidney damage manifested by an elevation in BUN, serum creatinine, and urine LDH levels and MDA, aldehyde oxidase and xanthin oxidase levels. Also, renal tissue from the cisplatin showed a significant decrease in glutathione, SOD and catalase. But the rats treated with the extracts has shown the protective effect on the kidney by normalizing these variations caused by cisplatin alone treatment.

**Wonqnekiat O et al., (2008)** were studied the protective effects of aged garlic extract (AGE) in cyclosporine A (CsA 50mg/kg/day for 10days). The extracts of AGE were administered orally 3 days before the administration of CsA and continued for 10 days along with CsA. BUN, serum
creatinine, creatinine clearance and renal histopathology were studied. The results of this study conclude the nephroprotective role of the aged garlic extract.

Wonqmekiat O et al., (2008) were studied the Shallot (Allium ascalonicum L) for the nephroprotective role against CsA (25mg/kg) induced nephrotoxic male wistar rats. CsA-induced nephrotoxicity was evidenced by increased BUN and serum creatinine, but decreased urea and creatinine clearance. The kidney of CsA treated rats exhibited severe vacuolations and tubular necrosis. CsA also induced oxidative stress, as indicated by increased renal MDA and reduced GSH concentrations. The rats treated with shallot extract along with CsA counteracted the deleterious effects of CsA on renal dysfunction, oxidative stress markers and morphological changes.

Jennifer J et al., (2008) were studied the lithium induced NDI, alterations in renal osmolyte concentrations and alteration by amiloride. Rats fed lithium (60mmol/kg dry food) over 4 weeks developed NDI. The medullary osmolyte content was restored to normal values. The reduced AQP2, AQP3 and urea transporter expression was significantly reversed following amiloride therapy.

Shai Efrati et al.,(2005) were studied the nephroprotective effect of N-acetylcystein in lithium induced renal failure. Renal failure induced in the rats with the administration of the lithium chloride in the drinking water for 3 weeks. The rats treated with lithium chloride have shown increased levels of serum creatinine, BUN and 24h urinary protein, osmolarity. The kidneys were excised for histopathological studies. The results of this study conclude the nephroprotective role in lithium induced renal failure in rats.

Vijaimohan K et al., (2010) were reported the protective role of a purified compound of Solanum triobatum in lithium carbonate induced multiple organ toxicity in rats. The lithium carbonate (150mg/kg body weight) was administered for a period of 30days. These rats showed decreased levels of reduced glutathione, SOD, catalase, GST, GPX activities and parallel decline in ATP in tissues. There are abnormal levels of cholesterol, triglycerides, phospholipids and fatty acids in the livers of these rats. Treatment with Solanum triobatum affords substantial protection in liver and heart by altering these parameters to normal values that were further confirmed by histopathological examination.
Jakob Nielsen et al., (2008) were studied the lithium induced alterations in the proteins by Bioinfomatic analysis. The data indicated that proteins involved in cell death, apoptosis, cell proliferation and morphology are highly affected by lithium. It was also demonstrated the members of several signaling pathways are highly affected by lithium treatment. This study provides a comprehensive analysis of the proteins affected by lithium treatment in the IMCD (inner medullary collecting ducts) and, as such, provides clues to potential lithium targets in the brain.

Shu-Huei Kaol et al., (2008) were reported that the lithium increased intracellular production of reactive oxygen species (ROS). Inhibition of intracellular ROS production by N-acetylcysteine resulted in a decreased HO-I expression in C6 glioma cells.

Wan-Loy Chu et al., (2010) were studied the protective effect of spirulina extract against cell death induced by free radicals. Spirulina extract did not cause cytotoxic effect on 3T3 cells within the range of concentrations tested (0-250 microgram /ml). The antioxidant activity was found to be good as Vitamin C and vitamin E.

Ihsan Yaman et al., (2010) were investigated the protective effect of Nigella sativa in GM induced nephrotoxicity in wistar albino rats. There is increased levels of plasma creatinine, urea, plasma MDA and NO and decreased levels of erythrocyte SOD and GSH-Px activies in GM treated rats. The rats treated with Nigella sativum and GM has shown altered levels of these markers to the normal values suggesting the nephroprotection.

Bibu KJ et al., (2009) were studied the therapeutic effect of ethanolic extract of Hygrophia spinosa in gentamicin-induced nephrotoxic model of kidney injury in male Sprague-Dawley rats. Serum creatinine, urea and kidney superoxide dismutase, lipid peroxidation, catalase and reduced glutathione were measured in the control and treated groups. Histopathological study was also performed. The results of this study suggested that this plant extract have nephroprotective activity.

Eslami SH et al., (2011) were reported the renoprotective effect of Erygnium caucasicum against gentamicin-induced renotoxicity. The studied parameters are BUN, serum urea and creatinine. The extract (400mg/kg) has showed significant renoprotective effect when compared with control group.
Sandeep D et al., (2010) were studied the nephroprotective effect of extracts of medicinal plants *Hemidesmus indicus* L and *Acorus calamus* L against cisplatin (12mg/kg body weight). The animals treated with extracts has shown decreased levels of lipid peroxidation and increased activities of the antioxidants in the renal tissue. The elevated levels of the serum creatinine were also reduced in animals treated with the extracts. The histopathological examinations also confirm the nephroprotective effects of this plant extracts.

Yapar K et al., (2009) were studied the protective role of royal jelly (RJ) and green tea (GT) extracts in cisplatin induced nephrotoxicity in rats. Cisplatin (7mg/kg i.p.) was administered as single dose. The studied parameters are BUN, serum creatinine, MDA, reduced glutathione levels and histopathological study. The results of this study concluded that 100mg/kg body weight doses of RJ and GT provided protection against cisplatin induced kidney damages.

Gholamreza Karimi et al., (2010) were reported the nephroprotective effect of *Portulaca oleracea* L. The nephrotoxicity was induced in rats with the administration of 4mg/kg i.p. for 5days. The studied parameters are BUN, serum creatinine and histopathological studies. The dose of aqueous and ethanolic extracts is 0.8 and 2g/kg body weight. The results of this study conclude the nephroprotective role of this plant extracts in cisplatin induced nephrotoxicity.

Naveen Tirkey et al., (2005) were reported the protective role of CMN in cyclosporine induced nephrotoxicity in rats. CsA(20mg/kg/day s.c) was administered for 21 days for the induction of nephrotoxicity. The studied parameters are TBARS, reduced glutathione content, SOD and catalase. Nitric levels were estimated in serum and tissue homogenates. CMN markedly reduced the elevated levels of TBARS and significantly attenuated renal dysfunction by increasing the antioxidant parameters. This concludes the nephroprotective effect of curcumin in rats.

Barbara Buffoli et al., (2005) were studied the nephroprotective activity of red wine polyphenol provinol (PV) in CsA induced nephrototoxic rats. CsA (15mg/kg/day s.c) was administered for 21 days. PV prevented the negative effects through a negative mechanism that involves the reduction of both oxidative stress and increased iNOS and NF-kB expression induced by cyclosporine. These results provide a pharmacological basis for the beneficial effects of plant – derived polyphenols against CsA –induced renal damage associated with CsA.
Ahmet Gokce et al., (2009) were reported the protective effect of Caffeic acid phenethyl ester (CAPE). CsA (15mg/kg/day) was administered for 10days sub cutaneously. CsA-CAPE (10micromole/kg/day) was administered for 11 days. The results of this study provide the experimental evidence for its nephroprotective effect on rats.

Sabahattin Ocak et al., (2007) were compared the beneficial effects of caffeic acid phenethyl ester, vitamin C, vitamin E and N-acetylcysteine on vancomycin –induced nephrotoxicity. They were divided the 30 rats into 6 groups. They measured the BUN, catalase, renal MDA, NO levels in the control and treated rats. The experimental data of this result suggest that vitamin E, as well as vitamin C, N-acetylcystein and CAPE could be useful for reducing the detrimental effects on vancomycin- induced toxicity in kidneys.