



INTERNATIONAL JOURNAL OF PHARMACY & LIFE SCIENCES
(Int. J. of Pharm. Life Sci.)

Antioxidant and Hepatoprotective activity of *Anaphyllum wightii* Schott.

J.N.Dharsana^{1*}, Sr. Molly Mathew², P.N. Baby¹, Sajith Kumar¹ and Diljit S. Kuttoor¹

1, Department of Pharmaceutical Chemistry, Academy of Pharmaceutical Sciences, Pariyaram Medical College, Kannur, Kerala - India

2, Malik Deenar College of Pharmacy, Kasargode, Kerala - India

Abstract

Herbal medicines have traditionally been used worldwide for the prevention and treatment of liver disease with fewer adverse effects. The tubers of *Anaphyllum wightii* plant were chosen and studied for their antioxidant activity *in vitro* and hepatoprotective activity *in vivo*. The antioxidant activity of the chloroform extract was examined *in vitro* using a 1,1-diphenyl-2-picrylhydrazyl (DPPH) free radical scavenging assay and super oxide scavenging assay. The extract had significant dose-dependent antioxidant activity in all *in vitro* experiments. The percentage of free radical scavenging by DPPH and super oxide scavenging assay values of *Anaphyllum wightii* and ascorbic acid (standard) were found to be 49.02±4.11, 60.20±3.43 and 72.78±3.97 respectively. Hepatoprotective activity of the plant extract was evaluated in a rat model of carbon tetrachloride (CCl₄) induced liver damage. CCl₄ significantly altered serum marker enzymes, total bilirubin, total protein, and liver weight. The extract caused these values to return to normal in rats with CCl₄ induced liver damage. This indicated the hepatoprotective potential of *Anaphyllum wightii* and was comparable to use of the standard drug silymarin. Thus, the present study revealed that *Anaphyllum wightii* have significant antioxidant and hepatoprotective activity.

Key-Words: *Anaphyllum wightii*, antioxidant, hepatoprotective, DPPH

Introduction

Plant drugs have been a major source for treatment of diseases for a long time. They have been used in the traditional medicine on the basis of experiences and practice. With the advent of modern systems of medicine need has been felt to investigate the active constituents present in these plants. Various molecules have been isolated, characterized and tested for their related pharmacological activities.

Reactive oxygen species (ROS) and reactive nitrogen species (RNS) are produced by normal physiologic processes and fulfill important functions in the body at minute or moderate concentrations. ROS and RNS are necessary for the maturation process of cellular structures and can act as weapons for the host's defence system. The importance of ROS production by the immune system is clearly evident in patients with granulomatous disease. These patients have a defective membrane-bound nicotinamide adenine dinucleotide phosphate (NADPH) oxidase system that precludes them from producing the superoxide anion radical (O₂⁻), thereby resulting in multiple and persistent infections^{1,2}.

Oxygen radicals have crucial action such as signal transduction, gene transcription and regulation of synthesis of cyclic guanosine monophosphate (cGMP) in cells^{3,4}.

Nitric oxide (NO) is a common signaling molecule and participates in virtually every one of the body's cellular and organ functions. Optimum amount of NO produced by endothelial cells are essential for regulating the relaxation and proliferation of vascular smooth muscle cells, leukocyte adhesion, platelet aggregation, angiogenesis, thrombosis, vascular tone and hemodynamics⁵. In addition, NO produced by neurons serves as a neurotransmitter⁶.

However, the generation of even slightly larger amounts of these essential compounds during metabolism or in response to environmental pollutants, radiation, chemicals, toxins, deep fried and spicy foods, and physical stress can cause massive physiologic problems by oxidation of bio-molecules (protein, amino acids, lipid, and DNA). Free radical presumably plays a major role in all pathologies. Free radicals are believed to be responsible for more than one hundred conditions like cancer, diabetes, atherosclerosis, arthritis, neuropathy, nephropathy, retinopathy, aging, compromised immunity, and cardiovascular diseases^{7,8,9,10}.

*** Corresponding Author**

E-Mail: dharsanaapsc@gmail.com

The liver has a central role in transforming and clearing chemicals and is closely related to the gastrointestinal tract, which makes it susceptible to drug toxicity, xenobiotic, and oxidative stress. Dysfunction of this organ results in impairment of energy metabolism and intracellular oxidant stress with excessive formation of ROS. CYP2E1 is a cytochrome P450 isoenzyme produced by the liver that also facilitates oxidative stress and cell injury^{11, 12}. Although Kupffer cells and recruited neutrophils in the liver are part of the host-defence system, these inflammatory cells initiate liver injury under certain circumstances, such as when excess free radicals are present^{13,14,15,16}.

Although the body has mechanisms to defend against the damaging properties of ROS^{17,18}, its capacity to control ROS can be overwhelmed, causing irreversible oxidative damage and various conditions that threaten the liver and other parts of the body. Several synthetic and semi-synthetic chemicals are often used to protect the liver from the detrimental effects of free radicals but they are not sufficiently effective and cause adverse reactions. Modern medicine includes many important bioactive molecules with antioxidant and hepatoprotective properties that were derived from plant. Hence, recent research has hastened to identify notable hepatoprotective agents from plant products that will reduce the harmful effects of and problems associated with free radicals while causing minimal adverse reactions.

As part of ongoing research, the current study examined the chloroform extract of *Anaphyllum wightii* for its use as a hepatoprotective agent to reduce the damage caused by ROS and RNS. No reports have described the plant's hepatoprotective activity. Thus, the present study sought to investigate the chloroform extract of *Anaphyllum wightii* for its hepatoprotective activity using a rat model of CCl₄ induced liver damage and different *in vitro* antioxidation experiments.

Material and Methods

Experimental animals

Healthy albino rats weighing 150-200g of either sex were used. The experimental protocol was approved by the Institutional Animal Ethics Committee (APSC/CPCSEA/-04/IAEC/2013) and animals were maintained under standard conditions in the animal house approved by Committee for the Purpose of Control and Supervision on Experiments on Animals (CPCSEA).

Collection and Authentication

Genus – *Anaphyllum beddomei* Engl. and *Anaphyllum wightii* Schott were reported from the high ranges of Western Ghats. The two species in this genus are

similar in appearance to those in the genus *Anaphyllopsis*. Genus *Anaphyllum wightii* is listed as an endemic and threatened genus of South India¹⁹. The species of the genus *Anaphyllum* are found in marshes. They are characterized by pinnate leaves and twisted spathe for the spadix. *Anaphyllum* is a genus of flowering plants in the Araceae family. The tribal communities (Kani Tribes, Malasars, Kadars, Pulayars, Madhuvarsetc) use these plants as food and as an antidote to snake bite. Leaves of *Anaphyllum beddomei* form a part of tribal diet. Arun *et al.* reported the use of the corms of *Anaphyllum wightii* (Keerikizhangu), as an antidote to snake bite along with some medicinal plants²⁰.

Plant material

Plant tubers were collected from Wynad hills, Wynad district, Kerala, India. The taxonomical identification of the plant was done by Dr. N. Sasidharan, Scientist-F, Programme co-ordinator, FE & BC division, Kerala Forest Research Institute, Peachi, and Trissur. The voucher specimen was preserved in Academy of Pharmaceutical sciences, Pariyaram Medical College, Kannur, and Kerala.

Preparation of plant extract

The collected plant tubers were dried at room temperature, pulverized by a mechanical grinder, sieved through 40 mesh. About 200g of powdered materials were extracted with chloroform. The extracted solution was filtered using a clean cloth and the filter paper. The extract was concentrated first in a rotary vacuum evaporator and then in a water bath. The extracted residue was weighed and the percent yield was 9.21% w/w. The extract was then frozen prior to examination of its potential antioxidant and hepatoprotective properties.

Chemicals

DPPH (2, 2-diphenyl-1-picrylhydrazyl), ascorbic acid, carbon tetra chloride, phosphate buffer saline (pH 6.3), EDTA, nitro blue tetrazolium (NBT), riboflavin and silymarin were purchased from Merck Pharmaceuticals.

DPPH Free Radical Scavenging Assay^{21,22,23}

1 ml of the test compound in various concentrations (50, 100, 150, 200 & 250 µg/ml) was added to 1 ml of 0.1 mM solution of DPPH in methanol. After 30 minutes, absorbance was measured at 517 nm, using a spectrophotometer (SHIMADZU, UV 1800). A 0.1 mM solution of DPPH in methanol was used as blank, whereas ascorbic acid was used as a reference standard. All tests were performed in triplicate. Percent inhibition was calculated using equation,

$$\text{Percent inhibition} = \left[\frac{(\text{Control} \times \text{Test})}{\text{Control}} \right] \times 100$$

Superoxide Scavenging Assay^{21,22,23}

1ml of the test compound in various concentrations (50,100,150,200& 250 µg/ml) was added to 0.2ml of EDTA, 0.1ml of NBT, 0.05 ml of riboflavin and 2.6 ml phosphate buffer. The control tubes were also set up where DMSO was added instead of the plant extracts. All the tubes were vortexed and the initial optical density was measured at 560 nm in a spectrophotometer. The tubes were illuminated using a fluorescent lamp for 30 minutes. The absorbance was measured again at 560 nm. The difference in absorbance before and after illumination was indicative of superoxide anion scavenging activity. All tests were performed in triplicate. Percent inhibition was calculated using equation,

Percent inhibition = $[(\text{Control} \times \text{Test}) / \text{Control}] \times 100$

Hepatoprotective activity

The hepatoprotective activity of chloroform extract of *Anaphyllum wightii* was determined using a rat model of carbon tetrachloride induced hepatotoxicity. After seven days of acclimatization, rats were divided into four groups consisting of three rats each. Treatment lasted for 8 days. Group I served as the normal control and received only normal saline (1 ml/kg, *i.p.* for eight days). Group II served as the toxic control and was administered carbon tetrachloride in liquid paraffin (CCl₄:liquid paraffin 1:2; 1 mL/kg, *i.p.*) once every 72 hours. Group III served as the positive control and was administered Silymarin (25 mg/kg/day, *p.o.*) along with carbon tetrachloride in liquid paraffin (CCl₄:liquid paraffin 1:2; 1 mL/kg, *i.p.*) once every 72 hours. Group IV was administered chloroform extract of *Anaphyllum wightii* (300 mg/kg, *p.o.*) daily and carbon tetrachloride in liquid paraffin (CCl₄:liquid paraffin 1:2; 1 mL/kg, *i.p.*) once every 72 hours. Twenty-four hours after the last dose, blood was taken from the retro-orbital plexus under sodium phenobarbital anesthesia and rats were dissected to remove the liver. Before blood was collected, the syringe was rinsed with heparin to prevent haemolysis/clotting. The blood samples were then centrifuged at 2,500 rpm at 37°C to separate serum and were used to estimate the biochemical markers of liver damage, *i.e.* SGOT, SGPT^{24, 25}, ALP²⁶, bilirubin²⁷ and total protein levels²⁸.

Results and Discussion**Antioxidant activity**

Several concentrations ranging from 50-250 µg/ml of the chloroform extracts of *Anaphyllum wightii* were compared for their antioxidant activity in different *in vitro* models. It was observed that free radicals were scavenged by the extracts in a concentration dependent manner up to the given concentration in all the models.

The absorbance of the extracts increased with the rise in their concentrations since the absorbance is directly proportional to the antioxidant property. As shown in Table 1 and 2 the chloroform extract of *Anaphyllum wightii* (from 22.98±2.11% to 49.02±4.11% for DPPH free radical scavenging assay and from 29.48±1.44% to 60.20±3.43% for super oxide scavenging assay) indicates better antioxidant power. The standard drug ascorbic acid showed 72.78±3.97% antioxidant power at 50 µg/ml concentration. The roles of antioxidants are to neutralize the excess of free radicals, to protect the cells against their toxic effects and to help prevent disease. Results of the current study indicated that the chloroform extract of *Anaphyllum wightii* has significant DPPH and super oxide scavenging activity. These pharmacological effects of the extract may at least in part be due to phenols and flavonoids components that were found in the chloroform extract of *Anaphyllum wightii*. Phenols are ubiquitous secondary metabolites in plants and have a wide range of therapeutic uses because of their antioxidant, antimutagenic, anticarcinogenic, and free radical scavenging activity^{29,30,31,32}. The antioxidant activity of phenolic compounds is mainly due to their redox properties, which can play an important role in absorbing and neutralizing free radicals, quenching single and triplet oxygen or decomposing peroxides³³. Flavonoids are a group of polyphenolic compounds with known properties, which include free radical scavenging and inhibition of hydrolytic and oxidative enzymes³⁴. Furthermore, these compounds have a strong affinity for iron ions (which are known to catalyse many processes and lead to the appearance of free radicals), so their antiperoxidative activity could also be attributed to a concomitant ability to chelate iron. Therefore, the phenol and flavonoid components in the chloroform extract of *Anaphyllum wightii* may have contributed directly to antioxidant action noted in this study.

Hepatoprotective activity

The hepatoprotective activity of the crude chloroform extract at a dose of 300 mg/kg body weight in rats with carbon tetrachloride-induced damage is shown in Table 3. For comparison, the table also shows the untreated group (control), carbon tetrachloride-treated group (induction control) and the group treated with the drug (silymarin). The control group (I) had serum SGPT of 23.1 ± 0.34 U/L, SGOT of 38.8 ± 0.39 U/L, ALP of 20.28 ± 0.04 mg/dL, total bilirubin of 1.06 ± 0.016 mg/dL, total protein of 12.14 ± 0.18 KA, and liver weight of 5.96 ± 0.18 g. In the group with CCl₄ induced liver damage (II), serum SGPT increased to 75.6 ± 0.61 U/L, SGOT increased to 86.4 ± 0.39 U/L,

ALP increased to 68.12 ± 0.14 mg/dL, total bilirubin increased to 6.38 ± 0.027 mg/dL, total protein increased to 7.87 ± 0.033 KA, and liver weight increased to 9.87 ± 0.51 g. Administration of Chloroform extract of *Anaphyllum wightii* in rats with CCl₄ induced liver damage resulted in gradual normalization of SGPT, SGOT, ALP, total bilirubin, total protein and liver weight compared to CCl₄ treated group.

In most developing countries, there is a high incidence of viral hepatitis. Identification of an efficient hepatoprotective drug derived from natural sources is an urgent necessity. The changes associated with CCl₄ induced liver damage are similar to those of acute viral hepatitis³⁵. CCl₄ is therefore a useful tool for inducing hepatic damage in experimental animals. The hepatotoxicity of CCl₄ is the result of its reductive dehalogenation. It is catalyzed by cytochromic P450 to produce the highly reactive metabolite trichloromethyl (CCl₃[•]) free radical. This then readily interacts with molecular oxygen to form the trichloromethyl peroxy radical (CCl₃OO[•]). These free radicals bind covalently to cellular proteins or lipids or extract a hydrogen atom from an unsaturated lipid, thereby initiating lipid peroxidation and consequently leading to liver damage. A substantial increase in the level of serum marker enzymes (SGOT, SGPT and ALP) and total bilirubin was noted in the CCl₄ control group. The return of elevated levels of serum enzymes to near normal values in group treated with the chloroform extract of *Anaphyllum wightii* or standard silymarin is an indication of the stabilization of the plasma membrane and the repair of hepatic tissue damage caused by CCl₄. A decrease in the level of total proteins in rats with CCl₄ induced liver damage is attributed to damage primarily in the endoplasmic reticulum. This results in a loss of P450 and subsequent decrease in protein synthesis. The rise in protein levels in treated groups suggests the stabilization of the endoplasmic reticulum and subsequent protein synthesis. In addition, CCl₄ led to a significant increase in liver weight because it blocks the secretion of hepatic triglycerides in plasma³⁶. Silymarin and the chloroform extract of *Anaphyllum wightii* were found to prevent an increase in liver weight in rats. These results suggest that the chloroform extract of *Anaphyllum wightii* offers hepatic protection by reducing damage or by preserving normal hepatic physiological mechanisms that have been disturbed by a hepatotoxin such as CCl₄. This finding indicates that *Anaphyllum wightii* has protective action *in vivo*.

Conclusion

The present study indicates that chloroform extract of *Anaphyllum wightii* has potential antioxidant activity *in vitro* and hepatoprotective activity *in vivo*. However, further studies are needed to examine underlying mechanisms of antioxidant activity *in vitro* and hepatoprotective activity *in vivo*. Studies also need to isolate the active compound(s) responsible for this pharmacological activity.

References

1. Valko M., Leibfritz D., Moncol J., Cronin M.T., Mazur M. and Telser J. (2007). Free radicals and antioxidants in normal physiological functions and human disease, *Int J Biochem Cell Biol*, 39:44-84.
2. Dröge W. (2002). Free radicals in the physiological control of cell function, *Physiol Rev*, 82:47-95.
3. Zheng M. and Storz G. (2000). Redox sensing by prokaryotic transcription factors, *Biochem Pharmacol*, 59:1-6.
4. Lander H.M. (1997). An essential role for free radicals and derived species in signal transduction, *FASEB J*, 11:118-124.
5. Ignarro L.J., Cirino G., Casini A. and Napoli C. (1999). Nitric oxide as a signaling molecule in the vascular system: An overview, *J Cardiovasc Pharmacol*, 34:879-886.
6. Freidovich I. (1999). Fundamental aspects of reactive oxygen species, or what's the matter with oxygen? *Ann N. Y. Acad Sci*, 893:13-18.
7. Kumpulainen J.T. and Salonen J.T. (1990). Natural antioxidants and Anti carcinogens in Nutrition, *Health and Disease, The Royal Society of Chemistry, UK*, 178-187.
8. Cook N.C. and Samman S. (1996). Flavonoids-chemistry, metabolism, cardio protective effects and dietary sources, *Nut Biochem*, 7:66-76.
9. Liao K. and Yin M. (2000). Individual and combined antioxidant effects of seven phenolic agents in human erythrocyte membrane ghosts and phosphatidylcholine liposome systems, Importance of the partition coefficient, *J Agric Food Chem*, 48:2266-2270.
10. Beckman K.B. and Ames B.N. (1998). The free radical theory of aging matures, *Physiological Rev*, 78:547-581.
11. Guengerich F.P., Kim D.H. and Iwasaki M. (1991). Role of human cytochrome P450 IIE1 in the oxidation of many low molecular weight cancer suspects, *Chem Res Toxicol*, 4:168-179.

12. Koop D.R.(1992).Oxidative and reductive metabolism by cytochrome P450 2E1,*FASEB J*, 6:724-730.
13. Jaeschke H. (2000). Reactive oxygen and mechanisms of inflammatory liver injury,*J Gastroenterol Hepatol*, 15:718-724.
14. Jaeschke H., Smith C.W., Clemens M.G., Ganey P.E. and Roth R.A. (1996). Mechanisms of inflammatory liver injury, Adhesionmolecules and cytotoxicity of neutrophils,*Toxicol Appl,Pharmacol*, 139:213-226.
15. Jaeschke H. and Smith C.W. (1997).Mechanisms of neutrophilinduced parenchymal cell injury, *J Leukoc Biol*, 61:647-653.
16. Laskin D.L. and Laskin J.D. (2001). Role of macrophages and inflammatory mediators in chemically induced toxicity, *Toxicology*, 160:111-118.
17. Halliwell B., Aeschbach R., Löliger J. and Aruoma O.I. (1995). The characterization of antioxidants, *Food Chem Toxic*, 33:601-617.
18. SiesH.(1993).Strategies of antioxidant defense, *Euro J Biochem*, 215:213-219.
19. Ahmedullah M. and Nayar M.P. (1987).Endemic plants of the Indian Region, Vol. 1,*Penisular India, Flora of India series IV, Botanical Survey of India*, 205-208.
20. Arun V., Liju V.B., Reena John J.V., Parthipan B. and Renuka C. (2007).Traditional remedies of Kani Tribes of Kottoor reserve forest, Agasthyaavanam,Vol 6, *Thiruvananthapuram, Kerala*, 589-594.
21. Ansari N.M., Houlihan L., Hussain B. and Pieroni A. (2005). Antioxidant Activity of Five Vegetables Traditionally Consumed by South-Asian Migrants in Bradford, Yorkshire, UK, *Phytother Res*, 19: 907-911.
22. Molyneux P. (2004). The use of stable free radical diphenylpicryl-hydrazyl (DPPH) for estimating antioxidant activity,*Songklanakar J Sciand technol*, 26:212-219.
23. Anandjiwala S. (2007).Antioxidant activity of stem bark of *Thespepsia populnea*,*Journal of Natural Remedies*, 7(1): 135-138.
24. Bergmeyer H.U., Horder M., Rej R. (1986). International Federation of Clinical Chemistry (IFCC) Scientific Committee,Analytical Section: Approved recommendation (1985) on IFCC methods for the measurement of catalyticconcentration of enzymes. Part 2. IFCC method for aspartate aminotransferase (L-aspartate: 2-oxoglutarate aminotransferase, EC 2.6.1.1),*J Clin Chem Clin Biochem*, 24:497-510.
25. Schumann G., Bonora R., and Ceriotti F. (2002). IFCC primary reference procedures for the measurement of catalytic activity concentrations of enzymes at 37°C. International Federation of Clinical Chemistry andLaboratory Medicine,Part 4, Reference procedure for the measurement of catalytic concentration of alanine aminotransferase,*Clin Chem Lab Med*, 40:718-724.
26. Kind R.N., and King E.J. (1954).Urinary excretion of acid phosphatase,*J Clin Path*, 7:322.
27. Jendrassik L. and Grof P. (1938).Vereinfachte photometrische methoden zur bestimmung des blutbilirubins, *Biochem Z*, 81:297.
28. Lowry O.H., Rosebrough N.J., Farr A.L. and Randall R.J. (1951).Protein measurement with the Folin phenol reagent,*JBio Chem*, 193:265-275.
29. Tanaka M., Kuie C.W., Nagashima Y. and Taguchi T. (1988). Applications of antioxidative Maillard reaction products from histidine and glucose to sardine products, *Nippon Suisan Gakkaishi*, 54:1409-1414.
30. Duh P.D., Tu Y.Y. and Yen G.C. (1999). Antioxidant activity of the aqueous extract of harn jyr (*Chrysanthemum morifolium*. Ramat),*Lebensmittel-Wissenschaft and Technologie*,32:269-277.
31. Gordon M.H. (1990). The mechanism of antioxidant action *in vitro*,*In: Food Antioxidants (Hudson B.JF, ed.)*,ElsevierApplied Science, London, 1-18.
32. Hare J.M. and Stamler J.S. (2005). NO/redox disequilibrium in the failing heart and cardiovascular system,*J Clin Invest*, 115:509-517.
33. Friedman., Scott E., Grendell., James H., McQuaid and Kenneth R. (2003).*In: Current diagnosis & treatment in gastroenterology*,Lang Medical Books/McGraw-Hill, New York, 664-679.
34. Yen G.C., Duh P.D. and Tsai C.L. (1993). Relationship between antioxidant activity and maturity of peanut hulls, *J Agric Food Chem*, 41:67-70.
35. Rubinstein D. (1962).Epinephrine release and liver glycogen levels after carbon tetrachloride administration,*Am J Physiol*203:1033-1037.

36. Aniya Y., Koyama T., Miyagi C., Miyahira M., Inomata C., Kinoshita S. and Ichiba T. (2005). Free radical scavenging and hepatoprotective actions of the medicinal herb

Crassocephalum crepietioides from Okinawa islands, *Biol Pharm Bul*, 28:19-23.

Table 1: Effect of chloroform extract of *Anaphyllum wightii* on in-vitro antioxidant activity by DPPH radicle scavenging assay

Concentration (µg/ml) of Chloroform extract	% Scavenging	Concentration (µg/ml) of Ascorbic acid	% Scavenging
50	22.98±2.11	10	35.63±2.32
100	31.36±2.14	20	48.69±2.43
150	35.64±3.34	30	53.67±2.52
200	42.02±3.17	40	61.91±3.57
250	49.02±4.11	50	72.78±3.97

Values are Mean ± SEM, n=3

Table 2: Effect of chloroform extract of *Anaphyllum wightii* on in-vitro antioxidant activity by superoxide scavenging assay

Concentration (µg/ml) of Chloroform extract	% Scavenging	Concentration (µg/ml) of Ascorbic acid	% Scavenging
50	29.48±1.44	10	35.63±2.32
100	31.89±1.07	20	48.69±2.43
150	46.03±2.88	30	53.67±2.52
200	56.28±2.73	40	61.91±3.57
250	60.20±3.43	50	72.78±3.97

Values are Mean ± SEM, n = 3

Table 3: Effect of chloroform extract of *Anaphyllum wightii* and Silymarin on serum bio chemical parameters in CCl₄ induced liver damage in rats

Treatment Group	SGPT(µ/L)	SGOT(µ/L)	ALP(KA)	Bilirubin(mg/d L)	Total Protein(mg/dL)	Liver Weight(g)
Normal (control)	23.1±0.34	38.8±0.39	20.28±0.04	1.06±0.016	12.14±0.18	5.96±0.18
CCl ₄	75.6±0.61	86.4±0.39	68.12±0.14	6.38±0.027	7.87±0.033	9.87±0.51
Chloroform Extract of AW	42.8±0.60	67.9±0.34	50.28±0.09	4.09±0.012	9.02±0.03	7.08±0.27
Silymarin	28.8±0.33	46.0±0.34	25.27±0.13	2.20±0.022	12.02±0.04	6.45±0.32

Values are mean ± STD and each group contained three rats. Drug treatment lasted for 8days.

How to cite this article

Dharsana J.N., Mathew M., Baby P.N., Kumar S. and Kuttoor D.S. (2014). Antioxidant and Hepatoprotective activity of *Anaphyllum wightii* Schott.. *Int. J. Pharm. Life Sci.*, 5(12):4049-4054.

Source of Support: Nil; Conflict of Interest: None declared

Received: 12.18.14; Revised: 30.19.14; Accepted:18.11.14