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Research Article

MODULATORY EFFECT OF SEMIPURIFIED FRACTIONS OF *BAUHINIA PURPUREA* L. BARK EXTRACT ON OXIDATIVE STRESS IN STZ-INDUCED DIABETIC RATS

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ABSTRACT: The role of oxidative stress in the development of diabetes mellitus and its vascular complications are extensively studied. Hyperglycaemia causes oxidative damage by generation of reactive oxygen species and results in the development of complications. The present study was undertaken with the objective of exploring the anti-hyperglycaemic potential of phenolic compounds enriched semipurified extract of *Bauhinia purpurea* bark in streptozotocin induced (STZ) diabetic rats for four weeks and to study oxidative stress and antioxidant status. Rats were rendered diabetic by single injection of streptozotocin (60 mg/kg body wt, ip). At the end of the treatment period, the level of blood glucose, serum biochemical markers, serum cholesterol levels and liver malondialdehyde, tissue antioxidant levels were measured. A marked rise was observed in the levels of fasting blood glucose, cholesterol, lipid peroxidative products and a significant decrease in tissue antioxidants (reduced glutathione) levels in STZ treated rats. Oral administration of two semi purified extracts B_1 and B_2 (100 and 50 mg/kg body wt each, p.o) decreased fasting blood glucose levels of STZ-treated diabetic rats significantly ($P < 0.01$), when compared with control rats. In addition, the extracts showed favorable effect ($P < 0.01$) on the reduced tissues antioxidants level, liver glycogen level, cholesterol level, with significant ($P < 0.01$) reduction of elevated lipid peroxidation products. Our study showed the antioxidant effect of phenolic compounds enriched semipurified extracts of *B. purpurea* in STZ induced experimental diabetes. The results also suggested that this polyphenolics rich extract could be potentially useful for hyperglycaemia treatment to correct the diabetic state.

Key words: *Bauhinia purpurea* barks, Oxidative stress, Semipurified fractions, Diabetic rats.

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INTRODUCTION

Bauhinia purpurea L. (BP) belonging to the family Caesalpiniaceae, is such a plant locally known as Kanchan, Rakta Kanchan *etc*., is native of Southern Asia, Southeast Asia, Taiwan, and China. The different plant parts are reported to be traditionally used for treatment of a variety of illness (Chopra *et al*., 1956; Asolkar *et al*., 2000; Parrota 2001; Janardhanan *et al.*, 2003; Kirthikar and Basu 2001).The leaves, stems, and roots are widely used to treat infections, pain, diabetes, jaundice, leprosy, and cough (Morais *et al*., 2005). The literature survey shows that there is scanty report of *in vivo* studies on the effect of semi purified fractions of *B. purpurea* crude ethanolic bark extract on oxidative stress in animal model. In milieu of these observations, the present study had been undertaken to evaluate the anti oxidant potential of two semipurified fractions of this plant's bark crude ethanolic extracts *in vivo.*

MATERIALS AND METHODS

Collection of Plant Materials

The barks of *B. purpurea* (BP) was collected at Shantiniketan campus of Visva-Bharati University, Birbhum district, West Bengal, India, during May 2013 and authenticated by botanist. The herbarium for future reference has been kept in Department of Veterinary Pharmacology and Toxicology, WBUAFS, Belgachia, Kolkata, India.

Preparation of semipurified fractions (SPF)

Semi-purified fractions from crude ethanolic extracts has been prepared following standard procedure of natural product chemistry **(**Harborne 1998; Sarkar *et al*., 2006; Brahmachari 2009). Crude ethanolic extract of *Bauhina purpuria* (BP) barks was prepared as reported earlier (Brahmachari *et al.*, 2015). An amount of 20 g of crude ethanolic extract of BP barks was placed into a stoppered conical flask. To it added 50 ml of 5% aqueous NaOH and shaken well, allowed to settle and filtered through Whatman filter No 1. This process was repeated twice. The filtrate was taken on a beaker and placed on ice bath. To it, dilute acetic acid was poured drop-wise to avoid heat production and the process continues unless solution becomes neutralized. Upon neutralization, the phenolic compounds precipitated out, and filtered off through Whatman No 1 filter paper and crude mass was dried at ambient conditions to obtain a dark pale yellow powder. Yield is 56.23% W/W of dry crude extract. This is partially purified fraction (PPF) from BP crude bark extract.

8 gram of PPF dissolved in minimum amount of ethyl acetate (EtOAc) was loaded in a suitable glass column pre-packed with silica gel (200 g; 60-120 mesh) and eluted sequentially with 2 lit petroleum ether (PE, BP 60-80°C), PE: EtOAc (1:1 *v/v*) and EtOAc to obtain 40 numbers of 50 ml batch at each of the eluents. Each eluent (PE/PE-EtOAc/EtOAc) was evaporated under reduced pressure (using a rotary evaporator) to obtain Fraction A, B and C. Each fraction was then quantitatively evaluated for total phenolic content (TPC). The total phenolic content (mg gallic acid equivalent) was found to be in the order C>B>>>A. The semi purified fractions B and C were selected for biological efficacy test. The average yield of SPF A, B and C were: A – 812 mg; B – 1.85 g; C – 3.93 g.

Estimation of total phenolic content

The concentration of phenolics in plant extracts was determined using

Table 1: Comparison of TPC Content in different semi purified fractions of *B.purpurea* bark with that of crude extract (*Dunnet's multiple comparison test followed by one way ANOVA*).

Groups	Mean Diff.	99% CI of diff.	Significant?	Summary
CEE vs. PPF	-155.8	-180.9 to -130.6	Yes	****
CEE vs. Fraction A	201.6	176.4 to 226.7	Yes	****
CEE vs. Fraction B	-353.4	-378.6 to -328.3	Yes	****
CEE vs. Fraction C	-455.8	-480.9 to -430.6	Yes	****

****: P<0.0001

CEE: Crude Ethanolic Extract; PPF: Partially purified fraction; Fraction A, B, C: Semipurified fractions obtained by column chromatography of PPF.

spectrophotometric method (Singleton *et al.*, 1999). Methanolic solution of the extract in the concentration of 1 mg/ml was used in the analysis. The reaction mixture was prepared by mixing 0.5 ml of methanolic solution of extract, 2.5 ml of 10% Folin-Ciocalteu's reagent dissolved in water and 2.5 ml 7.5% $NaHCO_3$. Blank was concomitantly prepared containing 0.5 ml methanol 2.5 ml 10% Folin-Ciocalteu's reagent dissolved in water and 2.5 ml of 7.5% of $NaHCO_3$. The samples were thereafter incubated in a thermostat at 45^0C for 45 min. The absorbance was determined using spectrophotometer at λ max= 765 nm. The samples were prepared in triplicate for each analysis and the mean value of absorbance was obtained. The same procedure was repeated for the standard solution of gallic acid (50-250 ug/ml) and the calibration line was construed. Based on the measured absorbance, the concentration of phenolics was read (mg/ml) from the calibration line; then the content of phenolics in extracts was expressed in terms of gallic acid equivalent (mg of GA/g of extract).

Chemicals and Reagents

Standards of phenolic acids (gallic acid), Folin-Ciocalteu's phenol reagent, Streptozotocin (STZ) were obtained from Sigma Chemicals Co., St Louis, MO, USA. Standard drug glibenclamide pure samples were procured from M/S Hindustan Chemicals & Pharmaceuticals, West Mumbai, India. All other solvents and chemicals were of analytical grade procured from SISCO Research Laboratories Private Limited, Mumbai, India, Hi-Media Laboratories Ltd, India and Merck India Ltd. All other chemicals and solvents were of analytical grade.

Animals

The study was conducted on Albino Wistar Rats of either sex, five to six weeks of age weighing between 150–200 g, housed in polypropylene cages at an ambient temperature of 25 ± 2°C with 12 h light and 12 h dark cycle. The rats were fed standard diets and water *ad libitum*. The animals were allowed to acclimatize to the laboratory environment for 1 week. All procedures complied with the standards for the care and use of animal subjects as stated in the guidelines laid by Institutional Animal Ethical Committee (IAEC), West Bengal University of Animal and Fishery

Sciences, West Bengal, India.

Acute Toxicity Study

Toxicity test was carried out according to Mukherjee *et al*. (1997). High concentration of the SPFs (5000mg/kg b.wt.) were prepared in normal saline and fed orally to group of three rats orally for 7 consecutive days. The rats were observed for any abnormal behavior such as diarrhea, salivation, respiratory distress, motor impairment, hyper excitability and incidence of mortality. At the end of seventh day the animals were sacrificed and the internal organs (liver, kidney, stomach etc.) were observed for gross morphological changes, weight in comparison to control. No abnormality was detected.

Induction of Type I diabetes

The rats were fasted overnight and administered freshly prepared STZ solution (60mg/kg body weight dissolved in cold citrate buffer, (0.1M, pH 4.5) intra peritoneally. Diabetes was identified by polydipsia, polyuria, and by measuring non fasting plasma glucose levels. Animals with postprandial glycemia over 225 mg/dL, 3 days after STZ administration, were considered diabetic. Whole blood was collected from the tail vein of the rats for glucose estimation in serum. Control rats received only citrate buffer. (Mondal *et al.*, 2012).

Experimental Design

The *in vivo* hypoglycemic and antioxidative effect of the SPFs was studied in STZ- induced diabetic rats. The rats (n = 3 per group) were divided into 7 groups: group 1(NC): normal rats treated with vehicle alone; group 2(DC): diabetic control treated with STZ solution (60 $mgkg^{-1}$ body weight dissolved in cold citrate buffer, 0.1M, pH 4.5) intra peritoneally; group 3 (PC): diabetic rats treated orally with glibenclamide @ 0.5mg/Kg BW; group 4, 5 diabetic rats were administered B1(Fraction C) @ 100 and 50 mg/kg respectively (B11 and B 21); groups 6 and 7 diabetic rats were administered B 2 (Fraction B) @ 100 and 50 mg/kg respectively (B 21 and B 22); the vehicle, standard drug, and extracts were administered orally to respective groups once daily for 28 days. FBS was estimated on 0, 14^{th} and 28^{th} day in blood collected from tail vein (using Reagent Kit). On day 28, the fasted animals were euthanized under ether inhalation. The fasted blood was collected by cardiac puncture with and without EDTA and processed for estimation of glucose, albumin, cholesterol, ALT, AST, using standard commercial reagents kits. Liver tissue was dissected out, washed with ice cold NSS and a 10% homogenate (w/v) was prepared in 0.1M, pH 7.4 PBS, centrifuged at 4°C at 5000g for 30 mins and the supernatant was stored at -20°C before analysis for MDA (Placer *et al*, 1966), NPSH (Sedlak and Lindsay 1969) and glycogen (Carroll *et al.*,1956).

Statistical Analysis

Data were statistically evaluated by using one-way analysis of variance (ANOVA), followed by Dunnett 't-test' using Graphad Prism Version 6 Statistical software (USA).

RESULTS AND DISCUSSION

Diabetes mellitus is the fastest growing metabolic disorder in the world and a major cause of morbidity in developing countries (King *et al.*, 1998). Diabetes mellitus is shown to be associated with increased oxidative stress, which could be a consequence of either increased production of free radicals or reduced antioxidant defenses (Rosen *et al.*, 2001).

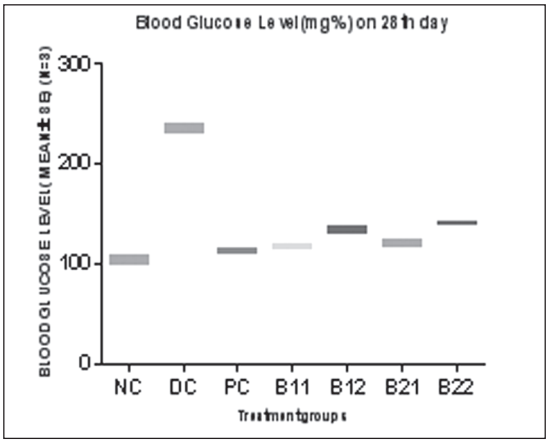

Fig. 1. Blood glucose level of rat on 28th day.

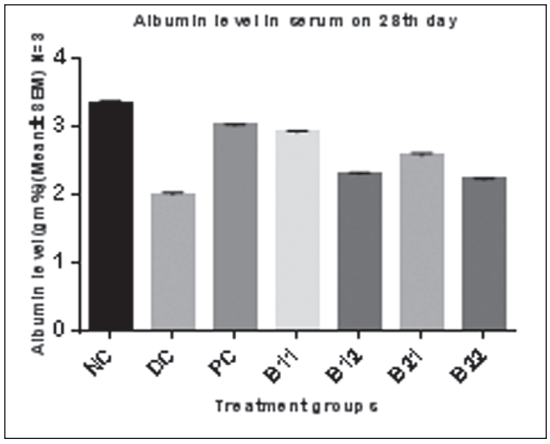

Fig. 2. Serum albumin level of rat on 28th day.

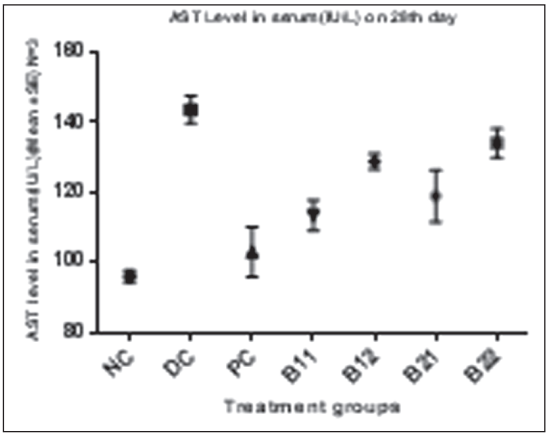

Fig. 3. AST level in serum of rat on 28th day.

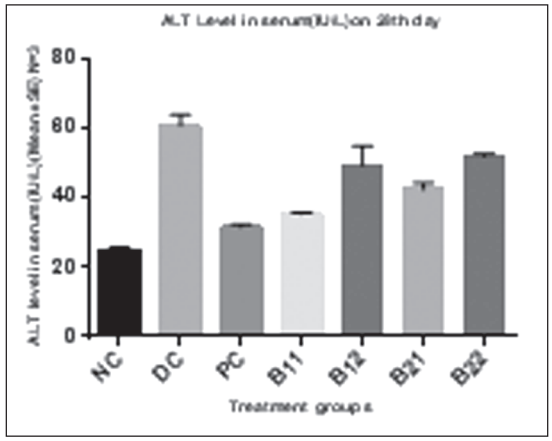

Fig. 4. ALT level in serum of rat on 28th day.

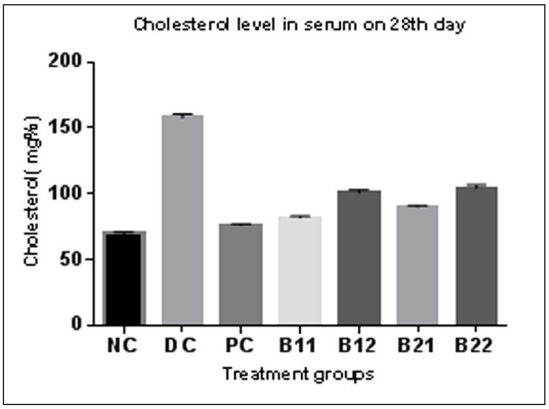

Fig. 5. Cholesterol level in serum of rat on 28th day.

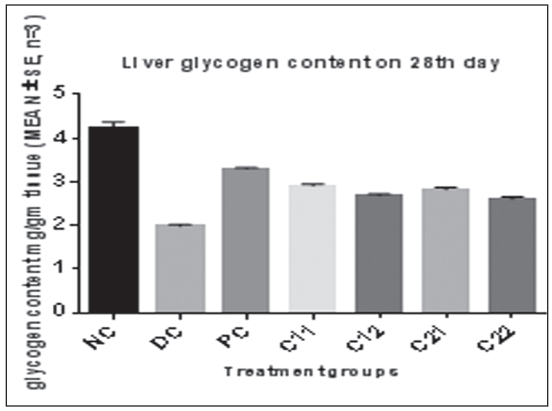

Fig. 6. Liver glycogen content of rat on 28th day.

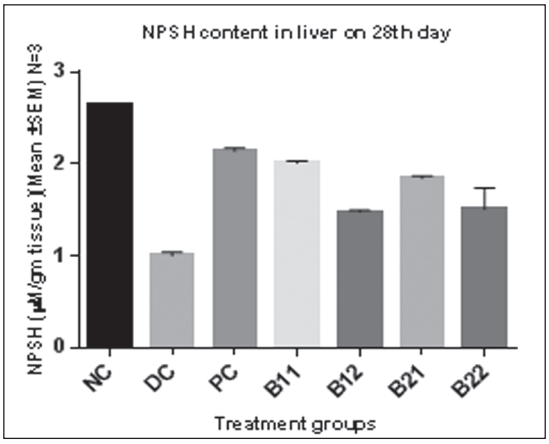

Fig. 7. NPSH content in liver of rat on 28th day.

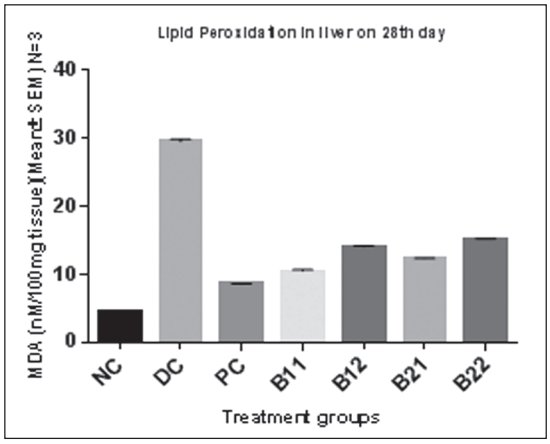

Fig. 8. Lipid peroxidation in liver of rat on 28th day.

Oxygen free radicals are formed disproportionately in diabetes mellitus by glucose oxidation, non-enzymatic glycation of proteins and the subsequent degradation of glycosylated proteins. Diabetic complications are also associated with overproduction of free radicals and accumulation of lipid peroxidation by-products. Enhanced oxidative stress has been well documented in both experimental and human diabetes mellitus (Baynes 1991). Thus attempt has been made to reduce the oxidative stress in patient with diabetes by supplementation with naturally occurring antioxidants (Bursell and King 1999). The major goals of antioxidant treatment have been to reduce oxidative stress by preventing or delaying the progression or reversing the complications of diabetes. Medicinal plants often contain substantial amounts of antioxidants such as polyphenols, flavonoids, anthocyanins and tannins.

During the present investigation it was observed that the total phenolic compound concentration has increased significantly ($P<0.0001$) by semi purification process (Table 1) as compared to crude extract.

Fig. 1,2,3,4 and 5 show the changes in the level of FBG, albumin, AST, ALT, cholesterol in normal and experimental groups of rats. There was a significant elevation in FBG, AST, ALT, cholesterol, and while the levels of albumin decreased during diabetes when compared to control group. Administration of semi purified extracts brought back the levels to near normal values though effects of all the dose levels of two semi purified fractions were not always comparable to that of standard drug glibenclamide in different parameters. Streptozotocin induced diabetic rats showed a significant decrease in liver glycogen content (Fig. 6), NPSH content (Fig. 7) and increase in MDA production (Fig. 8) compared to normal rats. Oral administration of phenol rich extract showed a significant improvement ($P<0.01$) in these parameters when compared to untreated diabetic rats.

The aim of the present study was to demonstrate the efficacy of polyphenolic extract in the reduction of FBG level as well as to determine the recovery in altered biochemical

variables indicative oxidative stress and various organ damages in rats with STZ induced diabetes. The high blood FBG levels were observed in STZ treated rats indicating the establishment of oxidative stress mediated diabetic state (Wright *et al.*, 2006). Administration of graded dose of polyphenolic semi purified extract significantly decreased FBG concentration when compared to the diabetic control. Oral administration of polyphenolic semipurified extract resulted in a significant decrease in serum cholesterol. Elevation of triglycerides level during liver injury could be due to increased availability of free fatty acids, decreased hepatic release of lipoprotein and increased esterification of free fattyacids **(**Sundari *et al.*, 2011**)**. The cholesterol contents in plasma registered a significant hike in diabetic control group, which was retrieved to near normalcy in polyphenolic semi purified extract treated groups. This observation indicates the lipid lowering potential of *B. purpurea.*

Increase in the plasma ALT and AST are observed in the condition in which pancreas, liver, kidney and heart are destroyed by STZ. Moreover, the activities of these enzymes have been used as indicators of tissue toxicity in experimental diabetes. Increased levels of AST, ALT were seen in STZ induced diabetic rats, over a four weeks period. Necrosis or membrane damage causes the release of enzymes into circulation and hence it can be measured in the serum. SGPT catalyses the conversion of alanine to pyruvate and glutamate and is released in a similar manner. Therefore SGPT is more specific to the liver, and is thus a better parameter for detecting liver injury. Elevated levels of serum enzymes are indicative of cellular leakage and loss of functional integrity of cell membrane in liver. Treatment with semi purified extracts showed potential hepato - protective activity as reported in earlier study (Portha *et al.***,** 1989). Protein metabolism is a major project of liver and a healthy functioning liver is required for the synthesis of the serum proteins except for γ globulins. Hypo-proteinemia is a feature of liver damage due to significant fall in protein synthesis as manifested by hypoalbuminaemia in diabetic rats .Glycogen synthesis in the rat liver and skeletal muscles is impaired during diabetes (Akatsuka *et al.,* 1983). The regulation of glycogen metabolism occurs by the multifunctional enzyme glycogen synthase and glycogen phosphorylase that play a major role in the glycogen metabolism (Janero 1990). The reduced glycogen store in rats with experimentally induced diabetes has been attributed to reduced activity of glycogen synthase and increased activity of glycogen phosphorylase. In the present study the experimental diabetic rats treated with polyphenolic semipurified extract and glibenclamide treated groups restored the level of hepatic glycogen may be by means of increasing the activity of glycogen synthase enzyme.

Measurement of tissue TBARS help to assess the extent of tissues damage and elevated MDA observed in liver of diabetic rat can be related to overproduction of lipid peroxidation by-products and diffusion from damaged tissues (Mukherjee *et al.,* 1994). Enhanced TBARS and declined antioxidants observed in the liver, of diabetic rats can be attributed to increased biomembrane lipid peroxidation process and thereby contributing to alteration in antioxidant status (McLennan *et al.*, 1991**).** The decrease in thiobarbituric acid reactive substance

(TBARS) of various tissues clearly showed the antioxidant property of semipurified extract. These findings suggest that the semi-purified extract may exert antioxidant activity and protect the tissue from lipid peroxidation.

In the current study, NPSH (Cellular glutathione) activities of diabetic rat liver was significantly reduced. This may be due to the production of reactive oxygen free radicals that can themselves reduce the activity of these enzymes. Streptozotocin induced diabetic rats exhibited high level of oxidative stress manifested by significantly ($P<0.01$) increased MDA production in liver with concomitant depletion in cellular glutathione content (Brahmachari *et al.,* 2015). The lowered glutathione level in diabetes has been considered an important indicator of increased oxidative stress (Du Thie *et al.,* 2000).

Antioxidant action of phenolic compounds is due to their high tendency to chelate metals. Phenolics possess hydroxyl and carboxyl groups, able to bind particularly iron and copper (Jun *et al.,* 2003).They may inactivate iron ions by chelating and additionally suppressing the superoxide-driven Fenton reaction, which is believed to be the most important source of ROS (Arora *et al.,* 1998, Rice-Evans *et al.,* 1997). According to Morgan *et al.* (1997), this general chelating ability of phenolic compounds is probably related to the high nucleophilic character of the aromatic rings rather than to specific chelating groups within the molecule. Phenolic antioxidants inhibit lipid peroxidation by trapping the lipid alkoxyl radical. This activity depends on the structure of the molecules, and the number and position of the hydroxyl group in the molecules (Millic *et al.,* 1997). They stabilize membranes by decreasing membrane fluidity (in a concentration-dependent manner) and hinder the diffusion of free radicals and restrict peroxidative reaction (Blokhina *et al.,* 2003, Arora *et al.,* 2000). According to Verstraeten *et al.* (2003), in addition to known protein-binding capacity of flavanols and procyanidins, they can interact with membrane phospholipids through hydrogen bonding to the polar head groups of phospholipids. As a consequence, these compounds can be accumulated at the membranes' surface, both outside and inside the cells. Through in this kind of interaction, as they suggest, selected flavonoids help maintain membranes' integrity by preventing the access of deleterious molecules to the hydrophobic region of the bilayer, including those that can affectmembrane rheology and those that induce oxidative damage to the membrane components. On the other hand, in vitro studies have shown that flavonoids can directly scavenge molecular species of active oxygen:·O2-superoxide, H_2O_2 - hydro genperoxide, OH-hydroxyl radical, $1O^{2\cdot}$singlet oxygen or peroxyl radical. Their antioxidant action resides mainly in their ability to donate electrons or hydrogen atoms (Arora *et al.,* 1998, Sakhiha *et al.,* 2000). Poly-phenols possess ideal structural chemistry for this activity and have been shown to be more effective *in vitro* than vitamins E and C on molar basis (Rice-Evans *et al.,* 1997).

Flavonoid rich fractions (FRF) fraction from red algae *A. spicifera* was potent anti-diabetic and antioxidant asset against STZ induced diabetes and oxidative tissue breakups (Vuppalapati *et al.,* 2016). It is possible that the delay in STZ induced oxidative stress in various tissues of polyphenolic extract treated rats is predominantly due to its antioxidant activity. Polyphenolic extract of *B. purpurea*

may also act by either directly scavenging reactive oxygen metabolites due to the presence of various antioxidant compounds or by increasing the level of endogenous antioxidant molecules or enzymes. Further, pharmacological and chemical studies are required to explore the mechanism of action of active ingredient(s) responsible for the antioxidant activity observed.

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REFERENCES

Akatsuka A, Singh TJ, Huang KP (1983) Comparison of liver glycogen synthase from normal and streptozotocin induced diabetic rats. Arch Biochem Biophy 220:426–34.

Arora A, Nair MG, Strasbur GGM (1998) Structure-activity relationships for antioxidant activities of a series of flavonoids in a liposomal system. Free Radic Biol Med 24: 1355.

Arora A, Byrem TM, Narim G, Strasbur GGM (2000) Modulation of liposomal membranes fluidity by flavonoids and isoflavonoids. Archives Biochem Biophy 373: 102.

Asolkar LV, Kakkar KK, Chakre OJ (2000) Supplement to Glossary of Indian Medicinal Plants, Part I (A-K), National Institute of Science Communication, New Delhi, India.

Baynes JW (1991) Role of oxidative stress in development of complications in diabetes. Diabetes 40: 405–412.

Blokhina O, Virolainen ENE, Fagerstedt KV (2003) Antioxidants, oxidative damage and oxygen deprivation stress: a review. Ann Bot 91: 179.

Brahmachari AK, Badyopadhyay SK, Mandal TK, Brahmachari G, Pattanayak S, De A, Das AK, Batabyal S (2015) Comparative evaluation of hypoglycemic effects of two different parts of *Bauhuniapurpurea*Linn plant in STZ-induced diabetic Albino Wistar rats.. Explor Anim Med Res 5(2): 120-132.

Brahmachari G (Editor) (2009) Natural Products: Chemistry, Biochemistry and Pharmacology, Alpha Science, Oxford, UK.

Bursell SE, King GL (1999) Can protein kinase C inhibition and vitamin E prevent the development of diabetic vascular complications? Diabetes Res Clin Pract 45: 169–182.

Carroll NV, Longley RW, Roe HJ (1956) The determination of glycogen in liver and muscle by use of anthrone reagent. J Biol Chem 220: 583-593.

Chopra RN, Nayar SL, Chopra IC (1956) Glossary of Indian Medicinal Plants, Publication and Information Directorate, Hill Side Road, New Delhi, India.

Du Thie G, Crozier A (2000) Plant derived polyphenolic antioxidants. Curr Opin Nutr Metab 3: 447–451.

Harborne J B (1998) Phytochemical methods. A guide to Modern Techniques of Plant Analysis, 3rd edn. Chapman and Hall, London, UK.

Janardhanan K, Vadivel V, Pugalenthi M (2003) Biodiversity in Indian Under-exploited/Tribal Pulses.

Cited in: Jaiwal PK, Singh RP (Eds.), Improvement Strategies for Leguminosae Biotechnology. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Janero DR (1990) Malondialdehyde and thiobarbituric acid reactivity as diagnostic indices of lipid peroxidation and peroxidative tissue injury. Free Rad Biol Med 9: 515–540.

Jun GCH, Maeder V, Funk F, Frey B, Sticher H, Frosserd E (2003) Release of phenols from *Lupinus albus* L. roots exposed to Cu and their possible role in Cu detoxification. Plant Soil 252: 301.

King H, Aubert R, Herman WH (1998) Global burden of diabetes, 1995-2025: Prevalence, numerical estimates and projections. Diabetes Care 21:1414–1431.

Kirthikar KR, Basu DB (2001) Indian Medicinal Plants, 3rd edn., Vol. 4. Oriental Enterprises Dehradun, India.

McLennan SV, Heffernan S, Wright L, Rae C, Fisher E, Yue DK (1991) Changes in hepatic glutathione metabolism in diabetes. Diabetes 40: 344–348.

Millic BL, Djilas SM, Canadanovic-Brunet JM (1998) Antioxidative activity of phenolic compounds on the metal-ion breakdown of lipid peroxidation system. Food Chem 61: 443.

Morgan JF, Klucas RV, Grayer RJ, Abian J, Becana M (1997) Complexes of iron with phenolic compounds from soybean nodules and other legume tissues: prooxidant and antioxidant properties. Free Radic Biol Med 22 : 861.

Morais SM, Dantas JDP, Silva ARA, Magalhães EF (2005) Plantas medicina isusada spelos índios Tapebas do Ceará. Rev Bras Farmacogn 15: 169–177.

Mondal S, Bhattacharya S, Biswas M (2012) Antidiabetic activity of *Areca catechu* leaf extracts against streptozotocin induced diabetic rats. J Adv Pharma Edu Res 2 (1): 10-17.

Mukherjee PK, Saha K, Pal M, Saha B (1997) Effect of Nelumbo nucifera rhizome extract on blood sugar level in rats. J Ethnopharmacol 58(3): 207-213.

Mukherjee B, Mukherjee JR, Chatterjee M (1994) Lipid peroxidation, glutathione levels and change in glutathione-related enzyme activities in streptozotocin-induced diabetic rats. Immunol Cell Biol 72: 109–114.

Parrota JA (2001) Healing Plants of Peninsular India. CABI Publishing, Oxford, UK.

Placer ZA, Cushman LL, Johnson BC (1966) Estimation of product of lipid peroxidation (malondialdehyde) in biochemical system. Anal Biochem 16: 359-364.

Portha B, Blondel O, Serradas P (1989) The rat models of non-insulin dependent diabetes induced by neonatal streptozotocin. Diabet Metab 15: 61–75.

Rajesh SV, Rajkapoor B, Kumar R, Raju K (2009) Effect of Clausena dentata (Willd.) M. Roem. against paracetamol induced hepatotoxicity in rats. Pak J Pharm Sci 22(1): 90-93.

Rosen P, Nawroth PP, King G, Moller W, Tritschler HJ, Packer L (2001) The role of oxidative stress in the onset and progression of diabetes and its complications: a summary of a Congress Series sponsored by UNESCO-MCBN, the American Diabetes Association and the German Diabetes Society. Diabetes Metab Res Rev 17: 189–212.

Rice-Evans CA, Miller NJ, Paganga G (1997) Antioxidant properties of phenolic compounds. Trends Plant Sci 2: 152.

Sakiha Ma Y, Mano J, Sano S, Asa Da K, Ya Makashi H (2000) Reduction of phenoxyl radicals mediated by mono dehydroascorbate reductase. Biochem Biophys Res Comm 279: 949.

Sarkar SD, Latif Z, Gray AI (Editors) (2006) Humana Natural product Isolation, 2nd edn. Press Inc. New Jersey.

Sedlak J, Lindsay RH (1968) Estimation of total protein bound and nonprotein sulfhydryl groups in tissue with Ellman's reagent. Anal Biochem 25: 192-205.

Singleton VL, Orthofer R, Lamuela-Raventos RM (1999) Analysis of total phenols and other oxidation substrates and antioxidants by means of Folin-Ciocalteu reagent. Methods Enzymol 299: 152-178.

Sundari K, Govindaraju G, Bharathi B (2011) Hepatoprotective effect of ethanolic extracts of Sphaeranthus indicus (Linn) on paracetamol-induced liver toxicity in rats. Int J Applied Biol Pharma Tech 2(2): 315-320.

Wright E Jr, Scism-Bacon JL, Glass LC (2006) Oxidative stress in type 2 diabetes: the role of fasting and postprandial glycaemia. Int J Clin Pract 60: 308–314.

Verstraeten SV, Keen CL, Schmitz HH, Fra Ga CG, Oteiza PI (2003) Flavan-3-ols and procyanidins protect liposomes against lipid oxidation and disruption of the bilayer structure. Free Radic Bio Med 34: 84.

Vuppalapati L, Velayudam R, Nazeer Ahamed KFH, Cherukuri S, Reddy BR (2016) The protective effect of dietary flavonoid fraction from *Acanthophora spicifera* on streptozotocin induced oxidative stress in diabetic rats. Food Science and Human Wellness, In Press, Corrected Proof — Note to users doi:10.1016/j.fshw.2016.02.002.

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