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In-vivo antioxidant activity of *Premna corymbosa* (Rottl.) against streptozotocin induced oxidative stress in Wistar albino rats

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ABSTRACT

The aim of the study was to investigate the *invivo* antioxidant activity of ethanolic extract *Premna corymbosa* (Rottl.) root against streptozotocin induced oxidative stress in different organs (liver, kidney, brain, heart and pancreas) of rats. Ethanolic extract of *P.corymbosa* (Rottl.) root was administered orally (200 mg/kg body weight) and the effect of extract on enzymatic antioxidants like superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione-S-transferase (GST) and polyphenol oxidase (PPO), non enzymatic like vitamin C, vitamin E and glutathione. Lipid peroxidation like basal, ascorbate and peroxide induced lipid peroxidation were also estimated. Glibenclamide was used as standard reference drug. A significant increase in the activities of superoxide dismutase, catalase, glutathione peroxidase, glutathione-S-transferase and reduced glutathione were observed in different organs of diabetic rats on treatment with 200 mg/kg body weight of *P.corymbosa* (Rottl.) root extract and glibenclamide for 30 days treatment. Both the treated groups showed significant decrease in lipid peroxidation, suggesting its role in protection against lipid peroxidation induced membrane damage. *P.corymbosa* (Rottl.) possesses antioxidant potential which may be used for therapeutic purposes mainly in the prevention of oxidative damage that occur during diabetes.

INTRODUCTION

Oxidative stress is a large increase in the cellular reduction potential of the cellular redox couples. Free radicals cause a chain reactions leading to consecutive oxidation. These radicals attack molecules like fat, protein, DNA, sugar etc. Antioxidants are beneficial components that neutralize free radicals before they can attack cell proteins, lipids and carbohydrates. The mechanism involves significant inhibition or delay in the oxidative process (Saha and Tamrakar, 2011). The disfunctioning of antioxidant enzymes has been implicated in several disorders including rheumatoid arthritis, reperfusion injury, cardio-vascular diseases, immune injury as well as diabetes mellitus (Pari and Latha, 2004). Hyperglycemia, which occurs during diabetes (both type 1 and type 2) and, to a lesser extent, during insulin resistance, causes oxidative stress.

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Oxidative stress may be important in diabetes, not just because of its role in the development of complications, but because persistent hyperglycemia, secondary to insulin resistance, may induce oxidative stress and contribute to beta cell destruction in type 2diabetes (King and Loeken, 2004). Moreover, diabetes also induces changes in the tissue content and activity of the antioxidant enzymes (Ugochukwu *et al.,* 2003). Many minor components of foods, such as secondary plant metabolites, have been shown to alter biological processes, which may reduce the risk of chronic diseases in diabetic humans (Gupta *et al.,* 2008). There are several oral hypoglycemic agents used therapeutically but certain adverse effects and weak effectiveness of them has led to the search for more effective agents. Therefore, herbal drugs are gradually gaining popularity in the treatment of diabetes mellitus. The major qualities of herbal medicine seem to be their supposed efficacy; low incidence of serious adverse effects (Shanmugasundaram *et al.,* 2011). *Premna corymbosa* (Rottl.) is a small-sized tree or large shrub and the plant is widely distributed throughout india in the

plains. It is commonly known as munna and is an ingredient of much ayurvedic preparation. The roots are useful in vitiated conditions of neuralgia, inflammations, cardiac disorders, hepatopathy, cough, asthma, bronchitis, leprosy, skin diseases, dyspepsia, flatulence, colic, anorexia, constipation, haemorrhoids, fever, diabetes and general debility (Karthikeyan and Deepa, 2008). The purpose of the present study was to evaluate the *invivo* antioxidant activity of *Premna corymbosa* (Rottl.) root extract in the different organs of strptozotocin induced rats.

MATERIAL AND METHODS

Plant Materials

The root of *Premna corymbosa* (Rottl.) were procured from the Trichur, Kerala and was identified by Botanist at Kottakkal Arya Vaidyasala Herbal garden, Kerala and the voucher specimen number is KPZ/ HG/ 259 A/ 09-10.

Successive Solvent Extraction

Premna corymbosa (Rottl.) root was washed well with water. They were air dried at 25^0C for 5 days in the absence of sunlight and powdered well using a mixer. The powdered medicinal plant material was taken and subjected to successive solvent extraction. About 25g of powdered plant material was extracted with 125ml ethanol using a separating funnel with occasional shaking for 16 hours. The extract was concentrated by Rotary flask evaporator (Buchi type).

Experimental animals

Adult Wistar albino rats weighing about 150-180 g were obtained from the animal house of Karpagam University, Coimbatore and were used for the study. Rats were housed in polycarbonate cages in a room with a 12-hour day-night cycle, at constant temperature of 22°C and humidity of 45-64%.During the experimental study rats were fed on pellets (Gulmohur rat feed, Lipton India, Bangalore) with free access to tap water.

The rats received humane care according to the criteria outlined in Principles of Laboratory Animal Care, 1985. All the study was approved by Institutional Animal Ethical Committee

Induction of diabetes

Rats were rendered diabetic by a single intraperitonial injection of freshly prepared streptozotocin (45mg/kg body weight) in 0.1M citrate buffer (pH 4.5) in a volume of 1ml/kg body weight using sterile 25G needle.

Diabetes was identified in rats by moderate polydipsia and marked polyuria. After 48 hrs of streptozotocin a dministration, blood glucose levels were estimated in rats following overnight fasting. Rats with a blood glucose level ranging between 200-300mg/dl were considered diabetic and used for the experiments. Five rats injected with 1ml citrate buffer with normoglycemia served as control.

Antioxidant study

The rats were divided into five groups of five animals each. The animals of group I served as the control. Group II was given a single intraperitoneal injection of STZ (45mg/kg body weight). Group III rats were given the standard drug, glibenclamide at a dose of 2mg/kg body weight and a single dose of STZ. Group IV was given *Premna corymbosa* (Rottl.) (200 mg/kg body weight) along with the single dose of STZ. Group V served as the control group which was given only the *Premna corymbosa* (Rottl.). The drug and the plant samples were given through oral gastric tube.

Estimation of antioxidant parameters in tissues

In the present study, antioxidant activity of *Premna corymbosa* (Rottl.) root extract was analysed in five organs namely liver, kidney, pancreas, brain and heart. The rats were sacrificed on 30^{th} day after the drug and STZ treatment by cervical dislocation after giving mild anaesthesia using chloroform. These tissues were used for estimation of protein (Lowry *et al*., 1951), enzymic antioxidants such as superoxide dismutase (Misra & Fridovich, 1972), catalase (Sinha, 1972), GPx (Rotruck *et al.,* (1973), GST (Habig *et al.,* (1981), polyphenol oxidase (Esterbauer *et al.,* 1977). Non enzymatic antioxidants such as vitamin C (Omaye *et al.,* (1973), vitamin E (Baker *et al.,* 1980) and glutathione (Moron *et al,* 1979) were estimated. Estimation of lipid peroxidation such as basal (Hogberg *et al*., (1974), ascorbate and peroxide induced lipid peroxidation (Devasagayam and Tarachand 1987) were also estimated.

RESULT AND DISCUSSION

Non Enzymatic antioxidants

Oxidative stress in diabetes mellitus revealed the reduction in the antioxidant status and glycation of proteins, inactivation of enzymes, and alteration in structural functions of collagen basement membrane (Nirmala *et al.,* 2011). Antioxidants are substances or nutrients in our foods which can prevent or slow the oxidative damage to our body. When our body cells use oxygen, they naturally produce free radicals (by-products) which can cause damage (Khalil *et al.,* 2012**).** Vitamin C plays a central role in the antioxidant protective system, protecting all lipids undergoing oxidation and diminishing the number of apoptotic cells (Sadi *et al.,* 2008) and it also regenerates the oxidized vitamin E (Chen *et al.,* 2005). Vitamin E, on the other hand, acts as a non-enzymatic antioxidant and reduces chain reactions of lipid peroxidation (Punithavatki *et al.,* 2008). Vitamin E is very effective in glycemic control, lowering the HbA1c levels (Ihara *et al.,* 2000).

Table (1-5) showed decreased levels of nonenzymatic antioxidant vitamin C and E was in diabetic rats, when compared to that of control rats. The levels of these antioxidants were significantly increased in different organs (liver, kidney, brain, heart and pancreas) of diabetic rats by treating with root extract of *P.corymbosa* (Rottl.). GSH has a multifaceted role in anti-oxidant defence. It is a direct scavenger of free radicals as well as a co-substrate for peroxide detoxification by glutathione peroxidases.

Oxidative stress in diabetes decreased the level of GSH in different organs of rat when compared to control. Significant

elevation of GSH level was observed in the *P.corymbosa* (Rottl.) root extract-treated diabetic rats.

This indicates that the extract can reduce the oxidative stress leading to less degradation of GSH due to less production of ROS in diabetic stage.

Enzymatic antioxidants

Oxidative stress is a condition of reduction in anti oxidative enzymes like SOD, CAT, GP_x and GST (Snehal *et al*., 2009). The antioxidant enzymes SOD and CAT play an important role in reducing cellular stress. SOD scavenges the superoxide radical by converting it to hydrogen peroxide and molecular oxygen, while CAT brings about the reduction of hydrogen peroxides and protects higher tissues from the highly reactive hydroxyl radicals (Ragini *et al.*, 2011).

From the table (6-10), activities of SOD, CAT, GPx and GST antioxidants were significantly decreased in different organs (liver, kidney, brain, heart and pancreas) of diabetic control rats due to inadequacy of the antioxidant defences in combating ROS mediated damage.

The decreased activities of CAT and SOD may be a response to increased production of H_2O_2 and O_2 by the aut-oxidation of glucose. These enzymes plays an important role in maintaining physiological levels of oxygen and hydrogen peroxide by hastening the dismutation of oxygen radicals and eliminating organic peroxides and hydroperoxides generated from inadvertent exposure to STZ (Pari and Latha, 2004). Treatment with root extract of *P.corymbosa* (Rottl.) increased the activity of these enzymes and may help to control free radicals when compared to diabetic rats. The effect produced by plant extract was comparable with that of standard drug glibenclamide.

Lipid peroxidation

Lipid peroxidation is a free radical mediated process leading to oxidative deterioration of polyunsaturated lipids. Under normal physiological conditions, low concentrations of lipid peroxides are found in plasma and tissues. Oxygen derived free radicals generated in excess in response to various stimuli could be cytotoxic to several tissues. Most of the tissue damage is considered to be mediated by these free radicals by attacking membranes through peroxidation of polyunsaturated fatty acids. The increase in oxygen free radicals in diabetes could be primarily due to increase in blood glucose levels, which upon auto-oxidation generate free radicals (Malini *et al.*, 2011). In the present study, Table (11-14) indicates, the levels of basal lipid peroxidation, ascorbate induced lipid peroxidation, peroxide induced lipid peroxidation were significantly increased in liver, kidney, brain and pancreas of STZ-induced diabetic rats which might be due to an increase in the generation of free radicals by STZ. The increased susceptibility of the tissues of the diabetic animals may be due to the activation of the lipid peroxidation system. The possible source of oxidative stress in diabetes includes shifts in redox balance resulting from altered carbohydrate and lipid metabolism, increased generation of reactive oxygen species (Laight *et al.*, 2000). Significantly decreased levels of lipid peroxidations in administration of root extract of *P.corymbosa* (Rottl.) in STZ-induced diabetic rat tissues when compared with diabetic control rats which may be responsible for scavenging free radicals liberated by STZ and thus enhance both enzymic and non-enzymic antioxidants in diabetic rats treated with root extract of *P.corymbosa* (Rottl.). *P.corymbosa* (Rottl.) extract alone treated rats maintain the similar enzymes alternation levels as a control rats.

Table. 1: Concentration of vitamin C, vitamin E and glutathione in liver of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* treated (Group IV)	*P.corymbosa* alone treated (Group V)
Vitamin C (mg/g of fresh tissue)	1.50±0.438[a]	0.65±0.36[b]	1.42±0.404[c]	1.37±0.501[d]	1.48±0.444[a]
Vitamin E (mg/g of fresh tissue)	6.54±0.460[a]	3.94±0.452[b]	5.84±0.4294[a]	5.77±0.480[a]	6.52±0.470[a]
Glutathione (µg/mg protein)	49.14±0.345[a]	30.41±0.501[b]	43.64±0.509[c]	42.45±0.466[d]	48.50±0.455[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 2: Concentration of vitamin C, vitamin E and glutathione in kidney of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* treated (Group IV)	*P.corymbosa* alone treated (Group V)
Vitamin C (mg/g of fresh tissue)	1.28±0.341[a]	0.48±0.071[b]	1.35±0.472[c]	1.26±0.459[a]	1.30±0.382[a]
Vitamin E (mg/g of fresh tissue)	4.68±0.514[a]	2.03±0.381[b]	4.19±0.451[c]	4.04±0.421[d]	4.54±0.458[a]
Glutathione (µg/mg protein)	48.40±0.428[a]	22.57±0.360[b]	45.47±0.48[c]	39.22±0.3213[d]	48.93±0.504[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 3: Concentration of vitamin C, vitamin E and glutathione in brain of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* treated (Group IV)	*P.corymbosa* alone treated (Group V)
Vitamin C (mg/g of fresh tissue)	1.04±0.519[a]	0.59±0.428[b]	0.83±0.375[c]	0.77±0.371[d]	0.99±0.420[a]
Vitamin E (mg/g of fresh tissue)	1.86±0.528[a]	0.87±0.494[b]	1.57±0.483[c]	1.46±0.454[d]	1.83±0.498[a]
Glutathione (µg/mg protein)	30.53±0.450[a]	18.08±0.49[b]	28.65±0.493[c]	27.29±0.587[d]	29.99±0.447[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 4: Concentration of vitamin C, vitamin E and glutathione in heart of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* treated (Group IV)	*P.corymbosa* alone treated (Group V)
Vitamin C (mg/g of fresh tissue)	0.90±0.487[a]	0.66±0.492[b]	0.87±0.493[c]	0.89±0.5101[a]	0.89±0.491[a]
Vitamin E (mg/g of fresh tissue)	2.49±0.438[a]	1.07±0.450[b]	2.12±0.470[c]	2.01±0.463[c]	2.31±0.473[a]
Glutathione (µg/mg protein)	31.62±0.486[a]	19.6±0.536[b]	29.92±0.514[cd]	29.35±0.578[c]	30.64±0.513[ad]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 5: Concentration of vitamin C, vitamin E and glutathione in pancreas of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* Treated (Group IV)	*P.corymbosa* alone treated (Group V)
Vitamin C (mg/g of fresh tissue)	1.17±0.466[a]	0.65±0.502[b]	1.08±0.463[a]	1.01±0.461[a]	1.00±0.547[a]
Vitamin E (mg/g of fresh tissue)	1.31±0.468[abc]	0.64±0.483[b]	1.12±0.514[bc]	1.00±0.402[ac]	1.07±0.461[a]
Glutathione (µg/mg protein)	31.62±0.486[a]	11.47±0.433[b]	17.29±0.319[c]	16.54±0.470[c]	19.70±0.536[d]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 6: Activities of antioxidant enzymes peroxidase, superoxide dismutase an catalase in liver of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+ Glibenclamide treated(Group III)	Diabetic+ *P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated (Group V)
Catalase (µmoles of H_2O_2 utilized /min/mg/protein)	60.25±0.035[a]	44.05±0.036[b]	55.70±0.322[c]	53.16±0.027[d]	59.90±0.409[a]
GPx(µg of GSH/mg of protein)	25.61±0.035[a]	12.36±0.496[b]	21.46±0.314[c]	20.35±0.031[d]	25.26±0.031[a]
SOD(Units/g tissue)	6.8±0.0497[a]	3.90±0.357[b]	6.34±0.040[ac]	6.06±0.067[c]	6.64±0.035[ac]
GST (µmoles of CDNBConjugate formed/mg of protein	9.72±0.031[a]	5.06±0.314[b]	9.43±0.450[ac]	8.89±0.545[c]	9.68±0.045[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 7: Activities of antioxidant enzymes peroxidase, superoxide dismutase and catalase in kidney of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated (Group III)	Diabetic+ *P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Catalase (µmoles of H_2O_2 utilized /min/mg/protein)	27.56±0.273[a]	14.20±0.0357[b]	26.27±0.049[c]	24.04±0.031[d]	26..99±0.049[a]
GPx (µg of GSH/mg of protein)	33.56±0.036[a]	10.76±0.519[b]	27.05±0.031[c]	26.75±0.031[c]	32.98±0.032[a]
SOD (Units/g tissue)	2.03±0.314[a]	0.84±0.040[b]	1.77±0.036[ac]	1.68±0.045[c]	1.92±0.035[ac]
GST(µmoles of CDNBConjugate formed/mg of protein	7.56±0.031[a]	4.01±0.035[b]	7.32±0.040[c]	7.17±0.040[d]	7.52±0.031[a]

Values are expressed as Mean ± S.D of five individual experiments. Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 8: Activities of antioxidant enzymes peroxidase, superoxide dismutase and catalase in brain of control and experimental groups

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated(Group III)	Diabetic+*P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Catalase (µmoles of H_2O_2 utilized /min/mg/protein)	15.23±0.403[a]	9.73±0.403[b]	14.6±0.403[a]	13.50±0.357[c]	16.21±0.375[d]
GPx (µg of GSH/mg of protein)	46.13±0.035[a]	19.63±0.049[b]	39.06±0.400[c]	40.87±0.049[d]	45.85±0.043[a]
SOD (Units/g tissue)	1.51±0.035[a]	0.68±0.049[b]	1.27±0.040[c]	1.18±0.049[d]	1.48±0.040[a]
GST(µmoles of CDNBConjugate formed/mg of protein	3.05±0.049[a]	1.02±0.035[b]	2.62±0.036[c]	2.52±0.040[d]	2.93±0.045[a]

Values are expressed as Mean ± S.D of five individual experiments. Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 9: Activities of antioxidant enzymes peroxidase, superoxide dismutase and catalase in heart of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated (Group III)	Diabetic+*P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Catalase (µmoles of H_2O_2 utilized /min/mg/protein)	17.70±0.329[a]	12.76±0.281[b]	16.20±0.361[c]	15.90±0.040[c]	17.50±0.315[a]
GPx (µg of GSH/mg of protein)	39.06±0.450[a]	27.84±0.045[b]	35.50±0.357[c]	33.28±0.045[d]	38.80±0.053[a]
SOD (Units/g tissue)	1.22±0.035[a]	0.71±0.036[b]	1.16±0.0361[ab]	1.13±0.285[ab]	1.22±0.141[a]
GST(µmoles of CDNBConjugate formed/mg of protein	4.38±0.049[a]	2.08±0.040[b]	3.87±0.045[c]	3.62±0.035[d]	4.00±0.058[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 10: Activities of antioxidant enzymes peroxidase, superoxide dismutase and catalase in pancreas of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated(Group III)	Diabetic+*P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated (Group V)
Catalase (µmoles of H_2O_2 utilized /min/mg/protein)	26.60±0.045[a]	16.32±0.040[b]	24.31±0.040[c]	25.51±0.040[d]	26.14±0.053[a]
GPx (µg of GSH/mg of protein)	30.00±0.058[a]	18.20±0.049[b]	26.30±0.031[c]	26.00±0.049[d]	29.77±0.040[a]
SOD (Units/g tissue)	1.08±0.062[a]	0.41±0.036[b]	0.90±0.040[c]	0.87±0.040[c]	1.02±0.040[a]
GST(µmoles of CDNBConjugate formed/mg of protein	2.00±0.079[a]	0.32±0.035[b]	1.65±0.031[ac]	1.50±0.447[c]	1.82±0.044[ac]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 11: Basal, ascorbate induced and peroxide induced lipid peroxidation in liver of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated (Group III)	Diabetic+*P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Lipid peroxidation (nano moles of MDA formed/ g tissue)	15.97±0.456[a]	31.99±0.460[b]	19.00±0.501[c]	22.17±0.474[d]	15.18±0.542[a]
Ascorbate induced lipid peroxidation (nano moles of MDA formed/ g tissue)	19.87±0.478[a]	39.76±0.492[b]	24.10±0.450[c]	27.52±0.452[d]	19.11±0.347[a]
Peroxide induced lipid peroxidation (nano moles of MDA formed/ g tissue)	14.42±0.501[a]	28.89±0.456[b]	17.75±0.470[c]	19.99±0.509[d]	13.98±0.483[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 12: Basal, ascorbate induced and peroxide induced lipid peroxidation in kidney of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated(Group III)	Diabetic+*P.corymbosa* treated (Group IV)	*P.corymbosa* alone treated(Group V)
Basal lipid peroxidation (nano moles of MDA formed/ g tissue)	12.35±0.462[a]	28.29±0.505[b]	18.00±0.522[c]	20.92±0.509[d]	11.87±0.565[a]
Ascorbate induced lipid peroxidation (nano moles of MDA formed/ g tissue)	15.33±0.492[a]	34.94±0.522[b]	22.20±0.542[c]	26.92±0.509[d]	15.05±0.332[a]
Peroxide induced lipid peroxidation (nano moles of MDA formed/ g tissue)	11.09±0.514[a]	25.41±0.506[b]	16.66±0.496[c]	18.93±0.439[d]	10.86±0.524[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 13 : Basal, ascorbate induced and peroxide induced lipid peroxidation in brain of control and experimental groups.

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated(Group III)	Diabetic+*P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Basal lipid peroxidation (nano moles of MDA formed/ g tissue)	15.70±0.545[a]	39.36±0.483[b]	17.13±0.465[c]	18.60±0.494[c]	15.67±0.519[a]
Ascorbate induced lipid peroxidation (nano moles of MDA formed/ g tissue)	19.59±0.456[a]	48.68±0.429[b]	21.13±0.465[c]	22.66±0.438[d]	19.59±0.429[a]
Peroxide induced lipid peroxidation (nano moles of MDA formed/ g tissue)	14.50±0.511[a]	35.48±0.561[b]	15.51±0.452[c]	16.72±0.462[d]	14.41±0.528[a]

Values are expressed as Mean ± S.D of five individual experiments.
Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

Table. 14: Basal, ascorbate induced and peroxide induced lipid peroxidation in pancreas of control and experimental groups

Particulars	Control (Group I)	Diabetic control (Group II)	Diabetic+Glibenclamide treated(Group III)	Diabetic+ *P.corymbosa* treated(Group IV)	*P.corymbosa* alone treated(Group V)
Basal lipid peroxidation (nano moles of MDA formed/ g tissue)	5.37±0.449[a]	11.4±0.498[b]	7.37±0.487[c]	7.76±0.522[c]	5.56±0.422[a]
Ascorbate induced lipid peroxidation (nano moles of MDA formed/ g tissue)	6.12±0.456[a]	13.77±0.447[b]	9.15 ±0.425[c]	9.95±0.483[c]	6.7±0.474[a]
Peroxide induced lipid peroxidation (nano moles of MDA formed/ g tissue)	4.51±0.490[a]	9.97±0.478[b]	6.7±0.439[c]	7.2±0.465[c]	4.89±0.50[a]

Values are expressed as Mean ± S.D of five individual experiments. Values not sharing a common superscript letter [(a-d)] differ significantly at p>0.05 (DMRT)

CONCLUSION

In conclusion, *P.corymbosa* (Rottl.) root extract offers a promising therapeutic value in prevention of oxidative stress that developed in diabetes. These effects could be mainly attributed to its antioxidant properties as shown by significant quenching impact on the extent of lipid peroxidation along with, enhancement of antioxidant defense systems in all the tissue selected. Further studies are needed in future to determine the main active ingredient having antidiabetic and antioxidant effects.

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