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ANTIDIABETIC EFFECT OF *PEDALIUM MUREX*: EFFECT ON LIPID PEROXIDATION IN ALLOXAN INDUCED DIABETES

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ABSTRACT
The antioxidant effect of an ethanol extract of *Pedalium murex* root*(PMEt)*, was studied in rats with alloxan-induced diabetes. Oral administration of *Pedalium murex* plant extract (100& 200 mg/kg body weight) for 3 weeks resulted in a significant reduction in blood glucose and an increase in plasma insulin. The ethanol extract also resulted in decreased free radical formation in tissues (liver and kidney) studied. The decrease in thiobarbituric acid reactive substances (TBARS) and hydroperoxides (HPx) and increase in the activities of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), reduced glutathione (GSH) and glutathione-S-transferase (GST) clearly showed the antioxidant properties of *Pedalium murex* ethanol extract in addition to its antidiabetic effect. The effect of *Pedalium murex* ethanol extract at 200 mg/kg body weight was better than glibenclamide a reference drug.
KEY WORDS: Enzymic antioxidants, Insulin, Lipid peroxidation, *Pedalium murex*, Alloxan, diabetes

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INTRODUCTION

Plants which are the major source of drugs in Indian system of medicine have the advantage of little or no side effect[1,2]. *Pedalium murex* L. (pedaliaceae) commenly known as "yanai nerungi" / "peru nerungi" is an erect ' much branched, foetid smelling succulent annual herb . The leaves contain pedalitin, diosmetin, dianatin, pedalin , dianatin-7-glucoronide and diosmetin-7-glucuronide[3]. The decoction of *Pedalium murex* and glycoside obtained from it showed mild diuretic activity[4]. The extract was devoid of anthelmintic, and anti cancer activities[5].

There are several species of medicinal plant popularly used in the treatment of diabetes mellitus[6]. Still there is a need to search more effective drugs with fewer side effects for the treatment of diabetes will be useful. Oxidative stress has been shown to have role in the elevation of diabetes and related problem[7]. In diabetes, protein glycation and glucose autoxidation may generate free radicals, which in turn catalyze lipid peroxidation[8]. Alloxan produces oxygen free radical in the body, which cause pancreatic injury[9,10] and could be responsible for, increased blood glucose level[11]. The free radicals react with biomembrane causing oxidative destruction of polyunsaturated fatty acids forming cytotoxic aldehydes by a process known as lipid peroxidation (LPO).The extent of LPO was measured in terms of thiobarbituric acid reactive substances (TBARS) and lipid hydroperoxides (HPX), which are the end products of LPO. Several studies in human and animal models, using TBARS assay have shown increased LPO in membranes and lipoproteins in the diabetic state[12,13] .

HPX formed by LPO have direct toxic effects on endothelial cells and also degrade to form hydroxyl radicals (OH*)[14]. The deleterious effects of superoxide radicals (O.− 2) and OH* in oxidative stress can be counteracted by antioxidant enzymes such as SOD, CAT and glutathione peroxidase (GPx). In addition to these enzymes, glutathione-S-transferase (GST) provides glutathione (GSH) and help to neutralize toxic electrophiles. There is evidence to show the role of free radicals in diabetes and studies indicate that tissue injury in diabetes may be due to free radicals[15,16]. Diabetes is becoming pandemic and despite the recent surge in new drugs to treat and prevent the condition, its prevalence continues to soar[17].Th us, the present study was undertaken to assess the antiperoxidative and antioxitant

effect of *Pedalium murex* in alloxan induced diabetic rats.

MATERIALS AND METHODS

Animals

Adult Wistar rats weighing 160-200 g of either sex were maintained in large polypropylene cages in a well-ventilated room temperature with natural day-night cycle, and fed balanced rodent pellet diet and water *ad libitum* throughout experimental period. They were quarantined for 1 week, prior to the experiments to acclimatize them to laboratory conditions. The study protocol was approved by the IAEC (Institutional Animal Ethics Committee of CPCSEA, New Delhi, Govt. of India).

Chemicals

Alloxan monohydrate, a most widely used chemical diabetogen was procured from Loba chemie, Mumbai, India. Chemically alloxan is 2, 4, 5, 6 tetra oxo hexahydro pyrimidine. Glibenclamide an oral hypoglycemic agent used in this experiment as standard drug purchased from Aventis Pharma. Ltd., Goa, India. 5, 5-dithio bis-2-nitro benzoic acid, and reduced glutathione were procured from SISCO Research Lab, Mumbai, India. Thiobarbituric acid, nitroblue tetrazolium and nicotinamide adenine dinucleotide were purchased from Loba Chemie, Mumbai, India. All chemicals and reagents used were of analytical grade.

Experimental design

In the experiment, a total of 36 rats (30 diabetic surviving rats, 6 normal rats) were used. The rats were divided into6 groups of 6 rats each. Two doses of ethanolic extracts (100 and 200 mg/kg body weight per day) and glibenclamide (600 μg/kg) were tested. All doses were started 48 h after Alloxan injection. Blood samples were drawn at weekly intervals till the end of study (i.e., 3 weeks). At the end of 3 weeks, all the rats were killed by decapitation under pentobarbitone sodium (60 mg/kg) anesthesia. Blood was collected in tubes containing potassium oxalate and sodium fluoride solution for the estimation of blood glucose and plasma was separated for assay of insulin. Liver and kidney were dissected out, washed in ice cold saline, patted dry and weighed

Induction of Diabetes

Experimental diabetes was induced by using alloxan monohydrate[19]. Rats were fasted for 12 hr and diabetes was induced by a single intraperitonial (i.p) injection of alloxan dissolved in a freshly prepared 0.15 M sodium acetate buffer (pH 4.5), at a dose of 150mg/kg body weight.

Biochemical analysis

Determination of blood glucose and plasma insulin

Fasting blood glucose was estimated by O-toluidine method[20]. Plasma insulin was estimated using enzyme-linked immunosorbent assay kit (Boehringer Mannheim, Germany).

Estimation of LPO

LPO in tissues were estimated colorimetrically by TBARS and HPX by the method of Nehius and Samuelson[21] (1968) and Jiang *et al.,*[22] (1992), respectively. In brief, 0.1 ml of tissue homogenate (Tris-HCl buffer, pH 7.5) was treated with 2 ml of (1 : 1 : 1 ratio) TBA-TCA-HCl reagent (TBA 37%, TCA 15% and 0.25 N HCl) and placed in water bath for 15 min, cooled and centrifuged at room temperature for 10 min at 1,000 rpm. The absorbance of clear supernatant was measured against reference blank at 535 nm and expressed as mmol/100 g tissue.

HPx were expressed as mmol/100 g tissue. 0.1 ml of tissue homogenate was treated with 0.9 ml of fox reagent (88 mg butylated hydroxytoluene, 7.6 mg xylenol orange and 9.8 mg ammonium ion sulphate were added to 90 ml of methanol and 10 ml 250 mmol/l sulphuric acid) and incubated at 37◦C for 30 min. The colour developed was read colorimetrically at 560 nm.

Determination of GST

The GST activity was determined spectrophotometrically by the method of Habig *et al*., (1974). The reaction mixture (3 ml) contained 1.0 ml of 100mmol/l phosphatebuffer (pH 6.5), 0.1 ml of 30 mmol/l 1-chloro-2,4-dinitrobenzene (CDNB) and 1.7ml of double distilled water. After pre-incubating the reaction mixture at 37◦C for 5 min, the reaction was started by the addition of 0.1 ml of tissue homogenate and 0.1 ml of GSH as substrate. The absorbance was followed for 5 min at 340 nm. Reaction mixture without the enzyme was used as blank. The activity of GST was expressed as mmoles of GSH-CDNB conjugate formed/min/mg protein using an extinction coefficient of 9.6 (mmol/l)−1.

Estimation of Protein

Protein was determined by the method of Lowry *et al*., [23] (1951) using bovine serum albumin as standard, at 660 nm.

RESULTS

In all groups prior to alloxan administration, the basal levels of blood glucose of the rats were not significantly different. However, 48 h after alloxan administration, blood glucose levels were significantly higher in rats selected for the study. In contrast, non-diabetic controls remained persistently euglycaemic throughout the course of the study.

Table 1 shows the effect of treatment with extracts on blood glucose levels. In all the *PMEt*-treated groups (all doses) a significant antihyperglycaemic effect was evident from first week onwards the decrease in blood sugar being maximum on completion of the third week in the group receiving 200 mg/kg/day of ethanolic *PMEt.* On the other hand, ethanolic- 100 mg/kg/day extracts-treated groups showed an antihyperglycaemic effect much later (i.e. on completion of the third week) in groups receiving 200 mg/kg per day. On the basis of these studies, doses of 100,200 mg/kg per day of ethanolic, *PMEt* was selected for further evaluation. TBARS and HPX (Table 2) from liver and kidney homogenate were significantly decreased and plasma insulin was significantly increased with *PMEt* treatment whereas diabetic control rats showed significantly increased levels of LPO products and decreased level of plasma insulin.

For studying the effect of *PMEt* on antioxidant status, the activities of SOD, CAT, GPx, GST and GSH were measured (Table3). They presented significant increases in *PMEt* treatment when compared with diabetic control rats. The extent of increase was higher in groups treated with aqueous *PMEt* than glibenclamide treated groups. Treatment with *PMEt* to normal animals did not show any significant Alterations.

DISCUSSION

Type II diabetes is one of the major health problems throughout the world especially in adults of age above 35 years in both sexes[24].In spite of the presence of number of synthetic oral antidiabetic drugs in the market, researchers are now diverted their attention to different herbs and medicinal plants in order to find out new active principle with less side effects and better antidiabetic activity[25]. Therefore, *Pedalium murex* Linn was selected for the present study in order to provide some help in patronizing indigenous drugs. In this context a number of other plants have also been reported to have antihyperglycaemic and insulin-release stimulatory effects[26,27].

The involvement of free radicals in diabetes and the role of these toxic species in LPO and the antioxidant defense system have been studied. The results show increased LPO in the tissues of diabetic group. The increase in oxygen free radicals in diabetes could be due to increase in blood glucose levels, which upon auto oxidation generate free radicals. Alloxan, a beta cytotxoin, destroys β cell by liberating oxygen free radicals, which cause lipid peroxide mediated pancreatic injury.Thus lipid peroxidation is one of the characteristic features of chronic diabetes[28]. There was an increased LPO in liver and kidney of diabetic rats[29,30].

Under in vivo conditions, GSH acts as an antioxidant and its decrease was Reported in diabetes mellitus[31].We have observed a significant decrease in GSH levels in liver and kidney during diabetes. The decrease in GSH levels represents increased utilization due to oxidative stress[32]. The depletion of GSH content may also lower the GST activity as GSH is required as a substrate for GST activity[33]. Depression in GPx activity was also observed in liver and kidney during diabetes. GPx has been shown to be an important adaptive response to condition of increased peroxidative stress[34].The increased GSH content in the liver and kidney of the rats treated with *PMEt* and glibenclamide may be one factor responsible for inhibition of LPO[35.]

SOD and CAT are the two major scavenging enzymes that remove toxic free radicals in vivo. Previous studies have reported that the activity of SOD is low in diabetes mellitus [35]. Reduced activities of SOD and CAT in liver and kidney have been observed during diabetes and this may result in a number of deleterious effects due to the accumulation of O.− 2 and H_2O_2[36]. Administration of *PMEt* having anthelamintic, hypoglycaemic,and anti cancer activity has been reported [5]. Any compound, natural or synthetic, with antioxidant properties, might contribute towards the partial or total alleviation of this damage. Therefore, removing O.− 2 and OH∗ is probably one of the most effective defenses against diseases [37].The result of the SOD and CAT activity suggest that *PMEt* contains a free radical scavenging activity,(Table 3,4) which could exert a beneficial action against pathological alterations caused by the presence of O.− 2, H2O2 and OH∗. This action could involve mechanisms related to Scavenging activity.

In conclusion, the present investigation show that *PMEt* possesses an antidiabetic effect in addition to antioxidant activity, which may be attributed to its protective action on LPO and to the enhancing effect on cellular antioxidant defense contributing to the protection against oxidative damage in alloxan diabetes.

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Table 1: Effect of 21 – days treatment with ethanol extract of the *Pedalium murex* root and root derived callus on glucose in normal and experimental rats

Groups	Day 0 (mg/dl)	2 nd day (mg/dl)	1 week (mg/dl)	2 week (mg/dl)	3 week (mg/dl)
Normal	86.83±5.67	89.83^{e}± 5.38	89.67^{d}± 5.57	87.33^{e}± 8.64	86.50^{e}± 6.38
Normal+root (100mg/kg)	89.33±8.57	86.50^{e} ±5.36	87^{d}± 5.80	88.67^{e}± 6.65	87.33^{e}± 7.39
Normal +root (200mg/kg)	94.33±5.47	88.67^{e} ± 6.5	86 ^{d}± 7.16	88.33^{e}± 6.71	91.33^{e} ± 5.32
Diabetic control (Alloxan 150mg/kg	85.67±7.97	275 ^{a}± 17.8	276.67^{a}±10.80	292.33^{a}±14.46	298.5^{a} ±14.34
Diabetic +root (100mg/kg)	87.67±6.35	269.83^{ab}±14.78	193.83^{bc} ±11.51	139.5^{c} ±6.25	108.5^{b} ±6.06
Diabetic + root (200mg/kg)	87.67±4.63	279.67^{a}± 19.9	188.17^{c}±14.86	127.83^{d}±6.27	87.83^{c} ± 7.05
Diabetic +glibenglamide 600µg/kg	89.83±6.71	256.67^{b}±11.66	205.83^{b}±15.21	187^{b}±5.76	112.67^{b}±7.55

Values are mean ± SD for six animals in each group .Values not sharing a common superscript differ significantly at p< 0.05.DMRT, Duncan's multiple range tests

Table 2: Changes in activities of superoxide dismutase (SOD), glutathione –S-transference (GST) in liver and kidney of normal control and experimental animals

GROUPS	GST (µ MOLES OF CDNB- GSH conjugate formed /min/mg protein)		SOD (NBT reduction /min/mg/protein)	
	Liver	kidney	Liver	kidney
CONTROL	6.14±0.35^{a}	5.14±0.31^{ab}	6.39±0.45^{a}	13.50±1.03^{a}
Control + root (100mg/kg)	6.13±0.52^{a}	5.36±0.51 ^{a}	6.37±0.50 ^{a}	14.37± 1.26^{a}
Control + root (200mg/kg)	6.05≠0.72 ^{a}	5.41≠0.53^{a}	6.47≠0.71^{a}	14.69≠1.04^{a}
Diabetic control	3.61≠0.35 ^{c}	2.39≠0.45^{d}	3.47≠0.72^{c}	9≠1.15^{c}
Diabetic+ root (100mg/kg)	5.6≠0.53^{a}	4.3≠0.34^{c}	5.42≠0.51^{b}	12.38≠1.43^{ab}
Diabetic+ root (200mg/kg)	6.09≠0.42^{a}	4.75±0.25^{b}	5.63±0.99^{ab}	13.58±1.04^{ab}
Diabetic+ root (600mg/kg)	4.58±0.38^{b}	3.82±0.12^{c}	5.37±0.58^{b}	12.21±0.94^{b}

Values are mean ± SD for six animals in each group .Values not sharing a common superscript differ significantly at p< 0.05.DMRT, Duncan's multiple range tests.

Table 3: Changes in levels of tissue thiobarbituric acid reactive substances (TBARS)and hydroperoxides (HPX)in normal and experimental animals

GROUPS	HPX m moles/100wet tissues		TBARS m moles /100g wet tissue	
	Liver	kidney	Liver	kidney
CONTROL	78.50±4.76 b	58.67±0.31^{ab}	0.79±0.081c	1.75±0.817^{cd}
Control + root (100mg/kg)	81.67±7.44^{b}	54.5± 4.5 ^{c}	0.77±0.098 ^{c}	1.69± 0.983^{d}
Control + root (200mg/kg)	82.17≠7.35 ^{b}	53.17≠5.67^{c}	0.78≠0.082^{c}	1.73≠ 0.109^{d}
Diabetic control	96≠4.73 ^{a}	74.17≠7.99^{a}	1.85≠0.115^{a}	3.78≠0.172^{a}
Diabetic+ root (100mg/kg)	75.83≠8.01^{b}	62.50≠3.89^{ab}	1.48≠0.117^{c}	1.67≠0.137^{c}
Diabetic+ root (200mg/kg)	75.57 ≠6.95^{b}	62.50±6.38^{bc}	1.26±0.089^{d}	1.74±0.075^{ab}
Diabetic+ glibenclamide (600mg/kg)	83.16±4.88^{b}	65±3.48^{b}	1.49±0.089^{b}	2.22±0.089^{b}

Values are mean ≠ SD for six animals in each group. Values not sharing a common superscript differ significantly at $p < 0.05$ DMRT, Duncan's multiple range tests

Table 4: Changes in activities of catalase in liver and kidney of normal control and experimental animals

Groups	Catalase (µmoles of H_2O_2 consumed/min/mg protein)	
	Liver	Kidney
Control	77.33± 8.16^{a}	33.67±6.15 ^{a}
Control + root (100mg/kg)	72.67±6.02 ^{a}	32.5±1.52^{a}
Control + root (200mg/kg)	73.67±5.16^{a}	35.67±1.63^{a}
Diabetic control	46.33±4.23^{c}	16.73±0.98^{d}
Diabetic+ root (100mg/kg)	61.5±6.16^{b}	22.5±3.78^{c}
Diabetic+root (200mg/kg)	61.83±4.99^{b}	26.67±2.16 ^{b}
Diabetic+ glibenclamide (600mg/kg)	51.33±5.28 ^{c}	22.67±2.07^{c}

Values are mean ≠ SD for six animals in each group. Values not sharing a common superscript differ significantly at $p < 0.05$ DMRT, Duncan's multiple range tests

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