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A Study on Potential Hypoglycemic and Hypolipidemic Effects of *Lepidium Sativum* (Garden Cress) in Alloxan Induced Diabetic Rats

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ABSTRACT

Hypoglycemic and hypolipidemic effects of *Lepidium sativum (Garden cress)* seed powder (3.0g/kg) was evaluated on diabetes and oxidative stress built up in alloxan induced diabetic male Wistar rats. Experimental animals were divided into six groups comprising of six animals each. Animals were supplemented with isoenergic diets (~3600) for a period of 45 days. Hyperlipidemia was induced by feeding high fat high cholesterol diet and diabetes was induced by single injection of alloxan (150mg/kg body weight) intraperitoneally. High fat feeding and alloxan induced diabetes resulted in marked alterations in blood glucose, lipid profile and antioxidant status in blood serum and hepatic tissue of albino rats. Garden cress treated rats showed a significant decrease ($p \leq 0.05$) in fasting blood glucose levels (FBG); glycosylated haemoglobin (Hb A_{1C} %); lipid profile (total cholesterol (TC), triglycerides (TG) and lipoprotein fractions (Low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C)) with a significant increase in high density lipoprotein cholesterol levels (HDL-C). A significant increase ($p \leq 0.05$) in thiobarbituric acid reactive substances (TBARS) levels with concomitant decrease in reduced glutathione (GSH) and antioxidant enzyme activity was also observed in diabetic control and HFHC diet fed experimental rats. *Lepidium sativum* neutralized the effect and restored the levels. *Lepidium sativum* seed powder thus lends credence for the prevention and management of diabetes mellitus and related complications.

Key words: Lepidium Sativum, Garden Cress, Alloxan, Antihyperglycaemic, Antihyperlipidemic, Oxidative stress, Blood glucose, Lipid peroxidation

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INTRODUCTION:

Diabetes mellitus has been a ubiquitous problem affecting human societies at all stages of development. It has become a major cause of morbidity and mortality in the present times. It is a clinical syndrome involving heterogeneous group of disorders, characterized by abnormally high blood glucose concentration, due to relative or absolute deficiency of insulin or resistance of the body cells to the action of insulin, often associated with derangement in carbohydrate, protein and lipid metabolism. The primary defect in fuel metabolism results in widespread, multi-organ complications that ultimately encompass virtually every system of the body and every specialty of medicine.

According to World Health Organization, approximately more than 100 million peoples were reported to have diabetes mellitus worldwide[1]. India is one of the leading countries with high number of people with diabetes mellitus and it is estimated that around 57 million peoples will be suffering from diabetes mellitus by the year 2025[2]. The prevalence of the disease has become so high to get for this country the sobriquet “Diabetes capital of the world”.

The relationship between diabetes and hyperlipidemia is a well recognized phenomenon. Hypercholesterolemia is common in diabetes, contributing to the high prevalence of accelerated atherosclerosis and coronary heart disease[3]. Insulin has important regulatory effects on lipid as well as on glucose metabolism and therefore, it is not unexpected that diabetes mellitus is associated with significant abnormalities in lipoprotein metabolism. Hyperlipidemia in diabetics results from altered lipoprotein metabolism due to increased production, decreased clearance and/or changes in lipoprotein composition caused by the changes in insulin, glucose and free fatty acid levels associated with diabetes mellitus[4]. Increased oxidized LDL or susceptibility to oxidation has also been shown in diabetes[8].

Increased oxidative stress as measured by indices of lipid peroxidation and protein oxidation has been shown to be increased in both insulin dependent and non- insulin dependent diabetes mellitus [5, 6, 7] even in patients without complications. The mechanism behind the apparent increased oxidative stress in diabetes is not clear. Accumulating evidence points to the number of inter-related mechanisms[9] increasing production of free radicals or decreasing antioxidant status. Under normal physiological conditions, enzymatic antioxidants form the first line of antioxidant defense mechanism to protect the organism from reactive oxygen species mediated oxidative damage[10]. However, in diabetes, hyperglycemia depresses the natural defense system and increased generation of free radicals leads to disruption of cellular functions and oxidative damage to membranes and may enhance susceptibility to lipid peroxidation[11].

Medicinal plants have been used as valuable therapeutic agents in traditional system. Before the advent of insulin and oral hypoglycemic drugs, the major form of treatment involved the use of herbs. Unfortunately, after the introduction of sulfonylurea and metformin about 50 years back no major lead has been obtained in this direction of finding a proper drug for diabetes but from the last two decades there has been a new trend in the preparation of different folk plant extracts and their acceptance by the users for diabetes especially in third world countries.

Lipidium Sativum (Garden cress) belonging to cruciferae family, widely grown in India, Europe and US is an underutilized crop. It has been considered as an important medicinal plant since the Vedic era. In Ayurveda, it is described as hot, bitter, galactogogue and aphrodisiac and claimed to destroy Vata (air) and Kapha (phlegm). In Unani system of medicine, seed powders and leaves of this plant have been reported to possess diuretics, aperients and aphrodisiac properties and are recommended in inflammation, bronchitis, rheumatism and muscular pain. It is also reported to be useful in the treatment of asthma, cough and bleeding piles[12]. The plant is also reported to possess antihemogglutinating, hypoglycemic, antihypertensive, diuretic, fracture healing properties and significant bronchodilatory activities[13,14,15,16]. *Lepidium sativum* seed powders are used in the management of diabetes mellitus with an average citation of 64 among 500 interviewed persons[17]. By administrating 15 gm seed powders per day the antidiabetic activity of *Lepidium Sativum* were tested on NIDDM as well as normal healthy subjects. In the treatment regimen spanning 21 days, hypoglycemic activity was reported[18]. Thus the present study was aimed at appraising the antiglycaemic; antihyperlipidemic and oxidative stress reducing potential of *L.sativum.*

MATERIALS AND METHODS

Lepidium Sativum seeds were ground to a fine powder and stored in air tight containers. Healthy albino wistar male rats 7 to 8 weeks old, weighing 150-200g were procured from the small animal house of Chaudhary Charan Singh Haryana Agriculture University Hissar (CCSHAU), India. Animals were given the standard pellet diet (Hindustan Liver Ltd., India) and water ad libitum during acclimatization period of 7 days. They were housed individually in the polypropylene cages with sterilized wood chip bedding in a specific pathogen free animal house room under the constant environmental condition with 12 hour light and dark cycle, 22±1 ^{0}C temperature and 50 ±10% relative humidity. The study protocol was approved by Institutional

Animal Ethics Committee (IAEC) of the University constituted as per the directions of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA).

Experimental Design

Animals were divided into six groups of six animals each. The groups were fed on the following diets for a period of 45 days: Group-A, Isoenergic normal fat diet; Group-B, High fat- high cholesterol diet (HFHC); Group-C, HFHC +Garden Cress seed powder (HFHC-GC (3.0g/kg)); Group-D, Isoenergic normal fat diet + Alloxan (150mg/kg bw.ip) (Diabetic Control); Group-E, HFHC + Alloxan (HFHC-Alloxan); Group-F, HFHC + Alloxan +Garden Cress seed powder (HFHC-Alloxan+ GC (3.0g/kg)). The diets were isoenergic (~3600C) and the composition of the various diets is given in Table 1. Oxidative stress was induced after three weeks of feeding experimental diets by giving intraperitoneal injection of ammonium acetate at a dose level of 125mg/kg body weight. The gain in body weight was recorded twice a week and food consumption was monitored daily. After the completion of feeding schedule, food was withheld and animals were provided only with water, *adlibitum* for overnight.

Table 1 -Composition of Diets (g/kg)

Component	**Isoenergic normal fat**	**High fat ,high cholesterol (HFHC)**	**HFHC + Garden Cress**	**Diabetic Control**	**Diabetic + HFHC**	**Diabetic+HFHC +Garden Cress**
	A	B	C	D	E	F
Casein (Vitamin & fat free)	200	200	200	200	200	200
Mineral mix 1	90	90	90	90	90	90
Vitamin mix 2	10	10	10	10	10	10
Cellulose	50	173.7	173.7	173.7	173.7	173.7
Sucrose	--	20	20	20	20	20
Corn starch	600	350	350	350	350	350
Oil	50	150	150	150	150	150
Coconut oil	50	122	122	122	122	122
Refined oil	--	28	28	28	28	28
Cholesterol	--	5	5	5	5	5
Bile salts	--	1.25	1.25	1.25	1.25	1.25
Garden Cress	--	--	3.0	--	--	3.0

1. Mineral mixture provided the following g/ kg/mix : Calcium carbonate (anhydrous), 357; Potassium phosphate monobasic,196; Potassium citrate,70.78; Sodium chloride,74; Potassium sulfate, 46.60; Magnesium oxide,24; Ferric citrate,6.06; Zinc carbonate,1.65; Managanous carbonate,0.63; Cupric carbonate, 0.30; Potassium iodate,0.01; Sodium selenate (anhydrous), 0.01; Ammonium paramolybdate.0.007; Sodium meta silicate,1.45; Chromium potassium sulfate,0.275; Lithium chloride,0.01; Boric acid,0.08; Sodium fluoride0.06; Nickel carbonate,0.03; Ammonium vandate,0.006; Powdered sucrose, 221.02.

2. Vitamin mixture provided the following (per Kg of diet): tocopherol, 50mg; L- ascorbic acid 0.05g; choline chloride, 0.75g; D- calcium pantothenate, 30 mg; inositol, 50 mg; menadione, 22 mg; niacin, 45 mg; p- amino benzoic acid,50 mg; pyridoxine. HCl, 10 mg; riboflavin, 10mg; thiamine. HCl, 10 mg; ritinyl acetate, 9 mg; ergocalciferol, 0.0025 mg; biotin, 0.2 mg; folic acid, 0.9 mg; vitamin B12, 0.013mg.

Induction of diabetes mellitus

Rats were made diabetic by a single intraperitoneal injection of alloxan monohydrate (150mg/kg body weight) [19].Alloxan was first weighed individually for each animal according to the body weight and then solubilized with 0.2ml saline (154mM NaCl) just prior to injection. Three days after alloxan injections, rats with plasma glucose levels of 140mg/dl were included in the study. Dietary treatment with garden cress seed powder was started 48 h after alloxan injection.

Biochemical Analysis

The blood was withdrawn from retrorbital plexus under mild ether anesthesia with heparinized capillary tubes into two pre-chilled vials, with one containing EDTA (1mg/ml) and the other as such without any added material. The blood samples were mixed thoroughly to prevent clotting. Thereafter, the animals were sacrificed by cervical decapitation. The liver was excised, washed with ice cold isotonic saline and weighed. A small part of the hepatic tissue was minced and used for enzyme activity assay and other biochemical evaluation. Samples were stored in vials at -25 ^{0}C until further biochemical analysis. All samples were coded prior to analysis. Biochemical analysis involved evaluation of blood glucose, lipid profile and enzyme assay of blood and hepatic tissue. The levels of haemoglobin and glycosylated haemoglobin were estimated using the methods of Drabkin and Austin[20], and Nayak and Pattabiraman[21] respectively. Plasma insulin level was assayed by enzyme-linked immuno sorbent assay kit (ELISA, Boerhringer Mannheim, Germany).

Serum was separated and analyzed for fasting blood glucose, total cholesterol, triglycerides, HDL-C using span diagnostic kit. LDL-C and VLDL-C were calculated by Friedewald's equation[22].

$$\text{VLDL-C} = \text{TG}/5$$

$$\text{LDL-C} = \text{TC} - (\text{HDL-C} + \text{VLDL-C})$$

For enzyme activity assay, 0.8-1.0g of hepatic tissue was minced and homogenized in 10 times its volume of 0.2M/L tris HCl (pH=8.0) containing 0.5M/L $CaCl_2$ using Potter Elevehjem apparatus at 0-4^{0}C using motor driven Teflon pestle rotated at 3000rpm. The homogenate was centrifuged at 10000x g for 30 minutes at 4^{0}C and 3/4th of the volume was carefully drawn using Pasteur's pipette. Enzyme assay involved, serum lipid peroxidation[23] and hepatic TBARS[24], red cell[25] and liver reduced glutathione (GSH)[26] and hepatic antioxidant enzymes; glutathione peroxidase (GSHPx)[27], catalase (CAT)[28] and superoxide dismutase (SOD)[29], Serum transaminases (SGPT and SGOT)[30] were evaluated.

Statistical Analysis

The data are expressed as Mean±SD. Statistical comparisons were performed by one way analysis of variance (ANOVA). The values with $p \leq 0.05$ were considered as statistically significant.

RESULTS AND DISCUSSION

In the present study hypoglycemic, hypolipidemic and antioxidative efficacy of garden cress seed powder was evaluated in alloxan induced diabetic male Wistar rats. Alloxan has been reported to cause significant reduction of insulin producing β- cells of islets of langerhans thus inducing hyperglycemia. The increased blood glucose level in diabetic rats as compared to normal control group may be due to increased glycogenolysis or gluconeogenesis. Satyanarayana *et al* [31], reported that alloxan at a dose of 140-200mg/kg body weight is non lethal. On the basis of the previous studies alloxan was administered at a dose of 150mg/kg body weight. This resulted in diminished secretion of insulin that was not sufficient to regulate the blood glucose level leading to hyperglycemia. Table 2 depicts a significant increase in fasting blood glucose level in diabetic control rats (225.0±2.98mg/dl) as compared to normal control reared on isoenergic normal fat diet (62.4±1.13mg/dl). However, supplementation of garden cress seed powder to experimental animals for 45 days resulted in decrease in fasting blood glucose concentration in group C (12.0±1.07µU/ml) and group F (10.5±0.47 µU/ml). On the contrary mean plasma insulin level significantly decreased in diabetic control rats (8.1±0.50) as compared to normal control ones (14.1±0.55).

Table: 2 Effect of different dietary treatments on blood glucose and insulin of albino rats

Dietary Groups	Blood Glucose (mg/dl)	Plasma Insulin (µU/ml)
Group-A	62.4±1.13	14.1±0.55
Group-B	128.7±1.85*	14.2±0.56**
Group-C	81.2±1.70*	12.0±1.07*
Group-D	225.0±2.98*	8.1±0.50*
Group-E	234.4±3.84*	9.2±0.36*
Group-F	111.7±1.77*	10.5±0.47*

Values are Mean ± SD of 6 rats in each group, *P≤0.05 Significant, **P>0.05 Non Significant
Group-A- Normal Control (Isoenergic normal fat), Group-B- Experimental (HFHC)
Group-C- Exp garden cress (HFHC+GC) , Group-D- Diabetic Control (Isoenergic normal fat+Alloxan) , Group-E- Diabetic Exp (HFHC+Alloxan), Group-F- Diabetic Exp Garden cress (HFHC+Alloxan+GC)

Concomitant feeding of garden cress tended to neutralize the effect. The increased mean insulin levels could be due to the stimulation of insulin secretion from remnant pancreatic β-cells which in-turn enhances glucose utilization by peripheral tissues. The results are in accordance with the previous study showing insulin release due to stimulatory effects[32].

In diabetes mellitus, high blood glucose reacts with hemoglobin which increases glycosylated hemoglobin, thereby decreasing hemoglobin levels. Glycosylated haemoglobin (HbA_{1c}) is a good measure to indicate the average blood glucose concentration over the preceding weeks while a single glucose determination gives a value which is true only at the time the blood sample is drawn[33]. HbA_{1c} is formed progressively and is irreversible over a period of time and is stable till the life of the RBC and is unaffected by diet, insulin or exercise on the day of testing[34]. Mean glycosylated haemoglobin significantly increased in alloxan induced diabetic rats (6.9±0.27%). Feeding of diets enriched with garden cress seed powder showed a marked decrease in mean glycosylated haemoglobin levels in group C (2.3±0.19%) and group F (4.6±0.22%). On the other hand, mean haemoglobin levels significantly decreased in diabetic controls (8.3±0.40 g/dl) as compared to normal controls (13.5±0.35 g/dl). A significant change was observed in animals supplemented with garden cress as shown in Table 3. The decreased level of total haemoglobin in diabetic rats is mainly due to increased formation of HbA_{1c}. A study has demonstrated that HbA_{1c} was found to increase in patients with diabetes mellitus and the amount of increase is directly proportional to fasting blood glucose[35].The increased HbA_{1c} levels in the diabetic control group indicate that erythrocytes are more prone to oxidative stress in diabetes. The longer the exposure of erythrocytes to hyperglycemia, the shorter is its life span. Administration of *L. sativum* reduced the glycosylation of haemoglobin by virtue of its normoglycaemic activity and thus increasing the levels of glycosylated haemoglobin indicating decreased glycation of protein. Therefore, prolonged intake of garden cress seed powder may further reduce HbA_{1c} levels and probably help in achieving better glycaemic control.

Table: 3 Effect of different dietary treatments on haemoglobin and glycosylated haemoglobin of albino rats

Dietary Groups	Glycosylated Haemoglobin ($Hb\ A_{1C}$ %)	Total Haemoglobin (g/dl)
Group-A	2.7±0.21	13.5±0.35
Group-B	3.0±0.24*	13.5± 0.48**
Group-C	2.3±0.19*	11.2±0.79*
Group-D	6.9±0.27*	8.3±0.40*
Group-E	6.0±0.49*	8.6±0.35*
Group-F	4.6±0.22*	9.3±0.58*

Values are Mean ± SD of 6 rats in each group, *P≤0.05 Significant, **P>0.05 Non Significant

Hyperlipidemia is a known complication of diabetes mellitus and coexists with hyperglycemia and is characterized by increased levels of cholesterol, triglycerides and marked changes in lipoprotein fractions[36]. A variety of derangements in metabolic and regulatory mechanisms due to insulin deficiency is responsible for the observed accumulation of lipids[37]. Impairment of insulin secretion results in enhanced metabolism of lipids from the adipose tissue to plasma. Furthermore it has also been shown that diabetic rats on treatment with insulin show normal lipids[38]. Control of hyperlipidemia is a prerequisite for the prevention of diabetic microangiopathy (retinopathy, nephropathy and neuropathy) and macroangiopathy (ischemic heart disease), cerebral vascular disease (CVD) and arteriosclerosis obliterans in diabetes[39].

In the present study the lipid abnormalities developed in alloxan induced diabetic animals were effectively countered by feeding garden cress. Table 4 elicits that total cholesterol and triglycerides were brought down significantly by garden cress feeding in experimental (118.3±1.06mg/dl); (134.1±2.26mg/dl) and diabetic rats (119.2±2.07mg/dl); (152.5±1.21mg/dl) respectively. The effect could be partly due to the control of hyperglycemia. Elevated LDL-C (58.8±4.40mg/dl); (9.0±7.457mg/dl) and VLDL-C (35.8±0.50); (48.7±3.44) and decreased HDL-C (34.7±6.58); (31.2± 3.58) levels in experimental and diabetic rats were markedly altered by garden cress supplementation. The precise mechanism underlying this effect probably appears to be anti-glycaemic effect which inhibits absorption and enhances excretion of lipids. Elevation in serum lipid profile (TC and TG) and lipoprotein fractions (LDL-C and VLDL-C) with concomitant decrease in HDL-C in alloxan induced diabetic animals is in agreement with previous studies regarding alteration of these parameters under diabetic condition32.

Table: 4 Effect of different dietary treatments on serum lipid profile of albino rats

Dietary Groups	TC (mg/dl)	TG (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)	HDL-C (mg/dl)
Group-A	81.6±1.61	77.8±1.79	14.1±2.38	15.6±0.96	51.3±5.54
Group-B	129.1±0.98*	178.8±2.76*	58.8±4.40*	35.8±0.50*	34.7±6.58*
Group-C	118.3±1.06*	134.1±2.26*	34.3±5.76*	26.8±3.40*	57.6±5.55*
Group-D	159.9±1.43*	244.0±1.83*	79.0±7.45*	48.7±3.44*	31.2± 3.58*
Group-E	171.0±1.87*	253.4±1.51*	91.2±8.30*	50.6± 3.17*	29.4±6.43*
Group-F	119.2±2.07*	152.5±1.21*	27.5±2.57*	30.5±2.86*	61.3±5.21*

Values are Mean ± SD of 6 rats in each group, *P≤0.05 Significant

Lipid peroxidation is one of the characteristic features in diabetes mellitus and red blood cells are more susceptible to lipid peroxidation in diabetes. Measurement of serum and hepatic thiobarbituric acid reactive substances (TBARS) is used as an index of lipid peroxidation and to assess the extent of tissue damage[40] as a result of high oxidative stress built ups. Serum and hepatic lipid peroxidation activity significantly increased in HFHC diet fed animals (41.7±2.37); (0.87±0.043), diabetic control (38.4±2.57); (0.83±0.028) and HFHC diet fed diabetic rats (48.4±2.00); (0.88±0.051) respectively. Inclusion of garden cress seed powder to HFHC diets reversed the trend indicating that seeds possesses anti-peroxidative activity (Table 5and 6). The results are consistent with other studies reporting an increase in TBARS levels in plasma, liver and kidney in experimentally induced diabetes mellitus[41, 42].

Glutathione; a tripeptide is an intracellular antioxidant and protects the cellular system from adverse effects of lipid peroxidation. Mean red cell reduced glutathione levels decreased significantly in serum and hepatic tissues (p≤0.05) in HFHC diet fed animals; diabetic control and HFHC diet fed diabetic rats as compared to the normal control fed isoenergic normal fat diet. Incorporating garden cress seed powder to HFHC diets reversed the effect and significantly increased (p≤0.05) the levels (Table 5 and 6). Lower levels of GSH in serum and hepatic tissue indicate increased utilization due to increased oxidative stress.

Table: 5 Effect of different dietary treatments on blood oxidative stress and serum transaminases status of albino rats

Dietary Groups	Serum TBARS (nM of TBARS/100ml)	Red Cell Reduced Glutathione (mM/100ml)	SGPT (IU/L)	SGOT (IU/L)
Group-A	23.0±1.90	45.6±3.56	10.5±0.90	7.4±0.52
Group-B	41.7±2.37*	37.8±2.48*	12.3±0.69*	7.1±0.24**
Group-C	33.8±1.67*	42.8±2.41*	11.1±1.31*	6.8±0.38*
Group-D	38.4±2.57*	33.7±1.67*	17.7±1.01*	13.5±1.15*
Group-E	48.4±2.00*	29.7±4.37*	19.1±1.06*	15.4±1.54*
Group-F	37.9±1.96*	40.3±3.84*	11.8±1.59*	9.6±0.91*

Values are Mean ± SD of 6 rats in each group, *P≤0.05 Significant, **P>0.05 Non significant

Table: 6 Effect of different dietary treatments on hepatic oxidative stress status of albino rats

Dietary Groups	Hepatic TBARS	Hepatic GSH
Group-A	0.76±0.048*	341.4±4.75*
Group-B	0.87±0.043*	275.0±5.03
Group-C	0.79±0.029*	332.6±6.19*
Group-D	0.83±0.028*	307.7±6.48*
Group-E	0.88±0.051*	314.4±4.74*
Group-F	0.79±0.053*	329.4±6.05*

Values are Mean ± SD of 6 rats in each group, *P≤0.05 Significant

Activities of enzymatic antioxidants viz. GSHPx, SOD, CAT and decreased in the liver of experimental animals fed HFHC diet; diabetic control and diabetic experimental animals. Activities of these enzymes significantly (p≤0.05) increased in garden cress treated animals as shown in Table 7. Previous studies reported that hyperglycemia induces polyol pathway that consumes NADPH which is necessary for GSHPx redox cycle[43]. GSHPx is a selenium containing antioxidant which plays a crucial role in scavenging free radicals by using GSH as substrate. Decreased GSHPx activity in HFHC diet fed animals and diabetic rats is probably due to insufficiency of GSH. SOD protects from oxygen free radicals by catalyzing the removal of superoxide radical, which damages the membrane and biological structures. Catalase is responsible for the detoxification of H_2O_2[44]. Decreased SOD and Catalase activity in experimental, diabetic control and diabetic experimental rats may result in detrimental effects due to accumulation of superoxide radical and H_2O_2.

Table: 7 Effect of different dietary treatments on hepatic antioxidant enzyme activities of albino rats

Dietary Groups	Glutathione Peroxidase (g of GSH utilized/min/mg protein)	Catalase (valuesx 10^{-3} units/mg protein)	Superoxide dismutase (units/mg protein)
Group-A	8.6±0.24	56.9±1.54	2.8±0.24
Group-B	6.3±0.40*	46.8±1.39*	1.7±0.40*
Group-C	7.4±0.62*	52.8±2.21*	3.0±0.28*
Group-D	5.4±0.27*	41.5±3.09*	1.5±0.37*
Group-E	4.7±0.24*	37.4±2.64*	1.4±0.24*
Group-F	6.8±0.55*	45.6±0.96*	2.9±0.36*

Values are Mean ± SD of 6 rats in each group, *$P \leq 0.05$ Significant

The mean values for transaminases activity are given in Table 5 as biochemical parameters of damage in liver function. HFHC diet fed animals, diabetic control and diabetic experimental animals showed a significant increase ($p \leq 0.05$) in the mean SGPT activity as compared to isoenergic normal fat fed group probably due to enhanced gluconeogenesis. Garden cress supplementation lowered the mean SGPT activity. There was a decreasing trend, though insignificant ($p \geq 0.05$), in mean SGOT activity of animals fed HFHC diets as compared to their control counter parts fed isoenergic normal diet. However, diabetic control and diabetic experimental animals showed a significant increase in SGOT activity as compared to normal controls. Feeding of garden cress enriched diets led to marked improvement in its activity.

CONCLUSIONS

The present study showed that the *Lepidium Sativum* efficiently regulated blood glucose and ameliorated lipid abnormalities associated with diabetes in alloxan induced diabetic rats possibly by virtue of various essential antioxidant, antidiabetic compounds and phytonutrients. Garden cress can thus contribute towards prevention and management of diabetes mellitus and its associated complications.

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