

Antidiabetic and antioxidant potential of the fruits of *Callistemon lanceolatus* DC

Das Sanjita^{1*}, Singh Uttam²

Department of Pharmaceutical Technology, Noida Institute of Engineering and Technology, Greater Noida (U.P), India.

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ABSTRACT

Aims: Literature survey shows that the leaves of *Callistemon lanceolatus* DC (Family: Myrtaceae) are having scientific data on antidiabetic activity, whereas for its fruits are also having the same chemical constituents. This initiated the present research work to evaluate of the antidiabetic and antioxidant properties of the fruits of *Callistemon lanceolatus*.

Materials and methods: The hypoglycemic activity of methanol extract of *Callistemon lanceolatus* fruits in alloxan induced diabetic in Wistar rats by administrating graded oral doses (200 and 400 mg/kg b.wt.) for 21 days. This study also includes biochemical examination of blood serum along with histopathological studies of pancreas and liver. The methanol extract showed strong antioxidant activity with DPPH radical scavenging experiment.

Results: Daily oral treatment with *Callistemon lanceolatus* methanol fruits extract and glibenclamide (10 mg/kg) for 3 weeks resulted in significantly reduction in blood glucose, serum cholesterol ($p < 0.01$), triglycerides, AST and ALT levels whereas HDL-cholesterol level was found to be improved ($p < 0.01$) as compared to diabetic control group. The pancreas and liver histology indicates significant recovery with the extract administration.

Conclusion: Present results indicate that *Callistemon lanceolatus* methanol fruits extract have prominent antidiabetic effect at dose 400 mg/kg in experimental diabetes, which may be due to its antioxidant property.

Key words: *Callistemon lanceolatus*, antidiabetic, antioxidant activity, hematological parameters, histopathology.

INTRODUCTION

Most of the drugs used in primitive medicine were obtained from plants and are the earliest and principal natural source of medicines. Plants are the main resource for new medicinal entities. There is no doubt that plants are a reservoir of potentially useful chemical compounds which serve as drugs, are provided newer leads and clues for modern design by synthesis (Evans, 2002). *Callistemon lanceolatus* DC (Family: Myrtaceae) commonly known as bottle brush, is frequently cultivated throughout India in gardens as ornamental plant. Hummingbirds love the flowers, and the plant is harder than most Bottlebrushes. Aqueous extracts of the leaves and flowers have antifungal and antibacterial activity. The extract also shows cholinesterase activity. The essential oils from leaves possess hepatoprotective, antimicrobial, fungitoxic, antinociceptive, anti-inflammatory and free radical scavenging activities (Rao et al., 2012; Jain et al., 2007; Kim et al., 2009; Kumar et al., 2011). New flavones of its leaves have also been reported to have antidiabetic activity (Kumar et al., 2011; Nazreen et al., 2012). Keeping in view of the growing concern towards the treatment of diabetes, the present research work is aimed at the evaluation of the antidiabetic and antioxidant properties of the fruits of *Callistemon lanceolatus*.

MATERIALS AND METHODS

Experimental animals:

The Wistar strains of albino rats, weighing about 150-250 g were used in the study. Animals were maintained under standard environmental conditions, i.e. ambient temperature of $22 \pm 2^\circ\text{C}$ and at 45-55% relative humidity, 12 h each of dark and light cycle and fed with a standard pellet rats diet ad libitum. Water was supplied ad libitum. All the experiments were conducted in strict compliance

*Corresponding author:

Das Sanjita

Department of Pharmaceutical Technology,
Noida Institute of Engineering and Technology,
Greater Noida (U.P), India.

*E-Mail: sanjita8@yahoo.co.in, uttamsingh.pharma@gmail.com

according to ethical principles and guidelines provided by Committee for the Purpose of Control and Supervision of Experiments on Animals (1121/ac/CPCSEA/07). The study was approved by Institutional Animal Ethical Committee (NIET/ IAEC/ 2011/ 24).

Collection and identification of plant material:

Plant fruits were collected from the college campus of Noida Institute of Engineering and Technology, Knowledge Park II, Greater Noida during September to November 2011. The plant was authenticated as *Callistemon lanceolatus* DC by Dr. Anjula Pandey (Principal Scientist), National Bureau of Plant Genetic Resources (NBPGR), Pusa Campus, New Delhi. A voucher specimen of the plant is preserved in the herbarium (NHCP/NBPGR/2011-25/).

Preparation of extract of the fruits of *Callistemon lanceolatus*:

Callistemon lanceolatus fruits were dried under shade. Dry fruits were uniformly grounded using a mechanical grinder to yield coarse powder. The powder (100 g) was extracted with methanol (300 ml) for 72 h. The extracts were filtered and evaporated to concentrate in water bath. The extract was cooled and kept in desiccator overnight. The methanol (5.605% w/w) extracts were weighed and used for the study.

Antidiabetic activity:

Rats were made diabetic by a single intraperitoneal injection of alloxan monohydrate (150mg/kg, i.p.; 5% w/v) in sterile saline. Twelve days after alloxan injection, rats with blood glucose level of >200 mg/dl were separated and used for the study. Blood glucose level was measured using blood glucose test strips with Easy Gluco™ Blood Glucose Meter at weekly intervals till the end of study (i.e. 3 weeks). The experiments were conducted on animal groups to see the effect of *Callistemon lanceolatus* methanol fruits extract (CLMFE) on diabetic rats. Six rats were used in each of the five groups that were as follows:

Group I. Normal control: received vehicle (distilled water).

Group II. Diabetic control: received vehicle (distilled water).

Group III. Diabetic rats treated with *Callistemon lanceolatus* methanol fruits extract (200 mg/kg body weight)

Group IV. Diabetic rats treated with *Callistemon lanceolatus* methanol fruits extract (400 mg/kg b.w.)

Group V. Diabetic rats treated with glibenclamide (10 mg/kg b.w.)

Vehicle (distilled water), CLMFE (200 mg/kg), CLMFE (400 mg/kg) and glibenclamide were administered once daily for 21 days from the day of induction. Blood was drawn from tip of the tail, and blood glucose level was estimated on 0, 7th, 14th, and 21st day of experiment with the help of Easy Gluco™ Blood Glucose Meter using strip method (Etuk, 2010; Kumar et al., 2011).

Hematological parameters:

After blood glucose estimation on day 21, whole blood was collected by cardiac puncture from rats under mild ether anesthesia. Blood was collected in a tube without anticoagulant for serum separation. Serum was separated by centrifugation at 2,500 rpm for 15 min, and utilized for biochemical studies. Serum cholesterol, triglycerides, HDL levels were evaluated in normal and alloxan-induced diabetic rats. TC, TG and HDL were analysed by kits (Roche Diagnostics GmbH, Mannheim, Germany) on Hitachi autoanalyser. Serum alanine transaminase (ALT or SGPT) and serum aspartate transaminase (AST or SGOT) were measured by autoanalyser (Erba Chem 7, Mannheim, Germany) (Ragavan et al., 2006).

Histopathology of liver and pancreas:

At the end of the 21 days, food was withdrawn from the rats and they were fasted overnight but the animals had free access to water. They were then euthanized under chloroform vapour and sacrificed. Internal organs including pancreas and liver were surgically removed. Thereafter, the tissues were placed in 10% formalin (diluted to 10% with normal saline) for 1 hr to rectify shrinkage due to high concentration of formalin. The tissues were dehydrated by ascending grades of isopropyl alcohol by immersing in 80% isopropanol overnight and 100% isopropyl alcohol for 1 hour. The wax impregnated tissues were embedded in paraffin blocks using the same grade wax. The paraffin blocks were cut with rotary microtome at 3 micron thickness. The sections were then melted in an incubator at 60°C and after 5 min the sections were allowed to cool. The sections were deparaffinised by immersing in xylene for 10 min in horizontal staining jar. After staining in hematoxylin, the sections were counter stained in 1% aqueous eosin (1 g in 100 ml tap water) for 1 min. Complete dehydration of stained sections was ensured by placing the sections in the incubator at 60°C for 5 min. When the sections were cooled, they were mounted in diphenylxylene (DPX) mount having the optical index of glass (the sections were wetted in xylene and inverted on to the mount and placed on the cover slip). The architecture was observed low power objective under microscope. The cell injury and over aspects were observed under high power dry objective (Ragavan et al., 2006).

Antioxidant activity by DPPH method:

The DPPH free radical is reduced to a corresponding hydrazine when it reacts with hydrogen donors. It is a discoloration assay, which is evaluated by the addition of the antioxidant to a DPPH solution in ethanol or methanol and decrease in absorbance is measured at 517 nm (Banu et al., 2011). The free radical scavenging activity was assayed spectrophotometrically (Blois, 1958) using deep blue 2, 2-diphenyl-1-picrylhydrazyl (DPPH) radical. The radical scavenging activity can be followed by a loss of absorbance at 525 nm. Sample stock solutions (1 mg/ml) were diluted to final concentrations of 50, 25, 12.5 and 6.25 mg/ml in methanol or DMSO. DPPH solution was prepared using 1.2 ml DPPH (0.2 mM in methanol), 3 ml methanol and 0.5 ml DMSO. DPPH solution (0.45 ml) was added to 0.05mL of sample solutions of different concentrations, shaken well by vortex, and allowed to react at room temperature. The absorbance values were measured after 10 min at 525 nm by UV/Vis spectrophotometer. The free radical scavenging activity of samples was calculated according to the formula:

$$\text{DPPH radical scavenging activity (\%)} = [1 - (\text{Abs}_{\text{sample}} - \text{Abs}_{\text{blank}}) / \text{Abs}_{\text{control}}] \times 100$$

Where Abs_{sample} is the absorbance of the experimental sample, Abs_{blank} is the absorbance of the blank;

Abs_{control} is the absorbance of the control. The ascorbic acid (vitamin C) was used as a positive control.

Each treatment was replicated thrice.

Statistical analysis:

All results were expressed as mean ± SEM. The data were analyzed using analysis of variance (ANOVA), and the group means were compared by Dunnett's test. Values were considered statistically significant with $P < 0.05$. GraphPad Instat was used for the analysis of data.

RESULTS AND DISCUSSION

Antidiabetic activity:

In diabetic control group there was significant ($P < 0.01$) increase in blood glucose levels from 257.5 to 383 mg/dl when compared to normal control. In the glibenclamide (10 mg/kg) and methanol extract (400 mg/kg) treated groups, the peak values of blood sugar significantly decreased from 255.16 to 112.33 mg/dl and from 253 to 92.5 mg/dl on the 21st day, respectively. In methanol extract (200mg/kg) treated group the blood glucose level decreased from 278 mg/dL to 153.83 mg/dL. Hence, in this study observations showed that the CLMFE (400 mg/kg) reduced the blood glucose level in diabetic rats but values did not return to those of normal controls. Therefore, CLMFE (400 mg/kg) possessed more significant ($P < 0.01$) antidiabetic activity while CLMFE (200 mg/kg) showed less significant antidiabetic activity, when compared with diabetic control. There was a marked reduction in blood glucose level (in 21 days) in alloxan induced diabetic animals. This effect of the CLMFE (400 mg/kg) is nearly equal to, if not better than, that of glibenclamide (10 mg/kg) (Fig. 1).

Alloxan has a destructive effect on the beta cells of the pancreas, which led to impaired glucose stimulated insulin release and insulin resistance, both of which are marked feature of type II diabetes (Jeloder et al., 2007). Oral hypoglycemic agents and insulin are currently available for treating diabetes mellitus. There is, however, a growing interest in herbal remedies due to the side effects associated with the existing drugs (Singh & Singh, 2010). The present investigation indicates the hypoglycemic and also protective effects of CLMFE on serum lipid profile of alloxan induced diabetic rats. It has been observed a significant ($P < 0.01$) decrease in blood glucose in CLMFE-treated diabetic rats, when compared with diabetic control rats.

Effect on hematological parameters:

Diabetes is also associated with altered lipid profile. There was a significant increase ($P < 0.01$) of serum total cholesterol (92.16–162.66), triglycerides (83.5–158.5), and significant decrease ($P < 0.01$) in HDL cholesterol (40.66–24) in diabetic rats as compared to that of normal control. Glibenclamide as well as both CLMFE regimens significantly decreased ($P < 0.01$) the levels of cholesterol and triglycerides. HDL cholesterol level was enhance (Figure 2) after 21 days of CLMFE treatment.

Diabetes is associated with hyperlipidemia. It is well known that insulin activates enzyme lipoprotein lipase, which hydrolyzes triglyceride under normal conditions. Destruction of β -cells leads to depletion of plasma insulin, which results in hyperlipidemia. The significant control of plasma lipid levels suggests that the CLMFE may produce its action by improving insulin secretion (Dewanjee et al., 2008).

Diabetogenic agents significantly increase the cholesterol and TG levels. The abnormally high concentration of serum lipids in diabetes mellitus is mainly due to an increase in the mobilisation of free fatty acids from the peripheral fat depots, since insulin inhibits the hormone-sensitive lipase. The marked hyperlipidemia that characterises the diabetic state may, therefore, be regarded as a consequence of the uninhibited actions of lipolytic hormones on the fat depots. Excess of fatty acids in plasma produced by alloxan promotes the liver conversion of some fatty acids to phospholipids and cholesterol. These two substances, along with excess of TG formed in the liver, may be discharged into lipoproteins in the blood. As a result, serum phospholipids are elevated (Rajagopal and Sasikala 2008). Administration of CLMFE to diabetic rats reversed all the above mentioned changes and improved the HDL levels. This effect may be due to low activity of cholesterol biosynthesis enzymes and/or low level of lipolysis which are under the control of insulin (Maiti et al., 2008).

There was a significant ($P < 0.01$) elevation in serum AST (SGOT) and ALT (SGPT) in the diabetic control animals as compared with normal control group. The administration of the CLMFE (200 mg/kg), CLMFE (400 mg/kg) and glibenclamide significantly ($P < 0.01$) decreased AST (SGOT) and ALT (SGPT) levels, when compared with diabetic control rats.

The present study showed significant effect of CLMFT on the liver of the diabetic rats (Fig. 3). The liver is regarded as the central metabolic organ in the body, with an important role in glucose and lipid homeostasis (Saravanan and Pari 2003). During diabetes, there is a decrease in liver weight due to enhanced catabolic processes such as glycogenolysis, lipolysis and proteolysis (Umesh et al., 2005) and therefore the quantification of glycogen, the primary intracellular storage form of glucose in liver can be considered as an important indicator of diabetes mellitus. Enzymes directly associated with the conversion of amino acids to keto acids are ALT and AST (Whitehead et al., 1999). The increase in the activities of serum AST, ALT indicated that diabetes may be induced hepatic dysfunction. Supporting our finding it has been reported (Larcan et al., 1979) that liver was necrotized in diabetic patients. Therefore, the increment of the activities of AST, ALT, in serum may be mainly due to the leakage of these enzymes from the liver cytosol into the blood stream, which gives an indication on the hepatotoxic effect of alloxan (Eidi and Eidi 2009).

Effect on Histopathology of pancreas:

After 21 days treatment period, the histopathological examination of the pancreas of the normal control rats showed round and elongated islets which were evenly distributed throughout the cytoplasm. In diabetic control rats, the cells were irregular, not well defined and necrosis of the cells could be clearly appreciated. Nuclear changes, karyolysis, disappearance of nucleus and in some places, residue of destroyed cells were visible. Relative reduction of size and number of islets especially around the central vessel and severe reduction of beta cells were clearly seen. The standard group showed a mild protection from alloxan induced changes in the pancreatic islets. CLMFE (200mg/kg) showed no significant changes in the cells when compared with the diabetic control. CLMFE (400 mg/kg) showed slight regeneration of beta cells as compared to that of the diabetic control group (Fig. 4).

Histological studies showed a degenerative effect on the pancreatic islet cells of diabetic rats. The results suggested restorative (protective) effect of the extract on pancreatic islet cells.

Effect on histopathology of liver:

Histology of the liver sections of normal control animals showed normal liver architecture with well brought out central vein, well-preserved cytoplasm and prominent nucleus and nucleolus. The alloxan-induced diabetic rat displayed feathery degeneration, micro and macro cellular fatty changes and inflammatory cells around portal tract. Glibenclamide treated animals showed a mild protection from alloxan-induced changes in the liver (Fig. 3). *Callistemon lanceolatus* methanol fruits extract (200 mg/kg) showed

less micro and macro cellular fatty changes in compare to diabetic control group.

Callistemon lanceolatus methanol fruits extract (400 mg/kg) showed no fatty degeneration and showed good protection against alloxan induced toxicity (Fig. 5).

Antioxidant activity:

The methanol extract showed strong antioxidant activity following DPPH radical scavenging experiment. DPPH radical scavenging activity of ascorbic acid showed 23±1.73µg/ml and scavenging activity of *Callistemon lanceolatus* methanol fruits extract (CLMFE) showed 18.66±2.33 at 6.25µg/ml concentration. At 12.50 µg/ml concentration scavenging activity of ascorbic acid was 63.33±2.33µg/ml while CLMFE showed 42.33±1.76. DPPH radical scavenging activity increased to 87.33±1.45µg/ml of ascorbic acid and 67.66±1.85 µg/ml of CLMFE at 25µg/ml. At 50µg/ml concentration ascorbic acid showed 90.33±2.90 µg/ml and CLMFE showed 83.67±2.72 µg/ml scavenging activity.

At 100µg/ml concentration scavenging activity of ascorbic acid was 91.73±2.89 µg/ml while CLMFE showed 84.41±3.32 µg/ml. Compared to ascorbic acid displaying 50% scavenging activity (RS50) at 10.4 µg/ml, the CLMFE exhibited RS50 at 17.0µg/ml on this test (Fig. 6). The effect of antioxidants on DPPH is thought to be due to their hydrogen donating ability (Shirwaikar et al., 2006). Though the DPPH radical scavenging abilities of the extracts were less than those of ascorbic acid, the study showed that the extracts have the proton-donating ability and could serve as free radical inhibitors or scavengers, acting possibly as primary antioxidants.

Increased production of high levels of oxygen free radicals has been linked to glucose oxidation and nonenzymatic glycation of proteins which contribute to the development of diabetic complications. Protective effects of exogenously administered antioxidants have been extensively studied in animal models within recent years, thus providing some insight into the relationship between free radicals, diabetes, and its complications (Maritim et al., 2003). In vitro and clinical studies may provide additional useful ways to probe the interconnections of oxidant stress and diabetes, and there is a need to continue to explore the mechanisms by which increased oxidative stress accelerates the development of complications in diabetes.

Increased oxidative stress is a widely accepted participant in the development and progression of diabetes and its complications. Diabetes is usually accompanied Mechanisms by which increased *Callistemon lanceolatus* oxidative stress is involved in the diabetic complications are partly known, including activation of transcription factors, advanced glycated end products (AGEs), and protein kinase C (Maritim et al., 2003).

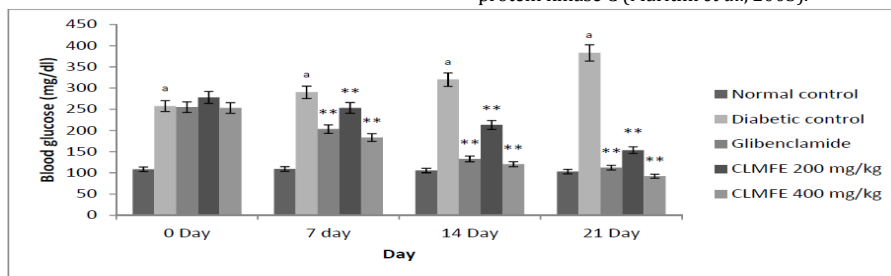


Fig. 1: The effect of CLMFE (200 mg/kg), CLMFE (400 mg/kg) and glibenclamide on blood glucose level in alloxaninduced diabetic rats at various days (on 0 day, 7th day, 14th day, and 21st day).

Each column represents mean ± SEM for six rats. aP<0.01 compared to normal controls, **P<0.01 compared to diabetic controls.

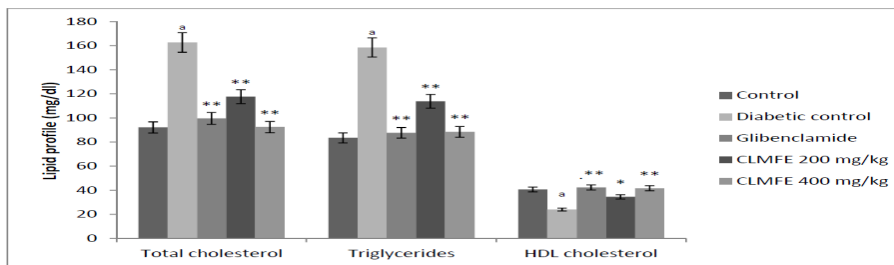


Fig. 2: Effect of CLMFE (200 mg/kg), CLMFE (400 mg/kg) and glibenclamide on serum lipid profile in alloxan induced diabetic rats.

Each column represents mean ± SEM for six rats. aP<0.01 compared to normal controls, *P<0.05, **P<0.01 compared to diabetic controls.

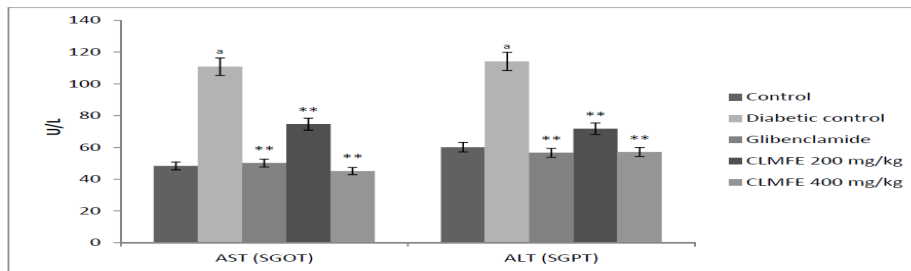


Fig. 3: Effect of CLMFE (200 mg/kg), CLMFE (400 mg/kg) and glibenclamide on liver parameters in alloxan induced diabetic rats. Each column represents mean \pm SEM for six rats. ^aP<0.01 compared to normal controls, ^{**}P<0.01 compared to diabetic controls.

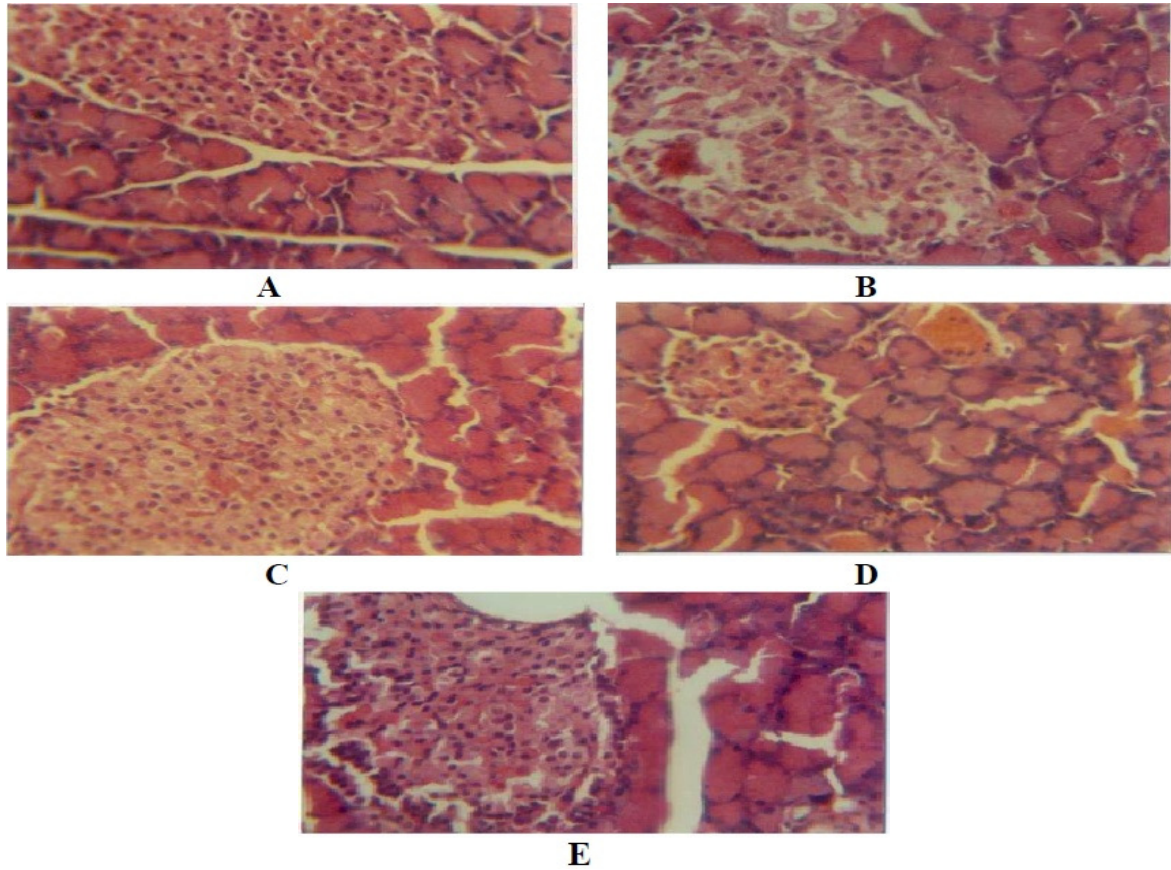
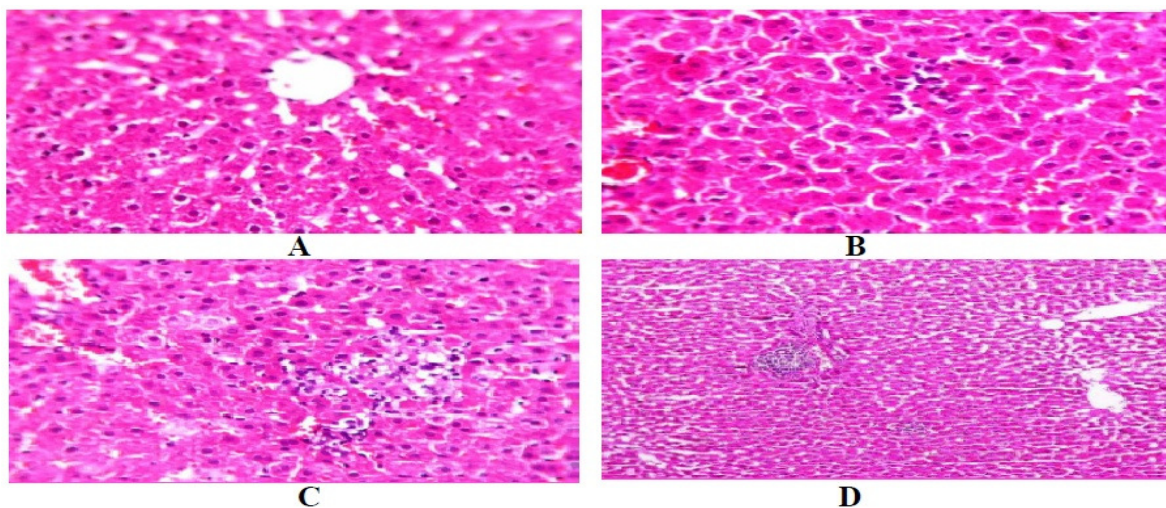


Fig. 4: Histological changes of rat pancreas of islets of langerhans. A) Normal control histological structure of rat pancreas showing normal islet; B) Diabetic control rat showing irregular cells, not well defined necrosis of cells is very clear; C) Glibenclamide (10 mg/kg) treated rat pancreas showing mild protection from alloxan induced changes in the pancreatic islets; D) CLMFE (200 mg/kg) showed no significant changes in cells were seen when compared with diabetic control; E) CLMFE (400 mg/kg) showed slight regeneration of β cells were seen when compared with diabetic control.



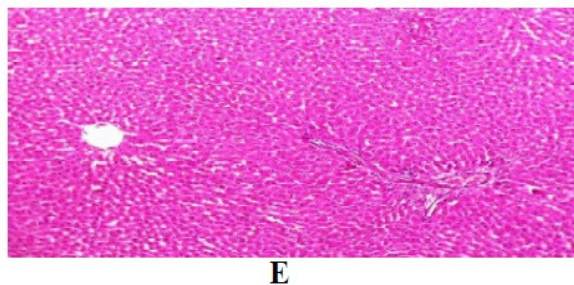


Fig. 5: Histopathological changes in liver of control and experimental rats. A) Normal control animals showed normal liver architecture; B) Diabetic control rat showing feathery degeneration, micro and macro cellular fatty changes and inflammatory cells around portal tract; C) Glibenclamide (10 mg/kg) treated rat liver showing a mild protection from alloxan-induced changes in the liver; D) CLMFE (200 mg/kg) showing less micro and macro cellular fatty changes in compare to diabetic control group; E) CLMFE (400 mg/kg) showing no fatty degeneration and showed good protection against alloxan induced toxicity.

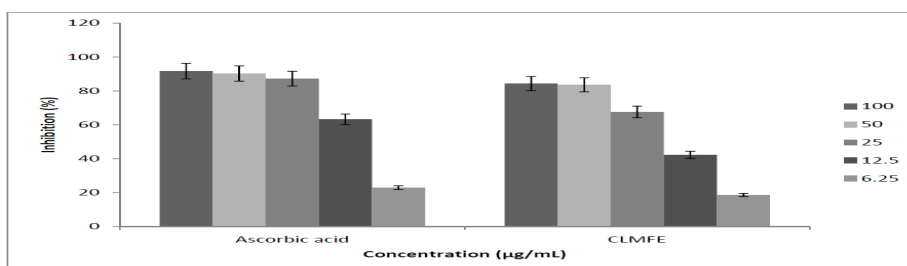


Fig. 6: DPPH radical scavenging activities of the extracts from *Callistemon lanceolatus*.

CONCLUSION

The results of this investigation revealed that the methanol extract of fruits of *Callistemon lanceolatus* possesses significant antidiabetic activity in alloxan-induced diabetic rats in a dose dependent manner. Alloxan has a destructive effect on the beta cells of the pancreas causing type II diabetes. Alloxan is a urea derivative which causes selective necrosis of the pancreatic islet β -cells. The possible mechanism of action of the methanol extract of fruits of *Callistemon lanceolatus* behind its antidiabetic activity may be through potentiation of pancreatic secretion of insulin from β -cell of islet and due to enhanced transport of blood glucose to the peripheral tissue. The extract showed to decrease cholesterol and TG level and increase HDL level. Diabetes is usually accompanied by increased production of free radicals or impaired antioxidant defenses. Experimental results supports the evidence the extract is capable of alleviating the augmented oxidative state associated with its antidiabetic activity.

REFERENCES:

- Banu S., Arunachalam G., Jayaveera K.N., Ashoka Babu V.L. and Premakumari K.B. Estimation of total phenolic content and *in vitro* antioxidant activity of *Barleria Montana*. *Der Pharmacia Lettre*, **2011**; 3(4): pp. 178-182.
- Blois M.S. Antioxidant determination by the use of a stable free radical. *Nature*, **1958**; 29: pp. 1199-1200.
- Dewanjee S., Bose S.K., Sahu R. and Mandal S.C. Antidiabetic effect of matured fruits of diospyros peregrine in alloxan-induced diabetic rats. *Int. J. Green Pharm.*, **2008**; 2: pp. 95-9.
- Eidi A. and Eidi M. Antidiabetic effects of sage (*Salvia officinalis* L.) leaves in normal and streptozotocin-induced diabetic rats. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, **2009**; 3: pp. 40-44.
- Etuk, E.U., 2010. Animals models for studying diabetes mellitus. *Agric. Biol. J. N. Am*, Vol. 1, Issue 2, pp. 130-134.
- Evans W.C. and Trease. *Textbook of Pharmacognosy*, XIII edition, Elsevier, London, **1998**.
- Jain A.K., Dubey S.K., Sikarwar M.S. and Jain S.K. Hepatoprotective activity of methanol extract of leaves of *Callistemon lanceolatus*. *Int. J. Plant Sci.*, **2007**; 2(2): pp. 185-186.
- Jeloder G., Mohsen M. and Shahram S. Effect of walnut leaf, coriander and pomegranate on blood glucose and histopathology of pancreas of alloxan induced diabetic rats. *Afr. J. Trad Complement Altern. Med.*, **2007**; 4(3): pp. 299-305.
- Kim J.H., Byun J.C., Bandi A.K.R., Hyun C.G. and Lee N.H. Compounds with elastase inhibition and free radical scavenging activities from *Callistemon lanceolatus*. *J. Med. Plant. Res.*, **2009**; 3(11): pp. 914-920.
- Kumar S., Kumar V. and Prakash O.M. Pharmacognostic study and anti-inflammatory activity of *Callistemon lanceolatus* leaf. *Asian Pac. J. Trop. Biomed.*, **2011**; pp. 177-181.
- Kumar S., Kumar V. and Prakash O.M. Antihyperglycemic, antihyperlipidemic potential and histopathological analysis of ethyl acetate fraction of *Callistemon lanceolatus* leaves extract on alloxan induced diabetic rats. *J. Exp. Integr. Med.*, **2011**; 1(3): pp. 185-190.
- Larcan A., Lambert H., Laprevote-Heully M.C. and Delorme N. Light and electron microscopic study of hepatic lesions in the course of hyperlactatemia in diabetic patients. *Diab. Metab.*, **1979**; 5: pp. 103-12.
- Maiti A., Dewanjee S., Jana G. and Mandal S.C. Hypoglycaemic effect of swietenia macrophylla seeds against type II diabetes. *Int. J. Green Pharm.*, **2008**; 2: pp. 224-7.
- Maritim A.C., Sanders R.A. and Watkins III J.B. Diabetes, oxidative stress, and antioxidants: A Review. *J. Biochem. Molecular Toxicology*, **2003**; 17(1): pp. 24-38.
- Nazreen S., Kaur G., Alam M.M., Shafi S., Hamid H. and Ali M., Alam M.S. New flavones with antidiabetic activity from *Callistemon lanceolatus* DC. *Fitoterapia*, **2012**; 83(8): pp. 1623-7.
- Rajagopal K. and Sasikala K. Antihyperglycaemic and antihyperlipidaemic effects of *Nymphaea stellata* in alloxan-induced diabetic rats. *Singapore Med. J.*, **2008**; 49(2): pp. 137-41.
- Ragavan B. and Krishnakumari S. Effect of *T. Arjuna* stem bark extract on histopathology of liver, kidney and pancreas of alloxan-induced diabetic rats. *African Journal of Biomedical Research*, **2006**; 9: pp. 189-197.
- Rao K.V.B., Paluri V., Ravichandran S., Kumar G. and Karthik L. Phytochemical composition and *in vitro* antimicrobial activity of methanol extract of *Callistemon lanceolatus* D.C. *Int. J. Pharm. Pharm. Sci.*, **2012**; 4(2): pp. 699-702.
- Saravanan R. and Pari L. Effect of Cogent db, a herbal drug, on serum and tissue lipid metabolism in experimental hyperglycaemic rats. *Diab. Obes. Metab.*, **2003**; 5: pp. 156-62.

20. Shirwaikar A., Prabhu K.S. and Punitha I.S.R. In vitro antioxidant studies of *Sphaeranthus indicus* (Linn). *Indian J. Exp. Biol.*, **2006**; 44: pp. 993-996.
21. Singh A.K. and Singh J. Evaluation of anti-diabetic potential of leaves and stem of *Flacourtia jangomas* in streptozotocin-induced diabetic rats. *Indian J. Pharmacol.*, **2010**; 42(5): pp. 301-305.
22. Umesh C.S., Yadav K, Moorthy K. and Najma Z.B. Combined treatment of sodium orthovanadate and *Mormodica charantia* fruit extract prevents alterations in lipid profile and lipogenic enzymes an alloxan diabetic rats. *Mol. Cell Biochem.*, **2005**; 268: pp. 111-20.
23. Whitehead M.W., Hawkes N.D., Hainsworth I. and Kingham J.G.C. A prospective study of the causes of notably raised aspartate transaminase of liver origin. *Gut*, **1999**; 45: pp. 129-33.

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