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# Research Article
# Protective Role of *Solandra longiflora* Flower Extracts on Streptozotocin Diabetes Induced Albino Rats

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## Abstract

**Background and Objective:** *Solandra longiflora* is a Neotropical genus of Solanaceae having phytochemical, ethnobotanical and ornamental importance. Its species contain small amounts of alkaloids such as atropine, noratropine, hyoscyamine, tropine and interestingly enough, these plants are said to be hallucinogenic. The aim was to evaluate the effect of methanol extract of the flower of *Solandra longiflora* on the prevention of oxidative stress caused by streptozotocin (STZ) was examined. **Materials and Methods:** Diabetes mellitus is a heterogeneous group of metabolic disorders characterized by high blood glucose levels. The pancreatic β-cells and their secretary hormone i.e., insulin are central in the pathophysiology of diabetes. Diabetes was induced in adult albino rats after administration of STZ (55 mg $kg^{-1}$ b.wt., i.p.). The effect of *Solandra longiflora* flower extract at SLFF 100 mg $kg^{-1}$ b.wt., SLFF 200 mg $kg^{-1}$ b.wt. and SLFF 300 mg $kg^{-1}$ b.wt. and SLFF 400 mg $kg^{-1}$ b.wt., at different concentrations orally for 28 days was investigated in the diabetic rats. **Results:** Insulin treated diabetic rats (6 U $kg^{-1}$, i.p., 28 days) served as the positive control, diabetic rats given normal saline served as diabetic control and non-diabetic rats were served as normal control. Serum creatine phosphokinase (CPK) increased in diabetic rats was significantly decreased progressively on insulin and SLFF treatments. The decrease in activities of superoxide dismutase (SOD) and catalase (CAT) and increase in lipid peroxidation (LPO) of erythrocytes as observed in diabetes was regained after insulin and SLFF treatments. However, there was a slight improvement in SOD, CAT and LPO of kidneys on SLFF different concentration treatment, though, not significant when compared to the standard. Despite increased CAT and SOD activities in the liver and heart, LPO was also increased in diabetic rats. Insulin and SLFF treatments significantly protected animals from cardiac damage. **Conclusion:** From this study, it was observed that SLFF prevent oxidative stress caused by STZ in the heart and erythrocytes. Thus, the use of this agent could help in the pharmaceutical industry.

Key words: Protective, *Solandra longiflora*, flower, streptozotocin, diabetes, rats

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

## INTRODUCTION

*Solandra longiflora* is a Neotropical genus of Solanaceae having phytochemical, ethnobotanical and ornamental importance. Its species contain small amounts of alkaloids such as atropine, noratropine, hyoscyamine, tropine and others[1], with a significant amount of alkaloids content in the leaves. Interestingly enough, these plants are said to be hallucinogenic, Mexican aborigines, i.e., the Huichols and possibly other groups, make use of these showy climbers on account of their medicinal and narcotic properties in rituals, witchcraft and sorcery. A tea from the juice of branchlets is the inebriant and the fruits and the roots are considered far more powerful than the leaves[2].

Despite their interest, no comprehensive work was done on the flower which as much remains to be done before a complete understanding of *Solandra* chemistry and ethnobotany is complete. The plants only consist of fallen flowers frequently epiphytic. It is a solitary funnel-shaped large up to 25-30 cm long and 10 cm wide at mouth corolla recurved and 5 lobed, 5 dark veins inside calyx tube, open light or creamy yellow with fragrant at night, the flower tolerates salt spray and saline soils, it contains alkaloids. It functions in digestion of substrate, stimulation of cell maturation, immune system and aid in intestinal transit and colonization resistance[3].

Thus, with the flower extract of *Solandra longiflora* this study is focusing on the potentials of the extracts on streptozotocin diabetes-induced albino rats. However, we are made to understand that this diseases diabetes mellitus has been a threat to mankind from time immemorial and it is now reeking havoc disproportionately worldwide[4].

It is a public health problem acknowledged as one of the most important killer diseases and a prominent cause of death in low and middle-income countries[5]. The life expectancy of diabetic patients is usually low compared to normal people[6]. DM is a no communicable disease in which there is a metabolic disorder of various aetiologies described by sustained hyperglycaemia with disorders of carbohydrate, fat and protein metabolism following defects in insulin secretion, insulin action, or both[6]. It is caused by the destruction of pancreatic β-cells or dysfunctional β-cell and insulin resistance which results in hyperglycaemia[7,8]. Over time, diabetic patients with poor glycaemic control undergo micro-and macrovascular complications including nephropathy, retinopathy, neuropathy and cardiovascular diseases[9-11]. These complications increase their suffering and are the major sources of expenses for patients with diabetes as well as increasing the financial burden of nations[12,13]. Above and beyond insulin are other therapeutic options for the treatment of type 1 diabetes which include transplantation of whole organ pancreas and isolated islets.

However, numerous agents that are currently used for the treatment of type 2 diabetes are facing limited efficacy and tolerability[14]. For instance, sulfonylureas induce β-cell death in isolated rodent and human islets while glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase-4 inhibitors have potential risks for pancreatitis, pancreatic and thyroid cancers[15,16].

Thus, the logical long-term solution to diabetic therapy is the restoration of β-cells since β-cell deficiency underlies both type 1 and type 2 diabetes[17]. The restoration of deficient β-cell mass by transplantation from exogenous sources or by endogenous regeneration of insulin-producing cells would undoubtedly be a worthwhile therapeutic goal that will significantly ameliorate diabetes and its complications[18,19].

Another approach to the treatment of diabetes is the application of medicinal plants with phytochemicals that cause beta-cell regeneration leading to normal blood glucose in animals and humans[20]. Many medicinal plants were found to be effective against various diseases including diabetes mellitus[21-25]. Several studies have confirmed that bioactive compounds such as alkaloids, flavonoids, phenols, tannins and terpenoids produce hypoglycemic, anti-inflammatory, antifungal and antimalarial effects[26-29]. However, there is limited documentation on the potential use of the *Solandra longiflora* flower in the treatment of diabetes mellitus. Therefore, this study aimed to evaluate the protective role of *Solandra Longiflora* flower extracts on streptozotocin diabetes-induced albino rats.

## MATERIALS AND METHODS

**Study area:** This study was carried out in Malaysia, Faculty of Resource Science and Technology, Universiti Malaysia Sarawak from 26th October to 14th June, 2020/2021.

**Animals:** Adult albino rats (180-200 g) were obtained from the Laboratory Animal Farm of Universiti Malaysia Sarawak. The animals were maintained on standard animal feed and provided with clean drinking water *ad libido*. The animals were kept under a condition of 20±3°C and acclimatized for a period of 7 days.

**Plant material:** The plants *Solandra longiflora* flower was procured from a reserved forest and were dried in a sample drying house. The methanol extracts were carried out through the conventional method, a total of 1000 g of the powdered sample was extracted using the cold-soaking method. This

was achieved by soaking the ground plant material in polar solvents methanol. The dried and ground flower was soaked in methanol in 5 L Erlenmeyer flasks at room temperature for 72 hrs. The resulting methanol solution was then filtered using filter paper and the residue was re-extracted with fresh methanol for another 72 hrs and filtered. All the extracts were combined and concentrated using the rotary evaporator (model Heidolph Laborota 4000 efficient) under reduced pressure to obtain the crude extract. At the end of the extraction process, the dry weight and yield of the crude extracts were determined.

**Chemical and drugs:** All chemicals and drugs used in this investigation were of analytical grade and were obtained from Sigma Chemical Co., St Louis, USA). Insulin and Streptozotocin (reference drug) was obtained from Medical Resource SDN BHD Kuching, Sarawak.

**Induction of diabetes:** To induce diabetes, STZ (Sigma), prepared freshly in citrate buffer, pH 4.5, was immediately injected intravenously (55 mg $kg^{-1}$) through tail vein[30]. The rats were monitored for plasma glucose levels at weekly intervals. The rats with a fasting glucose value of >250 mg $dL^{-1}$ were considered diabetic. Blood samples were drawn by retro-orbital venipuncture technique. Plasma was separated by centrifugation at 2000 rpm for 15 min. Glucose levels were measured by the O-Toluidine method using standard kits from Medical Resource SDN BHD Kuching, Sarawak. Based on the plasma glucose levels, uniformly diabetic rats were selected on day 30 after the injection of STZ.

**Treatment of animal:** Animals were divided into eight groups of five animals and treated as follows:

- **Group I:** Given citrate buffer and served as control (without STZ)

STZ induced diabetic rats were divided into four groups (Groups II–V).

- **Group II :** Diabetic control
- **Group III :** Positive control (insulin 6 U $kg^{-1}$ i.p.)
- **Group IV :** SLFF 100 mg $kg^{-1}$ b.wt.
- **Group V :** SLFF 200 mg $kg^{-1}$ b.wt.
- **Group VI :** SLFF 300 mg $kg^{-1}$ b.wt.
- **Group VII :** SLFF 400 mg $kg^{-1}$ b.wt.
- **Group VIII :** SLFF 500 mg $kg^{-1}$ b.wt.

Treatments were given orally for 28 days.

**Collection of samples:** The blood samples were collected at the start and 7th, 14th, 21st and 28th day of the experiment in two aliquots from retro-orbital plexus using micro-capillary technique[31]. In one of the aliquots, no anticoagulant was used. Serum was separated. In the second aliquots, heparin was used as an anticoagulant. Plasma was separated. The erythrocytes from heparinized blood were employed for membrane preparation by the method of Sonne[32] using 1 mM EDTA in a hypotonic buffer. The erythrocytes were washed thrice with ice-cold saline and suspended in 50% (v/v) of saline. After blood collection, all the rats were sacrificed by euthanasia. The organs such as liver, heart and kidney were excised immediately and kept on ice, homogenized and homogenates were centrifuged at 10,000×g for 10 min in a refrigerated centrifuge. Supernatants were collected and stored at 4°C.

**Estimation of antioxidant enzymes**

**Superoxide dismutase:** Superoxide dismutase (SOD) activity was determined in the homogenates and erythrocytes according to Muruganandan *et al.*[33]. A colourimetric assay involving the generation of superoxide by pyrogallol auto-oxidation and the inhibition of superoxide-dependent reduction of the tetrazolium dye, MTT (3-(4, 5-dimethylthiazol-2-yl) 2, 5-diphenyltetrazolium bromide) to its formazan by SOD was measured at 570 nm. The amount of MTT formazan was calculated by using a molar extinction coefficient E570 of 17,000 $M^{-1}$ $cm^{-1}$. One unit of SOD was defined as the amount of protein required to inhibit the MTT reduction by 50%.

**Catalase:** Catalase (CAT) activity was measured in homogenates and erythrocytes by the method of Pandya *et al.*[34]. The utilization of $H_2O_2$ by CAT in the samples was measured spectrophotometrically as the decrease in optical density at 254 nm. The substrate ($H_2O_2$) concentration was 20 mM for erythrocyte and cardiac tissue CAT measurement, while 2 mM for the renal CAT.

**Lipid peroxidation:** Lipid peroxidation (LPO) in tissue homogenates and erythrocytes was studied by measuring thiobarbituric acid reactive substances (TBARS). The results are expressed as nmol malondialdehyde (MDA) formed per ml packed erythrocyte and nmol MDA formed per g tissue per 30 min in heart, liver and kidney homogenates.

**Serum creatine phosphokinase:** Serum from animals was evaluated for creatine phosphokinase (CPK) activity using the kit obtained from medical resource SDN BHD Kuching, Sarawak.

**Statistical analysis:** Data were expressed as Mean±Standard Deviation for three determinations of each experiment. The analysis was done using the software-SPSS one-way ANOVA.

## RESULTS

Table 1 shows the protection of SLFF extract against cardiac injury, the serum creatine phosphokinase (CPK) activity is increased significantly in the STZ-induced diabetic rats as compared to the normal rats, the treatment of the diabetic rats with insulin (6 U $kg^{-1}$, i.p.) Methanol crude extract of SLFF of SLFF 100 mg $kg^{-1}$, b.wt., SLFF 200 mg $kg^{-1}$, b.wt., SLFF 300 mg $kg^{-1}$ b.wt., SLFF 400 mg $kg^{-1}$ b.wt., SLFF 500 mg $kg^{-1}$ b.wt., with the increase in concentration resulted in a significant reduction of serum phosphokinase activity of the diabetic rats when compared to the control insulin as shown with group VIII where the value reduced to 86.57±8.37, as when compared to the other groups with a value of 67.56±33.15, 189.89±5.36, 184.34±11.23, 134.42±23.11, 110.23±21.45 for group 1V, V, VI and VII. This indicates the serum phosphokinase reduced with increases in the SLFF concentration.

Table 2-5 indicated the antioxidant activity of the SLFF on the SOD, CAT and LPO of the Heart, Erythrocytes, Liver and Kidney. The STZ administration resulted in a significant elevation of cardiac SOD, CAT and LPO. With repeated administration of insulin (6 U $kg^{-1}$), SLFF of different concentrations of 100 mg $kg^{-1}$ b.wt., SLFF 200 mg $kg^{-1}$ b.wt., SLFF 300 mg $kg^{-1}$, b.wt., SLFF 400 mg $kg^{-1}$, b.wt., SLFF 500 mg $kg^{-1}$ b.wt., at higher concentration for 28 days, the cardiac LPO was significantly reduced comparable to normal control rats (13.16±5.66) as show in Table 2. It was observed that group IV of SLFF 100 mg $kg^{-1}$ b.wt., indicated a high value of CAT, SOD and LPO when compared to the insulin control and less when compared to diabetic control, also a concentration of 200, 300 and 400 mg $kg^{-1}$ b.wt., indicated decreases in the value of CAT, SOD AND LPO with 7.45±1.76, 21.14±1.64, 38.99±6.17 and 6.89±3.14, 20.56±2.39, 30.12±1.17 and 6.16±5.11, 17.33±3.36, 28.16±3.33**, respectively. From the resulting group, VIII gave better diabetic control of CAT, SOD and LPO of heart in STZ induced rats.

Table 3 shows the effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of erythrocytes in STZ-induced diabetic rats. With the administration of SLFF at different concentrations of 100, 200, 300, 400 and 500 mg $kg^{-1}$ b.wt., the LPO and SOD of erythrocytes was significantly reduced compared to the normal control rats. As shown with group VIII where the value of SOD reduced to 81.23±2.78 as when compared to the other groups with a value of 84.13±8.29, 87.12±5.17, 89.56±26.12, 98.11±6.34 and 84.12±12.43 for groups VII, VI, V and IV, respectively. With the increased concentration of SLFF, the CAT of erythrocyte significantly increased as shown in groups IV, V, VI, VII and VIII with values 1.49±1.12, 1.89±2.13, 1.89±2.13, 2.56±11.5, 3.12±3.45 and 5.13±2.98, respectively.

Table 4 shows the effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of the liver in STZ-induced diabetic rats. With the administration of STZ, there was a significant increase in the LPO, CAT and SOD in the diabetic control when compared to the normal control. As shown the CAT and LPO the result revealed a dose-dependent effect value at 200 mg $kg^{-1}$ b.wt., for group V with 97.45±13.48, 44.78±5.15, at 300 mg $kg^{-1}$ b.wt., it was also observed there was a progressive reduction with values of 88.67±16.32, 38.11±3.16 and at 400 mg $kg^{-1}$ b.wt., with 79.27±11.16, 35.24±2.78**, respectively and however, 500 mg $kg^{-1}$ b.wt., showed remarkable values of 73.11±34.12**, 34.33±6.77** when compared with the diabetic and insulin of 50.12±6.17, 26.44±8.33, respectively.

Despite the rise in the antioxidant enzyme activity in hepatic tissue, there was a significant elevation of LPO, However, administration of the SLFF show little or ineffective on the LPO while SOD and CAT enzyme activities were inhibited when compared to the controls.

Table 5 shows the effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of kidney in STZ-induced diabetic rats. There was a significant decrease in CAT and SOD in diabetes control rats of 19.65±7.83, 18.16±5.66, respectively when compared to the normal control. With increased administration of SLFF at 100, 200, 300, 400 and 500 mg $kg^{-1}$ b.wt., the CAT and SOD increased with 19.57±7.12, 18.18±6.78, 22.23±5.14, 18.65±6.33, 24.11±6.59, 19.23±7.32 and 26.12±13.2219.78±3.45, 28.33±11.34, 20.32±6.55**, respectively, with 500 mg $kg^{-1}$ b.wt., having the highest value, while LPO decreased significantly from 100, 200, 300, 400 and 500 with values of 51.17±6.14, 49.65±3.12, 43.13±6.37, 40.78±5.77 and 34.62±8.17 when compared to the values obtained from group VII CAT with the value 28.33±11.34 as compared to other groups with the values 26.12±13.22, 24.11±6.59, 22.23±5.14 and 19.57±7.12 for group VII, VI, V and IV respectively. The rise in the antioxidant enzyme activity in the hepatic tissue indicated also an elevation in the LPO and the SOD and CAT enzyme activities where a significant decrease was observed in the kidney. The significant elevation in LPO, there was a significant activity but not when compared to the control.

Table 1: Effect of SLFF given orally daily for 28 days on serum creatine phosphokinase of STZ-induced rats

| Groups | Treatments | Creatine phosphokinase (IU $1^{-1}$) |
|---|---|---|
| Group I | Control (citrate buffer) | 58.87±8.97 |
| Group II | Diabetes control | 187.77±66.13 |
| Group III | Insulin (6 U $kg^{-1}$ = i.p.) | 67.56±33.15 |
| Group IV | SLFF 100 mg $kg^{-1}$ b.wt. | 189.89±5.36 |
| Group V | SLFF 200 mg $kg^{-1}$ b.wt. | 184.34±11.23 |
| Group VI | SLFF 300 mg $kg^{-1}$ b.wt. | 134.42±23.11 |
| Group VII | SLFF 400 mg $kg^{-1}$ b.wt. | 110.23±21.45 |
| Group VIII | SLFF 500 mg $kg^{-1}$ b.wt. | 86.57±8.37 |

Values are Mean±SD, n = 5, *p>0.05 and **p<0.01 as compared to diabetes control

Table 2: Effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of heart in STZ-induced diabetic rats

| Groups | Treatments | CAT (Ua×$10^3$) | SOD, Ub | LPO, c |
|---|---|---|---|---|
| Group I | Control (citrate buffer) | 3.98±1.88 | 14.98±3.12 | 13.16±5.66 |
| Group II | Diabetes control | 7.86±2.65 | 21.19±4.87 | 40.33±7.76 |
| Group III | Insulin (6 U $kg^{-1}$) | 5.01±1.66* | 12.34±4.33* | 16.88±3.14 |
| Group IV | SLFF 100 mg $kg^{-1}$ b.wt. | 7.77±3.14 | 21.67±3.34 | 40.11±4.55 |
| Group V | SLFF 200 mg $kg^{-1}$ b.wt. | 7.45±1.76 | 21.14±1.64 | 38.99±6.17 |
| Group VI | SLFF 300 mg $kg^{-1}$ b.wt. | 6.89±3.14 | 20.56±2.39 | 30.12±1.17 |
| Group VII | SLFF 400 mg $kg^{-1}$ b.wt. | 6.16±5.11 | 17.33±3.36 | 28.16±3.33** |
| Group VIII | SLFF 500 mg $kg^{-1}$ b.wt. | 5.78±3.26** | 15.66±7.23 | 26.84±1.18** |

Values are Mean±SD, n = 5, *p>0.05, **p<0.01 as compared to diabetic control, Ua: Velocity constant per second, Ub: Amount of haemoglobin (mg) inhibiting MTT by 50% and c: nM of MDA produced/g tissue per minute

Table 3: Effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of erythrocytes in STZ-induced diabetic rats

| Groups | Treatments | CAT (Ua×$10^3$) | SOD, Ub | LPO, c |
|---|---|---|---|---|
| Group I | Control (citrate buffer) | 6.24±3.21 | 84.12±12.43 | 6.13±12.3 |
| Group II | Diabetes control | 4.01±1.16 | 55.03±14.36 | 9.49±1.36 |
| Group III | Insulin (6 U $kg^{-1}$) | 5.11±3.37* | 77.27±11.23 | 6.01±2.34* |
| Group IV | SLFF 100 mg $kg^{-1}$ b.wt. | 1.49±1.12 | 98.11±6.34 | 8.86±1.67 |
| Group V | SLFF 200 mg $kg^{-1}$ b.wt. | 1.89±2.13 | 89.56±26.12 | 8.47±5.77 |
| Group VI | SLFF 300 mg $kg^{-1}$ b.wt. | 2.56±11.5 | 87.12±5.17 | 6.78±11.23** |
| Group VII | SLFF 400 mg $kg^{-1}$ b.wt. | 3.12±3.45 | 84.13±8.29 | 6.15±7.87** |
| Group VIII | SLFF 500 mg $kg^{-1}$ b.wt. | 5.13±2.98** | 81.23±2.78 | 5..89±4.15** |

Values are Mean±SD, n = 5, *p>0.05, **p<0.01 as compared to diabetic control, Ua: Velocity constant per second, Ub: Amount of haemoglobin (mg) inhibiting MTT by 50% and c: nM of MDA produced/g tissue per minute

Table 4: Effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of liver in STZ-induced diabetic rats

| Groups | Treatments | CAT (Ua×$10^3$) | SOD, Ub | LPO, c |
|---|---|---|---|---|
| Group I | Control (citrate buffer) | 59.66±13.32 | 25.26±7.12 | 27.34±13.5 |
| Group II | Diabetes control | 101.11±14.56 | 35.11±2.31 | 50.12±6.17 |
| Group III | Insulin (6 U $kg^{-1}$) | 71.33±22.17* | 23.12±7.34 | 26.44±8.33* |
| Group IV | SLFF 100 mg $kg^{-1}$ b.wt. | 101.08±11.23 | 35.01±5.42 | 46.23±7.14 |
| Group V | SLFF 200 mg $kg^{-1}$ b.wt. | 97.45±13.48 | 34.76±4.11 | 44.78±5.15 |
| Group VI | SLFF 300 mg $kg^{-1}$ b.wt. | 88.67±16.32 | 33.86±7.12 | 38.11±3.16 |
| Group VII | SLFF 400 mg $kg^{-1}$ b.wt. | 79.27±11.16 | 32.79±1.32** | 35.24±2.78** |
| Group VIII | SLFF 500 mg $kg^{-1}$ b.wt. | 73.11±34.12** | 30.38±6.87** | 34.33±6.77** |

Values are Mean±SD, n = 5, *p>0.05, **p<0.01 as compared to diabetes control, Ua: Velocity constant per second, Ub: Amount of haemoglobin (mg) inhibiting MTT by 50% and c: nM of MDA produced/g tissue per minute

Table 5: Effect of SLFF given orally daily for 28 days on SOD, CAT and LPO of kidney in STZ-induced diabetic rats

| Groups | Treatments | CAT (Ua×$10^3$) | SOD, Ub | LPO, c |
|---|---|---|---|---|
| Group I | Control (citrate buffer) | 46.16±6.63 | 24.88±2.11 | 29.45±5.78 |
| Group II | Diabetes control | 19.65±7.83 | 18.16±5.66 | 55.11±8.68 |
| Group III | Insulin (6 U $kg^{-1}$) | 22.34±7.34* | 26.51±7.12 | 7.23±8.79 |
| Group IV | SLFF 100 mg $kg^{-1}$ b.wt. | 19.57±7.12 | 18.18±6.78 | 51.17±6.14 |
| Group V | SLFF 200 mg $kg^{-1}$ b.wt. | 22.23±5.14 | 18.65±6.33 | 49.65±3.12 |
| Group VI | SLFF 300 mg $kg^{-1}$ b.wt. | 24.11±6.59 | 19.23±7.32 | 43.13±6.37 |
| Group VII | SLFF 400 mg $kg^{-1}$ b.wt. | 26.12±13.22 | 19.78±3.45 | 40.78±5.77 |
| Group VIII | SLFF 500 mg $kg^{-1}$ b.wt. | 28.33±11.34 | 20.32±6.55** | 34.62±8.17 |

Values are Mean±SD, n = 5, *p<0.05, **p<0.01 as compared to diabetic control, Ua: Velocity constant per second, Ub: Amount of haemoglobin (mg) inhibiting MTT by 50% and c: nM of MDA produced/g tissue per minute

## DISCUSSION

The effect of *Solandra Longiflora* flower extract from Table 1 was observed to have shown a significant result on treatment for 28 days on serum creatine phosphokinase where a treated group of the induced rats the enzyme was gradually reduced with an increase in the concentration of the extract, from 100, 200, 300, 400 and 500 mg $mL^{-1}$ with 189.89±5.36, 184.34±11.23, 134.42±23.11 and 110.23±21.45 and 86.57±8.37, respectively when compared to the controls in group I, II and III.

The effect of STZ cause the diabetes rapid depletion of β-cells which causes the reduction of insulin release and hyperglycaemia causes oxidative damage by the generation of ROS and development of diabetic complications. Further, the STZ diabetic animals may exhibit most of the diabetic complications, namely, myocardial cardiovascular, gastrointestinal, nervous, vas deferens, kidney and urinary bladder dysfunctions[35].

Increased serum CPK level in diabetic rats indicates cardiac muscular damage. Elevated concentration of CPK was recovered by treatment with insulin and SLFF which suggest their cardioprotective effect. SLFF shows a significant effect when compared to Insulin

Table 2 shows the oral daily evaluation of the effects of the crude extract on the SOD, CAT and LPO of the induced rats, a significant result was observed concentration 500 mg $mL^{-1}$ with 5.78±3.26**, 15.66±7.23, 26.84±1.18** when compared with the standard drug insulin with Insulin (6U $kg^{-1}$) 5.01±1.66*, 12.34±4.33* and 16.88±3.14 on CAT, SOD and LPO, respectively.

These enzymes SOD and CAT are considered primary antioxidant enzymes since they are involved in the direct elimination of ROMs[36]. The effect of STZ in LPO, CAT and SOD activities was found to be tissue-dependent. Despite increased cardiac CAT and SOD activities in diabetic rats, an increase in LPO was observed. Higher LPO and low SOD and CAT activity indicate an oxidative stress condition. The effect on LPO, CAT and SOD was reversed by insulin and by the SLFF treatments in the heart as shown in Table 2, CAT from group IV, V, VI, VII and VIII, reduced significantly from treatment with 100 mg $mL^{-1}$ to 500 mg $mL^{-1}$ with 7.77±3.14, 7.45±1.76, 6.89±3.14, 6.16±5.11 and 5.78±3.26, respectively, the SOD as was reduced with 21.67±3.34, 21.14±1.64, 20.56±2.39, 17.33±3.36, 15.66±7.23 with increases in concentration, however, the LPO was observed to have a significant inhibition result at $p>0.05$ with 40.11±4.55, 38.99±6.17, 30.12±1.17, 28.16±3.33 and 26.84±1.18 but was less when compared to the values of the CAT and SOD this agrees with the report of Li *et al.*[37], in their study observed increase in heart LPO in diabetic rats.

The observation suggests that to overcome the oxidative damage in the heart, some other compensatory mechanisms exist in the heart in addition to antioxidant enzymes. Reversal of increased enzymes and inhibition of LPO appears to be due to free radical scavenger activity of SLFF in the heart. The erythrocyte LPO was significantly increased in diabetic controls with the reduction in antioxidant enzyme activities of SOD and CAT. Treatment with insulin and SLFF stimulated SOD and CAT to reverse oxidative damage to the erythrocyte membrane.

From the study in Table 3, it was observed that there was the little reduction of the SOD and LPO in the erythrocytes of the induced diabetic even after the treatment with SLFF in groups IV V. VI, VII and VIII with 98.11±6.34, 89.56±26.12, 87.12±5.17, 84.13±8.29, 81.23±2.78 and 8.86±1.67, 8.47±5.77, 6.78±11.23, 6.15±7.87, 5..89±4.15, respectively, when compared to the test control with 84.12±12.43 and 6.13±12.3. However, the CAT showed a significant reduction with 1.49±1.12, 1.89±2.13, 2.56±11.5, 3.12±3.45, 5.13±2.98 at $p>0.01$ when compared to the Insulin (6 U $kg^{-1}$) with 9.49±1.36 as well as the diabetic control with 6.01±2.34. This agrees with the report of Swamy *et al.*[38], where a significant increase in LPO, SOD and CAT in pancreas, heart and blood and an increase in glutathione peroxidase in kidney and pancreas in diabetes rats was observed.

The study revealed erythrocytes was significantly increased in diabetic rat control and with the administration of SLFF, the LPO, CAT and SOD was significantly reduced compared to the normal control rats.

Table 4 shows the effect of SLFF on SOD, CAT and LPO of the liver in STZ-induced diabetic rats, from the result it was observed there was little effect observed in all the four groups when compared with the test control.

With the administration of STZ, there was a significant increase in the LPO, CAT and SOD in the diabetic control when compared to the normal control, despite the rise in the antioxidant enzyme activity in hepatic tissue, there was a significant elevation of LPO, administration of the SLFF show little or ineffective on the LPO while SOD and CAT enzyme activities were inhibited when compared to the controls.

In Table 5, the effect of SLFF on SOD, CAT and LPO of the kidney of the induced rats was observed to have significant inhibition of the enzymes in all the groups with group VIII with higher inhibition rates of 28.33±11.34, 20.32±6.55, 41.62±8.17 SOD, CAT and LPO, respectively when compared to the test controls in group I, II and III.

The study revealed that CAT and SOD activities were significantly inhibited along with an elevation of LPO in the

kidney of STZ-treated diabetic animals which is not reversed by different treatments. Enzyme activities were significantly decreased this was observed in the kidney. The significant elevation in LPO. There was a significant activity but not when compared to the control. This was also reported by Jose *et al.*[39], where he stated that the significant increase in LPO, SOD and CAT and an increase in glutathione peroxidase in kidney and pancreas in diabetes rats was observed.

The experimental results indicated that LPO played a role in tissue injury in STZ-induced diabetic rats. SLFF reduced the LPO in the heart and erythrocytes, thus effectively protecting cell functions and structure. STZ-induced diabetic oxidative changes of cardiac and erythrocyte toxicity as observed was reversed by the significant stimulation of antioxidant defence mechanism in erythrocytes or compensatory elevation of antioxidant defence mechanism in cardiac tissue by SLFF. In STZ-induced diabetes, however, renal and hepatic toxicity was not prevented by SLFF. The results indicate that the methanol extract of the *Solandra longiflora* flower showed significant protection against the oxidative damage induced by STZ in the heart and erythrocytes of rats. SLFF may act as cardioprotective and a free radical scavenger agent.

However, the limitation was getting enough of the flower quantity and having access to a research machine to evaluate the chemical constituents of the extract will help to identify which chemical constituents are responsible for these activities.

Further studies should be cared out to unveil the phytochemicals endured in this plant material through extraction, isolation and characterization to get this active component using GCMS, NMR or higher upgraded research machinery.

## CONCLUSION

In conclusion, this study shows that the phytoconstituents in the *Solandra longiflora* flower exerts promising oxidative stress caused by STZ in the heart and erythrocytes of diabetic rats. From the above discussion, it concludes that methanol extract of *Solandra longiflora* flower at dose 400 and 500 mg $kg^{-1}$ exhibited significant anti-oxidative stress in STZ induced diabetic rats. These extracts also showed improvement in enzyme balance which was distorted as a result of STZ. Thus, the anti-oxidative agents can be explored for development as antihyperglycemic activity and further studies carried out in this direction to find out mechanisms of action may result ineffective treatment and control of diabetes.

## SIGNIFICANCE STATEMENT

This study discovered a promising safety effect of SLFF extract as is assured without any effect on the enzymes in the heart, erythrocytes, liver and kidney and can be beneficial for the treatment and speed up and cuticle menace endured diabetes patients. This study will help the researchers to uncover the critical areas behind the fact that diabetes doesn't heal and that of which many researchers were not able to explore. "Thus a new theory on the fact that medicinal plants extract as the major source for the treatment of diabetic may be arrived at".

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