



Journal of  
**Pharmacology and  
Toxicology**

ISSN 1816-496X



Academic  
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## Antihyperglycemic Effect of *Solanum surattense* Leaf-Extract in Streptozotocin Induced Diabetic Rats

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**Abstract:** In the present study, we have evaluated the validity of traditional usage of *Solanum surattense* as an antidiabetic agent. Adult male albino rats of Wistar strain, weighing 180-200 g, were made diabetic by administration of streptozotocin (STZ) (40 mg kg<sup>-1</sup> body weight) intraperitoneally. In a dose determination study, alcoholic leaf extract of *S. surattense* at 100, 200 and 300 mg kg<sup>-1</sup> body weight was orally administered for 15 days and the extract at 100 mg dose significantly reduced blood glucose and also found to reduce the increased lipid peroxidation marker in diabetic rats. Extending oral administration of 100 mg kg<sup>-1</sup> bw to diabetic rats, for 45 days, resulted in a significant decrease in blood glucose and an increase in plasma insulin level. In addition, diabetic rats showed significant reduction in the glycogen content and in the activities of glucose metabolizing enzymes such as glucokinase, glucose 6-phosphate dehydrogenase and an elevation in the activities of gluconeogenic enzymes such as glucose-6-phosphatase and fructose-1, 6-bisphosphatase. *S. surattense* extract administration to diabetic rats reversed these changes in a significant manner. Thus, the results show that *S. surattense* possesses antihyperglycemic activity and provide evidence for its traditional usage in the control of diabetes.

**Key words:** *Solanum surattense*, antihyperglycemia, streptozotocin, carbohydrate enzymes, insulin, hemoglobin

### INTRODUCTION

Diabetes mellitus is a pathologic condition, resulting in severe metabolic imbalances and non-physiologic changes in many tissues. In hyperglycemia, glucose auto-oxidation generates reactive oxygen species, such as superoxide radical (O<sub>2</sub><sup>•-</sup>), hydrogen peroxide and hydroxyl radical (OH<sup>•-</sup>). These species cause chronic oxidative stress in diabetic rats thereby depleting activities of the antioxidant defense system (Kakkar *et al.*, 1997). Hyperglycemia, defining established diabetes, can induce oxidative stress by various mechanisms; excessive levels of glucose reaching the mitochondria lead to an overdrive of the electron transport chain, resulting in overproduction of superoxide anions normally scavenged by mitochondrial superoxide dismutase. When the latter fails oxidative stress develops and it was recently proposed that this mechanism is responsible for the activation of all major pathways underlying the different components of vascular diabetic complications (glycation, protein kinase C activation, sorbitol pathway) (Nishikawa *et al.*, 2000).

The plant kingdom has become a target for the search of new drugs in recent years, because they synthesize a variety of metabolites with antioxidant potential which can play a major role in protection against molecular damage induced by Reactive Oxygen Species (ROS) (Cao *et al.*, 1997; Shukla *et al.*, 2000). Many plants traditionally are used for diabetes mellitus throughout the world. Few of the

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medicinal plant treatments for diabetes have received scientific scrutiny, for which World Health Organization (WHO) has also recommended attention (WHO, 1980) and it is pertinent to investigate the traditional antidiabetic plant. Though, many oral hypoglycemic agents are available for the treatment of diabetes mellitus, these synthetic agents can produce serious side effects. Therefore, search for safe and more effective natural medicines has stimulated a new wave of research in traditional practice.

*S. surattense* (synonym *S. xanthocarpum*) commonly known as Indian night shade or yellow berried night shade, is a prickly, diffusely bright-green, perennial shrub which grows in arid areas of India. The plant has been used traditionally for curing various ailments such as respiratory diseases, gonorrhoea, rheumatism, fever, asthma and diabetes in South Indian traditional medicine (Nadkarni, 1954; Kirtikar and Basu, 1975). The antidiabetic potential of the fruit was studied in diabetic rats (Gupta *et al.*, 2005; Kar *et al.*, 2006). The objective of the present study was to investigate the antihyperglycemic effect of the alcoholic leaf extract of *S. surattense* in STZ-diabetic rats.

## MATERIALS AND METHODS

### Chemicals

Streptozotocin was obtained from Sigma-Aldrich Company (St. Louis, Missouri, USA). All other chemicals used were of analytical grade obtained from E. Merck and HIMEDIA, Mumbai, India.

### Plant Material

Leaves of *Solanum surattense* were collected from local areas of Chidambaram Tamil Nadu, India during October 2006. The plant was botanically identified and authenticated in the Department of Botany, Annamalai University, Annamalainagar, Chidambaram, Tamil Nadu, India and a voucher specimen (AU 189) was deposited at the herbarium of botany.

### Preparation of Leaf Extract

The plant leaf was shade dried at room temperature ( $30\pm 2^\circ\text{C}$ ) and the dried leaf was ground into fine powder using pulverizer. The powdered part was sieved and kept in deep freezer until the time of use. One hundred gram of dry fine powder was suspended in 400 mL of ethanol for 72 h. The extract was filtered using a muslin cloth and concentrated at  $40^\circ\text{C}$ , refrigerated and used within two months.

### Animals

Male albino Wistar rats (weighing 180-200 g) were procured from the Central Animal House, Department of Experimental Medicine, Rajah Muthiah Medical College and Hospital, Annamalai University and maintained in an air-conditioned room ( $25\pm 1^\circ\text{C}$ ) with a 12 h light/12 h dark cycle. Feed and water were provided *ad libitum*. All the experimental study were conducted in the Department of Biochemistry, Faculty of Science, Annamalai University, in accordance with the National Institute of Health Guide for the Care and Use of Laboratory Animals (NIH, 1985) and the experimental study was approved by the Ethical Committee of Rajah Muthiah Medical College and Hospital (Reg No. 160/1999/CPCSEA), Annamalai University, Annamalainagar.

### Experimental Induction of Diabetes

The animals were made diabetic by an intraperitoneal injection of streptozotocin (STZ, 40 mg  $\text{kg}^{-1}$  body weight) in a freshly prepared citrate buffer (0.1 M, pH 4.5) after an overnight fast. STZ injected animals were given 20% glucose solution for 24 h to prevent initial drug-induced

hypoglycemic mortality. The animals exhibited massive glycosuria (determined by Benedict's qualitative test) and hyperglycemia within a few days. Diabetes was confirmed by measuring the fasting blood glucose concentration, 96 h after induction. Albino rats with a blood glucose level above 220 mg dL<sup>-1</sup> were considered diabetic and were used in the experiment.

#### **Preliminary and Dose Determination Study**

- Preliminary study was carried out to assess the effect of *S. surattense* extract on blood glucose levels in diabetic rats. For each test extract preparation (100, 200 and 300 mg kg<sup>-1</sup> bw) 6 animals were used. The extract was suspended in 2% gum acacia vehicle solution and fed by intubation. Blood was collected by retroorbital puncture after 12 h fasting and 2 h after giving the extract in gum acacia.
- Short duration (15 day) study was carried out to assess the effective dose of *S. surattense* extract on blood glucose levels in STZ diabetic rats. For each test the extract preparation (100, 200 and 300 mg kg<sup>-1</sup> bw) 6 animals were used.

#### **Long term (45 day) study with effective dose of *S. surattense* leaf extract**

The animals were randomly divided into five groups of six animals each.

- Group-I: Normal rats received 2% gum acacia only
- Group-II: Normal + leaf extract (100 mg kg<sup>-1</sup> bw) in 2% gum acacia
- Group-III: Diabetic control rats (STZ-40 mg kg<sup>-1</sup> bw)
- Group-IV: Diabetic + leaf extract (100 mg kg<sup>-1</sup> bw) in 2% gum acacia
- Group-V: Diabetic + glibenclamide (600 µg kg<sup>-1</sup> bw) in 2% gum acacia

After 45 days, the animals were fasted for 12 h, anesthetized between 8:00 am to 9:00 am using ketamine (24 mg kg<sup>-1</sup> b.wt, intramuscular injection) and sacrificed by decapitation. Blood was collected in tubes with a mixture of potassium oxalate and sodium fluoride (1:3) for the estimation of blood glucose and ethylenediaminetetra acetic acid (EDTA) for the estimation of hemoglobin, glycosylated hemoglobin. Liver was immediately dissected out, washed in ice-cold saline to remove the blood. Tissues were sliced into pieces and homogenised in an appropriate buffer (pH 7.0) in cold condition to give 20% homogenate (w/v). The homogenate were centrifuged at 1000 rpm for 10 min at 0°C in cold centrifuge. The supernatants were separated and used for various biochemical estimations.

#### **Biochemical Estimations**

Glucose was estimated by the method of Trinder using a reagent kit (Trinder, 1969). Hemoglobin (Hb) and glycosylated hemoglobin (HbA<sub>1c</sub>) were estimated by the methods Drabkin and Austin (1932) and Sudhakar and Pattabiraman (1981), respectively. TBARS was estimated by the method Nieuhas and Samuelson (1968). The insulin in the rat plasma was measured by the method of Burgi *et al.* (1988). Glucokinase, glucose-6-phosphatase, fructose-1,6-bisphosphatase and glucose 6-phosphate dehydrogenase (G6PD) were assayed by the methods of Brandstrup (1957), Koide and Oda (1959), Gancedo and Gancedo (1971) and Bergmeyer (1984), respectively. Glycogen content was determined as described by Morales *et al.* (1975).

#### **Statistical Analysis**

Statistical evaluation was done using one-way analysis of variance (ANOVA) followed by Duncan's Multiple Range Test (DMRT) by using statistical package of social science (SPSS) 10.0 for Windows. Student's t-test was performed in preliminary study. Significance level was set at p<0.05.

## RESULTS

### Effect of *S. surattense* on Blood Glucose Level in Normal and STZ-induced Diabetic Rats at 2 h

The diabetic rats showed a significant increase in blood glucose level. Oral administration of *S. surattense* leaf extract to diabetic rats at three different doses (Table 1) showed a significant reduction of glucose only at 100 mg.

### Effect of 15-day Oral Administration of *S. surattense* Extract on Body Weight, Blood Glucose and TBARS Levels in Normal and STZ-diabetic Rats

A reduction in body weight was observed in STZ-induced diabetic rats and animals treated with *S. surattense* extract showed an improvement when compared with diabetic rats (Table 2). Blood glucose and TBARS levels elevated significantly in STZ-diabetic rats, oral administration with *S. surattense* extract at 100, 200 and 300 mg kg<sup>-1</sup> bw decreased blood glucose and TBARS levels.

Since, *S. surattense* extract at 100 mg dose showed effective reduction in blood glucose level, this dose was used for long term (45 days) studies.

### Effect of 45 Day Oral Administration of *S. surattense* on Body Weight, Blood Glucose Level and Liver Glycogen Content in Normal and STZ-diabetic Rats

Diabetic rats showed decreased body weight, elevated blood glucose level and decreased glycogen content (Table 3). Oral administration of *S. surattense* and glibenclamide in diabetic rats showed an improvement in body weight, decreased blood glucose level and increased glycogen content.

Table 1: Effect of *S. surattense* on plasma glucose in STZ-diabetic rats: Dose determination study

Groups	Plasma glucose 0 h (mg dL <sup>-1</sup> )	Plasma glucose 2 h (mg dL <sup>-1</sup> )	Reduction (%)
Normal	69.60±2.17	64.23±5.14*	7.71
Diabetic control	252.25±14.80	243.90±16.67 <sup>NS</sup>	3.31
Normal + <i>S. surattense</i> (300 mg kg <sup>-1</sup> bw)	68.02±2.94	61.47±1.95*	9.62
Diabetic + <i>S. surattense</i> (100 mg kg <sup>-1</sup> bw)	252.49±17.70	230.82±13.77*	8.58
Diabetic + <i>S. surattense</i> (200 mg kg <sup>-1</sup> bw)	241.95±14.92	232.03±12.21 <sup>NS</sup>	3.81
Diabetic + <i>S. surattense</i> (300 mg kg <sup>-1</sup> bw)	247.83±12.92	238.56±13.02 <sup>NS</sup>	3.74
Diabetic + glibenclamide (600 µg kg <sup>-1</sup> bw)	250.09±13.60	223.31±16.27*	10.70

Values are means±SD for six rats. Comparison between 0 h to 2 h glucose-Student's t-test \* p<0.05 NS: No Significance

Table 2: Short duration (15 day) effect *S. surattense* on body weight, blood glucose and TBARS in normal and STZ-diabetic rats

Groups	Body weight (g)		Plasma glucose (mg dL <sup>-1</sup> )		TBARS (mmole dL <sup>-1</sup> )	
	Day 0	Day 15	Day 0	Day 15	Day 0	Day 15
Normal	178.33±5.16	196.66±2.58 <sup>a</sup>	69.60±2.17	72.40±6.08 <sup>a</sup>	0.181±0.01	0.171±0.01 <sup>a</sup>
Normal + <i>S. surattense</i> (300 mg kg <sup>-1</sup> bw)	181.66±6.83	194.16±8.61 <sup>a</sup>	68.02±2.94	60.04±6.07 <sup>a</sup>	0.158±0.01	0.161±0.01 <sup>a</sup>
Diabetic control	183.33±5.16	179.16±5.84 <sup>b</sup>	252.25±14.80	270.84±10.07 <sup>b</sup>	0.240±0.02	0.300±0.03 <sup>b</sup>
Diabetic + <i>S. surattense</i> (100 mg kg <sup>-1</sup> bw)	182.50±6.09	189.16±5.1 <sup>ab</sup>	252.49±17.70	213.35±12.77 <sup>c</sup>	0.249±0.03	0.218±0.02 <sup>c</sup>
Diabetic + <i>S. surattense</i> (200 mg kg <sup>-1</sup> bw)	181.38±6.54	185.48±7.04 <sup>ab</sup>	241.95±14.92	233.53±21.29 <sup>d</sup>	0.262±0.02	0.247±0.02 <sup>d</sup>
Diabetic + <i>S. surattense</i> (300 mg kg <sup>-1</sup> bw)	185.91±5.95	188.18±6.16 <sup>b</sup>	247.83±12.92	231.26±19.09 <sup>d</sup>	0.255±0.02	0.241±0.02 <sup>d</sup>
Diabetic + glibenclamide (600 µg kg <sup>-1</sup> bw)	183.12±6.11	190.13±6.03 <sup>ab</sup>	250.09±13.60	202.08±9.62 <sup>c</sup>	0.250±0.01	0.204±0.02 <sup>c</sup>

Values are means±SD for six rats. Values not sharing a common superscript differ significantly at p<0.05 (DMRT)

Table 3: Effect of *S. surattense* leaf-extract on body weight, blood glucose and liver glycogen content in normal and STZ-diabetic rats

Groups	Body weight (g)		Glucose (mg dL <sup>-1</sup> )		Glycogen (mg/100 g tissue)
	Day 0	Day 45	Day 0	Day 45	
Normal	179.33±4.16	201.79±8.40 <sup>a</sup>	68.65±2.07	75.32±7.59 <sup>a</sup>	51.01±4.65 <sup>a</sup>
Normal+ <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	185.20±5.10	202.93±7.00 <sup>a</sup>	67.02±4.94	74.45±7.76 <sup>a</sup>	53.28±4.85 <sup>a</sup>
Diabetic control	181.33±5.56	141.70±9.92 <sup>b</sup>	250.25±15.80	291.38±19.20 <sup>b</sup>	19.27±1.75 <sup>b</sup>
Diabetes + <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	184.50±5.09	170.04±5.50 <sup>c</sup>	247.95±17.92	140.57±12.82 <sup>c</sup>	30.60±2.79 <sup>c</sup>
Diabetes + glibenclamide (600 µg kg <sup>-1</sup> bw)	187.12±6.00	192.72±7.57 <sup>a</sup>	254.09±14.60	105.22±8.68 <sup>a</sup>	45.34±4.13 <sup>d</sup>

Values are means±SD of 6 rats from each group. Values not sharing a common superscript differ significantly at p<0.05 DMRT

Table 4: Effect of (45 day) *S. surattense* leaf-extract on insulin, hemoglobin and glycosylated hemoglobin in normal and STZ-diabetic rats

Groups	Insulin (µU mL <sup>-1</sup> )	Hemoglobin (g dL <sup>-1</sup> )	Glycosylated hemoglobin (mg g <sup>-1</sup> of Hb)
Normal	14.39±1.31 <sup>a</sup>	14.76±0.78 <sup>a</sup>	0.44±0.04 <sup>a</sup>
Normal + <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	14.29±1.30 <sup>a</sup>	13.99±0.87 <sup>a</sup>	0.40±0.03 <sup>a</sup>
Diabetic control	4.53±0.41 <sup>b</sup>	7.85±0.73 <sup>b</sup>	1.03±0.09 <sup>b</sup>
Diabetes + <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	8.97±0.80 <sup>c</sup>	10.12±1.11 <sup>c</sup>	0.54±0.05 <sup>c</sup>
Diabetes + glibenclamide (600 µg kg <sup>-1</sup> bw)	13.88±1.26 <sup>a</sup>	14.18±0.85 <sup>a</sup>	0.45±0.04 <sup>a</sup>

Values are means±SD of 6 rats from each group. Values not sharing a common superscript differ significantly at p<0.05 DMRT

Table 5: Effect of *Solanum surattense* leaf-extract on carbohydrate metabolic enzymes and gluconeogenic enzymes in the liver of normal and STZ-diabetic rats

Groups	Glucokinase (U <sup>®</sup> h <sup>-1</sup> mg <sup>-1</sup> protein)	Glucose 6-phosphate dehydrogenase (U <sup>#</sup> m <sup>-1</sup> g protein)	Glucose-6-phosphatase (U <sup>§</sup> min <sup>-1</sup> mg <sup>-1</sup> protein)	Fructose 1,6-bisphosphatase (U <sup>*</sup> h <sup>-1</sup> mg <sup>-1</sup> protein)
Normal	0.253±0.013 <sup>a</sup>	4.42±0.40 <sup>a</sup>	0.139±0.010 <sup>a</sup>	0.398±0.022 <sup>a</sup>
Normal + <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	0.248±0.016 <sup>a</sup>	4.64±0.42 <sup>a</sup>	0.119±0.018 <sup>a</sup>	0.376±0.036 <sup>a</sup>
Diabetic control	0.110±0.014 <sup>b</sup>	2.38±0.21 <sup>b</sup>	0.325±0.017 <sup>b</sup>	0.630±0.039 <sup>b</sup>
Diabetes + <i>S. surattense</i> (100 mg kg <sup>-1</sup> )	0.182±0.016 <sup>c</sup>	2.94±0.26 <sup>c</sup>	0.193±0.013 <sup>c</sup>	0.496±0.044 <sup>c</sup>
Diabetes + glibenclamide (600 µg kg <sup>-1</sup> bw)	0.225±0.012 <sup>d</sup>	3.85±0.35 <sup>d</sup>	0.141±0.014 <sup>d</sup>	0.446±0.041 <sup>a,d</sup>

U<sup>®</sup>-µ mole of glucose phosphorylated per hour. U<sup>#</sup>glucose 6-phosphate dehydrogenase is given in units of × 10<sup>4</sup> IU/mg of protein, U<sup>§</sup>-µmol of inorganic phosphate liberated/min/mg of protein. U<sup>\*</sup>-µmol of inorganic phosphate liberated/hour/mg of protein. Values are means±SD of 6 rats from each group. Values not sharing a common superscript differ significantly at p<0.05 DMRT

#### Effect of *S. surattense* on the Levels of Plasma Insulin, Hb and HbA<sub>1c</sub>

Plasma insulin and Hb decreased and HbA<sub>1c</sub> increased significantly in diabetic control rats (Table 4). Administration of *S. surattense* and glibenclamide in diabetic rats increased significantly plasma insulin and Hb and decreased HbA<sub>1c</sub>.

#### Effect of Oral Administration of *S. surattense* on Carbohydrate Metabolic Enzymes and Gluconeogenic Enzymes in the Liver of Normal and STZ-diabetic Rats

Diabetic rats showed decreased activities of glucokinase, glucose 6-phosphate dehydrogenase and increased activities glucose-6-phosphatase, fructose-1,6-bisphosphatase in the liver (Table 5). Administration of *S. surattense* and glibenclamide reversed these enzyme activities towards normalcy.

## DISCUSSION

The fundamental mechanism underlying hyperglycemia involves over production (excessive hepatic glycogenolysis and gluconeogenesis) and decreased utilization of glucose by the tissues. Maintaining the blood glucose concentrations within a narrow range is a critical physiological function which involves a dynamic equilibrium between endogenous glucose production and glucose utilization, requiring a series of cellular metabolic events, including a prominent role for hepatocytes (Klover and Mooney, 2004). The prevalence of diabetes is rapidly increasing and no satisfactory/effective therapy is available for its cure. In recent years, the use of medicinal plants is increasing due to the extraction and development of several successful drugs from plant as well as their use as traditional rural herbal remedies (Tiwari and Madhusudhana Rao, 2002).

STZ-induced diabetes is characterized by severe weight loss (Al-Shamaony *et al.*, 1994) which was observed in the present study. *S. surattense* and glibenclamide administration controlled the body weight loss in diabetic animals, though *S. surattense* did not normalize the body weight completely. The decrease in body weight in diabetic rats might be the result of protein wasting due to unavailability of carbohydrate as an energy source (Chen and Ianuzzo, 1982). The treated groups enhanced glucose metabolism and thus, improved the body weight in STZ-diabetic rats. The treatment with *S. surattense* at 100 mg dose for 45 days showed significant antihyperglycemic activity. Further, the antihyperglycemic activity was associated with increase in plasma insulin. Though the exact mechanism of action of the extract is not known, it could be due to increased pancreatic secretion of insulin from existing  $\beta$ -cells. A number of other plants have been reported to have antihyperglycemic and insulin release-stimulatory effects (Latha and Pari, 2004; Prakasam *et al.*, 2002; Kar *et al.*, 2006). The phytochemical screening showed the presence of alkaloids, flavonoids, tannins, glycosides, triterpenoids and sterols (Rahman *et al.*, 2003). The presence of flavonoids in the plant may exert its antihyperglycemic effect, as they are well known for antioxidant and antidiabetic properties (Vessal *et al.*, 2003; Coskum *et al.*, 2005; Abdel Nasser *et al.*, 2005). Glycogen is the primary intracellular storable form of glucose. Diabetes mellitus is known to impair the normal capacity of the liver to synthesize glycogen. The liver glycogen content is markedly decreased in diabetic animals (Bollen *et al.*, 1998) which are in proportion to insulin deficiency (Stalmans *et al.*, 1997). Diabetic rats treated with *S. surattense* brought back liver glycogen to near normal level, which could be due to increased secretion of insulin. During diabetes the excess of glucose present in blood, non-enzymatically react with hemoglobin to form glycosylated hemoglobin (Sheela and Augusti, 1992) and the extent of increase was directly proportional to fasting blood glucose levels (Al-Yassin *et al.*, 1981). Total hemoglobin decreased in diabetic rats, possibly due to the increased formation of HbA<sub>1c</sub>. The increase in hemoglobin and decrease in glycosylated Hb levels in animals receiving *S. surattense* is due to the decreased blood glucose levels.

The liver is an important organ that plays a pivotal role in glycolysis and gluconeogenesis. A partial or total deficiency of insulin causes derangement in carbohydrate metabolism that decreases activity of several key enzymes including glucokinase, phosphofructokinase and pyruvate kinase (Hikino *et al.*, 1989), resulting in impaired peripheral glucose utilization and augmented hepatic glucose production. In the present study, glucokinase activity was decreased in the liver of diabetic rats which may be due to a deficiency of insulin and treatment with *S. surattense* and glibenclamide elevated the activity of glucokinase. *S. surattense* administration increased insulin level which, in turn, activate glucokinase, thereby increasing the utilization of glucose leading to decreased blood sugar levels. Glucose-6-phosphatase and fructose-1,6-bisphosphatase, are the regulatory enzymes in gluconeogenic pathway. Activities of these enzymes were increased significantly in diabetic rats (Baquer *et al.*, 1998),

which may be due to the activation or increased synthesis of the enzymes contributing to the increased glucose production during diabetes by the liver. Treatment with *S. surattense* and glibenclamide decreased the activity of gluconeogenic enzymes significantly, which may be due to decreased blood sugar levels. This result was correlated with an earlier report of decreased gluconeogenic enzyme activity in experimental diabetic rats (Prakasam *et al.*, 2002). A decrease in the activity of glucose -6-phosphate dehydrogenase has been observed in diabetic rats (Pannerselvam and Govindasamy, 2002). Treatment with *S. surattense* increased the activity of the enzyme, via increased secretion of insulin which increases the influx of glucose into pentose monophosphate shunt in an attempt to reduce high blood glucose levels. This results in an increased production of the reducing agent, NADPH, with concomitant decrease in oxidative stress (Ugochukwu and Babady, 2002).

Thus, present results shows a sequential metabolic correlation between increased glycolysis and decreased glycogenolysis stimulated by *S. surattense* suggests the possible biochemical mechanism is via insulin secretion, through which glucose homeostasis are regulated.

### CONCLUSION

The ethanolic extract of *S. surattense* exhibits a significant antihyperglycaemic activity which may be due to the presence of phytochemicals like flavonoids, triterpenoids and sterols that is present in the leaf. Further studies are to be carried out to isolate the active constituent (s) responsible for the antidiabetic effect of *S. surattense*.

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