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Studies on Antidiabetic Activity of *Atylosia Albicans* in *Streptozotocin-Induced Diabetic Rats*

Sharvana bhava B S[1], Umasankar K[1], Kottai Muthu A[*2]

[1]Department of Pharmacology, Vaagdevi College of Pharmacy, Kakatiya University, Warangal, Telangana, India
[2]Department of Pharmacy, Faculty of Engineering & Technology, Annamalai University, Annamalai Nagar, Chidambaram, Tamilnadu, India

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ABSTRACT

The present study was aimed to investigate the Antidiabetic effect of *Atylosia Albicans* extracts in streptozotocin-induced diabetes in Wistar rats. Rats have fasted overnight, and the plant extracts at doses 100 and 200 mg/kg were administered for 14 days. Streptozotocin was used to induce diabetes mellitus. The standard hypoglycaemic agent selected for this work was Glibenclamide (10 mg/kg). The different test solutions at doses 100 and 200 mg/kg of petroleum ether, ethyl acetate and methanolic extracts respectively were given a respective group of rats. The anti-diabetic potential was assessed by determining oral glucose tolerance, fasting blood glucose, Blood glucose levels, glycated hemoglobin (HbA_1c), total cholesterol (TC), triglycerides (TG), high-density lipoproteins (HDL), low-density lipoproteins (LDL) and very-low-density lipoproteins (VLDL) were evaluated parameters. The methanolic extract with 200 mg/kg was more significant in anti-diabetic activity ($P<0.001$) on the 14^{th} day and also significantly decreased TG, TC, VLDL-C, LDL-C and HbA1c ($P<0.001$) when compared to diabetic control rats. From the obtained results, it can be revealed that the methanolic extract of *Atylosia Albicans* has great potent antidiabetic activity. The results suggest that *Triumfetta rotundifolia* has anti-diabetic activity, thereby justifying its traditional use.

*Corresponding Author

Name: Kottai Muthu A
Phone: 9443171712
Email: akottaimuthu@gmail.com

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INTRODUCTION

Type 2 diabetes mellitus (T_2DM), characterized by increased blood glucose levels and abnormality in carbohydrate metabolism, the leading cause of mortality globally, and a great economic burden (Upadhyay *et al.*, 2018). International Diabetes Federation (IDF) depicts nearly about 382 million patients who suffered from T_2DM in 2013 and it is expected to increase two folds by 2035 (Guariguata *et al.*, 2014). T_2DM developed because of insulin resistance and pancreatic dysfunction leads to increased blood glucose levels (Lacroix and Li-Chan, 2014). T_2DM and obesity can be correlated to insulin resistance and pancreatic β-cell apoptosis (Hossain *et al.*, 2016). The post-lunch blood sugar levels play a vital role in the onset and mounting complications of T_2DM (Chang *et al.*, 2004). Inhibition of α-amylase and α-glucosidase is one of the targets in the treatment strategies for managing post-lunch blood sugar (Bhandari *et al.*, 2008). The increased

blood sugar levels can also lead to increased non-enzymatic glycation of proteins and advanced glycation end products (AGE's) formation. The glycation modifications can damage T_2DM condition by progressing to microvascular and macrovascular complications (Vinson and Howard, 1996).

Many works proved that drugs from plant origin have great potential pharmacological benefits in managing T_2DM and its complications and also inhibit α-glucosidase and α-amylase (Rupasinghe *et al.*, 2017). The best choice for treating T_2DM is phytochemicals, which help in maintaining low blood sugar levels and enhance the production of antioxidants and insulin regulation (Patel *et al.*, 2012).

Imbalance of the body's defense mechanisms and generation of free radicals cause oxidative stress. Free radicals in the human body originate due to the most common exogenous factors like pollutants, smoking, alcohol, malnutrition, solar radiation, fertilizers, pesticides, drug side effects and great physical or psychological stress (Núñez-Sellés, 2005). Oxidative stress is also one of the etiological factors of diabetes and in the process of cancer, cardiovascular problems, hepatic failure, inflammation and senescence (Marx, 1987).

In India, the genus Atylosia, a member of *Papilionacea*e, holds many species abundant in the broadleaf evergreen forest areas of the Western Ghats and Malabar coast (Pundir and Singh, 1987). The species of Atylosia, a forage legume, by and large, are hardy perennial (Ariyanayagam and Spence, 1978), disease-resistant (Remanandan, 1980), and possess high protein content (Reddy *et al.*, 1979). The main purpose of this research work was to identify the *antidiabetic* potency of *Atylosia Albicans* aerial parts.

MATERIALS AND METHODS

Chemicals

A gift sample of Glibenclamide was acquired from Suzikem Drugs private limited, Hyderabad, Telangana, India. Streptozotocin was purchased from Sigma Aldrich (Germany). Total cholesterol and HDL kit, triglycerides kit and other chemicals purchased were of analytical grade.

Plant material

The aerial parts of *Atylosia Albicans* were collected during the rainy season in regions of Kerala. Dr.V.Chelladurai, Research Officer-Botany (Scientist-C) Central Council for Research in Ayurveda & Siddha, AYUSH, Govt. of India, authenticated the collected plant material.

Preparation of extract

The collected aerial parts were cleaned with distilled water and kept aside for drying (SatheeshKumar *et al.*, 2011). The dried material was weighed and powdered. Unit Quantity of powdered was packed in a muslin cloth and Soxhlation was performed with petroleum ether, ethyl acetate and methanol as solvents respectively according to the increasing order of polarity. Each extract was concentrated to dryness under reduced pressure at 40 °C using a rotary evaporator (SatheeshKumar *et al.*, 2010).

Animals

Wistar albino rats of either sex(150-200 g) were selected and purchased from Mahaveer Enterprises, Hyderabad. They were housed in rat cages and kept in standard conditions (12h light and 12h dark cycle, 25±2 °C, 35–60% relative humidity). They were fed with standard chow diet and water *ad libitum*. The study protocol was submitted and got approved by the Institutional Animal Ethical Committee (IAEC), Vaagdevi College of Pharmacy, Hanamkonda, Warangal, T.S.

Acute Toxicity

OECD Guideline No. 420 was followed for acute toxicity study using male Wistar rats (150-200 g). After the sighting study, the plant extract 2000 mg/ kg body weight was given to five animals. The animals were constantly looked for 14 days for mortality and gross behavior. No change in behavior and death were observed until the end of the study (OECD, 2001).

Oral glucose tolerance

Animals fasted overnight and oral glucose tolerance was performed. Eight groups of rats of six in each group were made. The control group rats received 1% sodium CMC (group-I), standard (group-II) treated with Glibenclamide (10 mg/kg). The different test solutions at doses 100 and 200 mg/kg of petroleum ether, ethyl acetate and methanolic extracts respectively were given to group-III to VIII. Blood samples were collected at 0 min before the glucose load and 30, 60 and 120 min after the glucose load by retro-orbital plexus puncture under mild ether anesthesia and blood glucose levels were estimated by glucometer (Nayak *et al.*, 2012).

Hypoglycemic activity

Overnight fasted Wistar rats were randomly divided into eight groups of six each. The control received 1% sodium CMC (group-I), standard (group-II) treated with Glibenclamide (10 mg/kg). The different test solutions were administered with 100 and

200 mg/kg doses of petroleum ether, ethyl acetate and methanolic extracts, respectively (group-III to VIII). Blood glucose levels were estimated after 0, 2, 4, and 6hrs of administration of extracts (Nayak *et al.*, 2012).

Induction of Diabetes by Streptozotocin

A single intraperitoneal injection of streptozotocin (STZ) at a dose of 60 mg/kg dissolved in suitable citrate buffer pH 4.5 was used to induce diabetes in selected and overnight fasted animals. The induced rats received 2% glucose solution for 24 hours, 5 hours after STZ injection, to avoid early drug-induced hypoglycemic deaths. The development of diabetes was verified after 72 hours of injection by determining the blood sugar levels. The animals having a blood sugar level above 250 mg/dl thought to be diabetes-induced ones and selected for the study (Nakhaee *et al.*, 2009).

Acute Study on Streptozotocin-induced Diabetic Rats

The animals were grouped into nine groups, each containing six animals and treated as summarized below,

SET 1- Normal control, 1% sodium CMC treated rats

SET II- Diabetic controls

SET III- Diabetic+glibenclamide (10 mg/kg, p.o)

SET IV- Diabetic+PEAA(100 mg/kg)

SET V- Diabetic+PEAA(200 mg/kg)

SET VI- Diabetic+EAAA(100 mg/kg)

SET VII- Diabetic+EAAA(200 mg/kg)

SET VIII- Diabetic+MAAA(100 mg/kg)

SET IX- Diabetic+MAAA(200 mg/kg)

The blood samples were withdrawn by retro-orbital plexus puncture at zero, second, fourth, sixth, and eighth hours of the treatment. The blood sugars using a glucometer were determined (Jangir and Jain, 2017).

Sub-acute Study

Nine sets of diabetic animals were made in a similar way as the acute study in this method.A single dose was administered in the morning, and the administration continued for 14 days. During this procedure, fasting blood sugar levels were checked on zero, seventh, and fourteenth days of the experiment using a glucometer (Kumavat *et al.*, 2012; Nayak *et al.*, 2012).

Estimation of lipid profile

Blood samples were collected from the test, standard and solventtreated groups, including normal animal as a reference after the 14^{th} day of treatment with the test fractions by sacrificing the rats by decapitation under ether anesthesia. Lipid profile studies such as TC, TG and High-density lipoprotein (Sepulveda, 2013) were determined by using the serum supernatant, which was separated by centrifugation and low-density lipoprotein and Very-low-density lipoprotein was calculated by Friedewald's equation (Friedewald *et al.*, 1972) described below,

VLDL - TG/5.

LDL - TC–(HDL+VLDL)

Estimation of Glycosylated Hemoglobin (HbA1c)

HbA1C was estimated by the laboratory method (ion exchange resin method) using the autoanalyzer.

Statistical Analysis

Results were articulated as Mean±SD and by applying One Way Analysis of Variance (ANOVA), statistical significance was calculated. $P<0.05$ was considered significant (Dunnett's test).

RESULTS AND DISCUSSION

Acute Oral Toxicity Study

Atylosia Albicans at a dose of 2 g/kg body weight, death of any animal was not observed in acute toxicity studies. From the results, test drugs at 100 and 200 mg/kg doses were chosen for efficacy studies.

Oral Glucose Tolerance Test(OGTT)

Significant changes in blood glucose levels were observed after glucose loading. Both extracts EAAA and MAAA 100 and 200 mg/kg doses reduced the blood sugar levels at 30min initially declined, followed by up to 120min. The extracts and glibenclamide significantly ($P<0.05$) decreased blood glucose levels (Table 1).

Hypoglycaemic Effect

The MAAA and EAAA at 200 mg/kg decreased the blood sugar level significantly ($P<0.05$) after 4 and 6 h of treatment when compared with glibenclamide ($P<0.01$) and 100 mg/kg dose also reduced the blood-glucose-lowering effect significantly ($P<0.05$) (Table 2).

Acute Antidiabetic Activity

EAAA and MAAA extracts showed dose-dependent reductions in blood glucose levels after 2 h of treatment, and the effect persisted up to 4 h (Table 3). The MAAA shows significant ($P<0.01$, $P<0.001$) reduction (33.39,41.36%) in blood glucose levels by 100 and 200 mg/kg respectively and EAAA

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Table 1: Oral Glucose Tolerance Test of Atylosia Albicans aerial part extracts in Normal Rats

Group	Dose (mg/kg)	Blood glucose (mg/dl)			
		0 min	30 min	60 min	120 min
Normal	0.1% Na CMC	89.45±2.95	97.12±3.90	107.02±4.52	94.72±3.50
Gliben-clamide	10	86.76±4.02	93.60±2.95 (3.62)	100.42±3.06* (6.16)	90.12±3.99* (4.85)
PEAA	100	88.22±3.56	96.96±3.94 (0.16)	106.39±2.89 (0.58)	93.65±3.62 (1.12)
	200	84.31±3.68	95.92±2.69 (1.23)	105.68±2.76 (1.25)	91.84±2.96 (3.04)
EAAA	100	86.02±3.39	95.33±2.34 (1.84)	103.32±3.48* (3.45)	91.98±3.09 (2.89)
	200	83.49±3.90	93.96±2.14 (3.25)	102.59±3.03* (4.13)	91.12±2.84 (3.80)
MAAA	100	87.62±2.86	94.80±3.22 (2.38)	102.42±2.10* (4.29)	91.36±2.92 (3.54)
	200	85.32±4.20	93.87±3.06 (3.34)	101.35±4.12* (5.29)	91.35±1.89 (3.55)

Data represents mean ± S.D. (n=6). *** P <0.001, ** P < 0.01, * P < 0.05, Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

Table 2: Hypoglycaemic activity of Atylosia Albicans aerial part extracts in Normal Rats

Group	Dose (mg/kg)	Blood glucose (mg/dl)			
		0 hrs	2 hrs	4 hrs	6 hrs
Normal	0.1% Na CMC	82.23±3.99	88.62±5.06	85.72±4.16	85.22±2.19
Glibenclamide	10	81.29±3.63	84.12±3.62* (5.02)	76.39±2.98** (10.88)	75.39±2.608** (11.53)
PEAA	100	81.36±4.86	88.23±3.68 (0.44)	84.26±3.56 (1.70)	84.12±3.10 (1.29)
	200	80.06±4.53	87.96±3.33 (0.74)	83.54±4.70 (2.54)	83.60±3.19 (1.90)
EAAA	100	81.20±4.01	88.43±3.29 (0.21)	83.29±3.49 (2.83)	82.99±2.69 (2.61)
	200	81.10±4.12	87.33±4.02 (1.45)	82.32±4.62* (3.96)	81.35±3.16* (4.54)
MAAA	100	81.10±3.98	87.96±3.26 (0.74)	82.69±3.50 (3.53)	80.92±2.86* (5.04)
	200	81.02±4.56	86.12±3.90 (2.82)	81.03±4.32 (5.47)*	79.32±3.72* (6.92)

Data represents mean ± S.D. (n=6). *** P < 0.001, ** P < 0.01, * P < 0.05,Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

Table 3: Acute anti-diabetic activity of Atylosia Albicans aerial part extracts in STZ induced Model

Group	Dose (mg/kg	Blood glucose (mg/dl)				
		0 hrs	2 hrs	4 hrs	6 hrs	8 hrs
Normal	0.1% Na CMC	83.12±3.69	83.16±4.32	83.29±4.63	85.19±5.23	80.59±4.32
Dia. Control	–	279.49±4.8	279.29±4.59	281.59±4.06	238.03±4.29	275.32±4.53
Glibenclamide	10	260.42±4.25	225.79±4.03* (19.15)	144.59±3.80*** (48.65)	221.12±3.99* (7.10)	269.36±3.59 (2.16)
PEAA	100	276.59±4.01	272.56±4.98 NS (2.40)	276.73±3.62 (1.72)	233.17±3.03 NS (2.04)	274.73±3.99 NS (0.21)
	200	271.63±4.34	269.86±3.62 NS (3.47)	270.97±4.32 (3.77)	230.86±4.29 NS (3.01)	273.59±3.63 NS (0.62)
EAAA	100	270.32 ±4.56	268.73±4.54 NS (3.78)	234.23±4.62* (16.81)	235.55±3.81 NS (1.04)	271.71±3.93 NS (1.31)
	200	269.12±3.92	265.83±3.06 NS (4.81)	220.45±4.53** (21.71)	231.39±3.60 NS (2.78)	270.59±4.52 NS (1.71)
MAAA	100	272.45±5.13	250.79±4.73 NS (10.20)	187.56±3.71** (33.39)	230.69±4.29 NS (3.08)	272.23±3.99 NS (1.12)
	200	267.39±3.98	246.66±3.76 NS (13.22)	165.12±3.23*** (41.36)	227.13±4.01* (4.57)	270.32±4.19 NS (1.53)

Data represents mean ± S.D. (n=6). *** P < 0.001, ** P < 0.01, * P < 0.05,Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

Table 4: Subacute anti-diabetic activity of Atylosia Albicans aerial part extracts in STZ induced Model

GroupDose (mg/kg)		Blood glucose (mg/dl)		
		0 day	7^{th} day	14^{th} day
Normal	0.1% Na CMC	84.39±5.61	78.63±4.98	76.53±4.58
Dia. Control	–	273.38±3.92	289.38±4.32	278.43±4.83
Glibenclamide	10	270.23±4.68	150.63±4.98*** (47.94)	110.42±3.81*** (60.34)
PEAA	100	269.63±3.69	257.06±4.32 (11.16)	240.39±4.68 (13.66)
	200	270.73±4.60	250.90±4.86 (13.29)	233.54±3.98 (16.12)
EAAA	100	272.38±4.93	245.12±4.83* (15.29)	220.58±4.89* (20.77)
	200	273.32±4.91	240.38±4.81* (16.93)	196.53±4.90* (29.41)
MAAA	100	272.37±4.63	213.38±4.63** (26.26)	190.26±4.93** (31.66)
	200	270.35±4.35	190.31±4.83*** (34.23)	150.34±4.98*** (46.00)

Data represents mean ± S.D. (n=6). *** P < 0.001, ** P < 0.01, * P < 0.05,Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

Table 5: Estimation of Lipid profile level AtylosiaAlbicans aerial part extracts in STZ induced model on 14 th day

Group	Dose (mg/kg)	Lipid profile(mg/dl)				
		TG	TC	HDL	LDL	VLDL
Normal	0.1% Na CMC	82.70±4.87	73.43±4.10	32.28±0.69	24.61±1.40	16.54±0.93
Dia. Control	–	188.26±5.23	173.53±5.30	12.53±0.79	123.35±1.62	37.65±0.81
Gliben-clamide	10	98.18±2.86***	89.28±3.59***	28.90±0.99***	40.75±1.84***	19.63±0.62***
PEAA	100	186.52±4.67	170.98±4.68	13.43±1.23	120.25±1.93	37.30±0.68
	200	184.24±4.73	168.78±3.99	12.99±0.63	118.95±1.32	36.84±0.32
EAAA	100	170.23±5.13*	164.78±3.86*	19.88±0.72*	110.86±1.87*	34.04±0.38*
	200	167.42±4.89**	161.48±4.60**	21.45±1.93**	106.55±1.68*	33.48±0.81*
MAAA	100	128.21±3.98**	113.79±3.23**	20.83±0.88**	67.32±1.60**	25.64±0.63**
	200	102.32±3.69***	110.68±4.78***	26.32±1.03***	63.90±1.56***	20.46±0.72***

Data represents mean ± S.D. (n=6). *** P < 0.001, ** P < 0.01, * P < 0.05,Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

Table 6: Estimation of HbA1c levels of Atylosia Albicans aerial part extracts in STZ induced model on 14 th day

Group	Dose (mg/kg)	HbA1c
Normal	0.1% Na CMC	5.12±1.23
Dia. Control	–	11.23±1.21
Glibenclamide	10	5.06±1.93***
PEAA	100	9.94±1.76
	200	8.82±1.54
EAAA	100	7.89±1.67*
	200	7.11±1.16**
MAAA	100	7.04±1.24*
	200	6.05±1.69**

Data represents mean ± S.D. (n=6). *** P < 0.001, ** P < 0.01, * P < 0.05, Significant compared to diabetic control analyzed by One-way ANOVA followed by Dunnett's test

also produced significant (P<0.05, P<0.01) reduction(16.81,21.71%) respectively when compared with that of 10 mg/kg of glibenclamide which produced maximum reduction of 48.65 after 4h. The PEAA extracts do not reduce blood glucose levels.

Sub-Acute Antidiabetic Activity

On day 14, MAAA treated rats showed more significant (P <0.01, P<0.001) decline (31.66 and 46.00%) in blood glucose at 100 and 200 mg/kg respectively when compared with 10 mg/kg of glibenclamide (P<0.001, 60.34%), EAAA also significantly (P <0.01) reduced (25.26, 34.68 %) but PEAA significantly did not altered the sugar levels. Dose-dependent reduction in blood sugar level at 7 days by treatment with MAAA and EAAA extracts but less significant reduction in blood glucose level was observed when compared with day 14 (Table 4).

Lipid profile

The serum lipid levels such as TG, TC, HDL, LDL and VLDL were measured in all groups of rats as depicted (Table 5). Serum levels of TG, TC, VLDL and LDL were increased, and HDL decreased in diabetic induced rats compared to that of the control rats. Treatment with MAAA and EAAA decreased elevated TG, TC, VLDL, LDL and HDL level restored, and glibenclamide reduced elevated TG, TC, VLDL, LDL significantly **(**P<0.001) respectively compared to disease control.

Glycosylated Hemoglobin

The glycosylated hemoglobin levels were increased in diabetic rats when compared with normal rats. HbA_1c levels by 200 mg/kg of EAAA and MAAA were decreased significantly (P<0.001) and 100 mg/kg also decreased significantly (P<0.01), but PEAA did not influence significantly compared to control (Table 6).

Around the globe, T2DM is one of the foremost endocrine diseases. Management of diabetes is a tough task with the synthetic drugs as they have many side effects. Day by day, the medicinal plant's importance has been increasing for remedy or reducing the risk of complications associated with it. In the current scenario, scientists have emphasized the herbal extracts and initiated wide research to observe their effectiveness and protective role in the diabetic animal models (Mondal *et al.*, 2016).

The extracts of *Atylosia Albicans* how significant anti-hyperglycemic activities in STZ-induced hyperglycemic by reducing the condition of diabetes mellitus, as indicated by parameters such as blood glucose, hemoglobinA_1C, lipid profile, antioxidants activities. Diabetes induced by STZ is the best widely accepted experimental design for screening antidiabetic drugs. T2DM occurs due to damage to the β-islet cells of the pancreas irreversibly by STZ, leading to decreased insulin emission. The pathological mechanisms of STZ induced T2DM are the generation of reactive oxygen species (ROS) followed by local oxidative stress, DNA methylation, and protein modifications (Szkudelski, 2001; Venkateshwarlu, 2014).

The basic underlying mechanism of increased sugar levels in T2DMis excessive generation and less usage of sugars by the cells. The routinely used marker for long term glycemic control is hemoglobin A_1c. Persistent hyperglycemia in diabetes manifests as an increased HbA_1C level as a result of glycation of hemoglobin. The increased level of HbA1C in diabetic patients was reported by up to 16% (Koenig *et al.*, 1976). The increased levels of HbA_1C well correlate with the complication such as diabetic retinopathy, nephropathy, and neuropathy. In addition, there is a relative insufficiency of insulin, leading to decreased protein synthesis in all tissues. As a result, the reduction of the synthesis of hemoglobin in diabetes (Prabhu *et al.*, 2008).

In the pathogenesis of diabetes mellitus, lipids also play a very crucial role. Diabetes is linked with thoughtful changes in serum lipid profile and an improved heart disease risk (Maghrani *et al.*, 2004). Hypertriglyceridemia and hypercholesterolemia are the most regular lipid abnormalities in diabetes. The enhanced lipid levels in diabetic animals might be due to the activation of lipoprotein lipase by insulin and hydrolysis of triglycerides. Raised movement of fatty acids into adipocytes and synthesis of triglycerides by insulin also inhibits lipolysis. If any insulin deficiency, hyperlipidemia is promoted because of no inhibition of lipolysis. InT_2DM state, the concentration of the serum-free acid is increased as a result of fat deposited, where the equilibrium of the free fatty acid esterification-triglyceride lipolysis cycle is displaced in support of lipolysis (Shirwaikar *et al.*, 2004). The lipoprotein pathway enhances the movement of triglycerides synthesized in the liver to muscle and adipose tissue. In addition, it also provides a pathway for the carrying of cholesterol from the liver to peripheral tissues, and coronary heart disease may be surmounted.

For the anticipation of atherosclerosis and ischemic diseases by a considerable improvement in the lipid profile is the most desired condition (Ahotupa, 2017). The administration of plant extracts showed a significant reduction in serum levels of TC, LDL, TG and VLDL, whereas a significant elevation in HDL levels (Venkateshwarlu *et al.*, 2013). Most research

works proved that increased blood sugar level is a potent risk factor for microvascular and macrovascular complications by excessive generation of free radicals (Hunt *et al.*, 1988).

CONCLUSION

Atylosia Albicans has little hypoglycaemic and potent anti-diabetic activities. This research work suggests that *Atylosia Albicans* can be used in the treatment of T2DM alone. It is further suggested that it can also be used as a supportive drug with other Ayurvedic drugs. Further evaluations must be carried out for bringing out the best formulations of it.

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