

**CHAPTER – VI**

***Costus pictus* D. Don**

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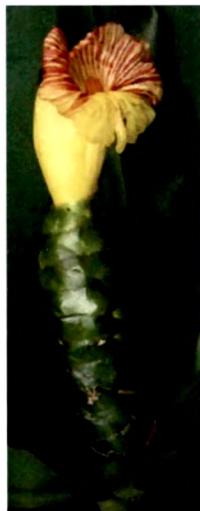
*Costus pictus* D. Don  
Plate No: 6



Habit



Stem



Flower

### ***Costus pictus*. D. Don**

**Family:** Costaceae

**Synonyms:** *Costus mexicanus* Liebm ex Petersen, *Costus congenitus* Rowle.

*Costus pictus*, an ornamental plant of Mexico, is newly introduced to India. It is an erect herb growing up to 2.7 meters tall, clumping. Stem horizontally striped at base; upper vegetative stem medium green with 0.5 cm long hairs at nodes with leaves; leaves narrowly lanceolate, dark green above, lighter green below; small leaves are present on the basal part; bracts green, with outer margin coloured maroon. Flowers yellow; lip with maroon striations, darker yellow stripe down the middle region; anther cream coloured.

This plant is distributed along the coast from Mexico to Costa Rica and is locally known as *cana agria* or *cana de jabali* in Mexico. In Mexico, it is used to treat diseases of the kidney (Martinez, 1996; Rzedowski and Rzedowski, 1976; Caceres *et al.*, 1987; Argueta *et al.*, 1994). It is reported to have effects on renal functions and its anti-inflammatory and hypoglycemic actions (Martinez, 1996; Maria, 2004). The practitioners in Mexico used an infusion of this plant in the treatment of renal disorders (Martinez, 1996; Argueta *et al.*, 1994), the plant also possesses diuretic activity (Comargo *et al.*, 2006).

The plant was reported to contain flavonoids, saponins, reduced sugars and tannins (Comargo *et al.*, 2006).

The present study was undertaken to test the plant for its antidiabetic activities, toxicity of the extract on normal rats and the effect of the extract on the antioxidant enzymes, non-enzymatic antioxidant, carbohydrate metabolizing enzymes and lipid profile. Pharmacognostic and phytochemical analysis of the leaves were also conducted.

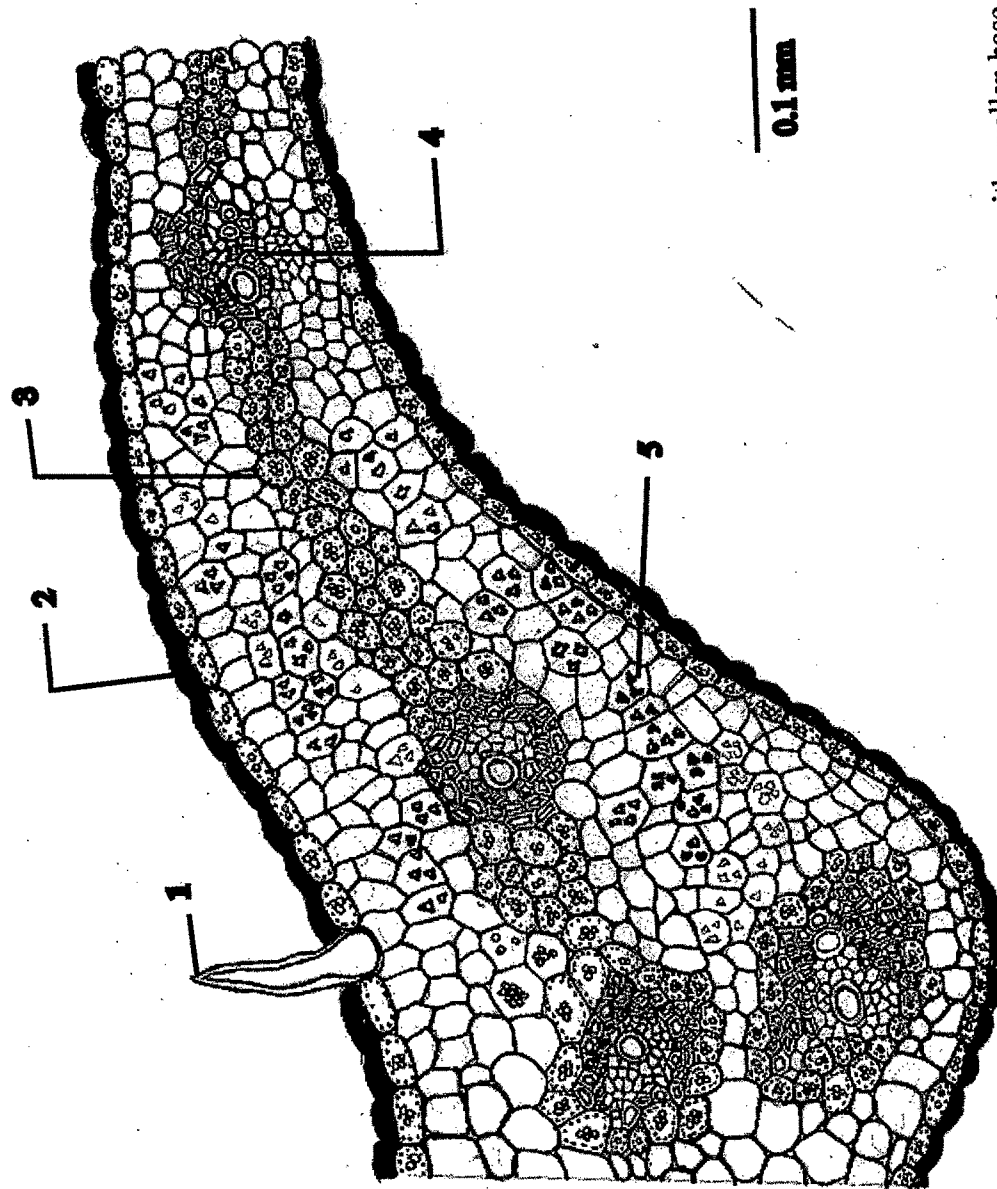
## **Results:**

### **Pharmacognosy:**

The leaves are amphistomatic bearing paracytic stomata (Fig. 2). Leaf constants such as stomatal index/mm<sup>2</sup> was 10.4 on the lower epidermis and 1.9 on the upper epidermis, stomatal frequency/mm<sup>2</sup> was 3.4 on the lower epidermis and 0.8 on the upper epidermis, vein islet number/mm<sup>2</sup> was 4 and vein termination number/mm<sup>2</sup> was 3.9 respectively.

In the T. S of leaf, epidermis contains barrel shaped cells, containing starch grains. Below the upper epidermis and above the lower epidermis there were two to three layers of parenchymatous hypodermis in between the two regions of parenchyma, were the mesophyll contain only spongy tissues and palisade layer was absent. The mesophyll was closely packed cells containing starch grains. On the upper epidermis simple unicellular, pointed non-glandular trichomes were present. The vascular bundle was encircled with sclerenchyma. The vein fibers are like shattered crystal pieces in structure present mostly in parenchyma cells (Fig. 1). The size of the cells were upper epidermal cells 40.3x16.5 µm; lower epidermal cells 31.6x16.5 µm; parenchyma cells 122x102 µm; sclerides 13.8x17.2 µm; xylem vessels 34.3x54.12 µm; trichomes 83.2x13.2 µm; stomata on the lower epidermal layer 34.9x23.1 µm; stomata on the upper epidermal layer 35.6x23.76 µm.

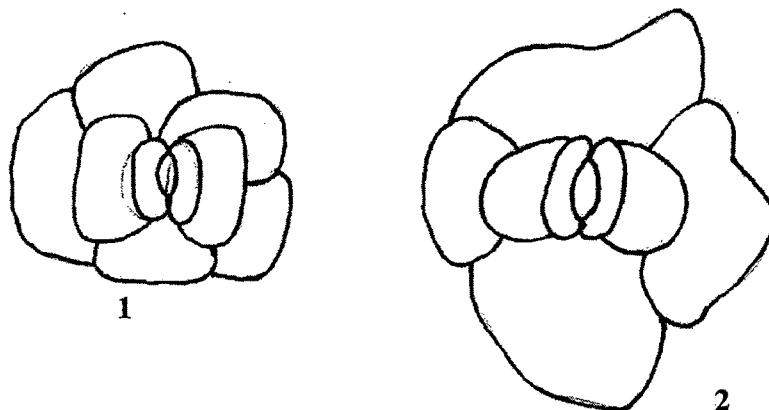
The powder analysis of the leaf showed fragments of trichomes, paracytic stomata, starch grains, mesophyll cells containing starch grains and fiber pieces (Fig 3).



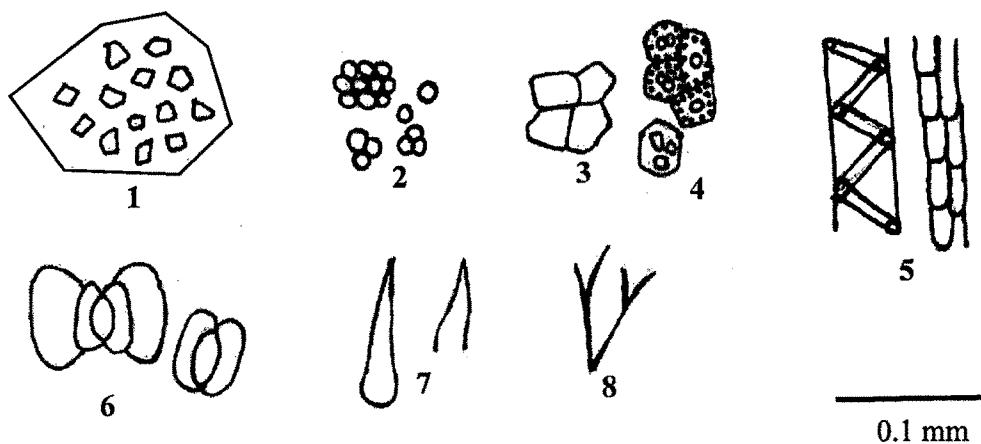
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Fig. 1 T. S of leaf *C. pictus*: 1. Unicellular trichome with swollen base,  
 2. Epidermis containing starch grains, 3. Starch grains,  
 4. Vascular bundle,  
 5. Vein fibers appearing like shattered crystal pieces

**Fig. 2**



**Fig. 3**



**Fig.2 *Costus pictus*:** Epidermal layer: 1. Stomata of upper epidermis, 2. Stomata of lower epidermis

**Fig. 3 Powder character:** 1. Fibres appearing like shattered crystal pieces, 2. Starch grains, 3. Parenchyma, 4. Chlorenchyma with starch grains, 5. Tracheids with spiral thickenings and epidermal cells, 6. Stomata 7. Fragments of trichomes, 8. Fibre pieces.

### Phytochemistry:

Kaempferol, 3', 4'-di OMe-quercetin and 4'-OMe-Kaempferol were the flavonoids located. Phenolic acids such as, gentisic, 2, 5 – dihydroxy benzoic acid, *o*-coumaric, vanillic, syringic, melilotic,  $\alpha$ -resorcylic, 3,5-dihydroxy benzoic acid, *p*-hydroxy benzoic acid, *cis* and *trans* - *p*-coumaric acid also were identified. Tannins, saponins, quinones and steroids were present and alkaloids and glycoflavones were absent.

### Pharmacology

Acute toxicity studies conducted revealed that the administration of graded doses of *C. pictus* leaf extract (up to a dosage of 1 gm/kg body weight/day) for 30 days produced no effect on the general behaviour or appearance of the animals and all the animals survived the test period. Body temperature, state of stool, body weight, water and food intake did not show any undesirable changes after the drug administration. Maximum glucose tolerance was observed after 2 hours of drug administration in normal glucose loaded rats (Table 1-9).

After the preliminary study, the alloxan induced diabetic rats were treated with the extract for 60 days and the parameters such as TBARS, non-enzymatic antioxidants such as GSH, vitamin C and vitamin E, activity of antioxidant enzymes such as SOD, CAT and GPX along with that the activity of carbohydrate metabolizing enzymes, toxicity parameters such as urea, uric acid and creatinine and plasma lipid levels were studied.

Significant hypoglycemic activity was observed in the leaf extract of 200mg, 400mg and glibenclamide in alloxan induced diabetic rats, the observed decrease was 57.09%, 57.2% and 57.0% respectively when compared with the untreated diabetic rats. The body weight gain was also improved by 24.7%, 23.3% and 24.9% in both the doses and glibenclamide when compared to the diabetic control rats (Table 10).

Table-11 to 14 present the changes observed in the level of TBARS, GSH, vitamin C and vitamin E in plasma, liver kidney and brain in control and experimental groups of rats. A significant increase in the level of TBARS was observed in plasma (56.1%), liver

(58.3%), kidney (57.7%) and brain (57.7%) of diabetic rats when compared with the normal control rats. The vitamin E level was found increased in plasma (34.2%) whereas the same decreased in liver (37.7%), kidney (59.2%) and brain (40.3%) of diabetic rats in comparison with the control rats. There was a significant decrease in the level of GSH and vitamin C in plasma (GSH: 42.2%; vitamin C: 57.8%), liver (GSH: 60%; vitamin C: 40%), kidney (GSH: 48.3%; vitamin C: 50%) and brain (GSH: 53.1%; vitamin C: 47.6%) of diabetic rats when compared with the control rats. After the treatment the level of TBARS in plasma liver kidney and brain was significantly ( $p < 0.05$ ) decreased and the percentage of decrease observed in 200mg, 400mg and glibenclamide was plasma (38.8%, 52.7% and 55.5%); liver (30.5%, 47.2% and 50%); kidney (40.1%, 45.9% and 52.5%); brain (36.6%, 57.7% and 55.1%) respectively when compared to diabetic control rats. There was decrease in the level of plasma vitamin E (24.0, 25.9 and 28.7) and in tissues the level was increased [ liver (22.09%, 22.63% and 32.7%); kidney (38.7%, 46.4% and 49.6%); brain (26.7%, 30.9% and 33.7%)] in the rats treated with extract 200mg, 400mg and glibenclamide respectively when compared with the untreated diabetic rats. There was a significant ( $p < 0.05$ ) increase in GSH (Plasma: 25.8%, 29.2% and 39.9%; liver: 48.2%, 51.6% and 57.6%; kidney: 42.5%, 45% and 45.3%; brain: 38.8%, 44.9% and 48.7%) and vitamin C (Plasma: 41.6%, 46.6 and 52.9%; liver: 30.7%, 31.8% and 34.7%; kidney: 38.7%, 42.3% and 46.4%; brain: 22.8%, 31.2% and 36.2%) observed in the rats treated with 200mg and 400mg extract and glibenclamide respectively when compared with the untreated diabetic rats.

The activity of SOD, CAT and GPX in liver, kidney and brain of control and experimental animals are given in table 15-17. Diabetic rats showed decreased activity of SOD, CAT and GPX in liver (64.1%, 57.8% and 53.9%) and brain (53.8%, 57.0% and 44.8%) respectively. In kidney the activity of GPX was increased (60.6%) and there was a decreased activity of SOD (56.9%) and CAT (47.2%). This diabetes induced changes were recovered significantly ( $p < 0.05$ ) after the treatment with the leaf extract 200mg, 400mg and glibenclamide. The level of SOD and CAT was increased [SOD (Brain: 46.5%, 48.5% and 49.0%; liver: 53.7%, 59.4% and 59.8%; kidney: 52.1%, 55.7% and 56.6% respectively) CAT (Brain: 32.2%, 43.7% and 45.2%; liver: 41.7%, 47.3% and



48.7; kidney: 38.1%, 46.6% and 53.02%)). The level of GPX in kidney was decreased (36.1%, 44.4% and 44.4%) and the same was increased in liver (37.8%, 45.8% and 48.7%) and brain (34.2%, 36% and 37.6%) respectively.

The activity of carbohydrate metabolizing enzymes depicted in table 18 explains that in diabetic rats there was a decreased activity of hexokinase (50%) but the activity of glucose-6-phosphatase (51.6%) and fructose-1, 6-bisphosphatase (53.7%) was increased in the liver. After the treatment the activity of hexokinase was increased by 38%, 45% and 45% and the percentage of decrease observed in glucose-6-phosphatase was 25.8%, 41.9% and 45.16% and in fructose- 1, 6 – bisphosphatase was 37.03%, 41.6% and 44.4% in 200mg 400mg leaf extract and glibenclamide respectively.

There was an increased level of urea (70.3%), uric acid (85.1%), creatinine (83.7%) and blood urea nitrogen (70.2%) in the plasma of diabetic rats (Table 19). This levels were decreased significantly ( $p < 0.05$ ) after the treatment with 200mg and 400mg of leaf extract and glibenclamide (urea: 40.4%, 45.1% and 48.6%; uric acid: 49.7%, 56.5% and 65.5%; creatinine: 72.09%, 77.2% and 78.6%; BUN: 40.5%, 45.1% and 48.7% respectively) when compared to the untreated diabetic rats.

Plasma cholesterol (28.0%), triglycerides (39.2%), free fatty acids (38.3%), phospholipids (57.6%), LDL-c (14.8%) and VLDL-c (39.0%) were increased significantly with a decreased level of HDL-c (43.1%) in diabetic rats (Table 20 and 21).

Administration of the *C. pictus* leaf extract (200mg and 400mg) and glibenclamide recovered the diabetic induced changes and decreased the level of lipids (cholesterol: 38.2%, 42.7% and 45.4; free fatty acid: 20.9%, 27.2% and 43.8; phospholipids: 16.1%, 30.1% and 32.8%; triglycerides: 42.8%, 47.9% and 54.1%; LDL-c: 49.8%, 56.5% and 62.2%; VLDL: 42.9%, 48.1% and 54.0 respectively) with an increase in HDL-c (52.7%, 58.1% and 63.4 respectively) when compared to the untreated diabetic rats.

### **Discussion:**

The results indicate that *C. pictus* leaf extract show a potent antidiabetic activity with a significant improvement in the body weight.

Lipid peroxidation has been implicated in a number of deleterious effects such as increased membrane rigidity, osmotic fragility, decreased cellular deformability, reduced erythrocyte survival and lipid fluidity as explained by Shobana *et al.*, (2000). In this study there was a significant elevated level of TBARS in plasma, liver, kidney and brain of diabetic rats which was recovered after the treatment this can be due to the ability of the extract to decrease the lipid peroxidation and there by activating the antioxidants to scavenge the free radical responsible for the lipid peroxidation during diabetes. These results are in agreement with the observations of previous researchers (Khan *et al.*, 1997). The level of GSH was decreased in plasma and tissues of diabetic rats. Gueeri (1995) explained that GSH is involved in the maintenance of normal cell structure and function probably through its redox and detoxification reactions. The glutathione depleted state may be due to the increased utilization of GSH by antioxidant enzymes such as glutathione peroxidase which scavenge  $H_2O_2$ , the leaf administration helped to restore the GSH level to near those of control rats.

Ascorbate has received much attention as a reducing agent since its discovery, and it has been recognized as an outstanding plasma antioxidant (Frie *et al.*, 1989). In the present experiment, concentration of vitamin C in plasma, liver, kidney and brain of diabetic rats were shown to decrease significantly. This result is in accordance with (Fang *et al.*, 1999). The decrease in vitamin C may be ascribed to its enhanced utilization due to increased oxidative stress caused by diabetes. Vitamin E is a major lipid soluble chain breaking antioxidant, in the present experiment, vitamin E level in diabetic rats gets increased in plasma and decreased in tissues. In this context, Asayam *et al.*, (1994) and Thompson *et al.*, (1992) also reported increased level of vitamin E in plasma. This can be due to increased peroxidation which was recovered after the treatment with *C. pictus* extract.

SOD scavenges the superoxide radical by converting it to  $H_2O_2$  and molecular oxygen (McCrod *et al.*, 1976). The observed decrease in the activity of SOD can be due to the inactivation of SOD by  $H_2O_2$  or by glycation of the enzyme. Similar result was reported by Sozmen *et al.*, (2001) in diabetic rats. The decreased activity of CAT observed in this

study can be due to increased production of reactive oxygen species. Chance *et al.*, (1952) explained that CAT is a heme protein, which catalyzed the reduction of hydrogen peroxides and protected the tissues from highly reactive hydroxyl radicals. GPX, an enzyme with selenium and GST works together with glutathione in the decomposition of  $H_2O_2$  or other organic hydroperoxides to non-toxic products at the expense of the GSH (Bruce *et al.*, 1982). Reduced activities of GPX may result from radical-induced inactivation and glycation of the enzyme (Hodgson and Fridovich, 1975). In this study the observed decrease in the activity of GPX can be due to the increased production of reactive oxygen species. In this context, other researchers also reported a decrease in the activity of antioxidant enzymes (SOD, CAT and GPX) in diabetic rats (Anuradha and Selvam, 1993; Stanely *et al.*, 2001). The activity of these enzymes were restored after the treatment and this result reflects the antioxidant potency of the extract, which by reducing blood glucose levels, prevents glycation and improves the activity of enzymatic and non enzymatic antioxidants.

Hexokinase, which brings about the first phosphorylation step of glucose metabolism, is reduced significantly in the diabetic group of rats (Nehal and Baquer, 1989). In the present study the observed decrease in the activity of hexokinase in the diabetic rat liver might be due to the diminished consumption of glucose in the system and increased blood sugar level. Liver plays an important role in the maintenance of blood glucose level by regulating its metabolism. The hepatic gluconeogenic enzymes, glucose-6-phosphatase and fructose-1, 6-bisphosphatase were increased significantly in diabetic rats. The increased activities of these two enzymes might be due to the activation or increased synthesis of the enzymes contributing to the increased glucose production during diabetes by the liver (Baquer *et al.*, 1998). In this present study *C. pictus* extract and glibenclamide treated groups restored the level of these carbohydrate metabolizing enzymes, this can be due to increased insulin secretion stimulated by the treatment.

The body cannot efficiently put the nitrogenous waste in to gaseous form and exhale it. Urea, uric acid and creatinine which are the major nitrogenous waste products are normally low in the blood since it is excreted in the urine continuously. When the kidney

fails to filter out this nitrogenous waste due to increased blood glucose level or due to kidney failure and these nitrogenous wastes get accumulated in the blood and this can lead to unconsciousness and death. The decreased level of these nitrogenous wastes after the treatment suggests that the plant extract can retrieve the renal damage caused due to diabetes.

In Table 20 and 21, it can be seen that diabetes provoked a rise in the levels of plasma lipids and a fall in the HDL-c, and that the extract and glibenclamide treatment restores these changes. Goodman and Gillman in 1985 explained that, the abnormal high concentration of serum lipids in the diabetic subjects is mainly due to an increase in the mobilization of free fatty acids from the peripheral fat deposit. The hypolipidemic effect of *C. pictus* could be explained as a direct result of the reduction in blood glucose concentration.

#### **Conclusion:**

The present findings suggest that the plant extract is non-toxic, since no marked changes were observed in the normal rats fed with the extract. Thus, at normal therapeutic doses, the extract was considered to be safe for long-term treatment in diabetic condition. The leaf extract showed potent antidiabetic activity and the dose 400mg/kg body weight was more effective than 200mg/kg body weight. 400mg dose was all most equally effective with the standard drug glibenclamide. Apart from this the plant extract also improved the activity of enzymatic and non-enzymatic antioxidants, thereby scavenging the free radical that initiates the lipid peroxidation. The decreased level of urea, uric acid and creatinine in the treated rats clearly shows that the plant extract, protects the diabetic rats from alloxan induced renal damage. The plant extract also lowered the plasma lipid levels, the antihyperlipidemic effect of the extract in particular can be considered as a possible therapeutic value. The result observed in all these parameters were statistically significant ( $p < 0.05$ ). Thus all these activities exhibited by the extract can be attributed to the presence of the active constituent of the plant. Longer duration studies of *C. pictus* extract and its isolated compounds are necessary to develop a potent antidiabetic drug

**Table: 1 Effect of *C. pictus* aqueous leaf extract on blood glucose levels in fasted normal rats.**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	1h	2h	3h
1	Control (received 2% gum acacia )	70.0 ± 2.8 <sup>a</sup>	69.05 ± 3.43 <sup>a</sup>	69.2 ± 3.4 <sup>a</sup>	68.6 ± 4.2 <sup>b</sup>
2	<i>C. pictus</i> 100mg	67.5 ± 5.3 <sup>a</sup>	67.6 ± 4.3 <sup>a</sup>	67.7 ± 4.9 <sup>a</sup>	66.7 ± 5.3 <sup>b</sup>
3	<i>C. pictus</i> 200mg	68.7 ± 3.8 <sup>a</sup>	68.1 ± 3.8 <sup>a</sup>	67.1 ± 3.6 <sup>b</sup>	66.7 ± 3.8 <sup>c</sup>
4	<i>C. pictus</i> 400mg	66.8 ± 2.7 <sup>a</sup>	65.9 ± 2.6 <sup>b</sup>	65.4 ± 2.9 <sup>c</sup>	65.0 ± 2.6 <sup>d</sup>
5	<i>C. pictus</i> 600mg	67.05 ± 2.9 <sup>a</sup>	66.2 ± 3.6 <sup>b</sup>	66.1 ± 3.5 <sup>c</sup>	65.8 ± 3.4 <sup>d</sup>
6	<i>C. pictus</i> 1000mg	67.45 ± 3.5 <sup>a</sup>	67.07 ± 3.4 <sup>b</sup>	66.7 ± 3.4 <sup>c</sup>	66.1 ± 3.3 <sup>d</sup>

**Table: 2 Effect of continuous administration of aqueous leaf extract of *C. pictus* on blood glucose levels in normal fasted rats.**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial Day	4 <sup>th</sup> Day	7 <sup>th</sup> Day	15 <sup>th</sup> Day
1	Control (received 2% gum acacia )	70.8 ± 1.8 <sup>a</sup>	70.1 ± 1.4 <sup>a</sup>	69.8 ± 1.5 <sup>a</sup>	68.7 ± 1.45 <sup>b</sup>
2	<i>C. pictus</i> 100mg	69.4 ± 2.1 <sup>a</sup>	69.0 ± 2.3 <sup>a</sup>	68.2 ± 1.6 <sup>b</sup>	67.3 ± 1.9 <sup>c</sup>
3	<i>C. pictus</i> 200mg	69.6 ± 2.6 <sup>a</sup>	69.0 ± 2.6 <sup>a</sup>	68.8 ± 2.9 <sup>b</sup>	68.1 ± 2.9 <sup>c</sup>
4	<i>C. pictus</i> 400mg	70.3 ± 1.8 <sup>a</sup>	69.6 ± 2.3 <sup>b</sup>	69.0 ± 1.9 <sup>b</sup>	68.3 ± 1.9 <sup>c</sup>
5	<i>C. pictus</i> 600mg	69.8 ± 2.2 <sup>a</sup>	69.2 ± 2.3 <sup>a</sup>	68.5 ± 2.6 <sup>b</sup>	67.8 ± 2.3 <sup>c</sup>
6	<i>C. pictus</i> 1000mg	69.05 ± 2.4 <sup>a</sup>	68.3 ± 2.4 <sup>a</sup>	67.8 ± 2.3 <sup>b</sup>	67.07 ± 2.4 <sup>c</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 3 Effect of *C. pictus* aqueous leaf extract on oral glucose tolerance in normal fasted rats (2g/kg body weight).**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	30 min	60 min	90 min
1	Control (received 2% gum acacia ) + glucose	68.9 ± 1.08 <sup>a</sup>	167.5 ± 3.4 <sup>b</sup>	164.5 ± 3.1 <sup>c</sup>	162.4 ± 2.9 <sup>c</sup>
2	<i>C. pictus</i> 100mg + glucose	67.6 ± 2.04 <sup>a</sup>	166.7 ± 3.3 <sup>b</sup>	163.6 ± 4.4 <sup>b</sup>	161.4 ± 4.1 <sup>c</sup>
3	<i>C. pictus</i> 200mg + glucose	69.4 ± 5.15 <sup>a</sup>	165.1 ± 5.7 <sup>b</sup>	162.9 ± 5.2 <sup>c</sup>	159.7 ± 3.3 <sup>d</sup>
4	<i>C. pictus</i> 400mg + glucose	69.1 ± 2.24 <sup>a</sup>	167.7 ± 3.4 <sup>b</sup>	164.8 ± 3.4 <sup>c</sup>	163.0 ± 2.9 <sup>d</sup>
5	<i>C. pictus</i> 600mg + glucose	69.5 ± 2.9 <sup>a</sup>	166.7 ± 3.5 <sup>b</sup>	164.5 ± 4.2 <sup>c</sup>	161.5 ± 2.8 <sup>d</sup>
6	<i>C. pictus</i> 1000mg + glucose	70.0 ± 1.9 <sup>a</sup>	168.0 ± 2.6 <sup>b</sup>	165.8 ± 1.7 <sup>c</sup>	163.3 ± 2.1 <sup>d</sup>

**Table: 4 Effect of *C. pictus* aqueous leaf extract on oral glucose tolerance test in normal fasted rats after 30 days of continuous drug administration (2g/kg body weight).**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	30 min	60 min	90 min
1	Control (received 2% gum acacia )	68.4 ± 1.5 <sup>a</sup>	167.0 ± 7.1 <sup>b</sup>	165.05 ± 6.8 <sup>b</sup>	162.4 ± 7.3 <sup>c</sup>
2	<i>C. pictus</i> 100mg	67.7 ± 2.2 <sup>a</sup>	166.5 ± 6.2 <sup>b</sup>	164.1 ± 6.4 <sup>b</sup>	162.0 ± 7.3 <sup>c</sup>
3	<i>C. pictus</i> 200mg	68.0 ± 3.1 <sup>a</sup>	164.5 ± 4.3 <sup>b</sup>	162.4 ± 4.2 <sup>b</sup>	160.2 ± 3.7 <sup>c</sup>
4	<i>C. pictus</i> 400mg	68.4 ± 3.09 <sup>a</sup>	167.1 ± 5.1 <sup>b</sup>	164.8 ± 5.09 <sup>c</sup>	162.6 ± 5.5 <sup>d</sup>
5	<i>C. pictus</i> 600mg	69.4 ± 2.9 <sup>a</sup>	167.6 ± 2.5 <sup>b</sup>	165.9 ± 2.2 <sup>c</sup>	164.1 ± 2.05 <sup>d</sup>
6	<i>C. pictus</i> 1000mg	67.9 ± 3.4 <sup>a</sup>	168.8 ± 3.08 <sup>b</sup>	167.1 ± 2.5 <sup>b</sup>	164.2 ± 3.5 <sup>c</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 5 Effect of continuous administration of *C. pictus* aqueous leaf extract on body weight changes in normal rats.**

Groups	Treatment (Dose/Kg body weight)	Body weight changes (gm)			
		Initial day	4 <sup>th</sup> Day	7 <sup>th</sup> Day	15 <sup>th</sup> Day
1	Control (received 2% gum acacia )	156.2 ± 1.7 <sup>a</sup>	157.4 ± 1.1 <sup>a</sup>	157.8 ± 0.6 <sup>a</sup>	158.3 ± 0.64 <sup>a</sup>
2	<i>C. pictus</i> 100mg	155.5 ± 0.6 <sup>a</sup>	156.2 ± 0.8 <sup>a</sup>	156.8 ± 0.3 <sup>a</sup>	157.6 ± 0.68 <sup>b</sup>
3	<i>C. pictus</i> 200mg	159.3 ± 2.7 <sup>a</sup>	159.8 ± 2.4 <sup>a</sup>	159.7 ± 2.8 <sup>a</sup>	160.25 ± 3.3 <sup>b</sup>
4	<i>C. pictus</i> 400mg	159.3 ± 2.6 <sup>a</sup>	160.2 ± 2.1 <sup>a</sup>	160.8 ± 2.07 <sup>a</sup>	161.7 ± 1.6 <sup>b</sup>
5	<i>C. pictus</i> 600mg	160 ± 1.9 <sup>a</sup>	160.2 ± 2.04 <sup>a</sup>	161.3 ± 2.2 <sup>b</sup>	162.2 ± 2.3 <sup>c</sup>
6	<i>C. pictus</i> 1000mg	159.5 ± 1.2 <sup>a</sup>	160.4 ± 1.61 <sup>a</sup>	161.2 ± 1.36 <sup>b</sup>	162.4 ± 1.2 <sup>c</sup>

**Table: 6 Effect of continuous administration of aqueous extract of *C. pictus* leaf on food intake in normal rats.**

Groups	Treatment (Dose/Kg body weight)	Food intake (g/week)		
		Initial day	4 <sup>th</sup> Day	15 <sup>th</sup> Day
1	Control (received 2% gum acacia )	76.2 ± 1.45 <sup>a</sup>	75.9 ± 1.3 <sup>a</sup>	74.9 ± 1.13 <sup>a</sup>
2	<i>C. pictus</i> 100mg	76.2 ± 1.7 <sup>a</sup>	75.9 ± 1.7 <sup>a</sup>	74.7 ± 1.5 <sup>b</sup>
3	<i>C. pictus</i> 200mg	76.6 ± 1.13 <sup>a</sup>	76.02 ± 1.01 <sup>a</sup>	74.8 ± 1.18 <sup>b</sup>
4	<i>C. pictus</i> 400mg	74.2 ± 1.01 <sup>a</sup>	73.5 ± 1.58 <sup>a</sup>	72.3 ± 1.6 <sup>b</sup>
5	<i>C. pictus</i> 600mg	76.2 ± 2.5 <sup>a</sup>	75.2 ± 2.38 <sup>a</sup>	73.7 ± 2.15 <sup>b</sup>
6	<i>C. pictus</i> 1000mg	76.5 ± 1.19 <sup>a</sup>	75.9 ± 0.86 <sup>a</sup>	74.9 ± 0.86 <sup>b</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 7 Effect of continuous administration of aqueous extract of *C. pictus* leaf on water intake in normal rats.**

Groups	Treatment (Dose/Kg body weight)	Water intake (L/week)			
		Initial day	4 <sup>th</sup> Day	7 <sup>th</sup> Day	15 <sup>th</sup> Day
1	Control (received 2% gum acacia )	4.6 ± 0.38	4.5 ± 0.46	4.8 ± 0.43	5.05 ± 0.44
2	<i>C. pictus</i> 100mg	5.0 ± 0.36	5.2 ± 0.30	5.3 ± 0.37	5.47 ± 0.18
3	<i>C. pictus</i> 200mg	4.6 ± 0.40	4.7 ± 0.36	4.8 ± 0.33	4.9 ± 0.35
4	<i>C. pictus</i> 400mg	4.5 ± 0.47	4.6 ± 0.40	4.6 ± 0.35	4.7 ± 0.36
5	<i>C. pictus</i> 600mg	4.7 ± 0.34 <sup>a</sup>	4.7 ± 0.45 <sup>a</sup>	4.8 ± 0.46 <sup>b</sup>	4.8 ± 0.51 <sup>b</sup>
6	<i>C. pictus</i> 1000mg	4.8 ± 0.40 <sup>a</sup>	4.7 ± 0.17 <sup>a</sup>	4.7 ± 0.10 <sup>a</sup>	5.0 ± 0.36 <sup>b</sup>

**Table: 8 Effect of *C. pictus* aqueous leaf extract on blood glucose level in alloxan diabetic rats.**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	1h	2h	3h
1	Control (received 2% gum acacia )	69.17 ± 2.86 <sup>a</sup>	68.07 ± 2.7 <sup>a</sup>	67.05 ± 3.1 <sup>a</sup>	65.5 ± 2.79 <sup>a</sup>
2	Diabetic control	258.3 ± 5.4 <sup>a</sup>	255.4 ± 5.4 <sup>a</sup>	253.1 ± 5.09 <sup>a</sup>	249.7 ± 4.1 <sup>a</sup>
3	Diabetic + <i>C. pictus</i> 200mg	258.8 ± 4.36 <sup>a</sup>	256.6 ± 4.3 <sup>b</sup>	255.0 ± 3.5 <sup>b</sup>	248.7 ± 2.32 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> 400mg	259.4 ± 3.89 <sup>a</sup>	257.1 ± 4.2 <sup>a</sup>	254.5 ± 4.2 <sup>b</sup>	252.0 ± 4.4 <sup>b</sup>
5	Diabetic + glibenclamide (600 µg/ kg body weight)	260.4 ± 3.6 <sup>a</sup>	258.4 ± 3.9 <sup>b</sup>	256.5 ± 3.4 <sup>b</sup>	254.5 ± 2.9 <sup>c</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)



**Table: 9 Effect of continuous administration of *C. pictus* aqueous leaf extract for 30 days on blood glucose level in alloxan diabetic rats.**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial day	4 <sup>th</sup> Day	7 <sup>th</sup> Day	15 <sup>th</sup> Day
1	Control (received 2% gum acacia )	68.07 ± 0.39 <sup>a</sup>	67.2 ± 0.46 <sup>a</sup>	66.5 ± 0.53 <sup>a</sup>	66.1 ± 0.49 <sup>a</sup>
2	Diabetic control	262.3 ± 5.7 <sup>a</sup>	263.4 ± 5.7 <sup>a</sup>	264.9 ± 6.3 <sup>a</sup>	266.7 ± 6.3 <sup>a</sup>
3	Diabetic + <i>C. pictus</i> 200mg	227.1 ± 4.6 <sup>a</sup>	224.6 ± 5.8 <sup>b</sup>	224.6 ± 5.7 <sup>b</sup>	223.3 ± 6.1 <sup>b</sup>
4	Diabetic + <i>C. pictus</i> 400mg	233.2 ± 5.1 <sup>a</sup>	230.4 ± 6.9 <sup>a</sup>	226.3 ± 7.2 <sup>b</sup>	224.5 ± 7.1 <sup>c</sup>
5	Diabetic + glibenclamide (600 µg/kg body weight)	234.5 ± 5.1 <sup>a</sup>	232.4 ± 5.21 <sup>a</sup>	230.7 ± 4.7 <sup>b</sup>	229.6 ± 4.9 <sup>b</sup>

**Table: 10 Effect of *C. pictus* on blood glucose level and body weight**

Groups	Treatment (Dose/Kg body weight)	Blood glucose (initial)	Blood glucose (final)	Body weight (initial)	Body weight (final)
1	Control (received 2% gum acacia )	79.00 ± 2.16	94.1 ± 4.9 <sup>a</sup>	183.3 ± 2.8	185.0 ± 2.6 <sup>a</sup>
2	Diabetic + control	255.00 ± 5.77	314.2 ± 28.4 <sup>b</sup>	178.0 ± 8.9	131.0 ± 2.6 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	264.50 ± 4.80	160.8 ± 11.14 <sup>c</sup>	172.5 ± 6.5	174.0 ± 6.7 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	265.00 ± 5.77	134.2 ± 6.6 <sup>d</sup>	169.5 ± 4.2	171.0 ± 4.8 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	297.50 ± 9.57	135 ± 17.3 <sup>d</sup>	172.8 ± 5.2	174.5 ± 4.4 <sup>d</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 11 Effect of *C. pictus* on TBARS, GSH, Vitamin C, and Vitamin E in plasma of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol /ml)	GSH (µg /dl)	Vitamin C (µg /dl)	Vitamin E (µg /dl)
1	Control (received 2% gum acacia )	1.58 ± 0.56 <sup>a</sup>	25.8 ± 2.5 <sup>a</sup>	1.9 ± 0.18 <sup>a</sup>	0.71 ± 0.02 <sup>a</sup>
2	Diabetic + control	3.6 ± 0.39 <sup>b</sup>	14.9 ± 2.3 <sup>b</sup>	0.8 ± 0.09 <sup>b</sup>	1.08 ± 0.01 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	2.2 ± 0.62 <sup>c</sup>	20.1 ± 3.25 <sup>c</sup>	1.37 ± 0.13 <sup>c</sup>	0.82 ± 0.02 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	1.7 ± 0.4 <sup>d</sup>	21.06 ± 2.3 <sup>c</sup>	1.5 ± 0.10 <sup>c</sup>	0.80 ± 0.02 <sup>c</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	1.6 ± 0.5 <sup>d</sup>	24.8 ± 2.7 <sup>d</sup>	1.7 ± 0.18 <sup>d</sup>	0.77 ± 0.02 <sup>d</sup>

**Table: 12 Effect of *C. pictus* on TBARS, GSH, Vitamin C, and Vitamin E in liver of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol /ml)	GSH (µg /dl)	Vitamin C (µg /dl)	Vitamin E (µg /dl)
1	Control (received 2% gum acacia )	1.5 ± 0.5 <sup>a</sup>	18.04 ± 2.2 <sup>a</sup>	0.75 ± 0.01 <sup>a</sup>	5.38 ± 0.13 <sup>a</sup>
2	Diabetic + control	3.6 ± 0.39 <sup>b</sup>	7.2 ± 2.5 <sup>b</sup>	0.45 ± 0.03 <sup>b</sup>	3.35 ± 0.19 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	2.5 ± 0.47 <sup>c</sup>	13.9 ± 1.7 <sup>c</sup>	0.65 ± 0.03 <sup>c</sup>	4.30 ± 0.24 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	1.9 ± 0.77 <sup>c</sup>	14.9 ± 1.2 <sup>c</sup>	0.66 ± 0.04 <sup>c</sup>	4.33 ± 0.10 <sup>c</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	1.8 ± 0.4 <sup>d</sup>	17.0 ± 2.4 <sup>d</sup>	0.69 ± 0.03 <sup>d</sup>	4.98 ± 0.17 <sup>d</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 13 Effect of *C. pictus* on TBARS, GSH, Vitamin C, and Vitamin E in Kidney of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol /ml)	GSH (µg /dl)	Vitamin C (µg /dl)	Vitamin E (µg /dl)
1	Control (received 2% gum acacia )	1.6 ± 0.47 <sup>a</sup>	14.9 ± 3.6 <sup>a</sup>	0.60 ± 0.02 <sup>a</sup>	3.68 ± 0.15 <sup>a</sup>
2	Diabetic + control	3.79 ± 0.33 <sup>b</sup>	7.7 ± 2.59 <sup>b</sup>	0.30 ± 0.05 <sup>b</sup>	1.50 ± 0.08 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	2.27 ± 0.58 <sup>c</sup>	13.4 ± 1.6 <sup>c</sup>	0.49 ± 0.01 <sup>c</sup>	2.45 ± 0.19 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	2.05 ± 0.69 <sup>d</sup>	14.0 ± 1.6 <sup>d</sup>	0.52 ± 0.02 <sup>d</sup>	2.80 ± 0.16 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	1.8 ± 0.66 <sup>d</sup>	14.1 ± 1.2 <sup>d</sup>	0.56 ± 0.01 <sup>d</sup>	2.98 ± 0.21 <sup>d</sup>

**Table: 14 Effect of *C. pictus* on TBARS, GSH, Vitamin C, and Vitamin E in Brain of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol /ml)	GSH(µg /dl)	Vitamin C (µg /dl)	Vitamin E (µg /dl)
1	Control (received 2% gum acacia )	1.6 ± 0.47 <sup>a</sup>	17.5 ± 2.5 <sup>a</sup>	0.84 ± 0.05 <sup>a</sup>	5.50 ± 0.22 <sup>a</sup>
2	Diabetic + control	3.79 ± 0.33 <sup>b</sup>	8.2 ± 2.5 <sup>b</sup>	0.44 ± 0.04 <sup>b</sup>	3.28 ± 0.10 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	2.4 ± 0.76 <sup>c</sup>	13.4 ± 1.6 <sup>c</sup>	0.57 ± 0.03 <sup>c</sup>	4.48 ± 0.10 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	1.6 ± 0.7 <sup>d</sup>	14.9 ± 2.3 <sup>d</sup>	0.64 ± 0.01 <sup>d</sup>	4.75 ± 0.10 <sup>c</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	1.7 ± 0.65 <sup>d</sup>	16.0 ± 2.3 <sup>d</sup>	0.69 ± 0.02 <sup>d</sup>	4.95 ± 0.13 <sup>c</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 15 Effect of *C. pictus* on SOD, CAT and GPX in brain of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	SOD U/mg protein	CAT U/mg protein	GPx U/mg protein
1	Control (received 2% gum acacia )	11.77 ± 0.21 <sup>a</sup>	166.6 ± 3.8 <sup>a</sup>	8.7 ± 0.63 <sup>a</sup>
2	Diabetic + control	5.4 ± 1.71 <sup>b</sup>	71.5 ± 10.0 <sup>b</sup>	4.8 ± 0.79 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	10.1 ± 0.35 <sup>c</sup>	105.5 ± 3.3 <sup>c</sup>	7.3 ± 0.7 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	10.5 ± 0.155 <sup>c</sup>	127 ± 13.6 <sup>d</sup>	7.5 ± 0.67 <sup>c</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	10.6 ± 0.37 <sup>c</sup>	130.5 ± 17.8 <sup>e</sup>	7.7 ± 0.97 <sup>d</sup>

**Table: 16 Effect of *C. pictus* on SOD, CAT and GPX in liver of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	SOD U/mg protein	CAT U/mg protein	GPx U/mg protein
1	Control (received 2% gum acacia )	12.0 ± 0.32 <sup>a</sup>	163 ± 24.36 <sup>a</sup>	8.9 ± 1.89 <sup>a</sup>
2	Diabetic + control	4.3 ± 0.68 <sup>b</sup>	68.7 ± 8.24 <sup>b</sup>	4.1 ± 0.78 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	9.3 ± 1.1 <sup>c</sup>	118 ± 9.3 <sup>c</sup>	6.6 ± 1.1 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	10.6 ± 0.22 <sup>c</sup>	130.5 ± 12.24 <sup>d</sup>	7.5 ± 0.6 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	10.7 ± 0.38 <sup>c</sup>	34 ± 13.25 <sup>d</sup>	8 ± 0.8 <sup>e</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 17 Effect of *C. pictus* on SOD, CAT and GPX in Kidney of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	SOD U/mg protein	CAT U/mg protein	GPx U/mg protein
1	Control (received 2% gum acacia )	11.7 ± 0.18 <sup>a</sup>	75.7 ± 7.17 <sup>a</sup>	3.8 ± 0.5 <sup>a</sup>
2	Diabetic + control	4.6 ± 1.17 <sup>b</sup>	32.6 ± 6.68 <sup>b</sup>	7.2 ± 0.8 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	9.6 ± 0.40 <sup>c</sup>	52.7 ± 6.25 <sup>c</sup>	4.6 ± 0.9 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	10.4 ± 0.29 <sup>c</sup>	61.1 ± 4.30 <sup>d</sup>	4.0 ± 0.59 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	10.6 ± 0.20 <sup>c</sup>	69.4 ± 5.69 <sup>d</sup>	4.0 ± 0.59 <sup>d</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 18 Effect of *C. pictus* on Hexokinase, Glucose-6-phosphatase and Fructose-1, 6-bisphosphatase of alloxan induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	Hexokinase (U <sup>a</sup> / mg protein)	Glucose-6-phosphatase (U <sup>b</sup> /mg protein)	Fructose-1, 6-bisphosphatase (U <sup>c</sup> /mg protein)
1	Control (received 2% gum acacia )	0.22 ± 0.01 <sup>a</sup>	0.15 ± 0.16 <sup>a</sup>	0.5 ± 0.02 <sup>a</sup>
2	Diabetic + control	0.11 ± 0.013 <sup>b</sup>	0.31 ± 0.057 <sup>b</sup>	1.08 ± 0.04 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	0.18 ± 0.015 <sup>c</sup>	0.23 ± 0.065 <sup>c</sup>	0.68 ± 0.02 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	0.2 ± 0.020 <sup>d</sup>	0.18 ± 0.024 <sup>d</sup>	0.63 ± 0.39 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	0.2 ± 0.023 <sup>d</sup>	0.17 ± 0.017 <sup>d</sup>	0.60 ± 0.55 <sup>d</sup>

Values are means ± S.D for six animals in each group. Enzyme units are expressed as units/mg protein

<sup>a</sup> µmol of glucose phosphorylated/h

<sup>b</sup> µmol of liberated / min

<sup>c</sup> µmol of pi liberated / min

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 19 Effect of *C. pictus* on plasma Urea, Uric acid, creatinine, and BUN on streptozotocin induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	Urea (mg/dl)	Uric acid (nmol/ml)	Creatinine (mg/dl)	Blood Urea Nitrogen (mg/dl)
1	Control (received 2% gum acacia )	19.6 ± 5.8 <sup>a</sup>	3.05 ± 0.4 <sup>a</sup>	0.7 ± 0.4 <sup>a</sup>	9.15 ± 2.7 <sup>a</sup>
2	Diabetic + control	66.0 ± 6.8 <sup>b</sup>	20.5 ± 0.87 <sup>b</sup>	4.3 ± 1.05 <sup>b</sup>	30.8 ± 3.19 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	39.3 ± 3.2 <sup>c</sup>	10.3 ± 0.48 <sup>c</sup>	1.2 ± 0.53 <sup>c</sup>	18.3 ± 1.4 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	36.2 ± 2.3 <sup>d</sup>	8.9 ± 0.56 <sup>d</sup>	0.98 ± 0.26 <sup>d</sup>	16.9 ± 1.11 <sup>d</sup>
5	Diabetic + Glibenclamide(600µg/kg body weight)	33.9 ± 2.2 <sup>e</sup>	7.06 ± 1.03 <sup>d</sup>	0.92 ± 0.48 <sup>d</sup>	15.8 ± 1.06 <sup>e</sup>

**Table: 20 Effect of *C. pictus* on plasma Cholesterol, Free fatty acid, Phospholipids and Triglycerides on streptozotocin induced diabetic rats.**

Groups	Treatment (Dose/Kg body weight)	Cholesterol (mg/dl)	Free fatty acids (mg/dl)	Phospholipids (mg/dl)	Triglycerides (mg/dl)
1	Control (received 2% gum acacia )	63.9 ± 10.9 <sup>a</sup>	50.9 ± 2.2 <sup>a</sup>	121.3 ± 1.17 <sup>a</sup>	59.9 ± 21.8 <sup>a</sup>
2	Diabetic + control	228 ± 27.3 <sup>b</sup>	132.8 ± 1.7 <sup>b</sup>	210.4 ± 6.5 <sup>b</sup>	152.6 ± 12.7 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	140.7 ± 41.5 <sup>c</sup>	105 ± 5.9 <sup>c</sup>	176.5 ± 4.4 <sup>c</sup>	87.2 ± 7.6 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	130.5 ± 27.18 <sup>d</sup>	96.6 ± 1.4 <sup>d</sup>	146.9 ± 6.2 <sup>d</sup>	79.4 ± 9.8 <sup>d</sup>
5	Diabetic + Glibenclamide (600µg/kg body weight)	124.4 ± 13.3 <sup>e</sup>	74.6 ± 2.7 <sup>e</sup>	141.2 ± 3.5 <sup>e</sup>	70 ± 14.1 <sup>e</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

**Table: 21 Effect of *C. pictus* on plasma HDL-C, LDL-C and VLDL-C in streptozotocin induced diabetic rats**

Groups	Treatment (Dose/Kg body weight)	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
1	Control (received 2% gum acacia )	39.9 ± 4.6 <sup>a</sup>	35.9 ± 7.1 <sup>a</sup>	11.9 ± 4.3 <sup>a</sup>
2	Diabetic + control	17.2 ± 6.6 <sup>b</sup>	242 ± 20 <sup>b</sup>	30.5 ± 2.5 <sup>b</sup>
3	Diabetic + <i>C. pictus</i> (200mg)	36.4 ± 2.2 <sup>c</sup>	121.4 ± 41.9 <sup>c</sup>	17.4 ± 1.5 <sup>c</sup>
4	Diabetic + <i>C. pictus</i> (400mg)	41.1 ± 8.7 <sup>d</sup>	105.2 ± 31.3 <sup>d</sup>	15.8 ± 1.9 <sup>d</sup>
5	Diabetic + Glibenclamide (600µg/kg body weight)	47 ± 13 <sup>e</sup>	91.4 ± 9.1 <sup>e</sup>	14.0 ± 2.8 <sup>e</sup>

Values are means ± S.D for six animals in each group.

Values not sharing a common superscript differ significantly at p<0.05.

Duncan's Multiple Range Test (DMRT)

