

CHAPTER – III

***Cassia alata* Linn.**



***Cassia alata* Linn**
Plate No: 3



Habit

***Cassia alata* Linn.**

Family: Caesalpiniaceae

Sanskrit name: Dadhrugna, Dvipagsti.

Commonly known as Roman Candle Bush, this is one of the many plants recommended for diabetes. It is a shrub growing up to 5 meters and the leaves are pinnately compound, coarse with obovate leaflets. Buds are covered with orange bracts which fall off when the flower opens. Fruit pod, oblong, thin, straight, seeds 50 or more.

Phytochemistry:

The roots contain an anthraquinone, alquinone (3-formyl-1, 2, 8-trihydroxyanthraquinone, $C_{15}H_8O_6$). The stem contains anthraquinones, alatonal ($C_{15}H_8O_6$, mp 300^0) and alatinone ($C_{15}H_{10}O_5$), together with dalbergin, 2, 6-dimethoxybenzoquinone, santal, luteolin, β -sitosterol and its β -D-glucoside. (Yadav & Kalidhar, 1994; Hemlata & Kalidhar, 1993).

The various compounds reported from leaves are flavonoids such as kaempferol, luteolin, rhamnetin glycoside, chrysoeriol glycoside, anthraquinones like emodin, aloe-emodin, chrysophanic acid, isochrysophanol, rhein, rhein methyl ester diacetate, 4, 5-dihydroxy-2-hydroxyanthrone, 4, 5-dihydroxy-1-hydroxyanthrone and physcion monoglucoside, β -sitosterol, lectins, dalbergin, daucosterol, deoxycoelulatin, and alatinone (Gupta & Singh, 1991; Yadav & Kalidar, 1994; Kelly *et al.*, 1994). It is reported that hydroxyl anthraquinone derivatives were concentrated in the leaves and flowers of *Cassia alata*. Kaemferol-3-O-gentiobioside, was quantified in various parts of the plant. The matured leaf was found to contain the highest content of this flavonols, the content ranged from 2.0 to 5.0%, the other parts studied were flower (sepal and petal), rachis, stem and seeds. But this compound was not detected in seeds.

The seeds contain two glycosides, chrysoeriol-7-O-(2''-O- β -D-mannopyranosyl)- β -D-allopyranoside and rhamnetin-3-O-(2''-O- β -D-mannopyranosyl)- β -D-allopyranoside (Gupta & Sing, 1991) and saturated fatty acids such as isopalmitic acid, palmitoleic acid,

myristoleic acid, tridecanoic, oleic acid, palmitic acid, myristic acid and caprylic / octanoic acid. They contain other compounds such as anthraquinones, chrysophanol, emodin, aloe-emodin and rhein also (Morah & Otumu, 1991).

Biological activities:

Traditionally the leaves are used to treat ringworm in Fiji, Tonga and Samoa. Bark is used to treat skin diseases, diarrhea, worms, parasitic skin diseases, scabies and eczema. In New Guinea, the leaves and wood sap are used as a remedy for constipation and in Fiji an infusion of the leaves is used to purify blood (Lans, *et al.*, 2000; Ponglux *et al.*, 1987). *Cassia alata* also finds use in preventative veterinary medicine and is used to deworm dogs (Cambie & Ash, 1994). In Orissa, the flowers are cooked as vegetable.

The leaf exhibited blood sugar lowering activity in streptozotocin- induced hyperglycemic animals while it had no effect on glucose levels in normoglycaemic animals (Palanichamy *et al.*, 1988). Antibacterial activity of the leaf was reported to be due to the presence of rhein. An infusion of the leaves showed a significant laxative activity at 800 mg/kg body weight. It is also used as a wash to cure eczema. Crushed leaves or roots are rubbed onto the skin to cure ringworm, and to control *Tinea imbricate*, a skin fungus. The leaf extract formulated in a polyethylene glycol base showed wound healing activity. A drug prepared from the leaves prevented the breaking of chromosome due to genotoxins (Palanichamy *et al.*, 1990; ogunti *et al.*, 1991; ogunti & Elujoba, 1993; Palanichamy & Nagarajan, 1990). It also showed purgative activity in healthy subjects with no side effects (May-Ave-Than *et al.*, 2002).

Aqueous and alcoholic extracts of leaf showed antibacterial activity on *Dermatophilus congolensis*, a gram positive bacterium that caused bovine dematophilosis; the alcoholic extract was more effective than the aqueous extract (Ali-emmanuel *et al.*, 2002).

The aqueous extract of the leaf showed antifungal activity on the skin of male and female patients, who were suffering from pityriasis versicolor, achronic superficial mycosis of the skin caused by the fungus malassezia furfur and had no side effects

(Damodaran & Venkataraman, 1994). Apart from which it also showed antihepatotoxic activity, antifungal activity (Effraim *et al.*, 1999; Sakharkar & patil, 1998). The saponin content in the aqueous extract was reported to be toxic since it decreased the levels of haemoglobin and erythrocyte count. In addition, it increased packed cell volume (Sodipo, *et al.*, 1998).

The hexane leaf extract was reported to be antianalgesic. Hexane and ethylacetate extract of the leaf exhibited anti-inflammatory activity. The chloroform extract of the leaf showed antimutagenic activity. The ethyl acetate extract also showed hypoglycemic activity. The pharmacological studies showed that all extracts (hexane, chloroform and ethyl acetate) of the leaf caused an immediate decrease in motor activity, enophthalmus, hyperemia, micturition and diarrhea. An excess dose of ethyl acetate extract of the leaf caused paralysis, screen grip loss and enophthalmus accompanied by drooping and closure of the eye lids. (Villasenor *et al.*, 2002).

Three cDNAs encoding very similar but unique isoforms of chalcone synthase (naringenin-chalcone synthase) [E. C. 2. 3. 1. 74] were isolated from a cDNA library prepared from RNA from root tissue of *C. alata*. Gene transcript for these three type III polyketide synthase was found to accumulate predominantly in roots. *C. alata* accumulates the flavonoids, quercetin, naringenin and kaempferol in roots, suggesting that the in planta function of these enzymes is the biosynthesis of root flavanoids (Samappito *et al.*, 2002).

Results:

Phytochemistry:

The leaves were found to possess various known quinones, flavonoids and phenolic acids. The flavonoids present were kaempferol, 3', 4'- OMe - Quercetin and 7, 3', 4'- tri OMe-Quercetin. The phenolic acids present were vanillic, syringic, 2-hydroxy benzoic, melilotic, *o*-coumaric and *p*-coumaric acids. Steroids, proanthocyanidins, saponins, and tannins were also present and alkaloids were absent.

Pharmacognosy:

Stomatal complex:

Stomatal Index/mm² was 7.6 on the upper epidermis and 11.26 on the lower epidermis, Stomatal Frequency/mm² was 5.5 on the upper epidermis and 8.5 on the lower epidermis, Trichome index/mm² was 1.85 on the upper epidermis and 3.22 on the lower epidermis, Trichome frequency/mm² was 1.2 on the upper epidermis and 2.2 on the lower epidermis, Palisade ratio was 8.05/mm², Vein Islet number/mm² was 5.2 and Vein Termination number/mm² was 12.8 respectively.

Anatomy:

Lower surface of the epidermis were papillose towards the lamina. The papillae appeared as outgrowths, where as in surface view they gave the impression of rounded bodies. Leaves were amphistomatic bearing anisocytic stomata and paracytic stomata. Adaxial and abaxial epidermal layers contained unicellular trichomes with swollen bases. The average size of the epidermal cell was 26.07x24.75µm on the lower surface and 40.26 x 21.45 µm on the upper surface. The size of the stomata on the upper surface was 16.5 x 10.56 µm and on the lower surface was 17.82 x 10.56 µm (Fig 1).

An arc shaped vascular bundle was located at the centre of the midrib which was surrounded by 2-3 layers of sclerenchyma cells in triangular patches. The parenchyma cells located above the sclerenchyma contained compound starch grains and calcium oxalate crystals. The size of the cells were: upper epidermal cells 13.86x16.50 µm; lower epidermal cells 17.49x14.19 µm; sclerides 12.88x13.12 µm; xylem vessels 25.41x25.41 µm; tracheids 13.53x15.18µm; trichomes 8.58x11.22 µm; crystals 22.4x25.08 µm; starch grains 6.6x4.1 µm; pith 29.04x30.36µm (Fig 2).

The vascular bundle of rachis was round in shape with sclerenchymatous pericyclic cap above each bundle. Epidermis contained unicellular trichomes. Compound starch grains were present on the parenchymatous cells, medullary rays, phloem and in the pith region. Tannin cells were seen on the hypodermis and sphaeraphides were seen only on the pith

region. The size of the cells were: upper epidermal cells 9.57x9.57 μm ; lower epidermal cells 10.50x9.80 μm ; sclerides 18.15x15.84 μm ; parenchyma 15.84x28.05 μm ; xylem vessels 58.08x39.60 μm ; tracheids 18.15x13.20 μm ; trichomes 36.30x8.25 μm ; crystals 22.4x25.08 μm ; starch grains 6.6x4.1 μm ; pith 41.91x42.57 μm (Fig 3).

Vascular bundle in the petiole was more or less oval shaped and opening on the upper side. The pith region contained irregular collenchyma cells. Starch grains and sphaeraphides were present on the cortex region and trichomes were unicellular with swollen base. The size of the cells were: upper epidermal cells 14.52x15.18 μm ; lower epidermal cells 15.18x16.50 μm ; collenchyma 11.88x14.19 μm ; parenchyma 38.61x33.99 μm ; xylem vessels 15.18x16.50 μm ; tracheids 17.22x12.14 μm ; trichomes 43.56x9.24 μm ; crystals 22.4x25.08 μm ; starch grains 6.6x4.1 μm ; pith 35.64x10.56 μm (Fig 4).

Leaf powdered contain vessels with scalariform pitting, tracheids with spiral thickenings, fragments of epidermis with papillae, palisade, paracytic stomata, anisocytic stomata, tannin filled cells, epidermal cells, collenchymatus cells, sclerenchyma cells, chlorenchyma cells, trichomes, rosettes of calcium oxalate crystals, starch grains. Powdered rachis contained vessel with scalariform pitting, spiral tracheids, rosettes of calcium oxalate crystals, trichomes and sclerenchyma (Fig. 5).

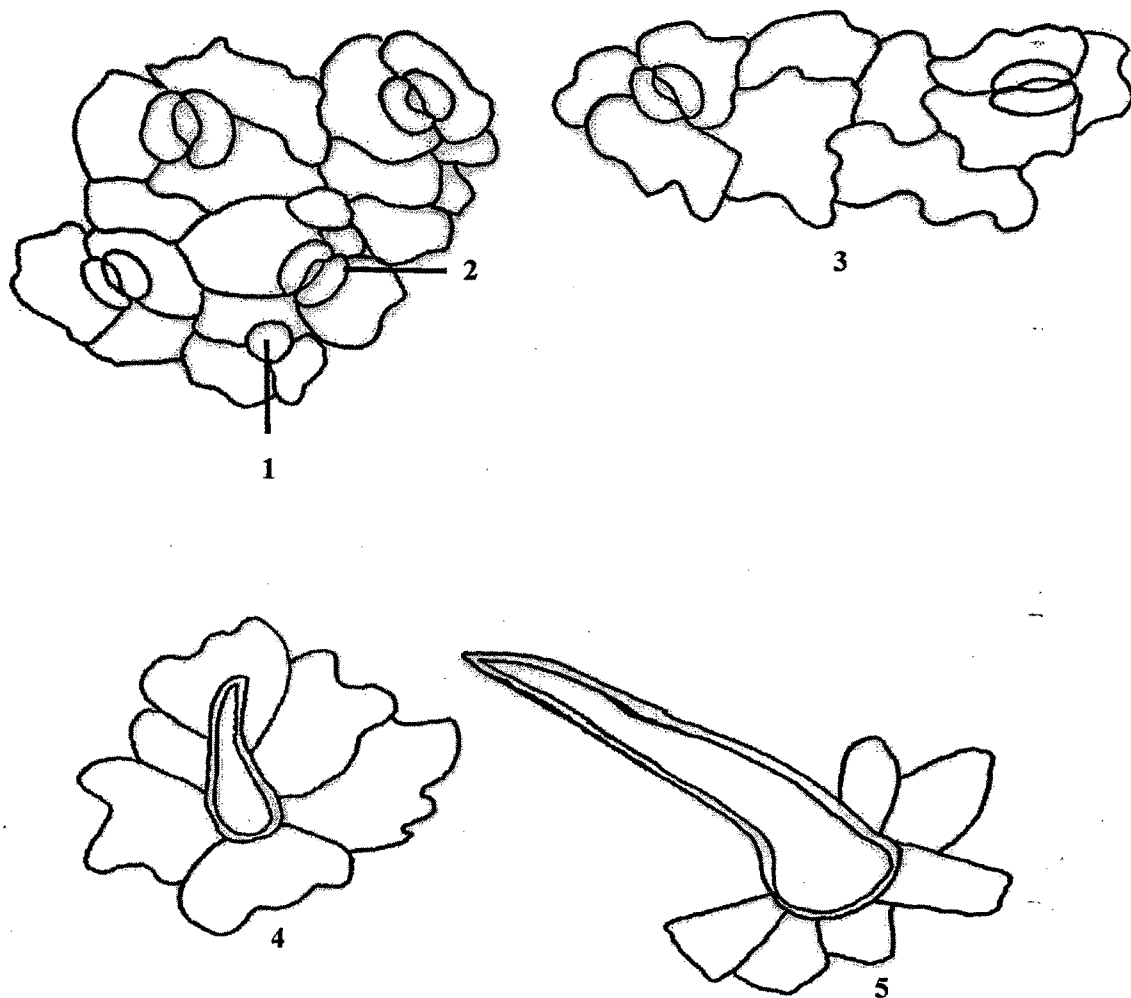


Fig. 1 Epidermal peel *Cassia alata*:

1. Paracytic stomata- Lower epidermis
2. Anisocytic stomata- Upper epidermis
3. Papillae
4. Unicellular trichome- Lower epidermis
5. Unicellular trichome- Upper epidermis

0.1 mm

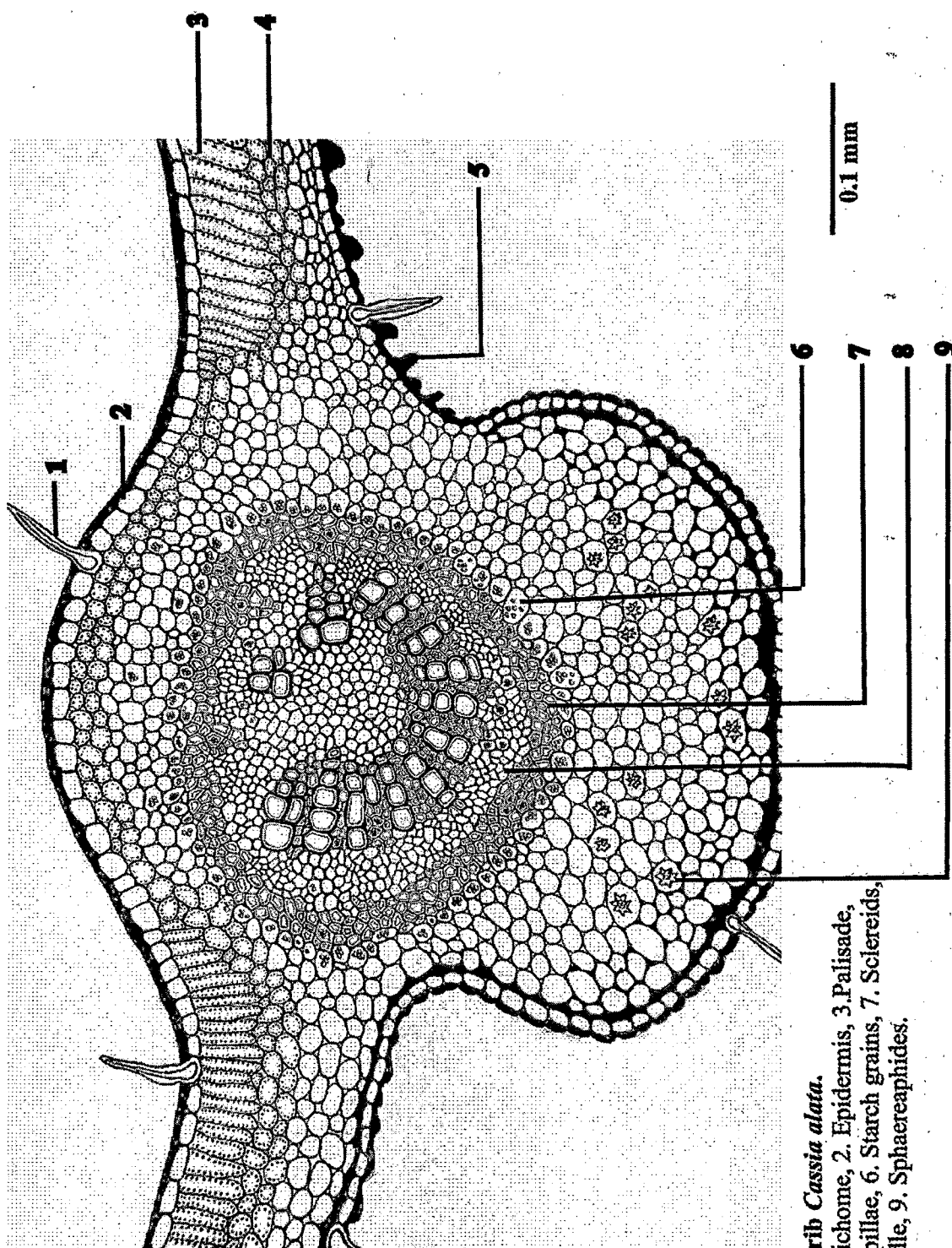


Fig 2: T. S Midrib *Cassia alata*.

1. Unicellular trichome, 2. Epidermis, 3. Palisade,
4. Spongy, 5. Papillae, 6. Starch grains, 7. Sclereids,
8. Vascular bundle, 9. Sphaerocarpophides.

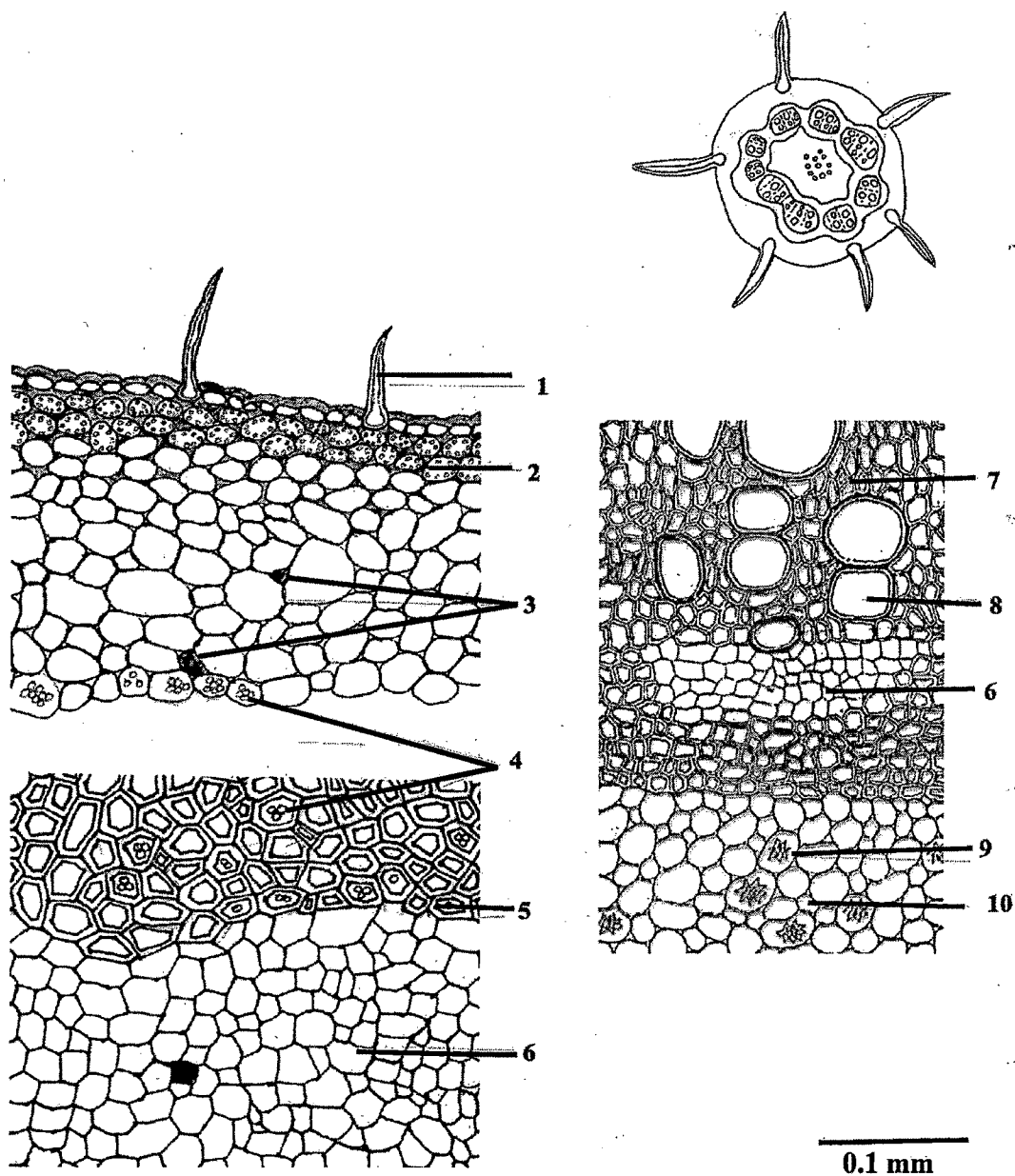


Fig. 3 T. S of Rachis *Cassia alata*: 1. Unicellular trichome, 2. Chlorenchyma, 3. Tannin cells, 4. Starch grains, 5. Sclereids, 6. Phloem, 7. Medullary rays, 8. Xylem, 9. Sphaeraphides, 10. Pith

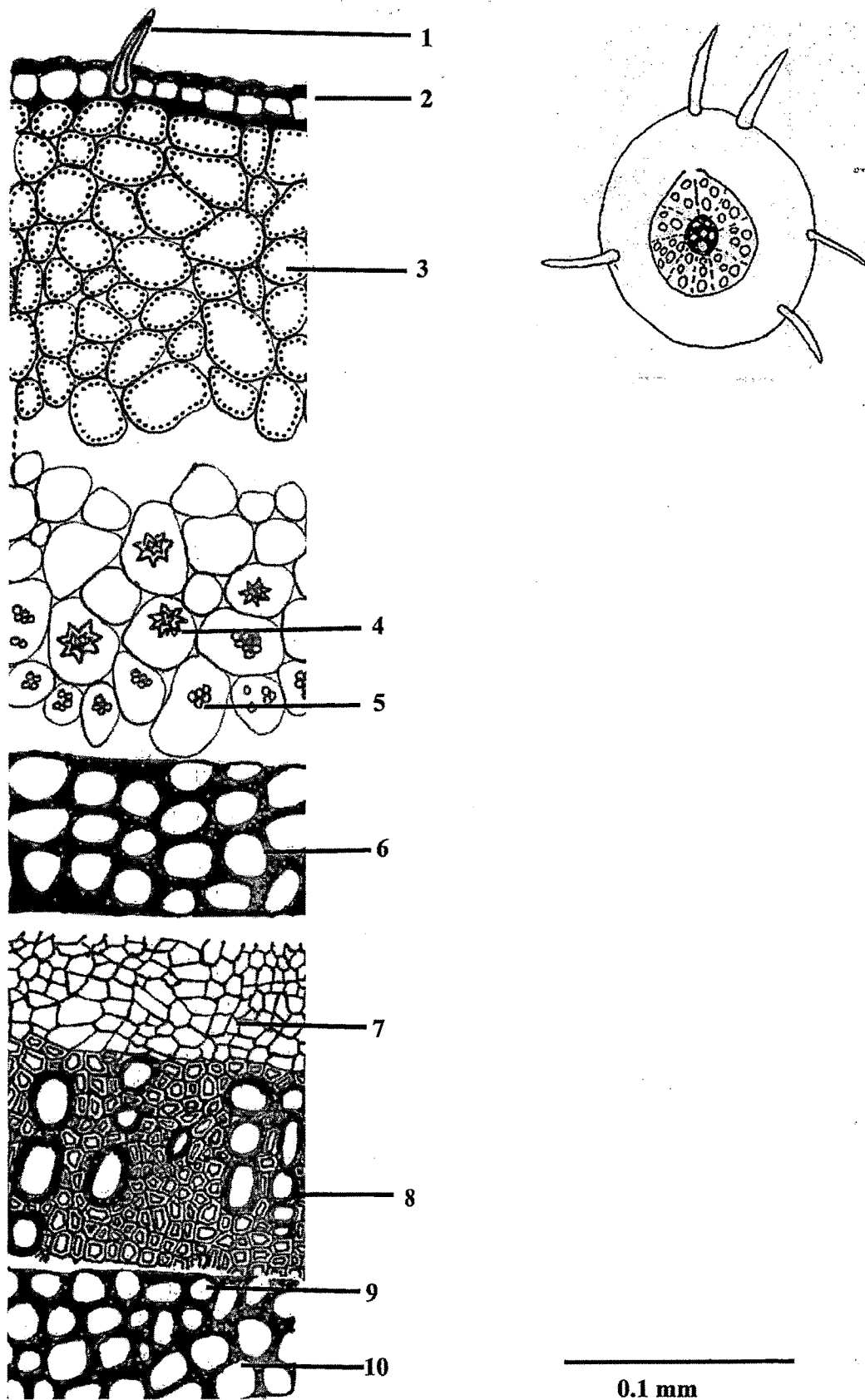


Fig. 4: T. S. Petiole *Cassia alata*, 1. Unicellular trichome, 2. Epidermis, 3. Chlorenchyma, 4. Sphaeraphides, 5. Starch grains, 6. Thick walled parenchyma, 7. Phloem, 8. Xylem, 9. Medullary rays, 10. Pith.

Fig. 5 Powder characters of *Cassia alata*

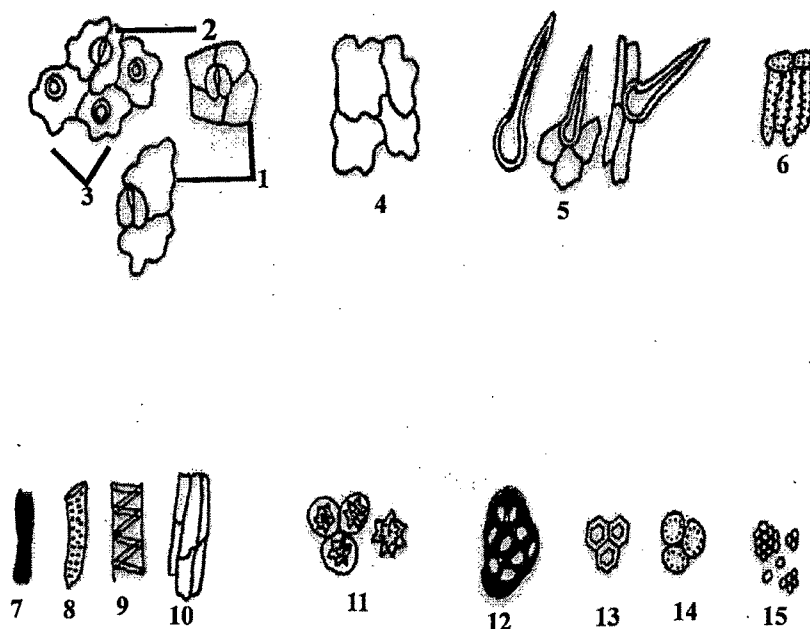
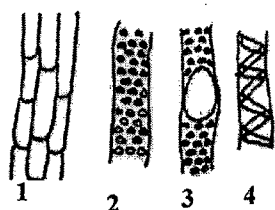


Fig. 6: Powdered Rachis of *Cassia alata*



0.1 mm

Fig. 5 Powder characters of *Cassia alata*:

1. Anisocytic stomata, 2. Paracytic stomata, 3. Papillae, 4. Parenchyma, 5. Unicellular trichomes. 6. Palisade, 7. Tannin filled cell, 8. Vessels with scalariform pitting, 9. Tracheids with spiral thickenings, 10. Fragments of epidermis, 11. Calcium oxalate crystals, 12. Collenchyma, 13. Sclerenchyma, 14. Chlorenchyma, 15. Starch grains.

Fig. 6: Powdered Rachis of *Cassia alata*:

1. Fragments of epidermis, 2. Saclariform pitting, 3. Reticulate thickening, 4. Spiral tracheids.

Pharmacology:

Experiments were carried out in three phases. In the first phase, the plant extracts (alcoholic, aqueous fresh leaf and aqueous dry leaf extract) were studied to determine the hypoglycemic activity (blood glucose lowering in normal rats), antihyperglycemic activity (blood glucose lowering in diabetes induced rats), glucose tolerance and the safety profile in normoglycemic rats.

Second and third phase were carried out to determine the antihyperglycemic activity and antioxidant activity of the aqueous fresh and dry leaf extract also, the efficacy of the extract to improve the lipid levels in streptozotocin induced and alloxan induced diabetic rats. The ability of the extracts to protect the diabetes induced renal damage was studied.

Phase I Results and Discussion:

The study showed that a peak hypoglycemia occurred in normal rats on the 2nd hour after the administration of the aqueous extracts (fresh and dry). On continuous treatment for 15 days a significant fall in the fasting blood glucose was noticed. There were no significant changes observed in body weight, food and water intake. Both the extracts showed maximum glucose tolerance in normal rats after 30 minutes and the doses 400 and 600 mg/kg body weight showed increased tolerance to glucose. Alcoholic extract showed maximum glucose tolerance after 1 hour. Same results were observed in all the three extracts even after continuous treatment of the extracts for 15 days in normal rats.

Since the aqueous extract with dose 400 and 600 mg/kg body exhibited better effect in normal rats, the same was taken as a standard dose and administered orally in a volume of 10 ml/kg body weight, along with this a standard drug glibenclamide was also treated in diabetes induced rats. The maximum blood glucose reduction was noted after 2 hours of drug administration. After 15 days of treatment on diabetic rats, when blood glucose were checked on 4th, 7th, and 15th day, there was a maximum reduction of blood glucose on 15th day, but all these effects were less than the standard drug glibenclamide.

Since both the aqueous extracts showed the same effects, aqueous extract of the dry leaf was tested for the levels of plasma TBARS, urea, and uric acid after 15 days of the treatment. And these levels were noted at the initial day, 4th, 7th and 15th day of the treatment. It was observed that the level of TBARS, urea and uric acid were decreased in the treated rats (Table 1-29)

The optimum time of action of the extract was 2 hours after the time of administration. This implies that the activity of the crude extract might be of short duration. However multiple doses were also effective in reducing plasma glucose concentration after a glucose challenge. The long term effect was also evident.

The mechanism of action is not known at present. The hypoglycemic effect may be due to any of the following reasons:

1. Increase in the release of pancreatic insulin.
2. Decrease absorption of glucose from the intestine or an increased uptake of glucose at the peripheral tissues.
3. Inhibition of dietary fiber or the inhibition of glucose transporter.

The pancreas is especially susceptible to the action of STZ and alloxan induced free radical damage, administration of *C.alata* leaf extract might reduce the level of plasma lipid peroxides. These results confirm the possibility that a major function of the extract is protection of vital tissues including liver, kidney, brain, and pancreas, thereby reducing the chances of diabetes. Reduction in the body weights were observed in the diabetic rats, but when the animals were treated with *C.alata* leaf extract, the decrease in body weight was minimized and an improvement in body weight was observed. The present study also indicated that *C.alata* can inhibit diabetic renal damage partially as seen from the elevated urea levels. Uric acid is considered as one of the non-enzymatic antioxidant, but increased production of uric acid means, increased free radical production due to activation of the xanthine oxidase enzyme system (Nemeth *et al.*, 2002). In our experiment uric acid levels were increased in diabetic rats. This may be due to metabolic disturbance in diabetes reflected in high activities of xanthine oxidase and lipid peroxidation.

Effect of aqueous dry leaf extract of *C. alata* on streptozotocin induced diabetes including their effect on antioxidant enzymes, non enzymatic antioxidants and lipid profile:

Result and Discussion Phase II:

The blood glucose level, body weight and hemoglobin percentage, in control and experimental rats, are presented in Table 30. It is observed that blood glucose levels of the treated (400mg, 600mg and glibenclamide) rats showed significant decrease (41%, 50.5% and 54.2%) and hemoglobin percentage was significantly increased (12.5%, 21.2 %and 22.2%) when compared to the untreated diabetic rats.

The levels of TBARS, GSH, Vitamin C, and Vitamin E in plasma, erythrocyte, liver and kidney of normal and experimental animals are shown in Table 31, 32, 33 and 34. Diabetic animals showed increased TBARS in plasma (42.1%), erythrocytes (54.6%), liver (69.1%) and kidney (65.9%). The level of GSH, and Vitamin C, were decreased (Plasma: 39.5% and 75.9%; erythrocyte: 35.6% and 43.7%; liver: 32.9% and 34.3; kidney: 41.6% and 42.8 respectively) when compared to the control rats. The vitamin E level was increased in plasma (31.4%) and decreased in erythrocytes (48.1%), liver (34.2%) and kidney (57.3%) when compared with the normal rats. Increased levels of TBARS in diabetic rats suggest an increase in oxygen free radicals. The decreased level of GSH in plasma, liver kidney and erythrocytes in diabetic rats, is due to the generation of oxygen radicals. An increased level of glucose cause utilization of GSH and thus diminishes GSH levels in the plasma during diabetes. Similar diminished levels of serum GSH in experimental diabetic rats was also reported earlier (Sajithlal *et al.*, 1998).

The observed decrease in Vitamin C in diabetic rats may be due to increased utilization of vitamin C due to the increased reactive oxygen species, or it can be due to decrease in GSH level, since GSH is required for the recycling of Vitamin C (Infers & Sies, 1988).

The elevated levels of α - tocopherol in the plasma of diabetic rats may play a protective role against increased peroxidation in diabetes. The decrease in the level of α - tocopherol

in the erythrocyte, liver and kidney in diabetic rats can be due to the increased utilization of α -tocopherol due to increased level of TBARS. Treatment with the leaf extract 400mg, 600mg and glibenclamide altered the diabetic induced changes in the TBARS and non-enzymatic antioxidants. The level of TBARS in plasma (22.3%, 30.5% and 33.6%), erythrocyte (16.5%, 22.2% and 23.7%), liver (51.5%, 59.2% and 63.4%) and kidney (52.1%, 59.7% and 61.8%) were decreased. The level of GSH and vitamin C was increased (plasma GSH: 21.2%, 28.2% and 30.8%; plasma vitamin C: 27.9%, 35.5% and 38.7%; erythrocyte GSH: 15.5%, 23.1% and 27.4%; erythrocyte vitamin C: 33.1%, 36.5% and 38.8%; liver GSH: 20.3%, 24.3% and 28.1%; liver vitamin C: 27.5%, 30% and 32%; kidney GSH: 24%, 34.3% and 37.5%; kidney vitamin C: 33.3%, 38.5% and 39.6%) and it is observed that there was a decrease in the plasma vitamin E (9.8%, 15.9% and 19.5%) and the level was increased in erythrocyte (22.2%, 31.7% and 33.3%), liver (29.1%, 30.2% and 32.9%) and kidney (48.2%, 52.6% and 53.8%) when compared with the untreated diabetic rats.

The activities of SOD, CAT and GPX which were measured in liver, kidney and erythrocyte, of normal and experimental rats are shown in Table 35, 36 and 37. Diabetic rats showed decreased activity of SOD (41% and 46.3%), CAT (49.8% and 47.5%) and GPX (40.5% and 40.3%) in liver and erythrocyte respectively, but in kidney, the activity of GPX was increased (38.7%) and the activity of SOD and CAT was decreased by 51.6% and 46.9% respectively when compared with the normal control rats. These variations in the antioxidants are because of raised blood glucose levels that induce oxidative stress via the generation of oxygen free radicals. *C. alata* extract 400mg, 600mg and glibenclamide recovered the diabetic induced changes in enzymatic antioxidants. The activity of SOD, CAT and GPX in liver and erythrocyte were increased (liver SOD: 27.9%, 35.6% and 39.5%; liver CAT: 37.8%, 43.5% and 46.9% and liver GPX: 34.3%, 37.4% and 39.1%; erythrocyte SOD: 25.4%, 33.3% and 38.9%; erythrocyte CAT: 40.1%, 41.5% and 42.9% and erythrocyte GPX: 26.8%, 33.7% and 37.3%) respectively. The activity of GPX in the kidney was decreased (18.72%, 29.1% and 31.7%) and the activity of SOD and CAT was increased by (42.5%, 47.7%, 48% and 35.4%, 40.7% and 42.7%) respectively when compared to the untreated diabetic rats.

Table 38 illustrates the level of plasma cholesterol, free fatty acids, phospholipids and triglycerides in normal and experimental rats. In the diabetic rats the lipid levels were increased (cholesterol: 71.3%, free fatty acids: 60.3%, phospholipids: 41.8% and triglycerides: 60.7%), administration of plant extract at 400, 600 mg kg⁻¹ and glibenclamide, decreased plasma cholesterol (24.3%, 37.1% and 40.7%); free fatty acids (16.6%, 25.7% and 41.9%), phospholipids (18.9%, 29.5% and 33.3%) and triglycerides (36.1%, 40.4% and 56.3%) when compared with the untreated diabetic rats.

The concentration of HDL, LDL and VLDL in the plasma of control and experimental rats are shown in Table 39. A decrease in HDL cholesterol (28%) and an increase in LDL (76.5%) and VLDL cholesterol (44.7%) in diabetic rats were observed when compared with the normal rats. The extract 400mg, 600mg and glibenclamide significantly increased the HDL cholesterol (11.3%, 14.4% and 17.4%) and decreased the level of LDL (33.8%, 50.5% and 56.8%) and VLDL cholesterol (26%, 29.4% and 33.4%) respectively. Lipid disorders are common in both insulin dependent and non insulin dependent diabetes mellitus and are related to the degree of glycemic control. The hypolipidemic effect of *C. alata* could be explained as a direct result of the reduction in blood glucose concentration.

Effect of aqueous fresh leaf extract of *C. alata* on alloxan induced diabetes including their effect on antioxidant enzymes, non enzymatic antioxidants and lipid profile:

Result and Discussion Phase III:

Fasting blood glucose concentrations at the initial and final stages observed in the study, confirm uncontrolled hyperglycemia in untreated diabetic rats, where as the leaf extract 400mg, 600mg and glibenclamide treatment remarkably decreased blood glucose concentration (51.6%, 56.7% and 57%) in diabetic rats (Table 40). The effect of the leaf extract in controlling hyperglycemia could be due to polyphenols or simple phenols present in the leaf.

The increased level of TBARS in plasma (56.1%), liver (58.3%), kidney (56.7%) and brain (56.7%) in diabetes indicates the activation of lipid peroxidation system. Lipid peroxide

mediated tissue damage has been observed in the development of type I and type II diabetes mellitus (Sundaram *et al.*, 1996). The level of GSH and Vitamin C was decreased in plasma (42.2% and 57.9%), liver (60.1% and 40%), kidney (51.7% and 55.9%) and brain (53% and 47.6%) of diabetic rats. Other workers have observed that elevation in glucose concentration may depress natural antioxidant defenses such as vitamin C and glutathione (Armstrong *et al.*, 1996 and yoshida *et al.*, 1995). The level of vitamin E was increased in plasma (33%) and decreased in liver (41.5%), kidney (59.5%) and brain (37.9%). Vitamin E is circulatory antioxidant and they are also called as non-enzymatic free radical scavengers. All these diabetes induced changes observed in TBARS and non-enzymatic antioxidants were recovered after the treatment (Table 41, 42, 43, and 44). The level of TBARS was decreased in plasma (33.3%, 43.1% and 50%), liver (36.1%, 47.2% and 52.7%), kidney (40.5%, 48.6% and 51.4%) and brain (32.4%, 51.4% and 54.1%). There was a significant increase observed in GSH and vitamin C in plasma (35.7%, 38.4%, 39.9% and 46.6%, 50%, 52.9%), liver (50%, 53.5%, 54.7% and 31.8%, 34.8%, 36.6%), kidney (50%, 53.5%, 54.7% and 41.2%, 43.4%, 56.4%) and brain (41%, 43.1%, 49.1% and 31%, 35.3%, 36%). It is observed that the level of vitamin E was decreased in plasma (22.6%, 27.4% and 24.5%) and increased in liver (24.5%, 31.3% and 29.7%), kidney (43.7%, 44.25 and 50.3%) and brain (24%, 30.7% and 31.4%) of the experimental rats treated with 400mg, 600mg and glibenclamide when compared with the untreated diabetic rats.

In this study a decrease in the activities of SOD and CAT in brain (53.8% and 56.9%), liver (64.2% and 57.9%) and kidney (60.7% and 56.9%) of diabetic rats were observed. SOD is a major defense for aerobic cells combating the toxic effects of superoxide radicals (Mc Crod *et al.*, 1976). CAT protects tissues from highly reactive hydroxyl radicals (Chance *et al.*, 1952). Therefore, a reduction in the activities of SOD and CAT in diabetic rats can lead to an excess availability of superoxide ($O_2^{\cdot-}$) and hydrogen peroxide (H_2O_2) in the biological system, which in turn generates hydroxyl radicals resulting in initiation and propagation of lipid peroxidation.

In diabetic rats the activity of GPX was decreased in brain (44.8%) and liver (53.9%), whereas in kidney there was an increased level of GPX (47.2%). Glutathione peroxidase

has a key role in enzymatic defense systems and acts on peroxides to remove them. In the present study it was observed that *C. alata* 400mg, 600mg and glibenclamide therapy could normalize the level of these antioxidant enzymes in plasma liver kidney and brain (Table 45, 46 and 47). The activity of SOD and CAT were increased in brain (47%, 49.5%, 49.5% and 38%, 43% and 45%), liver (58.6%, 58.6%, 59.8% and 40.1%, 48.2%, 48.7%) and kidney (54.5%, 55.7%, 56.6% and 38.9%, 48.4%, 53%) and the activity of GPX was decreased in kidney (36.1%, 43.1% and 44.4%) and increased in brain (32.4%, 36% and 37.6%) and liver (40.5%, 46.7% and 48.7%) respectively when compared with the untreated diabetic rats.

The variation in carbohydrate metabolizing enzymes such as hexokinase, glucose-6-phosphatase and fructose-1, 6-bisphosphatase in liver was studied. It is observed that the activity of hexokinase was decreased (50%) which may be due to insulin deficiency, the increased activities of glucose-6-phosphatase (51.6%) and fructose-1,6-bisphosphatase (49.1%) might be due to decreased insulin level because insulin decreases gluconeogenesis by decreasing the activities of key enzymes such as glucose-6-phosphatase, fructose-1,6-bisphosphatase, phosphoenol pyruvate carboxykinase and pyruvate carboxylase (Murray *et al.*,2000), treatment with the leaf extract 400mg, 600mg and glibenclamide significantly reduced the activities of glucose-6-phosphatase (41.9%, 41.9% and 45.2%) and fructose-1,6-bisphosphatase (35.2%, 41.6% and 44.4%), also it increased the activity of hexokinase (35.3%, 45% and 45%) (Table 48).

Kidney maintains optimum chemical composition of body fluids by acidification of urine and removal of metabolites. Wastes such as urea, uric acid, creatinine and ions, during renal diseases the concentrations of these metabolites increase in blood. In this study, the levels of urea (70.3%), uric acid (85%) and creatinine (83.7%) were elevated in diabetic rats, this condition was retrieved after the treatment with the extract 400mg, 600mg and glibenclamide, the level of urea (40%, 48.4% and 48.6%), uric acid (55.6%, 62.4% and 65.5%) and creatinine (72.1%, 79.1% and 79.1%) were decreased significantly. This indicates that the extract protects kidney from any renal injury there by reducing the causes of diabetes (table 49).

Abnormalities in lipoproteins are very common in both NIDDM and IDDM. Although lipoprotein alterations appear to be an intrinsic part of these disorders, such alterations are also induced by diabetes associated complications such as obesity and hyperlipidemia. In the present study, total cholesterol (71.9%), free fatty acids (61.7%), triglycerides (60.7%), phospholipids (42.3%), LDL (85.2%) and VLDL (60.9%) were increased in diabetic rats, along with a decrease in HDL (56.9%). The elevated levels of lipids observed in the diabetic rats were reduced (cholesterol: 40.4%, 44.3% and 45.6%, free fatty acid: 20.2%, 26.9% and 44.3%, phospholipids: 18.6%, 28.6%, 32.8%, triglycerides: 43.9%, 52%, 54.1%, LDL: 53.3%, 58.6% and 62.2%, VLDL: 43.8%, 52.1% and 54.1%) by the extract 400mg, 600mg and glibenclamide and the level of HDL was increased (56.6%, 59% and 63.4%) when compared with the untreated diabetic rats. These results suggest the beneficial effects of the natural extract in improving the imbalance in lipid metabolism are also comparable to those of glibenclamide (Table 50 and 51).

Conclusion:

It can be thus concluded that aqueous leaf extract of *C. alata* (both dry and fresh) exhibited hypoglycaemic effect in streptozotocin and alloxan induced diabetes in rats. The various effects of the extract on enzymatic and non enzymatic antioxidants are due to the reduction in the blood glucose concentration in diabetic rats by both the fresh and dry leaf extract showed the same effect and the extract at a dose of 600mg kg⁻¹ was more effective but less than the standard drug glibenclamide. Further studies are necessary to determine the exact nature of the active principles and the mechanism of action of the plant extract.

Table 1 Effect of *C. alata* dry 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)	66.4 ± 5.8 ^a	65.8 ± 7.6 ^a	65.4 ± 5.4 ^a	63.8 ± 4.6 ^b
2	<i>C. alata</i> 100mg	67.8 ± 4.8 ^a	66.2 ± 4.6 ^a	65.7 ± 7.6 ^a	62.8 ± 3.6 ^b
3	<i>C. alata</i> 200mg	68.6 ± 8.5 ^a	66.4 ± 3.8 ^a	62.6 ± 6.6 ^b	60.7 ± 5.8 ^c
4	<i>C. alata</i> 400mg	65.4 ± 2.6 ^a	61.2 ± 7.1 ^b	56.4 ± 4.6 ^c	52.6 ± 4.8 ^d
5	<i>C. alata</i> 600mg	65.4 ± 2.6 ^a	62.3 ± 5.8 ^b	56.8 ± 2.6 ^c	50.4 ± 3.2 ^d
6	<i>C. alata</i> 1000mg	67.3 ± 7.1 ^a	64.6 ± 4.7 ^b	61.4 ± 3.8 ^c	58.4 ± 2.6 ^d

Table 2 Effect of *C. alata* alcoholic 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)	67.2 ± 3.1 ^a	67.2 ± 4.2 ^a	66.8 ± 3.6 ^a	68.8 ± 4.2 ^a
2	<i>C. alata</i> 100mg	68.6 ± 3.2 ^a	67.4 ± 5.6 ^a	66.5 ± 4.2 ^a	63.8 ± 2.6 ^b
3	<i>C. alata</i> 200mg	67.4 ± 3.6 ^a	65.6 ± 4.2 ^a	64.6 ± 4.6 ^b	62.1 ± 5.4 ^c
4	<i>C. alata</i> 400mg	68.4 ± 3.2 ^a	63.4 ± 5.6 ^b	59.6 ± 4.1 ^c	58.7 ± 4.1 ^d
5	<i>C. alata</i> 600mg	67.8 ± 6.6 ^a	62.0 ± 3.6 ^b	60.0 ± 5.2 ^c	57.6 ± 3.8 ^d
6	<i>C. alata</i> 1000mg	68.0 ± 2.6 ^a	66.4 ± 5.8 ^a	65.6 ± 4.2 ^b	62.6 ± 4.8 ^c

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 continuous administration of dry aqueous extract of *C. alata* on blood glucose levels in normal fasted rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial Day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	67.8 ± 4.2 ^a	66.2 ± 3.6 ^a	65.6 ± 6.8 ^a	66.8 ± 6.2 ^a
2	<i>C. alata</i> 100mg	68.8 ± 5.8 ^a	66.4 ± 7.2 ^a	65.2 ± 4.2 ^b	64.2 ± 3.6 ^b
3	<i>C. alata</i> 200mg	65.6 ± 2.6 ^a	63.9 ± 7.4 ^a	62.4 ± 3.2 ^b	61.8 ± 4.4 ^b
4	<i>C. alata</i> 400mg	66.6 ± 2.2 ^a	62.3 ± 4.7 ^b	59.4 ± 5.5 ^c	57.8 ± 4.2 ^d
5	<i>C. alata</i> 600mg	67.4 ± 5.6 ^a	64.4 ± 7.2 ^b	61.6 ± 3.8 ^c	56.4 ± 4.5 ^d
6	<i>C. alata</i> 1000mg	68.4 ± 4.6 ^a	66.4 ± 3.6 ^b	64.8 ± 4.8 ^c	62.7 ± 3.2 ^d

Table 4 Effect of continuous administration of alcoholic extract of *C. alata* on blood glucose levels in normal fasted rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial Day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	68.2 ± 4.6 ^a	66.4 ± 7.4 ^a	66.2 ± 6.8 ^a	66.7 ± 3.6 ^a
2	<i>C. alata</i> 100mg	67.4 ± 5.4 ^a	66.2 ± 5.2 ^a	64.2 ± 4.6 ^b	61.4 ± 3.1 ^c
3	<i>C. alata</i> 200mg	68.6 ± 2.8 ^a	65.6 ± 4.2 ^b	63.3 ± 4.5 ^c	61.4 ± 3.6 ^c
4	<i>C. alata</i> 400mg	67.4 ± 3.8 ^a	63.0 ± 3.2 ^b	61.6 ± 4.8 ^c	59.4 ± 6.8 ^d
5	<i>C. alata</i> 600mg	66.4 ± 5.2 ^a	62.4 ± 2.8 ^b	58.6 ± 4.4 ^c	56.7 ± 3.5 ^d
6	<i>C. alata</i> 1000mg	67.4 ± 4.6 ^a	65.4 ± 5.2 ^b	63.2 ± 2.6 ^c	63.4 ± 4.8 ^d

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 *C. alata* dry 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	69.6 ± 8.5 ^a	168.4 ± 12.5 ^b	162.6 ± 8.8 ^a	154.6 ± 11.2 ^a
2	<i>C. alata</i> 100mg + glucose	68.6 ± 6.5 ^a	162.6 ± 8.6 ^b	156.4 ± 10.6 ^b	148.7 ± 8.0 ^c
3	<i>C. alata</i> 200mg + glucose	69.6 ± 4.8 ^a	146.4 ± 9.6 ^b	140.6 ± 12.4 ^b	138.4 ± 11.6 ^b
4	<i>C. alata</i> 400mg + glucose	68.8 ± 4.2 ^a	142.8 ± 13.6 ^b	124.6 ± 10.4 ^c	120.8 ± 12.4 ^c
5	<i>C. alata</i> 600mg + glucose	67.6 ± 5.2 ^a	136.4 ± 10.5 ^b	122.4 ± 11.6 ^c	108.6 ± 10.4 ^d
6	<i>C. alata</i> 1000mg + glucose	69.6 ± 3.8 ^a	144.6 ± 13.2 ^b	138.4 ± 8.6 ^c	132.8 ± 9.8 ^d

Table 6 Effect of *C. alata* alcoholic leaf extract on oral glucose tolerance in normal fasted rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	30 min	60 min	90 min
1	Control (received 2% gum acacia)	67.8 ± 4.6 ^a	176.4 ± 10.8 ^b	164.8 ± 9.6 ^c	158.0 ± 8.6 ^c
2	<i>C. alata</i> 100mg + glucose	68.8 ± 5.8 ^a	168.8 ± 8.8 ^b	160.4 ± 11.6 ^c	152.6 ± 13.5 ^c
3	<i>C. alata</i> 200mg + glucose	69.6 ± 4.2 ^a	152.4 ± 14.6 ^b	146.4 ± 10.8 ^c	142.8 ± 10.8 ^c
4	<i>C. alata</i> 400mg + glucose	68.2 ± 6.2 ^a	148.6 ± 13.2 ^b	138.4 ± 10.6 ^c	130.8 ± 11.6 ^d
5	<i>C. alata</i> 600mg + glucose	67.5 ± 6.8 ^a	139.4 ± 13.2 ^b	131.4 ± 12.6 ^c	124.6 ± 13.2 ^d
6	<i>C. alata</i> 1000mg + glucose	66.6 ± 8.6 ^a	146.4 ± 8.2 ^b	140.6 ± 10.6 ^c	130.6 ± 10.8 ^d

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 *C. alata* dry aqueous leaf extract on oral glucose tolerance test in normal fasted rats after 15 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)	66.8 ± 6.2 ^a	174.3 ± 12.4 ^b	173.8 ± 10.6 ^b	168.4 ± 8.6 ^b
2	<i>C. alata</i> 100mg	64.2 ± 3.6 ^a	168.6 ± 10.8 ^b	163.4 ± 8.6 ^b	158.2 ± 9.4 ^c
3	<i>C. alata</i> 200mg	61.8 ± 4.4 ^a	162.6 ± 11.4 ^b	159.6 ± 10.4 ^b	155.3 ± 11.8 ^b
4	<i>C. alata</i> 400mg	57.8 ± 4.2 ^a	124.6 ± 11.2 ^b	116.4 ± 12.4 ^b	108.4 ± 10.4 ^c
5	<i>C. alata</i> 600mg	56.4 ± 4.5 ^a	122.8 ± 8.6 ^b	118.4 ± 8.6 ^c	110.4 ± 9.4 ^d
6	<i>C. alata</i> 1000mg	62.7 ± 3.2 ^a	133.8 ± 7.6 ^b	126.4 ± 9.4 ^c	122.8 ± 10.4 ^d

Table: 8 Effect of alcoholic extract of *C. alata* leaf on oral glucose tolerance test in normal fasted rats after 15 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)	66.7 ± 3.6 ^a	171.6 ± 12.4 ^b	169.8 ± 10.6 ^b	165.8 ± 12.4 ^b
2	<i>C. alata</i> 100mg	61.4 ± 3.1 ^a	168.4 ± 13.6 ^b	164.2 ± 10.0 ^c	161.8 ± 8.8 ^d
3	<i>C. alata</i> 200mg	61.4 ± 3.6 ^a	164.6 ± 11.3 ^b	160.8 ± 11.4 ^c	158.4 ± 8.6 ^c
4	<i>C. alata</i> 400mg	59.4 ± 6.8 ^a	130.4 ± 11.4 ^b	124.8 ± 11.4 ^c	120.6 ± 10.2 ^c
5	<i>C. alata</i> 600mg	56.7 ± 3.5 ^a	126.4 ± 10.8 ^b	122.6 ± 9.4 ^c	118.2 ± 11.4 ^c
6	<i>C. alata</i> 1000mg	63.4 ± 4.8 ^a	136.8 ± 11.4 ^b	133.4 ± 12.1 ^c	129.6 ± 8.8 ^d

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. alata* dry aqueous extract on body weight changes in normal rats.

Groups	Treatment (Dose/Kg body weight)	Body weight changes (gm)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	153.7 ± 6.8 ^a	154.3 ± 5.8 ^a	156.3 ± 6.2 ^a	158.2 ± 10.4 ^a
2	<i>C. alata</i> 100mg	156.2 ± 4.2 ^a	157.3 ± 3.8 ^a	158.6 ± 2.6 ^a	160.4 ± 4.8 ^b
3	<i>C. alata</i> 200mg	155.4 ± 2.6 ^a	156.3 ± 2.6 ^a	157.4 ± 3.3 ^a	159.4 ± 4.1 ^b
4	<i>C. alata</i> 400mg	153.7 ± 4.3 ^a	154.0 ± 2.6 ^a	156.3 ± 2.7 ^a	160.2 ± 3.8 ^a
5	<i>C. alata</i> 600mg	155.5 ± 2.6 ^a	157.4 ± 4.6 ^a	158.3 ± 4.8 ^a	159.4 ± 4.6 ^b
6	<i>C. alata</i> 1000mg	154.5 ± 3.7 ^a	155.5 ± 4.6 ^a	157.2 ± 2.8 ^b	159.4 ± 6.8 ^c

Table: 10 Effect of continuous administration of *C. alata* alcoholic extract on body weight changes in normal rats.

Groups	Treatment (Dose/Kg body weight)	Body weight changes (gm)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	153.6 ± 6.8 ^a	154.3 ± 5.8 ^a	156.4 ± 6.2 ^a	158.4 ± 10.4 ^a
2	<i>C. alata</i> 100mg	156.4 ± 4.2 ^a	157.4 ± 3.8 ^a	158.6 ± 2.6 ^a	160.4 ± 4.8 ^b
3	<i>C. alata</i> 200mg	155.6 ± 2.6 ^a	156.4 ± 2.6 ^a	157.4 ± 3.3 ^a	159.4 ± 4.1 ^b
4	<i>C. alata</i> 400mg	153.8 ± 4.3 ^a	154.3 ± 2.6 ^a	156.3 ± 2.7 ^a	160.4 ± 3.8 ^a
5	<i>C. alata</i> 600mg	155.6 ± 2.6 ^a	157.6 ± 4.6 ^a	158.4 ± 4.8 ^a	159.4 ± 4.6 ^b
6	<i>C. alata</i> 1000mg	154.6 ± 3.7 ^a	155.6 ± 4.6 ^a	157.8 ± 2.8 ^b	159.6 ± 6.8 ^c

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 continuous administration of dry aqueous extract of *C. alata* leaf on food intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Food intake (g/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	76.4 ± 4.8 ^a	75.3 ± 3.4 ^a	75.6 ± 4.6 ^a	76.4 ± 3.8 ^a
2	<i>C. alata</i> 100mg	78.2 ± 3.6 ^a	79.6 ± 6.4 ^a	79.8 ± 5.8 ^a	80.6 ± 4.6 ^b
3	<i>C. alata</i> 200mg	75.3 ± 4.6 ^a	76.4 ± 5.2 ^a	76.8 ± 3.8 ^a	79.6 ± 4.8 ^b
4	<i>C. alata</i> 400mg	76.8 ± 2.6 ^a	77.4 ± 3.8 ^a	79.6 ± 4.2 ^a	81.6 ± 8.2 ^b
5	<i>C. alata</i> 600mg	74.8 ± 3.1 ^a	76.4 ± 3.2 ^a	77.4 ± 3.7 ^a	78.6 ± 4.2 ^b
6	<i>C. alata</i> 1000mg	73.6 ± 3.0 ^a	76.4 ± 3.6 ^a	76.8 ± 8.2 ^a	77.0 ± 6.2 ^b

Table: 12 Effect of continuous administration of alcoholic extract of *C. alata* leaf on food intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Food intake (g/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	74.3 ± 2.6 ^a	75.2 ± 2.8 ^a	75.8 ± 3.6 ^a	76.4 ± 2.8 ^a
2	<i>C. alata</i> 100mg	73.6 ± 3.8 ^a	76.4 ± 3.1 ^a	77.6 ± 2.1 ^a	77.4 ± 3.1 ^b
3	<i>C. alata</i> 200mg	75.6 ± 2.8 ^a	76.2 ± 2.6 ^a	77.8 ± 3.6 ^a	78.6 ± 4.8 ^b
4	<i>C. alata</i> 400mg	76.4 ± 3.6 ^a	77.4 ± 5.6 ^a	78.6 ± 4.2 ^a	78.8 ± 5.2 ^b
5	<i>C. alata</i> 600mg	75.8 ± 2.8 ^a	76.4 ± 3.1 ^a	78.0 ± 5.0 ^a	79.6 ± 3.2 ^b
6	<i>C. alata</i> 1000mg	74.4 ± 3.1 ^a	76.4 ± 3.6 ^a	77.1 ± 4.2 ^b	77.9 ± 5.2 ^b

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 continuous administration of dry aqueous extract of *C. alata* leaf on water intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Water intake (L/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	4.26 ± 0.35	4.28 ± 0.58	4.44 ± 0.36	1.48 ± 0.50
2	<i>C. alata</i> 100mg	4.12 ± 0.44	4.20 ± 0.38	4.22 ± 0.64	4.28 ± 0.20
3	<i>C. alata</i> 200mg	4.08 ± 0.26	4.16 ± 0.60	4.20 ± 0.72	4.20 ± 0.36
4	<i>C. alata</i> 400mg	4.14 ± 0.12	4.18 ± 0.22	4.20 ± 0.32	4.26 ± 0.44
5	<i>C. alata</i> 600mg	4.20 ± 0.18 ^a	4.22 ± 0.22 ^a	4.26 ± 0.16 ^a	4.30 ± 0.16 ^b
6	<i>C. alata</i> 1000mg	4.21 ± 0.22 ^a	4.26 ± 0.03 ^a	4.30 ± 0.42 ^a	4.36 ± 0.52 ^b

Table: 14 Effect of continuous administration of alcoholic extract of *C. alata* leaf on water intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Water intake (L/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	4.20 ± 0.58	4.12 0.62	4.26 ± 0.72	4.30 ± 0.80
2	<i>C. alata</i> 100mg	4.12 ± 0.22	4.16 ± 0.16	4.20 ± 0.20	4.22 ± 0.42
3	<i>C. alata</i> 200mg	4.10 ± 0.22	4.16 ± 0.26	4.22 ± 0.32	4.36 ± 0.28
4	<i>C. alata</i> 400mg	4.32 ± 0.16	4.36 ± 0.16	4.38 ± 0.28	4.40 ± 0.46
5	<i>C. alata</i> 600mg	4.26 ± 0.20 ^a	4.28 ± 0.16 ^a	4.29 ± 0.22 ^a	4.31 ± 0.31 ^b
6	<i>C. alata</i> 1000mg	4.22 ± 0.48 ^a	4.26 ± 0.49 ^a	4.31 ± 0.50 ^a	4.32 ± 0.64 ^b

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. alata* dry aqueous leaf extract on blood glucose level in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	1h	2h	3h
1	Control (received 2% gum acacia)	69.6 ± 5.25 ^a	68.7 ± 5.5 ^a	67.8 ± 3.6 ^a	66.8 ± 4.0 ^a
2	Diabetic control	245.8 ± 7.4 ^a	244.6 ± 10.6 ^a	242.6 ± 12.4 ^a	242.4 ± 12.0 ^a
3	Diabetic + <i>C. alata</i> 400mg	248.6 ± 12.4 ^a	246.6 ± 11.4 ^a	240.8 ± 8.6 ^b	232.8 ± 11.0 ^c
4	Diabetic + <i>C. alata</i> 600mg	246.4 ± 8.2 ^a	240.8 ± 8.8 ^b	236.4 ± 11.4 ^c	232.8 ± 12.8 ^d
5	Diabetic + glibenclamide (600 µg/ kg body weight)	252.4 ± 8.5 ^a	248.6 ± 11.5 ^b	244.8 ± 11.2 ^c	231.4 ± 11.6 ^d

Table: 16 Effect of continuous administration of *C. alata* dry aqueous leaf extract for 15 days on blood glucose level in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	68.4 ± 6.3 ^a	67.6 ± 4.3 ^a	67.6 ± 7.7 ^a	70.6 ± 6.4 ^a
2	Diabetic control	252.4 ± 11.4 ^a	258.6 ± 10.4 ^a	260.6 ± 8.8 ^a	262.8 ± 10.4 ^b
3	Diabetic + <i>C. alata</i> 400mg	28.6 ± 10.8 ^a	245.6 ± 7.5 ^a	243.4 ± 12.6 ^b	238.6 ± 11.4 ^c
4	Diabetic + <i>C. alata</i> 600mg	253.8 ± 6.8 ^a	249.6 ± 11.8 ^b	241.6 ± 9.6 ^c	233.2 ± 8.4 ^d
5	Diabetic + glibenclamide (600 µg/ kg body weight)	252.8 ± 8.5 ^a	246.4 ± 11.4 ^b	240.6 ± 4.8 ^c	32.4 ± 4.6 ^d

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. alata* dry aqueous leaf extract on TBARS in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol/ml)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	1.83 ± 0.3	1.80 ± 0.26	1.79 ± 0.33	1.75 ± 0.65
2	Diabetic control	2.58 ± 0.28 ^a	2.62 ± 0.36 ^b	2.68 ± 0.42 ^c	2.72 ± 0.66 ^d
3	Diabetic + <i>C. alata</i> 400mg	2.52 ± 0.36 ^a	2.50 ± 0.16 ^a	2.48 ± 0.60 ^b	2.22 ± 0.62 ^c
4	Diabetic + <i>C. alata</i> 600mg	2.50 ± 0.26 ^a	2.46 ± 0.36 ^a	2.38 ± 0.42 ^b	2.16 ± 0.16 ^c
5	Diabetic + glibenclamide (600 µg/ kg body weight)	2.55 ± 0.36 ^a	2.52 ± 0.46 ^a	2.38 ± 0.46 ^b	2.22 ± 0.16 ^c

Table: 18 Effect of *C. alata* dry aqueous leaf extract on body weight changes in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Body weight changes (gm)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	156 ± 10.6	157.6 ± 8.8	156.6 ± 10.8	158.6 ± 6.6
2	Diabetic control	160.4 ± 8.6 ^a	158.6 ± 6.6 ^a	155.4 ± 8.8 ^a	153.6 ± 6.8 ^b
3	Diabetic + <i>C. alata</i> 400mg	162.6 ± 4.8 ^a	163.6 ± 8.0 ^a	163.8 ± 5.8 ^a	165.5 ± 4.4 ^b
4	Diabetic + <i>C. alata</i> 600mg	158.4 ± 8.8 ^a	159.8 ± 6.6 ^a	160.6 ± 5.5 ^a	164.8 ± 8.8 ^b
5	Diabetic + glibenclamide (600 µg/ kg body weight)	158.6 ± 6.6 ^a	160.6 ± 13.1 ^a	163.8 ± 8.8 ^b	165.6 ± 10.8 ^c

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: 19 Effect of *C. alata* dry aqueous leaf extract on Urea level in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Blood Urea (mg/dl)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	17.8 ± 1.2 ^a	18.6 ± 1.8 ^a	18.4 ± 1.8 ^a	18.0 ± 1.2 ^a
2	Diabetic control	56.4 ± 1.8 ^a	60.2 ± 2.2 ^b	64.6 ± 3.6 ^c	68.4 ± 2.4 ^d
3	Diabetic + <i>C. alata</i> 400mg	58.6 ± 2.2 ^a	54.3 ± 2.6 ^b	52.8 ± 1.4 ^c	46.4 ± 1.8 ^d
4	Diabetic + <i>C. alata</i> 600mg	60.4 ± 3.1 ^a	56.4 ± 3.6 ^b	52.4 ± 4.2 ^c	42.4 ± 3.6 ^d
5	Diabetic + glibenclamide (600 µg/ kg body weight)	61.6 ± 3.0 ^a	54.2 ± 4.2 ^b	50.4 ± 3.6 ^c	41.6 ± 4.2 ^d

Table: 20 Effect of *C. alata* dry aqueous leaf extract on Uric acid level in streptozotocin diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Uric acid (n mol/ml)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	2.66 ± 0.02 ^a	2.74 ± 0.06 ^a	2.75 ± 0.08 ^a	2.77 ± 0.06 ^a
2	Diabetic control	13.82 ± 1.20 ^a	14.88 ± 1.26 ^b	16.42 ± 1.38 ^c	22.42 ± 3.82 ^d
3	Diabetic + <i>C. alata</i> 400mg	14.40 ± 0.52 ^a	12.64 ± 1.32 ^b	10.43 ± 1.22 ^c	8.40 ± 0.52 ^d
4	Diabetic + <i>C. alata</i> 600mg	14.62 ± 0.26 ^a	10.64 ± 1.22 ^b	8.64 ± 0.56 ^c	4.32 ± 0.36 ^d
5	Diabetic + glibenclamide (600 µg/ kg body weight)	13.82 ± 0.22 ^a	9.42 ± 0.62 ^b	7.46 ± 0.56 ^c	3.66 ± 0.36 ^d

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. alata* fresh aqueous leaf extract on blood glucose levels in normal fasted rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Fasting	1h	2h	3h
1	Control (received 2% gum acacia)	66.6 ± 0.9 ^a	66.3 ± 0.94 ^a	66.4 ± 1.6 ^a	65.3 ± 1.2 ^b
2	<i>C. alata</i> 100mg	66.8 ± 1.17 ^a	66.1 ± 0.99 ^a	65.9 ± 1.7 ^a	65.05 ± 1.8 ^b
3	<i>C. alata</i> 200mg	67.3 ± 1.50 ^a	66.2 ± 1.5 ^a	65.7 ± 1.16 ^b	65.0 ± 1.5 ^b
4	<i>C. alata</i> 400mg	67.07 ± 1.0 ^a	66.2 ± 1.5 ^a	65.2 ± 1.7 ^c	64.4 ± 1.6 ^c
5	<i>C. alata</i> 600mg	68.4 ± 1.57 ^a	67.5 ± 2.0 ^b	66.5 ± 1.6 ^c	65.8 ± 1.38 ^c
6	<i>C. alata</i> 1000mg	68.4 ± 1.57 ^a	66.8 ± 1.2 ^b	66.3 ± 1.45 ^c	65.3 ± 1.63 ^c

Table: 22 Effect of continuous administration of aqueous fresh extract of *C. alata* on blood glucose levels in normal fasted rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)			
		Initial Day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	67.7 ± 0.25 ^a	67.5 ± 0.25 ^a	66.8 ± 0.52 ^a	66.6 ± 0.56 ^a
2	<i>C. alata</i> 100mg	67.8 ± 0.71 ^a	67.2 ± 0.6 ^a	66.5 ± 0.30 ^b	50.5 ± 29.8 ^b
3	<i>C. alata</i> 200mg	68.0 ± 1.0 ^a	67.4 ± 1.07 ^a	66.6 ± 1.27 ^b	65.9 ± 0.7 ^b
4	<i>C. alata</i> 400mg	67.4 ± 0.86 ^a	66.2 ± 1.0 ^b	65.5 ± 0.70 ^c	65.0 ± 0.67 ^d
5	<i>C. alata</i> 600mg	68.6 ± 1.5 ^a	67.8 ± 1.2 ^b	67.3 ± 1.16 ^c	66.3 ± 1.08 ^d
6	<i>C. alata</i> 1000mg	70.05 ± 1.9 ^a	68.9 ± 2.01 ^b	68.1 ± 1.99 ^c	66.9 ± 2.1 ^d

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: 23 Effect of *C. alata* aqueous fresh 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	70.3 ± 1.8 ^a	165.3 ± 2.3 ^b	163.15 ± 1.9 ^a	158.4 ± 30 ^a
2	<i>C. alata</i> 100mg + glucose	67.5 ± 1.8 ^a	168.0 ± 2.3 ^b	161.9 ± 0.46 ^b	154.3 ± 1.2 ^c
3	<i>C. alata</i> 200mg + glucose	67.1 ± 1.9 ^a	154.8 ± 4.1 ^b	149.8 ± 3.4 ^b	145.3 ± 3.5 ^b
4	<i>C. alata</i> 400mg + glucose	67.5 ± 2.1 ^a	159.8 ± 2.7 ^b	155.9 ± 2.8 ^c	149.6 ± 3.1 ^c
5	<i>C. alata</i> 600mg + glucose	66.8 ± 1.7 ^a	165.8 ± 2.3 ^b	159.0 ± 0.85 ^c	152.8 ± 2.4 ^d
6	<i>C. alata</i> 1000mg + glucose	66.7 ± 1.5 ^a	167.0 ± 1.17 ^b	160.9 ± 1.05 ^c	153.6 ± 1.4 ^d

Table: 24 Effect of *C. alata* aqueous fresh leaf extract on oral glucose tolerance test in normal fasted rats after 15 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)	66.5 ± 1.6 ^a	169.9 ± 3.5 ^b	167.9 ± 3.9 ^b	164.7 ± 2.8 ^b
2	<i>C. alata</i> 100mg	66.9 ± 1.8 ^a	166.9 ± 0.55 ^b	160.7 ± 1.2 ^b	155.3 ± 2.47 ^c
3	<i>C. alata</i> 200mg	66.8 ± 1.6 ^a	163.8 ± 2.7 ^b	156.2 ± 1.6 ^b	150.5 ± 1.5 ^b
4	<i>C. alata</i> 400mg	67.05 ± 1.2 ^a	152.1 ± 12.1 ^b	146.4 ± 12.2 ^b	138.9 ± 12.2 ^c
5	<i>C. alata</i> 600mg	66.2 ± 1.6 ^a	149.8 ± 12.9 ^b	144.0 ± 12.2 ^c	136.5 ± 12.9 ^d
6	<i>C. alata</i> 1000mg	64.1 ± 2.0 ^a	160.5 ± 5.9 ^b	157.0 ± 4.4 ^c	152.8 ± 3.1 ^d

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: 25 Effect of continuous administration of *C. alata* aqueous fresh extract on body weight changes in normal rats.

Groups	Treatment (Dose/Kg body weight)	Body weight changes (gm)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	156.4 ± 1.3 ^a	157.2 ± 1.4 ^a	157.7 ± 1.6 ^a	158.5 ± 1.9 ^a
2	<i>C. alata</i> 100mg	156.4 ± 0.8 ^a	157.0 ± 0.71 ^a	157.4 ± 0.59 ^a	158.0 ± 0.63 ^b
3	<i>C. alata</i> 200mg	156.9 ± 1.3 ^a	157.2 ± 1.3 ^a	157.7 ± 1.35 ^a	158.4 ± 1.45 ^b
4	<i>C. alata</i> 400mg	155.9 ± 2.06 ^a	156.5 ± 2.12 ^a	157.1 ± 2.4 ^a	157.6 ± 2.3 ^b
5	<i>C. alata</i> 600mg	154.2 ± 1.08 ^a	154.6 ± 1.07 ^a	155.1 ± 1.06 ^a	155.6 ± 1.07 ^b
6	<i>C. alata</i> 1000mg	154.3 ± 1.89 ^a	154.8 ± 1.7 ^a	155.5 ± 1.5 ^a	156.0 ± 1.6 ^b

Table: 26 Effect of continuous administration of aqueous fresh extract of *C. alata* leaf on food intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Food intake (g/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	77.3 ± 0.8 ^a	76.4 ± 0.9 ^a	76.0 ± 0.67 ^a	75.3 ± 0.69 ^b
2	<i>C. alata</i> 100mg	76.6 ± 0.9 ^a	77.0 ± 0.9 ^a	77.5 ± 1.08 ^a	77.9 ± 0.98 ^b
3	<i>C. alata</i> 200mg	77.3 ± 1.7 ^a	77.5 ± 1.7 ^a	77.8 ± 1.65 ^a	77.8 ± 1.65 ^b
4	<i>C. alata</i> 400mg	74.1 ± 1.2 ^a	74.4 ± 1.3 ^a	74.7 ± 1.11 ^a	75.1 ± 1.2 ^b
5	<i>C. alata</i> 600mg	76.6 ± 1.07 ^a	77.1 ± 0.77 ^a	77.4 ± 0.75 ^a	77.7 ± 0.8 ^b
6	<i>C. alata</i> 1000mg	75.1 ± 0.9 ^a	75.5 ± 0.84 ^a	75.7 ± 0.8 ^b	76.1 ± 0.77 ^b

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: 27 Effect of continuous administration of aqueous fresh extract of *C. alata* leaf on water intake in normal rats.

Groups	Treatment (Dose/Kg body weight)	Water intake (L/week)			
		Initial day	4 th Day	7 th Day	15 th Day
1	Control (received 2% gum acacia)	4.2 ± 0.02	4.5 ± 0.44	4.55 ± 0.45	4.57 ± 0.046
2	<i>C. alata</i> 100mg	6.6 ± 5.01	4.17 ± 0.019	4.19 ± 0.019	4.21 ± 0.019
3	<i>C. alata</i> 200mg	4.09 ± 0.02	4.13 ± 0.019	4.15 ± 0.019	4.17 ± 0.022
4	<i>C. alata</i> 400mg	4.14 ± 0.01	4.16 ± 0.010	4.19 ± 0.011	4.21 ± 0.015
5	<i>C. alata</i> 600mg	4.13 ± 0.02 ^a	4.16 ± 0.028 ^a	4.18 ± 0.034 ^a	4.20 ± 0.028 ^b
6	<i>C. alata</i> 1000mg	4.15 ± 0.03 ^a	4.17 ± 0.025 ^a	4.19 ± 0.025 ^a	4.21 ± 0.025 ^a

Table: 28 Effect of *C. alata* aqueous fresh 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)	68.9 ± 1.04 ^a	68.1 ± 1.18 ^a	67.5 ± 1.2 ^a	66.9 ± 1.3 ^a
2	Diabetic control	249.4 ± 2.9 ^a	247.5 ± 2.58 ^a	246.4 ± 1.8 ^a	245.6 ± 1.5 ^a
3	Diabetic + <i>C. alata</i> 400mg	247.4 ± 1.16 ^a	246.4 ± 1.27 ^a	245.8 ± 1.3 ^b	245.1 ± 1.16 ^c
4	Diabetic + <i>C. alata</i> 600mg	247.6 ± 0.97 ^a	246.9 ± 1.11 ^b	246.2 ± 1.14 ^c	245.2 ± 0.89 ^d
5	Diabetic + glibenclamide (600 µg/ kg body weight)	251.3 ± 2.7 ^a	249.5 ± 2.60 ^b	247.7 ± 2.48 ^c	246.2 ± 2.35 ^d

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: 29 Effect of continuous administration of *C. alata* aqueous fresh leaf extract for 15 days on blood glucose level in alloxan diabetic rats.

Groups	Treatment (Dose/Kg body weight)	Blood glucose (mg/dl)		
		Initial day	4 th Day	7 th Day
1	Control (received 2% gum acacia)	68.4 ± 0.78 ^a	67.6 ± 0.90 ^a	66.8 ± 1.14 ^a
2	Diabetic control	252.6 ± 1.64 ^a	254.7 ± 1.55 ^a	256.6 ± 1.72 ^a
3	Diabetic + <i>C. alata</i> 400mg	230.5 ± 5.24 ^a	233.5 ± 4.18 ^a	235.5 ± 3.5 ^b
4	Diabetic + <i>C. alata</i> 600mg	251.3 ± 7.30 ^a	250.1 ± 7.86 ^b	247.6 ± 7.5 ^c
5	Diabetic + glibenclamide (600 µg/ kg body weight)	252.5 ± 2.8 ^a	250.4 ± 2.6 ^b	248.4 ± 2.7 ^c
				246.4 ± 2.64 ^d

Table: 30 Effect of *C. alata* dry leaf extract on Blood glucose level, Body weight and Hemoglobin percentage on control and STZ induced diabetic rats

Groups	Treatment (Dose/Kg body weight)	Blood glucose (Initial)	Blood glucose (Final)	Body weight (Initial)	Body weight (Final)	Hemoglobin percentage
1	Control (received 2% gum acacia)	68.46 ± 5.8	71.4 ± 6.6 ^a	156.4 ± 10.6	176.8 ± 4.8 ^a	15.30 ± 2.50 ^a
2	Diabetic control	252.4 ± 11.4	316.4 ± 10.8 ^b	160.4 ± 8.6	130.6 ± 5.5 ^b	10.8 ± 1.6 ^b
3	Normal + <i>C. alata</i> 1	66.4 ± 6.8	69.8 ± 6.4 ^a	160.8 ± 9.6	164.6 ± 4.6 ^c	15.26 ± 3.6 ^a
4	Normal + <i>C. alata</i> 2	69.8 ± 8.4	71.1 ± 8.4 ^a	156.0 ± 6.4	159.8 ± 8.8 ^c	15.18 ± 2.4 ^a
5	Diabetic + <i>C. alata</i> 1	248.6 ± 10.8	186.4 ± 14.2 ^c	162.6 ± 4.8	172.6 ± 4.8 ^d	12.35 ± 0.38 ^c
6	Diabetic + <i>C. alata</i> 2	253.8 ± 6.8	156.6 ± 9.6 ^d	158.4 ± 8.8	168.6 ± 6.6 ^e	13.70 ± 1.06 ^d
7	Diabetic + glibenclamide (600µg/kg body weight)	252.8 ± 8.5	144.8 ± 6.4 ^e	158.6 ± 6.6	172.6 ± 9.0 ^e	13.96 ± 1.83 ^d

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: 31 Effect of *C. alata* dry leaf extract on the levels of Plasma TBARS, GSH, Vit C and Vit E on control and STZ 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)	2.2 ± 0.6 ^a	24.5 ± 1.8 ^a	1.80 ± 0.16 ^a	1.08 ± 0.03 ^a
2	Diabetic control	3.8 ± 0.86 ^b	14.8 ± 0.9 ^b	0.98 ± 0.2 ^b	0.74 ± 0.02 ^b
3	Normal + <i>C. alata</i> 1	2.1 ± 0.44 ^a	26.4 ± 1.3 ^a	1.82 ± 0.32 ^a	1.09 ± 0.04 ^a
4	Normal + <i>C. alata</i> 2	2.0 ± 0.35 ^a	27.8 ± 1.8 ^a	1.83 ± 0.42 ^a	1.12 ± 0.03 ^a
5	Diabetic + <i>C. alata</i> 1	2.95 ± 0.43 ^c	18.8 ± 0.88 ^c	1.36 ± 0.64 ^c	0.82 ± 0.04 ^c
6	Diabetic + <i>C. alata</i> 2	2.64 ± 0.64 ^d	20.6 ± 1.2 ^d	1.52 ± 0.48 ^d	0.88 ± 0.26 ^c
7	Diabetic + glibenclamide (600 µg)	2.52 ± 0.73 ^c	21.4 ± 1.3 ^d	1.6 ± 0.36 ^d	0.92 ± 0.28 ^d

Table: 32 Effect of *C. alata* dry leaf extract on the levels of Erythrocyte TBARS, GSH, Vit C and Vit E on control and STZ 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)	1.76 ± 0.26 ^a	72.60 ± 0.38 ^a	1.76 ± 0.23 ^a	1.08 ± 0.05 ^a
2	Diabetic control	3.88 ± 0.64 ^b	49.69 ± 2.8 ^b	0.99 ± 0.12 ^b	0.56 ± 0.04 ^b
3	Normal + <i>C. alata</i> 1	1.74 ± 0.24 ^a	73.6 ± 4.8 ^a	1.76 ± 0.42 ^a	1.09 ± 0.04 ^a
4	Normal + <i>C. alata</i> 2	1.70 ± 0.28 ^a	74.5 ± 3.8 ^a	1.76 ± 0.39 ^a	1.10 ± 0.05 ^a
5	Diabetic + <i>C. alata</i> 1	3.24 ± 0.58 ^c	58.8 ± 4.7 ^c	1.48 ± 0.34 ^c	0.72 ± 0.04 ^c
6	Diabetic + <i>C. alata</i> 2	3.02 ± 0.64 ^d	64.6 ± 5.8 ^d	1.56 ± 0.47 ^c	0.82 ± 0.06 ^d
7	Diabetic + glibenclamide (600µg/kg body weight)	2.96 ± 0.52 ^d	68.4 ± 5.4 ^c	1.62 ± 0.38 ^d	0.84 ± 0.05 ^d

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: 33 Effect of *C. alata* dry leaf extract on the levels of Liver TBARS, GSH, Vit C and Vit E on control and STZ induced diabetic rats

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol/ml)	GSH (μ g/dl)	Vitamin C (μ g/dl)	Vitamin E (μ g/dl)
1	Control (received 2% gum acacia)	1.20 $\pm$ 0.16 ^a	13.82 $\pm$ 0.88 ^a	0.64 $\pm$ 0.06 ^a	5.26 $\pm$ 0.14 ^a
2	Diabetic control	3.88 $\pm$ 0.18 ^b	9.26 $\pm$ 0.88 ^b	0.42 $\pm$ 0.05 ^b	3.46 $\pm$ 0.44 ^b
3	Normal + <i>C. alata</i> 1	1.18 $\pm$ 0.09 ^a	14.08 $\pm$ 0.86 ^a	0.65 $\pm$ 0.04 ^a	5.32 $\pm$ 0.26 ^a
4	Normal + <i>C. alata</i> 2	1.16 $\pm$ 0.05 ^a	14.26 $\pm$ 0.78 ^a	0.66 $\pm$ 0.06 ^a	5.34 $\pm$ 0.44 ^a
5	Diabetic + <i>C. alata</i> 1	1.88 $\pm$ 0.10 ^c	11.62 $\pm$ 0.88 ^c	0.78 $\pm$ 0.07 ^c	4.88 $\pm$ 0.58 ^c
6	Diabetic + <i>C. alata</i> 2	1.58 $\pm$ 0.16 ^d	12.24 $\pm$ 0.56 ^d	0.60 $\pm$ 0.06 ^c	4.96 $\pm$ 0.72 ^d
7	Diabetic + glibenclamide (600 μ g)	1.42 $\pm$ 0.12 ^c	12.88 $\pm$ 0.62 ^d	0.62 $\pm$ 0.07 ^d	5.16 $\pm$ 0.52 ^c

Table: 34 Effect of *C. alata* dry leaf extract on the levels of Kidney TBARS, GSH, Vit C and Vit E on control and STZ induced diabetic rats

Groups	Treatment (Dose/Kg body weight)	TBARS (n mol/ml)	GSH (μ g/dl)	Vitamin C (μ g/dl)	Vitamin E (μ g/dl)
1	Control (received 2% gum acacia)	1.32 $\pm$ 0.05 ^a	7.26 $\pm$ 0.65 ^a	0.56 $\pm$ 0.16 ^a	3.42 $\pm$ 0.40 ^a
2	Diabetic control	3.88 $\pm$ 0.22 ^b	4.24 $\pm$ 0.38 ^b	0.32 $\pm$ 0.10 ^b	1.46 $\pm$ 0.58 ^b
3	Normal + <i>C. alata</i> 1	1.30 $\pm$ 0.26 ^a	7.30 $\pm$ 0.28 ^a	0.58 $\pm$ 0.12 ^a	3.40 $\pm$ 0.38 ^a
4	Normal + <i>C. alata</i> 2	1.32 $\pm$ 0.20 ^a	7.32 $\pm$ 0.35 ^a	0.59 $\pm$ 0.14 ^a	3.46 $\pm$ 0.52 ^a
5	Diabetic + <i>C. alata</i> 1	1.86 $\pm$ 0.10 ^c	5.58 $\pm$ 0.36 ^c	0.48 $\pm$ 0.13 ^c	2.82 $\pm$ 0.32 ^c
6	Diabetic + <i>C. alata</i> 2	1.56 $\pm$ 0.24 ^d	6.46 $\pm$ 0.58 ^d	0.52 $\pm$ 0.10 ^d	3.08 $\pm$ 0.21 ^c
7	Diabetic + glibenclamide (600 μ g)	1.48 $\pm$ 0.36 ^c	6.78 $\pm$ 0.50 ^d	0.53 $\pm$ 0.11 ^d	3.16 $\pm$ 0.28 ^d

Values are means $\pm$ 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: 35 Effect of *C. alata* dry leaf extract on the activity of SOD, CAT and GPx in the liver of control and STZ 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)	9.36 ± 0.68 ^a	72.55 ± 4.34 ^a	5.48 ± 0.43 ^a
2	Diabetic control	5.42 ± 0.37 ^b	36.42 ± 3.82 ^b	3.26 ± 0.25 ^b
3	Normal + <i>C. alata</i> 1	9.44 ± 0.36 ^a	74.60 ± 3.80 ^a	5.55 ± 0.38 ^a
4	Normal + <i>C. alata</i> 2	9.48 ± 0.88 ^a	75.42 ± 4.40 ^a	5.57 ± 0.41 ^a
5	Diabetic + <i>C. alata</i> 1	7.52 ± 0.39 ^c	58.64 ± 5.80 ^c	4.96 ± 0.54 ^c
6	Diabetic + <i>C. alata</i> 2	8.42 ± 0.58 ^d	64.5 ± 4.38 ^d	5.21 ± 0.36 ^d
7	Diabetic + glibenclamide (600µg)	8.96 ± 0.64 ^d	68.6 ± 5.26 ^c	5.35 ± 0.32 ^c

Table: 36 Effect of *C. alata* dry leaf extract on the activity of SOD, CAT and GPx in the kidney of control and STZ 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)	13.58 ± 0.96 ^a	34.82 ± 1.58 ^a	3.66 ± 0.48 ^a
2	Diabetic control	6.57 ± 0.72 ^b	18.46 ± 0.68 ^b	5.98 ± 0.57 ^b
3	Normal + <i>C. alata</i> 1	13.62 ± 0.83 ^a	35.26 ± 1.58 ^a	3.58 ± 0.24 ^a
4	Normal + <i>C. alata</i> 2	13.82 ± 0.74 ^a	35.76 ± 1.36 ^a	3.52 ± 0.57 ^a
5	Diabetic + <i>C. alata</i> 1	11.43 ± 0.68 ^c	28.56 ± 1.58 ^c	4.86 ± 0.38 ^c
6	Diabetic + <i>C. alata</i> 2	12.58 ± 0.73 ^d	31.16 ± 1.32 ^d	4.24 ± 0.37 ^d
7	Diabetic + glibenclamide (600µg)	12.64 ± 0.82 ^d	32.24 ± 1.24 ^d	4.08 ± 0.36 ^d

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: 37 Effect of *Cassia alata* dry leaf extract on the activity of SOD, CAT and GPx in the erythrocyte of control and STZ 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)	6.78 ± 0.58 ^a	176.56 ± 1.6 ^a	14.28 ± 1.58 ^a
2	Diabetic control	3.64 ± 0.64 ^b	92.58 ± 10.64 ^b	8.52 ± 0.52 ^b
3	Normal + <i>C. alata</i> 1	6.82 ± 0.64 ^a	178.4 ± 6.8 ^a	14.36 ± 1.02 ^a
4	Normal + <i>C. alata</i> 2	6.84 ± 0.88 ^a	179.5 ± 5.8 ^a	14.44 ± 0.88 ^a
5	Diabetic + <i>C. alata</i> 1	4.88 ± 0.56 ^c	154.62 ± 12.6 ^c	11.64 ± 5.8 ^c
6	Diabetic + <i>C. alata</i> 2	5.46 ± 0.64 ^d	158.26 ± 8.84 ^c	12.86 ± 0.78 ^c
7	Diabetic + glibenclamide (600µg)	5.96 ± 0.72 ^c	162.4 ± 10.4 ^d	13.60 ± 0.88 ^d

Table: 38 Effect of *C. alata* dry leaf extract on lipid profile in control and STZ 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)	31.42 ± 0.28 ^a	42.85 ± 3.82 ^a	12.42 ± 0.88 ^a
2	Diabetic control	22.62 ± 0.32 ^b	182.65 ± 10.82 ^b	22.46 ± 3.62 ^b
3	Normal + <i>C. alata</i> 1	30.62 ± 0.42 ^a	43.85 ± 3.25 ^a	13.82 ± 1.28 ^a
4	Normal + <i>C. alata</i> 2	31.82 ± 0.38 ^a	44.86 ± 2.82 ^a	13.96 ± 1.24 ^a
5	Diabetic + <i>C. alata</i> 1	25.52 ± 3.80 ^c	120.82 ± 9.62 ^c	16.62 ± 3.82 ^c
6	Diabetic + <i>C. alata</i> 2	26.42 ± 4.28 ^d	90.42 ± 6.60 ^d	15.86 ± 2.82 ^c
7	Diabetic + glibenclamide (600µg)	27.38 ± 1.22 ^d	78.82 ± 3.82 ^c	14.96 ± 2.42 ^c

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: 39 Effect of *C. alata* dry leaf extract on the level of Cholesterol, Free fatty acids, Phospholipids and Triglycerides on control and STZ 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)	60.46 ± 5.89 ^a	52.46 ± 2.52 ^a	122.40 ± 6.10 ^a	54.30±4.2 ^a
2	Diabetic control	210.52 ± 4.30 ^b	132.4 ± 5.82 ^b	210.4 ± 10.42 ^b	138.40±3.20 ^b
3	Normal + <i>C. alata</i> 1	62.46 ± 0.82 ^a	54.38 ± 4.45 ^a	120.42 ± 8.50 ^a	55.40±4.30 ^a
4	Normal + <i>C. alata</i> 2	61.82 ± 2.60 ^a	53.36 ± 5.26 ^a	121.40 ± 6.50 ^a	118.40±3.60 ^a
5	Diabetic + <i>C. alata</i> 1	159.42 ± 4.60 ^c	110.42 ± 4.28 ^c	170.60 ± 7.40 ^c	88.4±3.60 ^c
6	Diabetic + <i>C. alata</i> 2	132.42 ± 4.25 ^d	98.28 ± 4.20 ^d	148.36±12.40 ^d	82.42±3.80 ^c
7	Diabetic + glibenclamide (600µg/kg body weight)	124.8 ± 5.82 ^c	76.82 ± 3.60 ^c	140.28±11.30 ^c	60.40±6.6 ^d

Table: 40 Effect of *C. alata* fresh leaf extract on blood glucose level and body weight in alloxan induced diabetic rats

Groups	Treatment (Dose/Kg body weight)	Blood glucose (initial)	Blood glucose (final)	Body weight (initial)	Body weight (final)
1	Control (received 2% gum acacia)	82.3 ± 1.7	94 ± 4.9 ^a	186.3 ± 1.3	187.8 ± 1.7 ^a
2	Diabetic + control	257.0 ± 6.3	314 ± 28.3 ^b	190.5 ± 4.2	137.5 ± 8.7 ^b
3	Diabetic + <i>C. alata</i> (400mg)	268.0 ± 15.1	152 ± 8.4 ^c	157.8 ± 5.9	159.3 ± 5.7 ^c
4	Diabetic + <i>C. alata</i> (600mg)	283.5 ± 1.9	136 ± 5.16 ^d	181.8 ± 14.3	184.8 ± 15.9 ^d
5	Diabetic + Glibenclamide (600µg/kg body weight)	290.5 ± 10.2	135 ± 17.3 ^d	185.8 ± 3.3	187.0 ± 3.7 ^d

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: 41 Effect of *C. alata* fresh leaf extract 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 (μ g /dl)	Vitamin C (μ g /dl)	Vitamin E (μ g /dl)
1	Control (received 2% gum acacia)	1.58 $\pm$ 0.56 ^a	25.8 $\pm$ 2.5 ^a	1.9 $\pm$ 0.18 ^a	0.71 $\pm$ 0.010 ^a
2	Diabetic + control	3.6 $\pm$ 0.39 ^b	14.9 $\pm$ 2.3 ^b	0.8 $\pm$ 0.09 ^b	1.06 $\pm$ 0.017 ^b
3	Diabetic + <i>C. alata</i> (400mg)	2.4 $\pm$ 0.87 ^c	23.2 $\pm$ 2.5 ^c	1.5 $\pm$ 0.10 ^c	0.82 $\pm$ 0.013 ^c
4	Diabetic + <i>C. alata</i> (600mg)	2.05 $\pm$ 0.69 ^c	24.2 $\pm$ 3.03 ^d	1.6 $\pm$ 0.13 ^d	0.77 $\pm$ 0.029 ^d
5	Diabetic + Glibenclamide (600 μ g/kg body weight)	1.8 $\pm$ 0.66 ^d	24.8 $\pm$ 2.7 ^d	1.7 $\pm$ 0.18 ^d	0.80 $\pm$ 0.008 ^d

Table: 42 Effect of *C. alata* fresh leaf extract 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 (μ g /dl)	Vitamin C (μ g /dl)	Vitamin E (μ g /dl)
1	Control (received 2% gum acacia)	1.5 $\pm$ 0.52 ^a	18.04 $\pm$ 2.2 ^a	0.75 $\pm$ 0.01 ^a	5.53 $\pm$ 0.10 ^a
2	Diabetic + control	3.6 $\pm$ 0.39 ^b	7.2 $\pm$ 2.5 ^b	0.45 $\pm$ 0.03 ^b	3.23 $\pm$ 0.17 ^b
3	Diabetic + <i>C. alata</i> (400mg)	2.3 $\pm$ 0.71 ^c	14.4 $\pm$ 4.1 ^c	0.66 $\pm$ 0.02 ^c	4.28 $\pm$ 0.22 ^c
4	Diabetic + <i>C. alata</i> (600mg)	1.9 $\pm$ 0.79 ^d	15.5 $\pm$ 1.9 ^d	0.69 $\pm$ 0.01 ^d	4.70 $\pm$ 0.12 ^d
5	Diabetic + Glibenclamide (600 μ g/kg body weight)	1.7 $\pm$ 0.65 ^e	15.9 $\pm$ 3.5 ^d	0.71 $\pm$ 0.03 ^d	4.60 $\pm$ 0.46 ^d

Values are means $\pm$ 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: 43 Effect of *C. alata* fresh leaf extract 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 (μ g /dl)	Vitamin C (μ g /dl)	Vitamin E (μ g /dl)
1	Control (received 2% gum acacia)	1.6 $\pm$ 0.4 ^a	14.9 $\pm$ 3.6 ^a	0.68 $\pm$ 0.02 ^a	3.65 $\pm$ 0.19 ^a
2	Diabetic + control	3.7 $\pm$ 0.33 ^b	7.2 $\pm$ 2.5 ^b	0.30 $\pm$ 0.05 ^b	1.48 $\pm$ 0.10 ^b
3	Diabetic + <i>C. alata</i> (400mg)	2.2 $\pm$ 0.80 ^c	14.4 $\pm$ 4.1 ^c	0.51 $\pm$ 0.02 ^c	2.63 $\pm$ 0.13 ^c
4	Diabetic + <i>C. alata</i> (600mg)	1.9 $\pm$ 0.79 ^d	15.5 $\pm$ 1.9 ^d	0.53 $\pm$ 0.01 ^d	2.65 $\pm$ 0.19 ^d
5	Diabetic + Glibenclamide (600 μ g/kg body weight)	1.8 $\pm$ 0.66 ^e	15.9 $\pm$ 3.5 ^d	0.56 $\pm$ 0.01 ^e	2.98 $\pm$ 0.15 ^d

Table: 44 Effect of *C. alata* fresh leaf extract 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 (μ g /dl)	Vitamin C (μ g /dl)	Vitamin E (μ g /dl)
1	Control (received 2% gum acacia)	1.6 $\pm$ 0.70 ^a	17.5 $\pm$ 2.5 ^a	0.84 $\pm$ 0.04 ^a	5.45 $\pm$ 0.13 ^a
2	Diabetic + control	3.7 $\pm$ 0.65 ^b	8.2 $\pm$ 2.5 ^b	0.44 $\pm$ 0.04 ^b	3.38 $\pm$ 0.17 ^b
3	Diabetic + <i>C. alata</i> (400mg)	2.5 $\pm$ 0.62 ^c	13.9 $\pm$ 1.6 ^c	0.64 $\pm$ 0.01 ^c	4.46 $\pm$ 0.06 ^c
4	Diabetic + <i>C. alata</i> (600mg)	1.8 $\pm$ 0.66 ^d	14.4 $\pm$ 1.6 ^d	0.68 $\pm$ 0.02 ^d	4.88 $\pm$ 0.15 ^d
5	Diabetic + Glibenclamide (600 μ g/kg body weight)	0.17 $\pm$ 0.65 ^d	16.1 $\pm$ 2.3 ^e	0.69 $\pm$ 0.02 ^e	4.93 $\pm$ 0.28 ^d

Values are means $\pm$ 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: 45 Effect of *C. alata* fresh leaf extract 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)	11.7 ± 0.2 ^a	166 ± 34.8 ^a	8.7 ± 0.63 ^a
2	Diabetic + control	5.4 ± 1.7 ^b	71.5 ± 10.1 ^b	4.8 ± 0.79 ^b
3	Diabetic + <i>C. alata</i> (400mg)	10.2 ± 0.5 ^c	115.9 ± 10.9 ^c	7.1 ± 0.67 ^c
4	Diabetic + <i>C. alata</i> (600mg)	10.7 ± 0.4 ^d	125.6 ± 19.04 ^d	7.5 ± 0.67 ^d
5	Diabetic + Glibenclamide (600µg/kg body weight)	10.7 ± 0.37 ^d	130.5 ± 17.8 ^e	7.7 ± 0.97 ^e

Table: 46 Effect of *C. alata* fresh leaf extract 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)	12.01 ± 0.32 ^a	163.2 ± 24.3 ^a	8.9 ± 1.8 ^a
2	Diabetic + control	4.3 ± 0.68 ^b	68.7 ± 8.24 ^b	4.1 ± 0.78 ^b
3	Diabetic + <i>C. alata</i> (400mg)	10.4 ± 0.27 ^c	114.6 ± 11.3 ⁺	6.9 ± 0.67 ^c
4	Diabetic + <i>C. alata</i> (600mg)	10.4 ± 0.16 ^c	132.6 ± 14.5 ^d	7.7 ± 0.53 ^d
5	Diabetic + Glibenclamide (600µg/kg body weight)	10.7 ± 0.38 ^d	134 ± 13.2 ^d	8.0 ± 0.82 ^e

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: 47 Effect of *C. alata* fresh leaf extract 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 ^a	75.7 ± 7.17 ^a	3.8 ± 0.50 ^a
2	Diabetic + control	4.6 ± 1.17 ^b	32.6 ± 6.6 ^b	7.2 ± 0.8 ^b
3	Diabetic + <i>C. alata</i> (400mg)	10.1 ± 0.21 ^c	53.4 ± 4.8 ^c	4.6 ± 0.73 ^c
4	Diabetic + <i>C. alata</i> (600mg)	10.4 ± 0.20 ^c	63.2 ± 3.1 ^d	4.1 ± 1.06 ^d
5	Diabetic + Glibenclamide (600µg/kg body weight)	10.6 ± 0.21 ^d	69.4 ± 5.6 ^e	4.0 ± 0.59 ^d

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: 48 Effect of *C. alata* fresh leaf extract on Hexokinase, Glucose-6-phosphatase and Fructose-1, 6-bisphosphatase of alloxan induced diabetic rats

Groups	Treatment (Dose/Kg body weight)	Hexokinase (U ^a / mg protein)	Glucose-6-phosphatase (U ^b /mg protein)	Fructose-1, 6-Bisphosphatase (U ^c /mg protein)
1	Control (received 2% gum acacia)	0.22 ± 0.012 ^a	0.15 ± 0.01 ^a	0.55 ± 0.02 ^a
2	Diabetic + control	0.11 ± 0.013 ^b	0.31 ± 0.05 ^b	1.08 ± 0.04 ^b
3	Diabetic + <i>C. alata</i> (400mg)	0.17 ± 0.016 ^c	0.18 ± 0.009 ^c	0.70 ± 0.033 ^c
4	Diabetic + <i>C. alata</i> (600mg)	0.20 ± 0.026 ^d	0.18 ± 0.01 ^d	0.63 ± 0.032 ^d
5	Diabetic + Glibenclamide (600µg/kg body weight)	0.20 ± 0.023 ^d	0.17 ± 0.017 ^e	0.60 ± 0.05 ^e

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

^a µmol of glucose phosphorylated/h

^b µmol of liberated / min

^c µmol of pi liberated / min

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

Duncan's Multiple Range Test (DMRT)

Table: 49 Effect of *C. alata* fresh leaf extract on plasma Urea, Uric acid, creatinine, and BUN on alloxan 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)	19.60 ± 5.83 ^a	3.05 ± 0.41 ^a	0.70 ± 0.40 ^a	9.1 ± 2.7 ^a
2	Diabetic + control	66.04 ± 6.8 ^b	20.5 ± 0.87 ^b	4.3 ± 1.05 ^b	3.08 ± 3.19 ^b
3	Diabetic + <i>C. alata</i> (400mg)	39.6 ± 6.8 ^c	9.1 ± 1.16 ^c	1.2 ± 1.3 ^c	18.5 ± 3.19 ^c
4	Diabetic + <i>C. alata</i> (600mg)	34.1 ± 3.8 ^d	7.7 ± 1.01 ^d	0.9 ± 0.47 ^d	15.9 ± 1.81 ^d
5	Diabetic + Glibenclamide(600µg/kg body weight)	33.9 ± 2.27 ^d	7.06 ± 1.03 ^e	0.9 ± 0.48 ^d	15.8 ± 1.06 ^d

Table: 50 Effect of *C. alata* fresh leaf extract on plasma Cholesterol, Free fatty acid, Phospholipids and Triglycerides on alloxan 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 ^a	50.9 ± 2.2 ^a	121.3 ± 1.17 ^a	59.9 ± 21.8 ^a
2	Diabetic + control	228 ± 27.3 ^b	132.8 ± 1.7 ^b	210.4 ± 6.5 ^b	152.6 ± 12.7 ^b
3	Diabetic + <i>C. alata</i> (400mg)	136 ± 29.9 ^c	106 ± 8.5 ^c	171.1 ± 3.8 ^c	85.6 ± 19.07 ^c
4	Diabetic + <i>C. alata</i> (600mg)	127 ± 16.4 ^d	97 ± 1.46 ^d	150.3 ± 1.80 ^d	73.2 ± 18.1 ^d
5	Diabetic + Glibenclamide(600µg/kg body weight)	124 ± 13.3 ^e	74 ± 2.7 ^e	141.2 ± 3.56 ^e	70.09 ± 14.1 ^e

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: 51 Effect of *C. alata* fresh leaf extract on plasma HDL-C, LDL-C and VLDL-C on alloxan 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 ^a	35.9 ± 7.1 ^a	11.9 ± 4.3 ^a
2	Diabetic + control	17.2 ± 6.6 ^b	242 ± 20.04 ^b	30.5 ± 2.5 ^b
3	Diabetic + <i>C. alata</i> (400mg)	39.7 ± 3.4 ^c	113 ± 31.8 ^c	17.13 ± 3.8 ^c
4	Diabetic + <i>C. alata</i> (600mg)	42.1 ± 11.4 ^d	100.2 ± 19.2 ^d	14.6 ± 3.6 ^d
5	Diabetic + Glibenclamide(600µg/kg body weight)	47.05 ± 13.02 ^e	91.4 ± 9.1 ^e	14.0 ± 2.8 ^e

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)

