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AMELIORATION OF DIABETES BY *SWIETENIA MAHAGONI* IN STREPTOZOTOCIN INDUCED DIABETIC RATS

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Sweitenia mahagoni, anti-diabetic, liver glycogen, hyperglycemia

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ABSTRACT: *Sweitenia mahagoni* is a natural medicine used to treat various human ailments including diabetes. The present study is a comprehensive approach which targets more than one locus in the diabetic model to enumerate the anti-diabetic potential of *Swietenia mahagoni* as claimed by folklore. Oral administration of the aqueous extract of the leaf decreased the fasting blood glucose level (155.2±6.7) from its initial value (309.8±10.5) in diabetic rats. There was improvement in the content of innate antioxidant components viz., glutathione (0.570±0.01) when compared to positive control group (0.28±0.02). The activity liver health marker enzymes in the serum were also decreased in the treated group compared to untreated diabetic group. There was also reduction in body mass loss in treated groups. Thus it can be proposed that *Sweitenia mahagoni* leaf has potent anti-diabetic activity and the effect may be mediated through increasing the antioxidant strength, improving glycogen content in liver, balancing the lipid components in serum, decreasing the muscle protein catabolism and improved overall health.

INTRODUCTION: Diabetes is a chronic metabolic disease characterized by relative or absolute lack of insulin, inefficient insulin resulting in hyperglycaemia. Prolonged hyperglycaemia may lead to variety of secondary complications such as, retinopathy nephropathy, neuropathy and cardiovascular disease. Recent data on worldwide prevalence of diabetes shows 9.2% of women and 9.8% of men population are diabetic, with approximately 347 million people suffering from the disease worldwide in 2008 [1]. There are several different classifications of diabetes, the most common being type 1 and type 2 diabetes.

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Even though powerful anti-diabetic drugs are available for the management of diabetes, there has been no drug developed which has no side effect(s) and cost effective. Since the existing drugs for the treatment of diabetes do not satisfy our need completely, the search for new drugs continues. In recent years, herbal remedies for the unsolved medical problems have been gaining immense importance in the field of pharmacology.

Traditional knowledge with its holistic and systematic approach supported by scientific documentation can serve as an innovative and powerful discovery engine for newer, safer and affordable medicines [2].

Evaluation of plant products to treat diabetes mellitus is of growing interest as plants contain many bioactive substances with therapeutic potential and possibly works on multi-targets to ameliorate the disorder. *Swietenia mahagoni Jacq.*

is a small leafy, medium sized tree native to west indies. Around the world the plant is commonly called as West Indies mahogany, caoba, caoba dominicana or acajou. It is one of the species of genus Swietenia which belongs to chinaberry family, meliacea [3, 4, 5]. The plant has been used to treat many human ailments such as, hypertension, diabetes, astringent, diarrhea, malaria etc. traditionally. The fruit of the plant is used as powerful anti-hyperglycemic drug. Many potent bio-active components have been isolated and their potency has been confirmed in pre-clinical studies.

The oil of the plant seed is being used as an alternative body ointment therapy for a range of skin cuts, itches and wounds to ameliorate the healing process in African countries. Decoction of bark is used to increase appetite, as energizer in case of tuberculosis, to treat anemia, diarrhea, dysentery, fever and toothache. The decoction of leaf is used to treat nerve disorders, the infusion of seed to relieve from chest pain[6,7, 8]. The *in-vitro* anti-hyperglycemic potency of the leaf has been worked out in our laboratory. Based on the promising results obtained from the *in-vitro* hypeglycaemic assays the plant has been selected for assaying the *in-vivo* anti-diabetic potency in the wistar rats. The present study evaluates the anti-diabetic efficiency of the aqueous extract of *Swietenia mahagoni* leaf streptozotocin induced diabetic rat model.

MATERIALS AND METHODS:

Chemicals and reagents:

Alanine aminotransferase (ALT), Alkaline phosphatase (ALP), Aspartate aminotransferase (AST), Total protein, albumin, urea, creatinine, total bilirubin, tryglycerides, total cholesterol assay kits and GOD-POD glucose analysis kit were purchased from Aggappe Diagnostics, Ernakulam, India. Reduced glutathione (GSH), 5,5-dithio (bis) nitro benzoic acid (DTNB) were purchased from Sigma-Aldrich, Bangalore, India. All the chemicals and reagents used in the study were of analytical grade.

Collection and preparation of samples:

The leaf of *Swietenia mahogani* was collected from Mysore district of Karnataka, India and subsequently identified by Dr. G. R. Shivamurthy, Department of Studies in Botany, University of Mysore, Mysore, India. The collected sample was thoroughly washed under running water to remove adhering dirt and other foreign particles, dried overnight at 50^0C, powdered, passed through 60 mesh sieve and stored in airtight container at 4^0C till further use.

50 gm of leaf powder was extracted with water 500 ml of distilled water for 6 h at ambient temperature. The extract was centrifuged at 2,000 rpm and the supernatant was filtered through filter paper (Whatman no 4). The filtrate was freeze dried to obtain brown colored aqueous extract. The extract was named as MAE (Mahagoni aqueous extract) and stored at 4^0C till further use.

Experimental animals:

Healthy adult rats of Wistar strain weighing around 140-180g were procured from the animal house of University of Mysore, Mysore. The procured rats were kept in the poly-acrylic cages in the room maintained at standard conditions (25±2º C, 45 to 60 % RH and 12 h photo period) and acclimatized for about 15 days. The rats were provided with Pellet diet (procured from Amrut feeds, Pune, India) and water *ad-libitum*. Prior permission from the institutional animal ethical committee was obtained for the planned protocol on anti-diabetic study in the experimental rats (UOM/IAEC/03/2013).

Effect of MAE on normoglycaemia:

The rats were divided into two groups of 6 in each, and named as control and treated (The experiment was performed after the acclimatization and before diabetes induction). Both the groups were overnight fasted and treated group were administered with aqeous extract. The control group was administered with equal volume of physiological saline. Blood was withdrawn from the tail vain at 0, 30, 60, 90, 120 and 160 min of mahagoni powder administration and glucose levels were estimated using glucometer (Glucocord, india).

Experimental design for anti-diabetic activity:

30 male rats were weighed and randomly distributed into five groups of six rats each. The rats were divided into five groups using random block design to randomly segregate the animals

into different experimental groups. The segregated groups were named Group –I to V. Group-I to Group-V were taken as normal control, Diabetic control, Insulin treated, Glibinclamide treated and sample treated groups, respectively. Group III received glibinclimide (5 mg/Kg) for 45 days after the induction of diabetes whereas Group IV received insulin (10 units/kg b.w) for 45 days. The Group V received MAE at 500 mg/kg for 45 days.

Preparation of streptozotocin (STZ) solution:
The STZ solution was prepared by dissolving the commercial STZ in freshly prepared citrate buffer (0.1 M, pH 4.5). The concentration of prepared working solution was 20 mg/ml and the prepared solution was used immediately to avoid any chemical degradation [9].

Preparation of Glibenclamide solution:
Glibenclamide (Daonil®, 5 mg), an oral hypoglycemic drug was dissolved in distilled water (82.33 ml) to give a concentration of 60 µg/ml and administered orally at a dose of 600 µg/ kg daily for a period of 45 days.

Induction of diabetes in rats:
The streptozotocin at the dose of 45 mg/kg of the body weight was injected to rats of each group intraperitoneally, except for normal control group. After 30 hours of streptozotocin injection, glucose solution was supplemented to avoid any mortality due to hypoglycemia induced by sudden hyperinsulinemia. The diabetic state in the animals was confirmed by measuring the fasting blood glucose levels after 72 h of STZ injection. The animals with the blood glucose level above 200 mg/dl were considered diabetic. After the confirmation of diabetic state, the treatment was started.

Collection of blood samples:
To evaluate the blood glucose level at different interval of treatment in various groups the blood was collected from the tail vein using the artificial rat restrainer. About 100 µl of blood was collected on each sampling.

After the treatment for 45 days all the animals were decapitated. Blood was collected through cardiac puncture into the tubes coated with pre-coagulant. The coagulated blood was centrifuged to separate serum. The separated serum was used for various biochemical analyses.

Oral glucose tolerance test (OGTT) in Diabetic rats:
The experiment was performed in diabetic control and MAE treated groups at the end of the treatment. The two groups were overnight fasted and oral glucose load (2 gm/kg) was administered. The blood glucose level in the both the groups was analyzed at 0, 30, 60, 120 and 160 min after the administration of glucose.

Histological examinations:
For histological examinations, small pieces of liver, kidney were fixed in Bouin's solution for 24 h, dehydrated through graded concentration of ethanol, embedded in paraffin wax, sectioned at 5 µm thicknesses and stained with Mayer's hematoxylin and eosin (HandE) and photographed in complex microscope with camera.

Biochemical analysis:
Liver marker enzymes (ALP, SGPT & SGOT), total protein, albumin, urea, bilirubin, total cholesterol, tri-glycerides were analyzed using respective standard kits from Agappe diagnostics.

Antioxidant status:
Serum SOD:
The procedure adopted was that of Beauchamp and Fridovich [10] with minor modifications. The principle of SOD activity assay was based on the inhibition of nitroblue tetrazolium (NBT) reduction. Illumination of riboflavin in the presence of O_2 and electron donor like methionine generates superoxide anions and this has been used as the basis of assay of SOD. The reduction of NBT by superoxide radicals to blue colored formazan was followed at 560 nm. One unit of SOD activity is defined as that amount of enzyme required to inhibit the reduction of NBT by 50% under the specified conditions.

Catalase:
Enzyme activity of catalase was assayed by the method of Goth [11]. The principle was based on the colored stable complex formation of hydrogen peroxide with ammonium molybdate. In brief, 0.2

ml of serum was incubated with 1.0 ml of substrate (65 µ mol/ml hydrogen peroxide in 60 m mmol/l sodium potassium phosphate buffer, pH 7.4) at 37 °C for 60 sec. The enzyme reaction was stopped by addition of 1 ml ammonium molybdate (32.4 mmol/l) and the yellow colored complex was measured at 405 nm against blank 3.

$$\text{serum catalase activity(KU/l)} = \frac{\text{A(Sample)} - \text{A(Blank 1)}}{\text{A(Blank 2)} - \text{A (Blank 3)}} \times 271$$

Where, Blank 1. contained 1 ml substrate, 1 ml molybdate and 0.2 ml serum.

Blank 2. contained 1 ml substrate, 1 ml molybdate and 0.2 ml buffer.

Blank 3. contained 1.0 ml buffer 1.0 ml molybdate and 0.2 ml serum

Glutathione:

Reduced glutathione was determined by the method of Moron[12]. Reduced glutathione on reaction with DTNB (5,5'-dithiobis nitro benzoic acid) produces a yellow coloured product that absorbs light at 412nm. The serum (0.1ml) was made up to 1.0ml with 0.2M sodium phosphate buffer (pH 8.0). Two ml of freshly prepared DTNB solution was added and the intensity of the yellow color developed was measured in a spectrophotometer at 412nm after 10 minutes. Standard GSH corresponding to concentrations ranging between 2 and 10 nmoles were also prepared. The values are expressed as n moles GSH/g sample.

TBARS:

TBARS was estimated according to the method of Ohkawa[13].

Urine analysis: Amount of urine excreted was observed indirectly by the extent of soft bed wetting used in the cages.

RESULTS AND DISCUSSION:

Body and organ weights:

Body and organ weights are given in **Table 1** and **2**. From the table it was observed that the body weight of diabetic control rats was decreased significantly from their initial body weights. The percentage of decrease in body weights was reduced in insulin treated and glibenclamide treated groups when compared to diabetic control group. In MAE treated group decrease in body weights was reduced at a significant level when compared to diabetic control and the effect was more profound than the insulin and glibenclamide treated groups.

TABLE 1: WEIGHTS OF RATS OF THE GROUPS AT VARIOUS AT INTERVALS OF THE STUDY

Group	Initial	15th Day	45th day
C	184.7±2.1	197.5 ±4.8	215±4.13
DC	189.8±3.97	164±2.82	143±4.38
IN	186±5.40	168.3±6.03	165±5.17
Gli	198±2..82	160.4±6.58	156.8±5.25
MAE	199.9±2.89	178.7±4.59	173.5±3.58

Initial refers to 1st day of the treatment after diabetic induction and final values were taken before sacrifice. Values are the mean of values for six rats with standard deviation. C-Control, DC-diabetic control, In-insulin treated group, Gli-Glibenclamide treated group and MAE-Group treated with aqueous extract of *Swietenia mahagoni*

TABLE 2: ORGAN WEIGHTS IN GM

Group	Liver	Pancreas	Brain	kidney	Slpeen	Heart	Testis	Adrenal glands
C	6.5	0.61	1.35	1.61	0.53	0.73	2.48	0.03
DC	4.19	0.30	1.52	1.41	0.10	0.46	0.61	0.04
IN	6.58	0.41	1.57	1.78	0.30	0.623	1.92	0.03
Gli	4.77	0.44	1.09	1.806	0.08	0.461	1.32	0.04
MAE	8.95	0.52	1.255	1.572	0.54	0.76	2.53	0.3

C-Control, DC-diabetic control, In-insulin treated group, Gli-Glibenclamide treated group and MAE-Group treated with aqueous extract of *Swietenia mahagoni*

TABLE 3: FASTING BLOOD GLUCOSE OF THE GROUPS AT VARIOUS INTERVALS OF THE STUDY

	Initial	15th day	30th day	45th day
Control	74.2±1.7	76.7±1.6	85.5±4.8	91.0±2.1
DC	352.7±6.9	375.8±3.8	385.0±8.9	390.8±4.9

Gli	359.2±5.1	207.7±7.5	164.2±6.3	144.0±12.7
In	379.5±9.4	191.2±7.3	159.0±8.1	137.3±6.3
MAE	309.8±10.5	260.8±7.9	187.2±7	155.2±6.7

C-Control, DC-diabetic control, In-insulin treated group, Gli-Glibenclamide treated group and MAE-Group treated with aqueous extract of *Swietenia mahagoni*

TABLE 4: WATER AND FOOD CONSUMPTION

	Food (g/day)	Water (ml/day)
C	30±2.36	52.83±3.6
DC	44.83±2.36	105±3.84
IN	43.3±1.50	90.83±3.8
Gli	42.3±1.75	86.83±2.7
MAE	35.3±1.86	82.8±1.60

C-Control, DC-diabetic control, In-insulin treated group, Gli-Glibenclamide treated group and MAE-Group treated with aqueous extract of *Swietenia mahagoni*. The values are expressed in mean ± SD for each group, where (n=6). The values are taken at last 4 days of the experiment before the sacrifice.

Biochemical parameters:

Normoglycemic & oral glucose tolerance study: The results indicate that MAE treatment does not affect the basal glucose level in the serum of rats (**Fig.1.**) and treatment improved the oral glucose tolerance in diabetic rats (**Fig.2**). The effect was significant when compared to diabetic rats.

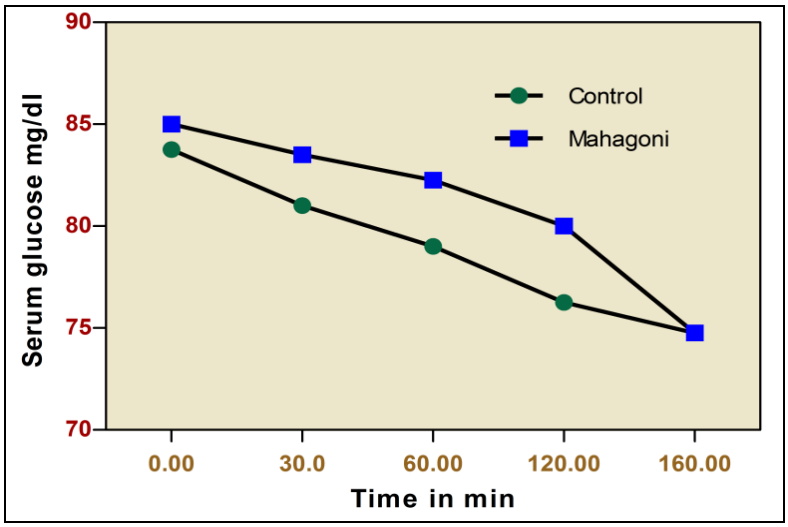


FIG.1: EFFECT OF MAHAGONI WATER EXTRACT TREATMENT ON NORMOGLYCAEMIA OF NORMAL RATS. The experiment was conducted on fasted rats (n=6) in each group.

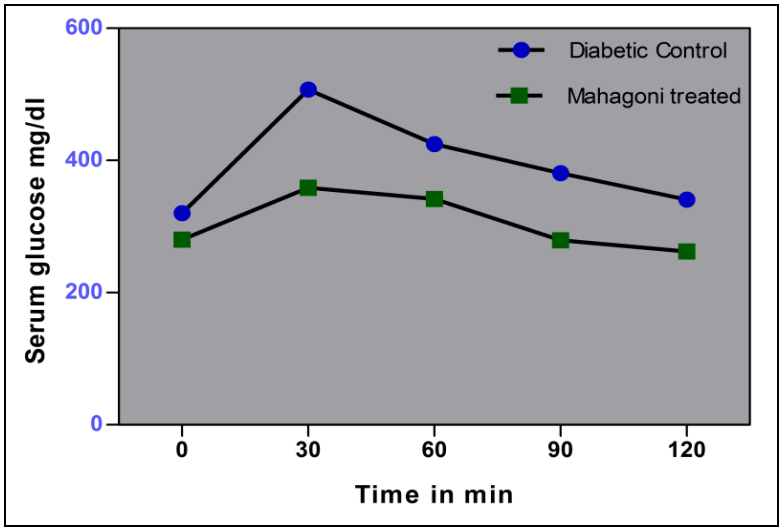


FIG.2: EFFECT OF MAHAGONI WATER EXTRACT TREATMENT ON ORAL GLUCOSE TOLERANCE OF DIABETIC RATS

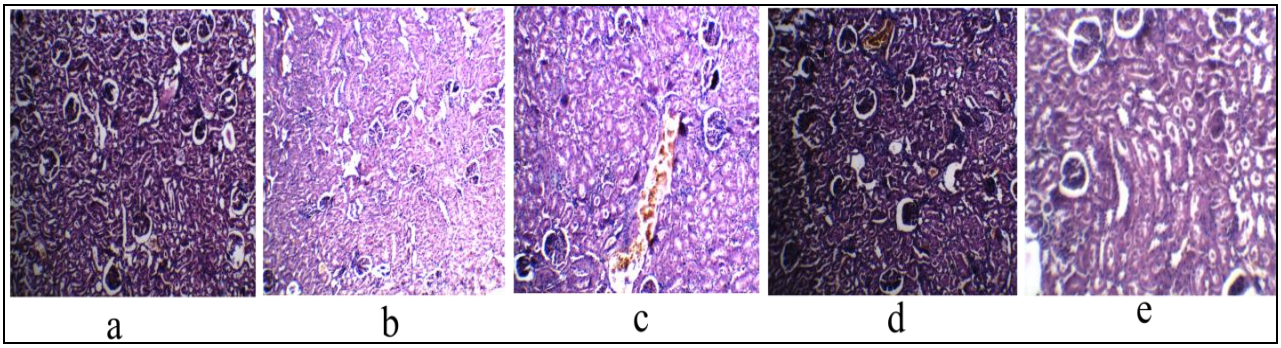


FIG.3: KIDNEY SECTIONS OF THE GROUPS

a-Normal control, b-Diabetic Control, c-Glibinclamide, d-Insulin and d-Mahagoni Aqueous extract. Photographs shown are taken at 10X resolution

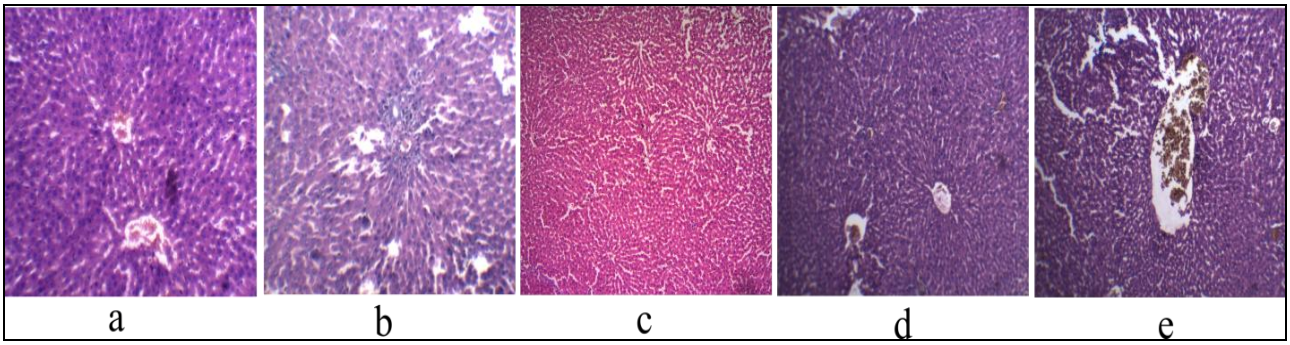


FIG.4: LIVER SECTIONS OF THE GROUPS

a-Normal control, b-Diabetic Control, c-Glibinclamide, d-Insulin and d-Mahagoni Aqueous extract. Photographs shown are taken at 10X resolution

Liver marker enzymes:

From the **Table 5** it can be observed that there is an elevation in liver marker enzymes viz., ALP, AST and AST in serum of diabetic rats. The activities of liver marker enzymes has significantly increased in diabetic control group when compared to control and all the other treated groups. It can be observed that treatment with glibenclamide, insulin and MAE has decreased the activities of ALT, AST and ALP, and the effect was more profound in MAE treated group.

Lipid parameter:

The lipid parameters of diabetic, normal control and treated groups are given in the **Table 5**. Results indicate that there is sever hyperlipidemia in the diabetic control group. The treatment with MAE has improved the level of HDL and decreased the level of triglycerides. Insulin and glibenclamide treatment also has improved the lipid status and the effect was more in group treated with insulin.

Kidney markers: The serum levels of bilirubin and urea were presented in **Table 5**. It can be noted that diabetic control group has lowest level of urea in the serum. In the groups treated with Insulin and glibenclamide and MAE, level of urea in serum has increased towards the normal. Level of bilirubin in the serum of diabetic rats was elevated beyond the normal range. In the serum of treated groups there was decrease in the level of bilirubin and decline was towards the normal control.

Antioxidant level:

The level antioxidant enzymes (Catalase and SOD) were decreased in serum of diabetic induced groups (**Table 5**). Treatment with MAE has improved the levels of antioxidant enzymes towards normal range. The level of lipid peroxides was highest in the serum of diabetic control group. Treatment with MAE decreased the level of lipid peroxides. The level of glutathione was decreased with diabetes induction. The glutathione level was improved with the treatment with MAE. Glibenclamide and insulin treatment has not shown significant amelioration effect on the level of glutathione.

Glycogen content in liver:

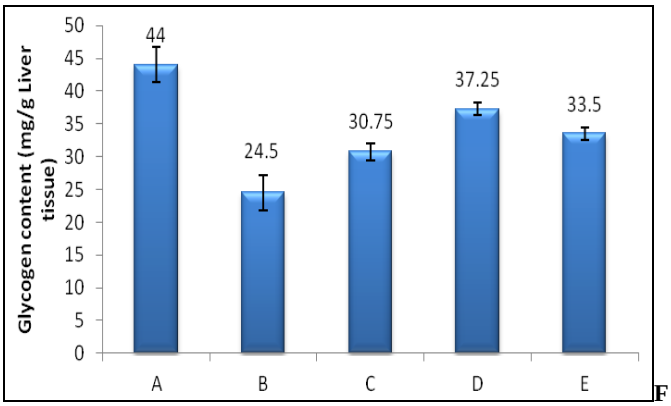
The glycogen content of the groups is given in Figure.5. There was severe decrease in the

glycogen content of liver can be noted in diabetic control group when compared to normal control. The groups treated with MAE and insulin has near normal glycogen levels.

TABLE 5: SERUM BIOCHEMICAL PARAMETERS

Groups	ALP (U/L)	SGPT (ALT) U/L	SGOT (AST) U/L	T.pro g/dL	Albumin g/dL	Urea mg/dL	T. Bilirubin mg/dL	TBARS ng/mgPro	GSH micM/mgPro	T.Cholesterol (mg/dl)	TGL (mg/dl)	SOD (activity / mg pro)	Catalase (activity KU/L)
Con	126.5 ±9.8	11.56 ±0.78	45.13 ±0.46	7.11 ±0.109	2.13 ±.088	47.94 ±2.86	0.225 ±0.011	18.3 ±0.88	0.771 ±0.008	46.8± 1.016	93.24 ±3.13	14.25 ±1.81	291 ±5.9
DC	206.75 ±4.9	40.9 ± 0.91	82.03 ±1.94	5.11 ±0.13	3.58 ±0.22	38.86 ±2.47	0.75 ±0.020	37.08 ±3.54	0.28 ±0.028	73.81 ±4.53	137.7 ±7.70	5.43 ±1.20	237 ±10.2
In	134.75 ±12.8	42.56 ±1.46	73.31 ±1.43	6.87 ±0.28	1.53 ±0.25	34.9 ±2.85	0.221 ±.09	39± 2.40	0.657 ±.009	54.31 ±3.16	115.51 ±5.59	7.25 ±0.15	115 ±8
Gli	181.5 ±15	17.1 ±0.92	56.01 ±2.74	6.19 ±0.11	2.34 ±0.14	35.43 ±2.48	0.251 ±0.007	16.65 ±1.87	0.617 ±0.15	55.11 ±2.92	108.86 ±8.63	3.46 ±0.62	228 ±8.2
MAE	192 ±11.2	26.08 ±1.88	50.9 ±1.13	7.15 ±0.15	2.42 ±0.34	35.43 ±2.77	0.24 ±0.02	21.63 ±1.98	0.570 ±0.01	50.61 ±1.77	101.83 ±3.33	10.91 ±1.97	261 ±4.8

C-Control, DC-diabetic control, In-insulin treated group, Gli-Glibenclamide treated group and MAE-Group treated with aqueous extract of *Swietenia mahagoni*. The values are the mean of results from each group with SD (where n=6).



IG.5: GLYCOGEN CONTENT IN LIVER A-Normal control, B-Diabetic Control, C-Glibinclamide, D-Insulin and E-MAE treated (The values are mean values for 6 rats in each group with ± SD

DISCUSSION: Streptozotocin is a cytotoxic compound which induces diabetes by damaging the β-cells of pancreas through mechanism involving the damage to cell membrane of β-cells resulting in degeneration. β-cell degeneration results in insulin insufficiency and hypeglycemia. The Streptozotocin induces diabetes which is similar to diabetes mellitus with non-ketosis hyperglycemia. The symptoms include dyslipidemia, loss of protein mass and body weight, damage to liver and other organs resulting in major disturbance of central metabolic balance [14].

Mahagoni treatment to diabetic rats resulted in decrease of fasting blood glucose level and the effect was comparable to the anti-diabetic drug glibenclamide. The improvement in hyperglycemia was also supported by the decrease of daily water and food intake by the diabetic rats.

In addition to reduction in the hyperglycemic state, the group treated with mahagoni showed better oral glucose tolerance as indicated by the oral glucose tolerance test performed at the end of treatment. Oxidative stress is worsened in the case of hyperglycemia and literature indicates the direct role of oxidative stress in the development of diabetic secondary complications. Increased oxidative stress is claimed to be triggered directly by the hyperglycemia [15, 16]. In the present study it was observed that the induction of diabetes by streptozotocin results in oxidative stress, wherein the innate antioxidant system is weakened.

In the serum of diabetic control rats, there was large depletion of antioxidants such as glutathione and anti-oxidant enzymes viz., SOD and catalase. Increase in the level of lipid peroxides was observed as a consequence of reduced anti-oxidants and increased oxidants in the serum of diabetic control rats. Treatment with mahagoni has improved the level of anti-oxidants and decreased the lipid peroxides, indicating overall amelioration of oxidative stress in diabetic rats.

In the normal physiology the glucose in the plasma will be taken up by muscle and adipose tissue, which forms the major reserve source of energy. Insulin plays a major role in glucose uptake into muscle and adipose tissue, lack of insulin or insufficient insulin results in failure of glucose uptake into muscle and adipose tissue[17] resulting in decreased stored energy. Decreased stored energy force the body to utilize the protein mass in the

muscle resulting in the loss of overall bodyweight. In the diabetic control rats there was a drastic fall in the body weight was observed during the study, where as treatment with the mahagoni decreased the loss of body weight in the diabetic rats indicating the insulin mimetic or insulin potentiating activity of the components in the extract of plant.

Glycogen is the gluco-polymer which is stored in the liver and is used at the time of starvation to provide instant energy to the body. In the sever diabetes, due to the inactive glycogen synthetic pathways and overactive breakdown pathways, glycogen content of the liver will be depleted[18]. Mahagoni treatment improved the liver glycogen content in sever diabetic rats indicating the affect on mahagoni on the pathways inducing glycogenesis.

In sever hyperglycemic condition lipid synthesis will be inhibited whereas the lipolysis will be aggravated. The condition results in increased triglycerides and LDL in the plasma[19]. The plasma lipid level in the mahagoni treated groups was ameliorated indicating the positive effect of the mahagoni extract on overall lipid metabolism. Although we have not measured the exact amount of urine volume excreted in the experimental groups, it was apparent when we observed the wetting of the soft bed used in the cages. It was observed that wetting was more in diabetic control when compared to treated groups and mahagoni treatment reduced the wetting gradually indicating the ameliorating affect of mahagoni on polyuric condition.

CONCLUSION: The present study is a comprehensive approach which targets more than one locus in the diabetic model to enumerate the anti-diabetic potential of *Swietenia mahagoni* as claimed by folklore medicine. From the results of the study it can be concluded the aqueous extract of *Swietenia mahagoni* normalizes blood glucose level, improves dyslipidemia and increases antioxidant strength in sever diabetic condition.

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REFERENCES:

1. Goodarz Danaei, Mariel M Finucane, Yuan Lu, Gitanjali M Singh, Melanie J Cowan, Christopher J Paciorek. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2·7 million participants. The Lancet 2011; 378: 31 – 40.
2. Azaizeh H, Fulder S, Khalil K and Said O: Ethnomedicinal knowledge of local Arab practioners in the Middle East region. Fitoterapia 2003; 74: 98-108.
3. Elbert Luther little: Atlas of United States trees.US department of agriculture forest service.Washington DC.1978; 3:5. (Book)
4. Lord Britton, Charles Frederick Millspaugh: TheBahama flora, New York.1920.
5. John Francis and k. John. Swietenia mahagoni Jacq. West Indies mahogany. Meliaceae. Mahogany family: New Orleans, LA: US Department of Agriculture, Forest Service, Southern Forest Experiment Station, Institute of Tropical Forestry. 1991:7. (SO-ITF-SM; 46)
6. K. Bacsal, ChavezL, I. Diaz, S. Espina, J. Javillo and H. Manzanilla: The Effect of *Swietenia Mahogani* (Mahogany) seed extract on Indomethacin-inducd gastric ulcers In female Sprague-Dawley rat. ActaMedica Philippina 1997; 33:127-139.
7. Miroslav M G: Elsevier's Dictionary of Trees. London, Elsevier Inc. 2005; 1:381.
8. Naveen YP, Divya Rupini G, Ahmed F and Urooj A: Pharmacological effects and active phytoconstituents of swietenia mahagoni: a review. J Integr Med 2014; 12(2):86-93.
9. Karunanayake EH, Hearse D J, Mellows G: The metabolic fate and elimination of streptozocin. Biochem Soc Trans 1975; 3: 410-14.
10. Beauchamp C and Fridovich I: Superoxide dismutase: Improved assays and an assay applicable to acrylamide gels. Anal Biochem Review 1971; 44: 276-287.
11. Goth L. A simple method for determination of serum catalase activity and revision of reference range: Clinica chemical acta 1991; 196: 143-152.
12. Moron M J, Depierre J W, Mannervik B: Levels of GSH, GR and GST activities in rat lung and liver. Biochem Biophys Acta 1979; 582: 67-78.
13. Ohkawa H, Ohishi N, Yagi K: Assay for lipid peroxides in animals tissues by thio-barbituric reaction. Anal Biochem 1979; 95: 351–8.
14. Wei M, Ong L, Smith MT, Ross FB, Schmid K, Hoey AJ: The streptozotocin-diabetic rat as a model of the chronic complications of human diabetes. Heart Lung Circ 2003; 12(1):44-50.
15. King GL, Loeken MR: Hyperglycemia-induced oxidative stress in diabetic complications. Histochem Cell Biol 2004; 122 (4):333-8. Epub 2004 Jul 15.
16. Ferdinando Giacco and Michael Brownlee: Oxidative stress and diabetic complications. Circ Res 2010; 107(9): 1058–1070.
17. Sophie EL and Jeremy M T: The molecular basis of insulin-stimulated glucose uptake: signalling, trafficking and potential drug targets. Journal of Endocrinology 2009; 203:1–18.

18. Martin Krssak, Attila Brehm, Elisabeth Bernroider, Christian Anderwald, Peter Nowotny, Chiara Dalla Man: Alterations in postprandial hepatic glycogen metabolism in Type 2 Diabetes. Diabetes 2004; 53(12): 3048-3056.

19. Bardini G, Rotella CM, Giannini S: Dyslipidemia and diabetes: reciprocal impact of impaired lipid metabolism and beta-cell dysfunction on micro- and macrovascular complications. Rev Diabet Stud 2012; 9(2-3):82-93.

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