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Antidiabetic activity on *Butea monosperma* (seeds) and *Ficus benghalensis* (aerial roots)

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Abstract
Antidiabetic activities of the extracts of *B. monosperma* (seeds) and *F. bengalensis* (aerial roots) were performed on alloxan induced diabetic rats and showed the antidiabetic activity. The administration of alloxan increased the various serum lipids. Treatment with the extracts of *F. bengalensis* decreased the lipid parameters significantly (P<0.01) while *B. monosperma* decreased the lipid parameters not significantly (P>0.05).

Keywords: Antidiabetic activity, *Butea monosperma, Ficus benghalensis*, lipid parameters

Introduction
Diabetes mellitus is a chronic metabolic disorder characterized by a high blood glucose concentration-hyperglycaemia (fasting plasma glucose >7.0 mmol) or plasma glucose >11.1 mmol/12 hour after a meal), caused by insulin deficiency, often combined with insulin resistance. Hyperglycaemia occurs because of uncontrolled hepatic glucose output and reduced uptake of glucose by skeletal muscle with reduced glycogen synthesis. When the renal threshold for glucose reabsorption is exceeded, glucose spills over into the urine (glycosuria) and causes an osmotic diuresis (polyurea), which in turn, results in dehydration, thirst and increased drinking (polydipsia). Insulin deficiency causes wasting through increased breakdown and reduced synthesis of proteins. Diabetic ketoacidosis is an acute emergency. It develops in the absence of insulin because of accelerated fat breakdown to acetyl-CoA, which in the absence of aerobic carbohydrate metabolism, is converted to acetoacetate and β-hydroxybutyrate (which causes acidosis) and acetone (a ketone) (Rang *et al.,* 2003).
There will be a 42% increase from 51 to 72 million in the developed countries and 170% increase from 84 to 228 million, in the developing countries. Thus, by the year 2025, over 75% of all people with diabetes will be in the developing countries, as compared to 62% in 1995. Madhumeha which has been correlated with diabetes mellitus has become a global problem in spite of advances in modern science. India has been projected by WHO as the country with the fastest growing population of diabetic patients. It is estimated that between 1995-2025 diabetic patients in India will increase by 95% (Mukherji *et al.*, 1999).

Antidiabetic Activity
Diabetes mellitus is a chronic metabolic disorder characterized by a high blood glucose concentration-hyperglycaemia (fasting plasma glucose >7.0 mmol) or plasma glucose >11.1 mmol/12 hour after a meal), caused by insulin deficiency, often combined with insulin resistance. Hyperglycaemia occurs because of uncontrolled hepatic glucose output and reduced uptake of glucose by skeletal muscle with reduced glycogen synthesis. When the renal threshold for glucose reabsorption is exceeded, glucose spills over into the urine (glycosuria) and causes an osmotic diuresis (polyurea), which in turn, results in dehydration, thirst and increased drinking (polydipsia). Insulin deficiency causes wasting through increased breakdown and reduced synthesis of proteins. Diabetic ketoacidosis is an acute emergency. It develops in the absence of insulin because of accelerated fat breakdown to acetyl-CoA, which in the absence of aerobic carbohydrate metabolism, is converted to acetoacetate and β-hydroxybutyrate (which causes acidosis) and acetone (a ketone) (Rang *et al.,* 2003).

Two major types of diabetes mellitus are:
Type I- Insulin dependent diabetes mellitus (IDDM)
Type II - Non insulin dependent diabetes mellitus (NIDDM)

Type 1- Insulin dependent diabetes mellitus, juvenile onset diabetes mellitus

There is β cell destruction in pancreatic islets; majority of cases are autoimmune (type 1A) antibodies that destroy β cells are detectable in blood, but some are idiopathic (type1B)-no β cell antibody is found. In all type 1 cases circulating insulin levels are very low, and patients are more prone to ketosis. This type is less common and has low degree of genetic predisposition.

Type 11- Non insulin dependent diabetes mellitus, maturity onset diabetes mellitus

There is no loss or moderate reduction in β cell mass; insulin in circulation is low, normal or even high, no anti β-cell antibody is demonstrable; has a high degree of genetic predisposition; generally has a late onset (past middle age). Over 90% cases are type 11DM. The causes may be:

Abnormality in glucose-receptor of β cells so that they respond at higher glucose concentration.

Reduced sensitivity of peripheral tissues to insulin: reduction in number of insulin receptor, down regulation, of insulin receptors. Many hypertensives are hyperinsulinemic but normoglycaemic; exhibit insulin resistance. Hyperinsulinemia per *se* has been implicated in causing angiopathy.

Excess of hyperglycemic hormones (glucagons etc.)/obesity: causes relative insulin deficiency- the β cells lag behind (Tripathy, 2003).

Materials and Methods Plants material

Butea monosperma seeds were procured from the Hansraj & Sons, Khari Baoli, and local market of Delhi. *F. bengalensis* aerial roots were collected from the campus of Jamia Hamdard Delhi.

Preparation of the extracts

Air-dried, powdered plant material was extracted exhaustively with methanol. The filtrate was dried in vacuum and stored in refrigerator for further studies. The dried extracts were suspended in 1% Tween 80 in water for the animal studies when necessary.

Animals

Wistar albino rats (150-200 g) were obtained from Central Animal Facility, Jamia Hamdard University and maintained in 25 ± 1 °C, with 55 ± 5% humidity with 12 hr light/dark cycle and allowed food and water *ad-libitum*. Animals were acclimatized to the conditions before start of the experiments. The Institutional Animal Ethics Committee approved the experiments. All the extracts and the standard drugs were administered orally.

Procedure

Diabetes was induced in animals by single intraperitoneal injection of Alloxan (150 mg/kg) freshly prepared in normal saline. Control rats received only the normal saline. Diabetes was confirmed by checking the blood glucose levels after 7 days. Animals showing blood glucose level above 250 mg/dl were selected for the study. Diabetic animals were randomly assigned to groups. Group I contained normal animals and served as normal control. Group II served as diabetic control (toxic). Groups I and II received vehicle during the experiments, while the Group III received the reference standard drug glibenclamide (3 mg/kg) and groups from IV to V received the extracts of *F. bengalensis*, and *B. monosperma* (250 mg/kg, and 250 mg/kg) respectively. Blood glucose level was measured at zero and two hr after drugs administration on 1st day and 8th day.

Estimation of blood glucose

The blood glucose level was estimated with glucose Test Kit. Enzymatic GOD-POD method. (Span diagnostics Ltd. Sachin).

Statistical analysis

Values are expressed as mean ± standard error of the mean. Statistical significance was calculated by using one way analysis of variance followed by Dunnetts't' test. The values were considered significantly different when the P- value was less than 0.05.

Table 1: Effect of different extracts of drugs on blood glucose of alloxan-induced diabetic rats

Group s	Treatment	Dose	Blood glucose level in mg/100 ml			
			1st day		8th day	
			0 hr	2 hr	0 hr	2 hr
I	Control (Vehicle)	10 ml/kg	84.57± 4.96*	82.56±4.69*	83±5.19*	81.56±3.64*
II	Control (Diabetic)	10 ml/kg	285.99±9.6	287.72±8.67	283.72±6.67	281.72±9.17
III	Standard (Glibenclamide)	3 mg/kg	270.42 ± 13.69*	203.13±13.69*	155.87±4.96*	153.13±3.69*
IV	Extract of *F. bengalensis*	250 mg/kg	253.86 ± 5.12*	240.61±7.25*	198.83±8.95*	196.61±6.25*
V	Extract of *B. monosperma*	250 mg/kg	259.40 ± 8.41*	250.29±5.07*	232.54±4.58*	232.29±5.07*

All values are Mean ± SEM; n=6

* P<0.01 when compared with diabetic control (group II)

Effect of extracts on serum lipid profile of alloxan-induced diabetic rats

Diabetes was induced in animals by single intraperitoneal injection of alloxan (150 mg/kg) freshly prepared in normal saline. Control rats received only the normal saline. Diabetes was confirmed by checking the blood glucose levels after 7 days of alloxan injection. Animals showing blood glucose level above 250 mg/dl were selected for the study. Diabetic animals were randomly assigned to different groups. Group I contained normal animals and served as normal control. II group served as diabetic control (Toxic). Groups I and II received vehicle during the experiments, while the, groups III received the reference standard drug glibenclamide (3 mg/kg). Group IV and V received extracts of *F. bengalensis and B. monosperema* at a dose of 250 mg/kg, and 250 mg/kg respectively for 8 days.

Estimation of Lipid profile

The body weight of animals was monitored to adjust the dose. Animals were fasted overnight and the blood was collected from tail vein of each animal. Serum was separated from the blood by centrifugation at 3000 rpm for 20 min. The

estimation of triglycerides, total cholesterol, and VLDL were carried out using the commercially available kits. The serum total cholesterol (TC), triglycerides (TG) and VLDL were estimated using commercially available kits (Span Diagnostics, India). The method given by the manufacturer along with the kit was followed. The VLDL were calculated using the following formulae (Friedewald *et al.,* 1972).

VLDL = TG/5

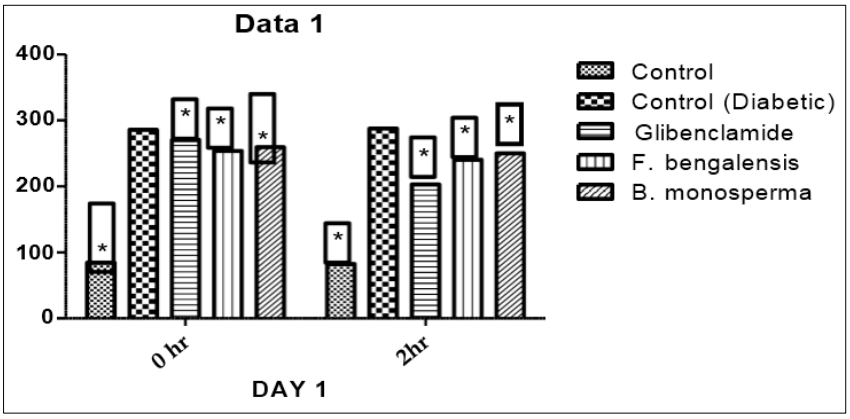

Fig 1

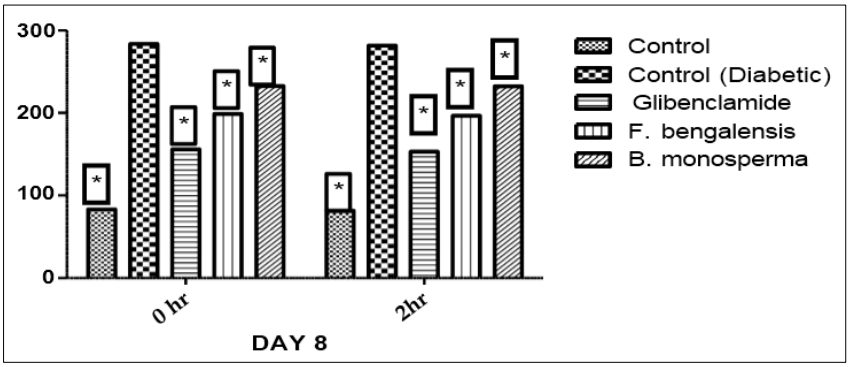

Fig 2

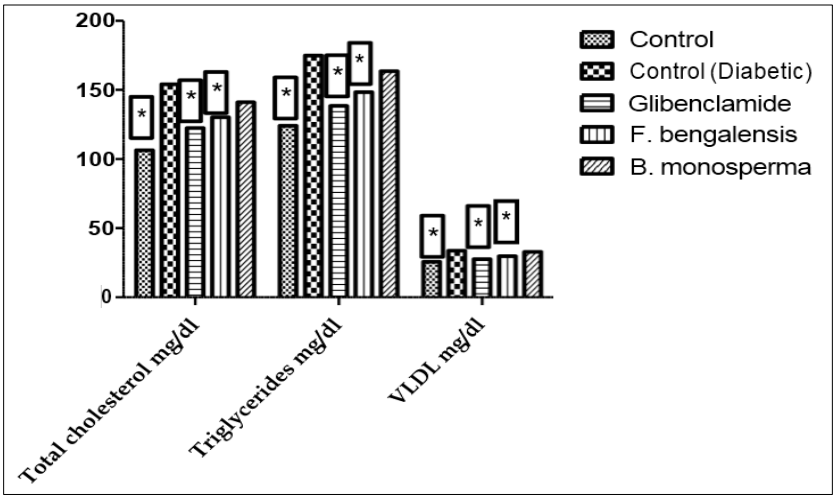

Fig 3

Table 2: Effect of different extracts of drugs on lipid profile of alloxan- induced diabetic rats

Group	Treatment	Total cholesterol mg/dl	Triglycerides mg/dl	VLDL mg/dl
I	Normal control	106.34±6.038*	124 ±2.596*	25.78±2.035*
II	Diabetic control	154±5.823	174.85±3.548	33.7±2.314
III	Standard	122.41±9.248*	138.36±2.958*	27.53 ±2.016*
IV	*F. bengalensis (*250 mg/kg)	130.29±3.590*	148.45±2.762*	29.7±1.321*
V	*B. monosperma* (250 mg/kg)	141.14±2.27ns	163.57±3.665ns	32.91±1.254ns

Values are Mean ± SEM; n=6

*P<0.01 when compared with diabetic control group

ns not significant when compared with diabetic control, group

Result and Discussion

Effect of 8 days treatment of extracts on blood glucose of alloxan-induced diabetic rats Extracts of *F. bengalensis and B. monosperma* decreases the blood glucose levels significantly (P<0.01) which is shown in Table 1.

Effect of 8 days treatment of extracts on serum lipid profile of alloxan-induced diabetic rats

The administration of alloxan increases the various serum lipids. Treatment with extracts of *F. bengalensis* decreases the lipid parameters significantly (P<0.01) while *B. monosperma* decreases the lipid parameters not significantly (P>0.05) which is shown in Table 2.

Antidiabetic activities of the extracts of *B. monosperma* (seeds) and *F. bengalensis* (aerial roots) were performed on alloxan induced diabetic rats and showed the antidiabetic activity. The administration of alloxan increased the various serum lipids. Treatment with the extracts of *F. bengalensis* decreased the lipid parameters significantly (P<0.01) while *B. monosperma* decreased the lipid parameters not significantly (P>0.05).

References

1. Akerele O, Stott G, Lu W. The role of traditional medicine in primary health care in China. The American journal of Chinese Medicine, Supplement No.1, 1987.
2. Alarcon AFJ, Roman RR, Perez GS, Aguilar CA, Contreras WCC, Flores SJL. Study of anti-hypoglycemic effects of plants used as antidiabetics. Journal of Ethnopharmacology. 1998; 61(2):101-110.
3. Atta-Ur-Rahman, Zaman K. Medicinal plants with hypoglycemic activity. Journal of Ethnopharmacology. 1989; 26:1-55.
4. Baily CJ, Day C. Traditional plant medicines as treatments for diabetes. Diabetes care. 1989; 12:553-564.
5. Coimbra TC, Danni FF, Blotta RM, Guedes MD, Graf RG. Plants employed in the treatment of diabetes mellitus: result of an ethnopharmacological survey in porto Alehre, Brazil. Fitoterapia. 1992; 63(4):320-322.
6. Demand Study for Selected Medicinal Plants. Ministry of Health and Family Welfare, Govt. of India Department of Indian systems of Medicinal and Homeopathy (ISM &H) World Health Organization(WHO), 2001-2002, 17.
7. Fransworth NR. World Economy of Medicinal plants. Economic botany. 1984; 38:4-13.
8. Hamberger M, Marston A, Hostettmann KK. Advances in Drug Research, 20, B., ed., Academic Press, London, 1991, 167-215.
9. Huupponen R. Adverse cardiovascular effects of sulphonylurea drugs: clinical significance. Medical toxicology. 1978; 2:190-209.
10. Ivorra MD, Paya M, Villar A. Hypoglycemic and insulin releasing effect of tormentic acid: a new hypoglycemic natural product. Planta Medica. 1988; 54:282-286.
11. Kalia AN. Textbook of Industrial Pharmacognosy. CBS Publishers, New Delhi. Chap, 2000, 2&3rd.
12. Kar A, Choudhary BK, Bandhopadhyay NG. Preliminary studies on the inorganic constituents of some indigenous hypoglycemic herbs on oral glucose tolerance test. Journal of Experimental Botany. 1999; 64:179-184.
13. Kinghorn AD, Balandrin MF. Human Medicinal Agents from Plants. American Chemical Society, Washington, DC, 1993.
14. Lantz TC. Examining the Potential Role of Co-operatives in the Ethical Commercialization of Medicinal Plants: Plant Conservation, Intellectual Property Rights, Ethics, and Devil's Club (*Oplopanax horridus*). British Columbia Institute for Co-operative Studies. Occasional, 2001, 3, 1.
15. Marles RJ, Farnsworth NR. Antidiabetics plants and their active constituents-1, Phytomedicine. 1995; 2:133-189.
16. Masood E. Medicinal plants threatened by over-use. Nature. 1997, 385, 570.
17. Mukharjee K, Ghosh NC, Datta T. *Coccinia indica* Linn. As potential hypoglycemic agent. Indian journal of Experimental biology. 1972; 10(5):347-349.
18. Pari L, Uma MJ. Hypoglycemic effect of *Musa sapientum* L. in alloxan- induced diabetic rats. Journal of ethnopharmacology. 1999; 68:321-325.
19. Pari L, Venkateswaran S. Effect of *Coccinia indica* on blood glucose, insulin and key hepatic enzyme in experimental diabetes. Pharmaceutical biology. 2002; 40:165-170.
20. Saifi AQ, Shinde S, Kwishwar WK, Gupta SR. Some aspects of Phytochemistry and hypoglycemic action of *Pterocarpus marsupium*. Journal of Research in India Medicine. 1971; 6(2):205-207.
21. Suffness M, Douros J. Journal of Natural Products. 1982; 45:1-14.
22. Sumana G, Suryawanshi S. Effect of *Vinca rosea* extracts in treatments of alloxan diabetes in male albino rats. Indian journal of experimental biology. 2001; 39:748-758.
23. Wagner H, Fransworth NR. Economic and Medicinal Plant Research. 1990; 4:3-16.