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ANTIDIABETIC, ANTIOXIDANT AND ANTI-HYPERLIPIDAEMIC ACTIVITY OF *CUCUMIS CALLOSUS* IN STREPTOZOTOCIN-INDUCED DIABETIC RATS

Sivaprasad Panda [1], Mainak Chakraborty [2], Poulami Majumder [2], Suchandra Mazumder [2], Sudipta Das [3], Pallab Kanti Haldar [*2]

College of Pharmaceutical Sciences [1], Puri-Konark Marine Drive Road, Puri-2, Odisha - 752002,India.
Department of Pharmaceutical Technology [2], Jadavpur University, Kolkata - 700032, West Bengal, India.
Netaji Subhas Chandra Bose Institute of Pharmacy [3], Chakdaha, Nadia - 741222, West Bengal, India.

Key words:

Antidiabetic; glibenclamide; streptozotocin; *Cucumis callosus*.

Correspondence to Author:
Pallab Kanti Haldar

Department of Pharmaceutical Technology, Jadavpur University Kolkata-700032, India.

Email: pallab_haldar@rediffmail.com

ABSTRACT: *Cucumis callosus* (Rottl.) Cogn. (Cucurbitaceae) is a highly branched very common prostate, perennial herb, distributed throughout India in the arid zones. **Objective:** To evaluate the antihyperglycemic potential of *C. callosus* fruit in experimental animal model. **Materials and methods:** Hyperglycemia was induced in rats by single intraperitoneal injection of STZ (55 mg/kg body weight). Three days after STZ induction, the hyperglycemic rats were treated with MECC at the doses of 200 and 400 mg/kg body weight (p.o.) daily for 15 days. Glibenclamide (0.5 mg/kg, orally) was used as reference drug. The fasting blood glucose levels were measured on every 5th day during the 15 days of treatment. Serum biochemical parameters including lipid content were estimated. **Results and discussion:** MECC at the doses of 200 and 400 mg/kg significantly ($P < 0.01$) and dose dependently reduced blood glucose levels towards normal as compared to that of STZ control group; the higher dose (400 mg/kg) being the most potent showing complete normalization of blood glucose levels. Serum biochemical parameters including lipid profile were significantly ($p < 0.01$) restored toward normal levels in MECC-treated rats as compared to STZ control group. **Conclusion:** The present study concludes that *Cucumis callosus* fruit demonstrated promising hypoglycemic action in STZ-induced diabetic rats substantiating its ethnomedicinal use.

INTRODUCTION: Hyper glycaemia, hyper lipidaemia and depressed antioxidants are the main common clinical features of the autoimmune disease and diabetes mellitus. Experimental diabetes mellitus can be induced by alloxan or streptozotocin [1].

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Severity of the disease complications closely correlates to glycosylated hemoglobin (HbA1c) levels, a parameter frequently employed as an index for monitoring the disease and for therapy prognosis [2]. Management of diabetes without any side effect is still a challenge to the medical community.

Thus searching for a new class of compounds is essential to overcome diabetic problems. The ethnomedicine and ethnopharmacology remedies have gained popularity amongst the people of developing countries, being safe, effective and inexpensive [3]. *Cucumis callosus* (Rottl.) Cogn.

(Cucurbitaceae) commonly called as 'Bitter cucumber' in English, 'Kachri' in Hindi, is a highly branched very common prostate, perennial herb, distributed throughout India in the arid zones. The fruits are smooth, ovoid, ellipsoid, green variegated stripes and have bitter pulp [4]. *C. callosus* is essentially a warm season crop and a long period of warm and humid climate is required [5]. On the basis of ayurvedic evidence fruit pulp of *C. callosus* is bitter, acrid, thermogenic, anthelmintic, liver tonic, cardio tonic, appetizer, expectorant and intellect promoting. Roots are used as emetic and purgative [6]. Traditionally its fruits and seeds are used for strong memory, remove vertigo, cooling, astringent, bilious disorder [7]. The *C. callosus* is used in treatment of diabetes mellitus by Srilankan ayurvedic and traditional physicians [8].

The aqueous and alcoholic extract of *C. callosus* (seed) has been reported for its antioxidant activity [9]. The tribal people of Balasore and Baripada (Odisha, India) traditionally use the fruits of *C callosus* during worship and for curing diabetes, epilepsy and diarrhoea [10]. Hence, the present study was aimed to evaluate antidiabetic activity of the methanol extract of *C. callosus* fruits.

MATERIALS AND METHODS:

Collection of plant materials:

The fruits of *C. callosus* were collected in the month of July 2007 from village area of Kendrapara and Balasore district, Odisha (India). The plant was authenticated by M. S. Mondal, Botanical Survey of India, Kolkata, India, and a voucher specimen (CNH/1-1(196)/2007/Tech-II/160) has been preserved in the Phytotherapy and Pharmacology Research Laboratory, Jadavpur University, Kolkata for future reference.

Extraction:

The fruits of *C. callosus* were shade dried and then powdered with a mechanical grinder. The powder (500 g) was successively extracted with petroleum ether (60–80°C) and methanol in a Soxhlet extraction apparatus. The solvents were completely removed under reduced pressure to obtain a dry mass. The yields of the petroleum ether and methanol extracts were found to be 2.8% and 9%, w/w, respectively. The extracts were stored in a vacuum desiccator for further use. The preliminary phytochemical screening of the methanol extract of *C. callosus* (MECC) showed the presence of tannins, flavonoids, and triterpenoids [11].

Animals:

Adult male Wistar albino rats weighing 180–200 g were used for the present investigation. They were housed in a clean polypropylene cage and were fed with a standard pellet diet (Hindustan Lever, Kolkata, India) and water *ad libitum*. The animals were acclimatized to laboratory conditions for 7 days prior to the experiment. All procedures described were reviewed and approved by the university animal ethics committee (367001/C/ CPCACA).

Oral Glucose Tolerance Test (OGTT):

The oral glucose tolerance test (OGTT) of MECC was performed at the doses of (200 and 400 mg/kg b.w.p.o) [12]. and blood glucose level was measured by one touch glucometer (accu-check). The glucose level was measured at 0, 30, 60, and 120 min after the administration of MECC.

Drugs and chemicals:

STZ was from Himedia, Mumbai, India. Glibenclamide was from Hoechst, India. Glibenclamide (Daonil®) tablets were powdered and made into a suspension in distilled water using 3%, v/v, aqueous Tween 80 as suspending agent. All other reagents used were of analytical grade obtained commercially.

Acute toxicity study:

MECC was administered orally to male Swiss albino mice to evaluate the acute toxicity as reported previously[13].

Induction of Experimental diabetes:

The rats were rendered diabetic by a single intraperitoneal dose of 55 mg/kg b.w. STZ freshly dissolved in ice cold 0.1 M citrate buffer (pH 4.5). After 72 h, fasting blood glucose (FBG) levels were measured and only those animals showing blood glucose level $\geq$ 225 mg/dl were considered for the present investigation. The day on which hyperglycemia had been confirmed was designated as day 0.

Experimental design and testing of fasting blood glucose level (FBG):

Thirty male Wistar albino rats (180–200 g) were divided into five groups (n = 6). The first group served as the normal non-diabetic control (saline control) group. Group II served as the diabetic control (STZ control). Groups III and IV received MECC 200 and 400 mg/kg b.w. (p.o.) daily for 15 consecutive days and group V received the reference drug glibenclamide (1 mg/kg, orally) daily for 15 consecutive days. Body weight and fasting blood glucose levels were measured at day 0, 5, 10 and 15 by using a digital balance and one touch glucometer (Accu-Chek®) [14]

Estimation of serum biochemical parameters:

After 15 days treatment, blood samples were drawn from overnight fasted rats by retroorbital venipuncture technique from light-ether anesthetized animals. The nonheparinized blood was allowed to coagulate before being centrifuged (4000 rpm for 20 min) and the serum separated. Serum levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDLC), high-density lipoprotein cholesterol (HDLC), glycosylated hemoglobin (HbA1C), aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) were estimated enzymatically using commercially available reagent kits (Erba Diagnostics and Span diagnostics Ltd.).

Statistical analysis:

All results are expressed as the mean ± SEM (standard error of the mean). The results were analyzed for statistical significance by one-way analysis of variance (ANOVA) followed by Dunnett's test using GraphPad Prism version 5.0 (GraphPad Software, USA).

RESULTS:

Acute toxicity:

The MECC did not show any toxic effect or death up to the dose of 2000 mg/kg, b.w., p.o. in mice.

Oral glucose tolerance (OGTT):

Effects of the MECC on glucose-loaded rats are shown in **Table 1.** Results of the OGTT strongly supported the improved ability of glucose tolerance with treatment of MECC and glibenclamide. Among the groups, the concentrations of blood glucose baseline (0 min) were not significantly different. Although plasma glucose levels were increased after loading with glucose, animals treated with MECC at 200 and 400 mg/kg showed slight increase when compared with the normal control group at 30, 60 and 120 min during OGTT. Glibenclamide significantly blocked ($p < 0.05$) the increase in blood glucose levels after glucose administration at 120 min.

Fasting blood glucose levels (FBG):

TABLE 1: EFFECT OF METHANOL EXTRACT OF *CUCUMIS CALLOSUS* (MECC) ON ORAL GLUCOSE TOLERANCE TEST IN NORMAL RATS.

	Oral glucose tolerance test (time in minutes)			
Group	**0**	**30**	**60**	**120**
Control glucose 4 g/kg	83.65 ± 2.60	137.35 ± 3.14	123.54 ± 3.48	114.26 ± 1.89
MECC (200 mg/kg)	90.53 ± 1.97	118.36 ± 2.78*	108.23 ± 2.87*	97.57 ± 1.63*
MECC (400 mg/kg)	87.56 ± 2.33	105.83 ± 1.71*	93.59 ± 1.83*	86.71 ± 2.26*
Glibenclamide	81.26 ± 3.21	82.35 ± 3.91*	75.56 ± 2.93*	70.39 ± 4.55*

Data are expressed as mean ± SEM (n=6); * p<0.05 when compared to normal control group

Fasting blood glucose levels measured in normal and STZ-induced diabetic rats after a single day and at the end of 5, 10, and 15 days of treatment are given in **Table 2**. Here, diabetic rats had a significant effect on blood glucose response after treat for 15 days. NC rats did not show any significant variation in the blood glucose throughout the experimental period. Administration of STZ (55 mg/ kg, i.p.) led to several fold elevation of blood glucose levels relative to that of the NC group, indicating stable hyperglycemia during the experimental period. MECC at the doses of 200 and 400 mg/ kg significantly ($p < 0.01$) reduced elevated glucose level towards normal control as compared to the diabetic control (STZ) group.

Effect on body weight:

TABLE 2: EFFECT OF MECC ON FASTING BLOOD GLUCOSE (FBG) LEVEL IN STZ INDUCED DIABETIC RATS.

	Fasting blood glucose level (mg/dl)			
Group	**Day 0**	**Day 5**	**Day 10**	**Day 15**
Normal Control	74±1.372	76±1.91	72±1.55	74±1.89
Diabetic Control STZ (50 mg/kg)	328±2.65[a]*	339±2.19[a]*	380±3.35[a*]	370±1.434[a]*
Diabetic + MECC (200 mg/kg BW)	375±1.35[b*]	253±1.89[b]*	167±1.67[b*]	151±1.73[b]*
Diabetic + MECC (400 mg/kg BW)	360±1.45[c]*	235±1.53[c]*	158±1.15c*	131±1.35[c]*
Diabetic + Glibenclamide	351±1.89[d]*	215±1.35[d]*	141±1.79d*	109±1.94[d]*

Each volume expressed as MEAN±SEM, where n=6, [a]* normal control group vs. diabetic control group ([b]*, [c]*, [d]* all treated group vs. diabetic control group [a]*) on corresponding day, p<0.05.

The effect of MECC on body weight of normal and diabetic animals is presented in **Table 3**. Normal control animals were found to be stable in their body weight but diabetic rats showed significant reduction in body weight during 15 days. STZ-induced body weight reduction, which was significantly (p < 0.05) reversed by MECC treatment.

Serum biochemical parameters:

TABLE 3: EFFECT OF MECC ON BODY WEIGHT IN STZ INDUCED DIABETIC RATS.

Group	**Body weight in gram**			
	Day 0	**Day 5**	**Day 10**	**Day 15**
Normal Control	180.81±1.35	182.21±2.40	183.4±1.89	184.1±1.43
Diabetic Control STZ (55 mg/kg)	179±1.18	156.1±2.29[a]*	148.10±1.55[a]*	135±1.84
Diabetic + MECC (200 mg/kg BW)	172±1.16	159.31±1.61[b]*	154.51±1.42[b]*	140±1.91[b]*
Diabetic + MECC (400 mg/kg BW)	182±1.77	161±1.82[c*]	155±1.63[c*]	146±1.44 [c*]
Diabetic + Glibenclamide	175±1.99	170.3±1.21[d*]	168±2.01[d*]	160.7±1.92[d*]

Each value is expressed as Mean ± SEM, where n=6, [a]* normal control group vs. diabetic control group. ([b]*, [c]*, [d]* all treated group vs. diabetic control group [a]*) on corresponding day, p<0.05.

Results of biochemical parameters are represented in **Table 4** and **4a**. MECC had a significant (p < 0.05) effect in lowering glycosylated haemoglobin (HbA1C) level in dose dependent manner after 15 days of treatment as compared to the NC group. There was a significant (p < 0.01) decrease in the level of serum HDL-cholesterol and significant (p< 0.05) increase in TC, LDLC and TGs level in diabetic rats when compared to normal control rats. Administration of MECC at 200 and 400 mg/kg and glibenclamide (1 mg/ kg) significantly (p < 0.05) brought their levels toward normal. The activities of serum enzymes AST, ALT, and ALP were found to be significantly (p < 0.05) increased in diabetic rats compared to normal rats. Oral administration of MECC at 200 and 400 mg/kg and glibenclamide at 1 mg/ kg for 15 days significantly (p < 0.05) normalized the enzymatic activities in diabetic rats.

TABLE 4: EFFECT OF MECC ON SERUM BIOCHEMICAL PARAMETERS IN NORMAL AND STZ INDUCED DIABETIC RATS

	Serum biochemical parameters				
Group	**Glycosylated haemoglobin**	**Total cholesterol**	**Triglyceride**	**HDL-Cholesterol**	**LDL-Cholesterol**
Normal Control	5.38±0.091	81.76±1.64	67.41±1.18	81.51±1.89	38.23±1.91
Diabetic Control (STZ, 55 mg/kg)	11.74±0.87[a]*	192.31±2.41[a]*	192.41±1.37[a]*	25.76±1.29[a]*	141.96±2.02[a*]
STZ + MECC (200 mg/kg BW)	7.42±0.07[b]*	128.40±1.35[b]*	79.29±1.50[b]*	47.8±1.71[b*]	83.67±1.65[b*]
STZ + MECC (400 mg/kg BW)	6.18±0.06[c]*	117.72±1.32[c]*	77.48±1.41[c]*	55.80±1.98[c]*	64.19±2.18[c*]
STZ+ Glibenclamide	5.60±0.04[d]*	102.11±1.79[d]*	74.56±2.10[d]*	64.0±1.83[d]*	45.72±2.28[d*]

Each value is expressed as Mean ± SEM, where n=6, [a]* normal control group vs. diabetic control group. ([b]*, [c]*, [d]* all treated group vs. diabetic control group [a]*) on corresponding day, p<0.05.

TABLE 4a: EFFECT OF MECC ON SERUM ENZYMATIC PARAMETERS IN NORMAL AND STZ INDUCED DIABETIC RATS.

Groups	SGOT (IU/L)	SGPT (IU/L)	ALP (IU/L)
Normal Control	37.10±1.19	35±1.37	157.61±1.54
Diabetic Control STZ (55 mg/kg)	62.15±1.29[a]*	56.23±2.18[a]*	279.31±1.34[a]*
Diabetic + MECC (200 mg/kg BW)	52.13±1.37[b]*	5170±1.45[b]*	113.81±1.69[b]*
Diabetic + MECC (400 mg/kg BW)	45±1.62[c]*	41.76±1.41[c]*	181.21±1.56[c]*
Diabetic + Glibenclamide	39.16±1.21	39.83±1.15[d]*	177.14±1.81[d]*

Each value is expressed as Mean ± SEM, where n=6, [a]* normal control group vs. diabetic control group. ([b]*, [c]*, [d]* all treated group vs. diabetic control group [a]*) on corresponding day, p<0.05

DISCUSSION: Diabetes mellitus is a chronic metabolic disorder caused by partial or complete deficiency of insulin, which causes a disturbance in the uptake of glucose as well as glucose metabolism and leads to several acute and chronic pathological complications. Phytotherapy has been highly accepted worldwide in the health care system for diabetes mellitus. In the present study, the hypoglycemic activity of MECC was evaluated in STZ-induced diabetic rats.

STZ- at the dose of 55 mg/kg, i.p., was used to induce hyperglycemia after performing a pilot study for optimized dose to elevate blood glucose >225 mg/dl. The use of lower dose of STZ (55 mg/kg) produced an incomplete destruction of pancreatic β cells even though the rats become permanently diabetic [14]. MECC exhibited significant reduction in blood glucose level in diabetic rats at the doses of 200 and 400 mg/ kg towards normal control. The hypoglycemic action of MECC may be due to augmentation of insulin release from existing β cells of the islets of langerhans. The plasma glucose lowering activity was compared with that of glibenclamide, the reference oral hypoglycemic which has been used for many years to treat diabetes mellitus, to stimulate pancreatic β cells [15].

From the results of this study, it appears that insulin producing β cells are still functioning and the stimulation of insulin release could be responsible for most of the metabolic function. It may be suggested that the mechanism of hypoglycemic action of MECC is similar to glibenclamide.

Induction of diabetes with STZ is associated with a characteristic loss of body weight, during the observation period of 14 days even though the food intake was more in diabetic rats than normal control animals. It was due to increased muscle wasting and loss of tissue proteins [16]. STZ-induced insulin deficiency may lead to decrease protein content in muscular tissue by proteolysis [17]. Diabetic rats treated with the MECC showed significant improvement in body weight as compared to the STZ control animals, Hence, MECC exhibited marked effect in controlling the loss of body weights of diabetic rats.

Oral administration of MECC decreased the level of HbA1C. Lower levels of total hemoglobin observed in diabetic rats might be due to the increased formation of HbA1C. Glycohemoglobin is formed throughout the circulatory life of red blood cells (RBCs) by the addition of glucose to the N-terminal of the hemoglobin β chain. This process, which is non enzymatic, reflects the average exposure of hemoglobin to glucose over an extended period [14].

Lipids play an important role in the pathogenesis of diabetes mellitus. It is well known that in uncontrolled diabetes mellitus, there is an increase in total cholesterol in blood, which may contribute to coronary artery diseases [16].

The most common lipid abnormalities in diabetes are hypertriglyceridemia and hypercholesterolemia. In this study, elevated levels of serum lipids such as TC, LDLC and TGs were found in diabetic rats. STZ produced various cardinal symptoms of

diabetes mellitus including hypoinsulinemia, a condition that is probably responsible for the elevation of serum cholesterol levels because the insulin has an inhibitory action on HMG-CoA reductase, a key enzyme that acts as rate limiting in the metabolism of cholesterol rich LDL particles [18]. In insulin-deficient diabetes, the concentration of serum fatty acids is elevated as a result of free fatty acid outflow from fat depots, where the balance of the free fatty acid esterification-TG lipolysis cycle is displaced in favor of lipolysis [19]. High-density lipoprotein (HDL) is an antiatherogenic lipoprotein. It transports cholesterol from peripheral tissues into the liver and thereby acts as a protective factor against coronary heart disease. The level of HDLC, which increased after MECC administration, might be due to the increase in the activity of lecithin cholesterol acyl transferase (LCAT), which may contribute to the regulation of blood lipids [19]. Oral administration of MECC reduced the elevated serum lipids such as TC, LDLC and TGs toward normal in diabetic rats.

Elevation of serum biomarker enzymes such as SGOT, SGPT, and SALP was observed in diabetic rats indicating impaired liver function, which was obviously due to hepatocellular necrosis. It has been reported that liver necrosis occurred in STZ-induced diabetic rats. Therefore, increase in the activities of AST, ALT and ALP gives an indication on the hepatotoxic effect of STZ. 14-days of treatment with MECC restored all the above-mentioned serum hepatic biochemical parameters toward the normal values in a dose-dependent manner, thereby alleviating liver damage caused by STZ-induced diabetes.

In this study, administration of MECC to STZ-induced hyperglycemic rats demonstrated prominent reduction in blood sugar level, normalization of serum biochemical profiles including lipid contents, comparing to STZ control rats. Therefore, it can be concluded that the MECC is remarkably effective against STZ-induced diabetes in Wistar albino rats thereby validating its ethnomedicinal usage. From the observed oral hypoglycemic activity of *Cucumis callosus* seed extract in STZ-induced diabetic rats, it can be further inferred that *Cucumis callosus* seed can serve as an interesting candidate in complementary and alternative medicine for the effective management of diabetes mellitus.

CONCLUSION: The result of the present investigation is quite encouraging and it explores the potent antidiabetic activity of MECC probably because of its secondary metabolite which is further potentiated by its antioxidant properties. Further investigation was going on to find out the molecular mechanism for which antidiabetic activity shown.

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