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ORIGINAL ARTICLE

Hypoglycemic potential of alcoholic root extract of *Cassia occidentalis* Linn. in streptozotocin induced diabetes in albino mice

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KEYWORDS

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Abstract *Objective:* *Cassia occidentalis* (CO) (family: Caesalpiniaceae) is a common weed which is widely used to treat inflammation, hepatotoxicity, antimalarial activities, sore eyes, hematuria, rheumatism, typhoid, asthma, leprosy and diabetes in folklore medicine in India. The present study was carried out to investigate the antidiabetic activity of ethanolic extract of *C. occidentalis* roots.
Methods: Root extract of *C. occidentalis* (RCO) was administered orally at two doses (250 and 500 mg/kg) to normal and streptozotocin (STZ) induced NIDDM. Fasting blood glucose (FBG) level, biochemical parameters like blood glucose, serum cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides (TG), total protein, urea, creatinine, serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT) levels and physical parameters like change in body weight, food intake, water intake and levels in liver were performed for the evaluation of hypoglycemic effects.
Results: Both the doses of RCO caused a marked decrease in FBG levels in STZ induced type 2 diabetic mice. RCO decreased the blood glucose, food intake, water intake, organ weight, serum cholesterol, TG, creatinine, SGOT and SGPT levels with significant value and increased the levels of HDL cholesterol and total protein with a significant value ($P < 0.05$–0.01). The decrease in body weight induced by STZ was restored with a significant value ($P < 0.01$) at both doses.
Conclusion: The results suggest that ethanolic roots extract of *C. occidentalis* Linn. possesses hypoglycemic potential for the NIDDM and support the traditional use of the roots of plant as hypoglycemic agent.

1. Introduction

Diabetes mellitus (DM) is a complicated, chronic metabolic disorder characterized by either deficiency of insulin

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production due to destructive lesions of pancreatic β-cells or by cellular resistance to insulin.[1] Among the diabetics, about 10% have IDDM (insulin deficient), while 90% have NIDDM (insulin resistant).[2] NIDDM begins with a period of insulin resistance with augmented pancreatic insulin secretion. As the disease progresses, pancreas loses its function and thus no longer able to meet peripheral demands. As a result, insulin fails to fulfil the body requirements.[3] DM has caused significant morbidity and mortality due to microvascular (retinopathy, neuropathy and nephropathy) and macrovascular (heart attack, stroke and peripheral vascular disease) complications.[4] The number of people suffering from the disease worldwide is estimated to be over 173 million and this figure is likely to be increased to 300 million or more by the year 2025.[5] Diabetes is increasing at an alarming rate with a projected 366 million people likely to be diabetic by the year 2030 as against 191 million estimated in 2000.[6] Approximately 300,000 deaths each year are attributed to diabetes. Its prevalence increases with age, from about 0.2% in persons less than 17 years of age to about 10% in persons aged 65 years.[7] Therapeutic options for diabetes are diet, exercise, oral hypoglycemic drugs and insulin therapy.[8] These drugs have been used as monotherapy or in different combinations so as to control diabetic condition. Considerable progress has been done in the treatment of diabetes by oral hypoglycemic agents, but search for newer drugs continues because of the various limitations of the synthetic drugs. Recently it has been identified by Indian Council of Medical Research (ICMR) that diabetes mellitus is one of the refractory diseases for which satisfactory treatment is not available in modern allopathic system of medicine and hence suitable herbal preparations are to be investigated which should be therapeutically effective in controlling diabetes.[9] Herbal drugs enjoy the advantages of comparatively less toxic than synthetic drugs, more harmony with the biological system and affordable to all classes of people.[10] Herbal preparations are the oldest and widely useful remedies known to mankind. India has one of the oldest, richest and most diverse cultures associated with the use of herbal medicines widely termed as ayurveda.[11] A large number of plant preparations have been reported to possess antidiabetic activity over last several decades. Researchers in India have documented the use of over 150 plants in various families with antidiabetic activity.[9]

Cassia occidentalis (family: Caesalpiniaceae) is a common weed scattered from the foothills of Himalayas to West Bengal, South India, Burma, and Sri Lanka. The plant is a diffuse (usually annual) under shrub with loosely spreading branches 60–150 cm long, found throughout India, up to an altitude of 1500 m.[12] *C. occidentalis* Linn. is an annual or perennial ayurvedic plant widely used in several traditional medicines to cure various diseases. This weed has been known to possess antibacterial, antifungal, antidiabetic, anti-inflammatory, antimutagenic and hepatoprotective activity. A wide range of chemical compounds including achrosin, aloe-emodin, emodin, anthraquinones, anthrones, apigenin, aurantiobtusin, campesterol, cassiollin, chryso-obtusin, chrysophanic acid, chrysarobin, chrysophanol and chrysoeriol have been isolated from this plant.[13] No study was conducted scientifically to prove the antidiabetic activity of roots of *C. occidentalis*. Hence the present study was conducted to prove the antidiabetic activity of *C. occidentalis* roots.

2. Materials and methods

All the experimental procedures and protocols used in the study were reviewed by the Institutional Animal Ethics Committee (IAEC) (Register Number: 536/02/a/CPCSEA) and were in accordance with the Committee for the purpose of Control and Supervision on Experiments on Animals (CPCSEA) guidelines, Government of India.

2.1. Animals

Healthy Swiss albino mice (25–30 g) of either sex of Wistar strain were obtained from a disease free animal house of Chaudhary Charan Singh, Haryana Agriculture University, Hisar, Haryana (India). They were housed in the animal house, Institute of Pharmaceutical Sciences, Kurukshetra University, Kurukshetra, Haryana (India). Animals were kept in standard polypropylene cages and maintained under controlled standard conditions of temperature (25 ± 5 °C), relative humidity (55 ± 5%), with 12/12 h light/dark cycle. They were fed with commercially available mice feed.

2.2. Drugs and chemicals

Streptozotocin was obtained from Himedia, and Metformin (MT) is a gift sample from Gnosis Pharma, Sirmour, Himachal Pradesh; HDL-cholesterol, TG, cholesterol, creatinine, urea, total protein, SGOT and SGPT estimation kits were obtained from ERBA Diagnostics Mannheim GmbH, Mallaustr, Germany. All other chemicals used were of AR grade.

2.3. Plant material

The roots of CO were collected from the banks of river Yamuna and surrounding local areas of Yamunanagar district during Sept–Oct, 2009. Then, collected roots were identified by Dr. B.D. Vashisht, (Head) Botany Department, Kurukshetra University, Kurukshetra, Haryana, India.

2.4. Extraction method

Roots were washed and cleaned thoroughly so as to remove any contamination. Then the washed plant parts were air dried in shade, powdered and passed through a sieve of mesh size no. 40. Thus the obtained coarse powder was subjected to Soxhlet extraction for 48 h using alcohol in the ratio of 70:30. The extract was distilled and last traces of solvent were removed by rotary evaporator under reduced pressure. Percentage yield of root extract was 10.2%. The extract obtained was preserved in air tight glass container at 4–8 °C for future studies.

2.5. Preliminary phytochemical study

Chemical tests were carried out on RCO extract for the qualitative determination of phytochemical constituents as described by Khandelwal.[14]

2.6. Drug solution

Extract was dissolved in tween 80 suspensions (0.5%, p.o.).

2.7. Acute toxicity study

Overnight fasted mice of either sex were divided into five groups of six mice in each group. The test extracts at increasing doses (125–2000 mg/kg, b.w.) were administered orally and toxicity was evaluated as per the Guidelines for non-clinical toxicity investigation of Herbal Medicine (Annexure-1) given by the Ministry of Health and Family Welfare, Government of India.[15] The animals were continuously observed for 2 h for behavioral, neurological or autonomic toxic effects and for any lethality or death after 24 and 72 h.

2.8. Oral glucose tolerance study

The effect of RCO extract was evaluated on glucose loaded animals. The blood glucose levels were monitored at various time intervals after single oral administration of extract.[16]

2.8.1. Experimental procedure

Overnight fasted mice were randomly divided into four groups, each group containing six animals. Glucose (2 g/kg, p.o.) was administered to each group.

- Group I: Vehicle control, tween 80 suspension (0.5%, p.o.).
- Groups II–III: RCO extract (250 and 500 mg/kg, p.o.).
- Group IV: MT (0.5 mg/kg, p.o.).

After overnight fasting blood samples were collected from tail vein at 0, 30, 60, 120 min after administration of the treatments. Blood glucose levels were determined by one touch electronic glucometer, using glucose strips.

2.9. Streptozotocin induced diabetic study

2.9.1. Induction of diabetes

150 mg/kg STZ (prepared using fresh cold citrate buffer pH 4.5) was injected in overnight fasted mice. Free access to 5% glucose solution, food and water was provided to counter the hypoglycemic shock. Determination of FBG level was done after 72 h and on 7th day of injection to confirm stable hyperglycemia. Mice showing FBG levels more than 150 mg/dl were selected for the antidiabetic study.[17]

2.9.2. Experimental procedure

Overnight fasted diabetic mice were divided into five groups of six mice each. Water was given ad libitum. Treatment was provided in the following manner:

- Group I: Vehicle control, tween 80 suspension (0.5% v/v, p.o.).
- Group II: Diabetic control, tween 80 suspension (0.5% v/v, p.o.).
- Groups III–IV: RCO extract (250 and 500 mg/kg, p.o.).
- Group V: MT (0.5 mg/kg, p.o.).

The effects of extract were studied in all the groups, for 21 days. Blood glucose levels were determined after two hours of various treatments, by withdrawing blood samples from the tail vein from overnight fasted animals on 0th, 7th, 14th and 21st by using elegance glucometer, using glucose strips.

2.9.3. Physical parameters

The changes in body weight, food and water intake were monitored on the 0th, 7th, 14th, and 21st day of treatment.

2.9.4. Collection of organs

Liver, kidney, pancreas, heart, lungs, and spleen were isolated from animals and were weighed.

2.9.5. Biochemical parameters

Blood glucose, serum cholesterol, HDL cholesterol, TG, total protein, urea, creatinine, SGOT and SGPT were estimated by using various kit methods.

2.10. Statistical analysis

Data obtained from pharmacological experiments, are expressed as mean ± SEM. Differences between control and treated groups were tested for significance using ANOVA followed by Dunnett's *t*-test, with $P < 0.05$ were considered as significant.

3. Results

3.1. Preliminary phytoconstituents

Preliminary phytochemical screening revealed the presence of flavonoids, glycosides, phytosterols, tannins and triterpenoids.

3.2. Acute toxicity study

Extract treated mice showed no lethality or any discernible behavioral changes up to 2000 mg/kg by oral route. No mortality was observed at this dose during 24 h observation period.

3.3. Oral glucose tolerance test

The effects of RCO extract were evaluated on the glucose loaded normal mice. The blood glucose levels were monitored at various time intervals after single administration of extract (Table 1). Decrease in blood glucose levels was observed during the first 60 min in MT and RCO at 250 mg/kg, then starts increasing at 120 min but then blood glucose levels were reduced constantly up to 270 min. These changes in blood glucose levels were significant ($^{**}P < 0.01$), ($^{*}P < 0.05$) when compared with vehicle control group. Decrease in glucose level was observed at both (250 and 500 mg/kg) the doses.

3.4. Streptozotocin induced diabetes in mice

The hypoglycemic effect of the extract on the FBG of diabetic mice is shown in Table 2. Administration of STZ (150 mg/kg, i.p.) led to increase in fasting hyperglycemia, which was

Table 1 Effect of RCO extract on the blood sugar level of glucose loaded mice.

Groups	*n*	0 min (mg/dl)	30 min (mg/dl)	60 min (glucose loading) (mg/dl)	120 min (mg/dl)	150 min (mg/dl)	270 min (mg/dl)
Vehicle control	6	85 ± 1.50	83.33 ± 1.50	82.4 ± 1.40	145 ± 1.60	140.83 ± 1.10	121.16 ± 2.90
RCO (250 mg/kg, b.w.)	6	89 ± 0.70	87 ± 0.90	84.5 ± 1.10	141.5 ± 1.50	132.16 ± 2.60*	111 ± 3.30**
RCO (500 mg/kg, b.w.)	6	86.83 ± 0.80	85.83 ± 0.60	88 ± 1.10**	138.33 ± 1.40**	122.66 ± 1.80**	100.5 ± 0.90**
MT (0.5 mg/kg, b.w.)	6	84.83 ± 1.60	79.5 ± 1.20	74.83 ± 1.50**	131.5 ± 1.10**	103 ± 2.20**	90.5 ± 1.90**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
* $P < 0.05$.
** $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

Table 2 Effect of RCO extracts on the blood glucose level of STZ-induced DM in mice.

Groups	*n*	0th day (g)	7th day (g)	14th day (g)	21st day (g)
Vehicle control	6	76.16 ± 4.80	80 ± 2.60	79.33 ± 3.10	80.66 ± 4.00
Diabetic control	6	185.16 ± 9.60$^{\wedge\wedge}$	187.33 ± 7.50$^{\wedge\wedge}$	188..83 ± 8.70$^{\wedge\wedge}$	191.83 ± 8.90$^{\wedge\wedge}$
RCO (250 mg/kg, b.w.)	6	179.5 ± 3.90	156.5 ± 4.40	137 ± 4.60**	114 ± 1.60**
RCO (500 mg/kg, b.w.)	6	178.16 ± 10.00	158.16 ± 10.20	128.16 ± 11.10**	102.83 ± 5.90**
MT (0.5 mg/kg, b.w.)	6	188.83 ± 19.20	151 ± 16.50	126 ± 13.80**	97.33 ± 5.00**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
** $P < 0.01$ *vs* diabetic control.
$^{\wedge\wedge}$ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

maintained over a period of 21 days. RCO extracts at both the doses showed significant ($^{**}P < 0.01$) decrease in FBG levels but effect at 500 mg/kg was superior.

3.5. Physical parameters

Table 3 shows the effect of RCO and MT on body weight of STZ-induced diabetic mice. Diabetic control mice showed decrease in body weight during the study. Administration of RCO extracts reversed the reduction in body weight. The result obtained was significant ($^{**}P < 0.01$) at both doses (250 and 500 mg/kg) but more significant at 500 mg/kg.

Polyphagia and polydipsia in diabetic animals lead to increase in food and water intake which is induced due to uptake of STZ (Table 4). Animals treated with doses (250 mg/kg and 500 mg/kg) of RCO extracts, showed significant ($P < 0.01$) decrease in food and water intake after 21 days of treatment.

3.6. Organ weight

As shown in Table 5, STZ-induced diabetes increased the weight of liver, kidney and pancreas. The increase in weights of these organs was reversed by administration of RCO extract and MT. The increase in weights is significant ($^{**}P < 0.01$), ($^{*}P < 0.05$) at both the doses but superior results were obtained at 500 mg/kg of RCO extract and MT.

3.7. Biochemical parameters

Serum cholesterol, TG and creatinine levels were increased with STZ induced diabetes. 250 and 500 mg/kg of RCO extract induced significant ($^{*}P < 0.05$) and ($^{**}P < 0.01$) decrease in the level of serum creatinine whereas serum cholesterol and TG were decreased significantly only at dose 500 mg/kg (Table 6). HDL cholesterol and total protein were decreased in diabetic mice. There was a significant ($^{**}P < 0.01$),

Table 3 Effect of RCO extracts on the body weight of STZ-induced DM in mice.

Group	*n*	0th day (g)	7th day (g)	14th day (g)	21st day (g)
Vehicle control	6	28.81 ± 0.10	29.24 ± 0.09	29.49 ± 0.10	29.69 ± 0.09
Diabetic control	6	25.23 ± 0.30	24.84 ± 0.30$^{\wedge\wedge}$	24.56 ± 0.30$^{\wedge\wedge}$	24.12 ± 0.30$^{\wedge\wedge}$
RCO (250 mg/kg, b.w.)	6	24.97 ± 0.20	25.62 ± 0.30	26.19 ± 0.20**	26.50 ± 0.20**
RCO (500 mg/kg, b.w.)	6	25.45 ± 0.40	26.25 ± 0.30**	26.29 ± 0.20**	27.63 ± 0.20**
MT (0.5 mg/kg, b.w.)	6	25.96 ± 0.30	27.00 ± 0.30**	27.85 ± 0.40**	28.10 ± 0.20**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
** $P < 0.01$ *vs* diabetic control.
$^{\wedge\wedge}$ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

Table 4 Effect of RCO extracts on the food and water intake of STZ-induced DM in mice.

Groups	*n*	0th day		7th day		14th day		21st day	
		Water intake (ml)	Food intake (g)	Water intake (ml)	Food intake (g)	Water intake (ml)	Food intake (g)	Water intake (ml)	Food intake (g)
Vehicle control	6	31.5 ± 1.20	6.13 ± 0.30	32.83 ± 0.40	6.41 ± 0.20	33.16 ± 0.70	6.86 ± 0.20	33.66 ± 0.60	7.44 ± 0.20
Diabetic control	6	43.0 ± 0.80 ^^	16.48 ± 0.60 ^^	45.83 ± 1.00 ^^	16.82 ± 0.60 ^^	44.16 ± 1.50 ^^	17.16 ± 0.60 ^^	47.33 ± 1.50 ^^	17.55 ± 0.50 ^^
RCO (250 mg/kg, b.w.)	6	44.0 ± 0.90	16.55 ± 0.20	45.16 ± 1.10	16.33 ± 0.20	42.66 ± 0.70	16.08 ± 0.20	40.83 ± 0.40**	15.72 ± 0.20**
RCO (500 mg/kg, b.w)	6	45.16 ± 0.60	15.58 ± 0.30	43 ± 0.50	15.21 ± 0.40**	38.33 ± 0.60**	14.86 ± 0.40**	37.5 ± 0.60**	14.52 ± 0.40**
MT (0.5 mg/kg, b.w.)	6	45.5 ± 0.60	15.97 ± 0.20	39.83 ± 0.60**	14.87 ± 0.20**	35.33 ± 0.50**	14.34 ± 0.10**	33 ± 0.50**	13.79 ± 0.10**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
** $P < 0.01$ *vs* diabetic control.
^^ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

(* $P < 0.05$) increase in total protein only at both the doses where as in HDL cholesterol no significant increase was observed.

Diabetic control mice have significantly increased levels of SGOT and SGPT levels (Table 7). Administration of RCO extracts for 21 days induced significant (** $P < 0.01$) decrease in SGOT and SGPT levels when compared to diabetic control mice.

4. Discussion

Diabetes mellitus is one of the most common chronic diseases associated with carbohydrate metabolism. It is also an indication of co-morbidities such as obesity, hypertension, and hyperlipidemia which are metabolic complications of both clinical and experimental diabetes.[6] In the present study the hypoglycemic activity of root extract of *C. occidentalis* was evaluated in Streptozotocin induced diabetic mice. STZ selectively destroys pancreatic insulin secreting β cells by causing diabetes close to type 2 diabetes in mice.[11] The continuous treatment of root extract for a period of 21 days has shown significant results in STZ induced mice. The number of functionally intact β cells in the islet organ is of decisive importance for the development course and outcome of DM. The renewal of β-cells in diabetes has been studied in several animal models. The total β-cell mass reflects the balance between the renewal and loss of these cells.[18] RCO extracts showed hypoglycemic activity by acting through one of the following mechanisms.

Like some hypothesis relates to the effects of plant extracts on the activity of pancreatic beta cells, increase in the inhibitory effect against insulinase enzyme, increase of the insulin sensitivity or the insulin like activity of the plant extracts. Other mechanisms may also be involved such as increase of peripheral utilization of glucose, increase of synthesis of hepatic glycogen or decrease of glycogenolysis, inhibition of intestinal glucose absorption, reduction of glycaemic index of carbohydrates and reduction of effect of glutathione.[4]

Total % reduction in glucose level was observed in normal mice when compared to the vehicle control group. The decrease in blood glucose level may be due to potentiation of insulin effect either by increase in pancreatic secretion of insulin from beta cells of islets of Langerhans or by increase in peripheral glucose uptake.[19] RCO extract treated animals have been shown to increase body weight as compared to diabetic control. Induction of diabetes by STZ leads to loss of body weight due to increased muscle wasting and loss of tissue proteins.[20] The decrease in body weight in diabetes was due to the increased muscle wasting and loss of tissue proteins.[21] The extract treated animals recovered the body weight significantly toward normal level which may be due to the lipid lowering activity of the extract or indirectly to the influence on various lipid regulation systems. The observed hypolipidemic effect may be due to inhibition of fatty acid synthesis and decreased cholesterogenesis.[22] MT is a biguanide which exerts its effects on glucose transport via acting through insulin-mediated enhanced peripheral glucose uptake.[23]

In both types of diabetes mellitus polyuria, polydipsia and polyphagia symptoms develop. When the glucose concentration in the blood is raised beyond the renal threshold, reabsorption of glucose in the proximal renal tubule is incomplete and part of the glucose remains in the urine

Table 5 Effect of RCO extracts on the organ weights of kidney, liver and pancreas of STZ induced DM in mice.

Groups	*n*	Kidney (g)	Liver (g)	Pancreas (g)
Vehicle control	6	0.53 ± 0.00	2.07 ± 0.01	0.05 ± 0.00
Diabetic control	6	0.94 ± 0.01$^{\wedge\wedge}$	2.72 ± 0.04$^{\wedge\wedge}$	0.18 ± 0.00$^{\wedge\wedge}$
RCO (250 mg/kg, b.w.)	6	0.79 ± 0.01**	2.24 ± 0.01**	0.16 ± 0.00*
RCO (500 mg/kg, b.w.)	6	0.72 ± 0.00**	2.19 ± 0.00**	0.13 ± 0.00**
MT (0.5 mg/kg, b.w.)	6	0.59 ± 0.00**	2.15 ± 0.01**	0.09 ± 0.00**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
* $P < 0.05$.
** $P < 0.01$ *vs* diabetic control.
$^{\wedge\wedge}$ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

Table 6 Effect of RCO extracts on the serum profile of STZ- induced DM in mice.

Groups	*n*	Serum cholesterol (mg/dl)	Serum HDL cholesterol (mg/dl)	Serum triglycerides(mg/dl)	Serum creatinine (mg/dl)	Serum total protein (g/dl)
Vehicle control	6	64.33 ± 2.90	35.27 ± 1.80	67.87 ± 1.90	0.59 ± 0.01	7.3 ± 0.20
Diabetic control	6	78.31 ± 3.90$^{\wedge\wedge}$	31.82 ± 1.40	91.64 ± 2.40$^{\wedge\wedge}$	1.76 ± 0.01$^{\wedge\wedge}$	3.4 ± 0.10$^{\wedge\wedge}$
RCO (250 mg/kg, b.w.)	6	69.12 ± 1.90	36.09 ± 2.20	86.69 ± 1.70	0.96 ± 0.01**	4.0 ± 0.10*
RCO (500 mg/kg, b.w.)	6	66.90 ± 2.30*	38.20 ± 1.50	78.54 ± 1.40*	0.80 ± 0.00**	4.4 ± 0.10**
MT (0.5 mg/kg, b.w.)	6	61.80 ± 3.70**	38.74 ± 1.90*	91.64 ± 6.50	0.71 ± 0.00**	6.4 ± 0.20**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: 0.5% v/v, tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
* $P < 0.05$ *vs* diabetic control.
** $P < 0.01$.
$^{\wedge\wedge}$ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

Table 7 Effect of RCO extracts on the SGOT and SGPT levels of STZ-induced DM in mice.

Groups	*n*	SGOT level (IU/L)	SGPT level (IU/L)
Vehicle control	6	93.3 ± 1.6	58.7 ± 4.5
Diabetic control	6	99.5 ± 1.9	122 ± 8.5$^{\wedge\wedge}$
RCO (250 mg/kg, b.w.)	6	82 ± 2.4**	89.5 ± 2.0**
RCO (500 mg/kg, b.w.)	6	75 ± 0.7**	80.5 ± 2.4**
MT (0.5 mg/kg, b.w.)	6	62 ± 1.6**	69.5 ± 3.2**

The values are mean ± SEM, *n* = number of animals used.
Vehicle control: (0.5% v/v); tween 80; RCO: Root extract of *Cassia occidentalis*; MT: Metformin.
** $P < 0.01$ *vs* diabetic control.
$^{\wedge\wedge}$ $P < 0.01$ *vs* vehicle control (One way ANOVA followed by Dunnett's, Multiple comparison test).

(glycosuria). This increases the osmotic pressure of the urine and inhibits the reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume will be replaced osmotically by water held in body cells, causing dehydration and increased thirst. The hormone insulin is also responsible for stimulating hunger. In order to cope up with high sugar level in blood; body produces insulin which leads to increased hunger. In the present study, RCO extract, showed a significant ($P < 0.01$) decrease in food and water intake after 21 days of treatment.[24]

STZ-induced diabetes increased the weight of liver, kidney, pancreas in normal mice. An alteration in the internal organ weights may primarily indicate toxicity or pathology occurring to these organs.[25] But treatment with RCO extracts has shown significant reduction in organ weight at both doses.

FBG levels have been reduced to a significant value which was earlier increased due to administration of STZ (150 mg/kg, i.p.). The decrease in FBG levels may be due to increase in the activity of enzymes responsible for utilization of glucose by insulin dependent pathway or regenerate b-cells in pancreatic islets.[8]

A significant reduction in serum cholesterol, HDL cholesterol, TG, serum total protein and creatinine was observed in STZ induced diabetic mice, when compared to vehicle control and MT treated mice. On administration of RCO extract to the diabetic mice serum profile levels were found to be restored to normal. Significant decrease in TG, HDL cholesterol, creatinine, cholesterol and total protein levels was observed. Significant reduction in SGOT and SGPT levels was found in RCO extract treated animals. Results were significant ($^{**}P < 0.01$) at both doses. The results of the study were satisfactory and revealed that the RCO extracts have exhibited hypoglycemic activity.

5. Conclusions

Ethanolic RCO extract exhibited significant hypoglycemic activities in STZ induced diabetic mice. The extract showed improvement in various body and serum parameters as well as regeneration of β cells of pancreas and so might be of value in diabetes. However, further phytochemical investigations are required to isolate and identify the hypoglycemic principles in the plant as well as elucidating their mechanism of action.

6. Conflict of interest

We declare that we have no conflict of interest.

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