Antidiabetic and antioxidant activity of *Rhizophora mucronata* leaves (Indian sundarban mangrove): An *in vitro* and *in vivo* study

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Abstract

Background: *Rhizophora mucronata* is a salt-tolerant true mangrove which is widely distributed in Indian mangrove forest and traditionally used to treat diabetes and other health ailments. **Aim:** The aim of this study is to elucidate the role of Indian variety of *R. mucronata* leaves on glucose impairing metabolism during diabetes by *in vitro* and *in vivo* methods. **Materials and Methods:** The ethanolic fraction of *R. mucronata* leaves extract (RHE) was assessed for DPPH (1,1-diphenyl-2-picrylhydrazyl) radical scavenging and *in vitro* anti-diabetic action through α-amylase and α-glucosidase activity assessment. Oral glucose tolerance test (OGTT) and insulin sensitivity test (IST) were assessed and their counteraction with RHE (100 and 200 mg/kg, p.o) and glibenclamide (10 mg/kg, p.o) in streptozotocin (STZ) (50 mg/kg, intravenous) induced hyperglycemic rats were also monitored for 28 days. The data were analyzed statistically using t-test. **Results:** RHE dose-dependently inhibited α-amylase and α-glucosidase enzymes and lowered the area under the curve (AUC) for glucose on both OGTT and IST. RHE also significantly ($p < 0.01$) controlled glycemic index and thereby reducing diabetic complications as assessed by lipid profiles, atherogenic index, and coronary index in STZ rats. **Conclusion:** RHE at doses of 100 and 200 mg/kg/day for 28 days provided a significant decrease in diabetes complications and metabolic impairment.

Keywords: α-amylase, α-glucosidase, diabetes, insulin, mangrove, *Rhizophora mucronata*

Introduction

Rhizophora mucronata Lam. (Rhizophoraceae), a true mangrove, is widely distributed along the delta of Indian Sunderbans (21°32 and 22°40 N and between 88°05 and 80°00 E).[1] The bark, root, leaves, fruit and flowers of *R. mucronata* have been traditionally used as medicine in the coastal areas of Asian subcontinents for treating health ailments such as diabetes,[2,3] diarrhea,[4] hepatitis,[5] inflammation,[6] and cognitive function3.[7] The perspective of its use as anti-diabetic medicine was supported with numerous scientific reports, but more information is still required.[8,9] Chemical identity of *R. mucronata* has also been carried out, and the presence of secolabdane diterpenoid (*rhizophorin A*),[10] phomoxanthone,[11] lupiol, beta-sitosterol,[12] gallic acid, coumarin, quercetin,[13] and tannins,[14] though antidiabetic principle(s) are yet to be confirmed. Diabetes is a metabolic disorder resulting from a defect in insulin secretion, insulin action, or both. Its global prevalence was about 8% in 2011 and is predicted to rise to 10% by 2030.[15] As metabolic syndrome is characterized by a combination of risk factors for cardiovascular diseases and diabetes that usually link to insulin deficit or dysfunction;[16] hence, it is essential to find out the role of newly develop antidiabetic principle(s) on that point of view. In the above context study was conducted to assess the effect of ethanolic fraction of Indian *R. mucronata* leaves on complications of diabetes glycemic control and lipid metabolism impairment in the *in vitro* and *in vivo* to elicit the underlying insulin facilitatory action of it.

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Materials and methods

Animals

Sixty male Wistar rats weighing 150–175 g were used for antihyperglycemic studies. The animals were kept in the departmental animal house, maintaining standard condition (room temperature 22°C ± 2°C, relative humidity 60%–65%, and 12 h light dark cycles), and fed with proper diet and water *ad libitum*. Ethical clearance for the animal study was obtained from the Institutional Animal Ethics Committee (RKC/IAEC/13/17/1).

Drug preparation

The fresh leaves of *R. mucronata* were collected from the forest area of Sunderban, South 24 Parganas, West Bengal, and identified from the Botanical Survey of India, West Bengal (CNH/55/2013/Tech. II/19). The shade-dried cleaned leaves were cut into small pieces, pulverized into coarse powder, and extracted with ethanol in Soxhlet apparatus. Thereafter, the solvent was removed under reduced pressure, and the extract was dried (RHE). RHE was mixed in ethanol (1 mg/ml), filtered, and spotted on a precoated silica gel plates (Merck, $60F_{254}$, 20 cm × 20 cm) using CAMAG Linomat 5 applicator and processed in a solvent system (toluene: ethylacetate: formic acid = 4.5:3:0.2) for 30 min. The densitometric scanning was performed on CAMAG TLC Scanner 3 at absorbance 280 nm (D2 lamp) operated by multi level win CATS planar chromatography manager.[9]

In vitro antioxidant activity

The radical scavenging activity of RHE was determined using 1,1-diphenyl-2-picrylhydrazyl (DPPH).[1,13] 0.1 ml of RHE (10–1000 μg/ml) was mixed with 3.9 ml of 0.135 mM DPPH solution, allowed to stand in dark for 30 min, and observed at 517 nm. Finally, 50% inhibitory concentration (IC_{50}) of RHE was determined. Furthermore, phenolics and flavonoids present in RHE was determined.[17-18] For phenolics assay folin-ciocalteu reagent was used and flavonoids assay aluminium chloride reagent was used. Total phenolic content was expressed as gallic acid equivalent (GAE) in μg/ml of RHE and flavonoids as quercetin equivalent (QE) in μg/ml of RHE.

In vitro antidiabetic activity
α-amylase inhibitory action

The α-amylase inhibition assay of RHE was determined using the chromogenic 3,5-dinitrosalicylic acid (DNSA) method.[19] Assay mixture composed of 0.4 ml of 0.02 M sodium phosphate buffer (pH 6.9), 0.02 ml of 0.04 units of porcine pancreatic α-amylase solution, and 0.08 ml of RHE (10–1000 μg/ml). The tubes were pre-incubated at 37°C for 10 min, and 0.5 ml of 1% (v/v) starch solution was added to each of the tube and incubated for another 15 min at 37°C. The reaction was terminated with 1 ml 96 mM DNSA reagent and placed in boiling water bath for 5 min. The reaction mixture was then diluted with 2 ml of distilled water and read at 540 nm. The results were expressed as IC_{50} of RHE.

α-glucosidase inhibitory action

The serial dilutions of RHE (10-1000 μg/ml) in 0.5 ml of phosphate buffer (pH 6.8) was mixed with 0.05 ml of 10 mM para-nitrophenyl-α-D-glucopyranoside and incubated in 25°C for 10 min. After preincubation, 0.02 ml of α-glucosidase (0.5 mg/ml) was added and heated at 25°C for 5 min. Finally, 0.3 ml of 50 mM sodium hydroxide was added, and the absorbance was measured at 410 nm.[19] The results were expressed as IC_{50}.

Induction of diabetes in rats

Streptozotocin (STZ) was dissolved in ice-cold citrate buffer (0.1 M, pH 4.5) and injected intravenously at the dose of 50 mg/kg in rats (except normal control).[3] The diabetic state (fasting blood glucose >180 mg/dl) was confirmed 3 days after STZ injection. A total of 30 rats were studied, which were randomly grouped under normal control, diabetic control, diabetic rats with glibenclamide, and diabetic rats with RHE. The experimental design was as follows (*n* = 6 in each group): Group I: Normal control (without STZ); Group II: Diabetic control; Group III: Diabetic with glibenclamide (10 mg/kg, p.o);[20] Group IV: Diabetic with RHE (100 mg/kg, p.o); and Group V: Diabetic with RHE (200 mg/kg, p.o). The test doses were selected on the basis of pilot studies.

Oral glucose tolerant test

Oral glucose tolerant test (OGTT) was performed in STZ diabetic rats after 7 days of STZ injection.[21] Basal blood glucose of rats was monitored in overnight fasting (16 h) rats. Group I–II rats were treated with distilled water, while, Group III with glibenclamide and Group IV–V treated with RHE in different single doses. Thereafter, glucose solution (1 g/kg) was given orally to all rats, and blood glucose was monitored at 30, 60, 90, and 120 min interval after glucose administration. The area under the glucose curve (AUC_{OGTT}) was calculated using the following linear trapezoidal rule:[22]

$$AUC_{OGTT} = 0.25 \times \text{(fasting value)} + 0.5 \times \text{(30 min value)} + 0.75 \times \text{(60 min value)} + 0.5 \times \text{(120 min value)}.$$

Insulin sensitivity test

STZ diabetic rats after 14 days of STZ injection (fasting blood glucose >250 mg/dl) were divided into five groups (*n* = 6) and treated as before. Basal blood glucose was monitored and human insulin (Human Insulatard, Novo Nordisk) was injected (30 U/kg body weight) subcutaneously. Blood glucose levels were noted at 30, 60, 90, and 120 min after insulin injection and area under the glucose curve (AUC_{IST}) was determined.[22,23]

Glucose monitoring and blood biochemistry

In a separate regimen, thirty animals were divided into five groups (*n* = 6), and treatment was continued once/day for 4 weeks, as stated before.[3] At day 28, all animals were sacrificed under deep anesthesia, and blood was drawn. The biochemical estimations of glucose, cholesterol, high-density lipoprotein (HDL)-cholesterol, and triglycerides in serum were done (Span Diagnostics, India). Low-density

lipoprotein (LDL)-cholesterol, very low-density lipoprotein (VLDL)-cholesterol, atherogenic index, and coronary risk index were calculated using the following formulas:[24]

VLDL = Triglycerides ÷ 5

LDL = Total cholesterol − (HDL-cholesterol + VLDL-cholesterol)

Atherogenic index = LDL-cholesterol ÷ HDL-cholesterol

Coronary risk index = Total cholesterol ÷ HDL-cholesterol.

Statistical analysis

The data were expressed as mean ± standard error of the mean, and differences between groups were assessed statistically using t-test. $P < 0.05$ was taken as level of statistical significance in all tests. SPSS version 17 (IBM, Chicago, USA) software was used for statistical analysis.

Results

In vitro assay

RHE exhibited rich sources of phenolic acids and flavonoids. High performance thin layer chromatography fingerprint quantified quercetin as a major ingredient (1.68%) in RHE. It had strong radical scavenging power. In the *in vitro* assay, RHE showed α-amylase and α-glucosidase inhibitory actions [Table 1].

Oral glucose tolerant test

RHE showed significant ($P < 0.001$) dose dependant reduction in blood glucose concentration on STZ-induced hyperglycemic OGTT rats [Table 2]. Maximum reduction was noted at 2 h time point (−31.24% and −49.57%, respectively), similar to standard glibenclamide treated group (−48.04%). RHE had lower AUC_{OGTT} than STZ diabetic rats [Figure 1].

Insulin sensitivity test

Exogenous insulin sharply lowered blood glucose concentration in all animals [Table 3]. RHE had significant and dose-dependent potentiating action on insulin sensitivity
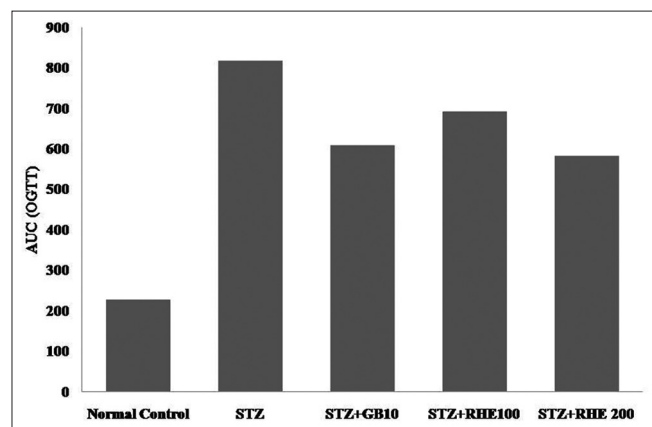
in diabetic rats. Maximum effect was observed at 1 h after exogenous insulin administration (−25.36% and −42.43%), better than glibenclamide-treated group (−23.57%). RHE had also lower AUC_{IST} than STZ diabetic rats [Figure 2].

Glucose monitoring and blood biochemistry

Administration of RHE continuously for 4 weeks resulted in a significant ($p < 0.01$) decrease of blood glucose level (−35.58% and −52.92%) in comparison to STZ-diabetic rat. Moreover, RHE lowered the triglycerides (−19.15% and − 31.74%), total cholesterol (−20.13% and −28.13%), LDL (-68.93% and -72.85%), VLDL (−19.18% and −31.68%), atherogenic index (−74.19% and −80%), and coronary risk index (−34.82% and − 47.85%), while enhanced HDL concentration (21.05% and 35.43%). However, glibenclamide did not show any significant lipid lowering action in STZ diabetic rats [Table 4].

Discussion

In South Asian countries including India, the leaves of *R. mucronata* are traditionally used in the treatment of diabetes.[2,3] Earlier studies, revealed that fresh leaves of *R. mucronata* juice contain quercetin (5,7,3',4'-tetrahydroxy

Table 1: *In vitro* assay, antioxidant and antidiabetic properties of ethanolic extract of *Rhizophora mucronata* leaves

In vitro assay	RHE
DPPH radical scavenging (IC_{50} μg/mL)	6.65±0.10
Phenolics (GAE μg/mL)	12.11±0.05
Flavonoids (QE μg/mL)	24.42±0.32
α-amylase inhibition (IC_{50} μg/mL)	41.10±0.35
α-glucosidase inhibition (IC_{50} μg/mL)	24.42±0.32

Values were mean±SEM when *n*=6 in each test. IC_{50}: 50% inhibitory concentration, GAE: Gallic acid equivalent, QE: Quercetin equivalent, Unit: μg/mL extract, RHE: Ethanolic extract of *Rhizophora mucronata* leaves, DPPH: 1,1-diphenyl-2-picrylhydrazyl, SEM: Standard error of mean

Figure 1: Area under the curve of oral glucose tolerance test in streptozotocin-diabetic rats. RHE: Ethanolic extract of *Rhizophora mucronata* leaves, GB: Glibenclamide, Unit: mg/dl, STZ: Streptozotocin
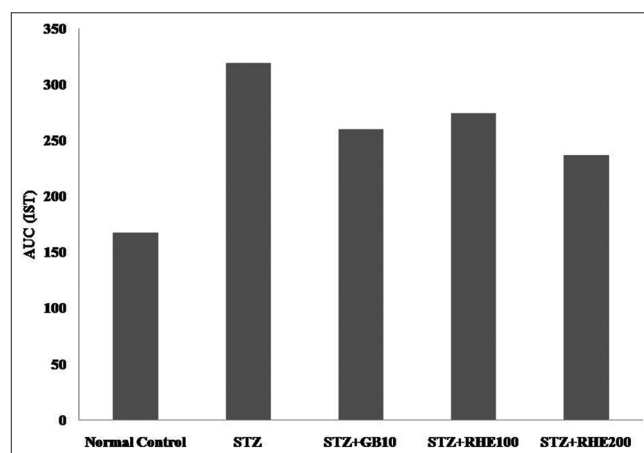

Figure 2: Area under the curve of insulin sensitivity test in streptozotocin-diabetic rats. RHE: Ethanolic extract of *Rhizophora mucronata* leaves, GB: Glibenclamide, Unit: mg/dl, STZ: Streptozotocin

Table 2: Oral glucose tolerance test in streptozotocin-induced hyperglycemic rats

Group	Dose	Fasting blood glucose (mg/100 ml of blood)				
		0 h	30 min	60 min	90 min	120 min
Normal control	2 mL/kg	66.33±1.62	99.16±2.62	168.8±4.15	145.5±4.55	71.16±2.13
STZ control	2 mL/kg	342.6±4.57[a,]*** (416.50)	387±3.73[a,]*** (290.27)	440.6±5.65[a,]*** (161.01)	423.3±4.15[a,]** (190.92)	412.5±5.99[a,]*** (479.67)
STZ + glibenclamide	10 mg/kg	339±3.88[b]	312±3.34[b,]*** (−19.37)	346.8±3.23[b,]*** (−21.28)	243±7.58[b,]*** (−42.59)	214.3±4.31[b,]*** (−48.04)
STZ + RHE	100 mg/kg	336±3.35[b]	347±4.46[b,]** (−10.33)	389.6±2.23[b,]** (−11.57)	314±3.53[b,]*** (−25.82)	283.6±3.11[b,]*** (−31.24)
	200 mg/kg	334.3±2.66[b]	301.8±4.34[b,]*** (−22.18)	325±6.85[b,]*** (−26.23)	237.1±6.03[b,]*** (−43.98)	208±5.31[b,]*** (−49.57)

Values were mean±SEM when n=6 in each group. Data in parenthesis indicate percentage change to same comparison, **$P<0.01$ and ***$P<0.001$. Statistical analysis were done using t-test. [a]Compared to normal control and [b]Compared to STZ control. RHE: Ethanolic extract of *Rhizophora mucronata* leaves, STZ: Streptozotocin, SEM: Standard error of mean

Table 3: Insulin sensitivity test in streptozotocin-induced hyperglycemic rats

Group	Dose	Fasting blood glucose (mg/100 ml of blood)				
		0 h	30 min	60 min	90 min	120 min
Normal control	2 mL/kg	91.5±2.70	70.5±2.06	109.5±1.83	65.8±1.40	54.5±1.56
STZ control	2 mL/kg	379.6±3.31[a,]*** (314.86)	180±3.43[a,]*** (155.32)	123±2.01[a,]*** (12.3)	90.6±2.82[a,]** (37.68)	83.1±4.12[a,]*** (52.47)
STZ + glibenclamide	10 mg/kg	374.6±3.49[b]	126.1±2.02[b,]*** (−29.94)	94±1.36[b,]*** (−23.57)	75.6±1.74[b,]** (−16.55)	64.8±1.57[b,]*** (−22.02)
STZ + RHE	100 mg/kg	377.3±3.79[b]	139.6±1.76[b,]*** (−22.44)	91.8±1.92[b,]*** (−25.36)	74.6±1.52[b,]*** (−17.66)	81.8±3.60[b] (−1.56)
	200 mg/kg	377.6±2.66[b]	114.1±3.34[b,]*** (−36.61)	70.8±1.77[b,]*** (−42.43)	69.5±2.14[b,]*** (−23.28)	63.6±1.33[b,]*** (−23.4)

Data in parenthesis indicated percentage change to same comparison, **$P<0.01$ and ***$P<0.001$. Values were mean±SEM when n=6 in each group. Statistical analysis was done using t-test. [a]Compared to normal control and [b]Compared to STZ control. STZ: Streptozotocin, SEM: Standard error of mean, RHE: Ethanolic extract of *Rhizophora mucronata* leaves

Table 4: Blood biochemistry in streptozotocin-induced hyperglycemic rats

Parameter	Normal control (2 mL/kg)	STZ control (2 mL/kg)	STZ + glibenclamide (10 mg/kg)	STZ + RHE (100 mg/kg)	STZ + RHE (200 mg/kg)
Glucose	70.16±2.10	475.8±5.38[a,]*** (578.16)	215.3±4.42[b,]*** (−54.74)	215.3±4.42[b,]*** (−54.74)	224±3.92[b,]*** (−52.92)
Triglycerides	67±2.29	172.3±2.85[a,]*** (157.16)	165.8±2.21[b] (−3.77)	139.3±1.47[b,]** (−19.15)	117.6±3.32[b,]*** (−31.74)
Total cholesterol	73.3±3.51	152.5±3.04[a,]*** (108.05)	137.3±1.56[b,]* (−9.96)	121.8±2.05[b,]*** (−20.13)	109.6±1.66[b,]*** (−28.13)
HDL-cholesterol	41.6±1.17	28.5±0.84[a,]*** (−31.49)	30.1±1.90[b] (5.6)	34.5±0.99[b,]*** (21.05)	38.6±0.88[b,]*** (35.43)
LDL-cholesterol	18.2±3.60	89.5±3.70[a,]*** (391.75)	74±2.18[b,]* (−17.3)	27.8±0.29[b,]*** (−68.93)	24.3±1.05[b,]*** (−72.85)
VLDL-cholesterol	13.4±0.45	34.4±0.57[a,]*** (156.71)	33.1±0.44[b] (−3.77)	27.8±0.29[b,]*** (−19.18)	23.5±0.66[b,]*** (−31.68)
AI	0.44±0.09	3.1±0.22[a,]*** (604.54)	2.3±0.25[b] (−25.8)	0.8±0.02[b,]*** (−74.19)	0.62±0.01[b,]*** (−80)
CRI	1.76±0.09	5.37±0.24[a,]*** (205.11)	4.6±0.29[b] (−14.33)	3.5±0.08[b,]*** (−34.82)	2.8±0.05[b,]*** (−47.85)

Values were mean±SEM when n=6 in each group, unit: mg/100 mL blood, Statistical analysis was done using t-test. [a]Compared to normal control and [b]Compared to STZ control. Data in parenthesis indicated percent change to same comparison. *$P<0.05$, **$P<0.01$ and ***$P<0.001$. STZ: Streptozotocin, SEM: Standard error of mean, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very-low-density lipoprotein, CRI: Coronary risk index, AI: Atherogenic index, RHE: Ethanolic extract of *Rhizophora mucronata* leaves. AI: Atherogenic index, CRI: Coronary risk index

flavonol).[9,13] This study substantiated that the ethanolic extract of *R. mucronata* leaves (RHE) are also rich in phenolics, flavonoids, and quercetin. Many works in the literature have shown the antioxidative, antidiabetic, and cardioprotective

activities of phenols and flavonoids. Quercetin is a potent antioxidant that inhibits pro-oxidant enzymes and decreases the oxidative damage in diabetes by improving the antioxidant response.[19] RHE showed promising antioxidant properties by inhibiting DPPH radical scavenging that may be due to high amount of quercetin on it.

Pancreatic α-amylase and intestinal α-glucosidase are the key enzymes in carbohydrate digestion from the initial step in hydrolysis of starch to the final product glucose. Therefore, activities of these enzymes correlates to an increase in postprandial glucose levels.[19] Hence, one therapeutic approach to treat diabetes is to retard the absorption of glucose through the inhibition of enzymes, such as α-amylase and α-glucosidase, in the digestive organs.[25,26] In this study, RHE showed inhibitory actions on both the enzymes.

Impaired glucose tolerance is a well-known clinical characteristic of diabetes mellitus. In most cases, impaired glucose tolerance represents a transient stage during the development of noninsulin-dependent diabetes mellitus.[19] RHE showed a significant dose-dependent reduction in blood glucose concentration similar to glibenclamide as evident by AUC of RHE in comparison to STZ diabetic rats. From this study, it may be inferred that RHE rats were more tolerant to oral glucose challenges, demonstrating antihyperglycemic activity of the plant.

Metabolic syndrome comprises several physiological disorders and those changes also affect the glucose transporters. The stimulation of insulin causes the translocation of glucose transporter 4 (GLUT-4) toward the plasma membrane thereby increasing glucose uptake, participating significantly in the control of glucose homeostasis.[27] Insulin resistance refers to the insensitivity of tissues to insulin action, i.e., the weaker glucose utilization of body after insulin action that results in hyperglycemia.[28] In this study, exogenous insulin sharply lowered blood glucose, whereas RHE-facilitated insulin sensitivity in diabetic rats, thereby supporting its insulin-mimetic action. Moreover, the high blood glucose level of STZ-induced type 1 diabetic rats indicates the development of insulin resistance. Regular administration of RHE for 4 weeks resulted in a significant attenuation of blood glucose and urinary albumin. According to the WHO, urinary excretion of albumin is a condition of metabolic syndrome.[29]

Insulin has important role in lipid metabolism and its deficiency, therefore, leads to hypercholesterolemia.[16] RHE treatment for 4 weeks has not only lowered cholesterol, triglycerides, and LDL concentration, but it also enhanced HDL-cholesterol which is known to play an important role in the transport of cholesterol from peripheral cells to liver and is considered to be a cardioprotective lipid. Glibenclamide, a known hypoglycemic agent, did not show any significant lipid lowering action in STZ diabetic rats. It is known that plant flavonoids, mainly quercetin help to enhance the number of pancreatic islets, protects β-cells from damage and facilitates in translocation of GLUT-4.[30] Thus, the presence of quercetin

in RHE may play a contributing factor to the observed antidiabetic activity through the above mentioned pathways.

Conclusion

Quercetin rich *R. mucronata* leaves ethanolic extract (RHE) lowers blood glucose and controls lipid impairment either through facilitating insulin action or by inhibiting α-amylo glucosidase pathways and may be useful in conventional antidiabetic therapy in the near future.

Conflicts of interest

There are no conflicts of interest.

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हिन्दी सारांश

भारतीय सुंदरवन मैंग्रोव पत्र का मधुमेह संबन्धित जटिलता में इन (राइजोफोरा म्यूक्रोनाटा) वीवो एवं इन विट्रो अध्ययन

अंजान अधिकारी, मोमिता राय, अनूप कुमार दास, तपस कुमार सूर

राइजोफोरा म्यूक्रोनाटा भारतीय सुंदरवन में पाया जाने वाला आम्रकुंज है (मैंग्रोव), जो पारंपरिक रूप से मधुमेह एवं अन्य स्वास्थ्य संबंधी उपचारों के लिए उपयोग किया जाता है। प्रस्तुत शोध कार्य में मधुमेह के दौरान ग्लूकोज के चयापचय प्रक्रिया हीनता में इस पौधे की पत्तियों द्वारा इनका इन वीवो एवं इन विट्रो प्रयोगिक क्रिया के माध्यम से अध्ययन किया गया। जिसके लिए २८ दिनों कि अवधि का निर्धारण किया गया। इस अध्ययन में मैंग्रोव (राइजोफोरा म्यूक्रोनाटा) की पत्तियों का इथैनोलिक सार (आरएचई) का प्रयोग डीपीपीएच रेडिकल स्क्वेजिंग द्वारा तथा इन विट्रो मधुमेह प्रतिरोधी क्रिया का परीक्षण अल्फा एमाइलेज व अल्फा ग्लूकोसिडेज प्रतिक्रिया द्वारा किया गया। इसके अतिरिक्त ओरल ग्लूकोज टॉलरेन्स परीक्षण तथा इंसुलिन सेन्सेटीविटी परीक्षण भी किया गया एवं इसका नियंत्रण (आरएचई) तथा ग्लिबेनक्लेमाइड द्वारा स्ट्रेप्टोज़ोटोसिन प्रेरित मधुमेह ग्रसित चूहों पर देखा गया। परीक्षण से ज्ञात हुआ कि (आरएचई) ने मात्रा आधारित अल्फा एमाइलेज व अल्फा ग्लूकोसिडेज का अवरोध किया तथा ग्लायसिमिक इंडेक्स का नियंत्रण किया परिणाम स्वरूप (आरएचई) ने मधुमेह संबन्धित उपद्रवों जो कि लिपिड प्रोफ़ाइल, ऐथ्रोजिनिक इंडेक्स तथा कोरोनरी इंडेक्स द्वारा जाँची गयी को कम किया ऐसा परिणाम प्राप्त हुआ। इस प्रकार अध्ययन द्वारा यह निष्कर्ष निकाला गया कि भारतीय सुंदरवन मैंग्रोव (राइजोफोरा म्यूक्रोनाटा) पत्र मधुमेह संबन्धित जटिलताओं को दूर करने में सक्षम है।