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EFFECT OF NON-POLAR FRACTION OF CINNAMOMUM TAMALA LEAVES ON INTESTINAL α-GLYCOSIDASE ENZYME IN NORMAL AND DIABETIC RATS

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ABSTRACT

Cinnamomum tamala (CT) is a member of the Lauraceae family and its leaves were used as food spices for flavor. The leaves are also used as an antidiabetic agent, and many more in the Indian Medicine System. This potential of CT leaves towards postprandial hyperglycemia is not known, so it is planned here for the same. Here, we have prepared hexane fraction of CT leaves (CTH, most active) and shown their effect on oral, glucose and sucrose tolerance and α- glycosidase activity for postprandial hyperglycemia. CTH @ 400 and 800 mg/kg body weight were given to normal and diabetic rats and blood glucose was monitored for 2 hrs. For the study of the mechanism, their effect was measured on alpha glycosidase activity and its effect on postprandial hyperglycemia. Results suggested that CTH reduced postprandial hyperglycemia by inhibiting Alpha glycosidase activity. Acarbose which is a well-known alpha glycosidase inhibitor, is used as a reference drug for postprandial hyperglycemia.

KEYWORDS: Cinnamomum tamala, anti-diabetes, α-glycosidase-inhibitor, Acarbose.

1. INTRODUCTION

Diabetes is characterized by increased blood sugar which can be by different means such as failure of insulin secretion from beta cells of the pancreas, increased insulin resistance, increased intestinal glucose absorption, etc. Glucose levels after a meal is related to the amount and digestion rate of consumed carbohydrates, one therapeutic approach for managing postprandial hyperglycemia is to retard digestion and subsequent absorption of dietary complex carbohydrates.[1] Postprandial hyperglycemia (High oral glucose tolerance test-OGTT) has been documented as one of the major risk factors for various diabetic complications including cardiovascular disease.[3] High LDL oxidation and rapid blood coagulation. Diabetes mellitus (DM) and associated complications are the major cause of disability. It induces the non-enzyme glycation of various proteins. The accumulated load of AGEs results in various cellular pathologies, resulting in diabetic complications involving kidney, neuron, or endothelial cell damage. This is primarily related to the reduction of the antioxidant defences in diabetics.[4] Currently, it covers about 4% of the world's population, but it is expected to significantly rise with the projected growth rate of 366 million patients by the year by 2030.[5-7]

In a fasting state, plasma glucose concentration is maintained almost stable, and dietary glucose delivery is required for maintenance of normal plasma glucose concentrations. After a meal, plasma glucose concentrations increase rapidly as glucose absorption increases to more than twice the rate of endogenous glucose production[8] which is known as the postprandial phase.

In a clinical setup, the oral glucose tolerance test (OGTT) has been mostly used to predict this situation.[9] This abnormal OGTT may be managed at 2 levels, either by prevention of longer hyperglycemic state in the blood or by reducing the free radical load. The altered carbohydrate metabolism at the intestinal level, which is mediated by intestinal α-glucosidase and pancreatic amylases are significant components.[10] The available anti-diabetic drugs are broadly divided into different groups, such as sulfonylurea group, responsible for enhanced insulin release from beta cells, biguanides group responsible for increased peripheral sensitivity of insulin receptors[11,12] and reduced hepatic glucose production[13] and inhibitors of intestinal enzymes, responsible for delayed absorption of ingested glucose. This group of drugs includes α- glycosidase inhibitors e.g. Acarbose, Miglitol.[14] Several herbal food supplements have been reported to be effective; however; these drugs have several adverse effects including gastrointestinal malfunctioning. Therefore, the search for novel drugs of plant origin is in demand of time. Some plant products have already been associated with this property of α-glycosidase inhibition such as Momordica charantia[15], Nigella sativa[16] etc. Here, we have observed the effect of the non-polar fraction (CTH) of Leaves of Cinnamomum tamala on postprandial hyperglycemia in normal and diabetic rats and α-glycosidase activity in the intestine homogenate model. CT leaves are an important constituent of Indian species. Earlier, we have reported its anti-diabetic and antioxidant properties[17-22] and immune-modulating potential.

2. MATERIALS AND METHODS

Alloxan was purchased from Central Drug House (CDH) and, a glucose kit (GOD/POD) was arranged from Hind Diagnostic, Varanasi, Acarbose (Glucoby), and other chemicals were of analytical grade.

2.1. Preparation of CTH: The coarse powder of CT leaves was extracted with hexane for 30 hrs in a continuous Soxhlet extractor. It was distilled under reduced pressure and desiccated till a constant weight was attained. The drug was dissolved in 20% Tween 20 in water for biological system and in DMSO for chemical system.

2.2. Preparation of Diabetic rats: The experimental protocols were approved by the Institute animal Ethics Committee. The inbreed albino rats of Charls Foster (CF) strain, (180±20 g body weight) were purchased from the central animal house of the Institute of Medical Sciences. They were acclimatized for 6 days in our laboratory conditions with a 12-h light/12-h dark cycle, and free access to water and food. Alloxan monohydrate solution (2 % in sterile normal saline) was injected through an intra-peritoneal route to each overnight fasted rat at a dose of 100 mg/kg BW. After one week, blood sugar was determined. Rats having blood sugar higher than 200 mg/dl, were considered diabetic and used for the experiments as per requirements.

2.3. Effect of CTH on Oral Glucose Tolerance Test (OGTT) and Oral Sucrose Tolerance Test (OSTT) in rats: The diabetic animals were divided into different groups, with 6 animals in each. Group1- experimental diabetic control- drug vector (5 ml/kg). Group2, 3 and 4 were treated with CTH 400 and 800 mg/Kg BW, and acarbose (5 mg/kg) respectively. After, 20 min of drug treatment, glucose (4 g/kg BW)/ sucrose (4 g/kg BW) solutions were orally given to all rats. At varying time intervals (30, 60 and 120 minutes), blood glucose was measured by GOD/POD method.[23]

2.4. Effect of CTH on intestinal α-glycosidase activity

For the preparation of the enzyme, a normal rat was sacrificed, and small intestine was dissected out. Its small pieces were taken in pre-cooled PBS, and the faecal material was gently removed. The clean tissue was dried on blotting paper, weighed, and homogenized in glass Teflon homogenizer. It was centrifuged at 12,000 g for 30 minutes, and clear supernatant (20%-w/v) was used as an enzyme source. To assess, 20µl of homogenate of small intestine (enzyme source) was mixed with 40 µl of test samples (CTH/Acarbose), and after 10 min incubation at 37°C, it was mixed with 140 µl substrate (sucrose 37 mM) and re-incubated for 60 min. The tubes were suspended in a boiling water bath for 10 min to stop the reaction and then centrifuged. The supernatant was used for glucose determination by the GOD/POD method and % inhibition was calculated with respect to the control (without the test sample).

3. STATISTICAL ANALYSIS

Results were articulated as mean ± SD, and all statistical comparisons were made using a one- way ANOVA test followed by post hoc analysis with the Dunnett test and p-values less than or equal to 0.05 were considered significant.

4. RESULTS

4.1. Effect of CTH on oral glucose and sucrose tolerance test

In the experimental diabetic control group, the glucose administration resulted in a 71% increase in blood sugar, after 30 min of oral glucose administration. However, in the treated group, this rise was 62% at 400 mg and 47% at 800 mg/kg body weight dose (Table 1). The acarbose (5mg/KG BW) treatment resulted to only 42% increase. Basal is the blood sugar measured just prior to glucose/sucrose load.

Table 1: Effect of hexane fraction of CT leaves (CTH) on glucose on OGTT in normal and diabetic rat.

Groups	Blood glucose in mg/dl (Mean±SD) and %Change			
	Basal	30 min	60 min	120 min
Normal control	93.0±8.50	122.5±7.21* (31.7±1.14 %)	118.6±14.6* (27.5±1.10 %)	104.4±5.9* (11.8±1.12 %)
CTH 400 mg/kg	84.24±5.20	108.66±6.92* (29±0.96 %)	102.0±5.80* (21±0.64 %)	96.56±4.86 (12.2±1.04%)
CTH 800 mg/kg	91.40±4.58	110.54±4.60* (21±1.10 %)	106.36±6.64* (16.36±1.06%)	96.20±5.74 (5.4±1.20 %)
Acarbose 5 mg/kg	94.20±4.22	112.14±2.32* (19±1.18 %)	110.64±3.58* (17.5±0.98%)	105.28±3.88 (11.8±0.86 %)
Diabetic Control	221.25±5.60	379.60±7.92** (71.4±0.60 %)	375.6±9.02** (69.6±1.22. %)	310.8±5.40** (40.2±1.24%)
CTH 400 mg/kg	245.22±7.28	398.10±6.84** (62.4±1.10 %)	372.80±6.28** (51.1±0.98 %)	377.21±5.45** (53.8±0.90%)
CTH 800 mg/kg	256.56±6.76	379.26±5.32** (47.82±1.00)	355.76±6.08** (38.1±1.06 %)	342.44±6.52** (33.6±1.18%)
Acarbose 5 mg/kg	261.00±4.52	372.20±2.02** (42.6±1.20 %)	360.44±3.65** (38±0.98 %)	345.30±2.82** (32.3±1.10 %)

*Values are significant p<0.05, ** Values are highly significant p<0.01

In sucrose treated diabetic rats, there was only a 46% rise in blood glucose in experimental diabetic control group, which was significantly lower than the glucose load. With CTH pre-treatment, this rise was further reduced to 28.8% at 400 mg and 19% at 800mg/Kg body weight (Table 2). In this condition, acarbose showed only a 13% rise. Thus, in both conditions, significant inhibition in the rise of blood glucose was noted.

Table 2: Effect of hexane fraction of CT leaves (CTH) on sucrose tolerance in normal and diabetic rats.

Groups (N=6)	Blood Glucose in mg/dl (% Change)			
	Basal	30 min	60 min	120 min
Normal Control	79.3±5.32	111.9±4.86** (41.4±1.32)	106.3±6.18 (34.1±1.46)	86.9±3.48 (10.1±1.10)
CTH 400	110.8±5.58	137±2.82** (24.5±1.21)	128.6±4.42 (16.3±1.18)	118.2±3.72 (7.2±1.08)
CTH 800	106.9±3.82	125.5±4.64** (17.9±1.34)	117.6±3.94 (9.3±1.20)	111.5±5.36 (3.7±1.08)
Acarbose 5	89.2±3.86	100±2.65* (12.1±1.10)	95.4±2.20 (6.7±1.24)	92.5±3.46 (3.3±1.36)
Diabetic Control	290±4.86	424.6±6.12** (46.2±1.44)	418.1±4.50 (44.1±1.22)	367.1±2.64 (26.5±1.22)
CTH 400	312.0±5.98	402.5±5.30** (28.8±1.26)	392.8±5.64 (25.6±1.34)	355.6±6.62 (13.7±1.20)
CTH 800	306.25±4.40	365.9±3.92** (19.2±1.30)	357.8±4.56 (16.6±1.28)	315.6±5.92 (2.9±0.98)
Acarbose 5	302.9±6.82	342.5±6.60** (12.8±1.10)	330.3±3.80 (9.2±1.20)	319.0±4.48 (5.6±1.12)

*Values are significant p<0.05, ** Values are highly significant p<0.01

4.2. Effect of CTH on isolated α-glycosidase enzyme preparation

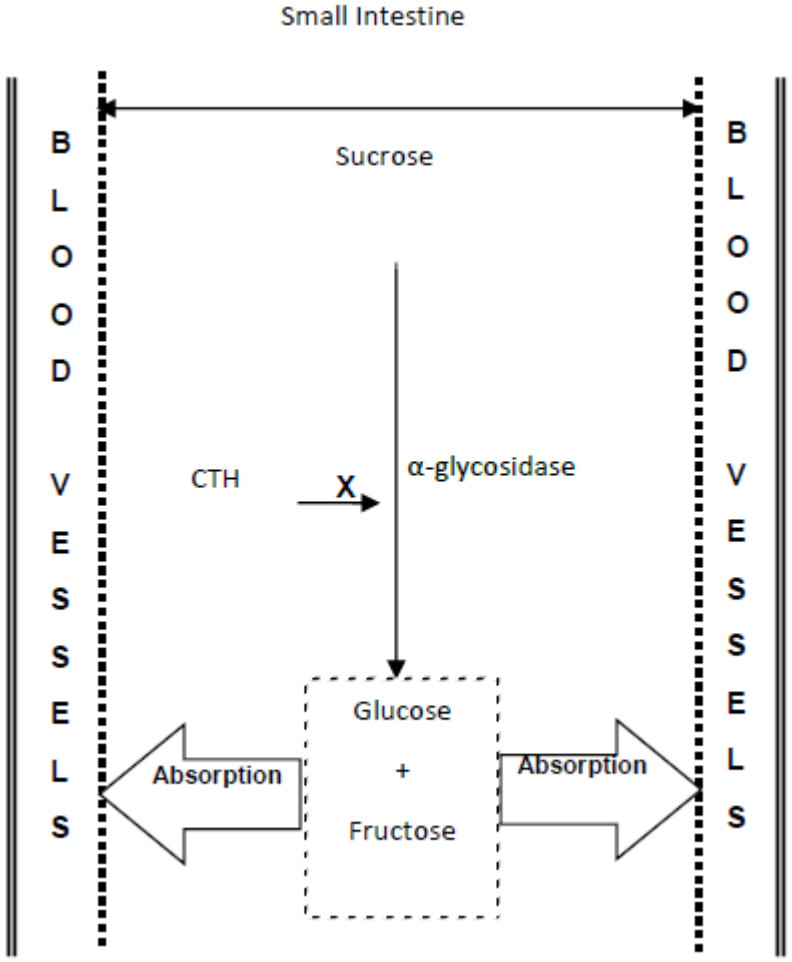
The effect of CTH and acarbose was tested on isolated intestinal enzyme preparation, to assess the specific inhibition, if any. The IC_{50} value of CTH was 841.114 µg/ml which was approximately, double to the IC_{50} value for acarbose (374.676 µg/ml) a known α-glycosidase inhibitor (Table 3). Thus, CTH and acarbose action are the same i.e. CTH inhibiting the activity of the α-glycosidase enzyme.

Table 3: Effect of hexane fraction of CT leaves (CTH) on α-glycosidase enzyme in small intestine homogenate in normal rat.

CTH (µg/ml)	% Inhibition	Acarbose (µg/ml)	% Inhibition
50	1.15±1.7	50.0	43.2±1.69**
100	5.55±1.7*	100	46.2±1.21**
200	9.00±1.66*	200	52.3±1.28**
400	22.10±1.98**	400	55.5±1.20**
800	40.20±1.42**	800	63.6±1.07**
1000	52.71±1.58**	1000	64.5±1.20**
IC_{50}= 841.114 µg/ml		IC_{50}= 374.676 µg/ml	

*Values are significant p<0.05, ** Values are highly significant p<0.01 This result explains the mechanism of the reduction of blood glucose after meals.

Pictorial Presentation for mechanism of action of CTH towards reduction of α-Glycosidase enzyme activity



5. DISCUSSION

Carbohydrate component of food is digested in the intestine through several hydrolytic enzymes, including α-glycosidase. It hydrolyses the 1, 4 alpha glycosidic linkage of disaccharides. Its activity is normally higher in diabetics, and it is associated with high postprandial hyperglycaemia.[24] Several allopathic drugs are available for inhibition of α- glucosidase, but they are associated with adverse effects on GI functioning. Some of them exclusively inhibit this enzyme and some also act as an insulin secretagogue. Voglibose is an inhibitor of carbohydrate absorption and reduces the postprandial glucose level without stimulating insulin secretion, whereas nateglinide improves PPG by insulin secretion.[18] In this process, food supplements also play an important role, which includes herbal medicine. Most of the spices have a definite role in the digestion and metabolism of food, thus indirectly affecting the blood glucose level. Earlier, we have reported the antioxidant and hypoglycaemic property of CT extract.[22]

Here, we have investigated its effect on OGTT, OSTT, and intestinal enzyme α glucosidase, which is responsible for the acute rise in blood glucose after meals. Its significant reducing effect on OGTT and OSTT indicates its hypoglycaemic effect. This could be

because of its direct effect on beta cells for more release of insulin and/or due to improved peripheral sensitivity of the insulin receptors, resulting to the high turnover of the existing insulin and/or due to inhibition of intestinal α-glucosidase activity, resulting to lower rate of digestion and absorption of ingested carbohydrates. The last factor appears to be more relevant because the maximum change was noted 30 min post glucose/sucrose treatment. This hypothesis was further confirmed by α -glucosidase activity in intestinal homogenate. The result was comparable with acarbose, a standard α -glucosidase inhibitor, and found that CTH significantly inhibited the activity of α - glucosidase in concentration dependent manner. This response was similar to acarbose. There is a major difference in blood glucose in OGTT and OSTT in alloxan-induced diabetic rats, and the difference is due to glucose and sucrose. Glucose absorbs in the intestine without any chemical modification but in sucrose, it first hydrolyses and then gets absorbed in intestine. Thus, enzyme involvement is only in sucrose not in glucose. Thus, it supports the action of CTH on α – glucosidase enzyme.

CONCLUSION

The CTH inhibits the rise in blood glucose after a meal (postprandial) by reducing the activity of the alpha glycosidase enzyme found in the small intestine. The action was found to be the same as acarbose which is a well-known alpha glycosidase inhibitor.

Abbreviations

CT: Cinnamomum tamala
CTH: Hexane fraction of Cinnamomum tamala leaves
OGTT: Oral Glucose Tolerance Test
OSTT: Oral Sucrose Tolerance Test
DM: Diabetic Mellitus
AGEs: Advanced glycation end products
GOD/POD: Glucose oxidase-peroxidase
DMSO: Dimethyl sulfoxide
IC: Inhibitory Concentration
GI: Gastrointestinal

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