Research Article

In vitro study on the antidiabetic effect on jasmine oil

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ABSTRACT

Aim: The aim of the study is to determine the *in vitro* antidiabetic activity of jasmine oil. **Introduction:** Jasmine is a genus of shrubs in *Oleaceae* family. Jasmine oil is used as an essential oil, and it is reported to have antimicrobial, antidepressant, and stimulatory properties. Diabetes mellitus (diabetes) is a group of metabolic disorders in which there is an elevated blood sugar level over a prolonged period. **Materials and Methods:** Jasmine oil was purchased from the market commercially and its *in vitro* antidiabetic activity was studied using inhibition of alpha-amylase activity and glucose uptake assay. **Results and Discussion:** Jasmine oil inhibited the activity of alpha-amylase in a dose-dependent manner. It also showed enhanced glucose uptake in rat L6 myogenic cells. These two activities will cause a decrease in the blood glucose level, which is essential in the treatment of diabetes. **Conclusion:** Jasmine oil showed *in vitro* antidiabetic activity.

KEY WORDS: Alpha-amylase, Antidiabetic, Glucose uptake assay, Jasmine oil

INTRODUCTION

Diabetes mellitus, commonly referred to as diabetes, is a group of metabolic disorder in which there is a high blood sugar level over a prolonged period.[1,2] It is considered as one of the most common diseases and it rises progressively. It is estimated that 366 million had diabetes mellitus in 2011, it can reach to 552 million in 2030.[3] Type 1 diabetes mellitus is caused by immune-mediated failure to produce insulin due to the loss of beta cells.[4] Type 2 diabetes mellitus is described as one of the most common diseases. In Type 2 there is a relative insulin deficiency, hyperglycemia, and insulin resistance.[5] Type 2 diabetes mellitus results from the interaction between genetic, behavioral, and environmental risk factors.[6]

Insulin is the main remedy for Type 1 diabetes mellitus.[5] The treatment for diabetes is to maintain a normal level of blood glucose. The available oral medications for the treatment of Type 2 diabetes as well as a combination of drugs are alpha-glucosidases inhibitors, SLCT -2 Inhibitors, biguanides, etc. Another choice is Metformin, which is an oral antidiabetic drug in biguanide class. It is a first-line drug for the treatment of Type 2 diabetes in overweight conditions.[7]

Herbs are available very easily and it has few side effects, cure many diseases such as diabetes mellitus in ancient medicine.[8] Traditional medicine is used by 60% of India's population. They are not only used in rural areas in developing countries but also in developed countries.

Jasmine is the large genus of shrubs in olive family (*Oleaceae*), and it contains nearly 200 species. It is popularly used as an ornamental plant and it is cultivated for its fragment flowers in a tropical and subtropical region. The flowers are white, fragment, and solitary. Jasmine oil is an essential oil. Essential oils are highly concentrated essence of aromatic plants and chemically derived from terpens and oxygenated compounds.[8,9] Jasmine oil has beneficial in the treatment of severe depression and soothes the nerves, producing a feeling of confidence, optimism, and euphoria, while revitalizing and restoring energy and improving memory.[10] The main components of jasmine oil are benzyl acetate, benzyl propionate, and beta lonaool. The properties of volatile oils are aromatic, antimicrobial, antidepressant, stimulatory, etc. Hence, our aim is to conduct an *in vitro* antidiabetic study on jasmine oil.

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MATERIALS AND METHODS

Jasmine oil was purchased from the market commercially and used for this study.

Alpha-Amylase Inhibition Assay

The jasmine oil with three different concentrations (10–30 µl) was used for the study. A total of 500 µl of oil and 500 µl of 0.02M sodium phosphate buffer (pH 6.9 with 0.006M sodium chloride) containing α-amylase solution (0.5 mg/ml) were incubated for 10 min at 25°C. After pre-incubation, 500 µl of 1% starch solution in 0.02M sodium phosphate buffer (pH 6.9 with 0.006M sodium chloride) was added to each tube at 5 s intervals. This reaction mixture was then incubated for 10 min at 25°C. 1 ml of DNSA color reagent was added to stop the reaction. These test tubes were then incubated in a boiling water bath for 5 min and cooled to room temperature. Finally, this reaction mixture was again diluted by adding 10 ml distilled water following which absorbance was measured at 540 nm.

Glucose Uptake Assay

L6 rat myogenic cells

L6 rat myogenic cells were cultured in hypotonic buffer (20 mM Tris-HCl, pH 8, 1 mM EDTA, 0.2 mM EGTA, 50 mM NaF, 0.7 µg/ml pepstatin, 10 mM sodium orthovanadate, and 50 mM benzamidine, 0.5 µg/ml leupeptin, 4 µg/ml aprotinin, and 2 mM phenylmethylsulphonyl fluoride). The cell suspension was centrifuged at 20,000 × g for 15 min and homogenate obtained. The supernatant was collected as the soluble fraction for further experiments.

Preparation of test solutions

Different concentrations of jasmine oil (10, 20, 30 and 40 µl) were prepared using the medium.

Assay procedure

L6 rat myogenic cells were seeded into 96-well plate with six wells left as blank wells and let growing to confluence; then cells were fully differentiated in DMEM with 2% FBS for 5 days. Before tests, the medium was replaced by RPMI1640 (2 g/L glucose) supplemented with 0.2% BSA. The medium was removed after 2 h, and the same medium containing Jasmine oil (10-40 µl), metformin (0.01 mM) the standard, and DMSO in absence or presence of insulin (1 µmol/L) was added to all wells including the blank. The glucose in the medium was determined by the glucose-oxidase method after 48 h treatment. The amount of glucose uptake by muscle cells was calculated by using the following formula:

Glucose uptake = Glucose concentration of blank wells – Glucose concentration of cell plated wells

Table 1: Percentage of inhibition of αlpha-amylase

Treatment	Concentration (µl)	Absorbance at 540 nm	% of Inhibition
Control	0	0.175±0.32	0
Jasmine oil	10	0.023±0.15*	13.1
	20	0.035±0.05*	20.9
	30	0.051±0.23*	29.1
	40	0.084±0.15*	48.0
Metformin	0.1	0.148±0.19*	84.5

Values are mean±SD expressed as (n=3); *P<0.001 as compared with vehicle control

Statistical Analysis

Results were expressed as mean ± SD. Statistical significance was determined by one-way analysis of variance and *post hoc* least significant difference test. $P < 0.05$ was considered significant.

RESULTS AND DISCUSSION

The high postprandial blood glucose in diabetes results in microvascular complications including retinopathy, nephropathy, neuropathy, and macrovascular complications refer to increased atherosclerosis-related events such as myocardial infarction and stroke.[11,12] One of the therapeutic approaches for controlling postprandial hyperglycemia in a diabetic patient is to prevent or decreasing absorption of carbohydrate after food intake. Complex starches, oligosaccharides, and disaccharides must be broken down into monosaccharides by α-amylase and α-glucosidases before they are absorbed in the duodenum and upper jejunum.[13] Recent advances in understanding the activity of intestinal enzymes helped in the development of newer pharmacological agents.[14]

In our study, we observed that the oil showed inhibition in the activity of α-amylase in a dose-dependent manner [Table 1]. The activity was compared with the standard drug metformin. As compared to metformin, the oil showed mild hypoglycemic activity. Inhibition of this enzyme slows the absorption of carbohydrates from the gastrointestinal tract and decreases the rate of rise of postprandial glucose.[15] This delay digestion and breakdown of starch may have beneficial effects on insulin resistance and glycemic index control in people with diabetes.[16]

Peripheral insulin resistance and impaired insulin secretion from pancreatic β-cells are two important features of type 2 diabetes mellitus. The occurrence of cardiovascular diseases in type 2 diabetic patients mainly due to insulin resistance mediated hyperglycemia and dyslipidemia.[17] The drugs such as metformin and pioglitazone causes amelioration in insulin resistance and control the hyperglycemia and abnormal lipid metabolism. But, this class of drugs has adverse effects such as lactic acidosis, gastrointestinal disturbance, liver toxicity, and cardiovascular risk.[18]

Table 2: Glucose uptake in L6 rat muscle cells

Treatment	Concentration (µl)	Glucose consumption (mg/100ml)	
		Absence of Insulin	Presence of Insulin
Control	0	1.78 ± 0.16	5.18 ± 0.23
Jasmine oil	10	1.98 ± 0.21	5.23 ± 0.30
	20	1.81 ± 0.12	5.25 ± 0.28
	30	1.70 ± 0.19	5.36 ± 0.21*
	40	1.56 ± 0.14*	5.41 ± 0.16*
Metformin	0.1 µM	4.10 ± 0.06*	6.70 ± 0.31*

Values are mean±SD expressed as (n=3); *P<0.05 as compared with vehicle control

Thus, drugs which improve the sensitivity of insulin without any adverse effects were reported to be useful for the long-term treatment in type 2 diabetes. The insulin sensitivity was studied by assessing the glucose uptake in rat L6 myogenic cells and the effect was compared with the standard drug metformin. The results showed that [Table 2] jasmine oil exhibited increase in the glucose uptake in muscle cells, revealing its insulin sensitivity. But glucose uptake of jasmine oil in muscle cells in the presence and absence of insulin was not as strong as that of metformin. However, the oil possessed enhanced glucose uptake in the presence of insulin, which might be useful in reversing the insulin resistance in diabetes mellitus.

CONCLUSION

The present study establishes the *in vitro* antidiabetic effect of jasmine oil.

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