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Hypoglycaemic activity of *Syzigium cumini* seeds: effect on lipid peroxidation in alloxan diabetic rats

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Abstract

Syzigium cumini, commonly known as 'jamun', is widely used in Indian folk medicine for the treatment of diabetes mellitus. Oral administration of 2.5 and 5.0 g/kg body weight of the aqueous extract of the seed for 6 weeks resulted in a significant reduction in blood glucose and an increase in total haemoglobin, but in the case of 7.5 g/kg body weight the effect was not significant. It also prevents decrease in body weight. The aqueous extract also resulted in decreased free radical formation in tissues studied. Thus the study shows that Jamun seed extract (JSEt) has hypoglycaemic action. The decrease in thiobarbituric acid reactive substances (TBARS) and increase in reduced glutathione (GSH), superoxide dismutase (SOD) and catalase (CAT) clearly show the antioxidant property of the JSEt. The effect of JSEt was most prominently seen in the case of animals given 5.0 g/kg body weight. JSEt was more effective than glibenclamide.

Keywords: *Syzigium cumini*; Diabetes; Extract; Glucose; Hypoglycaemic

1. Introduction

Diabetes mellitus was known to ancient Indian physicians as 'madumeha'. Many herbal products including several metals and minerals have been described for the care of diabetes mellitus in ancient literature (Nadkarni, 1992). Ayurveda is an ancient Indian form of medicine, which deals with plants and plant extracts. This indigenous form of medicine uses the active ingredients present in plants for treating diseases (Lewis and Elvin-Lewis, 1977). Plant drugs are frequently considered to be less toxic and more free from side effects than synthetic ones (Momin, 1987). Many herbs have been shown to have hypoglycaemic action in animals and humans (Twaij and Al-Badr, 1988; Gupta, 1994).

The jamun tree which is native to India, thrives easily in tropical climates and is found in many

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parts of our subcontinent. It is also found in South East Asia and Eastern Africa. The jamun is a large evergreen tree which grows widely in the Indogangetic plains and also in the Cauvery delta of Tamil Nadu (Indira and Mohan Ram, 1992). The jamun belongs to the family Myrtaceae and is called *Syzigium cumini* (Samba-Murthy and Subrahmanyam, 1989). Jamun seeds have been used by natives in the treatment of diabetes (Chopra et al., 1958).

A literature survey showed that a decoction of the dry leaves of the *S. cumini* possesses a hypoglycaemic effect (Coimbra et al., 1992). Mahapatra et al. (1985) did some preliminary studies on the glycaemic effect of *S. cumini* seeds and found that they produce hypoglycaemia. There is a report showing that oral administration of dried alcoholic extract of the seed has a hypoglycaemic effect and also reduces glycosuria (Indira and Mohan Ram, 1992). Thus the available reports show that very little work seems to have been done with respect to Jamun seeds other than its hypoglycaemic effects. In the present communication its role on glucose and lipid peroxide metabolism in alloxan diabetic rats is studied.

2. Materials and methods

2.1. Plant material

S. cumini (jamun) seeds were collected fresh from Nagercoil, Kanayakumari District, Tamil Nadu, India and dried. The plant was identified and authenticated at the Herbarium of Botany Directorate in Annamalai University. A voucher specimen (number 367) was deposited in the Botany Department of Annamalai University. The dried seed was ground by an electrical mill (mesh number 50) and the powdered seeds were kept separately in airtight containers in a deep freeze until the time of use.

2.2. Preparation of the aqueous extract

A suspension of 100 g of seeds in 200 ml of distilled water was stirred magnetically overnight (12 h) at room temperature. This was repeated three consecutive times. The residue was removed by filtration and the extract evaporated to dryness at a lower temperature ($< 40°C$) under reduced pressure in a rotary evaporator. The yield of the extract was 3% w/w. The residual extract was dissolved in normal saline and used in the study.

2.3. Experimental induction of diabetes in rats

Male albino Wistar rats (body weight 130–160 g) bred in the Central Animal House, Rajah Muthiah Medical College, Annamalai University, were used in this study. The animals were fed on a pellet diet (Hindustan Lever, India) and water ad libitum.

The rats were injected with alloxan monohydrate dissolved in sterile normal saline in a dose of 150 mg/kg body weight, intraperitoneally. Since alloxan is capable of producing fatal hypoglycaemia as a result of massive pancreatic insulin release, rats were treated with 20% glucose solution (15–20 ml) intraperitoneally after 6 h. The rats were then kept for the next 24 h on 5% glucose solution bottles in their cages to prevent hypoglycaemia (Gupta et al., 1984).

After a fortnight, rats with moderate diabetes having glycosuria (indicated by Benedict's test for urine) and hyperglycaemia with blood glucose range of 200–260 mg/100 ml were used for the experiment. Blood was collected from the eyes (venous pool).

2.4. Determination of blood glucose and haemoglobin

Fasting blood glucose was estimated by *O*-toluidine method (Sasaki et al., 1972). Haemoglobin was estimated by cyanmethaemoglobin method (Drabkin and Austin, 1932).

2.5. Determination of thiobarbituric acid reactive substances and reduced glutathione

Thiobarbituric acid reactive substances (TBARS) in tissues were estimated by the method of Nichans and Samuelsson (1968). Glutathione (GSH) was determined by the method of Patterson and Lazarow (1955).

2.6. Assay of antioxidant enzymes and protein

The activity of superoxide dismutase (SOD) in tissues was assayed by the method of Kakkar et al. (1984). Catalase (CAT) was assayed according to the method of Maehly and Chance (1954). Protein content of tissue homogenates was measured by the method of Lowry et al. (1951).

2.7. Experimental design

In the experiment a total of 84 rats (72 diabetic surviving rats, 12 normal rats) were used. Diabetes was induced in rats 2 weeks before starting the treatment. The rats were divided into seven groups as follows after the induction of alloxan diabetes. In the experiment 12 rats were used in each group.

Group 1, control rats injected with 0.5 ml of physiological saline;
group 2, diabetic rats;
group 3, diabetic rats given jamun seed extract (JSEt) (2.5 g/kg body weight) in aqueous solution daily using an intragastric tube for 6 weeks;
group 4, diabetic rats given JSEt (5.0 g/kg body weight) in aqueous solution daily using an intragastric tube for 6 weeks;
group 5, diabetic rats given JSEt (7.5 g/kg body weight) in aqueous solution daily using an intragastric tube for 6 weeks;
group 6, diabetic rats given glibenclamide orally (600 μg/kg body weight) as aqueous solution using an intragastric tube daily for 6 weeks; and
group 7, diabetic rats given protamine zinc insulin intraperitoneally (6 U/kg body weight) daily for 6 weeks.

During the second, fourth and sixth weeks of feeding, the body weight, urine sugar, and fasting blood glucose of all the rats were determined. Animals described as fasted were deprived of food for at least 12 h but allowed free access to drinking water. After 42 days, the rats were sacrificed by decapitation. Blood was collected in a tube containing potassium oxalate and sodium fluoride solution for the estimation of blood glucose. Tissues (liver, kidney and heart) were also collected in ice cold containers for various estimations.

The experiment was repeated on the same number of rats.

2.8. Statistical analysis

All the grouped data were statistically evaluated and the significance of various treatments was calculated using Student's t-test. All the results were expressed as mean $\pm$ S.D.

3. Results

Table 1 demonstrates the blood glucose, total haemoglobin, change in body weight, and urine sugar of normal and experimental animals. There was a significant elevation in blood glucose ($P < 0.001$) while the level of total haemoglobin ($P < 0.01$) decreased during diabetes when compared with the corresponding control group. Administration of JSEt at 2.5 and 5.0 g and glibenclamide tends to bring the values to near normal. JSEt at a dose of 7.5 g did not show any significant effect. JSEt (at 2.5 and 5.0 g) was more effective than glibenclamide.

Table 2 gives the concentration of TBARS in tissues of normal and experimental animals. There was a significant elevation in TBARS in liver and kidney ($P < 0.001$ and $P < 0.001$, respectively) during diabetes while heart showed a significant decrease ($P < 0.001$) when compared with the corresponding control group. Administration of JSEt at 2.5 and 5.0g and glibenclamide tends to bring the values to near normal. JSEt at a dose of 7.5 g did not show any significant effect. JSEt (at 2.5 and 5.0 g) was more effective than glibenclamide.

Table 3 shows the concentration of GSH in tissues of normal and experimental animals. There was a significant decrease in the concentration of GSH in liver and heart ($P < 0.001$ and $P < 0.001$, respectively) during diabetes while kidney did not show any significant change when compared with the corresponding control group. Administration of JSEt at 2.5 and 5.0 g and glibenclamide tends to bring the values to near normal. JSEt at a dose of 7.5 g did not show any significant effect. JSEt (at 2.5 and 5.0 g) was more effective than glibenclamide.

Table 1
Blood glucose, total haemoglobin, change in body weight and urine sugar of normal and experimental rats

Group	Fasting blood glucose (mg/100 ml)		Haemoglobin (g/100 ml)	Changes in body weight(g)	Urine sugar[a]
	Initial	Final			
Normal	78.4 ± 10.15	82.0 ± 9.28	15.9 ± 4.1	34.5 ± 3.0	−
Diabetic control	220.0 ± 9.25	289.5 ± 9.63 # #	11.9 ± 1.7 #	−16.5 ± 3.0 # #	+++
Diabetic + JSEt (2.5 g)	220.0 ± 8.83	112.0 ± 6.73**	13.6 ± 0.2*	4.8 ± 1.0**	+
Diabetic + JSEt (5.0 g)	225.0 ± 8.75	107.0 ± 8.42**	13.9 ± 1.3*	5.6 ± 1.1**	+
Diabetic + JSEt (7.5 g)	222.0 ± 7.56	282.5 ± 10.46[b]	11.2 ± 1.4[b]	0.5 ± 1.2[b]	+++
Diabetic + glibenclamide	230.5 ± 9.87	114.0 ± 8.45**	13.6 ± 0.4*	6.1 ± 1.1**	+
Diabetic + insulin	215.0 ± 11.47	92.0 ± 7.41**	14.6 ± 2.5*	7.2 ± 1.2**	+

Values are given as mean ± S.D. for 12 rats in each group.
Diabetic control was compared with normal.
Experimental groups were compared with diabetic control.
[a](+) Indicates 0.25% sugar and (+++) indicates more than 2% sugar.
[b]Not significant as compared with the diabetic control.
Values are statistically significant at # $P<0.01$ and # # $P<0.001$ as compared with normal; * $P<0.01$ and ** $P<0.001$ as compared with the diabetic control.

Table 4 demonstrates the activity of SOD in tissues of normal and experimental animals. There was a significant reduction in the activity of SOD in tissues like liver and kidney ($P < 0.001$ and $P < 0.001$, respectively) during diabetes while heart did not show any significant change when compared with the corresponding control group. Administration of JSEt at 2.5 and 5.0 g and glibenclamide tends to bring the values to near normal. JSEt at a dose of 7.5 g did not show any significant effect. JSEt (at 2.5 and 5.0 g) was more effective than glibenclamide.

Table 5 illustrates the activity of CAT in tissues of normal and experimental animals. There was a significant reduction in the activity of CAT in tissues like liver and kidney ($P < 0.001$ and $P <$ 0.001, respectively) during diabetes while heart did not show any significant change when compared with the corresponding control group. Administration of JSEt at 2.5 and 5.0 g and glibenclamide tends to bring the values to near normal. JSEt at a dose of 7.5 g did not show any significant effect. JSEt (at 2.5 and 5.0 g) was more effective than glibenclamide.

4. Discussion and conclusion

Alloxan causes a massive reduction in insulin release, by the destruction of the β-cells of the islets of Langerhans and inducing hyperglycaemia (Goldner and Gomori, 1943). In our present study we have observed that an aqueous extract of jamun seeds can reverse these effect. The possible mechanism by which JSEt brings about its hypoglycaemic action may be by potentiation of the insulin effect of plasma by increasing either the pancreatic secretion of insulin from β-cells of islets of Langerhans or its release from the bound form. In this context a number of other plants have also been observed to have hypoglycaemic and insulin-release stimulatory effects. (Twaij and Al-Badr, 1988; Gupta, 1994).

We have observed a decrease in total haemoglobin during diabetes and this may be due to the formation of glycosylated haemoglobin. The increase in the level of haemoglobin in animals given JSEt may be due to the decreased level of blood glucose.

We have also observed that when JSEt was administered to animals given alloxan, the weight

Table 2
Concentration of TBARS in normal and experimental rats

Group	Concentration of TBARS (mM/100 g wet tissue)		
	Heart	Liver	Kidney
Normal	0.43 ± 0.003	0.89 ± 0.005	1.24 ± 0.05
Diabetic control	0.12 ± 0.02**	1.47 ± 0.06**	1.76 ± 0.04**
Diabetic + JSEt (2.5 g)	0.40 ± 0.04*	0.96 ± 0.04*	1.33 ± 0.05*
Diabetic + JSEt (5.0 g)	0.41 ± 0.02*	0.94 ± 0.04*	1.36 ± 0.04*
Diabetic + JSEt (7.5 g)	0.13 ± 0.01[a]	1.44 ± 0.05[a]	1.75 ± 0.03[a]
Diabetic + glibenclamide	0.39 ± 0.05*	0.97 ± 0.03*	1.32 ± 0.03*
Diabetic + insulin	0.42 ± 0.03*	0.90 ± 0.03*	1.25 ± 0.03*

Values are given as mean ± S.D. for 12 rats in each group.
Diabetic control was compared with normal.
Experimental groups were compared with diabetic control.
[a]Not significant as compared with the diabetic control.
Values are statistically significant at ** $P<0.001$ as compared with the normal; * $P<0.001$ as compared with the diabetic control.

loss was reversed and the animals returned to near normal. The ability of the JSEt to protect body weight loss seems to be due to its ability to reduce hyperglycaemia.

Lipid peroxide-mediated tissue damage has been observed in the development of both type I and II diabetes. It has been observed that insulin secretion is closely associated with lipoxygenase-derived peroxides (Metz, 1984; Walsh and Pek, 1984). Increased concentration of lipid peroxide in the liver can result in decreased activity of cytochrome P_{450} and cytochrome b_5 and this may affect the drug metabolising-activity in chronic diabetes (Levin et al., 1973). Increased concentration of TBARS is also observed in kidney during diabetes. Nakakimura and Mizuno (1980) have reported that the concentration of lipid peroxides increases in the kidney of diabetic rats. Our study shows that administration of JSEt tends to bring the kidney and liver TBARS back to near normal.

Table 3
Concentration of GSH in normal and experimental rats

Group	GSH (mg/100 g wet tissue)		
	Heart	Liver	Kidney
Normal	48.6 ± 2.5	45.9 ± 1.7	28.9 ± 1.9
Diabetic control	28.9 ± 1.8**	25.2 ± 2.0**	27.8 ± 1.2
Diabetic + JSEt (2.5 g)	45.2 ± 1.6*	42.6 ± 1.8*	26.5 ± 1.3
Diabetic + JSEt (5.0 g)	45.7 ± 1.5*	43.1 ± 1.7*	27.1 ± 1.3
Diabetic + JSEt (7.5 g)	29.5 ± 1.9	25.6 ± 2.1	26.5 ± 1.5
Diabetic + glibenclamide	44.6 ± 2.1*	42.4 ± 2.0*	26.4 ± 1.4
Diabetic + insulin	47.5 ± 2.0*	44.8 ± 1.6*	28.0 ± 1.8

Values are given as mean ± S.D. for 12 rats in each group.
Diabetic control was compared with normal.
Experimental groups were compared with diabetic control.
Values are statistically significant at ** $P<0.001$ as compared with the normal; * $P<0.001$ as compared with the diabetic control.

The decrease in GSH levels in liver during diabetes is probably due to its increased utilisation by the hepatic cells. This may be due to an attempt by the hepatocytes to counteract the increased formation of lipid peroxides. We have also observed a decrease in GSH in heart during diabetes. Our studies shows that in diabetic rats there is decreased GSH in liver and heart while administration of JSEt helps in restoring these levels to near normal.

Reduced activities of SOD and CAT in liver and kidney have been observed during diabetes and this may result in a number of deleterious effects due to the accumulation of superoxide radicals ($O^{\bullet}_2$) and hydrogen peroxide (Searle and Wilson, 1980). Administration of JSEt results in

Table 4
Activity of SOD in normal and experimental rats

Group	SOD (U[a]/mg protein)		
	Heart	Liver	Kidney
Normal	11.60 ± 1.86	10.6 ± 0.15	16.5 ± 1.6
Diabetic control	10.45 ± 1.26	5.3 ± 0.30*	8.5 ± 0.4*
Diabetic +JSEt (2.5 g)	10.63 ± 0.77	8.4 ± 0.72**	13.4 ± 0.4**
Diabetic +JSEt (5.0 g)	10.67 ± 0.96	8.7 ± 0.67**	14.1 ± 0.4**
Diabetic +JSEt (7.5 g)	10.29 ± 1.17	5.4 ± 0.67	8.9 ± 0.3
Diabetic +glibenclamide	10.62 ± 1.09	8.1 ± 0.42**	12.9 ± 0.26**
Diabetic +insulin	11.45 ± 2.42	10.1 ± 0.32**	16.2 ± 0.38**

Values are given as mean ± S.D. for 12 rats in each group.
Diabetic control was compared with normal.
Experimental groups were compared with diabetic control.
[a]U, enzyme concentration required to inhibit the OD at 560 nm of chromogen production by 50% in 1 min
Values are statistically significant at * $P<0.001$ as compared with the normal; ** $P<0.001$ as compared with the diabetic control.

Table 5
Activity of CAT in normal and experimental rats

Diabetic control	7.80 ± 0.46	67.8 ± 2.1**	24.4 ± 0.8**
Group	CAT ($\times 10^{-3}$ U[a]/mg protein)		
	Heart	Liver	Kidney
Normal	8.95 ± 1.85	71.9 ± 5.5	27.1 ± 1.9
Diabetic control	7.70 ± 1.66	29.9 ± 1.7*	20.7 ± 05*
Diabetic+JSEt (2.5 g)	8.00 ± 0.48	67.8 ± 2.1**	24.4 ± 0.8**
Diabetic+JSEt (5.0 g)	8.15 ± 0.48	68.3 ± 2.2**	25.3 ± 0.7**
Diabetic+JSEt (7.5 g)	7.80 ± 0.46	30.5 ± 2.1	20.9 ± 0.8
Diabetic +glibenclamide	7.95 ± 0.38	67.4 ± 1.8**	24.2 ± 0.7**
Diabetic+insulin	8.45 ± 0.58	70.2 ± 0.7**	26.7 ± 0.7**

Values are given as mean ± S.D. for 12 rats in each group.
Diabetic control was compared with normal.
Experimental groups were compared with diabetic control.
[a]U, velocity constant/s.
Values are statistically significant at * $P<0.001$ as compared with normal; ** $P<0.001$ as compared with the diabetic control.

the activities of SOD and CAT returning to near normal. $O^{\bullet}_2$ and hydroxyl radicals (OH*) induce various injuries in the surrounding organs and play an important role in some clinical disorders. Any compound, natural or synthetic, with antioxidant properties, might contribute towards the partial or total alleviation of this damage. Therefore, removing $O^{\bullet}_2$ and OH* is probably one of the most effective defences of a living body against diseases (Lin et al., 1995). The results of SOD and CAT activity clearly shows that the JSEt contains a free radical-scavenging activity, which could exert a beneficial action against pathological alterations caused by the presence of $O^{\bullet}_2$ and OH*. This action, predominantly due to the extract, could involve mechanisms related to scavenging activity.

Thus the study shows that JSEt has a hypoglycaemic effect. The effect was more pronounced in the case of 5.0 g/kg body weight. The decreased levels of TBARS shows that JSEt can resist, to an extent, the formation of lipid peroxy radicals in a number of tissues, thereby protecting these tissues.

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