BLACK TEA (*CAMELLIA SINENSIS*) SUPPRESSES HYPERGLYCEMIA IN STZ-INDUCED DIABETIC RATS

Mayuko Kubo[1], Iwao Sakane[2], Shin-ichi Sawamura[2], Ken-ichi Yoshida[1], and Hitoshi Ashida[1]

1: Department of Biofunctional Chemistry, Faculty of Agriculture, Kobe University, 1-1 Roddodai, Nada, Kobe, Hyogo, 657-8501, Japan
2: Central Research Institute, Ito En, Ltd., Shizuoka 421-0516, Japan

Summary
Diabetes is difficult to cure in many cases and results in a diabetic coma or death in severe cases. In order to prevent diabetic mellitus and its complications, regulation of the daily blood glucose level is very important and leads to maintain the quality of life. In this study, we investigated the effects of black tea on anti-hyperglycemia in streptozotocin (STZ)-induced diabetic rats. Black tea improved STZ-induced hyperglycemia to the control level, and inhibited the increases in glycation of proteins and oxidative stress, which are associated with complications, in serum. Therefore, our findings suggest that black tea has the preventive effects on diabetes and its complications.

Keywords
Black tea, Streptozotocin (STZ), Diabetes mellitus, Hyperglycemia

Introduction

Black tea (*Camellia sinensis*) is a popular and a daily-consumed beverage worldwide: about 80% of tea in the world is manufactured as black tea [1]. We daily intake the major sources of polyphenolic compounds from tea [2]. Recent studies demonstrate that black tea and its components including polyphenols possess various biological properties such as antioxidant activity [3, 4], amelioration of mucosal injury [5]. Recently, the anti-diabetic effects of green tea have been much attention, but it is less understood whether black tea also has the same effects as green tea yet. Since hyperglycemia is a typical symptom of diabetes mellitus, the regulation of the daily blood glucose level is very important for prevention of diabetes and it's progress. In this study, suppressive effect of black tea on the blood glucose level, glycation of proteins, and oxidative stress were investigated in STZ-induced diabetic rats.

Materials and Methods

Two grams of dry black tealeaves (Uva) were added to 100 ml of boiling distilled water, steeped for 2 min, and filtered. The supernatant was used as a hot water extract of black tea. This condition is as well as we ordinary intake a cup of tea. Fresh extract was prepared dairy for rats. Animal treatments in this study conformed to the "Guidelines for the care and use of experimental animals, in Rokkodai Campus, Kobe University". Male Wistar/ST rats (6 weeks old and weighing 170-190 g, Japan SLC, Inc., Shizuoka, Japan) were acclimated to the circumstances for 7 days. They were separated into 4 groups; (1) control + water, (2) control + black tea, (3) STZ + water and (4) STZ + black tea, and allowed free access to commercial chow and tap water or black tea for 35 days. After 7 days (referred as day 0), insulin-deficient diabetes was induced to rats by a single intravenous injection of STZ (freshly prepared in 0.05M citrate buffer, pH 4.5) via the tail vein at a dose of 40 mg/kg body weight. Control animals were injected with an equal volume of vehicle buffer alone. Blood glucose level was measured on day 0, 14 and 28 with Dexter-Z II (Bayer, Pittsburgh, PA) after 12-h fasting. On day 28, animals were sacrificed, and serum was prepared. Serum was subjected to measurement of thiobarbituric acid-reactive substances (TBARS) [6] and fructosamine [7]. Statistical significance was analyzed by Student's *t*-test; $p<0.05$ was considered significant.

Results and Discussion

Black tea had preventive effects on hyperglycemia in STZ-induced diabetic rats. As shown in Table 1, black tea had no effects on non-diabetic rats. STZ caused hyperglycemia on day 14 as the symptoms of diabetes. When black tea was pre-treated to rats, STZ-induced hyperglycemia was significantly suppressed on day 14, and finally downregulated to the control level on day 28. This indicates that black tea would have the possibility to prevent STZ-induced diabetes.

Table 1. Black tea had preventive effects on hyperglycemia in STZ-induced diabetic rats.

		Blood glucose concentration (mgdl)		
		On day 0	On day 14	On day 28
Control	+Water	93.0 ± 3.8	102.7 ± 3.0	100.0 ± 3.4
	+Black tea	92.7 ± 7.4	92.5 ± 3.5	105.0 ± 4.0
STZ	+Water	94.5 ± 4.4	309.0 ± 21.7[a]	371.2 ± 45.9[a]
	+Black tea	90.9 ± 3.4	140.5 ± 27.2[b]	124.3 ± 17.0[b]

Data are represented as the mean±SE (n=6). Significant differences from control+water group, [a]$p<0.05$; from diabetic+water group, [b]$p<0.05$ by Student's t-test.

Since oxidative stress is involved in the risk for complications of diabetes, we measured the levels of thiobarbituric acid reactive substance (TBARS) and fructosamine in serum (Table 2). Black tea significantly suppressed both of them. This means black tea had inhibitory effects on the peroxidation of serum lipids and the non-enzymatic glycation of protein in diabetic animals. Therefore, black tea would control the development of complications in diabetic rats by down regulating of the blood glucose level. In conclusion, our findings strongly suggest that black tea has the preventive effects on STZ-induced diabetes and its complications, indicating that black tea is useful beverage for insulin-deficient diabetes mellitus.

Table 2. Black tea helped reduce the risk for complications of diabetes.

		Control		STZ	
		Water	Black tea	Water	Black tea
TBARS	(neq/ml)	1.60 ± 0.05	1.72 ± 0.04	2.70 ± 0.10[a]	2.34 ± 0.06[ab]
Fructosamine	(μmol/ml)	1.23 ± 0.02	1.25 ± 0.01	1.74 ± 0.04[a]	1.47 ± 0.03[ab]

Data are represented as the mean±SE (n=6). Significant differences from control+water group, [a]$p<0.05$; from diabetic+water group, [b]$p<0.05$ by Student's t-test.

References

[1] Krishnamoorthy K.K., 1991. In Proceeding of the International Symposium on Tea Science., pp.6-11.
[2] Hertog M.G. *et al.*, 1993. *Lancet.*, 342, 1007-1011.
[3] Rice-Evans C.A. and Miller N.J., 1996. *Biochem. Soc. Trans.*, 24, 790-795.
[4] Fadhel Z.A.and Amran S., 2002. *Phytother. Res.*, 16, 28-32.
[5] Maity S. *et al.*, 2002. *Eur. J. Pharmacol.*, 470, 103-112.
[6] Kikugawa K. *et al.*, 1992. *Anal. Biochem.*, 202, 249-255.
[7] Johnson R.N. *et al.*, 1983. *Clin. Chim. Acta.*, 127, 87-95.