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Effects of Korea Red Ginseng on Substance Metabolism and Endocrinic Function in Animals

Z.M. Zhou, Z.L. Li, S.Q. Nie, L.X. Li, X.M. Sun, Y.L. Wang,
E.H. Liang, G.Q. Li and L.X. Yang
Institute of Chinese Materia Medica, China Academy of Traditional Chinese Medicine

Panax ginseng has been used as tonics in traditional Chinese medicine for thousands of years. It was confirmed that *Panax ginseng* had wide effects on the different systems in humans, such as central nervous system, cardiac vascular system, endocrine system, immunoresponse system and substance metabolism, *etc.*

A large amount of chemical, pharmacological and biochemical research works have been carried out with morden science and technique by the scientists. It is known that *Panax ginseng* form China and Korea, *etc.* contains a series of compounds which have various pharmacological activities. Even different compound in Ginseng has contradictory action. For instance, Ginsenoside-Rb1 enhanced, while Ginsenoside-Rc repressed the RNA synthesis and the activities of RNA polymerases I & II. It is necessary to separate and purify the active compounds from Ginseng, and carry out the studies of their biochemistry and molecular pharmacology.

According to the characteristics of clinical use of Traditional Chinese Medicine, Gingseng is often used in complex prescription decoction, besides used in single decoction. The biological activity of Ginseng decoction actually represents the collective action of various compounds Ginseng contains.

In this report, the effects of Korea Red Ginseng (KRG) on the DNA, RNA and protein metabolism in mice have been observed, the influence of the drugs on the amount of plasma corticosterone in mice, and the effects on hypoglycemia and convulsion induced by insulin have also been described.

1. Effect on the incorporation of ^{3}H-TdR into DNA of spleen and testicle in mice

Male Kuming strain mice, weighing 20 ± 1.2g, were used. The preparation of KRG imported from Korea was solution of dry powder from the aqueous extracts of KRG crude drug. .0.5 g/kg (crude drug/body weight) KRG were administered p.o. into the mice, twice daily for 9 times. Then 1hr after the last dosing i.p. ^{3}H-TdR(3E 5Bq/20g) and 3 hr after injection, the mice were sacrificed, and spleen and testicle were taken off for measuring radioactivities incorporated into DNA by a filter-paper disk method with liquid sintillation spectrometer. The results of incorporation of ^{3}H-TdR into DNA in mice showed that there was no significant change in spleen and testicle of mice after been administrated by the preparation of KRG. It indicated that the KRG could not affect the DNA metabolism in these two organs under these condition (Table 1).

2. Effect on the incorporation of ^{3}H-UR into RNA of liver and kidney in mice

The preparation of KRG at the doses of 0.5 g/kg or 1.0 g/kg was given orally to mice twice a daily for 5 times. 1 hr after the last dosing, i.p. ^{3}H-UR (2.2E 5Bg/20g) and 3 hr after injection, mice were sacrificed to take off the liver and kidney for measuring radioactivities incorporated into RNA.

The results showed that the incorporation of ^{3}H-UR into RNA in all test groups tented to increase. The radioactivities in the liver of mice significantly increased after administration of KRG only at the dose level of 1.0 g/kg and no remarkable change was found in kidney of the mice given at the doses of 0.5 g/kg and 1.0 g/kg (Table 2).

Table 1. Effect of KRG on the Incorporation of ^{3}H-TdR into DNA of spleen and testicle in mice

Group	Dosage (g/ kg.po)*	Radioactivity (CPM×10⁴/g tissue) Spleen $\bar{X}\pm SD$	Testicle $\bar{X}\pm SD$	p
Control	N.S.	15.9±4.9 (9)#	3.40±1.82 (8)	
KRG	0.5×9	11.8±5.1 (10)	2.09±0.57 (9)	>0.05

* Equal to crude drug
# (n): Number of mice

Table 2. Effect of KRG on the incorporation of ^{3}H-UR into RNA of liver and kidney in mice

Group	Dosage (g/ kg.po)*	Radioactivity (CPM×10⁴/g tissue) Liver $\bar{X}\pm SD$	Kidney $\bar{X}\pm SD$
Control	N.S.	11.6±4.5 (9)#	3.80±0.77 (8)
KRG	0.5×5	15.2±5.0 (10)	4.60±0.93 (10)
KRG	1.0×5	19.7±5.5** (10)	5.60±2.48 (10)

* Equal to crude drug
** Compared with cotrol group: $p<0.01$
# (n): number of mice

Table 3. Effect of KRG on the amount of protein of liver and kidney in mice

Group	Dosage (g/kg.po)*	No. of Mice	Amount of Protein (mg/g tissue) Liver $\bar{X}\pm SD$	Kidney $\bar{X}\pm SD$
Control	N.S.	9	133.3±30.4	124.6±18.9
KRG	0.5×9	9	138.8±37.2	128.8±14.4
KRG	1.0×9	9	133.3±23.3	113.0±14.9

* Equal to crude drug
Compared with control group: KRG groups $p>0.05$

Table 4. Effect of KRG on plasma corticosterone in mice

Group	Dosage (g/kg.po)*	No. of mice	Amoiunt of Corticosterone (μg/dl) $\bar{X}\pm SD$
Control	N.S.	16	28.84±16.72
KRG	0.6	16	32.46± 9.08

* Equal to crude drug

3. Effect on the amount of protein in liver and kidney

The same proccedure as DNA incorporation test was used to give KRG to mice as described above. The samples of the liver and kidney were taken for measuring the amount of protein with Bradford method.

The results showed that the same amount of protein in the liver or kidney were obtained in the KRG test groups and the control group. So, the protein synthesis in the liver and kidney of mice can not be affected by the KRG at the dose of 0.5 g/kg or 1.0 g/kg p.o. (Table 3).

4. Effect on the plasma corticosterone

.6 g/kg of KRG were injected i.p. to the mice, the blood was drawn 30 min later and centrafugated to get plasma, the corticosterone was measured using radioimmunoassay method. No difference between KRG group and control group was observed. A single dose of 0.6 g/kg of KRG i.p. had no effects on the plasma corticosterone (Table 4).

5. Effect on the hypoglycemia and convulsion induced by insulin in rats

1) Effect of reincreasing blood sugar

The Wester rats were divided at random into two groups according to the value of blood sugar, insulin 4(IU/kg) was given by hypodermic injection, 30 min later, 5 g/kg of KRG was administered i.p. to one group, water was injected to the control group, and blood sugar concentration were measured 2,3, 4,5,6 after insulin injecting, respectively.

The range of blood sugar before insulin injection was 103-106 mg/dl, it decreased to the 40 mg/dl in the control group after insulin injecting, but in drug group, there was a significant reincreases at 2 hour phase, the drug has a reincreasing effect on the hypoglycemia by insulin (Table 5).

Table 5. Effect of KRG on the blood sugar after insulin injection

Group	Dosage (g/kg)*	No. of rats	Blood sugar# (mg/dl) $\bar{X} \pm SD$	P
Control		11	40.0±10.5	
KRG	5.0	10	67.3±23.0	<0.05

* Equal to crude drug
# 2 hrs after insulin injection

Table 6. Effect of KRG on the convulsion produced by insulin

Group	Dosage (g/kg)*	No. of rats	Convulsion time (min) $\bar{X} \pm SD$	P
Control		9	116.1±24.81	
KRG	5.0	10	188.8±49.82	<0.01

* Equal to crude drug
# 2 hrs after insulin injection

2) Anticonvulsant action

When the large dose of insulin (20 IU/kg s.c.) was given to the rats, it induced convulsion or shock. After insulin injecting s.c., 5 g/kg of KRG was given i.p., the time of onset of convulsion in the drug group was remarkably delayed; 116.1 ±24.48 min in the control group. 188.8 ± 49.2 min in the KRG group (Table 6).

Summarization of above research work as follows:

Pharmacological Actions	Result
DNA Metabolism	±
RNA Metabolism	+ +
Protein Metabolism	±
Plasma Corticosterone	±
Insulin-Induced Hypoglycemia and convulsion	
Reincreasing Blood Sugar	+
Anticonvulsant Action	+ +

Compared with Control group: + $p < 0.05$, + + $p < 0.01$