

Effect of Feeding Mixture of Soybean Peptides, L-Carnitine and Garcinia Cambogia Extract on Body Weight and Lipid Metabolism in Rats

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Abstract

This study was performed to investigate effects of the experimental mixture containing soybean peptides, L-carnitine and Garcinia Cambogia extract on body weight and lipid metabolism in rats. Male Sprague-Dawley rats (n=40) of eight weeks old were raised for four weeks with high fat diet (40% fat as calorie) to induce obesity. After induction of obesity, rats were feed control (C) diet, containing either 0.16% (+1D), 1.6% (+10D), 8% (+50D) of experimental mixture for eight weeks. Plasma aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activity and total protein and albumin concentration were not different among groups. The Body weight gain was significantly lower in experimental mixture diet group compared to control group. Weights of perirenal fat pad and epididymal fat pad in the +50D group were significantly lower than those in the +1D and +10D groups. Plasma total lipid and liver total cholesterol levels in the experimental groups were significantly lower than those in the control group. Fecal total lipid and total cholesterol excretions were highest in +50D group. These results suggest that the experimental mixture containing peptides, L-carnitine and Garsinia Canbogia extract is effective for reducing the body weight and adipose tissue weight which may be due to the modulation of lipid metabolism and the increased fecal excretion of lipid.

Key words: soybean peptides, L-carnitine, Garcinia Cambogia extract, body weight

INTRODUCTION

Korea has experienced rapid socioeconomic growth with prominent lifestyle transformation over the past several decades, therefore overweight and obesity represent a rapidly growing health threat (1). Obesity is defined as an excess of body weight that is mainly attributable to an increased body fat accumulation and induced by an imbalance of energy intake and expenditure (total energy expenditure includes energy expended at rest, in physical activity and for metabolism (2). According to the Korean National Nutrition Survey found that the prevalence of obesity (BMI 25.0 kg/m²) increased from 26.3% in 1998 to 30.6% in 2001 (3). Overweight carries an increased risk of health problems such as cardiovascular disease, insulin resistance, diabetes, hyperlipidemia, hypertension, gallbladder disease, certain cancers, and premature mortality (4). Therefore, the effect of dietary components on lipid metabolism have recently received considerable attention, thereby highlighting the importance of naturally occurring compounds as lipid me-

tabolism regulators. From this point of view, soybean peptides, L-carnitine and Garcinia Cambogia extract have been attracted public attention. Thus, identification of a safe and efficacious supplement (mixture containing soybean peptides, L-carnitine and Garcinia Cambogia extract) for weight management is extremely important for health professionals in treating obesity.

Soybean peptides may lower plasma cholesterol and triglycerides via several mechanisms. Possible mechanisms proposed for soybean peptides include a decrease in the intestinal absorption of cholesterol and/or bile acids (5,6), increased plasma cholesterol clearance through enhancing hepatic LDL-receptor activity (7,8), and changes in hepatic biotransformation of cholesterol (9). The total amount of neutral steroids excreted in feces have a tendency to increase (10). L-carnitine can be formed endogenously in the mammalian metabolism from the precursors lysine and methionine, mainly in the liver (11). L-carnitine has an important function in the fatty acid metabolism. It is a co-factor of carnitine palmitoyl transferase and needed for the transport of long-

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chain fatty acids into the mitochondrial, the site of β -oxidation (12).

The dried fruit rind of *Garcinia Cambogia* (Lewis and Neelakantan 1965) is novel source for (-)-hydroxycitric acid (HCA) (13). HCA is a potent competitive inhibitor of ATP-citrate lyase, which is an extramitochondrial enzyme catalyzing the cleavage of citrate to oxaloacetate and acetyl-CoA, thus resulting in the suppression of body fat accumulation through stimulation of carnitine palmitoyl transferase I activity and promotion of fatty acid oxidation (14).

In this study, we investigated the effect of feeding experimental mixture (soybean peptides, L-carnitine and *Garcinia Cambogia* extract) on body weight and lipid metabolism in rats. Body and fat pad weight; and total lipids, triglycerides and cholesterol levels in plasma and liver, lipids and protein excretions in fecal were compared and used to evaluate the relative antiobesity effect of the experimental mixture.

MATERIALS AND METHODS

Dietary mixture

Powder form experimental mixture containing soybean peptides, L-carnitine and *Garcinia Cambogia* extract was provided from the CJ Foods R & D (Seoul, Korea). This study was performed to investigate of feeding experimental mixture in rat. The compositions of the experimental diets are shown in Table 1.

Animals and dietary treatment

Rats were purchased from Korea Bio genomics Inc. (Daejeon, Korea). 40 of 8-week-old male Sprague-Dawley rats (Charles River) were used in this study and they were adapted on solid assorted fed (Rodent Laboratory Chow, Ralston Purina, St. Louis, MO) for 1 week prior to breeding on experimental diet. After 1 week of acclimatization, rats of weighing 329.1 ± 1.7 g were raised for 4 weeks with high fat diet (40% fat as calorie) to induce obesity. The compositions of high fat diets are shown in Table 2. After induction of obesity, rats weighing 560.8 ± 4.1 g were randomly divided into four groups (n=10) and were assigned to different dietary treatments. Diets containing different levels of HCA (1

dose 1.6 g, 10 dose 16 g and 50 dose 80 g/kg diet) were fed to 8-week-old rats to drink free. The compositions of the experimental diets are shown in Table 2. Mineral (AIN-93M) and vitamin (AIN-93M) mixes were purchased from Harlan Teklad (USA). Rats were fed experimental mixture-free diet (control group), with diet containing either 0.16% (group +1D), 1.6% (group +10D) or 8% (group +50D) of experimental mixture and tap water *ad libitum* for eight weeks. Food intake was measured three times per week and body weight gain was measured weekly.

Rats were housed one per cage under controlled temperature (23 $^{\circ}$ C) and the relative humidity (505%) with a 12-hour light/dark cycle and allowed free access to water and diets assigned to individual feeding groups (15,16).

Sampling (blood, liver and adipose tissue)

Rats were put in cage for 24 hours to collect feces during the last five days of the feeding period. Feces were collected and weighed daily, followed by lyophilization. The dried fecal sample were weighed, ground, and stored at -20 $^{\circ}$ C until analyzed (15,16).

At the end of the experiment, rats were deprived of food for 12 hours and then anesthetized using diethyl ether. A central longitudinal incision was made into the abdominal wall and blood samples were collected by cardiac puncture with syringes. Blood samples were centrifuged at 2,800 rpm for 30 minutes at 4 $^{\circ}$ C and the plasma was separated and stored at -20 $^{\circ}$ C until analyzed. Liver samples were excised, immediately frozen in liquid nitrogen and stored at -70 $^{\circ}$ C until analyzed. The adipose tissue (kidney, epididymal) and brown adipose tissue were dissected, rinsed with saline, and weighed. Care and treatment of experimental animals were in accordance with the guide for the care and use of laboratory animals of Ewha Womans University.

Analysis

Plasma lipid analysis: The plasma total lipid level was measured by Ferlito S et al. (17). Triglycerides (TG) and total cholesterol levels were determined by enzymatic colorimetric methods using commercial kits (Yeongdong pharmaceutical, Korea). HDL-cholesterol level was determined by enzymatic colorimetric methods using commercial kits (Asan pharmaceutical, Korea).

HDL-cholesterol was calculated and LDL-cholesterol according to the Friedwald-Fredrickson's formula was obtained (17). Modification of a rapid method of analysis of LDL-cholesterols by Friedwald calculation: (LDL-cholesterol)=[(total cholesterol-HDL cholesterol)-triacylglycerides/5]. AST and ALT levels were determined

Table 1. Composition of experimental mixture

Ingredients	Percentage (%)
Soybean peptides	20.8
L-carnitine	0.4
Garcinia Cambogia extract	6.2
Fiber	72.6
Total	100.0

Table 2. Composition of experimental diets

(unit: g/kg diet)

Ingredients	Dietary groups ¹⁾		Normal diet ²⁾		
	High fat diet	C	+1D	+10D	+50D
Corn starch	371.2	465.7	464.1	449.7	420.7
Casein	174.0	140.0	140.0	140.0	140.0
Dextrinized corn starch	155.0	155.0	155.0	155.0	155.0
Sucrose	100.0	100.0	100.0	100.0	100.0
Soybean oil	100.0	40.0	40.0	40.0	45.0
Fiber	50.0	50.0	50.0	50.0	10.0
Mineral mix ³⁾	35.0	35.0	35.0	35.0	35.0
Vitamin mix ⁴⁾	10.0	10.0	10.0	10.0	10.0
L-Cystine	1.8	1.8	1.8	1.8	1.8
Choline bitartrate	2.5	2.5	2.5	2.5	2.5
Experimental mixture	–	–	1.6	16.0	80.0
Cholesterol	0.5	–	–	–	–
Tert-butyl hydroquinone	0.008	0.008	0.008	0.008	0.008
Total	1,000	1,000	1,000	1,000	1,000
Total calorie (kcal)	4,463	3,601	3,598	3,565	3,589
Carbohydrates (% as calorie)	45.6	75.9	75.8	75.3	73.0
Protein (% as calorie)	14.1	14.1	14.2	14.6	15.7
Fat (% as calorie)	40.3	10.0	10.0	10.1	11.3

¹⁾Control rat were control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

²⁾Normal diet: Based on AIN 93 diet.

³⁾Mineral mixture: AIN-93M-MX (mg/kg diet): Calcium 5,000, Phosphorus 3,000, Magnesium 511, Sodium 1,033, Potassium 3,600, Chloride 1,613, Sulfur (inorganic) 300, Iron 45, Zinc 35, Manganese 10, Copper 6, Iodine 0.2, Molybdenum 0.15, Selenium 0.17, Silicon 5, Chromium 1, Fluoride 1, Nickel 0.5, Boron 0.5, Lithium 0.1, Vanadium 0.1.

⁴⁾Vitamin mixture: AIN-93M-VX (mg/kg diet): Nicotinic acid 30, Ca pantothenate 15, Pyridoxine 6, Thiamin 5, Riboflavin 6, Folic acid 2, Biotin 0.2, Vitamin B-12 25 µg, Vitamin K 860 µg, Vitamin E 75 IU, Vitamin A 4,000 IU, Vitamin D 1,000 IU, Choline 1,000.

by the method of Retiman-Frankel using commercial kits (Yeongdong pharmaceutical, Korea). Total proteins and albumin levels were determined by biuret reaction using commercial kits (Yeongdong pharmaceutical, Korea).

Liver analysis: Total lipid level in liver was extracted by Drouillard et al. (18). Triglycerides and total cholesterol levels in liver were determined by enzymatic colorimetric methods using commercial kits (Yeongdong pharmaceutical, Korea). Total protein in liver determined by the method of micro-Kjeldahl (19).

Fecal analysis: Total lipid, triglycerides and total cholesterol were determined using the same method described for liver. Fecal moisture content was estimated by drying the sample to a constant weight at 105°C. Total protein in fecal determined by the method of micro-Kjeldahl (19).

Statistical analysis: Data were expressed mean \pm SE. Data for the control, experimental mixture groups were analyzed by one-way ANOVA; $p \geq 0.05$ was taken as indicating no significant difference. Where ANOVA showed significance, differences among groups were evaluated by Duncan's multiple range test.

RESULTS

Body weight gain, food intake and food efficiency

Body weight gain, food intake and food efficiency of rats in the control and experimental groups are shown in Table 3. There were no significant differences in food efficiency both +1D and +10D experimental groups compared to the control group. But there were significantly reduced in the +50D group ($p < 0.05$). Experimental groups significantly reduced the weight gain compared to the control group ($p < 0.05$). Rats treated with 8% (group +50D) experimental mixture had significantly lower body weight gain than rats treated with 0.16% (group +1D) and 1.6% (group +10D) experimental mixture ($p < 0.05$). The food efficiency ratio of the +50D group was lower than that of the +1D and +10D group.

Tissue weights

Kidney and epididymal fat pad and brown fat weights are shown in Table 4. The kidney fat pad weight tended to be lower in experimental groups. Weight of epididymal fat pad in the +50D group were significantly lower than those in the +1D and +10D groups ($p < 0.05$).

Table 3. Food intake, body weight gain and energy efficiency in Sprague-Dawley rats fed diets containing different levels of experimental mixture¹⁾

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
Food intake (g/day)	25.51 ± 0.56 ^a	24.73 ± 0.60 ^a	25.56 ± 0.48 ^a	22.84 ± 0.58 ^b
Weight gain (g/two months)	129.00 ± 5.48 ^a	103.78 ± 7.95 ^b	103.13 ± 5.29 ^b	75.50 ± 10.32 ^c
Energy efficiency ³⁾	334.89 ± 0.004 ^a	277.05 ± 0.006 ^b	263.81 ± 0.003 ^{bc}	211.75 ± 0.007 ^c

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat were control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

³⁾Energy efficiency = Food efficiency ratio [(Body weight gain (g)/Food intake (g)) × total calorie (kcal)].

Table 4. Adipose tissue weights in Sprague-Dawley rats fed diets containing different level of experimental mixture¹⁾ (unit: g)

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
Perirenal fat pad	30.12 ± 1.96 ^a	26.47 ± 1.76 ^{ab}	27.63 ± 2.16 ^a	21.61 ± 2.05 ^b
Epididymal fat pad	21.55 ± 1.21 ^a	18.19 ± 1.26 ^{ab}	18.26 ± 1.15 ^{ab}	15.25 ± 1.54 ^b
Brown	0.55 ± 0.07	–	0.63 ± 0.04	–

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat fed control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

Table 5. Plasma AST and ALT activities and total protein and albumin concentration in Sprague-Dawley rats fed containing different level of experimental mixture¹⁾

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
AST (U/L)	76.75 ± 8.04	75.24 ± 13.74	68.74 ± 9.19	64.48 ± 6.05
ALT (U/L)	38.50 ± 5.15	26.50 ± 3.53	26.80 ± 2.28	30.90 ± 7.47
Total protein (g/dl)	6.56 ± 0.23	6.86 ± 0.20	7.11 ± 0.26	7.03 ± 0.19
Albumin (g/dl)	3.49 ± 0.09	3.22 ± 0.09	3.47 ± 0.10	3.32 ± 0.11

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat fed control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

However the experimental groups exhibited no reduction in the weights of brown fat compared to the control group.

Plasma aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities

Effects of experimental mixture on the plasma AST and ALT activities were measured. The plasma AST and ALT activities tended to be lower in experimental groups, though not significantly (Table 5).

Plasma total protein and albumin levels

Total protein and albumin levels in plasma are shown in Table 6. Total protein and albumin levels in plasma were not significantly different among all groups.

Plasma lipids levels

Total lipid, triglycerides, total cholesterol, HDL-cho-

lesterol and LDL-cholesterol levels in plasma are shown in Table 6. Total lipid level in the experimental groups were significantly lower than those in the control group (p<0.05). The plasma triglyceride and total cholesterol levels tended to be lower in the experimental groups. HDL-cholesterol and LDL-cholesterol levels were not significantly different among all groups.

Liver lipids levels

Total lipid, triglycerides, total cholesterol levels in plasma are shown in Table 7. Total lipid and triglyceride levels in liver were not significantly different among all groups. Total cholesterol levels tended to be lower in the control group compared to experimental groups. Total protein level in liver was not significantly different among all groups.

Table 6. Plasma total lipid, triglyceride, total cholesterol, HDL-cholesterol and LDL-cholesterol levels in Sprague-Dawley rats fed diets containing different level of experimental mixture¹⁾

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
Plasma lipids (mg/dL)				
Total lipid	382.50 ± 27.23 ^a	287.69 ± 15.94 ^b	274.89 ± 27.32 ^b	282.95 ± 34.83 ^b
Triglyceride	159.54 ± 17.99	148.27 ± 16.32	106.22 ± 11.83	128.55 ± 28.51
Total cholesterol	96.52 ± 5.79	88.58 ± 6.54	88.55 ± 9.76	94.37 ± 11.21
HDL-cholesterol	41.39 ± 3.31	38.36 ± 2.76	34.96 ± 3.22	37.87 ± 4.55
LDL-cholesterol	26.38 ± 5.29	21.49 ± 3.55	36.25 ± 4.95	35.56 ± 6.51

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat fed control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

Table 7. Liver total lipid, triglyceride, cholesterol and total protein levels in Sprague-Dawley rats fed diets containing different level of experimental mixture¹⁾

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
Liver lipids (mg/g wet weight)				
Total lipid	50.61 ± 6.93	57.04 ± 5.28	58.19 ± 3.15	52.29 ± 5.55
Triglyceride	3.15 ± 0.30 ^{ab}	3.97 ± 0.36 ^a	3.82 ± 0.27 ^a	2.28 ± 0.41 ^b
Total cholesterol	1.32 ± 0.09 ^b	2.04 ± 0.08 ^a	1.93 ± 0.08 ^a	1.90 ± 0.05 ^a
Total protein (g/g dry liver)	0.35 ± 0.04	0.37 ± 0.02	0.40 ± 0.01	0.38 ± 0.02

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat fed control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

Table 8. Feces weight, fecal total lipid, triglyceride, cholesterol and total protein excretions in Sprague-Dawley rats fed diets containing different level of experimental mixture¹⁾

	Dietary groups ²⁾			
	C	+1D	+10D	+50D
Feces weight (g/day)				
Wet weight	1.61 ± 0.19	2.32 ± 0.36	2.07 ± 0.25	2.15 ± 0.28
Dry weight	1.20 ± 0.14	1.37 ± 0.19	1.33 ± 0.15	1.42 ± 0.15
Fecal lipids and protein excretions (mg/g-fecal dry wt)				
Total lipid	16.98 ± 3.67 ^b	18.69 ± 2.97 ^{ab}	11.28 ± 1.44 ^b	22.14 ± 5.40 ^a
Triglyceride	0.02 ± 0.12	0.03 ± 0.21	0.05 ± 0.24	0.02 ± 0.09
Total cholesterol	6.41 ± 0.47	5.84 ± 0.68	3.16 ± 0.47	6.10 ± 0.68
Total protein	0.13 ± 0.05 ^a	0.05 ± 0.03 ^b	0.11 ± 0.05 ^{ab}	0.06 ± 0.03 ^{ab}

¹⁾Values are mean ± SEM (n=10). Means different letters differ (p<0.05).

²⁾Control rat fed control mixture diet for 8 wk (C), and experimental rats fed 1 dose (1.6 g/kg diet) of experimental mixture added (+1D), 10 dose (16 g/kg diet) of experimental mixture added (+10D) and 50 dose (80 g/kg diet) of experimental mixture added (+50D) for 8 weeks followed by 4 weeks of feeding diet containing high fat diet.

Fecal lipids and protein excretions

The effect of experimental mixture on the feces weight, fecal lipids and protein excretions is shown in Table 8. No significant differences in feces weight were observed between the control group and the experimental groups. Total lipid excretion was higher in the experimental groups except for +10D when compared to

control group. Triglyceride and cholesterol in feces were higher in rats fed experimental mixture compared with the control group. Total protein was not significantly different among all groups.

DISCUSSION

Our study was carried out to clarify whether the ex-

perimental mixture containing soybean peptides, L-carnitine and Garcinia Cambogia extract affects the body weight and lipid metabolism in rats. The results showed that body weight gain, levels of plasma lipids and cholesterol in rats fed experimental mixture were reduced by the experimental mixture supplementation.

Our study may provide an approach integrating the HCA (L-carnitine and Garcinia Cambogia extract) with biomedical sciences aimed at new food source discoveries (20). Also, chemotherapeutic potential of HCA-containing Garcinia Cambogia should be investigated further. The previous human study research in (20) based on HCA (L-carnitine and Garcinia Cambogia extract) intake suggested that human has higher absorption and basal metabolic rate than rodent. We were concerned about experimental animal that human differences in that HCA (L-carnitine and Garcinia Cambogia extract) intake affect by diet absorption. Diet containing of HCA in 1 dose 1.6 g/kg diet also examined to safety dosage (20). We observed that the experimental mixtures significantly reduced on body weight gain in the obese rats. Ali AA et al. (21) reported that soybean isoflavones in diets reduced the body weight gain compared to the control group. Shara M et al. (22) reported that HCA-containing Garcinia Cambogia has been shown to be active in suppressing appetite and body fat accumulation in experimental groups.

Moreover, acetyl-CoA production from glucose in humans has been reported to be approximately one-fortieth of that in rats because of low activity of ATP-citrate lyase in humans (23), and thus it is further unlikely that obese people would experience a suppression of body fat accumulation by HCA except in the setting of an unphysiological high-carbohydrate and low-fat diet containing very high level of HCA (24). These results suggested that a high level of experimental mixture might be required to decrease body fatness. Food efficiency was decreased in the experimental groups compared with the control group.

Weights of kidney and epididymal fat pad in the experimental groups were lower than that in the control group. However the experimental groups exhibited no reduction in the weights of brown fat compared to the control group. As a result, a significant suppression of body fat accumulation was observed in kidney and epididymal fat pad in the highest dietary level of experimental mixture. Saito M et al. (14) reported that epididymal fat pad weights were significantly lower in the highest HCA group than in the other groups. Effects of carnitine supplementation on the lipid metabolism are most likely to occur when there is intensive oxidation

of fatty acids (12).

AST can be generally found in the liver, cardiac muscle, kidneys, brain, pancreas, lungs, leukocytes, and erythrocytes, whereas, ALT is present in highest concentration in liver (25). In previous study (25,26), serum AST and ALT of experimental groups were found to be significantly lower than those of controls groups. Further studies to determine whether HCA effect in serum or liver of the AST and ALT levels also the expression of other function would provide greater insight into their potential mechanism. Usually the levels of AST and ALT are low in the blood, but are increased in case of diseases such as acute hepatitis or myocardial infarction. Those enzymes are released from cells into the blood due to the increased membrane permeability (26).

Total protein and albumin concentrations in the experimental groups were not significantly. Plasma total lipid concentrations in the experimental groups were significantly lower than the control group. Triglyceride concentration tended to be lower in the experimental groups compared to the control group. Whereas, plasma total cholesterol, HDL-cholesterol and LDL-cholesterol were not significantly different in the all groups.

Liver total lipid and triglyceride concentrations in all treated groups were not significantly different. Total cholesterol concentration was decreased in the experimental groups compared with that in the control group. Lin Y (27) reported that after five weeks of feeding, the soybean protein diet reduced plasma triglyceride by 13 and 9%, respectively, compared with the control diet. There have been occasional reports in the literature that carnitine supplements, especially in hyperlipidaemic individuals, reduce plasma lipid concentrations and can modify the lipid metabolism (28). It is therefore, reasonable that the hypocholesterolemic effect of HCA may stem in part from impaired cholesterol synthesis. Animal studies, however, have shown that the HCA diet supplementation in the diet depressed the hepatic activities of lipogenic and cholesterologenic enzyme such as fatty acid synthesis, glucose-6-phosphate dehydrogenase and HMG-CoA reductase (28). The excretion of total lipid, triglyceride and total cholesterol in feces of rats fed with experimental mixture was increased in compared to those of the control diet.

Experimental mixture increased fecal wet and dry weights by group (+1D) and group (+50D), respectively. Total protein was not significantly different among all groups. These results demonstrate that the experimental mixture was more effective than control diet in the excretion of total lipid, triglyceride and total cholesterol

via feces. Several studies have suggested that the hypocholesterolemic effect of vegetable proteins, in particular soybean protein, is largely attributable to higher fecal steroid excretion as a consequence of the reduction in intestinal absorption (29,30). In our observation, the experimental mixture containing peptides, L-carnitine and Garcinia Cambogia extract was effective for reducing the body weight and the effect may be due to the modulation of lipid metabolism and increase fecal excretion of lipid.

In conclusion, the addition of experimental mixture containing soybean peptides, L-carnitine and Garcinia Cambogia extract to rat diets had a beneficial effect on reducing body weight and body fat. The effect of experimental mixture was associated with lowering in the total lipids levels in plasma and liver, and decreasing AST and ALT activities. Further research is required to fully delineate the mechanisms that contribute to the obesity effects of the experimental mixture and its constituents.

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