Effects of *Garcinia cambogia* Extract Feeding on Body Weight and Lipid Profiles in Rats Fed a High-carbohydrate or High-fat Diet

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Abstract   This study was designed to evaluate the effects of high-carbohydrate (HC) or high-fat (HF) diet with hydroxycitric acid (HCA)-containing *Garcinia cambogia* in rats. Seventy male Sprague-Dawley rats were randomly divided into 7 groups and raised with experimental diets containing different HCA levels (0, 1.6, and 3.2%) and calorie sources (carbohydrate, fat) for 8 weeks. Energy intake was significantly reduced in rats fed a HC diet with HCA compared to the HC diet without HCA. Body weight gain was significantly reduced in HCA groups. In the diet groups, plasma total lipid and triglyceride (TG) levels of the HCA groups were significantly lower than those of the group without HCA. There were no significant differences in energy intake and plasma lipid profile in HF groups. These results suggest that HC diet with HCA was more effective in the reduction of energy intake, body weight gain, and plasma lipid contents than those of HF diet with HCA.

Keywords: *Garcinia cambogia*, hydroxycitric acid, obesity, high-fat diet, high-carbohydrate diet

Introduction

Obesity, particularly with visceral fat accumulation is a serious risk factor for so-called lifestyle-related diseases (1,2) and is a worldwide problem that complicates or contributes to serious diseases including hypertension, diabetes, coronary vasculature disease, cardiovascular disease, and stroke. (3,4) Therefore, if antiobesity foods and food ingredients are effective in reducing body fat accumulation, they may avert obesity, possibly leading to the prevention of lifestyle-related diseases (5).

Garcinia (family: Guttiferae) is a large genus of polygamous trees or shrubs, distributed in tropical Asia, Africa, and Polynesia (6). It consists of 180 species, of which ca. 30 species are found in India. Hydroxycitric acid (HCA) is contained in the rind of the Indian fruit, *Garcinia cambogia* (7). HCA has been reported to have an effect on lipid metabolism (2,8-10) and suppress food intake, body weight gain, and visceral fat accumulation (11-15). HCA inhibits *de novo* lipogenesis by blocking the extramitochondrial enzyme, adenosine 5'-triphosphate (ATP) citrate lyase, which catalyzes the cleavage of citrate to acetyl coenzyme A and oxaloacetate (16,17). This inhibitory action of HCA reduces the acetyl-CoA pool, thus limiting the availability of 2-carbon units required for the initial steps of fatty acid and cholesterol biosynthesis (18-22). This enzyme is particularly important during the hyperlipogenic nutritional state produced by a high carbohydrate diet. The reduction in the acetyl-CoA pool is proposed to decrease the concentration of malonyl-CoA, thus resulting in the suppression of body fat accumulation through the stimulation of carnitine palmitoyltransferase I activity and promotion of fatty acid oxidation (9,23-25). In our previous study, we investigated the effects of HCA on body weight gain and visceral fat accumulation in rats fed a high fat diet. Therefore, this study was designed to evaluate the effects of HCA, an inhibitor of *de novo* lipogenesis, in rats fed a high-carbohydrate diet or a high-fat diet with HCA.

Materials and Methods

Animals and housing   Eight-week old male Sprague-Dawley rats (CD Outbred, International Genetic Standard, Charles River Origin; Jung-Ang Lab. Animal, Inc., Seoul, Korea) were placed in individual stainless steel wire-mesh cages in a climate-controlled room. The room was maintained at 22-24°C and a relative humidity of 45±5%, with a 12-hr light/dark cycle. This study was conducted in compliance with the guidelines of the 'guide for the care and use of laboratory animals' (26).

Experimental design and diets   Before the experiments, the rats had *ad libitum* access to water and a pelleted rodent diet (Samyang Co., Seoul, Korea) for the first 6 days (adaptation period). At the end of the adaptation period, the rats weighed 289.72±0.86 g. They were stratified according to the body weight, randomly blocked into 7 treatment groups and raised with experimental diets containing different HCA (Super CitriMax® HCA-600-SXS; InterHealth Nutraceuticals, Benicia, CA, USA) levels (0, 1.6, and 3.2%) and calorie sources (carbohydrate, fat) for 2 months. The diet sources were a normal-fat diet (C group), a normal-fat diet including HCA 0% (S group), 1.6% (SL group), 3.2% (SH group) powder with 30% sucrose solutions or a high-fat diet including HCA 0% (F group), 1.6% (FL group), 3.2% (FH group) powder with tap water. Sclafani A reported that when rats were fed a carbohydrate source, differed in type (glucose, sucrose, or polysaccharide) and form (32% solution, powder, or gel), sucrose solution group gained the most weight (49).

The specifications of HCA were analyzed and showed in Table 1. The composition of the experimental diets is shown in Table 2. Corn starch and casein were purchased

from Daesang Co. (Seoul, Korea) and Scerma Co. (Coudeville, France). All the other materials were purchased from Dyets Inc. (Bethlehem, PA, USA).

The rats were fed the modified American Institute of Nutrition (AIN)-93G Diet (Table 2) (27). The lipid sources in the high-fat diet were lard and soybean oil. Also each

amount of protein, fiber, minerals, and vitamins/total calorie in the high-fat diet was equalized to that of the AIN-93G normal-fat diet (28). The rats were allowed free access to the experimental diets and deionized water or 30% sucrose solution during the experimental period.

Specimen collection Body weight was recorded weekly. To determine the food intake, the amount of food offered was weighed and the weights of scraps and waste were recorded 3 times/week. The food efficiency ratio was calculated as followed; food efficiency ratio=body weight change for experimental period/food intake for experimental period (29). Blood samples were collected directly from the heart using syringes treated with heparin. They were centrifuged at $1,660 \times g$ for 30 min at 4°C and were frozen at −80°C. The liver was removed, weighed, and cut into small pieces, which were frozen in liquid nitrogen and stored at −80°C until analysis. Perirenal and epididymal fat pads were removed and weighed after sacrifying the animals.

Measurements of plasma and liver lipid concentration The total lipids in the plasma and liver were assayed by the sulfo-phospho-vanillin method (30,31). Plasma concentrations of triglyceride (TG), total cholesterols and high-density

Table 1. Specifications of *Garcinia cambogia* extract

Characteristics	*Garcinia cambogia*
Content of HCA	60%
(-) HCA (mg/g)	600±50
Calcium (mg/g)	110±30
Potassium (mg/g)	160±40
Sodium (mg/g)	Less than 10
Calories	2 kcal/g
Appearance	Amorphous powder
Color	Cream/white
Loss on drying	<8%
pH (1 g/100 mL water)	6.0-9.0
Solubility (water)	>99%
Heavy metals	<20 ppm
Total microbial count	<3,000 CFU/g

Table 2. Composition of experimental diets (g/kg diet)

Ingredient	Group[1]						
	C	S	SL	SH	F	FL	FH
Cornstarch	397.486	397.486	370.826	344.156	290.586	263.926	237.256
Dextrinized cornstarch	132.0	132.0	132.0	132.0	90.0	90.0	90.0
Sucrose	100.0	100.0	100.0	100.0	70.0	70.0	70.0
Lard	-	-	-	-	100.0	100.0	100.0
Soybean oil	70.0	70.0	70.0	70.0	100.0	100.0	100.0
Casein	200.0	200.0	200.0	200.0	230.0	230.0	230.0
Fiber	50.0	50.0	50.0	50.0	60.0	60.0	60.0
Mineral mix[2]	35.0	35.0	35.0	35.0	41.0	41.0	41.0
Vitamin mix[3]	10.0	10.0	10.0	10.0	12.0	12.0	12.0
L-Cystine	3.0	3.0	3.0	3.0	3.5	3.5	3.5
Choline bitartrate	2.5	2.5	2.5	2.5	2.9	2.9	2.9
Tert-butyl hydroquinone	0.014	0.014	0.014	0.014	0.014	0.014	0.014
Garcinia cambogia extract powder (60% HCA)	-	-	26.66 (16.00)	53.33 (32.00)	-	26.66 (16.00)	53.33 (32.00)
Total (g)	1,000	1,000	1,000	1,000	1,000	1,000	1,000
Energy content (kcal)	3,736.2	3,736.2	3,693.6	3,650.9	4,371.1	4,328.4	4,285.8
30% Sucrose solution	-	+	+	+	-	-	-

These diets were based on the AIN-93G diet composition.

[1]C, control diet group: 7%(w/w) fat diet; S, high carbohydrate diet: 7%(w/w) fat diet+30% sucrose solution; SL, high carbohydrate diet+ HCA 1.6%; SH, high carbohydrate diet+HCA 3.2%; F, high fat diet: 20%(w/w) fat diet; FL, high fat diet+HCA 1.6%; FH, high fat diet+ HCA 3.2%.
[2]Mineral mix (AIN-93G-MIX) (g/kg mixture): Anhydrous calcium carbonate, 357; monobasic potassium phosphate, 196; sodium chloride, 74; potassium sulfate, 46.6; potassium citrate, tripotassium, monohydrate, 70.78; magnesium oxide 24, ferric citrate, 6.06; zinc carbonate, 1.65; manganous carbonate, 0.63; cupric carbonate, 0.3; potassium iodate, 0.01; anhydrous sodium selenate, 0.01025; ammonium paramolybdate 4-hydrate, 0.000795; sodium meta-silicate, 9 -hydrate, 1.45; chromium potassium sulfate, 12-hydrate, 0.275; boric acid, 0.0815; sodium fluoride, 0.0635; nickel carbonate, 0.0318; lithium chloride, 0.0174; ammonium vanadate, 0.0066; powdered sucrose, 221.026.
[3]Vitamin mix (AIN-93-VX) (g/kg mixture): Nicotinic acid, 3; Ca-pantothenate 1.6; pyrodoxine-HCl, 0.7; thiamin-HCl, 0.6; riboflavin, 0.6; folic acid, 0.2; D-biotin, 0.02; vitamin B12 (cyonocobalamine) (0.1% in mannitol), 2.5; vitamin E (all-rac-α-tocopheryl acetate) (500 IU/g), 15; vitamin A (all-trans-retinyl palmitate) (500,000 IU/g), 0.8; vitamin D₃ (cholecalciferol) (400,000 IU/g), 0.25; vitamin K/destrose mix (10 mg/g), 7.5; powdered sucrose 967.23.

Table 3. Calorie intake, 30% sucrose solution intake, body weight gain, calorie efficiency ratio in rats fed diets containing hydroxycitric acid with high sucrose or fat level

Group[1]	Calorie intake (kcal/day)	30% sucrose solution intake (mL/day)	Weight gain (g/8 weeks)	Weight gain/calorie intake (g/100 kcal)	Calorie intake as carbohydrate (%)	Calorie intake as fat (%)
C	102.45±1.93[2)b3)]		261.79±7.51[ab]	4.55±0.16[b]	63.66±1.20[b]	16.86±0.32[b]
S	114.00±2.14[a]	34.52±1.49[a]	286.05±10.29[a]	4.45±0.12[b]	76.86±1.55[a]	10.73±0.30[c]
SL	98.74±1.62[bc]	24.52±0.88[b]	222.12±4.32[c]	4.01±0.10[c]	74.19±1.21[a]	11.97±0.29[c]
SH	99.39±1.26[bc]	27.82±1.35[b]	207.16±9.85[c]	3.70±0.15[c]	75.28±1.17[a]	11.46±0.28[c]
F	99.34±1.70[bc]		284.36±9.45[a]	5.08±0.12[a]	39.66±0.68[c]	41.17±0.70[a]
FL	94.88±2.30[c]		254.44±11.10[b]	4.76±0.16[ab]	39.07±0.95[c]	41.59±1.01[a]
FH	100.38±3.26[bc]		252.76±12.50[b]	4.48±0.17[b]	38.46±1.25[c]	42.00±1.36[a]

[1)]C, control diet group: 7%(w/w) fat diet; S, high carbohydrate diet: 7%(w/w) fat diet+30% sucrose solution; SL, high carbohydrate diet+ HCA 1.6%; SH, high carbohydrate diet+HCA 3.2%; F, high fat diet: 20%(w/w) fat diet; FL, high fat diet+HCA 1.6%; FH, high fat diet+ HCA 3.2%.
[2)]Mean±SE ($n=10$).
[3)]Values with different alphabet within the column are significantly different at $p<0.05$ level by Duncan's multiple-range test.

lipoprotein (HDL) cholesterol and hepatic concentrations of TG and total cholesterols were measured using the commercial kit from Asan Pharm. Co. (Seoul, Korea).

Hepatic enzyme activity Hepatic activity of ATP citrate lyase (EC 4.1.3.8) was determined as described elsewhere (32,33). The protein content was measured by the method of Lowry. ATP citrate lyase activity was measured using the hydroxamate assay (34). The amount of acetylhydroxamate generated was quantitated spectrophotometrically at 520 nm. The difference in absorbance between the reaction evaluated in the presence and absence of CoA (Sigma-Aldrich, St. Louis, MO, USA) determined the ATP citrate lyase specific generation of acetylhydroxamate (33).

Hepatic carnitine palmitoyl transferase (CPT) activity was measured using the method of Bieber and Markwell (35) and Markwell *et al.* (36). CPT activity was quantified by measuring the release of CoA-SH from palmitoyl-CoA (Sigma-Aldrich) using the general thiol reagent 5,5-dithio-bis (2-nitrobenzoic acid) (DTNB). The difference in absorbance at 412 between the rates with and without carnitine provided the carnitine-dependent rate for the formation of CoA, which reflects CPT activity (37). The protein content of the enzyme solution was determined according to the Lowry method.

Statistical analysis All results are expressed as the mean ±standard error (SE). The data were analyzed by the one-way analysis of variance (ANOVA) and the differences between experimental groups were evaluated using Duncan's multiple-range tests at the $p<0.05$ level.

Results and Discussion

Calorie intake and body weight change of rats Daily calorie intake, body weight gain, and calorie efficiency ratio (body weight change/100 kcal consumed) are shown in Table 3 and weekly body weight change is shown in Fig. 1. Daily calorie intake was significantly higher in the S group than that of the SL and SH groups but there was no significant difference in the HF groups. The body weight gain of the HCA groups significantly decreased and that of
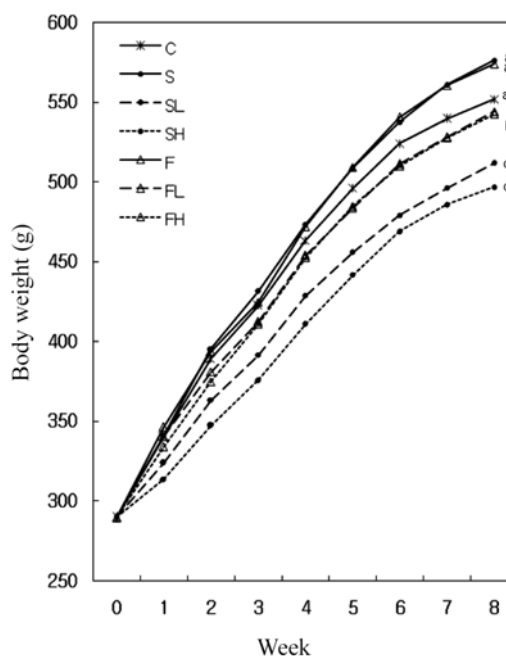

Fig. 1. Body weight changes during experimental period. Values with different letters are significantly different ($p<0.05$).

the SL and SH groups was significantly lower than that of the FL and FH groups, although there was no significant difference in energy intake of those groups. Calorie efficiency ratio in the S and F groups was significantly higher than that in the SL, SH, FL, and FH groups. HCA reduced body weight gain and adipose tissue weight compared with the control rats. This is similar to the reports on rats fed a HCA (1.8% of diet) with a 50% fructose diet after a 3 week period of food restriction (38). Soni *et al.* (39) also investigated the effects of HCA on body weight. That result showed that HCA-SX (Super CitriMax®) treatment resulted in a significant reduction in body weight at the end of 60 and 90 days. Asghar *et al.* (40) reported that when Zucker rats (5-week old) were supplemented with vehicle and super CitriMax (HCA-SX) in drinking water for 7 weeks, HCA-SX supplementation in obese rats reduced

Table 4. Adipose tissue weights in rats fed diets containing hydroxycitric acid with high sucrose or fat level (g/100 g BW)

Group[1]	Epididymal fat pad	Perirenal fat pad
C	2.84±0.15[ab2]	3.08±0.11[a]
S	3.19±0.17[a]	3.01±0.24[ab]
SL	2.46±0.13[b]	2.56±0.13[bc]
SH	2.42±0.17[b]	2.31±0.13[c]
F	3.22±0.16[a]	2.94±0.18[ab]
FL	3.13±0.12[a]	2.87±0.09[ab]
FH	2.91±0.13[a]	2.69±0.15[abc]

[1]C, control diet group: 7%(w/w) fat diet; S, high carbohydrate diet: 7%(w/w) fat diet+30% sucrose solution; SL, high carbohydrate diet +HCA 1.6%; SH, high carbohydrate diet+HCA 3.2%; F, high fat diet: 20%(w/w) fat diet; FL, high fat diet+HCA 1.6%; FH, high fat diet+HCA 3.2%.
[2]Mean±SE (*n*=10); Values with different alphabet within the column are significantly different at *p*<0.05 level by Duncan's multiple-range test.

body weight gain. Body weight gain of 6 and 10-14 week old rats was significantly reduced by oral administration of a nontoxic dose of (-)-hydroxycitrate (11 g/kg diet) during 13.6 weeks (12).

The weight of epididymal and perirenal fat pads is shown in Table 4. Epididymal fat pad in the SL and SH groups was decreased compared with the S group and was significantly lower than that of the FL and FH groups. Perirenal fat pad weight in the SL and SH groups was significantly lower than the C group. HCA-containing *G cambogia* has also shown to suppress body fat accumulation in experimental animals (5,11-13). Brandt *et al.* (38) reported that HCA (1.8% of diet) reduced visceral fat accumulation in rats that had *ad libitum* access to a 50% fructose diet during a 4 week period after they had a 3 week period of food restriction compared with control rats. According to Saito *et al.* (5), when diets containing different levels of HCA (0, 10, 51, 102, and 154 mmol/kg

diet) were fed to 6-week old rats for 92 or 93 days, the highest dose of HCA level (154 mmol/kg diet) showed significant decrease of epididymal fat accumulation in developing male rats, compared with the other groups. In this study HCA was effective in the reduction of body weight and body fat. Especially, a high carbohydrate diet with HCA was more effective than a high-fat diet with HCA in the reduction of body weight and body fat accumulation.

Plasma and liver lipid concentrations in rats Lipid concentrations in the plasma and liver are shown in Table 5. Plasma total lipid and triglyceride (TG) concentrations in SH group were significantly lower than S group. But HCA treatment did not affect the plasma total lipid, TG and total cholesterol levels in high-fat diet groups. The HDL-cholesterol concentration and HDL-cholesterol/total cholesterol ratio were not affected by HCA.

The level of hepatic total lipid in SH group was significantly higher than the C group. Total cholesterol concentrations in the SH and FH groups was significantly higher than the S and F groups.

The results of this study confirm previous data showing that high-sucrose diets can lead to an increase of TG level (41,42). As already mentioned, HCA decreased plasma total lipids and TG levels and increased the liver fat concentration in rats fed a high-carbohydrate diet. However groups of rats fed a high-fat diet did not show any significant differences from HCA. Sullivan *et al.* (22) reported that HCA reduced the fructose-induced increase in serum TG. Also, rats fed a high-glucose diet with HCA reduced the plasma TG level (43). Leonhardt and Langhans (44) demonstrated that in rats fed a 12% fat diet (76% carbohydrate, 9% protein, 12% fat) for 22 days, HCA reduced plasma triacylglycerol (C group 2.13±0.20 mmol/L, HCA group 1.63±0.20 mmol/L) and increased liver fat concentration (C group 5.8±0.2 g/100 g wet liver, HCA group 7.2±0.6 g/100 g wet liver). In another study, Brandt

Table 5. Plasma concentrations of total lipids (TL), triglyceride (TG), total cholesterol (T-C), HDL-cholesterol, and ratio of HDL-cholesterol/T-C and liver concentrations of TL, TG, and T-C in rats fed diets containing hydroxycitric acid with high sucrose or fat level

Group[1]	C	S	SL	SH	F	FL	FH
Plasma							
TL (mg/dL)	402.96±18.03[bcd2]	524.49±21.28[a]	465.58±20.07[ab]	411.46±22.62[bc]	342.51±25.32[de]	380.08±28.51[cde]	314.78±19.24[e]
TG (mg/dL)	243.26±18.56[bc]	357.86±28.03[a]	260.37±15.55[b]	205.65±19.95[bcd]	158.59±12.60[d]	186.61±28.04[cd]	142.29±18.14[d]
T-C (mg/dL)	104.72±8.14[ab]	114.79±8.48[a]	113.35±5.93[a]	110.18±9.69[ab]	93.71±6.88[ab]	98.93±7.12[ab]	87.22±5.82[b]
HDL-C (mg/dL)	49.25±2.25[NS3]	50.32±3.09	48.27±2.65	50.30±1.83	48.66±2.78	48.88±3.28	44.50±1.62
HDL-C/T-C	0.48±0.03[NS]	0.45±0.03	0.43±0.03	0.48±0.04	0.53±0.02	0.50±0.03	0.53±0.03
Liver (mg/g wet liver)							
TL	37.60±2.90[b]	39.68±3.81[ab]	44.92±4.95[ab]	53.74±6.62[a]	45.56±4.56[ab]	43.06±2.72[ab]	48.92±5.12[ab]
TG	14.67±1.43[NS]	15.42±1.08	17.12±1.72	17.15±1.35	17.59±0.89	17.90±1.00	16.39±1.12
T-C	1.30±0.07[cd]	1.18±0.05[d]	1.36±0.08[bcd]	1.53±0.07[b]	1.44±0.06[bc]	1.43±0.07[bc]	1.72±0.06[a]

TL=total lipids; TG=triglyceride; T-C=total-cholesterol; HDL-C=high-density lipoprotein cholesterol

[1]C, control diet group: 7%(w/w) fat diet; S, high carbohydrate diet: 7%(w/w) fat diet+30% sucrose solution; SL, high carbohydrate diet+ HCA 1.6%; SH, high carbohydrate diet+HCA 3.2%; F, high fat diet: 20%(w/w) fat diet; FL, high fat diet+HCA 1.6%; FH, high fat diet+ HCA 3.2%.
[2]Mean±SE (*n*=10); Values with different alphabet within the column are significantly different at *p*<0.05 level by Duncan's multiple-range test.
[3]Not significant at *p*<0.05 level by Duncan's multiple-range test.

Table 6. Hepatic ATP-citrate lyase and carnitine palmitoyl-transferase activities in rats fed diets containing hydroxycitric acid with high sucrose or fat level

Group[1]	ATP-citrate lyase (μmol hydroxamate/mg protein/30 min)	Carnitine palmitoyltransferase (nmol/mg protein/min)
C	0.26±0.06[NS2]	6.25±0.66 [NS]
S	0.28±0.05	6.22±0.56
SL	0.25±0.05	6.38±0.61
SH	0.17±0.03	6.58±0.71
F	0.20±0.06	7.04±0.53
FL	0.27±0.07	7.11±0.71
FH	0.27±0.07	6.95±0.58

[1]C, control diet group: 7%(w/w) fat diet; S, high carbohydrate diet: 7%(w/w) fat diet+30% sucrose solution; SL, high carbohydrate diet +HCA 1.6%; SH, high carbohydrate diet+HCA 3.2%; F, high fat diet: 20%(w/w) fat diet; FL, high fat diet+HCA 1.6%; FH, high fat diet+HCA 3.2%.
[2]Mean±SE (n=10); [NS]Not significant at $p<0.05$ level by Duncan's multiple-range test.

et al. (38) also showed that liver lipid content was significantly elevated in the HCA group compared with the control and pair-fed groups. On the other hand Chee *et al.* (45) reported that HCA had no effect on the plasma TG level in rats, but it increased the plasma TG and liver lipid contents in chickens. The fatty acids originating from hepatic *de novo* lipogenesis are secreted as TG in lipoproteins (mainly VLDL) into the circulation (15). A decrease in circulating TG levels with a concomitant increase in liver fat is usually caused by substances that markedly inhibit VLDL secretion (46). High dose of HCA-containing *G. cambogia* may increase hepatic lipid levels in HC diet groups. But adequate dose of HCA-containing *G. cambogia* may be useful for decreasing lipid levels. Further detailed mechanisms of HCA-containing *G. cambogia* on lipid levels in plasma and liver remain to be solved, especially substances that markedly inhibit VLDL secretion.

Hepatic enzyme activity in rats　　The activities of hepatic enzymes are shown in Table 6. The activities of hepatic ATP-citrate lyase and carnitine palmitoyl transferase (CPT) were not significantly influenced by HCA. But the activity of hepatic citrate lyase tended to be lower and CPT tended to be higher with increasing HCA level in the HC groups. Chee *et al.* (45) reported that pair-fed with HCA-treated free-feeding rats had significantly lower activities of ATP-citrate lyase and fatty acid synthetase in the liver. It has been suggested that because HCA inhibits the formation of acetyl-CoA in the cytoplasm, it may also inhibit the formation of the next compound, malonyl-CoA, in the pathway of fatty acid synthesis. Malonyl-CoA inhibits CPT, which is required for the oxidation of fat (39). Thus it has been suggested that HCA is proposed to decrease the concentration of malonyl-CoA, resulting in the suppression of body fat, the accumulation through stimulation of CPT activity and the promotion of fatty acid oxidation as a consequence of a high-carbohydrate diet (23,47,48). However our results showed that activities of hepatic CPT and ATP-citrate lyase did not show any significantly alterations among the groups although significant reduction

in body weight gain was noted.

In conclusion, this study showed effects of HCA-containing *G. cambogia* on body weight gain in high-fat and high-carbohydrate diet groups and on lipid profiles in rats fed a high-carbohydrate diet. Although further detailed mechanisms of HCA-containing *G. cambogia* on body weight remain to be solved, we suggest that HCA-containing *G. cambogia* may be used as a supplement to reduced body weight gain, especially in rats fed a high carbohydrate diet.

Acknowledgments

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