

## Effects of Capsaicin on Lipid Metabolism in Rats Fed a High Fat Diet<sup>1</sup>

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**ABSTRACT** Effects of capsaicin, a pungent principle of hot red pepper, were studied in experiments using male rats fed a diet containing 30% lard. Capsaicin was supplemented at 0.014% of the diet. The level of serum triglyceride was lower when capsaicin was present in the diet than when it was not. Levels of serum cholesterol and pre- $\beta$ -lipoprotein were not affected by the supplementation of capsaicin. The perirenal adipose tissue weight was lower when capsaicin was present in the diet than when it was not. Hepatic enzyme activities of glucose-6-phosphate dehydrogenase and adipose lipoprotein lipase were lower in rats fed the 30% lard diet than in those fed a nonpurified diet. Activities of these two enzymes were higher when capsaicin was added to the diet than when it was not. Hepatic acetyl-CoA carboxylase,  $\beta$ -hydroxyacyl-CoA dehydrogenase, and adipose hormone-sensitive lipase activities were not affected by capsaicin feeding. Lipid absorption was not affected by the supplementation of capsaicin. The perirenal adipose tissue weight and serum triglyceride were decreased as the level of capsaicin in the diet increased up to 0.021%. These results suggest that capsaicin stimulates lipid mobilization from adipose tissue and lowers the perirenal adipose tissue weight and serum triglyceride concentration in lard-fed rats. *J. Nutr.* 116: 1272-1278, 1986.

**INDEXING KEY WORDS** capsaicin • lipid metabolism • adipose tissue  
• serum triglyceride • high fat diet

Capsaicin is a pungent principle of hot red pepper that has been studied because of its importance in spices, food additives and drugs, which was recently reviewed by Suzuki and Iwai (1). The structure of capsaicin has been established as *N*-(4-hydroxy-3-methoxybenzyl)-8-methylnon-*trans*-6-enamide (2, 3). We have demonstrated that capsaicin is readily transported through the gastrointestinal tract and is absorbed via nonactive transport into the portal vein. Most of the absorbed capsaicin is excreted as metabolites via the urine within 48 h in rats (4, 5).

Recently, the effects of dietary components on lipid metabolism have received much attention. For example, vegetable proteins, dietary fiber and essential oils

appear to lower blood cholesterol (6-8). Recent studies have shown that capsaicin exerts a lipotropic effect similar to that of choline in rats (9, 10) and decreases total serum, myocardial and aortic cholesterol levels in turkeys (11).

The information about the effects of capsaicin on lipid metabolism is limited, and the mechanism of the effect of capsaicin is not clear. The present study investigates the effect of capsaicin on lipid metabolism in rats fed a high fat diet.

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## MATERIALS AND METHODS

**Animals and diets.** Wistar male rats (175–185 g body wt, Shizuoka Agricultural Cooperative Assoc. for Laboratory Animals, Hamamatsu, Japan) were individually housed in stainless steel wire-bottom cages in a room maintained at 22–24°C with about 50% relative humidity. The room was lighted from 0600 to 1800. Rats were fed each diet for 10 d. In all experiments, rats were fed a commercial nonpurified diet (MF, Oriental Yeast Co., Tokyo, Japan) (12) for 3 d to allow them to adjust to the new environment and then were starved for 1 d before starting experiments. Water was provided ad libitum.

The composition of experimental diets is given in table 1. A nonpurified diet (MF, Oriental Yeast Co., Tokyo, Japan) (12) was also fed to one group of rats. High fat diet contained only 10% casein, considered to be a low but adequate level since rats were adults. Capsaicin, the purity of which was determined as over 99% by the high-performance thin-layer chromatography method as described elsewhere (13), was purchased from E. Merck (Darmstadt, West Germany). Two experiments were conducted. In the first experiment, there were three dietary treatments: 1) nonpurified, 2) 30% lard, and 3) 30% lard plus 0.014% capsaicin. The dose of capsaicin used in this study was related to that usually ingested by rural Thai people (14). The same energy intake for the rats of three groups was maintained by adjusting the feed intake (about 15.4 g of nonpurified diet per rat, or about 10.5 g of the 30% lard or 30% lard plus 0.014% capsaicin diets per rat). The feed intake was higher in the nonpurified diet group than in the lard diets because of differences in energy density of the diets. The energy intakes of the three groups fed ad libitum were revealed in preliminary experiments. Rats were fed each diet for 10 d. There were 15 rats per group. In experiment 2, there were four dietary treatments: 1) 30% lard, 2) 30% lard plus 0.007% capsaicin, 3) 30% lard plus 0.014% capsaicin and 4) 30% lard plus 0.021% capsaicin. The additional amount of capsaicin in each experimental diet replaced starch as in experiment 1. The same energy intake for the rat of the four groups was maintained by adjusting the

TABLE 1

*Composition of experimental diets*<sup>1</sup>

Ingredients	High fat diet
	%
Casein <sup>2</sup>	10
Starch <sup>2</sup>	40
Sucrose <sup>2</sup>	10
Lard <sup>2</sup>	30
Soybean oil <sup>3</sup>	5
Mineral mixture <sup>4</sup>	2
Vitamin mixture <sup>5</sup>	1
Cellulose <sup>2</sup>	2
Capsaicin <sup>6</sup>	-

<sup>1</sup>Nonpurified diet was commercial (MF, Oriental Yeast Co., Tokyo, Japan) (12). <sup>2</sup>Oriental Yeast Co., Tokyo, Japan. <sup>3</sup>Wako Pure Chemical Ind., Osaka, Japan. <sup>4</sup>Supplied in milligrams/kilogram diet (except as noted): CaHPO<sub>4</sub> · 2H<sub>2</sub>O, 2.91 g; KH<sub>2</sub>PO<sub>4</sub>, 5.14 g; NaH<sub>2</sub>PO<sub>4</sub>, 1.87 g; NaCl, 0.93 g; calcium lactate, 7.02 g; iron citrate, 0.64 g; MgSO<sub>4</sub>, 1.43 g; ZnCO<sub>3</sub>, 22; MnSO<sub>4</sub> · 4-6H<sub>2</sub>O, 24; CuSO<sub>4</sub> · 5H<sub>2</sub>O, 6; KI, 2. <sup>5</sup>Supplied in milligrams/kilogram diet: thiamin · HCl, 12; riboflavin, 40; pyridoxine · HCl, 8; vitamin B-12, 0.005; ascorbic acid, 300; D-biotin, 0.2; menadione, 52; folic acid, 2; D-calcium pantothenate, 50; *p*-aminobenzoic acid, 50; nicotinic acid, 60; inositol, 60; choline chloride, 2000; all-*rac*- $\alpha$ -tocopheryl acetate, 50 and in IU/kilogram diet: retinyl acetate, 5000; cholecalciferol, 2500. <sup>6</sup>Added at 0.014% of diet in place of equal weight of starch in high fat plus capsaicin diet. Purchased from E. Merck, Darmstadt, West Germany.

feed intake (10.5 ± 0.2 g/d per rat in each group, mean ± SD). Thirty rats were used in this experiment.

**Sample collection.** At the end of experiments, rats were fasted for 16 h and lightly anesthetized with pentobarbital. Blood was collected by heart puncture for analyses of serum lipids, glucose, ketone bodies and lipoprotein. Immediately after blood sampling, the liver was excised, washed in chilled saline solution, blotted and cooled on crushed ice. A part of the liver was used for the assay of enzyme activities and the rest of the liver was kept at -30°C until analysis of lipid components. Perirenal adipose tissue was excised, rinsed, blotted and weighed. A part of adipose tissue was used for the enzyme assay and the measurement of adipose cell weight. The intestinal absorption rate of dietary lipid was monitored for 3 d, from d 5–7 of experiment 1, and determined by the method of balance experiments

(15). The fecal samples were dried, weighed and pulverized. Fecal lipids were extracted by the method of Folch et al. (21).

**Chemical assay.** Serum triglyceride was analyzed enzymically using a commercially available kit (Triglyceride G-Test Wako, Wako Chem. Ind., Osaka, Japan). Serum ketone bodies were also assayed enzymically by the method of Williamson et al. (16). Serum free fatty acid and cholesterol were measured according to the method of Itaya and Ui (17), and Pearson et al. (18), respectively. The analysis of serum lipoprotein was performed according to the method of Narayan et al. (19) as modified by Maruyama and Kobori (20). Liver lipids were extracted by the method of Folch et al. (21). Liver total lipids were determined gravimetrically. Liver triglyceride, free fatty acid and cholesterol were measured as described above for serum. DNA of perirenal adipose tissue was determined by the method of Leyva and Kelley (22).

**Enzyme assay.** Acetyl-CoA carboxylase (ACC; EC 6.4.1.2), glucose-6-phosphate de-

hydrogenase (G-6-PDH; EC 1.1.1.49) and  $\beta$ -hydroxyacyl-CoA dehydrogenase (HADH; EC 1.1.1.35) in the liver were assayed by the methods of Nakanishi and Numa (23), Kornberg and Horecker (24) and Osumi and Hashimoto (25), respectively. Hormone-sensitive lipase (HSL) and lipoprotein lipase (LPL; EC 3.1.1.34) activities of the perirenal adipose tissue were estimated as described by Rizack (26) and Salaman and Robinson (27), respectively.

**Statistical analysis.** In experiment 1, Student's *t*-test was used for statistical evaluation of the two means. Populations were tested using analysis of variance. When the *F*-test was significant at  $P < 0.05$ , comparison was made using Aspin-Welch method (28). In experiment 2, group means were tested by using standard one-way analysis of variance techniques (28) and Duncan's Multiple-Range test (29). These calculations were performed on a NEC PC-8801 mkII microcomputer (NEC Co., Tokyo, Japan) using N<sub>88</sub>-BASIC program (30).

TABLE 2  
Effects of capsaicin in rats fed high fat diets with and without capsaicin (experiment 1)<sup>1</sup>

	Dietary treatments			P-value, high fat vs. high fat plus capsaicin <sup>2</sup>
	Nonpurified	High fat	High fat plus 0.014% capsaicin	
Calorie intake, cal/10 d	549.0 ± 0.0	547.2 ± 0.7	548.6 ± 1.3	NS <sup>3</sup>
Gain in body weight, g/10 d	35.7 ± 1.6	18.8 ± 0.7	18.5 ± 1.6	NS
Liver weight, g	5.30 ± 0.13	5.29 ± 0.17	5.70 ± 0.09	NS
Epididymal fat pad weight, g	1.16 ± 0.07	2.22 ± 0.14	1.93 ± 0.09	NS
Perirenal fat pad weight, g	0.36 ± 0.06	2.23 ± 0.14	1.69 ± 0.13	< 0.01
Perirenal cell weight, mg/ $\mu$ g DNA	43.6 ± 0.1	43.8 ± 0.1	44.2 ± 0.3	NS
Fat absorption rate, %	99.6 ± 0.0	98.4 ± 0.1	98.9 ± 0.1	NS
Serum				
Triglyceride, mg/dl	26.1 ± 1.4	41.7 ± 4.4	29.3 ± 3.4	< 0.05
Free fatty acid, mg/dl	6.30 ± 0.27	6.51 ± 0.31	6.45 ± 0.46	NS
Ketone bodies, $\mu$ mol/dl	98.7 ± 3.3	141.5 ± 4.1	147.9 ± 4.0	NS
Cholesterol, mg/dl	104.9 ± 2.8	140.3 ± 12.1	140.2 ± 5.7	NS
Glucose, mg/dl	153.7 ± 8.0	103.0 ± 5.7	118.9 ± 6.5	NS
(Pre- $\beta$ + $\beta$ )/ $\alpha$ lipoprotein <sup>4</sup>	0.39 ± 0.02	0.40 ± 0.01	0.38 ± 0.01	NS
Liver				
Total lipid, mg/g liver	56.6 ± 1.8	82.5 ± 3.5	77.7 ± 2.7	NS
Triglyceride, mg/g liver	9.20 ± 0.44	27.8 ± 1.7	24.4 ± 1.1	NS
Free fatty acid, mg/g liver	3.95 ± 0.18	4.35 ± 0.08	4.34 ± 0.09	NS
Cholesterol, mg/g liver	3.73 ± 0.06	4.58 ± 0.07	4.85 ± 0.17	NS

<sup>1</sup>Mean  $\pm$  SEM of 15 rats. In Materials and Methods.

<sup>2</sup>The statistical significance of differences was calculated by the methods described in Materials and Methods.

<sup>3</sup>NS, not significant. <sup>4</sup>(Pre- $\beta$  lipoprotein +  $\beta$  lipoprotein)/ $\alpha$  lipoprotein.

## RESULTS

*Experiment 1.* Tables 2 and 3 summarized the results of experiment 1. There were no differences in gain of body weight or liver weight due to diet. Capsaicin supplementation tended to reduce epididymal adipose tissue weight and significantly reduced perirenal adipose tissue weight ( $P < 0.01$ ). The perirenal adipose cell weight was not affected by the supplementation of capsaicin. The lipid from the diets was well absorbed in each group, and the absorption rate was not affected by the supplementation of capsaicin. Serum triglyceride was lower in rats fed the purified diet containing capsaicin than in rats fed the same diet without capsaicin. There was no difference in serum triglyceride between the group fed the non-purified diet and the groups fed the high fat plus capsaicin diet. Serum free fatty acid was not altered by dietary treatment. Serum pre- $\beta$ -lipoprotein was not different due to diet. Liver total lipid, triglyceride and cholesterol in high fat diet groups were higher than in the nonpurified diet groups.

Activity of liver acetyl-CoA carboxylase, the rate-limiting enzyme of fatty acid synthesis (31), was lower in rats fed the high fat diet than in those fed the nonpurified diet. Liver G-6-PDH activity was significantly lower in rats fed the high fat diet without capsaicin than in those fed the nonpurified

diet. G-6-PDH activity was higher in rats fed capsaicin than in those fed the same diet without capsaicin ( $P < 0.02$ ). The liver HADH activity was higher in rats fed the two high fat diets than in rats fed the non-purified diet. The supplementation of capsaicin to the high fat diet did not influence enzyme activities of liver ACC and HADH, and perirenal adipose HSL but increased liver G-6-PDH and perirenal LPL activities.

*Experiment 2.* Since it has been found in experiment 1 that the supplementation of capsaicin affects the perirenal adipose tissue weight and serum triglyceride concentration, the relationship between the supplementation of capsaicin and metabolic parameters was investigated. Body weight gain was not significantly different among the four groups. There was a significant correlation between perirenal adipose tissue weight (% of body wt) and the dose-amount of capsaicin ( $r = -0.738$ ,  $P < 0.001$ ) (fig. 1). A similar relationship was also observed between the serum triglyceride concentration and the dose-amount of capsaicin ( $r = -0.444$ ,  $P < 0.02$ ) (fig. 2).

## DISCUSSION

Rats fed the nonpurified diet gained almost twice as much weight as those fed the lard diet. The ratio of protein to energy in the nonpurified diet was 3.5 times that in

TABLE 3

*Effects of capsaicin administration on various enzyme activities in rat liver and perirenal adipose tissue<sup>1</sup>*

	Nonpurified	High fat	High fat plus 0.014% capsaicin	<i>P</i> -value, high fat vs. high fat plus capsaicin <sup>2</sup>
<b>Liver</b>				
Acetyl-CoA carboxylase, <i>mU/g liver</i>	199.0 $\pm$ 14.9	74.7 $\pm$ 5.0	77.4 $\pm$ 10.2	NS <sup>3</sup>
Glucose-6-phosphate dehydrogenase, <i>U/g liver</i>	1.04 $\pm$ 0.63	0.76 $\pm$ 0.1	1.09 $\pm$ 0.05	< 0.02
$\beta$ -Hydroxyacyl-CoA dehydrogenase, <i>mU/g liver</i>	39.7 $\pm$ 2.9	53.0 $\pm$ 1.8	53.5 $\pm$ 2.9	NS
<b>Perirenal adipose tissue</b>				
Hormone-sensitive lipase, <i>free fatty acid <math>\mu</math>mol/<math>\mu</math>g DNA per h</i>	3.22 $\pm$ 0.25	4.92 $\pm$ 0.17	4.45 $\pm$ 0.16	NS
Lipoprotein lipase, <i>free fatty acid <math>\mu</math>mol/<math>\mu</math>g DNA per h</i>	23.0 $\pm$ 1.4	20.5 $\pm$ 0.3	25.5 $\pm$ 0.7	< 0.01

<sup>1</sup>Mean  $\pm$  SEM of 10 rats.  
in Materials and Methods.

<sup>2</sup>The statistical significance of differences was calculated by the methods described  
<sup>3</sup>NS, not significant.

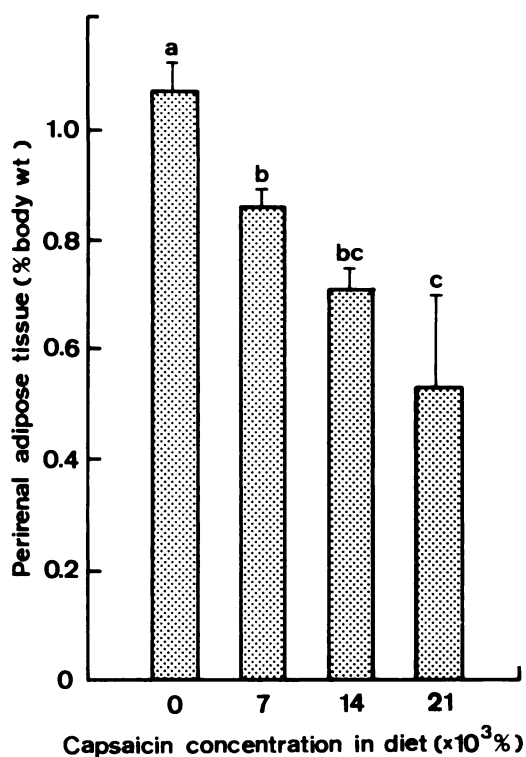


Fig. 1 Dose-response of capsaicin on perirenal adipose tissue weight. Values are means  $\pm$  SEM for individual values from 12 (0% capsaicin concentration in diet), 4 (0.007%), 10 (0.014%) or 4 (0.021%) rats. Means not sharing a common superscript letter are significantly different at  $P < 0.05$ .

the lard diet. Therefore, the rats fed the nonpurified diet might be so much more efficient in using the energy consumed, and the energy might be deposited as muscle.

The results of the present study showed that when similar amounts of diet were consumed the weight of perirenal adipose tissue and the serum triglyceride concentration were lower in rats fed the diet containing capsaicin than in those fed the diet without capsaicin. This effect on serum triglyceride concentration was not in conflict with the results reported by Sambaiiah and Satyanarayana (32) in rats fed ad libitum a diet containing 10% fructose and 0.015% capsaicin for 6 wk.

Perirenal fat pad weights were different in the three groups. On the other hand, the cell weights were similar in the three groups. These results suggest that existing perirenal cells are filled up with lipid (similar weight)

before additional perirenal cells are produced (fat pad weight gain).

It was reported that the absorption of lipid in rat intestine is inhibited by capsaicin (33). However, in the present study the gastrointestinal absorption of lipid was not inhibited by capsaicin as shown by the balance study method. Similar results were also obtained by Sambaiiah et al. (9). The data in table 2 indicate that the decrease of lipid accumulation in adipose tissue is not due to the decrease of gastrointestinal absorption of lipid in rats fed the high fat plus capsaicin diet.

The previous study using turkeys fed a high cholesterol diet for 23 d showed that capsaicin decreased total serum cholesterol (11), but there was no influence of capsaicin on the serum cholesterol in the present study using rats fed a diet without supplemental cholesterol for 10 d. The discrepant results may be due to differences in cholesterol intake or in animal species.

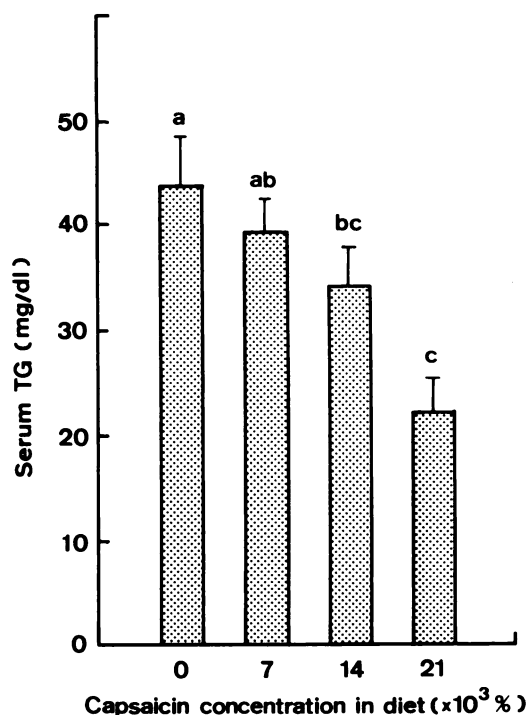


Fig. 2 Dose-response of capsaicin on serum triglyceride concentration. Values are means  $\pm$  SEM for individual values from 12 (0% capsaicin concentration in diet), 4 (0.007%), 10 (0.014%) or 4 (0.021%) rats. Means not sharing a common superscript letter are significantly different at  $P < 0.05$ .

It has been shown that the ingestion of fat markedly inhibits hepatic lipogenesis (34, 35). In the present experiments, ACC (a rate-limiting enzyme of fatty acid synthesis) (31) and G-6-PDH activities in rats fed the high fat diet were also lower than in rats fed the nonpurified diet. However, as seen in table 3, activities of both lipogenic enzymes were not inhibited by capsaicin, a result in opposition to that in fructose-fed rats (32). The mechanism of action of capsaicin on lipid metabolism in rats fed fructose is therefore different from that of rats fed lard. Serum triglyceride concentration of rats fed the high fat plus capsaicin diet was inversely related to the capsaicin level of the diet. Consequently, these results indicate that consumption of capsaicin facilitates lipid metabolism in rats fed a high fat diet. The stimulation of lipid metabolism by capsaicin is supported by the results obtained in perirenal adipose tissue weight, serum triglyceride concentration and adipose LPL activity.

There was no difference in serum ketone bodies, pre- $\beta$ -lipoprotein and liver lipids between high fat and high fat plus capsaicin groups. These results suggest that the surplus energy of the nutrients consumed with or without capsaicin accumulates in adipose tissue as lipid (triglyceride). Previous studies have shown that a free fatty acid was incorporated into an organ in proportion to the concentration of the fatty acid outside the organ (36, 37), and the rate of oxidation of that fatty acid automatically increased in the organ as its concentration increased (38, 39). Thus, it is possible that stimulation of lipid metabolism by capsaicin is attributable to fat mobilization from adipose tissue. However, its stimulation might not be accompanied by the induction of lipase, because HSL activity was not different between high fat and high fat plus capsaicin groups (table 3). Detailed study on the stimulation mechanism of capsaicin on lipid metabolism is now in progress.

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