Comparative Changes of Lipogenic-Related Enzyme Activities by Dietary Glycerol Tricaprylate, Tricaprate, Trilaurate and Trioleate in Rat Liver and Adipose Tissues

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Abstract The present study was undertaken to investigate the effects of the carbon chain length of dietary medium chain triacylglycerols on lipogenic-related enzyme activities in growing rats. Twenty-four Wistar strain male rats (8 weeks old) were allotted to 4 groups and fed the diets containing glycerol tri-homogeneous fatty acid (260 g/kg diet) composed of only caprylic acid (C8 : 0), capric acid (C10 : 0), lauric acid (C12 : 0) or oleic acid (C18 : 1). After four weeks, the activities of three lipogenic-related enzymes (glucose-6-phosphate dehydrogenase, G-6-PDH; citrate cleavage enzyme, CCE; and malic enzyme, ME) in liver and two adipose depots (perirenal and epididymal) were measured. Feeding of C8 : 0 diet resulted in significantly higher G-6-PDH and CCE activities in the liver than C10 : 0 feeding. In perirenal adipose tissue, G-6-PDH and ME activities were higher in C8 : 0 group than those in the other three groups. The activity of CCE in this adipose depot was lower in C8 : 0 group than in C10 : 0 and C12 : 0 groups. In epididymal adipose tissue, activities of G-6-PDH and ME were lower in C8 : 0 group than in C12 : 0 group. Fatty acid composition of perirenal adipose tissue demonstrated that significant amount of dietary C10 : 0 and C12 : 0 fatty acids was directly incorporated into adipose tissue, whereas C8 : 0 deposition was too small to be detected. These results suggest that dietary C8 : 0, C10 : 0 and C12 : 0 fatty acids differently affect lipogenic-related enzyme activity in both liver and adipose tissues in rats.

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Key words : Medium chain triacylglycerol, Lipogenic-related enzyme activity, Liver, adipose tissue, Rat

A number of studies have been undertaken to investigate the effects of dietary medium chain triacylglycerols (MCTs) on lipid metabolism. It has been reported that, dietary MCTs reduce serum cholesterol and serum triacylglycerol level^{21,25)} and fat accumulation in adipose tissue^{5,13)} than dietary long chain triacylglycerols (LCTs).

It has been demonstrated that dietary triacylglycerols suppress lipogenic and lipogenic-related enzyme activities in liver^{3,5)} and adipose tissue¹³⁾ whereas MCT is less effective than LCT²⁵⁾. Medium chain triacylglycerols which were used in these studies contained various medium chain fatty acids (MCFAs), such as caprylic acid (C8 : 0), capric acid (C10 :

0) and lauric acid (C12:0). It is still not known whether C8:0, C10:0 and C12:0 fatty acids identically affect lipogenic-related enzyme activities or not. The present study was undertaken to investigate the influence of the carbon chain length of dietary MCT on lipogenic-related enzyme activities in liver and adipose tissues by feeding each glycerol tri-homogeneous MCFAs in rats.

Materials and Methods

Animals and diets

Twenty-four Wistar strain male rats, aged 5 weeks, were purchased from SLC, Inc. (Hamamatsu, Japan) and fed the commercial laboratory chow for 3 weeks to reach 270 g of body weight. Through the experiment, room temperature and humidity were maintained at 24°C and 60%, respectively, with a 12 hours light-dark cycle. Then animals were randomly divided into 4 groups and fed the experimental diets for 4 weeks, which contained MCT or LCT (54% of total energy) composed of C8:0, C10:0, C12:0 or oleic acid (C18:1), respectively (Table 1). Such MCT and LCT contained more than 97% of synthesized glycerol tri-homogeneous fatty acid and were generously supplied by Kao Co. (Tokyo, Japan). Commercial corn oil was supplemented in each experimental diet (20 g/kg diet) for the source of essential fatty acids. All diets contained 22.3 kJ/g of gross energy and 54.2% of dietary energy was derived from fat, 27.2% was from protein and 18.6% was from carbohydrate. As a result, C18:1 diet contained less content of triacylglycerol compared with the other diets, when expressed as weight basis. Each animal was housed in an individual stainless steel wire cage and fed each experimental diet to take same amount of energy with free access to tap water.

Enzyme assay

Food deprivation was carried out for 15 hours before slaughter. After the animals were killed by exsanguination from the ab-

Table 1. Composition of experimental diets

Ingredients	C 8, C 10, C 12		C 18 : 1
	g/100 g		
Casein	21.0		21.0
DL-Methionine	0.3		0.3
Corn starch	10.0		10.0
Sucrose	10.0		10.0
Cellulose powder	25.0		27.0
Corn oil ¹	2.0		2.0
Mineral mix ²	4.0		4.0
Vitamin mix ³	1.5		1.5
Choline chloride	0.2		0.2
Triacylglycerol ⁴	26.0		24.0
	Source of energy (%)		
Fat	54.2		54.2
Protein	27.2		27.2
Carbohydrates	18.6		18.6

¹ Commercial corn oil. ^{2,3} AIN-76 mineral and vitamin mixtures (Oriental Yeast Co., Tokyo, Japan). ⁴ Each triacylglycerol contained more than 97% of synthesized triacylglycerols (Kao Co., Tokyo, Japan).

dominal aorta under pentobarbital anesthetization, liver, and perirenal and epididymal adipose tissues were collected and washed in ice-cold physiological saline. After weighing each tissue, 4 g of the liver or 2 g of the adipose tissue was homogenized in ice-cold 0.25 M sucrose/1 mM ethylenediaminetetraacetate buffer (pH=7.4) using a glass-Teflon homogenizer. Homogenate was centrifuged at 9800 rpm at 4°C for 15 minutes. Clear intermediate water-soluble phase was centrifuged again at 41000rpm at 4°C for 60 minutes. The supernatant was preserved at -80°C until assay for enzyme activities. Glucose-6-phosphate dehydrogenase (G-6-PDH ; EC 1.1.1.49) activity was assayed by the method of Löhr and Waller¹⁶⁾, citrate cleavage enzyme (CCE ; EC 4.1.3.8) activity by the method of Takeda *et al.*²³⁾, and malic enzyme (ME ; EC 1.1.1.40) activity by the method of Ochoa¹⁸⁾. Enzyme activities were expressed as rates of disappearance of substrate (n mol/min) per mg of protein. Protein content in the supernatant was determined by the method of

Lowry *et al.*¹⁷⁾.

Fatty acid composition

Total lipids of perirenal adipose tissue were extracted by the procedure of Folch *et al.*⁶⁾. Methyl-esterification of fatty acid was carried out by the procedure of Stoffel *et al.*²⁰⁾. The fatty acid composition of methyl esters was determined by gas liquid chromatography (Yanaco G.C. 180, Yanagimoto Seisakusho Co., Kyoto, Japan) using a glass column (4 m × 2 mm i.d.) packed with 5% Shinchrom E 71 on 80–100 mesh Shimalite AW (Shinwa Chemical Industries Ltd., Kyoto, Japan). A carrier gas was nitrogen. Injection temperature was maintained at 230°C and the oven temperature was programmed to increase from 160 to 240°C by 2°C/min. Fatty acids were identified by retention time comparison with authentic fatty acid methyl ester standards.

Statistical analysis

Statistical analysis was performed by one-way analysis of variance and differences between dietary groups were determined by Fisher's least significant difference test at $p < 0.05$ using GLM procedure of SAS¹⁹⁾. Differences in fatty acid composition between dietary groups were tested after arcsin transformation.

Results

Body weight, food intake and final liver weight are shown in Table 2. Final body weight was larger in C10:0 group than in C12:0 and C18:1 groups. Final liver weight

was significantly ($p < 0.05$) larger in C10:0 group than other three groups.

Weights of perirenal and epididymal adipose tissues are shown in Fig. 1. In both depots, there was no significant difference in tissue weight among the groups of C8:0–C12:0. Perirenal adipose tissue weight was significantly ($p < 0.05$) lower in C8:0 group than in C18:1 group. Epididymal adipose tissue weight was significantly ($p < 0.05$) lower in C8:0 and C12:0 groups than in C18:1 group but the difference between C10:0 and C18:1 group was not significant in this depot.

Lipogenic-related enzyme activities in liver are shown in Table 3. The activities of G-6-

Fig. 1. Weights of perirenal (open bars) and epididymal (hatched bars) adipose tissue in rats fed the different glycerol tri-homogeneous medium- or long-chain fatty acids.

Each bar represents mean for 6 rats.

a, b: Different letters within the same anatomical site denote significant differences ($p < 0.05$).

Table 2. Body weight, food intake and final liver weight in rats fed the different glycerol tri-homogeneous medium- or long-chain fatty acids

	Diets			
	C 8 : 0	C 10 : 0	C 12 : 0	C 18 : 1
Initial body weight (g)	274 ± 14	274 ± 14	274 ± 14	274 ± 12
Final body weight (g)	327 ± 5 ^{ab}	330 ± 5 ^a	315 ± 5 ^b	316 ± 5 ^b
Food intake (g/28 d)	463 ± 6 ^a	456 ± 6 ^{ab}	426 ± 6 ^c	443 ± 6 ^b
Final liver weight (g)	8.4 ± 0.3 ^b	9.3 ± 0.3 ^a	8.4 ± 0.3 ^b	7.9 ± 0.3 ^b

a, b, c: Values in the same row with different superscripts are significantly different ($p < 0.05$).

Dietary MCT and Lipogenic-Related Enzyme Activity

PDH, CCE and ME in C8 : 0 group was similar to those in C18 : 1 group. The activities of all three enzyme were significantly ($p < 0.05$) lower in C10 : 0 and C12 : 0 groups than in C18 : 1 group. The activity of G-6-PDH was significantly ($p < 0.05$) higher in C8 : 0 group than in C10 : 0 group but the difference between C8 : 0 and C12 : 0 group was not statistically significant. The activity of CCE was significantly ($p < 0.05$) lower in both C10 : 0 and C12 : 0 groups than in C8 : 0 group. The activity of ME was remarkably lower in C12 : 0 group than in C8 : 0 and C10 : 0 groups ($p < 0.05$).

In perirenal adipose tissue, G-6-PDH and ME activities were higher ($p < 0.05$) in C8 : 0 group than in C10 : 0 and C12 : 0 groups (Table 4).

Dietary C18 : 1 markedly suppressed ME activity in this adipose depot. The activity of CCE was significantly ($p < 0.05$) lower in C8 : 0 group than in C10 : 0 and C12 : 0 groups. The activity of this enzyme in C18 : 1 group was almost similar to C8 : 0 group.

In every group, the lipogenic-related enzyme activities were drastically lower in epididymal adipose tissue compared with perirenal adipose tissue (Table 4). The activity of G-6-PDH in epididymal adipose tissue was lower in C8 : 0 group than in C10 : 0 and C12 : 0 groups and was not different between C18 : 1 and C8 : 0 groups. The activity of ME was higher in C12 : 0 group than C8 : 0 group. On the other hand, no difference was found in CCE activities among all groups. The activities of G-6-PDH

Table 3. Lipogenic-related enzyme activities in the fasting state in liver of rats fed the different glycerol tri-homogeneous medium- or long-chain fatty acids for four weeks

	Diets			
	C8 : 0	C10 : 0	C12 : 0	C18 : 1
	(nmol/min/mg protein)			
G-6-PDH	15.3 $\pm$ 2.3 ^{bc}	8.0 $\pm$ 2.5 ^a	8.9 $\pm$ 2.3 ^{ab}	15.9 $\pm$ 2.3 ^c
CCE	8.0 $\pm$ 0.7 ^b	5.7 $\pm$ 0.7 ^a	5.3 $\pm$ 0.7 ^a	7.6 $\pm$ 0.7 ^b
ME	115 $\pm$ 10 ^{bc}	96 $\pm$ 10 ^b	51 $\pm$ 10 ^a	128 $\pm$ 10 ^c

^{a,b,c} : Values in the same row with different superscripts are significantly different ($p < 0.05$).

G-6-PDH, glucose-6-phosphate dehydrogenase ; CCE, citrate cleavage enzyme ; ME, malic enzyme.

Table 4. Lipogenic-related enzyme activities in the fasting state in perirenal and epididymal adipose tissue of rats fed the different glycerol tri-homogeneous medium- or long-chain fatty acids for four weeks

	Diets			
	C8 : 0	C10 : 0	C12 : 0	C18 : 1
	(nmol/min/mg protein)			
	Perirenal			
G-6-PDH	103.4 $\pm$ 9.8 ^b	69.9 $\pm$ 9.8 ^a	56.5 $\pm$ 9.8 ^a	46.6 $\pm$ 10.7 ^a
CCE	12.3 $\pm$ 2.0 ^a	18.6 $\pm$ 2.0 ^b	25.1 $\pm$ 2.0 ^c	14.4 $\pm$ 2.2 ^{ab}
ME	175 $\pm$ 13 ^d	118 $\pm$ 13 ^c	77 $\pm$ 13 ^b	31 $\pm$ 14 ^a
	Epididymal			
G-6-PDH	24.4 $\pm$ 5.3 ^a	43.5 $\pm$ 5.3 ^b	46.1 $\pm$ 5.3 ^b	26.3 $\pm$ 5.3 ^a
CCE	12.2 $\pm$ 1.8	12.8 $\pm$ 1.8	14.1 $\pm$ 1.8	10.4 $\pm$ 1.8
ME	29 $\pm$ 4 ^b	33 $\pm$ 4 ^{bc}	40 $\pm$ 4 ^c	18 $\pm$ 4 ^a

^{a,b,c,d} : Values in the same row with different superscripts are significantly different ($p < 0.05$).

G-6-PDH, glucose-6-phosphate dehydrogenase ; CCE, citrate cleavage enzyme ; ME, malic enzyme.

and ME were not different between the rats fed the C10:0 and C12:0 diets. The activity of ME was lower in C18:1 group than in any of MCT-fed groups.

As shown in Table 5, C8:0 content in perirenal adipose tissue was too small to be determined in C8:0-fed rats in spite of its high dietary level. On the other hand, C10:0 accounted for approximately 10% of total fatty acids in the adipose tissue of C10:0-fed rats. In rats fed the C12:0 diet, C12:0 content was remarkably high (18.2%). The content of palmitic acid, which is the major end product of biosynthesis of fatty acid, was higher in C8:0 group than in C10:0 and C12:0 groups. In addition, the contents of palmitoleic, oleic and

linoleic acids were significantly ($p < 0.05$) higher in C8:0 group than in C12:0 group. The rats fed C10:0 showed intermediate levels of the contents of these long-chain fatty acids between C8:0- and C12:0-fed rats. Oleic acid content was drastically high in C18:1 group. In addition, eicosamonoenoic acid content of C18:1 group was significantly ($p < 0.05$) higher than that of any other group.

Discussion

It has been well known that dietary fat inhibits hepatic and adipose tissue lipogenesis⁹. Acetate incorporation into fatty acids was significantly inhibited in liver slice from the rats fed corn oil, vegetable oil, hydrogenated

Table 5. Fatty acid composition of perirenal adipose tissue in rats fed the different glycerol tri-homogeneous medium- or long-chain fatty acids

	Diets			
	C8:0	C10:0	C12:0	C18:1
	g/100 g fatty acids			
	Tr	Tr	Tr	Tr
C8:0				
C10:0	1.4±0.2 ^b	9.8±0.4 ^c	1.2±0.1 ^b	0.1±0.0 ^a
C12:0	1.4±0.1 ^{ab}	1.9±0.0 ^b	18.2±0.9 ^c	0.6±0.2 ^a
C14:0	5.8±0.3 ^{bc}	5.5±0.2 ^b	7.0±0.3 ^c	3.1±0.2 ^a
C14:1(n-7)	0.6±0.1	0.4±0.1	0.6±0.0	0.1±0.0
C15:0	0.8±0.1 ^b	1.0±0.1 ^c	0.9±0.0 ^{bc}	0.6±0.1 ^a
C16:0	23.4±0.6 ^c	20.6±0.5 ^b	16.9±0.4 ^a	15.8±0.7 ^a
C16:1(n-7)	11.2±0.7 ^c	8.7±0.4 ^{ab}	7.7±0.2 ^a	9.5±0.4 ^b
C17:0	0.4±0.2 ^a	0.9±0.0 ^c	0.7±0.0 ^b	0.9±0.1 ^c
IsoC18:0	1.0±0.1 ^a	1.0±0.5 ^a	0.8±0.1 ^a	2.0±0.2 ^b
C18:0	7.7±0.3 ^b	8.4±0.5 ^b	5.7±0.2 ^a	6.5±0.2 ^a
C18:1(n-9)	24.2±0.5 ^b	20.1±0.4 ^a	17.9±0.5 ^a	40.2±1.6 ^c
C18:2(n-6)	17.3±0.7 ^b	16.9±0.4 ^b	15.1±0.5 ^a	16.0±0.3 ^{ab}
C18:3(n-3)	1.1±0.3 ^b	1.3±0.6 ^b	0.8±0.2 ^b	0.1±0.0 ^a
C20:0	0.5±0.1	0.5±0.1	0.3±0.0	0.3±0.1
C20:1(n-9)	1.6±0.4 ^a	1.8±0.8 ^a	2.2±0.2 ^a	4.1±0.2 ^b
C20:4(n-6)	0.3±0.1 ^{ab}	0.5±0.1 ^b	0.2±0.0 ^a	0.2±0.1 ^a
SFA	42.3±0.7 ^b	49.4±0.6 ^c	51.2±1.0 ^c	31.3±0.2 ^a
MUFA	37.8±0.7 ^b	31.0±1.1 ^a	28.7±0.6 ^a	53.9±1.1 ^c
PUFA	18.2±0.6 ^b	18.4±0.9 ^b	16.4±0.9 ^a	16.3±0.3 ^a
SFA/PUFA	0.75±0.03 ^b	0.94±0.01 ^c	1.08±0.01 ^d	0.45±0.01 ^a

^{a, b, c, d}: Values in the same row with different superscripts are significantly different ($p < 0.05$).

Tr, trace; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid.

vegetable oil or lard⁹⁾. Leveille¹⁵⁾ showed that incorporation of acetate or glucose into fatty acids *in vitro* was reduced in the epididymal adipose tissue of rats as dietary level of hydrogenated vegetable oil was increased from 10% to 30%. Furthermore, it has been suggested that, the inhibition of lipogenesis by dietary fat is not due to the reduction of carbohydrate intake²⁴⁾. In these studies, dietary fat was mainly composed of LCTs. On the other hand, it has been reported that dietary MCT is markedly less effective than LCT in suppression of lipogenesis in liver and adipose tissue of rats²⁵⁾. MCT-rich diet (34 or 37 g/100 g diet) diminished lipogenic-related enzyme activity compared with low-fat diet^{6,14)}. In contrast, Takase *et al.*²²⁾ reported stimulation of malic enzyme activity in liver of rats by dietary MCT (13 g/100 g diet). Dietary level of MCT used in the present study was 26 g/100 g diet but effects in more moderate condition (10 to 15 g/100 g diet) remains to be tested.

Dietary long-chain fatty acids are thought to be directly incorporated into adipose tissues, where these fatty acids inhibit lipogenesis. Fougelle *et al.* reported that medium-chain and long-chain fatty acids did not have the same inhibitory potency for the activity and the gene expression of acetyl-CoA carboxylase and fatty-acid synthase in liver and adipose tissue⁷⁾. In the present study, lipogenic-related enzyme activities in the liver of C18:1-fed rats were comparable to rats fed C8:0 diet, while C18:1 diet suppressed lipogenic-related enzyme activities in both perirenal and epididymal adipose tissues compared with MCT diets. It has been reported that fatty acids of the n-9 family such as oleic acid did not suppress the expression of genes coding for lipogenic enzymes in hepatocyte⁴⁾. The present results suggested that liver and adipose tissue lipogenesis might be regulated by dietary fats in different manner.

In the present study, C10:0 or C12:0 group showed significantly lower lipogenic-related

enzyme activities than C8:0 group in liver. It is well documented that MCFAs are easily absorbed and transported via portal vein into liver. MCFAs then can rapidly pass through inner mitochondrial membrane in hepatocytes, due to independence on action of carnitine. Because of these metabolic pathway, it has been suggested that MCFAs can be easily β -oxidized in liver, and they are utilized for energy as rapidly as glucose¹⁾. In addition, C8:0 has been suggested to be metabolized in mitochondria more rapidly compared with the other MCFAs, such as C10:0 and C12:0¹⁾, which may have affected hepatic energy flux followed by shift toward lipogenic state. These unique properties of C8:0 fatty acid resulted in markedly less effect on suppression of lipogenic-related enzyme activities^{10,25)}. According to the fatty acid composition in perirenal adipose tissue, it was shown that C10:0 and C12:0 were largely incorporated into adipose depot in rats fed C10:0 and C12:0 diets, respectively. However, C8:0 did not accumulate in the adipose depot of C8:0-fed rats (Table 5). These results suggest that relatively large amount of dietary C10:0 and C12:0 may be transported to adipose tissues via lymphatic circulation in the form of chylomicron. Therefore, the amount of C10:0 (in C10:0 group) or C12:0 (in C12:0 group) oxidized in liver should have been decreased compared with that of C8:0 in C8:0 group in the present study, which may account for lower lipogenic-related enzyme activity in liver.

Interestingly, lipogenic-related enzyme activities of epididymal adipose tissue were drastically low in every group compared with those of perirenal adipose tissue. It is considered that fat deposition in epididymal adipose tissue would be less active than perirenal adipose tissue in our experimental condition. It is not clear why there is such different lipogenic performance between these adipose depots. However, maturing stage of adipose

tissue may have been one of factors that induced the difference of lipogenic-related enzyme activities between depots.

In the present experiment, enzyme activities were measured in the animals of fasted condition. It has been reported that lipogenic-related enzyme activities vary greatly by feeding or fasting^{11,12)}. The effects of each MCTs on lipogenic-related enzyme activities in the fed state remain to be discussed.

Fatty acid profile showed a good reflection of de novo lipogenesis. The composition of palmitic acid, the major end product of biosynthesis of fatty acid, was high in C8:0 group (Table 5). In addition, it is assumable that higher contents of palmitoleic (C16:1) and oleic (C18:1) acids in C8:0 group may be resulted from the desaturation and chain elongation of palmitic acid. Some authors have proposed that Δ^9 -desaturase, which may affect fatty acid composition in the body, is regulated by different dietary fatty acids²⁾. The contents of C16:0, C16:1 and C18:1 was also high in the epididymal adipose tissue of C8:0 group (data not shown). These results suggest that the increase of composition of these endogenous fatty acids in adipose tissues by C8:0 feeding is attributed in part to the stimulation of hepatic, rather than adipose tissue, lipogenesis.

In conclusion, the present data suggested that C8:0, C10:0 and C12:0 fatty acids, so called MCT, should be reevaluated as different nutrients one another in view of its metabolic consequences, at least, in the point of different lipogenic effects. Furthermore, there may be tissue-specific regulatory mechanism of lipogenesis by the chain length of fatty acid. Lipogenic response can be varied with its anatomical site even in the same kind of tissues.

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単一の脂肪酸（カプリル酸，カプロン酸，ラウリン酸，オレイン酸）
からなるトリアシルグリセロールの給与がラット肝臓および
脂肪組織における脂肪酸合成関連酵素活性に及ぼす影響

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飼料中に添加した中鎖脂肪酸トリアシルグリセロール（MCT）における鎖長の違いが成長中ラットの脂肪酸合成関連酵素活性に及ぼす影響について検討した。8週齢のウィスター系雄ラットを4グループに分け，それぞれ単一の脂肪酸（カプリル酸；C8:0，カプロン酸；C10:0，ラウリン酸；C12:0，オレイン酸 C18:1）からなるトリアシルグリセロールを26%含む飼料で4週間飼育した。その後，肝臓，腎周囲脂肪組織，副こう丸脂肪組織を採取し，脂肪酸合成関連酵素であるグルコース-6-リン酸脱水素酵素（G-6-PDH），クエン酸開裂酵素（CCE）およびリンゴ酸酵素（ME）の各活性を測定した。C8:0区はC10:0区よりも肝臓におけるG-6-PDHおよびCCE活性が有意に高かった。またC8:0区とC18:1区は肝臓において同程度の酵素活性を示した。腎周囲脂肪組織において，C8:0区のG-6-PDH活性およびME活性は他の3区と比較して高かったが，CCE活性はC10:0区およびC12:0区よりも低かった。副こう丸脂肪組織においては，C8:0区のG-6-PDH活性およびME活性がC12:0区に対して低かった。また腎周囲脂肪組織中のトリアシルグリセロールの脂肪酸組成を調べたところ，C10:0区およびC12:0区ではそれぞれC10:0，C12:0の脂肪酸がかなり存在したのに対し，C8:0区の脂肪組織からはC8:0の脂肪酸はほとんど検出されなかった。以上の結果から，飼料源としてのC10:0およびC12:0の脂肪酸は，脂肪酸合成に及ぼす影響の点で，C8:0とはかなり異なる中鎖脂肪酸であると考えられた。

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