Original Article

Effect of dietary medium- and long-chain triacylglycerols (MLCT) on accumulation of body fat in healthy humans

Michio Kasai BSc[1], Naohisa Nosaka BSc[1], Hideaki Maki MSc[1], Satoshi Negishi PhD[1], Toshiaki Aoyama PhD[1], Masahiro Nakamura MSc[1], Yoshie Suzuki BSc[1], Hiroaki Tsuji MSc[1], Harumi Uto MSc[2], Mitsuko Okazaki PhD[3] and Kazuo Kondo MD, PhD[2]

[1] *Division of Healthcare Science Research Laboratory, Nisshin Oil Mills, LTD., Kanagawa, Japan.*
[2] *Institute of Environmental Science for Human Life, Ochanomizu University, Tokyo, Japan.*
[3] *Kagawa Nutrition University, Saitama, Japan.*

We investigated whether a structured medium- and long-chain triacylglycerols (MLCT) diet could decrease accumulation of body fat in healthy humans. The study was conducted under a double-blind randomized design. Ninety-three subjects participated in this study. However, 10 subjects could not consume the specified meal, and one subject wished to opt out. Consequently, the study included 82 subjects. The experimental subjects consumed the test bread, which was made with 14 g of MLCT containing 1.7 g MCFA, daily at breakfast during the study period of 12 weeks, and the control subjects consumed bread made with long-chain triacylglycerols (LCT). All subjects consumed the same standard packaged meals. Body composition parameters were body weight, total body fat and abdominal fat, and blood analyses included serum cholesterol, triacylglycerols and phospholipids. Significant decreases of body weight, the amount of body fat, subcutaneous and visceral fat were noted in the MLCT group as compared with those of the LCT group for 12 weeks ($P < 0.05$). Furthermore, a significant decrease in serum total cholesterol was noted in the MLCT group as compared with that of the LCT group at 8 weeks ($P < 0.05$). However, other serum parameters were not different between the MLCT and LCT groups. The results suggest that the daily intake of MLCT diet could result in a reduction in body weight and in accumulation of body fat, and, moreover, it could reduce serum total cholesterol.

Key words: medium-chain fatty acids (MCFA), long chain triacylglycerols (LCT), medium chain triacylglycerols (MCT), coconut, palm kernel oil, body weight, body fat, abdominal fat, obesity, serum cholesterol, Japan

Introduction

Obesity is a major health problem in developed and developing countries. It is an important risk factor of cardio-vascular disease[1,2] because of its close association with the increased prevalence of hypertension, diabetes mellitus and dyslipidaemias.[3,4] Obesity, which results from imbalance of energy metabolism, is associated with accumulation of excess body fat in the adipose tissue of the body. Excess calories from dietary fat is an important contributory factor to fat accumulation. Among the measures undertaken to prevent obesity, dietary fat restriction is often recommended. The bulk of the fatty acids found in usual western diets consist of molecules containing 12 or more carbon atoms. These long-chain fatty acids (LCFA), either saturated or unsaturated, originate from the long-chain triacylglycerols (LCT) contained in vegetable and/or animal oil and fat sources. In contrast, medium-chain fatty acids (MCFA) are composed of 8-10 carbon atoms, and are found in coconut and palm kernel oils. Medium-chain triacylglycerols (MCT) were introduced into clinical nutrition in the 1950s for the dietary treatment of malabsorption syndromes because of their rapid absorption and solubility.[5] MCT and LCT are differently hydrolyzed and absorbed in the gastro-intestinal system. MCT are hydrolyzed rapidly to MCFA which are transported directly to the liver via the portal vein and oxidized to ketones. In contrast, LCT are absorbed via the intestinal lymphatic ducts and transported in chylomicrons through the thoracic duct into the systemic circulation.[6] It is probably due to this metabolic mechanism of MCT that makes them candidates for the dietary treatment of obesity.[7] It has been reported that the body weight gain of rats fed an MCT containing diet is less than that of rats fed an LCT one,[8-10] possibly because the oxygen consumption of the former group is higher than that of the latter.[11] In addition, several clinical studies[5,12] show that the mean postprandial oxygen consumption after a meal containing 48 g MCT is higher than that after a meal containing 45g LCT. Furthermore, we showed[13] that the

Correspondence address: Michio Kasai, Division of Healthcare Science Research Laboratory, Nisshin Oillio Ltd, 1 Shinmei-cho, Yokosuka, Kanagawa, 239-0832, Japan.
Tel: 81 468 37 2416; Fax: 81 468 37 2509
Email: m-kasai@nisshin.oilliogroup.com
Accepted 9 July 2002

amount of body fat in healthy subjects who received a diet containing 10g MCT per day for 12 weeks was significantly decreased in comparison with that of subjects who received a diet containing 10g LCT. These results suggest that MCFA could be useful in the dietary treatment of obesity. However, it is difficult to substitute MCT for LCT in dietary fat for long-term dietary therapy, largely because the lower smoke point makes MCT difficult to use as a cooking oil as shown in the previous study.[14]

Recently, we formulated a new type of cooking oil composed of structured medium and long-chain triacylglycerols (MLCT).[14] MLCT are structured lipids that contain MCFA and LCFA in the same triacylglycerol. MLCT are better for cooking than simple physical mixtures of MCT and LCT because the smoke point of the former is higher than that of the latter. It has been reported by several researchers that a MLCT diet appears to lead to fat accumulation and postprandial thermogenesis similar to those for a MCT diet. Lee *et al*., reported[15] that there were no significant differences in the amount of body fat and body weight gain between structured triacylglycerols containing MCFA and mixtures of MCT and LCT given in the diet to rats for 3 weeks. Bendixen *et al.,* reported[16] that there were no significant differences in postprandial thermogenesis after the intake of test oil in healthy men between structured triacylglycerols containing MCFA and mixtures of MCT and LCT. On the other hand, Matsuo *et al.,* reported[17] that the mean postprandial oxygen consumption after a meal containing MLCT was higher than that after a meal containing LCT. Therefore, it is worthwhile to examine whether MLCT would be useful as a special cooking oil for dietary therapy.

The purpose of this research was to investigate the hypothesis that a MLCT containing diet could decrease body fat accumulation in healthy subjects in comparison with a LCT containing diet.

Methods

Subjects

The study commenced with 93 volunteers, ranging in age from 21 to 59 years, with mean body mass index (BMI) of 24.6 ± 0.3 kg/m^2. All the subjects were generally healthy and had no history of hypertension, diabetes or hyperlipidemia. Most of the subjects were classified as performing level 1 (mild) or level 2 (medium) daily physical activity according to the 6th edition of the Recommended Dietary Allowance for Japanese (Dietary Reference Intake).[18] The study was carried out in accordance with the Helsinki Declaration of 1995 (as revised 2000) and was approved by the Ethics Committee of Ochanomizu University. The procedures were explained in detail to all the volunteer subjects in advance, and all gave their signed informed consent before participating in the study. Among the 93 subjects who participated in the study, 10 were unable to consume the specified meal for more than 3 sequential days for various reasons, and one subject opted out of the study. The reasons were inability to consume the whole meals, and inability to consume the specified foods while on a long-term business trip. Therefore, the data for these subjects were excluded. Consequently, the study was carried out on 82 subjects, ranging in age from 21 to 59 years, with a mean BMI of 24.6 ± 0.3 kg/m^2 (Table 1). Statistical analysis was conducted using the data obtained from these 82 subjects.

Table 1. Characteristics of the study groups [a]

	Long-chain triacylglycerols diet group (N = 42)	Medium- and long-chain triacylglycerols diet group (N = 40)
Age (year)	37.0 ± 1.0	35.6 ± 1.5
Height (cm)	170.1 ± 1.1	170.5 ± 1.1
Body weight (kg)	71.2 ± 1.4	71.9 ± 1.4
BMI (kg/m^2)	24.6 ± 0.4	24.7 ± 0.4
Body fat weight (kg)[b]	16.7 ± 0.7	17.0 ± 0.9
Total fat area (cm^2)[c]	216.7 ± 10.9	218.6 ± 13.0
Subcutaneous fat area (cm^2)	145.7 ± 8.3	151.9 ± 10.1
Visceral fat area (cm^2)	71.0 ± 4.7	66.7 ± 5.0
Energy intake (kJ/day)	9074.9 ± 191.3	9153.9 ± 322.7
Fat intake (g/day)	70.2 ± 2.3	68.3 ± 3.6
Fat intake (% of energy)	29.1 ± 0.7	27.8 ± 0.8
Protein intake (g/day)	89.5 ± 2.7	86.7 ± 3.6
Protein intake (% of energy)	16.6 ± 0.4	15.9 ± 0.5
Carbohydrate intake (g/day)	280.3 ± 8.3	291.6 ± 10.3
Carbohydrate intake (% of energy)	51.8 ± 1.0	53.7 ± 0.8

[a] Mean ± SEM; N = 40 (medium- and long-chain triacylglycerols diet group; 36 men and 4 women) or 42 (long-chain triacylglycerols diet group; 39 men and 3 women). [b] Multiplied body weight by body fat (%) (Body fat weight = body weight x body fat (%)). [c] Sum of subcutaneous fat area and visceral fat area measured by CT scan.

Table 2. Fatty acid and triacylglycerol composition of test oils [a]

	Long-chain triacylglycerols[b]	Medium- and long-chain triacylglycerols
	% by weight	
Fatty acid		
8:0[c]	ND	9.7
10:0	ND	3.3
16:0	6.2	3.8
16:1	0.2	0.2
18:0	2.5	1.7
18:1	48.8	51.2
18:2	30.2	18.4
18:3	9.4	9.0
20:0	0.6	0.6
20:1	1.1	1.2
22:0	0.4	0.3
22:1	0.2	0.3
24:0	0.2	0.1
24:1	0.2	0.2
Total	100.0	100.0
Triacylglycerol		
L,L,L	100.0	55.1
L,L,M	ND	35.2
L,M,M	ND	9.1
M,M,M	ND	0.6
Total	100.0	100.0

[a]ND, not detected; L, long-chain fatty acids; M, medium-chain fatty acids. [b]Blended oil of rapeseed oil and soybean oil (7:3). [c]Number of carbon atoms: number of double bonds.

Test diets

The preparation of the test oil was based on the method of Akoh *et al.*,[19] The test oil was prepared by transesterification of 14% (wt/wt) MCT (Nisshin Oil Mills, Tokyo, Japan) and 86% (wt/wt) rapeseed oil (Nisshin Oil Mills). After the transesterification, the test oil was refined by the same method that is used for a common edible oil. A common edible oil (a blend of rapeseed and soybean oils; Nisshin Oil Mills), was used as LCT. The fatty acid compositions were determined by a gas-liquid chromatographic system (6890 series; Agilent Technologies, Palo Alto, CA) with a capillary column (Omegawax-320; Supelco, Bellefonte, PA), after methylation with sodium methoxide. The triacylglycerol compositions were determined by a gas-liquid chromatographic system (6890 series; Agilent Technologies, Palo Alto, CA) with a capillary column (DB-1HT; J&W Scientific, Folsom, CA). The fatty acid and triacylglycerol compositions of LCT and MLCT are given in Table 2. Bread containing LCT or MLCT was prepared and designated as LCT or MLCT containing diet, respectively. Fourteen grams of the MLCT contained 1.7g MCFA by a gas-liquid chromatographic method.

Test protocol

The study was carried out in a controlled, double-blind manner. The body weight, height, energy intake, lipid intake and daily physical activity of the subjects were determined before the beginning of the study (Table 3). The baseline data for energy and fat intake during the study was determined from a 3-day preliminary investigation conducted prior to the study.[18] The 82 subjects were randomized and assigned to either the group receiving the LCT containing diet (LCT) or the group receiving the MLCT containing diet (MLCT). The subjects were asked to consume 8778~10032 kJ energy and 64~70 g of total fat per day, and maintain their daily exercise at a fixed level, during the 12-week experimental period.

Before starting the study, all the subjects were given thorough instruction in dietary regulation. The test diet was given only at breakfast, and the daily intake of the test oil was set at 14 g. The subjects were asked to consume the test diet every day at breakfast throughout the study period. For lunch and dinner, the subjects consumed the same packaged meals throughout the 12 week period, prepared under the guidance of a dietitian. The average energy content of breakfast, lunch and dinner was about 1600, 2900 and 3600 kJ, respectively. The average total fat content of breakfast, lunch and dinner was about 16, 21 and 27 g, respectively. The subjects were also asked to consume 150 g/day fruit and 100 g/day vegetables (about 540 kJ) every day. Furthermore, they were also asked to consume side dishes or snacks containing between 210 and 1460 kJ of energy and 6g of fat, without fail every day. If the subjects were unable to consume their packaged meal lunch/dinner for any reason, they were asked to maintain the target intake of energy and total fat by eating food from the menu of a restaurant or fast-food outlet. Moreover, if they were also unable to consume this alternative food because of personal situations, individual directions were given on the basis of a menu provided in advance. The daily intake of alcoholic beverages was restricted to 25 ml of alcohol equivalent.

The subjects were instructed to record the contents of their daily meals, snacks and beverages in a diet diary for the entire test period. The diary was collected weekly to confirm the meal intake, and if necessary, the subject was immediately instructed afresh on how to adhere to the dietary regime. Daily intakes of energy, fat, protein, carbohydrate and fatty acids were calculated from the dietary records by the dietitian on the basis of the 4th Revision of the Standard Tables of Food Composition in Japan.[20]

Anthropometric measurements

All measurements were performed by investigators trained in anthropometric measurements. The subjects wore a swimsuit during the anthropometric measurements. Body weight and height were measured to the nearest 0.1 kg and 0.1 cm, respectively. Waist circumference was measured at the umbilical level. Maximum hip circumference was obtained at the level of the greatest posterior protuberance. Both waist and hip circumferences were measured to the nearest 0.1 cm in the standing position. All of these measurements were carried out at 0, 4, 8 and 12 weeks.

Blood sampling and clinical analysis

At 0, 4, 8 and 12 weeks, blood samples were collected from the subjects at 9:00 h after an overnight fast from 21:00 h on

Table 3. Intakes of energy, fat, fatty acids, protein, carbohydrate, cholesterol, and alcohol of men and women consuming either long-chain triacylglycerols or medium- and long-chain triacylglycerols diet for 12 weeks[a]

Dietary intake	week	Long-chain triacylglycerols diet group (N = 42)	Medium- and long-chain triacylglycerols diet group (N = 40)
Energy (kJ/day)	Before[b]	9074.9 ± 191.3	9153.9 ± 322.7
	1-12	9327.9 ± 47.4	9327.3 ± 45.5
Fat (g/day)	Before	70.2 ± 2.3	68.3 ± 3.6
	1-12	65.8 ± 0.2	65.7 ± 0.2
(% of total energy)	Before	29.1 ± 0.7	27.8 ± 0.8
	1-12	26.6 ± 0.1	26.6 ± 0.1
Saturated fatty acids[c] (g/day)	Before	13.0 ± 0.6	13.0 ± 0.8
	1-12	14.9 ± 0.1	14.4 ± 0.1
Monounsaturated fatty acids (g/day)	Before	18.2 ± 0.8	17.8 ± 1.2
	1-12	27.2 ± 0.1	27.9 ± 0.1
(n-6) polyunsaturated fatty acids (g/day)	Before	11.6 ± 0.5	10.9 ± 0.7
	1-12	14.5 ± 0.0	12.9 ± 0.0***
(n-3) polyunsaturated fatty acids (g/day)	Before	2.79 ± 0.14	2.60 ± 0.24
	1-12	3.35 ± 0.01	3.15 ± 0.01
Medium-chain fatty acids (g/day)	Before	0.20 ± 0.03	0.22 ± 0.03
	1-12	0.15 ± 0.01	1.95 ± 0.01***[d]
Protein (g/day)	Before	89.5 ± 2.7	86.7 ± 3.6
	1-12	85.1 ± 0.8	84.6 ± 0.7
(% of total energy)	Before	16.6 ± 0.4	15.9 ± 0.5
	1-12	15.3 ± 0.1	15.2 ± 0.1
Carbohydrate (g/day)	Before	280.3 ± 8.3	291.6 ± 10.3
	1-12	313.6 ± 2.5	312.7 ± 2.5
(% of total energy)	Before	51.8 ± 1.0	53.7 ± 0.8
	1-12	56.3 ± 0.2	56.2 ± 0.3
Cholesterol[e] (mg/day)	Before	410.2 ± 23.1	382.9 ± 23.6
	1-12	233.9 ± 1.1	233.7 ± 1.5
Alcohol (g/day)	Before	11.1 ± 2.1	12.8 ± 2.2
	1-12	7.4 ± 1.1	7.0 ± 1.0

Mean ± SEM; N = 40 (medium- and long-chain triacylglycerols diet group) or 42 (long-chain triacylglycerols diet group).[b] Based on data collected from 3-day food intake records. [c] Medium-chain fatty acids excluded. [d] One point seven grams out of 1.95g in the MCFA ingestion was supplied from test oil. [e] Determined by gas chromatography. ***Significantly different from long-chain triacylglycerols diet group, $P < 0.001$.

the previous day. The blood sampling and anthropometric measurements were carried out on the same day. Analyses of serum total cholesterol and triacylglycerols were carried out on a 7450 automated system (Hitachi, Tokyo, Japan) by enzymatic methods. Analyses of serum high density lipoprotein (HDL) cholesterol and low density lipoprotein (LDL) cholesterol were carried out on a Rapid electrophoresis scanning (REP) automated system (Helena Laboratories, Saitama, Japan) by agarose-gel electrophoretic methods.[21] Analysis of serum insulin was conducted using an ARC950 automated system (ALOKA, Tokyo, Japan) by enzymatic methods. Analyses of plasma glucose and serum total ketone bodies were carried out on a JCA-BM12 automated system (JEOL, Tokyo, Japan) by an enzymatic method. Analyses of serum AST, ALT and γ-GPT were carried out on a 7170 automated system (Hitachi) by UV, UV and colorimetric methods, respectively.

Measurement of body fat

At 0, 4, 8 and 12 weeks, body fat was measured by the air-replacement method with a MAB-1000 body densitometer (Nihon Kohden, Tokyo, Japan).

Measurement of fat by computed tomography (CT)

At 0, 4, 8 and 12 weeks, the subjects underwent CT scanning at Yokohama Red Cross Hospital (Yokohama, Japan) on a Pro Seed (GE Yokogawa Medical Systems, Tokyo, Japan). The subcutaneous and visceral fat areas were determined from the CT images at the umbilical level by the method of Tokunaga *et al.*[22]

Statistical analysis

Data are presented as means ± SEM. Differences in the measured values, changes in values, and change rates of anthropometric measurements, body composition, fasting blood profiles, and food intake between the MLCT and the

LCT groups were compared by repeated–measures analysis of variance (ANOVA). Diet group x period of interaction was included in the model as fixed effects. When significant differences were observed, comparison of means was carried out using Student's *t* test to examine the difference in treatment effects between the two groups. Data were also tested to compare the relations between the four periods of dietary regulation (i.e. 4, 8, and 12 weeks values against 0 week values) by using one-way ANOVA. If significant differences were detected, Scheffe's procedure for multiple comparisons was performed for evaluation of the differences among the four periods of dietary regulation. All analyses were performed using SPSS for WINDOWS (version 10.0J; SPSS Japan Inc., Tokyo, Japan). Statistical significance was set at $P < 0.05$.

Results

Energy consumption

Before the test, there were no differences between the LCT group and the MLCT group with regard to energy intake, major nutrient intake, dietary fat composition, dietary cholesterol and alcohol intakes (Table 3). On the other hand, during the 12 weeks study period, the intake of medium-chain fatty acids in the MLCT group was significantly higher than that in the LCT group, and the intake of (n-6) polyunsaturated fatty acids (PUFA) in the LCT group was significantly higher than that in the MLCT group. There were no other significant differences in nutrient intake or dietary lipid composition between both groups during the test study period. Moreover, there were also no differences in cholesterol and alcohol intake between both groups.

Anthropometric variables

Body weight and BMI at 4, 8, and 12 weeks in both groups decreased significantly during the study period (Table 4). However, the extent of the decrease in body weight and BMI was significantly greater in the MLCT group than in the LCT group at corresponding time points. Significant decreases in waist circumference at 4, 8 and 12 weeks and significant decreases in hip circumference at 8 and 12 weeks were noted in the MLCT group as compared with the LCT group. The waist-hip ratio (WHR) also decreased in both groups, but the difference was not significant.

Body fat analysis

The amount of body fat decreased significantly in both study groups (Table 5). A significant reduction in the amount of body fat was noted in the MLCT group than in the LCT group at 4, 8 and 12 weeks. The amount of body fat in both groups decreased after the consumption of the test diets for 12 weeks, and the changes in those parameters were almost parallel to the decreases noted in the body weight. The areas of subcutaneous and visceral fat in CT images also decreased significantly in both groups. The decrease in the area of subcutaneous fat was significantly greater in the MLCT

Table 4. Change in anthropometric measurement of men and women consuming either long-chain triacylglycerols or medium- and long-chain triacylglycerols diet for 12 weeks [a]

		Long-chain triacylglycerols diet group (N = 42)			Medium- and long-chain triacylglycerols diet group (N = 40)		
			Change			Change	
	Week		Delta [b]	% [c]		Delta	%
Body weight (kg)	0	71.2 ± 1.4			71.9 ± 1.4		
	4	69.5 ± 1.3***	–1.7 ± 0.2	–2.2 ± 0.3	69.4 ± 1.2***	–2.4 ± 0.2*	–3.3 ± 0.3 *
	8	68.7 ± 1.2***	–2.5 ± 0.3	–3.4 ± 0.4	68.4 ± 1.2***	–3.5 ± 0.3*	–4.8 ± 0.4 *
	12	67.9 ± 1.2***	–3.3 ± 0.4	–4.5 ± 0.5	67.3 ± 1.1***	–4.5 ± 0.4*	–6.1 ± 0.5 *
BMI (kg/m^2)	0	24.6 ± 0.4			24.7 ± 0.4		
	4	24.0 ± 0.3***	–0.6 ± 0.1	–2.2 ± 0.3	23.9 ± 0.4***	–0.8 ± 0.1*	–3.3 ± 0.3 *
	8	23.7 ± 0.3***	–0.9 ± 0.1	–3.4 ± 0.4	23.5 ± 0.4***	–1.2 ± 0.1*	–4.7 ± 0.4 *
	12	23.4 ± 0.3***	–1.1 ± 0.1	–4.5 ± 0.4	23.2 ± 0.4***	–1.5 ± 0.1*	–6.1 ± 0.5 *
Waist circumference (cm)	0	86.2 ± 1.0			86.9 ± 1.1		
	4	84.8 ± 1.0***	–1.3 ± 0.2	–1.5 ± 0.2	84.9 ± 1.1***	–2.0 ± 0.2*	–2.3 ± 0.3 *
	8	84.2 ± 1.0***	–2.0 ± 0.3	–2.3 ± 0.4	83.8 ± 1.1***	–3.1 ± 0.3*	–3.6 ± 0.4 *
	12	83.4 ± 1.0***	–2.8 ± 0.4	–3.2 ± 0.4	82.9 ± 1.1***	–4.0 ± 0.4*	–4.6 ± 0.5 *
Hip circumference (cm)	0	96.5 ± 0.6			97.7 ± 0.7		
	4	95.6 ± 0.6***	–0.9 ± 0.1	–1.0 ± 0.1	96.4 ± 0.6***	–1.3 ± 0.2	–1.4 ± 0.2
	8	95.0 ± 0.5***	–1.5 ± 0.2	–1.5 ± 0.2	95.4 ± 0.6***	–2.3 ± 0.2**	–2.3 ± 0.2**
	12	94.6 ± 0.5***	–2.0 ± 0.2	–2.0 ± 0.2	94.9 ± 0.6***	–2.9 ± 0.3**	–2.9 ± 0.3**
WHR [d]	0	0.892 ± 0.007			0.889 ± 0.007		
	4	0.887 ± 0.008	–0.005 ± 0.002	–0.6 ± 0.2	0.880 ± 0.008***	–0.009 ± 0.002	–1.0 ± 0.2
	8	0.885 ± 0.008***	–0.007 ± 0.003	–0.8 ± 0.3	0.877 ± 0.008***	–0.012 ± 0.002	–1.3 ± 0.3
	12	0.881 ± 0.008***	–0.011 ± 0.003	–1.2 ± 0.3	0.874 ± 0.009***	–0.015 ± 0.003	–1.7 ± 0.3

[a] Mean ± SEM; N= 40 (medium- and long-chain triacylglycerols diet group) or 42 (long-chain triacylglycerols diet group); [b] 4 -, 8 -, and 12 -week value minus the initial (0-week) value; [c] Change rate against the initial (0-week) value; [d] Waist-to-hip circumference ratio. ***Significantly different from the initial (0-week) value, $P < 0.001$; *Significantly different from long-chain triacylglycerols diet group, $P < 0.05$; **$P < 0.01$.

Group than in the LCT group at 8 and 12 weeks. Similarly, the decrease in the area of visceral fat in the MLCT group was also significantly greater than that in the LCT group at 8 and 12 weeks.

Blood chemistry

The serum total cholesterol concentration in both groups was significantly decreased after 4 weeks (Table 6). At 8 weeks, however, the decrease in serum total cholesterol was significantly greater in the MLCT group than in the LCT group (P = 0.07 at 12 weeks). The change in serum HDL cholesterol concentration did not differ significantly between the two groups. However, the decrease in serum LDL cholesterol in the MLCT group was greater than that in the LCT group. There was no significant difference in the serum triacylglycerols concentration between the two groups. Serum insulin and plasma glucose concentrations did not differ significantly in either of the two diet groups. There was no significant difference in the serum total ketone bodies concentration between the two groups. Moreover, serum AST, ALT and •-GPT concentrations also did not change significantly in the two diet groups (data not shown). The concentrations of serum total ketone bodies, AST, ALT and •-GPT were within their respective normal ranges in both groups.

Discussion

This study was conducted to investigate the effects on body weight and body fat in healthy humans of long-term ingestion of small amounts of MLCT in dietary fat (1.7g as MCFA). The results suggest that body fat accumulation in subjects who received 14 g of MLCT containing MCFA in the daily diet for a period of 12 weeks was lower than that in subjects who received 14 g of LCT in the diet for the same period. Many investigators have found in animal models that MCT are capable of exerting weight-reducing effects.[9-11,23-27] According to the previous reports, consumption of MCT-based dietary regimens result in a greater reduction of final body weight than consumption of LCT-based regimens. This has been confirmed in many experi-ments, performed mainly in rats. In these studies, the observed decreases in body weight resulted mainly from shrinkage of fat depots, leading to reduction in the relative fat content of the entire body.[9,24-26,28] In fact, Takeuchi *et al.*, reported[14] that the amount of body fat in rats fed the MLCT diet containing 4.8% (wt/wt) MCFA for 6 weeks was decreased in comparison with that in rats fed the LCT diet. There are a few reports comparing the effects of MCT and LCT in the diet on body weight and fat accumulation in humans.

Table 5. Change in the body fat composition of men and women consuming either long-chain triacylglycerols or medium- and long-chain triacylglycerols diet for 12 weeks [a]

		Long-chain triacylglycerols diet group (N = 42)			Medium- and long-chain triacylglycerols diet group (N = 40)		
			Change			Change	
	week		Delta [b]	% [c]		Delta	%
Body fat (%)	0	23.3 ± 0.7			23.4 ± 1.0		
	4	21.4 ± 0.8***	–1.9 ± 0.2	–8.5 ± 1.0	20.5 ± 1.1***	–2.9 ± 0.3**	–13.7 ± 1.5**
	8	20.2 ± 0.8***	–3.1 ± 0.3	–14.3 ± 1.6	19.2 ± 1.1***	–4.2 ± 0.3*	–19.9 ± 2.0*
	12	19.5 ± 0.8***	–3.8 ± 0.3	–17.0 ± 1.9	18.5 ± 1.1***	–4.9 ± 0.4*	–23.1 ± 2.5
Body fat weight (kg)[d]	0	16.7 ± 0.7			17.0 ± 0.9		
	4	15.0 ± 0.7***	–1.7 ± 0.2	–10.5 ± 1.1	14.4 ± 0.9***	–2.6 ± 0.2**	–16.4 ± 1.6**
	8	13.9 ± 0.6***	–2.7 ± 0.2	–17.0 ± 1.7	13.2 ± 0.9***	–3.8 ± 0.3*	–23.5 ± 2.2*
	12	13.3 ± 0.6***	–3.3 ± 0.3	–20.4 ± 2.1	12.6 ± 0.9***	–4.4 ± 0.4*	–27.4 ± 2.6†
Total fat area (cm^2)[e]	0	216.7 ± 10.9			218.6 ± 13.0		
	4	196.6 ± 10.3***	–20.1 ± 2.5	–9.6 ± 1.1	192.7±12.1***	–26.0 ± 3.3	–12.3 ± 1.4
	8	192.8 ± 10.3***	–23.9 ± 3.2	–11.5 ± 1.5	180.0±12.2***	–38.7 ± 3.8**	–18.8 ± 1.9**
	12	183.5 ± 10.0***	–33.2 ± 3.8	–15.8 ± 1.8	166.8±12.3***	–51.9 ± 5.1**	–25.0 ± 2.2**
Subcutaneous fat area (cm^2)	0	145.7 ± 8.3			151.9 ± 10.1		
	4	133.1 ± 7.9***	–12.7 ± 1.9	–9.1 ± 1.2	134.7 ± 9.7***	–17.3 ± 2.1	–12.5 ± 1.4
	8	128.5 ± 8.1***	–17.2 ± 2.3	–12.8 ± 1.6	125.9 ± 9.8***	–26.0 ± 3.3**	–18.8 ± 2.3**
	12	121.9 ± 7.7***	–23.9 ± 3.0	–17.0 ± 2.2	115.9±10.0***	–36.0 ± 4.2*	–25.5 ± 2.6**
Visceral fat area (cm^2)	0	71.0 ± 4.7			66.7 ± 5.0		
	4	63.5 ± 4.3***	–7.4 ± 1.3	–9.4 ± 2.1	58.0 ± 4.2***	–8.7 ± 1.8	–12.5 ± 2.4
	8	64.2 ± 4.4***	–6.7 ± 1.5	–8.7 ± 2.0	54.1 ± 4.0***	–12.7 ± 1.7*	–18.4 ± 2.2*
	12	61.7 ± 4.2***	–9.3 ± 1.9	–12.1 ± 2.2	50.9 ± 4.1***	–15.9 ± 2.0*	–23.6 ± 2.6*

[a]Mean ± SEM; N = 40 (medium- and long-chain triacylglycerols diet group) or 42 (long-chain triacylglycerols diet group); [b] 4-, 8-, and 12-week value minus the initial (0-week) value; [c] Change rate against the initial (0-week) value; [d] Multiplied body weight by body fat (%) (body fat weight = body weight x body fat (%)); [e] Sum of subcutaneous fat area and visceral fat area. *** Significantly different from the initial (0-week) value, $P < 0.001$; *Significantly different from long-chain triacylglycerols diet group, $P < 0.05$; ** $P < 0.01$.

Table 6. Concentration of lipids in serum, plasma glucose, total ketone bodies and hepatic enzymes in men and women consuming either long-chain triacylglycerols or medium- and long-chain triacylglycerols diet for 12 weeks [a]

		Long-chain triacylglycerols diet group (N = 42)			Medium- and long-chain triacylglycerols diet group (N = 40)		
			Change			Change	
	week		Delta[b]	%[c]		Delta	%
Total cholesterol (mmol/L)	0	5.28 ± 0.12			5.06 ± 0.14		
	4	4.58 ± 0.12**	–0.70 ± 0.06	–13.3 ± 1.1	4.25 ± 0.12**	–0.81 ± 0.07	–15.7 ± 1.2
	8	4.64 ± 0.13**	–0.64 ± 0.06	–12.2 ± 1.1	4.25 ± 0.13**	–0.81 ± 0.08	–15.8 ± 1.4†
	12	4.60 ± 0.11**	–0.68 ± 0.07	–12.6 ± 1.2	4.23 ± 0.12**	–0.83 ± 0.09	–16.1 ± 1.5
Chylomicron cholesterol (mmol/L)	0	0.02 ± 0.00			0.02 ± 0.00		
	4	0.01 ± 0.00	0.00 ± 0.00		0.01 ± 0.00	0.00 ± 0.00	
	8	0.03 ± 0.00*	0.01 ± 0.00		0.02 ± 0.00	0.00 ± 0.00	
	12	0.03 ± 0.00*	0.01 ± 0.00		0.03 ± 0.00*	0.01 ± 0.00	
VLDL cholesterol (mmol/L)	0	0.38 ± 0.03			0.34 ± 0.03		
	4	0.35 ± 0.04	–0.03 ± 0.03	1.1 ± 15.9	0.27 ± 0.03	–0.07 ± 0.03	–18.1 ± 8.7
	8	0.33 ± 0.03	–0.05 ± 0.03	–1.5 ± 9.0	0.31 ± 0.03	–0.03 ± 0.03	1.0 ± 7.7
	12	0.31 ± 0.03	–0.08 ± 0.02	–16.2 ± 4.4	0.25 ± 0.02*	–0.09 ± 0.02	–21.1 ± 5.8
LDL cholesterol (mmol/L)	0	3.46 ± 0.12			3.27 ± 0.15		
	4	2.90 ± 0.11**	–0.56 ± 0.06	–15.6 ± 1.8	2.69 ± 0.12**	–0.58 ± 0.08	–16.5 ± 2.1
	8	2.84 ± 0.12**	–0.62 ± 0.07	–17.8 ± 1.8	2.55 ± 0.11**	–0.72 ± 0.09	–20.5 ± 2.2
	12	2.89 ± 0.11**	–0.56 ± 0.07	–15.7 ± 1.7	2.57 ± 0.11**	–0.70 ± 0.09	–20.5 ± 1.9
HDL cholesterol (mmol/L)	0	1.43 ± 0.06			1.43 ± 0.04		
	4	1.32 ± 0.06	–0.11 ± 0.03	–7.3 ± 2.2	1.28 ± 0.05*	–0.15 ± 0.03	–10.3 ± 1.8
	8	1.44 ± 0.06	0.02 ± 0.04	3.1 ± 3.3	1.37 ± 0.06	–0.06 ± 0.07	–1.4 ± 5.7
	12	1.37 ± 0.06	–0.06 ± 0.03	–2.9 ± 2.2	1.39 ± 0.05	–0.05 ± 0.03	–2.9 ± 1.9
Triacylglycerol (mmol/L)	0	1.11 ± 0.09			1.03 ± 0.09		
	4	1.17 ± 0.09	0.07 ± 0.06	10.0 ± 5.1	0.97 ± 0.08	–0.06 ± 0.05	–1.0 ± 4.3
	8	1.04 ± 0.09	–0.07 ± 0.06	–2.9 ± 4.7	0.97 ± 0.09	–0.06 ± 0.07	0.0 ± 5.6
	12	1.05 ± 0.09	–0.05 ± 0.07	–1.1 ± 5.2	0.92 ± 0.07	–0.11 ± 0.06	–3.0 ± 5.4
Chylomicron triacylglycerol (mmol/L)	0	0.02 ± 0.00			0.01 ± 0.00		
	4	0.02 ± 0.00	0.00 ± 0.00		0.02 ± 0.00	0.00 ± 0.00	
	8	0.02 ± 0.00	0.00 ± 0.00		0.02 ± 0.00	0.00 ± 0.00	
	12	0.02 ± 0.00	0.00 ± 0.00		0.02 ± 0.00	0.00 ± 0.00	
VLDL triacylglycerol (mmol/L)	0	0.67 ± 0.09			0.55 ± 0.07		
	4	0.76 ± 0.08	0.09 ± 0.06	44.5 ± 17.4	0.55 ± 0.07	0.00 ± 0.04	20.4 ± 12.7
	8	0.61 ± 0.07	–0.06 ± 0.06	8.1 ± 10.3	0.53 ± 0.08	–0.01 ± 0.06	22.8 ± 14.6
	12	0.61 ± 0.08	–0.07 ± 0.07	6.2 ± 10.4	0.48 ± 0.08	–0.07 ± 0.05	11.3 ± 13.8
LDL triacylglycerol (mmol/L)	0	0.30 ± 0.01			0.35 ± 0.03		
	4	0.27 ± 0.02	–0.03 ± 0.02	–8.5 ± 4.4	0.30 ± 0.02	–0.06 ± 0.02	–9.5 ± 4.0
	8	0.28 ± 0.02	–0.02 ± 0.01	–5.6 ± 4.7	0.30 ± 0.02	–0.06 ± 0.03	–9.0 ± 5.1
	12	0.32 ± 0.03	0.01 ± 0.03	8.6 ± 12.9	0.31 ± 0.03	–0.04 ± 0.02	–8.8 ± 3.5
HDL triacylglycerol (mmol/L)	0	0.11 ± 0.01			0.11 ± 0.01		
	4	0.12 ± 0.01	0.01 ± 0.01	12.8 ± 8.9	0.11 ± 0.01	–0.01 ± 0.01	3.6 ± 6.3
	8	0.12 ± 0.01	0.01 ± 0.01	15.4 ± 10.2	0.13 ± 0.01	0.01 ± 0.01	19.9 ± 10.3
	12	0.12 ± 0.01	0.00 ± 0.01	9.4 ± 9.2	0.11 ± 0.01	0.00 ± 0.01	5.5 ± 7.5
Insulin (pmol/L)	0	42.2 ± 3.2			38.4 ± 2.4		
	4	41.9 ± 3.1	–0.34 ± 3.14	11.4 ± 7.9	37.5 ± 2.7	–0.91 ± 2.28	2.4 ± 6.6
	8	41.2 ± 3.0	–1.03 ± 2.91	9.6 ± 7.7	38.9 ± 3.2	0.54 ± 3.03	7.9 ± 9.2
	12	37.1 ± 2.8	–5.17 ± 2.67	–4.1 ± 5.8	35.5 ± 3.0	–2.90 ± 2.23	–3.8 ± 6.3
Plasma glucose (mmol/L)	0	5.11 ± 0.13			5.04 ± 0.08		
	4	4.77 ± 0.12**	–0.33 ± 0.09	–6.0 ± 1.7	4.80 ± 0.08*	–0.24 ± 0.09	–4.3 ± 1.6
	8	4.90 ± 0.09*	–0.21 ± 0.07	–3.5 ± 1.0	4.83 ± 0.08	–0.21 ± 0.08	–3.8 ± 1.6
	12	4.89 ± 0.10*	–0.21 ± 0.07	–3.6 ± 1.1	4.91 ± 0.07	–0.13 ± 0.05	–2.3 ± 1.0
Total ketone bodies (μmol/L) [d]	0	70.3 ± 10.0			84.7 ± 12.4		
	4	55.8 ± 6.5	–14.5 ± 11.8	21.9 ± 17.7	74.8 ± 10.3	–9.8 ± 12.9	19.5 ± 22.9
	8	73.0 ± 9.9	2.6 ± 11.9	53.5 ± 30.9	71.2 ± 9.7	–13.4 ± 9.4	1.2 ± 12.5
	12	60.8 ± 9.6	–9.5 ± 12.9	31.2 ± 24.8	73.0 ± 9.1	–11.7 ± 14.0	16.0 ± 12.9

[a] Mean ± SEM; N = 40 (medium- and long-chain triacylglycerols diet group) or 42 (long-chain triacylglycerols diet group); [b] 4-, 8-, and 12-week value minus the initial (0-week) value; [c]Change rate against the initial (0-week) value; [d]Include 3-hydoroxbutyric acid and acetoacetic acid.
*Significantly different from initial (0-week) value, $P < 0.05$; **$P < 0.01$; † Significantly different from long-chain triacylglycerols diet group, $P < 0.05$.

Yost *et al.,* placed 16 obese women on two different hypocaloric regimens (3352 kJ and 30% energy as fat) for 4 or 12 weeks.[29] The first regimen contained only LCT, whereas the second consisted of 6% LCT and 24% MCT. In their study, the recorded body weight loss was similar in both groups. Hill *et al.,* investigated 10 non-obese volunteers who were given a regimen providing 150% of the recommended dietary allowance for 6 days.[30] Fats were ingested as 40% LCT or 40% MCT in a randomized cross-over design. No significant change in body weight was recorded at the end of both diet protocols. However, we reported[13] that consumption of 10 g of MCT for 12 weeks had the effect of suppressing body fat accumulation in healthy men and women under test conditions, with total energy intake under the control of a dietitian. Moreover, in the present study, we found that body weight and the amount of body fat were decreased by intake when MLCT were consumed for 12 weeks.

This result differs from those of the previous studies described above. We suggest that the discrepancy is attributable to the different contents of MCT in the diets and to differences in the method of controlling the total energy intake. In this study, we used a much smaller quantity of MCFA than in the previous studies. Although 1.7g of MCFA in test oil is a very small amount, it is equivalent to 8 times the usual intake of about 0.2g per day by Japanese people (Table 3).[31] A large amount of MCT in the diet may increase de novo fatty acid synthesis[32] and enhance fatty acid elongation activity[33] by the liver. Geelen *et al.,* demonstrated[34] that MCT feeding increased the activities of acetyl-CoA carboxylase, fatty acid synthase and diacylglycerol acyltransferase resulting in lipid over-production. These changes would be expected to increase the production of hepatic triacylglycerols and secretion of very low density lipoprotein (VLDL). On the basis of these results, we considered that the high concentration of MCT used in the test diets in previous studies might lead to lack of difference in the effects of MCT and LCT diet groups. This consideration might be supported by the effects of diet containing MLCT on reduction of serum cholesterol concentration. Therefore, we suggest that about 2g of MCFA per day may be sufficient to accelerate lipid metabolism in humans.

The decreased accumulation of body fat following MLCT ingestion might be related to the increased thermic effect of MLCT. Recently, Matsuo *et al.,* reported[17] that the mean postprandial oxygen consumption after a meal containing MLCT was higher than that after a meal containing LCT. They suggested that the increased thermic effect of MLCT could be related to production and oxidation of ketone bodies. Synthesis of LCFA from acetyl-CoA in the liver requires a large amount of energy.[12,35] However, our results also revealed no difference in serum triacylglycerols and total ketone bodies after ingestion of diets containing LCT and MLCT. These results suggest that about 2g of MCFA is efficient for lipid oxidation. We speculate that the decrease in body fat accumulation after ingestion of MLCT might be related to peroxisomal β oxidation in brown adipose tissue[36] and a partial uncoupling of oxidative phosphorylation.[37] Therefore, daily intake of MLCT results in a significant reduction of body weight and body fat accumulation than does the intake of LCT.

Research has also been conducted on the effects of MCT on blood cholesterol metabolism,[25,30] but the results are divergent. Hashim *et al.,* reported[38] that in 8 healthy subjects, plasma total cholesterol concentrations after consumption of an MCT-supplemented diet for 2 weeks were slightly higher than those following intake of a corn oil-supplemented diet, but lower than those following consumption of a butter-enriched diet. Temme *et al*., also reported[39] in healthy subjects that the consumption of 10% MCFA diet (40% of total fat and 9100 kJ of energy per day) for 6 weeks resulted in greater elevation in blood LDL cholesterol concentration as compared with that resulting from consumption of high oleic acid diet. Swift *et al*., showed[40] that in 10 healthy subjects fed MCT at 1672 kJ for 6 days (weight maintenance energy requirements) experienced a 42% elevation in plasma triacylglycerols concentration and no effects on plasma concentrations of total cholesterol. However, in this study, the total cholesterol concentration in the MLCT group was lower than that in the LCT group. Takase *et al*., and Ecelbarger *et al.,* reported[41,42] that hepatic 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase activity in the rat was decreased by intake of an MCT containing diet, resulting in a decrease of serum total cholesterol. Therefore, we suggest that our results may be explained by a decrease in hepatic HMG-CoA reductase activity caused by intake of MLCT in the diet.

In conclusion, this study suggests that the intake of an MLCT diet containing MCFA over the long term may inhibit body weight and body fat gains. We used 14 g of MLCT as the test oil. This amount of oil is similar to that used in daily cooking in Japan. Therefore, MLCT may be useful as a special cooking oil for the dietary management of obesity.

Acknowledgement

The authors are grateful to the men and women who enthusiastically participated in this study. The authors acknowledge the technical guidance and advice given by F. Yamaki MD, S. Masuda, H. Takeuchi, S. Nakajima, and A. Seto. The authors are grateful to the Kanagawa branch of the Japan Anti-Tuberculosis Association for blood sampling, and to Yokohama Red Cross Hospital for undertaking the measurement of fat by computed tomography. The authors wish to thank K. Katori and T. Inuyama for the nutritional stewardship and the analysis, and T. Nagasawa and S. Arimoto for production of test oils.

References

1. Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen J. Puska P. Body weight, cardiovascular risk factors, and coronary mortality. 15-year follow-up of middle-aged men and women in eastern Finland. Circulation 1996; 93: 1372-1379.
2. Seidell J C, Verschuren W M M, van Leer E M, Kromhout D. Overweight, underweight, and mortality. A prospective study of 48, 287 men and women. Arch Intern Med 1996; 156: 958-963.

3. Higgins M, Kannel W, Garrison R, Pinsky J, Stokes J. Hazards of obesity- the Framinghan experience. Acta Med Scand 1988; 723 (Suppl): S23-S36.
4. Haffner S M, Stern M P, Hazuda H P, Pugh J, Patterson J K. Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids, and lipoproteins. Diabetes 1987; 36: 43-51.
5. Seaton T B, Welle S L, Warenko M K, Campbell R G. Thermic effect of medium-chain and long-chain triglycerides in man. Am J Clin Nutr 1986; 44: 630-634.
6. Bach A C, Babayan V K. Medium-chain triglycerides: An update. Am J Clin Nutr 1982; 36: 950-962.
7. Bach A C, Ingenbleek Y, Fery A. The usefulness of dietary medium-chain triglycerides in body weight control: fact of fancy? J. Lipid Res 1996; 37: 708-726.
8. Kaunitz H, Slanetz C A, Johnson R E, Babayan V K, Barsky G. Nutritional Properties of the triglycerides of saturated fatty acids of medium chain-length. J Am Oil Chem Soc 1958; 35: 10-13.
9. Hashim S A, Tantibhedyangkul P. Medium chain triglyceride in early life: Effects on growth of adipose tissue. Lipids 1987; 22: 429-434.
10. Geliebter A, Torbay N, Bracco E F, Hashim S A, Van Itallie T B. Overfeeding with medium-chain triglyceride diet results in diminished deposition of fat. Am J Clin Nutr 1983; 37: 1-4.
11. Baba N, Bracco E F, Hashim S A. Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium chain triglyceride. Am J Clin Nutr 1982; 35: 678-682.
12. Hill J O, Peters J C, Yang D, Sharp T, Kaler M, Abumrad N N and Greene H L. Thermogenesis in humans during overfeeding with medium-chain triglycerides. Metabolism 1989; 38: 641-648.
13. Tsuji H, Kasai M, Takeuchi H, Nakamura M, Okazaki M and Kondo K. Dietary medium-chain triacylglycerols suppress accumulation of body fat in a double-blind, controlled trial in healthy men and women. J Nutr 2001; 131: 2853-2859.
14. Takeuchi H, Kubota F, Itakura M, Taguchi N. Effect of triacylglycerols containing medium-and long-chain fatty acids on body fat accumulation in rats. J Nutr Sci Vitaminol 2001; 47: 267-269.
15. Lee K T, Akoh C C, Flatt W P, Lee J H. Nutritional effects of enzymatically modified soybean oil with caprylic acid versus physical mixture analogue in obese zucker rats. J Agric Food Chem 2000; 48: 5696-5701.
16. Bendixen H, Flint A, Raben A, Hoy C E, Mu H, Xu X, Bartels E M, Astrup A. Effect of 3 modified fats and a convetional fat on appetite, energy intake, energy expenditure, and substrate oxidation in healthy men. Am J Clin Nutr 2002; 75: 47-56.
17. Matsuo T, Matsuo M, Taguchi N, Takeuchi H. The thermic effect is greater for structured medium-and long-chain triacylglycerols versus long-chain triacylglycerols in healthy young women. Metabolisum 2001; 50: 125-130.
18. The Ministry of Health and Welfare. The 6th recommended dietary allowances for the Japanese. Tokyo: Dai-ichi shuppan publishing, 1999.
19. Akoh C C. Structured lipids enzymatic approach. Inform. 1995; 6: 1057-1061.
20. The Ministry of Science and Technology. The 4th revision of the standard table of food composition in Japan. Tokyo: Dai-ichi shuppan publishing, 1982.
21. Kido T, Kurata H, matsumoto A, Tobiyama R, Musha T, Hayashi K, Tamai S, Utsunomiya K, Tajima N, Fidge N, Itakura H, Kondo K. Lipoprotein analysis using agarose gel electrophoresis and differential staining of lipids. J Atheroscler Thromb 2001; 8: 7-13.
22. Tokunaga K, Matsuzawa Y, Ishikawa K, Tarui S. A novel technique for the determination of body fat by computed tomography. Int J Obes 1983; 7: 437-445.
23. Bach A C, Bois-Joyeux B, Chanez M, Delhomme B, Schirardin H, Peret J. Metabolic effects of medium-or long-chain triglycerides and high-protein, carbohydrate-free diets in Zukcer rat. Metabolism 1984; 33: 951-958.
24. Hwang SG, Yano H, Kawashima R. Influence of dietary medium- and long-chain triglycerides on fat deposition and lipogenic enzyme activities in rats. J Am Coll Nutr 1993; 12: 643-650.
25. Kaunitz H, Slanetz C A, Johnson R E, Babayan V K, Barsky G. Relation of saturated, medium-and long-chain triglycerides to growth, appetite, thirst and weight maintenance requirements. J Nutr 1958; 64: 513-524.
26. Ling P R, Hamawy K J, Moldawer L L, Istfan N, Bistrian B R, Blackburn G L. Evaluation of the protein quality of diets containing medium- and long-chain triglyceride in healthy rats. J Nutr 1986; 116: 343-349.
27. Turkenkopf I J, Maggio C A, Greenwood M R C. Effect of high fat weanling diets containing either medium-chain triglycerides or long-chain triglycerides on the development obesity in the Zucker rat. J Nutr 1982; 112: 1254-1263.
28. Hill J O, Peters J C, Lin D, Yakubu F, Greene H, Swift L. Lipid accumulation and body fat distribution is influenced by type of dietary fat fed to rats. Int J Obes 1993; 17: 223-236.
29. Yost T J, Eckel R H. Hypocaloric feeding in obese women: metabolic effects of medium-chain triglyceride substitution. Am J Clin Nutr 1989; 49: 326-330.
30. Hill J O, Peters J C, Swift L L, Yang D, Sharp T, Abumrad N. and Greene H L. Changes in blood lipids during six days of overfeeding with medium or long chain triglycerides. J Lipid Res 1990; 31: 407-416.
31. The ministry of health and welfare. The 1999 results of national nutrition survey of the Japanese. Tokyo: Dai-ichi shuppan, 2001; 65-99.
32. Levcille G A, Pardini R S, Tillotson J A. Influence of medium-chain triglycerides on lipid metabolism in the rats. Lipids. 1967; 2: 287-294.
33. Kritchevsky D, Tepper S A. Influence of medium-chain triglyceride (MCT) on cholesterol metabolism in rats. J.Nutr. 1965; 86: 67-72.
34. Geelen M J H, Schoots W J, Bijleveld C and Beynen A C. Dietary medium-chain fatty acids raise and (n-3) poly-unsaturated fatty acids lower hepatic triacylglycerol synthesis in rats. J Nutr 1995; 125: 2449-2456.
35. Papamandjaris A A, MacDougall D E and Jones P J H. Medium chain fatty acid metabolism and energy expenditure: Obesity treatment implications. Life Sci 1998; 62: 1203-1215.
36. Rothwell N J, Stock M J. Stimulation of thermogenesis and brown fat activity in rats fed medium chain triglyceride. Metabolism1987; 36: 128-130.
37. Baba N, Bracco E F, Hashim S A. Role of brown adipose tissue in thermogenesis induced by overfeeding a diet containing medium chain triglyceride. Lipids 1987; 22: 442-444.

38. Hashim S A, Arteaga A, Van Itallie TB. Effect of a saturated medium-chain triglycerid on serum-lipids in man. Lancet 1960; 1: 1105-1108.
39. Temme EHM, Mensink RP, Hornstra G. Effect of medium chain fatty acids (MCFA), myristic acid, and oleic acid on serum lipoproteins in healthy subjects. J Lipid Res 1997; 38: 1746-1754.
40. Swift L L, Hill J O, Peters J C, Greene H L. Plasma lipids and lipoproteins during 6 d of maintenance feeding with long-chain, medium-chain, and mixed-chain triglycerides. Am J Clin Nutr 1992; 56: 881-886.
41. Takase S, Mrimoto A, Nakanishi M, Muto Y. Long-term effect of medium-chain triglyceride on hepatic enzymes catalyzing lipogenesis and cholesterogenesis in rats. J Nutr Sci Vitaminol 1977; 23: 43-51.
42. Ecelbarger G L, Lasekan J B, Ney D M. In vivo triglyceride secretion and hepatic and plasma lipids in rat fed medium-chain triglycerides, tripelargonin, or corn oil. J Nutr Biochem 1991; 2: 260-266.