Effect of Resistant Maltodextrin on Digestion and Absorption of Lipids

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In order to confirm the mechanism how postprandial elevation of triacylglycerol is suppressed by resistant maltodextrin (Fibersol-2), we conducted the following experiments. Firstly, Rats were fed a high-fat diet with resistant maltodextrin at 0% (control), 2.5% or 5% for 5 weeks to determine the lipid amount excreted in the feces of the last three days. The total lipid and triacylglycerol excreted in rat feces were significantly increased in a dose-dependent manner by the ingestion of resistant maltodextrin. Secondly, 10 healthy adult subjects were administrated a beverage containing 5 g resistant maltodextrin or placebo for 10 days, and crossed over after an 11-day washout. Total lipid excreted in feces was determined by collecting all the feces for the last three days. Fecal weight and fecal lipid amount of the subjects increased significantly with the ingestion of resistant maltodextrin compared to the placebo ingestion. Thirdly, oil emulsion prepared with resistant maltodextrin was assessed for its hydrolysis rate by lipase. The hydrolysis rate of lipid by lipase was not affected by resistant maltodextrin. Lastly, micelle emulsion was prepared with or without resistant maltodextrin, and their stability was compared. Resistant maltodextrin inhibited the decomposition of micelles and stabilized micellar structure. From these results, it was suggested that resistant maltodextrin suppresses lipid absorption and promotes the excretion of lipid into feces by delaying the release of fatty acids from micelles in the lipid absorption process. No inhibitory effect on lipase activity was observed by resistant maltodextrin.

Key words —— resistant maltodextrin, blood triacylglycerol (triglyceride), lipid (fat), postprandial elevation, excretion, lipase

INTRODUCTION

Postprandial elevation of blood triacylglycerol is known to relate closely to the onset and development of arteriosclerosis and coronary artery diseases.[1, 2] It is desirable that postprandial elevation of blood triacylglycerol is controlled for the prevention of these diseases. In recent years, oolong-tea polyphenols, coffee oligosaccharides, and others have been found to inhibit the digestion and absorption of lipid in food by inhibiting lipase activity and facilitate lipid excretion. The food products formulated with these substances are known to suppress the rapid rise in blood triacylglycerol after meals.[3, 4]

In addition, it is also known that high-viscous water-soluble dietary fibers, such as psyllium affect digestion and absorption of nutrients. Digestion and absorption of lipid are also delayed by high-viscous soluble dietary fibers.[5] However, resistant maltodextrin (RMD) and hydrolyzed guar gum, which are non-viscous soluble dietary fibers and do not form a gel in the digestive tract, have also been reported to suppress postprandial elevation of triacylglycerol.[6, 7] Hydrolyzed guar gum has been reported to physically inhibit the formation of lipid-bile acid emulsion without inhibiting lipase activity.[8] As for RMD, its suppressive effect on postprandial rise in triacylglycerol was observed in animal and human studies,[6, 9, 10] however, its mechanism has not been clarified yet. We conducted this study to investigate the RMD mechanism to suppress the postprandial elevation of triacylglycerol.

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MATERIALS AND METHODS

Material —— RMD (Fibersol-2, Matsutani Chemical Industry Co, Ltd., Hyogo, Japan) was obtained by heating corn starch with a small amount of hydrochloric acid, following enzymatic hydrolysis with α-amylase and glucoamylase, and fractionation of dietary fiber.[11] RMD is a maltodextrin with an average molecular weight of 2000, but it is not readily hydrolyzed by amylases and is resistant to digestion since it has 1–2 and 1–3 glucosidic linkages in addition to 1–4 and 1–6 glucosidic linkages. The largest part of RMD is therefore dietary fiber, which reaches the large intestine. Dietary fiber in RMD is measureable by the AOAC Official Method 2001.03.[12] RMD used in this study contained 89.8% of dietary fiber by that method.

Fecal Lipid Excretion (Rat) —— The study was planned and conducted in accordance with the guide for the care and use of laboratory animals and for the alleviation of pain and distress, published by the Japanese Ministry of Environment (Public Notice No. 88, 2006). After one week of preliminary breeding, 4-week-old SD male rats (Jcl: SD, Clea Japan, Inc., Tokyo, Japan) were divided into 3 groups (7 rats/group) and fed for 5 weeks one of the following diets; high-fat diet as control (HF: 43.95% corn starch, 20.0% casein, 10.0% sucrose, 7.0% soy oil, 3.5% blended minerals, 1.0% blended vitamins, 14.0% lard, 0.3% L-cysteine and 0.25% coline bitartrate), HF with 2.5% RMD (2.5% RMD), and HF with 5% RMD (5% RMD). Each group was freely fed each diet and tap water, and the feed consumption (3 times per week) and body weight (once a week) were measured. All feces during the last 3 days of the feeding period were collected and freeze-dried to measure fecal weight. The freeze-dried feces were milled and homogenized, and total lipid concentration was measured according to the method by Folch *et al.*[13] Fecal triacylglycerol was determined by an enzymatic method (TG-E Test Wako, Wako Pure Chemical Industries, Ltd., Osaka, Japan), suspending freeze-dried feces in isopropyl alcohol. Weight of abdominal adipose tissue was also recorded at the end of the feeding period.

Fecal Lipid Excretion (Human) —— The study was conducted in the spirit of the Helsinki Declaration, by submitting related documents to an independent organization, the Ethical Committee of Medical Corporation Fukuhara Clinic, and obtained their approval in advance. Before conducting this study, we obtained spontaneously-written agreements from the participants, who had been fully informed and understood the purpose, content, method, predictable adverse effects, *etc*. The backgrounds of these 10 subjects (5 males and 5 females) are shown in Table 1.

Soft drinks containing 5 g of RMD (test beverage) or no RMD (placebo control) were prepared with lemon flavor, citric acid, and coloring. Both were filled in an unmarked plastic bottle with a 280 ml net volume, and confirmed before the experiment that both beverages were not distinguishable in appearance or taste.

In this double-blind crossover study, the subjects were randomly divided and assigned to two groups, and given either the test or placebo beverage at every meal time, three times per day for 10 days. All feces during the last 3 days were collected, as the fecal collection period. After an 11-day washout, the treatment with the crossover beverage was conducted in the same manner. The meals during the treatment periods were prepared to control the nutrient amounts. During both treatments, the subjects took the same meals in the same order to consume a uniform amount of nutrients. Fecal lipid concentration was measured according to the

Table 1. Backgrounds of Subjects

	All subjects	Male subjects	Female subjects
Number of subjects (N)	10	5	5
Age (years old)	39.7 ± 17.3	44.0 ± 10.1	35.4 ± 4.9
Body height (cm)	162.6 ± 7.2	167.6 ± 3.0	157.6 ± 1.3
Body weight (kg)	56.0 ± 8.3	61.5 ± 2.8	50.6 ± 2.9
BMI (kg/m^2)	21.1 ± 2.2	21.9 ± 1.1	20.3 ± 0.8
Systolic blood pressure (mmHg)	120.0 ± 17.9	126.1 ± 9.6	113.8 ± 5.8
Diastolic blood pressure (mmHg)	73.8 ± 12.6	76.7 ± 7.4	70.8 ± 3.5
Pulse (beats/min)	69.5 ± 7.6	68.0 ± 2.5	71.0 ± 4.3

Mean ± SEM.

analytical method by Kamer et al.[14] Average daily lipid excretion in the feces was calculated by using the obtained fecal weight and lipid concentration.

Effect on Lipase Activity —— Lipid emulsion was prepared as follows; 0.5 g sodium taurocholate and 5 g corn oil were added to 20 ml of a 20% RMD solution or distilled water, and homogenized at 21500 rpm for 3 min with a homogenizer (Ultra-Turrax T-25 Basic, IKA-Werke GmbH & Co. KG., Staufen, Germany). After a 2-hr rest period, 150 μl of the emulsion was taken from the upper layer and then suspended in 600 μl Tris-HCl buffer (pH 8.5). Then 300 μl of porcine pancreatic lipase (Elastin Products Co. Inc., Owensville, MO, U.S.A.) diluted to 400 unit/ml with Tris-HCl buffer (pH 8.5) was added to the diluted emulsion at 37°C, and the concentration of released fatty acid was measured at 0, 20, 30 and 60 min by an enzymatic method (NEFA C-Test Wako, Wako Pure Chemical Industries, Ltd.).

Stabilization of Micelle —— Micelle emulsion was prepared as follows; 0.5 g of sodium taurocholate and 2 g of 2 : 1 (molar ratio) oleic acid-monoolein mixture were added to 20 ml of a 20% RMD solution or distilled water, and homogenized at 21500 rpm for 3 min by using a homogenizer (described above). The micelle emulsion was let stand still at 37°C and the condition of the emulsion was observed visually at 0 (start), 60, 120 and 180 min with the measurement of absorbance and average particle diameter. As the indicator of transmittance, absorbance at 500 nm was measured for 1/400 dilution with 0.1% sodium dodecyl sulphate (SDS). Average particle diameter was measured using a particle size distribution analyzer (Microtrack, Nikkiso Co., Ltd., Tokyo, Japan).

Statistical Analysis —— Results were described as mean ± standard error of the mean (SEM). The significance of differences between groups was

assessed by the one-way analysis of variance (ANOVA) and Turkey's multiple comparisons, and for the comparison between two groups, a paired t-test was carried out. For all tests, $p < 0.05$ was used as the critical level of significance. A statistical software program, SPSS (Version 11, SPSS Japan, Tokyo, Japan) was used for the analysis.

RESULTS

Fecal Lipid Excretion (Rat)

No significant differences were found in cumulative feed consumption, weight gain, or body weight at the end of the feeding period. In Table 2, fecal dry weight and fecal lipid data for the last 3 days are shown along with weight of abdominal adipose tissue at the end of the feeding period. Fecal dry weight increased significantly corresponding to the dose of RMD, where the fecal dry weight of 5% RMD (2.38 ± 0.12 g/3 days) was about 30% larger than that of HF (1.81 ± 0.06 g/3 days). For the fecal lipid concentrations, no differences were found in either total lipid or triacylglycerol. However, the total excreted amount of total lipid and triacylglycerol was significantly larger in 5% RMD compared to HF, reflecting the significant increases in total fecal amount. The 5% RMD group excreted about 50% more total lipid, and about 30% more triacylglycerol compared to HF. Correspondingly, the amount of abdominal adipose tissue significantly decreased in the RMD groups compared to HF, showing negative correlation with the dose of RMD.

Fecal Lipid Excretion (Human)

The intakes of energy and fat during the treatment periods are shown in Table 3, with no significant differences between placebo and test. The results of fecal weight, lipid concentration and lipid

Table 2. Fecal Amount, Fecal Lipids and Amount of Abdominal Adipose Tissue (Rat)

	HF	2.5% RMD	5% RMD	p-value
Fecal dry weight (g/3 days)	1.81 ± 0.06[a]	2.27 ± 0.10[b]	2.38 ± 0.12[b]	0.002
Fecal lipid concentration				
Total lipid (mg/g)	123.0 ± 13.5	135.4 ± 11.0	145.4 ± 20.1	0.596
Triacylglycerol (mg/g)	108.6 ± 2.2	102.1 ± 3.0	106.7 ± 2.1	0.195
Lipid excretion				
Total lipid (mg/3 days)	220.4 ± 21.6[a]	304.7 ± 24.6[ab]	339.2 ± 40.8[b]	0.034
Triacylglycerol (mg/3 days)	196.1 ± 6.0[a]	232.0 ± 14.4[ab]	254.9 ± 14.7[b]	0.012
Abdominal adipose tissue weight (g)	20.8 ± 1.7[a]	16.2 ± 1.0[b]	14.4 ± 0.9[b]	0.006

$n = 7$ each, mean±SEM. Values not sharing a superscript letter in a same row differed significantly ($p < 0.05$).

Table 3. Energy Intake and Fat Intake During the Treatment Periods (Human)

	Placebo period	Test period
Energy intake (kcal/day)	1866.6 ± 96.8	1858.0 ± 115.4
Fat intake (g/day)	54.9 ± 3.1	55.0 ± 2.9
Energy ratio from fat (%)	26.5 ± 1.8	26.8 ± 2.7

$n = 10$, mean ± SEM.

Table 4. Fecal Amount and Fecal Lipid (Human)

	Placebo period	Test period	p-value
Fecal wet weight (g/day)	73.6 ± 13.1	129.7 ± 24.5*	0.023
Fecal lipid concentration (mg/g)	11.1 ± 1.3	11.3 ± 1.5	0.918
Lipid excretion (g/day)	0.77 ± 0.1	1.44 ± 0.1*	0.034

$n = 10$, mean ± SEM, *: $p < 0.05$.
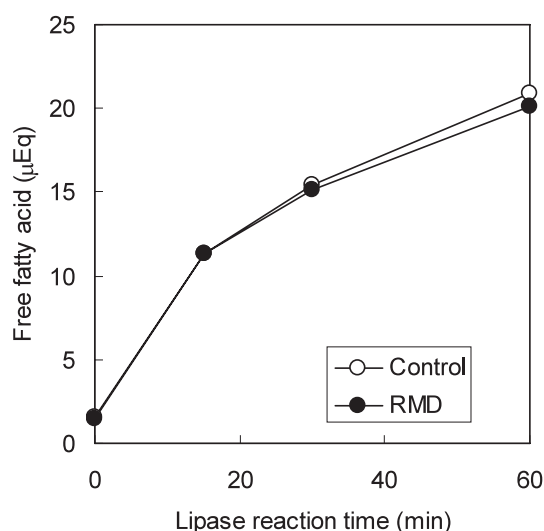
Fig. 1. Release of Fatty Acids by Lipase Hydrolysis from Emulsion Prepared with or without RMD

–○–: Control (0.5 g sodium taurocholate, 5 g corn oil and 20 ml water), –●–: RMD (emulsion prepared with 20% resistant maltodextrin solution instead of water).

amount per day are shown in Table 4. For the fecal lipid concentration, no significant differences were found between placebo and test, however, the fecal weight was significantly larger ($p = 0.023$) in the test beverage treatment (129.7 ± 24.5 g/day), compared to the placebo (73.6 ± 13.1 g/day). Correspondingly, the excreted lipid amount was significantly larger ($p = 0.034$) for the test beverage treatment (1.44 ± 0.1 g/day) than that of the placebo treatment (0.77 ± 0.1 g/day).

Effect on Lipase Activity

The release rate of fatty acids by lipase from the emulsion is shown in Fig. 1. The release of free fatty

acids from micelle was observed over time as the lipase-catalyzed reaction. RMD did not inhibit the release of fatty acids from the emulsion.

Stabilization of Micelle

The control micelle emulsion prepared without RMD was unstable, separating fatty acids and monoacylglycerol to form an upper oil layer in 180 min. However, the micelle emulsion prepared with RMD showed no separation of oil at 180 min. The changes in absorbance at 500 nm of micelle emulsion and average particle diameter of micelle are shown in Fig. 2. Absorbance of the control micelle emulsion decreased to less than 50% in 60 min, while that of the micelle emulsion containing RMD decreased about 20%. Similarly, the decrease of average particle diameter in 180 min was also smaller in the micelle emulsion with RMD, with an 80% decrease for the control micelle emulsion and a 40% decrease for the micelle emulsion with RMD.

DISCUSSION

In this study we conducted a series of experiments in order to investigate the mechanism of RMD to suppress postprandial elevation of blood triacylglycerol.

Polyphenols have been reported to suppress postprandial elevation of triacylglycerol by promoting lipid excretion into feces,[15, 16] so we considered that RMD would also have a similar function; promotion of lipid excretion. We conducted the animal and human experiments to confirm the effect of RMD on the lipid excretion into feces.
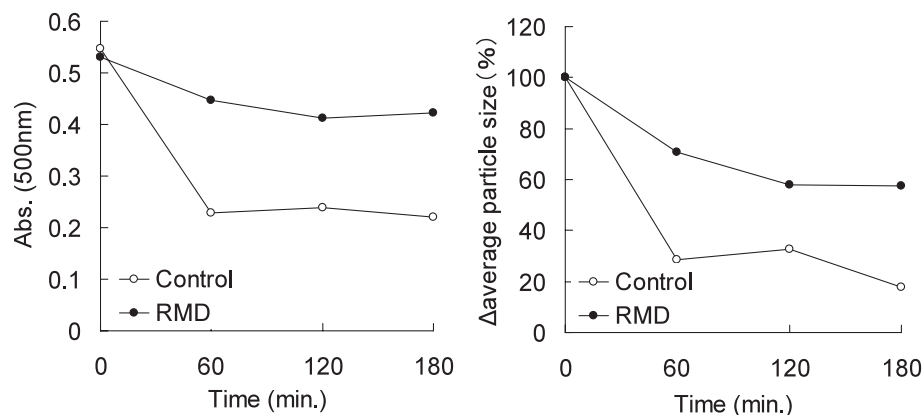
Fig. 2. Stability of Micelle Prepared with or without RMD

Left: absorbance at 500 nm of the 1/400 micelle 0.1% SDS solution; right: rate of average particle size change (start: 100%). –○–: Control (0.5 g sodium taurocholate, 2 g 2 : 1 oleic acid/monoolein mixture and 20 ml water), –●–: RMD (micelle prepared with 20% resistant maltodextrin solution instead of water).

In the animal experiment, we employed a high-fat diet containing 21% fat instead of the standard rat diet AIN93, which contains 7% fat. As a result, total lipid and triacylglycerol excreted in feces were increased by ingestion of RMD dose-dependently, showing significant differences between the control and 5% RMD.

In the human study, the meal provided to subjects contained 55 g of fat per day (about 26% energy from fat) based on the National Nutrition Survey[17] for the average nutritional intake of modern Japanese. Since the recommended daily energy intake from fat for Japanese is 20–25%,[18] 26% energy from fat in this study was slightly high, adapting to the actual eating patterns of Japanese. Consistent with the animal study, the concentration of fecal lipid did not change, however, the total excretion of lipid increased as increasing total fecal weight. It has been reported that lipid excretion in feces increased by the administration of oolong tea polyphenols.[16] In the polyphenol study, fecal lipid concentration (estimated from the reported data) seemed to stay in similar levels as shown in our current study, suggesting a possibility that there may be some upper limit for fecal lipid concentration while keeping normal fecal shape and conditions.

The fecal lipid excretion during the placebo treatment in this study (0.77 ± 0.1 g/day) matches well with a previous report.[15] Fecal lipid excretion is considered to correspond with the lipid amount not absorbed in the small intestine, because lipid is not hydrolyzed or utilized by intestinal bacteria, unlike protein or carbohydrates.[19] Since the lipid excretion was significantly increased by the ingestion

of RMD, it has been verified that RMD was associated with the reduction of lipid absorption.

Our assumption from animal experiments that RMD increases the excretion of lipids in feces has been verified in human for the first time in this study, although the increase in fecal weight by the supplementation of RMD has been reported in a large number of human studies.[20, 21]

Then we conducted a further investigation to illustrate the mechanism of how RMD suppresses lipid absorption. The process of lipid digestion and absorption consists of two processes; the digestion process and the absorption process. In the former, lipids are hydrolyzed by lipase to fatty acids and monoacylglycerol. In the latter, the produced fatty acids and monoacylglycerol form micelles with bile acids and phospholipids. The micelles are transported through the digestive tract and broken on the brush-border membrane. Fatty acids and monoacylglycerol are released from the broken micelles, to be absorbed into the body through the intestinal mucosa. In the lipid absorption process, stabilization of micelle is considered to be effective to delay the release of fatty acids and monoacylglycerol.

As it was observed that RMD does not affect the hydrolysis rate of lipid by lipase—the lipid digestion process, we examined the association of RMD in the lipid absorption process. We prepared model micelle with fatty acids, monoacylglycerol and taurocholic acid to observe its stability. The release of fatty acids and monoacylglycerol from the micelle containing RMD was delayed, showing small changes in the absorbance and average particle diameter compared to the control micelle. By adding

RMD, the stabilization of micelle was observed.

In conclusion, the results in the present study supported our hypothesis that the mechanism for RMD to suppress the elevation of postprandial triacylglycerol is the promotion of the lipid excretion into feces by delaying the release of fatty acids and monoacylglycerol from micelles.

In the food industry, maltodextrin has been used as an emulsion stabilizer based on its physical property. Similarly, or superior to regular maltodextrin, RMD also has a property to stabilize emulsion, because RMD has a high ratio of 1–6 glucosidic bonds—branched structure.[22] As shown in this study, RMD would work as an emulsion stabilizer even in the digestive tract, for RMD is not hydrolyzed by digestive enzymes and passes through the small intestine. In order to confirm that the stabilization of micelle found in the *in vitro* experiment is consistent with *in vivo*, we will conduct further investigations such as *in situ* experiments.

For clinical treatment, a lipase inhibitor that promotes lipid excretion is used as a medicine for obesity, however, because of the strong lipase inhibitory action; it also causes undesirable symptoms such as steatorrhea, loose stools, diarrhea, malabsorption of fat-soluble vitamins, *etc*. RMD did not affect the lipid concentration in feces in either animal or human without experiencing steatorrhea. Further, deficiency of fat soluble vitamins was not observed in the human study, in which subjects ingested 60 g per day of RMD for 3 months.[23] From these evidences, we believe that the mechanism of RMD to promote fat excretion is not too strong and would not cause any adverse effects from the daily consumption of RMD.

REFERENCES

1) Nikkilä, M., Solakivi, T., Lehtimäki, T., Koivula, T., Laippala, P. and Aström, B. (1994) Postprandial plasma lipoprotein changes in relation to apolipoprotein E phenotypes and low density lipoprotein size in men with and without coronary artery disease. *Atherosclerosis*, **106**, 149–157.

2) Karpe, F. (1999) Postprandial lipoprotein metabolism and atherosclerosis. *J. Intern. Med.*, **246**, 341–355.

3) Hara, Y., Moriguchi, S., Kusumoto, A., Nakai, M., Ono, Y., Abe, K., Ohta, H., Shibata, H., Kiso, Y. and Egawa, K. (2004) Suppressive effect of oolong tea polymerized polyphenols-enriched oolong tea on postprandial serum triglyceride elevation. *Japanese Pharmacology & Therapeutics*, **32**, 335–342.

4) Kumao, T., Fujii, S., Ozaki, K. and Takao, I. (2000) Effect of diets with mannooligosaccharides from coffee mannan on fat in blood serum in healthy volunteers. *Japanese Journal of Food Engineering*, **6**, 301–304.

5) Khossousi, A., Binns, C. W., Dhaliwal, S. S. and Pal, S. (2008) The acute effects of psyllium on postprandial lipaemia and thermogenesis in overweight and obese men. *Br. J. Nutr.*, **99**, 1068–1075.

6) Kishimoto, Y., Oga, H., Tagami, H., Okuma, K. and Gordon, D. T. (2007) Suppressive effect of resistant maltodextrin on postprandial blood triacylglycerol elevation. *Eur. J. Nutr.*, **46**, 133–138.

7) Kondo, S., Xiao, J. Z., Takahashi, N., Miyaji, K., Iwatsuki, K. and Kokubo, S. (2004) Suppressive effects of dietary fiber in yogurt on the postprandial serum lipid levels in healthy adult male volunteers. *Biosci. Biotechnol. Biochem.*, **68**, 1135–1138.

8) Kondo, S., Xiao, J. Z., Iwatsuki, K., Kokubo, S. and Higashiguchi, T. (2004) Effect of partially hydrolyzed guar gum on fat emulsion and lipase activity. *Jomyaku Keicho Eiyo* (*The Journal of Japanese Society for Parenteral and Enteral Nutrition*), **19**, 71–76.

9) Hironaka, T., Kishimoto, Y., Matsubara, H. and Matsuoka, Y. (2008) Inhibitory effect of tea containing resistant maltodextrin on the elevation of serum triglyceride after intake of lipid. *Japanese Pharmacology & Therapeutics*, **36**, 445–451.

10) Kishimoto, Y., Hayashi, N., Yamada, T., Yuba, K. and Yamamoto, K. (2009) Favorable effect of resistant maltodextrin on postprandial blood glucose, insulin and triglyceride levels. *Japanese Pharmacology & Therapeutics*, **37**, 277–283.

11) Ohkuma, K., Matsuda, I., Katta, Y. and Hanno, Y. (1990) Pyrolysis of starch and its digestibility by enzymes—Characterization of indigestible dextrin—. *Denpun Kagaku*, **37**, 107–114.

12) AOAC International (2005) AOAC Official Method 2001.03. Dietary Fiber Containing Supplemented Resistant Maltodextrin (RMD). In *Official Methods of Analysis of AOAC International*, 18th Ed., AOAC International, Gaithersburg, MD, U.S.A.

13) Folch, J., Lees, M. and Sloane-Stanly, G. H. (1957) A simple method for the isolation and purification of total lipides from animal tissues. *J. Biol. Chem.*, **226**, 497–509.

14) Van de Kamer, J. H., Ten Bokkel Huinink, H. and Weyers, H. A. (1949) Rapid method for the determination of fat in feces. *J Biol. Chem.*, **177**, 347–355.

15) Kumao, T., Fujii, S., Asakawa, A., Takehara, I. and

Fukuhara, I. (2006) Effect of coffee drink containing mannooligosaccharides on total amount of excreted fat in healthy adults. *J. Health Sci.*, **52**, 482–485.

16) Hsu, T. F., Kusumoto, A., Abe, K., Hosoda, K., Kiso, Y., Wang, M. F. and Yamamoto, S. (2006) Polyphenol-enriched oolong tea increases fecal lipid excretion. *Eur. J. Clin. Nutr.*, **60**, 1330–1336.

17) Ministry of Health, Labour and Welfare of Japan (2006) *National Health and Nutrition Survey in Japan, 2004*, Dai-Ichi Shuppan Publishing, Tokyo, pp. 52–53.

18) Ministry of Health, Labour and Welfare of Japan (2005) *Dietary Reference Intakes for Japanese, 2005*, Dai-Ichi Shuppan Publishing, Tokyo.

19) Kondo, T. and Nakae, Y. (1997) Examination of fat in feces. *Kensa to Gijutsu* (*Modern Medical Laboratory*), **25**, 435–440.

20) Satouchi, M., Wakabayashi, S., Ohkuma, K., Fujiwara, K. and Matsuoka, A. (1993) Effect of indigestible dextrine on bowel movements. *The Japanese Journal of Nutrition and Dietetics*, **51**, 31–37.

21) Yamamoto, Y., Nishida, T. and Sone, Y. (2000) The effect of ingestion of beverage supplemented with indigestible dextrin on human defecation. *Journal of Nutritional Food.*, **3**(2), 29–36.

22) Okuma, K. and Matsuda, I. (2003) Production of indigestible dextrin from pyrodextrin. *Journal of Applied Glycoscience*, **50**, 389–394.

23) Nomura, M., Nakajima, Y. and Abe, Y. (1992) Effects of long-term administration of indigestible dextrin as soluble dietary fiber on lipid and glucose metabolism. *Journal of Japanese Society of Nutrition Food Science*, **45**, 21–25.