Eur J Nutr (2007) 46:133–138
DOI 10.1007/s00394-007-0643-1

ORIGINAL CONTRIBUTION

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Suppressive effect of resistant maltodextrin on postprandial blood triacylglycerol elevation

Received: 18 August 2006
Accepted: 18 January 2007
Published online: 13 February 2007

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Abstract *Background* As the physiological functions of soluble dietary fibre, the favourable efficacy, such as attenuating the absorption of saccharides or lipids, is expected. Resistant maltodextrin, a soluble dietary fibre, was investigated and found that it delays the glucose absorption and attenuates the postprandial rise in the blood glucose levels, however, the efficacy of resistant maltodextrin on lipid metabolism is not yet reported. *Aim of the study* We conducted an animal experiment and a human experiment to investigate the effect of resistant maltodextrin on postprandial blood triacylglycerol elevation. *Methods* 1. Rats were fed corn oil with or without resistant maltodextrin and the postprandial changes in triacylglycerol were examined. 2. We then conducted a dietary loading experiment on 13 healthy adult male and female subjects using a meal containing approximately 50 g fat. A beverage not containing resistant maltodextrin was used as a placebo; subjects consumed the loading meal and a beverage containing either 5 g or 10 g resistant maltodextrin; blood was periodically collected to see the changes in serum constituents. *Results* 1. The corn oil administration experiment using rats showed that resistant maltodextrin dose-dependently suppressed elevation of blood triacylglycerol levels after corn oil administration. 2. The dietary loading experiment on 13 healthy subjects with 5 or 10 g of resistant maltodextrin showed that; in each administration group, resistant maltodextrin significantly suppressed postprandial elevation of blood triacylglycerol, RLP-cholesterol and insulin. *Conclusion* These results indicate that resistant maltodextrin ingested with fatty meals suppresses the postprandial elevation of blood triacylglycerol levels.

Key words resistant maltodextrin – postprandial elevation of blood triacylglycerol (triglyceride) – RLP-cholesterol (remnant-like particle cholesterol) – insulin

Background

Since the 1980s, various studies have addressed the relationship between fasting blood triacylglycerol levels and risk of developing cardiovascular disease, and elevation of blood triacylglycerol levels is currently regarded as an independent risk factor for coronary artery disease [1, 2, 6, 16]. Recent research has also asserted an intimate relationship between postprandial elevation of blood triacylglycerol levels and the development or onset of arteriosclerosis and coronary artery disease [9, 13, 15]. In this light, to suppress not only fasting blood triacylglycerol levels but postprandial elevation of blood triacylglycerol levels has been considered as effective to prevent the development of cardiovascular diseases.

At present, food ingredients reported to suppress postprandial elevation of blood triacylglycerol levels include globin hydrolysate, tea catechins, oolong tea polyphenols and partially hydrolyzed guar gum [4, 5, 19]. Dietary fibre has also long been known to suppress lipid absorption, and partially hydrolyzed guar gum, a kind of soluble dietary fibre, is also reported to have the effect of suppressing postprandial elevation of blood triacylglycerol levels [10]. Similarly, resistant maltodextrin, a soluble dietary fibre, has been reported to delay absorption of carbohydrates and to suppress postprandial elevation of blood glucose levels when consumed with a meal [17, 21, 22], however, its effect on the postprandial elevation of triacylglycerol levels is still unknown. We therefore carried out an animal experiment and a human experiment to investigate the effect of resistant maltodextrin on postprandial elevation of triacylglycerol levels.

Experimental methods

Experimental substance

The resistant maltodextrin used (Fibersol–2, Matsutani Chemical Industry Co, Ltd., Hyogo, Japan) was obtained by adding a trace amount of HCl to cornstarch and heating, carrying out enzymatic hydrolysis with α-amylase and glucoamylase, and fractionating and refining the dietary fibre fraction [14]. Resistant maltodextrin is a kind of dextrin or maltodextrin of ~2,000 molecular weight in average, however, it is resistant to digestion i.e. dietary fibre because in addition to 1–4 and 1–6 glucosidic linkages, resistant maltodextrin has 1–2 and 1–3 glucosidic linkages that are not cleaved by amylases. Therefore most part of resistant maltodextrin reaches the large intestine. The dietary fibre content of the resistant maltodextrin served for this experiment was 89.5% by AOAC Official Method 2001.03 "Dietary Fiber in Foods Containing Resistant Maltodextrin, High MW RMD by 985.29 (IDF and SDF) and Low MW RMD by Liquid Chromatography".

Corn oil administration experiment in rats

Seven-week-old male SD rats (Jcl:SD, Clea Japan, Inc., Tokyo, Japan) were raised prefatorily for 1 week and then used in the lipid oral administration experiment. After overnight fasting, rats were separated into 4 groups (10 animals/group) such that the mean body weight of each group was the same, and rats were then given an oral administration of 1 g corn oil plus either of 0.01 g, 0.1 g, or 1 g resistant maltodextrin suspended in a 5% aqueous solution of gum Arabic, with solution volume comprising 20 ml/kg body weight. The control group was given 1 g corn oil suspended in 20 ml/kg body weight of plain 5% gum Arabic solution. Blood was collected from the caudal vein before administration and at 1, 2, 3, 4, 5, 6 and 7 h after administration, and the blood triacylglycerol levels were measured by an enzymatic method (Triglyceride E-test Wako, Wako Pure Chemical Industries, Ltd., Osaka, Japan).

Dietary loading experiment in human subjects

Experimental beverage

A 350 ml carbonated beverage containing either 5 g or 10 g resistant maltodextrin (hereinafter called "FS5 g beverage" or "FS10 g beverage") was used. A 350 ml carbonated beverage not containing added resistant maltodextrin was used as a control (placebo) beverage. To make the beverages outwardly indistinguishable, each experimental beverage was sweetened with sweeteners (aspartame (L-phenylalanine compound), acesulfame potassium and sucralose), the taste was modified by acidulants and flavours, and colouring was added.

Experimental method

This experiment was conducted in accordance with the spirit of the Helsinki Declaration. Prior to the experiment, participants were provided with a thorough explanation of the experiment, and subjects comprised totally 13 healthy adults (11 males and 2 females) from whom informed, voluntary consent to experimental participation was obtained in writing. The mean ± SEM physical data for subjects were as follows; age: 36.5 ± 2.8 years old, height: 173.5 ± 1.8 cm, body weight: 69.2 ± 2.1 kg, and BMI: 23.0 ± 0.8. The experiment was designed as a single-blind crossover in which a one-week recovery period

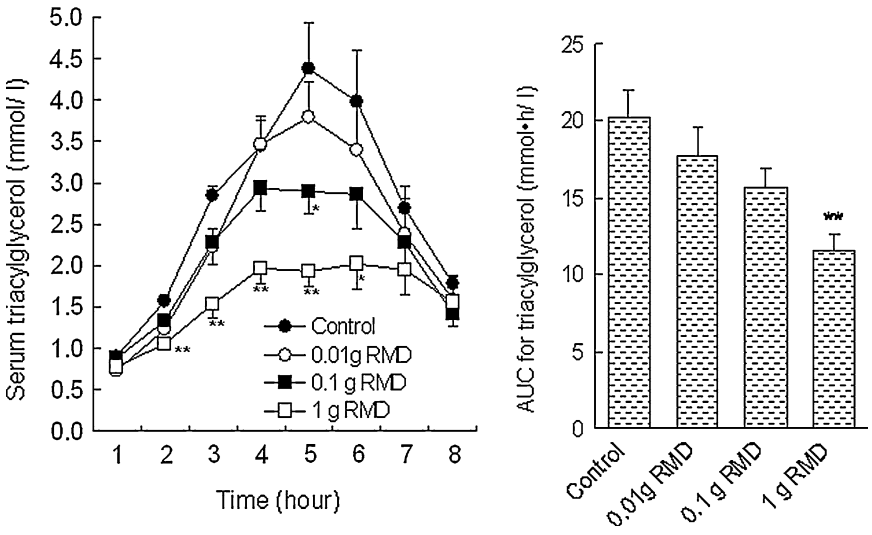

Fig. 1 Postprandial changes in the blood triacylglycerol levels (left) and the AUC up to 6 h with the pre-experiment values used as the baseline (right) after the administration of corn oil with or without resistant maltodextrin (RMD) in rats. ●: control (1 g corn oil); $n = 10$, ○: 1 g corn oil + 0.01 g RMD; $n = 10$, ■: 1 g corn oil + 0.1 g RMD; $n = 10$ and □: 1 g corn oil + 1 g RMD; $n = 10$. Mean ± SEM. *: $p < 0.05$, **: $p < 0.01$: significantly different from the control value at the given time, determined by a one-factor ANOVA (Dunnett)

was established, and the experimental beverages were then switched. Subjects were assigned randomly to 3 groups, and the experiment was conducted with a different order of experimental beverage intake in each group. On the day prior to the experiment, subjects were directed to consume the evening meal by 9 p.m., and aside from water, other food intake was prohibited until the start of the experiment. On the day of the experiment, the experiment was begun at 10 a.m. Blood was collected prior to the start of the experiment, subjects were then instructed to consume the loading meal and the experimental beverage within 20 min, and blood was collected at 0.5, 1, 2, 3, 4, 5 and 6 h postprandially. The loading meal used was 1 commercial hamburger and 135 g of fried potatoes. Their total nutritional values were as follows; energy value: 4,043.6 kJ, protein: 34.0 g, fat: 49.5 g, carbohydrate: 96.2 g. The interval from the start of the experiment to the end of the experiment was spent in a sitting position at rest, and aside from small amounts of drinking water, food and liquid fasting was enforced. Blood was collected from the cubital vein, and biochemical assays carried out measured triacylglycerol (Determiner-C TG, Kyowa Medex Co., Ltd., Tokyo, Japan), remnant-like particle lipoprotein cholesterol (RLP-cholesterol) (RLP-Cholesterol JIMRO II, Otsuka Pharmaceutical Co., Ltd., Tokyo, Japan) free fatty acids (Nescauto NEFA-V2, Alfresa Pharma Corporation, Osaka, Japan), blood glucose (Quick Auto II GLU-HK (S), Shino-Test Corporation, Tokyo, Japan), insulin (Architect Insulin, Abbott Japan Co., Ltd., Tokyo, Japan) and lipoprotein fraction [20].

Statistical analysis

Test results were indicated as mean ± SEM, and the area under the curve (AUC) was calculated by the trapezoidal method from blood concentrations up to 6 h after the meal loading, with the pre-experiment value taken as the baseline. Significance was evaluated by the iterative calculation analysis of variance (ANOVA) or Dunnett one-factor ANOVA. The statistical software used was SPSS (Ver. 13.0J, SPSS Japan, Tokyo, Japan), and the level of significance in each case was taken as 5% or lower, two tailed.

Results

Corn oil administration experiment in rats

Figure 1 presents blood triacylglycerol levels and the AUC for each group after corn oil administration. In the control group, blood triacylglycerol levels demonstrated a pattern of elevation after administration, reaching a peak at 4 h and declining thereafter. Triacylglycerol levels in the group given 0.01 g additional resistant maltodextrin did not differ significantly versus controls. In the 0.1 g addition group, triacylglycerol levels were significantly lower versus controls at 4 h after administration, and in the 1 g addition group, triacylglycerol levels were significantly lower versus controls at 1, 2, 3, 4 and 5 h after administration. Results from comparison of AUC for each group showed that AUC decreased dose-dependently as the amount of added resistant maltodextrin increased, and in the 1 g addition group, a significant difference versus controls was observed.

Dietary loading experiment in human subjects

Figure 2-a presents mean changes at each observation point, with the pre-experiment value of blood triacylglycerol levels serving as a baseline. Pre-experi-

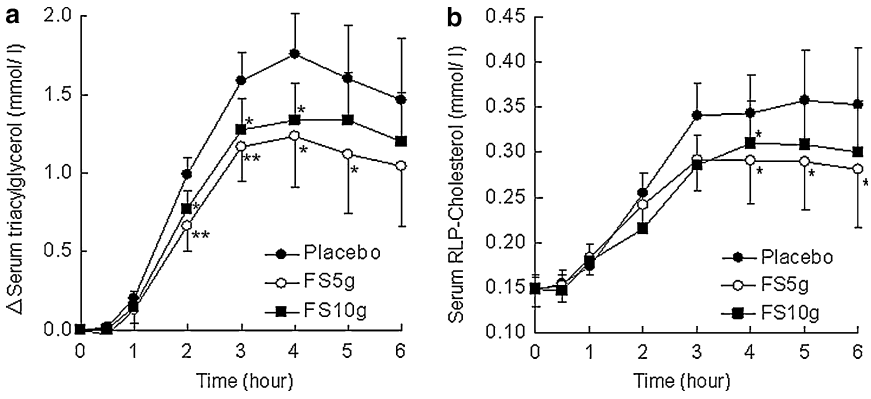

Fig. 2 (**a**) (left). Postprandial changes (differential from the pre-experiment values) in the blood triacylglycerol levels after the administration of the placebo, FS5 g (5 g RMD) or FS10 g (10 g RMD) beverage with the lipid-loading meal in human. (**b**) (right). Postprandial changes in the serum RLP-cholesterol levels after the administration of the placebo, FS5 g (5 g RMD) or FS10 g (10 g RMD) beverage with the lipid-loading meal in human. ●: placebo, ○: FS5 g and ■: FS10 g, $n = 13$, Mean ± SEM. *: $p < 0.05$, **: $p < 0.01$: significantly different from the placebo value at the given time, determined by a one-factor ANOVA (Dunnett)

ment values of blood triacylglycerol levels were 1.29 ± 0.15 mmol/l in the placebo beverage group, 1.46 ± 0.24 mmol/l in the FS5 g beverage group and 1.20 ± 0.19 mmol/l in the FS10 g beverage group; no significant intergroup differences were observed. In the placebo beverage group, blood triacylglycerol levels increased after the meal loading, reaching a peak 4 h thereafter. The FS5 g beverage group also demonstrated a similar pattern of a peak 4 h after the meal loading, however, the quantitative change at each point in time was less than that in the placebo beverage group, and values at 2, 3, 4 and 5 h after the loading meal were significantly lower than those in the placebo beverage group. In the FS10 g beverage group, triacylglycerol levels reached a peak 5 h after the meal loading, and as in the case of the FS5 g beverage group, values at each point in time were lower than those in the placebo beverage group, and a significant difference was observed at 2, 3 and 4 h after meal loading. The AUC for the FS5 g beverage group and the FS10 g beverage group were 4.99 ± 1.18 mmol·h/l and 5.48 ± 0.90 mmol·h/l respectively, significantly lower compared to the value for placebo beverage group, 6.85 ± 0.95 mmol·h/l; amounting to 72.8% and 79.9% respectively, versus the placebo beverage group.

The changes in the proportion of chylomicron in the lipoprotein fractions determined by electrophoresis matched the pattern pertaining to triacylglycerol levels, and values in the resistant maltodextrin-added beverage groups were lower than those in the placebo beverage group at each point in time. In contrast, both α-lipoprotein corresponding to HDL and β-lipoprotein corresponding to LDL decreased after ingesting the loading meal, however, no significant differences between the groups were observed.

Figure 2-b presents the time course of mean values of RLP-cholesterol levels. The FS5 g beverage group demonstrated significantly lower values than the placebo beverage group at 3, 4 and 6 h after meal loading, and the FS10 g demonstrated significantly lower values at 3 h after meal loading. The AUC for the FS5 g beverage group (0.634 ± 0.168 mmol·h/l) and the FS10 g beverage group (0.667 ± 0.160 mmol·h/l) decreased respectively to 75.9% and 79.8% of the AUC for placebo beverage group (0.836 ± 0.144 mmol·h/l), but significant differences were not observed.

By ingesting the loading meal, free fatty acid levels decreased to the half of the initial level 1 h after the meal loading, then increased again and recovered to the initial level 6 h after the meal loading. No significant intergroup differences were observed in the time courses in free fatty acid levels after loading meal ingestion.

Figure 3 presents changes in serum insulin levels after meal loading. The peak value of blood glucose levels occurred 30 min after meal loading in each group, and no significant intergroup differences were observed. Similarly, peak values of insulin occurred 30 min after loading meal ingestion, at 294.6 ± 56.6 pmol/l and 288.8 ± 40.9 pmol/l in the FS5 g beverage group and FS10 g beverage group respectively, and each of these values was significantly lower than the 30-min value of 401.7 ± 56.0 pmol/l in the placebo beverage group.

Discussion

A persistent state of high postprandial blood triacylglycerol and RLP-cholesterol levels have been implicated in causation of arteriosclerosis [8, 9, 13, 15], and RLP-cholesterol in particular is readily ingested by macrophages in the arterial walls, which is regarded to promote the formation of macrophage foam cells and facilitate the initial lesion formation in arteriosclerosis. Consequently, reduction of postprandial triacylglycerol and RLP-cholesterol levels is regarded as effective for preventing arteriosclerosis [12, 18]. There are numerous reports of food ingredients that have a suppressive effect on elevation of postprandial triacylglycerol and RLP-cholesterol, and we investigated the effect of resistant maltodextrin, a water-soluble dietary fibre.

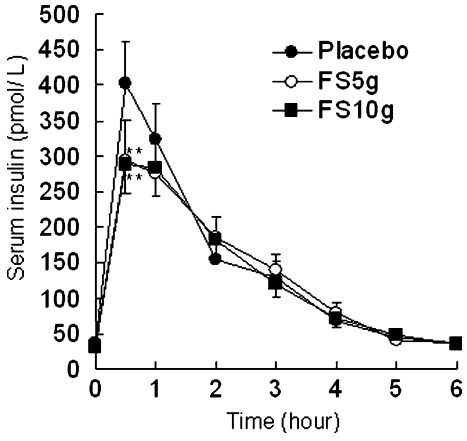

Fig. 3 Postprandial changes in the serum insulin levels after the administration of the placebo, FS5 g (5 g RMD) or FS10 g (10 g RMD) beverage with the lipid-loading meal in human. ●: placebo, ○: FS5 g and □: FS10 g, $n = 13$, Mean ± SEM. *: $p < 0.05$, **: $p < 0.01$: significantly different from the placebo value at the given time, determined by a one-factor ANOVA (Dunnett)

We first conducted an oral administration experiment of corn oil to rats. Resistant maltodextrin was added to the administered corn oil in an amount of 1, 10 or 100 wt%, and the results of this investigation showed that resistant maltodextrin dose-dependently suppressed elevation of postprandial blood triacylglycerol. Based on the result, we next conducted a dietary loading experiment on human subjects.

The dosage of resistant maltodextrin for the human experiment was decided as 10% of fat content in the loading meal; 5 g resistant maltodextrin to 50 g fat in the loading meal, because 10% resistant maltodextrin to the administered fat was effective in the animal experiment. 10 g resistant maltodextrin was also tested as a high dosage. We selected a commercial hamburger and fried potatoes among ordinary diets consumed in our daily lives in order to evaluate the efficacy of resistant maltodextrin on fatty meals. Fast foods such as hamburgers and fried potatoes are one of typical western style meals in Japan, and the relationship between obesity and the intake of such kind of fast foods is a subject of public interest worldwide [11]. As the result, resistant maltodextrin significantly suppressed elevation of blood triacylglycerol levels after meal loading at each administered amount.

We also determined changes with time in the ratio of chylomicron by lipoprotein fractionation using agarose-gel electrophoresis. The changes in chylomicron were correlated with the transitioning blood triacylglycerol levels, however, the ratios of HDL and LDL were affected by the increasing chylomicron; decreased. From these results, we concluded that the triacylglycerol elevated postprandially was mainly from chylomicron, and that resistant maltodextrin suppressed the elevation of food-derived chylomicron. Soluble dietary fibres such as pectin or psyllium are known to delay the digestion and absorption of nutrients by forming gel and prolonging the gastric retention time [7], however, it is not the case with resistant maltodextrin because the viscosity of resistant maltodextrin solution is extremely low and it does not form a gel. Polyphenols, globin hydrolysate, etc. are reported to suppress postprandial elevation of triacylglycerol by inhibiting pancreatic lipases [4, 5, 19], however, it is also not applicable to resistant maltodextrin because we experimentally confirmed that resistant maltodextrin does not inhibit lipases. A mechanism other than prolonging gastric retention time by gel formation or inhibition of lipase is required for the suppressive effect of resistant maltodextrin on postprandial triacylglycerol. As one of possible mechanisms for the suppression of postprandial triacylglycerol, the inhibition of micelle formation is considered for resistant maltodextrin as well as antihyperlipemic drugs or food ingredients inhibiting micelle formation. Further research is required to explain the mechanism of action for resistant maltodextrin, including the examination of micelle formation activity.

The peak value of postprandial blood glucose levels in the placebo beverage group was 6.14 ± 0.21 mmol/l, and despite approximately 100 g carbohydrates contained in the loading meal, a high elevation in blood glucose levels was not observed. Apart from the amount of carbohydrates ingested, the extent of postprandial rise in blood glucose levels is influenced by nutrients consumed at the same time, and the presence of lipids in particular is reported to suppress postprandial rise in blood glucose levels [3]. We believe that the presence of lipids in the loading meal in our experiment minimized the rise in postprandial blood glucose levels. At the same time, notwithstanding the lack of a high elevation in blood glucose, insulin levels were conspicuously elevated 30 min after meal loading, and resistant maltodextrin suppressed this insulin spike. In the prior researches, resistant maltodextrin is reported to suppress both postprandial rise in blood glucose and secretion of insulin [21, 22]. The result obtained that resistant maltodextrin suppressed elevation of insulin without affecting blood glucose is an extremely interesting.

In this study, we confirmed that resistant maltodextrin suppresses postprandial rise in triacylglycerol and RLP-cholesterol levels. We believe that resistant maltodextrin is useful to prevent cardiovascular diseases by improving high levels of posprandial triacylglycerol and RLP-cholesterol, which are known to promote arteriosclerosis.

In the future, we intend to clarify the mechanism of action of resistant maltodextrin and investigate its usefulness in a long-term, high fat diet experiment using animals.

Conclusion

We investigated the effect of resistant maltodextrin on the postprandial elevation of triacylglycerol in rats and humans. The conclusions derived are as follows.

1. In a corn oil administration experiment using rats, ≥ 10% to the lipid in meal of resistant maltodextrin suppressed postprandial rise in blood triacylglycerol levels dose-dependently.
2. In a dietary loading experiment among human subjects using a lipid-loading meal, a beverage containing 5 g or 10 g dissolved resistant maltodextrin was ingested with the meal; each administered amount of resistant maltodextrin was found to significantly suppress postprandial rise in blood triacylglycerol, RLP-cholesterol and insulin levels.

References

1. Carlson LA, Bottiber LE, Ahfeldt PE (1979) Risk factors for myocardial infarction in the Stockholm prospective study. A 14-year follow-up focusing on the role of plasma triglycerides and cholesterol. Acta Med Scand 206:351–360
2. Carlson LA, Bottiger LE (1985) Risk factors for ischaemic heart disease in men and women. Results of the 19-year follow-up of the Stockholm Prospective Study. Acta Med Scand 218:207–211
3. Gannon MC, Nuttall FQ, Westphal SA, Seaquist ER (1993) The effect of fat and carbohydrate on plasma glucose, insulin, C-peptide, and triglycerides in normal male subjects. J Am Coll Nutr 12:36–41
4. Hara Y, Moriguchi S, Kusumoto A, Nakai M, Ono Y, Abe K, Ohta H, Shibata H, Kiso Y, Egawa K (2004) Suppressive effect of oolong tea polymerized polyphenols-enriched oolon tea on postprandial serum triglyceride elevation. Jpn Pharmacol Ther 32:335–342
5. Inagaki H, Fujita H, Arakawa K, Sugiyama K, Hashizume S, Okuda H (2002) Suppressive effect of tea beverage including globin digest on postprandial serum triglyceride elevation. J Nutr Food 5:131–144
6. Iso H, Naito Y, Sato S, Kitamura A, Okamura T, Sankai T, Shimamoto T, Iida M, Komachi Y (2001) Serum triglycerides and risk of coronary heat disease among Japanese men and women. Am J Epidemiol 53:490–499
7. Jenkins DJ, Wolever TM, Leeds AR, Gassull MA, Haisman P, Diliwari J, Goff DV, Metz GL, Alberti KG (1978) Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity. Br Med J 1:1392–1394
8. Kane JP, Chen GC, Hamilton RL, Hardman DA, Malloy MJ, Havel RJ (1983) Remnants of lipoproteins of intestinal and hepatic origin in familial dysbetalipoproteinemia. Arteriosclerosis 3:47–56
9. Karpe F (1999) Postprandial lipoprotein metabolism and atherosclerosis. J Intern Med 246:341–355
10. Kondo S, Xiao J-Z, Takahashi N, Miyaji K, Iwatsuki K, 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
11. Maddock J (2004) The relationship between obesity and the prevalence of fast food restaurants: state-level analysis. Am J Health Promot 19(2):137–143
12. Nakajima K, Saito T, Tamura A, Suzuki M, Nakano T, Adachi M, Tanaka A, Tada N, Nakamura H, Campos E (1993) Cholesterol in remnant lipoproteins in human serum using monoclonal anti apo B-100 and A-I immunoaffinity mixed gels. Clin Chim Acta 223:53–71
13. Nikkila M, Solakivi T, Koivula T, Laippala P, Astrom 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
14. Ohkuma K, Matsuda I, Katta Y, Hanno Y (1990) Pyrolysis of starch and its digestibility by enzymes-Characterization of indigestible dextrin-. Denpun Kagaku 37:107–114
15. Patsch JR, Miesenbock G, Hopferwieser T, Muhlberger V, Knapp E, Dunn JK, Gotto AM Jr, Patsch W (1992) Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state. Arterioscler Thromb 12:1336–1345
16. Teramoto T (2002) Hyperlipidemia - pathophysiology, management and therapy. Nankodo, Tokyo, pp 76–77
17. Tokunaga K, Matsuoka A (1999) Effects of a FOSHU (food for specifed health use) containing indigestible dextrin as functional component on glucose and fat metabolisms. J Jpn Diab Soc 42:61–65
18. Tomono S, Kawazu S, Kato N, Ono T, Ishii C, Ito Y, Shimizu M, Shimoyama M, Nakano T, Nakajima K (1994) Uptake of remnant like particles (RLP) in diabetic patients from mouse peritoneal macrophages. J Atheroscler Thromb 1:98–102
19. Unno T, Suzuki Y, Nozawa A, Sagesaka Y, Kakuda T, Egawa K, Kondo K (2005) Suppressive effect of tea catechins on elevation of postprandial serum triglycerides. Jpn J Nutr Assess 22:207–212
20. Urata T, Sakurabayashi I, Kawai T (1979) Fractional determination method of lipoprotein - lipid by electrophoresis. Rinsho Kensa 23:871–880
21. Wakabayashi S (1993) The effects of Indigestible dextrin on sugar tolerance: III. Improvement in sugar torelance by indigestible dextrin on the impaired glucose tolerance model. Folia Endocrinol 69:594–608
22. Yuasa N, Yasue M, Ikeda M, Shinoda Y, Sato K, Kondo A (2004) The effects of tea beverage containing indigestible dextrin on postprandial blood glucose level after single intake and the safety in continuous intakes. J Jpn Council Adv Food Ingr Res 7:83–93