JOURNAL OF AGRICULTURAL AND FOOD CHEMISTRY

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pubs.acs.org/JAFC

Effects of a Meal Rich in 1,3-Diacylglycerol on Postprandial Cardiovascular Risk Factors and the Glucose-Dependent Insulinotropic Polypeptide in Subjects with High Fasting Triacylglycerol Concentrations

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ABSTRACT: It was previously reported that compared to triacylglycerol (TAG) oil, diacylglycerol (DAG) oil improves postprandial lipid response. However, the effects of DAG oil on postprandial hyperglycemia and incretin response have not yet been determined. In this study, the effects of DAG oil on both postprandial hyperlipidemia and hyperglycemia and the response to the glucose-dependent insulinotropic polypeptide (GIP) were studied. This randomized, double-blind, crossover study analyzed data for 41 individuals with high fasting triacylglycerol concentrations. The subjects ingested test meals (30.3 g of protein, 18.6 g of fat, and 50.1 g of carbohydrate) containing 10 g of DAG oil (DAG meal) or TAG oil (TAG meal) after fasting for at least 12 h. Blood samples were collected prior to and 0.5, 2, 3, 4, and 6 h after ingestion of the test meal. Postprandial TAG concentrations were significantly lower after the DAG meal compared with the TAG meal. Postprandial TAG, insulin, and GIP concentrations were significantly lower after the DAG meal compared with the TAG meal in 26 subjects with fasting serum TAG levels between1.36 and 2.83 mmol/L. DAG-oil-based meals, as a replacement for TAG oil, may provide cardiovascular benefits in high-risk individuals by limiting lipid and insulin excursions.

KEYWORDS: *diacylglycerol, postprandial, lipids, insulin, glucose-dependent insulinotropic polypeptide (GIP)*

INTRODUCTION

It has been suggested that atherogenesis might represent a postprandial phenomenon,[1] and both postprandial hyperlipidemia and hyperglycemia have been identified as risk markers for coronary artery disease.[2] A recent study reported that insulin resistance might be involved with the acute metabolism of dietary fats.[3] Exaggerated nonfasting concentrations of triacylglycerols (TAG), via higher peak concentrations or delayed clearance, are frequently found even in diabetic patients with normal fasting TAG levels.[4] However, few countermeasures have been devoted to nonfasting hypertriglyceridemia, despite growing recognition that postprandial TAG concentrations may be more significant than fasting concentrations in the assessment of cardiovascular disease risk.[5]

Postprandial TAG concentrations and insulin secretion are highly influenced by fasting TAG concentrations[6] and by the nature of the dietary fats in the meal.[7] Diacylglycerol (DAG) oil, which is an edible oil, was reported to suppress postprandial hyperlipidemia. Postprandial levels of serum TAG were less elevated after the ingestion of DAG oil in animals, compared with the ingestion of triacylglycerol (TAG) oil with the same fatty acid composition.[8,9] DAG oil consumption in humans suppressed increases not only in postprandial TAG but also in remnant lipoproteins (RLP), compared with TAG oil.[10−13]

However, the focus of these studies was on the effects of DAG oil on postprandial lipid metabolism and not glucose metabolism.

The objectives of the current study were therefore to investigate the effects of DAG oil on postprandial lipemia, glycemia, and incretin response with high fasting TAG concentrations.

MATERIALS AND METHODS

Ethics. This study was carried out in accordance with the Declaration of Helsinki and approved by the ethical Committee of Saitama Souka Hospital (Saitama, Japan). All participants received a full explanation of the study and provided written informed consent, under the supervision of the physicians in charge.

Subjects. Women and men, 35−60 years old, with a fasting serum TAG level of 1.36 (120 mg/dL) to 2.83 mmol/L were recruited from the Kanto region of Japan. These inclusion criteria were based on the onset point of increased risk of coronary heart disease according to the results of a Japanese cohort study.[14]

Subjects were excluded if they had underlying disorders that affect lipid metabolism, including cardiorespiratory dysfunction, renal damage, hepatic disease, or diabetes mellitus, as judged by blood and urine data collected at the time of a screening visit or from medical histories. Subjects taking antihyperlipidemic or antihyperglycemic agents, or using food products that affected lipid or glucose metabolism, were also excluded. In addition, premenopausal women were excluded to avoid alterations in metabolism that might be caused by the hormonal cycle. A total of 11 females and 31 males participated in the study.

Test Diets. Diaclyglycerol oil was prepared by esterifying glycerol with fatty acids from soybean and rapeseed oil according to Watanabe's method.[15] The final product was then refined by

Received: November 23, 2011
Revised: February 14, 2012
Accepted: February 20, 2012
Published: March 5, 2012

deodorization and bleaching. The levels of glycidol fatty acid esters and 3-monochloro-1, 2-propanediol fatty acid esters, which have been currently demonstrated to be contaminants derived from food processing,[16] were undetectable in the refined DAG oil, using the method developed by Shiro et al.[17] and DGF Standard Methods,[18] respectively. The product contained 1,3-DAG and 1,2-DAG isomers in a ratio of 7:3 with a minimum total DAG content of approximately 80%. TAG oil was prepared by mixing rapeseed, safflower, and perilla oils to give a final fatty acid composition similar to that of the DAG oil. The fatty acid compositions of the DAG and TAG oils are shown in Table 1. The oils were processed to produce mayonnaise-like food products and added to the test meals (DAG or TAG meal). The content of DAG oil (DAG meal) or TAG oil (TAG meal) in the test meal was 10 g.[11] The meals consisted of a sandwich, shrimp salad, consommé soup, and barley tea. The average total energy was 2089 kJ, with a macronutrient profile of 30.3 g of protein, 18.6 g of fat and 50.1 g of carbohydrate.[11]

Table 1. Composition of DAG and TAG Oils

	DAG oil	TAG oil
glyceride[a] (%)		
TAG	13.1	98.4
DAG	86.4	1.6
MAG	0.5	0.0
fatty acid (%)		
C16:0	2.6	5.3
C18:0	1.0	2.1
C18:1	38.0	37.4
C18:2	48.7	46.4
C18:3	8.8	7.0
others	0.9	1.8

[a]DAG, diacylglycerol; TAG, triacylglycerol; MAG, monoacylglycerol.

Study Protocol. The study had a randomized, double-blind, crossover design. The following measurements were conducted at the screening visit, after overnight fasting for at least 12 h: anthropometric parameters (height, weight, temperature), blood pressure, serum lipids (TAG, nonesterified fatty acid (NEFA), total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol), plasma glucose, hemoglobin A1c, hepatic function (serum alanine aminotransferase, aspartate aminotransferase, γ-glutamyl transpeptidase, alkaline phosphatase, lactate dehydrogenase, and total protein), serum albumin, serum uric acid, serum creatinine, serum total bilirubin, serum urea nitrogen, and urine analysis by urine test strip. The test meals were given in random order after overnight fasting for at least 12 h, with an interval of 2 weeks between meals. Subjects were instructed to maintain their habitual diet and physical activity during the study period and to record their meal contents for 3 days before each visit. Alcohol intake was prohibited for 1 day before each visit.

Blood samples were collected before the ingestion of the meals. to provide baseline values, and at 0.5, 2, 3, 4, and 6 h after the ingestion of the meals. Blood was centrifuged at 1500*g* for 15 min at 4 °C to separate the serum and plasma. The concentrations of serum TAG, apoB-48, insulin, and lipids in lipoproteins (very low density lipoprotein (VLDL), LDL, HDL), plasma glucose, and glucose-dependent insulinotropic polypeptide (GIP) were analyzed. Triacylglycerols were measured using a standard enzymatic colorimetric assay (Sekisui Medical Co., Ltd., Tokyo, Japan). The lipoprotein fractions were separated by ultracentrifugation according to the method reported by Hatch et al.[19] Plasma glucose was measured by a glucose hexokinase method (Shino-Test Corp., Tokyo, Japan). Enzyme-linked immunosorbent assay (ELISA) kits were used for serum insulin (Fujirebio Inc., Tokyo, Japan) with an intraassay CV of 2.54% and an interassay CV of 1.87%, serum apoB-48 (Fujirebio Inc., Tokyo, Japan) with an intraassay CV of 5.80−9.31% and an interassay CV of 3.58−8.28%, and plasma total GIP (Millipore Corp., Bedford, MA, USA) with an intraassay CV of 4.79−6.30% and an interassay CV of 2.20−4.97%. The samples were analyzed by SRL Inc. (Tokyo, Japan).

Statistical Analysis. All results are presented as the mean ± SD unless otherwise stated. Areas under the curves were calculated using the trapezoidal rule and are presented as postprandial responses. Fasting values were compared using paired *t* tests. Differences in blood parameter responses were compared using mixed linear models with repeated measures. Time, treatment, and interaction time × treatment were considered as fixed factors, and subjects were considered as a random effect. Data were analyzed using SPSS 15.0 (SPSS Inc., Chicago, IL, USA). A p value of <0.05 was considered to be significant in all analyses.

RESULTS

Except for one subject who was not able to participate in the ingestion test for personal reasons, all 11 females and 30 males completed the study. Data for these 41 subjects were therefore used for the full analysis set (FAS). The subjects satisfied the inclusion criteria of fasting serum TAG levels of 1.36−2.83 mmol/L at the screening visit, whereas 15 subjects did not satisfy the TAG levels at the ingestion test. Twenty-six subjects were therefore included in the per protocol set (PPS).

The characteristics of the subjects in the FAS and PPS are shown in Table 2. By gender, although height and weight were significantly smaller in females than in males, there was no significant difference in BMI. Also, there were no significant differences between the groups in terms of daily intake of energy, protein, fat, or carbohydrates for 3 days before each visit.

Table 2. Characteristics of Subjects

	full analysis set[a] (N = 41)	per protocol set[a] (N = 26)
male/female	30/11	18/8
age (years)	48.2 ± 7.8	49.2 ± 7.7
height (cm)	165.5 ± 9.2	165.2 ± 9.4
weight (kg)	70.0 ± 11.7	70.2 ± 12.4
BMI[b] (kg/m^2)	25.4 ± 2.9	25.6 ± 3.1

[a]Values are the mean ± SD. [b]BMI, body mass index.

Table 3 shows the concentrations of TAG, apoB-48, VLDL-TAG, LDL-TAG, HDL-TAG, glucose, insulin, and GIP before and 0.5, 2, 3, 4, and 6 h after the ingestion of the meals in the FAS. The concentrations of TAG were significantly lower after the DAG meal compared with the TAG meal. On the incremental areas under the curves (IAUCs) after the meals, the concentrations of not only TAG but also VLDL-TAG and LDL-TAG were significantly lower after the DAG meal compared with the TAG meal. The suppressive effect of DAG oil on postprandial TAG in the PPS was remarkable (IAUCs; $p < 0.001$). Furthermore, the IAUCs of apoB-48, HDL-TAG, insulin, and GIP after ingestion of the DAG meal were significantly lower than those after the TAG meal (Table 4).

DISCUSSION

The main finding of the present study was that DAG oil could reduce not only postprandial lipid but also insulin and GIP secretions in subjects with high fasting TAG concentrations. DAG oil suppressed postprandial serum lipid levels, compared with TAG oil, when administered in the form of an emulsion in prior studies.[10,12,13] This study used a test meal designed by Tomonobu et al.[11] to examine the effects of DAG oil on both

Table 3. Changes in Postprandial Concentration of Serum Lipids, Insulin, Plasma Glucose, and Glucose-Dependent Insulinotropic Polypeptide after Ingestion of Test Meals: Full Analysis Set Population[a]

							p value of ANOVA			
	0 h	0.5 h	2 h	3 h	4 h	6 h	group	time	group × time	IAUC
TAG (mmol/L)										
DAG meal	1.81 ± 0.70	1.83 ± 0.59	2.27 ± 0.71	2.38 ± 0.87	2.26 ± 0.89	1.89 ± 0.74	0.818	0.000	0.023	
TAG meal	1.74 ± 0.70	1.79 ± 0.63	2.36 ± 0.79	2.44 ± 0.95	2.34 ± 0.97	1.88 ± 0.78				
ΔDAG meal	0	0.02 ± 0.22	0.45 ± 0.38	0.57 ± 0.46	0.44 ± 0.46	0.07 ± 0.38	0.041	0.000	0.022	2.10 ± 1.58*
ΔTAG meal	0	0.05 ± 0.17	0.62 ± 0.34	0.70 ± 0.46	0.59 ± 0.47	0.13 ± 0.35				2.69 ± 1.48
apo-B48 (mmol/L)										
DAG meal	0.028 ± 0.013	0.030 ± 0.012	0.046 ± 0.016	0.047 ± 0.017	0.043 ± 0.018	0.036 ± 0.017	0.972	0.000	0.385	
TAG meal	0.027 ± 0.014	0.029 ± 0.015	0.048 ± 0.016	0.047 ± 0.018	0.044 ± 0.018	0.036 ± 0.016				
ΔDAG meal	0	0.003 ± 0.008	0.018 ± 0.011	0.019 ± 0.013	0.016 ± 0.014	0.008 ± 0.013	0.211	0.000	0.409	0.081 ± 0.050
ΔTAG meal	0	0.003 ± 0.006	0.021 ± 0.007	0.021 ± 0.010	0.017 ± 0.011	0.009 ± 0.011				0.087 ± 0.040
VLDL-TAG (mmol/L)										
DAG meal	1.12 ± 0.60	1.17 ± 0.50	1.55 ± 0.64	1.66 ± 0.79	1.51 ± 0.80	1.19 ± 0.64	0.656	0.000	0.106	
TAG meal	1.08 ± 0.59	1.17 ± 0.54	1.65 ± 0.70	1.72 ± 0.85	1.58 ± 0.83	1.19 ± 0.66				
ΔDAG meal	0	0.05 ± 0.21	0.43 ± 0.33	0.54 ± 0.40	0.39 ± 0.40	0.07 ± 0.32	0.063	0.000	0.138	1.95 ± 1.39*
ΔTAG meal	0	0.09 ± 0.15	0.56 ± 0.31	0.64 ± 0.40	0.50 ± 0.37	0.11 ± 0.30				2.38 ± 1.25
LDL-TAG (mmol/L)										
DAG meal	0.44 ± 0.13	0.42 ± 0.12	0.43 ± 0.12	0.43 ± 0.12	0.45 ± 0.12	0.42 ± 0.12	0.742	0.000	0.326	
TAG meal	0.43 ± 0.14	0.41 ± 0.13	0.44 ± 0.12	0.43 ± 0.12	0.44 ± 0.14	0.42 ± 0.13				
ΔDAG meal	0	−0.02 ± 0.04	−0.01 ± 0.04	−0.01 ± 0.06	0.01 ± 0.05	−0.02 ± 0.05	0.183	0.000	0.341	0.08 ± 0.09**
ΔTAG meal	0	−0.02 ± 0.05	0.01 ± 0.07	0.00 ± 0.05	0.02 ± 0.08	−0.01 ± 0.07				0.13 ± 0.12
HDL-TAG (mmol/L)										
DAG meal	0.22 ± 0.05	0.22 ± 0.05	0.24 ± 0.05	0.25 ± 0.05	0.25 ± 0.05	0.23 ± 0.05	0.921	0.000	0.133	
TAG meal	0.22 ± 0.07	0.21 ± 0.06	0.25 ± 0.07	0.25 ± 0.05	0.26 ± 0.07	0.23 ± 0.06				
ΔDAG meal	0	0.00 ± 0.02	0.02 ± 0.03	0.03 ± 0.04	0.03 ± 0.04	0.01 ± 0.04	0.349	0.000	0.086	0.13 ± 0.12
ΔTAG meal	0	−0.01 ± 0.02	0.03 ± 0.03	0.03 ± 0.04	0.04 ± 0.05	0.01 ± 0.05				0.16 ± 0.13
glucose (mmol/L)										
DAG meal	5.07 ± 0.54	6.52 ± 0.86	5.20 ± 0.88	5.00 ± 0.44	4.84 ± 0.37	4.74 ± 0.38	0.592	0.000	0.327	
TAG meal	5.08 ± 0.63	6.70 ± 1.04	5.24 ± 1.00	4.92 ± 0.58	4.86 ± 0.43	4.73 ± 0.39				
ΔDAG meal	0	1.46 ± 0.68	0.14 ± 0.64	−0.07 ± 0.43	−0.23 ± 0.46	−0.33 ± 0.48	0.683	0.000	0.280	2.06 ± 1.35
ΔTAG meal	0	1.63 ± 0.74	0.16 ± 0.77	−0.15 ± 0.52	−0.22 ± 0.48	−0.34 ± 0.52				2.27 ± 1.45
insulin (pmol/L)										
DAG meal	54.0 ± 25.8	326.4 ± 149.7	160.3 ± 84.8	80.1 ± 69.8	43.7 ± 32.0	32.3 ± 15.9	0.217	0.000	0.407	
TAG meal	51.7 ± 26.2	344.7 ± 155.1	181.4 ± 132.6	73.9 ± 64.4	45.9 ± 33.4	32.2 ± 15.5				
ΔDAG meal	0	272.4 ± 140.3	106.2 ± 76.5	26.1 ± 53.7	−10.3 ± 30.0	−21.7 ± 19.7	0.098	0.000	0.482	438.8 ± 188.0
ΔTAG meal	0	293.0 ± 148.2	129.8 ± 121.1	22.2 ± 54.0	−5.8 ± 26.8	−19.5 ± 16.8				487.6 ± 274.0
GIP (pmol/L)										
DAG meal	14.9 ± 16.4	108.0 ± 47.2	96.3 ± 28.9	54.2 ± 31.4	31.8 ± 22.4	14.8 ± 11.3	0.501	0.000	0.752	
TAG meal	15.4 ± 22.2	108.5 ± 40.0	102.2 ± 40.6	53.7 ± 33.2	30.5 ± 22.3	14.5 ± 9.0				
ΔDAG meal	0	93.1 ± 42.2	81.4 ± 31.0	39.3 ± 33.3	17.0 ± 26.7	−0.1 ± 16.0	0.839	0.000	0.625	268.3 ± 110.8
ΔTAG meal	0	93.1 ± 33.8	86.8 ± 38.3	38.3 ± 37.3	15.1 ± 27.8	−0.9 ± 20.2				274.0 ± 110.3

dx.doi.org/10.1021/jf204825p | J. Agric. Food Chem. 2012, 60, 2490–2496

Table 3. continued

[a]Values are the mean ± SD ($N = 41$). Statistical differences are based on repeated-measures ANOVA. Significantly different from the TAG meal: *, $p < 0.05$; **, $p < 0.01$. Participants ingested either a triacylglycerol meal (TAG meal) or a diacylglycerol meal (DAG meal). TAG, triacylglycerols; VLDL, very low density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein; GIP, glucose-dependent insulinotropic polypeptide; IAUCs, incremental areas under the curves.

postprandial lipid and glucose metabolism, thereby reducing postprandial TAG as well as insulin and GIP levels compared with TAG oil.

Previous studies have demonstrated that TAG resynthesis is reduced in the small intestinal epithelium after the intake of DAG intake, compared with after TAG intake, indicating a possible mechanism for the suppressive effect of DAG oil on postprandial TAG.[20] The lymphatic transport of chylomicrons has also been shown to be significantly delayed and reduced after 1,3-DAG, a major constituent of DAG oil, ingestion,[21] presumably as a result of poor re-esterification of fatty acids with 1-monoacylglycerol in the intestinal mucosa.[22] The concentration of apoB-48 in chylomicrons in the current study was significantly lower after the DAG meal, compared with the TAG meal.

Furthermore, the TAG concentrations of VLDL and LDL in lipoprotein fractions were also lower after the DAG meal compared with the TAG meal. The deposition of oxidatively modified LDL in the intima is an important initial event in atherogenesis,[23] and oxidized VLDL may also be involved.[24] Some studies have suggested that postprandial LDL particles are more easily oxidized than fasting LDL.[25] Similarly, postprandial VLDL particles may be more prone to oxidation than fasting VLDL. This could be partially attributed to elevated postprandial TAG levels, which would result in competition for lipolysis between chylomicrons and VLDL. This in turn could cause increased plasma residence time of VLDL, thereby increasing the risk of interactions with the cardiac wall. However, relationships between the suppressive effect of DAG oil on postprandial VLDL and the concentration of oxidized VLDL have not been reported. Clearly, additional studies designed to clarify these relationships need to be conducted.

Shimotoyodome et al. reported that fat administration, in combination with glucose, augmented the concentrations of insulin and GIP in animals compared with glucose alone and that those concentrations were lowered by replacing fat with DAG oil from TAG oil.[26] Meanwhile, they reported that pluronic L-81, an inhibitor of chylomicron formation, significantly attenuates fat-induced plasma TAG, as well as GIP response.[26] This suggests that fat-induced GIP secretion is associated with chylomicron formation in the intestinal mucosa. In this study, the concentrations of both insulin and GIP were lower after the DAG meal, compared with the TAG meal, and the concentration of apoB-48 in chylomicrons was also significantly lower after the DAG, compared with the TAG meal. The lowering of GIP after the ingestion of DAG oil in this study may be attributed to retarded chylomicron formation. It is possible that DAG oil decreased postprandial GIP secretion, thereby reducing insulin secretion compared with TAG oil.

A number of earlier studies reported that hypertriglyceridemic subjects had peripheral hyperinsulinemia and increased peripheral insulin resistance.[27−29] Gama et al. reported that postprandial GIP concentrations were greater in hypertriglyceridemic subjects than in normotriglyceridemic subjects.[6] This suggests that the postprandial elevation in GIP concentrations in the hypertriglyceridemic patients might be related to their hypertriglyceridemia and/or hyperinsulinemia. In the current study, the concentrations of GIP and insulin were lower after the ingestion of the DAG meal compared with after the ingestion of the TAG meal. The suppressive effects of DAG meal on postprandial GIP and insulin may contribute to

Table 4. Changes in Postprandial Concentration of Serum Lipids, Insulin, Plasma Glucose, and Glucose-Dependent Insulinotropic Polypeptide after Ingestion of Test Meals (1.36 mmol/L < TG < 2.83 mmol/L): Per Protocol Set Population[a]

							p value of ANOVA			
	0 h	0.5 h	2 h	3 h	4 h	6 h	group	time	group × time	IAUC
TAG (mmol/L)										
DAG meal	2.00 ± 0.45	1.94 ± 0.35	2.43 ± 0.50	2.55 ± 0.62	2.44 ± 0.63	2.01 ± 0.50	0.623	0.000	0.047	
TAG meal	1.89 ± 0.52	1.93 ± 0.47	2.55 ± 0.60	2.70 ± 0.76	2.60 ± 0.81	2.05 ± 0.63				
ΔDAG meal	0	−0.06 ± 0.17	0.43 ± 0.38	0.55 ± 0.45	0.44 ± 0.45	0.01 ± 0.39	0.005	0.000	0.206	1.92 ± 1.52***
ΔTAG meal	0	0.05 ± 0.18	0.66 ± 0.33	0.81 ± 0.45	0.71 ± 0.47	0.16 ± 0.39				3.08 ± 1.44
apo-B48 (mmol/L)										
DAG meal	0.031 ± 0.014	0.031 ± 0.012	0.048 ± 0.016	0.049 ± 0.016	0.046 ± 0.017	0.038 ± 0.016	0.810	0.000	0.066	
TAG meal	0.027 ± 0.013	0.030 ± 0.015	0.048 ± 0.016	0.050 ± 0.017	0.047 ± 0.016	0.038 ± 0.016				
ΔDAG meal	0	0.000 ± 0.006	0.017 ± 0.010	0.018 ± 0.011	0.015 ± 0.012	0.008 ± 0.012	0.004	0.000	0.826	0.074 ± 0.042**
ΔTAG meal	0	0.003 ± 0.006	0.021 ± 0.007	0.023 ± 0.009	0.020 ± 0.010	0.011 ± 0.012				0.095 ± 0.038
VLDL-TAG (mmol/L)										
DAG meal	1.26 ± 0.41	1.24 ± 0.32	1.67 ± 0.47	1.79 ± 0.60	1.64 ± 0.58	1.27 ± 0.45	0.469	0.000	0.019	
TAG meal	1.20 ± 0.47	1.28 ± 0.42	1.81 ± 0.58	1.93 ± 0.74	1.78 ± 0.72	1.33 ± 0.57				
ΔDAG meal	0	−0.02 ± 0.16	0.41 ± 0.34	0.53 ± 0.41	0.38 ± 0.39	0.01 ± 0.33	0.006	0.000	0.377	1.80 ± 1.32***
ΔTAG meal	0	0.08 ± 0.16	0.61 ± 0.29	0.74 ± 0.40	0.59 ± 0.36	0.13 ± 0.32				2.69 ± 1.24
LDL-TAG (mmol/L)										
DAG meal	0.46 ± 0.13	0.43 ± 0.11	0.44 ± 0.12	0.45 ± 0.12	0.48 ± 0.12	0.44 ± 0.12	0.735	0.000	0.294	
TAG meal	0.45 ± 0.14	0.42 ± 0.12	0.45 ± 0.11	0.45 ± 0.11	0.48 ± 0.12	0.43 ± 0.10				
ΔDAG meal	0	−0.03 ± 0.04	−0.02 ± 0.04	−0.01 ± 0.06	0.02 ± 0.05	−0.02 ± 0.05	0.393	0.000	0.217	0.07 ± 0.08**
ΔTAG meal	0	−0.03 ± 0.04	0.01 ± 0.07	0.00 ± 0.06	0.03 ± 0.09	−0.02 ± 0.07				0.14 ± 0.14
HDL-TAG (mmol/L)										
DAG meal	0.24 ± 0.05	0.23 ± 0.04	0.25 ± 0.05	0.26 ± 0.05	0.26 ± 0.05	0.24 ± 0.05	0.842	0.000	0.564	
TAG meal	0.23 ± 0.06	0.22 ± 0.05	0.25 ± 0.05	0.26 ± 0.04	0.27 ± 0.07	0.25 ± 0.05				
ΔDAG meal	0	−0.01 ± 0.02	0.01 ± 0.03	0.02 ± 0.03	0.02 ± 0.04	0.00 ± 0.04	0.112	0.000	0.464	0.10 ± 0.11*
ΔTAG meal	0	−0.01 ± 0.02	0.02 ± 0.03	0.03 ± 0.04	0.04 ± 0.06	0.02 ± 0.05				0.17 ± 0.13
glucose (mmol/L)										
DAG meal	5.10 ± 0.46	6.59 ± 0.73	5.27 ± 0.80	5.05 ± 0.47	4.88 ± 0.39	4.72 ± 0.41	0.316	0.000	0.229	
TAG meal	5.14 ± 0.59	6.95 ± 1.02	5.39 ± 1.03	5.02 ± 0.53	4.85 ± 0.41	4.71 ± 0.34				
ΔDAG meal	0	1.49 ± 0.58	0.17 ± 0.57	−0.05 ± 0.39	−0.22 ± 0.40	−0.38 ± 0.47	0.517	0.000	0.177	2.06 ± 0.90
ΔTAG meal	0	1.81 ± 0.78	0.25 ± 0.81	−0.12 ± 0.37	−0.29 ± 0.42	−0.43 ± 0.49				2.46 ± 1.16
insulin (pmol/L)										
DAG meal	57.7 ± 28.3	343.9 ± 157.6	180.9 ± 86.8	96.1 ± 82.6	49.7 ± 38.3	36.1 ± 17.4	0.051	0.000	0.255	
TAG meal	56.8 ± 28.1	387.3 ± 158.6	212.7 ± 147.0	90.9 ± 74.8	52.9 ± 38.2	35.9 ± 17.5				
ΔDAG meal	0	286.2 ± 146.4	123.1 ± 75.2	38.3 ± 62.6	−8.1 ± 35.2	−21.7 ± 20.2	0.053	0.000	0.217	485.9 ± 192.3*
ΔTAG meal	0	330.4 ± 151.9	155.9 ± 137.1	34.1 ± 64.9	−4.0 ± 32.4	−21.0 ± 17.3				573.2 ± 288.4
GIP (pmol/L)										
DAG meal	13.4 ± 5.5	113.0 ± 48.1	101.5 ± 28.2	60.4 ± 35.0	36.7 ± 25.9	17.0 ± 12.4	0.266	0.000	0.029	
TAG meal	11.8 ± 6.8	113.4 ± 30.8	110.4 ± 42.6	63.9 ± 37.3	36.0 ± 25.4	16.2 ± 9.3				
ΔDAG meal	0	99.6 ± 47.5	88.1 ± 25.7	47.1 ± 34.1	23.4 ± 25.8	3.6 ± 12.9	0.022	0.000	0.460	298.3 ± 117.4*
ΔTAG meal	0	101.7 ± 32.0	98.6 ± 39.9	52.1 ± 34.4	24.2 ± 23.2	4.4 ± 8.1				318.8 ± 107.0

dx.doi.org/10.1021/jf204825p | J. Agric. Food Chem. 2012, 60, 2490–2496

Table 4. continued

[a]Values are the means ± SD (N = 26). Statistical differences are based on repeated-mesures ANOVA. Significantly different from the TAG meal: *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$. Participants ingested either a triacylglycerol meal (TAG meal) or a diacylglycerol meal (DAG meal). TAG, triacylglycerols; VLDL, very low density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein; GIP, glucose-dependent insulinotropic polypeptide; IAUCs, incremental areas under the curves.

the amelioration of hyperinsulinemia in hypertriglyceridemic patients.

Previous postprandial studies showed that high-fat meals have the potential to induce β-cell dysfunction and insulin resistance in healthy individuals[30,31] and in subjects with type 2 diabetes[32] or metabolic syndrome.[33] Lopez et al. hypothesized that the transition from normal to impaired glucose tolerance, and then to overt diabetes, hinges on the nature of the postprandial lipid excursions.[34] The authors reported that postprandial TAG and insulin concentrations were lower after the ingestion of a monounsaturated fatty acid meal than after the ingestion of a saturated fatty acid meal in subjects with high fasting TAG concentrations, but there was no effect on glucose responses.[7] In the current study, the concentrations of TAG and insulin were lower after the ingestion of a DAG meal compared with after the ingestion of a TAG meal.

The present study demonstrated the efficacy of DAG oil by single-dose oral test. A further study in the evaluation of postprandial metabolism by long-term intake of DAG oil should be conducted in the future.

In conclusion, the results of the present study confirm that DAG oil is superior to TAG oil in terms of reducing postprandial lipid and insulin secretion in subjects with high fasting triacylglycerol concentrations. These data also suggest that DAG-oil-based meals, as a replacement for TAG oil, may provide cardiovascular benefits in high-risk individuals by limiting lipid and insulin excursions.

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Funding

This study was supported by Kao Corp.

Notes

The authors declare no competing financial interest.

ABBREVIATIONS USED

DAG, diacylglycerol; TAG, triacylglycerol; VLDL, very low-density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein; MAG, monoacylglycerol; SD, standard deviation; ANOVA, analysis of variance; IAUCs, incremental areas under the curves; GIP, glucose-dependent insulinotropic polypeptide; RLP, remnant lipoproteins; ELISA, enzyme-linked immunosorbent assay; FAS, full analysis set; PPS, per protocol set

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