Effects of different nutrient intakes on daytime triacylglycerolemia in healthy, normolipemic, free-living men[1–3]

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ABSTRACT

Background: Postprandial studies with standardized mixed meals have shown that ingestion of high-carbohydrate diets is associated with elevated plasma triacylglycerol (TG) concentrations.

Objective: We evaluated the effects of different nutritional components on daytime triacylglycerolemia in 58 healthy, free-living, normolipemic men.

Design: Capillary TG (TGc) was self-measured at 6 fixed time points over 3 d. Daytime TGc profiles were calculated as areas under the curve (AUCs) for absolute and incremental changes in TGc concentrations (TGc-AUC and ΔTGc-AUC, respectively). Food intake was recorded in a diary.

Results: The mean ($\pm$SD) fasting TGc concentration, TGc-AUC, and ΔTGc-AUC were 1.20 ± 0.41 mmol/L, 24.1 ± 6.9 mmol·h/L, and 7.3 ± 4.5 mmol·h/L, respectively. Mean total energy intake was $10\,881 \pm 2536$ kJ/d. Total intakes of fat, carbohydrate, and protein were 95 ± 25 (33% of energy), 304 ± 69 (48% of energy), and 101 ± 27 (16% of energy) g/d, respectively. Fasting TGc concentrations and TGc-AUC were not related to dietary intake. The mean ΔTGc-AUC was significantly related to total carbohydrate ($r = 0.38$, $P < 0.005$), protein ($r = 0.29$, $P < 0.05$), and energy ($r = 0.28$, $P < 0.05$) intakes. Fat intake (as a % of energy) was negatively associated with the mean ΔTGc-AUC ($r = -0.30$, $P < 0.05$). When the study group was subdivided into tertiles on the basis of fat intake (27.2%, 33.5%, and 39.1% of energy, respectively), carbohydrate intake was 50.9%, 48.1%, and 44.6% of energy, respectively. ΔTGc-AUC was significantly lower at the highest tertile of fat intake (4.8 ± 4.3 mmol·h/L) than at the lowest (8.2 ± 4.0 mmol·h/L) and intermediate (8.9 ± 4.3 mmol·h/L) tertiles ($P < 0.05$ for each).

Conclusion: ΔTGc-AUC is associated with the carbohydrate content of the diet in free-living men. *Am J Clin Nutr* 2001; 74:171–8.

KEY WORDS Diet, carbohydrates, triacylglycerol, postprandial lipemia, daytime triacylglycerolemia, free-living men

INTRODUCTION

Plasma triacylglycerol (TG) concentrations vary considerably throughout the day, mainly because of food intake (1). The effects of different nutritional components on postprandial TG metabolism have been widely investigated in metabolic ward studies. When tested after oral fat loads, the magnitude of post-prandial lipemia increases stepwise with the fat content of the meal (2, 3); however, this finding does not necessarily apply to the usual daily situation. Food intake usually consists of a mixture of different nutritional components, and acute oral tests with mixed meals have shown that the degree of postprandial lipemia is significantly influenced by the carbohydrate content of the meal (4–6). For example, both fasting and postprandial TG concentrations in healthy volunteers were significantly higher 2 wk after consumption of high-carbohydrate, low-fat diets than after consumption of low-carbohydrate, high-fat diets (7). Because postprandial hypertriacylglycerolemia has been linked to atherosclerosis in different patient groups (8–14), the relation between high-carbohydrate diets and TG concentrations is important, especially because high-carbohydrate, low-fat diets are recommended to reduce the risk of coronary heart disease (CHD) (15).

Generally, postprandial studies are performed with acute oral tests in fasting subjects and extremely high or extremely low proportions of carbohydrate and fat proportions are compared (6, 7). However, in the free-living situation, subjects consume food as part of 3–6 eating occasions throughout the day and intakes are generally much lower than those used in postprandial studies (2, 9–14, 16–18). It is important to realize that this standardization in postprandial studies leads to an artificial situation that does not necessarily reflect the free-living situation (19, 20). In the current study we evaluated the effects of different nutrient intakes on daytime triacylglycerolemia in healthy, normolipemic, free-living men.

SUBJECTS AND METHODS

Subjects

Healthy normolipemic men aged 20–65 y were recruited by advertisement. Exclusion criteria were as follows: a fasting

plasma cholesterol concentration >6.5 mmol/L; a fasting plasma TG concentration >2.3 mmol/L; a body mass index (BMI; in kg/m^2) >30; smoking, renal, or liver disease; diabetes mellitus; the use of lipid-lowering medication; and a family history of premature myocardial infarction or type 2 diabetes mellitus. On the morning of inclusion, blood pressure and waist-to-hip ratio were measured. Body fat mass was estimated by bioelectrical impedance analysis (RJL Systems, Detroit) according to instructions provided by the manufacturer (21, 22). All subjects gave written, informed consent before participating. The study was approved by the Medical Ethical Committee of the University Medical Center (Utrecht, Netherlands).

Self-measurements of TGc

Capillary TG (TGc) was self-measured with a TG-specific point-of-care testing device (Accutrend GCT; Roche Diagnostics, Mannheim, Germany) (23, 24) after the subjects received instructions from the same investigator. Subjects were instructed to wash and dry their hands thoroughly before each measurement. A drop of blood (30 μL) obtained from the finger with a lancing device was applied to the test strip in the device. Subsequently, the TG concentration from the capillary blood sample was measured by a process of dry chemistry and colorimetry. If there was not enough blood on the test strip, subjects were asked to repeat the measurement. The reference range for TGc is 0.80–6.86 mmol/L. In a previous study, CVs for different TGc concentrations ranged from 3.3% to 5.3% (25). The correlation coefficient between capillary and plasma TG concentrations with the device used is 0.94 according to enzymatic methods (25). Similar results were obtained in our laboratory (23, 26).

Subjects were instructed to measure their TGc concentrations on 3 different days (preferably Monday, Wednesday, and Friday; not on weekends) at the following 6 time points: fasting, before and 3 h after lunch and dinner, and at bedtime. The 3-h postprandial measurements were performed exactly 3 h after the meals, regardless of the intake of snacks, and the results were recorded in a diary. Subjects were requested to refrain from heavy physical activity, although normal daily activities such as riding a bike to work, were allowed. When one or more measurements were missing for a day, the data for that particular day were not used to create an average daytime TGc profile. The mean daytime TGc profile was used for statistical analysis.

Dietary intake

Dietary intake and time of intake were recorded in the same diary in which TGc concentrations were recorded. Subjects received no recommendations concerning the frequency and composition of the meals and were requested to consume their usual diet during the study. Dietary intake was recorded according to 3 major eating occasions: breakfast and snacks (in the morning), lunch, and dinner. In addition, snacks consumed in the afternoon and evening were also recorded. Quantities of intake were estimated according to instructions given by a dietitian and by using a table with standardized portion sizes (27). Other details, such as illness, were also recorded in the diary. The diaries were evaluated by a trained physician together with each subject. Foods consumed were converted into nutrients by using the Dutch Nutrient Database (28). Dietary intakes were compared with Dutch recommendations for a healthy diet and with the average diet in the Netherlands (27, 28). Dietary

intakes were calculated per eating occasion, per day, and as an average of 2 or 3 d.

Analytic determinations

On the morning of inclusion, fasting blood was collected for measurement of plasma lipid, apolipoprotein (apo), insulin, and glucose concentrations. Plasma cholesterol and TG concentrations were measured in duplicate by colorimetric assay with the CHOD-PAP and GPO-PAP kits (Roche, Germany), respectively (14, 29). HDL cholesterol was determined according to the method of Gidez (30). Plasma apo B was measured with nephelometry by using apo B monoclonal antibodies (OSAN 14/15; Behring Diagnostics NV, Marburg, Germany). Plasma apo A-I was measured with nephelometry by using apo A-I monoclonal antibodies (OUED 14/15; Behring Diagnostics NV). Glucose was measured by glucose oxidase dry chemistry (Vitros GLU slides; Johnson & Johnson, Clinical Diagnostics, Rochester, NY) and colorimetry, and insulin was measured by using a competitive radioimmunoassay with polyclonal antibodies. The HOMA (homeostasis model assessment) index [(glucose $\times$ insulin)/22.5] was calculated to estimate insulin sensitivity (31).

Statistics

Data are given as means ±SDs. Daytime TGc profiles were calculated as areas under the curve (AUCs) for absolute and incremental changes in TGc concentrations (TGc-AUC and ΔTGc-AUC, respectively), after correction for fasting TGc concentrations. Dietary intakes and TGc-AUC were calculated by using averages over 2 or 3 d. Differences in dietary intakes or TGc-AUC between 3 separate days were tested by paired t test. Univariate regression analysis was used to study associations between TGc-AUC and other variables. Stepwise multiple regression analysis was performed with TGc-AUC and ΔTGc-AUC as dependent variables and with the significantly associated variables identified by univariate regression analysis as independent variables. The study group was subdivided into tertiles on the basis of fat and carbohydrate intakes expressed as a percentage of total energy intake. Tertiles were compared by using repeated-measures analysis of variance with post hoc Fisher's least-significant-different test followed by Bonferroni correction of the P value. Comparisons by the same method were also made between the 3 major eating occasions and between the 3 groups. Plasma TG and insulin concentrations and the HOMA index were analyzed after logarithmic transformation because of the nonparametric distribution of these variables. SPSS (version 9.0; SPSS Inc, Chicago) was used for the statistical analysis. TGc-AUC and ΔTGc-AUC were calculated with PRISM (version 3.0; Graph Pad Software, San Diego) by using nonlogarithmically transformed TGc concentrations. Statistical significance was set at $P < 0.05$ (two sided).

RESULTS

Subject characteristics

Baseline characteristics of the subjects are given in **Table 1**. A total of 63 men were screened. Two subjects were excluded because they had a positive family history of myocardial infarction, 2 because they had an elevated BMI (>30), and 1 because he was a current smoker; thus, 58 subjects were included in the

TABLE 1
Baseline characteristics of the study group[1]

	Value
Age (y)	36.6 ± 13.2 (20–60)
Weight (kg)	78.3 ± 9.2 (59–108)
BMI (kg/m²)	23.1 ± 2.3 (18.3–28.4)
Waist-to-hip ratio	0.86 ± 0.07 (0.73–1.02)
Fat mass	
(kg)	13.4 ± 3.4 (4.5–23.4)
(%)	17.0 ± 4.0 (7.6–25.8)
Blood pressure (mm Hg)	
Systolic	122 ± 12 (90–155)
Diastolic	77 ± 7 (60–95)
Glucose (mmol/L)	4.80 ± 0.79 (3.12–6.40)
Insulin (pmol/L)	48.42 ± 16.86 (30–102)
HOMA index	1.75 ± 0.74 (0.69–3.70)
Cholesterol (mmol/L)	4.85 ± 0.92 (3.05–6.24)
Plasma triacylglycerol (mmol/L)	1.12 ± 0.47 (0.54–2.08)
HDL cholesterol (mmol/L)	1.26 ± 0.30 (0.61–2.17)
Apolipoprotein A-I (g/L)	1.34 ± 0.19 (0.88–1.78)
Apolipoprotein B (g/L)	0.95 ± 0.22 (0.42–1.38)

[1] $\bar{x}$ ± SD; range in parentheses. n = 58. HOMA, homeostasis model assessment.

study. Fasting plasma cholesterol and TG concentrations met the inclusion criteria. None of the subjects was diabetic or used lipid-lowering medication.

Daytime TGc profiles and dietary intakes

The mean daytime TGc profile of the study group is shown in **Figure 1**. In 16 subjects, mean daytime TGc profiles were based on 2 instead of 3 d of data because of missing TGc values on 1 d. The mean fasting TGc concentration was 1.20 ± 0.41 mmol/L. TGc increased significantly in the morning, resulting in a prelunch concentration (1.50 ± 0.58 mmol/L) that was significantly different from fasting. TGc concentrations increased significantly from 1.50 ± 0.58 mmol/L before lunch to 1.89 ± 0.66 mmol/L 3 h after lunch, an increase of 0.39 ± 0.61 mmol/L. The largest increase in TGc between 2 adjacent time points was from 1.66 ± 0.57 mmol/L before dinner to 2.35 ± 0.91 mmol/L after dinner (P < 0.005), an increase of 0.69 ± 0.75 mmol/L. At bedtime there was a slight decline in TGc concentrations, from 2.35 ± 0.91 mmol/L 3 h after dinner to 2.28 ± 0.88 mmol/L (NS). All daytime TGc concentrations were significantly greater than the fasting concentration. Mean TGc-AUC and ΔTGc-AUC were 24.1 ± 6.9 and
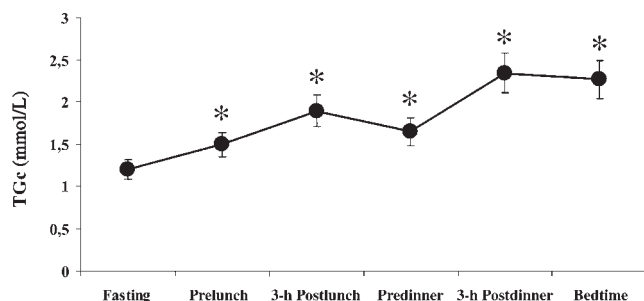

FIGURE 1. Mean (±SEM) daytime capillary triacylglycerol (TGc) concentrations in 58 healthy men during the different eating occasions over 2 or 3 d. *Significantly different from fasting, P < 0.05.

7.3 ± 4.5 mmol·h/L, respectively. There were no significant differences between fasting TGc concentrations, TGc-AUC, and ΔTGc-AUC on the different measurement days (data not shown).

The mean absolute food intake and intake as a percentage of energy are shown in **Table 2**. Forty-four subjects (76% of the study group) consumed alcohol on one or more of the 3 study days. There were no significant differences in dietary intake between the different measurement days (data not shown). Mean intakes per eating occasion are shown in **Table 3**. All subjects ingested food during the 3 major eating occasions. There was little variation in intakes of afternoon and evening snacks (data not shown). The highest energy and fat intakes occurred at dinner. The absolute carbohydrate intake was not significantly different between the 3 major eating occasions.

Energy intakes and relative intakes of alcohol, protein, carbohydrate, and fat at the different eating occasions are shown in **Figure 2**. Fat intake as a percentage of energy was higher at dinner than at lunch (39% compared with 32%, respectively; P < 0.001), whereas carbohydrate intake as a percentage of energy was higher at lunch than at dinner (51% compared with 39%, respectively; P < 0.001).

Associations between fasting TGc concentrations, ΔTGc-AUC, and nutrient intakes

There were no significant associations between fasting TGc concentrations, TGc-AUC, and absolute and relative intakes of carbohydrate, energy, protein, and fat (data not shown). In contrast, ΔTGc-AUC was associated with absolute intakes of carbohydrate, energy, and protein (**Figure 3**). The best correlation was with carbohydrate intake. There was no correlation between ΔTGc-AUC and intakes of alcohol (r = 0.10) and fat (r = 0.07). Fat intake (r = −0.30, P < 0.05), saturated fat intake (r = −0.29, P < 0.05), and polyunsaturated fat intake (r = −0.26, P < 0.05)

TABLE 2
Dietary intakes of subjects[1]

	Intake
Energy (kJ)	10 881 ± 2536
Total fat	
(g)	95 ± 25
(% of energy)	33.3 ± 5.6
Saturated fat	
(g)	35.5 ± 9.2
(% of energy)	12.5 ± 2.1
MUFA	
(g)	36.4 ± 11.2
(% of energy)	12.7 ± 3.0
PUFA	
(g)	14.8 ± 4.7
(% of energy)	5.2 ± 1.4
Carbohydrate	
(g)	304 ± 69
(% of energy)	47.9 ± 5.4
Protein	
(g)	101 ± 27
(% of energy)	15.9 ± 2.4
Alcohol	
(g)	18.3 ± 21.9
(% of energy)	4.9 ± 25.4
Cholesterol (mg)	208 ± 84

[1] $\bar{x}$ ± SD of 2 or 3 d; n = 58. MUFA, monounsaturated fat; PUFA, polyunsaturated fat.

TABLE 3
Dietary intakes of subjects per eating occasion[1]

	Breakfast	Lunch	Afternoon snack	Dinner	Evening snack	P (ANOVA)
Total energy (kJ)	2227 ± 924	2922 ± 980[2]	873 ± 587	3419 ± 789[2,3]	1440 ± 1022	<0.001
Total fat (g)	17.6 ± 10.3	25.4 ± 12.0[2]	7.4 ± 7.7	34.9 ± 12.5[2,3]	10.5 ± 9.8	<0.001
Saturated fat (g)	6.4 ± 3.9	9.0 ± 4.5[2]	3.2 ± 3.9	13.0 ± 5.4[2,3]	3.9 ± 3.5	<0.001
MUFA (g)	5.6 ± 3.9	8.9 ± 5.0[2]	2.7 ± 2.9	14.9 ± 6.1[2,3]	4.4 ± 4.6	<0.001
PUFA (g)	3.0 ± 1.9	4.3 ± 2.5[2]	0.8 ± 1.1	5.2 ± 2.3[2]	1.5 ± 1.7	<0.001
Carbohydrate (g)	76 ± 29	87 ± 29	30 ± 19	78 ± 23	34 ± 23	NS
Protein (g)	18.8 ± 9.6	30.0 ± 12.8[2]	5.1 ± 4.9	40.1 ± 12.3[2,3]	6.4 ± 6.6	<0.001

[1] $\bar{x}$ ± SD of 2 or 3 d; n = 58. MUFA, monounsaturated fat; PUFA, polyunsaturated fat. Statistical comparisons were made only between breakfast, lunch, and dinner.
[2] Significantly different from breakfast, $P < 0.05$.
[3] Significantly different from lunch, $P < 0.05$.

as a percentage of energy were negatively associated with ΔTGc-AUC. Carbohydrate and protein intakes as a percentage of energy ($r = 0.21$ and 0.13, respectively) showed no significant association with ΔTGc-AUC. Fasting insulin was the only nondietary variable associated with ΔTGc-AUC ($r = 0.29$, $P < 0.05$). Stepwise multiple regression analysis with ΔTGc-AUC as the dependent variable showed that carbohydrate intake (standardized β = 0.13) was the only variable included in the model, explaining 13% of the variation ($P < 0.005$).

Fasting TGc ($r = 0.76$, $P < 0.001$), relative fat mass ($r = 0.33$, $P < 0.05$), absolute fat mass ($r = 0.30$, $P < 0.05$), fasting insulin ($r = 0.29$, $P < 0.05$), and systolic blood pressure ($r = 0.29$, $P < 0.05$) were the nondietary variables that correlated with TGc-AUC. Stepwise multiple regression analysis showed that fasting TGc (standardized β = 0.79) was the best predictor of TGc-AUC, predicting 62% of the variation.

ΔTGc-AUC in subjects by tertiles of fat and carbohydrate intakes

Characteristics of the study group by tertiles of carbohydrate intake as a percentage of energy are shown in **Table 4**. There were no significant differences with respect to age, anthropometric variables, insulin sensitivity, or fasting plasma lipid concentrations between the tertiles. Total energy intake and absolute carbohydrate intake were also not significantly different between

the tertiles. Fat intake as a percentage of energy and absolute fat intake were significantly higher in the lowest tertile than in the highest tertile. There were no significant differences in fasting TGc, TGc-AUC, and ΔTGc-AUC between the tertiles.

Characteristics of the study group by tertiles of fat intake as a percentage of energy are shown in **Table 5**. There were no significant differences with respect to age, anthropometric variables, fasting plasma lipids, fasting apolipoproteins, or insulin sensitivity between the tertiles. Carbohydrate intake as a percentage of energy was significantly lower in the highest tertile than in the other 2 tertiles. Absolute fat and carbohydrate intakes were also significantly different between the tertiles, with carbohydrate intake being the highest in the intermediate tertile. Total energy intake was also the highest in the intermediate tertile. Fasting TGc concentrations and TGc-AUC were not significantly different among the tertiles. ΔTGc-AUC was significantly lower in the highest tertile than in the other 2 tertiles.

DISCUSSION

The results of the present study provide information on the effects of dietary intake on daytime triacylglycerolemia in free-living men. Ambulatory daytime TGc profiles were determined with a novel technique, measurement of serial TGc (23, 24, 26). In a recent report we showed that daytime TGc profiles of 48 healthy male subjects were associated with insulin sensitivity, body composition, and diet (26). In the current study, we evaluated in detail the effects of dietary intake on daytime triacylglycerolemia in a larger group of men.

Postprandial studies have shown that high-carbohydrate, low-fat diets increase fasting and postprandial TG concentrations (4, 5, 7) and postprandial hypertriacylglycerolemia has been linked to CHD in normolipemic individuals (8–12). Given the repetition of meals consumed in the free-living situation and the relatively long duration of the postprandial state, an important remodeling of HDL cholesterol and LDL particles occurs throughout the day and these changes have been shown to be associated with an increased risk of CHD (9, 12, 32–36). Nevertheless, high-carbohydrate, low-fat diets are generally recommended to reduce the risk of CHD (15). Postprandial studies, however, may not provide a realistic impression of the free-living situation; daytime TGc profiles may help to overcome this problem. The easy operation of the TG analyzer used in the current study enables subjects to determine their own daytime TGc profile through a simple finger prick (23, 24).
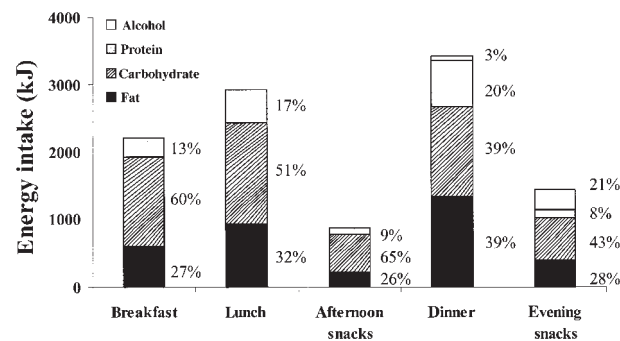

FIGURE 2. Mean energy intakes and relative intakes of alcohol, protein, carbohydrate, and fat during the different eating occasions over 2 or 3 d. Both relative fat and relative carbohydrate intakes were significantly different between the 3 eating occasions ($P < 0.01$ for all comparisons). The relative protein intake was significantly greater at dinner than at breakfast ($P < 0.05$).
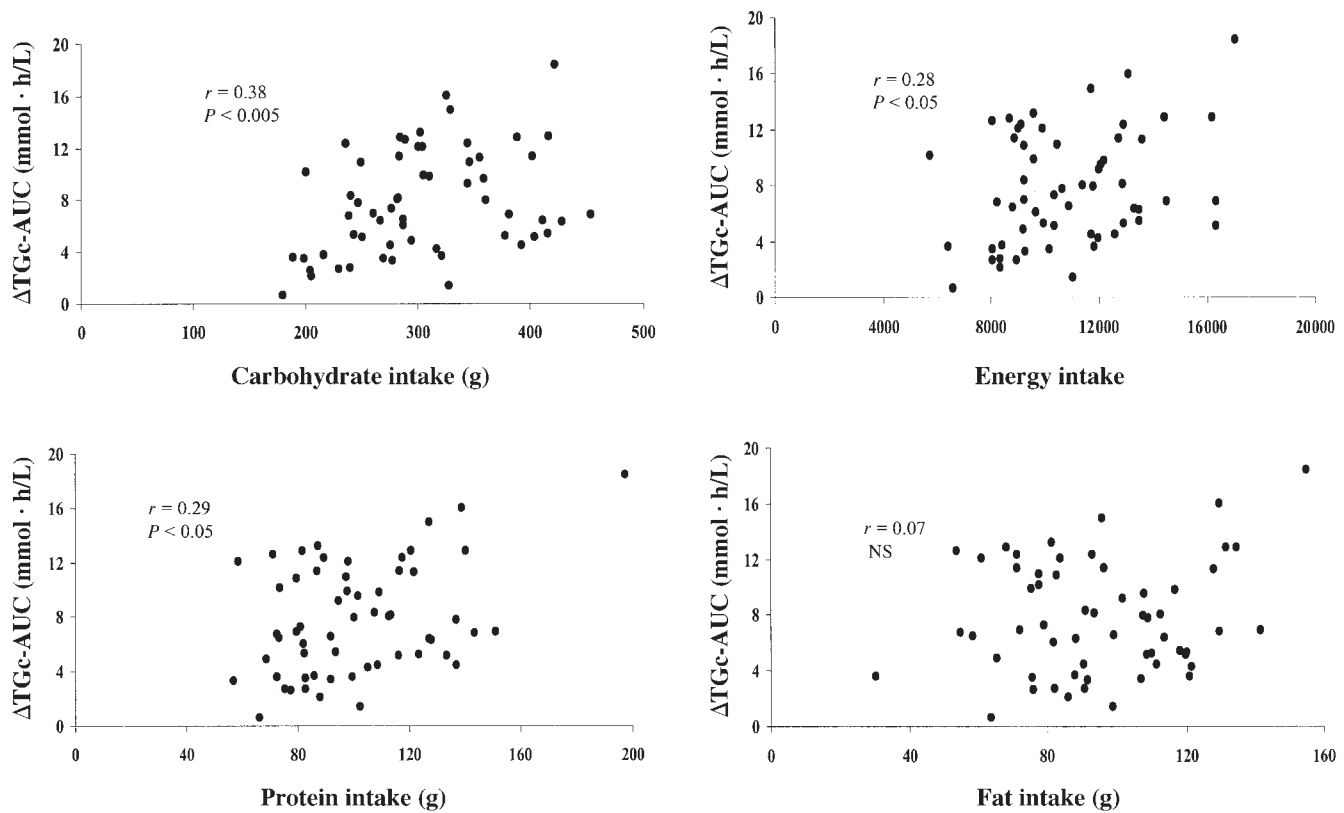
FIGURE 3. Correlation between mean daytime incremental triacylglycerol concentrations calculated as areas under the curve (ΔTGc-AUC) and mean daily intakes of carbohydrate, energy, protein, and fat. Note that when the outlier with the highest value of total protein intake was deleted, the correlation coefficient was $r = 0.25$ ($P = 0.07$).

TABLE 4
Characteristics of the study group by tertile of carbohydrate intake[1]

	Low ($n = 19$)	Intermediate ($n = 20$)	High ($n = 19$)	P (ANOVA)
		Tertile of carbohydrate intake[2]		
Carbohydrate (g/d)	280 ± 70	305 ± 71	328 ± 62	NS
Total energy (kJ/d)	11344 ± 2874	10938 ± 2573	10360 ± 2147	NS
Total fat				
(g/d)	106.1 ± 22.4	95.1 ± 27.9	83.7 ± 18.9[3]	0.017
(% of energy)	36.1 ± 4.5	32.7 ± 5.2	31.1 ± 6.1[3]	0.033
Protein				
(g/d)	112.4 ± 29.4	99.9 ± 25.9	90.9 ± 20.8[3]	0.016
(% of energy)	17.0 ± 2.4	15.6 ± 1.5	15.1 ± 2.7[3]	0.042
Plasma TG (mmol/L)	1.21 ± 0.39	1.00 ± 0.42	1.16 ± 0.58	NS
Fasting TGc (mmol/L)[4]	1.29 ± 0.38	1.14 ± 0.36	1.15 ± 0.45	NS
TGc-AUC (mmol · h/L)	25.4 ± 6.3	21.8 ± 6.5	25.2 ± 7.6	NS
ΔTGc-AUC (mmol · h/L)	7.3 ± 5.1	5.9 ± 4.6	8.9 ± 3.3	NS
Cholesterol (mmol/L)	5.24 ± 0.90	4.62 ± 0.91	4.69 ± 0.88	NS
HDL cholesterol (mmol/L)	1.31 ± 0.26	1.27 ± 0.37	1.19 ± 0.27	NS
HOMA index	1.77 ± 0.80	1.72 ± 0.74	1.77 ± 0.72	NS
Age (y)	33.5 ± 13.0	35.7 ± 14.1	34.7 ± 12.8	NS
BMI (kg/m^2)	23.0 ± 2.2	22.8 ± 2.2	23.5 ± 2.6	NS
Waist-to-hip ratio	0.85 ± 0.05	0.87 ± 0.07	0.87 ± 0.1	NS
Fat mass (kg)	13.5 ± 2.7	13.7 ± 4.0	12.9 ± 4.8	NS

[1] $\bar{x}$ ± SD. TG, triacylglycerol; TGc-AUC, area under the curve for daytime capillary TG; ΔTGc-AUC, incremental TGc-AUC; HOMA, homeostasis model assessment.
[2] Low tertile: 42.1 ± 1.8% of energy; intermediate tertile: 47.5 ± 2.0% of energy; high tertile: 54.1 ± 2.8% of energy.
[3] Significantly different from tertile 1, $P < 0.05$.
[4] Self-measured.

TABLE 5
Characteristics of the study group by tertile of fat intake[1]

| | Tertile of fat intake[2] | | | |
	Low (n = 19)	Intermediate (n = 20)	High (n = 19)	P (ANOVA)
Total fat (g/d)	74.8 ± 19.8	109.0 ± 22.4[3]	100.3 ± 18.6[3]	<0.001
Total energy (kJ/d)	10372 ± 2440	12383 ± 2492[3]	9809 ± 1967[4]	0.002
Carbohydrate				
(g/d)	307 ± 64	349 ± 64	254 ± 44[3,4]	<0.001
(% of energy)	50.9 ± 5.9	48.1 ± 3.8	44.6 ± 4.5[3,4]	<0.001
Protein				
(g/d)	94.1 ± 20.4	111.4 ± 31.7	97.1 ± 24.1	NS
(% of energy)	15.6 ± 1.6	15.2 ± 2.2	16.9 ± 2.8[4]	0.046
Plasma TG (mmol/L)	1.26 ± 0.59	0.92 ± 0.29	1.21 ± 0.43	NS
Fasting TGc (mmol/L)[5]	1.20 ± 0.46	1.10 ± 0.25	1.28 ± 0.45	NS
TGc-AUC (mmol · h/L)	25.1 ± 7.4	24.4 ± 6.6	22.8 ± 7.0	NS
ΔTGc-AUC (mmol · h/L)	8.2 ± 4.0	8.9 ± 4.3	4.8 ± 4.3[3,4]	0.008
Cholesterol (mmol/L)	5.00 ± 0.83	4.72 ± 1.06	4.86 ± 0.88	NS
HDL cholesterol (mmol/L)	1.21 ± 0.28	1.27 ± 0.31	1.28 ± 0.34	NS
HOMA index	1.64 ± 0.74	1.85 ± 0.71	1.76 ± 0.79	NS
Age (y)	36.2 ± 13.8	34.5 ± 14.4	33.4 ± 11.8	NS
BMI (kg/m^2)	23.5 ± 2.4	22.9 ± 2.4	22.9 ± 2.2	NS
Waist-to-hip ratio	0.88 ± 0.08	0.84 ± 0.08	0.86 ± 0.05	NS
Fat mass (kg)	13.7 ± 4.0	13.1 ± 4.5	13.3 ± 3.4	NS

[1] $\bar{x}$ ± SD. TG, triacylglycerol; TGc-AUC, area under the curve for daytime capillary TG; ΔTGc-AUC, incremental TGc-AUC; HOMA, homeostasis model assessment.

[2] Low tertile: 27.2 ± 2.9% of energy; intermediate tertile: 33.5 ± 1.5% of energy; high tertile: 39.1 ± 3.7% of energy.

[3] Significantly different from tertile 1, $P < 0.05$.

[4] Significantly different from tertile 2, $P < 0.05$.

[5] Self-measured.

We recently showed that daytime TGc profiles are closely related to postprandial lipemia assessed by oral fat-loading tests (23). In some postprandial studies, more modest fat loads reflecting more closely typical daily intakes were investigated; in these studies, maximal TG responses occurred 2–3 h postprandially (2, 37). In our study, TGc concentrations were measured 3 h after lunch and dinner. We previously showed that TGc-AUC estimated on the basis of 6 measurements was not significantly different from that determined from hourly measurements (26). This finding suggests that these 6 time points represented the daylong study period.

Food intake in the free-living situation is difficult to measure. We used food diaries to assess food intakes, although we are well aware that underreporting is often a problem with these diaries. A key issue in our study was whether all types of food were equally underreported. Selective underreporting of fat intake has been observed in obese individuals (38, 39). Additionally, obese subjects are more likely than are normal-weight subjects to underreport their food intake and women underreport more than do men (40, 41). However, we studied healthy nonobese men who were carefully instructed and were health conscious. Furthermore, the food diaries were evaluated by a trained physician in consultation with each participant. Therefore, underreporting was likely not a major problem in our study.

The mean BMI of the study group was relatively low compared with the average of the Dutch population (42). However, the aim of the current study was to evaluate dietary effects on TG metabolism in healthy nonobese men. The nutrient intakes of the subjects were similar to Dutch guidelines and were representative of the intakes of the Dutch population (27, 28). In the current observational study, ΔTGc-AUC was associated with carbohydrate, protein, and energy intakes but not with fat and alcohol intakes. Previous reports of the relation between the type of fat consumed and TG concentrations suggest that chronic ingestion of unsaturated fat reduces fasting and postprandial TG concentrations (43–46).

The Lyon Diet Heart Study showed that a Mediterranean diet has cardioprotective effects in patients after myocardial infarction (47). The cardioprotective effects of this diet may be due in part to improved TG metabolism (47). In contrast with the effects of unsaturated fat intakes, chronic ingestion of saturated fat increases postprandial TG concentrations (48, 49). Nevertheless, the nonphysiologic large doses of fat used in these studies may have influenced the results. In the current study, ΔTGc-AUC was not associated with intakes of total fat, saturated fat, or unsaturated fat. However, carbohydrate intake was the dietary variable that was most associated with ΔTGc-AUC; the relatively low r value suggests that a large variation in ΔTGc-AUC is not simply described by the carbohydrate intake. However, after inclusion of the nondietary variables, carbohydrate intake was still the single variable most associated with ΔTGc-AUC. The relatively low r value between carbohydrate intake and ΔTGc-AUC may have been because we studied a homogeneous group of healthy nonobese men who had relatively small differences in carbohydrate intake.

The effects of diets with extremely high or extremely low proportions of carbohydrate have been compared in metabolic ward studies (6, 7). We obtained similar results when we subdivided the study group into low, intermediate, and high tertiles of fat and carbohydrate intakes as a percentage of energy. Analysis by tertiles of fat intake showed that the mean ΔTGc-AUC was significantly lower in the highest tertile of fat intake (with the lowest proportion of carbohydrate). Analysis by tertiles of carbohydrate intake showed that the mean ΔTGc-AUC was not significantly different between the 3 tertiles of carbohydrate intake.

In a previous study, consumption of high-carbohydrate diets for 2 wk increased both fasting and postprandial TG concentrations in healthy men (7). Several studies focused on the mechanism of carbohydrate-induced hypertriacylglycerolemia, accompanied by elevated insulin concentrations (4, 50). Given the relation between insulin, VLDL-TG production, and TG concentrations, the increase in TG concentrations in response to high-carbohydrate diets may be the result of increased hepatic VLDL-TG production secondary to increased insulin concentrations (4, 50). It was also previously suggested that high-carbohydrate diets reduce VLDL-TG clearance but do not increase VLDL-TG secretion or de novo lipogenesis. Furthermore, the presence of chylomicron remnants in the fasting state may contribute to elevated TG concentrations by competing for VLDL-TG lipolysis and by providing a source of fatty acids for hepatic VLDL-TG synthesis (51). It was recently shown that high-carbohydrate diets stimulate fatty acid synthesis from carbohydrate and that plasma TG concentrations increase in proportion to the amount of fatty acid synthesis. This effect was not related to BMI and insulin (52). Meals with a high glycemic index increase TG concentrations, especially in subjects with fasting hypertriacylglycerolemia (53); however, we had no information on the glycemic index of the diets.

Protein intake was also associated with ΔTGc-AUC. Proteins are degraded into amino acids, which are further metabolized. Most of the amino groups of surplus amino acids are converted into urea, whereas their carbon skeletons are transformed into acetyl-CoA, acetoacetyl-CoA, pyruvate, or one of the intermediates of the citric acid cycle. Hence, fatty acids, ketone bodies, and glucose can be synthesized from amino acids (54). Therefore, increased VLDL-TG production may be responsible for the relation between protein intake and ΔTGc-AUC. Moderate alcohol intake induces transient increases in plasma TG in normolipemic individuals (55, 56). However, this study group had a mean alcohol intake of only 18 g/d, which is less than moderate, and only 76% reported alcohol use on one or more of the study days. This could explain why no significant correlation between alcohol intake and ΔTGc-AUC was found.

The total TGc-AUC was only related to nondietary variables such as fasting TGc, body composition, systolic blood pressure, and fasting insulin. Because these variables are all components of the insulin resistance syndrome, we suggest that the total TGc-AUC is associated with insulin sensitivity.

In this observational study we showed that the carbohydrate content of the diet is the variable most associated with the ΔTGc-AUC. This finding is important because high-carbohydrate diets are recommended to reduce the risk of CHD, especially in patients with fasting hypertriacylglycerolemia. In these patients, high-carbohydrate diets may be harmful because of the detrimental effects of such diets on TG metabolism as observed in this study.

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