Self-Monitoring of Plasma Triglyceride Levels to Evaluate Postprandial Response to Different Nutrients

C. Iovine, A. Gentile, A. Hattemer, D. Pacioni, G. Riccardi, and A.A. Rivellese

Self-monitoring of plasma triglycerides (TG) may be a very useful tool to monitor, on a daily basis, the TG responses to different nutrients, particularly carbohydrates (CHO) and fat, whose influence on postprandial TG levels is not very well known. Therefore, the aim of the present study was to evaluate the TG response of hypertriglyceridemic patients to a similar amount of calories deriving from different sources of CHO and fat. Thirty-nine hypertriglyceridemic patients were randomly assigned to 1 of 2 experimental groups. In 1 group (the fat group), patients were given a standard meal plus a fat supplement of 300 kcal derived from different types of fat (butter, sunflower margarine, olive oil) for dinner, once a week for 3 weeks. In the other group (the CHO group), patients consumed the same standard meal plus a supplement of 300 kcal derived from different types of CHO (bread, coke, fruit). In both groups, patients measured their plasma TG before and 3 hours after each meal by Accutrend GCT (ROCHE, Mannheim, Germany). A subgroup of patients (n = 18) also performed TG determinations 2 hours after the test meals. The 3-hour TG increments were not significantly different between the different test meals (f = 0.671; *P* = .52); instead, the TG increments induced by fat supplements were significantly higher than those induced by the CHO supplements (f = 14.31; *P* = .0001). Similar results were also obtained 2 hours after the test meals. In conclusion, this study shows that the 2- and 3-hour TG responses to fat are higher compared with that induced by carbohydrate. This point, especially if confirmed by experiments with more frequent after meal measurements and of longer duration, should be taken into account in defining the best dietary approach to lower plasma TG levels throughout the whole day.

SELF-MONITORING OF plasma triglyceride (TG) levels allows the evaluation of a patient's metabolic condition on a daily basis[1] and can therefore be a useful tool to monitor cardiovascular disease risk due to hypertriglyceridemia.[2] Plasma TG levels are usually measured in the fasting state, a condition limited to a few hours and thus inappropriate to estimate TG concentrations throughout the day. Furthermore, self-monitoring of plasma TG is also useful to educate patients to modify their diet accordingly. This is not yet totally feasible because the dietary factors with a major influence on TG[3] have not yet been thoroughly studied, especially those acting in the postprandial phase, for the obvious difficulties to utilize traditional laboratory measurements.

In particular, while the influence of the most important nutrients, carbohydrates (CHO) and fat, on fasting TG has been extensively studied,[3-9] their influence on postprandial TG is almost unknown, with very few exceptions,[10-12] which, however, seems to suggest a completely different behavior between fasting and nonfasting conditions, at least in some patients.[11]

Furthermore, postprandial TG response may be influenced to variable extents not only by the different nutrients (CHO *v* FAT), but also by the different types of CHO (slow or fast absorbed) and Fat (saturated *v* unsaturated).[13] Of course, the knowledge of these different responses could help doctors and patients, particularly hypertriglyceridemic patients, to choose foods that induce a lower postprandial TG response. This could be of particular clinical relevance, considering the relationship between postprandial lipemia and the risk of cardiovascular disease.[14]

Therefore, the aim of this study was to evaluate the TG response of hypertriglyceridemic patients to a similar amount of calories deriving from different sources of CHO (bread, fruit, coke) and fat (butter, margarine, olive oil). The TG response to different foods was self-monitored directly by the patients, using the Accutrend GCT (ROCHE).

MATERIALS AND METHODS

Thirty-nine patients with primary mild hypertriglyceridemia (fasting plasma TG >1.69 mmol/L <2.81 mmol/L at the time of selection), aged 49.5 ± 6.8 years (mean ± SD), with a body mass index (BMI) of 28.2 ± 3.1 kg/m^2, participated in the study after giving their informed consent.

Patients had no disease other than hypertriglyceridemia and were not on any drug treatment influencing lipid metabolism. After a 3-week run-in period to get accustomed to the self-monitoring of plasma TG (using Accutrend GCT, ROCHE, Mannheim, Germany), patients were randomly assigned to 1 of 2 experimental groups. In 1 group (the fat supplement group, n = 19), patients were given a standard meal (kcal 800, CHO 50%, Fat 30%, protein 20%) plus a fat supplement of 300 kcal derived from different types of fat (in turn, 40 g butter, rich in saturated fat; 40 g sunflower margarine, rich in polyunsaturated fat; 35 g olive oil rich in monounsaturated fat) to be consumed as dinner (at 7 PM), once a week for 3 weeks. In the other group (the CHO supplement group, n = 20), patients consumed the same standard meal plus a supplement of 300 kcal given by different types of CHO (110 g bread, rich in complex CHO; 770 g coke, rich in sucrose; 700 g fruit, rich particularly in fructose) at the same time and always once a week for 3 weeks. The dry weights of fat and CHO supplements were similar. The standard meals and the supplements were prepared in the metabolic kitchen of the university hospital and were given to the patients to be consumed at home. As to the rest of the diet, no specific indications were given to the patients, who continued their habitual diets as well as their habitual physical exercise.

From the Department of Clinical and Experimental Medicine, Federico II University, Naples, Italy; and Evaluation Patient Care, Roche Diagnostics GmbH, Mannheim, Germany.

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Address reprint requests to A.A. Rivellese, MD, Department of Clinical and Experimental Medicine, Federico II University, School of Medicine, Via S. Pansini 5, 80131, Naples, Italy.

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Table 1. Main Characteristics of the Subjects

	Fat Supplements (n = 19)	CHO Supplements (n = 20)	*P*
Sex M/F	16/3	17/3	
Age (yr)*	50 ± 7	50 ± 7	.98
BMI (kg/m^2)*	27 ± 2	29 ± 3	.16
S-cholesterol (mmol/L)*	6.34 ± 1.01	6.05 ± 1.01	.39
HDL-cholesterol (mmol/L)*	0.98 ± 0.23	0.96 ± 0.18	.77
S-triglycerides (mmol/L)*	2.67 ± 0.69	2.46 ± 0.56	.29

*Mean ± SD.

Patients in both groups were instructed to measure their TG levels just before the test meal and 3 hours after. This time-point was chosen as representing the postprandial peak in a previous experiment performed by our group in hypertriglyceridemic subjects (unpublished data) as well as in other studies.[1] A subgroup of patients (n = 18) also performed TG determinations 2 hours after the test meal.

Plasma TG was measured by Accutrend GCT on capillary blood. This system had shown good agreement with the glycerol phosphate oxidase–peroxidase amino phenazone (GPO-PAP) laboratory method, the correlation coefficient being $r = .97$.[15] In the same study, the mean difference between patients' measurements and those of professional users was 0.57% ± 9.55%, indicating that lay users are able to achieve reliable results with this system. Even in our hands, Accutrend GCT had shown a good correlation with the laboratory method ($r = .98$) (unpublished data). Furthermore, the interassay coefficient of variation, evaluated on a control serum given to patients during the present study, was 2.4% ± 0.8%. The study was approved by the Ethics Committee of the "Federico II" University.

Statistical Analysis

The TG response after the test meal is expressed as the variation between postmeal (2 and 3 hours) and premeal values (Δ TG). Differences between the 2 groups were evaluated by the unpaired *t* test; differences between test meals and between fat and CHO supplements were analyzed by the 2-way analysis of variance.[16] All statistical analysis on TG was performed after logarithmic transformation. A *P* value $<.05$ (2-tailed) was considered significant. Data are expressed as mean ± SD.

RESULTS

Of the 39 patients participating in the study, 20 were given fat and 19 CHO supplements. Their age, BMI, and plasma lipids were similar (Table 1). Body weight did not change throughout the experiment (fat group, 73.2 ± 9.4 kg at the beginning and 73.0 ± 9.5 kg at the end; CHO group, 76.6 ± 7.6 kg at the beginning and 76.6 ± 7.2 at the end). Despite the known variability of TG within and between subjects, values before the consumption of the test meals were very similar as shown in Table 2. However, to avoid any possible influence due to even minimal differences in pretest values, postprandial TG responses are expressed as the variation between postmeal and premeal values (Δ TG).

TG responses after the various test meals are shown in Table 3. The 3-hour TG increments were not significantly different between the different test meals ($f = 0.671$, $P = .52$) (Table 3); on the other hand, the TG increments induced by fat supplements (0.53 ± 1.26 mmol/L for butter, 0.79 ± 0.85 mmol/L for margarine, and 0.95 ± 1.15 mmol/L for olive oil) were significantly higher than those induced by the CHO supplements (−0.16 ± 0.96 mmol/L for bread, −0.12 ± 0.76 mmol/L for coke, and 0.09 ± 1.13 mmol/L for fruit) ($f = 14.31$; $P = .0001$) (Table 3).

Very similar results were obtained 2 hours after the test meals in the subgroup of patients who also performed this evaluation (Table 3); again, there was no difference between test meals ($f = 0.28$, $P = .75$), while the increments induced by fat supplements were significantly higher than those induced by CHO supplements ($f = 7.50$, $P = .009$).

DISCUSSION

The most important results of this study are: (1) the absence of substantial differences in the acute postprandial TG response to different types of fat and CHO and (2) an overall significantly higher acute postprandial TG response to fat compared with the one induced by CHO.

This second result is just the opposite of what happens for fasting TG. As a matter of fact, most studies comparing the effect of CHO and fat on fasting TG, either in acute or in more prolonged (weeks/months) conditions and in different metabolic conditions, have shown that CHO-rich diets induce a significant increase in fasting TG (an average of 20% in the different studies) in comparison with fat-rich diets.[5-9,17]

On the basis of this effect as well as of that on postprandial blood glucose, some investigators have criticized the use of CHO-rich diets as the best dietary approach to prevent cardiovascular diseases.[18] Of course, this opinion could change if the results on postprandial TG, obtained by us in acute conditions, were also confirmed in experiments of longer duration. As a matter of fact, the increase in fasting TG induced by CHO could be well balanced by the decrease in postprandial TG response induced by CHO. Moreover, considering that humans usually spend a longer period of time in postprandial conditions than in the fasting state and that postprandial TG levels may represent, according to some recent data, an independent cardiovascular risk factor,[14] lower TG levels in the postprandial

Table 2. Serum Triglycerides (mmol/L) Before the Different Test Meals in the Two Groups of Subjects

Fat Supplements (n = 19)			CHO Supplements (n = 20)		
1-way ANOVA			1-way ANOVA		
Butter	3.81 ± 1.42	f = 0.058	Bread	3.94 ± 1.34	f = 0.06
Margarine	3.82 ± 1.17	*P* = .94	Coke	4.20 ± 1.68	*P* = .93
Olive oil	3.91 ± 1.31		Fruit	4.03 ± 1.41	

*NOTE. Values are mean ± SD.

Table 3. 2-and 3-Hour Triglyceride Response in the Two Groups of Patients

	2 Hours*	3 Hours
ΔTG (mmol/L) during CHO supplements		
Bread	−0.17 ± 0.76	−0.16 ± 0.96
Coke	0.11 ± 1.24	−0.12 ± 0.76
Fruit	0.17 ± 1.30	0.09 ± 1.13
ΔTG (mmol/L) during fat supplements		
Butter	0.81 ± 0.64	0.53 ± 1.26
Margarine	0.89 ± 0.78	0.79 ± 0.85
Olive oil	0.61 ± 0.97	0.95 ± 1.15
2-way ANOVA		
Fat *v* CHO	f = 7.50, *P* = .009	f = 14.31, *P* = .0001
Between test-meals	f = 0.28, *P* = .75	f = 0.671, *P* = .52

Abbreviation: ΔTG, variation between after meal and premeal TG values.

*In a subgroup of 18 patients (mean ± SD).

phase could be even more important from a clinical point of view than lower fasting TG levels.

Our data refer to acute conditions and cannot be extrapolated to chronic conditions. However, it is important to remember that in one of the few studies in which the effects of a high CHO diet and a high fat diet were evaluated also on postprandial TG, in type 1 diabetic patients, the results obtained were very similar to ours: postprandial TG levels were significantly lower after 4 weeks on a high-CHO diet than on a high-fat diet.[11]

The possible explanations for the different effects of fat and CHO on plasma TG in fasting and nonfasting conditions are rather complex, and our study was not aimed to find the possible underlying mechanisms. At any rate, the most obvious explanation could be that during the postprandial period, the main determinant of TG levels is represented by the amount of exogenous fat, which induces a higher formation of chylomicrons. On the other hand, fasting TG are the direct expression of endogenous TG synthesis; the latter are regulated mainly by other factors,[19] the most important being plasma insulin concentration, which is generally increased during high-CHO diets.[8]

Because TG response to CHO-rich foods could be faster than that to fat-rich foods, in a subgroup of patients we also evaluated TG levels after 2 hours. The results were very similar to those found after 3 hours. It could be argued that the TG response to CHO supplements could be even faster, which would not allow us to detect an increase that occurred within the first 2 hours after meal intake. In any case, it has to be considered that our subjects are hypertriglyceridemic and that in these patients, plasma TG response to either CHO or fat-rich meals are reported to be slower, with peaks generally found after 2 to 3 hours.[20]

A further finding of our study was that there were no substantial differences in the acute TG response to the different types of CHO and fat. Other studies have reported some differences in relation to different types of fat, with higher TG levels after intake of saturated fat and lower TG concentration after intake of unsaturated, particularly n3 polyunsaturated fats,[14,21-23] which were not studied in our experiment. Moreover, these data refer especially to chronic feeding with different types of fat[14,21] and are particularly evident on chylomicrons and their remnants and not on total plasma TG.[23] A recent study has reported a lower acute TG response after stearic and palmitic fatty acids in comparison with unsaturated ones (both oleic and linoleic).[24] However, this study examined the response to specific fatty acids and not naturally fat-rich foods, such as butter, margarine, or olive oil, which contain a mixture of different fatty acids.

Another aspect of our study to be emphasized is that self-monitoring of TG is well accepted by patients and may represent a valid and simple method to have information on the TG profile throughout the day. This may be a very important achievement, because the great variability of TG in the same individual during the day and over different days is very well known.[25] Still more variable is the TG response to a given diet among hypertriglyceridemic individuals.[26] Therefore, to have a more precise indication of an individual's TG response to a diet, it is very important to be able to make repeated measurements, which is achievable only by self-monitoring. This information would really help both the doctor and the patient to modify dietary habits for the better according to the results obtained.

In conclusion, this study shows that the 2- and 3-hour postprandial TG responses to fat are higher compared with that induced by CHO. This point, especially if confirmed by experiments with more frequent after meal measurements and of longer duration, should be taken into account in defining the best dietary approach to lower plasma TG levels throughout the day.

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