Postprandial lipid metabolism

Gabriele Riccardi, Lutgarda Bozzetto and Giovanni Annuzzi

Department of Clinical and Experimental Medicine, Federico II University, Naples, Italy

Abstract

Atherogenic effects of postprandial lipoproteins are supported by many studies comparing patients with advanced clinical signs or early markers of cardiovascular disease with subjects without vascular disease. Postprandial lipoprotein abnormalities are more frequent in individuals with type 2 diabetes and other states of insulin resistance, which could be a major factor accounting for the higher rate of cardiovascular diseases observed in these conditions. In patients with type 2 diabetes multiple abnormalities of lipoproteins of both endogenous and exogenous origin are observed also in the presence of good blood glucose control and normal fasting triglyceridaemia, and a direct role of insulin resistance in the development of postprandial dyslipidaemia has been demonstrated. Clinical and experimental studies have demonstrated that a high fat consumption is associated with impaired insulin sensitivity and may worsen postprandial hypertriglyceridemia in individuals with type 2 diabetes and/or hyperlipidaemia. In addition to the amount of fat also the physical structure of the food in which fat is present is able to influence postprandial lipaemia. Data on the effects of dietary fatty acid (FA) modifications in humans are scarce. These studies have provided some evidence that dietary polyunsaturated FA, particularly those of the n-3 class, induce an attenuated postprandial lipaemic response as compared to saturated FA (SFA) and monounsaturated FA (MUFA); however, the data are often conflicting and their interpretation is, generally, controversial. Very little is known in patients with type 2 diabetes, in whom both insulin sensitivity and postprandial lipaemia are particularly relevant. In a medium term (3 weeks) study in type 2 diabetic patients, a MUFA rich diet did not modify insulin sensitivity as compared to the SFA rich diet. The MUFA diet induced a higher early peak of chylomicrons and a significantly lower postprandial response of small VLDL after a standard test meal, rich in saturated fat. In conclusion, postprandial lipoprotein abnormalities are a frequent feature of type 2 diabetes and of other conditions clustering with insulin resistance. The total amount of fat, more than the specific type of fatty acid, seems to influence postprandial lipoproteins. Data are needed on long term interventions as well as on the interactions between dietary fat and other food components or physical structure.

Keywords: *postprandial lipoproteins; metabolism of dietary fat*

Abbreviations: *Apo: apolipoprotein*; *FFA: free fatty acid*; *LPL: lipoprotein lipase*; *MUFA: monounsaturated fatty acid*; *PUFA: polyunsaturated fatty acid*; *SFA: saturated fatty acid*; *VLDL: very low-density lipoprotein.*

Introduction

After a meal, the ingested fat is emulsified with bile acids and phospholipids in the small intestine, then it is hydrolysed by pancreatic enzymes to cholesterol, free fatty acids (FFAs) and monoglycerides, which are absorbed from the small intestine into mucosal cells. Once reabsorbed, the lipids are re-esterified to form cholesterol esters and triglycerides. These are combined with free cholesterol, several apolipoproteins (ApoA, ApoB48) and phospholipids into chylomicrons, which are secreted into the lymphatic system and, via the thoracic duct, reach the blood.

In capillary beds, chylomicrons bind to lipoprotein lipase (LPL) on the luminal surface of the capillary endothelial cells. LPL hydrolyses chylomicron triglycerides producing FFAs, which freely diffuse into cells where they are either oxidized for energy utilization or re-esterified for storage. The depletion of triglycerides results in the reduction of the size of the lipoproteins; the modified particles are then referred to as remnants (1). Chylomicron remnants are cholesteryl ester enriched and maintain their content of ApoB48 and ApoE. The liver is the major organ that removes remnants from the blood (2). Receptors for chylomicron remnants recognize ApoE on the surface of the lipoproteins

Table 1. Studies evaluating postprandial lipaemia (PL) in patients with cardiovascular disease (CVD)

Study	Subjects	CVD	PL alteration
Groot et al. (4)	20 M vs 20 controls	Severe coronary stenosis	↑ TG and chylomicron remnants
Patsch et al. (5)	61 M vs 40 controls	Severe coronary stenosis	↑ TG 6–8 h after test meal
Karpe et al. (6)	32 M with AMI vs 10 controls	Coronary lesion progression	↑ Chylomicron remnants (Sf 20–60)
Weintraub et al. (7)	85 M+F vs 85 controls	Severe stenosis	↑ Chylomicron remnants (Sf 20–60)
Lupattelli et al. (8)	16 M vs 16 controls	Claudication	↑ TG

F: female; AMI: acute myocardial infarction; M: male; TG: triglycerides.

and this leads to a rapid absorption by the hepatocytes.

Postprandial dyslipidaemia and cardiovascular risk

Zilversmit postulated in 1979 that atherogenesis is a postprandial phenomenon and that chylomicrons or chylomicron remnants per se could cause atherosclerosis (3); thereafter, many data have been produced supporting the atherogenic effects of postprandial lipoproteins (4). Studies have mainly compared postprandial responses of plasma lipoproteins in patients with different forms of atherosclerosis and in control subjects without vascular disease. In these studies postprandial lipoprotein abnormalities have been reported both in patients with advanced clinical signs of coronary heart disease or peripheral arterial disease (Table 1) (4–8) and in people with early markers of vascular disease such as increased intima-media thickness (Table 2) (9–13).

Postprandial lipid metabolism and atherogenesis

Different mechanisms have been proposed relating postprandial lipid metabolism to atherosclerosis

Table 2. Relationship between postprandial lipaemia (PL) and early atherosclerosis: cross-sectional studies in subjects with increased carotid intima-media thickness

Study	Subjects	PL alteration
Ryu et al. (9)	20 M+F	↑ TG peak
Sharrett et al. (10)	229 cases vs 373 controls	↑ TG and VLDL
Karpe et al. (11)	30 M	↑ TG 6 h after test meal
Boquist et al. (12)	96 M	↑ TG 2 h after test meal
Karpe et al. (13)	50 M	↑ Remnants TRL fasting and postprandial

F: female; TG: triglycerides; M: male; VLDL: very low-density lipoprotein; TRL: triglyceride rich lipoproteins.

(14). Remnants of very low-density lipoprotein (VLDL) and chylomicrons could induce atherosclerosis by promoting the cellular response that leads to inflammation within the vessel wall. An early event in this process is the adhesion of monocytes to endothelial cells (15). In diabetic patients, who have a delayed removal of remnant lipoproteins in the postprandial phase, monocytes show increased adhesiveness to endothelial cells. When there is accumulation and retention of chylomicron remnants in the subendothelial extracellular space (this can be stimulated by an increased and prolonged postprandial triglyceride response), monocytes differentiate into macrophages and take up the retained remnant particles. If these macrophages, in the presence of an excess of chylomicron remnants, cannot efficiently remove these lipoproteins from the extracellular space, they continue to take up lipids and become foam cells, thus initiating the atherosclerotic process.

Postprandial hypertriglyceridaemia is also a major determinant of oxidative stress and impaired endothelial function, which have an important role in the advancement of atherosclerotic lesions; macrophages, stimulated by postprandial lipoproteins, secrete toxic substances (oxidizing agents, cytokines) that may harm the arterial wall and facilitate the inflammatory response. Furthermore, macrophage-secreted cytokines stimulate the growth of smooth-muscle cells and their migration to the subendothelial space; this leads to the formation of a fibrous cap over the atherosclerotic plaque, which protrudes in the arterial lumen and impairs blood flow.

Remnants of triglyceride-rich lipoproteins may also be associated with a thrombophilic state activating factor VII, an enzyme involved in the production of thrombin through the conversion of fibrinogen to fibrin (16). The FFAs produced by lipolysis of triglyceride-rich lipoproteins at the level

of the endothelium may activate the contact system of coagulation and subsequently factor VII. This is particularly dangerous since it may exacerbate thrombogenesis in the presence of a damaged atherosclerotic plaque; the fibrin clot will trap platelets which, in turn, may contribute to enlarge the thrombus, eventually occluding the arterial lumen.

Postprandial dyslipidaemia in type 2 diabetes

Postprandial lipoprotein abnormalities are more frequent in individuals with type 2 diabetes and other states of insulin resistance. Since these abnormalities are associated with an increased risk of coronary heart disease, they could be a major factor accounting for the higher rate of cardiovascular diseases observed in these conditions.

Patients with type 2 diabetes have high plasma triglyceride levels throughout the whole day, as shown by a study from this group (17) that compared the daily triglyceride profile in a population of patients with type 2 diabetes with that of a non-diabetic control group during their everyday life (Fig. 1). Many of these patients, despite normal fasting triglyceride plasma levels, had triglyceride concentrations above 200 mg dl^{-1} during the postprandial phase; this indicates that most patients with type 2 diabetes have plasma triglycerides above the desired level for several hours after the meals. Moreover, this study suggests that optimal triglyceride values at fasting are not always a good predictor of a normal triglyceride metabolism in the postprandial period (Fig. 2).

The type of postprandial lipoprotein abnormalities present in type 2 diabetes has been defined in
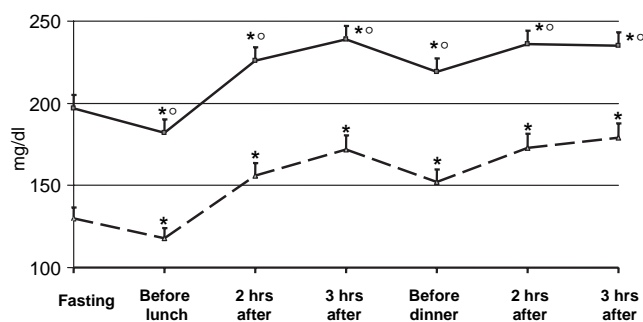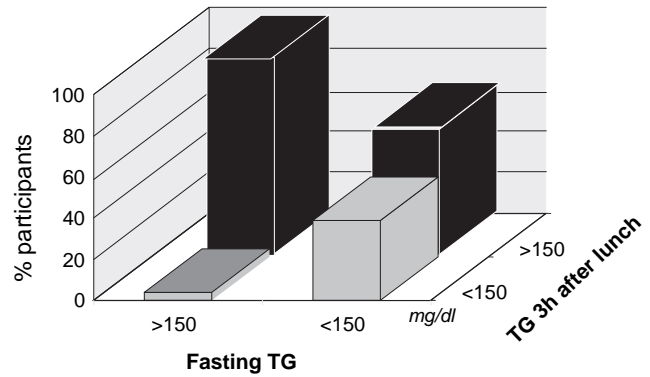
Fig. 2. Postprandial triglycerides (TG) (3 h after lunch) by fasting TG levels in patients with type 2 diabetes. [Modified from Iovine et al., 2004 (17).]

another study by this group (18), which evaluated exogenous and endogenous lipoprotein responses to a standard fat-rich test meal in patients with type 2 diabetes (with optimal fasting triglyceridaemia and optimal blood glucose control) and in non-diabetic individuals. As expected, blood glucose concentrations were higher in diabetic patients while fasting and remained higher during the postprandial period in comparison with the control group. The plasma insulin response to the meal was more prominent in diabetic patients than in control subjects. In these patients multiple abnormalities of lipoproteins of both endogenous and exogenous origin were observed (Fig. 3). In particular, the number of particles was increased in diabetic patients, as was their triglyceride and cholesterol content; this increase was more marked in the late postprandial phase. Therefore, patients with type 2 diabetes have postprandial abnormalities of triglyceride-rich lipoproteins even when their blood glucose control is good and fasting plasma triglyceride levels are normal.

Role of insulin resistance

Insulin resistance, or compensatory hyperinsulinaemia, as well as hyperglycaemia (which represent the characteristic features of type 2 diabetes) have been suggested as major determinants of the postprandial hyperlipaemia observed in patients with type 2 diabetes. The controversies on this issue are mainly attributable to the strong interrelationships among insulin resistance, hyperinsulinaemia and hyperglycaemia that make it difficult to assess these factors independently of each other. To evaluate the possible role of insulin resistance per se on the development of the postprandial lipid abnormalities

Fig. 1. Plasma triglyceride daily profile (4 days) in patients with type 2 diabetes ($n = 145$, solid line) and controls ($n = 30$, dashed line). Mean $\pm$ SEM. *$p < 0.0001$ vs fasting, °$p < 0.0001$ vs controls. [Modified from Iovine et al., 2004 (17).]

generally found in patients with type 2 diabetes, a standard fat-rich test meal was administered to patients with this condition and to a control group, during a hyperinsulinaemic glycaemic clamp (19). After the meal, there was a greater increase in triglycerides and cholesterol in the large VLDL particles in diabetic subjects than in controls, which was more evident in the late postprandial phase; moreover, these particles were richer in ApoB48. Altogether, these data indicate that insulin resistance, more than hyperglycaemia and hyperinsulinaemia, plays a key role in the development of postprandial lipid abnormalities. It is possible that the increase in large VLDL was attributable to an increased hepatic VLDL synthesis during the postprandial period, possibly due to the greater flux of FFA to the liver; this hypothesis is supported by the higher FFA plasma levels in the late postprandial period observed in diabetic subjects compared with controls, and by the strong direct correlation between FFA levels and changes in large VLDL triglyceride concentrations 6 h after the meal (Fig. 4). However, it cannot be ruled out that VLDL abnormalities were also induced by a decrease in the direct inhibitory effect of insulin on the synthesis of these particles.

Effects of the amount of fat and combination with other food constituents

There is consistent evidence that a high fat intake is associated with both insulin resistance and postprandial lipid abnormalities. Clinical and experimental studies have demonstrated that a high fat consumption is associated with impaired insulin sensitivity and may worsen postprandial hypertriglyceridaemia in individuals with type 2 diabetes and/or hyperlipidaemia.

Van Oostrom et al. (20) measured the levels of triglycerides and FFA in six healthy men before and after the ingestion of four study meals: fat, glucose, mixed meal and water. There was a significant meal and time interaction for triglycerides and FFA ($p < 0.005$). The fat test increased triglyceride concentrations from 1.04 ± 0.26 mmol l^{-1} to a maximum value of 2.29 ± 0.86 mmol l^{-1} at 4 h ($p < 0.005$). The mixed meal also increased triglyceride concentrations from 0.91 ± 0.45 mmol l^{-1} at baseline to a maximum value of 1.82 ± 1.34 mmol l^{-1} at 2 h ($p = 0.04$). Because of the small number of subjects, the incremental triglyceride response after the fat meal was not significantly different from that after the mixed meal (5.32 ± 2.33 compared with 3.08 ± 1.44 mmol $h^{-1} l^{-1}$, respectively), whereas
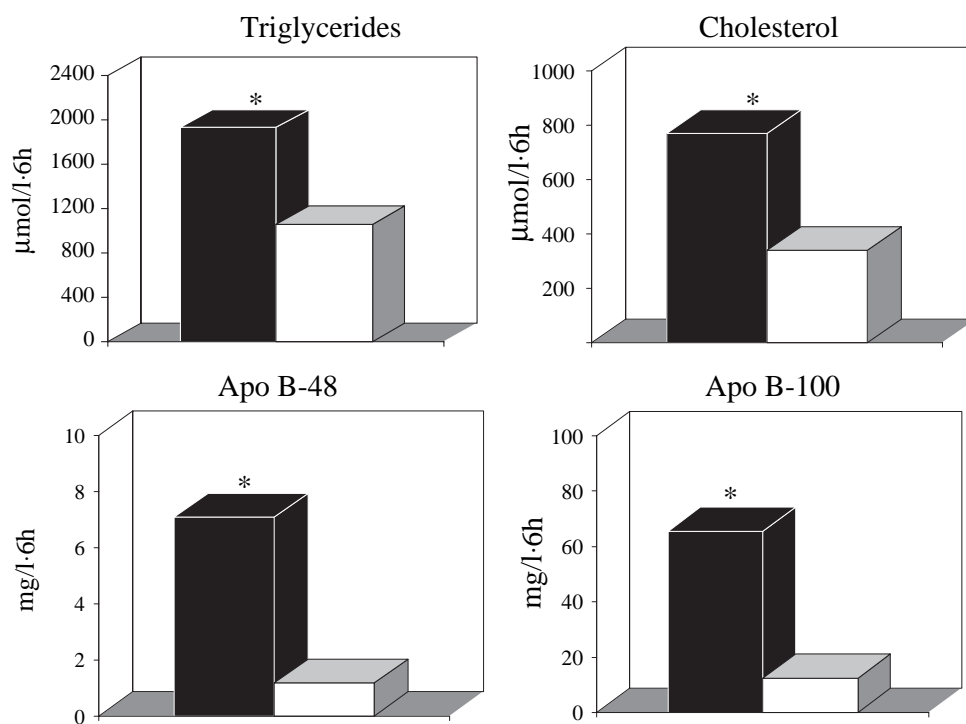

Fig. 3. Incremental area under the curve of lipids and apolipoprotein B (ApoB) in large very low-density lipoprotein after a standard meal in patients with type 2 diabetes (black) and controls (white). *$p < 0.05$ vs controls. [Modified from Rivellese et al., 2004 (18).]
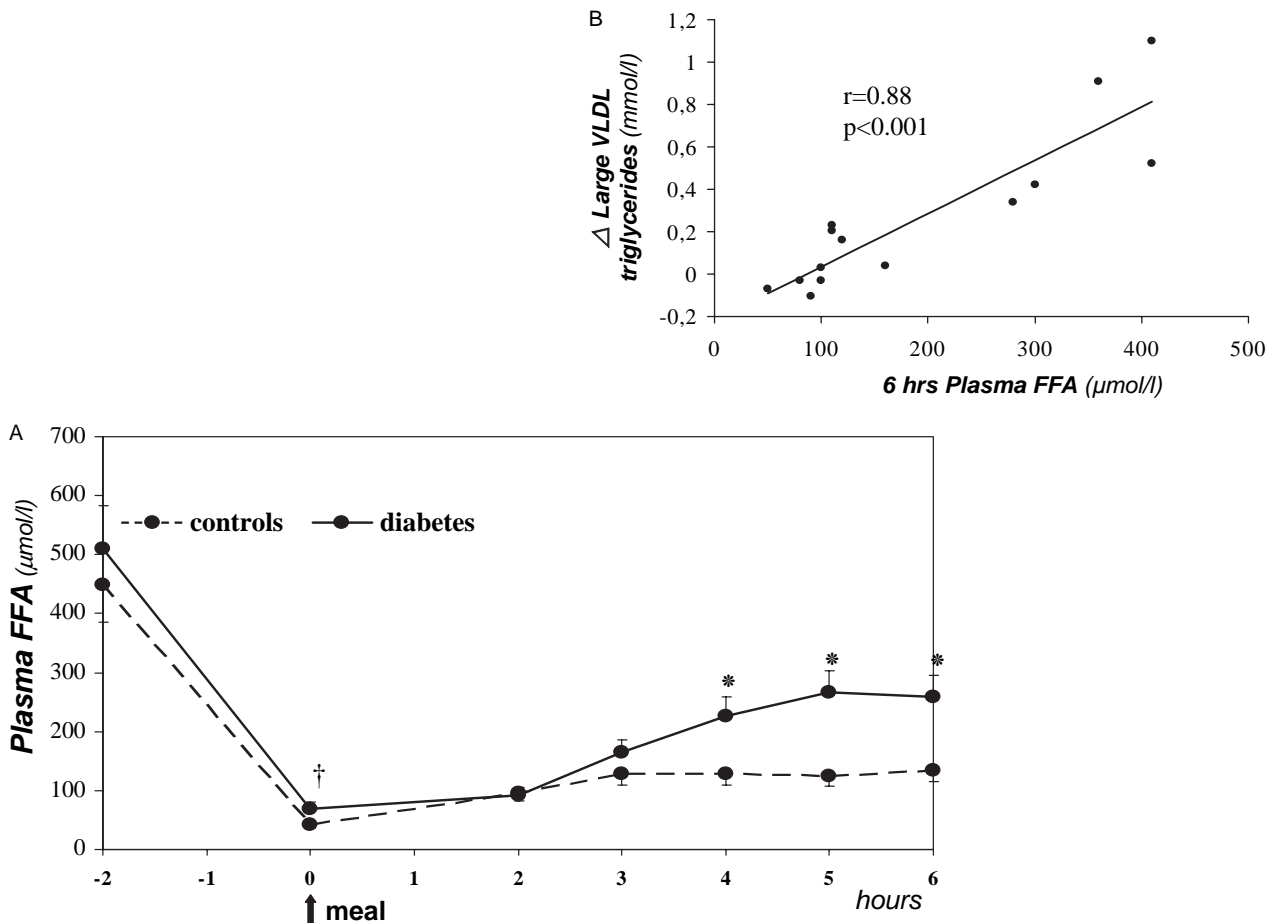
Fig. 4. (A) Free fatty acid (FFA) plasma concentrations before and after a standard meal consumed during a hyperinsulinaemic glycaemic clamp in patients with type 2 diabetes (solid line) and in non-diabetic controls (dashed line). †$p < 0.001$, *$p < 0.05$ vs controls. (B) Correlation between plasma FFA concentration and change in large (VLDL) triglyceride concentration 6 h after the meal. [Modified from Annuzzi et al., 2004 (19).]

the incremental triglyceride responses after both tests were significantly higher than after either water or glucose ($p < 0.01$). FFA concentrations after the fat test reached a peak at 5 h ($p = 0.02$) and decreased thereafter. The incremental FFA response after the fat test was the highest ($p < 0.05$ for all comparisons). This study shows that in insulin-sensitive subjects the postprandial triglyceride and FFA responses are dependent on the amount of oral fat and are reduced when glucose is added to the fat load.

Not only the amount of fat but also the physical structure of the food in which fat is present can influence postprandial lipaemia. In a group of diabetic patients, Clemente et al. (21) compared postprandial responses of plasma glucose, insulin, total cholesterol, high-density lipoprotein-cholesterol, triglycerides and FFAs to three fat-rich meals, with similar composition but different physical structure (liquid, semisolid and solid). Plasma

triglycerides increased after each of the three meals, reaching a plateau after 4–5 h. The time to peak was shorter with the milk-based meal (liquid) than after mozzarella cheese (semisolid) ($p < 0.03$) or butter (solid) ($p < 0.01$). With the butter meal the curve of postprandial plasma triglycerides was shifted to the right, indicating that postprandial lipaemia was delayed compared with the other two meals (Fig. 5). These data suggest that while the physical structure of fat-rich foods has no major effect on postprandial plasma triglyceride concentrations, it can influence the timing of the triglyceride peak.

Effects of different fatty acids

Test-meal studies

The effects of modifications of dietary fatty acids on postprandial lipaemia are very controversial.
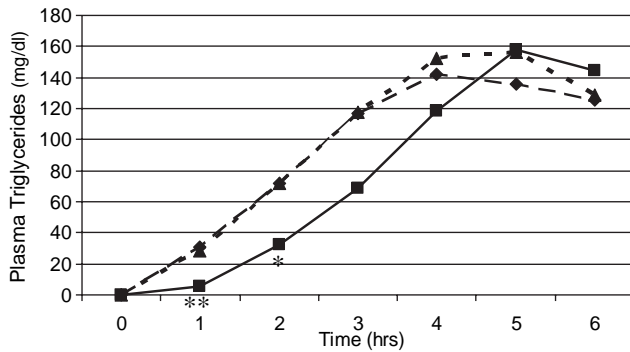
Fig. 5. Postprandial plasma triglyceride increments after the milk (◆), butter (■) and mozzarella (▲)-based meals. **p <0.0002, *p <0.05 vs milk. [From Clemente et al., 2003 (21).]

Data on the influence of dietary fatty acid modifications on postprandial lipid metabolism in humans are scarce and mainly concern the acute effects of test meals with different fatty acid composition. These studies have provided some evidence that polyunsaturated fatty acids (PUFAs), particularly those of the n-3 class, induce an attenuated postprandial lipaemic response compared with saturated fatty acids (SFAs) and mono-unsaturated fatty acids (MUFAs); however, the data are often conflicting and their interpretation is, in general, controversial.

Thomsen et al. (22) evaluated in a group of healthy subjects the postprandial responses of glucose, insulin, fatty acids and triglycerides to an energy-free soup consumed with 50 g carbohydrate (control meal), the control meal plus 100 g butter, and the control meal plus 80 g olive oil. Triglyceride concentrations in plasma, in the chylomicron-rich and in the chylomicron-poor fraction were higher at several time-points after the butter meal than after the olive oil and control meals.

Opposite results were shown by Mekky et al. (23), who compared the effects of mixed meals containing a reasonable amount of fat (40 g) in the form of common fat sources such as olive oil (n-9 MUFA source), sunflower oil (n-6 PUFA source) or butter (saturated fatty acids source) on postprandial lipaemia, chylomicrons, ApoB48 and ApoB100 triglyceride rich-lipoproteins and non-esterified fatty acids. The areas under the curves of serum triglycerides after the olive oil and sunflower oil meals were higher than after the butter fat meal.

Dietary interventions
The long-term effects of two diets with different fatty acid compositions on the lipid response to a standard meal were evaluated by Silva et al. (24). In a controlled sequential design, 51 healthy young subjects followed an SFA-rich diet for 8 weeks, after which half of the subjects followed a moderate MUFA diet and half followed a high MUFA diet for 16 weeks. The total areas under the postprandial response curves and the peak postprandial ApoB48 concentrations were more markedly reduced on the high than on the moderate MUFA diet, suggesting a dose response to dietary MUFA. In contrast to the ApoB48 findings, subjects did not show any significant difference in the patterns or the magnitude of plasma postprandial triglyceride responses between MUFA and SFA diets, suggesting that the fat composition of the habitual diet may have different effects on the number of postprandial triglyceride-rich lipoproteins and on their lipid composition.

Concerning the effects of different fatty acids on both insulin sensitivity and postprandial lipaemia, very few studies have been carried out in patients with type 2 diabetes, in whom these two abnormalities are particularly relevant. In a recent study we compared the medium-term (3 weeks) effects of MUFA- and SFA-rich diets on insulin resistance and postprandial lipid tolerance in patients with type 2 diabetes, evaluating the role of LPL in adipose tissue. Adipose tissue plays a central role in type 2 diabetes and insulin resistance, and could be essential in the postprandial removal and partitioning of exogenous fat. Under the conditions of the experiment, the MUFA-rich diet did not modify insulin sensitivity compared with the SFA-rich diet. The MUFA diet induced a higher early peak of chylomicrons and a significantly lower postprandial response of small VLDL lipoproteins after a standard test meal (rich in saturated fat) compared with the SFA diet. These changes were associated with increased LPL activity in the subcutaneous abdominal adipose tissue during the MUFA diet, both during fasting and postprandially.

Evidence in the real world
From the above studies it appears that the differences in the effects of the various fatty acids on postprandial lipaemia are small and influenced by the interaction with food structure. To explore this issue in the real world, the relationship between dietary habits and postprandial triglyceride response was evaluated in a non-selected population of patients with type 2 diabetes in free-living conditions (25). The study group consisted of 140

patients recruited from a population-based sample referring to the same health district of the province of Naples (Italy). These patients were asked to perform four daily triglyceride profiles, twice weekly, on two non-consecutive days, excluding weekends. Triglyceride self-measurements were performed while fasting, before lunch, and 2 and 3 h after lunch, using an Accutrend GCT. Usual dietary intakes, defined as the average intake over the previous year, were estimated with the use of a 138-item semi-quantitative food-frequency questionnaire. Except for the energy intake (high, considering the high body mass index and the very low level of physical activity) and the fibre intake (too low), the diet followed by this diabetic population seemed consistent with current dietary recommendations, particularly in relation to the amount and type of dietary fat. The postprandial triglyceride response (3 h after meal minus preprandial values) was significantly correlated with the intake (g per day) of animal proteins ($r = 0.20$, $p = 0.02$), total fat ($r = 0.21$, $p = 0.01$), animal fat ($r = 0.19$, $p = 0.03$) and vegetable fat ($r = 0.19$, $p = 0.03$). No correlation was found with energy intake, alcohol or carbohydrate intake. These data indicate that in a free-living population of patients with type 2 diabetes from a southern Italian region, the main dietary determinant of postprandial triglyceride response is the amount of dietary fat, independently of the different types of fat.

Conclusions

Postprandial lipoprotein abnormalities are a frequent feature of type 2 diabetes and of other conditions clustering with insulin resistance. The total amount of fat seems to be more important than the specific type of fatty acid consumed in relation to the dietary influence on postprandial lipoprotein abnormalities. Nevertheless, data on long-term interventions are still required, in addition to further studies aiming towards a better understanding of the interactions between dietary fat and other food components (fibre, carbohydrates, proteins, etc.) or physical structure.

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Gabriele Riccardi
Department of Clinical and Experimental Medicine
Federico II University
Via Sergio Pansini 5
IT-80131 Naples Italy
Tel: +39 081 746 2117
Fax: +39 081 546 6152
E-mail: gabriele.riccardi@unina.it