Postprandial lipid metabolism: effects of dietary fatty acids

BY CHRISTINE M. WILLIAMS

*Hugh Sinclair Unit of Human Nutrition, Department of Food Science and Technology,
University of Reading, Reading RG6 6AP*

Current dietary recommendations for fatty acid intakes aimed at reducing the prevalence of CHD are largely based on the cholesterol hypothesis and on the well-established effects of specific fatty acid classes on fasting total cholesterol, LDL and HDL concentrations (Department of Health, 1991, 1994). Increasing recognition of the potential atherogenic and thrombogenic consequences of elevated triacylglycerol-rich lipoproteins (TRL; Cohn, 1994; Havel, 1994; Karpe *et al.* 1994; Hodis & Mack, 1995; Karpe & Hamsten, 1995) has revealed the paucity of dietary studies which evaluate postprandial as well as fasting lipoproteins.

The need for a greater understanding of effects of diet on postprandial lipoproteins is emphasized by the fact that most individuals are in a postprandial state for the greater part of every 24 h period. A typical 24 h triacylglycerol profile of a normal individual eating three or four meals and snacks per day is shown in Fig. 1, and illustrates that triacylglycerol levels are elevated above fasting values for approximately 17 h of each 24 h period. Only in the early morning between 03.00 and 06.00 hours, do triacylglycerol levels reflect the values measured after a 12 h fast, the measurement usually made in most epidemiological studies. It is clear that the fasting state does not reflect total 24 h triacylglycerol values and is not the normal nutritional state for most individuals, at least as far as lipid metabolism is concerned.

POSTPRANDIAL TRIACYLGLYCEROL-RICH LIPOPROTEINS AS A LIPID RISK FACTOR

The involvement of postprandial TRL (chylomicrons (CM) and VLDL) in the pathogenesis of atherosclerosis and thrombosis is dealt with by other speakers in the symposium (Karpe, 1997; Miller, 1997; Sniderman *et al.* 1997). A brief outline of the relationships between CM and VLDL, and cholesterol-rich lipoproteins (LDL-cholesterol and HDL-cholesterol), is given here to illustrate how an elevated postprandial lipaemic response may be related to other lipid abnormalities of the 'metabolic syndrome', such as low HDL, raised fasting triacylglycerols and a preponderance of the LDL-3 (small dense) subtype.

The processes thought to be involved in the relationship between TRL and cholesterol-rich lipoproteins, and which may explain the link between elevated triacylglycerols and accelerated atherogenesis, have been reviewed elsewhere (Sethi *et al.* 1993; Patsch, 1994) and an outline summary of these events is shown in Fig. 2.

An exaggerated response to a fat-containing meal may be due to overproduction of CM or VLDL, or may reflect slower clearance of these particles by the rate-regulatory step in triacylglycerol hydrolysis and removal, which is mediated by lipoprotein lipase (*EC* 3.1.1.34; LPL). Accumulation of partially-hydrolysed remnant particles may also contribute to the elevated triacylglycerol response, since due to their poor recognition by hepatic receptors, there is reduced uptake of these cholesterol-enriched particles by receptor-mediated pathways. The net consequence of these defects is increased retention of TRL within the circulation, providing greater opportunity for neutral-lipid exchange,
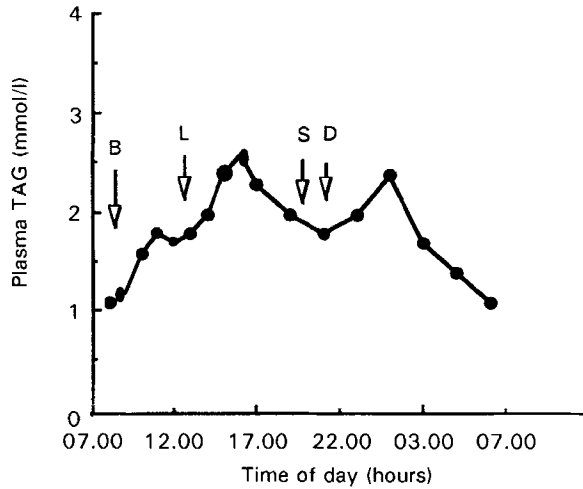
Fig. 1. Plasma triacylglycerol (TAG) responses (24 h) in a 27-year-old man given a breakfast (B), lunch (L), snack (S) and dinner (D) at the times indicated.
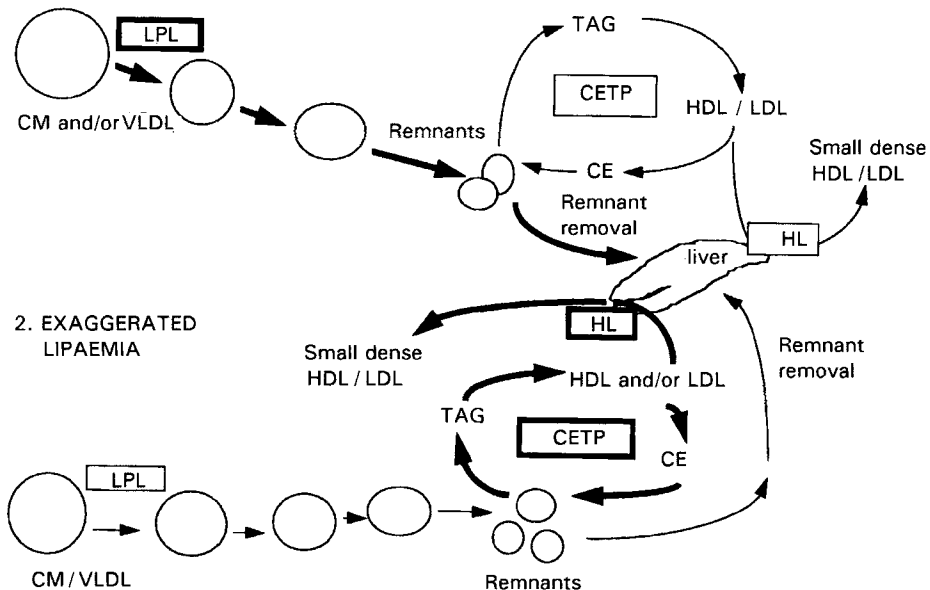

Fig. 2. Diagrammatic representation of the atherogenic consequences of impaired triacylglycerol (TAG) tolerance. 1. Normal lipaemia: (━), TAG-rich lipoprotein (TRL) production rates and clearance and remnant removal rates; (—), exchange with HDL and LDL and hepatic lipase (*EC* 3.1.1.3; HL) action. 2. Exaggerated lipaemia: (—), clearance of TRL and removal of remnants; (—), exchange with HDL and LDL and HL action. CM, chylomicron; CE, cholesteryl ester; CETP, CE transfer protein; LPL, lipoprotein lipase (*EC* 3.1.1.34).

catalysed by cholesteryl ester transfer protein (CETP), which mediates the reciprocal transfer of triacylglycerol and cholesterol between TRL and HDL and LDL. The net consequence of these transfers is triacylglycerol accumulation on HDL and LDL, and cholesterol on CM and VLDL and their remnants. The triacylglycerol-enriched HDL and LDL act as good substrates for the hepatic lipase (*EC* 3.1.1.3; HL) enzyme, resulting in formation of small dense HDL (HDL$_3$) and LDL (LDL-3). The former are catabolized rapidly, resulting in reduced circulating HDL levels. The small dense LDL and the cholesterol-enriched remnant particles are reputed to have greater atherogenic potential due to their prolonged retention within the circulation (Hazzard & Bierman, 1976; Nigon *et al.* 1991), their ability to induce foam cell formation (Parthasarathy *et al.* 1989; Kinoshita *et al.* 1990; Georgopoulos *et al.* 1994) and, in the case of LDL, its greater susceptibility to undergo oxidation (Chait *et al.* 1993). CM remnants have also been shown to penetrate the endothelium in much the same way as LDL and HDL (Mamo & Wheeler, 1994). As discussed elsewhere (Griffin, 1997), the importance of CETP and HL in determining quantitative and qualitative changes in HDL and LDL are the subject of debate and ongoing work, and for the purpose of the present account most emphasis will be placed on the role of dietary fatty acids in modulating postprandial triacylglycerol response, since present evidence suggests it is elevated triacylglycerol concentrations which drive the events described previously.

EFFECTS OF DIETARY FATTY ACIDS ON POSTPRANDIAL LIPAEMIA

In order to evaluate the effect of meal fatty acid composition on the extent of postprandial lipaemia, a number of investigators have compared triacylglycerol responses to single meals of varying fatty acid composition. Some have used circulating markers of intestinally-derived lipoproteins such as retinyl esters and apolipoprotein (apo) B-48 to discriminate intestinal (CM) and hepatic (VLDL) contributions to the postprandial lipaemic response to meals. Variations in postprandial lipaemia in response to meals of different fatty acid composition could reflect differences in the rate of synthesis, secretion or clearance of the intestinally-derived CM and the rate of production and clearance of their remnants. It is also possible that hepatic uptake of fatty acids released from CM, and the nature of the fatty acids in CM remnants presented to the liver could influence, acutely, the synthesis and secretion of VLDL, and thereby the hepatic contribution to the triacylglycerol response to meal consumption. Although some studies have attempted to address the relative contributions of CM and VLDL, the limitation of retinyl esters as specific markers of CM (Krasinski *et al.* 1990), and the paucity of studies using apo B-48 (Foger & Patsch, 1993), make it difficult to draw conclusions regarding the involvement of CM and VLDL in fatty acid-induced variations in postprandial lipaemia. Similar limitations apply to studies which have attempted to address the question of adaptational responses to diets of different fatty acid composition. In these studies, subjects fed on diets for periods ranging from 2 to 8 weeks are usually given test meals of standard fatty acid composition to evaluate effects of background diet on postprandial lipaemia. In these models, any effect of diet could reflect adaptational responses in enterocytic and hepatic enzymes involved in CM and VLDL synthesis, as well as in the lipoprotein and hepatic lipases which regulate clearance of triacylglycerol and remnant particles respectively. Other possible loci for adaptational effects of dietary fatty acids include receptors, such as the remnant and LDL receptors which regulate the uptake and removal of CM and VLDL remnant particles.

Table 1. *Effect of test-meal fatty acid (FA) composition on postprandial lipaemia*

Reference	Type	Fats used	FA composition	Fat	TAG	RP/RE	apo B-48
						Lipaemia measured by	
			Test meal				
Weintraub et al. (1988)	Mixed meal consisting of solid foods	Dairy fats (SFA) v. safflower oil (n-6 PUFA)	P:S 0·08 P:S 1·38	50 g/m² (surface area)	NS	CM and non-CM NS between meals	—
Harris et al. (1988)	Liquid-formula mixed meal	Peanut and coconut oil (SFA) v. MaxEPA or salmon oil	g/100 g total fats as: SFA 37, MUFA 41, PUFA 18 SFA 25, MUFA 48, PUFA 23 (PUFA 23 = n-6 PUFA 3, n-3 PUFA 20)	50 g	NS	—	—
Demacker et al. (1991)	Mixed meals consisting of solid foods	Butterfat (SFA) v. sunflower oil, margarine (n-6 PUFA)	g/100 g total fats as: SFA 21, MUFA 12, PUFA 3, P:S 0·14 SFA 10, MUFA 9, PUFA 18, P:S 1·8	Total fat intake for the day 90 g	Plasma and d < 1·019 TAG NS	—	d < 1·019 apo B-48 reduced 48 % on high-PUFA-meal diet

				Plasma AUC	CM AUC	apo B-48
Zampelas et al. (1994c)	Mixed meal consisting of solid foods	Palm–coconut–olive–sunflower oil mix (SFA) Maize oil (n-6 PUFA) Fish oil (n-3 PUFA)	g/100 g total fats as: SFA 16, MUFA 12, PUFA 4 SFA 6, MUFA 10, PUFA 17 SFA 8, MUFA 7, PUFA 16	40 g	Plasma AUC significantly reduced n-3 PUFA non-significant n-6 PUFA	NS	CM apo B-48 AUC significantly reduced for n-3 PUFA meal
de Bruin et al. (1993)	Fat emulsions	Cream (SFA) Olive oil (MUFA) Soyabean oil (n-6 PUFA)	g/100g total fats as: Not given SFA 14, MUFA 66, PUFA 30 SFA 14, MUFA 23, n-6 PUFA 65	25 g/m² (surface area)	Plasma, CM and non-CM TAG NS between oils	Soyabean oil CM AUC significantly lower than olive oil	Olive oil, 2 and 4 h apo B-48 measurements significantly higher than cream and soyabean oil
Zampelas et al. (1995)	Mixed meals consisting of solid foods	Proportions of oils used: coconut 40, palm 20, olive 30, sunflower 10 (SFA) Coconut 20, palm 20, olive 55, sunflower 5 (SFA) Olive oil 100	% meal energy as: SFA 17, MUFA 12, PUFA 5 SFA 12, MUFA 17, PUFA 5 SFA 5, MUFA 24, PUFA 4	40 g	NS	NS	NS

PUFA, polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; SFA, saturated fatty acids; P:S, polyunsaturated fat:saturated fat ratio; CM, chylomicrons; RP/RE, retinyl palmitate or retinyl ester; AUC, area under the curve; TAG, triacylglycerols; d, density; apo, apolipoprotein.

Although a number of animal feeding studies have used tracer techniques and measurements of tissue enzymes and receptor activities to investigate mechanisms involved in effects of specific fatty acids on CM metabolism (Groot *et al.* 1988; Levy *et al.* 1991), the present review mainly addresses human studies, most of which are limited to measurement of circulating postprandial lipid responses to meals.

ACUTE EFFECTS OF DIETARY FATTY ACIDS ON POSTPRANDIAL LIPAEMIA

There are five studies reported in the literature which have measured postprandial lipaemic responses to meals of different fatty acid composition (Harris *et al.* 1988; Weintraub *et al.* 1988; de Bruin *et al.* 1993; Zampelas *et al.* 1994*c*, 1995) and one which has studied combined effects of both meal and background diet (Demacker *et al.* 1991; Table 1). Comparison of the findings from these studies is difficult, because even when similar comparisons are being made, e.g. SFA (saturated fatty acids) *v.* *n*-6 polyunsaturated fatty acids (PUFA), the nature of the fats and oils used has not always been the same. Similarly the amount of fat used in the test meal and the duration of postprandial follow-up after the meal are frequently different. In one instance, liquid-formula meals have been given (Harris *et al.* 1988) rather than solid foods and in one case, lipid emulsions (de Bruin *et al.* 1993), without addition of carbohydrate or protein, have been used. In most instances, lipaemic response has been evaluated by measurement of plasma and sometimes CM triacylglycerol response.

Weintraub *et al.* (1988) compared postprandial lipoprotein responses to meals comprising predominantly SFA or *n*-6 PUFA. The SFA meal consisted of scrambled egg, cheese, bread and a milk shake, whereas the *n*-6 PUFA meal contained turkey, bread and a milk shake made with safflower oil. No differences in postprandial retinyl palmitate or triacylglycerol responses to the two meals were observed. This is in slight contrast to findings from our study (Zampelas *et al.* 1994*c*) in which subjects were given three test meals which were identical apart from the oils used, which were mixed (SFA), maize (*n*-6 PUFA) and fish oil (long-chain *n*-3 PUFA). The mixed oil was formulated to represent the current UK dietary fatty acid intake. The meals consisted of rice with vegetables, salad and a milk shake and the test oils were added as salad dressing and in a milk shake. Postprandial triacylglycerol response to the *n*-6 PUFA (maize oil) meal was lower than that to the mixed-oil meal but the major difference found was in response to the meal containing long-chain *n*-3 PUFA (fish oil), where marked attenuation in postprandial triacylglycerol levels was seen. Measurement of apo B-48 in the triacylglycerol-rich fraction confirmed that the attenuation represented reduced circulation of CM particles (Peel *et al.* 1993). In an earlier study, Harris *et al.* (1988) found no difference in postprandial triacylglycerol response to liquid test meals containing either a mixture of peanut oil and cocoa butter (saturated-fat meal) or MaxEPA (fish-oil meal). A recent study (Yahia *et al.* 1996) has shown that addition of only small amounts of long-chain *n*-3 PUFA to a standard fat-containing test meal can significantly reduce the postprandial lipaemic response compared with the same meal without added long-chain *n*-3 PUFA, and suggests that the fatty acids in fish oil alter the rate of synthesis and/or removal of CM triacylglycerol, independently of the remaining fatty acid substrate supplied.

There is also some disparity in the findings of studies which have compared effects of monounsaturated fatty acids (MUFA) and other major fatty acid classes on postprandial lipaemia. de Bruin *et al.* (1993) reported that the lipaemic response (postprandial triacylglycerol or retinyl palmitate measurement) was slightly higher when lipid emulsions containing olive oil were fed, than when cream or sunflower oil emulsions were fed. We

compared postprandial triacylglycerol, apo B-48 and retinyl ester responses in sixty volunteers given test meals in which SFA were progressively replaced by monounsaturated fatty acids (MUFA), and found no significant difference in plasma or CM fraction triacylglycerol, apo B-48 or retinyl ester responses (Zampelas et al. 1994a; Jackson et al. 1995). The three test meals fed contained 12, 17 and 24% dietary energy as MUFA, representing respectively current UK, current Mediterranean and previous Mediterranean MUFA intakes. This study suggests that varying meal MUFA composition across a range which reflects worldwide dietary variation has no effect on postprandial lipaemic response.

Based on this small number of studies it is concluded that similar postprandial lipaemic responses are observed when meals consisting of predominantly SFA, MUFA or n-6 PUFA are fed, but when meals containing significant amounts of n-3 PUFA are given, postprandial lipaemia is attenuated.

EFFECTS OF BACKGROUND DIETARY FATTY ACID INTAKE ON POSTPRANDIAL LIPAEMIA

There are a number of different types of studies which have evaluated the impact of altered background diet on postprandial lipaemia. Four studies used a whole-diet approach in which total dietary fat was altered (Harris & Connor, 1980; Harris et al. 1988; Weintraub et al. 1988; Demacker et al. 1991; Table 2), whereas four studies used capsule supplements (Brown & Roberts, 1991; Harris & Windsor, 1991; Williams et al. 1992; Harris & Muzio, 1993; Table 2) and one study incorporated microencapsulated oils into conventional foods (Lovegrove et al. 1997; Table 2), to partially alter background dietary fatty acid intake. One other study has compared effects of different PUFA- and MUFA-containing diets on fasting and postprandial triacylglycerol values but the limited data presented make the findings difficult to compare with other studies and are not included in the table (Lichtenstein et al. 1993).

In those studies which have altered total background diet, most investigators have carried out randomized controlled studies with measurement of subjects responses to a standard meal at the end of a control and a test-diet period. In some instances, investigators have chosen to measure response to a test meal which has the same fatty acid composition as the background diet being fed. This is the case with the study of Demacker et al. (1991) who altered the diets of free-living subjects so that either butterfat or safflower oil was the predominant fat used in cooking and in meals prepared at their work place. After 3 weeks on each diet, subjects responses to a breakfast, lunch and dinner were monitored over a 24 h period. On each occasion the fat content and composition of the test meals given were the same as those of the background diet. In this type of study it is difficult to distinguish the effect of altering the background diet from that of altering the test-meal fatty acid composition. However, this particular study suggests that when a diet and a test meal consisting largely of n-6 PUFA are fed, postprandial triacylglycerol and apo B-48 responses are lower than those when a diet and a meal rich in SFA are fed.

The careful study of Weintraub et al. (1988) was carried out in a metabolic unit with subjects randomized to receive three different background diets for 3 weeks over a 12-week period. The responses of the subjects to two different test meals were evaluated at the end of each dietary period. Lipaemic responses of the subjects were reduced when the background diet consisted of n-6 or n-3 PUFA, although it was only the reduction with n-3 PUFA which was statistically significant when a saturated-fat meal was used as the test meal. The attenuating effects of the n-6 PUFA diet were greater when an n-6 PUFA test meal was given than when a SFA test meal was given, supporting the findings of Demacker et al. (1991), which indicates there may be synergism between the adaptational response

Table 2. *Effect of long-chain dietary n-3 polyunsaturated fatty acid (PUFA) intake on postprandial lipaemia*

| Reference | Background diet | | | | Postprandial lipaemia | | |
	Dietary fats compared	Long-chain n-3 PUFA (g/d)	Duration (d)	Test meal typical fat	TAG		RP/RE
Fish diets							
Harris & Connor (1980)	Salmon oil	24	14	n-3 PUFA	55 % ↓ peak TAG		—
Weintraub et al. (1988)	Details not given, long-chain n-3 PUFA diet contained 30 g/100g total diet fat as fish oil	9	25	SFA	46 % ↓ peak TAG		47 % ↓ RP AUC
Harris et al. (1988)	Peanut oil and cocoa butter v. safflower and maize-oil v. MaxEPA	28	14	SFA	38 % ↓ peak TAG		—
Fish oil capsules or enriched foods							
Brown & Roberts (1991)	Olive oil v. fish oil	1·6	4·2	SFA	28 % ↓ CM AUC		18 % CM RP AUC Non-CM RP AUC, NS
Harris & Windsor (1991)	Olive oil v. fish oil	2·3	28	SFA	30 % ↓ plasma TAG AUC		—
Williams et al. (1992)	Fish oil supplements v. usual diet	2·8	42	SFA	32 % ↓ plasma TAG		—
Harris & Muzio (1993)	Olive oil v. fish oil	approximately 5·0	28	SFA	31 % ↓ plasma TAG AUC		43 % ↓ CM RP 27 % ↓ VLDL RP
Lovegrove et al. (1997)	Control foods v. n-3 PUFA-enriched foods	approximately 1·6	21	SFA	NS		NS

SFA, saturated fatty acid; CM, chylomicron; RP, retinyl palmitate; RE, retinyl esters; TAG, triacylglycerol; AUC, area under curve.

and acute-meal mechanism. Harris *et al.* (1988) also found hypolipaemic effects of a diet rich in fish oil in a study in which they fed either a control diet consisting of peanut oil and cocoa butter or a diet consisting of salmon oil or MaxEPA as background.

Data obtained from these diet studies suggest that the dietary fats fed in the background diet may markedly influence subjects' responses to a standard fat-containing meal and that, in particular, diets enriched with fish oils containing long-chain *n*-3 PUFA significantly increase triacylglycerol tolerance.

In the light of the convincing evidence for potent triacylglycerol-lowering effects of dietary long-chain *n*-3 PUFA, one question which needs to be addressed is the level at which these fatty acids need to be fed in the normal human diet in order to maintain optimally low levels of postprandial triacylglycerols. Useful information is available from studies which have used capsule preparations (Table 2). From these studies it would appear that diets containing 1·6–2·8 g long-chain *n*-3 PUFA result in 30 % reduction in postprandial lipaemia. Higher levels than this, e.g. 5 g/d, can achieve an even greater degree of reduction. This level of supplementation is considerably higher than habitual levels of intake of long-chain *n*-3 PUFA found in most human diets, since in the UK average levels of intake are approximately 0·2 g/d (Gregory *et al.* 1991). This compares with intake levels of 12 g/d for linoleic acid, the major *n*-6 PUFA, and 1·3 g/d for α-linolenic acid, the precursor of *n*-3 PUFA. If, as might be expected, future dietary strategies for prevention of CHD consider the need for reductions in fasting and postprandial triacylglycerol levels, an important question which must be addressed is the form (either precursor or long-chain), and level, at which the *n*-3 PUFA should be provided in the diet.

EFFECTS OF HABITUAL FATTY ACID INTAKE ON POSTPRANDIAL LIPAEMIA

Although the dietary studies described previously are designed to address the question of adaptational responses to altered diet, it is likely that these responses develop over many weeks or even months, depending on the site and mechanism involved. It is possible that many of the studies conducted to date have been too short to reveal the full extent of changes in lipoprotein metabolism which occur when fatty acid intake is markedly altered in human diets. This is particularly so in the light of the large number of sites through which fatty acids may mediate effects on triacylglycerol metabolism. This means that differences may not become evident in short-term dietary studies where the different diets have only been fed for a limited period of time (usually weeks). This conclusion is supported by a recent study we have carried out which measured postprandial responses to standard meals in subjects living in southern and northern Europe whose diets closely reflected the typical diets of their country (Williams *et al.* 1995; Zampelas *et al.* 1997). We found the pattern of lipaemic response to identical meals was very different in southern than northern Europeans and that, in particular, subjects from southern Europe showed a marked early rise in triacylglycerol with rapid return to fasting values, whereas northern Europeans showed a slow sluggish rise in triacylglycerols which did not return to fasting values until 8 or 9 h after the meal (Fig. 3). The most noticeable difference between the two groups was the lower triacylglycerol concentrations in the late postprandial period in the southern European group. Whether these findings have any significance to differences in risk of CHD between these two populations is not known, but they are consistent with the observation that elevations in late postprandial triacylglycerol values, between 6 and 10 h after a meal, have been identified as a significant risk factor for CHD in a number of case–control studies (Groot *et al.* 1991; Patsch *et al.* 1992), and in a study of the offspring of subjects with CHD (Uiterwaal *et al.* 1994).
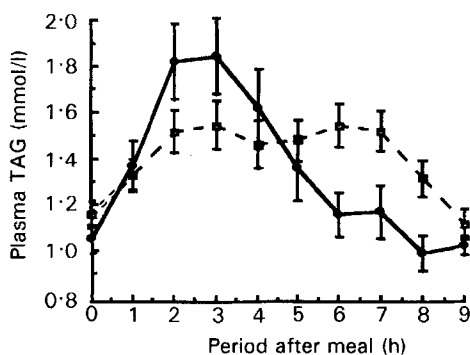
Fig. 3. Postprandial plasma triacylglycerol (TAG) responses to standard mixed test meals containing 40 g fat in young male subjects from: (●—●), southern (n 30); (□--□), northern Europe (n 30). (From Williams *et al.* 1995.)

MECHANISM INVOLVED IN FATTY ACID-MEDIATED ALTERATIONS IN POSTPRANDIAL LIPAEMIA

A detailed discussion of the mechanisms involved in fatty acid-mediated alterations in postprandial lipaemia is beyond the scope of the present review, although some consideration of possible sites has been given previously. Human test-meal studies suggest variations in meal fatty acid composition have modest effects on postprandial lipaemia, apart from marked attenuation of triacylglycerol responses when long-chain n-3 PUFA are fed. Longer-term diet studies suggest that n-6 PUFA may have moderate triacylglycerol-lowering effects, but marked hypolipaemic effects of long-chain n-3 PUFA are evident from a number of diet and supplementation studies. Animal studies have shown effects of meal fatty acid composition on postprandial lipaemia to depend in part on differences in CM size, with the PUFA producing CM of larger size which appear to act as better substrates for the LPL enzyme (Groot *et al.* 1988; Levy *et al.* 1991). There is also some evidence from animal studies (Table 3) to suggest that PUFA and MUFA increase the activity of LPL and thereby enhance triacylglycerol clearance. Animal studies are generally in agreement that diets containing large amounts of PUFA and MUFA significantly increase either post-heparin LPL (Groot *et al.* 1988; van Heek & Zilversmit, 1990) or adipose tissue LPL activity (Levy *et al.* 1991; Murphy *et al.* 1993) and one study has shown increased gene expression of LPL in epididymal fat pads of animals fed on fish oils (Murphy *et al.* 1993). However, human studies have generally failed to find increased post-heparin LPL activity when high-PUFA diets are fed (Harris *et al.* 1988; Weintraub *et al.* 1988; Nozaki *et al.* 1991), although one study has shown elevated post-heparin LPL activity 9 h following meals enriched with long-chain n-3 PUFA (Zampelas *et al.* 1994*b*; Table 3). Conflicting findings in these type of studies may be due to lack of precision and specificity of the post-heparin LPL measurements. Post-heparin LPL measurements include both LPL from skeletal muscle and adipose tissue, but animal studies suggest the activating effects of long-chain n-3 PUFA are localized to adipose tissue, so that these non-specific measurements may fail to detect single-site responses. However, further evidence that the effects of long-chain n-3 PUFA do not reflect activation of LPL was provided by Harris & Muzio (1993), who found that enrichment of diets with fish oil fatty acids, although markedly reducing postprandial responses to standard meals, did not accelerate removal of an artificial lipid emulsion when this was given intravenously. These findings

Table 3. *Effects of dietary fatty acids on indices of lipoprotein lipase (EC 3.1.1.34; LPL)*
activity

Reference	Species	Diets	LPL measurement
Animal studies			
Groot *et al.* (1988)	Rat	Meals containing either palm or sunflower oil	Post-heparin LPL 10 and 12 h after meal significantly higher in sunflower oil-fed rats
van Heek & Zilversmit (1990)	Rabbit	Diets containing either 140 g coconut oil or 140 g olive oil/kg	Post-heparin LPL three times higher in olive oil-fed animals after 49 d
Levy *et al.* (1991)	Rat	Diets containing MCT, coconut or safflower oils	Adipose tissue LPL activity significantly higher in safflower oil-fed animals
Murphy *et al.* (1993)	Rat	Diets containing mixed, maize or fish oils	Adipose tissue LPL activity higher (NS) and LPL mRNA significantly higher in fish oil-fed animals
Human studies			
Weintraub *et al.* (1988)		Background diets typified by SFA, *n*-6 PUFA or *n*-3 PUFA	Post-heparin LPL activity higher (NS) on *n*-3 PUFA diet
Harris *et al.* (1988)		Background diets typified by SFA, *n*-6 PUFA or *n*-3 PUFA	Post-heparin LPL and HL activities not significantly different for the three diets
Nozaki *et al.* (1991)		Diet supplemented with 10 g long-chain *n*-3 PUFA/d Hypertriacylglycerolaemic subjects	Post-heparin LPL and HL activities not significantly different with *n*-3 PUFA supplementation

MCT, medium-chain triacylglycerols; SFA, saturated fatty acids; PUFA polyunsaturated fatty acids; HL, hepatic lipase (*EC* 3.1.1.3).

have directed attention towards the enterocyte as a possible site of action of the acute effects of long-chain *n*-3 PUFA and studies using CaCo-2 cells have confirmed the inhibitory effects of these fatty acids on lipogenic enzymes involved in CM synthesis (Chautan *et al.* 1990; Murthy *et al.* 1990). The different patterns of triacylglycerol response observed in the comparison of northern and southern Europeans (Williams *et al.* 1995; Zampelas *et al.* 1997) also indicate potential effects of fatty acids on the gastrointestinal processing of dietary fat and the need for further studies of effects of fatty acids on the molecular processes involved in CM synthesis, assembly and secretion.

CONCLUSION

An exaggerated postprandial lipaemic response is becoming increasingly recognized as an important lipid risk factor for CHD. Relatively few studies of effects of meal and background dietary fatty acid composition on postprandial lipoprotein responses have been conducted, in contrast to the situation for fasting lipoproteins measurements, where many hundreds of studies have been reported in the literature.

Meal and background dietary fatty acid intake can influence both the extent and the pattern of postprandial triacylglycerol responses to fat ingestion, with the greatest effect observed for the long-chain *n*-3 PUFA which have potent triacylglycerol-lowering properties, both when added to individual meals, and when included in the habitual diet.

Further studies are required to determine the optimum type and balance of dietary fatty acids which can maintain a low postprandial response to meal consumption. Mechanisms underlying modulatory effects of dietary fatty acids need further investigation.

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