The effect of low and moderate fat intakes on the postprandial lipaemic and hormonal responses in healthy volunteers

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Present literature indicates that whereas an acute fat intake of 5 g does not elicit a postprandial triacylglycerolaemic response, 20 g of fat does. Since 67 % of fat intake occasions involve fat doses of less than 20 g, the present study examined the effect of a relatively low-fat (LF) meal (0·2 g/kg body weight; mean 14 g) on postprandial triacylglycerol (TAG) metabolism, compared with a high-fat (HF) meal (0·6 g/kg body weight; mean 43 g), a fat dose which is more typical of laboratory studies. Plasma- and chylomicron-TAG concentrations increased significantly ($P \leq 0{\cdot}001$) following both meals, and the increase was significantly ($P \leq 0{\cdot}02$) greater after the HF meal. The postprandial areas under the curves and maximal postprandial TAG concentrations for plasma- and chylomicron-TAG were significantly higher following the HF meal ($P \leq 0{\cdot}05$). Postprandial plasma insulin and gastric inhibitory polypeptide concentrations increased significantly ($P \leq 0{\cdot}001$) after each meal, but there was no difference between the two meals. These data show that modest amounts of fat in a meal will elicit a measurable postprandial TAG response. Since postprandial lipaemia affects the composition and concentration of the TAG- and cholesterol-rich lipoproteins, controlling dietary TAG supply may influence the metabolic fate of these lipoproteins.

Postprandial lipaemia: Triacylglycerols: Dietary fat

The acute effect of fat ingestion on the postprandial lipaemic response has been extensively investigated. The amount of fat used in test meals, in general, has varied between 40 and 100 g (Redgrave & Carlson, 1979; Groot & Scheek, 1984; Cohen *et al.* 1988; Cohn *et al.* 1988*a*; Bonanome & Grundy, 1989; Krasinski *et al.* 1990; Rifai *et al.* 1990; Zampelas *et al.* 1994*a*,*b*; Roche & Gibney, 1996). However, a recent study in this laboratory (K Harrington, personal communication) of 106 free-living healthy adult males, observed over a 7 d period involving more than 3000 eating occasions, has shown that on 26 % of occasions fat ingestion is below 5 g, on 41 % of occasions fat ingestion is between 5 and 20 g and on only 33 % of occasions is fat intake above 20 g. De Castro (1987) has also reported data which reveal that most eating occasions involve fat intakes of between 12 and 30 g. In planning a study of the effect of moderate fat intakes on the postprandial lipaemic response, note was taken of the literature prevailing at that time.

Cohen *et al.* (1988) showed that the magnitude of postprandial lipaemia was directly related to fat ingestion in studies involving doses of 40, 80 and 120 g fat. Dubois *et al.* (1994) found significant differences in postprandial lipaemia with doses of 31 and 43 g fat. Chen *et al.* (1992) examined the postprandial triacylglycerol (TAG) response in subjects with non-insulin dependent diabetes mellitus with doses of fat to provide either 25 % or 45 % of dietary energy. More recent publications have used doses as low as 5 g compared with doses of 40 or 80 g fat (Jeppesen *et al.* 1995) and doses from 20 to 80 g fat (Murphy *et al.* 1995). Since the completion of the present study, Dubois *et al.* (1998) have extended this area to examine the effects of 0, 15, 30, 40 and 50 g fat where the lowest fat dose (15 g) elicited a small increase in postprandial plasma TAG concentrations. Previous work in this laboratory has shown that 11 g fat can elicit a postprandial TAG response of sufficient magnitude to stimulate reverse cholesterol transport (O'Flaherty & Gibney, 1994). Clearly, a need exists to clarify the capacity of lower doses of fat to elicit a postprandial lipaemic response, given that some 67 % of eating occasions involve fat intakes of less than 20 g.

Murphy *et al.* (1995) showed that doses of 20 g fat were capable of eliciting a plasma gastric inhibitory polypeptide (GIP) response. GIP augments insulin-mediated stimulation of lipoprotein lipase (*EC* 3.1.1.34), the enzyme that

Abbreviations: AUC, area under the curve; CM, chylomicron; C_{MAX}, maximum postprandial concentration; GIP, gastric inhibitory polypeptide; HF, high fat; LF, low fat; NEFA, non-esterified fatty acid; TAG, triacylglycerol.

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catalyses plasma TAG clearance. Therefore the postprandial responses of insulin and GIP, as well as plasma lipid concentrations were investigated in the present study. The low and high fat doses (0·2 and 0·6 g fat/kg body weight) yielded mean acute intakes of 14 g for the low-fat (LF) meal and 43 g for the high-fat (HF) meal. This represents the range of fat intakes which are commonly consumed on a single eating occasion.

Experimental methods

Study design

This study was approved by the Ethics Committee of the Federated Dublin Voluntary Hospitals. Eight healthy subjects, four male and four female, participated in the study. None was taking any medication or nutritional supplements which would interfere with lipid metabolism. The subjects' mean age was 28·4 (range 22–36) years and the mean BMI was 24·1 (range 19·9–27·2) kg/m^2. Subjects refrained from drinking alcohol and taking strenuous exercise for 24 h before each postprandial investigation and avoided smoking during these investigations. They took a light dinner on the evening before the studies and reported to the laboratory between 08.00 and 09.00 hours, following a 12 h overnight fast. A 21 gauge, 32 mm venous cannula catheter (Abbot Ireland Ltd, Dublin, Republic of Ireland) was inserted into the antecubital vein of the forearm and a fasting sample was collected. Following meal ingestion, blood samples were drawn at 2, 4, 6 and 8 h for lipid analysis and additionally at 3, 5 and 7 h for insulin and GIP analysis. Blood was drawn into vacutainer tubes (containing 0·12 ml 0·3 M-EDTA K_3). After each blood sample was taken the cannula was rinsed with 1 ml normal saline and the first 10 ml blood drawn at each sampling time point was discarded.

Test meal composition

The test meals were liquid blends of skimmed milk and soyabean oil. The LF meal provided 0·2 g fat, 0·2 g carbohydrate and 0·18 g protein per kg body weight while the HF meal contained 0·6 g fat, 0·2 g carbohydrate and 0·18 g protein per kg body weight. Because of variation in body weight, the range of fat intakes for the LF meal was 10–18 g (mean 14 g) while for the HF diet it was 31–56 g (mean 43 g). The average energy intake was 979 kJ for the LF meal and 2051 kJ for the HF meal.

Biochemical analysis

Plasma samples were immediately centrifuged at 2500 rev./min for 15 min, the plasma was harvested, divided into portions and frozen (−20°) immediately for subsequent analysis. Plasma for chylomicron (CM) separation was stored overnight (2–5°). The plasma CM fraction was separated using the method of Grundy & Mok (1976) with some modification, as previously described (Roche *et al.* 1998). In a 4·7 ml Optiseal polyallomer centrifuge tube (Beckman Instruments Inc., Palo Alto, CA, USA), 2·35 ml plasma was overlaid with 2·35 ml saline ($\rho = 1{\cdot}006$ g/ml). The CM fraction was isolated by ultracentrifugation (100 000 rev./min for 24 min ($5{\cdot}29 \times 10^6$ ***g***.min), at 4°) (Beckman Optima TLX ultracentrifuge with a TLA 100.4 rotor, Beckman Instruments Inc.). The CM fraction was harvested and stored (−20°) for subsequent analysis.

Enzymic colorimetric assays were used to determine plasma TAG, CM-TAG (PAP 500; Biomerieux, France) and plasma non-esterified fatty acid (NEFA) (NEFAC; Wako Chemical GmbH, Neuss, Germany) concentrations. Plasma insulin concentrations were measured by radioimmunoassay (Kabi Pharmacia (Ireland) Ltd, Dublin, Republic of Ireland). Plasma GIP was measured by radioimmunoassay (Alam *et al.* 1992); this analysis was kindly carried out by Professor K. Buchanan (Wellcome Research Laboratories, Royal Victoria Hospital, Belfast, UK).

Data were analysed by repeated measures ANOVA using Data Desk 4.1 (Data Description Inc., Ithaca, NY, USA). The repeated measures model was designed to investigate the effect of several independent variables, including subject, sex, meal and time, on the postprandial response. All the data, with the exception of plasma NEFA, were transformed to their natural log to render their distribution normal. The NEFA data were transformed using the square root to normalize their distribution. Summary postprandial variables of the postprandial response including the area under the curve (AUC) (Mathews *et al.* 1990), the incremental AUC, maximum postprandial concentrations (C_{MAX}) and time to C_{MAX}, were analysed by ANOVA. Multiple regression analysis was used to explore factors influencing GIP AUC.

Results

Postprandial plasma TAG, CM-TAG and NEFA values are presented in Table 1. The concentrations of TAG in plasma and the CM fraction were significantly ($P \leq 0{\cdot}0001$) increased during the postprandial response phase. There were significantly greater increases in plasma- and CM-TAG concentrations ($P = 0{\cdot}01$ and $P = 0{\cdot}02$ respectively) following the HF meal; the *post-hoc* least significance difference test showed that this difference between meals occurred at 4 and 6 h ($P \leq 0{\cdot}05$). Postprandial plasma NEFA concentrations were significantly ($P \leq 0{\cdot}0001$) reduced during the postprandial phase, but there was no difference between meals. The repeated measures model took account of the sex mix in the study design; there was no significant effect of sex on plasma TAG or NEFA concentrations. CM-TAG concentrations were significantly ($P = 0{\cdot}03$) greater in the men; however, there was no sex × meal interaction, which demonstrates that sex did not influence the difference seen between meals.

The plasma insulin and GIP responses are presented in Table 2. The concentrations of both variables increased significantly during the postprandial response phase ($P = 0{\cdot}0001$ and $P = 0{\cdot}0003$ respectively), but there was no significant difference between meals. Sex had no effect on either of these variables. The summary variables of the postprandial response (AUC, incremental AUC, C_{MAX} and time to C_{MAX}) are presented in Table 3. The postprandial plasma TAG AUC, incremental AUC and C_{MAX}

Table 1. Postprandial plasma triacylglycerol (TAG), chylomicron-TAG (CM-TAG) and non-esterified fatty acid (NEFA) responses to the ingestion of a high-fat (HF) or low-fat (LF) test meals by healthy volunteers‡

(Mean values and standard deviations for eight subjects)

		Time after test meal (h)										Statistical significance of effect of: (ANOVA, $P =$)		
		0		2		4		6		8				
	Meal	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Meal (M)	Time (T)	M×T interaction
Plasma TAG (mmol/l)	HF	0·78	0·35	0·81	0·36	1·24*	0·50	1·19*	0·60	0·81	0·42	0·0102	0·0001	NS
	LF	0·67	0·45	0·63	0·42	0·92*†	0·40	0·82*†	0·48	0·66	0·38			
CM-TAG (mmol/l)	HF	0·25	0·19	0·30	0·22	0·69*	0·45	0·59*	0·43	0·26	0·22	0·0171	0·0001	NS
	LF	0·20	0·24	0·22	0·20	0·43*†	0·30	0·32†	0·33	0·19	0·21			
NEFA (mmol/l)	HF	0·44	0·15	0·27	0·16	0·52	0·19	0·67*	0·23	0·64*	0·28	NS	0·0001	NS
	LF	0·52	0·25	0·27*	0·14	0·46	0·18	0·63	0·20	0·83*	0·29			

Mean values were significantly different from fasting values, *$P < 0·05$.
Mean values were significantly different from those for the HF meal, †$P < 0·05$.
‡ For details of meals and procedures, see p. 26.

values were significantly greater following the HF meal than after the LF meal ($P = 0·03$, $P = 0·05$ and $P = 0·02$ respectively). Sex had no effect on the summary variables for the postprandial plasma TAG response. The CM-TAG AUC and C_{MAX} were significantly greater following the HF meal than after the LF meal ($P = 0·03$ and $P = 0·01$ respectively). For both of these variables CM-TAG levels were greater in males than in females, but these differences did not reach statistical significance ($P \geq 0·06$), and there was no sex×meal interaction which demonstrates that sex did not influence the difference between meals. The summary variables of the postprandial GIP and insulin responses were not significantly different between meals.

In the case of GIP, it was clear that there was a tendency toward a higher and more prolonged response after the HF meal and given the large between-subject variation for this variable, a regression analysis was completed to examine the determinants of GIP AUC (Table 4). Fasting plasma GIP concentrations (GIP-0) explained 39 % of the GIP AUC variation (r^2 0·39, $P = 0·0076$). When 'subject' was included in the model the strength of the equation increased substantially (r^2 0·756, $P < 0·0001$ for GIP-0 and $P = 0·007$ for subject). CM-TAG AUC had a significant, albeit minor, influence on the regression equation (r^2 0·828, $P = 0·0015$ for GIP-0; $P = 0·0004$ for subject; $P = 0·0325$ for CM-TAG AUC). The effects of several other variables were investigated but these three were the important factors determining the postprandial plasma GIP AUC.

Discussion

The results of the present study show that a dose of fat more akin to the normal level of fat consumed on a single eating occasion in free-living subjects, elicits a small but

Table 2. Postprandial insulin and gastric inhibitory polypeptide (GIP) responses to the ingestion of a high-fat (HF) or low-fat (LF) test meals by healthy volunteers†

(Mean values and standard deviations for eight subjects)

	Insulin (μU/ml)				GIP (ng/l)			
	HF		LF		HF		LF	
Time after test meal (h)	Mean	SD	Mean	SD	Mean	SD	Mean	SD
0	8·62	3·78	8·56	1·67	19·4	8·6	18·1	7·5
1	12·10*	7·27	11·75*	5·0	30·6	15·0	27·5	9·3
2	8·64	2·66	8·38	2·19	36·3*	21·0	28·8	11·9
3	8·91	4·52	6·85	1·88	49·4*	40·1	34·3*	13·7
4	6·94	1·60	6·26*	1·72	34·4*	18·8	38·8*	16·6
5	6·27*	2·65	5·38*	1·16	43·8*	39·4	26·4	12·2
6	6·47	2·51	4·86*	1·84	38·8*	30·6	23·1	10·0
7	4·95*	1·73	5·10*	1·22	31·3	13·0	28·8	15·1
8	4·79*	1·70	5·48*	1·70	25·6	10·8	23·1	8·4
Statistical significance of effect of: (repeated measures ANOVA, $P =$)								
Meal (M)		NS				NS		
Time (T)		0·0001				0·0003		
M×T interaction		NS				NS		

Mean values were significantly different from fasting values, *$P < 0·05$.
† For details of meals and procedures, see p. 26.

Table 3. Summary postprandial data for plasma triacylglycerols (TAG), chylomicron-TAG (CM-TAG), insulin and gastric inhibitory polypeptide (GIP) following ingestion of high-fat (HF) and low-fat (LF) test meals by healthy volunteers†

(Mean values and standard deviations for eight subjects)

		Area under the curve				Time to maximum observed value (h)		Maximum observed value (units)	
		Total (units/8 h)		Incremental (units/8 h)					
	Meal	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Plasma TAG (mmol/l)	HF	8·07*	3·46	1·84*	1·23	4·50	0·93	1·37*	0·57
	LF	6·07	3·37	0·73	0·46	4·75	1·04	0·94	0·41
CM-TAG (mmol/l)	HF	3·67*	2·44	1·66	1·31	4·50	0·93	0·78*	0·46
	LF	2·32	2·06	0·69	0·45	4·00	1·07	0·44	0·30
Insulin (μU/ml)	HF	61	23	53	26	1·75	0·89	12·8	6·7
	LF	56	10	43	22	1·63	1·06	12·2	4·7
GIP (ng/l)	HF	287	159	419	275	2·75	1·39	58	38
	LF	221	67	296	108	3·00	1·07	44	15

Mean values were significantly different from those for the LF meal, *$P < 0·05$.
† For details of meals and procedures, see p. 26.

significant rise in postprandial TAG level. At the outset of this study, the prevailing literature showed that 5 g fat did not elicit a significant lipaemic response (Jeppesen *et al.* 1995), but that a dose of 20 g fat did (Murphy *et al.* 1995). In the present study, fat intakes of 14–15 g significantly increased plasma TAG concentrations, although the postprandial response was small and short-lived. Recently, Dubois *et al.* (1998) examined the postprandial TAG response to a fat-free meal and to meals containing 15, 30, 40 and 50 g fat. The 15 g fat dose elicited a modest but significant rise in CM-TAG 2 h after the meal, although the AUC for the postprandial CM-TAG response was not significantly increased, whereas the greater fat doses (30, 40 and 50 g) significantly increased the postprandial CM-TAG AUC, in a dose-dependent fashion. This research group previously reported that modest differences in fat intake (31 g and 42 g) significantly influenced postprandial triacylglycerolaemia (Dubois *et al.* 1994). However, in contrast, Murphy *et al.* (1995) found no significant difference in plasma TAG response between intakes of 20 g and 40 g fat but did observe a significantly higher postprandial TAG response when 80 g fat was used. These contrasting findings probably reflect different experimental conditions between the studies.

Table 4. Multiple regression modelling of the area under the plasma gastric inhibitory polypeptide (GIP) curve (GIP AUC) and the effects of fasting GIP levels (GIP-0), subject, fat intake (g) and the area under the chylomicron-triacylglycerol curve (CM-TAG AUC) in postprandial data from eight subjects

Dependent variable = GIP AUC Independent variables	$P =$	r^2 adjusted
(i) GIP-0	0·0076	39·0 (F 9·96)
(ii) GIP-0	< 0·0001	75·6 (F 22·7)
Subject	0·007	
(iii) GIP-0	0·0015	82·8 (F 23·4)
Subject	0·0004	
CM-TAG AUC	0·0325	

Dubois *et al.* (1998) demonstrated that the incremental increase in the AUC for serum TAG per unit weight of fat ingested following meals containing 30, 40 and 50 g was constant (0·090, 0·089 and 0·091 mmol.h/l respectively) and much greater than that following a meal containing 15 g fat (0·043 mmol.h/l). Therefore the TAG-clearing capacity of the normolipaemic subjects was not exceeded by the 15 g fat meal but it was exceeded by the higher fat meals, in proportion to the amount of fat ingested. Postprandial lipaemia not only represents the flux of dietary TAG within the circulation, but it is a key metabolic event which determines the composition and metabolic fate of HDL and LDL (Roche & Gibney, 1995). In coronary artery disease, the preponderance of small dense LDL particles and low HDL-cholesterol concentrations are all associated with an elevated postprandial TAG response, whereas an attenuated response has beneficial effects, such as reverse cholesterol transport (O'Flaherty & Gibney, 1994). Dubois *et al.* (1998) showed that although 15 g fat caused relatively unimportant changes in postprandial TAG concentrations, this fat dose was capable of affecting the composition and concentration of HDL and LDL. The present study supports the finding that 14–15 g fat can stimulate a postprandial lipaemic response. Therefore controlling dietary TAG not only determines the magnitude of the postprandial TAG response, but it can be associated with the beneficial effects on LDL and HDL metabolism, such as reverse cholesterol transport.

Sex has been shown to affect the magnitude of the postprandial response, that is, peak postprandial TAG concentrations were greater in males than females (Kashyap *et al.* 1983; Cohn *et al.* 1988*b*). However, in those studies fasting plasma TAG levels were also greater in the male subjects. Since fasting TAG concentration is a strong predictor of the magnitude of the postprandial response, this may explain the greater postprandial TAG concentrations. Alternatively the difference in postprandial TAG concentrations may be due to the greater levels of lipoprotein lipase in women than men (Kashyap *et al.* 1983; Weintraub *et al.* 1987). Lipoprotein lipase stimulates the clearance of

postprandial TAG. In the present study, the study cohort was composed of both men and women, therefore sex was included as an independent factor in the statistical model used to analyse the data. Neither fasting nor postprandial plasma TAG concentration was significantly affected by sex; however, both fasting and postprandial CM-TAG concentrations were significantly greater in the male subjects, as illustrated in Fig. 1. The males had higher CM-TAG concentrations than the females, while the HF meal generated a greater postprandial lipaemic response than the LF meal, in both groups.

The pattern of postprandial NEFA response was similar to that previously reported (Gibney & Daly, 1994; Murphy *et al.* 1995), that is, an initial decline followed by a gradual rise, but there were no significant differences between the HF and LF meals. The postprandial insulin response was not affected by the level of fat provided in the test meals. Murphy *et al.* (1995) also showed no significant effect of 20, 40 or 80 g fat on postprandial insulinaemia. However, Dubois *et al.* (1998) demonstrated that the insulin AUC was significantly greater following a 50 g fat meal, compared with a 15 g fat meal. In the present study, the postprandial GIP response was not significantly different between meals, although plasma GIP concentrations were elevated for a longer time following the HF meal. There is some evidence to suggest that the postprandial plasma GIP response to fat is dose-dependent. Murphy *et al.* (1995) demonstrated that the postprandial GIP response was significantly greater following an 80 g fat meal, but there was no significant difference between the 20 and 40 g fat doses.

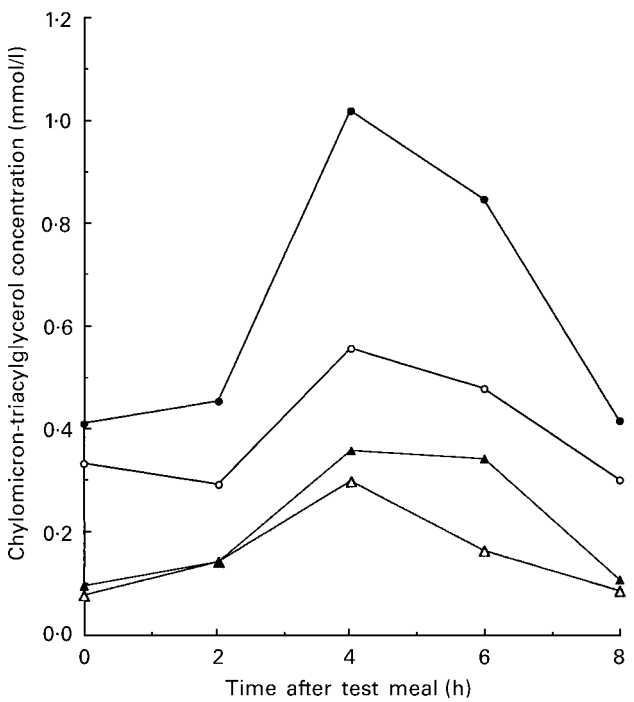

Fig. 1. Postprandial chylomicron-triacylglycerol concentrations following high-fat (HF) and low-fat (LF) test meals in male and female subjects. (○), LF meal, males; (●), HF meal, males; (△), LF meal, females; (▲), HF meal, females. Values are means for four subjects. For details of meals and procedures, see p. 26.

Regression analysis was completed to identify the significant determinants of GIP AUC; this showed that fasting GIP concentrations and between-subject variation were the most important factors, and CM-TAG AUC to a lesser extent. Clearly non-dietary factors play a greater role in determining the GIP response to fat ingestion than does the dose of fat ingested. Although chronic high-fat diets have been found to lead to an increased postprandial sensitivity in GIP release in response to oral glucose loads, these findings would not support the concept that chronic rather than acute fat intake influences fasting plasma GIP (Knapper *et al.* 1996). The effect of fat intake on plasma GIP response and the significance of postprandial GIP response to CM and NEFA metabolism is poorly understood and warrants investigation.

In conclusion, low doses of fat (0·2 g/kg) were found to elicit a weak and short-lived postprandial TAG response and this would indicate that for the majority of eating occasions, where fat intake is known to be at or about this level, some degree of lipaemia will be encountered. This has implications in the area of periodicity of eating and its influence on plasma cholesterol through activation of reverse cholesterol transport via postprandial inter-lipoprotein lipid exchange (Mann, 1997).

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