Invited commentary

Dietary factors and postprandial lipaemia

When we eat a meal containing, say, 100 g carbohydrate, the glucose that is absorbed enters a pool of free glucose in the body of about 12 g. It does not, however, lead to an 8-fold increase in the plasma glucose concentration because co-ordinated mechanisms come into play that suppress the entry of endogenous glucose into the plasma, and stimulate the removal of glucose from plasma (Fery *et al.* 1990). It is not always appreciated that exactly the same arguments can be applied to fat metabolism. A typical intake of dietary fat for those of us eating Western diets is about 100 g/d. About 95 % of this is in the form of triacylglycerol (TG). Therefore we eat about 30–50 g fat in a notional 'typical' meal, depending, of course, on how we distribute the fat intake amongst our daily meals. The amount of TG circulating in the plasma is approximately 3 g (taking a typical plasma TG concentration as 1 mmol/l). Therefore, a superficial analysis suggests that the plasma TG concentration will increase 10-fold after a meal. In fact, in healthy people the rise is usually considerably less than 2-fold. This is, again, because the ingestion of a meal sets in train a series of co-ordinated changes in fat metabolism that tend both to suppress the entry of endogenous TG into the plasma, and to increase TG disposal from the plasma (Frayn, 1993).

The magnitude of the increase in plasma TG following a meal (postprandial lipaemia) is now recognized to be an important risk marker for CHD (Groot *et al.* 1991; Patsch *et al.* 1992). Impaired postprandial lipid metabolism (increased postprandial lipaemia) seems to be a feature of the atherogenic lipoprotein phenotype, the dyslipidaemic phenotype associated with insulin resistance (Griffin & Zampelas, 1995; Jeppesen *et al.* 1995*b*), which is a much more prevalent factor underlying CHD than is simple elevation of the plasma cholesterol concentration (Lamarche *et al.* 1995). Few would argue that elevated TG concentrations themselves have direct atherogenic potential; it seems more likely that they are a marker for some underlying disturbance of lipid metabolism. In principle, disruption of any aspect of the co-ordinated response discussed earlier might lead to alterations in postprandial lipaemia. The associated risk of CHD appears to be related to the persistence in the circulation of remnant particles derived from the chylomicrons that carry the dietary TG into the plasma (Karpe *et al.* 1994): these may represent the actual 'atherogenic particle' (Havel, 1994; Weintraub *et al.* 1996).

There is, therefore, great interest in understanding the factors that contribute to determination of plasma TG concentrations in the postprandial state. Many factors are known to influence the magnitude of postprandial lipaemia, including age and sex (Cohn *et al.* 1988), obesity (Lewis *et al.* 1990) and diabetes (Akanji *et al.* 1992), and the amount of fat in the test meal (Dubois *et al.* 1998). The presence of sucrose or fructose in the test meal also substantially potentiates the response (Cohen & Schall, 1988; Jeppesen *et al.* 1995*a*; Abraha *et al.* 1998).

These factors all affect the response quantitatively rather than qualitatively. In most studies it is found that dietary TG enters the circulation relatively slowly (compared with glucose from dietary carbohydrate), plasma TG concentrations reaching a peak somewhere between 2 and 5 h following a fat-rich meal. It is common to imagine a smooth, single peak in plasma TG concentration as is usually seen in figures in papers examining this process. However, graphs of mean values can often hide variations in the shapes of curves between and within individual subjects. Although some studies specifically report that the shape of the curve was monophasic in individual subjects (e.g. Potts *et al.* 1994), in other studies a variety of types of curve, with from one to three peaks, has been reported (Cohn *et al.* 1989). In this issue of the Journal, Shishehbor, Roche and Gibney publish the interesting observation that the amount of carbohydrate given with a fixed amount of fat determines whether a mono- or biphasic response in plasma TG concentration is observed (Shishehbor *et al.* 1998). Shishehbor *et al.* (1998) argue that the pattern of response observed reflects the rate of intestinal absorption of fat. Certainly there are reasons for believing this to be a complex process, open to influence by many factors. Shishehbor *et al.* (1998) discuss the long-established finding that ingestion of a meal, or even sham-feeding, can liberate into the circulation fat presumed to be previously stored in the small-intestinal wall or the lymphatics (Mendeloff, 1954; Peel *et al.* 1993; Fielding *et al.* 1996). The rate of delivery of dietary fat into the circulation must be a function of many processes including intestinal digestion and absorption, the synthesis of chylomicron particles in the enterocyte and their export into the lacteals, and the rate of lymphatic flow. As nutritionists we tend to be ignorant of many of these factors.

The findings of Shishehbor *et al.* (1998) therefore emphasize once again the complexity of events in the postprandial period. Those who undertake further work to elucidate the complex, co-ordinated physiological and metabolic events underlying postprandial lipid metabolism will now have to bear in mind the amount of dietary carbohydrate as a further important determinant of the nature, although not necessarily the magnitude, of postprandial lipaemia.

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References

Abraha A, Humphreys SM, Clark ML, Matthews DR & Frayn KN (1998) Acute effect of fructose on postprandial lipaemia in diabetic and non-diabetic subjects. *British Journal of Nutrition* **80**, 169–175.

Akanji AO, Nzegwu AA & Agbedana EO (1992) Some determinants of postprandial lipaemia in Nigerian diabetic and non-diabetic subjects. *British Journal of Nutrition* **68**, 153–162.

Cohen JC & Schall R (1988) Reassessing the effects of simple carbohydrates on the serum triglyceride responses to fat meals. *American Journal of Clinical Nutrition* **48**, 1031–1034.

Cohn JS, McNamara JR, Cohn SD, Ordovas JM & Schaefer EJ (1988) Postprandial plasma lipoprotein changes in human subjects of different ages. *Journal of Lipid Research* **29**, 469–479.

Cohn JS, McNamara JR, Krasinski SD, Russell RM & Schaefer EJ (1989) Role of triglyceride-rich lipoproteins from the liver and intestine in the etiology of postprandial peaks in plasma triglyceride concentration. *Metabolism* **38**, 484–490.

Dubois C, Beaumier G, Juhel C, Armand M, Portugal H, Pauli A-M, Borel P, Latgé C & Lairon D (1998) Effects of graded amounts (0–50 g) of dietary fat on postprandial lipemia and lipoproteins in normolipidemic adults. *American Journal of Clinical Nutrition* **67**, 31–38.

Fery FD, Attellis NP & Balasse EO (1990) Mechanisms of starvation diabetes: study with double tracer and indirect calorimetry. *American Journal of Physiology* **259**, E770–E777.

Fielding BA, Callow J, Owen RM, Samra JS, Matthews DR & Frayn KN (1996) Postprandial lipemia: the origin of an early peak studied by specific dietary fatty acid intake during sequential meals. *American Journal of Clinical Nutrition* **63**, 36–41.

Frayn KN (1993) Insulin resistance and lipid metabolism. *Current Opinion in Lipidology* **4**, 197–204.

Griffin BA & Zampelas A (1995) Influence of dietary fatty acids on the atherogenic lipoprotein phenotype. *Nutrition Research Reviews* **8**, 1–26.

Groot PHE, van Stiphout WAHJ, Krauss XH, Jansen H, van Tol A, van Ramshorst E, Chin-On S, Hofman A, Cresswell SR & Havekes L (1991) Postprandial lipoprotein metabolism in normolipidemic men with and without coronary artery disease. *Arteriosclerosis and Thrombosis* **11**, 653–662.

Havel RJ (1994) Postprandial hyperlipidemia and remnant lipoproteins. *Current Opinion in Lipidology* **5**, 102–109.

Jeppesen J, Chen Y-DI, Zhou M-Y, Wang T & Reaven GM (1995*a*) Effect of variations in oral fat and carbohydrate load on postprandial lipemia. *American Journal of Clinical Nutrition* **62**, 1201–1205.

Jeppesen J, Hollenbeck CB, Zhou M-Y, Coulston AM, Jones C, Chen Y-DI & Reaven GM (1995*b*) Relation between insulin resistance, hyperinsulinemia, postheparin plasma lipoprotein lipase activity, and postprandial lipemia. *Arteriosclerosis, Thrombosis, and Vascular Biology* **15**, 320–324.

Karpe F, Steiner G, Uffelman K, Olivecrona T & Hamsten A (1994) Postprandial lipoproteins and progression of coronary atherosclerosis. *Atherosclerosis* **106**, 83–97.

Lamarche B, Després J-P, Moorjani S, Cantin B, Dagenais GR & Lupien P-J (1995) Prevalence of dyslipidemic phenotypes in ischemic heart disease (Prospective results from the Québec Cardiovascular Study). *American Journal of Cardiology* **75**, 1189–1195.

Lewis GFO, Meara NM, Soltys PA, Blackman JD, Iverius PH, Druetzler AF, Getz GS & Polonsky KS (1990) Postprandial lipoprotein metabolism in normal and obese subjects: comparison after the vitamin A fat-loading test. *Journal of Clinical Endocrinology and Metabolism* **71**, 1041–1050.

Mendeloff AI (1954) The effects of eating and of sham feeding upon the absorption of vitamin A palmitate in man. *Journal of Clinical Investigation* **33**, 1015–1021.

Patsch JR, Miesenböck G, Hopferwieser T, Mühlberger V, Knapp E, Dunn JK, Gotto AMJ & Patsch W (1992) Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state. *Arteriosclerosis and Thrombosis* **12**, 1336–1345.

Peel AS, Zampelas A, Williams CM & Gould BJ (1993) A novel antiserum specific to apolipoprotein B-48: application in the investigation of postprandial lipidaemia in humans. *Clinical Science* **85**, 521–524.

Potts JL, Humphreys SM, Coppack SW, Fisher RM, Gibbons GF & Frayn KN (1994) Fasting plasma triacylglycerol concentrations predict adverse changes in lipoprotein metabolism after a normal meal. *British Journal of Nutrition* **72**, 101–109.

Shishehbor F, Roche HM & Gibney MJ (1998) The effect of acute carbohydrate load on the monophasic or biphasic nature of the postprandial lipaemic response to acute fat ingestion in human subjects. *British Journal of Nutrition* **80**, 411–418.

Weintraub MS, Grosskopf I, Rassin T, Miller H, Charach G, Rotmensch HH, Liron M, Rubinstein A & Iaina A (1996) Clearance of chylomicron remnants in normolipidaemic patients with coronary artery disease: case control study over three years. *British Medical Journal* **312**, 935–939.