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Milk-fed calves digest as monogastric animals since the abomasum is the only functional gastric pouch. Replacement proteins are less digestible than milk protein and their effects on pancreatic secretion are not elucidated. In this study, the kinetics of pancreatic secretion were compared in calves fed with diets based either on milk or soyabean protein.

Five 60–120-day-old Holstein calves were fitted with two catheters, one into the pancreatic duct and another in the duodenum. The pancreatic juice was continuously collected and simultaneously reintroduced into the intestine after removing samples at 15 min intervals, from 1 h before to 6 h after the morning meal. Calves were fed at 830 and 1630 hours with a milk substitute diet, the protein of which was provided either by a skim milk powder (milk diet) or by an alcohol-extracted soyabean concentrate (soyabean diet) after a 4 day adaptation period.

With the milk diet, the pancreatic outflow decreased by 80% between the first 15 and 30 min postfeeding, remained low for 2 h and increased thereafter up to the prefeeding level which was reached about 4 h after the meal. Protein and trypsin outflows showed similar patterns except that the low postfeeding level lasted only 1 h. With the soyabean diet, compared to the milk diet, prefeeding juice flow was 32% lower; 45 min after the meal, the volume was decreased by 59% but remained low for 4 h. It increased thereafter up to the level obtained with the milk diet, 6 h after the meal. In opposition to the milk diet, protein and trypsin outflows increased, especially during the first hour postfeeding. Globally during the 6 postfeeding hours, the pancreatic juice secretion was 26% lower and protein and trypsin flows were increased, respectively, by 50 and 140% as compared to the milk diet.

These changes with diet could be partially controled by gut regulatory peptides. The replacement of milk by soyabean protein has been previously shown to induce a decrease of prefeeding plasma secretin and an increase of postfeeding plasma cholecystokinin. The lower prefeeding level of pancreatic juice flow and the higher postfeeding level of protein output are consistent with these variations. Therefore, digestive processes appear to adapt to the faster gastric emptying of the soyabean diet. Nevertheless, this increase in enzyme secretion is not the only nutritional adaptation implied since the soyabean protein remained less efficiently digested than milk protein.

Effects of different amounts of dietary triglycerides (0–50 g) on postprandial lipemia in healthy human subjects.

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Postprandial events can play key roles in the development of coronary heart disease. Generally, the amount of fat present in the test meals (70–140 g) greatly exceeded that usually ingested during a meal (usually called 'oral-fat load'). In line with our recent studies [Dubois et al (1994) *Am J Clin Nutr* 60, 374-382; Dubois et al (1994) *J Lipid Res* 35, 1993-2007], the present study was aimed at comparing for the first time the postprandial responses to five different amounts of dietary triglycerides in the range of usual fat intake (0–50 g/meal) in healthy humans.

Eight healthy males ingested on separate days in a random order five mixed meals (640–1 090 kcal) containing either 0, 15, 30, 40 or 50 g emulsified triglycerides as sunflower oil. Vitamin A was added to

the meals (50 000 UI/meal). Blood samples were obtained in the fasting condition and every hour for 7 h after the meal intake. Serum and lipoproteins (chylomicrons + large remnants, very low density lipoproteins [VLDL] + small remnants, low density lipoproteins [LDL] and high density lipoproteins [HDL]) were isolated by ultracentrifugation. Triglycerides, free and esterified cholesterol, phospholipids, glucose, insulin, apo A1 and B were assayed.

The chylomicron and serum triglyceride responses were proportional to the amounts of fat ingested and peaked after 2–3 h. The peak values and 0–7 h AUCs were significantly higher than those of the no-fat meal for the 30, 40 and 50 g-fat meals only. The chylomicron cholesterol did not exhibit marked changes after the different test meals; thus, marked changes in the chylomicron lipid composition were only observed after the 30, 40, 50 g-fat meals. Chylomicron retinyl-palmitate was influenced by the amount of ingested triglycerides. The 15 g-fat meal did not significantly change postprandial serum phospholipid, free and esterified cholesterol concentrations over fasting baseline or postprandial Og-fat meal values. On the contrary, significant increases in plasma free cholesterol and phospholipids and decreases in esterified cholesterol were observed after the 30, 40, 50 g-fat meals. At the same time, different reponses were observed after the meals for LDL or HDL free and esterified cholesterol.

In conclusion, the present data show that i) changes in triglyceride intake (15–50 g) markedly affect chylomicron secretion, postprandial lipemia and lipoprotein responses; ii) 15 g triglycerides per meal seems to be a threshold level avoiding postprandial triglyceridemia and lipoprotein changes; and iii) postprandial lipid data may provide useful information for setting dietary guidelines.

Involvement of neurotensin in the control of the postprandial motor response of the colon to food intake in rats. S Pellissier [1], O Eribon [1], J Chabert [1], D Gully [2], M Roche [1] *([1] University of Savoie, Laboratory of Physiology, Le Bourget du Lac; [2] Sanofi Recherche, Toulouse, France)*

Neurotensin is a neurohormone which has been detected in the digestive tract of various species including rats. It is released in response to food intake by the endocrine cells of the terminal intestine. The objective of the present study was to investigate the involvement of neurotensin in the control of the postprandial motor response of the colon in awake rats. The experiment used 14 male Wistar rats equipped with insulated wire electrodes inserted in the wall of the proximal and the distal colon. During the interdigestive state, the myoelectrical activity of the colon was characterized by long spike bursts (LSB). They appeared with a higher frequency on the proximal colon (1.4 ± 0.4/min) than on the distal colon (0.5 ± 0.3/min). In contrast, their duration was longer on the distal colon (30 ± 5 s) than on the proximal colon (14 ± 2 s). Intravenous administration of neurotensin (5 µg/kg) in the fasted rat induced an increase of spike burst frequency similar to that induced by food intake. In contrast, SR48692, a specific neurotensin receptor antagonist, had no significant effect. A 30 min daily meal (4 ± 0.5 g) induced a biphasic colonic motor response. Phase I was characterized by an immediate increase of LSB frequency (+100%) and duration (+25%) on the whole colon. This corresponded to the 'gastrocolonic reflex'. It lasted 30 min. During phase II only the LSB frequency remained elevated for 3 h. The specific blockade of the neurotensin receptor by SR48692 (200 and 800 µg/kg iv) 30 min before feeding induced a complete inhibition of phase I on the distal colon and a partial inhibition of phase II on the proximal colon. It was concluded that neurotensin modulated the early postprandial motor response of the distal colon and the late response of the proximal colon.