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.