test meal corresponded to a meal in accordance with dietary guidelines (30–35% total energy provided by fat), and the 40 g test meal was closer to the western diet.

Eight normolipemic males ingested on separate days and in a random order a full meal containing 4 slices French bread, 60 g wheat semolina (cooked and hydrated with 120 ml water), 2 cooked egg whites, 1 fat-free yogurt, a cup of coffee and the tested fat emulsion. The 30 g fat test meal provided 3 240 kJ (775 kcal with 13.3% proteins, 50.7% carbohydrates and 35.9% fat), and the 40 g fat test meal provided 3 649 kJ (873 kcal with 11.8% proteins, 44.8% carbohydrates and 43.4% fat). The amount of phospholipids was less than 0.2 g per meal and no cholesterol was present in the test-meals.

Blood samples were obtained in the fasting condition and after the meal intake *via* an iv catheter every hour for 7 h. Lipoproteins (chylomicrons, VLDL + chylomicrons remnants, LDL and HDL) were isolated by ultracentrifugation. Lipid parameters (triglycerides, free and esterified cholesterol, phospholipids) were determined on serum and on each class of lipoproteins using enzymatic procedures. Insulin was determined by an immuno-enzymatic method and apoproteins (A1, B) were determined by laser-nephelometry on serum.

The serum and chylomicron triglyceride response was strictly proportional to the amount of fat ingested and peaked after 2–3 h. After the 30 g fat test-meal, there were no significant changes over baseline in phospholipid, free and esterified cholesterol concentrations in serum for 7 h. Inversely, after the 40 g fat test-meal, serum phospholipids and free cholesterol significantly increased and esterified cholesterol decreased postprandially. At the same time, significantly different responses were observed after both meals for LDL-free cholesterol, VLDL and LDL esterified cholesterol and HDL phospholipids: the variation of the range was higher with the 40 g fat meal as compared to the 30 g fat meal. Insulin, ApoA1 and ApoB responses were comparable with the 2 test-meals.

The present data show that limited changes in fat intake (30 instead of 40 g fat) markedly affects postprandial lipemia and lipoprotein responses in normolipidic human subject. This might be taken into account for planning future postprandial studies, and suggests that postprandial lipid data may be useful tools for setting dietary guidelines.

Selective storage and mobilization of individual fish oil n–3 polyunsaturated fatty acids in adipose tissue. T Raclot, E Mioskowski, R Groscolas (*Centre d'Écologie et Physiologie Énergétiques, CNRS, 23, rue Becquerel, 67087 Strasbourg, France)*

The post-intake bioavailability, and thus the biological effects, of dietary n–3 polyunsaturated fatty acids (n–3 PUFAs) may depend on their storage in and mobilization from adipose tissue triglycerides (TG). Both aspects of the metabolism of the 4 major n–3 PUFAs (20:5n–3, 22:6n–3, 22:5n–3 and 18:4n–3) were studied in rats fed a high-fat (20%) fish oil (40% n–3 PUFAs) diet or a control diet (4% fat; 2% n–3 PUFAs in fat) for 4 weeks. According to the n–3 PUFA, 13–32% of its ingested mass was stored in fat reserves at the end of fish oil feeding. However, the storage was selective. The *in vivo* relative incorporation (% in TG / % in diet) increased significantly according to: 20:5n–3 (0.25) < 18:4n–3 (0.37) < 22:6n–3 (0.49) < 22:5n–3 (0.78). Thus, 20:5n–3 was 3-fold less incorporated than 22:5n–3. The *in vitro* relative mobilization of n–3 PUFAs (% in released FFA / % in TG) was determined by incubating pieces of adipose tissue under conditions of stimulated lipolysis (norepinephrine 10^{-6} M; bovine albumin 4% by weight in the medium). Relative mobilization was also selective, decreasing significantly according to 20:5n–3 (2.88) > 18:4n–3 (1.51) > 22:6n–3 (1.08) > 22:5n–3 (0.91). Thus, 20:5n–3 was almost 3 fold more mobilized than total fatty acids, including 22:5n–3 and 22:6n–3. This confirms previous results obtained by incubating isolated fat cells [Raclot and Groscolas (1993) *J Lipid Res* 34, 1515-1526]. The *in vivo* relative incorporation into adipose tissue was inversely and significantly related to the *in vitro* relative mobilization. The same results were obtained from rats fed the control diet, and from retroperitoneal and subcutaneous adipose tissues.

In conclusion, n–3 PUFAs are efficiently but selectively stored in adipose tissues of growing rats, whatever their dietary intake. The selectivity of the mobilization of individual n–3 PUFAs could contribute to their differential storage. The most preferential mobilization of 20:5n–3 could contribute to its maintenance in the circulation during or shortly after its supplementation, which could substain its various biological effects. On the other hand, the long-term storage of this fatty acid in fat stores is probably limited.