

each. Diet 1 was 30 % fat (8 % SFA, 12 % oleate, 6 % linoleate and 1 % linolenate), 55 % carbohydrate, 200 mg cholesterol. Diet 2 was 34 % fat (11 % SFA), 51 % carbohydrate and no change in oleate, linoleate, linolenate and cholesterol. Baseline diet (2 400 kcal, 35 % fat, 13 % SFA, 52 % carbohydrate) was provided before each diet for 4 weeks. Samples obtained at the end of each period were assessed for plasma lipids and fatty acids of the phospholipids and cholesteryl esters. In comparison with baseline diet, diets 1 and 2 caused a decrease in total C, LDL-C and triglycerides (TG) ( $P < 0.001$ ); HDL-C was not modified, apoA-I/apoB ratio was increased ( $P < 0.001$ ). Plasma TG was lower after diet 2 than after diet 1, whereas HDL-C was higher ( $P < 0.05$ ). In phospholipids, myristate, oleate, linoleate, EPA and DHA were increased in diet 2 versus baseline ( $P < 0.01$ ) and diet 1 ( $P < 0.05$ ); in cholesteryl esters, linolenate was increased with diets 1 and 2 ( $P < 0.05$ ).

These data suggest that a diet 34 % fat with 11 % SFA and 51 % carbohydrate has beneficial effects on plasma lipid and fatty acid profiles. This diet seems to be more effective than a diet 30 % fat with 8 % SFA and 55 % carbohydrate.

**Altered apolipoprotein AI metabolism in subjects with type IIA heterozygous familial hypercholesterolemia: a kinetic study.** R. Frénais, C. Maugeais, K. Ouguerram, V. De Mallmann, T. Magot, J.M. Bard, M. Krempf (CRNH and Clinique d'endocrinologie et maladies métaboliques, Hôtel Dieu, 44093 Nantes, cedex 01, France).

Patients with type IIA heterozygous familial hypercholesterolemia (FH) demonstrate an increase in LDL-cholesterol, associated with an elevated cardiovascular risk. Whereas alterations in apolipoprotein (apo) B100 metabolism are well established, potential effects of FH

on apo AI metabolism remain to be elucidated. Six healthy subjects and six FH patients (plasma cholesterol  $167 \pm 24$  and  $425 \pm 29$  mg/dL, respectively,  $P < 0.001$ ) received a priming dose ( $10 \cdot 10^{-6}$  mol/kg) followed by a continuous 14-h [ $^2\text{H}_3$ ]-leucine infusion ( $10 \cdot 10^{-6}$  mol/kg/h). Apo AI concentration was similar in FH patients compared to controls ( $113 \pm 18$  versus  $123 \pm 18$  mg/dL, respectively, NS). Data were analysed using a mono compartmental model (SAAM II modelling software). The HDL-apo AI fractional catabolic rate (FCR) and absolute production rate (APR) were increased in FH subjects ( $0.36 \pm 0.16$  versus  $0.18 \pm 0.04$  pool/day,  $P < 0.05$ , and  $17.7 \pm 7.7$  versus  $10.1 \pm 2.0$  mg/kg/day,  $P < 0.05$ ), these parameters being correlated ( $r^2 = 0.955$ ,  $P < 0.001$ ). HDL-triglyceride concentration was higher ( $20 \pm 8$  versus  $6 \pm 2$  mg/dL,  $P < 0.01$ ), whereas HDL-cholesterol concentration was decreased ( $37 \pm 7$  versus  $56 \pm 16$  mg/dL,  $P < 0.05$ ) in FH patients. Both FCR and APR of HDL-apo AI were negatively correlated with plasma cholesterol concentration ( $r = 0.351$ ,  $P < 0.05$  and  $r = 0.327$ ,  $P < 0.05$ , respectively). Plasma and HDL-triglyceride concentrations were correlated with the FCR and the APR of HDL-apo AI ( $P < 0.05$ ). Thus, our results suggest that patients with heterozygous FH up-regulate apo AI production in response to an hypercatabolism, which may itself be related to changes in HDL composition.

**The hypocholesterolemic effect of soybean is modified by dietary iron content in the rainbow trout.** S.J. Kaushik, G. Corraze, A. Mitrenko (Laboratoire de Nutrition des Poissons, Inra, 64310 St-Pée-sur-Nivelle, France).

Teleost fish are generally known to have high plasma cholesterol levels. Recent studies have confirmed the hypocholesterolemic effect of dietary soybean in

Type of SPC	High iron				Low iron		
	0	75	95	100	75	95	100
Fishmeal replacement level (%)							
Triglycerides (g/L)	2.60 <sup>bc</sup>	3.65 <sup>a</sup>	2.82 <sup>b</sup>	2.96 <sup>b</sup>	3.88 <sup>a</sup>	3.26 <sup>a</sup>	3.73 <sup>a</sup>
Cholesterol total (g/L)	3.82 <sup>a</sup>	2.83 <sup>b</sup>	2.19 <sup>c</sup>	1.89 <sup>d</sup>	2.62 <sup>b</sup>	2.52 <sup>b</sup>	2.48 <sup>b</sup>

rainbow trout or European seabass, similar to that of terrestrial vertebrates. In mammals, a dietary excess of iron has been shown to increase plasma triglyceride and cholesterol levels. Given the known variability in the iron content of soybean and in the general context of research on the replacement of dietary fishmeal by plant protein sources in fish-feeds, studies were aimed at verifying the possible effects of dietary iron on plasma lipid concentrations in the rainbow trout. Two trials were undertaken with rainbow trout grown in freshwater at 18 °C. In a first study involving the total replacement of fishmeal by soyprotein concentrates (SPC), it was found that the hypocholesterolemic effect of soyprotein was reduced when the iron content of the SPC was high. In the second trial, a definite dose-response of plasma cholesterol levels to dietary SPC levels was found with the high iron SPC, whereas no such dose-response was observed with the low iron SPC. The hypercholesterolemic effect of iron appears to be dose-dependant.

**Low density lipoprotein oxidation in children.** L. Iughetti<sup>a</sup>, C. Volta<sup>a</sup>, E. Maggi<sup>c</sup>, G. Bellomo<sup>c</sup>, S. Bernasconi<sup>b</sup> (<sup>1</sup>Clinica Pediatrica, Università di Parma e <sup>b</sup>Modena, <sup>c</sup>Clinica Medica I, Università di Pavia, Italy).

Several studies have shown that LDL oxidation plays an important role in the pathogenesis of atherosclerosis. Oxidized LDL (oxLDL) are more atherogenic than their native forms because oxidative modifications generate molecular epitopes that provide chemotactic stimuli for monocyte recruitment. They are more avidly taken up by macrophages, thus forming foam cells, which are cytotoxic for endothelial cells. Moreover it has been demonstrated that an in vivo modified LDL could induce an auto-antibody response. Both an enhanced LDL oxidation and high autoantibody anti-oxLDL levels have been shown in several patients characterized by accelerated atherosclerosis as subjects affected by diabetes, essential hypertension, severe carotid atherosclerosis and uremia.

As data exist for children, we studied the oxidation susceptibility of LDL in vitro and the existence of anti-oxLDL autoantibodies in vivo in 18 children and adolescents (9 males, 9 females; age 11.02 ± 4.6 years) and in 27 normal adult subjects (13 males, 14 females, age 39.0 ± 11.1 years). In all subjects we evaluated:

1) LDL oxidation, triggered by the addition of CuSO<sub>4</sub> and continuously monitored spectrophotometrically at 234 nm to follow the formation of conjugated dienes. The lag-phase (the most important parameter of oxidability) was significantly more accelerated in children than in