LDL to determine the relative quantity of each fraction.

Triglyceride concentrations were significantly higher in the NIDDM group. Total cholesterol and LDL apoB were not statistically different between the two groups. Fractions 2 and 3 were significantly decreased and fraction 4 significantly increased in the NIDDM group compared to controls (fig 1).

LDL repartition showed an atherogenic profile in the NIDDM hypertriglyceridemic group. Total cholesterol and LDL apoB were not different but dense LDL were predominant in the NIDDM group.

Endogenous labelling of cholesterol using ^{13}C-acetate perfusion: development of an in vivo study protocol for cholesterol reverse transport in humans.

K Ouguerram, P Maugeais, C Maugeais, M Krempf, T Magot *(Laboratoire de nutrition humaine, hôpital Laënnec, 44035 Nantes cedex 01, France)*

Three normocholesterolemic subjects were submitted to ^{13}C-acetate continuous administration (1.5 µmol/kg/min) for 7 h after one bolus of 90 µmol/kg. During the study, blood samples were drawn at 30 min, 1 h, 1 h 30, 2 h and then hourly for 7 h. Samples were used for separation of very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL) by ultracentrifugation. Lipids were extracted by the Folch method. They were subjected to chromatography on a silicic acid microcolumn to separate the cholesteryl esters from unesterified cholesterol. Samples were derivatizated with acetic anhydride-pyridine and enrichments were measured by gas-liquid chromatography coupled to spectrometry (GC-C-IRMS). The enrichment curves of lipoprotein unesterified cholesterol were similar, attesting to rapid exchanges of unesterified cholesterol between lipoproteins. At the end of the experiment, the isotopic enrichment of free cholesterol was 0.07%, that of esterified cholesterol was 0.016, 0.007, 0.003%, respectively, for HDL, VLDL and LDL.

The order of labelling apparition in lipoprotein esterified cholesterol (HDL, VLDL and LDL) was in agreement with previous data: esterification of cholesterol in HDL and its delivery to apolipoprotein B-containing lipoproteins. A multicompartmental model of cholesteryl ester metabolism was previously developed in normocholesterolemic subjects using radioisotopes. This model integrated the essential role of cholesteryl ester movement from HDL to apoB-containing lipoproteins (about 50% of HDL cholesteryl ester/h) in reverse cholesterol transport. In this model cholesteryl ester appeared essentially by esterification (6%/h of unesterified cholesterol) and disappeared exclusively by LDL uptake (about 3%/h). The simulation of this model under these new labelling conditions led to curves very similar to our experimental data.

This protocol, using stable isotopes permitted us to quantify cholesterol movement involved into reverse cholesterol transport.

Kinetic study of HDL2 and HDL3 metabolism using endogenous labelling of apolipoprotein AI by stable isotopes in normolipidemic subjects.

K Ouguerram, C Collober, C Maugeais, M Krempf, T Magot *(Laboratoire de nutrition humaine, hôpital Laënnec, 44035 Nantes cedex 01, France)*

Apolipoprotein AI plays a major role in cholesterol efflux from extrahepatic tissues to the liver. It was therefore considered interesting to measure the characteristics of the high density lipoprotein (HDL) apolipoprotein AI turnover.

The endogenous labelling of apolipoprotein AI was carried out in six normolipidemic subjects through the administration of D3 leucine. Four volunteers were perfused for

14 h (10 µmol/kg/h) after one bolus of 10 µmol/kg and two were submitted to a bolus (53 µmol/kg). Blood samples were drawn during the study. HDL2 (1.063 < d < 1.125) and HDL3 (1.125 < d < 1.21) were isolated by ultracentrifugation and apolipoprotein AI by electrophoresis. The isotopic enrichment of apolipoprotein AI was measured by mass spectrometry. The plateau of very low density lipoprotein (VLDL) apolipoprotein B-100 was used as a precursor pool estimation for apolipoprotein AI and the data were analyzed by monoexponential regression. For the bolus study, the precursor pool was plasma leucine which was used in the model as a forcing function. The production rate was calculated as the product of FCR and apolipoprotein AI mass.

The apolipoprotein AI kinetic curves were similar in HDL2 and HDL3 in the six volunteers for the two methods of administration. For our analysis, no kinetic heterogeneity was required in HDL.

The FCR of apolipoprotein AI was 20 ± 5%/day and the production rate was 10.2 ± 2.6 mg/kg/day. These results were in agreement with those previously obtained with radioisotopes.

LDL-apheresis: a comparison between double-membrane filtration (DF) and dextran sulfate-cellulose adsorption (DSC).

JD Lalau, P Morinière (CHU, 80054 Amiens, France)

The two most widely available techniques of LDL-apheresis – DF and DSC – have been comparatively tested in terms of the decrease after apheresis in plasma lipoprotein values but not in terms of mass transfer. This latter would provide more accurate information on the efficacy and selectivity of removal. For this purpose, we studied long-term apheresis treatment of seven patients with severe familial hypercholesterolemia, of whom three had, in addition, elevated plasma lp(a) levels. Ninety exchanges were performed at biweekly intervals using DF (Evaflux, Kuraray) or DSC (Kaneka columns). We calculated the removal coefficient (Cpost-Cpre/Cpre, where Cpost and Cpre are the post- and the pre-apheresis plasma concentrations) and the mass transfer (C x ΣV, where C is the plasma concentration and ΣV the sum of the volumes of rejected plasma and of rinsing fluids for DF and the volume of rinsing and regenerating fluids for DSC). The results are shown in the table.

In conclusion, calculating mass transfer revealed information that could not be appreciated using the classical determination of removal coefficient: DF removed LDL-CT more efficiently than DSC while DSC seemed to be more efficient for lp(a) removal and was more selective than DF, removing less HDL-CT.

Parameters	Removal coefficient %		Mass transfer mmol, g for lp(a)	
	DF	DSC	DF	DSC
LDL-CT	55.1 ± 2.5	54.7 ± 2.5	8.1 ± 0.9	5.8 ± 0.5**
HDL-CT	45.7 ± 3.3	11.2 ± 2.7*	1.3 ± 0.1	0.16 ± 0.04
Lp(a)(n = 14)	70 ± < 1	70 ± < 1	1.75 ± 0.24	2.08 ± 0.36

* $P < 0.02$; ** $P < 0.001$.