**Table I.** Data for 22 boys (9 controls; 13 DMD patients) (Gottrand *et al*).

| *Mean ± SD* | *Controls* | *DMD* | *p* |
|---|---|---|---|
| REE (kcal/h) | 54.7 (3.2) | 49 (6.2) | 0.02 |
| REE/FFM (kcal/kg/h) | 2.1 (0.2) | 2.3 (0.3) | NS |
| RQ | 0.83 (0.04) | 0.88 (0.03) | 0.004 |
| FFM (kg) | 26.1 (4.2) | 22 (3.8) | 0.04 |
| MM (kg) | 12 (4) | 3 (1) | 0.0001 |
| 3-MH (μmol/g) | 220 (41) | 583 (169) | 0.001 |
| DEI (kcal/d) | 2 038 (374) | 1 624 (401) | 0.02 |
| Carbohydrates (g/d) | 250 (51) | 195 (47) | 0.04 |
| Proteins (g/d) | 67 (16) | 56 (16) | NS |
| Lipids (g/d) | 85 (18) | 69 (20) | NS |

from bioelectrical impedance analysis (BIA 101, RJL systems, Detroit, MI) using Schaeffer's formula (*Pediatr Res* (1994) 35, 617-24). Their muscle mass (MM) was estimated from 3 d creatinin urea. The 3-methyl histidine (3-MH) level was measured to estimate muscle breakdown. Diet records for one week were analysed for daily energy intake (DEI) and diet composition (table I).

Obesity in DMD is associated neither with high caloric intake nor with a different diet composition when compared with controls. The low REE (probably explained by the decrease of FFM) and the lack of lipid oxidation (suggested by the elevated RQ observed in patients with DMD) could be risk factors leading to the development of obesity in DMD patients.

**Lecithin: cholesterol acyltransferase activity and chemical composition of $HDL_2$ and $HDL_3$ in patients with chronic renal failure treated by haemodialysis.**
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The aim of the present study was to correlate lipid and apo A-I amounts in $HDL_2$ and $HDL_3$ with plasma lecithin/cholesterol acyltransferase (LCAT, EC 2.3.1.43) activity in CRF patients treated by haemodialysis.

Fifty-eight patients (34 women, 24 men) with a mean age of 42 ± 13 years were investigated. This population was divided into 3 groups according to the duration of their haemodialysis: G1 < 1 year; G2 1–5 years; G3 5–13 years. These patients presented hypertriglyceridemia (1.45 ± 0.68 g/l) but had a normal plasma total cholesterol level (1.45 ± 0.55 g/l). A control group was composed of 22 adults (13 men, 9 women; mean age 40 ± 7 years). The total cholesterol (TC) and unesterified cholesterol (UC) of $HDL_2$ and $HDL_3$ were evaluated by gas-liquid chromatography (Gambert *et al* (1979) *J Chromatogr* 162, 1-6; (1982) *Biochim Biophys Acta* 713, 1-9), apo A-1 was evaluated by electroimmuno-diffusion on agarose gel and the plasma LCAT activity was assayed (Glomset *et al* (1964) *Biochim Biophys Acta* 89, 266-271).

In $HDL_3$, the UC and triacylglycerol (TG) proportions were higher, whereas the proportions of phospholipids (PL) were lower. Those of the cholesteryl esters (CE) were similar in the 3 groups compared with the

**Table I.** Data correlating apo A-I amounts in $HDL_2$ and $HDL_3$ with plasma LCAT activity (Mekki *et al*).

| | *$HDL_3$-apo A-I (arbitrary units)* | *$HDL_2$-apo A-I (arbitrary units)* | *Plasma LCAT activity (nM cholesteryl esters released/ml/h)* |
|---|---|---|---|
| Control group | 72.4 ± 23.0 | 71.0 ± 8.3 | 60.0 ± 10.1 |
| G1 | 34.0 ± 23.0* | 32.6 ± 25.4* | 19.6 ± 8.5* |
| G2 | 31.7 ± 19.0* | 30.5 ± 22.2* | 15.0 ± 7.2* |
| G3 | 20.9 ± 8.4* | 25.6 ± 24.8* | 10.7 ± 9.9** |

Values are means ± SD. Means are pair-compared using Student's *t* test. * (G1, G2, G3 *vs* control; $p < 0.001$); ** (G3 *vs* G1; $p < 0.01$).

control group. In $HDL_2$, a low CE level and a high amount of UC, TG and PL were observed in the 3 groups compared with the control group.

The decrease in plasma LCAT activity contributed to the alteration of the $HDL_2$ fraction and might participate in plasma hypertriglyceridemia in CRF (table I). An inverse correlation between the $HDL_2$-cholesterol and LCAT activity was noted in G1 ($r = 0.68$, $p < 0.05$) and G3 ($r = 0.78$, $p < 0.001$) groups. In contrast, inverse relationships were observed, only in G3, between the LCAT activity and TG-$HDL_3$ ($r = -0.87$, $p < 0.01$), PL-$HDL_3$ ($r = -0.65$, $p < 0.05$) and UC-$HDL_3$ ($r = -0.97$, $p < 0.001$).

**LPL gene polymorphism influences the serum triglyceride response to dietary intervention in obese people.** R Jemaa, S Tuzet, F Fumeron, D Betoulle, M Apfelbaum *(INSERM U 286, faculté X-Bichat, BP 416, 75870 Paris cedex 18, France)*

Obesity is one of the most prevalent metabolic abnormalities in Western society, and is commonly associated with hypertriglyceridemia. A hypocaloric diet is accompanied by a decrease in plasma lipid levels. The individual variations in response to diet modifications are likely attributable to both environmental and genetic factors.

Lipoprotein lipase (LPL) plays a key role in the catabolism of triglyceride-rich lipoproteins. Several studies have reported associations between serum lipid levels (triglycerides, HDL-cholesterol) and restriction fragment length polymorphisms (RFLPs) of the LPL gene (using Hind III and Pvu II enzymes) (Heinzmann *et al* (1991) *Hum Genet* 86, 578; Chamberlain *et al* (1989) *Atherosclerosis* 79, 85).

The aim of the present study was to assess the risk of hypertriglyceridemia associated with the *H2* allele in obese people and to compare the lipid, lipoprotein and apolipoprotein concentrations according to Hind III and Pvu II polymorphisms before and after a hypocaloric diet.

Unrelated outpatients ($n = 120$) were selected on the basis of 120% ideal body weight according to tables of the Metropolitan Life Insurance Co (1959), *ie* a body mass index (BMI) ≥ 25.8 kg/m$^2$ for women and ≥ 26.4 kg/m$^2$ for men. Diabetic patients or patients using drugs known to modify serum lipid levels were excluded. The obese patients ate a hypocaloric diet (1 824 ± 311