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

kcal/d) containing 42% of energy as fat, 19.4% as protein and 37% as carbohydrates for 3 months.

Two restriction length polymorphisms (RFLPs) (Pvu II and Hind III) were determined by enzymatic digestion of DNA from leukocytes after PCR amplification. Digestion with Hind III generated 2 alleles, *H1* and *H2* with frequencies of 0.28 and 0.72, respectively. Digestion with Pvu II generated 2 alleles, P1 and P2, with frequencies of 0.49 and 0.51, respectively.

Subjects with *H2H2* genotypes fed a spontaneous diet had significantly higher serum TG (1.21 ± 0.73 g/l) and VLDL (0.76 ± 0.61 g/l) than *H1H1* or *H1H2* subjects (0.93 ± 0.47 and 0.53 ± 0.39 g/l). Following the hypocaloric diet, subjects with *H2H2* genotypes reduced their total (18%) and VLDL-TG (28%) more than subjects with *H1H1* or *H1H2* genotypes (4% and 9%).

In Pvu II genotypes, no differences between lipid related variables were observed in obese subjects irrespective of the diet (spontaneous diet or hypocaloric diet).

In conclusion, the serum TG and VLDL levels differed in obese people and depend, at least in part, on genetic factors. The response of circulating lipids to the hypocaloric diet also depends on genetic factors. This signifies that the benefits from such diets are not identical for all obese people.

Resting metabolic rate, diet-induced thermogenesis and body composition in lean and obese men. M Dabbech, A Boulier, M Apfelbaum, R Aubert *(INSERM U 286, faculté X-Bichat, BP 416, 75870 Paris cedex 18, France)*

The existence and significance of a defect in postprandial thermogenesis in obesity is a matter of considerable controversy (Shetty *et al* (1981) *Clin Sci* 60, 519-25; Nair *et al* (1983) *Clin Sci* 65, 307-12; Swaminathan *et al* (1985) *Am J Clin Nutr* 42, 177-81). Conflicting results have been reported for almost every factor affecting diet-induced thermogenesis (DIT) that has been investigated, but few studies specify the relationship between postprandial thermogenesis and body composition (Segal *et al* (1987) *Am J Physiol* 252, E110-E117; Segal *et al* (1989) *Am J Physiol* 256, E573-E579). To further clarify the independent relationship of body composition parameters to energy expenditure (EE), resting metabolic rate (RMR) and DIT were studied in 8 lean (body mass index (BMI) = 21.7, age = 22.5 years) and 10 obese (BMI = 29.6, age = 27 years) men. The groups were matched for fat-free mass (FFM) in order to study the relationship between thermogenesis and body fat independent of FFM. Body composition was assessed by bioelectrical impedance analysis. Metabolic rates were measured by indirect calorimetry. The baseline RMR was measured for 20 min. The DIT was assessed for 345 min, following a 4 055 kJ mixed meal.

The FFM was not significantly different for the 2 groups (obese: 64.11 kg, non-obese: 63.84 kg). The FM was significantly greater in obese than in lean men (23.94 *vs* 8.3 kg respectively, $p < 0.001$).

The RMR was significantly higher in obese than in lean subjects. When adjusted for the differences in FFM, the RMR values were not significantly different for the 2 groups. There was a significant correlation between RMR and FFM ($R = 0.53$, $p < 0.05$). The RMR was significantly correlated with the FFM and FM combined ($r = 0.74$, $p < 0.01$, DER (kcal/24 h) = 13.5 FFM + 12.8 FM + 942).

After ingestion of the meal, the energy expenditure rates showed a significant increase ($p < 0.0001$) in all subjects.

The DIT (the integrated postprandial area above the baseline level) was significantly greater for the lean than the obese men