

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 ( $1.21 \pm 0.73$  g/l) and VLDL ( $0.76 \pm 0.61$  g/l) than *H1H1* or *H1H2* subjects ( $0.93 \pm 0.47$  and  $0.53 \pm 0.39$  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 Boulrier, 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) =  $13.5 \text{ FFM} + 12.8 \text{ FM} + 942$ ).

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

(518 vs 350 kJ respectively,  $p < 0.05$ ). The DIT was significantly and negatively correlated with the percentage body fat ( $r = -0.51$ ,  $p < 0.005$ ). The DIT was highly correlated with the FFM and FM combined ( $r = 0.71$ ,  $p < 0.01$ ,  $\text{DIT (kJ)} = 17.5 \text{ FFM} - 11.3 \text{ FM} - 500.9$ ).

These results support 2 conclusions. First that the DIT was significantly higher for lean men than obese men of similar FFM. Second, that body composition is a significant determinant of the magnitude of the DIT.

**Deformability, membrane lipid composition and cytosolic cation contents of red blood cells in non-insulin dependent diabetic patients.** P Miossec<sup>1</sup>, M Garnier<sup>2</sup>, J Pariès<sup>1</sup>, F Zhiri<sup>2</sup>, M Dufilho<sup>3</sup>, MG Pernollet<sup>3</sup>, JR Attali<sup>1</sup>, P Valensi<sup>1</sup> (<sup>1</sup> *Diabetology-Nutrition*; <sup>2</sup> *Toxicology, Jean-Verdier Hospital, Bondy*; <sup>3</sup> *URA CNRS 1482, Necker Hospital, Paris, France*)

Many studies have clearly demonstrated hemorheological abnormalities in diabetic patients. The erythrocyte rigidity is higher, and is associated with changes in the membrane lipid composition. The aim of this work was to study, simultaneously, the variations in intra-erythrocyte cation concentrations and membrane lipids in 28 poorly controlled ( $\text{HbA}_{1\text{C}} = 8.8 \pm 3.6\%$ ,  $N < 6\%$ ) non-insulin-dependent diabetic patients (NIDDs) (age  $54.9 \pm 6.4$  years,  $\text{BMI } 27.53 \pm 4.2$ ) compared with 26 control subjects (identical age and BMI). The red blood cell rigidity index (RI), measured on a Hanss' hemorheometer, was higher in the NIDDs than in controls ( $11.8 \pm 2$  vs  $10.4 \pm 2.2$ ,  $p < 0.03$ ), the RI correlated with post-prandial glycemia ( $r = 0.51$ ,  $p < 0.02$ ). Compared with the controls, in the NIDDs free cholesterol (FC) was lower ( $-16\%$ ,  $p < 0.01$ ), sphingomyelins (SP) were higher ( $+42\%$ ,  $p < 0.01$ ), free cholesterol/phospholipid ratio (FC/PL) was lower ( $-26\%$ ,  $p < 0.05$ ). In NIDD, intra-erythrocyte

sodium ( $\text{Na}_i$ ) was higher ( $29.4 \pm 10.3$  vs  $18 \pm 10.1$  mEq/l,  $p < 0.001$ ), while cytosolic potassium and magnesium were not significantly different. Intra-erythrocyte ionized calcium ( $\text{Ca}_i$ ) was higher ( $105 \pm 22.2$  vs  $69 \pm 8$  nmol/l,  $p < 0.001$ ) as well as the calcium influx estimated by incorporation of  $^{45}\text{Ca}^{2+}$  ( $V_i$   $^{45}\text{Ca}^{2+}$ ) ( $89 \pm 28.8$  vs  $47 \pm 20$   $\mu\text{mol/l/cell/h}$  ( $p < 0.001$ )). In the NIDDs,  $\text{Na}_i$  correlated with RI ( $r = 0.41$ ;  $p < 0.02$ ) and with fasting glycemia ( $r = 0.45$ ,  $p < 0.05$ );  $\text{Na}_i$  also correlated with membrane lipids: FC ( $r = -0.41$ ,  $p < 0.05$ ); phosphatidylethanolamine (PE) ( $r = -0.43$ ,  $p < 0.05$ ); phosphatidyl choline (PC) ( $r = -0.58$ ,  $p < 0.001$ ); FC/PL ( $r = 0.48$ ,  $p < 0.01$ ).  $V_i$   $^{45}\text{Ca}^{2+}$  correlated with  $\text{HbA}_{1\text{C}}$  ( $r = 0.51$ ,  $p < 0.02$ ), PC ( $r = 0.41$ ,  $p < 0.05$ ) and SP ( $r = 0.42$ ,  $p < 0.05$ ). In poorly controlled NIDDs the increase in  $\text{Na}_i$  and  $\text{Ca}_i$  may be in part responsible for the reduction in red cell deformability and may be the result of modifications in the membrane lipid constitution. The increase in  $\text{Ca}_i$  may result, at least in part, from the augmentation of  $V_i$   $^{45}\text{Ca}^{2+}$ . The changes in cation and lipid constitution of red blood cells correlate with glycemic control.

**Anthropometric characteristics of obese subjects with nocturnal oxygen desaturation.** D Cassuto<sup>1</sup>, G Panotopoulos<sup>1</sup>, E Orvoën-Frija<sup>2</sup>, B Saci<sup>2</sup>, A Basdevant<sup>1</sup>, B Guy-Grand<sup>1</sup> (<sup>1</sup> *Dept of Nutrition*; <sup>2</sup> *Dept of Pneumology, Hôtel-Dieu, 75181 Paris cedex, France*)

Nocturnal oxygen desaturation (NOD) can be observed in obese subjects (OB) with or without Obstructive Sleep Apnea Syndrome (OSAS). The aim of this prospective study was to clarify the anthropometric characteristics of OB with NOD with or without SAS.

Patients hospitalized in the Nutrition Unit ( $n = 66$ ; 45 women, body mass index (BMI)