120  A NOVEL MUTATION IN THE LIPOASE MATURATION FACTOR 1 (LMF-1) GENE RESPONSIVE OF SEVERE HYPERTRIGLYCERIDEMIA R. Spina1, M.L. Argo2, N. Vivona1, M. Ditta3, F. Fayer1, M. Mina4, V. Valentini5, D. Pollacca1, D. Noto6, A.B. Cefai7, A. Nota7, M.R. Averna8, A. Camera8, D. Bini1, L. Magi8

The proband is a 41-year-old Tunisian man who was referred to our attention because of severe hypertriglyceridemia first noted at the age of 32 in the course of the acute episode of pancreatitis. Since then plasma TG levels have been between 7.86 and 22.40 mmol/L and he had been suffering from two more episodes of acute pancreatitis. After the last episode, he was noted to tolerate well the diet recommended to control hypertriglyceridemia. The analysis of the LMF-1 gene shows the presence of a homozgyous G to A substitution in the exon 9 (c.1391G>A), leading to a premature stop codon (W464X) responsible of a truncated LMF-1 protein. In conclusion, LMF-1 should be considered an important candidate gene in those cases of severe hypertriglyceridemia that remain unexplained by mutations in the current set of candidate genes.

121  LIPOA: A POSSIBLE LINK WITH MIGRAINE E. Iacoviello, P. Cappellani, A. Giustini, A. Bellia, M. Villo, A. Pappalardo, G. Vigano

Background: Migraine, a common multifactorial neurovascular disorder, has been suggested to be an independent risk factor for stroke and data from literature evidenced that elevated lipoprotein(a) (Lp(a)) concentrations represent a risk factor for stroke. Aim of the present study was to evaluate the role of Lp(a) in affecting migraine, so possibly contributing to identify a biomarker of predisposition to the disease.

Materials and Methods: Lp(a) levels have been determined in 138 migraine patients (110 females and 28 males), among whom 90 with aura and 28 healthy subjects (87 females and 25 males), comparable for age and gender. Plasma levels of Lp(a) have been determined by an ELISA method.

Results: Median value of Lp(a) was 104 (1-2110) mg/dL in migraine patients and 103 (9-495) mg/dL in the control group (p=0.8). A significant difference among tertiles of Lp(a) concentrations between patients and controls was found (p=0.04). In particular, a significant difference in the high tertile of Lp(a) between patients and controls was observed (p=0.001). Moreover, abnormal Lp(a) levels, defined as a concentration >50th percentile, have been found to significantly predispose to migraine (OR 3.4, 95% CI 1.57-7.55, p=0.002), after adjustment for age, gender and traditional risk factors. No difference in Lp(a) concentrations was observed between patients with aura and without aura, and no relationship was found between abnormal Lp(a) concentrations and headache intensity.

Conclusions: The present study evidences a role for Lp(a) in affecting the risk of migraine, so providing information on a novel possible mechanism involved in the predisposition to the disease.