was to identify sequence variants in the GPIHBP1 gene in a group of patients with SHTG.

Methods: We studied 70 patients with fasting plasma TG > 1000 mg/DL, in at least on one occasion, referred to different Lipid Units in Spain. The average age was 40 years, 80 % were men, 20 % suffered from DM2, 23 % were obese and 39 % had suffered from episodes of acute pancreatitis. The detection of genetic variants was carried out by high resolution melt curve analysis or HRM and its verification by direct sequencing.

Results: In exon 1 we detected the polymorphisms: p.Leu4= (rs61747644) with a frequency of 19.3% and p.Cys14Phe (rs11538389) with a frequency of 9.3%. In exon 2 we identified the p.Val46= (rs11538388) variant with a frequency of 46%. We did not find any variant in exon 3. Finally, only one patient had the p.Gly155= (rs139851434) mutation, in exon 4, in the heterozygous state.

Conclusions: The identified four variants previously described in genomic data bases. Only one of them was a missense polymorphism whereas the two other common variants and the mutation consisted of synonymous changes.

P2.2.185.

POSTPRANDIAL EFFECTS OF DIFFERENT DAIRY PRODUCTS ON BLOOD LIPIDS IN LEAN AND OVERWEIGHT SUBJECTS

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Aim: Postprandial lipemia is a risk factor for cardiovascular disease. Dairy products differ in nutrient content and food matrix, and little is known about how different dairy products affect triglycerides (TG) and other blood lipids postprandially. The aim was to investigate how isocaloric high-fat meals with different dairy products affect postprandial blood lipid levels.

Methods: A randomized controlled cross-over study was conducted with two different groups and four meals. One group consisted of 34 lean subjects (body mass index (BMI) 18.5–25 kg/m² and waist circumference < 80/94 cm for women and men respectively) and the other of 13 overweight and obese subjects (BMI > 25 kg/m² and waist circumference > 80/94 cm). All meals included three slices of white bread with jam and either butter, hard cheese, whipped cream or sour cream, corresponding to 45 grams of fat. Blood samples were taken fasting before ingesting the meals and after 2, 4 and 6 hours. Incremental area under the curve (Iauc) was calculated and data were analyzed using a linear mixed model.

Results: Sour cream induced a significantly greater TG-Iauc compared to all other dairy products within the lean group (vs butter, p=0.001; vs cheese, p<0.05; vs sour cream, p<0.001). Among the overweight subjects, sour cream induced a significantly greater TG-Iauc compared to butter only (p<0.05).

Conclusions: Isocaloric meals with the same amount of fat from different dairy products induce divergent postprandial effects on TG, with sour cream generating the greatest TG-Iauc. Disparate milk fat globule membranes between products may be an explanation.

P2.2.186.

IS ENDOTHELIAL LIPASE A SUPPORTING ACTOR OF LIPOPROTEIN LIPASE IN OBESITY?

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Aim: Lipoprotein lipase (LPL) and endothelial lipase (EL) are involved in lipoproteins metabolism. In insulin-resistant states (IR), with visceral adipose tissue (AT) expansion and cardiac steatosis, these enzymes behavior is altered. Peroxisome Proliferator Activated Receptors (PPARs) could be partly responsible of these alterations. Our aim was to evaluate LPL and EL activity in AT and heart, their association with lipoprotein profile and the role of PPARs expression in an obesity model.

Methods: Male Wistar rats were fed with standard diet (Control) or High Fat Diet (HFD) during 14 weeks. Glucose, lipoprotein profile and insulin resistance markers were measured in serum. Histological studies were performed in heart and epididymal AT. Mrna levels of LPL and EL were assessed by real time PCR. Activities of LPL and EL were assessed by radiometric assays. Cardiac and AT PPARs expression were measured by Western Blot.

Results: EL activity was increased in AT (p=0.002) and heart (p<0.001) from HFD. LPL activity was lower in both tissues from HFD than controls (p=0.002 and p<0.001). An inverse association between both lipases was observed in AT (r=-0.53, p=0.004) and heart (r=-0.64, p<0.001). PPAR γ expression in AT was decreased in HFD, associated with LPL activity (r=-0.505, p=0.014) and EL activity (r=-0.714, p=0.001). No differences were found in cardiac PPAR δ expression between groups.

Conclusions: The increase in EL activity could be an alternative pathway for free fatty acids uptake in tissues with decreased LPL activity. In AT, PPAR γ could be involved in the regulation of the enzymes.

P2.2.187.

GROWTH HORMONE (GH) AND TRIGLYCERIDE-RICH LIPOPROTEIN (TRL) METABOLISM: EFFECT OF ONE MONTH OF DISCONTINUED GROWTH HORMONE TREATMENT IN GROWTH HORMONE DEFICIENT PATIENTS

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Aim: Growth hormone (GH) deficiency is associated with increased cardiovascular mortality. Insulin resistant states and lipid profile alterations with hypertriglyceridemia, decreased HDL cholesterol, increased small and dense LDL and postprandial hyperlipidemia are commonly observed in GH deficient patients but GH specific function in triglyceride-rich lipoprotein (TRL) metabolism regulation remains unclear.

Methods: Lipids and apolipoproteins (apo) profiles were investigated in fasting and postprandial states in plasma and TRL fractions during a test meal before (M0) and after one month of discontinued GH treatment (M1) in GH deficient patients (n=10).

Results: After one month of discontinued GH treatment, GH levels remained unchanged but IGF-1 decreased significantly (-12,12±2,36nmol/L). We observed a significant weight loss (-1,31±0,45kg) together with body composition significant changes : lean mass decrease and fat mass increase (-1,17±0,41kg and +0,36±0,16kg respectively), and insulin resistance parameters modifications with a significant HOMA-index reduction (-0,45±0,19) associated with both significant decreased fasting insulin and C-peptide (-2,04±0,78µmol/L and -0,15±0,05nmol/L respectively) but fasting plasma glucose remained unchanged. Fasting plasma triglycerides and apoC-III levels decreased significantly (-0,41±0,16mmol/L and -23,07±6,93mg/L respectively), main other fasting lipid parameters assessed in the plasma fraction (total, LDL and HDL cholesterol, but also apoA1, apoB, apoB48 and apoC-II) were unchanged. Triglycerides but also apoC-II and apoC-III levels assessed in TRL fraction decreased significantly both in fasting and postprandial states after GH discontinuation.

Conclusions: Lipid profiles alterations both in fasting and postprandial states were observed in GH deficient patients after GH treatment discontinuation suggesting a specific role of GH in TRL metabolism specifically altered in this pathology.