CI: 0.98–1.17, p=0.141), whereas Lp(a) was independently associated with CAD (OR: 1.02, 95% CI: 1.00–1.03, p=0.015).
Conclusions: Serum RLP-C levels were significantly associated with the presence and severity of CAD in patients with FH. However, the clinical usefulness of measuring RLP-C levels beyond that of measuring TG levels should be further assessed.

P6270
Hepatic FDG uptake and visceral adipose tissue volume in individuals with hereditary hyperlipidaemia syndromes

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Background: Familial combined hyperlipidaemia (FCH) and heterozygous familial hypercholesterolaemia (heFH) are the more common hereditary hyperlipidaemia syndromes. However, there are several differences in their phenotype, especially in the prevalence of non-alcoholic fatty liver disease and obesity.
Purpose: To compare the degree of 18F fluorodeoxyglycose (FDG) uptake in the liver, as a marker of metabolic activity that is influenced by insulin resistance and obesity, and the visceral adipose tissue (VAT) volume between individuals with FCH and heFH.
Methods: In total 42 statin naïve individuals (14 FCH, 14 heFH and 14 non-dyslipidemic controls) were enrolled. Blood lipid profile was obtained from all study patients. All participants underwent integrated FDG positron emission tomography (PET)/computed tomography (CT). Target to background ratio of the liver (TBRLIVER derived from SUVmax/blood pool SUVmean) was used for the quantification of FDG uptake in the hepatic tissue. Volume of VAT was measured from CT images between the proximal (cephalic) end of the L1 and distal (caudal) end of the L3 vertebrae.
Results: FCH individuals exhibited higher TBRLIVER values compared to heFH individuals (3.25±0.46 versus 2.65±0.33, $p<0.001$) and controls (3.25±0.46 versus 2.41±0.27, $p<0.001$). In addition, FCH individuals demonstrated higher VAT volumes compared to heFH patients and controls (2233.7±776.5 versus 1120.8±815.2 versus 806.3±716.8 cm^3, $p<0.001$). There was a significant correlation between TBRLIVER and triglyceride levels (R=0.6, $p<0.001$). Moreover, significant associations of VAT volume with triglyceride and non-HDL levels were observed (R=0.45, p=0.003 and R=0.39, p=0.042, respectively). Finally, a strong correlation was observed between TBRLIVER and VAT volume (R=0.68, $p<0.001$).
Conclusion: Individuals with FCH are characterized by higher liver FDG uptake and visceral fat volume compared to those with heFH. These findings potentially reflect different pathophysiological mechanisms involved in the expression of each phenotype.

P6271
Variants of CETP, LIPC, and SCARB1 genes in Korean patients with very high HDL-cholesterol levels

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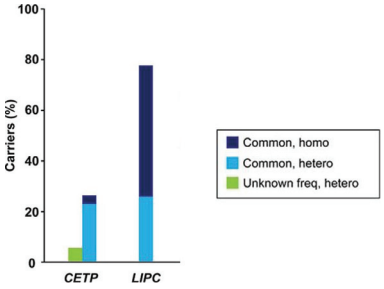
Background: Although the HDL-C levels may be manifesting polygenic effects, there are no sufficient data on genetic variants associated with very high HDL-cholesterol, particularly in Asians.
Purpose: The aim of the present study was to evaluate the prevalence and characteristics of variants of CETP, LIPC, and SCARB1 genes in Korean patients with very high HDL-cholesterol.
Methods: Forty-two patients (mean age: 54 years; male: 38%) with HDL-cholesterol >100 mg/dL (mean: 110 mg/dL) were selected from 13,545 Korean subjects enrolled in a cohort for cardiovascular genome study. Three above-mentioned candidate genes were sequenced by targeted next-generation sequencing. Prediction of functional changes were conducted using SIFT, PolyPhen-2 and Mutation Taster, and matched against public databases.
Results: Four rare variants in CETP and SCARB1 were found in four patients. These four variants are all novel and heterozygous, and two variants in two patients (4.8%) of them were suspected to be pathogenic. Five common variants (two in CETP and three in LIPC) in two genes were discovered in 34 patients. Among the common variants, c.A1196G (p.D399G) in CETP and c.A644G (p.N215S) in LIPC were identified in 13 (31.0%) and 32 (76.2%) individuals, respectively, and had disease-causing probability. No common variants were identified in SCARB1 in the current analysis.
Conclusions: Rare homozygous variants in three target genes were very infrequent in the study subjects, whereas 5% of patients showed putative pathogenic rare heterozygous variants in CETP or SCARB1. Most of the study population was affected by common variants of CETP or LIPC.



Prevalence of carriers

P6272
The relationship between plasma levels of lipoprotein(a) [Lp(a)], PCSK9 and their complex in hypercholesterolemic patients depends on the apolipoprotein(a) phenotype

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Purpose of the study: The aim of this study was to investigate plasma levels of lipids, lipoprotein(a) [Lp(a)], protein convertase subtilisin-kexin type 9 (PCSK9) and Lp(a)-PCSK9 complex, lipoprotein subfractions in hypercholesterolemic patients with different apo(a) phenotype.
Materials and methods: The study included 205 patients with total cholesterol (TC) >7.5 mmol/L and low density lipoprotein cholesterol (LDL-C) >4.9 mmol/L from 18 to 76 years (53±11), 79 (38%) male, 63 (31%) took statins. Coronary heart disease (CHD) was verified in 32 patients. Lipid parameter, Lp(a) and PCSK9 plasma level, apo(a) phenotypes were measured in blood serum. Depending on apo(a) phenotype all patients were divided into two subgroups: high-molecular weight (HMW) and undetectable apo(a) phenotype (subgroup 1, n=145) and low-molecular weight (LMW) apo(a) phenotype (subgroup 2, n=60). Plasma level of Lp(a)-PCSK9 complex was measured in 80 statin-naive patients without CHD. Lipoprotein subfractions were determined by "Lipoprint" Systems.
Results: The subgroups were comparable by clinical characteristics and lipids. The mean level of PCSK9 also did not differ between the subgroups 368±126 and 343±118 ng/mL, $p>0.05$. In the whole group the PCSK9 level correlated with TC (r=0.17, $p<0.01$), LDL-C (r=0.17, $p<0.01$), triglycerides (TG) (r=0.22, $p<0.005$). We found a weak relationship between the concentration of Lp(a) and PCSK9 (r=0.16, $p<0.005$), which was enhanced in the subgroup of patients with LMW apo(a) (r=0.36, $p<0.005$) and disappeared in subgroup 1 (r=0.15, p=0.36) regardless of the of hypolipidemic medications.
In the LMW apo(a) subgroup Lp(a) and TG levels (r=0.53, $p<0.005$; r=0.31, $p<0.05$, respectively) were independent predictors of PCSK9 concentration, independent of age, sex, TC, LDL-C and use of statins. In subgroup 1 concentration of PCSK9 was associated with age, TC concentration and statins ($p<0.05$ for all). In the whole group there was a significant correlation of Lp(a)-PCSK9 complex concentrations with TC (r=0.40, $p<0.005$), and large subfractions of intermediate density lipoproteins (r=0.30, $p<0.05$) and LDL-1 r=0.30, $p<0.05$ but not with Lp(a) or PCSK9 levels. The correlation of Lp(a)-PCSK9 complex with large LDL-1 and IDL-C concentrations was significant only in LMW apo(a) subgroup (r=0.59, $p<0.005$ and r=0.40, $p<0.05$, respectively).
Conclusions: A positive relationship between concentrations of Lp(a) and PCSK9 as well as between Lp(a)-PCSK9 complex and large apoB100-containing lipoprotein subfractions was observed only in hypercholesterolemia patients with LMW apo(a) phenotype. These facts illustrate the difference in metabolism and pathophysiological properties of Lp(a) with small and large apo(a) isoforms.

P6273
Impact of lipoprotein apheresis on thrombotic parameters in patients with refractory angina and raised lipoprotein(a)

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Background: Raised lipoprotein(a) [Lp(a)] is a cardiovascular risk factor and common in patients with refractory angina. The apolipoprotein(a) component of Lp(a) exhibits structural homology with plasminogen, and can enhance thrombosis and impair fibrinolysis.
Purpose: To assess the effect of Lp(a) lowering with lipoprotein apheresis in patients with refractory angina and raised Lp(a) on markers of thrombosis and fibrinolysis.
Method: In a prospective, single-blind, crossover trial, 20 patients with refractory angina and raised Lp(a)>500mg/L, were randomised to three months of weekly lipoprotein apheresis or sham, followed by crossover. Blood taken before and after apheresis/sham was assessed using the Global Thrombosis Test, to assess