significant higher in patients with impaired fasting glucose than in normal controls and again significantly lower than in type 2 diabetic patients.

**Table 2. Distribution of HDL-C levels of the study groups**

<table>
<thead>
<tr>
<th>HDL-C</th>
<th>Healthy Controls (n=8544)</th>
<th>Patients with impaired fasting glucose (n=4100)</th>
<th>Patients with type 2 diabetes mellitus (n=1586)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30 mg/dL (%)</td>
<td>6.0</td>
<td>7.7</td>
<td>13.3</td>
</tr>
<tr>
<td>31-35 mg/dL (%)</td>
<td>9.2</td>
<td>11.9</td>
<td>28.4</td>
</tr>
<tr>
<td>36-40 mg/dL (%)</td>
<td>14.7</td>
<td>17.3</td>
<td>37.0</td>
</tr>
<tr>
<td>41.45 mg/dL (%)</td>
<td>17.3</td>
<td>17.6</td>
<td>12.2</td>
</tr>
<tr>
<td>&gt; 46 mg/dL (%)</td>
<td>52.8</td>
<td>45.5</td>
<td>0</td>
</tr>
</tbody>
</table>

**W7-P-003 ASSOCIATIONS OF LPL VARIANTS, HIND III AND PVU II, WITH PLASMA LEVELS OF LIPIDS, LIPOPROTEINS, AND INSULIN RESISTANCE, IN A TYPE 2 DIABETIC POPULATION**


**Objective:** To look for associations among LPL intronic variants 8 (Hind III) and 6 (Pvu II) and levels of plasma lipids, lipoproteins, insulin and HOMA indices in 874 patients.<br>

**Methods:** Biochemical and genetic analysis were determined in 118 type 2 diabetic patients. Lipids and lipoproteins were measured after ultracentrifugation, in a Cobas Mira S 89 (Roche), small dense LDL cholesterol in the Lipoprint System (Quantimatrix) and systemic insulin levels in an Immulite One (DPC). Genotyping was carried out by PCR amplification of genomic DNA (isolated from EDTA whole blood) and analysis of restriction fragments.

**Results:** Carriers of the H- allele had lower levels of plasma triglycerides (p<0.005), VLDL-triglycerides (p<0.005), apo B100 (p<0.05), apo CII (p<0.005) and higher levels of LDL cholesterol (p<0.05). Concerning the carbohydrate metabolism related markers, H- carriers and P- carriers showed lower levels of insulin and HOMA index (p<0.05). All statistical analyses were done after adjustment for age, sex, BMI, smoking and hypertension.

**Conclusions:** In our population, rare alleles of the LPL intronic variants analyzed are associated with lower levels of variables related with atherogenic risk; concerning lipid and carbohydrate metabolism, for P- allele, or only carbohydrate metabolism, for F- allele. Therefore, they could have a favorable effect.

**W7-P-004 HEPATIC LIPASE GENE C-480T POLYMORPHISM AND AGE MODULATE THE RISK OF ACUTE MYOCARDIAL INFARCTION AND SUDDEN CARDIAC DEATH THE HELSINKI SUDDEN DEATH STUDY**

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**Objective:** The hepatic lipase (HL) C-480T polymorphism has been found to be associated with diminished lipase activity, dyslipidemia, and atherosclerosis, but the role of this polymorphism in atherosclerosis has not been studied at the vessel-wall level. We examined the association of the HL C-480T polymorphism with acute myocardial infarction (AMI) and sudden cardiac death (SCD) in middle-aged men who died suddenly.

**Methods:** 700 unselected sudden or violent out-of-hospital deaths of middle-aged white Finnish men in the Helsinki Sudden Death Study, which comprised two large autopsy series, collected 10 years apart during 1981 to 1982 and 1991 to 1992 (average age 55 years, range 33 to 70 years), was analyzed.

**Results:** Genotype information was obtained for 682 men. In adjusted logistic regression analysis, men with the TT genotype had an increased risk for SCD (n=278, odds ratio [OR] 2.6, P=0.04), AMI (n=84, OR=2.6, P<0.06) and AMI without thrombus (n=44, OR = 4.1, P=0.009) compared with CT and CC genotypes combined. Further, the interaction analysis revealed that the HL C-480T polymorphism was more significantly associated with SCD and AMI in men < 53 years old but not with that in older men (P<0.005 and P<0.05, respectively, for age-by-genotype interaction). In men < 53 years old, the subjects with TT genotype had higher risks for AMI and SCD (OR=2.6, OR=0.008 and OR = 11.2, P>0.001, respectively) compared with CT and CC genotypes combined.

**Conclusions:** The results from this autopsy series suggest that HL C-480T polymorphism may be associated with increased risk of SCD and AMI in men, especially in early middle age.

**W7-P-005 HEPATIC LIPASE PROMOTER VARIANT (C-514>T) INFLUENCES PLASMA LEVELS OF LDL CHOLESTEROL IN A LARGE CAUCASIAN POPULATION**

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**Objective:** The importance of a hepatic lipase (HL) gene for plasma HDL cholesterol (HDL-C), levels determination has been suggested in population studies. We have evaluated the influence of HL promoter polymorphism (C-514>T) on plasma lipid levels in Czech Caucasian population. Lipid levels have been analysed in 1997 and in 2001 in all individuals.

**Methods:** C-514>T polymorphism in HL gene was analysed by PCR and restriction analysis in representatively selected 1173 males (aged 49.2 ± 10.8 years) and 1369 females (aged 48.8 ± 10.6 years) of Czech origin.

**Results:** In the population, there were 38.9% of the T-514 allele carriers. Variation in the HL gene affects the plasma HDL cholesterol levels showing a higher levels in TT homozygotes than in CC homozygotes with CT heterozygotes having the medium value both in males (1.40 ± 0.36 mmol/l, 1.27 ± 0.32 mmol/l vers. 1.22 ± 0.33 mmol/l, p<0.001 for linear trend); and females (1.58 ± 0.43 mmol/l, 1.51 ± 0.36 mmol/l, 1.48 ± 0.36 mmol/l, p<0.05 for linear trend). This association was not influenced by age and/or smoking status.

Additionally, C-514>T variant influenced BMI in females. CC homozygotes have higher BMI (27.6 ± 5.76 kg/m²) than TT homozygotes (25.9 ± 6.01 kg/m²) (p<0.05). After adjustment for BMI, the association between HL variant and HDL cholesterol in females disappeared. Other analysed lipid parameters (total cholesterol, LDL cholesterol, triglycerides) have not been associated with HL variant.

**Conclusions:** We conclude that C-514>T variation in the regulatory part of hepatic lipase gene play an important role in genetic determination of plasma HDL cholesterol levels. After adjustment for BMI the effect of this variant is detectable in males but not in females.

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**W7-P-006 FAMILIAL LECITHIN-CHOLESTEROL ACYLTRANSFERASE DEFICIENCY: CHARACTERISTICS OF THE CARRIERS OF A NEW LCAT MUTATION FROM A POLISH FAMILY**

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Familial LCAT deficiency (FLD) is a rare genetic disorder associated with corneal opacities, anaemia and proteinuria with renal failure. The aim of this study was to provide a biochemical characteristics and molecular analysis of a new LCAT mutation in a Polish family. Serum lipids were measured enzymatically, LCAT activity by using endo- and exogenous substrates; genetic analysis included PCR and sequencing of DNA. We examined two siblings, presenting classical symptoms of FLD, in whom a new Val309Met mutation in the exon 6 of LCAT gene was identified. Both patients were homogeneous carriers of a newly discovered mutation. They displayed low total (219 mg/dl and 294 mmol/l) and HDL-cholesterol concentrations (0.52 and 0.48mmol/l), low percentage of cholesteryl esters (11.1% and 12%) and decreased ape AI, ape AII, apo B and Lp(a) serum levels. LCAT activity against exogenous proteoliposomes was 0, by Stokke Norum method was 10% of normal and LCAT concentration was undetectable by immunanassay. Plasma CETP activity was at low limits of.