PolyPhen-2 and SIFT predicted as "potentially harmful" the effect of the following variants: pArg50Cys observed in 9 subjects (9.37%), p. Arg198Glu present in 1 subject (1.05%) and p.Asp440Lys found in 6 patients (6.25%).
We observed differences in the distribution of allele frequencies for c.502-256G>A and c.502-133G>A variants, being more frequent in hypercholesterolemic individuals than in 1000 Genomes, with statistical significance ($p<0.05$).
Conclusion: We have identified 14 variants in ABCG5 gene in subjects affected of primary hypercholesterolemia. The 16.7% of the studied subjects presented a change predicted as pathogenic by bioinformatic analysis. The p.Arg50Cys variant, that we have found in 9 subjects, has been associated with gallstones. We have also identified by the first time the p.Asn285Ser variant.

40 - Inherited dyslipidemias

EAS-0381.
DYSLIPIDEMIA REGISTRY OF THE SPANISH ATHEROSCLEROSIS SOCIETY

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Objectives: Introduction: Registries are useful tools in biomedical research that allow sharing information, a better knowledge of clinical practice, and facilitate the improvement of diseases management. Since May 2013, a National Registry of Dyslipidemias launched by the Spanish Atherosclerosis Society (SEA) is operating in Spain. Lipids Units around the country collect information of the main dyslipidemias attending lipid units.

Objective: To develop an 'on line' application that would allow all lipids units of the SEA to introduced clinical information in a uniform database, and to study the status and type of enrolment after operating during 8 months.
Methods: SEA and a Spanish Software company, named Infozara, created an 'on line' platform (www.infozara/rihad.es) that allows to each lipid unit to introduce clinical information: demographic data, familial and personal history of cardiovascular disease and risk factors, anthropometric measurements, lipid values without and with treatment, genetic analysis, diagnosis and treatments.
Results: To January 08 st, 46 lipids units have joined the application, and 1481 cases have been included. The most common dyslipidemias introduced so far are: familiar hypercholesterolemia (FH) with (FH+) or without LDLR, APOB or PCSK9 mutation (FH-), disbetalipoproteinemia (DLP), familiar combined hyperlipidemia (FCHL) and polygenic hypercholesterolemia (PH).
Conclusion: At present, FH+, FH- and FCHL are the dyslipidemias most commonly introduced in the SEA Dyslipidemias Registry. These figures probably indicate the high level of specialization of the Lipid Clinics involved, and anticipate that the registry is going to give relevant scientific knowledge about dyslipidemias of our country.

	FH+	FH-	FCHL	HP	DLP	OTHERS	TOTAL
MALES	329	61	110	28	25	203	**756**
FEMALES	342	86	67	27	19	184	**725**
TOTAL	**671**	**147**	**177**	**55**	**44**	**387**	**1481**

40 - Inherited dyslipidemias

EAS-0292.
CORONARY CALCIUM SCORING AND ATHEROMA ASSESSMENT IN DYSBETALIPOPROTEINEMIC PATIENTS

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Objectives: Dysbetalipoproteinemia results from a defect in the clearance of VLDL and chylomicron remnants due to homozygous Apo E2 variant or heterozygous Apo E mutation. Whereas these patients are considered at high cardiovascular risk, little is known about the exact prevalence of ischemic cardiovascular complications and subclinical atheroma. We systematically investigated atheroma severity by coronary calcium scoring (CAC), carotid intima media thickness (IMTc), and ankle brachial index (ABI). Additionnaly we investigated our ability to predict the atheroma intensity according conventional cardiovascular risk factors.

Methods: Observational study of 31 dysbetalipoproteinemic patients older than 40 (60.9±10.6 years old, men 58%). Systematic clinical and lipid assessment, full apo E sequencing, CAC, IMTc and ABI measurements.
Results: 7 patients (22.6%) had a history of myocardial infarction. Following risk stratification by Agatston score, three groups were identified: low cardiovascular risk (CAC=0, n=12), intermediate cardiovascular risk ($0<CAC\leq100$, n=9) and high cardiovascular risk (CAC>100, n=10). The 7 patients in secondary cardiovascular prevention were all found in the third group (6/7 had CAC>300). Among CVRF, only age was increased in patients with the higher score (cf Table). Framingham risk score, IMTc and carotid stenosis failed to identify the patients with the highest CAC. Conversely, ABI was inversely correlated with CAC score (p=0.06), and a higher prevalence of stenosis in the lower limbs was found in the group with CAC>100 (p=0,01).

	CAC=0	0<CAC≤100	CAC>100	p univariate	p multivariate
Age(years)	56.7±11.0	59.7±8.4	67.0±9.9	0.06	0.09
Hypertension(%)	41.7	55.6	100	0.01	0.70
Renal Failure(%)	0	22.2	50.0	0.02	0.90
Diabetes(%)	41.7(n=5)	44.4(n=4)	70.0(n=7)	0.37	

Conclusion: 32% of the patients in this cohort were in secondary prevention and/or with high CAC score however 39% were free of any obvious atheromatous lesions. Conventional CVRF, alone or combined in risk score were unable to identify patients with the most or less severe atheromatous lesions. A systematic screening for sub-clinical atheroma by measuring CAC score and ABI seems necessary in dysbetalipoproteinemic patients older than 40 in order to adjust cardiovascular prevention and follow-up.

41 - Secondary dyslipidaemia

EAS-0501.
EFFECTS OF TUMOR BEARING ON SERUM AND LIVER LIPID LEVELS IN RCN-9-IMPLANTED RATS

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Objectives: To investigate the effects of a bearing of RCN-9, a transplantable rat colon adenocarcinoma, on lipid metabolism, changes in serum and liver lipid levels were investigated in RCN-9-implanted rats. In the tumor-bearing state, decreases in food intake and body weight are seen as a result of a cancer-induced cachexia; therefore, a pair-feeding study was

conducted to investigate the effects of food restriction on tumor-bearing in RCN-9-implanted rats.

Methods: F344 rats were divided into three groups (n=7) of similar body weights, with the first group receiving a subcutaneous implantation of 5×10^6 RCN-9 cells suspended in phosphate-buffered saline (PBS(-)) in the back to produce a solid tumor (tumor-bearing group), with the second group receiving a sham injection of PBS(-) alone, this group being designated normal rats (control group). Normal rats of the last group were given the same amount of diet which group of the tumor-bearing rats ate on the day before, and this group designates pair-fed group. The rats of each group were maintained for a further 21 days.
Results: Both the serum and liver thiobarbituric acid-reactive substances (TBARS) values in the tumor-bearing group were significantly higher than those in the control group. In the pair-feeding study, there were no significant differences between the control and pair-fed groups with regard to the serum and liver TBARS values. The serum and liver TBARS values in the pair-fed group were not significantly different from those of the tumor-bearing group. The solid tumor could be observed 6-7 days after RCN-9 implantation, and continued to grow with time.
Conclusion: In RCN-9-implanted rats, serum and liver TBARS values undergo enhancement, resulting in abnormal lipid metabolism, and that the serum and liver TBARS value-enhancing actions might be due, at least in part, to the tumor-bearing effect itself and not to the suppression of food intake accompanying the tumor-bearing state.

41 - Secondary dyslipidaemia

EAS-0234.
BEYOND LIPID PLASMA CONCENTRATION: FUNCTIONAL EVALUATION OF HDL AND VLDL IN PATIENTS WITH CHRONIC KIDNEY DISEASE UNDER HEMODIALYSIS

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Objectives: Chronic kidney disease is associated with chronic inflammation and atherosclerosis. Its characteristic dyslipidemia consists in moderately increased triglycerides (TG) and low HDL. Qualitative alterations in lipoproteins may affect their functionality. Objective: To assess functions of VLDL and HDL in patients on hemodialysis (HD).

Methods: We studied 15 HD -of various etiologies- and 10 healthy controls (C), of both sexes. Ages, HD: 61±17 years and C: 32±10, $p<0.001$. Fasting serum lipid-lipoprotein profile and cholesteryl ester transfer protein (CETP) activity were measured. VLDL was isolated and its capacity as lipoprotein lipase (LPL) substrate was determined by an *in vitro* lipolysis assay, incubating VLDL with bovine LPL and determining the constant Km -inverse to affinity-. Furthermore, LDL and HDL were isolated for the *in vitro* evaluation of HDL antioxidant effect on LDL, expressed as % of inhibition of LDL oxidation. All results were adjusted by age.
Results: HD showed increased TG (143±88mg/dL vs. 92±32) and decreased HDL-cholesterol (41±15mg/dL vs. 60±15) $p<0.001$. HD had higher VLDL-cholesterol content evidenced by the cholesterol/TG ratio (HD: 0.36±0.10 vs. C: 0.22±0.11 $p<0.004$). CETP activity was higher in HD: 247 (131-417%ml/h) vs C: 181 (92-54) $p<0.007$ and it was positively associated with VLDL-cholesterol ($r=0.33$ $p<0.001$). VLDL from HD presented lower affinity for LPL (Km HD: 4.0±3.0 mM vs. Km C: 2.1±0.9, $p<0.05$), and Km was associated with VLDL-cholesterol content ($r=0.38$, $p<0.001$). Although HDL inhibited LDL oxidation in all cases, HDL antioxidant capacity was lower in HD (HD: 22±23% vs. C: 54±33, $p<0.05$).
Conclusion: VLDL from patients under HD presented less lipolysis capability as substrate of LPL, associated with its enrichment in cholesterol induced by increased CETP activity. In addition, HDL from HD was less efficient in protecting LDL from oxidation. Thus, in HD, VLDL and HDL not only present quantitative alterations but also an impaired functionality, aggravating the atherogenic frame.

42 - Polygenetic and multifactorial dyslipidemias

EAS-0707.
NON-HDL-C AND APOB GOALS EQUIVALENT TO LDL-C TARGETS IN MIXED DYSLIPIDEMIC PATIENTS

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Objectives: Latest IAS recommendations highlight the importance of non-HDL-c, the most atherogenic fraction.

The increase in atherogenic particles derived from triglyceride-rich lipoproteins means an additional cardiovascular risk from this associated to LDL-c. Measurement of non-HDL-c and apoB estimate total burden of atherogenic particles.
The aim of this study was to establish equivalence between LDL-c targets and non-HDL-c and apoB in mixed dyslipidemia.
Methods: A total of 373 patients diagnosed with mixed dyslipidemia were studied (61.93% male; 18-69 aged).
Lipid profile was measured, including cholesterol, triglycerides and apoB in lipoproteins fractions isolated by ultracentrifugation.
Statistical analysis was performed using SPSS 20.0
Results: Non-HDL-c is highly correlated with LDL-c ($R^2>0.905$), nevertheless this correlation decreases when Tg≥500mg/dL ($R^2=0.579$). A similar behaviour is observed between LDL-c and apoB: $R^2>0.704$ if Tg<500mg/dL and $R^2=0.475$ if Tg≥500mg/dL.
Equivalences between LDL-c target and non-HDL-c or apoB were calculated by:
non-HDL-c = 8.422 + 1.027*LDL-c + 0.08*Tg ($R^2=0.908$)
apoB = 21.486 + 0.684*LDL-c – 0.010*Tg ($R^2=0.710$)

LDL-c	Tg	non-HDL-c	apoB
70	150	92,31	67,87
	200	96,31	67,37
	250	100,31	66,87
	300	104,31	66,37
	500	120,31	64,37
100	150	123,12	88,39
	200	127,12	87,89
	250	131,12	87,39
	300	135,12	86,89
	500	151,12	84,89

Conclusion: In our mixed hyperlipidemic population the non-HDL-c goals set at 30 mg/dL higher than LDL-c goals is valid only for Tg=250 mg/dL. However Tg>500 mg/dL increase the equivalence to 50 mg/dL. In addition, very high triglycerides levels get worse LDL-c and non-HDL-c correlation, as well as non-HDL-c and apoB correlation.