Conclusion: Our results suggest a lack of sympatho-vagal balance activation in the first 5 min of tilt test in subjects with vasovagal syncope. CI and VD syncope groups show a lower decrease of HF indices in the Early5. Moreover, CI group shows a lower increase of LF indices. A perspective of this work is the set of cut-off values from data recorded over the first 5 min of tilt test to estimate the risk and the type of vasovagal reaction, based on a bigger population.

Figure (abstract 235) – LFnu and HFnu variations (%) in NEG and CI

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Effects and mechanisms of n-3 polyunsaturated fatty acids in the fetal pulmonary circulation

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The effect of n-3 polyunsaturated fatty acids in the pulmonary circulation is not well defined. This question is clinically relevant in respiratory failure associated with pulmonary hypertension. To investigate this effect we have realised a randomized, placebo-controlled comparative study on sixty-two chronically prepared lamb fetuses.

Methods: Catheters and ultrasonic flow transducer were placed through a left thoracotomy in the lamb fetuses to determine aortic, pulmonary, and left atrial pressures and left pulmonary artery blood flow. We compared the pulmonary vascular responses to 120 min of Omegaven (lipid emulsions enriched in n-6 polyunsaturated fatty acids) or Intralipide (lipid emulsions enriched in n-3 polyunsaturated fatty acids) infusion. Then we investigated the effects of Omegaven on the pulmonary circulation after nitric oxide synthase inhibition by L-nitro-arginine, potassium channel blockade by tetraethylammonium, cytochrome P450 epoxygenase inhibition by (methylsulfonyl)-2- (2-propynyloxy)-benzenehexanamide, and cyclo-oxygenase inhibition by Ibuprofen.

Results: Pulmonary artery and aortic pressures as well as blood gases and plasma lactate concentrations did not change during either fat emulsion infusion. Left pulmonary blood flow increased by 30% and pulmonary vascular resistance decreased by 29% during Omegaven infusion, whereas they did not change during Intralipide infusion. This pulmonary vascular response to Omegaven was not altered by L-nitro-arginine or Ibuprofen infusion. At the opposite, Omegaven induced pulmonary vasodilatation was abolished by tetraethylammonium and markedly attenuated by (methylsulfonyl)-2- (2-propynyloxy)-benzenehexanamide.

Conclusion: Lipid emulsion containing n-3 polyunsaturated fatty acids may induce a potent and sustained vasodilatation in the fetal lung. This pulmonary vasodilator response is mediated through production of vasoactive mediators by cytochrome P450 epoxygenase and through activation of potassium channels.

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Phosphatidylcholine distribution between lipoprotein fractions in patients with CAD

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Background and objectives: Recent data have shown that phospholipids (mainly phosphatidylcholine) transfer activity is associated to metabolic syndrome and coronary artery disease (CAD). This transfer activity could modulate the phosphatidylcholine (PC) distribution between lipoprotein subclasses.

Methods: Ninety two subjects were recruited (47 CAD patients and 45 age- and sex- matched controls). Anthropometric measurements, lipid profile and PC analysis were performed.

Results: Despite from an abnormal lipid profile: elevated serum triglycerides and LDL-C, and low HDL-C (but total cholesterol level was not significantly different between controls and CAD patients), serum PC concentrations were slightly higher in CAD patients than in controls (respectively 2.53±0.84 and 2.36±0.43 mmol/L, ns). The level of PC was significantly higher in patients, compared to controls, concerning the (VLDL+LDL) fractions (p<0.01). In concomitance, HDL-PC was decreased in CAD patients in comparison with controls (p<0.05). Furthermore, serum PC level was colosly correlated to waist/hip ratio only in CAD patients (p=0.0013). However, PC concentrations were less closely associated to BMI in the same group (p=0.035).

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Conclusion: Serum phospholipid status is modulated in patients with CAD. This involves a significant modulation of the PC distribution between lipoprotein subclasses and probably reflects an abnormality in the phospholipid transfer activity (PLTP activity) representing a useful marker of cardiovascular risk.

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Lack of association between estrogen receptor α gene polymorphisms and myocardial infarction in Tunisian population

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Introduction: Coronary artery disease (CAD) is a multifactorial disease, its expression probably being influenced by the interaction of genetic and environmental risk factors. Genetic variation in the estrogen receptor-alpha gene (ERα) has been associated with CAD in several populations worldwide, but results are still controversial. In the present study, we examined the association between the c.454-397T>C: Pvull restriction site (rs2234693) and c.454-351A>G:Xbal restriction site (rs9340799) polymorphisms (SNPs) and myocardial infarction (MI) in a sample of the Tunisian population.

Material and methods: The case group included 294 patients with MI, and the control group comprised 275 individuals with angiographically normal coronary arteries and without signs or symptoms of MI. ESR1 Pvull and Xbal polymorphism was determined by polymerase chain reaction (PCR)-restriction fragment length polymorphism (RFLP) method.

Results: Genotype distributions of the SNPs in the control and patient groups were consistent with those expected for samples in Hardy-Weinberg equilibrium (p> 0.05). The Genotype distribution and the relative allelic frequencies of the –397T>C and – 351A>G polymorphisms were not significantly different between the control and patient groups (p=NS). Moreover, the odds ratio for MI associated with the c.454-397C (OR=1.02, 95% CI 0.81-1.30), and c.454-351G (OR=1.24, 95% CI 0.98-1.58) variants failed to reach statistical significance.

Conclusion: In the current study, we were unable to demonstrate a significant association of the ESR1 c.454-397T>C and c.454-351A>G polymorphisms with MI. These results do not support the extent of risk associated with developing MI previously reported for the ESR1 c.454-397T>C and c.454-351A>G polymorphisms.