INTRODUCTION: Exercise training reduces atherogenesis in animals model. However, the mechanisms underlying the anti-atherogenic effects of exercise remain unclear. In the present study, we investigated the role of a long-term exercise training on the lipid parameters, the endothelial signal transduction cascade leading to eNOS activation and the plaque morphology in ApoE-/- mice. METHODS: 10-11 week old apoE-/ mice were divided into 2 groups treated with a lipid-rich diet: the exercise group which underwent a 6-month swimming protocol (50 min/day; 5 days/week) and the sedentary group. Total cholesterol, HDL and non-HDL cholesterol, phospholipids and triglyceride levels were determined enzymatically at sacrifice. The aortic levels of total eNOS and total Akt were assayed by Western blotting. To assess plaque morphology in aortic sinus, we quantified smooth muscle cell (SMC) content in the fibrous cap and we explored plaque inflammation by macrophage quantification (immunohistochemistry analysis). PRELIMINARY RESULTS AND DISCUSSION: Up-to-date a part of data is still in processing. According to the litterature and to our previous published results, swimming did not modify blood lipid profile in ApoE-/- mice. We showed that arterial Akt and eNOS protein expression was not different between exercise and sedentary ApoE-/ mice. Thin fibrous cap with little proliferation of SMC and macrophage accumulation in the atherosclerotic plaque are two accepted predictors of both plaque vulnerability and thrombogenicity. SMC content in fibrous cap was higher in exercise group compared with sedentary group. Macrophage plaque content was decreased with swimming suggesting a better plaque quality. CONCLUSION: Our data demonstrated that a long-term swimming exercise enhances atherosclerotic plaque morphology independently of a blood lipid modification and a change in eNOS protein expression.

Keywords: Lipid Metabolism, Swimming, Atherosclerotic Disease