Abstract

To investigate molecular mechanism of action of nicotinic acid in the treatment of atherosclerosis, we have used genetically modified mice, LDLR k.o. mice and PumaG double deficient mice. Mice were fed high fat diet for sixteen weeks and treated with or without 0.3% nicotinic acid in drinking water. Afterwards mice were sacrificed and the hearts were used for determination of the atherosclerotic lesions by defining the plaque area and areas immunostained for macrophages.

Further we aimed at clearing the mechanism of action of nicotinic acid. By using fluorescence microscopy we found area with expression of gene GPR109A in peritoneal exudates cells. Next, by setting the limit of intracellular calcium I was measuring the response of peritoneal cells to the nicotinic acid with subsequent addition of chemokines – mcp-1, rantes, il-8, mip-1- α. The goal was to lower the level of chemokines and thus to reduce the inflammation.