ABSTRACT

Atherosclerosis, or sclerosis of arteries, is a degenerative disease of arteries. Sometimes it is called „the disease of 20th century“.

ApoE/LDL – receptor double knockout mice represent a new animal model for study of atherogenesis, which is characterized by severe hyperlipidaemia and atherosclerosis.

Statins (or competitive inhibitors 3-hydroxyl-3-methyl-glutaryl-coenzym Areductase) currently belong to the most efficient and the most useful hypolipidemic drugs for all over the world. They decrease mainly levels of total cholesterol and LDL cholesterol.

The aim of this rigorous thesis was to describe the expression of endoglin and SMAD 2 in atherosclerotic plaques in apoE/LDL-receptor deficient mice. Moreover we wanted to determine the effect of atorvastatin treatment on the expression of both endoglin and SMAD 2.

ApoE/LDLR-deficient mice on were subdivided into 2 groups. The control group of animals was fed with the western type diet. The same atherogenic diet was used in ATV group, where atorvastatin was added to the atherogenic diet at the dosage of 100 mg/kg per day.

The results of this thesis confirmed the expression of endoglin and SMAD 2 in atherosclerotic lesions in ApoE/LDLR-deficient mice. The expression of endoglin was located on the aortic vascular endothelium and in other smaller vessels and capillaries of surrounding myocardium. SMAD 2 expression was visible in whole atherosclerotic lesion (intima, and endothelium). Atorvastatin treatment resulted in a strong hypolipidemic effect.

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In addition western blot analysis showed significant increase of the expression of both endoglin and SMAD 2 after atorvastatin treatment.

These results indicate that endoglin and SMAD 2 could be potencional target of atorvastatin effect and suggesting these proteins could have antiatherogenic properties.