Atherosclerosis and its complications are still the leading cause of death in Poland and in the world. *Smooth muscle cells* (SMCs) are the dominant component of the arterial wall. The dominant physiological function of SMCs is shrinkage, which helps to maintain adequate vascular wall pressure and enables blood flow regulation through the organs. In the process of atherogenesis, SMCs change the phenotype from "contractile" to "synthetic," which becomes an important contributor to the development of atherosclerotic lesions in the artery wall. It has been shown that fatty acids, by specific receptors, can modulate the phenotype of different cell types, including selected cell types involved in the development of atherosclerotic lesions (macrophages, endothelial cells). The present project aims to investigate the role of the GPR120 fatty acid receptor in vivo modulation of smooth muscle cell phenotype in vitro in cultured smooth muscle cells and in vivo on animal models of atherosclerosis in apoE knockout mice. Evaluation of the potential effects of the GRP120 receptor on mechanisms involved in regulation of cell phenotype will contribute to a better understanding of atherogenesis and possibly further research into the use of the GRP120 receptor pathway for the prevention or treatment of atherosclerosis.