Atherosclerosis as a chronic inflammatory vascular disease is the base of many cardiovascular diseases in the world. In Poland, it is estimated that atherosclerotic lesions are responsible for approximately 180,000 deaths annually. Atherosclerosis is a disease associated with the development of chronic degenerative-productive changes in the intima and media of medium and large-sized arteries. The development of the disease is associated with the formation of atherosclerotic plaque, which involves damage to the vascular endothelium, allowing accumulation of lipids and cells such as leukocytes and vascular smooth muscle cells. The structure of the plaque directly affects its stability. Stable atherosclerotic plaque is rich in extracellular matrix proteins that prevent its rupture.

The aim of the study is to determine whether the stiffness of the substrate of cells affects the release of interleukin-6 and its receptor, and whether these changes are related to the accumulation of collagen in cultures of vascular smooth muscle cells both under normal and hyperlipidemic conditions. It is also planned to determine whether IL-6 as a pro-inflammatory factor may affect the process of collagen accumulation. The different stiffness of the substrate reflects changes in the physical properties that occur in the blood vessels in the process of atherosclerosis. The research will be carried out on a human internal thoracic artery smooth muscle cells. *In vivo* studies will be carried out in a apolipoprotein E (*ApoE* $^{-/-}$) deficient mouse model, characterized by the spontaneous development of atherosclerosis.

The expected research results will allow to assess whether IL-6 is involved in collagen metabolism in vascular smooth muscle cells. This will determine whether controlling the level of IL-6 may constitute a new therapeutic target in atherosclerosis that would allow to influence the formation of stable atherosclerotic plaque.