Cardiovascular diseases (CVD) are the most common cause of death and the main reason of disability in Poland. Atherosclerosis and the subsequent cardiovascular complications, such as myocardial infraction, stroke, and ischemic heart failure, are the major cause of death in the Western world. In 2011 about 45.5% of all deaths were caused by CVD. The medical care expenses increase with each year. Only in 2014, 13.3 million zloty was devoted for the program of treatment and prevention of CVD. Thus the CVD seem to be not only social but also economical problem.

The risk factors of atherosclerosis are well known, including hypertension, prolonged inflammation, high blood glucose and/or serum total and low-density lipoprotein cholesterol levels (collectively called as metabolic syndrome symptoms). Lipids have a central role in the pathogenesis of plaques, but the mechanistic links between lipids and atherogenesis are not fully understood. It is known that lipid metabolism in vascular smooth muscle cells (VSMC) is altered in hyperlipidemic and/or hyperglycemic environment. Increased expression of the lipogenic genes as well as enhanced uptake of lipoproteins and fatty acids from plasma cause excessive lipid accumulation in VSMC. This causes serious adverse effects on cell function (lipotoxicity) and can contribute to the development of insulin resistance. On the other hand, impairment of the insulin-dependent pathways in VSMC change glucose metabolism and thus contributes the pathological alteration of cell phenotype.

One of the major regulators of cellular metabolism is stearoyl-CoA desaturase (SCD). SCD is the key-enzyme which controls many signalling pathways involved in regulation energy balance of the cell. Recent studies showed that SCD ablation significantly improves many aspects of the metabolic syndrome including increased insulin sensitivity and glucose tolerance. Consequently, SCD become a potential therapeutic target in the treatment of metabolic syndrome symptoms. Our preliminary data indicate that SCD1 and SCD4 are implicated in arterial remodelling. Therefore, the main aim of the project is to investigate the role of SCD in the VSMC metabolism and phenotypic plasticity. We designed *in vivo* and i*n vitro* studies to investigate the role of SCD1 and SCD4 in lipid-dependent regulation of VSMC function by molecular analysis of SCD-related signalling pathways. We will investigate the role of SCD in regulation of morphology and mechanical properties of thoracic aorta in physiology and pathological conditions. Subsequently, we will establish the functional role of SCD in the regulation of VSMC metabolism by analyses of (1) lipogenesis, (2) lipolysis, (3) β-oxidation, (4) intracellular lipid levels, (5) glucose uptake and parameters of insulin signaling pathway, (6) AMPK pathway, (7) the rate of fatty acids uptake. Successful achievement of this project will contribute to extend the knowledge in the field of VSMC metabolism to maintain proper vascular wall function or to restore arterial function after pathological events.