In the developed countries, cardiovascular diseases (CVDs) are the main cause of death and the inglorious, leading position belongs to ischemic heart disease. There are many factors that can accelerate the disease development, but the one, which contributes the most is atherosclerosis. It causes gradual narrowing of blood vessels and decreased blood flow. Finally, the most affected vessels may clog. It is extremely dangerous in case of occlusion of one or even more coronary arteries, which are supplying cardiac muscle with oxygen and nutrients carried by blood. Such event is called myocardial infarction. In consequence, cardiac cells in affected area are seriously damaged and in the end they die. Within next few days inflammation is developing, as well as the processes involved in remodeling and later healing of damaged tissue are triggered. In the ischemic lesion a scar tissue made of collagens produced by activated fibroblasts is formed. Unfortunately, most frequently it is becoming stiff and rigid. Then, this structure - instead of maintaining the integrity of the heart - disturbs heart contractility leading to arrhythmia, expansion of the injury and finally to the heart failure. Despite existing innovative pharmacological and surgical therapies, treatment of ischemic heart disease is still a demanding issue for medicine, because the results of the applied therapy are not always satisfactory. During a period of several past years, thanks to implementation of efficient medical approaches, such as timely revascularization or bypass grafting, the severity of MI decreased. Withal, in numerous cases relapsing cardiac events and subsequent progression of heart failure, likewise rejection of numerous patients from standard treatments because of disease complexity, are tremendous dilemma.

The main aim of this project is to investigate a role of macrophages in cardiac muscle in processes following ischemic heart injury. According to current state of art, there are several populations of such cells. Some of them are contributing to protection of myocardium, whereas others may excessively fuel inflammation and in consequence aggravate heart damage. However, subsets of local cardiac macrophages are not well described and characterized regarding their role in above mentioned processes. In this research project we will investigate how particular populations of these cells influence the development of inflammation after myocardial infarction, and how they contribute to remodeling and regeneration of heart muscle after ischemic injury. Particularly, we want to investigate how important for protective activities of cells of interest is the enzyme heme oxygenase-1 (HMOX1). Some literature data indicates, that HMOX1 plays a pivotal role in maintenance of cellular homeostasis, stimulation of growth, differentiation and preventing so called “programmed cell death”.

We are convinced, that the results which will be obtained in this project will allow us to better characterize different subsets of cardiac macrophages and better understand their role in processes following myocardial infarction: development and resolution of immune responses, as well as governing heart remodeling and regeneration. Earning better recognition of myocardial infarction pathology is inevitable for development of new methods of heart disease prevention and treatment based on regulation of inflammatory response in patients suffering from heart diseases.