Coronary artery disease is the most frequent cause of death in Europe among cardiovascular diseases. It is caused by atherosclerosis of the arteries that supply blood to cardiac muscle (so-called coronary arteries). Atherosclerotic plaque that narrows the artery prevents heart cells from receiving sufficient blood supply containing, among others, essential oxygen. This leads to ischemia of the heart muscle and in extreme cases to necrosis of heart tissue. Invasive treatment of this illness is based upon widening the artery using an expandable balloon and then implanting a miniature, metal spring (a stent) in the place of the narrowing. Even with the use of state-of-the-art stents, within a few months from the procedure subsequent narrowing of the artery (a restenosis) occurs in about 5-15% of cases. This happens due to an inadequate reaction of the organism to artery injury caused by expanding the balloon and implanting a foreign object – the stent. It also leads to a recurrence of cardiac ischemia and chest pain, which results in the necessity of carrying out a subsequent balloon angioplasty and either implanting another stent or performing coronary artery bypass grafting. Considering the fact that about 130 000 coronary artery stent implantations are performed in Poland every year, restenosis is associated with immense costs for the healthcare and a risk of complications for patients undergoing another, invasive procedure. To date, a few dozen risk factors of restenosis have been identified, which only vaguely allow to predict the risk of subsequent narrowing of the vessel in the area of stent implantation. Those factors include stent type, stent length, plasma concentration of certain proteins, and concomitant diseases, such as diabetes.

As previous studies - including our own - showed, certain predispositions to restenosis are genetically conditioned. Slight changes in DNA sequence, so-called single nucleotide polymorphisms, are responsible for the majority of genetic diversity in the population. They have a major impact on the risk of developing diseases such as neoplasms, coronary artery disease, and the organism's reaction to stent implantation analyzed by our team. Unfortunately, thus far only a small number of polymorphisms potentially significant in restenosis have been studied.

In our research we will analyze several polymorphisms of potential significance in the recurrence of coronary artery narrowing, which have not yet been studied in this context. We will assay the polymorphisms in full blood drawn from patients who underwent stent implantation in the Silesian Center for Heart Diseases and consented to give a few dozen milliliters of blood for research. Thus, we will investigate whether the presence of certain polymorphisms predisposes patients to restenosis and the necessity of subsequent invasive treatment of the same coronary artery.

We believe that our research will allow to discover new polymorphisms impacting the development of this complication. Because of that physicians will be able to more precisely predict the risk of its occurrence, which perhaps will simplify the decisions regarding alternative treatment in cases of patients with high restenosis risk. The results of our study will also lead to a better understanding of biological mechanisms involved in restenosis, thus increasing the chances of finding a way to reduce the incidence of in-stent restenosis.