Cancer is one of the greatest medical problems of nowadays world, affecting the increasing group of people every year. The disease can attack various body organs such as colon, lung and kidney. In the case of women, the most common cancer is breast cancer. However, this is not a homogeneous disease. Breast cancers can differ in e.g. the growth rate and response to therapy, and the basis for these differences are hidden inside the cells, i.e. they can be observed on the molecular level. One of the most aggressive breast cancers is so called triple negative breast cancer (TNBC). In this subtype three important factors used for treatment of other breast cancer subtypes – i.e. estrogen receptor, progesterone receptor and growth factor called HER2 – do not occur in cancer cells. Therefore, no effective, targeted therapy is available for this subtype of cancer and the usable one is a highly cytotoxic chemotherapy that is linked to dangerous side effects and usually does not eliminate the disease and allows the recurrence. Additionally, TNBC usually affects younger patients and is characterised by high aggressiveness, leading to poor prognosis.

Although we know some of the differences in the composition of triple-negative breast cancer cells and other subtypes, we still do not know what causes an extremely high aggressiveness of the disease. It is claimed that cancer cells can change from non-aggressive into aggressive, because of a process called epithelial-mesenchymal transition (EMT). It is a natural process, playing important roles in animals during embryonic development and wound healing. However, when the situation in the cell becomes pathological EMT can allow the cell to change. Cells undergoing EMT become motile and can live without support of other cells. Moreover, they become less differentiated than other cells of the organism.

EMT occurs due to the function of a number of factors present in the cells (proteins), from which one of the most important are the factors from the SNAIL family. SNAIL proteins are claimed to be the EMT driving factors and their excess is observed in many cancers. For example it was recognized that SNAIL proteins can lead to formation of aggressive breast cancer cells. Additionally, in the cell they regulate various processes, that lead to e.g. loosening of cell-cell matrix or changes in the metabolism. What is also interesting, such changes are also regulated by the so called SWI/SNF remodelling complex.

The SWI/SNF remodelling complex is a group of about 15 proteins that work together to regulate cell processes. This control is conducted on the epigenetic level. It means that the SWI/SNF complex allows or inhibits production of specific factors in the cell. These, subsequently, arrange themselves into specific pathways that affect how a cell behaves, what it is consisted of, what are the features of the cell and how it interacts with the tissue environment at the particular moment. The regulation performed by the SWI/SNF complex affects a wide range of processes and disruption of its function was observed in a few cancers e.g. colon cancer, breast cancer, pancreatic cancer, ovarian cancer or melanoma.

We hypothesise, that cancer's aggressiveness can be caused by cooperation of SWI/SNF complex and SNAIL family of proteins. In planned research we would like to check, whether these factors can directly interact with each other and influence each other's abundance in the cell. We suppose, that this interaction of SWI/SNF complex and SNAIL proteins can lead to tumour progression and metastasis, if any change in the level of the partners occur. In the research we will use the model of TNBC, a specific cancer cell line. We assume, that our research will lead to the better understanding of the molecular mechanisms that lead to cancer metastasis and in further perspective can allow discovery on the new therapy for TNBC patients.