The aim of the project is to evaluate a role of inflammation in progression of ductal breast cancer *in situ* (DCIS), a non-invasive type of breast cancer (BCa), to one of its most aggressive forms, the triple negative (ER-/PR-, HER2-), invasive ductal carcinoma (IDC). Investigation into a possible role of a molecular mechanism (NFκB/COX2→HIF1α signalling) activated by inflammation in DCIS→ IDC development will be carried out in an *in vitro* model of the disease. An inflammatory environment will be created by specific reagents (IL1β and TNFα cytokines) and its impact on biological function of cancer cells will be studied. Behaviour of cells in terms of degree of their aggressiveness (rate of growth, proliferative dominance among other tumour cells and ability to migrate) will be assessed in relation to the key cell characteristics known to be of a great clinical significance such as HER2 status.

In the final step of the study, we will use another *in vitro* system (more 'physiological'), where instead of synthetic reagents, human inflammatory cells (macrophages) will be used to create an inflammatory environment. This will enable to verify a biological significance of the obtained results. The proposed project aims to reveal one of the mechanisms likely to be responsible for the progression of DCIS to HER2-negative IDC. At present, there is no specific therapies for this type of BCa. It is foreseen that the results will not only further our knowledge of the pathophysiology of BCa but also might assist in identification of new therapeutic targets in this devastating disease.