In our project we head to determine if altered expression of group of genes consisting of host gene *MCM7* and three microRNAs that belong to the miR-106b-25 cluster causes intense proliferation, enhance migration and apoptosis resistance of kidney cancer cells and contributes to tumor progression. What is more, we want to identified group of genes that is involved in mentioned cellular processes and is commonly regulated by all three microRNAs of miR-106b-25 cluster. In our research we focus on the most common subtype of kidney cancer, called clear cell renal cell carcinoma (ccRCC) that accounts for 2-3% of all human cancers. ccRCC can be efficiently treated when tumor is detected at early stage of the disease. However, asymptotic course determines late diagnosis and makes treatment very difficult.

Insufficient treatment methods in the late stage of cancer clearly show that there is the urgent need to develop methods allowing for early disease detection. There is also a necessity to identify markers that could serve as a source of information about the progress of the disease and markers that would become the objective of new targeted therapies allowing to tailor treatment to the individual needs of particular patients.

Recently great promise is associated with the use of microRNA molecules in diagnostics. Those small RNA molecules regulate gene activity in cells by "switching on" or "switching off" target genes. Incorrect quantity of these molecules leads to an imbalance in the genes work through their incorrect adjustment.

The genes encoding the microRNAs can be placed on the chromosome in the close distance together within the host gene or as a single, independent unit. When they form a group, they can undergo common co-transcription and co-regulation, what suggest that they may be involved in the same processes. One example of such group is the host gene *MCM7* and three microRNA molecules: miR-25-3p, miR-93-5p, miR-106b localized within it. Studies carried out on other cancers of epithelial origin showed that increased level of these molecules cause intense proliferation, migration and apoptosis of tumor cells. What is more, by “switching off and on” those molecules the cellular processes can be controlled.

We assume that same situation occurs also in ccRCC. To the best of our knowledge this phenomena has not been studied so far in this type of cancer. We hope that our research will fulfill this gap in knowledge and will bring new insight into the disorders on the molecular level in the course of ccRCC. What is more, results of our proposal would be the basis for developing new diagnostic method in detection or even treatment of mentioned subtype of cancer.

To carry out our project we will use tissue samples derived from ccRCC patients. We will analyze expression of *MCM7*, miR-106b and genes commonly regulated by all three molecules from miR-106b-25 cluster. Altered expression of two others (namely, miR-25-3p and miR-93-5p) was demonstrated in our preliminary results. Then, by using ccRCC cells cultured in the laboratory, we will induce changes in quantities of all three microRNA molecules and *MCM7* gene to check how this affects the expression of miR-106b-25 targeted genes and cells proliferation, migration and apoptosis and whether it is tumor-stage dependent.

We believe that our proposal combines promises for using microRNA molecules as both, markers for the early disease detection (when ccRCC can be efficiently treated) and as potential therapeutic target. We propose that basic research of our project will head to expand the knowledge about the molecular disorders contributed to the ccRCC progression and will be the first step in developing new repair tools.