One of the most difficult problems of oncology is the resistance to treatment that limits the success of cancer therapy. Developing resistance to treatment causing tumor relapse may be a result of a change in cell phenotype due to Epithelial to Mesenchymal Transition (EMT). This process is a key element of metastasis in which epithelial cells acquire mesenchymal cell characteristics. Cells acquire features that facilitate their migration, invasion of surrounding tissues, and metastasis. The epitellial-mesenchymal transition process is also considered to be responsible for acquiring a stem cell phenotype that is insensitive to treatment and may be the source of possible tumor recurrence after therapy.

The results obtained by our team indicate that low level of MCPIP1 (Monocyte Chemotactic Protein-1 Induced Protein) in tumor cells correlates with the acquisition of mesenchymal cell features, tumor growth and lung metastasis. Therefore, the purpose of the project is to evaluate the role of MCPIP1 protein in the regulation of cell phenotype, its effect on tumor microenvironment, and the influence on resistance to antitumor therapy. The planned studies assume a comprehensive analysis of the behavior of the MCPIP1 protein during the EMT process and assess its impact on the level and location of the regulators of EMT. In our studies, we will examine whether the presence or the level of MCPIP1 protein is important for obtaining stem cell features, acquiring resistance to therapy and the impact on the microenvironment. We will also evaluate the role of MCPIP1 for the EMT process and the acquisition of therapy resistance in *in vivo* model. In our study, we will use mouse immunodeficient models and tissue specific animal models without the expression of the *Zc3h12a* gene coding MCPIP1. All experiments using animals and tissues from patients will be carried out according to the guidelines of the Bioethics Commission. In our research, we will use a number of techniques and methods of molecular and cellular biology and biochemistry.

The obtained results may help to better understand the role of MCPIP1 in tumor biology, show the mechanisms responsible for the EMT process, and resistance to treatment. Results from the project will be presented at national and international conferences on cancer biology and oncology. They will also be published in international journals with a high citation index. The results will be used in master's and doctoral dissertations conducted at the Department of General Biochemistry. The proposed research, according to our knowledge, is innovative on a global scale and has not been performed by any research team yet.