Autophagy is a process responsible for recycling the basic building blocks of cells (e.g., amino acids, nucleotides) by breaking down unwanted cellular elements, such as protein aggregates, damaged organelles or pathogens. This process is triggered when cell is under the influence of stress factors, for example, reduced oxygen concentration (hypoxia) or glucose deficiency. Therefore, it is a phenomenon that occurs in tumor cells, including colorectal cancer (CRC).

Autophagy in CRC has a dual role – it is attributed with properties that promote tumor development (e.g., through increased levels of LC3 protein) or inhibit tumor growth (e.g., by increasing sensitivity to chemotherapy through downregulated *ATG5* expression). The controversy over the effect of autophagy on CRC cells may be related to regulatory processes. Regulation of autophagy occurs at many levels, including gene transcription by transcription factors – for example estrogen receptor beta (ERβ). The link between ERβ and autophagy has already been demonstrated, e.g., in breast cancer, however, this issue in CRC is still unexplored. 17β-estradiol (E2), and its receptor in colonic epithelium, ERβ, have a protective role in CRC, mainly because of studies that showed a lower incidence of CRC among post-menopausal women receiving hormone replacement therapy compared to men of the same age and in animal studies proving suppressive role of ERβ in CRC.

Our preliminary studies support the hypothesis that there is a relationship between ERβ and autophagy-related genes in CRC. Our main research goal is to determine how ERβ affects the autophagy process in CRC. We intend to restore ERβ expression in CRC cell lines, and then induce autophagy in them by exposure to reduced oxygen concentration. We will also establish a functional characterization of the ERβ-mediated autophagy in CRC cells in 2D and 3D culture. Accurate selection of ERβ-dependent autophagy genes will be possible by using a variety of advanced methods, including next-generation sequencing.

Understanding ERβ-dependent autophagy in CRC will allow to: characterize the molecular mechanisms related to ERβ and autophagy in CRC cells, identify new biomarkers for the diagnostic process of patients with CRC, and recognize new proteins that may become targets in personalized medicine.