It is now recognized that adipose tissue is a multifunctional organ which secretes many hormones called adipokines. Studies focused on adipokines and cancer etiopathogenesis have shown that alterations in adipokines affect cell proliferation, apoptosis, tumour invasion, and angiogenesis. Several epidemiological studies have demonstrated a relationship between specific circulating adipocytokines and cancer risk. So far, only one adipokine – leptin has been comprehensively studied in relation to ovarian cancer. Our preliminary data shows that two adipokines: apelin, and chemerin are expressed in human epithelial ovarian cancer cells and granulosa cell tumors, moreover their expression is significantly higher in compare to non-cancer cells. We also demonstrate the stimulatory action of apelin and chemerin on ovarian cancer cell proliferation. A key role in the ovarian cancer pathogenesis plays also hormones and growth factors. 17β-estradiol and insulin-like growth factor (IGF-1) are well known stimulators of ovarian cancer progression. Moreover, potential crosstalk between IGF signaling and estrogen signaling may play important role in regulation of functions in the ovarian cancer cells. **These observations raised the hypothesis that: apelin and chemerin as an adipokines produced by ovarian cancer cells plays an important role in ovarian cancer progression. Thus, the main aim of this project is to recognize the molecular mechanism of apelin and chemerin in biology of ovarian cancer and identify whether apelin and chemerin may interact with 17β-estradiol and IGF-1 and regulate ovarian cancer progression.**

Although all cell types of the human ovary may undergo neoplastic transformation, the vast majority (85%) of tumors are derived from ovarian surface epithelium (OSE) and 5-10% from ovarian granulosa cells. For this reason, one human epithelial ovarian cancer cell line (OVCAR-3) and one human ovarian granulosa cell line (COV434) will be used as an *in vitro* model of ovarian cancers. In project, we intend to identify the effect of 17β-estradiol and IGF-1 on apelin and chemerin stimulated proliferation in epithelial cancer cell line (OVCAR-3) and granulosa tumor cell line (COV434). Next, we will investigate how apelin and chemerin effect on ERα, ERβ, GRP30 and IGF-1R expression. Finally, determine apelin and chemerin action on ovarian cancer cell apoptosis and whether 17β-estradiol or IGF-1 are involved in these actions. It is increasingly evident that three-dimensional (3D) cell culture models are better to provide cell-cell communication. Therefore due to improved structures that resemble *in vivo* architecture, we will perform all analysis in traditional two-dimensional (2D) monolayer cell culture and also in spheroid culture (3D).

Results of this project will add new information about the knowledge of apelin and chemerin in ovarian cancer. Molecular biology and biotechnology based methods allow us to better understand the role of these two adipokines on processes of proliferation and apoptosis in ovarian cancer. Characterization of the actions of apelin and chemerin with 17β-estradiol and IGF-1 will extend our knowledge about the biology of ovarian cancer. Moreover, these results offer a path for further research, which enable the development of new treatment strategies for this tumor type.