Epithelial ovarian cancer (EOC) is one of the main cause of death among all gynecological malignancies among women worldwide, taking a huge death toll each year. The increasing resistance to commonly used therapeutics, as well as enhanced invasive potential of ovarian cancer cells leads to high mortality rate, reaching over 50%. The simultaneous occurrence of both of these phenomenons is recognized as the main cause of many systemic therapies failure. In order to improve the situation, the intensive search for potential therapeutic targets and novel strategies of targeted therapies is being conducted worldwide. Many of these studies focus on the thorough investigation of interplay and co-relation between cancer cells chemoresistance and ability to create metastases. Lately, greater emphasis is put on genetic and epigenetic changes, which allows cancer cells to change their morphology, phenotype and gain the ability to migrate and create metastases. The most essential process in the whole "metastatic cascade" is epithelial to mesenchymal transition (EMT), as it grants cancer cells migration capabilities and allows for local invasion to surrounding extracellular matrix. EMT in turn is determined by several transcription factors, termed as "master regulators", among which the most essential are those belonging to SNAIL, TWIST and ZEB families.

In this regard the very interesting are SNAIL transcription factors, with the emphasis on SNAIL 1 and SNAIL 2, as they are not only the main EMT-inducing factors but can also perform additional roles, independently of EMT induction, such as immune modulation, regulation of cell cycle or preservation of stem cells capabilities. SNAIL-mediated induction of cell movement plays crucial role in many physiological processes, such as gastrulation, formation of mesoderm, tissue and organs generation or wound healing. While these functions are crucial for embryonic development, they may become fatal in pathological situations, such as tumor progression. It has been shown that expression of SNAIL 1/2 is enhanced in many types of cancers, including ovarian cancer and their level correlates with increased migration, invasion and metastasis of cancer cells. Moreover, since EMT provides a mechanism of escape to a new niche and ensures cell survival in conditions of stress, it has been suggested that SNAIL 1/2, as a major EMT-inducers, might also be involved in the development of ovarian cancer drug resistance. So far, sparse studies performed in this direction, points that in fact SNAIL 1/2 are involved in the development of ovarian cancer resistance to taxanes or platinum compounds. Still very little is known about the exact mechanisms of SNAIL 1/2 participation in ovarian cancer cells response to chemotherapeutic agents. Current findings expose the complexity of EMT-chemoresistance relationship and point at controversies regarding the role of SNAIL transcription factors in the regulation of these phenomenons.

In order to clarify SNAIL 1/2 implication in development of ovarian cancer cells chemoresistance in regard to their metastatic potential, the following questions should be answered in the first place:

1) **Are the EMT-inducing factors – SNAIL 1/2 crucial in acquiring and sustaining the ovarian cancer cells resistance to cisplatin?**

2) **What is the impact of STAT3 and AKT signaling proteins on SNAIL-mediated chemoresistance and invasiveness? Could they be used as potential therapeutical targets abolishing drug resistance and limiting metastatic potential of ovarian cancer cells *via* regulation of SNAIL 1/2 level and activity?**

3) **Can SNAIL 1/2 transcription factors be used as predictive factors, determining the patients' response to chemotherapy?**

The study is designed to bring answers to these questions by thorough investigation of SNAIL 1/2 level, activity and cellular localization in various ovarian cancer cell lines, as well as in EOC cells isolated from tumors and ascites fluid and primary EOC cell lines. The modulation of SNAIL 1/2 level will be performed either directly by their silencing (transfection), or indirectly by inhibiting the activity of STAT3 and AKT signaling proteins. What is more, the level of epithelial/mesenchymal phenotype markers, as well as metastatic capacity of ovarian cancer cells will be evaluated. Cells will be also treated with cisplatin and SNAIL 1/2 level and activity, as well as ovarian cancer cells metastatic capacity will be also measured after cisplatin treatment and analyzed in regard to cells' viability, apoptosis and proliferation.

Success in providing answer for above-mentioned questions will definitely systemize and broaden the knowledge regarding significance of SNAIL 1/2 in the development of ovarian cancer chemoresistance and simultaneous occurrence of invasiveness. This may bring very needed clues for developing novel therapies in the future.