

Cancer development and progression vary depending on tumor type, localization, invasion, immunogenicity and the ability of immune system to become activated. There are frequent interactions between tumor cells and immune cells, occurring locally at the site of primary tumor or distally through paracrine signalling of various mediators and cytokines.

The main subject of this PhD thesis is to study key factors and aspects of immune response in cancer patients.

In the first part, we analyzed immune cells infiltrating tumor tissues of ovarian cancer patients at different stages of disease. We focused on the dynamics of immune response, primarily on frequency of individual T lymphocyte populations in peripheral blood and tumor infiltrating T lymphocytes in tumors of early and advanced stages of ovarian cancer. We found that during disease progression there is a gradual decrease of proinflammatory Th17 and Th1 immune responses and a specific recruitment of regulatory T cells to the tumor site, which results in a significant immune suppression in the tumor microenvironment.

In the second part, we demonstrated that the character of immune response in HPV-positive head and neck cancer patients is very different from the patients with tumors not associated with HPV infection. In HPV-positive patients, significantly higher frequency of immune cells, predominantly CD8⁺ T cells, is observed in the tumor tissue. These cells seem to have a higher potential for activation as confirmed by IFN γ and IL-17 production. Furthermore, patients with HPV-positive tumors had significantly lower expression of Cox-2 mRNA and higher expression of PD1 mRNA compared to HPV-negative tumors.