Mucosal surface of the gut is in continuous contact with foreign compounds derived from diet as well as from commensal or pathogenic microorganisms. Thousands of years of symbiosis resulted in tight cooperation between the host and its microbiota. Microbiota composition and metabolism actively influence host's physiological as well as pathological processes. Chronic inflammation is characterized by prolonged active inflammatory response associated with tissue damage. This status results from accumulation of defects in various factors including gut barrier functions as well as mechanisms of innate and adaptive immunity. It's commonly accepted that chronic inflammatory diseases of the gastrointestinal tract like IBD, are associated with an increased risk of CAC development.

Two publications related to this thesis deal with modulatory effects of peroral administration of components of commensal bacteria or probiotics on intestinal inflammation. Using acute or chronic model of DSS-induced colitis, we demonstrated that oral treatment of BALB/c mice with membranous fraction of the commensal, Parabacteroides distasonis, as well as with lysate of probiotic bacterium Lactobacillus casei DN-114 001 significantly reduces the severity of intestinal inflammation. Moreover, the treatment was associated with reduction of local pro-inflammatory cytokines and with improvement of barrier function, and led to stabilization of the intestinal microbial community.

Sequentially, we studied the role of negative regulator of TLR signaling – IRAK-M and gut microbiota in the development of CAC and related molecular mechanisms. In the associated study, we investigated the role of the Wnt signaling pathway in gut epithelium homeostasis and during malignant transformation. We found that gut microbiota promotes tumorigenesis by increasing the exposure of gut epithelium to carcinogens and that negative regulation by IRAK-M is essential for colon cancer resistance.