Dynamic molecular interactions between the microbiota and the intestinal mucosa play an important role in the establishment and maintenance of mucosal homeostasis. Aberrant host-microbiota interaction could lead to many diseases such as inflammatory bowel disease. The aim of our study was to evaluate the commensal and probiotic bacteria activities and their ability to induce pathological or exert beneficial effects.

The most important trigger for immune system development is an exposure to microbial components. Here, we show that there is a time window at about three weeks of age, which enables the artificial colonization of germ free mice by a single oral dose of cecal content. The delayed colonization by either inoculation or co-housing causes permanent changes in immune system reactivity, which may downgrade the results of experiments performed on first generation of colonized animals.

In this thesis we report that even non-living commensal bacteria such as *Parabacteroides distasonis* (mPd) or well known probiotics such as *L. casei* DN-114 001 (Lc) possess anti-inflammatory effects in experimental model of colitis. The mechanisms that this effect is achieved by the lysate of *L. casei* DN-114 001 comprise: a) improvement in the gut barrier function, b) correction of the dysbiosis, and c) modulation of the mucosal immune response. Unlike the oral treatment with Lc, mPd leads to increase of specific antibodies in serum.

These complex immunomodulatory properties of bacterial lysates may lead to the development of new therapeutic approaches for treatment of chronic intestinal inflammation and also highlight the importance of individualizing and characterizing the potential capacity of bacteria as immunomodulatory agents. Moreover, oral administration of sterile bacteria, in contrast to living bacteria, may be safer in severely ill or immunocompromised patients.

Next, we demonstrated that metabolic activity of certain commensal microbes substantially influences the process of colitis associated cancer. We showed that antibiotic treatment changes the microbiota composition, and that this change is responsible for the beneficial effect on tumorigenesis.

Therefore, understanding this host-microbiota crosstalk could bring new strategies in therapy and prevention of specific disorders associated with intestinal dysbiosis and disruption of mucosal homeostasis.