Gastrointestinal (GI) microbes play important roles in the health and disease of the host. There are many documented evidences which demonstrated that disturbance of intestinal microbiota is linked to the risk of developing infectious, inflammatory and allergic diseases. Human intestine is home for a complex consortium of $10^{13}$-$10^{14}$ microbial cells. Interactions between the intestinal microbes, pathogens and host lead to exclusion of toxins (mycotoxins) and pathogens (colonization resistance), interference in disease progression as demonstrated in the prevention of oral infection, dental caries, diarrhoeas (Antibiotic Associated Diarrhoea, Travellers’ Diarrhoea and Rotavirus Diarrhoea), postoperative infection, respiratory infection and certain cancers. The group of beneficial intestinal microbes termed probiotics alter intestinal epithelial cell tight junction and immunological functions, via Toll-like receptor mediated cytokines. Probiotics are also involved in the digestion and transformation of dietary components, and energy metabolism. They promote intestinal peristaltic movement and modulate GI microbiota through competition for GI surface receptors and production of antimicrobial substances. Lately, laboratory and clinical studies demonstrated gut-brain axis communication and intestinal microbial (both pathogens and probiotics) modulation of host psycho-neuroimmunological functions, in relation to depression, anxiety and memory dysfunction, via regulation of hormone production and neurotransmission. These open up many possibilities for moderating intestinal microbiota as an approach in disease prevention and treatment. Composition and succession of the intestinal microbiota of people in various geographical regions have been characterized. Such perspective studies provide markers for the stage of health and positive guidance for microbial colonization through interference. Diet is a major factor in determining intestinal microbiota profile, health and diseases.

**Abstract**

**Selected Publications**


