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Fungal chitin treatment restores the anaerobic bacteria and reduces the intestinal inflammation in mice

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The gastrointestinal (GI) microbiota acts a natural barrier to colonization and proliferation of opportunistic pathogens, thereby decreasing the risk of intestinal infection and disease. Deregulation of the dynamic crosstalk between the microbiota, intestinal epithelial cells and immune cells is critically involved in the development of inflammatory bowel disease. Clinical and experimental studies have shown that either Candida albicans or Candida glabrata aggravates the intestinal inflammation-induced by dextran sulfate sodium (DSS) in mice, and, conversely, that DSS induced-colitis promotes the fungal colonization. C. glabrata is an opportunistic yeast pathogen that has adapted to colonize all segments of the human GI tract. The fungal cell wall is the predominant site of interaction between the fungus and its host. C. glabrata cell wall consists of a complex structure of polysaccharides, proteins, and lipids, but its composition is dynamic, responding to changes in the local environment. Expansion of the fungal wall during growth involves permanent remodeling of the cell wall polysaccharide network, which is comprised of three major types of polysaccharide: mannans, β-glucans, and chitin. Chitin is a homopolymer of β1,4-N-acetylglucosamine (GlcNAc) and is essential for biological functions in fungi, including cell division, forming the primary septum of all septa, hyphal growth, and virulence. Deregulation of chitin biosynthesis is a potential mechanism of virulence and resistance to antifungal treatments.In the present study, we investigated the impact of C. glabrata colonization on the diversity of the gut microbiota in a DSS-induced colitis model, and assessed how the C. glabrata cell wall is remodeled in order to persist in the gut environment. We also analyzed the effect of fungal chitin treatment on C. glabrata-host interactions in the DSS mouse model. This study provides evidence that inflammation of the gut alters the microbial balance and leads to C. glabrata cell wall remodeling through an increase in chitin, which is involved in promoting persistence of C. glabrata in the gut while the oral administration of chitin to mice reduced the overgrowth of aerobic bacteria and C. glabrata as well the production of inflammatory parameters through stimulation of intestinal receptors.

Biography

Samir Jawhara is a researcher in Lille University Hospital, France (Inserm U995/2). He was a research fellow in Cleveland Clinic (Cleveland, OH), before returning to Lille University Hospital in 2012. He received his PhD in immunology-microbiology in 2006 from Lille University. The major research interests of his lab are related to host-yeast interactions in the intestine and their impact on intestinal inflammation. He is particularly interested in defining the functions of pattern recognition receptors (PRR), such as mannose binding lectin (MBL), Galectin-3, and Toll-like receptors in the intestine during disease. They also interested in how the human pathogenic yeast Candida albicans may manipulate host innate immune pathways during the intestinal inflammation to cause disease.

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