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The Effects of *F. prausnitzii* on *Candida albicans* Growth and Pathogenicity and Related Mechanisms

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Abstract

Inflammatory bowel disease (IBD), including ulcerative colitis (UC) and crohn's disease (CD), is characterized by chronic relapsing inflammatory disorder of the gastrointestinal tract. The pathogenesis of the disease is complex. It is believed that genetic, environmental, intestinal microbes and immune factors are involved in the occurrence and development of IBD, especially the intestinal flora disorder has become a research hotspot in recent years.

The reduction of *Faecalibacterium prausnitzii* (*F. prausnitzii*) and the increase of the opportunistic pathogen *Candida albicans* are one of the important features of intestinal flora disorder in patients with IBD.

Many studies have shown that *Faecalibacterium prausnitzii* and its supernatant can improve intestinal inflammation, regulate dysbacteriosis, maintain the homeostasis of the intestinal microenvironment, etc. *C. albicans* (CA) are about 65% of the total fungi in the gastrointestinal tract. The mycelial phase of *Candida albicans* has strong pathogenic ability. Hydrolytic enzyme production capacities (Ep, Sap, and Ha) such as extracellular phospholipase (Ep), secreted aspartyl proteinase (Sap) and hemolytic factor (Haemolysin, Ha) further enhance their pathogenicity.

Intestinal bacteria and fungi influence each other in many ways, such through physical interactions, chemical exchanges, changes in the environment, or alteration of the host immune response to impact each other's survival or virulence.

Keywords: *Inflammatory Bowel Disease; Faecalibacterium prausnitzii; Candida albicans; Antimicrobial Peptides; Inflammasomes*

Aim of the Study

F. prausnitzii and its supernatant were co-cultured with *Candida albicans in vitro* to observe the effects of *F. prausnitzii* and its supernatant on *Candida albicans* growth, hyphal and hydrolytic enzyme production capacities. To investigate whether *F. prausnitzii* and its supernatant can inhibit the growth and pathogenic ability of *Candida albicans* by stimulating the expression of inflammasomes (NLRP6) and antimicrobial peptides (LL-37/BD-2/BD-3) and tight junction proteins (ZO-1, occludin) in intestinal epithelial cell.

Methods

1. *F. prausnitzii* and its supernatant were co-cultured with CA under anaerobic environment *in vitro* for 2h, 4h, 6h, 8h, 10h.
2. The YPD solid plate was used to observe the growth and reproduction ability of CA.

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3. RT-PCR analysis of CA Hyphae-Specific Genes (BCR1, CDC24b, ECE1, HGC1, HWP1 and EFG1) mRNA expression, Scanning electron microscopic observation of the production of CA hyphae, the activity of CA hydrolytic enzyme production capacities (Ep, Sap, and Ha) were detected using the specific plates.
4. *F. prausnitzii* and its supernatant were co-cultured with IEC for 6h, then join CA to continue co-cultivation for 6h. Western blot analysis of expression levels of inflammasomes and antimicrobial peptides (BD-2, BD-3, LL-37) and tight junction proteins (occludin, zo) in IEC the level of expression.

Results

1. It was found that the amount of *C. albicans* with *F. prausnitzii* and its supernatant co-cultured were significantly decreased compared with the control group by plate counting method.
2. It was found that the expressions of Hyphae-Specific Genes (BCR1, CDC24b, ECE1, HGC1, HWP1 and EFG1) and production of hyphal phase CA were inhibited and hydrolytic enzyme production capacities (Ep, Sap, and Ha) were decreased when *F. prausnitzii* and its supernatant were co-cultured with CA.
3. Western blot analysis showed that *F. prausnitzii* and its supernatant may inhibit the growth of CA by stimulating IEC secretion of NLRP6, ASC, IL-1β/IL-18 and antibacterial peptides BD-2 and BD-3, but caspase-1 and LL-37 had no significant effect.
4. Western blot analysis showed that *F. prausnitzii* and its supernatant may enhance the intestinal mucosal barrier function by promoting the expression of IEC tight junction protein (occludin, zo-1).

Conclusion

F. prausnitzii and its supernatant have inhibitory effects on CA. The direct effects are as follows: inhibiting CA growth and reproduction, inhibiting the expression of CA Hyphae-Specific Genes (BCR1, CDC24b, ECE1, HGC1, HWP1 and EFG1), reducing production of hyphal phase CA, reducing the production of CA hydrolytic enzyme production capacities (Ep, Sap, and Ha). The indirect effect is by stimulating the expression of NLRP6, ASC, IL-1β/IL-18, BD-2, BD-3 and the tight junction proteins occludin and ZO-1 in IEC to inhibition the growth of CA.

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