

14<sup>th</sup> International Conference on **Microbial Interactions & Microbial Ecology**  
 &  
 11<sup>th</sup> Edition of International Conference on **Advances in Microbiology and Public Health**  
 August 19-20, 2019 Vienna, Austria

## Enterocyte tight junctions as a target for *Candida albicans* translocation through the gut barrier

Fabienne Bon<sup>1</sup>, Tracy Paradis<sup>1</sup>, Alicia Loiselet<sup>1</sup>, Amandine Ducreux<sup>1</sup>, Christophe D'Enfert<sup>2</sup> and Frederic Dalle<sup>1,3</sup>

<sup>1</sup>UMR A PAM-University Bourgogne-Franche-Comte, France

<sup>2</sup>Institut Pasteur, France

<sup>3</sup>Centre Hospitalier Universitaire, France

Formerly a commensal resident of the gut microbiota in healthy humans, *Candida albicans* is an opportunistic pathogen causing infections ranging from superficial to the more life-threatening disseminated infections, especially in the ever-growing population of vulnerable patients in hospital settings. Because overwhelming evidences suggest that the gastrointestinal tract is the main source of disseminated *C. albicans* infections, deciphering the cellular and molecular mechanisms of the interaction of *C. albicans* with enterocytes is necessary to better understand the switch from commensalism to pathogenicity of this fungus and to improve the management of disseminated candidiasis. Different routes of the gut barrier translocation by *C. albicans* have been reported, including transcytosis through epithelial cells and the paracellular route where the fungus can cross the gut barrier without damaging the tissue barrier. The intestinal epithelium indeed consists in a monolayer of enterocytes where adjacent cells are jointed each other by cell junctions. Among those, tight junctions ensure integrity and impermeability of the intestinal barrier, limiting invasion of the gut barrier by *Candida albicans*. We propose to investigate the modulation of tight junction integrity during *Candida albicans* interaction with enterocytes and specify the mechanisms allowing the fungus to cross the gut barrier through the paracellular route.

1. Znaidi S, van Wijlick L, Hernández-Cervantes A, Sertour N, Desseyn J L, Vincent F, Atanassova R, Gouyer V, Munro C A, Bachellier-Bassi S, Dalle F, Jouault T, Bougnoux M E and d'Enfert C (2018) Systematic gene overexpression in *Candida albicans* identifies a regulator of early adaptation to the mammalian gut. *Cell Microbiol.* 20(11):e12890.
2. Albac S, Schmitz A, Lopez-Alayon C, d'Enfert C, Sautour M, Ducreux A, Labruère-Chazal C, Laue M, Holland G, Bonnin A and Dalle F (2016) *Candida albicans* is able to use M cells as a portal of entry across the intestinal barrier in vitro. *Cell Microbiol.* 18(2):195-210.
3. Bohringer M, Pohlers S, Schulze S, Albrecht-Eckardt D, Piegsa J, Weber M, Martin R, Hünninger K, Linde J, Guthke R and Kurzai O (2016) *Candida albicans* infection leads to barrier breakdown and a MAPK/NF-κB mediated stress response in the intestinal epithelial cell line C2BBel. *Cell Microbiol.* 18(7):889-904.
4. Dalle F, Wächtler B, L'Ollivier C, Holland G, Bannert N, Wilson D, Labruère C, Bonnin A and Hube B (2010) Cellular interactions of *Candida albicans* with human oral epithelial cells and enterocytes. *Cell Microbiol.* 12(2):248-71.

### Biography

Bon Fabienne is a Research Teacher at the University of Burgundy. Her work aims to improve knowledge on the pathophysiology of *Candida albicans* and more specifically on the interaction of this agent with enterocyte cells. The objective is to understand the cellular and molecular mechanisms involved in the passage of *Candida albicans* from commensalism to the pathogenic state.

fabienne.bon@iut-dijon.u-bourgogne.fr