

From attachment to damage: defined genes of *Candida albicans* mediate adhesion, invasion and damage during interaction with oral epithelial cells.

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Abstract

Candida albicans frequently causes superficial infections by invading and damaging epithelial cells, but may also cause systemic infections by penetrating through epithelial barriers. *C. albicans* is an unusual pathogen because it can invade epithelial cells via two distinct mechanisms: induced endocytosis, analogous to facultative intracellular enteropathogenic bacteria, and active penetration, similar to plant pathogenic fungi. Here we investigated the molecular basis of *C. albicans* epithelial interactions. By systematically assessing the contributions of defined fungal pathways and factors to different stages of epithelial interactions, we provide an expansive portrait of the processes and activities involved in epithelial infection. We strengthen the concept that hyphal formation is critical for epithelial invasion. Importantly, our data support a model whereby initial epithelial invasion per se does not elicit host damage, but that *C. albicans* relies on a

combination of contact-sensing, directed hyphal extension, active penetration and the expression of novel pathogenicity factors for further inter-epithelial invasion, dissemination and ultimate damage of host cells. Finally, we explore the transcriptional landscape of *C. albicans* during the early stages of epithelial interaction, and, via genetic analysis, identify ICL1 and PGA34 as novel oral epithelial pathogenicity factors.

Involved units

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