Distinct roles of *Candida albicans*-specific genes in host-pathogen interactions.

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Details



Abstract

Human fungal pathogens are distributed throughout their kingdom, suggesting that pathogenic potential evolved independently. Candida albicans is the most virulent member of the CUG clade of yeasts and a common cause of both superficial and invasive infections. We therefore hypothesised that C. albicans possesses distinct pathogenicity mechanisms. In silico genome subtraction and comparative transcriptional analysis identified a total of 65 C. albicans specific genes (ASGs) expressed during infection. Phenotypic characterisation of six ASG-null mutants demonstrated that these genes are dispensable for in vitro growth, but play defined roles in host pathogen interactions. Based on these analyses, we investigated two ASGs in greater detail. An orf19.6688Δ mutant was found to be fully virulent in a mouse model of disseminated candidiasis and to induce higher levels of the proinflammatory cytokine, IL-1β, following incubation with murine macrophages. A pga16Δ mutant, on the other hand exhibited attenuated virulence. Moreover we provide evidence that secondary filamentation events (multiple hyphae emerging from a mother cell and hyphal branching) contribute to pathogenicity: PGA16 deletion did not influence primary hypha formation or extension following contact with epithelial cells; however, multiple hyphae and

hyphal branching were strongly reduced. Significantly, these hyphae failed to damage host cells as effectively as the multiple hyphae structures formed by wild type C. albicans cells. Together, our data show that species-specific genes of a eukaryotic pathogen can play important roles in pathogenicity.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Mikrobielle Immunologie Ilse Jacobsen Mehr erfahren

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