Granulocytes govern the transcriptional response, morphology and proliferation of *Candida albicans* in human blood.

Fradin C, De Groot P, MacCallum D, Schaller M, Klis F, Odds FC, Hube B (2005) Granulocytes govern the transcriptional response, morphology and proliferation of *Candida albicans* in human blood. *Mol Microbiol* 56(2), 397-415.

Details



Abstract

Survival in blood and escape from blood vessels into tissues are essential steps for the yeast Candida albicans to cause systemic infections. To elucidate the influence of blood components on fungal growth, morphology and transcript profile during bloodstream infections, we exposed C. albicans to blood, blood fractions enriched in erythrocytes, polymorphonuclear or mononuclear leukocytes, blood depleted of neutrophils and plasma. C. albicans cells exposed to erythrocytes, mononuclear cells, plasma or blood lacking neutrophils were physiologically active and rapidly switched to filamentous growth. In contrast, the presence of neutrophils arrested C. albicans growth, enhanced the fungal response to overcome nitrogen and carbohydrate starvation, and induced the expression of a large number of genes involved in the oxidative stress response. In particular, SOD5, encoding a glycosylphosphatidylinositol (GPI)-anchored superoxide dismutase

localized on the cell surface of C. albicans, was strongly expressed in yeast cells that were associated with neutrophils. Mutants lacking key genes involved in oxidative stress, morphology or virulence had significantly reduced survival rates in blood and the neutrophil fraction, but remained viable for at least 1 h of incubation when exposed to erythrocytes, mononuclear cells, plasma or blood lacking neutrophils. These data suggest that C. albicans genes expressed in blood were predominantly induced in response to neutrophils, and that neutrophils play a key role during C. albicans bloodstream infections. However, C. albicans is equipped with several genes and transcriptional programmes, which may help the fungus to counteract the attack of neutrophils, to escape from the bloodstream and to cause systemic infections.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Leibniz-HKI-Autor*innen



Bernhard Hube

Details

Awards

Number of citations (Google scholar): 240; Selected as “recommended” Factor 3.0 by Faculty of 1000, “of special interest” and “of outstanding interest” (3x) by Curr Opin Microbiol and as “exceptional presentation” in Nat Rev Microbiol

Identifier

PMID: 15813733