Induction of ERK-kinase signalling triggers morphotype-specific killing of *Candida albicans* filaments by human neutrophils.

Wozniok I, Hornbach A, Schmitt C, Frosch M, Einsele H, Hube B, Löffler J, Kurzai O (2008) Induction of ERK-kinase signalling triggers morphotype-specific killing of *Candida albicans* filaments by human neutrophils. *Cell Microbiol* 10(3), 807-820.

Details

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Abstract

Candida albicans is among the most important fungal pathogens in humans. Morphological plasticity has been linked to its pathogenic potential as filamentous forms are associated with tissue invasion and infection. Here we show that human neutrophils discriminate between yeasts and filaments of C. albicans. Whereas filaments induced targeted motility, resulting in the establishment of close contact between neutrophils and fungal cells, yeast forms were largely ignored during coincubation. In transwell assays, C. albicans filaments induced significantly higher migratory activity in neutrophils than yeasts. Neutrophil motility based on actin rearrangement was essential for killing of C. albicans filaments but not involved in killing of yeast forms. Using inhibitors for MAP-kinase cascades, it was shown that recognition of C. albicans filaments by neutrophils is mediated via the MEK/ERK cascade and independent of JNK or p38 activation. Inhibition of the ERK signalling pathway abolished neutrophil migration induced by C. albicans filaments and selectively impaired the ability to kill this morphotype. These data show that invasive

filamentous forms of C. albicans trigger a morphotype-specific activation of neutrophils, which is strongly dependent on neutrophil motility. Therefore, human neutrophils are capable of sensing C. albicans invasion and initiating an appropriate early immune response.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Fungal Septomics Oliver Kurzai Mehr erfahren

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