

Role of pH-regulated antigen 1 of *Candida albicans* in the fungal recognition and antifungal response of human neutrophils.

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Abstract

Candida albicans is an opportunistic human-pathogenic fungus, which can cause superficial but also life-threatening invasive infections. The pH-regulated antigen 1 (Pra1) of *C. albicans* is a surface-associated and secreted protein highly expressed in the hyphal form. Pra1 can bind to complement receptor 3 (CD11b/CD18) and can mediate adhesion to and migration of human phagocytes. Here, we investigated the role of Pra1 in the activation of human neutrophils. A *C. albicans* mutant strain lacking Pra1 (*pra1*Δ) supported neutrophil migration to a lower extent than did the parental wild-type strain. A Pra1-overexpressing *C. albicans* strain enhanced neutrophil migration and adherence. While inactivated hyphae of the Pra1-overexpressing mutant with surface-associated Pra1 enhanced the production and release of reactive oxygen species, myeloperoxidase, lactoferrin, and interleukin 8 by neutrophils, such responses were reduced when stimulated with inactivated hyphae of the *pra1*Δ strain. However, Pra1-overexpressing living hyphae, which secrete large amounts of Pra1, also caused a reduced neutrophil activation,

indicating that the release of extracellular Pra1 can inhibit the activation of these innate immune cells. Similarly, soluble recombinant Pra1 inhibited the neutrophil responses elicited by cell-wall bound Pra1. Finally, fungal cells lacking Pra1 were more efficiently killed by neutrophils. In conclusion, surface-exposed Pra1 plays a role in the recognition of *C. albicans*, especially hyphal cells, by human neutrophils and enhances neutrophil antimicrobial responses. However, the fungus can counteract some of these defense mechanisms by releasing soluble Pra1.

Beteiligte Forschungseinheiten

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