

The glycosylphosphatidylinositol-anchored protease Sap9 modulates the interaction of *Candida albicans* with human neutrophils.

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Abstract

Human polymorphonuclear neutrophils (PMNs) play a major role in the immune defense against invasive *Candida albicans* infection. This fungal pathogen produces a set of aspartic proteases that directly contributes to virulence properties such as adhesion, tissue invasion, and immune evasion. We show here that, in contrast to other secreted proteases, the cell surface-associated isoform Sap9 has a major impact on the recognition of *C. albicans* by PMNs. SAP9 is required for the induction of PMN chemotaxis toward *C. albicans* filaments, an essential prerequisite of effective PMN activation. Furthermore, deletion of SAP9 leads to a mitigated release of reactive oxygen intermediates (ROI) in human PMNs and decreases *C. albicans*-induced apoptosis triggered by ROI formation. In confrontation assays, killing of a SAP9 deletion mutant is reduced in comparison to wild-type *C. albicans*. These data clearly implicate Sap9 protease activity in the initiation of protective innate immunity and suggest novel molecular mechanisms in *C. albicans*-host interaction leading to neutrophil activation.

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