

The dual Function of the fungal toxin candidalysin during *Candida albicans*-macrophage interaction and virulence.

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Abstract

The dimorphic fungus *Candida albicans* is both a harmless commensal organism on mucosal surfaces and an opportunistic pathogen. Under certain predisposing conditions, the fungus can overgrow the mucosal microbiome and cause both superficial and life-threatening systemic infections after gaining access to the bloodstream. As the first line of defense of the innate immune response, infecting *C. albicans* cells face macrophages, which mediate the clearance of invading fungi by intracellular killing. However, the fungus has evolved sophisticated strategies to counteract macrophage antimicrobial activities and thus evade immune surveillance. The cytolytic peptide toxin, candidalysin, contributes to this fungal defense machinery by damaging immune cell membranes, providing an escape route from the hostile phagosome environment. Nevertheless, candidalysin also induces NLRP3 inflammasome activation, leading to an increased host-protective pro-inflammatory response in mononuclear phagocytes. Therefore, candidalysin

facilitates immune evasion by acting as a classical virulence factor but also contributes to an antifungal immune response, serving as an avirulence factor. In this review, we discuss the role of candidalysin during *C. albicans* infections, focusing on its implications during *C. albicans*-macrophage interactions.

Involved units

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