Fungal biotin homeostasis is essential for immune evasion after macrophage phagocytosis and virulence.

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Details

Abstract

Biotin is an important cofactor for multiple enzymes in central metabolic processes. While many bacteria and most fungi are able to synthesize biotin de novo, Candida spp. are auxotrophic for this vitamin and thus require efficient uptake systems to facilitate biotin acquisition during infection. Here we show that C. glabrata and C. albicans use a largely conserved system for biotin uptake and regulation, consisting of the high-affinity biotin transporter Vht1 and the transcription factor Vhr1. Both species induce expression of biotin-metabolic genes upon in vitro biotin depletion and following phagocytosis by macrophages, indicating low biotin levels in the Candida-containing phagosome. In line with this, we observed reduced intracellular proliferation of both Candida cells pre-starved of biotin and deletion mutants lacking VHR1 or VHT1 genes. VHT1 was essential for the full virulence of C. albicans during systemic mouse infections, and the lack of VHT1 led to

reduced fungal burden in C. glabrata-infected brains and C. albicans-infected brains and kidneys. Together, our data suggest a critical role of Vht1-mediated biotin acquisition for C. glabrata and C. albicans during intracellular growth in macrophages and systemic infections.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Mikrobielle Immunologie Ilse Jacobsen Mehr erfahren

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