

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

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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. This article is protected by copyright. All rights reserved.

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Leibniz-HKI-Authors



Stefanie Allert

[Details](#)



Katja Graf

[Details](#)



Teresa Sofie Hartung

[Details](#)



Bernhard Hube

[Details](#)



Ilse Denise Jacobsen

[Details](#)



Lydia Kasper

[Details](#)



Maria Joanna Niemiec

[Details](#)



Marcel Sprenger

[Details](#)



Stephanie Wisgott

[Details](#)

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