

# 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.

## Involved units

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## **Topics**

[Interactions with immune cells \(MPM\)](#)

## **Awards**

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