

The fungal pathogen *Candida glabrata* does not depend on surface ferric reductases for iron acquisition.

Gerwien F, Safyan A, Wisgott S, Brunke S, Kasper L, Hube B (2017) The fungal pathogen *Candida glabrata* does not depend on surface ferric reductases for iron acquisition. *Front Microbiol* 8, 1055.

[Details](#)



Abstract

Iron acquisition is a crucial virulence determinant for many bacteria and fungi, including the opportunistic fungal pathogens *Candida albicans* and *C. glabrata*. While the diverse strategies used by *C. albicans* for obtaining iron from the host are well-described, much less is known about the acquisition of this micronutrient from host sources by *C. glabrata* - a distant relative of *C. albicans* with closer evolutionary ties to *Saccharomyces cerevisiae*, which nonetheless causes severe clinical symptoms in humans. Here we show that *C. glabrata* is much more restricted than *C. albicans* in using host iron sources, lacking, for example, the ability to grow on transferrin and hemin/hemoglobin. Instead, *C. glabrata* is able to use ferritin and non-protein-bound iron (FeCl_3) as iron sources in a pH-dependent manner. As in other fungal pathogens, iron-dependent growth requires the reductive high affinity (HA) iron uptake system. Typically highly conserved, this uptake mechanism normally relies on initial ferric reduction by cell-surface ferric reductases. The *C. glabrata* genome contains only three such putative ferric reductases, which were found to be

dispensable for iron-dependent growth. In addition and in contrast to *C. albicans* and *S. cerevisiae*, we also detected no surface ferric reductase activity in *C. glabrata*. Instead, extracellular ferric reduction was found in this and the two other fungal species, which was largely dependent on an excreted low-molecular weight, non-protein ferric reductant. We therefore propose an iron acquisition strategy of *C. glabrata* which differs from other pathogenic fungi, such as *C. albicans*, in that it depends on a limited set of host iron sources and that it lacks the need for surface ferric reductases. Extracellular ferric reduction by a secreted molecule possibly compensates for the loss of surface ferric reductase activity in the HA iron uptake system.

Involved units

[Microbial Pathogenicity Mechanisms Bernhard Hube](#) [Read more](#)

Leibniz-HKI-Authors



Sascha Brunke

[Details](#)



Franziska Gerwien

[Details](#)



Bernhard Hube

[Details](#)



Lydia Kasper

[Details](#)



Abu Safyan

[Details](#)



Stephanie Wisgott

[Details](#)

Topics

[Nutrient acquisition in infections](#)

Identifier

doi: 10.3389/fmicb.2017.01055

PMID: 28642757