A TRP1-marker-based system for gene complementation, overexpression, reporter gene expression, and gene modification in *Candida glabrata*.

Sprenger M, Brunke S, Hube B, Kasper L (2021) A TRP1-marker-based system for gene complementation, overexpression, reporter gene expression, and gene modification in *Candida glabrata*. *FEMS Yeast Res* 20(8), foaa066.

Details

Abstract

Although less prevalent than its relative Candida albicans, the yeast Candida glabrata is a successful pathogen of humans which causes life-threatening candidiasis. It is thus vital to understand the pathogenicity mechanisms and contributing genes in C. glabrata. However, gene complementation as a tool for restoring the function of a previously deleted gene is not standardized in C. glabrata, and it is less frequently used than in C. albicans. In this study, we established a gene complementation strategy using genomic integration at the TRP1 locus. We prove that our approach can not only be used for integration of complementation cassettes, but also for overexpression of markers like fluorescent proteins and the antigen ovalbumin, or of potential pathogenicity-related factors like the biotin transporter gene VHT1. With urea amidolyase

Dur1,2 as an example, we demonstrate the application of the gene complementation approach for the expression of sequence-modified genes. With this approach we found that a lysine-to-arginine mutation in the biotinylation motif of Dur1,2 impairs urea-dependent growth of C. glabrata and C. albicans. Taken together, the TRP1-based gene complementation approach is a valuable tool for investigating novel gene functions and for elucidating their role in the pathobiology of C. glabrata.

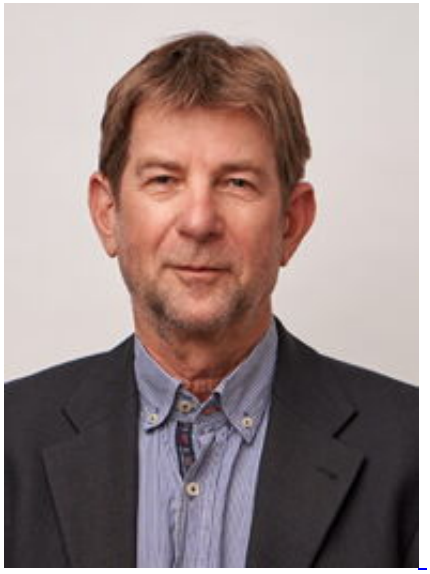
Involved units

Microbial Pathogenicity Mechanisms Bernhard Hube Read more

Leibniz-HKI-Authors

Sascha Brunke

Details



Bernhard Hube

Details

Lydia Kasper

Details

Marcel Sprenger

Details

Topics

Damage to the host

Evolution & adaptation in pathogenicity

Identifier

doi: 10.1093/femsyr/foaa066

PMID: 33289831