

Systematic phenotyping of a large-scale *Candida glabrata* deletion collection reveals novel antifungal tolerance genes.

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Abstract

The opportunistic fungal pathogen *Candida glabrata* is a frequent cause of candidiasis, causing infections ranging from superficial to life-threatening disseminated disease. The inherent tolerance of *C. glabrata* to azole drugs makes this pathogen a serious clinical threat. To identify novel genes implicated in antifungal drug tolerance, we have constructed a large-scale *C. glabrata* deletion library consisting of 619 unique, individually bar-coded mutant strains, each lacking one specific gene, all together representing almost 12% of the genome. Functional analysis of this library in a series of phenotypic and fitness assays identified numerous genes required for growth of *C.*

glabrata under normal or specific stress conditions, as well as a number of novel genes involved in tolerance to clinically important antifungal drugs such as azoles and echinocandins. We identified 38 deletion strains displaying strongly increased susceptibility to caspofungin, 28 of which encoding proteins that have not previously been linked to echinocandin tolerance. Our results demonstrate the potential of the *C. glabrata* mutant collection as a valuable resource in functional genomics studies of this important fungal pathogen of humans, and to facilitate the identification of putative novel antifungal drug target and virulence genes.

Beteiligte Forschungseinheiten

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