Competition of *Candida glabrata* against *Lactobacillus* is Hog1 dependent.

Beyer R, Jandric Z, Zutz C, Gregori C, Willinger B, Jacobsen ID, Kovarik P, Strauss J, Schüller C (2018) Competition of *Candida glabrata* against *Lactobacillus* is Hog1 dependent. *Cell Microbiol* 20(12), e12943.

Abstract

Candida glabrata is a common human fungal commensal and opportunistic pathogen. This fungus shows remarkable resilience as it can form recalcitrant biofilms on indwelling catheters, has intrinsic resistance against azole antifungals and is causing vulvo-vaginal candidiasis. As a nosocomial pathogen, it can cause life-threatening bloodstream infections in immune-compromised patients. Here, we investigate the potential role of the high osmolarity glycerol response (HOG) MAP kinase pathway for C. glabrata virulence. The C. glabrata MAP kinase CgHog1 becomes activated by a variety of environmental stress conditions such as osmotic stress, low pH and carboxylic acids and subsequently accumulates in the nucleus. We found that CgHog1 allows C. glabrata to persist within murine macrophages but it is not required for systemic infection in a mouse model. C.glabrata and Lactobacilli co-colonize mucosal surfaces. Lactic acid at a concentration produced by vaginal Lactobacillus spp. causes CgHog1 phosphorylation and accumulation in the nucleus. In addition, CgHog1 enables C. glabrata to tolerate different

Lactobacillus spp. and their metabolites when grown in co-culture. Using a phenotypic diverse set of clinical C. glabrata isolates, we find that the HOG pathway is likely the main quantitative determinant of lactic acid stress resistance. Taken together, our data indicate that CgHog1 has an important role in the confrontation of C. glabrata with the common vaginal flora.

Beteiligte Forschungseinheiten

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