A family of glutathione peroxidases contributes to oxidative stress resistance in *Candida albicans*.

Miramón P, Dunker C, Kasper L, Jacobsen ID, Barz D, Kurzai O, Hube B (2014) A family of glutathione peroxidases contributes to oxidative stress resistance in *Candida albicans*. *Med Mycol* 52(3), 223-239. PubMed

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Projects

The role of filamentation in the pathogenesis of candidiasis
Details

Abstract

Candida albicans is a well-adapted human commensal but is also a facultative pathogen that can cause superficial and systemic infections. Its remarkable capacity to thrive within the human host relies on its ability to adapt and respond to the local environment of different niches. *C. albicans* is able to cope with oxidative stress in a coordinated fashion via upregulation of different protective mechanisms. Here, we unravel the role of a family of glutathione peroxidase (GPx), designated Gpx31, Gpx32, and Gpx33, in oxidative stress resistance. We show that GPx activity in *C. albicans* is induced upon exposure to peroxides and that this enzymatic activity is required for full resistance to oxidative stress. The GPx activity relies on the presence of GPX31, with no apparent contribution from GPX32 and GPX33 during in vitro short-term (3 h) exposure to peroxides. However, a triple gpx31-33Δ/Δ mutant exhibited a more pronounced sensitivity than a single gpx31Δ/Δ mutant on solid media in the presence of oxidants, suggesting that GPX32 and GPX33 may be involved in long-term adaptation to oxidative stress. Interestingly, reintegration of a single allele of GPX31 was sufficient to restore the wild-type phenotype in both the single and triple mutants. We found that mutants lacking GPX31-33 were more susceptible to killing by phagocytic cells, suggesting that GPxs are required for full resistance to innate immune effector cells. Despite the sensitivity to oxidative stress and phagocytes, these mutants were not affected in their virulence in the chicken embryo model of candidiasis.

Identifier

doi: 10.1093/mmy/myt021 PMID: 24625675