Cooperative role of MAPK pathways in the interaction of *Candida albicans* with the host Epithelium.

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Abstract

Candida albicans is an important human fungal pathogen responsible for tens of millions of infections as well as hundreds of thousands of severe life-threatening infections each year. MAP kinase (MAPK) signal transduction pathways facilitate the sensing and adaptation to external stimuli and control the expression of key virulence factors such as the yeast-to-hypha transition, the biogenesis of the cell wall, and the interaction with the host. In the present study, we have combined molecular approaches and infection biology to analyse the role of C. albicans MAPK pathways during an epithelial invasion. Hog1 was found to be important for adhesion to abiotic surfaces but was dispensable for damage to epithelial cells. The Mkc1 cell wall integrity (CWI) and Cek1 pathways, on the other hand, were both required for oral epithelial damage. Analysis of the ability to penetrate nutrient-rich semi-solid media revealed a cooperative role for Cek1 and Mkc1 in

this process. Finally, cek2Δ (as well as cek1Δ) but not mkc1Δ or hog1Δ mutants, exhibited elevated β-glucan unmasking as revealed by immunofluorescence studies. Therefore, the four MAPK pathways play distinct roles in adhesion, epithelial damage, invasion and cell wall remodelling that may contribute to the pathogenicity of C. albicans.

Involved units

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