

***Candida albicans* infection leads to barrier breakdown and a MAPK/NF-κB mediated stress response in the intestinal epithelial cell line C2BBE1.**

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Abstract

Intestinal epithelial cells (IEC) form a tight barrier to the gut lumen. Paracellular permeability of the intestinal barrier is regulated by tight junction proteins and can be modulated by microorganisms and other stimuli. The polymorphic fungus *Candida albicans*, a frequent commensal of the human mucosa has the capacity of traversing this barrier and establishing systemic disease within the host. Infection of polarized C2BBE1 IEC with wild-type *C. albicans* led to a transient increase of transepithelial electric resistance (TEER) before subsequent barrier disruption, accompanied by a strong decline of junctional protein levels and substantial, but considerably delayed cytotoxicity. Time-resolved microarray-based transcriptome analysis of *C. albicans* challenged IEC revealed a prominent role of NF-κB and MAPK signaling pathways in the response to infection. Hence, we inferred a gene regulatory network based on differentially expressed NF-κB and MAPK pathway components and their predicted transcriptional targets. The network model predicted activation of

GDF15 by NF- κ B was experimentally validated. Furthermore, inhibition of NF- κ B activation in *C. albicans* infected C2BBel cells led to enhanced cytotoxicity in the epithelial cells. Taken together our study identifies NF- κ B activation as an important protective signaling pathway in the response of epithelial cells to *C. albicans*.

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