Candida albicans infection leads to barrier breakdown and a MAPK/NF-κB mediated stress response in the intestinal epithelial cell line C2BBe1.

Böhringer M, Pohlers S, Schulze S, Albrecht-Eckardt D, Piegsa J, Weber M, Martin R, Hünniger K, Linde J, Guthke R, Kurzai O (2016) *Candida albicans* infection leads to barrier breakdown and a MAPK/NF-κB mediated stress response in the intestinal epithelial cell line C2BBe1. *Cellular Microbiology* 18(7), 889-904.

Details

PubMed

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-κB was experimentally validated. Furthermore, inhibition of NF-κB activation in C. albicans infected C2BBe1 cells led to enhanced cytotoxicity in the epithelial cells. Taken together our study identifies NF-κB activation as an important protective signaling pathway in the response of epithelial cells to *C. albicans*.

Involved units

Fungal Septomics Oliver Kurzai Read more

Microbiome Dynamics Gianni Panagiotou Read more

Leibniz-HKI-Authors



Daniela Albrecht-Eckardt

Details



Michael Böhringer

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Reinhard Guthke

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Kerstin Hünniger

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Oliver Kurzai

Details



Jörg Linde

Details



Ronny Martin

Details



Sylvie McNamara

Details



Susann Pohlers

Details



Michael Weber

Details

Topics

Networks of host- fungal pathogen interaction

Identifier

doi: 10.1111/cmi.12566

PMID: 26752615