Dual-species transcriptional profiling during systemic candidiasis reveals organ-specific host-pathogen interactions.

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Abstract

Please see the corrigendum:

Scientific Reports 6: Article number: 36055; published online: November 03 2016; updated: December 19 201610.1038/srep36055 [Cross Ref]

This Article contains an error in the legend of Table 1.

“Values indicate absolute log2-FC.”

should read:

“Values indicate absolute fold-change.”

Abstract:

Candida albicans is a common cause of life-threatening fungal bloodstream infections. In the murine model of systemic candidiasis, the kidney is the primary target organ while the fungal load declines over time in liver and spleen. To better understand these organ-specific differences in host-pathogen interaction, we performed gene expression profiling of murine kidney, liver and spleen and determined the fungal transcriptome in liver and kidney. We observed a delayed transcriptional immune response accompanied by late induction of fungal stress response genes in the kidneys. In contrast, early upregulation of the proinflammatory response in the liver was associated with a fungal transcriptome resembling response to phagocytosis, suggesting that phagocytes contribute significantly to fungal control in the liver. Notably, C. albicans hypha-associated genes were upregulated in the absence of visible filamentation in the liver, indicating an uncoupling of gene expression and morphology and a morphology-independent effect by hypha-associated genes in this organ. Consistently, integration of host and pathogen transcriptional data in an inter-species gene regulatory network indicated connections of C. albicans cell wall remodelling and metabolism to the organ-specific immune responses.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Mikrobielle Immunologie Ilse Jacobsen Mehr erfahren

Microbiome Dynamics Gianni Panagiotou Mehr erfahren

Leibniz-HKI-Autor*innen

Sascha Brunke

Details

Theresia Conrad

Details

Betty Hebecker

Details

Bernhard Hube

Details

Ilse Denise Jacobsen

Details

Mario Kapitan

Details

Jörg Linde

Details

Sebastian Vlaic

Details

Themenfelder

Pathomechanismen an der Epithelbarriere

Awards

Please note the corrigendum

Identifier

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