A functional link between hyphal maintenance and quorum sensing in *Candida albicans*.

Polke M, Sprenger M, Scherlach K, Albán-Proaño MC, Martin R, Hertweck C, Hube B, Jacobsen ID (2017) A functional link between hyphal maintenance and quorum sensing in *Candida albicans*. *Mol Microbiol* 103(4), 595-617.

Details

PubMed

Abstract

Morphogenesis in Candida albicans requires hyphal initiation and maintenance, and both processes are regulated by the fungal quorum sensing molecule (QSM) farnesol. We show that deletion of C. albicans EED1, which is crucial for hyphal extension and maintenance, led to a dramatically increased sensitivity to farnesol, and thus identified the first mutant hypersensitive to farnesol. Furthermore, farnesol decreased the transient filamentation of an eed1Δ strain without inducing cell death, indicating that two separate mechanisms mediate quorum sensing and cell lysis by farnesol. To analyze the cause of farnesol hypersensitivity we constructed either hyperactive or deletion mutants of factors involved in farnesol signaling, by introducing the hyperactive RAS1(G13V) or pADH1-CYR1(CAT) allele, or deleting CZF1 or NRG1 respectively. Neither of the constructs nor the exogenous addition of dB-cAMP was able to rescue the farnesol hypersensitivity, highlighting that farnesol mediates its effects not only via the cAMP pathway. Interestingly, the eed1Δ strain also displayed increased farnesol production. When eed1Δ was grown under continuous medium flow conditions, to remove accumulating QSMs from the

supernatant, maintenance of eed1Δ filamentation, although not restored, was significantly prolonged, indicating a link between farnesol sensitivity, production, and the hyphal maintenance-defect in the eed1Δ mutant strain.

Beteiligte Forschungseinheiten

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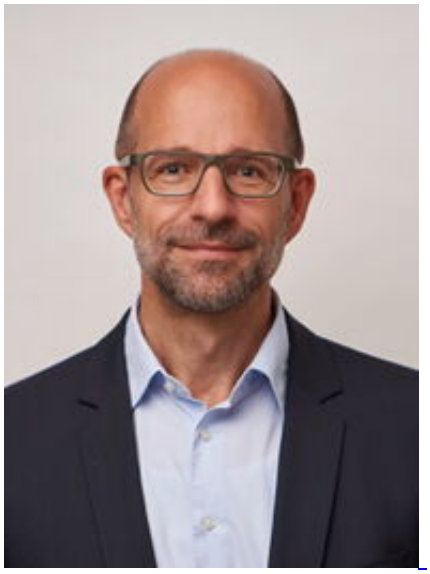

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Awards

Wissenschaftspreis der Stiftung der DMykG e.V.

Identifier

doi: 10.1111/mmi.13526

PMID: 27623739