

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

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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 eed1 Δ filamentation, although not restored, was significantly prolonged, indicating a link between farnesol sensitivity, production, and the hyphal maintenance-defect in the eed1 Δ mutant strain.

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