The effects of the *Pseudomonas aeruginosa* quorum-sensing molecule 3-oxo-C12-homoserine lactone on clinical *Candida albicans* biofilm development

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Abstract

The dimorphic yeast *Candida albicans* is the most common cause of systemic human fungal infections. Its virulence is linked to its ability to switch between budded and hyphal growth states, termed the budded-to-hyphal transition (BHT), as well as its ability to form biofilms on surfaces such as indwelling medical devices. In nature, *C. albicans* is commonly found in biofilms in association with other bacteria, including *Pseudomonas aeruginosa.* Previous work has shown that laboratory *C. albicans* strains can behave different from clinical *C. albicans* when treated with small molecules to prevent biofilm formation. The naturally occurring *C. albicans* quorum sensing molecule farnesol has been shown to inhibit hyphae formation as well as biofilm formation in laboratory strains. A quorum sensing molecule from *P. aeruginosa,* 3-oxo-C12-homoserine lactone (3OC12HSL) has also been suggested to inhibit hyphae formation and biofilm formation. Clinical isolates have been shown to respond differently to other inhibitory small molecules due to genetic variation, but farnesol and 3OC12HSL have not been tested with clinical isolates. Using a BHT assay and a separate biofilm assay, both molecules were tested with 11 clinical isolates previously shown to form strong biofilms. Both farnesol and 3OC12HSL were able to inhibit the BHT and biofilm formation in the 11 clinical isolates tested. Since these molecules naturally occur in the organisms, they may be good candidates for fungal therapies in order to prevent *C. albicans* biofilm formation.