

Fluconazole and lipopeptide surfactin interplay during *Candida albicans* plasma membrane and cell wall remodeling increases fungal immune system exposure.

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Rok wydania

2020

Czasopismo

Pharmaceutics

Numer woluminu

12

Strony

314/1-314/22

DOI

10.3390/pharmaceutics12040314

Kolekcja

Naukowa

Język

Angielski

Typ publikacji

Artykuł

Streszczenie

Recognizing the β -glucan component of the *Candida albicans* cell wall is a necessary step involved in host immune system recognition. Compounds that result in exposed β -glucan recognizable to the immune system could be valuable antifungal drugs. Antifungal development is especially important because fungi are becoming increasingly drug resistant. This study demonstrates that lipopeptide, surfactin, unmasks β -glucan when the *C. albicans* cells lack ergosterol. This observation also holds when ergosterol is depleted by fluconazole. Surfactin does not enhance the effects of local chitin accumulation in the presence of fluconazole. Expression of the CHS3 gene, encoding a gene product resulting in 80% of cellular chitin, is downregulated. *C. albicans* exposure to fluconazole changes the composition and structure of the fungal plasma membrane. At the same time, the fungal cell wall is altered and remodeled in a way that makes the fungi susceptible to surfactin. In silico studies show that surfactin can form a complex with β -glucan. Surfactin forms a less stable complex with chitin, which in combination with lowering chitin synthesis, could be a second anti-fungal mechanism of action of this lipopeptide.

Słowa kluczowe

Candida albicans, cell wall, β -glucan, lipopeptide, surfactin, fluconazole

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Adres publiczny

<http://dx.doi.org/10.3390/pharmaceutics12040314>

Strona internetowa wydawcy

<http://www.mdpi.com/journal/metals>