Candida and Candidiasis

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POSTER ABSTRACT BOOK

MICROBIOLOGY SOCIETY

BLOCK A

Differential modulation of complosome by colonizing and VVC-associated *Candida (C) albicans* strains in vaginal epithelial cells

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Abstract

Introduction

C. albicans is a common component of the vaginal microbiota but can also cause vulvovaginal candidiasis (VVC). Intracellular complement system, also known as complosome, contributes to local immune responses, however its involvement in VVC remains elusive.

Methods

An *in vitro* infection model using human vaginal epithelial cells (VECs) was employed to compare a colonizing *C. albicans* strain from a healthy woman with a pathogenic strain from a VVC patient. Flow cytometry assessed the intracellular C3, C5, their products C3a, C3b, C5a, and receptors C5aR1 and C3aR. ELISA quantified C3a and C5a release. RNA-seq analyzed transcriptomic changes and a functional assay evaluated Cathepsin D activity in VECs.

Results

Neither strain alter C3 levels but both increased C3a and C3b compared to uninfected VECs; however, VVC strain induced lower C3b production. While colonizing strain did not affect intracellular C5 or C5a, VVC strain caused marked reduction of both. Only colonizing strain substantially increased intracellular C5aR1. In contrast, C3aR similarly increased in VECs infected with both strains compared to those uninfected. A greater C3a release was induced by colonizing strain, with no C5a increase after infection with both strains. Expression of Cathepsin genes and Cathepsin D activity were reduced in VECs infected with VVC strain compared to those infected with colonizing strain.

Conclusion

The VVC strain compromises C5a/C5aR1 axis and reduces C3a/C3aR axis through the regulation of Cathepsins. Our study provides the first evidence linking the complosome to VVC pathogenesis, highlighting the C5a/C5aR1 axis as a potential therapeutic target in fungal infections.