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Fungal Genomics and Biology

Perspective

A Note on Vaginal Candidiasis

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INTRODUCTION

Candida vaginitis is one of the most common infections of the female genital tract with a high incidence. Candida is part of the typical flora in numerous women and is frequently asymptomatic. Therefore, candidal vulvovaginitis requires both the presence of candida in the vagina/vulva as well as the signs of irritation, itching, dysuria, or inflammation. Candida vaginitis affects about 75% of sexually active women, with 10% having repeated bouts. The most prevalent predisposing factors include pregnancy, diabetes, and antibiotic therapy, with C. *Albicans* being the most common causative agent. The research on vulvovaginal candidiasis species distribution and in vitro susceptibility to antifungal medications was inspired by reports of fluconazole resistance in Candida species. Mostly the following species were recognized in the patients with vulvovaginal candidiasis: C. *albicans* 87.5%, C. *glabrata* 8.6% and 3.9% included C. *krusei*, C. *famata*, C. *tropicalis* and S. *cerevisiae*. In women of reproductive age, vulvovaginal candidiasis is a prominent concern. While cell-mediated immunity is considered to be the most effective host defense against mucosal candidal infections, animal models and clinical investigations have indicated that adaptive immunity has no protective impact against VVC induced by putative immune regulatory mechanisms. However, natural defensive mechanisms and variables linked to infection susceptibility have remained mysterious. That is, until recently, when it was discovered, using a live challenge model in humans, that protection against vaginitis is associated with a non-inflammatory innate presence, whereas symptomatic infection is associated with a neutrophil infiltrate in the vaginal lumen and an increased fungal burden. As a result, rather than a supposedly inadequate adaptive immune response, symptomatic vaginitis is now thought to be caused by an aggressive innate reaction. Antibiotic usage, pregnancy, diabetes mellitus, immune suppression as in AIDS or HIV patients, frequent sexual intercourse, spermicide, intrauterine devices, and vaginal douching are the most prevalent risk factors that contribute to an imbalance in the vaginal microbiota.

DESCRIPTION

A vast spectrum of virulence characteristics and fitness traits enhance C. *Albicans* ability to infect so different host habitats. The morphological transition between yeast and hyphal forms, cell surface expression of adhesions and invasions, thigmotropism, development of biofilms, phenotypic switching, and production of hydrolytic enzymes are all considered as virulence factors. Rapid adaptability to changes in ambient pH, metabolic flexibility, potent food acquisition systems, and robust stress response machines are also fitness traits. Dimorphism refers to the transition between yeast and hyphal growth forms, and it has been suggested that both growth forms are crucial for pathogenicity. It's been proven that the hyphal form is more invasive than the yeast form. The smaller yeast form, on the other hand, is thought to be the one that is predominantly engaged in spreading. Virulence is often reduced in mutants that are unable to generate hyphae under in vitro conditions. Hypha development, on the other hand, is connected to the expression of a group of genes that encode virulence factors but aren't engaged in hyphal formation. The hyphal wall protein Hwp1, the agglutinin-like sequence protein Als3, the secreted aspartic proteases Sap4, Sap5, and Sap6, and the hypha-associated proteins Ece1 and Hyr1 are examples of hypha-associated proteins. HGC1, which codes for a hypha-specific G1 cyclin-related protein, causes cells to proliferate properly in yeast form but fail to generate hyphae. Despite this, hgc1/mutant cells express at least four hypha-associated genes (HWP1, ECE1, HYR1 and ALS3). The discovery that an hgc1 mutant was attenuated in a mouse model of systemic infection supported the concept that hyphal production is a key virulence feature in and of itself.

CONCLUSION

VC can be effectively treated with a variety of antifungal medications. Various traditional vaginal formulations (creams, gels, suppositories, powder, ointment, etc.) for VC are already available, but their efficiency is restricted due to the vaginal epithelium's self-cleaning function. To solve this issue, persistent

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and intimate contact with the vaginal mucosa is required, which can be achieved using mucoadhesive polymers. Mucoadhesive polymers have a high affinity for mucosal tissues and can attach to them for a long time. This unique characteristic of these polymers boosts the retention period of various formulations on mucosal tissues greatly. Liposomes, Nano and micro particles, micro-emulsions, bio-adhesive gel, and tablets are among the new formulations now being employed to manage and treat VC.