P358

Influence of Laminarin in colonisation process of *Candida albicans*

P. S. Bonfim-Mendonça,[1] F. K. Tobaldini,[2] I. M. Batalini,[1] I. R. G. Capoci,[1] J. S. R. Godoy,[1] E. S. Kioshima,[1] M. Negri[1] and T. I. E. Svidzinski[1]

[1]*Universidade Estadual de Maringá, Maringá, Brazil and* [2]*Universidade Estadual de Maringá/Universidade do Minho, Maringá, Brazil*

Objective *Candida albicans* is responsible for the majority of cases of vulvovaginal candidiasis (VVC), one of the most important candidal virulence factors is the ability to adhere to host surfaces. Chemotherapies that seek to improve the host immune response are an alternative to control fungal infections. β-glucans are polymeric carbohydrates that have been reported to modulate human inflammatory responses *in vitro* and *in vivo*. The aim of this study was to determine the influence of Laminarin (LAM) a β-glucan on *C. albicans* virulence, namely colonisation of HeLa cells.

Methods To assess the role of LAM in the cell colonization process, HeLa cells were previously treated or not with 3 mg mL^{-1} of LAM (β-glucan extracted from *Laminarina digitata*) for 30 min at 37 °C,

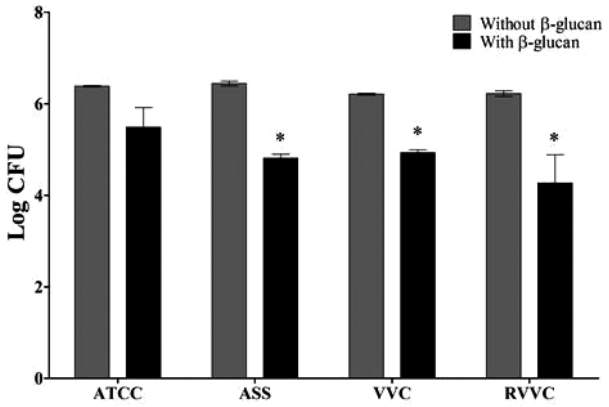

Figure 1. Adhesion of *Candida albicans* from VVC in Hela cells before and after treatment with Laminarin. *p<0.05, significance difference among treated and untreated group.

5% CO_2. Three clinical isolates (5V, 7V and 9V) obtained from female vaginal secretions and one reference strain (ATCC 90028) were used in the study. These strains were separated according to symptoms presented by the patients. Colonization assays were assessed for 2 h incubation at 37 °C, 5% CO_2, with $2x10^5$ HeLa cells mL^{-1} treated or not with LAM and $1x10^7$ yeast mL^{-1} of different clinical isolates of *C. albicans*. After colonization assays, adherent *C. albicans* cells were harvested by detaching the cervical cells monolayer to evaluation of viable cells (colony forming units).

Results In this study, LAM significantly decreased the interaction of VVC clinical isolates with Hela cells (Figure 1). For ASS and VVC isolates, there was a similar reduction in the number of viable cells during colonization process, approximately one log ($P < 0.05$). Moreover, RVVC isolate showed a reduction more expressive, approximately two log ($P < 0.05$).

Conclusion The pathogenesis of VVC involves the initial adherence of the yeast to the vaginal mucosa, followed by asymptomatic colonisation, ultimately leading to infection (symptomatic vaginitis). This study was able to show that LAM a β-glucan can negatively modulate the process of interaction between HeLa cells and *Candida albicans*. These results show that this carbohydrate might be a promising agent for preventing the first contact between yeast and vaginal epithelium, and consequently the development of VVC.