

P179 - *Candida albicans* and *Candida glabrata* sharing a live in vaginal environment  
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## Abstracts of poster presentations

Candidal vulvovaginitis (CV) is an infection of the mucous membranes of the vagina by *Candida*. Up to 75% of woman will have this infection at same point in their lives and approximately 5% will have recurring episodes. *Candida* species are found naturally in the vagina, and are usually harmless. However, conditions in the vagina may change resulting in an environment that facilitates *Candida* infection. However, it remains unclear what specific changes in the vaginal environment, or amongst the *Candida*, are key to promoting CV.

The current study examined the interaction and expression of virulence factors by two frequent *Candida* species colonising the vagina, namely *Candida albicans* and *C. glabrata*. *In vitro* infection studies were performed using a reconstituted human vaginal epithelium (RHVE), which facilitated examining the effect of altered environmental factors on *Candida* virulence. Confocal laser scanning microscopy showed that in single species infection, *C. albicans* was an extensive colonizer and invader of the RHVE, which was in direct contrast to *C. glabrata*. However, increased colonization and invasion of the RHVE by *C. glabrata* was evident in dual species infection. Furthermore, in dual species infection, expression of the *C. glabrata* epithelial adhesin (EPA) family of genes was considered less relevant to the infection process than expression of *C. albicans* virulence genes (*HWP1*; *ALS* and Phospholipases B and D family). Interestingly, up-regulation of *ALS3* and *HWP1* by *C. albicans* was evident in dual species infection. When the vaginal environment was modified by changing like pH or increasing progesterone concentration, decreased RHVE colonization by both species occurred together with reduced hyphal production by *C. albicans* and down regulation of *HWP1*. In conclusion, this work demonstrated that dual species infection of RHVE results in enhanced pathogenicity, which in turn, is attenuated by environmental changes.