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Host-microbe interaction in *Candida* mucosal infections: a complex balance

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INTRODUCTION. Vulvovaginal candidiasis (VVC) is a very common mucosal infection in women of childbearing age caused primarily by *C. albicans*, characterized by powerful inflammatory response associated with infiltration of non-protective neutrophils. *C. albicans* can also asymptomatically colonize the vaginal mucosa as part of the resident microbiota in healthy women. The loss of the epithelial tolerance triggers the onset of the disease with increased fungal burden and virulence. However, the tolerance threshold to *C. albicans* varies among women and the causes of such variability are still unknown. The scope of our work is to understand how epithelial cell tolerance to *C. albicans* breaks down, focusing on fungal-intrinsic factors and host-pathogen interaction.
MATERIALS AND METHODS. We characterized several traits of *C. albicans* isolates obtained from symptomatic and asymptomatic women both in culture medium or after infection of vaginal epithelial cells A-431, to determine the intrinsic pathogenic potential of these strains as well as their pathogenic activity during the interaction with vaginal epithelial cells. We analyzed strains by Multi Locus Sequence Typing (MLST), sequencing of the gene encoding the candidalysin toxin, quantification of cell-wall epitope exposure, rate of fungal growth and propensity for filamentation. Moreover, *C. albicans*-induced damage of the epithelial cells was evaluated; IL-1beta production and *C. albicans* shedding together with exfoliated epithelial cells were also tested.
RESULTS. The two sets of isolates from symptomatic and asymptomatic women did not differ in genetic profile or behaviour in culture media (i.e. MLST profile, rate of growth, filamentation) but they showed relevant differences when interacting with vaginal epithelial cells. Indeed, unlike the isolates from healthy colonized women, the VVC isolates showed a significantly greater propensity to induce *C. albicans* shedding in association with exfoliated epithelial cells.
DISCUSSION AND CONCLUSIONS. Our results point towards the exclusion of the involvement of fungal intrinsic factors in host-*C. albicans* balance at mucosal level; rather, fungal traits that arise when interacting with the host correlate with the likelihood of symptomatic infection in VVC.