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Secreted aspartic proteases from *Candida parapsilosis* regulate its virulence

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Candida parapsilosis is an opportunistic fungal pathogen responsible for approximately 30% of candidaemia episodes in new-borns and 10%–15% of *candida* infections in adults worldwide. Fungal extracellular hydrolytic enzymes, especially secreted aspartyl proteinases (SAP) are important virulence factors of *Candida* species, that contribute to the development of disseminated candidiasis. Although, several studies have investigated the role of *C. albicans* secreted aspartyl proteinases in its virulence, less is known about the precise role of *C. parapsilosis* secreted aspartyl proteinases (SAPPs) in host innate immune evasion, including complement. Thus, we aimed to study the function of *C. parapsilosis* secreted aspartyl proteinases during host attack. To investigate the role of SAPP genes, a deletion mutant strain (SAPP-/-) was generated, lacking all functional CpSAPP open reading frames (ORFs). To examine the effect of complement proteins on the growth of the *C. parapsilosis* GA1 (wild-type) and SAPP-/- strains, their growth was measured in YPD and YCB liquid medium supplemented with 20% of either normal human plasma (NHP) or heat inactivated human plasma (HiP) at 30 and 37 . In order to examine the effect of gene deletion on viability and appearance, we also tested their growth on different complex, minimal and stressor containing media. Virulence properties of the mentioned strains were also tested using an epithelial cell line, the human monocytic cell line THP-1 and human macrophages (PBMC-DMs). *In silico* analyses were also performed to evaluate efficiencies of HIV protease inhibitors against SAPP'S. Our results clearly indicated similar growth rates of the SAPP-/- strain and the wild-type strain in both YPD and YCB medium regardless of the temperature used whereas, addition of NHP significantly inhibited the growth of the mutant strain only. Deletion of CpSAPPs neither affect biofilm formation nor the morphological attributes of C. parapsilosis. Although, the SAPP-/- strain showed significant differences in its virulence when compared to the wild type. THP-1 and PBMCDM's killed and phagocytosed SAPP-/- cells more efficiently. Mutant cells also induced less damage to human epithelial cells, THP-1 cells and to PBMC-DMs compared to the wild type strain. Furthermore, when examining host cytokine responses, the wild type strain induced higher levels of IL-1β, TNF-α and IL-8 than the SAPP-/- mutant strain indicates SAPP mediated immune modulation during *C. parapsilosis* infection.

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