Protective effect of inactivated blastoconidia in keratinocytes and human reconstituted epithelium against C. Albicans infection

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Candida albicans is commensal yeast that colonizes skin and mucosa; however, it can become an opportunist pathogen by changing from blastoconidia (commensal form) into hypha (pathogenic form). Each form activates a different cytokines response in epithelial cells. Little is known about the commensal role of C. albicans in the innate immunity. This work studied whether stimulation with C. albicans blastoconidia induces protection in keratinocytes and/or in a reconstituted human epithelium (RHE) infected with C. albicans. For this, inactivated C. albicans blastoconidia was used to stimulate keratinocytes and RHE prior to infection with C. albicans. Blastoconidia induced different cytokine expression profiles; in the case of RHE it decreased interleukin (IL)-1? and IL-10 and increased IL-8, tumor necrosis factor ? (TNF-?), and interferon ? (IFN-?). A significant increase in the expression of human ?-defensins (HBD) 2 and HBD3 was observed in blastoconidia stimulated keratinocytes and RHE, associated with impaired growth and viability of C. albicans. Additionally, blastoconidia stimulation decreased the expression of virulence factors in C. albicans

that are associated with filamentation (EFG1, CPH1 and NRG1), adhesion (ALS5), and invasion (SAP2). Blastoconidia stimulated RHE was significantly less damaged by C. albicans invasion. These results show that the commensal form of C. albicans would exert a protective effect against self-infection.