Polymorphonuclear leukocytes (PMNs) induce protective Th1-type cytokine epithelial responses in an *in vitro* model of oral candidosis.

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Abstract

The immune response and the anticandidal activity of keratinocytes and polymorphonuclear leukocytes (PMNs) play a key role in host defence against localized Candida albicans infection. An established model of oral candidosis based on reconstituted human oral epithelium (RHE) was supplemented with PMNs to study the effect of these immune cells during experimental oral candidosis. Infection of RHE with C. albicans induced a strong expression of the chemokine interleukin-8 (IL-8) and the cytokine granulocyte-macrophages colony-stimulating factor (GM-CSF), and a moderate stimulation of interleukin-1alpha (IL-1alpha), interleukin-1beta (IL-1beta), interleukin-6 (IL-6), interferon gamma (IFN-gamma) and tumour necrosis factor alpha (TNF-alpha) by keratinocytes. This immune response was associated with chemoattraction of PMNs to the site of infection, whereas uninfected RHE failed to induce cytokine expression or to attract PMNs. Growth of the pathogen and tissue damage of C. albicans-infected RHE were significantly reduced when PMNs were applied to the apical epithelial surface or when PMNs migrated through a

perforated basal polycarbonate filter of the model. Notably, protection against epithelial tissue damage was also observed when PMNs were placed on the basal side of non-perforated filters, which prevented PMN migration into the RHE. Addition of PMNs enhanced a Th1-type immune response (IFN-gamma, TNF-alpha), down-regulated the expression of the Th2-type cytokine interleukin-10 (IL-10), and was associated with protection against Candida-induced tissue damage. This PMN-supplemented model of oral candidosis mimics the in vivo situation, and provides a promising tool for studying the immunological interactions between keratinocytes and C. albicans, as well as the influence of PMNs on C. albicans pathogenesis.

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