THE EFFECTS OF IL-17 AND IFN-G ON HUMAN GINGIVAL FIBROBLASTS

Mahanonda R, Jitprasertwong P, Sa-Ard-Iam N, Yongvanitchit K and Pichyangkul S

Periodontitis is a bacterial disease characterized by chronic gingival inflammation, which leads to the loss of tooth-supporting tissues. Dense infiltrates of activated memory T cells and high levels of T cell cytokines were consistently detected in periodontitis lesions. Although T cells have been considered to be central to both progression and control of chronic inflammatory periodontal diseases, the precise contribution in local immunoregulation has not been fully clarified. Recent observations showed the presence of IL-17 and IFN-g in the inflamed periodontal tissues. Therefore, in this study we investigated the immunoregulatory role of IL-17 and IFN-g on human gingival fibroblasts which were obtained from clinically healthy periodontal tissues. Dose dependent production of IL-8 was detected in fibroblasts cultured with IL-17 but not with IFN-g. However, IFN-g significantly enhanced IL-8 production when combined with IL-17. The expression of ICAM-1 (CD54) and HLA-DR on fibroblasts was also studied. Our prelimary results demonstrated that unlike IL-17, IFN-g up-regulated both ICAM-1 and HLA-DR expression. Further work on the combination of the two cytokines on cell surface expression are being investigated.

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HIGH RATES OF APOPTOSIS IN CULTURE POSITIVE LESIONS OF BURULI ULCER

Walsh DS, Meyers W, Portaels F, Lane JE and Myint KS

Tissue damage in Buruli ulcer, a disease caused by *Mycobacterium ulcerans*, may be mediated by 1 or more bacterial toxins that trigger apoptosis. Here, we analyzed sections of paraffin-embedded, untreated Buruli ulcer lesions (n = 3) and normal appearing adjacent skin (n = 9) for apoptosis by an indirect immunofluorescent terminal deoxynucleotide-transferase mediated dUTP-digoxigenin nick end labeling (TUNEL) assay. All samples were cultured, and most were analyzed with a *M. ulcerans*-specific polymerase chain reaction (PCR). TUNEL (+) bodies were numerous in all 3 ulcer samples, and minimal or absent in normal appearing skin adjacent to ulcers. Cultures for *M. ulcerans* were positive only in the 3 ulcer samples, whereas PCR was positive in the ulcers as well as 4 of 6 adjacent, non-ulcerated tissue samples. High numbers of TUNEL (+) bodies in frank, *M. ulcerans* culture positive ulcers, but not in nearby PCR positive, but culture negative normal appearing skin, strengthen the evidence that apoptosis may be a key tissue destruction factor that is directly linked to the presence of viable *M. ulcerans*.

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