B-5. Expression of antigen presenting function-associated surface molecules on interferon γ-treated gingival fibroblasts and periodontal ligament fibroblasts

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It is becoming increasingly clear that human gingival fibroblasts(HGF) may play a role in regulating immune responsiveness in inflammatory periodontal lesions. Stimulation of HGF with locally-secreted T cell cytokine IFN γ induces human leukocyte antigen class II(HLA II) expression on HGF, which is one of the characteristic feature of professional antigen presenting cells(pAPC). However, IFN γ treated HGF and other nonprofessional antigen presenting cells(npAPC) are known to be ineffective or less effective antigen presenter to resting T cells. This study, therefore, was undertaken in an effort to elucidate the differences in expression of cell surface molecules between npAPC in periodontal tissues, such as HGF and periodontal ligament fibroblasts(PDLF), and pAPC such as monocytes/macrophages. Using flow cytometry, the levels of cell surface expression of HLA-D, ICAM-1, LFA-3, and B7-1, which are involved in antigen presentation, were determined in HGF, PDLF and human myelomonocytic cell line THP-1. IFNγ clearly induced HLA-D expression on both of fibroblasts and monocytes dose dependently. However, expression level on monocytes were 4 to 5 times higher than that on fibroblasts, and induction rate was faster in monocytes than in fibroblasts. The levels of ICAM-1 expression on fibroblasts and monocytes were enhanced by IFN γ in a dose dependent manner. On the other hand, the expression of LFA-3 molecule, which could be detected in fibroblasts and monocytes without cytokine stimulation, was no more enhanced by addition of IFNγ. B7-1, important costimulatory molecule in T cell activation and proliferation, was not detected on both of fibroblasts and monocytes even when stimulated with IFNγ, except on monocytes fully differentiated by pretreatment of PMA and treated by IFNγ. These results suggest that delayed expression of HLA-D and absence of B7-1 on IFNγ-treated fibroblasts may at least in part be involved in the ineffectiveness of fibroblasts as primary APC. And it is postulated that although periodontal fibroblasts may not serve as primary APC in normal periodontium, sustained expression of HLA II on ubiquitous fibroblasts in inflammatory lesions may perpetuate immune responses and produce chronic inflammation and tissue injury.