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Title: Interferon γ (IFNγ) decreases apoptosis rate of antigen primed T helper lymphocytes from bronchoalveolar lavage (BAL)

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Body: Background: We have previously found that IFNγ levels in BAL supernatants of interstitial lung diseases (ILD) patients were associated with T cell number and increased CD4/CD8 ratio. We suggested that Th, but not Tc, are the principal IFNγ local source. However, alternative explanation is possible. According to some experimental data [McKinstry et al, 2010], IFNγ protects primed T cells from contraction during immune response, i.e. from massive apoptosis. Methods: IFNγ levels were tested by ELISA in BAL supernatants from sarcoidosis (PS), subdivided due to disease form, extrinsic allergic alveolitis (EAA), idiopathic pulmonary fibrosis (IPF), nonspecific interstitial pneumonia (NSIP) and silicosis (n=33, 9, 11, 8, 7 resp.). BAL cells were stained for major subsets. AL apoptosis was examined with use of flow cytometry by both cell cycle analysis (sub-G1 peak) and cell light scatter properties. Results: IFNγ level was significantly increased in Loefgren's syndrome (11.7±2.9), progressive sarcoidosis (5.5±1.4), IPF (6.7±1.3) and silicosis (6.6±2.3 pg/ml, all results as median±SEM). In chronic sarcoidosis (2.6±1.4) and NSIP (3.3±1.7) IFNγ results were comparable to controls (1.7±1.4pg/ml). There was no association between IFN level and total BAL lymphocyte apoptosis, as well as Tc apoptosis. However, Th (CD4+) apoptosis rate was remarkably negatively correlated with IFNγ levels ($R_s=-0.25$, $p<0.01$). Conclusions: Our data are consistent with view on IFNγ as a survival factor for primed Th. Experimental results were supported herein by clinical data. Additionally, IFNγ levels obtained in IPF, indicate the need to reevaluate this disease as Th2 disorder.