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Title: The death receptors (DRs) expressed on alveolar lymphocytes (AL) in interstitial lung diseases (ILD) participate in apoptosis regulation

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Body: Background. The number of AL, regulated by their local proliferation and apoptosis, contributes to the activity of immune process in ILD. Ligation of death receptors (DRs) by specific ligands on AL seems to be a potent mechanism of apoptosis induction. Aim. Evaluation of DRs expression on AL. The assessment of DR role in AL apoptosis in ILD. Methods. Bronchoalvolar lavage (BAL) carried out in sarcoidosis (PS), extrinsic allergic alveolitis (EAA), idiopathic interstitial pneumonias (IIP) and controls (n=46,7,23,13 resp.). AL staining for Fas, Fas Ligand (FasL), DR3, DR4 and TNFα receptors (CD120A and B). BAL supernatant soluble Fas (sFas), sFasL, TRAIL and TNFα levels measured by ELISA. Results. In general, common Fas and CD120B apearance on AL coexists with low expression of DR3, DR4 and CD120A. AL apoptosis rate was sign. positively correlated with TNFα level as well as with DR4, FasL, CD120A expression and ($p<0.00001$) CD120A/CD120B ratio; sign. negative correlation was found for sFas. Remarkably declined FasL and CD120A expression was shown in ILD with low AL apoptosis rate, as Loefgren syndrome, progressive PS and EAA (e.g. CD120A+: 5.6±1.5% in PS and 3.3±2.1% in EAA vs 11.0±6.7% in controls, $p<0.01$, median±SEM), Increased percentage of AL FasL+, high TRAIL and TNFα levels were characteristic for IIP, the disorder with frequent AL apoptosis. Conclusions. DRs participate in AL number regulation, however different mechanisms may drive the process in specific ILD. TNFα proapoptotic effect on AL is probably dependent on the imbalance of its receptors (CD120A and B). Fas/FasL system seems to be active by FasL membrane-bound form, but not soluble one.