Idiopathic pulmonay fibrosis (IPF), extrinsic alergic alveolitis (EAA) and sarcoidosis are interstitial lung diseases (ILD) of distinct pathogenesis. While IPF represents primarily fibrosing lung disorder of unknown etiology with dominant Th2 cytokine milieu, sarcoidosis is a systemic disease presenting with noncaseating granulomas and Th1 cytokine pattern. EAA develops after repeated exposure to inhalation antigens, and can present either with a granulomatous formation or progressive pulmonary fibrosis according to the stage of the disease and relationship between antigen exposure and immune system status.
Th1/Th2 imbalance is dominant feature of these ILDs and we suppose that the susceptibility to them could be genetically encoded in cytokine gene polymorphisms. Cytokine gene polymorphisms could influence cytokine protein expression, which might lead to imbalance of Th1/Th2 immune reactions. Enhanced Th2 type cytokine production might induce the alternative activation of alveolar macrophages (AM), with consequent stimulation of colagen production by fibroblasts. the way from gene to protein leads through messenger RNA (mRNA) expression. Results of the pilot studies suggest that expression profiling could help to identify pathways relevant to pathogenesis of these disorders. The aim of our study was to ellucidate possible pathogenic pathways of ILDs, from cytokine genes through mRNA to localized proteins expressions. We think that this kind of approach could help us to better understand ILDs pathogenesis.