

Rheumatoid arthritis (RA) is a chronic autoimmune disease that is associated with formation of autoantibodies, activation of inflammatory cascade and up-regulation of several cytokines. These processes lead to persistent synovial inflammation, joint damage and systemic manifestations. The aim of this diploma thesis is to characterize the role of a novel cytokine interleukin-20 (IL-20) in the pathogenesis of RA and to investigate its involvement in different stages of the disease as a potential surrogate biomarker. In this work, several methods including Enzyme-Linked Immunosorbent Assay (ELISA), Immunohistochemistry and Real-Time quantitative Polymerase Chain Reaction (RT-qPCR) have been employed.

We demonstrated increased expression of IL-20 in the synovial tissue of RA compared with control osteoarthritis (OA) patients. Along with the up-regulation at sites of inflammation, concentrations of IL-20 were higher in the synovial fluid compared with circulating levels of IL-20. Furthermore, serum and synovial fluid IL-20 levels significantly correlated with RA disease activity. Synthesis of IL-20 was significantly increased in peripheral blood mononuclear cells (PBMCs) and synovial fibroblasts upon stimulation with some TLR ligands and pro-inflammatory cytokines. Although not regulating PBMCs functions *in vitro*, IL-20 stimulated expression of IL-8 in RA synovial fibroblasts. Taken together, IL-20 represents significant cytokine involved in the pathology of RA, may reflect disease severity and potentially might be a good therapeutic target for RA.