CHARACTERISATION OF CD8+ T CELL SUBSETS IN THE SYNOVIAL FLUID AND PERIPHERAL BLOOD OF RHEUMATOID ARTHRITIS PATIENTS

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Background and objectives Detailed information on CD8+ T cells in rheumatoid arthritis (RA) is still reduced. However, studies on animal models of arthritis from the authors’ team and others suggest a major potential role of CD8+ T cells in the pathogenesis of chronic polyarthritis.

In the present study the authors characterised the phenotype and cytokine-production profile of CD8+ T cells from peripheral blood (PB) and synovial fluid (SF) of RA patients.

Materials and methods Unstimulated CD8+ T cells from the PB and SF of 40 patients with established RA were analysed by flow-cytometry for cell surface markers expression and intracellular cytokine production, and compared to the ones present in the PB of 15 healthy donors.

Results 40% of the total T cells infiltrating the SF were CD8+. The SF was characterised by a significant (p<0.05) accumulation of mature CD45Ro+CD8+ T cells when compared to the RA and control PB. The majority of these CD8+ T cells infiltrating the SF presented an effector phenotype (CD62L−CD27− short-term effector, or CD62L−CD27+ central effector), and the presence of the activation markers CD69 and CD25 were significantly (p<0.05) higher in the SF than in the PB of RA patients or controls. The expression of the pro-inflammatory homing chemokine receptors CCR7 and CXCR4 were significantly (p<0.05) increased in the short-term effector CD62L−CD27−CD8+ T cells and in the SF central memory CD62L+CD27+CD8+ T cells when compared to RA PB. The intracellular expression of the pro-inflammatory cytokines IFN-γ, interleukin-6, interleukin-17 and tumour necrosis factor-α in central memory CD62L+CD8+ T cells were significantly (p<0.05) higher in the SF when compared to RA PB, and absent in controls.

Conclusions The present study presents strong evidence for a marked role of CD8+ T cells in RA pathogenesis. The authors propose that activated effector CD8+ T cells, home into the SF, where they might contribute to articular destruction and maintenance of a chronic pro-inflammatory environment through the production of cytokines and cytotoxic agents.