Cell Communication and Signaling BioMed Central

Meeting abstract

Open Access

Induction of CCL13 expression in synovial fibroblasts underlines a significant role of oncostatin M in rheumatoid arthritis

C Hintzen*[1], S Quaiser[2], T Pap[3], PC Heinrich[4] and HM Hermanns[1]

Address: [1]Rudolf-Virchow-Center, University of Würzburg, Würzburg, Germany, [2]Department of Biochemistry, University Hospital RWTH, Aachen, Germany, [3]Division of Molecular Medicine of Musculoskeletal Tissue, Department of Orthopedics, University of Münster, Münster, Germany and [4]Department of Biochemistry and Molecular Biology, University of Freiburg, Freiburg, Germany

* Corresponding author

from 12th Joint Meeting of the Signal Transduction Society (STS). Signal Transduction: Receptors, Mediators and Genes
Weimar, Germany. 29–31 October 2008

Published: 26 February 2009

Cell Communication and Signaling 2009, **7**(Suppl 1):A107 doi:10.1186/1478-811X-7-S1-A107

This abstract is available from: http://www.biosignaling.com/content/7/S1/A107

Rheumatoid arthritis (RA) is a severe, chronic disease characterized by a profound inflammatory response that leads to both joint destruction as well as extraarticular symptoms with a significant impact on both morbidity and mortality. The cause and pathologic process underlying RA have not yet been fully elucidated. However, it is clear that both the cellular immune system and the cytokine network are subject to profound dysregulations. The success of an anti-proinflammatory cytokine therapy directed against either tumor necrosis factor alpha (TNFα) or, to a lesser extent, interleukin-1 (IL-1), has highlighted the significance of these cytokines in the pathological progession of RA. Nevertheless, these therapeutical strategies are not beneficial to all patients, suggesting that other cytokines like IL-6, oncostatin M, IL-4, IL-13, IFNγ are also present in the synovial fluid of chronically inflamed joints. Here we demonstrate that among a number of cytokines present in the synovial fluid of RA patients, only OSM induces a prolonged expression of the chemokine CCL13. The expression of CCL13 appears to be tissue-specific since neither in dermal nor lung or cervix fibroblasts CCL13 expression could be observed. We identify human synovial fibroblasts (HSF) as well as synovial fibroblasts of RA patients (RASFs) as producers of CCL13. The expression of CCL13 is mediated through STAT5 activation and p38 MAPK-dependent pathways and results in increased migration of monocytic cells.