is expressed on early myeloid and lymphoid progenitors and is activated by its soluble ligand, Flt3L. The highly differentiated cellular pattern in rheumatoid arthritis (RA) synovium made the authors hypothesise that Flt3L, with its ability to induce proliferation and differentiation, could be of importance in induction and/or progression of arthritis.

Material and methods Patients with active RA, psoriatic arthritis (PsA), spondyloarthritis (SpA), osteoarthritis (OA), gout and healthy donors (HD) were included in this study. Soluble (s)Flt3L levels in synovial fluid (SF) and serum were determined by ELISA. Expression of membrane-bound (m) Flt3L and Flt3L receptor (CD135) in peripheral blood mononuclear cells (PBMC) and SFMC were assessed by FACS. In addition, immunohistochemical analysis of Flt3L and CD135 was performed in RA, PsA, gout, OA and HD synovial tissues.

Results SF levels of Flt3L in RA (n=103), PsA (n=33), and SpA (n=32), OA (n=8) and gout (n=43) were significantly higher compared to paired serum. In addition, Flt3L levels were significantly higher in RA, PsA and SpA SFs compared to gout SF. In peripheral blood (PB) monocytes, B cells and mDC the expression of mFlt3L in RA was higher compared to HD. Flt3L receptor expression was confined to monocytes and mDC and higher in RA SF compared to PB. Immunohistochemistry and immunofluorescence data showed the presence of Flt3L and CD135 in RA ST. Interestingly, microarray data of RA synovial tissue showed that CD135 expression is increased in patients with high inflammatory gene profile compared to low inflammatory gene profile. There is no different in Flt3L expression between RA and OA confirming the ELISA data. In addition, anti-TNF therapy reduced Flt3L serum levels in RA patients.

Conclusion The data presented in this study point to inflammatory role for Flt3L/CD135 system. Moreover, as the Flt3L/CD135 system is implicated in the generation of DC and B cells, inflammatory cells important in RA pathogenesis, this system might be of importance in RA. Achieving a detailed understanding of Flt3L function(s) in arthritis may lead to the development of novel immunotherapies for RA and other immune-mediated inflammatory diseases.

A52 FMS-LIKE TYROSINE KINASE 3 LIGAND/CD135 IN ARTHRITIS: A NEW INFLAMMATORY SYSTEM IN RA?

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Background Fms-like tyrosine kinase 3 ligand (Flt3L) is a potent endogenous growth factor for myeloid dendritic cells (mDC) and plasmacytoid dendritic cells (pDC). Its administration to mice and humans leads to dramatic increases of various DC subsets while Flt3L–/– mice show reduced DC numbers. Flt3L and its receptor (CD135) have been poorly studied in the setting of autoimmune diseases in general. Typically, CD135