only slightly. Importantly, levels of IL-17 in synovial fluid of ACPA+ RA patients were significantly higher than in ACPA– RA or OA patients.

Conclusions Mast cells are the main cell population producing IL-17 in all three disorders analysed. Possibly, selective activation of mast cells in ACPA+ RA patients is responsible for the increased IL-17 levels in this group. These data are relevant for studies aiming to inhibit IL-17 production in the treatment of arthritis.

A86 MAST CELLS ARE THE MAIN IL-17 PRODUCING CELLS IN SYNOVIUM OF ANTI-CITRULLINATED PROTEIN ANTIBODY-POSITIVE AND -NEGATIVE RHEUMATOID ARTHRITIS AND OSTEOARTHRITIS PATIENTS

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Background and objectives Recently, evidence is raised that rheumatoid arthritis (RA) can be divided in two main syndromes on the basis of the presence of anti-citrullinated protein antibodies (ACPA). Mast cells have been implicated to play a functional role in mouse models of arthritis as well as human RA, especially in autoantibody-positive disease. Interleukin 17 (IL-17) has also been implicated to play an important role in RA. Recent data in 10 RA patients indicate that mast cells are the main producers of IL-17 in synovial tissue, whereas T cells have also been implicated as a prominent IL-17 producing subset. Therefore, the authors aimed to identify whether expression of IL-17 by mast cells and T cells in synovium is different in ACPA+ and ACPA– RA and osteoarthritis (OA) patients.

Materials and methods Synovial tissue of ACPA+ (n=25) and ACPA– (n=34) RA and OA (n=29) patients was stained for IL-17 in combination with CD117 (mast cells), CD3 (T cells) and CD68 (macrophages) or isotype controls. Concentrations of IL-17 in synovial fluid of ACPA+ (n=30) and ACPA– (n=29) RA and OA (n=14) patients were determined by ELISA.

Results The number of IL-17+ cells was comparable in ACPA+ RA, ACPA– RA and OA patients. The vast majority of IL-17+ cells were mast cells in all three groups (median ranged from 93% to 97%), whereas T cells and macrophages contributed