into the Risk-RA clinical research program, and followed-up by our multidisciplinary rheumatology team. A total of 64 patients with complete US records were included between years 2015–2016. Our patient demographics were 84% (n 54/64) females, mean age 49 (range 22–82) years, median relative ACPA (times cut-off) 26 (range 1–174) titer, median visual analogue scale (VAS) pain 30, median VAS patient global 28 and median C-reactive protein was 1 (0–20) at inclusion. Hand (Wrists, MCP's, PIP's, DIP's) and feet joints were US-evaluated for synovial hypertrophy, hyperemia and bone erosions. The presence of wrist (compartments 1–6) and finger (flexor and extensor) tenosynovitis were assessed. Data from inclusion→follow-up visits until September 2017 were analysed. SPSS software version 25 was used (Univariate, Chi-square, T-test and Mann Whitney U-test) for comparisons.

Results At inclusion, none of the 64 patients had any signs of active joint inflammation. However, ultrasound changes for tenosynovitis were seen in 7 out of 64 patients, 3 of who also presented with mild hypertrophy without Doppler activity and one patient with mild Doppler hyperemia (without hypertrophy), and none with bone erosions at inclusion. Among all tendons evaluated bilaterally, tenosynovitis of the Extensor Carpi Ulnaris (ECU) wrist tendons (4 of 7 patients) and the 2nd finger flexor-tendons (3 of 7 patients) were most commonly affected.

Of the 57 patients without US-tendon changes, one had mild Doppler hyperemia (without hypertrophy). Patients with US-tendon changes were 86% (n 6/7) females, had mean age 56 years, median VAS pain 42, median VAS global health 20, mean 2.7 mg/L CRP, median relative ACPA titer 70 in comparison to patients without US-tendon changes, 75% females, mean age of 48 years, median VAS pain 24, median VAS global health 28, mean 2.7 mg/L CRP and median ACPA titer of 23. The numerical difference in pain and relative ACPA titer were non-significant ($p>0.05$).

After follow up for mean 18 months (range 1–18), 7 out of 7 (100%) with US-tendon changes at inclusion and 18 out of 57 (32%) without US-tendon changes developed arthritis. Patients with US-tendon changes compared to those without tenosynovitis at inclusion, developed arthritis within 12 and 11 mean months follow-up, respectively.

Conclusions Our study shows that tenosynovitis is a specific marker for arthritis development in ACPA-positive patients with musculoskeletal symptoms. The role of inflammatory spreading from tendons (synovial sheath) to synovial tissue within joints need to be further investigated.

Disclosure of interest None declared

P020

TOLL-LIKE RECEPTOR 9 INFLUENCES INFLAMMATORY ARTHRITIS AND OSTEOCLASTOGENESIS

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Introduction Release and insufficient removal of endogenous nucleic acids may be involved in triggering autoimmune reactions important in the initiation of systemic autoimmune

diseases including rheumatoid arthritis (RA). Nucleic acid sensing molecules, such as the endosomal Toll-like receptors (TLRs) 7 and 9, have been linked to pathogenic autoimmune processes, but their role in RA is less clear.

Objectives To gain more insight into the role of TLR9 in autoimmune arthritis, TLR9 inhibition was investigated in rats with pristane-induced arthritis (PIA). To further investigate TLR9 involvement, streptococcal cell wall (SCW) arthritis was induced in TLR9^{-/-} mice.

Methods Arthritis was induced in mice with SCW lysates and in rats with the mineral oil pristane. Rats were treated with a TLR9 antagonist, starting before disease induction. Arthritis was scored using established scoring systems, inflammation and bone erosion was quantified by histological analysis of the paws. Levels of α -1-acid-glycoprotein (AGP), rheumatoid factor (RF) and IL-6 in sera were analysed. The role of TLR9 in osteoclast differentiation was investigated *in vitro*.

Results In PIA, which is T cell-dependent, the TLR9 antagonist reduced arthritis severity by ~50%. This was accompanied by a reduction of AGP, IL-6 and RF in the sera of these animals. In addition, TLR9 inhibition led to reduced inflammation, bone erosion and cartilage degradation in the paws. Moreover, the T cell-dependent chronic phase of SCW arthritis was significantly suppressed in TLR9^{-/-} mice. Remarkably, TLR7 and TLR9 mRNA levels strongly differed in the course of *in vitro* osteoclastogenesis. Whereas TLR7 expression did not change throughout osteoclastogenesis, expression of TLR9 was higher in precursor cells than in mature osteoclasts and stimulation with a TLR9 agonist (CpG) completely inhibited osteoclastogenesis.

Conclusions The results suggest a crucial role for TLR9 in the T cell-dependent phases of PIA and SCW arthritis and thus an important involvement of the DNA (CpG) recognising TLR9 in the induction of arthritogenic autoimmune reactions. In addition, TLR9 also seems to play a role in the initiation of osteoclast differentiation which needs to be further elucidated in future experiments.

Disclosure of interest None declared

P021

MEMBRANE TNF EXPRESSION ON MONOCYTES AND DIFFERENTIATION OF MONOCYTES INTO M2-M1 MACROPHAGES: 2 NEW BIOMARKERS OF RHEUMATOID ARTHRITIS

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Introduction Three monocyte subsets have been described based on their CD14 and CD16 expression profiles, the sub-population CD14⁺CD16⁺ being expanded in rheumatoid arthritis (RA) patients. Macrophages contribute *in situ* to the RA pathogenesis. They can display various states of activation or « polarisation ». Two distinct states of polarisation for macrophages have been recognised: the 'classically activated macrophage phenotype' (M1) and the 'alternatively activated macrophage phenotype' (M2). To sum-up, M1 are considered to be pro-inflammatory and M2 to be regulatory and anti-inflammatory.

Objectives Here, we have assessed monocytes subsets and their capacity of differentiation into M2 or M1 macrophages in RA patients and controls.