Soluble ICAM-1 (sICAM-1) and CXCL13 concentrations in sera from RA patients and comparing with osteoarthritis (OA) patients. The serum Syndecan-4 is higher in RA patients than in OA patients. Results: The mean age of the 60 patients were 54.8±11.6 years. Serum Syndecan-4, one of the members of heparan sulphate proteoglycans (HSPGs), has been shown to be involved in regulating inflammatory responses, angiogenesis, and cell migration. Its role has been proved in animal arthritis models, however not clearly elucidated in rheumatoid arthritis (RA) patients.

Methods: The concentrations of syndecan-4 in sera and synovial fluid of RA and osteoarthritic (OA) patients were detected by ELISA. In another cohort of 60 RA patients, the association analysis was performed. All the RA patients were with disease duration more than 6 months and with DAS28-3.2 and at least 3 months. They were further treated with TNFi receptor Fc fusion protein and MTX 10mg per week for 12 weeks.

Results: The correlation between serum sICAM-1 and CXCL13 concentrations at baseline and 12 weeks based on different response criteria. The comparisons of sICAM-1 and CXCL13 concentrations at baseline and 12 weeks based on different response criteria.
7. Spondyloarthritis - etiology, pathogenesis and animal models

**AB0113** ANTIBODIES AGAINST HELICOBACTER PYLORI ANTIGENS IN PATIENTS WITH PSSORIATIC ARTHRITIS AND PSORIASIS

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**Background:** Psoriasis (Ps) and Psoriatic Arthritis (PsA) are inflammatory diseases of unknown etiology. Helicobacter pylori (Hp) infection has been hypothesized as one of the microbial agents that can lead to development of immune-mediated psoriatic disease, but the nature of the specific Hp antigens involved remains unclear.

**Objectives:** To assess antigen specific antibody responses against immunodominant Hp antigens in patients with psoriatic diseases.

**Methods:** Ninety-one patients with Ps (35 females; median age 51.9, age range 25-87), 47 patients with PsA (25 females; median age 52.9, age range 25-87) and 60 demographically matched healthy controls (HC) were studied. Reactivity to Hp-specific antigens were tested by Western immunoblotting (in combination with line immunoassay for anti-CagA and anti-VagA antibody testing) (EUROIMMUN AG, Lubeck, Germany).

**Results:** Positivity against Hp was comparable between PsA (38.3%), Ps (39.6%) and HCs (50%). Anti-p66-UreB, anti-p54-flagelin and anti-p29-UreA abs were more frequent in psoriatic patients compared to healthy controls (p66: 94.4% in Ps vs 69.7% in PsA vs 33.3% in HC, p=0.017; p54: 66.7% in Ps vs 33.3% in HC, p=0.02; p29 72.2% in Ps vs 45.5% in HC, p=0.044) and anti-p29-UreA abs were detected in higher frequency in PsA patients compared to HC (94.4% vs 45.5%, p=0.002).

Reactivities against the remaining Hp antigens were comparable between Ps and PsA patients and HC.

**Conclusion:** Antibody responses against p66-UreB, p29-UreA, and p54-flagelin are more prevalent in patients with psoriatic disease, suggesting their potential involvement in PsA and Ps.

**Disclosure of Interests:** Eleini Patrikou: None declared, George Ethymiou: None declared, Christos Liaskos: None declared, Niki Ntavari: None declared, Efterpi Zirioú: None declared, Theodora Simopoulou: None declared, Thomas Schepers: Employee of: Employee of EUROWIMMUN AG, Lubeck, Germany, Wolfgang Meyer Employee of: Employee of EUROIMMUN AG, Lubeck, Germany, Angeliki Roussakis-Schulze: None declared, Dimitrios Bogdanos: None declared

**DOI:** 10.1136/annrheumdis-2020-eular.5383

**AB0114** IL12P40/IL23P40 BLOCKADE WITH USTEKINUMAB DECREASES THE INFLAMMATORY INfiltrATE AND MODULATES MOLECULAR PATHWAYS IN THE SYNOVIIUM OF PSORIATIC ARTHRITIS PATIENTS

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**Background:** Psoriatic arthritis (PsA) is a chronic inflammatory joint disease within the spondyloarthritides (SpA) spectrum. TNF and IL17/IL23 pathways play a key role in SpA pathogenesis. Blocking of IL12p40/IL23p40 has been shown to effectively reduce disease activity in PsA [1,2]. It is however incompletely understood how IL12p40/IL23p40 blockade affects local inflammatory processes.

**Objectives:** To investigate the cellular and molecular pathways affected by IL12p40/IL23p40 blockade with ustekinumab in PsA patients (pts).

**Methods:** Eleven male PsA pts with at least 1 inflamed knee or ankle joint, who were scheduled to start ustekinumab treatment, were included in a 24-week single-center open-label study. All pts received ustekinumab 45 mg/sc according to standard care at week (W) 0, 4 and 16. Besides clinical outcomes, need of arthroscopic synovial tissue (ST) biopsy samples were obtained from an inflamed knee or ankle joint at baseline (BL), W12 and W24, ST samples were analyzed by immunohistochemistry (IHC), RNA sequencing and real-time quantitative polymerase chain reaction (qPCR) analysis.

**Results:** Paired BL and W12, and paired BL, W12 and W24 ST samples were available of 9 and 6 pts, respectively. Two pts only underwent BL ST sampling (pt refusal; withdrawal after the W12 clinical visit). Two pts were excluded after W12 because of treatment adjustments. Of 1 pt no ST was obtained at W24 due to technical difficulties. Eight pts finished 24 weeks of clinical follow-up. No serious adverse events were observed. At W12 6/11 pts met ACR20, 2/11 met ACR50 and 1/11 met ACR70 improvement criteria, at W24 this was 3/8, 2/8 and 1/8 pts, respectively. Significant improvements between BL and W12 and/or W24 were seen in clinical (TJC, PASI, BASDAI) and serological markers (CRP and ESR).

Table 1. IHC showed a significant decrease in staining macrophages, a biomarker of an inflammatory response in peripheral SpA, of BL [2-3] vs W12 1.5(0-2]=0.020, but not W24 1(0.5-2.5]=ns. Other synovial infiltrating cells were not significantly decreased. Significant downregulation of MMP3 (p=0.047) and IL-23p19 (p=0.046), but not IL6, TNF or IL12p40 were seen with qPCR analysis at W12. RNA seq analysis showed 178 significantly differentially expressed genes between BL and W24 (FDR 0.1). Gene ontology and KEGG terms enrichment analyses identified overrepresentation of MAPK and PI3K-Akt signaling pathways among the down-regulated genes and WNT signalling pathway among the up-regulated genes. Gene expression was confirmed by qPCR analysis.

**Conclusion:** Ustekinumab treatment reduced synovial inflammation and modulated specific molecular pathways, however inflammation was not completely resolved. Future studies comparing histological and gene expression data between different treatments targeting IL17/IL23 axis will show which changes are treatment-specific and which reflect downregulation of local inflammation.

**References:**

Work was financially supported by an unrestricted grand of Janssen Pharmaceutica.

**Disclosure of Interests:** Renée Fiechter: None declared, Henriëtte de Jong: None declared, Leonieke van Mens: None declared, Inka Fluri: None declared, Sander Tas: None declared, Dominique Baeten Employee of: UCB Pharma, Nataliya Yeremenko: None declared, Marleen G.H. van de Sande Grant/research support from: Novartis, Eli lilly, UCB, Jansen, Consultant of: Abbvve, Novartis, Eli lilly, MSD

**DOI:** 10.1136/annrheumdis-2020-eular.4518

**AB0115** SECUKINUMAB THERAPY DOES NOT AFFECT NEUTROPHIL HOST DEFENCE IN PSORIATIC ARTHRITIS

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**Background:** Psoriatic arthritis (PsA) is a chronic inflammatory joint disease associated with disease activity. sICAM-1 is an independent predictor of TNFi response in csDMARDs refractory RA patients.

**Objectives:** To evaluate the role of neutrophils in PsA patients treated with secukinumab.

**Methods:** 30 PsA patients (mean age 58, range 32-71) on secukinumab treatment were included in a single-centre retrospective study. Paired blood samples were obtained at baseline (BL), W12 and W24. Flow cytometry, ELISA and qPCR were used to determine neutrophil subsets, MMP-9, IL-8, myeloperoxidase and the neutrophil extracellular trap marker MBP14.

**Results:** At BL, the number of total neutrophils and neutrophil subsets (CD16+ and CD66b+) were comparable between BL and W12. However, a significant increase in myeloperoxidase (p=0.01) and a decrease in MBP14 (p=0.04) were observed at W24. MMP-9 levels did not change significantly between BL and W12, but increased significantly at W24 (p<0.001).

**Conclusion:** Secukinumab treatment did not affect neutrophil host defence in PsA patients.

**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2020-eular.1689