Conclusion: sICAM-1 and CXCL13 are elevated in RA patients and correlated with disease activity. sICAM-1 is an independent predictor of TNFi response in csDMARDs refractory RA patients.

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7. Spondyloarthritis - etiology, pathogenesis and animal models

AB0113

ANTIBODIES AGAINST HELICOBACTER PYLORI ANTIGENS IN PATIENTS WITH PSORIATIC 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, Lübeck, 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 HC, p=0.017; p54: 66.7% in Ps vs 33.3% in HC, p=0.012; 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.

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AB0114

IL12P40/IL23P40 BLOCKADE WITH USTEKINUMAB DECREASES THE INFLAMMATORY INFILTRATE AND MODULATES MOLECULAR PATHWAYS IN THE SYNOVIUM OF PSORIATIC ARTHRITIS PATIENTS

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Background: Psoriatic arthritis (PsA) is a chronic inflammatory joint disease within the spondyloarthritis (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, needle 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 sublining macrophages, a sensitive biomarker of an inflammatory response in peripheral SpA, of BL 2[1-3] vs W12 1.5[0-2] p=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 W12 (FDR 0.1). Gene ontology and KEGG terms enrichment analyses identified overrepresentation of MAPK and PI3K-Akt signalling pathways among the down-regulated genes and WNT signalling pathway among the up-regulated genes. Gene expression was confirmed by qPCR analysis.

Table 1.

	Baseline (n=11)	Week 12 (n=11)	Week 24 (n=8)	p-value*	p-value**
TJC	1 (0-5)	1 (1-2)	0 (0-0.75)	0.168	0.034
SJC	2 (1-3)	2 (1-3)	1.5 (1-2)	0.394	0.715
VAS patient global	28 (16-55)	22 (12-46)	45 (2.8-50.5)	0.423	0.779
PASI	3.2 (1.2-7.4)	0.8 (0-1.8)	0 (0-2.4)	0.028	0.046
BASDAI	3.2 (2.4-4.7)	1.9 (1-3.8)	2.1 (0.4-4.2)	0.008	0.161
CRP (mg/L)	8.5 (1.7-16.4)	1.6 (0.6-3)	1.0 (0.6-7.0)	0.008	0.012
ESR (mm/hr)	20 (8-35)	6 (2-17)	8 (2-20.5)	0.008	0.017

TJC/SJC tender/swollen joint count; VAS visual analogue scale; PASI psoriasis area severity index; BASDAI Bath Ankylosing Spondylitis Disease Activity Index; CRP C-reactive protein; ESR erythrocyte sedimentation rate;

All values are presented as median (IQR) p-value for the comparison of baseline and week 12 (*) or week 24 (**)

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:

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AB0115

SECUKINUMAB THERAPY DOES NOT AFFECT NEUTROPHIL HOST DEFENCE IN PSORIATIC ARTHRITIS

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