Table. Familial risks and heritability of AS by type of first-degree relative (FDR) and sex

	Overall	Male-male	Male-female	Female-male	Female-female
Relative with AS	Familial risk, odds ratio (95% CI)				
Sibling	19.5 (16.9 - 22.4)	19.9 (16.1 - 24.7)	20.9 (16.0 - 27.4)	20.5 (16.0 - 26.5)	21.0 (13.3 - 33.2)
Parent	19.1 (17.2 - 21.3)	18.3 (15.5 - 21.6)	20.8 (16.3 - 26.4)	14.1 (11.3 - 17.5)	34.2 (26.1 - 44.9)
Child	20.6 (18.2 - 23.3)	20.5 (16.8 - 25.1)	16.0 (12.2 - 21.0)	19.9 (15.0 - 26.4)	26.2 (18.5 - 37.2)
Any FDR	19.4 (18.1 - 20.8)	19.0 (17.2 - 21.1)	18.2 (15.9 - 21.0)	17.7 (15.4 - 20.3)	26.9 (22.0 - 32.9)
>1 relative	68.0 (51.3 - 90.1)				
	Heritability (95% CI)				
Siblings	0.77 (0.73 - 0.80)	0.80 (0.74 - 0.85)	0.73 (0.67 - 0.78)	0.77 (0.72 - 0.83)	0.71 (0.61 - 0.81)

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FRI0370 ACCELERATED EXTRACELLULAR MATRIX REMODELING IN PSORIATIC ARTHRITIS & VALUE OF ECM-RELATED BIOMARKERS

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Background: With our increasing understanding of cross-talk between the immune system (immune cells, cytokines) and extracellular matrix (ECM) of the affected tissues, ECM destruction and turnover has gained a lot of attention in inflammatory arthritis. ECM mainly consists of interstitial matrix (rich in type I and III collagens) and basement membrane (mainly composed of type IV collagen), both of which are affected in inflammatory arthritis. We hypothesize that inflammatory milieu in Psoriatic Arthritis (PsA) leads to accelerated ECM destruction and remodeling, and the release of ECM metabolites into circulation, that can be measured in serum as biomarkers of tissue remodeling and may give insight to pathological process at the tissue level.

Objectives: This prospective study aimed to use serological biomarkers for evaluation of the extent of damage to, and changes in turnover of ECM in patients with PsA and to investigate their relation to inflammatory biomarkers and disease activity.

Methods: Patients with PsA (n=145) fulfilling the CASPAR criteria and above 18 years of age with any disease activity were recruited through outpatient department in Aalborg University Hospital, Denmark. Exclusion criteria were pregnancy, treatment with biological DMARD, or with oral corticosteroids and other comorbidities. Clinical disease parameters were recorded, and blood samples were collected at baseline and 24 weeks follow-up. Inflammation-related ECM remodelling was measured by serological biomarkers of type I, III and IV collagen formation (Pro-C1, Pro-C3 and Pro-C4, respectively) and MMP-mediated degradation (C1M, C3M and C4M respectively). The chronic inflammation was measured by CRPM (MMP degraded metabolite of CRP). All biomarkers were measured by competitive ELISAs and levels were compared to the reference levels of healthy individuals. The biomarker data was log₂ transformed for normalization and standard parametric statistical methods were applied.

Biomarkers (ng/ml)	PsA patients			Reference values*			Statistics	
	Mean	SD	n	Mean	SD	n	Difference	P Value
C1M	50.00	32.18	145	24.8	10.8	29	25.2	<0.0001
C3M	10.94	3.15	145	9.69	2.59	127	1.25	0.0004
C4M2	29.57	10.51	145	21.33	5.64	89	8.24	<0.0001
Pro-C1	79.20	38.85	145	104.77	54.17	119	-25.57	<0.0001
Pro-C3	11.49	3.56	145	11.28	3.56	111	0.21	ns
Pro-C4	304.07	120.17	145	204.88	63.54	127	99.19	<0.0001

Table 1. Comparison of ECM turnover biomarkers in PsA patients (at baseline) and healthy individuals
(*internal laboratory data)

	Log ₂ ΔC1M	Log ₂ ΔC3M	Log ₂ ΔC4M	Log ₂ ΔPro-C1	Log ₂ ΔPro-C3	Log ₂ ΔPro-C4
ΔCRP	0.621±	0.53±	0.341±	-0.062	0.098	0.267±
Log ₂ ΔCRPM	0.436±	0.615±	0.415±	0.097	0.208	0.393±
ΔASDAS	0.202*	0.21*	0.207*	0.042	0.104	0.204*
ΔBASDAI	0.032	0.102	0.148	0.03	0.057	0.117
ΔBASMI	0.015	0.005	0.107	-0.015	-0.061	0.175*
ΔPASI	-0.076	-0.033	-0.001	-0.09	-0.053	0.008
ΔLEI	-0.024	0.027	0.246*	0.108	-0.043	0.094
ΔSPARCC	0	0.152	0.163	0.018	-0.049	0.174
ΔSJC	-0.015	-0.144	0.153	-0.011	0.019	0.062
ΔTJC	-0.039	0.037	0.062	0.006	-0.006	0.077
ΔVAS-Pain	0.07	0.104	0.138	0.075	0.087	0.198*

Results: We found that there was an increase in the degradation of interstitial matrix (represented by C1M and C3M) which was uncompensated with either decreased (Pro-C1) or unaltered (Pro-C3) formation in patients with PsA as compared to healthy individuals. On the contrary, there was a compensated increase in basement membrane remodeling in patients with PsA as represented by increase in both C4M and Pro-C4 in comparison to healthy individuals (Table 1). Interestingly, there was a strong correlation between baseline C4M and Pro-C4 (r=0.703, p<0.0001), and 24-week changes in both biomarkers (r=0.468, p<0.0001) pointing towards coupling between the two processes which was either lost or was weak in case of interstitial collagens. As baseline values and changes in C1M, C3M, C4M and Pro-C4 showed a strong correlation with changes in inflammatory biomarkers, we believe that ECM remodeling in PsA is, at least partly, driven by inflammation. Furthermore, baseline values and changes in these ECM turnover biomarkers correlated positively with changes in composite disease activity scores (Table 2) that may indicate their clinical importance as potential diagnostic or disease activity markers.

Conclusion: Chronic inflammation underlying PsA pathology results in an increased amount of ECM turnover that correlates with clinical progression and can be measured by serological biomarkers.

REFERENCES:

None

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FRI0371 ALTERED EXPRESSION OF LNCRNA NR_003542 AND NR_026756 IN ANKYLOSING SPONDYLITIS MIGHT BE ASSOCIATED WITH THE DISEASE ACTIVITY

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Background: lncRNAs have been recognized as powerful regulators of numerous genes and pathways in the pathogenesis of inflammatory and autoimmune diseases, including SLE, RA, T1DM, MS, autoimmune thyroid disease, Sjögren's syndrome psoriasis, and Crohn's disease[1]. The role of lncRNAs in the pathogenesis of Ankylosing Spondylitis (AS) is just beginning to be investigated

Objectives: To determine whether the altered expression of lncRNA NR_003542 and NR_026756 in peripheral blood of AS patients could provide insights into the pathogenesis of AS.

Methods: Peripheral blood mononuclear cells (PBMCs) lncRNA NR_003542 and NR_026756 expression were measured in 30 active AS (AAS)group, 30 inactive AS (IAS) group and 30 healthy control (HC) subjects. Clinical characteristics of the subjects are summarized in Table1.

Results: 1. Clinical and laboratory characteristics of the three groups (Table 1)