

Table 1. Unadjusted means (SD) of SF-36 PCS and MCS

	Months	RA (mean (SD))	PsA (mean (SD))	p-value
SF-36 PCS	0	31.3 (10.0)	31.6 (9.7)	0.31
	3	36.7 (11.1)	37.3 (11.1)	0.16
	6	37.9 (11.3)	38.1 (10.9)	0.64
SF-36 MCS	0	45.6 (11.6)	46.3 (11.7)	0.08
	3	48.1 (10.9)	48.3 (10.9)	0.60
	6	48.8 (10.8)	49.1 (10.7)	0.49

Table 2. ANCOVA analyses with adjustment for age, gender and years since diagnosis

	Months	Estimated marginal means (95% CI)		P value
		RA	PsA	
SF-36 PCS	0	32.3 (31.9–32.7)	31.6 (31.0–32.2)	0.06
	3	37.9 (37.4–38.4)	36.8 (36.1–37.6)	0.02
	6	39.3 (38.8–39.9)	37.6 (36.9–38.4)	0.001
SF-36 MCS	0	46.3 (45.8–46.8)	46.2 (45.5–46.9)	0.90
	3	48.3 (47.8–48.9)	48.3 (47.6–49.0)	0.92
	6	49.5 (49.0–50.0)	48.9 (48.1–49.7)	0.21

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OP0110 ASSOCIATION OF PHARMACOLOGICAL BIOMARKERS WITH TREATMENT RESPONSE AND LONG-TERM DISABILITY IN PATIENTS WITH PSORIATIC ARTHRITIS: RESULTS FROM OUTPASS

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Background: Up to 40% of patients with inflammatory arthritis on TNF- α inhibitor (TNFi) treatment fail to respond either due to primary inefficacy or loss of response. One explanation is immunogenicity leading to the development of anti-drug antibodies (ADAb) and subsequent low drug levels. Few data exist on whether such pharmacological tests correlate with treatment response in psoriatic arthritis (PsA). The clinical utility of whether such tests should be incorporated into practice is in question.

Objectives: To identify (i) whether the presence of ADABs/drug levels predict treatment response and disability in TNFi-treated PsA patients (ii) the factors associated with drug levels (iii) a drug level threshold for optimal therapeutic response.

Methods: 75 patients were available from the Outcomes of Treatment in PsA Study Syndicate (OUTPASS) [n=49 adalimumab; n=26 etanercept], a national UK prospective observational cohort. Serum samples were collected at 3, 6 and 12 months following initiation of TNFi therapy. ADABs were measured using radioimmunoassay (RIA) and random (non-trough) drug levels using ELISA assays at 3, 6 and 12 months. Disease activity (DAS28) scores were measured at each visit. Patient self-reported adherence to TNFi was measured at each time-point. Generalised estimating equation (GEE) was used to test the association between ADABs and drug levels, both biomarkers and treatment response [as assessed by change in DAS28 score between pre-treatment and 12 months post-treatment (Δ DAS28)]. Health assessment Questionnaire (HAQ) and the association between longitudinal/baseline factors with drug levels.

Results: 264 serial samples were suitable for pharmacological testing (n=174 adalimumab; n=90 etanercept). Mean age was 51 \pm 12 years; 61% were female; median BMI 28.9 (IQR 26.0–34.9). 20% (n=10/49) of adalimumab-treated patients were positive for ADABs, but none were detected in etanercept-treated patients. There was no significant association between etanercept drug levels and Δ DAS over 12 months [β = -0.039 (95% CI -0.31, 0.23), p=0.77]. Using GEE, adalimumab drug levels were significantly associated with Δ DAS28 over 12 months [β =0.055 (95% CI: 0.011, 0.099) p=0.014] and inversely with HAQ scores over 12 months [β = -0.022 (95% CI: -0.043, -0.00063)]. Δ DAS28 was not independently associated with ADAB level [β = -0.0015 (95% CI: -0.0031, 0.000047), p=0.057]. Adalimumab concentrations between 4.5–8.5 mg/L were associated with an optimal treatment response at 6 months using concentration-effect curves. Factors that were significantly associated with adalimumab drug levels were ADAB level [β = -0.0073 (95% CI: -0.0014, 0.18), p<0.0001] and BMI [β = -0.15 (-0.29, -0.00450, p=0.043] in the final GEE model (adjusting for age, gender, adherence, BMI).

Conclusions: TNFi drug-level testing in adalimumab-initiated PsA patients may be useful in determining treatment response and disability over 12 months; interestingly, both the presence of ADABs and BMI were inversely associated with

drug levels. Identification of a drug level threshold for optimal response may help tailor adalimumab therapy for PsA patients in the future.

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OP0111 THE ASSOCIATION BETWEEN SONOGRAPHIC ENTHESITIS AND RADIOGRAPHIC JOINT DAMAGE IN PSORIATIC ARTHRITIS

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Background: Enthesitis is a common clinical finding and a key pathogenic feature in psoriatic arthritis (PsA). Ultrasound is emerging as a preferred method to assess enthesitis. Little is known about the relation between the presence of enthesitis and the severity of joint damage in patients with PsA.

Objectives: Our objective was to examine the association between sonographic enthesitis and the severity of radiographic features of damage in the peripheral and axial joints in PsA.

Methods: A cross-sectional study was conducted in consecutive patients with PsA. The Madrid Sonography Enthesitis Index (MASEI) scoring system was used to quantify the extent of sonographic enthesal abnormalities in 12 enthesal sites. Total MASEI was further categorized into: bone scores (enthesophytes, erosions) and soft tissue scores (structural changes, vascularization, bursitis). Radiographic joint damage in the peripheral joints and spine was assessed independently of the ultrasound results using the modified Steinbrocker score, Modified New York Criteria for sacroiliitis and the modified Stoke Ankylosing Spondylitis Spine Score (mSASSS). Additionally, the presence of ankylosis, arthritis mutilans and periostitis in the hands or feet was determined. Linear and logistic regression models were used to assess the association between MASEI score and the radiographic features of joint damage after controlling for age, sex, BMI, PsA duration and the use of DMARDs and biologic medications.

Results: 222 patients were included (58% men) with mean (s.d.) age of 55.9 (12.9) years and PsA duration of 16.7 (12.4) years. The mean MASEI score was 15.6 (12.6). The mean modified Steinbrocker score was 18.1 (32.3), mSASSS was 1.7 (7.3) and 37% had sacroiliitis. Multivariate regression analyses found an association between higher scores of MASEI scores and peripheral joint damage: modified Steinbrocker score (β 9.26, p<0.0001), joint ankylosis (Odds Ratio (OR) 2.09, p=0.0001) and arthritis mutilans (OR 1.73, p=0.005). The association between MASEI scores and periostitis was of borderline statistical significance (OR 1.29, p=0.06). Similarly, an association was found in multivariate analyses between higher MASEI scores and axial damage as measured by mSASSS (β 1.55, p<0.0001) and sacroiliitis (OR 1.36, p=0.02). Sub-analysis showed that the MASEI bone score were more strongly associated with radiographic damage outcomes than the MASEI soft tissue score.

Conclusions: The severity of sonographic enthesitis is a marker of radiographic peripheral and axial joint damage in PsA. The association was found with both erosive and bone formation lesions. These findings highlight the potential role of enthesitis in the pathogenesis of articular damage in PsA.

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OP0112 IN PSORIATIC ARTHRITIS FATIGUE IS DRIVEN BY INFLAMMATION, DISEASE DURATION, AND CHRONIC PAIN: AN OBSERVATIONAL DANBIO REGISTRY STUDY

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Background: Fatigue has been identified as one of the most significant symptoms and an outcome of great importance to patients with psoriatic arthritis (PsA), but the association between underlying components of experienced fatigue and the disease has been sparsely investigated. [1]

Objectives: To describe the degree of fatigue in a PsA population. Secondly, to explore which components of inflammation and non-inflammatory factors contribute to experienced fatigue.

Methods: The study was designed as a cross-sectional survey including patients

registered in DANBIO, the Danish nationwide rheumatologic registry, from December 2013 to June 2014. Principal component analysis was used to identify clustering factors associated with fatigue

Results: A total of 1,062 PsA patients were included in the study. The median Visual Analog Scale (VAS) fatigue score was 57mm. Patients with moderate to severe fatigue (VAS score ≥ 57) had higher scores of pain, DAS28, HAQ, patient global assessment, and more tender and swollen joints ($p < 0.001$) (Table 1). In the principal component analysis the clinical co-variables were reduced to 3 components explaining 63% of experienced fatigue (figure 1); The first component, contributing to 31%, was mainly constituted by inflammatory factors as swollen and tender joints, doctors-global evaluation, higher CRP, and pain score, whereas the second component mainly consisted of contributions from age and disease duration, explaining 17% of experienced fatigue. The third component, contributing to 15%, consisted of patient pain, tender joint count, increasing age, and by concomitant low CRP. The remaining 37% was considered residuals.

Characteristics	Fatigue Non to mild VAS score <57 n=520		Fatigue Moderate to severe VAS score ≥ 57 n=542		p-value
	n	n	n	n	
Female, n (%)	253 (48.7%)	520	358 (66.1%)	542	<0.001
Age, yrs	53.0 (44.0–62.0)	520	52.0 (42.8–60.0)	542	0.070
Disease duration, yrs	6.0 (3.0–11.5)	449	5.0 (2.0–10.0)	456	0.022
C-reactive protein, mg/L	3.0 (1.0–6.0)	421	4.0 (2.0–7.0)	464	0.008
Patient global assessment, 0–100 mmVAS	27.0 (15.0–43.0)	520	75.5 (61.0–86.0)	542	<0.001
Doctors global assessment, 5-grade Likert scale	7.0 (3.0–15.0)	432	14.0 (7.0–14.0)	438	<0.001
PDQ pain score	9.0 (6.0–14.0)	520	17.0 (13.0–23.0)	542	<0.001
DAS28-CRP	2.3 (1.8–2.9)	400	3.5 (2.6–4.4)	418	<0.001
HAQ, 0–3	0.4 (0.1–0.8)	507	1.1 (0.8–1.6)	530	<0.001

All values are median (Q1-Q3) unless otherwise indicated. VAS; visual analogue scale, PDQ; pain assessment questionnaire, DAS28-CRP; disease activity score-C-reactive protein, HAQ; health assessment questionnaire.

Component 1

Clinical inflammatory manifestations

- Pain score: 0.41
- Swollen joints: 0.77
- Tender joints: 0.73
- Doctors VAS: 0.82
- Crp: 0.41
- Disease duration: -0.08
- Age: -0.05

Component 2

Chronification

- Pain score: -0.38
- Swollen joints: 0.21
- Tender joints: -0.01
- Doctors VAS: 0.07
- Crp: 0.08
- Disease duration: 0.74
- Age: 0.66

Component 3

Chronic pain

- Pain score: 0.59
- Swollen joints: -0.08
- Tender joints: 0.35
- Doctors VAS: -0.20
- Crp: -0.61
- Disease duration: 0.04
- Age: 0.43

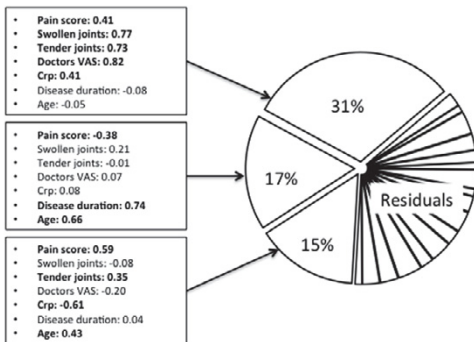


Figure 1

Conclusions: Clinical inflammatory disease activity, chronification, as well as pain in the absence of inflammation were all identified as important factors explaining 63% of moderate to severe fatigue in the current PsA population.

References:

[1] Orbai AM, Mease PJ, de WM et al. Report of the GRAPPA-OMERACT Psoriatic Arthritis Working Group from the GRAPPA 2015 Annual Meeting. J Rheumatol 43; 965–9.

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OP0113 METABOLIC SYNDROME AND LIVER STIFFNESS IN PSORIATIC ARTHRITIS AND PSORIASIS PATIENTS: A CASE-CONTROL STUDY

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Background: Psoriatic arthritis (PsA) and psoriasis (PsO) are commonly associated to various comorbidities, among which metabolic syndrome (MetS) has been demonstrated to be more prevalent in these groups with respect to the general population. However, few data are available regarding the comparison between PsA/PsO. Besides, a possible consequence of MetS is the development of a non-alcoholic fatty liver disease (NAFLD), which can progress to fibrosis, the latter rarely assessed in PsA/PsO.

Objectives: The aim of this case-control study was: 1) to compare prevalence of MetS in PsA and PsO 2) to evaluate the presence of liver fibrosis in these two groups using hepatic elastography.

Methods: Forty-three consecutive PsA patients classified according to CIASsification criteria for Psoriatic Arthritis (CASPAR), and 33 consecutive PsO patients without history/manifestations of arthritis, attending the Rheumatology and Dermatology Units of University of Padua, were studied. Exclusion criteria were: conditions which may cause liver fibrosis other than NALFD (eg viral hepatitis, autoimmune or genetic liver disease), alcohol consumption >20 grams/day, active smoking, daily use of non-steroidal anti-inflammatory drugs. Anamnestic, laboratory (cholesterol, triglycerides, uric acid, fasting glucose, insulin, albumin, transaminase) and metrological (blood pressure, waist circumference, height, weight) data were collected. MetS was defined according to the criteria of National Cholesterol Education Program's Adult Treatment Panel III report. Insulin resistance was quantified through HOMA (Homeostatic Model Assessment). All patients underwent hepatic elastography to evaluate liver stiffness; values >7 kPa were taken as indicator of liver fibrosis. PsO severity was assessed through Psoriasis area severity index (PASI). Differences in variables between PsA/PsO were compared through non parametric Mann-Whitney test, and Chi-square test for categorical variables. Correlations between variables were evaluated through Spearman test.

Results: PsA and PsO patients showed similar characteristics (mean age 60,2±8,4 vs 54,5±19,6 years, 74,4% vs 63% males, arthritis/PsO duration 12,6±8,5 vs 18,2±14,2 years). The only variables which differ in PsA/PsO groups were Body Mass Index (BMI) (25,7±3,4 vs 29,1±6,3), PASI (5±4,6 e 1,5±2,5) and serum uric acid (4,9±1,5 vs 5,7±1,4 mg/dL), all higher in PsO (p-values 0,0092, 0,0355 and 0,0001 respectively). Prevalence of MetS and liver fibrosis in the 2 groups were similar: 34,9% and 30,8% in PsA vs 33,3% and 27,6% in PsO (p=ns). Among all correlation studied, only serum uric acid, liver stiffness and PASI correlated with other variables (Table). Most interestingly, liver stiffness very well correlated with serum uric acid in PsO ($p < 0,0001$ $r=0,73$).

Table. Correlations found between serum uric acid, liver stiffness and PASI with other variables studied

Variables	Uric acid		Liver Stiffness		PASI		
	PsA	PsO	PsA	PsO	Variables	PsA	
BMI	n.s	p=0,019 r=0,41	BMI	n.s	p=0,020 r=0,43	Waist circumference	p=0,019 r=0,36
Waist circumference	n.s	p<0,0001 r=0,59	Waist circumference	n.s	p<0,0001 r=0,69	HDL cholesterol	p=0,012 r=-0,38
PASI	p=0,001 r=0,47	n.s	Fasting glucose	p=0,037 r=0,33	n.s	HOMA	p=0,044 r=0,31
HDL cholesterol	p=0,004 r=-0,42	p=0,015 r=-0,42	Glycated hemoglobin	p=0,027 r=0,35	p=0,022 r=0,42	Serum uric acid	p=0,001 r=0,47
Triglycerides	n.s	p=0,005 r=0,48	Insulin	p=0,014 r=0,38	p<0,0001 r=0,64		
Fasting glucose	n.s	p<0,0001 r=0,66	HOMA	p=0,018 r=0,37	p<0,0001 r=0,71		
Serum insulin	n.s	p<0,0001 r=0,74	Serum uric acid	n.s	p<0,0001 r=0,73		
Glycated hemoglobin	n.s	p<0,0001 r=0,63	Triglycerides	n.s	p=0,016 r=0,44		
HOMA	p=0,025 r=0,34	p<0,0001 r=0,80					
Liver stiffness	n.s	p<0,0001 r=0,73					

BMI=Body mass Index; HDL=High Density Lipoprotein; PASI= Psoriasis area severity index; HOMA= homeostatic model assessment

Conclusions: We observed a similar prevalence of MetS and hepatic stiffness in PsA and PsO. The correlation found between uric acid level and hepatic stiffness could lie on the fact that uric acid seems to favour insulin resistance, hypertension, dyslipidemia and other MetS risk factors. MetS could be therefore one of the major determinants to liver fibrosis in PsA and PsO, thus highlighting how comorbidities are not only coexisting conditions, but are strongly linked to each other and need to be treated as well as the skin and joint aspect.

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