Abstract THU0300 – Table 1. Fixed-effect NMA data for ACR20 and Δ HAQ-DI in TNFi-N patients with PsA – comparison of bDMARDs or apremilast vs tofacitinib 5 and 10 mg BID^{a,b}

Treatment	ACR20, OR (95% CrI) ^{a,c}		Difference in Δ HAQ-DI (95% CrI) ^{a,c}	
	vs tofacitinib 5 mg BID	vs tofacitinib 10 mg BID	vs tofacitinib 5 mg BID	vs tofacitinib 10 mg BID
Abatacept 125 mg	1.0 (0.4, 2.4)	0.7 (0.3, 1.6)	NA	NA
Adalimumab 40 mg Q2W	1.4 (0.8, 2.2)	0.9 (0.5, 1.5)	-0.1 (-0.2, 0.1)	0.0 (-0.1, 0.1)
Apremilast 30 mg BID	0.9 (0.5, 1.6)	0.6 (0.3, 1.0)	0.04 (-0.1, 0.2)	0.1 (-0.1, 0.3)
Apremilast 20 mg BID	0.7 (0.4, 1.3)	0.5 (0.3, 0.8)	NA	NA
Certolizumab 400 mg Q4W + 200 mg Q2W	1.6 (0.8, 3.2)	1.1 (0.5, 2.1)	NA	NA
Etanercept 25 mg BW	3.7 (1.7, 8.1)	2.4 (1.1, 5.3)	-0.9 (-1.3, -0.6)	-0.9 (-1.2, -0.5)
Golimumab 50 mg Q4W	4.9 (1.7, 19.4)	2.7 (1.1, 6.9)	-0.1 (-0.2, 0.1)	-0.03 (-0.2, 0.1)
Golimumab 100 mg Q4W	3.2 (1.4, 8.4)	2.1 (0.9, 5.5)	-0.2 (-0.3, 0.02)	-0.1 (-0.3, 0.1)
Infliximab 5 mg/kg	3.6 (1.7, 7.9)	2.4 (1.1, 5.0)	NA	NA
Ixekizumab 80 mg Q4W	1.4 (0.7, 2.7)	0.9 (0.5, 1.8)	-0.1 (-0.2, 0.1)	-0.01 (-0.2, 0.2)
Ixekizumab 80 mg Q2W	1.6 (0.8, 3.1)	1.0 (0.5, 2.1)	-0.2 (-0.3, 0.01)	-0.1 (-0.3, 0.1)
Secukinumab 150 mg QW three Q4W	2.5 (1.2, 4.9)	1.6 (0.8, 3.3)	-0.01 (-0.2, 0.2)	0.04 (-0.2, 0.3)
Secukinumab 300 mg QW then Q4W	2.2 (1.0, 5.3)	1.5 (0.6, 3.5)	-0.1 (-0.3, 0.2)	0.0 (-0.2, 0.2)
Ustekinumab 45 mg Q12W	1.0 (0.5, 1.8)	0.6 (0.3, 1.2)	-0.03 (-0.2, 0.1)	0.02 (-0.1, 0.2)
Ustekinumab 90 mg Q12W	1.2 (0.6, 2.3)	0.8 (0.4, 1.5)	-0.1 (-0.2, 0.1)	-0.02 (-0.2, 0.1)

^aResults which do not include the null value are bold and italicized

^bSLR-identified clinical manuscripts published between earliest available record from each database and 9 Oct 2017

^cA binary outcome, where an OR of 1 indicates that there was no difference in the odds of an ACR20 response between tofacitinib and comparator, an OR <1 favours tofacitinib and an OR >1 favours comparator

^dA continuous outcome (higher score indicates worse disability), where a value of 0 indicates that there was no difference in the mean change in HAQ-DI from baseline between tofacitinib and comparator, a difference <0 favours comparator and a difference >0 favours tofacitinib

^eTo ensure robustness, each outcome was 50 000 burn-in and 150 000 samples (from three chains), and it was confirmed that all models converged

^fSLR-identified study population arms (including placebo arms) ranged from n=30 to n=219 for ACR20

^gSLR-identified study population arms (including placebo arms) ranged from n=30 to n=206 for Δ HAQ-DI

ACR20, American College of Rheumatology 20 response; bDMARD, biologic disease-modifying antirheumatic drug; BID, twice daily; BW, bi-weekly; CrI, credible interval; HAQ-DI, Health Assessment Questionnaire-Disability Index; NA, not applicable; NMA, network meta-analysis; OR, odds ratio; PsA, psoriatic arthritis; Q2W, every 2 weeks; Q4W, every 4 weeks; SLR, systematic literature review; TNFi-N, tumour necrosis factor inhibitor-naïve

Conclusions: Based on the NMA of published RCTs in TNFi-N patients with PsA, tofacitinib 5 and 10 mg BID had similar efficacy vs many, but not all, bDMARDs and APR in improving ACR20 and Δ HAQ-DI.

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THU0301 A NOVEL ROLE FOR THE PSORIATIC ARTHRITIS IMPACT OF DISEASE QUESTIONNAIRE (PSAID)

K. Johnson, J. Ye, V. Chandran, D. Gladman. *Rheumatology, Toronto Western Hospital, University of Toronto, Toronto, Canada*

Background: Psoriatic arthritis (PsA) is an inflammatory arthritis that occurs in about 30% of patients with Psoriasis (Ps). Recently, a new Patient Reported Outcome Measure (PROM), Psoriatic Arthritis Impact of Disease (PsAID) was specifically developed for PsA Patients. The two versions of the PsAID, PsAID-9 and PsAID-12, are scored on a Numeric Rating Scale (NRS) of 0–10. The Minimal Disease Activity (MDA) is a composite outcome measure for PsA patients, which uses the Health Assessment Questionnaire (HAQ) as one criterion. However, the HAQ does not correlate well with measures of disease activity as PsA disease duration increases, and its use in the assessment of disease activity has been questioned.

Objectives: Our objectives were to 1) validate the PsAID within our patient cohort, 2) determine if the PsAID can replace any of the other PROMs administered in the clinic, and 3) determine if the PsAID can replace the HAQ in the MDA.

Methods: Patients were recruited from a large psoriatic arthritis clinic. All patients completed the PsAID and 10 other PROMs. Various measures of disease activity were recorded by a physician at each visit. Descriptive statistics (mean, median, SD, min, max) were calculated for all PROMs. PsAID cut-offs for use in the MDA were generated based on Remission (REM) and Low Disease Activity (LDA) disease states in the Clinical Disease Activity for Psoriatic Arthritis Index (cDAPSA).

Results: 115 patients completed the PsAID. There were 70 males, 45 females, with a mean PsA duration of 18.7 (± 11.6) years. Mean scores of PsAID-9 and PsAID-12 were 3.4 (± 2.4) and 3.2 (± 2.3) respectively. The PsAID correlated moderately well with 9 of the PROMs administered in the clinic ($R^2=0.51-0.78$). Four

PsAID cutoffs were generated for use in the MDA: REM PsAID-9, REM PsAID-12, LDA PsAID-9, and LDA PsAID-12. All four versions of the PsAID MDAs had a sensitivity greater than 85% with the HAQ MDA, and three versions of the PsAID MDA had a specificity greater than 85% with the HAQ MDA.

Conclusions: Our cohort had lower mean PsAID scores than previously reported series suggesting that our patients are monitored carefully. The only moderate correlations with other PROMs suggest that the PsAID cannot replace any of these PROMs. The high sensitivity and specificity of the PsAID MDA with the HAQ MDA suggest that the PsAID is an effective replacement for the HAQ in the MDA.

Disclosure of Interest: None declared

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THU0302 SURVIVAL OF DISEASE-MODIFYING DRUGS (DMARD) IN PATIENTS WITH RECENT DIAGNOSIS OF PSORIATIC ARTHRITIS IN DAILY CLINICAL PRACTICE

^{1,2,3}D. Freitas Nuñez, L. León², Z. Rosales^{1,2}, J. Font¹, C. Lajas¹, E. Pato¹, L. Rodríguez^{1,2}, B. Fernández¹, J.-A. Jover¹, L. Abasolo^{1,2}. ¹Rheumatology, Hospital Clínico San Carlos; ²Fundación para la Investigación Biomedica; ³Complutense University, Madrid, Spain

Background: Psoriatic arthritis (PsA) is a chronic inflammatory disease that benefits from DMARDs, in this regard knowing more about these therapies is a great step forward in the management of these patients in daily clinical practice.

Objectives: To evaluate the survival of DMARDs used in recent diagnosis PsA patients as well as the causes of discontinuation and to analyse the possible associated factors.

Methods: Retrospective longitudinal observational study. **Subjects:** Inception cohort of patients from January 2010 to , December 2014 and followed up to December 2016, diagnosed with PsA according to ICD-10 code. **Main outcome:** discontinuation of conventional synthetic DMARDs (csDMARDs) and biological originator DMARDs (boDMARDs) due to: Adverse drug reactions (ADRs); Improvement or remission; Inefficacy; Patient's decision and Physician's decision. **Covariables:** sociodemographic and clinical. **Statistical analysis:** To estimate DMARDs discontinuation rates, survival techniques were used, expressing the incidence rate (IR) per 100 patients*year with their respective CI at 95%. Multivariate Cox regression models were performed to analyse the factors associated with DMARDs discontinuation and the results were expressed in Hazard ratio (HR) and 95% CI.

Results: 191 patients with recent diagnosis of PsA were included, with a 379.70 Patients*year follow-up. 50.3% were male, the mean age at diagnosis was 50 ± 14.6 years old. 46.6% of the patients had a history of cutaneous psoriasis. The HLA-B27 was positive in 20% of patients. 50% of the patients started a DMARDs at the first visit. Throughout the follow-up, all patients received csDMARDs and 23 used boDMARDs. The median DMARD per patient was 2^{1,3}. Methotrexate (MTX) was the most used drug 69.7%. According to the treatment regimen, 30% were on combination therapy, the most frequent was antiTNF + MTX (33%). 103 discontinuations were recorded with a IR 27.13 [22.36–32.90] within these, 44 were related with ADRs (IR 11.59 [8.62–15.57]), 24 (IR 6.32 [3.58–11.13]) were due to inefficiency, 9 (IR 2.37 [1.23–4.55]) were registered after remission, 12 (IR 3.16 [1.79–5.56]) by decision of the patient and 12 (IR 3.16 [1.79–5.56]) by doctor's decision. The DMARDs median survival was 1.8 years [1.4–2.7]. Table 1 shows the discontinuation rates for each type of DMARDs and the multivariate analysis for the factors associated with DMARDs discontinuation is in table 2.

Table 1:

Table 1	Patients*year	Events(n)	IR	95%CI
MTX orally	243.46	53	21.77	16.63–28.49
Sc MTX	49.90	18	36.07	22.73–57.25
Salazopyrine	84.80	30	35.37	24.73–50.59
Leflunomide	21.83	10	45.80	24.64–85.13
Antimalarials	25.34	8	31.56	15.78–63.12
boDMARDs	57.56	25	43.43	29.35–64.28

Table 2

	HR	95% CI	P
Women	1.51	0.61–3.09	0.436
Age at diagnosis	0.97	0.97–1.01	0.267
Distress level	1.87	1.00–3.48	0.049
C Reactive protein ≥ 1.05 gr/dL	1.85	0.99–3.48	0.055
Combination therapy	2.36	1.46–3.80	0.000
boDMARDs	1.05	0.52–2.11	0.886
MTX vs rest DMARDs	0.53	0.31–0.91	0.021

Conclusions: In our study, the DMARD discontinuation rate was 27.13, mainly related with ADRs. We have also found some psychological, clinical and therapy regimen factors that can modify the DMARDs survival on PsA. We observed that MTX, presented the longest survival independent of the rest of the factors.

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