were biologic-naïve or prior TNF α inhibitor (TNFi)-treated (DISCOVER-1), GUS 100 mg, given every 4/8 weeks (Q4W/Q8W), demonstrated efficacy for joint & skin symptoms, physical function & quality of life vs PBO; AEs were consistent with GUS safety in PsO.

Objectives: Assess GUS efficacy & safety in PsA through 1 year.

Methods: Adults with active PsA (≥ 3 swollen ≥ 3 tender joints; CRP ≥ 0.3 mg/dL) despite standard therapies were eligible. Approx. 30% of pts could have previously received ≤ 2 TNFi. Pts were randomized 1:1:1, stratified by W0 DMARD [Y/N] & prior TNFi (Y/N) use, to GUS 100mg Q4W; GUS 100 mg at W0, W4 & Q8W; or PBO. At W24, PBO pts crossed over to GUS 100 mg Q4W (PBO X Q4W). W48 marked the last dose of study agent. ACR response rates at W52, based on nonresponder imputation (NRI) for missing data and as observed in pts still on study agent at W24, are shown. Observed data for additional endpoints are shown. AEs through W60 are reported.

Results: 362/381 (95%) randomized pts continued study agent at W24 (125 Q4W, 123 Q8W, 114 PBO X Q4W), 347/381 (91%) pts completed treatment & 343/381 (90%) completed study. NRI ACR20 response rates were maintained at W52 (Q4W 73%, Q8W 60%; Fig 1A). Similar responses patterns were seen for the more stringent ACR50/70 criteria (Fig 1C,E). Observed ACR responses, overall (Fig. 1B,D,F) and in pts with (Fig 2A,C,E) & without (Fig 2B,D,F) prior TNFi use, were also maintained at W52. Improvements in other clinical outcomes were also maintained at W52, and responses for pts crossing over from PBO X Q4W at W24 were generally consistent with other GUS-treated pts by W52 (Table 1). Through W24, 4 (2%) GUS- and 5 (4%) PBO-treated pts had serious AEs; no GUS-treated and 2 (2%) PBO-treated pts had a serious infection. Through W60, serious AEs and serious infections occurred in 4% & 1%, respectively, of all 369 GUS-treated pts; no GUS-treated pt died or had IBD, opportunistic infections or active TB, or anaphylactic or serum sickness-like reactions.

Table 1. Observed Efficacy¹

	GUS Q4W		GUS Q8W		PBO (W0-24) X (W24-52)	
	W24	W52	W24	W52	W24	W52
Data are % unless otherwise stated						
Dactylitis at W0, n	37	37	49	44	47	43
Resolution	64.9	78.4	67.3	79.5	61.7	81.4
Enthesitis at W0, n	71	70	71	64	71	63
Resolution	49.3	62.9	40.8	56.3	31.0	69.8
$\geq 3\%$ BSA psoriasis, IGA ≥ 2 at W0, n	88	88	81	75	68	66
IGA 0/1 + ≥ 2 -grade decrease	76.1	83.0	58.0	69.3	17.6	81.5 ²
PASI75	87.5	94.3	76.5	80.0	20.6	84.8
PASI90	63.6	76.1	50.6	66.7	13.2	72.7
PASI100	45.5	64.8	25.9	48.0	7.4	62.1
HAQ-DI, n	125	124	123	114	114	104
Mean change	-0.4	-0.5	-0.3	-0.4	-0.1	-0.4
SF-36 scores, n (mean change)	124	124	123	114	114	104
Physical Component - PCS	6.6	8.5	6.5	7.3	2.7	6.9
Mental Component - MCS	3.8	4.9	3.0	5.1	1.8	4.2
MDA, n	125	124	123	112	114	103
MDA response	31.2	40.3	23.6	33.9	12.3	31.1
VLDA, n	125	124	123	114	113	104
VLDA response	9.6	16.9	4.1	12.3	1.8	14.4

¹Randomized pts still on study agent at W24; 2n=65

Conclusion: GUS Q4W & Q8W maintained improvements in joint symptoms through 1 year in pts with active PsA who were biologic-naïve or previously TNFi-treated. In pts continuing in the study, improvements in skin symptoms, dactylitis, enthesitis, physical function & quality of life were also maintained through 1 year. GUS 100 mg Q4W & Q8W were safe and well-tolerated through study completion and consistent with GUS safety in PsO.¹

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[1] <https://www.janssenlabels.com/package-insert/product-monograph/prescribing-information/TREMFYA-pi.pdf>.

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SAT0398

PERSISTENCE OF USTEKINUMAB (UST) OR TNF INHIBITOR (TNFi) TREATMENT IN PSORIATIC ARTHRITIS (PSA): INSIGHTS FROM THE LARGE, PROSPECTIVE, MULTINATIONAL, REAL-WORLD PSABIO COHORT

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Figure 1. Kaplan-Meier graphs showing treatment persistence for a) UST vs TNFi; b) line of treatment; c) UST/MTX and persistence of UST at baseline; d) UST/MTX and extent of skin involvement at baseline

Patients who did not stop/switch initial treatment were censored

Background: Several biologic DMARDs (bDMARDs) exist for PsA, TNFi and UST being the earliest on European markets. When bDMARDs are insufficiently effective, later-line bDMARDs typically have shorter persistence. Treatment persistence reflects a mix of effectiveness and adverse events (AEs), and persistence data are limited in PsA.

Objectives: Comparative analysis of 1-year persistence of UST and TNFi within the prospective PsABio cohort.

Methods: PsABio is an observational, multinational study of PsA patients (pts) treated with 1st to 3rd line UST or TNFi at their rheumatologist's discretion.¹ Treatment persistence (up to 15 months of follow-up) was defined as time between start of first bDMARD treatment in PsABio, and either stop or switch to another bDMARD, or withdrawal.

Persistence of UST and TNFi is shown by Kaplan-Meier curves and compared using Cox regression analysis, with propensity score (PS) to adjust for baseline imbalanced demographic and disease-related covariates (age, sex, bDMARD line, BMI, Clinical Disease Activity index for Psoriatic Arthritis [cDAPSA], 12-item PsA Impact of Disease [PsAID-12], Fibromyalgia Rapid Screening Tool [FIRST] score, co-treatments with MTX, NSAIDs, glucocorticoids, cardiovascular/metabolic comorbidities, dactylitis, enthesitis and body surface area [BSA]). Factors including concomitant MTX use and skin involvement: <3%, 3–10% and >10%, were added to the Cox model to investigate their impact on the PS-adjusted treatment effect.

Results: Of 438 and 455 pts who started UST and TNF, respectively, 121 (28%) and 134 (29%) stopped or switched treatment before Month 15, with differences (as expected) according to treatment line (Fig. 1a, b). Reasons for stop/switch were related to safety/AEs in 12% (UST) and 28% (TNFi), and effectiveness (joints, nails or skin) in 77% (UST) and 69% (TNFi) of pts.

The observed mean time on drug was 397 days for UST and 385 days for TNFi pts (1st line 410/397 days, 2nd 390/382 days, 3rd 381/338 days). Fig. 1b shows similar persistence for all drugs and treatment lines, except for lower persistence in TNFi 3rd line vs 1st/2nd. In PS-adjusted Cox analysis, no statistically significant difference between UST and TNFi persistence was seen; hazard ratio (HR; 95% CI) for stop/switch bDMARD (UST vs TNFi) was 0.82 (0.60, 1.13). In the model, bDMARD monotherapy (without MTX) and extensive skin involvement (BSA >10%), showed significantly better persistence for UST (HR 0.61 [0.42, 0.90] and 0.41 [0.19, 0.89] respectively; unadjusted Kaplan-Meier graphs shown in Fig. 1c, d). MTX co-therapy and low BSA did not affect the PS-adjusted treatment effect. Other factors added to the PS-adjusted Cox model did not show significant effects.

Conclusion: In this real-world PsA cohort undergoing bDMARD treatment, persistence was generally comparable for UST and TNFi, but some clinical situations led to better drug persistence with UST compared to TNFi – particularly monotherapy, more extensive skin involvement, and in 3rd-line treatment. Our data emphasise the importance of skin involvement for pts with PsA.

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[1] Gossec L, et al. *Ann Rheum Dis*. 2018;77(suppl 2):Abstract AB0928

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SAT0399

RISK OF OSTEOARTHRITIS IN AN INCIDENT COHORT OF PEOPLE WITH PSORIATIC ARTHRITIS: A POPULATION-BASED COHORT STUDY

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Background: Psoriatic arthritis (PsA) is a chronic inflammatory arthritis that causes pain, stiffness and swelling around the joints. PsA is reported to affect between 10 and 40% of individuals with psoriasis¹ and in the majority of patients presents after, or synchronously with, psoriasis onset.² Osteoarthritis (OA) is a common form of non-inflammatory arthritis related to joint degeneration and typically commences late in the fifth decade. PsA and OA have long been considered two distinct arthropathies, however they do have some overlapping features and symptoms and in certain circumstances it can be difficult to differentiate between them, particularly in the small joints of the hands or spine.³

Objectives: To determine the risk of a diagnosis of osteoarthritis in psoriatic arthritis patients compared to patients with psoriasis and a general population cohort.

Methods: Incident PsA patients aged 18–89 years at diagnosis were identified from the UK Clinical Practice Research Datalink between 1998 and 2014. All PsA patients were matched to a cohort of patients with psoriasis and a general population cohort (with no psoriasis or PsA) at a 1:4 ratio based on index date, year of birth, sex and general practice. The baseline prevalence of OA of any site was calculated as a percentage for each study cohort and then those prevalent cases were excluded from the numerators and denominators of the incident calculations. The incidence of OA was calculated and relative risks (RR_{adj}), adjusting for body mass index (BMI), were calculated using conditional Poisson regression.

Results: In total, 6,783 incident PsA patients were identified. The baseline prevalence of OA ranged from 22.1% (CI₉₅ 21.1–23.1) in the PsA cohort to 12.6% (CI₉₅ 12.2–13.0) and 11.0% (CI₉₅ 10.6–11.3) in the psoriasis and general population cohorts respectively. The incidence of OA was significantly higher in the PsA cohort compared to the psoriasis and general population cohorts after adjusting for BMI (RR_{adj} 1.68 CI₉₅ 1.46–1.93 and RR_{adj} 1.86 CI₉₅ 1.62–2.14 respectively) (Tables 1 and 2).

Conclusion: An increased risk of OA was observed in patients with PsA compared to patients with psoriasis alone and those in the general population. Further work is needed to determine whether this reflects a true increase in OA risk or misdiagnosed PsA and the extent to which it can be explained by differences in the opportunity for OA diagnosis between cohorts.

Table 1. Incidence of osteoarthritis in the PsA, psoriasis and general population cohorts

	Cases	Median age (years) at diagnosis (IQR)	Person years	Incidence rate per 10,000 person years	CI ₉₅
Any OA[†]					
General population	1374	59 (51 - 66)	125,798	109.2	(103.5 - 115.0)
Psoriasis	1432	59 (53 - 67)	122,279	117.1	(111.0 - 123.2)
PsA	464	59 (51 - 66)	28,574	162.4	(147.6 - 177.2)

[†]including spondylitis