Table 1. Characteristic of the patients with axial spondyloarthritis (n=140).

Indicator	Results		
	ankylosing spondylitis	psoriatic arthritis without spinal involvement	psoriatic arthritis with spinal involvement
Quantity	48	47	45
Male, n (%)	30 (62,5)	19 (40,4)	25 (55,6)
Age, years (mean±SD)	41,6 ± 11,0	53,32 ± 11,0	46,9 ± 10,4
Disease duration, years (mean±SD)	14,8 ± 9,4	17,1 ± 8,6	13,6 ± 5,9
BMI, kg/m ² (mean±SD)	25,0 ± 4,2	29,2 ± 5,0	29,3 ± 4,0
Overweight, n (%)	18 (37,5)	35 (74,5)	38 (84,4)
Included obesity, n (%)	7 (14,6)	19 (40,4)	18 (40)
Hypertension, n (%)	11 (22,9)	32 (68,1)	21 (46,7)
Diabetes and prediabetes, n (%)	4 (8,3)	13 (27,7)	8 (17,8)
Dyslipidemia, n (%)	4 (8,3)	13 (27,7)	16 (35,6)
Coronary heart disease, n (%)	2 (4,2)	8 (17,0)	3 (6,7)
Diseases of the gastrointestinal tract, n (%)	19 (39,6)	20 (42,6)	16 (35,6)

Conclusion: The prevalence of diseases of the gastrointestinal tract is approximately equal in all three groups, which is probably due to the use of non-steroidal anti-inflammatory drugs and glucocorticoids. The prevalence of the other pathology presented is significantly higher in the groups of psoriatic arthritis and does not significantly differ depending on the involvement of the spine in the pathological process. Due to the high prevalence of cardiovascular disease among patients with psoriatic arthritis, careful monitoring and timely administration of therapy is necessary.

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FRI0335

THE EFFECT OF TOFACITINIB ON RESIDUAL PAIN IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Current treatments for PsA have proven effective in reducing patient (pt)-reported pain;^{1,2} however, residual pain often remains. Tofacitinib is an oral Janus kinase inhibitor for the treatment of PsA.

Objectives: This descriptive analysis evaluated the effect of tofacitinib, adalimumab and placebo on residual pain in pts with PsA whose inflammation was attenuated after 3 months of therapy.

Methods: Data were included from OPAL Broaden (NCT01877668), a randomised, double-blind, placebo-controlled Phase 3 trial of 12 months' duration in pts with PsA.³ Pts were randomised to receive tofacitinib 5 mg twice daily (BID), tofacitinib 10 mg BID, adalimumab 40 mg subcutaneous injection once every 2 weeks or placebo. This analysis assessed pts with 'residual pain' at Month (M)3. Residual pain was considered as pain in pts with complete attenuation of inflammation at M3, defined by a swollen joint count (SJC) of 0 and CRP levels <6 mg/L. Pain was measured by a visual analogue scale (VAS; 0 ["no pain"] – 100 mm ["most severe pain"]). Changes in pain from baseline to M3 and residual pain (VAS pain reported at M3) were assessed.

Results: Demographics and baseline disease characteristics have previously been reported in the primary study, and were generally similar between treatment groups.³ At M3, 100/422 (23.7%) pts with PsA had achieved SJC of 0 and CRP <6 mg/L. At M3, more tofacitinib-treated (tofacitinib 5 mg BID, n=23/107 [21.5%]; tofacitinib 10 mg BID, n=33/104 [31.7%]) and adalimumab-treated pts (n=31/106 [29.2%]) achieved SJC of 0 and CRP <6 mg/L vs placebo (PsA: n=13/105 [12.4%]). Baseline pain appeared numerically higher in tofacitinib-treated pts (tofacitinib 5 mg BID, 54.7 mm; tofacitinib 10 mg BID, 58.4 mm) vs adalimumab-treated pts (47.7 mm) and placebo (50.4 mm). In pts who achieved SJC of 0 and CRP <6 mg/L at M3, improvements in pain from baseline to M3 appeared numerically greater in pts receiving tofacitinib vs those receiving placebo (Figure 1a). When considering absolute (residual) pain at M3, mean residual pain

was similar across treatment groups (ranging from 22.7–29.2 mm; Figure 1b), despite a higher baseline pain in tofacitinib treatment groups.

Conclusion: Changes from baseline in pain and absolute pain at M3 suggest that in pts with PsA whose inflammation has been completely attenuated, tofacitinib might have an effect on residual pain not obviously attributable to inflammation. However, the sample population was small, and there were large standard deviations. To confirm these results and to understand the mechanisms by which tofacitinib may improve residual pain, a meta-analysis will be performed using individual participant data from pts with rheumatic disease who have participated in tofacitinib randomised controlled trials.

References:

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Figure 1. Change from baseline in a) pain to Month 3 and b) absolute pain at Month 3 in patients with PsA and SJC=0 and CRP <6 mg/L by treatment group

Only patients who reported SJC of 0 and CRP <6 mg/L at Month 3 were included in this analysis. Mean pain is measured by a visual analogue scale from 0–100 mm where 0 = "no pain" and 100 = "most severe pain"

*One patient did not have baseline pain data

BID, twice daily; CRP, C-reactive protein; PsA, psoriatic arthritis; Q2W, once every 2 weeks; SC, subcutaneous injection; SD standard deviation; SJC, swollen joint count

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FRI0336

CONCOMITANT USE OF BIOLOGIC DMARDS WITH CSDMARDS INCREASES DRUG RETENTION RATE AND IMPROVES TREATMENT RESPONSE IN THE PSORIATIC ARTHRITIS. HUR-BIO REAL LIFE RESULTS

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Background: For inflammatory arthritis, drug retention is accepted as an important indicator of the effectiveness and safety of biological drugs.

Objectives: The objective of this study was to determine of the effects first and overall bDMARD drug retention rate during concomitant csDMARD in psoriatic arthritis (PsA).

Methods: HUR-BIO (Hacettepe University Rheumatology Biologic Registry) is a prospective, single center database of biological treatments since 2005. All PsA patients (469) who enrolled in HUR-BIO registry and prescribed at least once biologic DMARD (bDMARD) were included in the study. The subjects were divided into two groups depending on whether or not to use csDMARDs (methotrexate, sulphasalazine or leflunomide) at the last control visit. Demographic, clinical and therapeutic data were collected from this database. Baseline disease activity before the first bDMARD initiation was assessed with DAPSA and PsAID-12.

Table. Demographics, baseline and follow-up clinical characteristics of PsA patients

	With concomitant csDMARD n=288	Without concomitant csDMARD n=167	p
Age (mean, SD)	48.9 (12.1)	45.1 (12.4)	0.002
Female n (%)	204 (70.8)	113 (67.7)	0.48
PsA disease duration (med, IQR)	7 (13)	7 (14.5)	0.53
Age at PsA diagnosis (mean, SD)	39 (11.7)	40.8 (12.9)	0.81
BMI (mean, SD)	29.6 (6.0)	29.2 (5.9)	0.53
Axial involvement n (%)	68 (34.0)	37 (44.6)	0.09
HLA-B 27 (+) n (%)	21/76 (27.6)	17/69 (24.6)	0.68
Duration of use first bDMARD (months) (med, IQR)	22.4 (51.3)	14.1 (31.4)	0.003
Duration of use overall bDMARD (months) (med, IQR)	56.8 (69.4)	24.4 (57.7)	<0.001
Switching bDMARD (+) n (%)	148 (52.5)	71 (42.8)	0.04
Initial DAPSA ¹ (med, IQR)	19.4 (11.7)	17.3 (10.2)	0.04
Initial PsAID ² (med, IQR)	5.7 (3)	5.6 (2.8)	0.55
Final visit PsAID ³ (med, IQR)	2.8 (3.8)	2 (5.7)	0.41
Final visit DAPSA ⁴ (med, IQR)	10.2 (12.4)	12.5 (12.5)	0.01
Remission (n, %)	61 (23.9)	27 (17.6)	0.13
Low disease activity	111 (43.5)	59 (38.6)	
Moderate disease activity	72 (28.2)	58 (37.9)	
High disease activity	11 (4.3)	9 (5.9)	

¹: n=249, ²: n=214, ³: n=109, ⁴: n=408

p<0.001

p=0.003

Results: HUR-BIO PsA registry included 469 PsA patients. Baseline, clinical characteristics and follow-up parameters were given in Table. The using overall bDMARD were adalimumab 294 (62.0%), etanercept 135 (28.8%), infliximab 119 (25.4%), certolizumab pegol 107 (22.8%), secukinumab 67 (14.3%), golimumab 58 (12.4%), ustekinumab 25 (5.3%) and tofacitinib 11 (2.3%). Two hundred eighty eight (61.4%) patients used concomitant cDMARDs [methotrexate 176 (37.5%), leflunomide 94 (20.0%), sulphasalazine 35 (7.5%) and two csDMARD combination 17 (3.6%)]. The first-year retention rate of first bDMARD with or without concomitant csDMARDs were 88% and 80%, respectively. The median duration of first bDMARD retention with or without concomitant csDMARDs were 131.7 and 91.4 months, respectively (Figure). The first-year retention rate of overall bDMARDs with or without concomitant csDMARDs were 92% and 85%, respectively. The median drug retention rate of overall bDMARD in using csDMARD and not using csDMARD were 141.5 and 131.5 months, respectively. Retention rates (both for first bDMARD and overall bDMARDs) were significantly higher in concomitant csDMARDs using group (p=0.003 for first bDMARD retention, p<0.001 for overall bDMARDs retention, log-rank; Figure). In concomitant csDMARD using group, no differences were identified methotrexate or other csDMARDs.

Conclusion: In this study, csDMARDs, either methotrexate or leflunomide/sulphasalazine have additional effect for both retention rate and treatment response of bDMARDs. On the other hand, using bDMARD monotherapy is relatively higher than rheumatoid arthritis (1).

Figure. Drug retention rate of the first bDMARD and overall bDMARDs according to concomitant csDMARD use (csDMARD:conventional synthetic Disease Modifying Antirheumatic Drug)

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FRI0337

REAL WORLD SECUKINUMAB DRUG-SURVIVAL IN PSORIATIC ARTHRITIS

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Background: Secukinumab (Cosentyx) is a human IgG1 monoclonal antibody that selectively binds to and neutralizes IL-17A. Several prospective randomized control trials have demonstrated the efficacy and safety of secukinumab in PsA, but there is a paucity of real life data in the PsA population.

Objectives: To prospectively study secukinumab's safety, efficacy, and tolerability in the cohort of PsA patients from the Israeli registry of inflammatory diseases.

Methods: PsA patients fulfilling the CASPAR criteria from the Israeli registry of inflammatory diseases were included in the analysis, from 2010 to November 2019. The primary end point was secukinumab drug survival compared to other available TNFi drug. Bivariate and multivariate analysis of the factors that affect the drug event free survival was done by cox regression analysis. Drug survival according to treatment line (all treatment lines, or 2nd and above treatment lines) was examined using Kaplan-Meier curves.

Results: Four hundred and four PsA patients were included, which had 709 treatment episodes (initiations) during the study period. Ninety patients had been treated with secukinumab (22%). Secukinumab treated patients were significantly older at time of initiation of treatment, and disease duration was longer. Secukinumab was more likely to be a second, third or fourth line of treatment than the TNFi. Time to an inefficacy event was longer for secukinumab than any other anti-TNF treatment. As a first line treatment secukinumab had drug survival similar to other TNFi. As a second or third line treatment, secukinumab had a better drug survival than other TNFi. Methotrexate did not have a significant effect on inefficacy event rate in combination treatment with secukinumab. Secukinumab, as infliximab and golimumab, was as effective in the higher BMI group as it was in the normal weight to obese groups. Smokers (current or past) did better on secukinumab than on TNFi. Secukinumab had a similar rate of adverse events compared to TNFi.

Conclusion: In this multicenter real world study, secukinumab had a comparable drug survival to TNFi. As a second and beyond line of treatment secukinumab had a better drug survival and lower HR for an inefficacy event. IL-17 inhibition is an effective mechanism of action to treat PsA in real life, and should be used more frequently as a first and second line treatment.

Tables and a graph: