Figure. PASI 75/90/100 response rates up to week 52 across treatments and time points

Response rates were calculated in patients with BSA $\geq 3\%$ at baseline. Black symbols and corresponding p-values were analysed using nonresponse imputation for missing responses. Error bars represent two-sided 95% CI based on the normal approximation for binomial proportions. P-values are based on Cochran-Mantel-Haenszel test (with prior anti-TNF use and baseline body weight as stratification factors) for nonresponse imputation data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ vs placebo. BSA, body surface area; CI, confidence interval; PASI, Psoriasis Area and Severity Index; Q4W, every 4 weeks; Q12W, every 12 weeks; TIL, Itrixizumab; TNF, tumour necrosis factor.

Conclusion: PASI75/90/100 response rates progressively improved with treatment; PASI 75 responses were significantly improved vs placebo as early as W4 (TIL 200mg Q12W), and all response rates were significantly improved vs placebo at W24. Response rates continued to improve through W36 and were sustained through W52. These results demonstrate TIL significantly reduced psoriasis disease activity and was generally well tolerated in a mixed population of anti-TNF-naïve and -experienced patients with PsA and BSA $\geq 3\%$ through W52.

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SAT0418

EFFECT OF TOFACITINIB TREATMENT ON ACTIVE MRI SACROILIITIS AND DISEASE ACTIVITY REDUCTION IN PSORIATIC ARTHRITIS PATIENTS. DATA FROM CLINICAL PRACTICE

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Background: Axial involvement in psoriatic arthritis (PsA) is quite common. Tofacitinib (TOFA) is an oral Janus kinase inhibitor. There is no data on the use of TOFA in PsA patients (pts) with axial involvement, nor is there any data on its effect on active MRI sacroiliitis (MRI-SI). There are only preliminary results of a randomized clinical trial on TOFA efficacy on active SI in AS (1).

Objectives: To study the effect of TOFA therapy on active MRI-SI in PsA pts.

Methods: 40 pts (F/M – 23/17) with active PsA fulfilling the CASPAR criteria were examined. No patients with inflammatory back pain (IBP) were specifically selected. Median (Me) age 41.0 [35.0; 50.0] yrs, Me PsA duration 6.0 [3.0; 10.0] yrs. Pts underwent a standard clinical examination of PsA activity: Me tender joint count 19 [12; 24], swollen joint count 11 [8; 16], patient's global disease activity measured by Visual Analogue Scale (VAS) 70 [50; 80], patient's pain VAS 65 [50; 75], Me activity indexes: DAPSA 44.2 [37.8; 55.3], BASDAI 6.0 [4.2; 7.0], ASDAS 3.8 [2.8; 4.4]. Me CRP 21.3 [3.2; 72.3] mg/L, ESR 28 [12; 52] mm/h. Enthesitis was observed in 65.9% of pts, dactylitis in 53.7% of pts. Apart from a standard clinical examination, all 40 pts underwent sacroiliac joint (SIJ) MRI on scanner Siemens General Electric 1.5 TESLA. Bone marrow edema/osteitis on MRI (STIR) with one lesion on two consecutive slices or at least two lesions on a single slice, was considered active MRI-SI. MRI results were evaluated by 2 independent readers (radiologist and rheumatologist). TOFA was given in 5 mg tablets bds over a period of 6 months, after which 35 patients underwent SIJ MRI. Me [Q25; Q75], Pierson- χ^2 tests were performed. All $p < 0.05$ were considered to indicate statistical significance.

Results: Prior to TOFA therapy, active MRI-SI was detected in 14 of 40 (35%) pts: bilateral in 9 pts, unilateral in 5 pts. At the end of 6 months therapy, active MRI-SI was detected in 4 of 35 (11.4%) pts observed: in 1 pt with baseline bilateral MRI-SI and in 2 pts with unilateral MRI-SI. 1 pt showed negative dynamics, that is, development of active MRI-SI (absent at baseline). The decrease in number

of active MRI-SI patients is statistically significant ($p = 0.017$; Pearson- χ^2). At baseline, inflammatory changes were detected in 23 of 80 (28.8%) SIJs, after 6 months of therapy they were found in 5 of 70 (7.1%) SIJs observed. Decrease in number of SIJs with active inflammation is statistically significant ($p = 0.001$; Pearson- χ^2). At baseline, Me BASDAI 6.0 [4.2; 7.0], Me ASDAS 3.8 [2.8; 4.4]. After 6 months of treatment, Me BASDAI 1.4 [0.6; 3.2], Me ASDAS 1.5 [1.0; 2.1] ($p = 0.001$ for both comparisons).

Conclusion: JAK inhibition using TOFA therapy shows high efficacy in reducing active MRI-SI and decreasing activity of axial involvement in PsA. More extensive studies are needed.

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SAT0419

ASSOCIATION OF ACTIVE MRI SACROILIITIS WITH DACTYLITIS AND WORK PRODUCTIVITY IMPAIRMENT IN PSORIATIC ARTHRITIS PATIENTS. POSITIVE EFFECTS OF TOFACITINIB TREATMENT. DATA FROM CLINICAL PRACTICE

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Background: Psoriatic arthritis (PsA) is a heterogeneous disease with multiple manifestations and choosing among treatments can be a complex decision. Patients (pts) with axial involvement and pts having dactylitis are more likely to develop a severe disease (1, 2). Tofacitinib (TOFA), an oral Janus kinase inhibitor, showed efficacy in treating PsA pts with dactylitis (3). However, its efficacy in treating PsA pts with active sacroiliitis (SI) and dactylitis has not been studied.

Objectives: To study the effect of TOFA therapy in PsA pts having active SI on MRI (MRI-SI) and dactylitis.

Methods: 40 pts (M/F – 23/17) with active PsA fulfilling the CASPAR criteria were examined. Median (Me) age 41.0 [35.0; 50.0] yrs, Me PsA duration 6.0 [3.0; 10.0] yrs. A standard clinical examination of PsA activity was performed: Me tender joint count 19 [12; 24], swollen joint count 11 [8; 16], patient's global disease activity measured by Visual Analogue Scale (VAS) 70 [50; 80], patient's pain VAS 65 [50; 75], Me activity indexes: DAPSA 44.2 [37.8; 55.3], BASDAI 6.0 [4.2; 7.0], ASDAS 3.8 [2.8; 4.4]. CRP 21.3 [3.2; 72.3] mg/L, ESR 28 [12; 52] mm/h. Enthesitis was observed in 65.9% of pts with Me LEI index 1 [0; 1], dactylitis in 53.7% of pts, Me digits with dactylitis 1 [0; 2]. Apart from a standard clinical examination, MRI of sacroiliac joints (SIJs) was performed in all 40 pts using MRI scanner Siemens General Electric 1.5 TESLA. Bone marrow edema/osteitis on MRI (STIR) considered active MRI sacroiliitis (MRI-SI), was evaluated by 2 independent readers (radiologist and rheumatologist). TOFA was given in 5 mg tablets bds over a period of 6 months, after which 35 patients underwent SIJ MRI. Me [Q25; Q75], Pierson- χ^2 tests were performed. All $p < 0.05$ were considered to indicate statistical significance.

Results: Prior to TOFA therapy, MRI-SI was detected in 14 of 40 (35.0%) pts. At the end of 6 months therapy, MRI-SI was detected in 4 of 35 (11.4%) pts: in 3 pts with baseline SI; 1 pt showed negative dynamics, that is, development of active SI (absent at baseline). The decrease in number of active MRI-SI pts is statistically significant ($p = 0.017$; Pearson- χ^2). Significant differences between baseline symptoms in patients with MRI-SI ($n = 14$) and without it ($n = 26$) were defined by **number of digits with dactylitis:** 2 [0; 4] and 0 [0; 2] ($p = 0.047$), **by ESR:** 47 [26; 76] and 20 [6; 37] mm/h ($p = 0.025$), and **by WPAI overall work impairment due to health index:** 80 [60; 84]% and 20 [0; 60]% ($p = 0.033$), respectively; these parameters were higher in MRI-SI subgroup. After 6 months of therapy number of digits with dactylitis, ESR and WPAI indexes were significantly lower as compared with the baseline ones (Table 1).

Table 1

Dynamics of symptoms in TOFA treated pts with or without MRI-SI

Symptoms	MRT-SI (+)			MRT-SI (-)		
	(n=14)			(n=26)		
	Baseline	After 6 months	p	Baseline	After 6 months	p
Number of digits with dactylitis	2 [0; 4]	0 [0; 0]	0.008	0 [0; 2]	0 [0; 0]	0.003
ESR (mm/h)	47 [26; 76]	12 [6; 16]	0.006	20 [6; 37]	8 [6; 16]	0.001
WPAI (%)	80 [60; 84]	0 [0; 20]	0.007	20 [0; 60]	0 [0; 10]	0.128

After 6 months of TOFA therapy, no differences were found between groups of pts with and without MRI-SI in the number of digits with dactylitis ($p=0.47$), in ESR ($p=0.79$) and in WPAI ($p=0.93$).

Conclusion: In PsA pts significant association of active MRI-SI was found with dactylitis, high ESR level and WPAI. Use of TOFA in pts with both active MRI-SI and dactylitis demonstrated its high efficacy in reduction of SI inflammation and dactylitis; it also significantly improved pts' work productivity. These findings are important for personalized approach to treatment of PsA.

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SAT0420

EFFICACY OF NON-TUMOUR NECROSIS FACTOR BIOLOGICS AND TARGETED SYSTEMIC DISEASE MODIFYING ANTI-RHEUMATIC DRUGS IN THE TREATMENT OF PSORIATIC ARTHRITIS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: Psoriatic arthritis (PsA) is a systemic, inflammatory condition presenting in approximately 30% of patients with psoriasis and associated with functional impairment and a reduced health-related quality of life. Current treatment guidelines recommend non-steroidal anti-inflammatory drugs, conventional Disease Modifying Anti-Rheumatic Drugs (cDMARDs) and Tumour Necrosis Factor α inhibitors (TNFi). Recent research has focused on alternative biologic medications which target interleukin (IL) 6, 12/23, 17A, 23 and T Cell co-stimulation, as well as targeted synthetic DMARDs (tsDMARDs) including Janus Kinase inhibitors (JAKi) and Phosphodiesterase 4 inhibitors (PDE4i). Evidence of the safety and efficacy, measured using the American College of Rheumatology-20 (ACR20), has been demonstrated leading to the inclusion of several biologics and tsDMARDs in guidelines. However, it can be argued that ACR50, indicating a 50% improvement in disease, is a more clinically relevant outcome measure.

Objectives: To conduct a systematic review and meta-analysis of the efficacy (ACR50 response) of non-TNFi biologics and tsDMARDs in the treatment of PsA. **Methods:** A systematic literature search of Embase, MedLine and Web of Science was undertaken to identify randomised controlled trials (RCTs) investigating efficacy and safety of non-TNFi biologics and tsDMARDs published in English from the inception of the databases to September 2019. The Cochrane Risk of Bias tool was used to assess methodological rigour of included trials. A meta-analysis was performed using a random effects model to estimate odds ratios of ACR 50 response vs placebo. A subgroup analysis was performed using patients with previous TNFi exposure.

Results: 21 RCTs were eligible with 6389 participants. Evaluation periods ranged from 12 to 24 weeks. JAKi, PDE4i, IL6i, IL12/23i, IL17Ai and IL23i treatments were more efficacious than placebo for ACR50 response ($p<0.001$) (Figure 1). Only tofacitinib (JAKi), secukinumab (IL17Ai) and ixekizumab (IL17Ai) were able to

demonstrate efficacy at the ACR50 level in participants with prior TNFi exposure ($p<0.0001$) (Figure 2). All treatments demonstrated an adequate safety profile.

Figure 1. Forest plot of achieving American College of Rheumatology-50 with treatment versus placebo M-H, Mantel-Haenszel; CI, confidence interval; JAKi, Janus Kinase inhibitor; PLA, placebo; df, degrees of freedom; IL6i, Interleukin 6 inhibitor; IL12/23i, Interleukin 12/23 inhibitor; IL17Ai, Interleukin 17A inhibitor; TNFi, Tumour necrosis factor inhibitor; IL23i, Interleukin 23 inhibitor

Figure 2. Forest plot of achieving American College of Rheumatology-50 with treatment versus placebo in tumour necrosis factor inhibitor-exposed subgroup M-H, Mantel-Haenszel; CI, confidence interval; JAKi, Janus Kinase inhibitor; PLA, placebo; df, degrees of freedom; PDE4i, Phosphodiesterase 4 inhibitor; IL6i, Interleukin 6 inhibitor; IL12/23i, Interleukin 12/23 inhibitor; IL17Ai, Interleukin 17A inhibitor; IL23i, Interleukin 23 inhibitor

Conclusion: Non TNFi biologics and tsDMARDs are able to demonstrate 50% improvement with adequate safety profiles. These therapies are often used in patients who are inadequate responders to TNFi but there is less robust data in this specific patient group. Studies with clinically relevant primary endpoints should be considered in this patient population.