PsAID9 domains for FIL vs PBO, including pain ($p<0.0001$; **Figure 1b**). A significant improvement in SF-36 PCS, but not in MCS, with FIL vs PBO was observed. The mean change (SD) from baseline in PCS at week 16 was 7.4 (6.6) vs 2.4 (6.6) for FIL vs PBO, respectively (LS mean of group difference 4.67 [2.58, 6.76], $p<0.0001$).

Conclusion: Compared with PBO, FIL significantly improved disease impact in patients with active PsA, as measured by the PsA-specific PsAID9 total score and individual domain scores. Significant improvement in SF-36 PCS score was also seen with FIL.

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SAT0368

GO-DACT: A RANDOMIZED DOUBLE-BLIND PLACEBO-CONTROLLED PROOF-OF-CONCEPT TRIAL OF GOLIMUMAB PLUS METHOTREXATE (MTX) VERSUS MTX MONOTHERAPY, IN IMPROVING DACTYLITIS, IN MTX NAÏVE PSORIATIC ARTHRITIS PATIENTS

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Background: Psoriatic arthritis (PsA) dactylitis is associated with an increased risk of erosions and higher disease activity. Dactylitis treatment

strategies are however controversial due to the absence of evidence from randomized controlled trials studying dactylitis as a primary outcome.

Objectives: To assess the efficacy of golimumab plus MTX versus placebo plus MTX for active dactylitis in PsA patients, in a phase 3b trial.

Methods: GO-DACT was a proof-of-concept multicentric, investigator-initiated randomized, double-blind, placebo-controlled, parallel-design trial, conducted in 13 Portuguese Rheumatology Centers. PsA patients, naïve for MTX and biologic disease modifying anti-rheumatic drugs (bDMARDs), with active dactylitis, were randomly allocated to either golimumab in combination with MTX or MTX monotherapy. The primary endpoint was the change from baseline in the dactylitis severity score (DSS) assessed at week 24. Key secondary endpoints included DSS response and the magnetic resonance imaging (MRI) dactylitis score, as well as composite indexes of PsA activity.

Results: 44 patients were centrally randomized, 21 to golimumab plus MTX and 23 to placebo plus MTX, for 24 weeks, and 1 patient from each arm dropped out. Due to favorable results on a planned interim analysis recruitment was halted. The median MTX dose in the golimumab plus MTX group was 15mg/week and in the MTX monotherapy group 20mg/week. The median baseline DSS was 6 in each arm. Patients treated with golimumab plus MTX experienced significantly greater improvements in the DSS at week 24 (median change of 5) as compared to the MTX group (median change of 2) ($p=0.026$). At week 24, 12 (60.0%) patients treated with golimumab plus MTX and 4 (18.2%) with MTX, achieved the DSS70 response ($p<0.05$). Significant differences were also observed in the median changes from baseline to week 24 in MRI dactylitis score, Disease Activity Score 28 (DAS28), Disease Activity Index for PsA (DAPSA), PsA Disease Activity Score (PASDAS) and Target Nail Psoriasis Severity Index (tNAPSI), favoring the golimumab and MTX association arm. Likewise, higher proportions of patients treated with golimumab plus MTX achieved DSS50 responses and the American College of Rheumatology 20/50 responses, at week 24. There were no new safety issues for golimumab during this trial.

Conclusion: GO-DACT suggests additional benefits from the combination of golimumab and MTX as first-line bDMARD therapy versus MTX monotherapy, in the treatment algorithm of PsA active dactylitis.

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Not applicable

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SAT0369

SECUKINUMAB IMPROVES GRAPPA-OMERACT CORE DOMAINS OF PSORIATIC ARTHRITIS REGARDLESS OF PREVIOUS EXPOSURE TO A TNF INHIBITOR

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Background: Secukinumab, a fully human interleukin 17 (IL-17A) monoclonal antibody, has demonstrated efficacy in psoriatic arthritis (PsA) in phase 3 clinical trials.¹⁻⁴ The PsA core domain set, updated by the

Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) and endorsed by Outcome Measures in Rheumatology (OMERACT), aims to improve and standardize the assessment of PsA outcomes.⁵

Objectives: To report the efficacy of secukinumab vs placebo across individual PsA core domains at week 16 in patients naive to tumor necrosis factor (TNF) inhibitors or who were inadequate responders (TNF-IR), using pooled data from 4 phase 3 FUTURE studies.

Methods: Patients with active PsA participated in the phase 3 FUTURE 2, 3, 4, and 5 trials.¹⁻⁴ Data were pooled for secukinumab 150 mg (load vs. no load), 300 mg, or placebo at the end of the 16-week double-blind period. Efficacy was assessed using multiple clinical and laboratory measures to evaluate the updated GRAPPA-OMERACT PsA core domain set for disease activity, pain, function, and quality of life (Table 1).

Results: A total of 2049 patients were included (1436 TNF naive and 613 TNF-IR). Baseline demographics and disease characteristics were broadly similar in all treatment groups. Efficacy results for the core inner circle domains are shown in Table 1.

Table 1. Pooled Efficacy Results Across GRAPPA-OMERACT PsA Core Domains by TNF Inhibitor Status

PsA Core Domains	TNF inhibitor status	Improvement in Patients With PsA at Week 16 in FUTURE Trials							
		Secukinumab 300 mg n = 461		Secukinumab 150 mg n = 572		Secukinumab 150 mg, no load n = 335		Placebo n = 681	
		Naive	TNF-IR	Naive	TNF-IR	Naive	TNF-IR	Naive	TNF-IR
Musculoskeletal disease activity	SJC6, ≥ 50% improvement	73.7 (233/316)	57.9 (84/145)	64.7 (258/399)	56.1 (87/173)	65.2 (159/244)	44.0 (40/91)	42.8 (204/477)	31.4 (64/204)
	SJC6 resolution	41.5 (131/316)	18.6* (27/145)	29.3 (117/399)	22.0 (38/173)	25.0 (61/244)	16.7* (17/91)	16.8 (80/477)	12.3 (25/204)
	TJC6, ≥ 50% improvement	67.1 (212/316)	48.7 (72/145)	56.9 (227/399)	47.4 (82/173)	58.6 (143/244)	44.0 (40/91)	42.8 (204/477)	31.4 (64/204)
	TJC6 resolution	34.4 (111/316)	15.8* (24/145)	24.4 (93/399)	11.6* (20/173)	24.4 (59/244)	9.9* (10/91)	27.4 (129/477)	12.2 (25/204)
	LEI resolution	56.9 (181/316)	44.9 (66/145)	46.4 (175/399)	40.5 (71/173)	44.8 (106/244)	32.8* (34/91)	32.3 (154/477)	22.1 (45/204)
Skin disease activity	LDI resolution	63.2 (204/316)	40.1 (58/145)	48.0 (182/399)	49.2 (85/173)	45.0 (106/244)	40.0* (41/91)	32.9 (154/477)	26.6 (53/204)
	PASI 75	68.6 (221/316)	52.7 (77/145)	58.9 (228/399)	52.9 (91/173)	57.9 (133/244)	47.4 (43/91)	40.8 (194/477)	28.9 (60/204)
	IGA mod 2011 0/1	51.4 (164/316)	36.6* (54/145)	44.3 (162/399)	31.8 (53/173)	33.1 (82/244)	28.9 (31/91)	28.3 (133/477)	19.5 (39/204)
Pain	PSA pain VAS, ≥ 3-point improvement*	72.2 (228/316)	56.6 (85/145)	72.4 (289/399)	57.1 (114/173)	58.2 (176/244)	51.8 (53/91)	51.8 (247/477)	41.2 (84/204)
	PGA, mean change from BL (95% CI)	-21.11 (19.64, -22.58)	-19.64 (19.64, -22.58)	-17.38 (19.64, -22.58)	-16.08 (19.64, -22.58)	-17.84 (19.64, -22.58)	-13.48 (19.64, -22.58)	-7.46 (19.64, -22.58)	-5.13 (19.64, -22.58)
Patient global	HAQ-DI, MCID ≥ 0.3	60.4 (191/316)	49.7 (85/145)	54.9 (204/399)	41.8 (72/173)	54.9 (176/244)	41.8 (53/91)	51.8 (247/477)	36.3 (72/204)
	SF-36 PCS, MCID ≥ 2.5	68.4 (216/316)	50.7 (85/145)	61.9 (247/399)	53.8 (93/173)	65.6 (169/244)	50.5 (49/91)	45.3 (216/477)	34.3 (70/204)
HRQOL	SF-36 MCS, MCID ≥ 2.5	49.4 (156/316)	46.2 (71/145)	51.1 (204/399)	47.4 (93/173)	51.2 (125/244)	45.1* (41/91)	42.1 (201/477)	36.3 (72/204)
	FACIT-Fatigue, mean change from BL (95% CI)	6.07 (6.07, 6.07)	6.07 (6.07, 6.07)	5.36 (6.07, 6.07)	5.13 (6.07, 6.07)	5.67 (6.07, 6.07)	4.78 (6.07, 6.07)	2.01 (6.07, 6.07)	1.21 (6.07, 6.07)
Systemic inflammation	Elevated CRP (≥ 10 mg/L)	76.6 (252/316)	62.2 (87/145)	67.1 (257/399)	51.8 (93/173)	79.6 (229/244)	44.8 (53/91)	38.3 (181/477)	21.2 (45/204)
	Resolution	23.3 (74/316)	37.8 (58/145)	32.9 (142/399)	48.2 (80/173)	24.4 (55/244)	55.2 (60/91)	61.7 (296/477)	78.8 (159/204)

BL, baseline; CRP, C-reactive protein; FACIT-Fatigue, Functional Assessment of Chronic Illness Therapy-Fatigue; HAQ-DI, Health Assessment Questionnaire-Disability Index; HRQOL, health-related quality of life; IGA, Investigator's Global Assessment; IR, inadequate responder; LDI, Leeds Dactylitis Index; LEI, Leeds Enthesitis Index; MCID, minimal clinically important difference; MCS, mental component summary; PASI 75, 75% reduction in the Psoriasis Area and Severity Index; PCS, physical component summary; PGA, Patient Global Assessment; PsA, psoriatic arthritis; SJC6, swollen joint count based on 66 joints; TJC6, tender joint count based on 68 joints; TNF, tumor necrosis factor; VAS, 100-mm visual analog scale.

All P values vs placebo were $P < .05$ except where indicated. All P values were for hypothesis generation. No adjustment was made for multiple comparisons. * $P < .05$; ** $P < .01$; *** $P < .001$. No MCID has been defined in PsA. * Least-squares mean change from BL; n is the number of patients with measures at both BL and week 16.

Conclusion: Secukinumab demonstrated efficacy compared with placebo across GRAPPA-OMERACT PsA core domains in the phase 3 clinical trials program, regardless of prior TNF inhibitor use.

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SAT0370

IMBALANCE OF TH17 AND REGULATORY T CELLS IN PATIENTS WITH PSORIATIC ARTHRITIS AND RE-BALANCE BY LOW-DOSE IL-2

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Background: Psoriatic arthritis (PsA) is an T lymphocytes-mediated inflammatory condition [1]. Although regulatory T cells (Tregs) isolated from blood and psoriatic skin have been showed a functional deficiency in suppressing effector T-cells in PsA [2], absolute quantitative status of peripheral Treg or Th17 cells is still unclear. On the other hand, recent studies have revealed that low-dose IL-2 alleviates some of autoimmune disease activity by upregulating Treg cells [3, 4], which is expected to control the development of PsA.

Objectives: To assess the absolute numbers of peripheral lymphocyte subpopulations and the efficacy and safety of low-dose IL-2 therapy in PsA patients.

Methods: Total 95 PsA patients and 106 age- and sex-matched healthy controls were recruited. Of them, 22 cases received the treatment of low-dose IL-2 at 0.5 million IU per day for 5 days subcutaneously. The absolute numbers of lymphocyte subgroups and CD4⁺T subsets in peripheral blood were measured by flow cytometry. The clinical manifestations and laboratory indicators as well as the levels of peripheral lymphocyte and CD4⁺T subsets were compared before and after the treatment.

Figure 1: Comparison of absolute numbers of peripheral CD4⁺T cell subsets between PsA patients (n=95) and healthy controls (n=106). The level of Th17 was significantly increased while that of Tregs reduced in patients with PsA. Data were calculated and compared with Mann-Whitney U test. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Figure 2: Comparative analysis the changes of CD4⁺T cell subsets in patients with PsA between pre- and post-treatment (n=22). Low-dose IL-2 markedly raised the absolute numbers of Treg and moderately increased the level of Th17 cells in PsA patients. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Figure 3: Comparative analysis the changes of PsA signs and symptoms between pre- and post-treatment (n=22). Data were presented as median(range) and calculated and compared with Mann-Whitney *U* test. (A-I) show that low-dose IL-2 treatment provided a significant decrease in 28 tender joint count, DAS28-ESR index, VAS index, PHGA index, DLQI index and HAQ index. **P* < 0.05, ****P* < 0.001.

Results: Notably, the absolute numbers of lymphocyte subpopulations in peripheral blood such as NK, CD4⁺T, Th17 and Tregs in PsA patients were lower than those of healthy controls (*P* < 0.05). The absolute number of Treg was significantly and negatively correlated with the levels of disease indicators, including DAS28, the number of tender joints, visual analogue scale, physician's global assessment, dermatology life quality index, and health assessment questionnaire (*P* < 0.05). After low-dose IL-2 treatment, compared with the baselines, there was a significant increase in the absolute numbers of NK (*P* < 0.05), CD4⁺T (*P* < 0.01), Th17 (*P* < 0.01) and Treg cells (*P* < 0.001). Interestingly, IL-2 markedly raised the level of Tregs in PsA patients even higher than that of healthy donors (*P* < 0.001), leading to re-balance of Th17 and Tregs. Further, low-dose IL-2 treatment rapid reduced the disease activity such as DAS28, the number of tender joints, visual analogue scale, physician's global assessment, dermatology life quality index, and health assessment questionnaire (*P* < 0.05).

Conclusion: Patients with PsA had an imbalance between pro- and anti-inflammatory cells, particularly the reduction of the absolute number of Tregs. Low-dose IL-2 combination treatments restored the decreased number of Treg cells and lowered disease activity indicators of these patients without over-treatment and evaluable side effect. Further studies are needed to evaluate the long-term immunoregulatory ability of IL-2 treatment.

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SAT0371

CARDIOVASCULAR IMPACT OF HYPERURICEMIA IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Inflammatory joint diseases (IJD) such as psoriatic arthritis (PsA) have an increased risk of cardiovascular disease (CVD) since inflammation plays a pivotal role in the pathogenesis of coronary artery disease (CAD), heart failure (HF) and atrial fibrillation (Afib)¹. Ischemic heart disease and HF are the main causes of the increased and premature mortality among patients with IJD². Additionally, patients with PsA have a prevalence of hyperuricemia (HUC) of 32%, 3 times greater as compared with the general population, which may be related to increased cell turnover as well as the release of pro-inflammatory cytokines and tumor necrosis factor³.

Prolonged exposure to high levels of uric acid (UA) has been shown to result in oxidative stress causing endothelial dysfunction, ionic channel changes, atrial and ventricular remodeling⁴. There is experimental evidence indicating that uric acid stimulates renin-angiotensin-aldosterone system (RAAS), and it is associated with an increase in cardiac tissue xanthine oxidase activity, all of which induce cardiomyocyte hypertrophy, myocardial oxidative stress, interstitial fibrosis and impaired diastolic relaxation⁵.

Objectives: The aim of this study is to assess the correlation of HUC and the clinical expression of CVD in patients with PsA.

Methods: This is a retrospective cohort study using the 2016 National Inpatient Sample (NIS) of adults diagnosed with PsA based on ICD-10 codes, to detect the prevalence of cardiovascular (CV) conditions such as CAD, Afib, and HF with preserved ejection fraction (HFpEF) in patients with concomitant HUC or gout versus age matched controls. Chi square was used for point prevalence and multivariate linear regression adjusted for age, gender, race, CAD, diabetes mellitus, HTN, hyperlipidemia (HLD), smoking, chronic kidney disease (CKD) and Charlson comorbidity index for prevalence odds ratio (POR). We used STATA-15 for statistical analysis.

Results: We identified 37,315 patients with PsA, of whom 2,165 had concomitant HUC or gout (5.80%). Mean age was 61 years, 57% were females. Our results showed that PsA with concomitant HUC or gout compared to PsA without HUC or gout was associated with a higher rate of Afib (17.8% vs 6.1%, *p* < 0.001), CAD (35.1% vs 19.4%, *p* < 0.001) and HFpEF (7.2% vs 3.1%, *p* < 0.001). Furthermore, patients with PsA and HUC/gout appeared to have more risk of developing Afib (POR 1.79; 95%-CI 1.31-2.45; *p* < 0.001) and HFpEF (POR 1.56; 95%-CI 1.08-2.26; *p* = 0.018), compared to patients with normal uric acid after multivariate-adjustment for risk factors. No statistical difference in CAD was identified between the two groups (POR 1.21; 95%-CI 0.94-1.55; *p* = 0.131) after multivariate linear regression adjustment for confounders.

Conclusion: This study showed that HUC is independently associated with CVD, mainly with Afib and HFpEF in patients with PsA. It remains to be seen if a treat to target approach with normalization of UA in patients with PsA will result in improved CV outcomes. We believe that our findings merit further investigation and that this study adds weight to the hypothesis of UA as a potential risk factor for CVD. Prospective studies are needed to establish the role of serum uric acid level as a biomarker or predictor for CVD, including CAD, Afib and HFpEF in patients with PsA.

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