Stable treatment with 1 csDMARD was required. Primary endpoints comparing tofacitinib vs PBO were ACR20 response rate and change from baseline in Health Assessment Questionnaire Disability Index (Δ HAQ-DI) at M3. Secondary endpoints included: ACR20 response rates and Δ HAQ-DI through M12; pts achieving ACR50, ACR70, $\geq 75\%$ improvement of PASI and PsARC at all time points; and changes from baseline in LEI, Dactylitis Severity Score and SPARCC Enthesitis Index. Radiographic progression was assessed by van der Heijde-modified Total Sharp Score (mTSS).

Results: 96.9% of pts were white and 53.3% were female; mean age was 47.9 years. 96.2% and 88.4% of pts completed M3 and M12, respectively. At M3, tofacitinib 5 and 10 mg BID significantly improved ACR20 response rates (50.5% [$p \leq 0.05$] and 60.6% [$p < 0.0001$] vs 33.3%; Fig 1A) and Δ HAQ-DI (-0.35 [$p \leq 0.05$] and -0.40 [$p < 0.001$] vs -0.18; Fig 1B) vs PBO, with responses maintained to M12 (Fig 1C&D). Greater efficacy was also seen for adalimumab vs PBO. Tofacitinib 5 and 10 mg BID were superior to PBO for ACR20 response rates at Week 2 (22.4% [$p < 0.001$] and 31.7% [$p < 0.0001$] vs 5.7%; Fig 1C). Secondary endpoints supported primary findings (data not shown). $> 91\%$ of pts were radiographic non-progressors at M12 (defined as an increase from baseline in mTSS ≤ 0.5). M12 safety findings were similar between groups (Fig 1E). The most common adverse events were upper respiratory tract infection (7.5–10.6%), nasopharyngitis (7.5–11.5%) and headache (3.8–10.6%).

Figure 1. A, ACR20 response rates with tofacitinib vs placebo at M3. B, Δ HAQ-DI with tofacitinib vs placebo at M3. C, ACR20 response rates up to M12. D, Δ HAQ-DI up to M12. E, Safety summary (safety analysis set; all causality)

	Up to M3	Up to M12
	Placebo (N=108)	Placebo to tofacitinib 5 mg BID (N=52)
AEs, n (%)	37 (35.2)	34 (65.4)
SAEs, n (%)	1 (1.0)	0 (0.0)
Discontinuation due to AEs, n (%)	1 (1.0)	2 (3.8)
Deaths, n (%)	0	1 (1.9)
AEs of special interest, n (%) [day of onset]		
Serious infection	0	2 (3.8) [102, 391]
Herpes zoster (all non serious)	0	0
Malignancy	0	0
MACE	0	1 (1.9) [109]

Nominal $p < 0.05$, $^{**}p < 0.001$, $^{***}p < 0.0001$ vs placebo; † Achieved statistical significance at $p < 0.05$ per the pre-specified step-down testing procedure; dashed line indicates the end of the placebo-controlled period; † All patients who received ≥ 1 dose of study medication; † Cardiac arrest; † Placebo groups progressed to tofacitinib 5 mg or tofacitinib 10 mg at M3; AE, adverse event; BID, twice daily; M, month; MACE, major adverse cardiovascular event; n, number of patients with event; Q2W, every 2 weeks; SAE, serious adverse event; SC, subcutaneous

Conclusions: In TNFi-naïve pts with active PsA, tofacitinib was superior to PBO in ACR20 response rates and Δ HAQ-DI at M3, with superiority vs PBO as early as Week 2 for ACR20, which was maintained to M12. No new safety risks were identified vs previous studies in other indications.

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OP0217 USTEKINUMAB IS SUPERIOR TO TNF INHIBITOR TREATMENT IN RESOLVING ENTHESITIS IN PSA PATIENTS WITH ACTIVE ENTHESITIS- RESULTS FROM THE ENTHESIAL CLEARANCE IN PSORIATIC ARTHRITIS (ECLIPSA) STUDY

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Background: IL-23 is considered to play an important role in the development of enthesitis. Ustekinumab (UST), a combined inhibitor of IL-12/IL-23 shows efficacy in psoriatic arthritis (PsA), which is driven by enthesial and synovial disease, while it has no therapeutic role in diseases driven by synovitis alone, such as rheumatoid arthritis. We therefore speculated that inhibition of IL-23 is particularly effective in enthesitis-driven PsA patients.

Objectives: To compare the efficacy of UST with tumor necrosis factor inhibitor (TNFi) treatment in clearing enthesitis in PsA patients.

Methods: ECLIPSA is a prospective randomized-controlled open study. Patients with PsA with active enthesitis were randomized 1:1, receiving either standard doses of UST (arm 1) or TNFi (arm 2). At baseline the following parameters were assessed: age, gender, BMI, disease duration, previous DMARDs, use of corticosteroids, use of NSAIDs, swollen and tender joint count, VAS-pain, VAS-global, NAPS, PASI, MASES, SPARCC, LDI, BASDAI, BASFI, HAQ-DI, SF-36, FACIT-F, ESR and CRP. Primary endpoint was a SPARCC of 0 after 6 months. Patients were seen every 3 months and followed for a total of 6 months. In order to investigate the effects of study treatment over time we used 2x3 mixed design ANOVA models for both physician's and patient's reported outcomes. Furthermore, exploratory logistic regression was used to predict a SPARCC of 0 at month 6 from baseline SPARCC, PASI, NAPS, FACIT-F and BASDAI while additionally accounting for age, gender, PsA duration and study treatment.

Results: 51 patients (UST=25; TNFi=26) were screened and 47 patients (UST=23; TNFi=24) were enrolled with 4 patients not presenting signs of active enthesitis at baseline. Mean $\pm$ SD age was 59.11 $\pm$ 12.16 years and mean $\pm$ SD disease duration was 6.4 $\pm$ 7.79 years. Mean $\pm$ SD SPARCC at baseline was 4.87 $\pm$ 2.69 in the UST group and 3.88 $\pm$ 2.52 in the TNFi group. Other baseline characteristics were similar between both groups with exception of HAQ-DI, BASFI and SF-36 mental scale. In regards to the effect of study treatment (TNFi vs. UST) and time, the corresponding ANOVAs suggested an important interaction of both factors for measures of enthesitis (MASES and SPARCC), patient-reported disease activity (BASDAI and BASFI), physical well-being (SF-36 physical component summary scale), and PASI all $p < 0.044$ with superiority of UST. However, TNFi was superior to UST with respect to improvement of fatigue (FACIT-F), $p < 0.001$. After 6 months, 17 out of 24 UST patients (70.8%) and 10 out of 26 TNFi patients (38.4%) reached the primary endpoint (SPARCC=0). Logistic regression predicting enthesitis-free state of disease was significantly related to study treatment only, with patients receiving UST being more likely to show no signs of enthesitis at month 6 (OR=0.037; $p=0.005$).

Conclusions: These results show that UST is superior to TNFi in resolving the enthesitis component of disease in PsA patients with active enthesial disease. Based on these data more stratified treatment approaches can be designed in PsA patients, where enthesitis-driven patients are targeted by IL-23/IL-17 pathway inhibitors, while more systemic disease manifestations of PsA, such as patients with polyarticular disease or those with high-level fatigue are targeted by TNFi.

Disclosure of Interest: None declared

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OP0218 EFFICACY AND SAFETY RESULTS OF GUSELKUMAB, AN ANTI-IL23 MONOCLONAL ANTIBODY, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS OVER 24 WEEKS: A PHASE 2A, RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED STUDY

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Objectives: To evaluate the efficacy, safety, and tolerability of guselkumab (GUS), a fully human monoclonal antibody against the p19 subunit of IL-23, in patients (pts) with active psoriatic arthritis (PsA).

Methods: In this double-blind, placebo-controlled, multicenter study, pts with active PsA and $\geq 3\%$ body surface area (BSA) of plaque psoriasis despite current or previous treatment with standard-of-care therapies, including those previously exposed to anti-TNF α agents, were randomized 2:1 to receive GUS 100 mg subcutaneously (SC) or placebo (PBO) at wks 0, 4, and every 8 wks (q8w) thereafter through wk44. At wk16, pts from either group with $< 5\%$ improvement from baseline in both swollen and tender joint counts were eligible for early escape to open-label ustekinumab. At wk24, all remaining PBO pts crossed-over to receive GUS 100 mg, and then received GUS at wk28, and q8w thereafter through wk44. The primary endpoint was ACR 20 response at wk24. Major