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POS0195

GUSELKUMAB TREATMENT MODULATES CORE PSORIATIC ARTHRITIS GENE EXPRESSION IN TWO PHASE 3 CLINICAL TRIALS (DISCOVER-1 AND -2)

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Background: Guselkumab (GUS), an interleukin-23 p19-subunit monoclonal antibody, demonstrated efficacy compared with placebo (PBO) in reducing signs and symptoms of psoriatic arthritis (PsA) in the phase 3 DISCOVER-1 & 2 studies.^{1,2}

Objectives: To evaluate gene expression in the blood of PsA patients (pts) in the DISCOVER-1 & -2 studies and the impact of GUS on the expression of these genes.

Methods: Pts were treated with GUS 100mg every 4 weeks (Q4W); GUS 100 mg at W0, W4, then Q8W; or matching PBO. Whole transcriptome profiling by RNA-sequencing was performed using the Novaseq platform on blood samples obtained from a subset of 673 pts with PsA at baseline across the 2 DISCOVER studies, as well as from 21 demographically (age, sex, and ethnicity) matched healthy controls procured independently of the clinical program. A subgroup (N=227) also had serial blood samples (W0/W4/W24) evaluated; the subgroup pts were selected based on having baseline characteristics (demographics, disease activity, medication use) representative of the overall cross-study PsA population. Significance of differentially expressed genes (DEGs) between PsA and healthy controls was defined by a false discovery rate (FDR) <0.05 based on a log-linear model using edgeR. Top genes were defined by significance and |logFC| >1. For cell type analysis, genes that changed with GUS treatment were tested for enrichment using Cibersort. Gene enrichment scores were calculated using Gene Set Variation Analysis (GSVA).

Results: To define disease genes, we compared genes at baseline in pts with active PsA vs. healthy control whole blood transcriptomes and detected 355 upregulated and 314 downregulated (top genes shown in Table 1), defined here as core disease genes. Upregulated genes were largely related to neutrophils, monocytes, macrophages, and extracellular matrix, whereas downregulated genes were related to T cells. The upregulated disease genes were significantly decreased and the downregulated disease genes were significantly increased by GUS treatment vs. PBO at W4 and W24 (Fig 1). Upon stratification by Psoriasis Area and Severity Index 75% response and American College of Rheumatology 20% response, changes in core disease gene expression from W0 were statistically significant among responders, but not in non-responders, at W4 and W24 (data not shown). We then performed the second differential expression analysis comparing baseline to W4 and W24 for both PBO and GUS treatment arms to define genes that change with treatment arm over time. At W4 and W24 we

found many DEGs from baseline with GUS treatment and none with PBO. These included genes related to B-, T-, NK-, and plasma cells (increased by GUS) and neutrophils, monocytes, eosinophils, and macrophages (decreased by GUS), suggestive of a partial normalization of immune cell composition in whole blood. **Conclusion:** Using whole transcriptome profiling, we detected DEGs in blood samples obtained from PsA pts vs. healthy controls, suggesting a dysregulation of immune cell profiles in PsA. The majority of these disease-associated genes were modulated by GUS, with directionality toward a normalization of whole blood transcriptomic signatures.

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Table 1. Top DEGs derived from PsA vs. healthy whole blood transcriptomes.

Upregulated in PsA				Downregulated in PsA			
Gene	logFC	logCPM	FDR	Gene	logFC	logCPM	FDR
ADGRG7	5.92	-0.90	0.02101	AK8	-1.36	-1.06	1.61E-07
ADAMTS2	4.06	0.82	0.006466	FTCD	-1.48	-1.74	1.67E-05
PGF	3.21	-0.68	0.006466	GPR15	-1.54	1.81	1.67E-05
PCSK9	3.21	-2.96	0.023872	CHRM3	-1.54	-2.62	9.6E-08
OLAH	2.76	0.75	0.004539	RFPL4AL1	-1.69	-3.34	0.009738
MAOA	2.55	-0.26	0.005463	SPACA3	-1.85	-3.23	0.000216
SLC2A14	2.30	0.59	0.022594	VANGL2	-1.95	-1.79	9.6E-08
MMP1	2.25	-1.16	0.004745	RFPL4A	-2.04	-1.28	0.004539
DAAM2	2.12	4.31	0.024628	GLYATL2	-2.77	-2.78	1.93E-15
				BCAR1	-3.13	-2.58	6.24E-26

Bold indicates positive change. CPM = counts per million.

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Figure. Change from baseline in median GSVA scores for upregulated and downregulated genes in PsA for GUS and PBO treatment arms from DISCOVER-1 and 2.

*, +, ‡ p < 0.05, 0.01, 0.001, respectively. Wilcoxon test performed between GUS (Q4W, dashed line; Q8W, solid line) and PBO.

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POS0196

UPADACITINIB IN PATIENTS WITH PSORIATIC ARTHRITIS REFRACTORY TO BIOLOGIC DISEASE-MODIFYING ANTIRHEUMATIC DRUGS: 56-WEEK DATA FROM THE PHASE 3 SELECT-PSA 2 STUDY

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Background: Upadacitinib (UPA) is an oral Janus kinase inhibitor currently under evaluation for the treatment of psoriatic arthritis (PsA). Previous 24-week results from the SELECT-PsA 2 study in patients with PsA and prior inadequate response to ≥ 1 biologic disease-modifying antirheumatic drug (bDMARD) demonstrated UPA efficacy with a safety profile consistent with that observed in rheumatoid arthritis.¹

Objectives: To evaluate the 56-week efficacy and safety of UPA in the SELECT-PsA 2 study.

Methods: Patients were randomized to 56 weeks of blinded treatment with UPA 15 or 30 mg once daily (QD), or placebo (PBO) switched to UPA 15 or 30 mg QD at Week 24. Efficacy endpoints included proportions of patients achieving 20/50/70% improvement in American College of Rheumatology (ACR) criteria (ACR20/50/70), 75/90/100% improvement in the Psoriasis Area and Severity Index (PASI75/90/100), resolution of dactylitis and enthesitis, and minimal disease activity (MDA). Non-responder imputation was used for missing data. Treatment-emergent adverse events (TEAEs) were summarized for events occurring while on UPA and ≤ 30 days after last dose (for those who discontinued).

Results: Of 641 patients who received ≥ 1 dose of study drug, 74.7% completed 56 weeks of treatment. Clinical improvements based on the proportion of patients achieving ACR20/50/70 and MDA (Figure 1), PASI75/90/100, and resolution of dactylitis and enthesitis were generally maintained through 56 weeks of UPA treatment. Week 56 results for patients who switched from PBO to UPA at Week 24 had a similar trajectory to those for patients originally randomized to UPA. Overall, improvements observed with UPA 15 mg were similar to or approached those with UPA 30 mg over 56 weeks. Dose-dependent increases were observed for exposure-adjusted event rates (EAERs) of serious infections, herpes zoster (HZ), hepatic disorders, hematologic lab-related adverse events, and creatine phosphokinase (CPK) elevations, but not for exposure-adjusted incidence rates (EAIRs) of major adverse cardiovascular events (MACE), venous thromboembolic events (VTEs), or malignancies (Table 1). Generally, rates of TEAEs were lower with UPA 15 mg versus 30 mg.

Conclusion: In patients with PsA and prior inadequate response to ≥ 1 bDMARD, UPA efficacy was maintained over 56 weeks with no new safety signals.

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Table 1. Safety through Week 56

Event	UPA 15 mg QD (n=290; PY=419.4)	UPA 30 mg QD (n=308; PY=423.5)
EAER, events/100 PY (95% CI)		
Infection	89.7 (81.0–99.2)	113.6 (103.9–124.2)
Serious infection	2.6 (1.5–4.7)	6.1 (4.2–9.0)
Opportunistic infection ^a	0.7 (0.2–2.2)	0.9 (0.4–2.5)
HZ	3.8 (2.3–6.2)	8.5 (6.1–11.8)
Active TB	0	0
Gastrointestinal perforation (adjudicated)	0	0
Hepatic disorder	4.8 (3.1–7.4)	17.7 (14.1–22.2)
Anemia	2.1 (1.1–4.1)	5.4 (3.6–8.2)
Neutropenia	1.0 (0.4–2.5)	3.1 (1.8–5.3)
Lymphopenia	0.7 (0.2–2.2)	2.4 (1.3–4.4)
CPK elevation	5.2 (3.5–8.0)	8.7 (6.3–12.1)
Renal dysfunction	0.5 (0.1–1.9)	0.2 (0.0–1.7)
EAIR, n/100 PY (95% CI)		
NMSC ^b	1.2 (0.5–2.9)	1.0 (0.4–2.5)
Malignancy other than NMSC ^c	1.2 (0.5–2.9)	1.2 (0.5–2.9)
Lymphoma ^d	0.5 (0.1–1.9)	0
MACE (adjudicated)	0.2 (0–1.7)	0.2 (0–1.7)
VTE (adjudicated)	0.2 (0–1.7)	0.2 (0–1.7)

^aExcludes TB and HZ. ^bUPA 15 mg: 4 cases of BCC and 1 case of SCC of the skin; UPA 30 mg: 3 cases of BCC and 3 cases of SCC of the skin. ^cUPA 15 mg: 2 cases of prostate cancer, and single cases of malignant melanoma, ovarian cancer, and rectal cancer; UPA 30 mg: single cases of basosquamous carcinoma (considered NMSC after medical review), malignant melanoma, oropharyngeal SCC, and rectal adenocarcinoma, as well as endometrial cancer and ovarian cancer (in the same patient). ^dUPA 15 mg: 2 events of treatment-emergent abnormal lymphocyte morphology; abnormal lymphocytes were not reported in subsequent laboratory testing BCC, basal cell carcinoma; CI, confidence interval; NMSC, non-melanoma skin cancer; PY, patient-years; SCC, squamous cell carcinoma; TB, tuberculosis

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Figure Efficacy through Week 56 (non-responder imputation)

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POS0197

RESPONSIVENESS OF ULTRASOUND SYNOVITIS AND CLINICAL OUTCOMES IN PSORIATIC ARTHRITIS TREATED WITH SECUKINUMAB: DATA FROM THE ULTIMATE TRIAL

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Background: Power Doppler ultrasonography is a sensitive imaging tool to assess synovitis in psoriatic arthritis (PsA).^{1,2} ULTIMATE (NCT02662985) is the first large, randomised, double-blind placebo-controlled phase IIIb study in PsA, using ultrasound to evaluate early response to secukinumab on synovitis. The use of the standardised and reliable global EULAR-OMERACT composite ultrasound synovitis score at patient level (GLOESS) as the primary endpoint showed the early and significant benefit of secukinumab vs. placebo on synovitis at week 12.³

Objectives: To investigate the responsiveness and discriminative validity of GLOESS compared to clinical outcomes on joints at week 12 and report ultrasound and clinical efficacy data up to week 24.

Methods: This is a 52-week study with a 12-week double-blind, placebo-controlled period followed by 12-week open-label (OL) and 6-month OL extension. All placebo patients were switched to secukinumab (300 or 150 mg) at week 12.³ Discriminative validity of GLOESS was analysed post-hoc: within-group responsiveness was assessed by comparing its standardised response mean (SRM) to that of core set of ACR response in the initial secukinumab group over 12 and 24 weeks. Mean change from baseline up to week 12 of GLOESS was determined with mixed-effect model repeated measures analysis (MMRM) and from week 12-24 as observed. 24 week efficacy outcomes included ACR responses, HAQ-DI, PASI response and resolution of dactylitis. These outcomes were exploratory and reported either according to non-responder imputation (ACR response), or as observed (HAQ-DI, PASI response and resolution of dactylitis).

Results: Of 166 patients enrolled, a total of 155 patients (93%) completed 24 weeks of treatment (secukinumab, 79 patients 95%; placebo, 76 patients 92%). Mean change from baseline to week 24 in GLOESS was similar in the initial secukinumab and placebo-secukinumab groups. A continued improvement in GLOESS was observed in the secukinumab group, with catch-up improvement in the placebo group after switch to active therapy (Figure 1). Both SRM of GLOESS and ACR core components over 12 and 24 weeks were high (Table 1). Similar

clinical response rates were reported for clinical joint count, skin, dactylitis and function at week 24 in secukinumab and placebo switchers groups (Table 1).

Conclusion: These analyses highlight the responsiveness and high discriminative validity of GLOESS in PsA, resembling that of key clinical PsA manifestations and physical function.

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Table 1. Responsiveness of GLOESS - analysed by standardised response mean and efficacy outcomes at week 24

Standardised response mean (Initial secukinumab)	Efficacy of secukinumab				
	Week 0–12 (N = 83)	Week 0–24 (N = 83)	Responders (%)	Secukinumab week 0–24 (N = 83)	Placebo switchers week 12–24 (N = 83)
GLOESS	1.05	1.06	ACR20	87	74
Tender joint count	1.03	1.35	ACR50	64	49
Swollen joint count	0.91	1.18	ACR70	34	23
Patient global assessment of disease activity	1.37	1.55	HAQ-DI [†]	80	63
Physician global assessment of disease activity	1.59	2.39	PASI 75 [†]	72	59
Pain	1.32	1.53	PASI 90 [†]	62	45
HAQ-DI score	1.32	1.32	Resolution of dactylitis (LDI=0)	67	59

[†]HAQ-DI response is defined as an improvement of at least 0.35 score points compared to baseline (change ≤ -0.35).[†]N value for PASI response in secukinumab and placebo groups are 36 and 33; respectively. PASI response was calculated for patients with BSA ≥ 3 %. BSA, Body Surface Area; LDI, Leeds Dactylitis Index

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POS0198

EFFICACY AND SAFETY OF DEUCRAVACITINIB, AN ORAL, SELECTIVE TYROSINE KINASE 2 INHIBITOR, IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: RESULTS FROM A PHASE 2, RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

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*P < 0.05. Data presented as MMRM up to week 12. At week 12, mean difference (SE) = -3.16 (1.19), 95% CI = [-5.52, -0.83], P = 0.004. Data presented as observed from week 12 to 24. Open label period from week 12-24 (shaded area). No significant difference was observed in comparison of GLOESS score at week 24 between treatment groups as tested using t-test. At week 24, mean difference = 0.36, 95% CI = [-3.62, 3.74], P = 0.93.

Figure 1. Mean change from baseline in GLOESS by treatment through Week 24