Figure. ACR & IGA Responses at W24 by Select BL Characteristics

Figure 1

Table 1. ACR & IGA Responses at Weeks 24 & 52 & by Select BL Characteristics

	Guselkumab Q4W		Guselkumab Q8W	
	N=373		N=375	
	Week 24	Week 52	Week 24	Week 52
ACR20, %	62	72	60	70
SJC (<10/10-15/>15)	68/59/53	79/61/67	57/66/60	68/68/76
TJC (<10/10-15/>15)	74/67/56	73/76/69	62/60/60	75/68/68
csDMARD use (none/any/MTX)	66/60/63	80/68/68	62/59/57	73/68/68
ACR50, %	34	49	31	45
SJC (<10/10-15/>15)	41/32/20	58/39/38	34/28/26	46/40/49
TJC (<10/10-15/>15)	51/41/24	58/53/43	40/33/26	52/46/43
csDMARD use (none/any/MTX)	36/33/35	53/46/48	36/29/27	51/42/40
ACR70, %	16	27	16	27
SJC (<10/10-15/>15)	22/10/7	32/20/24	18/10/19	30/23/26
TJC (<10/10-15/>15)	29/19/9	34/32/22	27/15/14	35/28/24
csDMARD use (none/any/MTX)	21/13/14	30/26/27	21/14/14	34/24/23
IGA 0/1, %	71	80	66	71
BSA % with psoriasis (≥3-<10/>10-20/>20)	61/71/80	76/87/79	62/64/72	67/72/74
csDMARD use (none/any/MTX)	84/64/67	87/77/78	72/63/64	77/68/68

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AB0527

CORRELATIONS BETWEEN REDUCTIONS IN FATIGUE SEVERITY AND IMPROVEMENTS IN PHYSICAL FUNCTION AND CLINICAL RESPONSE IN PATIENTS WITH PSORIATIC ARTHRITIS: RESULTS FROM THE PHASE 3 DISCOVER PROGRAM

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Background: In patients (pts) with psoriatic arthritis (PsA), fatigue is a major driver of perceived impact of disease and has been identified as an important domain to be assessed in clinical trials.^{1,2} The association between fatigue and other PsA domains (eg, physical function) or clinical response is not well understood.

Objectives: Fatigue was measured with the Functional Assessment of Chronic Illness Therapy (FACIT)-Fatigue questionnaire in the pivotal DISCOVER-1 and DISCOVER-2 Phase 3 studies of guselkumab (GUS) vs placebo (PBO). This post hoc analysis explores the correlation between FACIT-Fatigue and physical function and clinical response in the DISCOVER program.

Methods: This analysis used pooled data from pts (N=1120) treated with GUS or PBO. In DISCOVER-1 and DISCOVER-2, 381 pts with active PsA (swollen joint count [SJC] ≥3, tender joint count [TJC] ≥3, C-reactive protein [CRP] ≥0.3mg/dL) and 739 pts with active PsA (SJC ≥5, TJC ≥5, CRP ≥0.6mg/dL) and inadequate response to standard therapies, respectively, were randomized 1:1 to GUS 100mg Q4W; GUS 100mg at W0, W4, then Q8W; or PBO. PBO pts switched to GUS 100mg Q4W at W24. The FACIT-Fatigue questionnaire has 13 items that assess self-reported fatigue/tiredness over the last 7 days. Items are scored from 0 (very much fatigued) to 4 (not at all fatigued). FACIT-Fatigue response was defined as an increase of ≥4 points from baseline. Physical function was evaluated with the Health Assessment Questionnaire-Disability Index (HAQ-DI). HAQ-DI response was defined as a decrease of ≥0.35 points from baseline. Clinical response was defined as achievement of ≥20%

improvement in the American College of Rheumatology (ACR 20) criteria. Relationships between FACIT-Fatigue and HAQ-DI at W8/W16/24 were assessed by Pearson correlation coefficients. Mean changes in HAQ-DI scores at W8/W16/24 were summarized in FACIT-Fatigue responders and nonresponders. A logistic regression model was applied to estimate odds ratios (ORs) for achievement of HAQ-DI and ACR 20 response by FACIT-Fatigue response status at each visit. A multiple linear regression model was used to evaluate the association between FACIT-Fatigue and HAQ-DI at W24 after adjusting for SJC, TJC, CRP, and pt assessment of pain.

Results: FACIT-Fatigue and HAQ-DI scores and changes from baseline were negatively correlated at W8, W16, and W24 (Table 1). Mean changes in HAQ-DI were -0.31, -0.43, and -0.48 at W8, W16, and W24, respectively, in FACIT-Fatigue responders and -0.06, -0.07, and -0.09, respectively, in FACIT-Fatigue nonresponders. FACIT-Fatigue responders were significantly more likely than nonresponders to achieve HAQ-DI and ACR 20 response (OR [95% CI] at W8, 2.8 [2.2-3.7] and 2.4 [1.9-3.1]; at W16, 3.6 [2.8-4.7] and 2.9 [2.3-3.7]; and at W24, 4.4 [3.4-5.7] and 3.2 [2.5-4.2], respectively; Figure 1). Correlations between FACIT-Fatigue and HAQ-DI remained significant after adjusting for SJC, TJC, CRP, and pt assessment of pain.

Conclusion: In pts with PsA, fatigue response is a clinically meaningful predictor of improvements in physical function and achievement of ACR 20 response, reinforcing the importance of assessing fatigue in PsA disease management.

REFERENCES:

- [1] Leung YY et al. *J Rheumatol*. 2020;96:46-9.
- [2] Gudu T et al. *Joint Bone Spine*. 2016;83:439-43.

Table 1. Correlation* of FACIT-Fatigue and HAQ-DI

Visit	HAQ-DI and FACIT Fatigue Scores	Changes From Baseline in HAQ-DI and FACIT-Fatigue Scores
W8	-0.61 (p<0.0001)	-0.42 (p<0.0001)
W16	-0.60 (p<0.0001)	-0.47 (p<0.0001)
W24	-0.62 (p<0.0001)	-0.50 (p<0.0001)

*Determined by Pearson correlation coefficient; p-values derived from hypothesis tests of correlation $\rho=0$ (ie, no correlation).

Figure. HAQ-DI and ACR 20 Responders by FACIT-Fatigue Responder Status (Y/N)

Figure 1

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AB0528

COMPARABLE SAFETY PROFILE OF GUSELKUMAB IN PSORIATIC ARTHRITIS AND PSORIASIS: RESULTS FROM PHASE 3 TRIALS THROUGH 1 YEAR

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Background: DISCOVER 1&2 (PsA) and VOYAGE 1&2 (PsO) are Phase 3 trials of guselkumab (GUS).

Objectives: Compare safety results through up to 1yr of GUS in PsA and PsO pts.

Methods: In DISCOVER, 1120 pts with active PsA despite standard therapy were treated. Most pts were biologic-naïve; ~30% in DISCOVER 1 had previous exposure to 1-2 TNFi. Concomitant MTX (57%), oral corticosteroids (17%), and NSAIDs (64%) were permitted. Pts were randomized to SC GUS 100mg at W0, W4, then Q8W; GUS 100mg Q4W; or PBO. At W24, PBO patients were switched to GUS 100mg Q4W. In VOYAGE, in which concomitant MTX use was prohibited, 1245 pts with moderate to severe PsO were treated and randomized to SC GUS 100mg at W0, W4, W12, then Q8W; or PBO at W0, W4, W12, with crossover to GUS at W16, W20, then Q8W. AEs and laboratory parameters, analyzed by National Cancer Institute-Common Terminology Criteria for AEs [NCI-CTCAE] toxicity grades, were summarized through the PBO-controlled periods and 1yr.

Results: Safety profiles were generally consistent across the GUS PsO and PsA clinical programs (Table 1). Time-adjusted incidence rates for numbers of AEs, serious AEs, serious infections, malignancy, MACE and AEs leading to d/c were generally similar between PsO and PsA. No cases of anaphylaxis or opportunistic infections were reported. Proportions of pts with decreased neutrophil counts and elevations in hepatic transaminases were slightly higher in PsA vs PsO. These abnormalities were mostly of NCI-CTCAE Grade 1 or 2 (<LLN-1000/mm³ for neutrophils; <5.0 x ULN for AST/ALT), generally transient, required no medical interventions, resolved spontaneously, and did not lead to interruption or d/c of treatment. Through 1yr, proportions of pts with ALT/AST elevations in PsA trials were slightly higher for GUS Q4W than Q8W and in pts with vs without baseline MTX use.

Conclusion: The GUS safety profile was generally consistent in PsA and PsO GUS-treated pts through 1yr of the DISCOVER and VOYAGE trials.