Patient-Reported Outcomes in Participants With Active and Erosive Psoriatic Arthritis Receiving Guselkumab: Results Through Week 48 of a Phase 3b, Randomized Double-Blind, Placebo-Controlled Study

Proton Rahman,[1] Philip J. Mease,[2,3] Christopher T. Ritchlin,[4] Elizabeth Adamson,[5] Soumya D. Chakravarty,[5,6] Alexa P. Kollmeier,[7] Weihong Hu,[8] Karissa Lozenski,[6] Arthur Kavanaugh,[9] Laura C. Coates[10]

[1]Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. Johns, NL, Canada; [2]Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; [3]University of Washington School of Medicine, Seattle, WA, USA; [4]Department of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester, NY, USA; [5]Johnson & Johnson, Horsham, PA, USA; [6]Drexel University College of Medicine, Philadelphia, PA, USA; [7]Johnson & Johnson, San Diego, CA, USA; [8]Johnson & Johnson, Spring House, PA, USA; [9]Center for Innovative Therapy, Division of Rheumatology, Allergy, Immunology, University of California San Diego, La Jolla, CA, USA; [10]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Botnar Research Centre Oxford, UK

Scan the QR code.
The QR code is intended to provide scientific information for individual reference, and the information should not be altered or reproduced in any way.

Key Takeaways

- **In APEX, GUS-randomized pts showed numerically increased LSM improvements in fatigue and overall disease impact from W4 to W48**
- **Among GUS-randomized pts, achievement of CMI in FACIT-Fatigue and PsAID-12 (responders) as early as W12 was associated with less SD progression than nonresponders**

Background

- Guselkumab (GUS), a fully human, dual-acting[1] monoclonal antibody that selectively inhibits the interleukin-23p19 subunit, has demonstrated multidomain efficacy in patients with active psoriatic arthritis (PsA)
- In the Phase 3b, multicenter, randomized, double-blind, placebo (PBO)-controlled APEX study, GUS treatment improved PsA signs/symptoms and significantly inhibited structural damage (SD) progression compared with PBO at Week (W) 24 in biologic-naïve participants (pts) with active and erosive PsA[2]

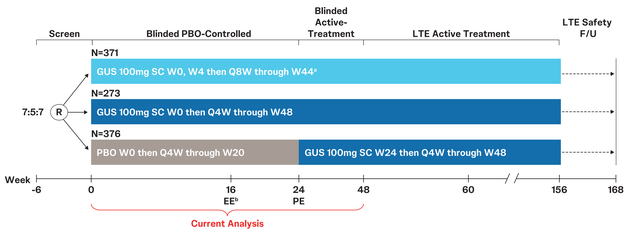
Objective

The current analysis evaluates the effects of GUS through W48 using patient-reported outcomes (PROs) assessing fatigue and impact of PsA on physical and psychological aspects, and their association with SD progression at W24 and W48

APEX Study Design

Inclusion Criteria

- ✓ Biologic-naive
- ✓ Age ≥18 years
- ✓ Active PsA ≥6 months (despite prior csDMARD, apremilast, NSAID); CASPAR criteria met
- ✓ ≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL
- ✓ **≥2 erosive joints on hand/foot radiographs**



[a]PBO SC W0 then Q8W through W48 administered to maintain blinding. [b]EE if ≤20% improvement from baseline in both TJC and SJC at W16, EE pts may initiate/increase dose of permitted medication up to the maximum dose, at the investigator's discretion. **CASPAR**=CASPAR classification criteria for Psoriatic Arthritis; **CRP**=C-reactive protein; **csDMARD**=conventional synthetic disease-modifying antirheumatic drug; **EE**=early escape; **F/U**=follow-up; **LTE**=long-term extension; **NSAID**=nonsteroidal anti-inflammatory drug; **PE**=primary endpoint; **Q4W/Q8W**=every 4/8 weeks; **R**=randomization; **SC**=subcutaneous; **SJC**=swollen joint count; **TJC**=tender joint count.

Methods

Endpoints	Statistical Analysis
LSM improvements from baseline through W48 for: **FACIT-Fatigue** • 13-item questionnaire evaluating patient fatigue/impact on daily function over the last 7 days (total score range, 0-52; lower scores reflect more severe fatigue) **PsAID-12** • 12-item questionnaire assessing the impact of PsA on physical and psychological domains of patient health[a] (total score range, 0-10; higher scores indicate a higher impact of disease)	• LSM improvements and p-values were based on MMRM and not multiplicity controlled; they are considered nominal and may not be used to claim statistical significance • Pts were considered to have a change of 0 if they initiated/increased the dose of oral corticosteroids or csDMARDs or initiated protocol-prohibited PsA therapies through W24, or discontinued study agent for any reason other than ND/MD through W48 • Data affected by ND/MD were not used • After applying these rules, remaining missing data were accounted for in the analysis model
LSM changes in **PsA modified vdH-S score** at W24 and W48 among GUS-randomized pts were analyzed by response status (CMI) • FACIT-Fatigue CMI: ≥4 • PsAID-12 CMI: ≥3	• For change in vdH-S scores, data affected by ND/MD were not used; missing data were imputed using MI

[a]Includes pain, fatigue, skin problems, work/leisure activities, functional capacity, discomfort, sleep disturbance, coping, anxiety, embarrassment, social participation, and depression. **CMI**=clinically meaningful improvement; **FACIT**=Functional Assessment of Chronic Illness; **LSM**=least squares mean; **PsAID-12**=Psoriatic Arthritis Impact of Disease 12-item questionnaire; **ND**=major disruption (Ukraine/Russia crisis); **MI**=multiple imputation; **MMRM**=Mixed Model for Repeated Measures; **ND**=natural disaster (COVID-19 site access restrictions); **vdH-S**=van der Heijde-Sharp.

Results

Baseline demographics were well balanced among treatment groups

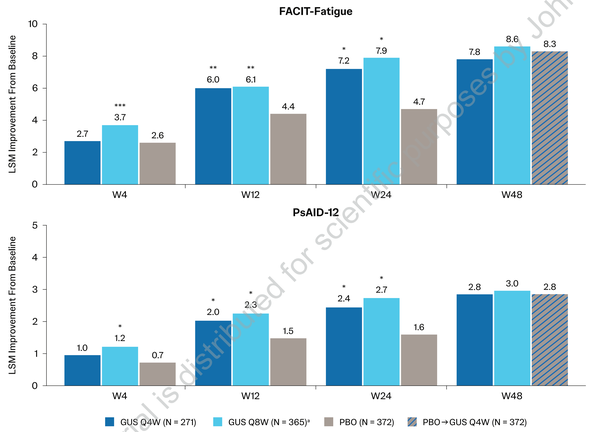
- Mean baseline disease characteristics were consistent across treatment groups

	GUS Q4W (N=273)	GUS Q8W (N=371)	PBO (N=376)	Total (N=1020)
Demographics				
Age, yrs	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)	53.0 (13.0)
Male	55%	54%	57%	55%
White	85%	82%	83%	83%
BMI, kg/m^2	29.4 (6.0)	29.0 (5.6)	28.9 (5.7)	29.1 (5.7)
PsA Characteristics				
PsA Disease Duration, yrs	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)	7.3 (7.2)
SJC (0-66)	11.6 (9.4)	12.1 (8.5)	11.8 (8.9)	11.9 (8.9)
TJC (0-68)	21.2 (14.6)	20.6 (13.4)	20.5 (13.9)	20.7 (13.9)
Patient Assessment of Pain (VAS; 0-10cm)	5.9 (2.2)	5.9 (2.1)	5.9 (2.1)	5.9 (2.1)
Patient's Global Assessment (VAS; 0-10cm)	5.9 (2.2)	5.9 (2.1)	5.9 (2.0)	5.9 (2.1)
Physician's Global Assessment (VAS; 0-10cm)	6.4 (1.6)	6.4 (1.6)	6.2 (1.7)	6.3 (1.7)
CRP, mg/dL	1.7 (2.9)	1.5 (2.0)	1.7 (2.5)	1.6 (2.5)
Dactylitis (1-60)[a]	10.8 (11.5)	11.0 (12.8)	10.2 (10.5)	10.6 (11.6)
Enthesitis LEI (1-6)[b]	3.2 (1.8)	3.0 (1.7)	3.0 (1.6)	3.1 (1.7)
HAQ-DI (0-3)	1.2 (0.7)	1.2 (0.6)	1.2 (0.7)	1.2 (0.7)
FACIT-Fatigue (0-52)	30.8 (11.3)	31.1 (10.2)	31.2 (11.0)	31.1 (10.8)
PsAID-12 (0-10)	5.1 (2.2)	5.2 (2.0)	5.1 (2.0)	5.1 (2.1)

Data shown are mean (SD) unless otherwise noted. [a]Among pts with dactylitis at baseline: GUS Q4W, N=159; GUS Q8W, N=143; PBO, N=167. [b]Among pts with enthesitis at baseline: GUS Q4W, N=157; GUS Q8W, N=214; PBO, N=218. ***BMI****=body mass index,* ***HAQ-DI****=Health Assessment Questionnaire-Disability Index,* ***LEI****=Leeds Enthesitis Index,* ***VAS****=visual analog scale.*

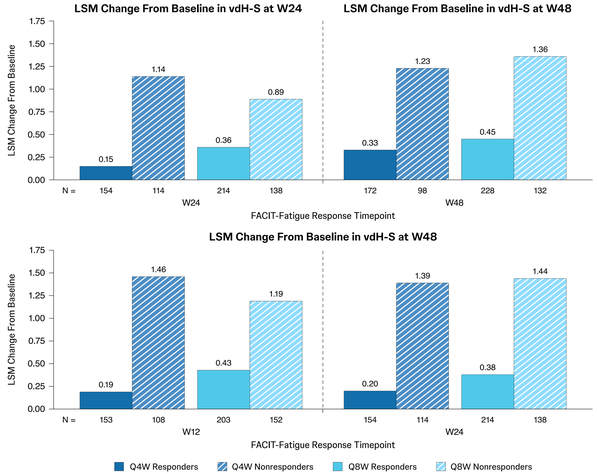
LSM improvements in FACIT-Fatigue and PsAID-12 through W48 among GUS-randomized pts

- GUS-randomized pts achieved greater LSM improvements from baseline in FACIT-Fatigue and PsAID-12 compared to PBO through W24
- At W48, PBO→GUS Q4W pts achieved comparable LSM improvement to those of GUS-randomized pts

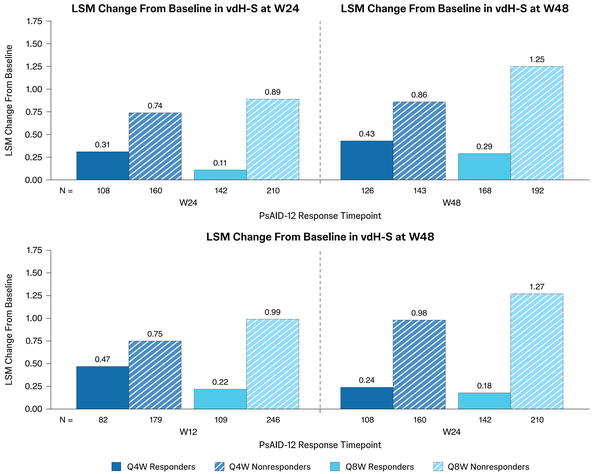


*Nominal *p=0.001, **p=0.01, ***p=0.05. [a]At W48, N=369.*

LSM change from baseline in vdH-S at W24 and W48 by FACIT-Fatigue response among GUS-randomized pts



LSM change from baseline in vdH-S at W24 and W48 by PsAID-12 response among GUS-randomized pts



Material is distributed for scientific purposes by Johnson & Johnson Innovative Medicine and is not for promotional use.

PRESENTED AT: American College of Rheumatology (ACR) Convergence 2026; November 6–11, 2026; Orlando, FL, USA. **REFERENCES: 1.** Sachen KL, et al. *Front Immunol.* 2025;16:1532852. **2.** Mease PJ, et al. *Ann Rheum Dis.* 2025;84:1983-94. **ACKNOWLEDGMENTS:** Medical writing support was provided by Alyssa Cortina, Pharm D, under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298-1304). Sponsored by Johnson & Johnson. **DISCLOSURES: PR** has received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; has received meeting attendance/travel support from Johnson & Johnson; and has received research grants from Johnson & Johnson and Novartis. **PJM** has received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, Sun Pharma, Takeda, and UCB; has received grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; and has received speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **CTR** has been a consultant for AbbVie, Eli Lilly, Johnson & Johnson, Novartis, and UCB; and has received grant/research support from Johnson & Johnson, Novartis, and UCB. **EA, SDC, APK,** and **KL** are employees of Johnson & Johnson, and may own stock or stock options in Johnson & Johnson. **WH** is an employee of Cytel, Inc. providing statistical support (funded by Johnson & Johnson). **AK** has received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB. **LCC** has received grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB; has worked as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; and has been paid as a speaker for AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Johnson & Johnson, Medac, Novartis, Pfizer, and UCB.