Achievement of DAPSA/cDAPSA Low Disease Activity With Guselkumab in Active and Erosive Psoriatic Arthritis Across Participant Subgroups in APEX

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Key Takeaways

- **In APEX, GUS-treated pts had greater response rates for achieving cDAPSA/DAPSA LDA/REM through W24 vs PBO-treated pts**
  - **Response rates numerically increased through 1 year for GUS-randomized pts**
- **Achievement of cDAPSA/DAPSA LDA/REM with GUS was generally consistent with both dosing regimens and across diverse pt subgroups defined by baseline demographics, disease characteristics, concomitant csDMARD use, and structural damage**

Background

Psoriatic arthritis (PsA) is a heterogenous, chronic, inflammatory disease characterized by peripheral arthritis, axial involvement, dactylitis, enthesitis, psoriatic skin, and nail disease

Guselkumab (GUS) is a fully human, dual-acting,[1] monoclonal antibody that selectively inhibits the interleukin (IL)-23p19 subunit and is approved to treat moderate-to-severe plaque psoriasis (PsO), active psoriatic arthritis (PsA), and moderately-to-severely active Crohn's disease and ulcerative colitis[2]

In the ongoing phase 3b, randomized, double-blind, placebo (PBO)-controlled APEX study of participants with active and erosive PsA, GUS demonstrated efficacy vs PBO in improving signs and symptoms of PsA and significantly inhibiting structural damage progression at Week (W) 24[3]

Objectives

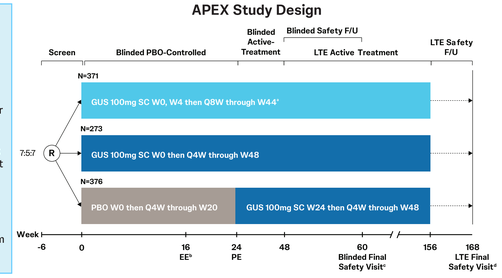
This post hoc analysis evaluated the consistency of GUS treatment effect in achieving low disease activity (LDA)/remission (REM) using the Disease Activity Index for PsA (DAPSA) and clinical DAPSA (cDAPSA) at W24 across baseline subgroups in APEX study

Methods

Key Inclusion Criteria

- Biologic-naïve
- ≥18 years
- Active PsA (≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL) for ≥6 months (despite prior csDMARDs, apremilast, NSAIDs); CASPAR criteria met
- **≥2 erosive joints on hand/foot radiographs**
- Active plaque PsO (≥1 PsO plaque ≥2 cm and/or nail PsO)

APEX Study Design



DAPSA[4]	cDAPSA[4,5]
REM	
≤4	≤4
LDA	
≤14	≤13
ModDA	
≤28	≤27
HDA	
>28	>27
Components	
• SJC • TJC • Pt Pain • PtGA • CRP	• SJC • TJC • Pt Pain • PtGA

[a]PBO SC W8 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. [c]Final safety visit for those who do not enter LTE. [d]Final safety visit for those who entered LTE. ***BID***=twice daily, ***BMI***=body mass index, ***CASPAR***=Classification Criteria for Psoriatic Arthritis, ***cDAPSA***=Clinical Disease Activity Index for Psoriatic Arthritis, ***CRP***=C-reactive protein, ***csDMARDs***=conventional synthetic disease-modifying antirheumatic drugs, ***DAPSA***=Disease Activity Index for Psoriatic Arthritis, ***EE***=early escape, ***GLMM***=generalized linear mixed model, ***GUS***=guselkumab, ***HDA***=high disease activity, ***JC***=joint count, ***JSN***=joint space narrowing, ***LDA***=low disease activity, ***LTE***=long-term extension, ***MD***=major disruption, ***ModDA***=moderate disease activity, ***MRI***=magnetic resonance imaging, ***NRI***=nonresponder imputation, ***NSAIDs***=nonsteroidal anti-inflammatory drugs, ***PASI***=Psoriasis Area and Severity Index, ***PBO***=placebo, ***PE***=primary endpoint, ***PsA***=psoriatic arthritis, ***PsO***=psoriasis, ***Pt***=patient, ***PtGA***=patient's global assessment, ***Q4W***=every 4 weeks, ***Q8W***=every 8 weeks, ***R***=randomization, ***REM***=remission, ***SC***=subcutaneous, ***SJC***=swollen joint count, ***TJC***=tender joint count, ***vdH-S***=van der Heijde–Sharp, ***W***=week.

Endpoints

- cDAPSA and DAPSA LDA/REM (score ≤13 or ≤14, respectively) through W48
- cDAPSA and DAPSA LDA/REM at W24 by baseline subgroups
  - Demographics (Sex, BMI)
  - PsA characteristics (PsA duration, SJC, TJC, CRP, PASI)
  - Concomitant medication use (csDMARDs)
  - Radiographic features (total PsA-modified vdH-S, erosion, and JSN scores; number of joints with erosions and JSN)

Statistical Analyses

- Response rates and p-values from W4–24 were based on GLMM; p-values were not multiplicity controlled, are considered nominal, and may not be used to claim statistical significance
- Pts were considered nonresponders at a post-baseline visit through W24 if they had previously initiated/increased oral corticosteroids or csDMARDs or initiated protocol-prohibited PsA therapies and through W48 if they discontinued study agent for any reason other than ND/MD. Data affected by ND/MD were not used. After applying these rules, missing data due to ND/MD were accounted for in the analysis model, and other missing data were imputed using NRI
- Efficacy analyses included all randomized pts except those from Ukrainian sites unable to support key study operations due to major disruption (mFAS, N=1020)

Results

Baseline characteristics of pts with active and erosive PsA were balanced across treatment groups

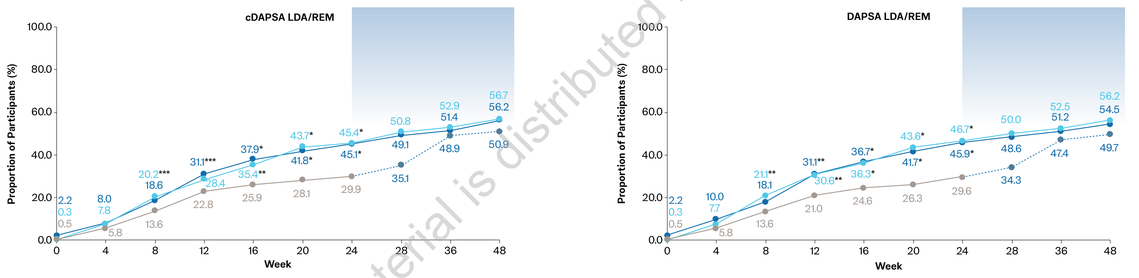
Table 1. Baseline characteristics

Baseline Characteristics	GUS Q4W (N = 273)	GUS Q8W (N = 371)	PBO (N = 376)
Demographics			
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)
Female	45.4%	46.4%	43.4%
PsA Characteristics			
PsA disease duration, years	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)
SJC [0–66]	11.6 (9.4)	12.1 (8.5)	11.8 (8.9)
TJC [0–68]	21.2 (14.6)	20.6 (13.4)	20.5 (13.9)
HAQ-DI [0–3]	1.2 (0.7)	1.2 (0.6)	1.2 (0.6)
Patient assessment of pain [VAS; 0–10cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.1)
Patient's global assessment - arthritis [VAS; 0–10cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.0)
Physician's global assessment [VAS; 0–10cm]	6.4 (1.6)	6.4 (1.6)	6.2 (1.7)
CRP, mg/dL[a]	0.7 (0.4; 1.5)	0.8 (0.4; 1.6)	0.8 (0.4; 1.8)
DAPSA	46.4 (24.3)	46.1 (22.1)	46.0 (22.8)
cDAPSA	44.7 (23.5)	44.6 (21.7)	44.3 (22.3)
PsO Characteristics			
%BSA	15.0 (19.2)	16.5 (21.9)	16.3 (21.5)
PASI [0-72]	7.6 (8.3)	8.3 (10.1)	8.2 (9.5)

[a]Values are mean (SD) unless otherwise noted.
BSA=body surface area, ***CRP***=C-reactive protein, ***cDAPSA***=clinical disease activity index for psoriatic arthritis, ***DAPSA***=disease activity index for psoriatic arthritis, ***GUS***=guselkumab, ***HAQ-DI***=health assessment questionnaire–disability index, ***N***=number of patients, ***PASI***=Psoriasis Area and severity index, ***PBO***=placebo, ***PsA***=Psoriatic Arthritis, ***Q4W***=every 4 weeks, ***Q8W***=every 8 weeks, ***SD***=standard deviation, ***SJC***=swollen joint count, ***TJC***=tender joint count, ***VAS***=visual analog scale.

Through W24, greater proportions of GUS-treated pts achieved cDAPSA and DAPSA LDA/REM vs PBO

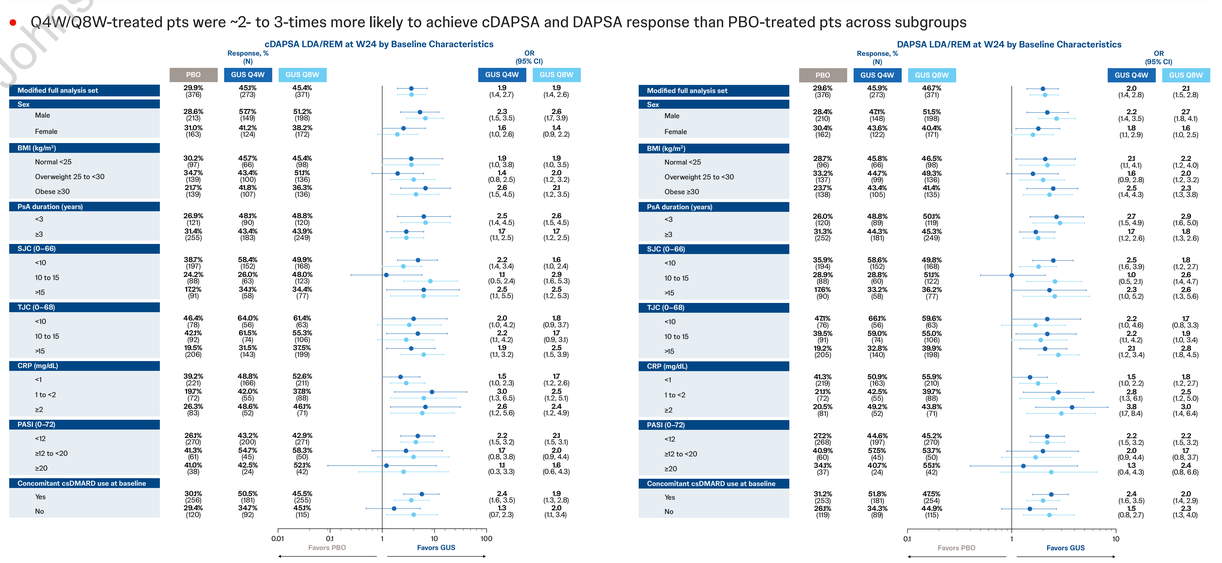
- Response rates for GUS-randomized pts increased through W48



*Nominal. *p≤0.001, **p<0.01, ***p<0.05. Post-baseline (W4–48) response rates based on GLMM; observed data were reported at W0. cDAPSA: GUS Q4W N=273; GUS Q8W N=369 (W0), N=370 (W4–24), N=371 (W28–48); PBO → GUS Q4W N=375 (W0), N=376 (W4–48). DAPSA: GUS Q4W N=273 (W0), N=270 (W4–24), N=273 (W28–48); GUS Q8W N=369 (W0–24), N=371 (W28–48); PBO → GUS N=375 (W0), N=372 (W4–24), N=376 (W28–48).*
cDAPSA=clinical Disease Activity Index for Psoriatic Arthritis, ***DAPSA***=Disease Activity Index for Psoriatic Arthritis, ***GLMM***=generalized linear mixed model, ***GUS***=guselkumab, ***LDA***=low disease activity, ***PBO***=placebo, ***pts***=patients, ***Q4W***=every 4 weeks, ***Q8W***=every 8 weeks, ***REM***=remission, ***W***=week.

At W24, GUS treatment effect in achieving cDAPSA and DAPSA LDA/REM was consistent across subgroups

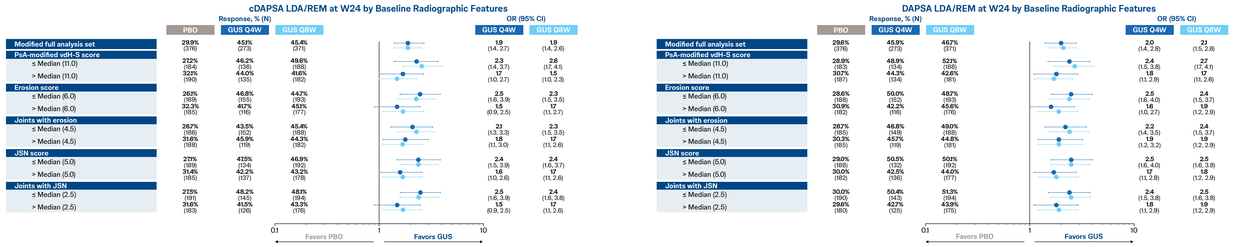
- Q4W/Q8W-treated pts were ~2- to 3-times more likely to achieve cDAPSA and DAPSA response than PBO-treated pts across subgroups



BMI=body mass index, ***cDAPSA***=clinical Disease Activity Index for Psoriatic Arthritis, ***CI***=confidence interval, ***CRP***=C-reactive protein, ***csDMARD***=conventional synthetic disease-modifying antirheumatic drugs, ***DAPSA***=Disease Activity Index for Psoriatic Arthritis, ***GUS***=guselkumab, ***LDA***=low disease activity, ***OR***=odds ratio, ***PASI***=Psoriasis Area and Severity Index, ***PBO***=placebo, ***PsA***=psoriatic arthritis, ***Q4W***=every 4 weeks, ***Q8W***=every 8 weeks, ***REM***=remission, ***SJC***=swollen joint count, ***TJC***=tender joint count, ***TNFi***=tumor necrosis factor inhibitor, ***W***=week.

At W24, GUS treatment effect in achieving cDAPSA and DAPSA LDA/REM was consistent across subgroups defined by baseline radiographic features

- Q4W/Q8W-treated pts were ~2- to 2.5-times more likely to achieve cDAPSA and DAPSA response than PBO-treated pts



CI=confidence interval, ***cDAPSA***=clinical Disease Activity Index for Psoriatic Arthritis, ***DAPSA***=Disease Activity Index for Psoriatic Arthritis, ***GUS***=guselkumab, ***JSN***=joint space narrowing, ***LDA***=low disease activity, ***N***=number of patients, ***OR***=odds ratio, PBO=placebo, ***PsA***=psoriatic arthritis, ***Q4W***=every 4 weeks, ***Q8W***=every 8 weeks, ***pts***=patients, ***REM***=remission, ***vdH-S***=van der Heijde–Sharp score, ***W***=week.

PRESENTED BY: Alejandro Lizano Corzo at Florida Society of Rheumatology (FSR); July 16-19, 2026; Maui, HI, USA. **REFERENCES: 1.** Sachen KL. *Front Immunol.* 2025;16:1532852. **2.** Tremfya: Package insert. Horsham, PA: Janssen Biotech, Inc.; 2025. **3.** Mease PJ, et al. *Ann Rheum Dis.* 2025;84:1983–1994. **4.** Schoels M. *Ann Rheum Dis.* 2016;75:811–818. **5.** Helliwell PS. Arthritis Care Res. 2014;66:749–756. **ACKNOWLEDGMENTS:** Medical writing support was provided by Teresa Tartaglione, PharmD, of Certara, Radnor, PA under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298–1304). Layout design and formatting for this presentation were provided by Mugdha Rokade of SIRO Medical Writing Pvt., Ltd., India (funded by Johnson & Johnson). This presentation was sponsored by Johnson & Johnson. **DISCLOSURES: CTR:** Grant/research support: Johnson & Johnson, Novartis, and UCB; Consultant: AbbVie, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **LCC:** Consultant: AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; Grants/research support: AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson,Novartis, Pfizer and UCB; Manuscripts: supported by the National Institute for Health Research (NIHR) Oxford Biomedical Research Centre (BRC). The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR, or the Department of Health; Speakers bureau: AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Johnson & Johnson, Medac, Novartis, Pfizer, and UCB. **PJM:** Consultant: AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Immagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB; Grant/research support: AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; Speakers bureau: AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **APK, SDC, SF, KL,** and **ALC (Presenter)** Employee: Johnson & Johnson, Shareholder: Owns stock or stock options in Johnson & Johnson. **WH:** Employee: Cytel providing statistical support (funded by Johnson & Johnson). **AO:** Consultant/Advisory Boards: AbbVie, Amgen, Bristol Myers Squibb, CorEvitas, Gilead, GSK, Johnson & Johnson, Eli Lilly, Novartis, Pfizer, UCB; Grant/research support: AbbVie to Penn, Pfizer to Penn, Novartis to Penn, Amgen to Forward/NDB; other funding: NIAMS, Rheumatology Research Foundation, National Psoriasis Foundation, University of Pennsylvania. **PR:** Consultant: AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; Grant/research support: Johnson & Johnson and Novartis; Meeting attendance/travel support: Johnson & Johnson. **PREVIOUSLY PRESENTED AT:** CCR-East: April 30–May 3, 2026; Destin, Florida, USA. **PREVIOUSLY PRESENTED AT:** CCR-East: April 30–May 3, 2026; Destin, Florida, USA.

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