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

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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 (pts) 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]

Objective

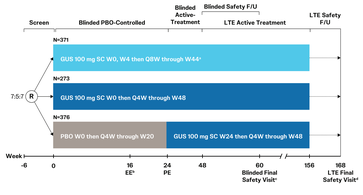
This post hoc analysis evaluated the consistency of GUS treatment effect in achieving low disease activity (LDA)/remission (REM) using the Disease Activity Index for PsA (DAPSA) and clinical DAPSA (cDAPSA) at W24 across baseline subgroups in APEX study

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

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)

*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 entered LTE. **BMI**=body mass index, **CASPAR**=ClASsification criteria for Psoriatic Arthritis, **cDAPSA**=clinical DAPSA, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease modifying antirheumatic drugs, **DAPSA**=Disease Activity Index for PsA, **EE**=early escape, **F/U**=follow-up, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **HDA**=high disease activity, **JSN**=joint space narrowing, **LDA**=low disease activity, **LTE**=long-term extension, **MD**=major disruption involving Ukraine and neighboring countries/territories beginning 24 February 2022, **mFAS**=modified full analysis set, **ModDA**=moderate disease activity, **ND**=natural disaster: site closure, site access restrictions, or lockdowns due to the COVID-19 pandemic, **NRI**=nonresponder imputation, **NSAIDs**=nonsteroidal anti-inflammatory drug, **PBO**=placebo, **PE**=primary endpoint, **PASI**=Psoriasis Area and Severity Index, **PsA**=psoriatic arthritis, **PsO**=psoriasis, **PtGA**=patient's global assessment, **Pt Pain**=patient's assessment of pain, **Q4W/Q8W**=every 4/8 weeks, **R**=randomization, **REM**=remission, **SC**=subcutaneous, **SJC**=swollen joint count, **TJC**=tender joint count, **vdH-S**=van der Heijde-Sharp, **W**=week.

Results

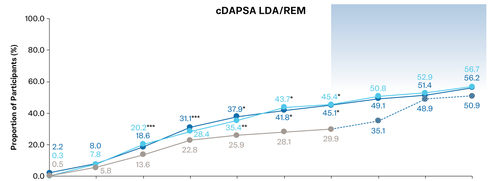
Baseline characteristics of pts with active and erosive PsA were balanced across treatment groups

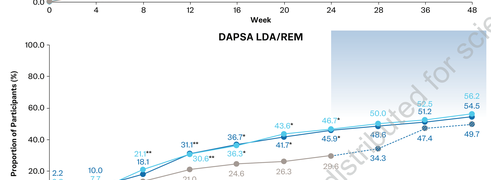
Baseline Characteristics	GUS Q4W (N=273)	GUS Q8W (N=371)	PBO (N=376)
Demographics			
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)
Female	45.4%	46.4%	43.4%
PsA Characteristics			
PsA disease duration, years	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)
SJC [0–66]	11.6 (9.4)	12.1 (8.5)	11.8 (8.9)
TJC [0–68]	21.2 (14.6)	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; 0–10 cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.1)
Patient's global assessment - arthritis [VAS; 0–10 cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.0)
Physician's global assessment [VAS; 0–10 cm]	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)

*Values are mean (SD) unless otherwise noted. **BSA**=body surface area, **cDAPSA**=clinical DAPSA, **CRP**=C-reactive protein, **DAPSA**=Disease Activity Index for PsA, **GUS**=guselkumab, **HAQ-DI**=health assessment questionnaire disability index, **PASI**=Psoriasis Area and Severity Index, **PBO**=placebo, **PsA**=psoriatic arthritis, **Q4W/Q8W**=every 4/8 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

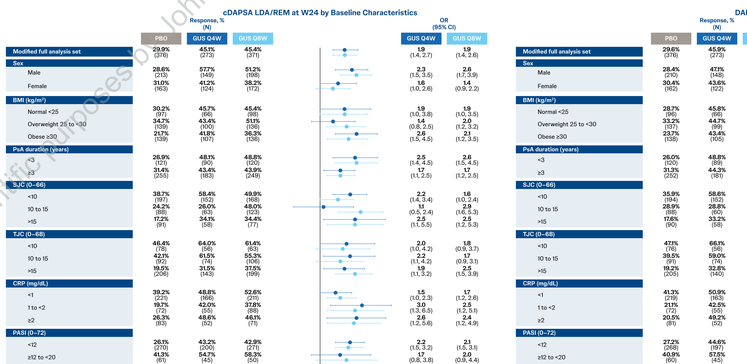


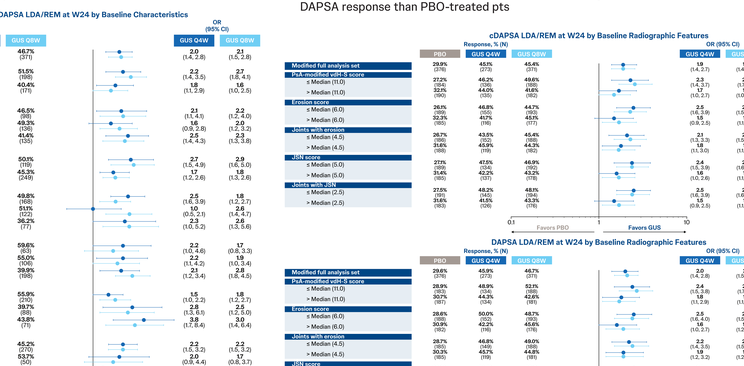


*Nominal. *p≤0.001, **p<0.01, ***p<0.05. Post-baseline (W4-48) response rates based on GLMM; observed data were reported at W0. cDAPSA: GUS Q4W N=273; GUS Q8W N=369 (W0), N=370 (W4–24), N=371 (W28–48); PBO → GUS Q4W N=375 (W0), N=369 (W4–48). DAPSA: GUS Q4W N=273 (W0), N=270 (W4–24), N=273 (W28–48); 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 DAPSA, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **LDA**=low disease activity, **PBO**=placebo, **Q4W/Q8W**=every 4/8 week, **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 DAPSA, **CI**=confidence interval, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease modifying antirheumatic drugs, **DAPSA**=Disease Activity Index for PsA, **GUS**=guselkumab, **LDA**=low disease activity, **OR**=odds ratio, **PASI**=Psoriasis Area and Severity Index, **PBO**=placebo, **PsA**=psoriatic arthritis, **Q4W/Q8W**=every 4/8 week, **REM**=remission, **SJC**=swollen joint count, **TJC**=tender joint count, **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

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

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**

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PRESENTED AT: The American College of Rheumatology (ACR) Convergence 2026; November 6–11, 2026; Orlando, Florida, USA. **REFERENCES: 1.** Sachen KL. *Front Immunol*. 2022;13:1532252. **2.** TremfyaPackage 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;66:1749–756. **ACKNOWLEDGMENTS:** Medical writing support was provided by Teresa Tartaglione, PharmD, of Certara, Radnor, PA under the direction of the author/s in accordance with Good Publication Practice guidelines (*Ann Intern Med*. 2022;175:1298–1304). Layout design and formatting for this encore presentation were provided by Ganesh Karpe (SIRO Medical Writing Pvt. Ltd., India). This presentation was sponsored by Johnson & Johnson. **DISCLOSURES: CTR:** Grant/research support: AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, 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, Bristol Myers Squibb, Celgene, 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:** ACR-East; April 30–May 3, 2026; Destin, Florida, USA.

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