Guselkumab Efficacy on Improvements in American College of Rheumatology Response Components and Association With Structural Damage Progression in Participants With Active and Erosive Psoriatic Arthritis: Results From a Phase 3b, Randomized Double-Blind, Placebo-Controlled Study

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Key Takeaways

- In APEX, GUS-randomized pts demonstrated higher rates of achieving ≥50% and ≥70% improvements in ACR components through W24 compared with PBO-randomized pts
- Across multiple ACR domains, improvements of ≥50% and ≥70% at W24 were associated with reduced structural damage progression at W24 and W48 for both GUS dosing regimens compared with nonresponders
- GUS-randomized pts achieving ≥50%/≥70% improvement in SJC, TJC, PGA, and CRP at W8 had less structural damage progression at W48

Background

Guselkumab (GUS) is a fully human, dual-acting[1] monoclonal antibody that selectively inhibits the interleukin-23p19 subunit

In APEX, GUS demonstrated efficacy across clinical endpoints and significantly inhibited structural damage progression at Week (W) 24 in participants (pts) with active and erosive psoriatic arthritis (PsA)[2]

Objective

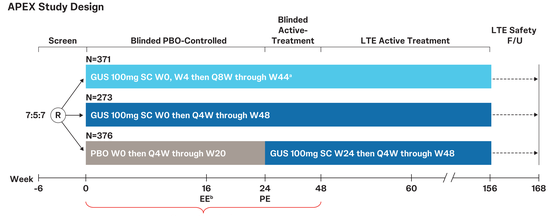
Here we evaluate the efficacy of GUS in improving individual components of the American College of Rheumatology (ACR) response criteria[3] through W24 and the association of these outcomes with structural damage progression through 1 year

APEX Methods

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**

APEX Study Design



Endpoints	Statistical Analysis
Proportion of pts achieving ≥50% and ≥70% improvement in ACR response components through W24: • SJC • TJC • Pt Pain • PtGA • PGA • HAQ-DI • CRP	• Pts who initiated/increased csDMARDs or oral corticosteroids or initiated protocol-prohibited medication/therapies for PsA through W24 or discontinued study intervention for any reason other than ND/MD through W48 were considered nonresponders – Data affected by ND/MD were not used • After applying these rules, missing data and/or data not used due to ND/MD were imputed using MI; other missing data were imputed using NRI • The average proportion of pts achieving response, over 200 MI datasets, is presented • P-values were based on the combined standardized Wilson-Hilferty transformation of the CMH test statistic stratified by the randomization strata levels, from each MI dataset
LSM changes in PsA modified vdH-S score among GUS-randomized pts at W24 and W48 were analyzed by W8 and W24 response status: • ≥50% improvement in ACR response components • ≥70% improvement in ACR response components	• For change in vdH-S scores, data affected by ND/MD were not used; missing data were imputed using MI • Included all GUS-randomized pts except those from Ukrainian sites unable to support key study operations

*[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. **ACR50/ACR70**=≥50%/≥70% improvement in ACR response criteria; **CASPAR**=CASsification criteria for Psoriatic ARthritis; **CMH**=Cochran-Mantel-Haenszel; **CRP**=C-reactive protein; **csDMARD**=conventional synthetic disease-modifying antirheumatic drug; **EE**=early escape; **F/U**=follow-up; **HAQ-DI**=Health Assessment Questionnaire-Disability Index; **LSM**=least squares mean; **LTE**=long-term extension; **MD**=major disruption (Ukraine/Russia crisis); **MI**=multiple imputation; **ND**=mutual disaster (COVID-19 site access restrictions); **NRI**=nonresponder imputation; **NSAID**=nonsteroidal anti-inflammatory drug; **PBO**=placebo; **PE**=primary endpoint; **PGA**=Physician's Global Assessment; **Pt Pain**=Patient-reported Pain; **PtGA**=Patient's Global Assessment (arthritis); **Q4W/Q8W**=every 4/8 weeks; **R**=randomization; **SC**=subcutaneous; **SJC**=swollen joint count; **TJC**=tender joint count; **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)
Pt Pain (VAS; 0-10 cm)	5.9 (2.2)	5.9 (2.1)	5.9 (2.1)	5.9 (2.1)
PtGA (arthritis, VAS; 0-10cm)	5.9 (2.2)	5.9 (2.1)	5.9 (2.0)	5.9 (2.1)
PGA (VAS; 0-10cm)	6.4 (1.6)	6.4 (1.6)	6.2 (1.7)	6.3 (1.7)
HAQ-DI (0-3)	1.2 (0.7)	1.2 (0.6)	1.2 (0.7)	1.2 (0.7)
CRP, mg/dL	1.7 (2.9)	1.5 (2.0)	1.7 (2.5)	1.6 (2.5)

*Data shown are mean (SD) unless otherwise noted. **BMI**=body mass index.*

Across all ACR response components, response rates for achieving ≥50% improvement were greater for GUS Q4W/Q8W compared with PBO through W24

- Separation from PBO occurred as early as W8-12 (nominal p<0.05)

Proportions of Pts Achieving ≥50% Improvement in ACR Response Components Through W24

GUS Q4W						
Week	4	8	12	16	20	24
SJC	29.3	52.6†	63.8*	70.6*	73.2*	74.4*
TJC	16.8	39.0*	53.5*	58.3*	64.8*	64.0*
Pt Pain	14.7	28.7‡	37.4‡	43.1*	46.6*	51.0*
PtGA	14.7	29.1†	36.3‡	41.3*	44.7*	51.3*
PGA	17.9‡	34.2‡	51.0*	57.8*	63.4*	66.1*
HAQ-DI	16.1†	24.2‡	23.9	30.1†	33.4‡	35.9*
CRP	26.2†	36.0*	38.3*	38.4*	38.9*	39.2*

GUS Q8W						
Week	4	8	12	16	20	24
SJC	32.4‡	52.0†	65.7*	68.7*	77.1*	74.8*
TJC	21.1‡	37.4‡	53.0*	62.7*	69.0*	68.0*
Pt Pain	12.4	28.7‡	36.3‡	41.4‡	47.5*	49.1*
PtGA	15.7	29.3†	38.2†	43.6*	47.6*	52.2*
PGA	18.1†	34.9*	47.2*	58.9*	67.0*	66.1*
HAQ-DI	14.3†	22.2‡	27.9‡	32.8*	32.3†	34.2†
CRP	30.2*	38.0*	39.7*	42.4*	43.2*	45.4*

PBO						
Week	4	8	12	16	20	24
SJC	25.3	41.4	48.3	52.1	58.3	56.8
TJC	14.9	26.4	38.2	42.3	47.6	47.5
Pt Pain	12.2	21.5	29.1	30.0	29.8	31.8
PtGA	11.7	19.7	26.7	28.6	30.9	30.5
PGA	12.0	23.4	33.7	40.5	40.7	42.6
HAQ-DI	8.2	15.0	21.2	21.1	23.5	23.8
CRP	16.5	21.0	23.0	22.0	24.4	26.2



*Nominal *p=0.001; †p=0.01; ‡p=0.05 vs PBO.*

Greater proportions of GUS Q4W/Q8W pts achieved ≥70% improvement compared with PBO across all ACR response components through W24

- Separation from PBO occurred as early as W8 (nominal p<0.05)

Proportions of Pts Achieving ≥70% Improvement in ACR Response Components Through W24

GUS Q4W						
Week	4	8	12	16	20	24
SJC	15.8	31.6†	39.9‡	55.3*	59.4*	60.6*
TJC	6.2	17.3‡	28.2‡	36.1*	41.9*	44.1*
Pt Pain	3.3	12.9	21.1‡	26.4‡	29.7*	30.1†
PtGA	4.4	14.0†	18.5	25.7*	29.3†	31.5*
PGA	5.9‡	17.2‡	30.4*	37.8*	46.3*	48.4*
HAQ-DI	8.1†	12.5†	13.2	18.7‡	21.2*	21.6‡
CRP	12.0‡	20.2*	23.6*	23.8*	22.9†	25.3†

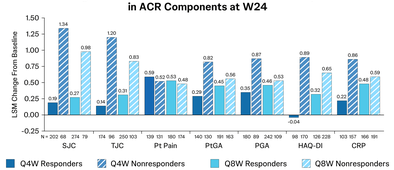
GUS Q8W						
Week	4	8	12	16	20	24
SJC	14.9	32.5*	45.6*	48.8*	59.5*	61.0*
TJC	8.7	17.9*	31.4*	37.3*	43.5*	49.3*
Pt Pain	6.5‡	14.8‡	22.7‡	27.9‡	30.1*	31.5*
PtGA	5.4‡	13.2‡	21.1‡	26.0†	30.6*	31.0*
PGA	4.9‡	18.9†	31.1*	40.1*	43.9*	45.3*
HAQ-DI	5.7	9.2	15.9‡	17.0	20.0*	21.4‡
CRP	13.9†	23.9*	24.6*	28.7*	29.5*	28.4*

PBO						
Week	4	8	12	16	20	24
SJC	10.6	20.9	31.7	35.0	42.0	40.2
TJC	5.9	9.4	18.3	21.5	28.4	27.0
Pt Pain	3.2	10.0	13.8	18.7	18.4	20.3
PtGA	2.4	6.5	13.5	17.1	19.0	18.4
PGA	2.1	10.5	15.9	22.4	24.3	29.3
HAQ-DI	3.2	6.4	10.5	12.3	10.4	13.9
CRP	7.3	10.3	10.3	11.8	14.5	15.7

*Nominal *p=0.001; †p=0.01; ‡p=0.05 vs PBO.*

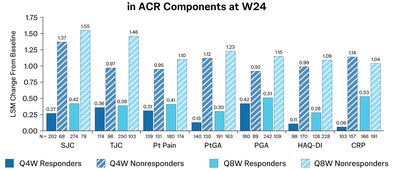
Among GUS-randomized pts, LSM changes in vdH-S score at W24 were smaller for those achieving ≥50% improvement in SJC, TJC, PtGA, PGA, HAQ-DI, and CRP at W24 than in nonresponders

LSM Change From Baseline in vdH-S at W24 by ≥50% Improvement in ACR Components at W24



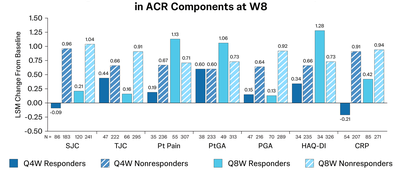
At W48, LSM changes in vdH-S score were smaller for ≥50% responders at W24 across all components vs nonresponders

LSM Change From Baseline in vdH-S at W48 by ≥50% Improvement in ACR Components at W24



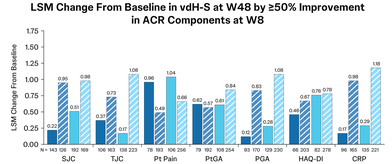
GUS-randomized pts achieving responses at W8 had smaller LSM changes in vdH-S score for SJC, TJC, PGA, and CRP at W48 vs nonresponders

LSM Change From Baseline in vdH-S at W48 by ≥70% Improvement in ACR Components at W8



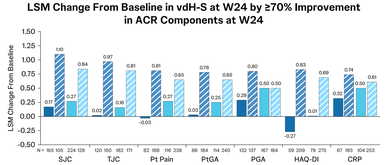
Achieving ≥50% improvement in SJC, TJC, PGA, and CRP at W8 was associated with less structural damage progression at W48

LSM Change From Baseline in vdH-S at W48 by ≥50% Improvement in ACR Components at W8



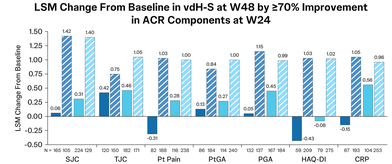
Among GUS-randomized pts, LSM changes in vdH-S score at W24 were smaller for those achieving ≥70% improvement in SJC, TJC, Pt Pain, PtGA, HAQ-DI, and CRP at W24 than in nonresponders

LSM Change From Baseline in vdH-S at W24 by ≥70% Improvement in ACR Components at W24



LSM changes in vdH-S score at W48 were smaller in all components for GUS-randomized pts achieving ≥70% improvement at W24 vs nonresponders

LSM Change From Baseline in vdH-S at W48 by ≥70% Improvement in ACR Components at W24



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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. **3.** Felson DT. *Arthritis Rheumatol.* 1995; 38:727-35. **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** receives consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, Merck, Novartis, Pfizer, and UCB; meeting attendance/travel support from Johnson & Johnson; and research grants from Johnson & Johnson, and Novartis. **LCC** receives grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer and UCB; works as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; and receives speaker fees for AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Johnson & Johnson, Medac, Novartis, Pfizer, and UCB. **PJM** receives 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; grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; and speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **SF, SDC, APK,** and **KL** are employees of Johnson & Johnson and own stock/stock options in Johnson & Johnson. **WH** is an employee of Cytel, Inc. providing statistical support (funded by Johnson & Johnson). **FRW** receives clinical research support from AbbVie, Bristol Myers Squibb, Johnson & Johnson, Novartis, and Pfizer; consulting fees from AbbVie, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, and UCB; and speaking fees from AbbVie, Bristol Myers Squibb, Johnson & Johnson, and UCB. **AK** receives consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB. **CTR** receives speaker fees from AbbVie, Eli Lilly, Johnson & Johnson, Novartis, and UCB; and grant/research support from Johnson & Johnson, Novartis, and UCB.