P72888

APEX: Guselkumab Response and Inhibition of Structural Damage Progression in Active Psoriatic Arthritis by Participant Baseline Characteristics

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Background

Guselkumab (GUS), a fully-human monoclonal antibody able to bind to the CD64-receptor and simultaneously inhibit the IL-23p19 subunit, is approved for moderate-to-severe plaque psoriasis, active psoriatic arthritis (PsA), and moderately-to-severely active Crohn's disease and ulcerative colitis

The ongoing phase 3b, randomized, double-blind, placebo (PBO)-controlled **APEX study (NCT04882098)** is further evaluating GUS effects on clinical and radiographic progression outcomes in **participants (pts) with active and erosive PsA**

APEX met primary (American College of Rheumatology ≥20% improvement [ACR20]) and major secondary (PsA-modified van der Heijde-Sharp [vdH-S] score change from baseline) endpoints, such that **GUS Q4W and Q8W demonstrated significantly higher rates of clinical improvement and significant inhibition of structural damage progression** vs PBO at Week(W)24[1]

Objective

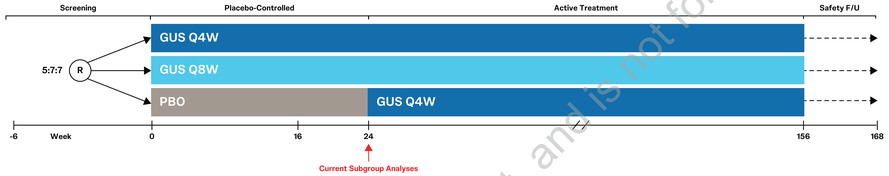
Evaluate consistency in GUS clinical response and radiographic progression inhibition across subgroups of pts of high clinical interest

***Q4W/Q8W**=every 4/8 weeks*

APEX Study Design and Analysis Methods

Inclusion Criteria

- ✓Biologic-naïve adults ≥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 radiographs of hands/feet**
- ✓Active plaque psoriasis



Endpoints

- Proportions achieving ACR20/50[b], PASI 90[c], and MDA responses
- LSM change in PsA-modified vdH-S score

High Clinical Interest Baseline Subgroups[a]

Demographics (Sex, BMI)
PsA Characteristics (Duration[d], SJC, TJC, CRP)
Medication Use (MTX[e])

Statistical Analysis

Pts who discontinued study intervention for any reason except natural disaster/major disruption; initiated/increased dose of csDMARD or corticosteroid; or initiated prohibited therapies prior to W24 were considered nonresponders[f]

Based on an ANCOVA model[g]

[a]29 subgroups were predefined to evaluate treatment consistency over baseline demographics (n=7), disease characteristics (n=12), and medication use (n=5); those of high clinical interest are reported here. [b]Average proportion of pts over 200 MI datasets. [c]Among pts who had ≥3% BSA psoriatic involvement and an IGA score of ≥2 (mild) at baseline. [d]Predefined PsA duration subgroups categories were <1/≥1 to <3/≥3 years, however, <3/≥3 presented due to small sample size in <1 year category. [e]Predefined as csDMARD use at baseline, however, MTX component presented separately based on MTX representing the majority of csDMARD use at baseline. [f]Data impacted by, or missing due to, major disaster/major disruption were imputed using MI (ACR20/50) or were not explicitly imputed (PASI 90/MDA); other missing data were imputed using NRI. [g]Explanatory model variables: baseline vdH-S score, treatment group, and randomization stratification level; data impacted by natural disaster/major disruption and missing data imputed using MI. ***ANCOVA****=analysis of covariance,* ***BMI****=body mass index,* ***BSA****=body surface area.* ***CASPAR****=ClASsification criteria for Psoriatic Arthritis,* ***CRP****=C-reactive protein,* ***csDMARD****=conventional synthetic disease modifying antirheumatic drug,* ***FU****=follow-up,* ***IGA****=Investigator's Global Assessment,* ***LSM****=least squares mean,* ***MDA****=minimal disease activity,* ***MI****=multiple imputation,* ***MTX****=methotrexate,* ***NRI****=nonresponder imputation,* ***NSAID****=nonsteroidal anti-inflammatory drug,* ***PASI 90****=90% improvement in Psoriasis Area and Severity Index,* ***R****=randomization,* ***SJC****=swollen joint count,* ***TJC****=tender joint count.*

Key Takeaways

- **GUS-treated biologic-naïve pts with active and erosive PsA demonstrated significantly greater clinical improvement and significant inhibition of structural damage progression vs PBO at W24**
- **GUS treatment effect was generally consistent across diverse subgroups of pts defined by baseline demographics, disease characteristics, and concomitant MTX use**
  - Benefit in achieving ACR20, ACR50, PASI 90, and MDA was similar regardless of sex, BMI, PsA duration, joint involvement, CRP, and MTX use at baseline
  - Inhibition of structural damage progression was observed across subgroups

Results

Similar proportions of pts comprised baseline characteristic subgroups across treatment arms

- Pts had active and erosive PsA: median disease duration=5 years, SJC=9, TJC=16, and CRP=0.8 mg/dL

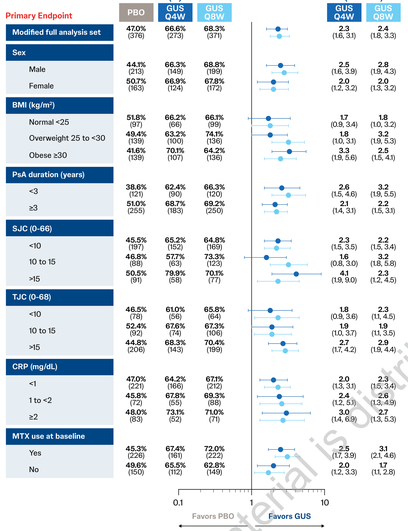
Modified full analysis set[a]	PBO N=376	GUS Q4W N=273	GUS Q8W N=371	Total N=1020
Sex				
Male	57%	55%	54%	55%
Female	43%	45%	46%	45%
BMI, kg/m^2				
Normal <25	26%	24%	27%	26%
Overweight ≥25 to <30	37%	37%	37%	37%
Obese ≥30	37%	39%	37%	37%
PsA disease duration, yrs				
<3	32%	33%	32%	32%
≥3	68%	67%	68%	68%
SJC (0-66)				
<10	52%	56%	46%	51%
10 to 15	23%	23%	33%	27%
>15	24%	21%	21%	22%
TJC (0-68)				
<10	21%	21%	17%	19%
10 to 15	24%	27%	29%	27%
>15	55%	52%	54%	54%
CRP, mg/dL				
<1	59%	61%	57%	59%
1 to <2	19%	20%	24%	21%
≥2	22%	19%	19%	20%
MTX use at baseline				
Yes	60%	59%	60%	60%
No	40%	41%	40%	40%

[a]All randomized pts except those from Ukraine sites rendered unable to support key study operations due to major disruptions (N=1020).

GUS treatment effect on joint disease activity was consistent across subgroups

- Aligned with primary endpoint results, GUS-treated pts had approximately 2- to 4-times higher odds of achieving ACR20 response than PBO-treated pts

ACR20 Response at W24 by Baseline Characteristics

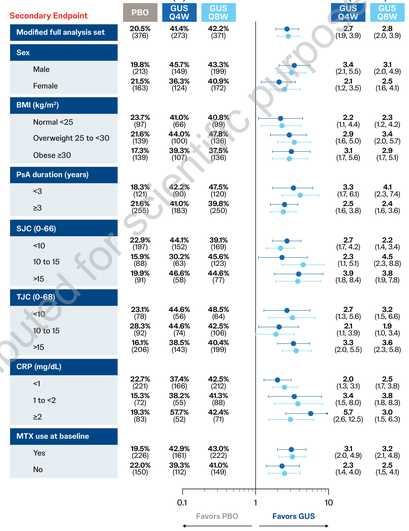


CI=confidence interval, ***OR****=odds ratio.*

GUS effect on the more stringent ACR50 response was also consistent across subgroups

- Aligned with overall ACR50 results, GUS-treated pts had approximately 2- to 6-times higher odds of achieving ACR50 response than PBO-treated pts

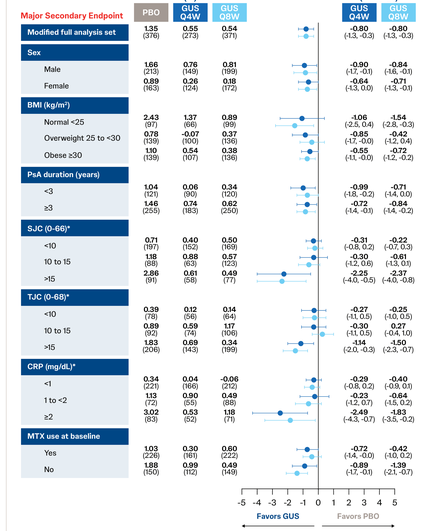
ACR50 Response at W24 by Baseline Characteristics



Significant inhibition of structural damage progression with GUS was generally consistent across baseline pt subgroups

- Concordant with known risk factors, PBO-treated pts with SJC >15 & CRP ≥2 mg/dL exhibited notably higher degrees of structural damage progression, leading to even more robust GUS effects in these groups

Modified vdH-S Score at W24 by Baseline Characteristics

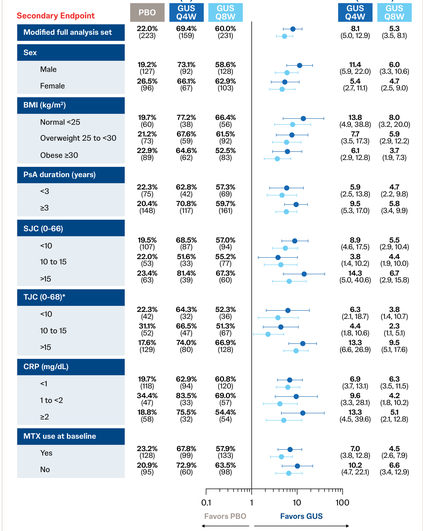


**Interaction p-value <0.05 for SJC GUS Q4W and Q8W; TJC GUS Q8W; CRP GUS Q4W.*

Treatment effect of GUS on achieving PASI 90 response was generally consistent across subgroups

- Comparable to overall PASI 90 results, GUS-treated pts had approximately 2- to 14-times higher odds of achieving PASI 90 response than PBO-treated pts

PASI 90 Response at W24 by Baseline Characteristics[a]

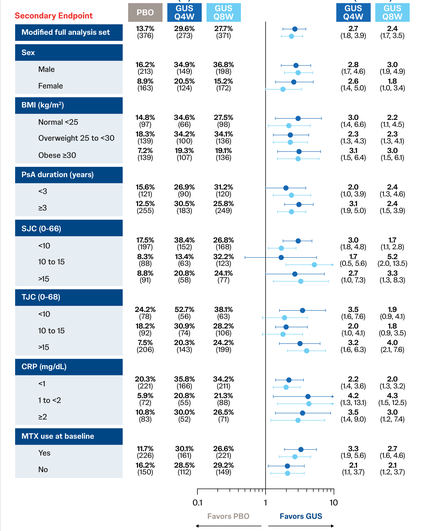


[a]Among pts who had ≥3% BSA psoriatic involvement and an IGA score of ≥2 (mild) at baseline. *Interaction p-value =0.05 for TJC GUS Q8W.*

GUS effect on achievement of the MDA composite endpoint remained consistent across subgroups

- Aligned with overall MDA results, GUS-treated pts had approximately 2- to 5-times higher odds of achieving MDA than PBO-treated pts

MDA Response at W24 by Baseline Characteristics



PRESENTED BY: April W Armstrong at American Academy of Dermatology (AAD) Annual Meeting; March 27–31, 2026; Denver, CO, USA. **REFERENCES: 1.** Mease PJ, et al. *Ann Rheum Dis.* 2025;84:1983–94. **ACKNOWLEDGMENTS:** Medical writing support was provided by Alyssa Cortina, PharmD, of Johnson & Johnson, under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298–1304). This presentation was sponsored by Johnson & Johnson. **DISCLOSURES: AWA:** has served as a speaker, consultant, and/or investigator for AbbVie, Amgen, Arcutis, Bristol Myers Squibb, Dermavant Sciences, Eli Lilly and Company, Galderma, Incyte, Johnson & Johnson, Leo, Novartis, Pfizer, Regeneron, Sanofi, Takeda, and UCB. **LCC:** has received grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB; has received consulting fees from 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. **PJM:** has received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Immagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Sopre, SUN Pharma, Takeda, and UCB; has received grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sanofi, Takeda, and UCB; and has received speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **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. **CTR:** has received grant/research support from AbbVie, Amgen, and UCB; and has received consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, and UCB. **SDC, APK, KL,** and **BZ:** are employees of Johnson & Johnson and may own stock/stock options in Johnson & Johnson. **YJ:** is an employee of Cytel providing statistical support (funded by Johnson & Johnson).