Guselkumab Response and Inhibition of Structural Damage Progression in Active Psoriatic Arthritis Across APEX Participant Subgroups

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Background

- **Guselkumab (GUS)**, a fully-human monoclonal antibody able to bind to the CD64-receptor and simultaneously inhibit the interleukin (IL)-23p19 subunit, is indicated for moderate-to-severe plaque psoriasis, active psoriatic arthritis (PsA), and moderately-to-severely active Crohn's disease/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 every 4 weeks (Q4W) and every 8 weeks (Q8W) demonstrated significantly higher rates of clinical improvement and significant inhibition of structural damage progression** vs PBO at Week(W)24

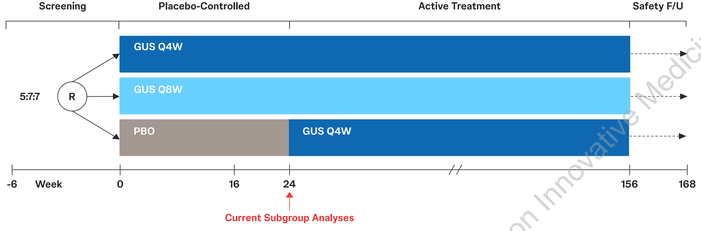
Objectives

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

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[a]	High Clinical Interest Baseline Subgroups[b]	Statistical Analysis
• Proportion achieving ACR20/50 response	**Demographics** (Sex, BMI) **PsA Characteristics** (Duration[c], SJC, TJC, CRP) **Medication Use** (MTX[d])	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[e]
• LSM change in PsA-modified vdH-S score		Based on an ANCOVA model[f]
• LSM change in PsA-modified vdH-S score	**Radiographic Features** (Total vdH-S, erosion, and JSN scores; joints with erosions/JSN)	Based on an ANCOVA model[f]

*Over 200 MI datasets. †29 subgroups were predefined to evaluate treatment consistency over baseline demographics (n=7), disease characteristics (n=12), medication use (n=5), and radiographic features (n=5); those of high clinical interest are reported here. ‡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. §Predefined as csDMARD use at baseline, however, MTX component presented separately based on MTX representing the majority of csDMARD use at baseline. ¶Data impacted by, or missing due to, natural disaster/major disruption were imputed using MI; other missing data were imputed using NRI. [a]Exploratory 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. **ACR**=American College of Rheumatology; **ANCOVA**=analysis of covariance; **BMI**=body mass index; **CASPAR**=CASsification criteria for Psoriatic ARthritis; **CRP**=C-reactive protein; **csDMARD**=conventional synthetic disease modifying antirheumatic drug; **FU**=follow-up; **GUS**=guselkumab; **JSN**=joint space narrowing; **LSM**=least squares mean; **MI**=multiple imputation; **MTX**=methotrexate; **NRI**=nonresponder imputation; **NSAID**=nonsteroidal anti-inflammatory drug; **PBO**=placebo; **PsA**=psoriatic arthritis; **pts**=participants; **Q4W**=every 4 weeks, **Q8W**=every 8 weeks; **R**=randomization; **SJC**=swollen joint count; **TJC**=tender joint count; **vdH-S**=van der Heijde-Sharp; **W**=week.

Results

Similar proportions of pts comprised baseline characteristic subgroups across treatment arms

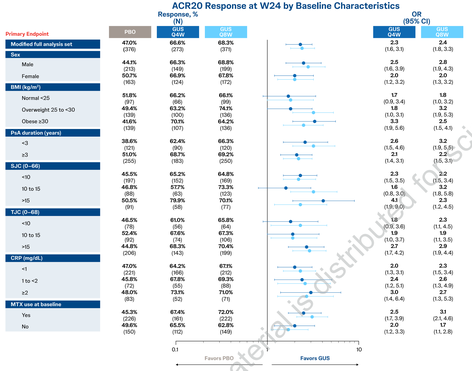
- Pts with active and erosive PsA: median disease duration=5 years, swollen joint count (SJC)=9, tender joint count (TJC)=16, and C-reactive protein (CRP)=0.8 mg/dL

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/m²				
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%

*All randomized pts except those from Ukraine sites rendered unable to support key study operations due to major disruptions (N=1020). **BMI**=body mass index; **CRP**=C-reactive protein; **GUS**=guselkumab; **MTX**=methotrexate; **PBO**=placebo; **PsA**=psoriatic arthritis; **pts**=participants; **Q4W**=every 4 weeks; **Q8W**=every 8 weeks; **SJC**=swollen joint count; **TJC**=tender joint count; **yrs**=years.

GUS treatment effect on joint disease activity was consistent across subgroups

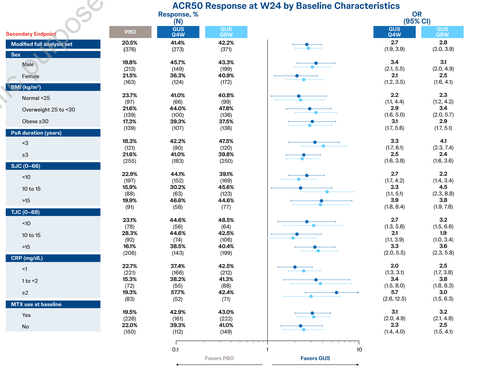
- Aligned with primary endpoint results, GUS-treated pts had approximately 2- to 4-times higher odds of achieving ACR20 response than PBO-treated pts



ACR=American College of Rheumatology; *BMI*=body mass index; *CI*=confidence interval; *CRP*=C-reactive protein; *GUS*=guselkumab; *MTX*=methotrexate; *OR*=odds ratio; *PBO*=placebo; *PsA*=psoriatic arthritis; *Q4W*=every 4 weeks; *Q8W*=every 8 weeks; *SJC*=swollen joint count; *TJC*=tender joint count; *W*=week.

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

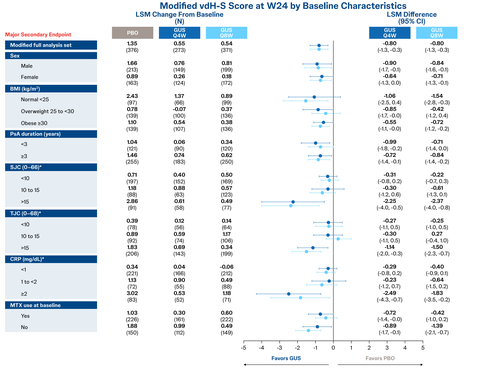
- Aligned with overall ACR50 results, GUS-treated pts had approximately 2- to 6-times higher odds of achieving ACR50 response than PBO-treated pts



ACR=American College of Rheumatology; *BMI*=body mass index; *CI*=confidence interval; *CRP*=C-reactive protein; *GUS*=guselkumab; *MTX*=methotrexate; *OR*=odds ratio; *PBO*=placebo; *PsA*=psoriatic arthritis; *Q4W*=every 4 weeks; *Q8W*=every 8 weeks; *SJC*=swollen joint count; *TJC*=tender joint count; *W*=week.

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

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



*Interaction p-value =0.05 for SJC GUS Q4W and Q8W, TJC GUS Q8W; CRP GUS Q4W, BMI=body mass index, CI=confidence interval, CRP=C-reactive protein, GUS=guselkumab, LSM=least squares mean, MTX=methotrexate, PBO=placebo, PsA=psoriatic arthritis, Q4W=every 4 weeks, Q8W=every 8 weeks, SJC=swollen joint count, TJC=tender joint count, vdH-S=van der Heijde-Sharp, W=week.

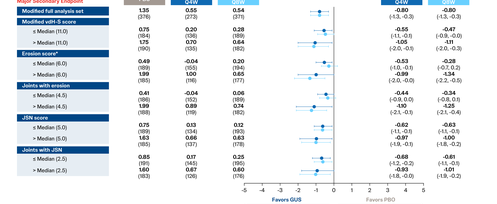
Baseline radiographic joint damage was of moderate degree and similar across treatment groups

	PBO (N=374)	GUS Q4W (N=271)	GUS Q8W (N=371)	Total (N=1016)
Baseline Radiographic Features				
Total vdH-S score [0–528]	11.5 [5.0–27.5]	11.0 [4.5–26.5]	11.0 [4.5–25.0]	11.0 [5.0–26.5]
Erosion score [0–320]	6.0 [2.5–13.0]	5.5 [2.5–13.0]	6.0 [2.5–14.0]	6.0 [2.5–13.5]
JSN score [0–208]	5.0 [1.5–14.0]	5.5 [1.5–14.9]	5.0 [1.0–14.5]	5.0 [1.5–14.5]

*Values are median [interquartile range]. **GUS**=guselkumab, **JSN**=joint space narrowing, **PBO**=placebo, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **vdH-S**=van der Heijde-Sharp.

Inhibition of structural damage progression with GUS was largely consistent regardless of baseline radiographic features

- PBO-treated pts with a vdH-S erosion score >6 and >4.5 erosive joints at baseline had the most radiographic progression at W24



*Interaction p-value =0.05 for erosion score GUS Q8W; CI=confidence interval; GUS=guselkumab; JSN=joint space narrowing; LSM=least squares mean; PBO=placebo; Q4W=every 4 weeks; Q8W=every 8 weeks; vdH-S=van der Heijde-Sharp; W=week.

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 effects were generally consistent across diverse subgroups of pts defined by baseline demographics, disease characteristics, medication use, and radiographic features of interest**
  - Benefit in ACR20/50 clinical improvement was similar regardless of sex, BMI, PsA duration, joint involvement, CRP, and MTX use at baseline
  - Inhibition of radiographic progression observed across clinical and radiographic feature subgroups

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