

SOLSTICE: Efficacy of Guselkumab in Participants With Active Psoriatic Arthritis and Inadequate Response and/or Intolerance to One Prior Tumor Necrosis Factor Inhibitor Across Baseline Subgroups

Joseph F. Merola,¹ Philip J. Mease,^{2,3} Alexis Ogdie,⁴ Christopher T. Ritchlin,⁵ Jose U. Scher,⁶ Oyediran Adelakun,⁷ Evan Leibowitz,⁷ Yanli Wang,⁸ Yevgeniy Krol,⁷ Alice B. Gottlieb,⁹

¹Department of Dermatology and Department of Medicine, Division of Rheumatology, UT Southwestern Medical Center and O'Donnell School of Public Health, Dallas, TX, USA; ²Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; ³University of Washington School of Medicine, Seattle, WA, USA; ⁴Division of Rheumatology, University of Pennsylvania School of Medicine, Philadelphia, PA, USA; ⁵Department of Medicine, Allergy/Immunology, and Rheumatology, University of Rochester Medical Center, Rochester, NY, USA; ⁶New York University Grossman School of Medicine, New York, NY, USA; ⁷Johnson & Johnson, Horsham, PA, USA; ⁸Johnson & Johnson, Spring House, PA, USA; ⁹Department of Dermatology, UT Southwestern Medical Center, Dallas, TX, USA

Background

Guselkumab (GUS), a fully human, dual-acting,¹ monoclonal antibody that selectively inhibits the interleukin (IL)-23p19 subunit, is approved to treat moderate-to-severe plaque psoriasis (PsO), active psoriatic arthritis (PsA), and moderately to severely active Crohn's disease and ulcerative colitis²

SOLSTICE is an ongoing Phase 3b, multicenter, randomized, double-blinded placebo (PBO)-controlled study evaluating the efficacy and safety of GUS in participants with active PsA who had an inadequate response (IR; inadequate efficacy and/or intolerance) to one prior tumor necrosis factor inhibitor (TNFi)

At Week (W)24, GUS demonstrated significant improvements across PsA signs and symptoms, including joint and skin outcomes, compared with placebo³

Objective

This analysis assessed the consistency of GUS clinical responses at W24 across SOLSTICE participant subgroups

Methods

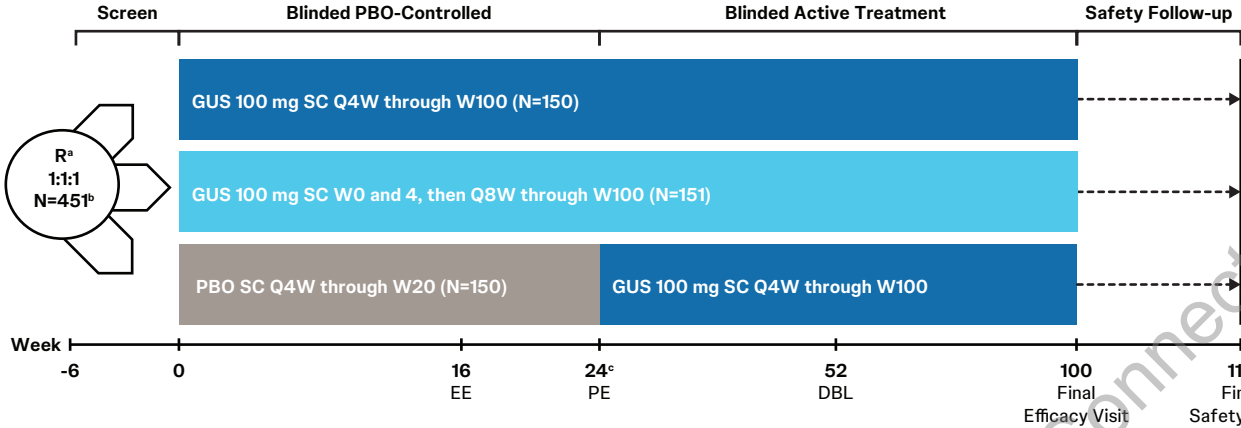
Inclusion Criteria

✓ Age ≥18 years

✓ Active PsA (≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL); CASPAR criteria met

✓ Inadequate response and/or intolerance to 1 prior TNFi therapy

✓ Active (≥1 PsO plaque ≥2 cm and/or nail PsO) or history of PsO



Baseline Subgroups

Demographics (sex and BMI)
Disease characteristics (PsA duration, SJC, TJC, PASI)
Medication history (concomitant csDMARD use, reason for d/c of prior TNFi)

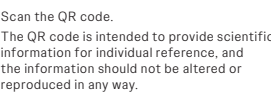
Clinical Responses

Primary Endpoint: ACR20
Select major secondary endpoints: ACR50, MDA, IGA 0/1*

Statistical Analysis:

• Participants were considered nonresponders if they discontinued study agent for any reason other than ND/MD, initiated or increased the dose from baseline of csDMARDs or oral corticosteroids, or initiated protocol-prohibited PsA therapies through W24

• Missing data due to ND/MD were imputed using MI. Other missing data imputed using NRI. Response rates shown are the average proportion of participants achieving response, over the 200 MI datasets.



Scan the QR code.
The QR code is intended to provide scientific information for individual reference, and the information should not be altered or reproduced in any way.

Results

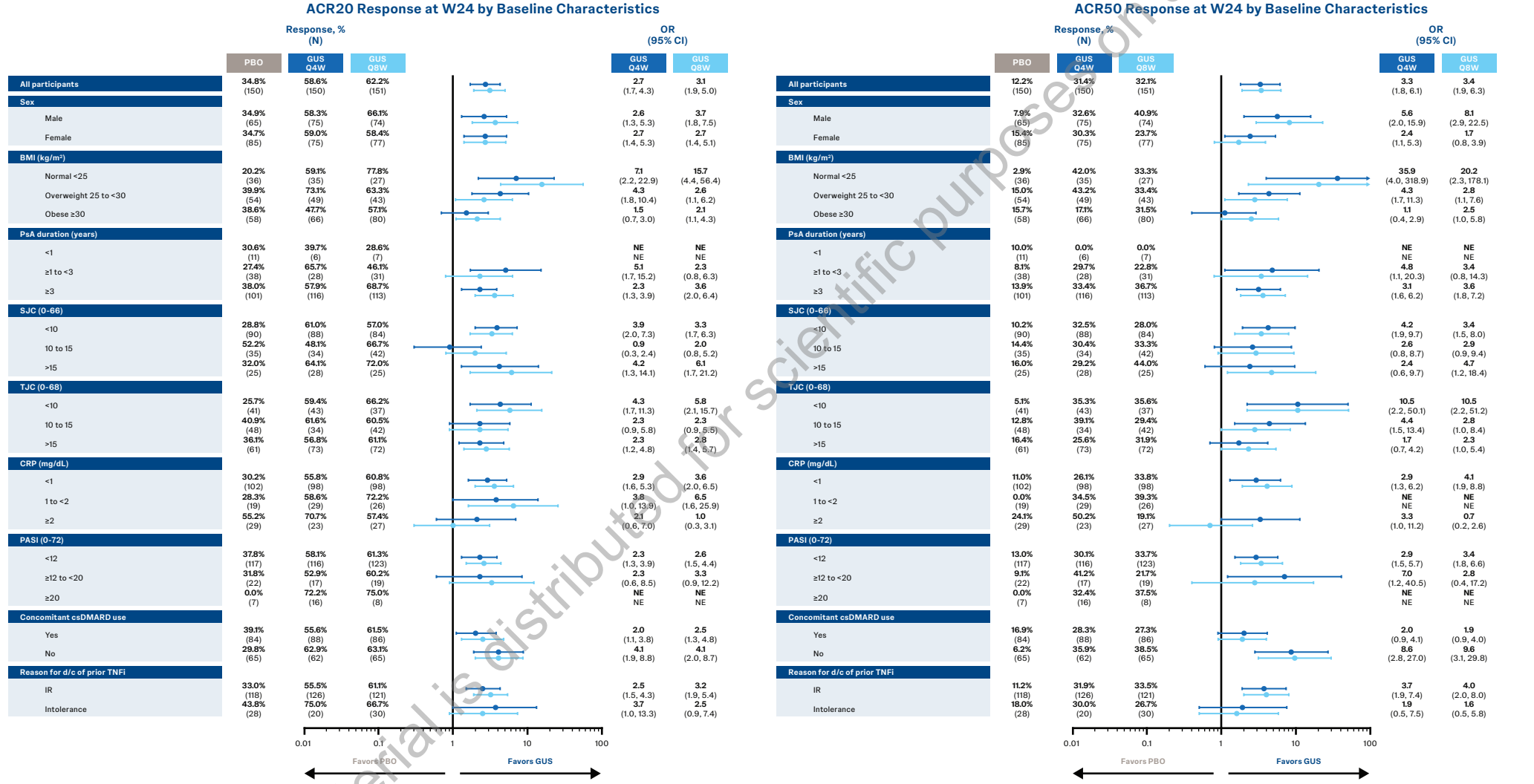
Overall baseline demographics and disease characteristics were well balanced across treatment groups

- Mean baseline disease assessments were consistent with moderately-to-severely active PsA

| | PBO (N=150) | GUS 100 mg Q4W (N=150) | GUS 100 mg Q8W (N=151) |
|---|----------------|------------------------------|------------------------------|
| Demographics | | | |
| Age, yrs | | | |
| Mean | 49.2 | 50.6 | 51.9 |
| Sex | | | |
| Male, % | 43 | 50 | 49 |
| Female, % | 57 | 50 | 51 |
| BMI, kg/m² | | | |
| Mean | 30.0 | 30.0 | 30.9 |
| Disease Characteristics | | | |
| PsA Disease Duration, yrs | | | |
| Mean | 7.0 | 8.8 | 8.3 |
| SJC (0-66) | | | |
| Mean | 10.2 | 10.7 | 10.3 |
| TJC (0-68) | | | |
| Mean | 16.8 | 18.1 | 17.1 |
| CRP, mg/dL | | | |
| Mean | 14 | 1.2 | 1.3 |
| PASI (0-72), N | | | |
| Mean | 146 | 149 | 150 |
| Participants With a BSA ≥3% and IGA ≥2 | | | |
| Mean | 6.0 | 7.3 | 6.7 |
| Participants, N (%) | | | |
| PsO Disease Duration, yrs | | | |
| Mean | 16.5 | 18.7 | 17.3 |
| BSA, % | | | |
| Mean | 13.9 | 17.0 | 15.7 |
| PASI (0-72) | | | |
| Mean | 9.0 | 10.4 | 10.1 |
| IGA score | | | |
| Mild (2), % | 45 | 48 | 44 |
| Moderate (3), % | 49 | 40 | 51 |
| Severe (4), % | 6 | 12 | 6 |
| Medication History | | | |
| Concomitant csDMARD use at baseline | | | |
| Yes, % | 56 | 59 | 57 |
| Reason for d/c of prior anti-TNF | | | |
| Inadequate Response, % | 79 | 84 | 80 |
| Intolerance, % | 19 | 13 | 20 |

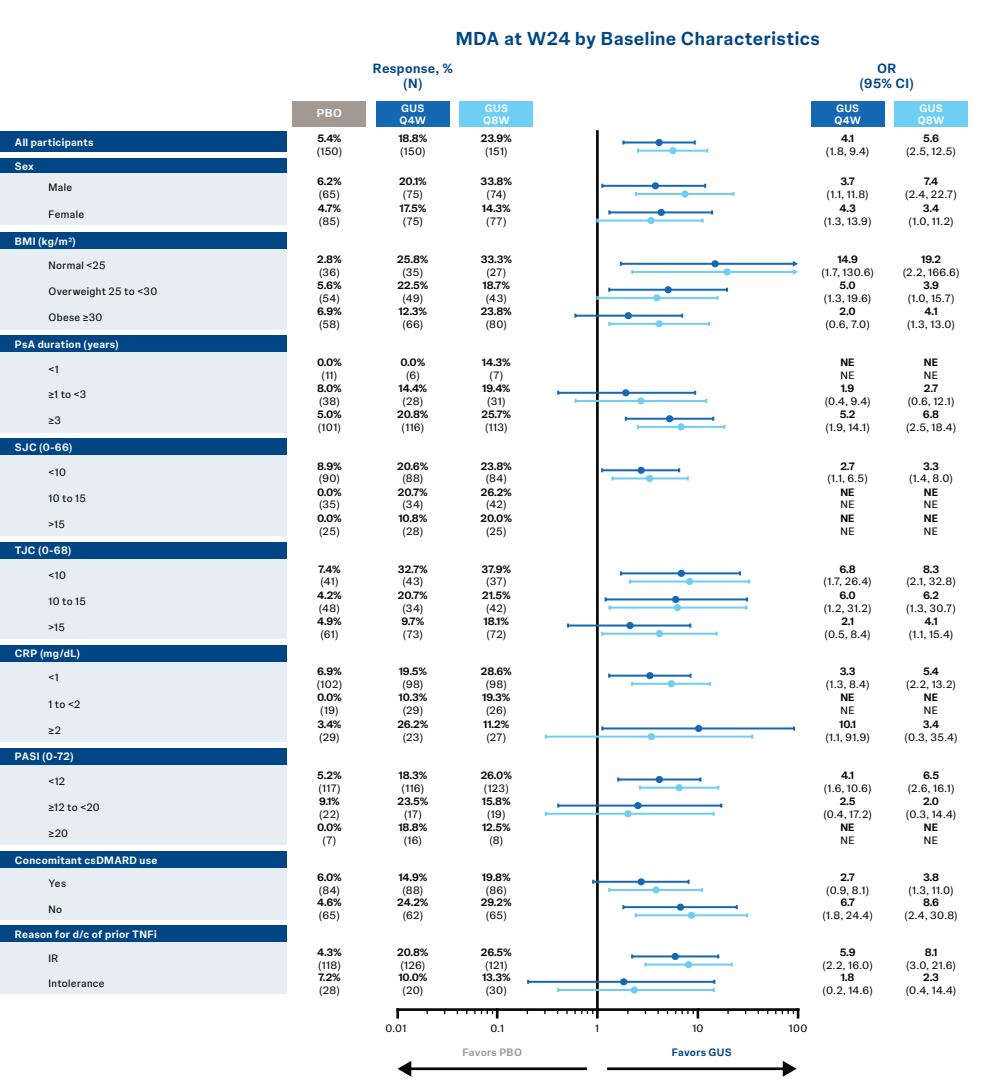
GUS treatment effect on ACR20/50 responses was generally consistent across subgroups regardless of dosing regimen

- There may have been a slight trend for potentially lower ACR20/50 response rates in participants with BMI ≥30 kg/m²; however, these results may be confounded by the small sample sizes and relatively high placebo response in this subgroup

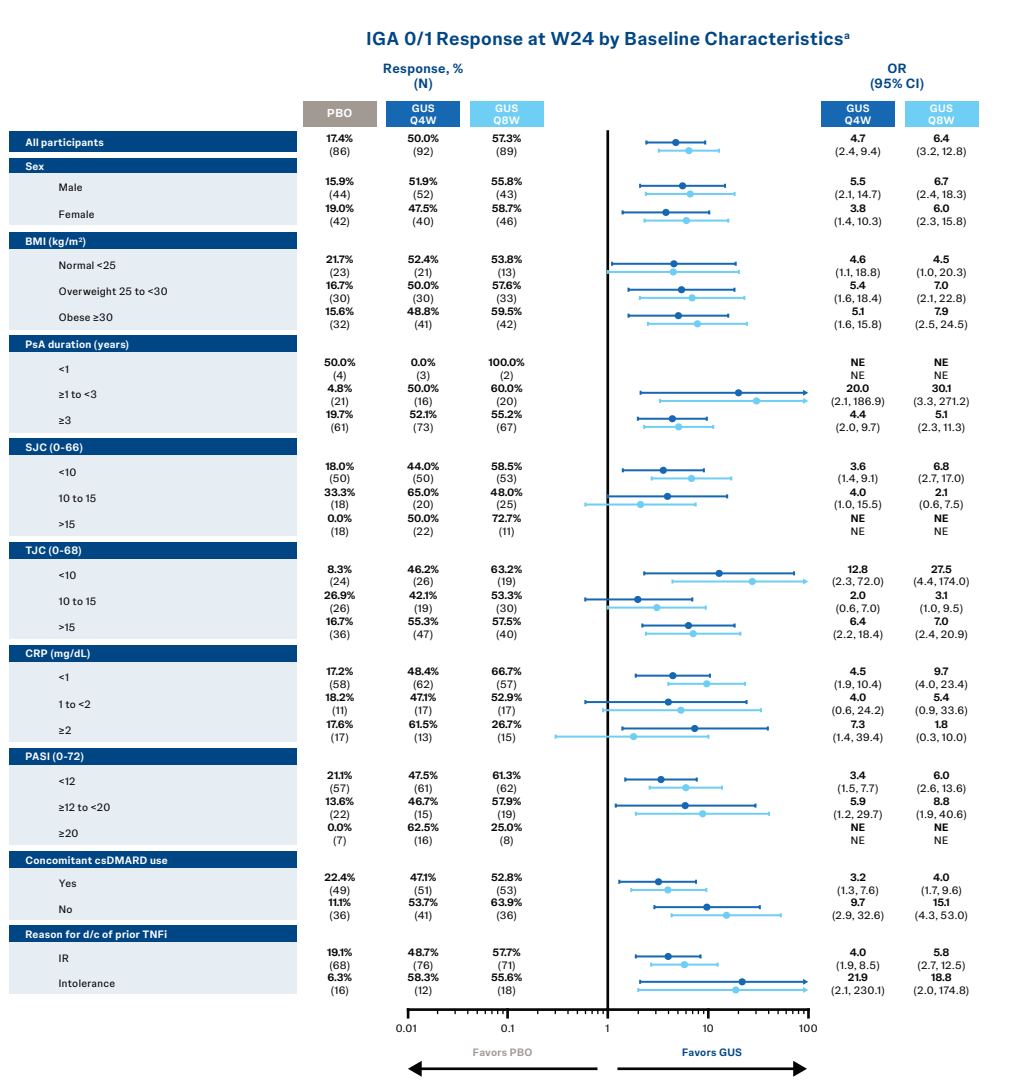


CI=confidence interval; OR=odds ratio; NE=not evaluable

GUS effect on achievement of MDA remained consistent across subgroups for both dosing regimens



Treatment effect of GUS on achieving clear or almost clear skin was also consistent across subgroups with both Q4W and Q8W dosing



*IGA 0/1 response was evaluated among participants who achieved a ≥2 grade reduction from baseline and who had a baseline BSA of PsO ≥3% and an IGA ≥2