

Efficacy and Safety of Guselkumab in Participants With Active Psoriatic Arthritis and Inadequate Response/Intolerance to One Prior Tumor Necrosis Factor Inhibitor Through 1 Year of the SOLSTICE Study

Alice B. Gottlieb,^{1*} Joseph F. Merola,² Philip J. Mease,^{3,4} Christopher T. Ritchlin,⁵ Jose U. Scher,⁶ Kimberly P. Lafferty,⁷ Soumya D. Chakravarty,^{7,8} Yanli Wang,⁹ Yevgeniy Krol,⁷ Christopher Russo,⁷ Stacey Fitch,⁷ Alexis Ogdie¹⁰

¹UT Southwestern Medical Center, Department of Dermatology, Dallas, TX, USA; ²Department of Dermatology and Department of Medicine, Division of Rheumatology, UT Southwestern Medical Center and O'Donnell School of Public Health, Dallas, TX, USA; ³Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; ⁴University of Washington School of Medicine, Seattle, WA, 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; ⁸Drexel University College of Medicine, Philadelphia, PA, USA; ⁹Johnson & Johnson, Spring House, PA, USA; ¹⁰University of Pennsylvania School of Medicine, Philadelphia, PA, USA.

*Presenting author

Background

- Guselkumab (GUS) is a fully human dual-acting¹ monoclonal antibody inhibiting 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 colitis²
- PsA is a chronic, heterogeneous, inflammatory disease primarily affecting the joints and skin^{3,4}
- In SOLSTICE, a phase 3b, multicenter, randomized, placebo (PBO)-controlled study, GUS 100 mg every 4 weeks (Q4W) and every 8 weeks (Q8W) significantly improved PsA signs and symptoms through week (W) 24 in participants (pts) with inadequate response (IR; inadequate efficacy or intolerance) to 1 prior tumor necrosis factor inhibitor (TNFi)⁵

Objective

Report efficacy and safety findings for GUS Q4W and Q8W through W52 of SOLSTICE in a dedicated TNFi-IR pt population with active PsA

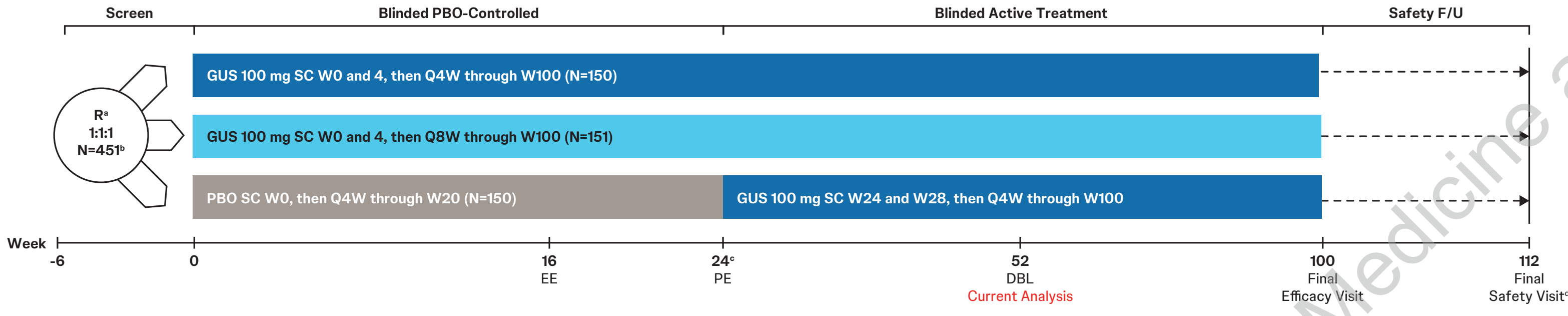
Methods

Key inclusion criteria:

- Age ≥18 years
- Active PsA (≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL); CASPAR criteria met
- Inadequate efficacy and/or intolerance to 1 prior TNFi therapy
- History of or active PsO (≥1 plaque ≥2 cm and/or nail PsO)

Endpoints at W52:

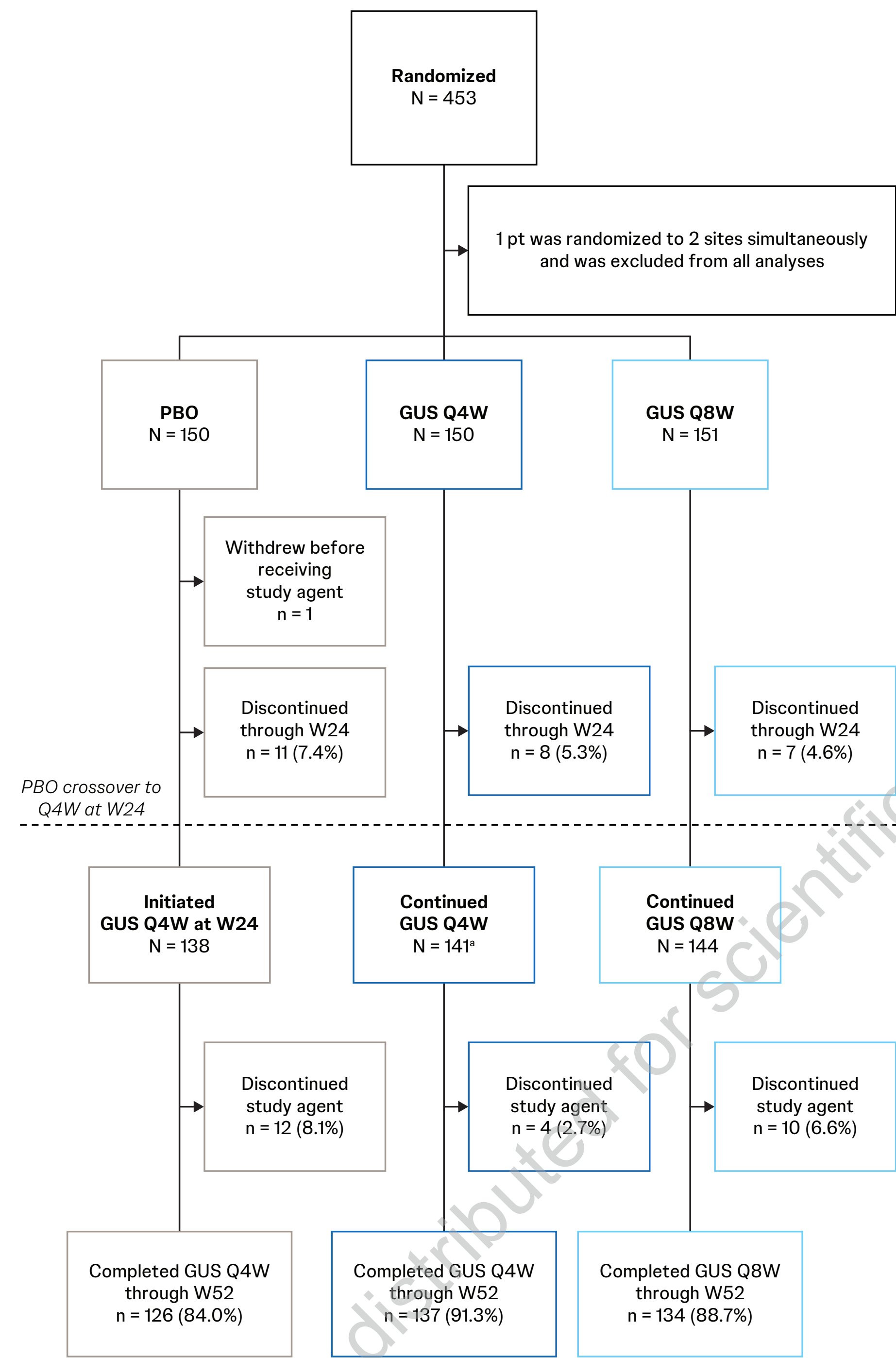
- ACR20
- ACR50
- ACR70
- IGA 0/1 response: score 0 or 1 plus ≥2-grade improvement
- PASI 90
- MDA



*Randomization was stratified by baseline use of csDMARDs. Total number randomized=453, the full analysis set of 451 excludes 1 pt who was double randomized. Crossover: Final safety F/U at W112 is 12W after final study agent administration. ACR20=≥20% in American College of Rheumatology response criteria, CASPAR=CASPAR criteria for Psoriatic Arthritis, CRP=C-reactive protein, csDMARDs=conventional synthetic disease-modifying antirheumatic drugs, DBL=database lock, EE=early escape, F/U=follow-up, GUS=guselkumab, IGA=Investigator's Global Assessment of PsO, MD=major disruption involving Ukraine and neighboring countries/territories beginning 24 February 2022, MDA=Minimal Disease Activity, MI=Multiple imputation, ND=natural disaster, site closure, site access restrictions, or lockdowns due to the COVID-19 pandemic, NRI=nonresponder imputation, PASI90=≥90% improvement in Psoriasis Area and Severity, PBO=placebo, PE=primary endpoint, PsA=psoriatic arthritis, PsO=psoriasis, Pts=participants, Q4W=every 4 weeks, Q8W=every 8 weeks, R=randomization, SC=subcutaneous, SJC=Swollen joint count, TJC=Tender joint count, TNFi=tumor necrosis factor inhibitor, W=week.

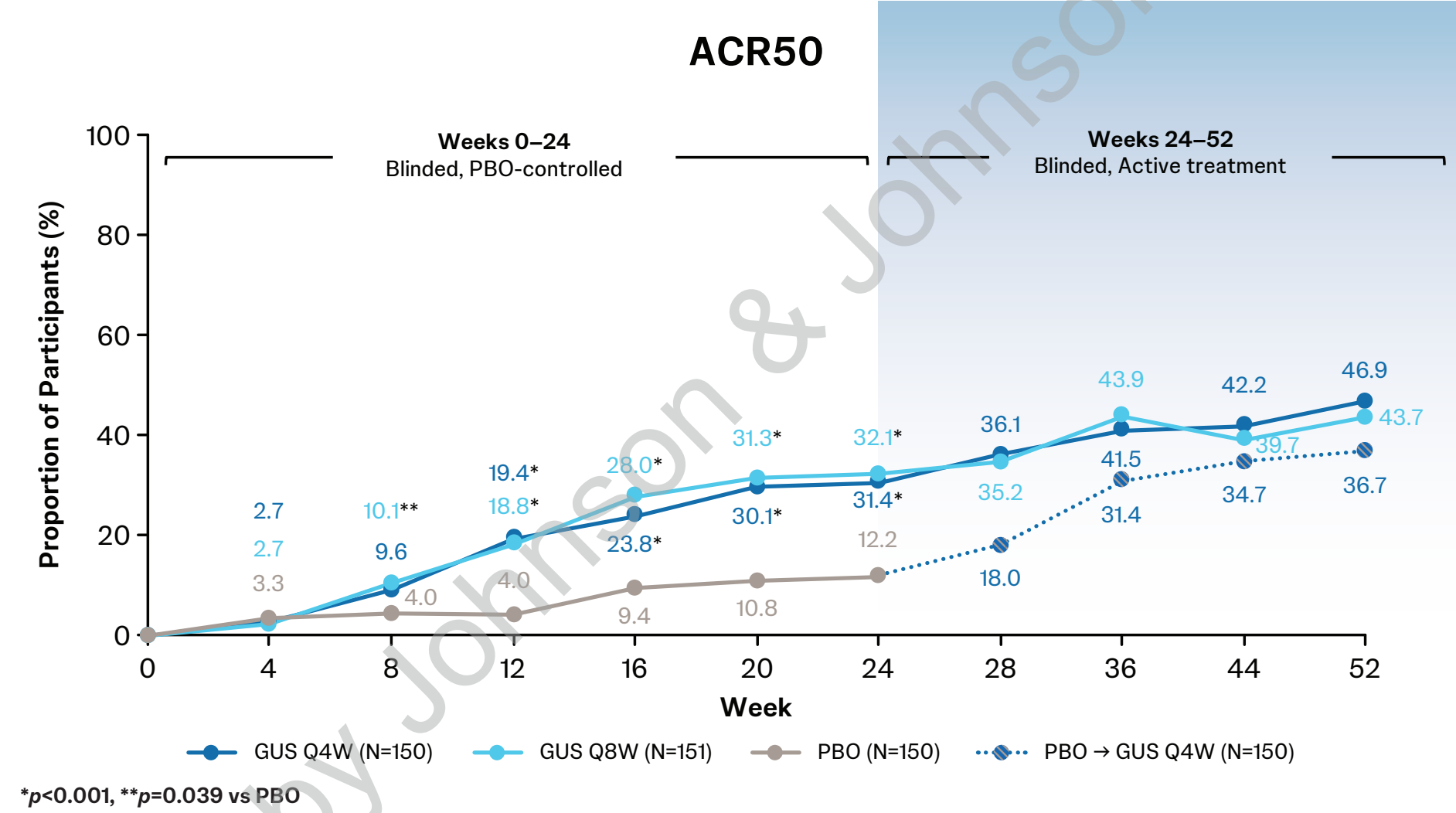
Results

Of 451 analyzed pts, 88.0% completed treatment through W52



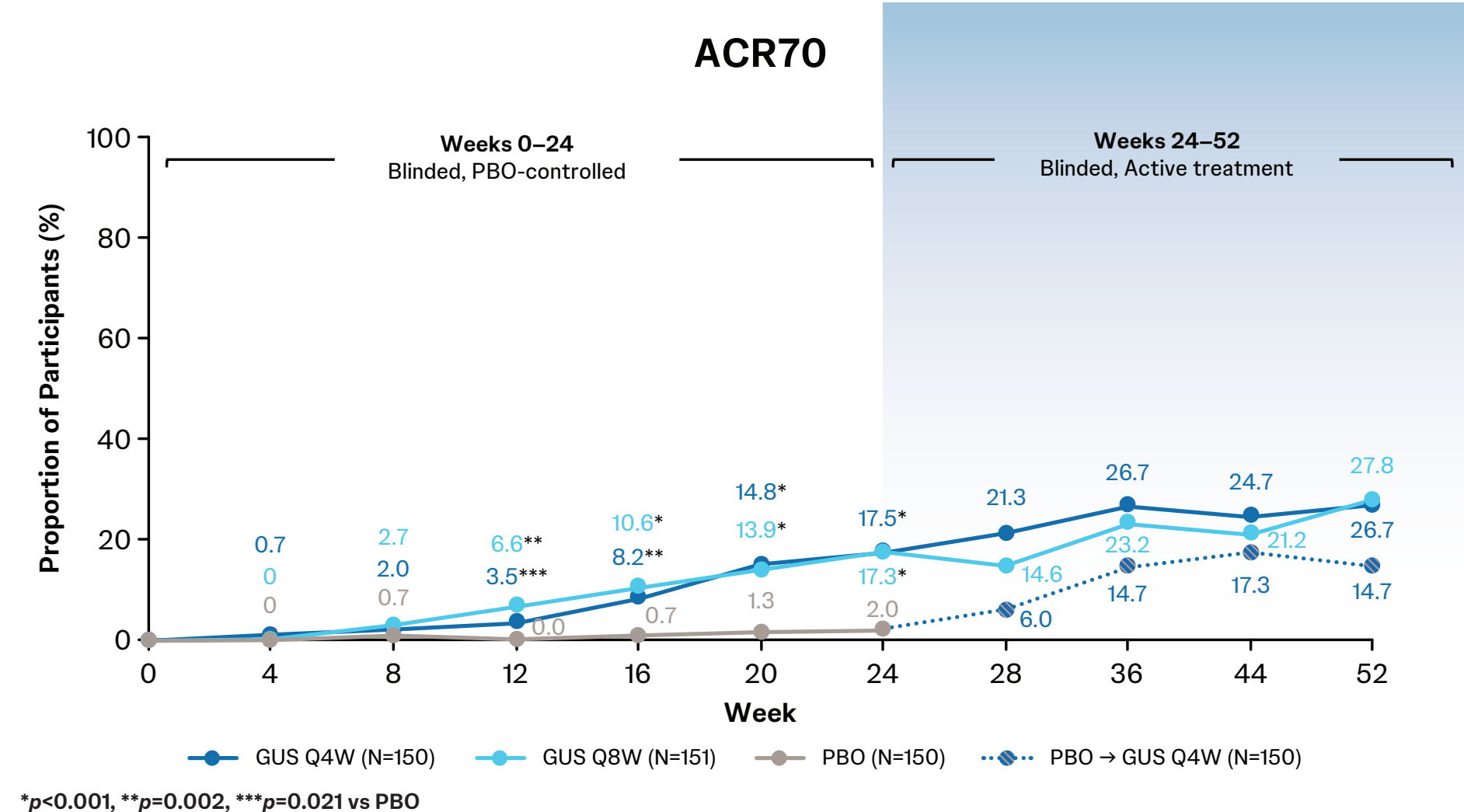
*One additional pt discontinued study agent after receiving their W24 dose. GUS=guselkumab, PBO=placebo, pts=participants, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

ACR50 response rates increased numerically from W24–W52 in GUS-randomized pts



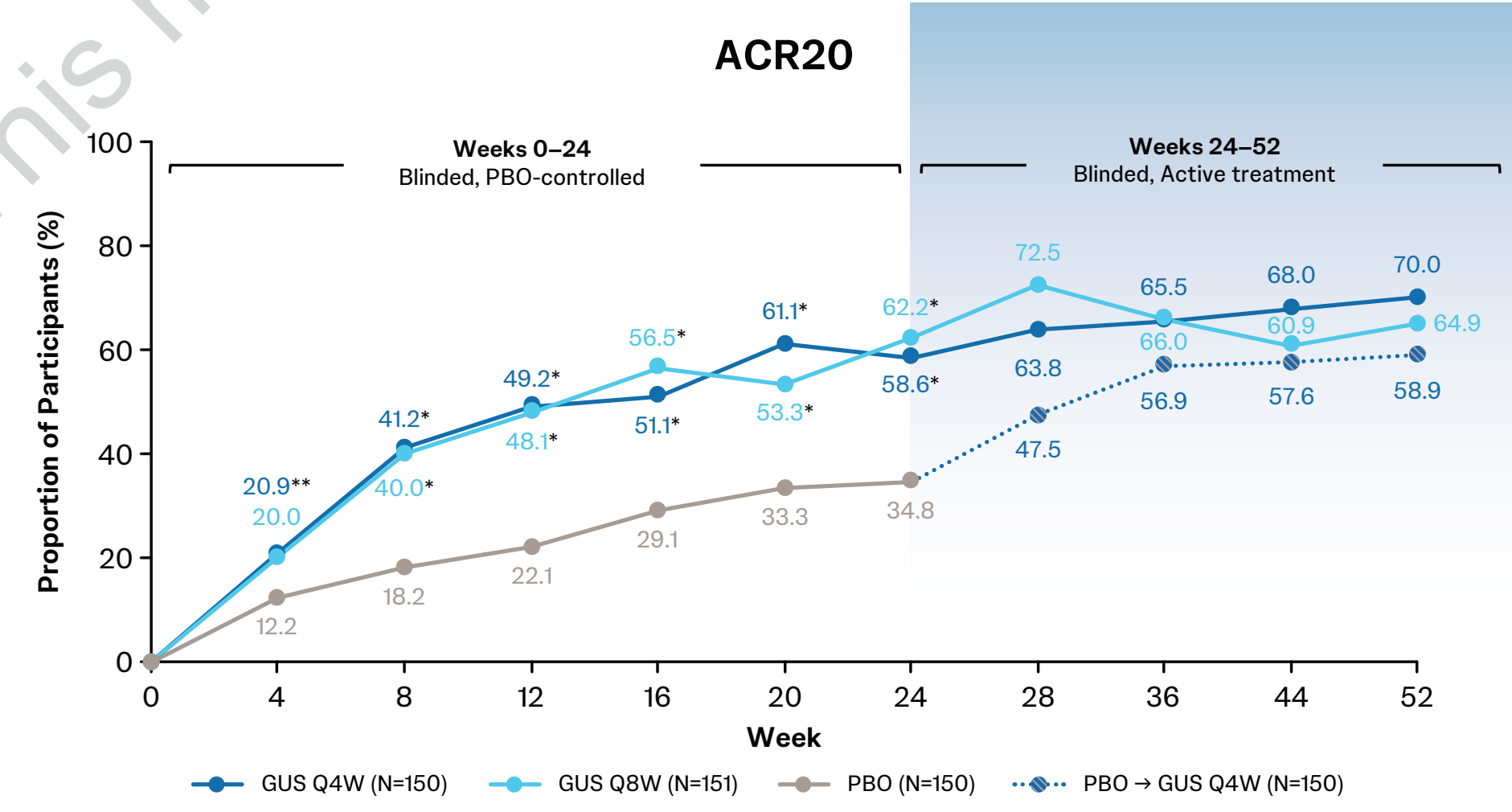
ACR50 was weekly controlled at W16 and W24; all other p values are nominal. Post hoc analysis (W4, W8, W12, W20, and W28–W52) used methods consistent with primary and major secondary endpoint analysis. ACR=American College of Rheumatology, GUS=guselkumab, PBO=placebo, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

ACR70 response rates increased numerically from W24–W52 in GUS-randomized pts



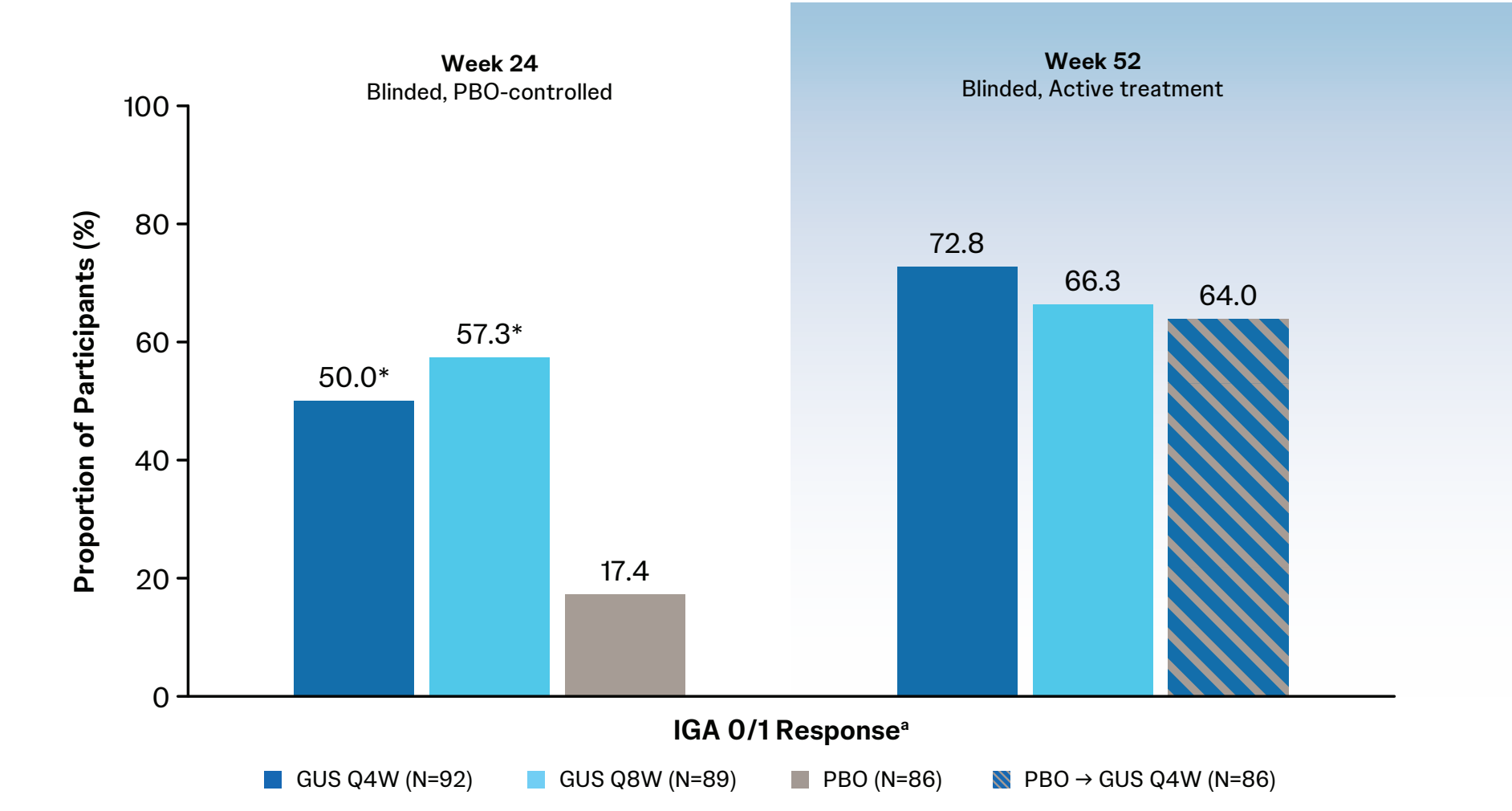
ACR70 was weekly controlled at W24; all other p values are nominal. Post hoc analysis (W4, W8, W12, W20, and W28–W52) used methods consistent with primary and major secondary endpoint analysis. ACR=American College of Rheumatology, GUS=guselkumab, PBO=placebo, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

ACR20 response rates increased numerically from W24–W52 in GUS-randomized pts



ACR20 was weekly controlled at W16 and multiplicity-controlled at W24; all other p values are nominal. Post hoc analysis (W4, W8, W12, W20, and W28–W52) used methods consistent with primary and major secondary endpoint analysis. ACR=American College of Rheumatology, GUS=guselkumab, PBO=placebo, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

Response rates for achieving almost clear or clear skin numerically increased from W24–W52 in GUS-randomized pts



*Among pts with baseline BSA ≥3% and IGA ≥2 (mild). Post hoc analysis used methods consistent with primary and major secondary endpoint analysis. BSA=body surface area, GUS=guselkumab, IGA=Investigator's Global Assessment of PsO, PBO=placebo, pts=participants, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.



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.

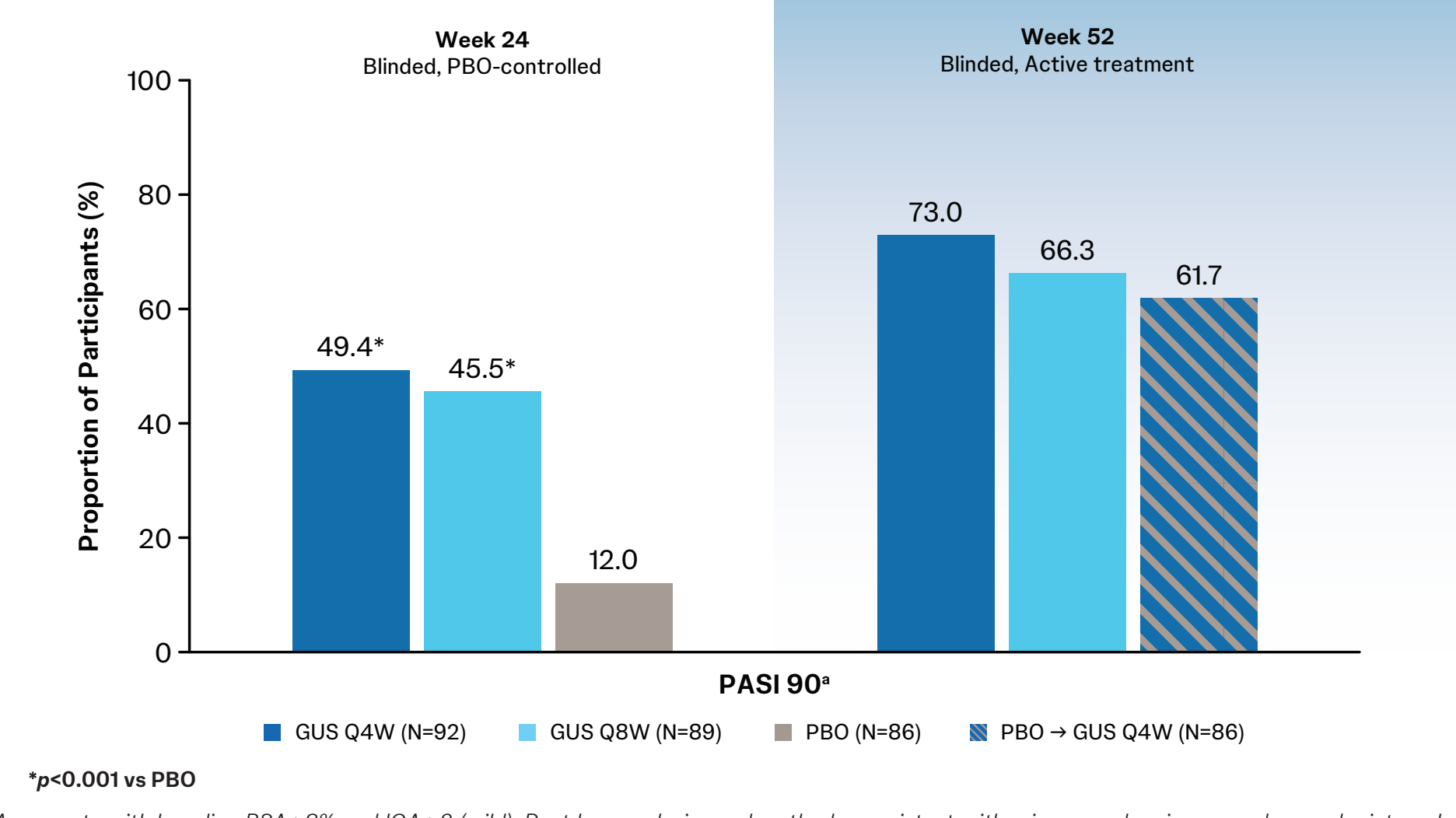
Key Takeaways

- In the SOLSTICE TNFi-IR PsA population, response rate for achieving improvements in joint (ACR20/50/70) and skin (IGA 0/1; PASI 90) outcomes were sustained or numerically increased from W24–W52 among GUS-randomized pts
- MDA response rates numerically increased from W24–W52 in GUS-randomized pts
- Similar efficacy observed for both GUS Q4W and Q8W
- The GUS safety profile from W24–W52 was consistent with that during the PBO-controlled period; no new safety signals were identified

Statistical Analyses

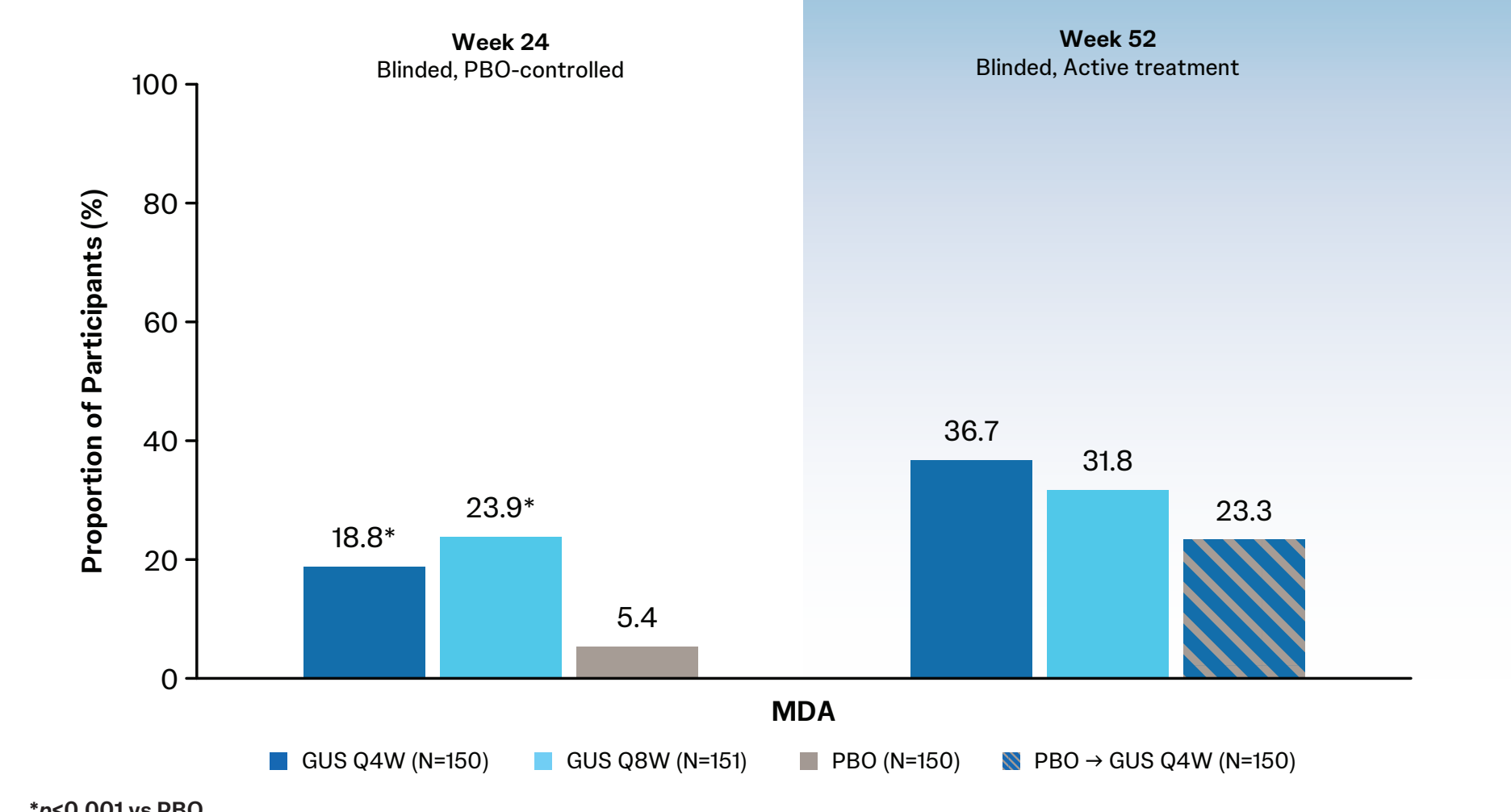
- Pts were considered nonresponders through W24 if they increased dose/initiated csDMARDs or oral corticosteroids or initiated protocol-prohibited PsA therapies, and through W52 if they discontinued study agent for any reason other than ND/MD
- Data impacted by ND/MD were imputed using MI; other missing data were imputed using NRI. Response rates shown are the average proportion achieving response, over 200 MI datasets

In the Q4W and Q8W groups, PASI 90 response rates indicated improvements in psoriatic skin disease from W24–W52



*p<0.001 vs PBO. Among pts with baseline BSA ≥3% and IGA ≥2 (mild). Post hoc analysis used methods consistent with primary and major secondary endpoint analysis. BSA=body surface area, GUS=guselkumab, IGA=Investigator's Global Assessment of PsO, PASI=Psoriasis Area and Severity Index, PBO=placebo, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

MDA response rates numerically increased from W24–W52 in GUS-randomized pts



*p<0.001 vs PBO. Post hoc analysis used methods consistent with primary and major secondary endpoint analysis. GUS=guselkumab, MDA=minimal disease activity, PBO=placebo, Q4W=every 4 weeks, Q8W=every 8 weeks, W=week.

Frequencies of AEs and SAEs were similar across treatment groups

	GUS Q4W W0–24	GUS Q8W W0–24	PBO W0–24	GUS Q4W W0–52	GUS Q8W W0–52	PBO → GUS Q4W W24–52*
Safety Analysis Set, N ^a	150	151	149	150	151	138
Mean weeks of follow-up	24.0	23.7	23.6	50.7	50.2	27.4
Mean number of GUS administrations	5.7	3.8	0	12.1	6.6	6.7
Pts with ≥1 of the following:						
AE	70 (46.7)	81 (53.6)	72 (48.3)	97 (64.7)	101 (66.9)	64 (46.4)
Events/100 PYs	178.1	212.2	207.0	170.1	200.4	181.0
SAE	2 (1.3)	4 (2.6)	6 (4.0)	5 (3.3)	10 (6.6)	2 (1.4)
Events/100 PYs	2.9	7.2	8.8	3.4	9.6	2.8
AE leading to discontinuation of study agent	1 (0.7)	2 (1.3)	3 (2.0)	1 (0.7)	2 (1.3)	2 (1.4)
Events/100 PYs	1.4	1.4	4.4	0.7	1.4	2.8
Infections	35 (23.3)	43 (28.5)	44 (29.5)	50 (33.3)	63 (41.7)	39 (28.3)
Events/100 PYs	55.6	78.0	74.9	47.3	73.7	70.4
Opportunistic infections	0	0	0	0	0	0
Events/100 PYs	0	0	0	0	0	0
Injection site reactions	1 (0.7)	2 (1.3)	1 (0.7)	6 (4.0)	3 (2.0)	1 (0.7)
Events/100 PYs	2.8	2.9	1.5	7.5	2.8	5.5
Safety events of interest W0–W24:	• 2 x serious infections (pyelonephritis and laryngitis) • 1 x malignancy (basal cell carcinoma) • 1 x MACE • 2 x VTEs (DVT and PE in same pt) • 1 x Death (MACE pt)			Safety events of interest W24–W52: • 1 x serious infections (cellulitis) • 2 x malignancy (gastric cancer and colon cancer [fatal]) • 1 x death (colon cancer)		

Data reported as n (%) unless otherwise noted. Includes all pts who received ≥1 study agent administration. *Pts are counted only once for any given event, regardless of the number of times they actually experienced the event. Includes only pts who received GUS Q4W following crossover at W24. AEs are coded using Medical Dictionary for Regulatory Activities Version 27.0. AE=Adverse event, DVT=deep vein thrombosis, GUS=guselkumab, IGA=Investigator's Global Assessment of PsO, MACE=Major adverse cardiovascular death (cardiovascular death, nonfatal myocardial infarction, and nonfatal stroke), PBO=placebo, PE=pulmonary embolism, Pts=participants, PY=patient-year, Q4W=every 4 weeks, Q8W=every 8 weeks, SAE=Serious adverse event, VTE=Venous thromboembolism events, W=week.