Durable Inhibition of Structural Damage Progression and Improvements in Joint Disease Activity with Guselkumab in Active and Erosive Psoriatic Arthritis: Week 48 Results from APEX

Christopher T. Ritchlin,[1] Philip J. Mease,[2,3] Laura C. Coates,[4] Alexa P. Kollmeier,[5] Bei Zhou,[6] Yusang Jiang,[6] Karen Bensley,[6] Koeun Im,[7] Rattandeep Batra,[8] Karissa Lozenski,[9] Steven Fakharzadeh,[9,10] Soumya D. Chakravarty,[9,11] Proton Rahman,[12] Désirée van der Heijde[13]

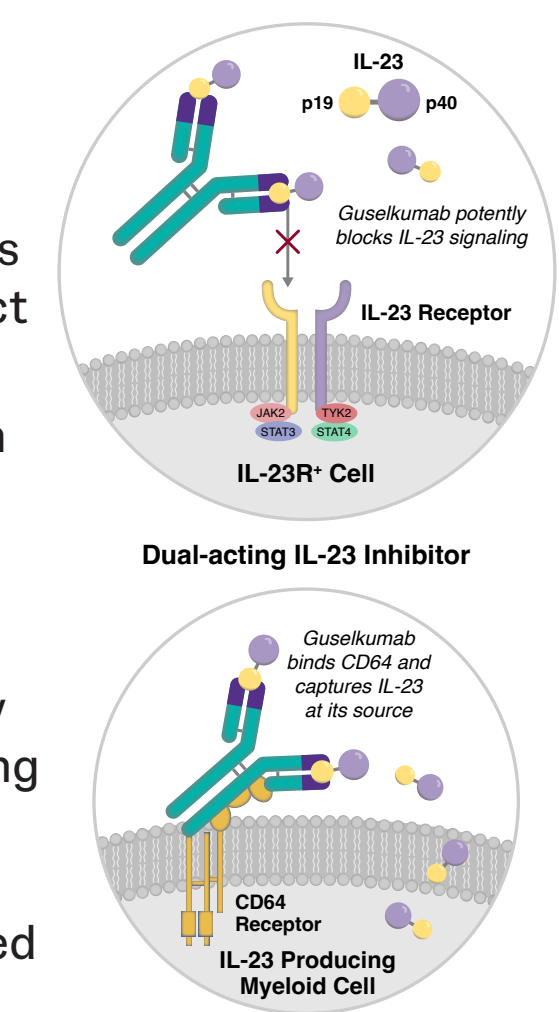
[1]Department of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester, NY, USA; [2]Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; [3]University of Washington School of Medicine, Seattle, WA, USA; [4]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Botnar Research Centre, Oxford, UK; [5]Johnson & Johnson, San Diego, CA, USA; [6]Johnson & Johnson, Spring House, PA, USA; [7]Johnson & Johnson, Cambridge, MA, USA; [8]Johnson & Johnson, Toronto, Canada; [9]Johnson & Johnson, Horsham, PA, USA; [10]Department of Dermatology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA; [11]Drexel University College of Medicine, Philadelphia, PA, USA; [12]Craig L Dobbin Genetics Research Centre, Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. John's, NL, Canada; [13]Leiden University Medical Center, Leiden, The Netherlands

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.

Background

Guselkumab (GUS) for Psoriatic Arthritis (PsA)

- PsA is a chronic, heterogeneous, inflammatory disease affecting joints and skin that can substantially impact health-related quality of life[1,2]
  - Structural damage resulting from chronic inflammation leads to poorer outcomes[3]
- GUS is a fully human, dual-acting monoclonal antibody that selectively inhibits interleukin (IL)-23 by targeting its p19 subunit, and is able to bind cluster of differentiation (CD)64 expressed by immune cells in inflamed tissues[4]
- GUS is indicated for treating active PsA, moderate-to-severe plaque psoriasis, and moderately-to-severely active Crohn's disease and ulcerative colitis[5]


IL-23
p19
p40
Guselkumab potently blocks IL-23 signaling
IL-23 Receptor
JAK2
TYK2
STAT3
STAT4
IL-23R+ Cell
Guselkumab binds CD64 and captures IL-23 at its source
CD64 Receptor
IL-23 Producing Myeloid Cell

Dual-acting IL-23 Inhibitor

APEX (NCT04882098)

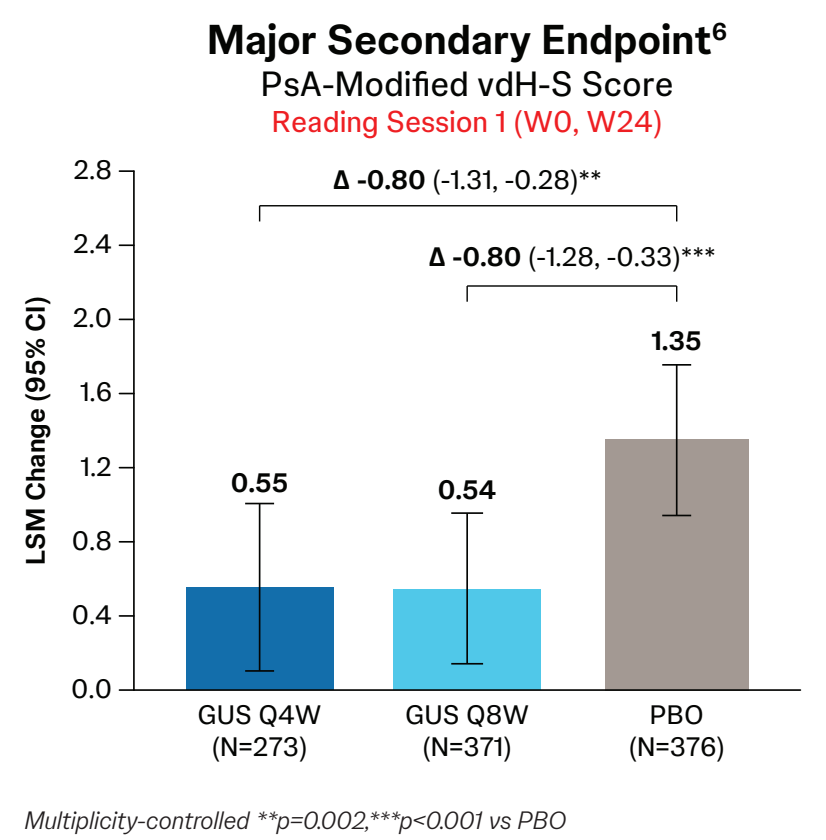
- An ongoing phase 3b, randomized, double-blind, placebo (PBO)-controlled study aimed to further evaluate GUS effects on clinical and radiographic progression outcomes in participants (pts) with active and erosive PsA

Introduction

APEX Results Through Week (W)24[6]

The study met its **primary** and **major secondary endpoints** demonstrating:

- **Significantly higher American College of Rheumatology (ACR)20 rates** with both GUS every (Q)4W and Q8W vs PBO at W24
- **Significant inhibition of structural damage progression** with both GUS Q4W and Q8W vs PBO at W24 based on initial readout (Reading Session 1: W0, W24 radiographs)



*Multiplicity-controlled **p=0.002,***p<0.001 vs PBO*
***CI**=confidence interval, **GUS**=guselkumab, **LSM**=least squares mean, **PBO**=placebo, **PsA**=psoriatic arthritis, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **vdH-S**=van der Heijde-Sharp, **W**=week.*

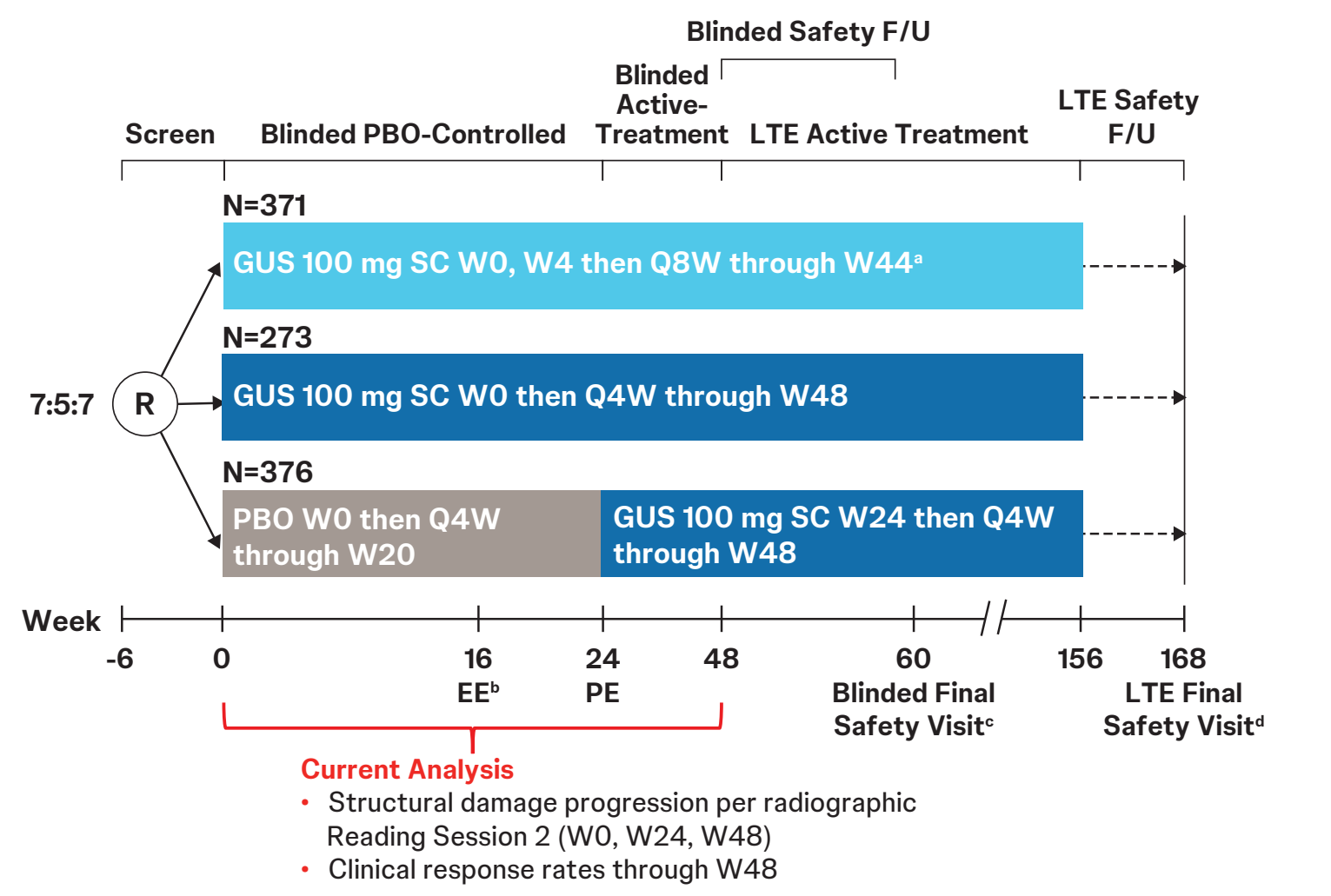
Objectives

Evaluate structural damage progression in APEX based on Reading Session 2, using radiographs from W0, W24, and W48, and report clinical response rates through W48

APEX – Study Design

Key Inclusion Criteria

- Biologic-naïve
- ≥18 years
- Active PsA for ≥6 months (despite prior csDMARDs, apremilast, and NSAIDs); CASPAR criteria met
  - ≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL
- **≥2 erosive joints on hand/foot radiographs**
- Active plaque psoriasis (PsO) (≥1 PsO plaque ≥2 cm and/or nail PsO)


Blinded Safety F/U
Screen
Blinded PBO-Controlled
Blinded Active-Treatment
LTE Active Treatment
LTE Safety F/U
7:5:7
R
N=371
GUS 100 mg SC W0, W4 then Q8W through W44[a]
N=273
GUS 100 mg SC W0 then Q4W through W48
N=376
PBO W0 then Q4W through W20
GUS 100 mg SC W24 then Q4W through W48
Week
-6
0
16
EE[b]
24
PE
48
60
Blinded Final Safety Visit[c]
156
168
LTE Final Safety Visit[d]
Current Analysis
Structural damage progression per radiographic Reading Session 2 (W0, W24, W48)
Clinical response rates through W48

- **Modified full analysis set (mFAS):** All randomized pts excluding those from Ukraine sites rendered unable to support key study operations due to major disruptions; employed as the main efficacy analysis set
- **Safety analysis set:** All pts who received ≥1 administration of any study intervention

*[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. [c]Final safety visit for those who do not enter LTE. [d]Final safety visit for those who entered LTE. **CASPAR**=ClASsification criteria for Psoriatic ARthritis, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease modifying antirheumatic drugs, **EE**=early escape, **F/U**=follow-up, **GUS**=guselkumab, **LTE**=long-term extension, **NSAIDs**=nonsteroidal anti-inflammatory drugs, **PBO**=placebo, **PE**=primary endpoint, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **R**=randomization, **SC**=subcutaneous, **SJC**=swollen joint count, **TJC**=tender joint count, **W**=week.*

Outcomes & Endpoints

Structural Damage Progression	PsA-modified vdH-S score	LSM change W0→24, W24→48, W0→48 (Reading Session 2) GUS maintenance of no radiographic progression[a] W24→48
Joint Disease Activity	ACR20/50/70	Response rates W0→48 GUS maintenance of ACR20/50 responses W24→48
Physical Function	HAQ-DI CMI[b]	Response rates W0→48 GUS maintenance of response W24→48
Skin Disease Activity	PASI 90[c]	Response rates W0→48
Overall Disease Activity	MDA	Achievement rates W0→48 GUS maintenance of status W24→48
Safety	AEs	Number (%) of pts and exposure-adjusted incidence rates (events/100 PY) W0→48

*[a]Defined as change from baseline in total PsA-modified vdH-S score of ≤0 and ≤0.5. [b]Defined as ≥0.35-point score improvement from baseline in HAQ-DI among pts with a baseline score ≥0.35. [c]Defined as ≥90% improvement from baseline in PASI among pts with ≥3% BSA Psoriatic Involvement and an IGA score ≥2 at baseline. **ACR**=American College of Rheumatology, **AE**=adverse event, **BSA**=body surface area, **CMI**=clinically meaningful improvement, **GUS**=guselkumab, **HAQ-DI**=Health Assessment Questionnaire-Disability Index, **IGA**=Investigator's Global Assessment, **LSM**=least squares mean, **MDA**=minimal disease activity, **PASI**=Psoriasis Area and Severity Index, **PsA**=psoriatic arthritis, **pts**=participants, **PY**=participant-years, **vdH-S**=van der Heijde-Sharp, **W**=week.*

Participants

Baseline characteristics of APEX pts with active and erosive PsA were generally balanced across treatment groups

- Background PsA medication use and treatment completion through W48 (>90%) were consistent across treatment groups

Baseline Characteristics	GUS Q4W (N=273)	GUS Q8W (N=371)	PBO (N=376)	Total (N=1,020)
Demographics				
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)	53.0 (13.0)
Male	55%	54%	57%	55%
Weight, kg	85.6 (20.1)	83.2 (17.4)	83.1 (18.2)	83.8 (18.5)
BMI, kg/m²	29.4 (6.0)	29.0 (5.6)	28.9 (5.7)	29.1 (5.7)
Disease Characteristics				
PsA disease duration, years	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)	7.3 (7.2)
SJC (0–66)[a]	9.0 (6.0; 14.0)	10.0 (6.0; 14.0)	9.0 (6.0; 15.0)	9.0 (6.0; 14.0)
TJC (0–68)[a]	16.0 (10.0; 27.0)	17.0 (11.0; 26.0)	16.6 (10.0; 25.5)	16.1 (10.0; 26.0)
HAQ-DI (0–3)	1.2 (0.7)	1.2 (0.6)	1.2 (0.7)	1.2 (0.7)
CRP, mg/dL[a]	0.7 (0.4; 1.5)	0.8 (0.4; 1.6)	0.8 (0.4; 1.8)	0.8 (0.4; 1.6)
Enthesitis / Dactylitis	58% / 44%	59% / 39%	59% / 45%	58% / 43%
Mean LEI (1–6) / DSS (1–60)	3.2 / 10.8	3.0 / 11.0	3.0 / 10.2	3.1 / 10.6
% BSA with PsO	15.0 (19.2)	16.5 (21.9)	16.3 (21.5)	16.0 (21.0)
PASI (0–72)	7.6 (8.3)	8.3 (10.1)	8.2 (9.5)	8.1 (9.4)
Radiographic Characteristics				
PsA-modified vdH-S score (0–528)[b]	25.1 (47.5)	24.2 (42.5)	23.9 (40.2)	24.3 (43.0)
Erosion score (0–320)	11.9 (24.6)	11.4 (21.0)	11.4 (19.6)	11.5 (21.5)
JSN score (0–208)	13.2 (23.8)	12.8 (22.6)	12.5 (21.6)	12.8 (22.5)

*Data shown are mean (SD) unless otherwise noted. [a]Values are median (IQR). [b]Data shown are from reading session 2. For pts with missing W48 radiographs, W0/24 radiographs were not re-read during reading session 2; W0/24 data from reading session 1 was used if available. The same readers were used for reading session 1 and 2. **BMI**=body mass index, **BSA**=body surface area, **CRP**=C-reactive protein, **DSS**=Dactylitis Severity Score, **GUS**=guselkumab, **HAQ-DI**=Health Assessment Questionnaire-Disability Index, **IQR**=interquartile range, **JSN**=joint space narrowing, **LEI**=Leeds Enthesitis Index, **PASI**=Psoriasis Area and Severity Index, **PBO**=placebo, **PsA**=psoriatic arthritis, **PsO**=psoriasis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **SD**=standard deviation, **SJC**=swollen joint count, **TJC**=tender joint count, **vdH-S**=van der Heijde-Sharp, **W**=week.*

Key Takeaways

In the phase 3 APEX study of biologic-naïve adults with active and erosive PsA:

✓ **Through W48, GUS Q4W and Q8W dosing regimens demonstrated robust and durable:**

- ✓ **Inhibition of structural damage progression; ~90% of GUS pts with no radiographic progression at W24 maintained this status at W48**
- ✓ **Improvements in clinical and pt-reported outcomes, with ~40% achieving minimal disease activity (MDA)**

✓ **Safety was consistent with the established GUS safety profile across indications**

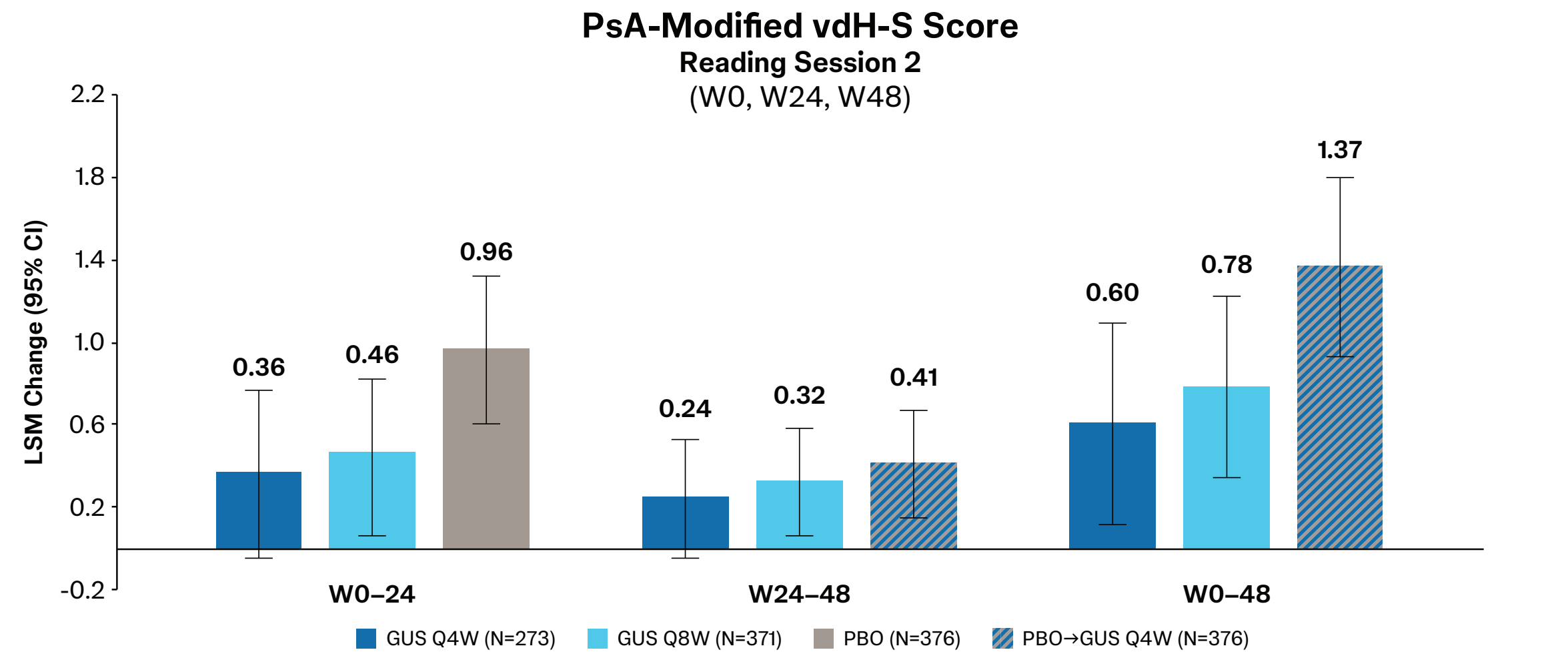
- ✓ **GUS AE rates were similar to PBO through W24 and were generally stable with continued GUS through W48**

Longer-term APEX data further substantiate GUS benefit for preserving joint health, as the only selective IL-23i to demonstrate significant and durable inhibition of structural damage progression

Results

Inhibition of structural damage progression with GUS was durable through W48

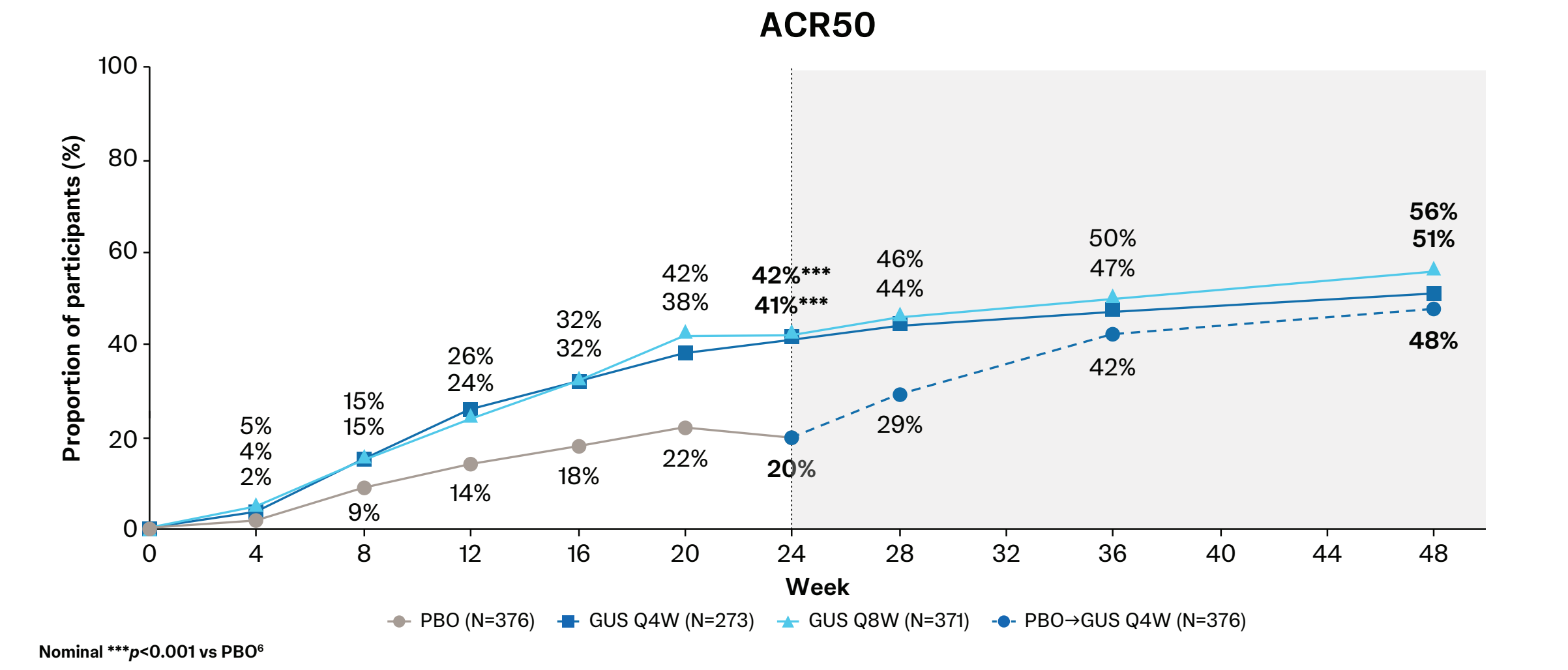
- Treatment with GUS from W24–48 reduced the rate of radiographic progression among PBO-randomized pts



*For pts with missing W48 radiographs, W0/24 radiographs were not re-read during reading session 2; W0/24 data from reading session 1 was used if available. The same readers were used for reading session 1 and 2. Score change was assessed in the mFAS according to the Adjusted Treatment Policy Estimand: irrespective of background PsA medication or adherence to study intervention; in situations when ND/MD occurred, observed data collected after ND/MD was not used; data for visits after ND/MD and missing data were imputed using full conditional specifications MI. LSM change and CIs based on combining ANCOVA model (explanatory variables: baseline modified vdH-S score, treatment group, and randomization stratification level) results from each MI dataset. **ANCOVA**=Analysis of Covariance, **CI**=confidence interval, **GUS**=guselkumab, **LSM**=least squares mean, **mFAS**=modified full analysis set, **MI**=multiple imputation, **ND/MD**=natural disaster or major disruption, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **vdH-S**=van der Heijde-Sharp, **W**=week.*

>50% of GUS-randomized pts achieved ACR50 response at W48

- PBO-randomized pts achieved comparable ACR50 rates after starting GUS

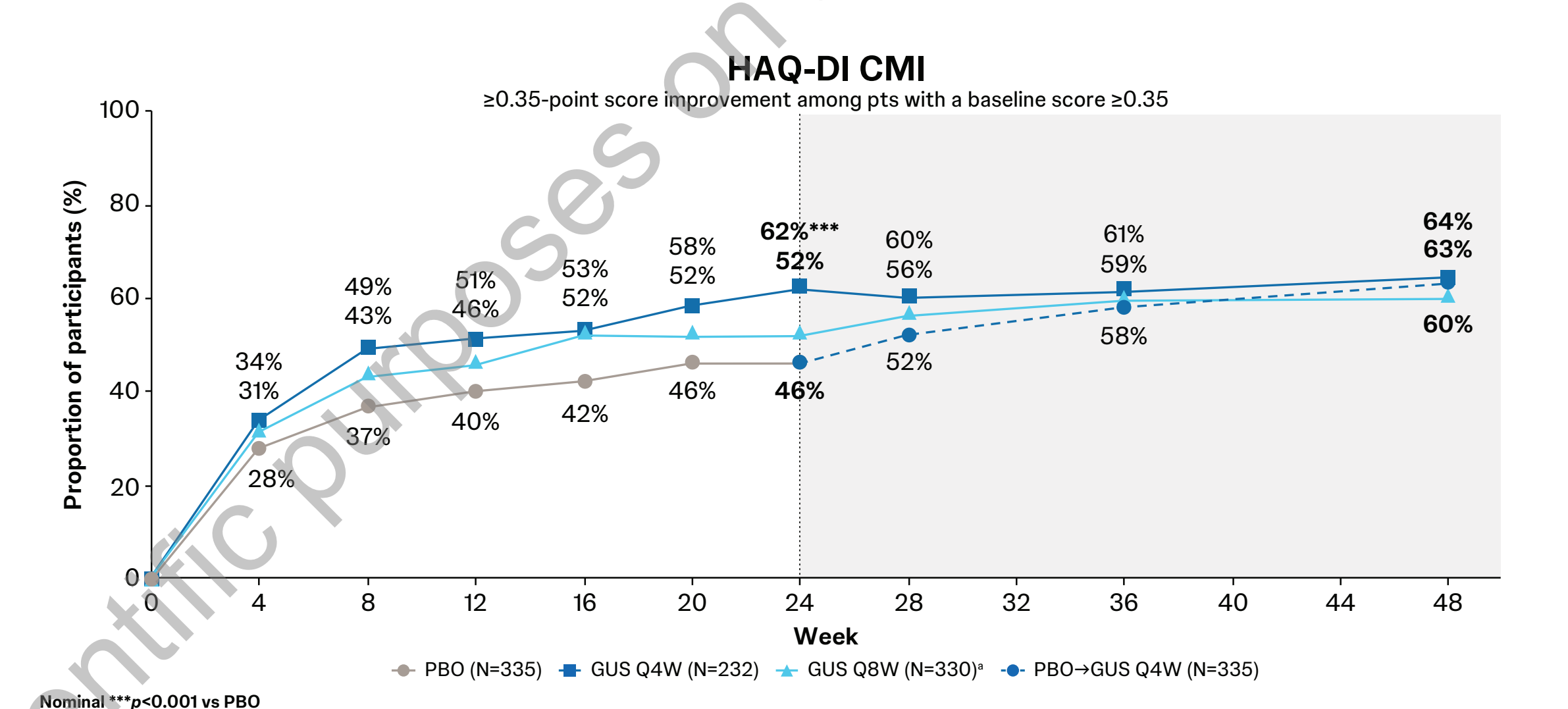


Nominal ***p<0.001 vs PBO[6]

*Shown are the average proportion of mFAS pts with ACR50 response, over the 200 MI datasets. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were treated as nonresponders; after W24, these pts were not treated as nonresponders. Data from pts who discontinued study intervention or with severe treatment noncompliance due to ND/MD were imputed using MI at all subsequent timepoints or at the next timepoint, respectively. Other missing data were imputed using NRI. **ACR**=American College of Rheumatology, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GUS**=guselkumab, **mFAS**=modified full analysis set, **MI**=multiple imputation, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

>60% of GUS-randomized pts reported CMI in physical function at W48

- PBO-randomized pts achieved comparable HAQ-DI CMI rates after starting GUS

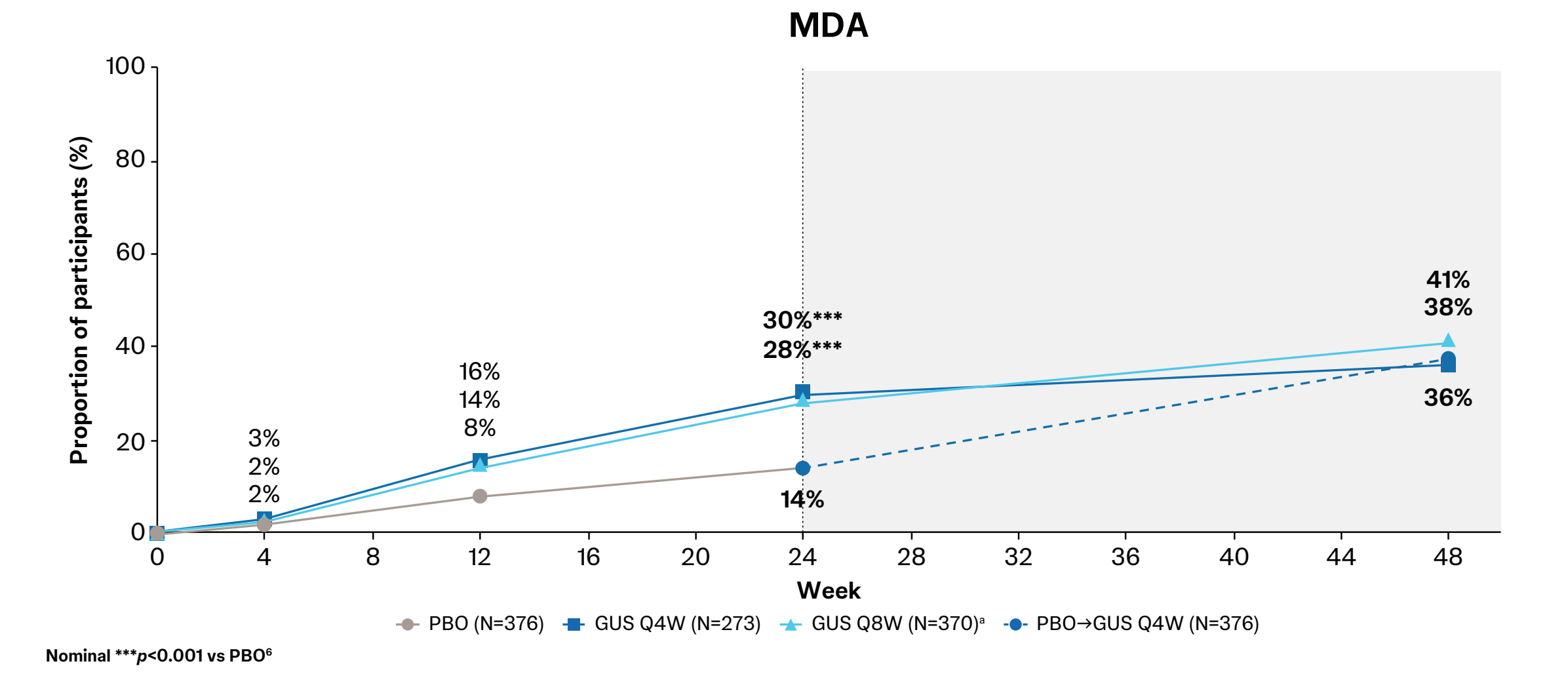


Nominal ***p<0.001 vs PBO

Response rates and p values based on GLMMs adjusted for treatment group, visit, treatment group visit, and randomization stratification level. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were also considered nonresponders; after W24, these pts were not treated as nonresponders. Missing data from ND/MD was handled within the model. Other missing data were imputed using NRI. [a]N=331 from W28–48. **CMI**=clinically meaningful improvement, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **HAQ-DI**=Health Assessment Questionnaire-Disability Index, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

~40% of GUS-randomized pts achieved MDA at W48

- PBO-randomized pts achieved comparable MDA rates after starting GUS



Nominal ***p<0.001 vs PBO[6]

Response rates and p values based on GLMMs adjusted for treatment group, visit, treatment group visit, and randomization stratification level. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were also considered nonresponders; after W24, these pts were not treated as nonresponders. Missing data from ND/MD was handled within the model. Other missing data were imputed using NRI. [a]N=371 at W48. **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **MDA**=minimal disease activity, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

AE rates were consistent across groups at W24 and generally stable through W48

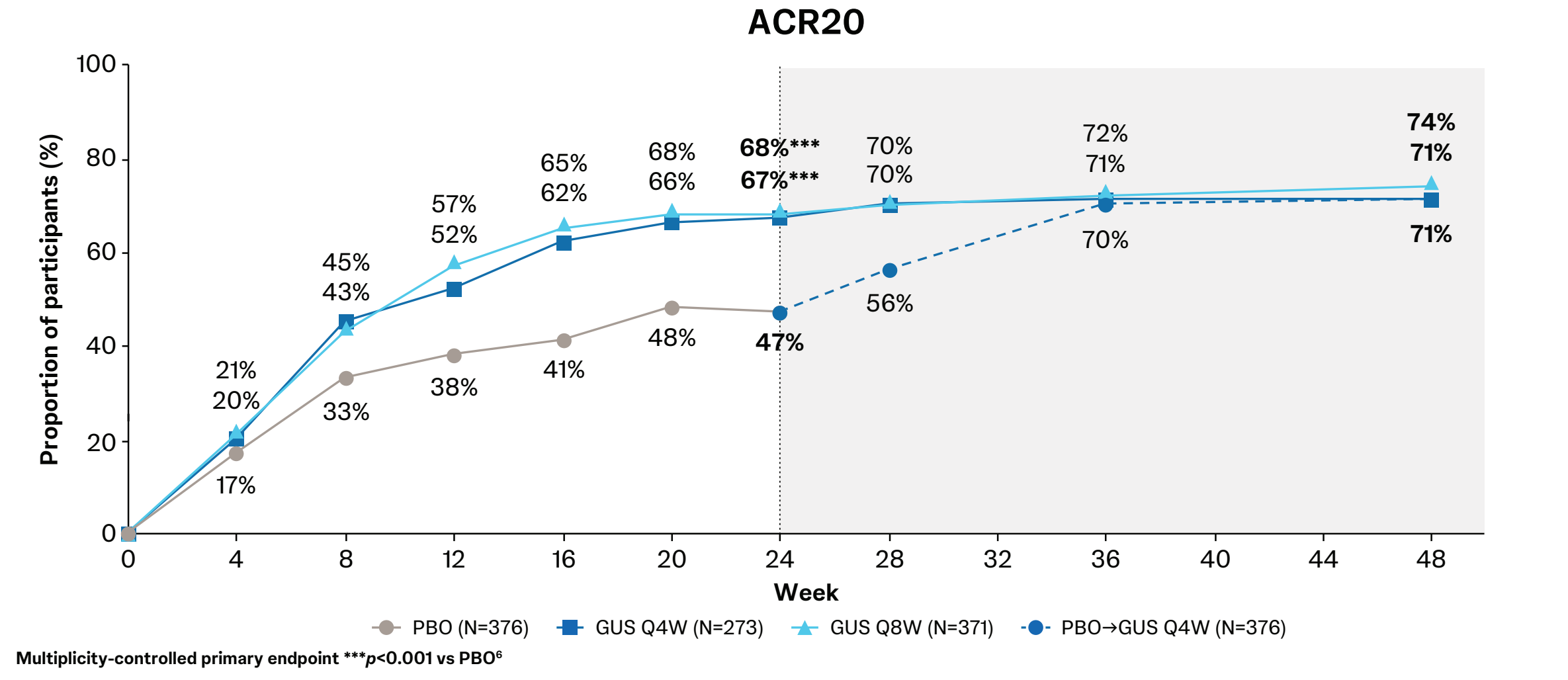
- In GUS-randomized pts, exposure-adjusted AE rates did not increase through W48

	PBO-Controlled (W0–24)			W24–48	W0–48	
AEs Through W48	GUS Q4W (N=280)	GUS Q8W (N=388)	PBO (N=386)	PBO → GUS Q4W (N=372)	GUS Q4W (N=280)	GUS Q8W (N=388)
Mean weeks of follow-up	24.2	24.2	24.1	23.5	47.3	46.9
Total PY of follow-up	130	180	179	168	254	349
Any AE	108 (39%)	166 (43%)	146 (38%)	127 (34%)	142 (51%)	214 (55%)
Incidence/100 PY (95% CI)	168 (146, 191)	163 (145, 183)	174 (155, 195)	156 (138, 176)	147 (132, 163)	148 (136, 162)
SAE	5 (2%)	12 (3%)	10 (3%)	16 (4%)	10 (4%)	21 (5%)
Incidence/100 PY (95% CI)	4 (1, 9)	9 (5, 14)	7 (3, 12)	14 (9, 21)	4 (2, 7)	9 (6, 12)
AE leading to D/C	2 (1%)	7 (2%)	1 (<1%)	8 (2%)	4 (1%)	10 (3%)
Incidence/100 PY (95% CI)	2 (<1, 6)	4 (2, 8)	1 (<1, 3)	7 (4, 12)	2 (<1, 4)	3 (1, 5)
Infection	52 (19%)	91 (23%)	81 (21%)	50 (13%)	84 (30%)	131 (34%)
Incidence/100 PY (95% CI)	55 (43, 70)	66 (54, 79)	57 (47, 69)	39 (30, 50)	50 (41, 59)	58 (51, 67)
Serious infection	2 (1%)	5 (1%)	1 (<1%)	4 (1%)	2 (1%)	6 (2%)
Incidence/100 PY (95% CI)	2 (<1, 6)	3 (1, 6)	1 (<1, 3)	2 (1, 6)	1 (<1, 3)	2 (1, 4)

*Data shown are n (%) unless otherwise noted. AEs were coded using MedDRA Version 27.0. CIs were based on an exact method assuming that the observed number of events follows a Poisson distribution. **AE**=adverse event, **CI**=confidence interval, **GUS**=guselkumab, **D/C**=discontinuation, **MedDRA**=Medical Dictionary for Regulatory Activities, **PBO**=placebo, **PY**=participant-year, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **SAE**=serious AE, **W**=week.*

GUS demonstrated high ACR20 response rates that were durable through W48

- PBO-randomized pts achieved comparable ACR20 rates after starting GUS

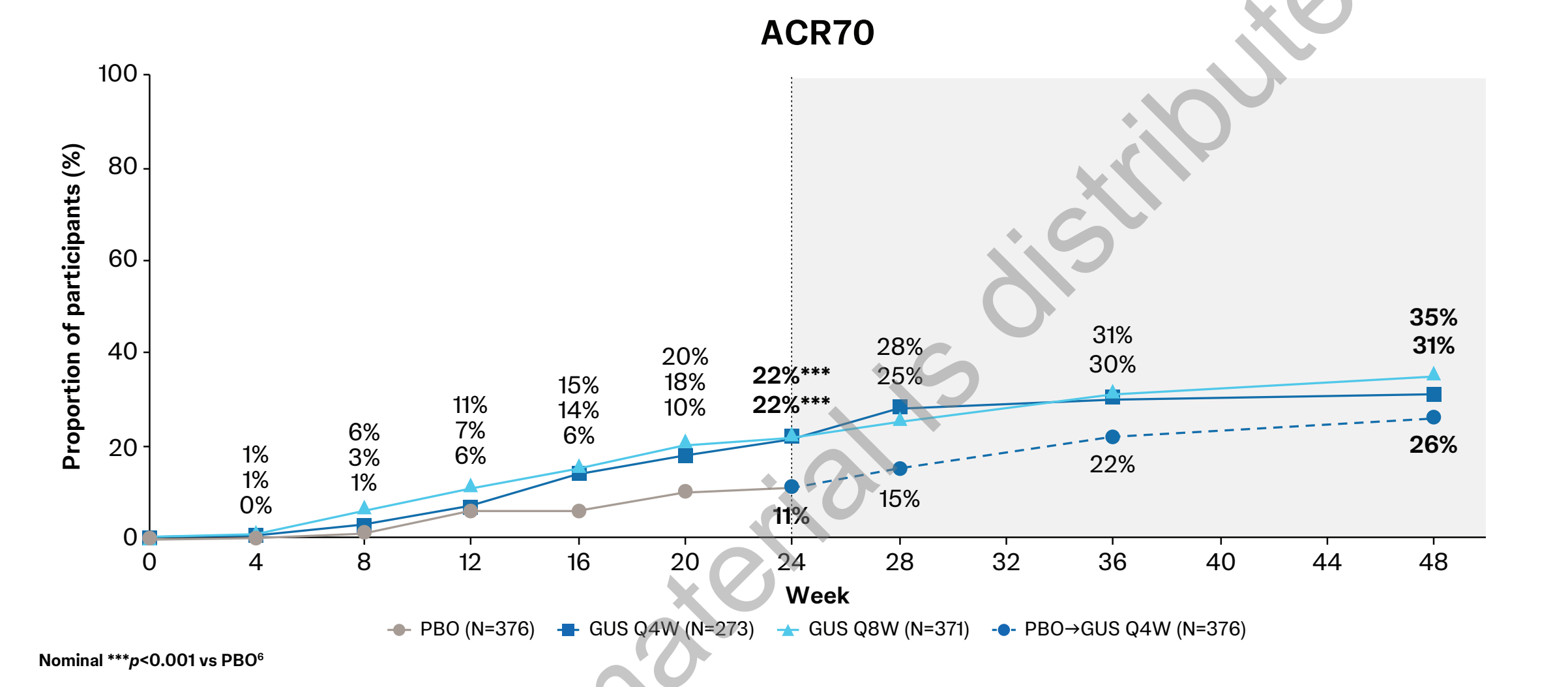


Multiplicity-controlled primary endpoint ***p<0.001 vs PBO[6]

*Shown are the average proportions of mFAS pts with ACR20 response, over the 200 MI datasets. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were treated as nonresponders; after W24, these pts were not treated as nonresponders. Data from pts who discontinued study intervention or with severe treatment noncompliance due to ND/MD were imputed using MI at all subsequent timepoints or at the next timepoint, respectively. Other missing data were imputed using NRI. **ACR**=American College of Rheumatology, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GUS**=guselkumab, **mFAS**=modified full analysis set, **MI**=multiple imputation, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

>30% of GUS-randomized pts achieved ACR70 response at W48

- PBO-randomized pts achieved comparable ACR70 rates after starting GUS

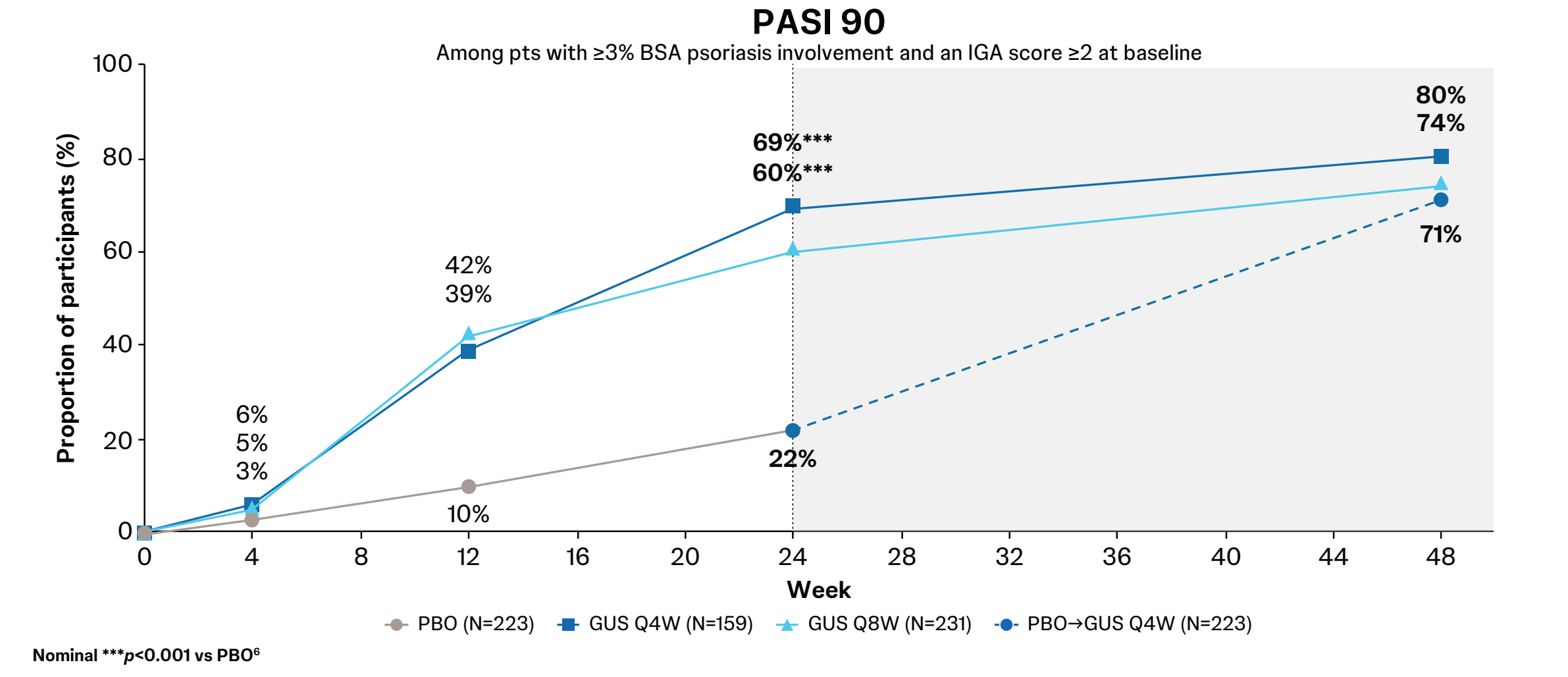


Nominal ***p<0.001 vs PBO[6]

*Shown are the average proportion of mFAS pts with ACR70 response, over the 200 MI datasets. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were treated as nonresponders; after W24, these pts were not treated as nonresponders. Data from pts who discontinued study intervention or with severe treatment noncompliance due to ND/MD were imputed using MI at all subsequent timepoints or at the next timepoint, respectively. Other missing data were imputed using NRI. **ACR**=American College of Rheumatology, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GUS**=guselkumab, **mFAS**=modified full analysis set, **MI**=multiple imputation, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

~75% of GUS-randomized pts achieved clear/almost clear skin at W48

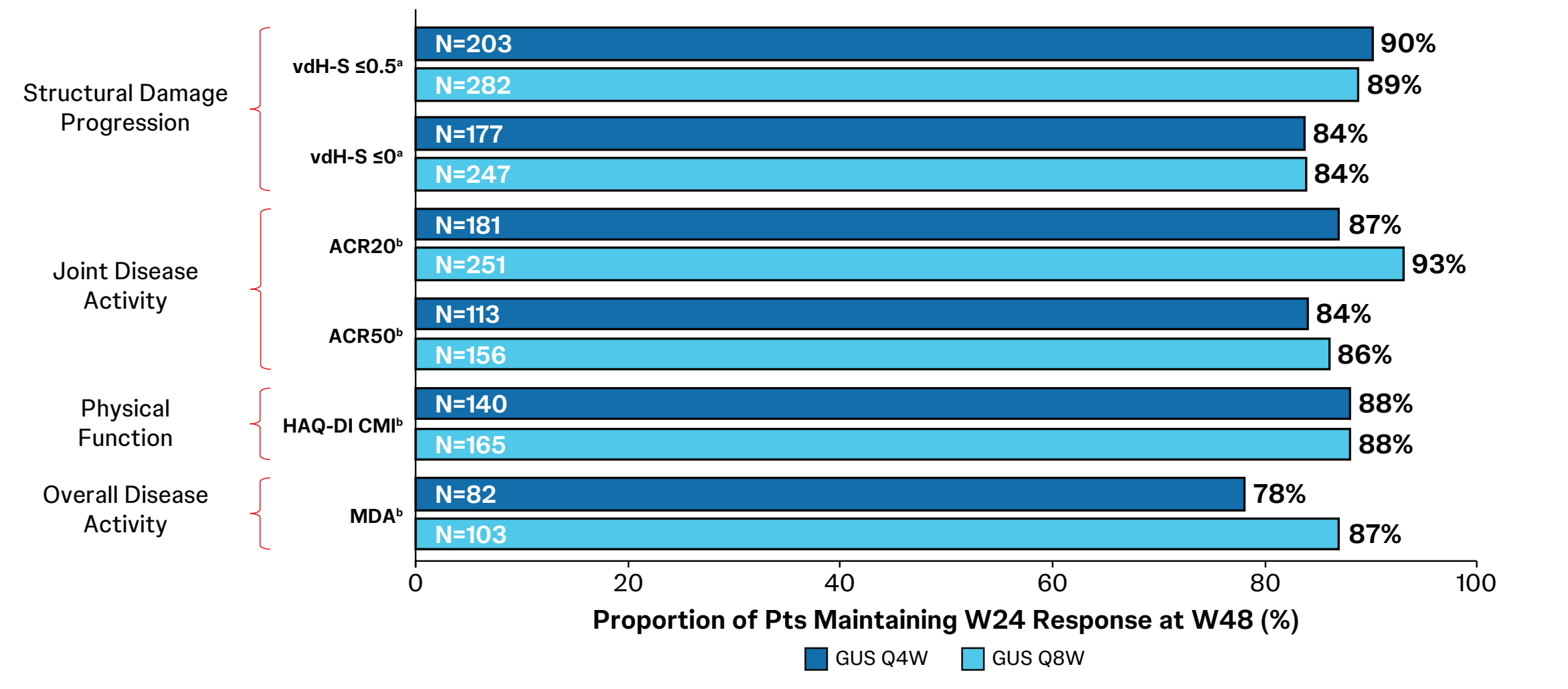
- PBO-randomized pts achieved comparable PASI 90 rates after starting GUS



Nominal ***p<0.001 vs PBO[6]

Response rates and p values based on GLMMs adjusted for treatment group, visit, treatment group visit, and randomization stratification level. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were also considered nonresponders; after W24, these pts were not treated as nonresponders. Missing data from ND/MD was handled within the model. Other missing data were imputed using NRI. **BSA**=body surface area, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **IGA**=Investigator's Global Assessment, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PASI**=Psoriasis Area and Severity Index, **PBO**=placebo, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **W**=week.*

~80–90% of GUS-randomized pts maintained W24 responses through W48



[a]Shown are the average proportions of mFAS pts maintaining no radiographic progression, over the 200 MI datasets. For pts with missing W48 radiographs, W0/24 radiographs were not re-read during reading session 2; W0/24 data from reading session 1 was used if available. The same readers were used for reading sessions 1 and 2. Score change was assessed in the mFAS according to the Adjusted Treatment Policy Estimand: irrespective of background PsA medication or adherence to study intervention; in situations when ND/MD occurred, observed data collected after ND/MD was not used; data for visits after ND/MD and missing data were imputed using full conditional specifications MI. [b]Response rates based on GLMMs adjusted for treatment group, visit, treatment group visit, and randomization stratification level. Through W48, pts who discontinued study intervention for any reason except ND/MD were treated as nonresponders. Through W24, pts who initiated/increased dose of oral corticosteroid or csDMARD or initiated protocol prohibited therapies for PsA were also considered nonresponders; after W24, these pts were not treated as nonresponders. Missing data from ND/MD was handled within the model. Other missing data were imputed using NRI. **ACR**=American College of Rheumatology, **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **CMI**=clinically meaningful improvement, **GLMM**=generalized linear mixed model, **GUS**=guselkumab, **HAQ-DI**=Health Assessment Questionnaire-Disability Index, **MDA**=minimal disease activity, **mFAS**=modified full analysis set, **MI**=multiple imputation, **ND/MD**=natural disaster or major disruption, **NRI**=nonresponder imputation, **PsA**=psoriatic arthritis, **pts**=participants, **Q4W**=every 4 weeks, **Q8W**=every 8 weeks, **vdH-S**=van der Heijde-Sharp, **W**=week.*

PRESENTED BY: Anthony Todd at Congress of Clinical Rheumatology – East 2026 (CCR – East); April 30–May 3, 2026; Destin, FL.

REFERENCES: 1. Gladman DD, et al. *Q J Med.* 1987;62:127–141. **2.** Ritchlin CT. *J Rheumatol.* 2008;35:1434–1437. **3.** van der Heijde D, et al. *Arthritis Res Ther.* 2020;22:18. **4.** Sachen KL, et al. *Front Immunol.* 2025;16:1532852. **5.** Janssen Biotech, Inc. *US FDA website.* Revised March 2025. **6.** Mease PJ, et al. *Ann Rheum Dis.* September 16, 2025.

ACKNOWLEDGMENTS: Medical writing support was provided by Kelly Koch, PharmD of Johnson & Johnson, under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298–1304). Layout design and formatting for this presentation were provided by Amit Kavle of SIRO Medical Writing Pvt., Ltd, India (funded by Johnson & Johnson). This presentation was sponsored by Johnson & Johnson.

DISCLOSURES: CTR: Received grant/research support from AbbVie, Amgen, and UCB; and consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, and UCB. **PJM:** Consultant for AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB; received grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Sana, and UCB; speaker for AbbVie, Amgen, Eli Lilly, Johnson & Johnson, and UCB. **LCC:** Received grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB; worked as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly, Gilead, Galapagos, 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. **APK, BZ, YJ, KB, KI, RB, SF, SDC,** and **AT:** Employees of Johnson & Johnson; may own stock/stock options in Johnson & Johnson. **KL:** Employee of Johnson & Johnson; owns stock/stock options in Johnson & Johnson and Bristol Myers Squibb. **PR:** Received 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. **DvdH:** Received consulting fees from AbbVie, Alfasigma, ArgenX, Bristol Myers Squibb, Eli Lilly, Grey-Wolf Therapeutics, Johnson & Johnson, Novartis, Pfizer, Takeda, and UCB; and is associate editor of Annals Rheumatic Diseases, editorial board member Journal of Rheumatology, and director of Imaging Rheumatology BV.

Previously presented at: The 19th Annual Rheumatology Winter Clinical Symposium (RWCS); February 11–14, 2026; Maui, HI, USA.

This material is distributed for scientific purposes on J&J Medical Connect, and is not for promotional use