Achievement of DAPSA/cDAPSA Low Disease Activity With Guselkumab in Active and Erosive Psoriatic Arthritis Across Participant Subgroups in APEX

Christopher T Ritchlin[1], Laura C Coates[2], Philip J Mease[3,4], Alexa P Kollmeier[5], Weihong Hu[6], Soumya D Chakravarty[7,8], Stacey Fitch[7], Karissa Lozenski[7], Alexis Ogdie[9], Proton Rahman[10]

[1]Department of Medicine, Allergy/Immunology and Rheumatology, University of Rochester Medical Center, Rochester, NY, USA; [2]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Botnar Research Centre, Oxford, UK; [3]Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; [4]University of Washington School of Medicine, Seattle, WA, USA; [5]Johnson & Johnson, San Diego, CA, USA; [6]Johnson & Johnson, Spring House, PA, USA; [7]Johnson & Johnson, Horsham, PA, USA; [8]Drexel University College of Medicine, Philadelphia, PA, USA; [9]University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [10]Faculty of Medicine, Division of Rheumatology, Memorial University of Newfoundland, St. Johns, NL, Canada

Key Takeaways

- In APEX, GUS-treated pts had greater response rates for achieving cDAPSA/DAPSA LDA/REM through W24 vs PBO-treated pts
  - Response rates numerically increased through 1 year for GUS-randomized pts
- Achievement of cDAPSA/DAPSA LDA/REM with GUS was generally consistent with both dosing regimens and across diverse pt subgroups defined by baseline demographics, disease characteristics, concomitant csDMARD use, and structural damage

Background

- Psoriatic arthritis (PsA) is a heterogeneous, chronic, inflammatory disease characterized by peripheral arthritis, axial involvement, dactylitis, enthesitis, psoriatic skin, and nail disease
- Guselkumab (GUS) is a fully human, dual-acting,[1] monoclonal antibody that selectively inhibits 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 colitis[2]
- In the ongoing phase 3b, randomized, double-blind, placebo (PBO)-controlled APEX study of participants (pts) with active and erosive PsA, GUS demonstrated efficacy vs PBO in improving signs and symptoms of PsA and significantly inhibiting structural damage progression at Week (W) 24[3]

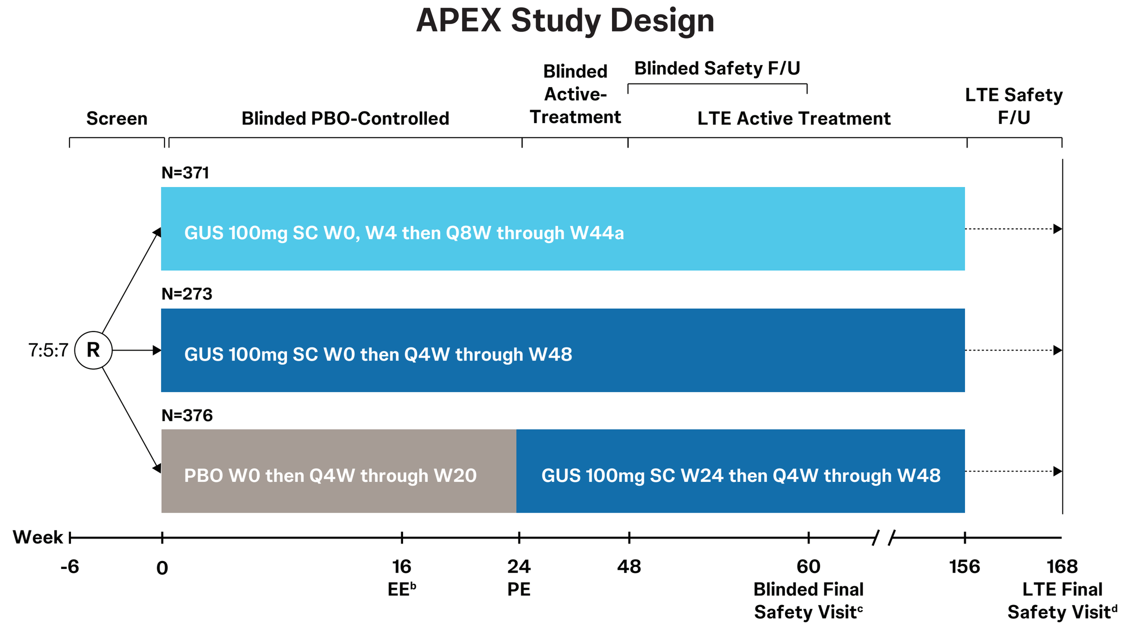
Objective

This post hoc analysis evaluated the consistency of GUS treatment effect in achieving low disease activity (LDA)/remission (REM) using the Disease Activity Index for PsA (DAPSA) and clinical DAPSA (cDAPSA) at W24 across baseline subgroups in APEX study

Methods

APEX Study Design

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



DAPSA[4]	cDAPSA[4,5]
REM	
≤4	≤4
LDA	
≤14	≤13
ModDA	
≤28	≤27
HDA	
>28	>27
Components	
• SJC	• SJC
• TJC	• TJC
• Pt Pain	• Pt Pain
• PtGA	• PtGA
• CRP	

Endpoints

- cDAPSA and DAPSA LDA/REM (score ≤13 or ≤14, respectively) through W48
- cDAPSA and DAPSA LDA/REM at W24 by baseline subgroups
  - Demographics (Sex, BMI)
  - PsA characteristics (PsA duration, SJC, TJC, CRP, PASI)
  - Concomitant medication use (csDMARDs)
  - Radiographic features (total PsA-modified vdH-S, erosion, and JSN scores; number of joints with erosions and JSN)

Statistical Analyses

- Response rates and p-values from W4-24 were based on GLMM; p-values were not multiplicity controlled, are considered nominal, and may not be used to claim statistical significance
- Pts were considered nonresponders at a post-baseline visit through W24 if they had previously initiated/increased oral corticosteroids or csDMARDs or initiated protocol-prohibited PsA therapies and through W48 if they discontinued study agent for any reason other than ND/MD. Data affected by ND/MD were not used. After applying these rules, missing data due to ND/MD were accounted for in the analysis model, and other missing data were imputed using NRI
- Efficacy analyses included all randomized pts except those from Ukrainian sites unable to support key study operations due to major disruption (mFAS, N=1020)

aPBO SC W8 then Q8W through W48 administered to maintain blinding. bEE 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. cFinal safety visit for those who do not enter LTE. dFinal safety visit for those who entered LTE. **BMI**=body mass index, **CASPAR**=CIASsification criteria for Psoriatic Arthritis, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease modifying antirheumatic drugs, **EE**=early escape, **F/U**=follow-up, **GLMM**=generalized linear mixed model, **HDA**=high disease activity, **JSN**=joint space narrowing, **LTE**=long-term extension, **MD**=major disruption: involving Ukraine and neighboring countries/territories beginning 24 February 2022, **mFAS**=modified full analysis set, **ModDA**=moderate disease activity, **ND**=natural disaster: site closure, site access restrictions, or lockdowns due to the COVID-19 pandemic, **NRI**=nonresponder imputation, **NSAIDs**=nonsteroidal anti-inflammatory drug, **PE**=primary endpoint, **PASI**=Psoriasis Area and Severity Index, **PtGA**=patient's global assessment, **Pt Pain**=patient's assessment of pain, **Q4W/Q8W**=every 4/8 weeks, **R**=randomization, **SC**=subcutaneous, **SJC**=swollen joint count, **TJC**=tender joint count, **vdH-S**=van der Heijde-Sharp.

Results

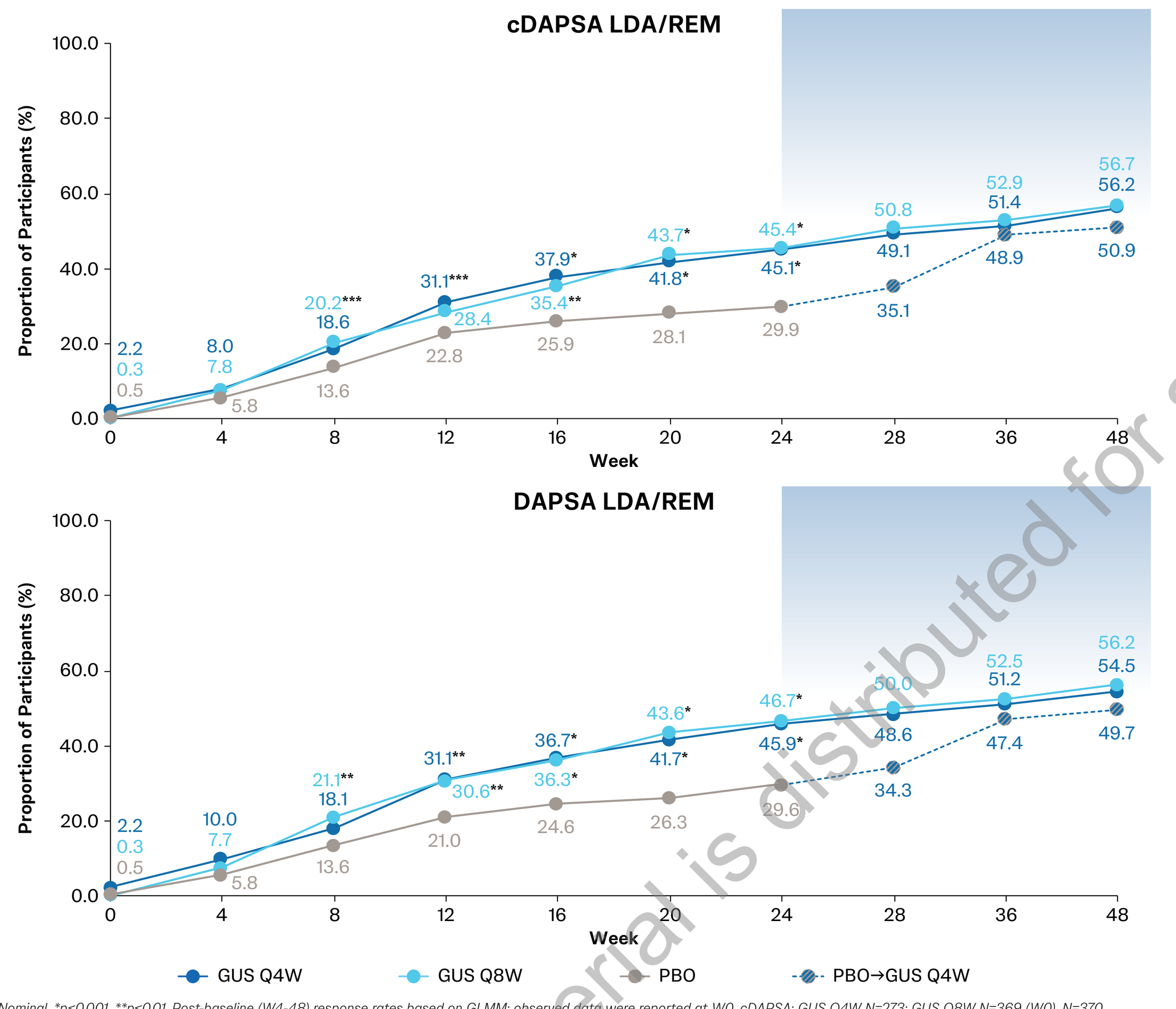
Baseline characteristics of pts with active and erosive PsA were balanced across treatment groups

Baseline Characteristics	GUS Q4W (N = 273)	GUS Q8W (N = 371)	PBO (N = 376)
Demographics			
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)
Female	45.4%	46.4%	43.4%
PsA Characteristics			
PsA disease duration, years	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)
SJC [0-66]	11.6 (9.4)	12.1 (8.5)	11.8 (8.9)
TJC [0-68]	21.2 (14.6)	20.6 (13.4)	20.5 (13.9)
HAQ-DI [0-3]	1.2 (0.7)	1.2 (0.6)	1.2 (0.6)
Patient assessment of pain [VAS; 0-10cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.1)
Patient's global assessment - arthritis [VAS; 0-10cm]	5.9 (2.2)	5.9 (2.1)	5.9 (2.0)
Physician's global assessment [VAS; 0-10cm]	6.4 (1.6)	6.4 (1.6)	6.2 (1.7)
CRP, mg/dL[a]	0.7 (0.4; 1.5)	0.8 (0.4; 1.6)	0.8 (0.4; 1.8)
DAPSA	46.4 (24.3)	46.1 (22.1)	46.0 (22.8)
cDAPSA	44.7 (23.5)	44.6 (21.7)	44.3 (22.3)
PsO Characteristics			
%BSA	15.0 (19.2)	16.5 (21.9)	16.3 (21.5)
PASI [0-72]	7.6 (8.3)	8.3 (10.1)	8.2 (9.5)

aValues are mean (SD) unless otherwise noted.
HAQ-DI=health assessment questionnaire disability index, **SD**=standard deviation, **VAS**=visual analog scale.

Through W24, greater proportions of GUS-treated pts achieved cDAPSA and DAPSA LDA/REM vs PBO

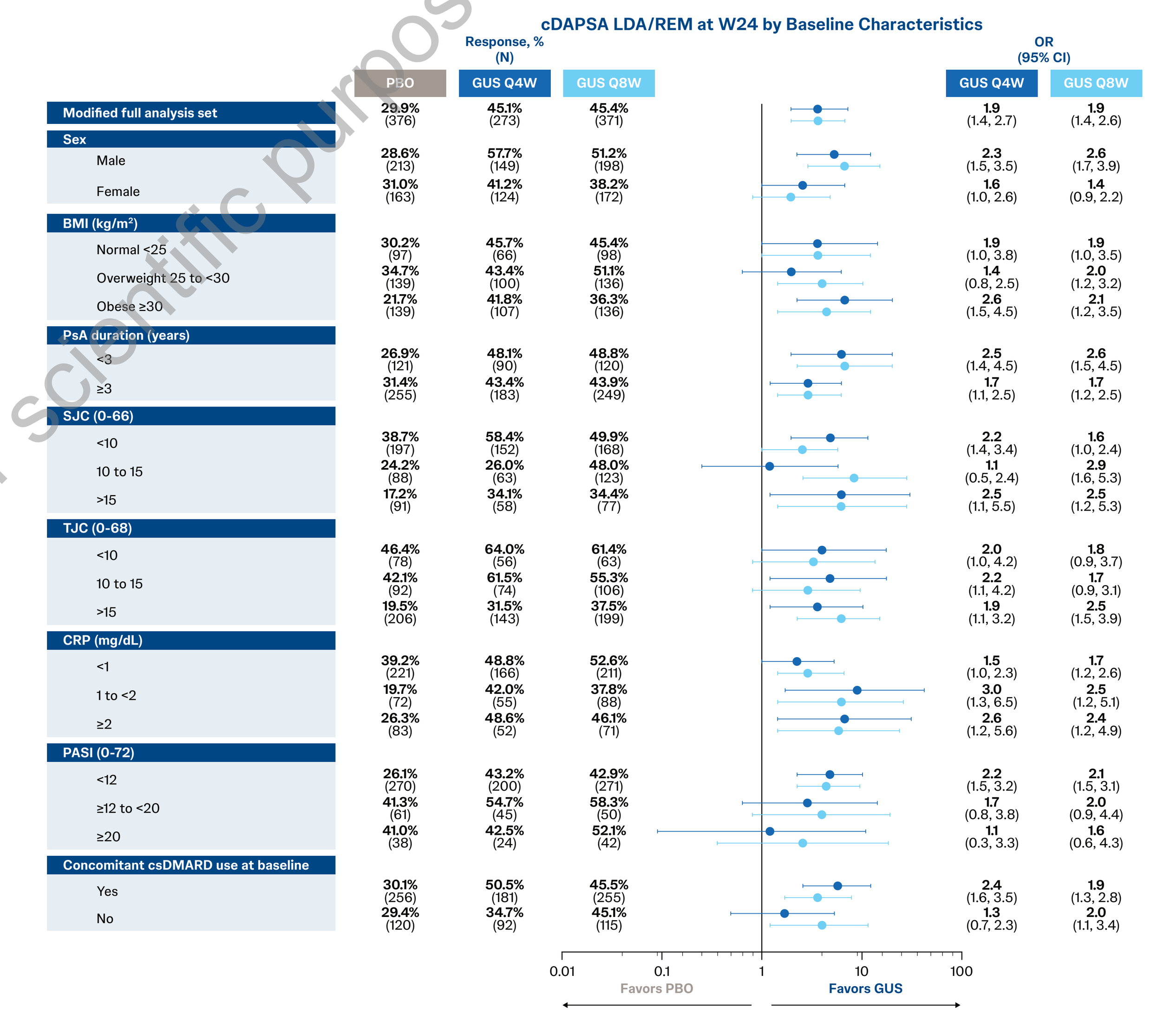
- Response rates for GUS-randomized pts increased through W48

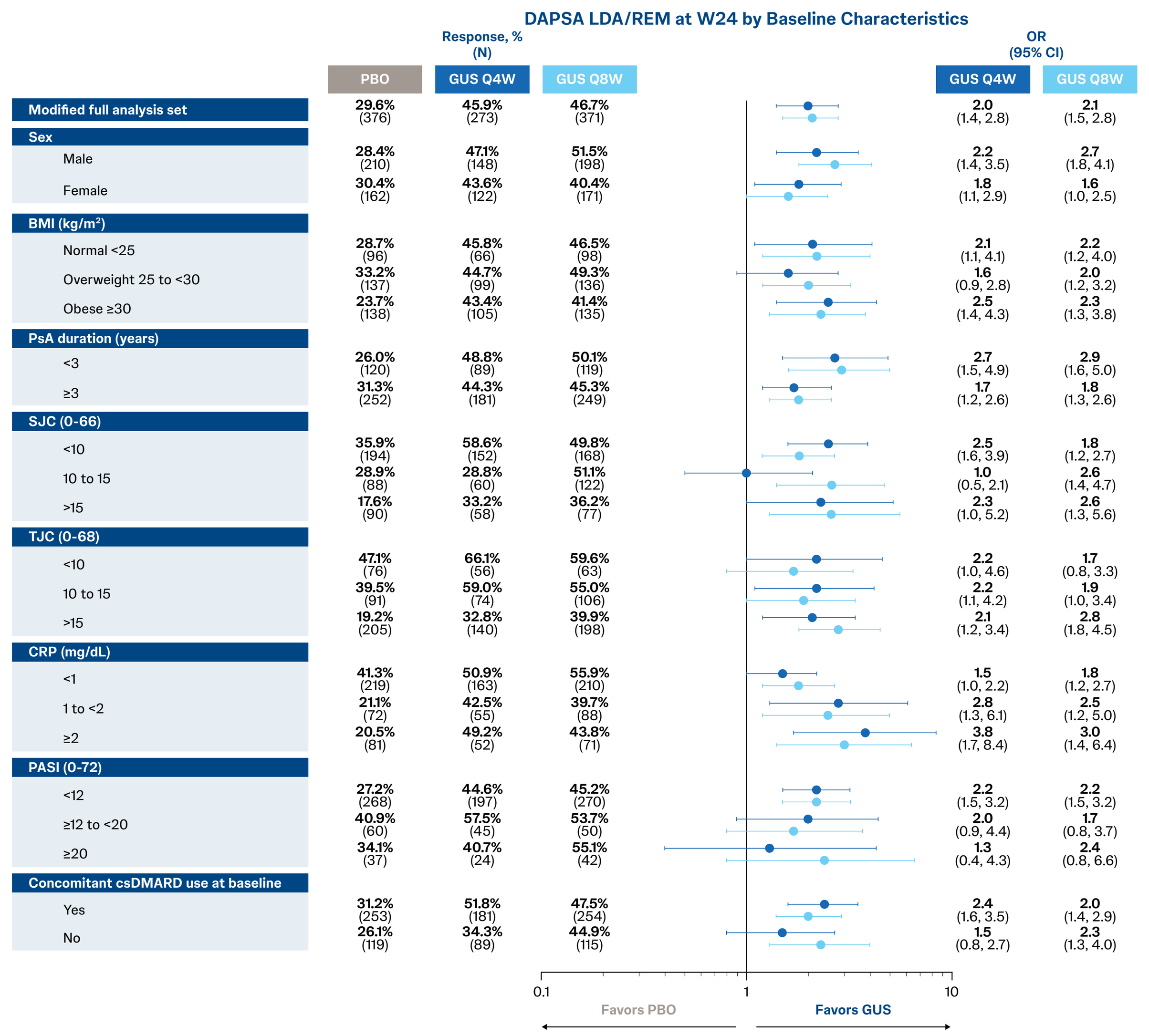


Nominal. *p≤0.001, **p<0.01. Post-baseline (W4-48) response rates based on GLMM; observed data were reported at W0. cDAPSA: GUS Q4W N=273; GUS Q8W N=369 (W0), N=370 (W4-24), N=371 (W28-48); PBO → GUS Q4W N=375 (W0), N=376 (W4-48). DAPSA: GUS Q4W N=273 (W0), N=270 (W4-24), N=273 (W28-48); GUS Q8W N=369 (W0-24), N=371 (W28-48); PBO → GUS N=375 (W0), N=372 (W4-24), N=376 (W28-48).

At W24, GUS treatment effect in achieving cDAPSA and DAPSA LDA/REM was consistent across subgroups

- Q4W/Q8W-treated pts were ~2- to 3-times more likely to achieve cDAPSA and DAPSA response than PBO-treated pts across subgroups

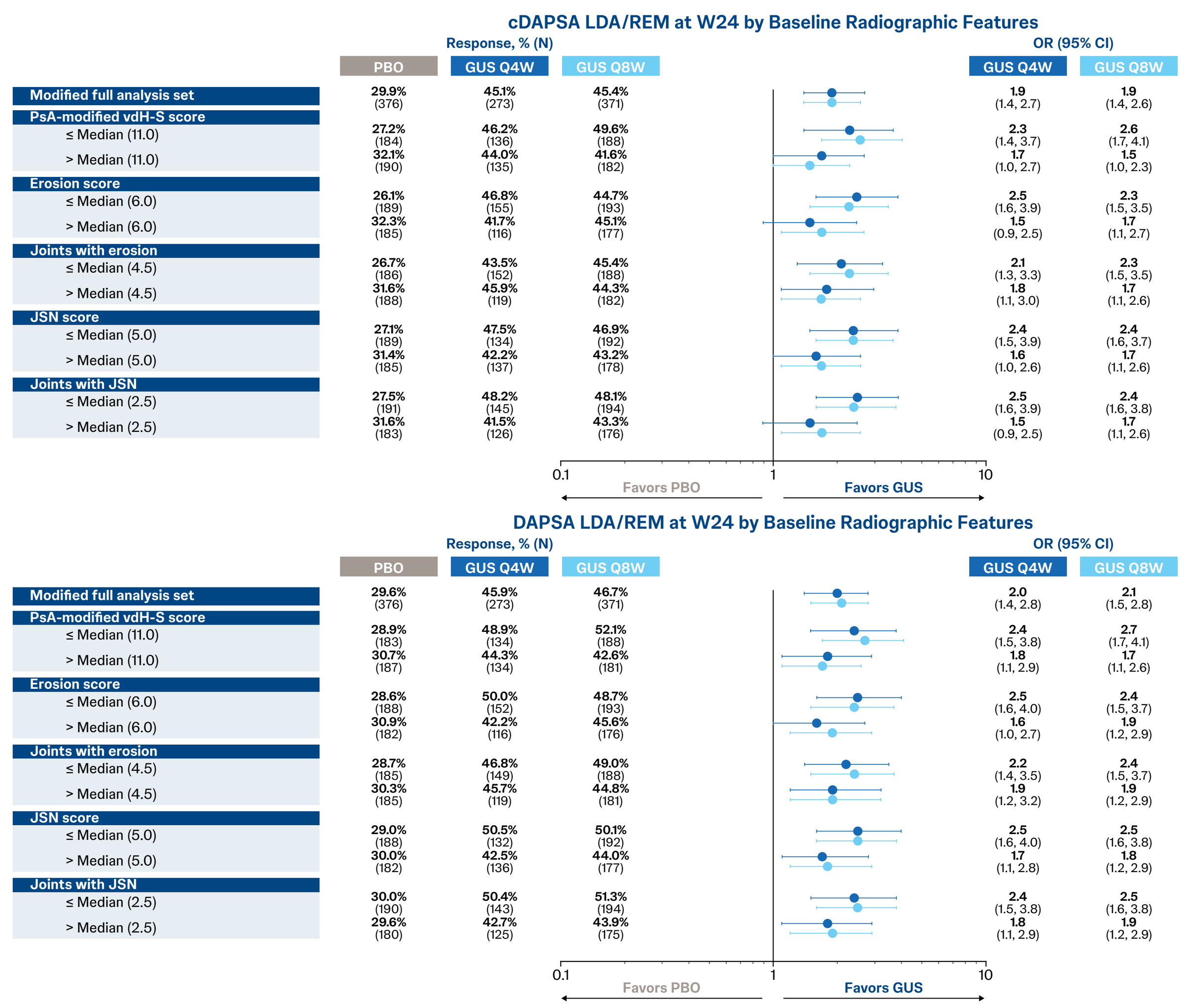




CI=Confidence interval, OR=Odds ratio.

At W24, GUS treatment effect in achieving cDAPSA and DAPSA LDA/REM was consistent across subgroups defined by baseline radiographic features

- Q4W/Q8W-treated pts were ~2- to 2.5-times more likely to achieve cDAPSA and DAPSA response than PBO-treated pts



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