Structural Damage Progression and Clinical Response to Guselkumab in Participants With Active and Erosive Psoriatic Arthritis: Post Hoc Analysis at Week 24 of the Phase 3b, Randomized, Double-Blind, Placebo-Controlled APEX Study

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Conflicts of Interest

CTR: is a consultant for AbbVie, Eli Lilly, Johnson & Johnson, Novartis, and UCB; receives grant/research support from Johnson & Johnson, Novartis, and UCB.

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PJM: is a consultant for AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB; receives grant/research support from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; is a paid speaker for AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB.

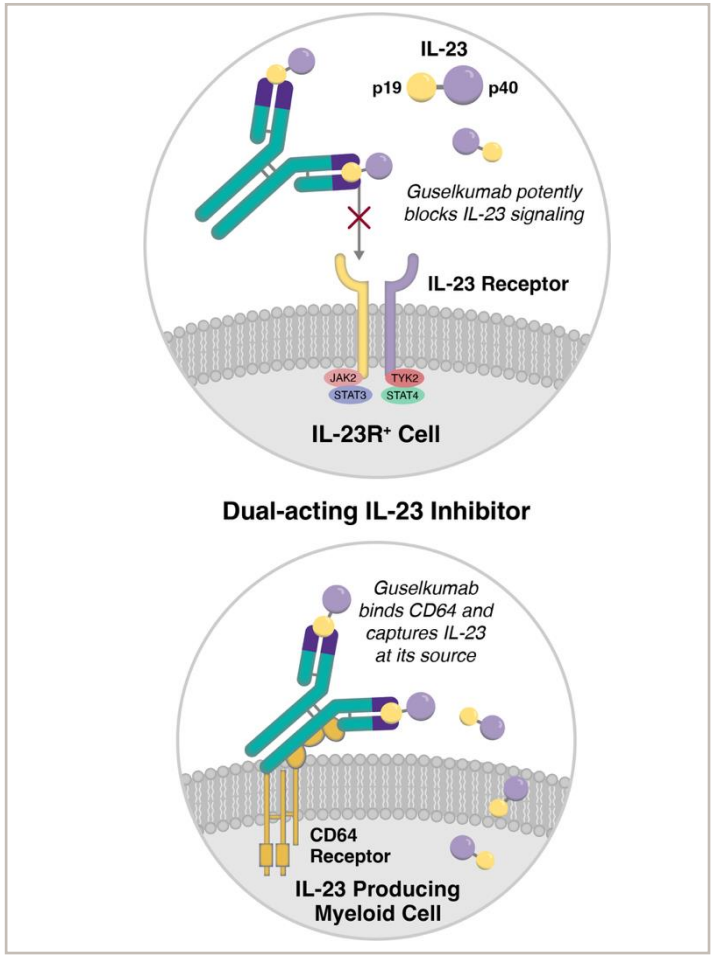
Background & Objective

Guselkumab inhibition of structural damage progression in PsA

- Uncontrolled inflammation in psoriatic arthritis (PsA) increases the risk of structural joint damage, which is associated with long-term disability and greater impairments in physical function and health-related quality of life
- Guselkumab (GUS) is a fully human, dual-acting,[1] monoclonal antibody that selectively inhibits the interleukin (IL)-23p19 subunit
- In the phase 3b APEX study, GUS demonstrated efficacy across PsA domains and significantly inhibited structural damage progression compared with placebo (PBO) in participants (pts) with active and erosive PsA at Week (W) 24[2]

Objective

- **Evaluate the relationship between clinical response status and structural damage progression at W24 among GUS-randomized pts in APEX**

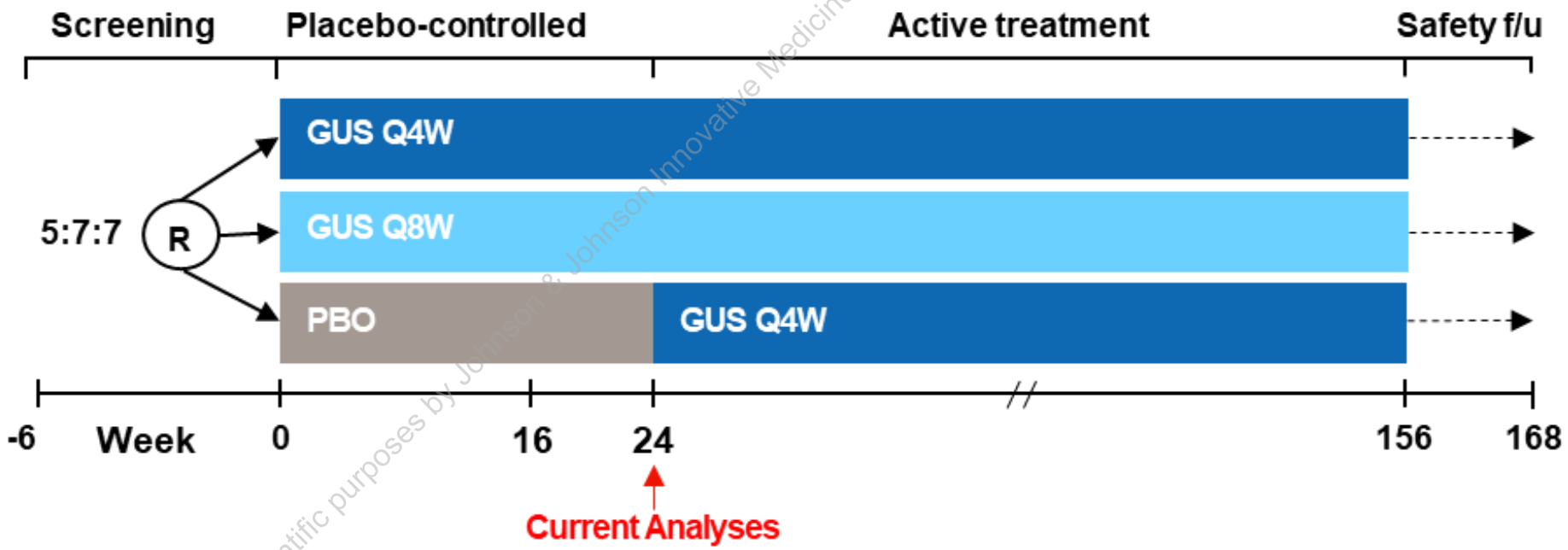


1. Sachen KL, et al. *Front Immunol*. 2025;16:1532852. 2. Mease PJ. *Ann Rheum Dis*. 2025;84:1983-4.

APEX Study Design and Methods

Key inclusion criteria:

- ✓ Biologic-naïve
- ✓ ≥18 years
- ✓ Active PsA ≥6 months (despite prior csDMARDs, apremilast, NSAIDs); CASPAR criteria met
- ✓ ≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL
- ✓ **≥2 joints with erosions** on baseline radiographs of hands/foots (centrally read)
- ✓ Active plaque PsO (≥1 PsO plaque ≥2 cm and/or nail PsO)



CASPAR=CIASsification criteria for Psoriatic Arthritis, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease modifying antirheumatic drugs, **F/U**=follow-up, **NSAIDs**=nonsteroidal anti-inflammatory drug, **PsO**=psoriasis, **Q4W/Q8W**=every 4/8 weeks, **R**=randomization, **SJC**=swollen joint count, **TJC**=tender joint count.

APEX Methods

Endpoints assessed at W24

LSM change from baseline in PsA-modified vdH-S score by clinical response status at W24, as assessed by:

- ACR20/50/70
- DAPSA LDA ≤14/REM ≤4[1]
- cDAPSA LDA ≤13/REM ≤4[1,2]
- MDA[3]
- HAQ-DI ≤0.5
- Pt-Reported Pain ≤15 mm on VAS

Statistical Analyses

- Pts were considered clinical nonresponders if they initiated/increased oral corticosteroids, csDMARDs, initiated protocol-prohibited PsA therapies, or discontinued study agent for any reason other than ND/MD
  - Data affected by ND/MD were not used
- For change in vdH-S scores, data affected by ND/MD were not used; missing data were imputed using MI

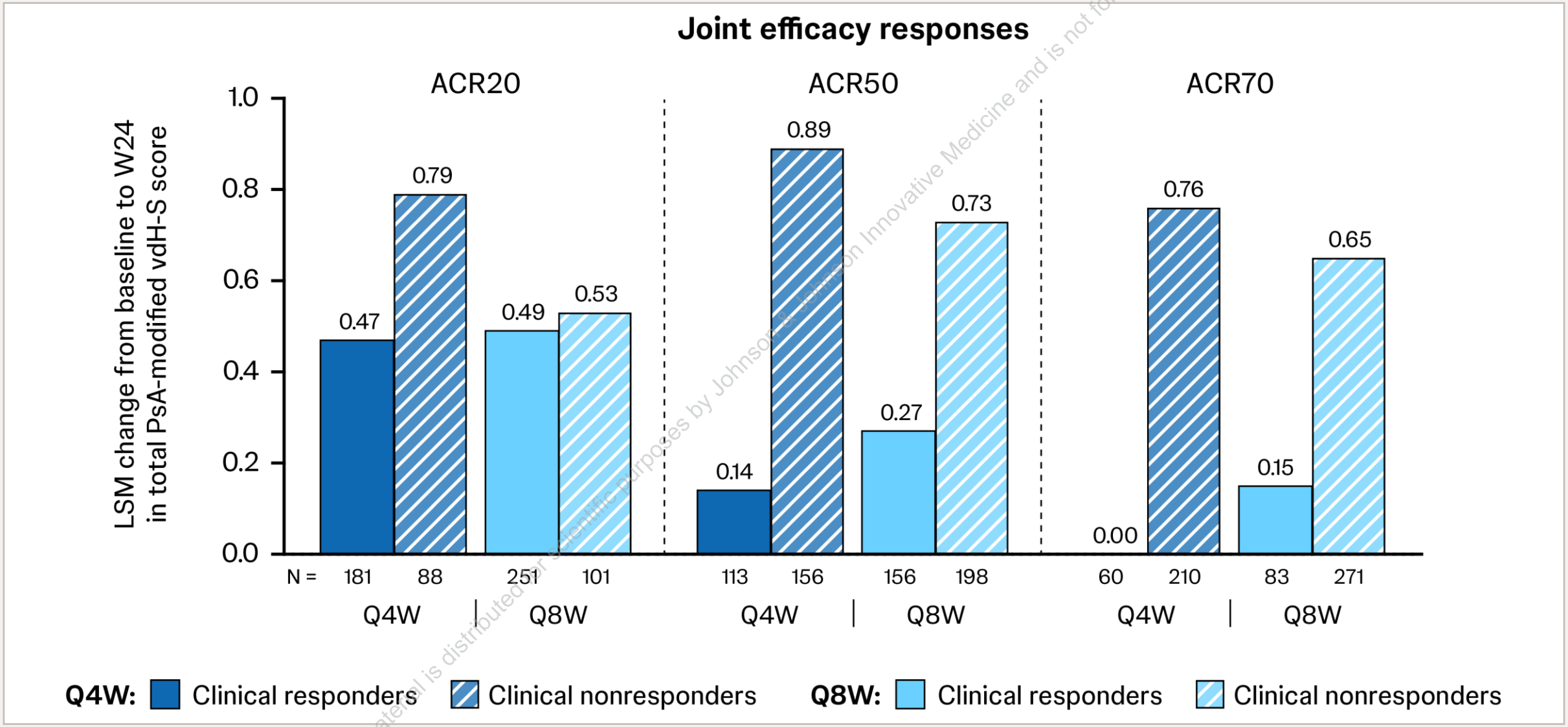
1. Schoels M. *Ann Rheum Dis*. 2016;75:811-8. 2. Helliwell PS. *Arthritis Care Res*. 2014;66:749-756. 3. Coates LC. *J Rheumatol*. 2016;43:371-375. **ACR20/50/70**=≥20%/≥50%/≥70% improvement in the American College of Rheumatology criteria, **cDAPSA**=clinical Disease Activity Index for PsA, **DAPSA**=Disease Activity Index for PsA, **HAQ-DI**=Health Assessment Questionnaire-Disability Index, **LDA**=low disease activity, **LSM**=least squares mean, **MD**=major disruption (Ukraine/Russia Crisis), **MDA**=minimal disease activity, **MI**=multiple imputation, **ND**=natural disaster (COVID-19 site access restrictions), **REM**=remission, **VAS**=visual analog scale, **vdH-S**=van der Heijde-Sharp.

Baseline characteristics were well balanced across treatment groups

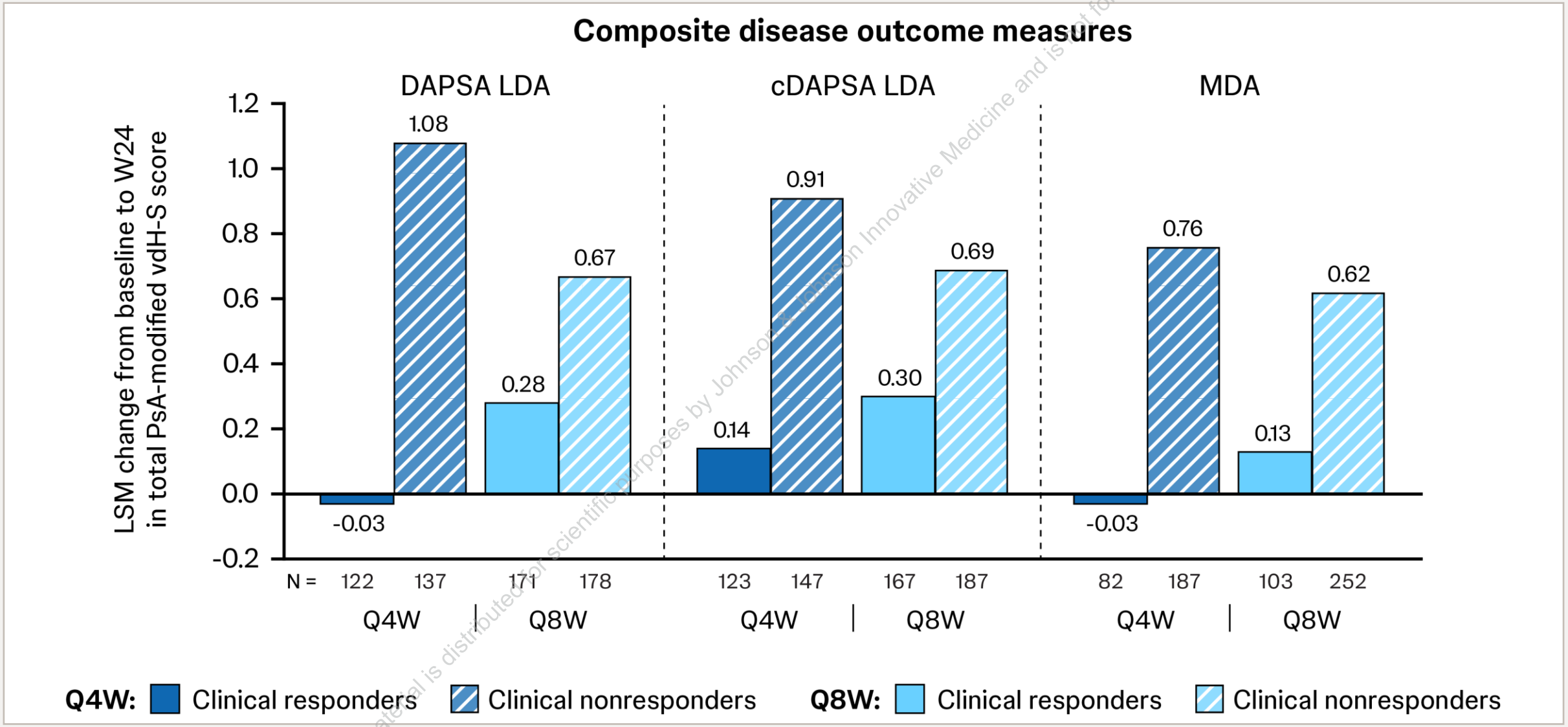
	GUS Q4W N=273	GUS Q8W N=371	PBO N=376	Total N=1020
Baseline Demographics				
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)	53.0 (13.0)
Male	55%	54%	57%	55%
Weight, kg	85.6 (20.1)	83.2 (17.4)	83.1 (18.2)	83.8 (18.5)
BMI, kg/m^2	29.4 (6.0)	29.0 (5.6)	28.9 (5.7)	29.1 (5.7)
PsA Characteristics				
PsA disease duration, years	7.5 (7.1)	7.2 (7.6)	7.2 (6.9)	7.3 (7.2)
SJC [0-66]	11.6 (9.4)	12.1 (8.5)	11.8 (8.9)	11.9 (8.9)
TJC [0-68]	21.2 (14.6)	20.6 (13.4)	20.5 (13.9)	20.7 (13.9)
Pt-reported pain (VAS; 0-100 mm)	59.2 (22.2)	59.3 (21.0)	59.4 (20.6)	59.3 (21.1)
HAQ-DI [0-3]	1.2 (0.7)	1.2 (0.6)	1.2 (0.7)	1.2 (0.7)
DAPSA	46.4 (24.3)	46.1 (22.1)	46.0 (22.8)	46.1 (22.9)
cDAPSA	44.7 (23.5)	44.6 (21.6)	44.3 (22.3)	44.5 (22.4)
CRP, mg/dL	1.7 (2.9)	1.5 (2.0)	1.7 (2.5)	1.6 (2.5)
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
Radiographic Characteristics				
PsA-modified vdH-S score [0-528]	27.7 (47.6)	26.7 (43.4)	26.8 (42.2)	27.0 (44.1)
Erosion score [0-320]	13.7 (24.3)	13.4 (21.9)	13.4 (20.7)	13.5 (22.1)
JSN score [0-208]	14.0 (24.2)	13.3 (22.8)	13.4 (22.4)	13.5 (23.0)

Values are reported as mean (SD) for pts with nonmissing data unless otherwise noted. **BMI**=body mass index, **DSS**=Dactylitis Severity Score, **JSN**=joint space narrowing, **LEI**=Leeds Enthesitis Index, **SD**=standard deviation.

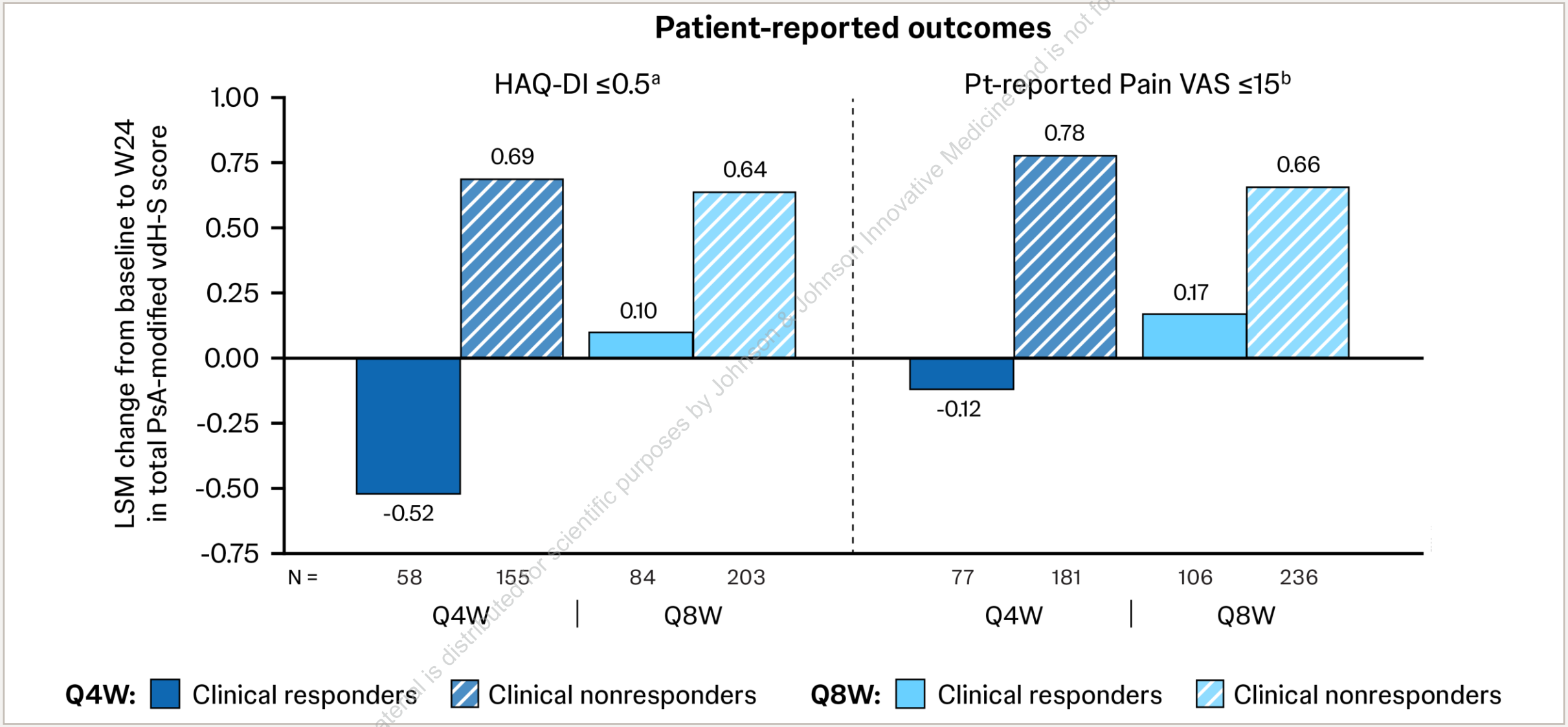
At W24, GUS-randomized pts who achieved response in joint efficacy measures had numerically lower levels of structural damage progression



At W24, GUS-randomized pts who achieved low or minimal disease activity by composite indices had numerically lower levels of structural damage progression



At Week 24, GUS-randomized pts who achieved normalized physical function and minimal pain had numerically lower levels of structural damage progression



[a]Among pts who had HAQ-DI score >0.5 at baseline. [b] Among pts who had pt-reported pain VAS >15 at baseline.

Key Takeaways

- In the APEX population of pts with active and erosive PsA, GUS treatment significantly inhibited structural damage progression vs PBO at W24[2]

- Among pts receiving GUS Q4W and Q8W, achievement of clinical response was associated with less structural damage progression at W24 across PsA domains:
  - Joint efficacy: ACR20/50/70
  - Composite disease measures: DAPSA LDA, cDAPSA LDA, MDA
  - Pt-reported outcomes: normalized physical function and minimal pain

- Additional analyses beyond W24 will determine the maintenance of structural damage inhibition among clinical responders and continue assessment of clinical nonresponders

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