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

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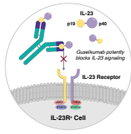
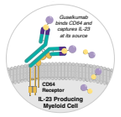
Background

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 CD64 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]





APEX (NCT04882098)

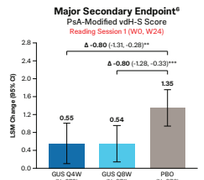
- An ongoing phase 3b, randomized, double-blind, placebo (PBO)-controlled study aimed to further evaluate GUS effects on clinical and radiographic progression outcomes in participants (pts) with active and erosive PsA

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 **s=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.*

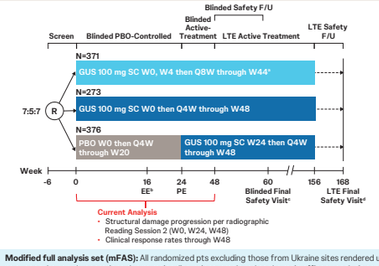
Objectives

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, NSAIDs); CASPAR criteria met
  - ≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL
- **≥2 erosive joints on hand/foot radiographs**
- Active plaque PsO (≥1 PsO plaque ≥2 cm and/or nail PsO)



- **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

**PBO SC W8 then Q8W through W48 administered to maintain blinding. †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. ‡Final safety visit for those who do not enter LTE. §Final safety visit for those who entered LTE. CASPAR=ClASification criteria for Psoriatic ARthritis; CRP=C-reactive protein; csDMARDs=conventional synthetic disease-modifying antirheumatic drugs; EE=early escape; F&I=follow-up; GUS=guselkumab; LTE=long-term extension; NSAIDs=nonsteroidal anti-inflammatory drugs; 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; 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	• Adverse events (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; 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

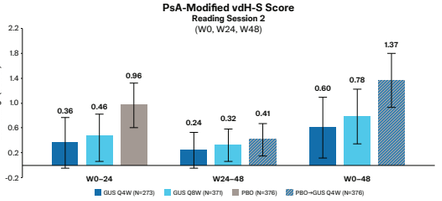
Baseline Characteristics	GUS Q4W (N=273)	GUS Q8W (N=371)	PBO (N=376)	Total (N=1020)
Demographics				
Age, years	52.2 (13.2)	53.2 (12.9)	53.5 (13.0)	53.0 (13.0)
Male	55%	54%	57%	55%
Weight, kg	85.6 (20.1)	83.2 (17.4)	83.1 (18.2)	83.8 (18.5)
BMI, kg/m²	29.4 (6.0)	29.0 (5.6)	28.9 (5.7)	29.1 (5.7)
Disease 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, W024 radiographs were not re-read during reading session 2; W024 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; 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.

Results

Inhibition of structural damage progression with GUS was durable through W48

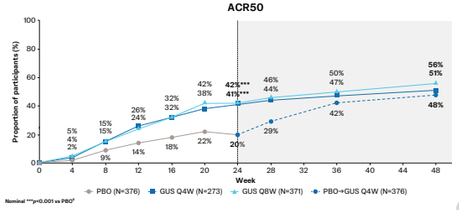
- Treatment with GUS from W24–48 reduced the rate of radiographic progression among PBO-randomized pts



For pts with missing W48 radiographs, W024 radiographs were not re-read during reading session 2; W024 data from reading session 1 was used if available. The same readers were used for reading session 1 and 2. Data shown are the average proportion of mFAS pts with structural damage progression by the Adjusted Treatment Policy Estimand irrespective of background PsA medication or adherence to study intervention, in situations when NDMD occurred; observed data collected after NDMD was not used; data for visits after NDMD 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 square means; mFAS=modified full analysis set; MI=multiple imputation; NDMD=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

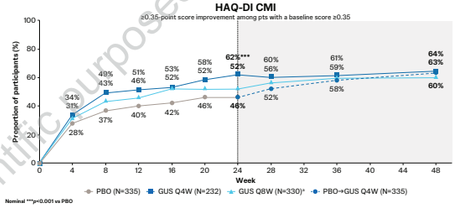
- PBO-randomized pts achieved comparable ACR50 rates after starting GUS



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 NDMD 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 NDMD 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; NDMD=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

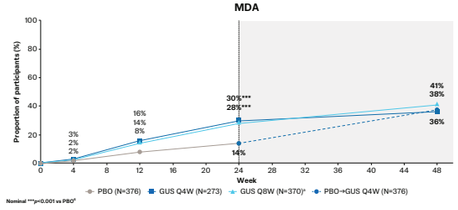
- PBO-randomized pts achieved comparable HAQ-DI CMI rates after starting GUS



*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 NDMD 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 NDMD was handled within the model. Other missing data were imputed using NRI. *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; NDMD=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



*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 NDMD 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 NDMD was handled within the model. Other missing data were imputed using NRI. *N=376 from W28–48. csDMARD=conventional synthetic disease modifying antirheumatic drug; GLMM=generalized linear mixed model; GUS=guselkumab; MDA=minimal disease activity; NDMD=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

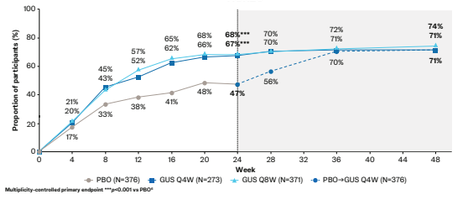
- In GUS-randomized pts, exposure-adjusted AE rates did not increase through W48

	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

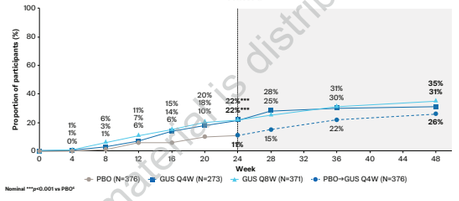
- PBO-randomized pts achieved comparable ACR20 rates after starting GUS



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 NDMD 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 NDMD 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; NDMD=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

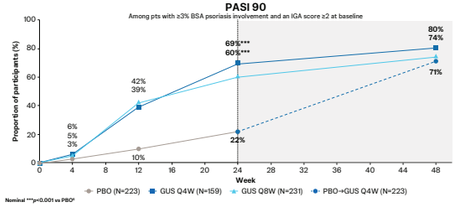
- PBO-randomized pts achieved comparable ACR70 rates after starting GUS



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 NDMD 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 NDMD 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; NDMD=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



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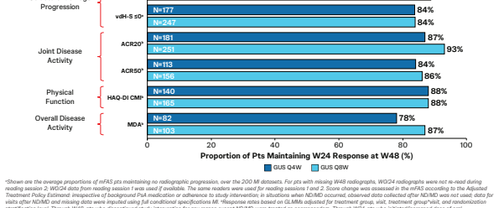
~80–90% of GUS-randomized pts maintained W24 responses through W48



**Shown are the average proportions of mFAS pts maintaining no radiographic progression, over the 200 MI datasets. For pts with missing W48 radiographs, W024 radiographs were not re-read during reading session 2; W024 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 NDMD occurred; observed data collected after NDMD was not used; data for visits after NDMD and missing data were imputed using full conditional specifications MI. †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 NDMD 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 NDMD 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; NDMD=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.*

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

PRESENTED AT: 4th Annual Maui Derm Caribbean 2026; San Juan, Puerto Rico; January 7–10, 2026. **REFERENCES:** 1. Gladman DD, et al. *Q J Med.* 1997;62:127–141. 2. Ritchlin CT, *J Rheumatol.* 2008;35:1659–1437. 3. van der Heijde D, et al. *Arthritis Res Ther.* 2020;22:88. 4. Sachen KL, et al. *Front Immunol* 2025;16:1532852. 5. Johnson & Johnson. *US FDA* website. Revised March 2025. 6. Mease PJ, et al. *Ann Rheum Dis.* September 16, 2025; doi:10.1016/j.ard.2025.08.006. **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 reformatting was done by Ganesh Karpe 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, Innagene, 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, Eli Lilly, Johnson & Johnson, 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, and SDC:** 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 6th Inflammatory Skin Disease Summit (ISDS); New York, NY, USA; November 12–15, 2025.