12-Month Persistence and Multi-Domain Effectiveness of Guselkumab in Adults With Active Psoriatic Arthritis: Real-World Data From the PPD CorEvitas Psoriatic Arthritis/Spondyloarthritis Registry

Philip J. Mease,[1,2] Alexis Ogdie,[3] John Tesser,[4] Timothy P. Fitzgerald,[5] Elizabeth Adamson,[5] Soumya D. Chakravarty,[5,6] Robert R. McLean,[7] Taylor S. Blachley,[7] Melissa Eliot,[7] Skyler S. Peterson,[7] Aaron Broadwell,[8] Kurt Oelke,[9] Arthur Kavanaugh,[10] Joseph F. Merola[11]

[1]Rheumatology Research, Providence Swedish Medical Centre, Seattle, WA, USA; [2]University of Washington School of Medicine, Seattle, WA, USA; [3]University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [4]Arizona Arthritis & Rheumatology Associates, P.C., Phoenix, AZ, USA; [5]Johnson & Johnson, Horsham, PA, USA; [6]Drexel University College of Medicine, Philadelphia, PA, USA; [7]Thermo Fisher Scientific, Waltham, MA, USA; [8]Rheumatology and Osteoporosis Specialists, Shreveport, LA, USA; [9]Rheumatic Disease Centre, Glendale, WI, USA; [10]Centre for Innovative Therapy, Division of Rheumatology, Allergy, and Immunology, University of California San Diego, La Jolla, CA, USA; [11]Department of Dermatology, and Department of Medicine, Division of Rheumatology, UT Southwestern Medical Centre and O'Donnell School of Public Health, Dallas, TX, USA

Background

Guselkumab (GUS), a fully human, dual-acting interleukin-23p19 subunit inhibitor, has demonstrated significant efficacy in treating psoriatic arthritis (PsA) in Phase 3 clinical trials[1-3]

- GUS was approved by the United States Food and Drug Administration in July 2020 for adults with active PsA (dosing regimen: GUS 100 mg subcutaneously at Week [W]0, W4, then every 8 weeks)[4]

Real-world data on GUS persistence and effectiveness are available from the prospective, multicenter, observational PPD™ CorEvitas™ PsA/Spondyloarthritis Registry of adults with rheumatologist-diagnosed active PsA[5]

In a previous analysis of CorEvitas data, persistence through 6 months (M) of on-label GUS therapy was associated with significant improvements in PsA signs and symptoms[5]

Objective

To assess real-world effectiveness and persistence of on-label GUS at 12M in participants (pts) with active PsA

Methods

CorEvitas PsA/SpA Registry

- Prospective, multicenter, observational registry of adults in the US with rheumatologist-diagnosed active PsA
- Collects data from healthcare providers and pts at the time of outpatient clinical rheumatology encounters
- This analysis included data from GUS initiators (October 12, 2017 –July 31, 2025)

Study Population

- **GUS on-label initiators**
  - CorEvitas registry pts with PsA who initiated GUS after FDA approval for active PsA (July 13, 2020) using the FDA-approved (on-label) dosing regimen (GUS 100 mg subcutaneously at W0, W4, then Q8W), either as monotherapy or in combination with a csDMARD
  - Had a valid baseline visit associated with GUS initiation and a 12M follow-up visit
- **GUS on-label initiators**
  - Pts who maintained on-label use of GUS through the 12M visit

Effectiveness Endpoints Evaluated in GUS On-Label Persisters

- **Primary outcome:** Mean change (95% CI) in cDAPSA score from baseline to 12M visit
- **Secondary outcomes** (in order of multiplicity-controlled testing):
  Mean (95% CI) change from baseline to 12M visit in:
  - Physician Global Assessment of arthritis+PsO (0–100)
  - Patient-reported pain (Patient Pain; 0–100)
  - % BSA with PsO (0–100%)
- For primary and secondary outcomes, paired t-tests were used to determine statistical significance (α=0.05)
  - To control for multiplicity, a fixed-sequence statistical strategy was used to test primary and secondary outcomes in a predefined order, all at the same significance level (α=0.05)
- **Other outcomes** (not multiplicity-controlled) included:
  - Proportions of pts achieving cDAPSA LDA/REM among pts with moderate or high disease activity at baseline

BSA=body surface area, cDAPSA=clinical Disease Activity Index for PsA, CI=confidence interval, csDMARD=conventional synthetic disease modifying antirheumatic drug, FDA=Food and Drug Administration, GUS=guselkumab, LDA=low disease activity, M=month, PsA=psoriatic arthritis, PsO=psoriasis, pts=participants, Q8W=every 8 weeks, REM=remission, SpA=spondyloarthritis, US=United States, W=week.

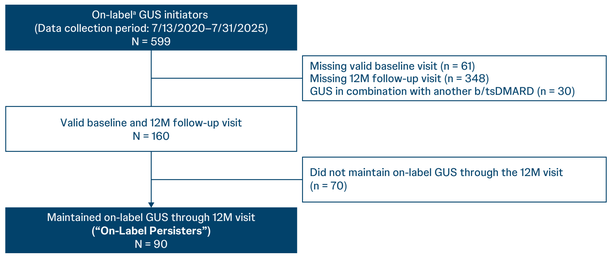
Key Takeaways

In this real-world population of pts with longstanding, active, and largely treatment-refractory PsA:

- **GUS on-label persisters demonstrated statistically significant improvements in clinical measures of joint and skin disease activity and patient-reported outcomes at 12M**
- **50% of pts with moderate/high disease activity at baseline achieved low disease activity/remission at 12M with on-label, persistent GUS therapy**

Results

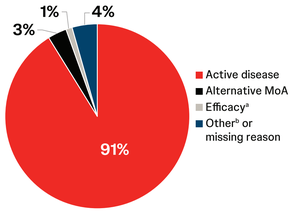
Of 160 on-label GUS initiators[a] with eligible baseline and 12M visits, 56% maintained on-label use through 12M



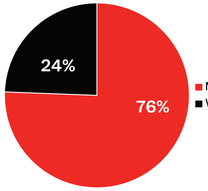
*[a]CorEvitas PsA/SpA Registry pts who initiated GUS after US FDA approval (7/13/2020) using the FDA-approved dosing regimen (100 mg at W0, W4, then Q8W). **bDMARD**=biologic disease modifying antirheumatic drug, **FDA**=Food and Drug Administration, **GUS**=guselkumab, **M**=month, **PsA**=psoriatic arthritis, **pts**=participants, **Q8W**=every 8 weeks, **SpA**=spondyloarthritis, **tsDMARD**=targeted synthetic disease modifying antirheumatic drug, **US**=United States, **W**=week.*

The majority of GUS on-label persisters initiated GUS due to active disease and as monotherapy

91% initiated GUS due to active disease



76% initiated GUS as monotherapy



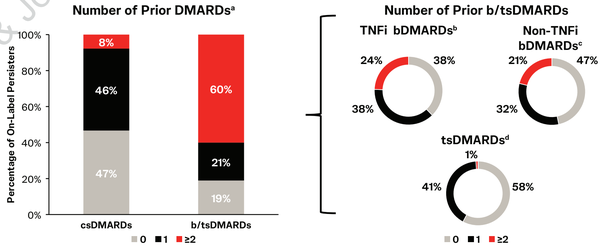
*Percentages may not add to 100% due to rounding. [a]Efficacy reasons defined as inadequate initial response or failure to maintain initial response. [b]Other reasons defined as fear of future side effect, temporary interruption, pt preference, to improve compliance, to improve tolerability, frequency of administration, route of administration, and pt doing well. No pts cited safety/intolerability (serious side effect, minor side effect) or insurance (co payat cost, denied by the insurance) as their reason for initiating GUS. The sum of all reason categories may total more than 100% given that pts could provide up to 3 reasons (3 pts provided multiple reasons). [c]Defined as any csDMARD confirmed to be initiated as of GUS baseline visit. Concomitant therapy may have started prior to or concurrently with GUS initiation. **csDMARD**=conventional synthetic disease modifying antirheumatic drug, **GUS**=guselkumab, **MoA**=mechanism of action, **pts**=participants.*

Baseline characteristics of GUS on-label persisters

Baseline Characteristics		GUS On-Label Persisters (N=90)
Demographics		
	Age, yrs	51.3 (13.5)
	Female	58%
	Race, White	86%
	Ethnicity, non-Hispanic	85%
	BMI,[a] kg/m^2	32.1 (6.9)
	Normal/underweight, <25 kg/m^2	11%
	Overweight, ≥25 to <30 kg/m^2	29%
	Obese, ≥30 kg/m^2	60%
Related Conditions[b]		
	IBD[c]	7%
	Crohn's disease	0%
	Ulcerative Colitis	1%
	Uveitis	1%
PsA Characteristics		
	Yrs since PsA diagnosis	7.0 (7.6)
	Median (IQR)	4.0 (1.0, 11.0)
	History of PsO[d]	97%
	% BSA, 0–100[e,f]	7.1 (12.0)
	Median (IQR)	2.0 (1.0, 8.0)
	Axial PsA[g]	44%
Disease Activity		
	Tender joint count, 0–68[h]	7.4 (12.2)
	Swollen joint count, 0–66[h]	3.0 (6.9)
	Physician Global Assessment of arthritis+PsO, VAS 0–100[e,i]	38.4 (23.8)
	Physician Global Assessment of arthritis, VAS 0–100[i]	32.5 (23.2)
	Investigator's Global Assessment of PsO[e,k]	
	Clear/Almost Clear	14%/12%
	Mild/Moderate/Severe	40%/25%/8%
	cDAPSA[e]	21.5 (18.2)
	REM, ≤4	5%
	LDA, >4 to ≤13	25%
	Moderate, >13 to ≤27	51%
	High, >27	19%
	MDA[l]	15%
	VLDA[e]	2%
PRO Measures		
	Patient Pain, VAS 0–100[e]	55.7 (23.8)
	Patient Global Assessment of arthritis+PsO, VAS 0–100[e,i]	50.8 (23.6)
	HAQ-DI, 0–3[e]	0.9 (0.6)

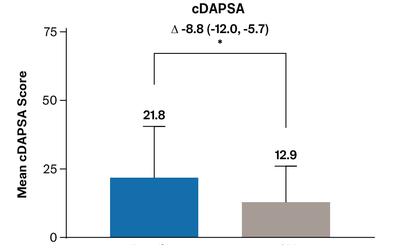
*Data shown are mean (SD) unless otherwise noted. Percentages may not add to 100% due to rounding. [a]N=89. [b]Related conditions are considered at all points up to and including the baseline visit and therefore represent any past or current presence of these conditions. [c]Includes Crohn's disease, ulcerative colitis, possible IBD, and other IBD. [d]Evidence of current PsO or a personal history of PsO does not include family history of PsO. [e]Limited to pts with history of PsO. [f]N=82. [g]Axial involvement defined by physician-reported PsA diagnosis and either (1) diagnosis of axial SpA or ankylosing spondylitis, (2) physician indicated spinal involvement or completed any of the mobility measurements (includes occiput-to-wall distance, lateral lumbar flexion and lumbar flexion [Schöber]), or (3) any of the following criteria for diagnosing axial SpA: inflammatory back pain; ≥3 months back pain (age of onset <45 years); low back pain and stiffness for >3 months which improves with exercise and is not relieved by rest; limitation of motion of the lumbar spine in both the sagittal and frontal planes; active (acute) inflammation on MRI highly suggestive of sacroiliitis associated with SpA; and sacroiliitis grade ≥2 bilaterally or grade 3–4 unilaterally by radiograph. [h]N=85. [i]N=84. [j]N=87. [k]N=83. [l]N=86. **BMI**=body mass index, **BSA**=body surface area, **cDAPSA**=clinical Disease Activity Index for PsA, **GUS**=guselkumab, **HAQ-DI**=Health Assessment Questionnaire Disability Index, **IBD**=inflammatory bowel disease, **IQR**=interquartile range, **LDA**=low disease activity, **MDA**=minimal disease activity, **MRI**=magnetic resonance imaging, **PRO**=patient-reported outcome, **PsA**=psoriatic arthritis, **PsO**=psoriasis, **pts**=participants, **REM**=remission, **SD**=standard deviation, **SpA**=spondyloarthritis, **VAS**=visual analogue scale, **VLDA**=very low disease activity, **yrs**=years.*

The majority of GUS on-label persisters were b/tsDMARD-experienced, and 60% had received ≥2 prior b/tsDMARDs



*Percentages may not add to 100% due to rounding. Counts do not include pt's current therapy. [a]Pts could have received prior csDMARDs and/or b/tsDMARDs. [b]TNFi bDMARDs: adalimumab, certolizumab pegol, etanercept, etanercept-szzs, golimumab, infliximab, infliximab-dyyb. [c]Non-TNFi bDMARDs: abatacept, anakinra, irekizumab, rituximab, secukinumab, tocilizumab, ustekinumab, risankizumab-rzaa. [d]tsDMARDs: apremilast, tofacitinib, upadacitinib. **bDMARD**=biologic DMARDs, **csDMARDs**=conventional synthetic DMARDs, **DMARDs**=disease modifying antirheumatic drugs, **GUS**=guselkumab, **pts**=participants, **TNFi**=tumor necrosis factor inhibitor, **tsDMARD**=targeted synthetic DMARDs.*

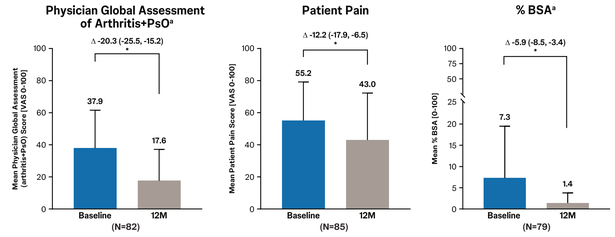
Among GUS on-label persisters, significant mean improvements from baseline to 12M were observed in the primary endpoint: cDAPSA



***p<0.001, based on paired t-tests.**

*Δ represents mean change (95% CI) measured as 12M minus baseline. Error bars represent SDs. **cDAPSA**=clinical Disease Activity Index for PsA, **CI**=confidence interval, **GUS**=guselkumab, **M**=month, **SD**=standard deviation.*

Among GUS on-label persisters, significant mean improvements from baseline to 12M were observed in all major secondary endpoints: Physician Global Assessment, Patient Pain, and % BSA



***p<0.001, based on paired t-tests.**

*Δ represents mean change (95% CI) measured as 12M minus baseline. Error bars represent SDs. [a]Evaluated in pts with history of PsO. **BSA**=body surface area, **CI**=confidence interval, **GUS**=guselkumab, **M**=month, **PsO**=psoriasis, **pts**=participants, **SD**=standard deviation, **VAS**=visual analogue scale.*

Half of GUS on-label persisters with moderate/high disease activity at baseline achieved LDA/REM at 12M

cDAPSA at GUS initiation[a]	cDAPSA LDA/REM at 12M[a]
Moderate/High (n=54)	**27/54 (50%)**
Moderate (n=38)	21/38 (55%)
High (n=16)	6/16 (38%)

*[a]REM: cDAPSA ≤4; LDA: cDAPSA >4 to ≤13; moderate: cDAPSA >13 to ≤27; high: cDAPSA >27. **cDAPSA**=clinical Disease Activity Index for PsA, **GUS**=guselkumab, **LDA**=low disease activity, **M**=month, **PsA**=psoriatic arthritis, **REM**=remission.*

Strengths and Limitations

Strengths

- Observational design of the CorEvitas PsA/SpA Registry captures real-world practice patterns and data on pts seen in routine clinical practice across several regions of the US
- More representative of the US PsA patient population than clinical trial populations
- Standardized data collection instruments and methods across all sites
- Primary and major secondary endpoints were controlled for multiplicity

Limitations

- Modest sample size
- May not be generalizable to regions outside the US
- Pt selection based on a 12M follow-up period requirement and further restricting to those who persist at follow-up may introduce time and selection biases

M=month, PsA=psoriatic arthritis, pt=participant, SpA=spondyloarthritis, US=United States.

Accepted as abstract publication only at EULAR 2026; June 3–6, 2026; London, UK. Poster intended for Johnson & Johnson Medical Connect only. **REFERENCES: 1.** Deodhar A, et al. *Lancet.* 2020;395:1115–1125. **2.** Mease PJ, et al. *Lancet.* 2020;395:1126–1136. **3.** Mease PJ, et al. *Ann Rheum Dis.* 2025;84:1983–1994. **4.** Tremfya: Package insert. Horsham, PA: Janssen Biotech, Inc.; 2025. **5.** Mease PJ, et al. *Rheumatol Ther.* 2023;10:1479–501. **ACKNOWLEDGMENTS:** Medical writing support was provided by Erin Bekes, PhD, of Certara under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298–1304) and funded by Johnson & Johnson. Layout design and formatting for this encore presentation were provided by Ganesh Karpe (SIRO Medical Writing Pvt. Ltd., India). This study is sponsored by the PPD™ Clinical research business of Thermo Fisher Scientific and funded by Johnson & Johnson. PPD™ CorEvitas™ Clinical Registries has been supported through contracted subscriptions in the last two years by AbbVie, Amgen, Inc., Arena, Boehringer Ingelheim, Bristol Myers Squibb, Chugai, Eli Lilly and Company, Genentech, GSK, Johnson & Johnson, LEO Pharma, Novartis, Ortho Dermatologics, Pfizer, Inc., Sun Pharmaceutical Industries Ltd., and UCB S.A. **DISCLOSURES: PJM:** Consultant for AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Imagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, Sun Pharma, Takeda, and UCB; grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, and UCB. **AO:** Consulting fees from AbbVie, Amgen, Bristol Myers Squibb, CorEvitas, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Merck, Novartis, Oruka, Pfizer, Spyre, Takeda, and UCB; advisory board fees from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Novartis, Pfizer, and UCB; grants from AbbVie, Pfizer, and Novartis (all to University of Pennsylvania) and Amgen (to Forward/NDB); and other funding from NIAMS, Rheumatology Research Foundation, National Psoriasis Foundation, and University of Pennsylvania. **JT:** Advisory boards/consultant for AbbVie, AstraZeneca, GSK, Immpact Bio, Johnson & Johnson, Novartis, Sanofi-Genzyme, Setpoint, and UCB; speaker bureaus for: AbbVie, Aqtual, Argenix, AstraZeneca, Bendcare, Biogen, GSK, Johnson & Johnson, Pfizer, and Sanofi/Genzyme. **TPF, EA,** and **SDC:** Employees of Johnson & Johnson; may own stock/stock options in Johnson & Johnson. **RRM** and **TSB:** Employee of PPD CorEvitas Clinical Registries, Thermo Fisher Scientific and stockholder of Thermo Fisher Scientific. **ME** and **SSP:** Employees of PPD CorEvitas Clinical Registries, Thermo Fisher Scientific. **AB:** Consulting fees from AbbVie, Amgen, Aurinia, Celgene, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and Sanofi/; speakers fees from AbbVie, Amgen, Eli Lilly, Horizon, Johnson & Johnson, Mallinckrodt, Novartis, Pfizer, Radius, Sanofi/Regeneron, and UCB. **KO:** Speaker's bureau for AbbVie, AstraZeneca, BMS, Johnson & Johnson, and UCB. **AK:** Consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Pfizer, and UCB; **JFM:** Consultant and/or investigator for AbbVie, Amgen, AstraZeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Johnson & Johnson, Moonlake, Novartis, Oruka, Pfizer, Regeneron, Sanofi, Sun Pharma, and UCB. **PREVIOUSLY PRESENTED AT:** Rheumatology Winter Clinical Symposium (RWCS); February 11–14, 2026; Maui, HI, USA.