Real-World On-Label Treatment Persistence Through 24 Months in Biologic-Naïve and Biologic-Experienced Patients With Psoriatic Arthritis: Comparison of Guselkumab Versus Targeted Synthetic Disease-Modifying Antirheumatic Drugs

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Key Takeaways

- **First real-world claims analysis comparing on-label persistence over 24 months in pts with active PsA initiating GUS vs tsDMARDs, including bio-naïve and bio-experienced pts**
- **Pts initiating GUS demonstrated significantly higher short-term and long-term on-label persistence than those initiating tsDMARDs in the overall, bio-naïve, and bio-experienced groups**
- **Findings were consistent for subgroup comparisons (GUS vs apremilast and vs JAKi) and sensitivity analyses**

Background

Subcutaneous (SC) guselkumab (GUS; fully human, dual-acting selective interleukin (IL)-23p19-subunit inhibitor) and oral targeted synthetic disease-modifying antirheumatic drugs (tsDMARDs; apremilast, tofacitinib, upadacitinib) are approved for patients (pts) with active psoriatic arthritis (PsA)[1–4]

- US Food and Drug Administration (FDA)-approved dosing regimen[1] (on-label): GUS 100 mg at Week 0, Week 4, then every 8 weeks[1]

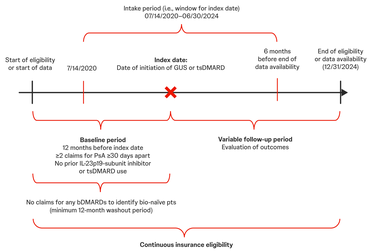
Persistence on therapy is key to maintaining long-term disease control in active PsA. However, real-world comparisons of long-term on-label persistence between pts receiving GUS and tsDMARDs are lacking

Objectives

This study used health plan claims data to compare on-label treatment persistence in pts with PsA newly initiating GUS or a tsDMARD (apremilast; Janus kinase inhibitors [JAKi; tofacitinib, upadacitinib]), including biologic-naïve (bio-naïve) and biologic-experienced (bio-experienced), through 24 months

Methods

IQVIA PharMetrics® Plus database (07/14/2019–12/31/2024) Study Design



GUS=guselkumab; **IL**=interleukin; **PsA**=psoriatic arthritis; **pts**=patients; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

Statistical Analyses

- **Baseline demographic and disease characteristics (12 months pre-index):**
  - Balanced between the GUS and tsDMARDs separately for the overall, bio-naïve, and bio-experienced populations using propensity score-weighting (overlap weights)
- **On-label persistence up to 24 months post-index:**
  - No treatment discontinuation or dose modification relative to FDA-approved labeling
  - Proportion of pts determined using **weighted** KM curves
  - GUS vs tsDMARDs comparison using **weighted** Cox proportional hazard models
- **Subgroups:**
  - Balancing and on-label persistence analyses for overall population replicated for GUS vs tsDMARD subgroups (i.e., apremilast, JAKi)

Days between administration or refill[a]	GUS	Apremilast	Upadacitinib	Tofacitinib
Primary analysis				
2x[1–4]	112 days	60 days	60 days	60 days
Sensitivity analyses				
1x[1–4]	56 days	30 days	30 days	30 days
Fixed gap	112 days	112 days	112 days	112 days

[a]Primary analysis was conducted based on 2x duration of time between administration per label. Sensitivity analyses were conducted based on 1x duration of time between administration per label as well as a fixed discontinuation gap of 112 days. **GUS**=guselkumab; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

Censoring and Imputations

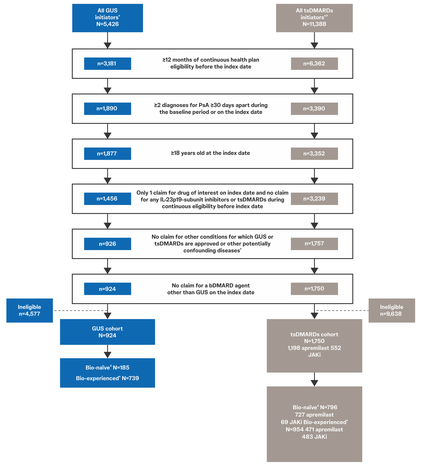
Censoring: On earliest of first off-label claim or last day of index agent supply preceding end of follow-up period if discontinuation was not observed

Days of supply imputation rule	GUS	Apremilast	Upadacitinib	Tofacitinib
Medical Claims[1–4]				
1st claim	28 days	Not found in medical claims[b]	Not found in medical claims[b]	Not found in medical claims[b]
2nd + claims	56 days	Not found in medical claims[b]	Not found in medical claims[b]	Not found in medical claims[b]
Pharmacy Claims				
1st claim	28 days	28 days	28 or 30 days[d]	30 or 90 days[c]
2nd + claims	Based on time to next claim[a]	30 or 90 days[c]	28 or 30 days[d]	30 or 90 days[c]

[a]28 days if time to next claim >42 days; 56 days if time to next claim 42–70 days; 84 days if time to next claim >70 days; if there is no next claim, days of supply of the previous claim was carried forward or imputed as 90 days if the previous claim was a pharmacy claim with days of supply >90 days, and as 30 days if the previous claim was a medical claim for outpatient procedure codes for apremilast, upadacitinib, and tofacitinib; there are no medical claims and no inpatients were made. If the second or later claim was a pharmacy claim with days of supply >90 days, days of supply were imputed to 30 days. If the second or later claim was a pharmacy claim with days of supply ≤90 days, days of supply were imputed to 90 days. [b]For pharmacy claims, if days of supply were equal to 28 days, no imputation was applied and the value was retained as 28 days; if days of supply were not equal to 28 days, days of supply were imputed to 30 days. **GUS**=guselkumab.

Results

The GUS and tsDMARD cohorts, respectively, included 924 and 1,750 pts overall, 185 and 796 bio-naïve pts, and 739 and 954 bio-experienced pts



[a]1st GUS or tsDMARD claim during intake period (7/14/2020–6/30/2024). [b]The tsDMARD cohort was defined as pts with an index claim for a tsDMARD (i.e., apremilast, upadacitinib, or tofacitinib). [c]Other specific conditions include atopic dermatitis, inflammatory bowel disease, uveitis, hidradenitis suppurativa, ankylosing spondylitis, other inflammatory arthritides (i.e., gout, calcium pyrophosphate dihydrate crystal deposition disease, non-radiographic spondyloarthritis, post-infectious and reactive arthritis), other spondyloarthritides, relapsing polychondritis, rheumatoid arthritis, systemic connective tissue disorders, or unclassified connective tissue disease. [d]Bio-naïve pts are defined as those with no medical or pharmacy claims for any other bDMARD prior to the initiation of GUS or a tsDMARD. [e]Bio-experienced pts are defined as those with ≥1 least one medical or pharmacy claim for any other bDMARD during the period of continuous eligibility prior to the initiation of GUS or a tsDMARD. **bDMARD**=biologic disease-modifying antirheumatic drug; **GUS**=guselkumab; **IL**=interleukin; **PsA**=psoriatic arthritis; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

Weighted baseline demographic and clinical characteristics were similar between the GUS and tsDMARD cohorts

- Balancing was achieved for all comparisons (i.e., GUS vs tsDMARD in bio-naïve, GUS vs tsDMARD in bio-experienced, GUS vs apremilast, and GUS vs JAKi)

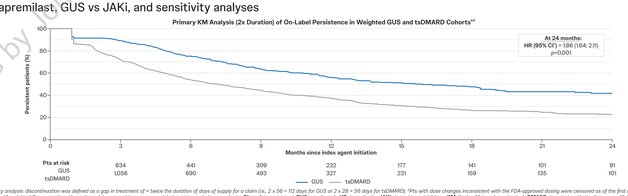
Weighted Baseline Demographics and Clinical Characteristics[a]	GUS (N=924)	tsDMARD (N=1,750)
Demographics		
Age at index date (years), Mean ± SD [median]	50.3 ± 12.0 [50.7]	50.3 ± 12.0 [50.6]
Female	59.1	59.1
Insurance type at index date		
Preferred provider organization	45.8	45.8
Health maintenance organization	39.0	39.3
Other[b]	15.2	14.9
Year of index date		
2020	6.4	6.4
2021	25.8	25.8
2022	28.8	28.8
2023	26.4	26.4
2024	12.6	12.6
Characteristics		
Months between latest observed PsA diagnosis and index date, Mean ± SD [median]	1.1 ± 1.5 [0.5]	1.1 ± 1.6 [0.5]
Quan-CCI, Mean ± SD [median]	0.8 ± 1.4 [0.0]	0.8 ± 1.5 [0.0]
Comorbidities		
Hyperlipidemia	41.6	41.6
Osteoarthritis	34.0	34.0
Diabetes	20.9	18.2
Peripheral vascular disease	4.0	4.2
Psoriasis	80.6	80.6
Smoking	16.2	13.9
Medication Use[c]		
bDMARDs[d]	64.3	64.3
csDMARDs[e]	34.3	34.3
Corticosteroids	70.5	72.0

*Data are % unless otherwise noted. [a]Propensity score using overlap weighting. [b]Includes plan subservice, consumer directed health care, indemnity/traditional, and unknown plan type. [c]During 12 months before index date. [d]Includes tumor necrosis factor inhibitors (i.e., adalimumab, etanercept, certolizumab pegol, golimumab, infliximab, adalimumab-fkjp/adbm), abatacept [biosimilar], and infliximab [biosimilar]; anti-IL-12/23 (i.e., ustekinumab); anti-cytotoxic T-lymphocyte antigen-4 (i.e., abatacept); anti-IL-6/17 (i.e., SC secukinumab, ixekizumab and IV secukinumab). [e]Includes methotrexate, sulfasalazine, leflunomide, hydroxychloroquine, azathioprine, cyclosporine, and mycophenolate. **bDMARD**=biologic disease-modifying antirheumatic drug; **csDMARD**=conventional synthetic disease-modifying anti-rheumatic drug; **GUS**=guselkumab; **IL**=interleukin; **PsA**=psoriatic arthritis; **Quan-CCI**=Quan Charlson Comorbidity Index; **SD**=standard deviation; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

Pts treated with GUS vs tsDMARDs were significantly (1.86x) more likely to remain persistent with on-label treatment through 24 months

- **Proportion of pts with on-label persistence at 24 months:** GUS (41.6%) vs tsDMARD (22.7%)
- **Median time to discontinuation:** GUS (15.9 months) vs tsDMARD (7.2 months)
- Trends were consistent in other comparisons: GUS vs tsDMARD in bio-naïve, GUS vs tsDMARD in bio-experienced, GUS vs apremilast, GUS vs JAKi, and sensitivity analyses

Primary KM Analysis (2x Duration) of On-Label Persistence in Weighted GUS and tsDMARD Cohorts[a,b]



[a]Primary analysis: discontinuation was defined as a gap in treatment of > twice the duration of days of supply for a claim (i.e., 2 x 56 = 112 days for GUS or 2 x 28 = 56 days for tsDMARD). [b]Pts with dose changes inconsistent with the FDA-approved dosing were censored as of the first dose change. [c]A weighted Cox proportional hazards model was used to compare on-label persistence between cohorts. **CI**=confidence interval; **GUS**=guselkumab; **HR**=hazard ratio; **JAKi**=Janus kinase inhibitors; **KM**=Kaplan-Meier; **pts**=patients; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

GUS was associated with significantly higher on-label persistence than tsDMARDs at all assessed time points (3, 12, 18, and 24 months), overall and in both biologic-naïve and biologic-experienced pts

On-label persistence through 24 months in weighted GUS and tsDMARD cohorts[a,b,c] – Primary analysis (2x duration)

	3 months	12 months	18 months	24 months
GUS vs tsDMARDs – Overall KM Persistence, % (95% CI)				
GUS (N=924)	89.2 (83.5; 93.0)	56.1 (49.7; 62.0)	47.8 (40.5; 54.8)	41.6 (32.9; 50.1)
tsDMARD (N=1,750)	72.6 (68.6; 76.1)	37.3 (32.2; 42.3)	26.4 (20.4; 32.8)	22.7 (16.0; 30.2)
HR (95% CI)[d]	2.65 (2.10; 3.35)*	1.88 (1.64; 2.16)*	1.90 (1.67; 2.17)*	1.86 (1.64; 2.11)*
GUS vs tsDMARDs – Bio-naïve KM Persistence, % (95% CI)				
GUS (N=185)	91.0 (73.5; 97.2)	65.6 (51.5; 76.5)	53.9 (38.0; 67.4)	43.1 (22.1; 62.6)
tsDMARD (N=796)	73.8 (67.4; 79.1)	43.6 (35.5; 51.4)	28.5 (18.9; 38.8)	25.5 (15.5; 36.8)
HR (95% CI)[d]	3.08 (1.81; 5.25)*	2.21 (1.61; 3.03)*	2.18 (1.63; 2.92)*	2.04 (1.54; 2.69)*
GUS vs tsDMARDs – Bio-experienced KM Persistence, % (95% CI)				
GUS (N=739)	88.8 (82.4; 93.0)	53.5 (46.2; 60.2)	46.7 (38.5; 54.4)	41.6 (32.2; 50.7)
tsDMARD (N=954)	71.7 (68.6; 76.3)	34.7 (28.3; 41.2)	24.6 (16.5; 33.5)	20.8 (11.6; 31.8)
HR (95% CI)[d]	2.64 (2.02; 3.45)*	1.87 (1.59; 2.19)*	1.91 (1.64; 2.22)*	1.89 (1.63; 2.19)*

*p<0.001 based on the chi-square test. [a]Primary analysis: discontinuation was defined as a gap in treatment of > twice the duration of days of supply for a claim (i.e., 2 x 56 = 112 days for GUS or 2 x 28 = 56 days for tsDMARD). [b]Pts with dose changes inconsistent with the FDA-approved dosing were censored as of the first dose change. [c]Overlap weights were used to obtain a balanced sample. Weights were estimated using a multivariable logistic regression model. Baseline covariates included several demographic and clinical characteristics. [d]A weighted Cox proportional hazards model was used to compare on-label persistence between cohorts. **CI**=confidence interval; **GUS**=guselkumab; **HR**=hazard ratio; **KM**=Kaplan-Meier; **pts**=patients; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

GUS was associated with significantly higher on-label persistence vs apremilast and vs JAKi at each assessed time point (3, 12, 18, and 24 months)

On-label persistence through 24 months in weighted GUS and tsDMARD subgroups[a,b,c] – Primary analysis (2x duration)

	3 months	12 months	18 months	24 months
GUS vs Apremilast – Overall KM Persistence, % (95% CI)				
GUS (N=924)	88.7 (82.9; 92.6)	55.0 (48.1; 61.4)	46.0 (38.0; 53.7)	39.4 (29.6; 48.9)
Apremilast (N=1,198)	70.3 (65.5; 74.6)	35.6 (29.5; 41.7)	24.4 (17.4; 32.1)	21.3 (13.7; 30.0)
HR (95% CI)[d]	2.83 (2.23; 3.60)*	1.99 (1.72; 2.30)*	1.99 (1.74; 2.29)*	1.94 (1.69; 2.22)*
GUS vs JAKi – Overall KM Persistence, % (95% CI)				
GUS (N=924)	88.5 (82.7; 92.4)	54.1 (46.8; 60.8)	47.7 (39.4; 55.5)	41.9 (32.0; 51.5)
JAKi (N=552)	75.2 (68.4; 80.7)	41.0 (33.3; 48.5)	30.6 (21.2; 40.6)	25.2 (14.2; 37.8)
HR (95% CI)[d]	2.09 (1.59; 2.75)*	1.52 (1.28; 1.81)*	1.58 (1.34; 1.86)*	1.57 (1.34; 1.85)*

*p<0.001 based on the chi-square test. [a]Primary analysis: discontinuation was defined as a gap in treatment of > twice the duration of days of supply for a claim (i.e., 2 x 56 = 112 days for GUS or 2 x 28 = 56 days for tsDMARD). [b]Pts with dose changes inconsistent with the FDA-approved dosing were censored as of the first dose change. [c]Overlap weights were used to obtain a balanced sample. Weights were estimated using a multivariable logistic regression model. Baseline covariates included several demographic and clinical characteristics. [d]A weighted Cox proportional hazards model was used to compare on-label persistence between cohorts. **CI**=confidence interval; **GUS**=guselkumab; **HR**=hazard ratio; **JAKi**=Janus kinase inhibitors; **KM**=Kaplan-Meier; **tsDMARD**=targeted synthetic disease-modifying antirheumatic drug.

Strengths and Limitations

- **Strengths**
  - PsA pts were identified using a case finding algorithm validated in US claims data[5]
  - A long follow-up window allowed robust assessment of both short-term and long-term persistence outcomes
  - Baseline characteristics for the GUS and tsDMARD cohorts were balanced across all comparisons
- **Limitations**
  - Claims data do not ensure treatments are taken as prescribed
  - Differences in route of administration may limit the direct comparability of treatment persistence across cohorts
  - Treatment effectiveness and reasons for discontinuation could not be assessed using claims data

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PRESENTED AT: The American College of Rheumatology (ACR) Convergence 2026; November 6–11, 2026; Orlando, FL, USA. **REFERENCES: 1.** Tremfya: Package insert. Janssen Biotech, Inc. 2020; **2.** Otezla: Package insert. Amgen Inc. 2021. **3.** Rinvoq: Package insert. AbbVie Inc. 2023. **4.** Xeljanz: Package insert. Pfizer Inc. 2021. **5.** Lee H. *Pharmacoepidemiology and Drug Safety.* 2020;29:404–13. **ACKNOWLEDGMENTS:** This presentation was sponsored by Johnson & Johnson. Layout design and formatting for this encore presentation were provided by Samita Warang of SIRO Medical Writing Pvt. Ltd., India, (funded by Johnson & Johnson). **DISCLOSURES: PJM:** Consulting fees: AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Immagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, Sun Pharma, Takeda, and UCB; Grants: AbbVie/Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; Speaker fees: AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **JW:** Research funding: AbbVie, Merck, and Pfizer; Consulting fees: AbbVie, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **TPF, SDC, EA, PS,** and **TN:** Employee of Johnson & Johnson; may own stock or stock options in Johnson & Johnson. **BE, AV, YW, SSa, HH, PL,** and **DP:** Employees of Analysis Group, Inc., a consulting company that has received research funding from Johnson & Johnson. **SSe:** Research funding: Eli Lilly; consulting fees: AbbVie, Johnson & Johnson and UCB; speaker fees: Johnson & Johnson. **JFM:** Consultant and/or investigator: AbbVie, Amgen, AstraZeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Johnson & Johnson, Moonlake, Novartis, Oruka, Pfizer, Regeneron, Sanofi, Sun Pharma, UCB. **PREVIOUSLY PRESENTED AT:** Rheumatology Winter Clinical Symposium (RWCS); February 11–14, 2026; Maui, HI, USA.