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ORIGINAL RESEARCH ARTICLE

Comparison of Real-World On-Label Treatment Persistence in Patients with Psoriatic Arthritis Receiving Guselkumab Versus Subcutaneous Tumor Necrosis Factor Inhibitors

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Abstract

Background Treatment persistence among patients with psoriatic arthritis (PsA) is essential for achieving optimal treatment outcomes. Guselkumab, a fully human interleukin-23p19-subunit inhibitor, was approved by the United States (US) Food and Drug Administration for the treatment of active PsA in July 2020, with a dosing regimen of 100 mg at week 0, week 4, then every 8 weeks. In the Phase 3 DISCOVER-1 and DISCOVER-2 studies of patients with active PsA, 94% of guselkumab-randomized patients completed treatment through 1 year and 90% did so through 2 years (DISCOVER-2). Real-world evidence is needed to compare treatment persistence while following US prescribing guidelines (i.e., on-label persistence) for guselkumab versus subcutaneous (SC) tumor necrosis factor inhibitors (TNFis).

Methods Adults with PsA receiving guselkumab or their first SC TNFi (i.e., adalimumab, certolizumab pegol, etanercept, or golimumab) between 14 July 2020 and 31 March 2022 were identified in the IQVIA PharMetrics® Plus database (first claim defined the treatment start date [index date]). Baseline characteristics and biologic use (biologic-naïve/biologic-experienced) were assessed during the 12-month period preceding the index date. Baseline characteristics were balanced between cohorts using propensity-score weighting based on the standardized mortality ratio approach. The follow-up period spanned from the index date until the earlier of the end of continuous insurance eligibility or end of data availability. On-label persistence, defined as the absence of treatment discontinuation (based on a gap of 112 days for guselkumab or 56 days for SC TNFi) or any dose escalation/reduction during follow-up, was assessed in the weighted treatment cohorts using Kaplan-Meier (KM) curves. A Cox proportional hazards model, further adjusted for baseline biologic use, was used to compare on-label persistence between the weighted cohorts.

Results The guselkumab cohort included 526 patients (mean age 49.8 years; 61.2% female) and the SC TNFi cohort included 1953 patients (mean age: 48.5 years; 60.2% female). After weighting, baseline characteristics were well balanced with a mean follow-up of 12.3–12.4 months across cohorts; 51.5% of patients in the guselkumab cohort and 16.7% in the SC TNFi cohort received biologics in the 12-month baseline period. Respective rates of treatment persistence at 3, 6, 9, and 12 months were 91.2%, 84.1%, 75.9%, and 71.5% for the guselkumab cohort versus 77.3%, 61.6%, 50.0%, and 43.7% for the SC TNFi cohort (all log-rank $p < 0.001$). At 12 months, patients in the guselkumab cohort were 3.0 times more likely than patients in the SC TNFi cohort to remain persistent on treatment ($p < 0.001$). Median time to discontinuation was not reached for the guselkumab cohort and was 8.9 months for the SC TNFi cohort.

Conclusion This real-world study employing US commercial health-plan claims data to assess on-label treatment persistence in PsA demonstrated that, at 12 months, guselkumab was associated with a 3 times greater likelihood of persistence compared with SC TNFi.

Previous Presentations: Data from this study were presented, in part, at the Congress of Clinical Rheumatology held 7–10 September 2023 in San Diego, CA, as a poster presentation.

Extended author information available on the last page of the article

Graphical abstract

Comparison of Real-World On-Label Treatment Persistence in Patients with Psoriatic Arthritis Receiving Guselkumab versus Subcutaneous TNF Inhibitors

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Drugs - Real World Outcomes

Why was this research needed?

Treatment persistence among patients with psoriatic arthritis (PsA) is essential for achieving optimal treatment outcomes.

Guselkumab is a medicine for PsA. In clinical trials, 94% of patients with active PsA taking guselkumab completed treatment through 1 year and 90% did so through 2 years.

Real-world evidence is needed to compare treatment persistence while following US prescribing information (i.e., on-label persistence) for guselkumab versus subcutaneous (SC) tumor necrosis factor inhibitors (TNFi).

How was this research done?

IQVIA PharMetrics® Plus claims database: Biologic-naïve and biologic-experienced adults with PsA receiving guselkumab or for whom it was their first time receiving a SC TNFi

'Weighted cohorts': Guselkumab and SC TNFi cohorts balanced for baseline characteristics during the 12-month period preceding the index date

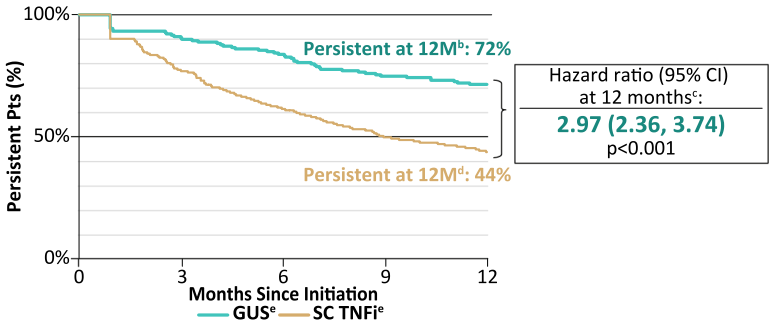
'On-label Persistence': Absence of treatment discontinuation or any dose escalation/reduction during follow-up

On-label persistence in weighted treatment cohorts

- Duration of persistence (Kaplan-Meier curves)
- % of patients persistent at 12 months compared between cohorts (Cox proportional hazards model)

What did this research tell us?

Patients in the guselkumab cohort were significantly (~3 times) more likely than those in the SC TNFi cohort to remain persistent[a] on treatment at 12 months



[a] Based on a gap of 112 days for guselkumab or 56 days for SC TNFi. [b] 52% of GUS pts were biologic-experienced at baseline; median time to discontinuation not reached. [c] Weighted Cox proportional hazard model was used to compare risk of discontinuation between the GUS and SC TNFi cohorts. Models were adjusted for baseline use of bDMARDs. [d] 17% of SC TNFi pts were biologic-experienced at baseline; median time to discontinuation: 8.9 months. [e] To obtain a balanced sample, weights were calculated based on propensity score. Baseline covariates included demographic and clinical characteristics assessed.

What could have been improved?

- **Results may not be generalizable** to uninsured patients, those with other types of health insurance, or people living outside the US
- **Claims data from health plan databases:** No assurance medication taken as prescribed; no information on treatment effectiveness or reasons for discontinuation; analysis relies on correct diagnosis, pharmacy, and procedure codes; coding errors can result in misclassification.

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PEER REVIEWED INFOGRAPHIC

Key Points

In July 2020, the United States (US) Food and Drug Administration approved guselkumab, a fully human interleukin-23p19-subunit inhibitor, for the treatment of active psoriatic arthritis (PsA) based on a dosing regimen of 100 mg at week 0, week 4, then every 8 weeks.

This retrospective study of patients with PsA in a US commercial health plan claims database, identified using a validated algorithm, compared on-label treatment persistence between those receiving guselkumab or their first subcutaneous (SC) tumor necrosis factor inhibitor (TNFi) during the study period. Persistence was defined as the absence of treatment discontinuation (based on a gap of 112 days for guselkumab or 56 days for SC TNFi) or any dose escalation/reduction during follow-up.

After applying propensity-score weighting based on the standardized mortality ratio approach, although more patients with PsA were biologic-experienced in the guselkumab (51.5%) versus SC TNFi (16.7%) cohort, patients receiving guselkumab were 3 times more likely than those receiving their first SC TNFi to be persistent on treatment through 1 year. Rates of persistence at 1 year were 71.5% and 43.7%, respectively, in the guselkumab and SC TNFi cohorts.

1 Background

Psoriatic arthritis (PsA), a multidomain, systemic inflammatory disease occurring in ~30% of patients with psoriasis [1–4], is estimated to affect over 7.5 million adults in the United States (US) [5]. Manifestations include peripheral arthritis, skin and nail psoriasis, axial disease, enthesitis, and dactylitis [6]. Additionally, inflammatory bowel disease (IBD) and uveitis are considered PsA-related conditions, and obesity, cardiometabolic conditions, and mood disorders are commonly associated comorbidities [7]. PsA can result in a considerable burden on patients' daily living and profoundly impact their health-related quality of life (HRQoL) [8–10].

Conventional pharmacological treatments for PsA include nonsteroidal anti-inflammatory drugs, glucocorticoids, and conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs), including methotrexate, which have shown modest efficacy in relieving certain PsA symptoms but are unable to delay radiographic progression [1, 2]. Biologic DMARDs (bDMARDs) indicated for PsA such as subcutaneous (SC) tumor necrosis factor inhibitors (TNFis) and interleukin (IL)-12/23, IL-17A, and IL-23 inhibitors have demonstrated clinical benefit across multiple disease domains [1, 2]. Following its approval in 2017 for the treatment of moderate-to-severe plaque psoriasis in adults, on 13 July 2020, guselkumab became the first and only fully human IL-23p19-subunit inhibitor to receive approval from the US Food and Drug Administration (FDA) for the treatment of active PsA [11]. The FDA-approved dosing regimen of 100 mg SC injection at Weeks 0, 4, and every 8 weeks thereafter for adults with active PsA was based on the results of the Phase 3, randomized, placebo-controlled DISCOVER-1 and DISCOVER-2 clinical trials that demonstrated the efficacy and favorable benefit-risk profile of guselkumab in this population [12, 13].

As patients with chronic conditions often require lifelong treatment, maintaining pharmacological treatment persistence for those with PsA is crucial for achieving optimal outcomes and improving HRQoL [8, 14–16]. Retrospective studies using real-world claims data show that persistence rates for SC and intravenous (IV) TNFi in patients with PsA newly initiated on biologics range from 51% to 56% at 12 months [14] and 18% to 22% at 24 months [17]. Due to its recent approval, real-world persistence data for guselkumab in patients with active PsA are limited. In the context of the DISCOVER-1 and DISCOVER-2 clinical trials, completion rates for patients randomized to guselkumab were 94% through 1 year and 90% through 2 years [18, 19].

Real-world data, such as those collected in an administrative health claims database, can provide evidence about treatment persistence in routine clinical care, which may differ from findings derived from stringently controlled clinical trials. This study utilized health plan claims data from a population of commercially insured patients with PsA in the US to compare persistence on treatment, based on the FDA-approved dosing regimen, between those receiving guselkumab and those receiving an initial SC TNFi.

2 Methods

2.1 Data Source

IQVIA PharMetrics® Plus [20] is a health plan claims database of predominantly commercially insured patients that contains fully adjudicated claims for inpatient and outpatient services, as well as outpatient prescription drugs, dates of service, demographic variables (e.g., age, gender, and geographic region), and start and stop dates of health plan enrollment. Information is available for more than 210

million unique enrollees across the US, with more than 95 million individuals having both medical and pharmacy benefits (40 million individuals in most recent years), and offers a diverse representation of geographic zones, employers, payers, healthcare providers, and therapeutic specialties.

IQVIA PharMetrics® Plus data are de-identified and comply with the Health Insurance Portability and Accountability Act (HIPAA) regulations. Therefore, no Institutional Review Board review was required. Data for this study were collected between 14 July 2019 and 30 September 2022 to allow for 12 months of data availability for the assessment of patient characteristics prior to guselkumab FDA approval (i.e., 13 July 2020) for the treatment of active PsA based on a dosing regimen of 100 mg at week 0, week 4, then every 8 weeks.

2.2 Study Design and Study Population

Adults with PsA who received an index biologic, i.e., guselkumab or first SC TNFi (adalimumab, certolizumab pegol, etanercept, or golimumab), between 14 July 2020 and 31 March 2022 (i.e., 6 months before the end of data availability) were included in the study (Fig. 1). The rationale for ending the intake period 6 months before the data cutoff was to allow patients in both cohorts to have sufficient follow-up time and an opportunity to discontinue the index medication. The date of the first claim for guselkumab or SC TNFi was defined as the index date, and baseline characteristics were assessed during the 12-month period of continuous insurance eligibility before the index date (i.e., baseline period). Based on a validated US claims-based algorithm for identifying PsA described by Lee and colleagues [21], patients were required to have ≥ 2 diagnoses for PsA (International Classification of Diseases, Tenth Revision [ICD-10] code: L40.5x) ≥ 30 days apart during the baseline period or on the index date as well as ≥ 1 prescription claim for guselkumab or an SC TNFi, the first of which defined the index date. For biologic agents to be covered by a commercial health plan, a patient must meet prior authorization criteria, which ensure that the medication is being prescribed for the intended use defined by FDA labeling [11]. Given that the index biologics evaluated as part of this study have been FDA-approved for the treatment of active PsA, a claim for the index biologic was considered a proxy for active disease.

Patients were identified as either biologic-naïve or biologic-experienced based on prior bDMARD use (other than guselkumab, SC TNFi, or IV TNFi) during the 12-month baseline period. However, patients were excluded if they had received ≥ 1 claim for any of the drugs under study (i.e., guselkumab or SC TNFi) or IV TNFi (i.e., infliximab or golimumab) any time during the period of continuous eligibility before the index date, in order to compare patients receiving either guselkumab or their first SC TNFi rather than patients cycling among TNFi therapies due to inadequate response or intolerance. In addition, patients were excluded if they initiated more than one index biologic on the index date or had ≥ 1 diagnosis for other potentially confounding rheumatic

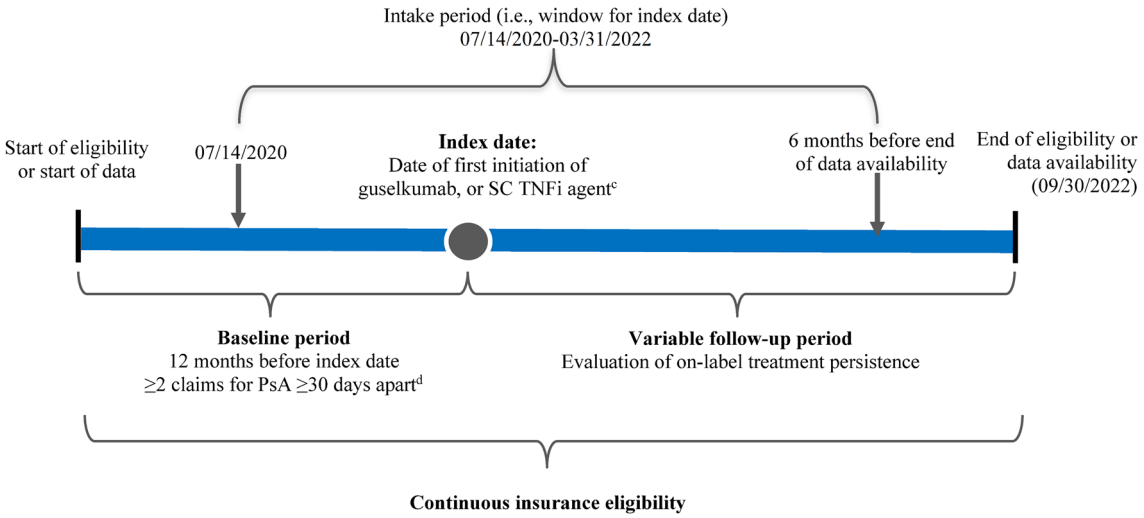

Fig. 1 Study design schema. [a,b]*ICD-10* International Classification of Disease, 10th revision, *PsA* psoriatic arthritis, *SC* subcutaneous, *TNFi* tumor necrosis factor inhibitor, *US* United States. [a]A validated algorithm for identifying patients with PsA in US claims data was used [21]. [b]Patients could be biologic-naïve or biologic-experienced during the 12-month baseline period but were naïve to treatment with guselkumab or TNFi agents. [c]The SC TNFi cohort included patients receiving a first SC TNFi. [d]Diagnoses for PsA include claims on the index date. Patients with PsA were identified based on ≥ 2 PsA diagnoses (ICD-10 code L40.5x) ≥ 30 days apart and ≥ 1 prescription claim for a PsA-related medication

diseases in the baseline period. The latter included ankylosing spondylitis (ICD-10 code: M45.x), other inflammatory arthritides (i.e., gout [ICD-10 codes: M10, M1A], calcium pyrophosphate dihydrate crystal deposition disease [ICD-10 codes: M11.20, M11.80], non-radiographic axial spondyloarthritis [ICD-10 code: M45.A], post-infectious and reactive arthritis [ICD-10 code: M02.x]), other spondyloarthropathies (ICD-10 code: M48), rheumatoid arthritis (ICD-10 codes: M05, M06, M08, M12), systemic connective tissue disorders (ICD-10 codes: M30–M35.x), relapsing polychondritis (ICD-10 code: M94.1), or unclassified connective tissue disease (ICD-10 code: L94.9). On-label persistence with the index agent was evaluated during the follow-up period, which spanned from the index date until the earlier of the end of continuous insurance eligibility or end of data availability.

2.3 Outcome Measures and Statistical Analyses

The outcome for this study was on-label persistence with the index biologic, defined as the absence of treatment discontinuation, and patients were censored at any dose escalation/reduction that was inconsistent with the respective FDA label dosing instructions for each agent. The primary definition of treatment discontinuation corresponded to a gap spanning twice the longest duration of time between administrations as per the FDA label [11, 22–25], incorporating the mode of days of supply observed in the data for each individual SC TNFi agent. Specifically, while adalimumab and certolizumab pegol dosing is indicated every 14 days, the mode of days of supply observed in the data was 28 days, as each claim typically contains two injections. Similarly, while etanercept is indicated for administration every 7 days, the mode of days of supply was 28 days owing to each claim typically comprising four injections. The longest dosing interval according to the FDA labels was selected for each agent considering that shorter dosing intervals are associated only with loading doses. As such, a 112-day gap (2 × 56 days) defined guselkumab discontinuation and a 56-day gap (2 × 28 days) defined SC TNFi discontinuation in the primary analyses. In sensitivity analyses, two additional definitions of discontinuation were assessed. In the first sensitivity analysis, the treatment gap was defined as the longest duration of time between administrations as per the FDA label (i.e., 56-day gap to define guselkumab discontinuation and 28-day gap to define SC TNFi discontinuation) [11, 22–25]. To evaluate persistence based on the same gap definition for all index biologics, the second sensitivity analysis employed a fixed 112-day gap to define discontinuation. If discontinuation was not observed, patients were censored at the earliest of the date of first observed off-label claim (i.e., the first observed dose escalation or reduction relative to the dosing instructions given by the respective FDA label, due to either a change in time between administrations or change in the number of injections per claim) or the last day of index agent supply preceding the end of the follow-up period.

Given the potential discrepancy between observed days of supply and the interval between claims in administrative databases, caused by restrictions on the maximum days of supply imposed by some health plans [26], the days of supply were conservatively imputed for both medical and pharmacy claims. For guselkumab medical claims, the days of supply were imputed as 28 days for the first claim and 56 days for all later claims, per the FDA label [11]. For guselkumab pharmacy claims, the days of supply were imputed as 28 days for the first claim with days of supply > 30 days, whereas second or later claim imputations were based on the time to next claim. For claims with missing days of supply or days of supply < 56 days, the days of supply were imputed as 28 days if the time to next claim was < 42 days, 56 days if the time to next claim was 42–70 days, and 84 days if the time to next claim was > 70 days. For SC TNFi medical claims, the days of supply were imputed as 28 days based on the mode of days of supply typically observed in pharmacy claims; no imputations were made for SC TNFi pharmacy claims, as the days of supply were largely consistent with respective FDA labels [22–25].

Baseline demographic and clinical characteristics were balanced between patients in the guselkumab and SC TNFi cohorts using propensity-score (PS) weighting calculated over the selection of treatment (i.e., guselkumab or an SC TNFi) based on the standardized mortality ratio (SMR) weighting approach. In this approach, each patient was attributed a weight based on the average treatment effect among the treated: 1 for patients in the guselkumab cohort and PS/(1-PS) for the SC TNFi cohort [27, 28]. Weights were then normalized using the mean weight so that the sample size of the weighted cohorts was the same as that of the unweighted cohorts. Covariates utilized in the SMR weighting included several baseline patient characteristics (see Online Supplemental Material [OSM] Methods). The balance of baseline demographic and clinical characteristics post-weighting was evaluated using standardized differences, whereby standardized differences < 10% indicated balance [29]. Due to the difference in prior bDMARD use during the 12-month baseline period in unweighted cohorts, this variable was not included in the SMR weighting as its inclusion meant there was not sufficient overlap in the PS distribution between treatment cohorts [30]. Utilizing the SMR-weighted treatment cohorts, Kaplan-Meier (KM) analysis was performed to assess the proportion of patients with on-label treatment persistence over time, up to 12 months following the index date. For example, in weighted cohorts, a patient with a weight of 1.5 who discontinues treatment would contribute an equivalent of

1.5 events. Cox proportional hazard models were used to compare on-label persistence between the SMR-weighted treatment cohorts at specific time points during follow-up, adjusting for prior bDMARD use in the 12-month baseline period, which was the only baseline demographic or clinical characteristic that remained imbalanced after weighting based on standardized differences > 10%.

3 Results

After applying the study's selection criteria, the guselkumab cohort included 526 patients, among whom 48.5% were biologic-naïve and 51.5% were biologic-experienced during the 12-month baseline period, and the SC TNFi cohort included 1953 patients, among whom 87.9% were biologic-naïve and 12.1% were biologic-experienced (Fig. 2).

3.1 Baseline Characteristics

Baseline demographic and clinical characteristics for the unweighted and weighted cohorts are reported in Table 1. After implementation of weighting, baseline characteristics were well balanced between patients in the guselkumab and SC TNFi cohorts, except for prior bDMARD use during the baseline period (51.5% in the guselkumab cohort and 16.7% in the SC TNFi cohort; Table 1). After weighting, the mean age was 49.8 years in the guselkumab cohort and 49.2 years in the SC TNFi cohort, and 61.2% and 61.1% of the cohorts, respectively, were female. A psoriasis diagnosis was observed in 89.4% of patients in the guselkumab cohort and 88.0% of those in the SC TNFi cohort. The most common comorbidity was hyperlipidemia (38.8% in the guselkumab cohort and 36.1% in the SC TNFi cohort), and in the guselkumab and SC TNFi cohorts, respectively, 3.2% and 3.4% of patients had IBD, while 0.4% and 0.5% had uveitis. The use of csDMARDs during the baseline period was observed in 22.4% and 24.1% of patients in the guselkumab and SC TNFi cohorts, respectively; targeted synthetic DMARDs were used by 18.1% and 18.4%, respectively, of the patients in those cohorts. Among patients with biologic use during the 12-month baseline period, 84.1% of those in the guselkumab cohort and 92.4% of those in the SC TNFi cohort received one prior biologic, while 15.9% and 7.6%, respectively, received ≥ 2 prior biologics.

3.2 On-Label Persistence

The mean follow-up times were 12.3 months in the guselkumab cohort and 12.4 months in the SC TNFi cohort. Using the primary definition of discontinuation, which reflects a gap of twice the duration of time between administrations per the FDA label, the KM rates of on-label persistence in weighted cohorts at 3, 6, 9, and 12 months were 91.2%, 84.1%, 75.9%, and 71.5%, respectively, for the guselkumab cohort and 77.3%, 61.6%, 50.0%, and 43.7%, respectively, for the SC TNFi cohort (all log-rank $p < 0.001$; Table 2). Relative to patients in the SC TNFi cohort, those in the guselkumab cohort were 3.0 times more likely to remain persistent on treatment at 12 months (95% confidence interval [CI] 2.4–3.7; $p < 0.001$; Fig. 3). The median time to discontinuation was not reached for the guselkumab cohort, and, in contrast, was 8.9 months for the SC TNFi cohort.

Consistent results were observed with each sensitivity analysis conducted. For the first sensitivity analysis, in which the discontinuation gap was defined as the longest duration between administrations according to the FDA label, the KM rates of on-label persistence in weighted cohorts at 3, 6, 9, and 12 months were 87.1%, 76.4%, 66.7%, and 59.2%, respectively, for the guselkumab cohort versus 72.3%, 55.0%, 41.5%, and 33.5%, respectively, for the SC TNFi cohort (all log-rank $p < 0.001$; OSM Table 1). At 12 months, patients in the guselkumab cohort were 2.4 times more likely to remain persistent on treatment than those in the SC TNFi cohort (95% CI: 2.0–2.9; $p < 0.001$; OSM Fig. 1). The median time to discontinuation was 18.4 months for the guselkumab cohort versus 6.9 months for the SC TNFi cohort. For the second sensitivity analysis, employing a fixed gap of 112 days, the KM rates of on-label persistence in weighted cohorts at 3, 6, 9, and 12 months were 91.2%, 84.1%, 75.9%, and 71.5%, respectively, for the guselkumab cohort versus 82.6%, 68.7%, 58.9%, and 52.1%, respectively, for the SC TNFi cohort (all log-rank $p < 0.001$; OSM Table 2). At 12 months, patients in the guselkumab cohort were 2.4 times more likely to remain persistent on treatment (95% CI: 1.9–3.0; $p < 0.001$; OSM Fig. 2). While not reached in the guselkumab cohort, the median time to discontinuation was 13.8 months for the SC TNFi cohort.

4 Discussion

This retrospective claims-based study represents the first comparative analysis of real-world on-label treatment persistence for guselkumab, a selective IL-23p19-subunit inhibitor, versus SC TNFi for the treatment of PsA in the US. Results showed that patients treated with guselkumab were significantly more likely (~3 times) than those treated

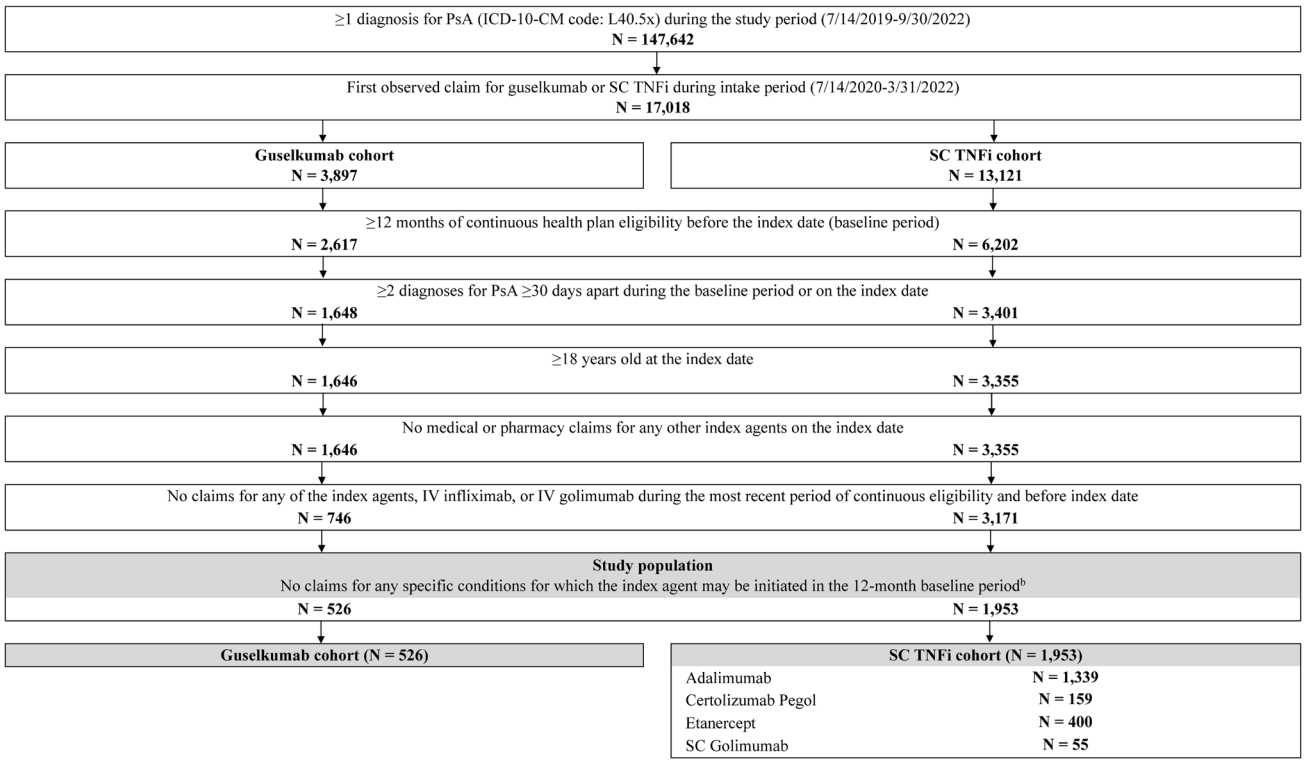

Fig. 2 Identification of the study population of patients with PsA receiving guselkumab or an SC TNFi.[a]*ICD-10* International Classification of Disease, 10th revision, *IV* intravenous, *PsA* psoriatic arthritis, *SC* subcutaneous, *TNFi* tumor necrosis factor inhibitor. [a]The SC TNFi cohort is defined as patients with an index claim for an SC TNFi (i.e., adalimumab, certolizumab pegol, etanercept, or golimumab). [b]Specific conditions for which the index agent may be initiated included ankylosing spondylitis, calcium pyrophosphate deposition disease, gout, non-radiographic axial spondyloarthritis, other spondyloarthropathies, post infectious and reactive arthropathies, relapsing polychondritis, rheumatoid arthritis, systemic connective tissue disorders, or unclassified connective tissue disease

with SC TNFi to remain persistent with treatment at 12 months.

Based on a therapy exposure gap of twice the duration of time between administrations, 72% of patients with PsA who received guselkumab were persistent on treatment after 12 months compared with only 44% of patients who received an SC TNFi. Furthermore, the median time to treatment discontinuation was 9 months in the SC TNFi cohort whereas more than half of the patients in the guselkumab cohort continued treatment beyond 12 months. The current results are consistent with findings from previous studies investigating the real-world drug survival of SC TNFi in patients with PsA, i.e., 12-month persistence rates of ~40% to 50% and estimated durations of persistence of ~8 months, using US Medicare and commercial insurance claims data across various definitions of the therapy exposure gap (i.e., 60- or 90-day fixed gap), patient clinical characteristics (e.g., biologic-naïve or biologic-experienced and with co-occurring rheumatic disease), and geographic regions [31–33]. Significantly higher rates of treatment persistence with guselkumab versus a first SC TNFi are also aligned with a series of recent studies describing persistence among biologic-naïve and biologic-experienced patients with psoriasis who initiated biologics in US and non-US clinical practice, whereby guselkumab demonstrated high persistence and showed higher rates of persistence and disease remission relative to other biologics, including TNFi, IL-17A inhibitors, and IL-12/23 inhibitors [34–36]. Notably, in a recent analysis that used the same insurance claims database to compare on-label treatment persistence among patients with PsA initiated on guselkumab or an SC IL-17A inhibitor, those who initiated guselkumab were ~2 times more likely to remain persistent at each time point up to 12 months compared with those who initiated SC IL-17A inhibitors (data presented at the Rheumatology Winter Clinical Symposium on 14–17 February 2024 in Maui, Hawaii).

For patients with PsA receiving biologics, long-term persistence on treatment can be an integral determinant of optimal outcomes [8, 14–16]. However, previous studies have documented a general pattern of poor persistence on biologics among this population in clinical practice [31, 32, 37]. Within 2 years of treatment initiation, one study showed that the overall discontinuation rate was ~80%

Table 1 Demographic and clinical characteristics in the 12-month baseline period

Mean ± SD [median] or n (%)	Unweighted cohorts			Weighted cohorts[a]		
	Guselkumab	SC TNFi	Std. diff., %	Guselkumab	SC TNFi	Std. diff., %
	N = 526	N = 1953		N = 526	N = 1953	
Age at index date (years)	49.8 ± 11.7 [50.7]	48.5 ± 11.4 [49.6]	10.6	49.8 ± 11.7 [50.7]	49.2 ± 11.6 [50.3]	5.2
Female	322 (61.2)	1176 (60.2)	2.1	322 (61.2)	1193 (61.1)	0.3
US region of residence at index date						
South	276 (52.5)	864 (44.2)	16.5	276 (52.5)	1008 (51.6)	1.7
Midwest	119 (22.6)	460 (23.6)	2.2	119 (22.6)	443 (22.7)	0.2
Northeast	78 (14.8)	335 (17.2)	6.3	78 (14.8)	290 (14.9)	0.1
West	53 (10.1)	292 (15.0)	14.8	53 (10.1)	209 (10.7)	2.0
Unknown	0 (0.0)	2 (0.1)	4.5	0 (0.0)	3 (0.1)	5.5
Insurance type at index date						
Preferred provider organization	403 (76.6)	1463 (74.9)	4.0	403 (76.6)	1501 (76.9)	0.6
Health maintenance organization	65 (12.4)	270 (13.8)	4.4	65 (12.4)	245 (12.6)	0.6
Other[b]	58 (11.0)	220 (11.3)	0.8	58 (11.0)	207 (10.6)	1.4
Medicare Advantage enrollment						
Not enrolled	511 (97.1)	1923 (98.5)	9.0	511 (97.1)	1910 (97.8)	4.1
Enrolled	15 (2.9)	30 (1.5)	9.0	15 (2.9)	43 (2.2)	4.1
Relationship of patient to the primary beneficiary at index date						
Self	306 (58.2)	1169 (59.9)	3.4	306 (58.2)	1205 (61.7)	7.2
Spouse	120 (22.8)	409 (20.9)	4.5	120 (22.8)	405 (20.7)	5.1
Child	5 (1.0)	48 (2.5)	11.7	5 (1.0)	21 (1.1)	1.2
Unknown	95 (18.1)	327 (16.7)	3.5	95 (18.1)	322 (16.5)	4.2
Year of index date						
2020	89 (16.9)	458 (23.5)	16.3	89 (16.9)	332 (17.0)	0.3
2021	346 (65.8)	1225 (62.7)	6.4	346 (65.8)	1284 (65.8)	0.0
2022	91 (17.3)	270 (13.8)	9.6	91 (17.3)	336 (17.2)	0.2
Time between latest observed PsA diagnosis to index date (months)	1.4 ± 1.7 [0.8]	0.9 ± 1.2 [0.6]	31.0	1.4 ± 1.7 [0.8]	1.2 ± 1.6 [0.7]	9.6
Baseline Quan-CCI	0.6 ± 1.4 [0.0]	0.5 ± 1.2 [0.0]	7.2	0.6 ± 1.4 [0.0]	0.6 ± 1.4 [0.0]	0.6
Prior conditions						
Hyperlipidemia	204 (38.8)	616 (31.5)	15.2	204 (38.8)	706 (36.1)	5.5
Osteoarthritis	130 (24.7)	694 (35.5)	23.7	130 (24.7)	521 (26.7)	4.4
Diabetes	94 (17.9)	256 (13.1)	13.2	94 (17.9)	312 (16.0)	5.1
IBD[c]	17 (3.2)	85 (4.4)	5.9	17 (3.2)	67 (3.4)	1.2
Peripheral vascular disease	6 (1.1)	40 (2.0)	7.2	6 (1.1)	23 (1.2)	0.2
Uveitis	2 (0.4)	12 (0.6)	3.3	2 (0.4)	9 (0.5)	1.5
Psoriasis[d]	470 (89.4)	1348 (69.0)	51.7	470 (89.4)	1718 (88.0)	4.4
Smoking	70 (13.3)	192 (9.8)	10.9	70 (13.3)	237 (12.2)	3.5
Any prior PsA treatment	385 (73.2)	1255 (64.3)	19.4	385 (73.2)	1012 (51.8)	45.3
bDMARDs[e]	271 (51.5)	236 (12.1)	93.5	271 (51.5)	327 (16.7)	78.9
1	228 (84.1)	218 (92.4)	77.5	228 (84.1)	302 (92.4)	64.3
≥ 2	43 (15.9)	18 (7.6)	36.1	43 (15.9)	25 (7.6)	34.3
csDMARDs[f]	118 (22.4)	894 (45.8)	50.8	118 (22.4)	471 (24.1)	4.0
tsDMARDs[g]	95 (18.1)	316 (16.2)	5.0	95 (18.1)	359 (18.4)	0.8
Prior non-narcotic analgesics use	22 (4.2)	48 (2.5)	9.6	22 (4.2)	65 (3.3)	4.5
Prior corticosteroids use	375 (71.3)	1294 (66.3)	10.9	375 (71.3)	1316 (67.4)	8.5
Prior opioids use	182 (34.6)	538 (27.5)	15.3	182 (34.6)	639 (32.7)	4.0

bDMARD biologic disease-modifying anti-rheumatic drug, *CCI* Charlson comorbidity index, *csDMARD* conventional synthetic disease-modifying anti-rheumatic drug, *CTLA* cytotoxic T lymphocyte-associated antigen, *IBD* inflammatory bowel disease, *ICD-10* International Classification of Disease, 10th revision, *IL* interleukin, *IV* intravenous, *PsA* psoriatic arthritis, *SC* subcutaneous, *SD* standard deviation, *Std. Diff.* standardized

Table 1 (continued)

difference, *TNFi* tumor necrosis factor inhibitor, *tsDMARD* targeted synthetic disease-modifying anti-rheumatic drug, *US* United States. [a]Propensity score weighting based on the standardized mortality ratio weighting approach was used to adjust for differences in baseline characteristics between the guselkumab and SC TNFi cohorts. Weights were estimated using a multivariable logistic regression model. Baseline covariates included all demographic and clinical characteristics reported in this table with the exception of baseline use of bDMARDs, which was included in the adjusted Cox proportional hazard models. [b]Point-of-service, consumer directed health care, indemnity/traditional, and unknown plan type. [c]Unclassified IBD, Crohn's disease, and ulcerative colitis. [d]Defined based on ICD-10 code L40.x (excluding L40.5). [e]IL-17A inhibitors (i.e., secukinumab and ixekizumab), IL-12/23 inhibitor (i.e., ustekinumab), anti-CTLA-4 agent (i.e., abatacept), and IL-23p19-subunit inhibitor (i.e., risankizumab). The proportion of patients with 1 and ≥ 2 bDMARDs is reported among those with any bDMARD use. [f]Methotrexate, leflunomide, cyclosporine, mycophenolate, and azathioprine. [g]Apremilast, deucravacitinib, and Janus kinase inhibitors (i.e., upadacitinib, baricitinib, and tofacitinib)

Table 2 On-label persistence through 12 months in weighted guselkumab and SC TNFi cohorts: [a]primary analysis (gap of twice the duration of time between administrations as per FDA label)

Cox proportional hazards model[b]	3 months	6 months	9 months	12 months
Patients at risk, n (%)[c]				
Guselkumab (N = 526)	368 (70.0)	263 (50.0)	155 (29.5)	84 (16.0)
SC TNFi (N = 1953)	1051 (53.8)	744 (38.1)	452 (23.1)	299 (15.3)
Hazard ratios (95% CI)	3.41 (2.41; 4.80)	3.30 (2.51; 4.33)	3.06 (2.41; 3.88)	2.97 (2.36; 3.74)
Chi-square p value	< 0.001*	< 0.001*	< 0.001*	< 0.001*
KM persistence, % (95% CI)				
Guselkumab	91.2 (82.8; 95.6)	84.1 (76.7; 89.4)	75.9 (68.3; 81.9)	71.5 (63.2; 78.3)
SC TNFi	77.3 (73.1; 80.9)	61.6 (56.8; 66.1)	50.0 (44.4; 55.3)	43.7 (37.3; 49.8)
Log-rank test p-value	< 0.001*	< 0.001*	< 0.001*	< 0.001*

bDMARD biologic disease-modifying anti-rheumatic drug, *CI* confidence interval, *FDA* Food and Drug Administration, *KM* Kaplan-Meier, *SC* subcutaneous, *TNFi* tumor necrosis factor inhibitor. *Denotes statistical significance based on a threshold of $p < 0.05$. [a]Propensity score weighting based on the standardized mortality ratio weighting approach was used to adjust for differences in baseline characteristics between the guselkumab and SC TNFi cohorts. Weights were estimated using a multivariable logistic regression model. Baseline covariates included all demographic and clinical characteristics reported in Table 1, with the exception of baseline use of bDMARDs, which was included in the adjusted Cox proportional hazard models. [b]Cox proportional hazard models were used to compare risk of discontinuation between the weighted guselkumab and SC TNFi cohorts. Models were adjusted for baseline use of bDMARDs. [c]Patients at risk of having the event are patients who have not had the event and have not been lost to follow-up at that point in time

among biologic-naïve patients with PsA treated with injectable biologics, including SC TNFi [17]. In a Multinational Assessment of Psoriasis and Psoriatic Arthritis patient survey, 59% of patients with PsA were not receiving any therapy or were receiving only a topical therapy, despite 50% to 75% of patients self-reporting severe symptoms [38].

As a comprehensive measure, treatment persistence reflects many factors including treatment effectiveness and safety, disease severity and activity, the presence of comorbidities, as well as patient and physician preferences and awareness of biologics [14, 16, 37, 39]. Indeed, studies of patients with PsA treated with biologics, including TNFi, reported that inadequate symptom control and adverse effects were the most common predictors of switching or discontinuing treatments [38, 40, 41]. Recent US registry data from a largely treatment-refractory population of patients with active PsA show that after 6 months of persistent treatment with guselkumab, patients experienced significant improvements in peripheral joint and skin disease and patient-reported pain, with nearly 80% of patients maintaining on-label persistence with guselkumab at 6 months, which is consistent with the findings from the current study [42]. Though further analysis using long-term data is warranted, the high real-world persistence on guselkumab observed in this study aligns with the high patient retention rates of 94% through 1 year and 90% through 2 years in Phase 3 trials [18, 19].

Several methodological considerations strengthen the results reported in this study. The study sample comprised commercially insured patients whose baseline demographic and clinical characteristics were balanced between the guselkumab and SC TNFi cohorts using SMR weighting and for whom payer prior authorization requires that medications be used in accordance with FDA prescribing information. The use of a case-finding algorithm for PsA validated in US claims data, coupled with the prior authorization processes, provides confidence that this analysis included patients with active PsA, despite the lack of clinical measures of disease

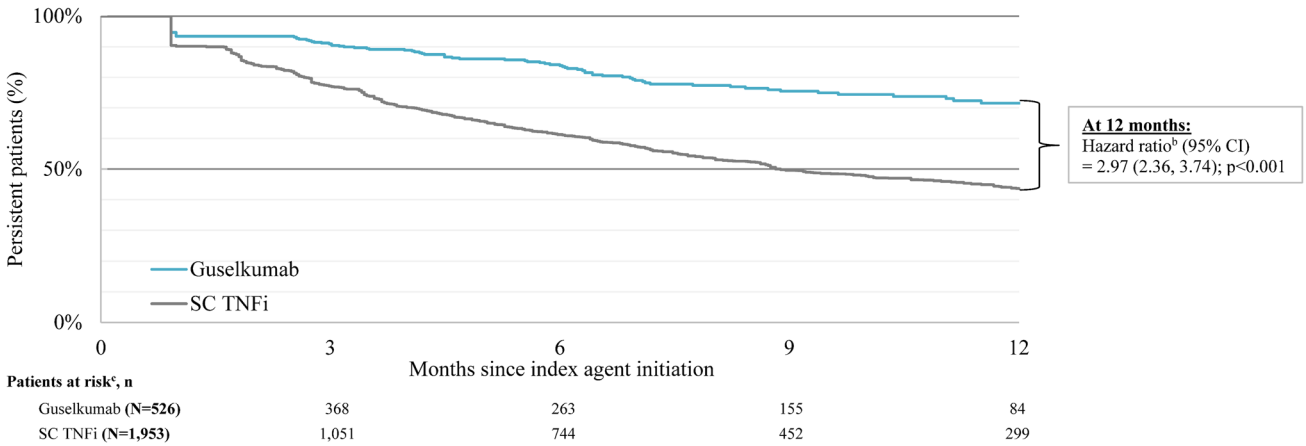

Fig. 3 Kaplan-Meier analysis of on-label persistence in weighted guselkumab and SC TNFi cohorts: [a]primary analysis (gap of twice the duration of time between administrations as per FDA label). *bDMARD*biologic disease-modifying anti-rheumatic drug, *CI*confidence interval, *FDA* Food and Drug Administration, *SC* subcutaneous, *TNFi* tumor necrosis factor inhibitor. [a] Propensity score weighting based on the standardized mortality ratio weighting approach was used to adjust for differences in baseline characteristics between the guselkumab and SC TNFi cohorts. Weights were estimated using a multivariable logistic regression model. Baseline covariates included all demographic and clinical characteristics reported in Table 1, with the exception of baseline use of bDMARDs, which was included in the adjusted Cox proportional hazard models. [b] Cox proportional hazard models were used to compare risk of discontinuation between the weighted guselkumab and SC TNFi cohorts. Models were adjusted for baseline use of bDMARDs. [c] Patients at risk of having the event are patients who have not had the event and have not been lost to follow-up at that point in time

activity in this type of data source. The imputation of days of supply for certain agents was conducted based on the FDA label recommendations using the time to next claim as a proxy for days of supply, a common technique based on a previously published algorithm [26]. Although misclassification of off-label status for some patients may have occurred, more conservative estimates of the therapy exposure gap were employed in sensitivity analyses, the results of which were consistent with findings of the primary analysis. Specifically, significantly higher rates of guselkumab than SC TNFi persistence were also observed when exposure gaps to define treatment discontinuation were either the longest duration between administrations according to the FDA label or a fixed gap of 112 days. As such the potential misclassification resulting from the imputation of days of supply is expected to be minimal.

4.1 Limitations

In addition to the strengths highlighted above, the findings from this study should be interpreted in the context of some limitations, most of which are inherent to the use of health plan claims databases. As analyses depend on correct diagnosis, procedure, and drug codes, coding inaccuracies may have led to case misidentification. Claims data do not necessarily ensure that the medication was taken as prescribed, and neither evaluation of treatment effectiveness, reasons for discontinuation, nor date of death were available in the database. Relatedly, given the use of claims data and a 12-month baseline period, bDMARD exposure prior to the start of the baseline period was unknown. Therefore, misclassification could have occurred, but this is expected to be minimal as only an additional 3% of patients would have been reclassified as bio-experienced by extending the period for evaluating prior bDMARD use from the 12-month baseline period to the entire period of continuous insurance eligibility prior to the index date. In addition, certain clinical (e.g., patient- or clinician-reported outcomes of disease severity or relative burden of psoriasis vs. arthritis) or patient (e.g., race, ethnicity) characteristics that might influence outcomes were not available or may have been under-reported in the database, which may have resulted in residual confounding. Finally, results may not be generalizable to uninsured patients with PsA, those with other types of health insurance, or patients with PsA outside of the US.

5 Conclusions

This study was the first to use predominantly commercial health plan claims data to compare real-world persistence of guselkumab and SC TNFi per US FDA-approved labels among patients with PsA in the US. Results showed that over a 12-month period, guselkumab was associated with a 3 times greater likelihood of persistence than an initial SC TNFi. These findings are consistent with previous real-world studies reporting high persistence for patients treated with guselkumab, and support the high patient retention rates observed in clinical trials. Given the complexity and chronicity of PsA as a heterogenous disease, and the benefit of maintaining long-term disease control on optimizing clinical outcomes and HRQoL, long-term treatment persistence should be considered as part of the shared decision making in treatment selection.

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Declarations

Funding This study was funded by Janssen Scientific Affairs, LLC, a Johnson & Johnson company. Authors who are employees of the study sponsor were involved in several aspects of the research, including designing the study. All authors were involved in interpretation of data and drafting or revising the writing of the manuscript.

Conflict of Interest IL, NS, and SDC are employees of Janssen Scientific Affairs, LLC, a Johnson & Johnson company and stockholders of Johnson & Johnson. NS received salary support from the Childhood Arthritis and Rheumatology Research Alliance and owns or has owned stock in AbbVie, Gilead, Iovance, Jazz Pharmaceuticals, Novavax, and Viatris. RZ was an employee of Janssen Scientific Affairs, LLC, a Johnson & Johnson company at the time of study conduct. PL, DP, BE, LM, LHY, and SS are employees of Analysis Group, Inc., a consulting company that has received research funding from Janssen Scientific Affairs, LLC, a Johnson & Johnson company. JAW received research funding from Pfizer, Merck, AbbVie and served as a consultant for AbbVie, Janssen, Eli Lilly, Novartis, UCB. PM received research funding from AbbVie, Acelyrin, Amgen, Bristol Myers Squibb, Eli Lilly, Janssen, Novartis, Pfizer, Sun Pharma, and UCB, served as a consultant for AbbVie, Acelyrin, Aclaris, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Eli Lilly, Galapagos, Gilead, GlaxoSmithKline, Inmagene, Janssen, Novartis, Pfizer, Sun Pharma, UCB, and Ventyx, and received speaker fees from AbbVie, Amgen, Eli Lilly, Janssen, Novartis, Pfizer, and UCB.

Author Contributions JAW contributed to the conceptualization, investigation, methodology, supervision, validation, visualization, writing of the original draft, and its review and editing. IL contributed to the conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualization, writing of the original draft, and its review and editing. RZ contributed to the conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualization, writing of the original draft, and its review and editing. NJS contributed to the conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualization, writing of the original draft, and its review and editing. LM contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. BE contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. LHY contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. SS contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. PL contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. DP contributed to the conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing of the original draft, and its review and editing. SDC contributed to the conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualization, writing of the original draft, and its review and editing. PM contributed to the conceptualization, investigation, methodology, supervision, validation, visualization, writing of the original draft, and its review and editing. All authors have reviewed and approved the final submitted manuscript and agree to be accountable for all aspects of the work.

Data Availability The data that support the findings of this study are available from IQVIA PharMetrics® Plus. Restrictions apply to the availability of these data, which were used under license for this study.

Ethics Statement Data were de-identified and comply with the patient requirements of the Health Insurance Portability and Accountability Act (HIPAA) of 1996; therefore, no review by an Institutional Review Board was required per Title 45 of CFR, Part 46.101(b)(4) (https://www.hhs.gov/ohrp/regulations-and-policy/regulations/45-cfr-46/#46.101).

Consent to Participate Not applicable.

Consent for Publication Not applicable.

Code Availability All analyses were conducted using SAS Enterprise Guide 7.1 (SAS Institute, Cary, NC, US). The SAS programs are proprietary materials of Analysis Group, Inc.; therefore, restrictions apply to the access of these codes, which cannot be made available publicly.

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