Clinical, Cosmetic and Investigational Dermatology

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Baseline Clinical and Metabolic Predictors of 52-Week Durability of Secukinumab Response in Moderate-to-Severe Plaque Psoriasis

Jiayue Wang[1], Ping Li[1,2]

[1]Beijing University of Chinese Medicine, Beijing, People's Republic of China; [2]Beijing Hospital of Traditional Chinese Medicine, Capital Medical University, Beijing Institute of Traditional Chinese Medicine, Beijing, People's Republic of China

Correspondence: Ping Li, Beijing University of Chinese Medicine, Beijing, People's Republic of China, Email bjtcmzyyy@yeah.net

Background: Durability of response to IL-17A blockade (secukinumab) varies, representing a major clinical challenge. Psoriasis is inherently linked to immunometabolic dysfunction. We hypothesized that simple, routine metabolic indices could predict long-term treatment stability, offering crucial pre-treatment stratification tools.

Methods: This was a 52-week prospective, single-center cohort study of 118 patients with moderate-to-severe plaque psoriasis initiating secukinumab. We defined Durable Response (DR) as PASI90 at Week 12 maintained as absolute PASI ≤ 2 until Week 52. Multivariable logistic regression and Cox models were used to identify independent predictors of DR and drug survival.

Results: Independent predictors of durable response were biologic-naïve status (adjusted OR = 3.52, 95% CI: 1.58–7.85) and a lower baseline TG/HDL-C ratio (adjusted OR = 0.61, 95% CI: 0.47–0.79). Conversely, obesity (BMI≥30) (OR = 0.42) and higher baseline PASI (OR = 0.91) were associated with reduced odds of DR. Conventional systemic inflammatory markers (CRP, NLR) showed no significant difference. Drug survival was significantly higher in DRs (92.6% vs 78.3% at Week 52) and was independently reduced by psoriatic arthritis.

Conclusion: The routine baseline TG/HDL-C ratio is a strong, independent predictor of sustained secukinumab efficacy. These easily accessible clinical and metabolic features capture the immunometabolic milieu influencing IL-17A inhibition durability. Integrating the TG/HDL-C ratio into pre-treatment assessment can support patient stratification and optimize long-term management strategies for plaque psoriasis.

Keywords: psoriasis, secukinumab, treatment durability, PASI90 maintenance, metabolic predictors, TG/HDL-C ratio

Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disease that imposes a substantial burden on patients with moderate-to-severe plaque psoriasis.[1] Globally, the prevalence of psoriasis is rising, affecting approximately 2–3% of the population, with a significant disease burden observed in China.[2,3] Beyond cutaneous manifestations, psoriasis is frequently associated with systemic comorbidities, most notably psoriatic arthritis (PsA), which affects up to 30% of patients and further impacts quality of life.[4]

With the advent of biologic therapies, particularly IL-17A inhibitors such as secukinumab, treatment outcomes have improved dramatically. Secukinumab works by selectively neutralizing Interleukin-17A (IL-17A), a key pro-inflammatory cytokine that drives keratinocyte hyperproliferation and the recruitment of neutrophils via a feed-forward inflammatory loop.[5] However, therapeutic response remains highly heterogeneous.[6] While some patients achieve rapid, profound, and durable skin clearance, others exhibit only partial improvement or experience a gradual loss of response over time.[7] Understanding this variability represents an important unmet need in psoriasis management.

Previous studies have suggested that clinical features, including biologic-naïve status and baseline disease severity, may be related to treatment outcomes.[8] Psoriasis also frequently coexists with metabolic abnormalities such as obesity

https://doi.org/10.2147/CCID.S588137
Received: 11 December 2025
Accepted: 23 February 2026
Published: 3 March 2026

and dyslipidemia, which may interact with inflammatory pathways and potentially affect treatment response.[9] Emerging evidence suggests a "metabolic-immune nexus" where systemic metabolic dysfunction—characterized by insulin resistance and dyslipidemia—can amplify Th17 differentiation and IL-17A production, potentially creating an inflammatory environment that is more resistant to biologic blockade.[10]

However, the role of routine metabolic indicators, including composite lipid ratios, in predicting the stability of biologic treatment response has not been fully clarified. This study aims to identify baseline clinical and metabolic factors associated with the long-term durability of secukinumab treatment, focusing on the maintenance of PASI90 response and drug survival over 52 weeks. Clarifying these predictors may support more individualized care and improve long-term management strategies for plaque psoriasis.

Methods

Study Design and Participants

This was a prospective, single-center, observational cohort study conducted at the Beijing Hospital of Traditional Chinese Medicine, Capital Medical University. The study was conducted in accordance with the principles of the Declaration of Helsinki.[11] The study protocol was approved by the Institutional Ethics Committee of the hospital (Reference Number: 2019BL02-010-02), and written informed consent was obtained from all participants.

Inclusion criteria were: (a) Adults aged 18–65 years, regardless of sex; (b) Clinically diagnosed with moderate-to-severe plaque psoriasis, with a baseline PASI score ≥10; (c) Voluntarily agreed to participate and signed the informed consent form. Exclusion criteria were: (a) Pregnant or breastfeeding women; (b) Known hypersensitivity to any component of the study medication; (c) Coexistence of other dermatologic conditions that could interfere with the assessment of treatment efficacy. Patients were permitted to use stable doses of topical agents; however, concomitant systemic antipsoriatic therapies were prohibited during the observation period.

The total sample size of 118 patients was determined by the availability of eligible patients during the recruitment period (convenience sampling). Although a formal a priori power calculation was not performed, post-hoc analysis indicated that the sample size was sufficient to detect significant associations in the multivariable analysis.

Patient Grouping

Patients achieving PASI90 at week 12 were stratified into two response-trajectory groups:

Durable Responders (DR)

Patients who achieved PASI90 at week 12 and maintained absolute PASI ≤ 2 at all subsequent predefined visits (weeks 24, 36, and 52). No treatment modifications for insufficient efficacy were required during the observation period. These individuals represent a consistently stable response trajectory.

Non-Durable Responders (Non-DR)

Patients who also reached PASI90 at week 12 but subsequently demonstrated instability in disease control during weeks 12–52. Instability was defined by the presence of any of the following: (a) Objective loss of response: Either loss of PASI90 at any follow-up visit accompanied by an absolute PASI ≥ 5, or a rise of ≥ 3 absolute PASI points from the individual nadir, persisting for at least two consecutive visits. (b) Treatment modification prompted by inadequate efficacy: This included dose escalation, shortened dosing intervals, addition of systemic therapy, or switching to another biologic.

Statistical Analysis

All statistical analyses were performed using SPSS version 26.0 and Python 3.9. Continuous variables were presented as mean ± standard deviation (SD) or median with interquartile range (IQR), and categorical variables as counts and percentages. Between-group comparisons were conducted using the Student's *t*-test or Mann–Whitney *U*-test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate. Variables with a univariate association at $p < 0.10$ were included in a multivariable logistic regression model to identify independent predictors of achieving a durable response. Time-to-event outcomes, defined as loss of PASI 90

maintenance, were evaluated using Kaplan–Meier survival curves and compared using the Log rank test. Cox proportional hazards models were applied to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for factors associated with response durability. A two-sided $p < 0.05$ was considered statistically significant.

Results

Baseline Characteristics

A total of 118 patients with moderate-to-severe plaque psoriasis completed the 52-week follow-up and were included in the analysis. Among them, 72 patients (61.0%) were categorized as **DR**, and 46 (39.0%) as Non-DR. At baseline, the two groups were comparable in age, sex distribution, and disease duration (Table 1). The DR group had a lower mean BMI (27.8 vs 29.9 kg/m^2, $p = 0.028$) and a lower baseline PASI score (12.5 vs 15.1, $p = 0.045$). A greater proportion of patients in the DR group were biologic-naïve at treatment initiation (52.8% vs 32.6%, $p = 0.027$). A family history of psoriasis was more

Table 1 Baseline Demographic and Clinical Characteristics of the Study Population Stratified by Response Durability

Characteristics	Total (n=118)	DRs (n=72)	Non-DRs (n=46)	P value
Demographic characteristics				
Age (years), mean ± SD	49.7 ± 14.1	48.3 ± 13.8	51.9 ± 14.5	0.173
Sex, n (%)				0.468
Male	74 (62.7%)	47 (65.3%)	27 (58.7%)	
Female	44 (37.3%)	25 (34.7%)	19 (41.3%)	
BMI (kg/m^2), mean ± SD	28.7 ± 5.3	27.8 ± 5.1	29.9 ± 5.4	0.028
Disease characteristics				
Disease duration (years), mean ± SD	16.4 ± 10.2	15.5 ± 9.7	17.8 ± 10.9	0.231
Baseline PASI, mean ± SD	13.9 ± 7.1	12.5 ± 6.2	15.1 ± 7.9	0.045
Psoriatic arthritis, n (%)				0.049
Yes	25 (21.2%)	11 (15.3%)	14 (30.4%)	
No	93 (78.8%)	61 (84.7%)	32 (69.6%)	
Family history of psoriasis, n (%)				0.039
Yes	36 (30.5%)	17 (23.6%)	19 (41.3%)	
No	82 (69.5%)	55 (76.4%)	27 (58.7%)	
Treatment history				
Biologic-naïve status, n (%)				0.027
Yes	53 (44.9%)	38 (52.8%)	15 (32.6%)	
No	65 (55.1%)	34 (47.2%)	31 (67.4%)	
Comorbidities, n (%)				
Hypertension				0.118
Yes	41 (34.7%)	20 (27.8%)	21 (45.7%)	
No	77 (65.3%)	52 (72.2%)	25 (54.3%)	

(*Continued*)

Table 1 (Continued).

Characteristics	Total (n=118)	DRs (n=72)	Non-DRs (n=46)	P value
Type 2 diabetes mellitus				0.062
Yes	19 (16.1%)	8 (11.1%)	11 (23.9%)	
No	99 (83.9%)	64 (88.9%)	35 (76.1%)	
Nonalcoholic fatty liver disease				0.082
Yes	31 (26.3%)	15 (20.8%)	16 (34.8%)	
No	87 (73.7%)	57 (79.2%)	30 (65.2%)	

Note: P-values < 0.05 are indicated in bold.
Abbreviations: BMI, Body Mass Index; PASI, Psoriasis Area and Severity Index; NAFLD, Nonalcoholic Fatty Liver Disease; SD, Standard Deviation.

frequently reported in the Non-DR group (41.3% vs 23.6%, p = 0.039). The prevalence of hypertension showed a higher trend in Non-DR patients (41.3% vs 27.8%, p = 0.118), although the difference did not reach statistical significance. Variables including BMI, baseline PASI, concomitant psoriatic arthritis, family history of psoriasis, and biologic-naïve status were identified as potential covariates and were therefore included in subsequent multivariable analyses.

Baseline Metabolic Characteristics

Analysis of laboratory parameters is presented in Table 2. Regarding lipid metabolism, the DR group demonstrated significantly lower triglyceride levels (1.52 ± 0.75 mmol/L vs 1.92 ± 0.85 mmol/L, p = 0.007), lower LDL-C levels (2.71

Table 2 Comparison of Baseline Metabolic and Inflammatory Parameters Between Durable Responders and Non-Durable Responders

Laboratory Parameters	DRs	Non-DRs	P value
TC (mmol/L), mean ± SD	4.91 ± 0.89	4.76 ± 0.96	0.382
TG (mmol/L), mean ± SD	1.52 ± 0.75	1.92 ± 0.85	0.007
HDL-C (mmol/L), mean ± SD	1.26 ± 0.30	1.15 ± 0.31	0.058
LDL-Cl (mmol/L), mean ± SD	2.71 ± 0.75	3.18 ± 0.78	0.009
TG/HDL-C ratio, mean ± SD	3.05 ± 1.52	3.98 ± 2.01	0.003
CRP (mg/L), median [IQR]	3.3 [1.7, 6.2]	4.1 [2.1, 7.5]	0.21
NLR, mean ± SD	2.25 ± 0.90	2.40 ± 1.00	0.394
ALT (U/L), median [IQR]	27 [20, 37]	30 [22, 42]	0.255
AST (U/L), median [IQR]	24 [20, 31]	26 [21, 34]	0.318
Cr (μmol/L), mean ± SD	75.8 ± 17.5	77.6 ± 19.3	0.584
BUN (mmol/L), mean ± SD	5.3 ± 1.5	5.5 ± 1.7	0.487
UA (μmol/L), mean ± SD	338 ± 82	356 ± 89	0.241
HbA1c (%), mean ± SD	5.6 ± 0.5	5.9 ± 0.7	0.032

Abbreviations: TC, Total Cholesterol; TG, Triglycerides; HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; CRP, C-reactive protein; NLR, Neutrophil-to-lymphocyte ratio; HbA1c, Glycated hemoglobin; UA, Uric acid; ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; Cr, Creatinine; IQR, Interquartile Range.

Variable	Adjusted OR	95% CI	P-value
Biologic-naïve status	3.52	1.58–7.85	0.002
Baseline TG/HDL-C ratio	0.61	1.47–0.09	< 0.001
Obesity	0.42	0.18–0.96	0.039
Baseline PASI score	0.91	0.86–0.96	0.003
HbA1c	0.92	0.42–1.61	0.563
Family History of Psoriasis	0.75	0.33–1.64	0.504
Psoriatic Arthritis	0.68	0.28–1.67	0.306

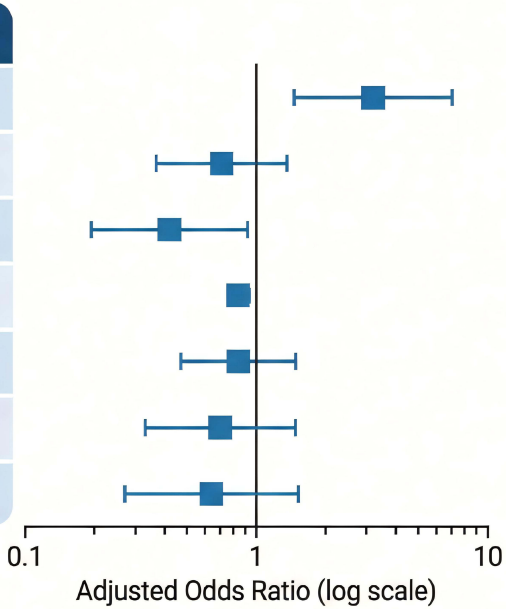

Figure 1 Forest plot of multivariable logistic regression analysis identifying independent predictors of durable response (DR) at Week 52. Odds ratios (OR) and 95% confidence intervals (CI) are presented. Biologic-naïve status and a lower TG/HDL-C ratio were significant predictors of sustained efficacy.

± 0.75 mmol/L vs 3.18 ± 0.78 mmol/L, p = 0.009), and a lower TG/HDL-C ratio (3.05 ± 1.52 vs 3.98 ± 2.01, p = 0.003) compared with the Non-DR group. In terms of glucose metabolism, HbA1c levels were also lower in DR patients (5.6% ± 0.5% vs 5.9% ± 0.7%, p = 0.032). No significant differences were observed in systemic inflammatory markers, including CRP and NLR, between the two groups.

Independent Predictors of Durable Response

Variables with a p value < 0.05 in univariate analyses were entered into the multivariable logistic regression model. As shown in Figure 1, biologic-naïve treatment status (adjusted OR = 3.52, 95% CI: 1.58–7.85) and a lower baseline TG/HDL-C ratio (adjusted OR = 0.61, 95% CI: 0.47–0.79) were identified as independent predictors of achieving a durable response. In contrast, obesity (adjusted OR = 0.42, 95% CI: 0.18–0.96) and higher baseline PASI scores (adjusted OR = 0.91, 95% CI: 0.86–0.97) were independently associated with reduced odds of durable response. The Hosmer–Lemeshow goodness-of-fit test yielded a p value of 0.62, indicating an adequate model fit. The model demonstrated good discriminative performance, with an area under the receiver operating characteristic curve of 0.82 (95% CI: 0.76–0.88).

Durability of Treatment Response

Kaplan–Meier analysis demonstrated that durable responders maintained PASI90 significantly longer than non-durable responders throughout the follow-up period (log-rank P < 0.001) (Table 3). By week 52, the event-free survival rate for maintaining PASI90 was 88.1% (95% CI: 79.0–93.5) in the DR group, compared with 71.4% (95% CI: 58.6–81.2) in the Non-DR group. The median duration of PASI90 maintenance was not reached within the 52-week follow-up for DRs, indicating that most patients sustained their high-level responses. In contrast, the estimated median duration for Non-DRs

Table 3 Survival Analysis of PASI 90 Maintenance Efficacy (Durability)

Group	Median Maintenance Time (Weeks)	52-Week Maintenance Rate (95% CI)	Log-Rank P value
DR	Not reached	88.1% (79.0–93.5)	<0.001
Non-DR	46.2 (40.1–51.0)	71.4% (58.6–81.2)	

https://doi.org/10.2147/CCID.S588137

Table 4 Drug Retention Rates (Drug Survival) at Follow-Up Time Points

Group	W12	W24	W36	W48	W52
DR	72 (100%)	71 (98.6%)	67 (93.1%)	67 (93.1%)	67 (93.1%)
Non-DR	46 (100%)	39 (85.4%)	35 (76.1%)	34 (73.9%)	34 (73.9%)

Notes: Data represent n (%). Comparison between groups at Week 52: Log-rank P = 0.006.

was 46.2 weeks (95% CI: 40.1–51.0). Explicitly, the median maintenance time refers to the time point at which the cumulative probability of sustaining a PASI 90 response drops to 50%.

Drug Survival Analysis

Kaplan–Meier estimates showed a significantly higher drug survival rate in the DR group (Table 4). By week 52, drug survival was 92.6% (95% CI: 84.8–96.6) in DRs, compared with 78.3% (95% CI: 66.4–86.6) in Non-DRs (log-rank P = 0.006). To identify independent predictors of drug survival, a multivariable Cox proportional hazards model was constructed, adjusting for age, sex, obesity, baseline PASI, and biologic-naïve status. As illustrated in Figure 2, durable response was the strongest independent protective factor (adjusted HR = 0.28, 95% CI: 0.14–0.55; P < 0.001). Biologic-naïve status also conferred a protective effect (adjusted HR = 0.52, 95% CI: 0.29–0.93; P = 0.027), whereas the presence of psoriatic arthritis significantly increased the risk of discontinuation (adjusted HR = 2.10, 95% CI: 1.16–3.79; P = 0.014).

Discussion

In this 52-week follow-up study, several baseline features showed clear associations with the durability of secukinumab response. Patients with a lower TG/HDL-C ratio were more likely to maintain PASI 90 throughout treatment. This finding is consistent with previous work suggesting that lipid metabolism may influence inflammatory pathways in psoriasis and modify biologic treatment outcomes.[12] Biologic-naïve status also favored long-term control, while higher BMI and higher baseline PASI were linked to weaker durability—patterns that have been reported in other secukinumab

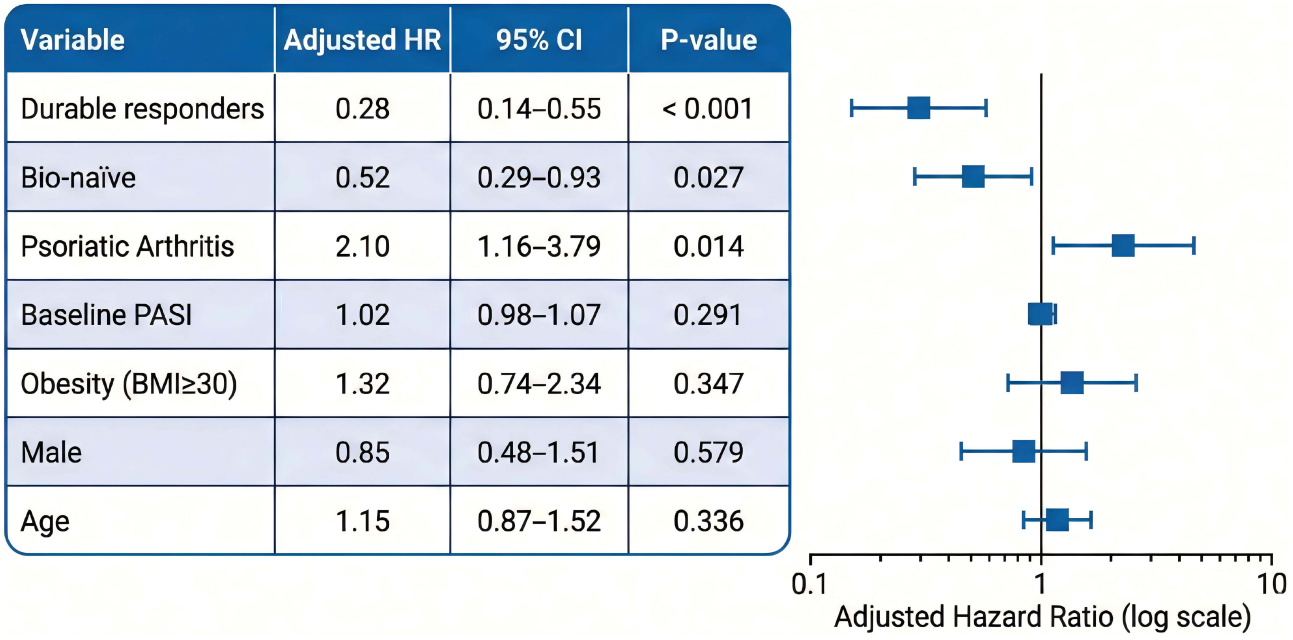

Figure 2 Forest plot of multivariable Cox proportional hazards model identifying predictors of drug survival over 52 weeks. Hazard ratios (HR) and 95% confidence intervals (CI) are presented. Achieving a durable response was the strongest protective factor against drug discontinuation.

https://doi.org/10.2147/CCID.S588137

cohorts.[13] Patients who achieved durable response showed longer maintenance of PASI 90 and better drug survival over one year.

The identification of the TG/HDL-C ratio as a superior predictor compared to traditional inflammatory markers (such as CRP or NLR) is a key finding of this study. While CRP and NLR are valuable indicators of acute inflammation, they are subject to high intra-individual variability due to short-term stressors or infections. In contrast, the TG/HDL-C ratio reflects a chronic, structural metabolic dysfunction closely linked to insulin resistance.[10] We propose that this ratio serves as a surrogate for a "pro-inflammatory metabolic milieu". High triglyceride levels and dysfunctional HDL may perpetuate a low-grade inflammatory state that continuously fuels the Th17/IL-17 axis, creating an "inflammatory reserve" that counteracts the neutralizing capacity of secukinumab over time.[14]

Furthermore, our findings regarding metabolic predictors may extend beyond secukinumab. Given the shared mechanism of action, similar interactions between metabolic health and drug durability have been observed with other IL-17 inhibitors like ixekizumab, suggesting a potential class effect where metabolic optimization could be crucial for long-term efficacy across the IL-17 inhibitor class.[15] The study contributes to the growing evidence that metabolic health is not only a comorbidity issue but may also shape therapeutic trajectory in psoriasis. Unlike many earlier studies focusing mainly on clinical severity or immune biomarkers, our results suggest that routine metabolic indices deserve greater attention in long-term management. Several limitations should be noted. The study was conducted at a single center and included a modest sample size, which limits generalizability. The observational design also prevents causal interpretation. Longer follow-up and external validation, especially across patients receiving other biologic classes such as IL-23 inhibitors, will be important for confirming these patterns.[11]

Conclusions

In conclusion, this study demonstrates that the routine baseline TG/HDL-C ratio is a strong, independent predictor of sustained secukinumab efficacy in patients with moderate-to-severe plaque psoriasis. These easily accessible clinical and metabolic features capture the immunometabolic milieu influencing IL-17A inhibition durability. Incorporating the TG/HDL-C ratio into pre-treatment assessment can support patient stratification and optimize long-term management strategies. Future research should investigate whether therapeutic interventions targeting metabolic dysregulation can improve the durability of response to biologic therapies.

Data Sharing Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Acknowledgments

The authors thank all patients who participated in this study and the clinical staff of the Beijing Hospital of Traditional Chinese Medicine for their assistance in patient management and data collection. The authors also acknowledge the statistical and methodological support provided by colleagues in the Department of Dermatology. No external funding or material support influenced the design, conduct, or reporting of this study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors have no conflicts of interest in this work.

References

1. Armstrong AW, Blauvelt A, Callis Duffin K, et al. Psoriasis. *Nat Rev Dis Primers*. 2025;11(1):45. doi:10.1038/s41572-025-00630-5
2. Yu K, Cao C, An F, et al. Analysis and contrast of psoriasis disease burden trends in China and globally from 1990 to 2021. *Front Public Health*. 2025;13:1541292. doi:10.3389/fpubh.2025.1541292
3. Zhang H, Chen M, Cui R, et al. Prevalence of psoriatic arthritis in Chinese population with psoriasis: a multicenter study conducted by experienced rheumatologists. *Chin Med J*. 2023;136(12):1439–1447. doi:10.1097/CM9.0000000000002683
4. Ghoreschi K, Balato A, Enerbäck C, Sabat R. Therapeutics targeting the IL-23 and IL-17 pathway in psoriasis. *Lancet*. 2021;397(10275):754–766. doi:10.1016/S0140-6736(21)00184-7
5. Mastorino L, Dapavo P, Cariti C, et al. Drug survival, safety, and effectiveness of secukinumab for up to 5 years in patients with psoriasis and psoriatic arthritis: a long-term real-life experience. *J Pers Med*. 2024;14(7):718. doi:10.3390/jpm14070718
6. Megna M, Ruggiero A, Torta G, et al. Efficacy and safety of bimekizumab in real-world psoriasis management: data from patients who are biologic-naive vs. biologic-experienced. *Clin Exp Dermatol*. 2024;49(10):1186–1189. doi:10.1093/ced/llae147
7. Foulkes A, McCabe C, Spanopoulos D, et al. Patient characteristics of biologic-experienced and biologic-naive patients with chronic plaque psoriasis receiving bimekizumab. *Br J Dermatol*. 2025;193(Suppl 1):P116. doi:10.1093/bjd/ljaf085.144
8. Bellinato F, Maurelli M, Geat D, et al. Managing the patient with psoriasis and metabolic comorbidities. *Am J Clin Dermatol*. 2024;25(4):527–540. doi:10.1007/s40257-024-00857-0
9. Li M, Gan Y, Cheng H, Wang Z. Correlation between cardiometabolic index and psoriasis: a cross-sectional analysis using NHANES data. *Front Physiol*. 2025;16:1552269. doi:10.3389/fphys.2025.1552269
10. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human participants. *JAMA*. 2025;333(1):71–74. doi:10.1001/jama.2024.21972
11. Kumar A, Bharti A, Shyama, Prasad B. Assessment of lipid abnormalities and atherogenic ratios in clinical subtypes of psoriasis: a hospital-based study. *Int J Pharm Qual Assur*. 2025;16(2):376–381.
12. Schielein MC, Tizek L, Ziehfreund S, et al. Clinical characteristics associated with response to biologics in the treatment of psoriasis: a meta-analysis. *JAMA Dermatol*. 2025;161(7):807–815.
13. Smith A, Karahasan A, Yi D, et al. Biologic therapy and cardiometabolic risk in psoriasis: a retrospective review. *Dermatol Ther*. 2025;15(1):201–212. doi:10.1007/s13555-024-01327-5
14. Cariti C, Dapavo P, Mastorino L, et al. Comparison of Secukinumab and Ixekizumab in psoriasis: a real-life cohort study on the efficacy and drug survival of 445 patients. *J Eur Acad Dermatol Venereol*. 2022;36(3):e233–e235. doi:10.1111/jdv.17766
15. Azimi SI, Jafarzadeh A, Goodarzi A. Systematic review of comparative studies on emerging psoriasis treatments: comparing biologics with biologics, small molecule inhibitors with small molecule inhibitors, and biologics with small molecule inhibitors. *Inflammopharmacology*. 2025;33(5):2377–2407. doi:10.1007/s10787-025-01758-2