Taylor & Francis
Taylor & Francis Group

Expert Review of Clinical Pharmacology

ISSN: 1751-2433 (Print) 1751-2441 (Online) Journal homepage: www.tandfonline.com/journals/ierj20

Biosimilars for chronic plaque psoriasis: a 2026 update

Martina Maurelli , Andrea Danese , Paolo Gisondi & Giampiero Girolomoni

To cite this article: Martina Maurelli , Andrea Danese , Paolo Gisondi & Giampiero Girolomoni (2026) Biosimilars for chronic plaque psoriasis: a 2026 update, Expert Review of Clinical Pharmacology, 19:5, 447-455, DOI: 10.1080/17512433.2026.2667342

To link to this article: https://doi.org/10.1080/17512433.2026.2667342

Published online: 03 May 2026.

EXPERT REVIEW OF CLINICAL PHARMACOLOGY
2026, VOL. 19, NO. 5, 447–455
https://doi.org/10.1080/17512433.2026.2667342

Taylor & Francis
by informa

REVIEW

Biosimilars for chronic plaque psoriasis: a 2026 update

Martina Maurelli, Andrea Danese, Paolo Gisondi and Giampiero Girolomoni

Section of Dermatology and Venereology, Department of Medicine, University of Verona, Verona, Italy

ABSTRACT

Introduction: Biosimilars have emerged as a key strategy to improve access to biologic therapies in moderate-to-severe psoriasis, addressing long-standing limitations related to treatment costs and availability.

Areas covered: This narrative review summarizes the current evidence on biosimilars approved for psoriasis, including tumor necrosis factor inhibitors and ustekinumab, focusing on efficacy, safety, interchangeability, and pharmacoeconomic impact. A literature search was conducted in PubMed/MEDLINE and Embase to identify relevant clinical trials, real-world studies, and pharmacoeconomic analyses published between January 2015 and January 2026. Available data consistently demonstrate that biosimilars are highly similar to their reference biologics, with no clinically meaningful differences in efficacy, safety, or immunogenicity. Pharmacoeconomic studies across Europe confirm substantial cost savings and reduced cost-per-responder, supporting broader and earlier access to biologic therapy.

Expert opinion: Biosimilars should be considered as first-line biologic options when appropriate, not only as cost-saving alternatives. Their wider use may enable earlier intervention, potentially reducing long-term disease burden and improving health system sustainability.

ARTICLE HISTORY
Received 31 March 2026
Accepted 27 April 2026

KEYWORDS
Plaque psoriasis; systemic treatment; biologics; biosimilars; pharmacoeconomy

1. Introduction

Psoriasis is a chronic inflammatory immune-mediated cutaneous disease, affecting 2–3% of the general population in Western countries [1,2]. Genetic predisposition and acquired environmental factors contribute to disease pathogenesis. HLA-C*06:02 risk allele is the major genetic susceptibility factor associated to psoriasis [2–4]. Environmental factors, such as streptococcal infection, psychological stress, skin trauma, medications, obesity, smoking, alcohol consumption and air pollution, have been reported as potential triggers in predisposed individuals [4–10].

The activation and expansion of Th17 lymphocytes, under the guidance of IL-23 released by activated myeloid cells, is crucial for the development of skin lesions. Plasmacytoid dendritic cells, natural killer cells, and macrophages secrete cytokines, such as IFN-α, IL-1β and TNF-α, leading to the activation of myeloid dendritic cells in the early phases of the disease. These induce the activation and expansion of autoreactive T lymphocytes. Activated dendritic cells produce IL-12 and IL-23, which regulate the differentiation and expansion of T helper lymphocytes (Th1, Th17, Th22), which produce the pathogenetic cytokines, IL-17, TNF, IFN-γ and IL-22 [11]. These cytokines act on keratinocytes to promote the release of autocrine growth factors and then hyperproliferation as well as the release of chemokines and cytokines, which amplify and perpetuate inflammation, finally producing the psoriasis plaque [1,4].

Moderate-to-severe psoriasis is treated with systemic drugs, which include conventional systemic drugs, biologicals and small molecules (Table 1). Biologics are in general very effective and have changed dramatically the possibility of effective and sustained disease control [12]. However, a substantial proportion of patients may have limited access to therapy due to economics, health policies and clinical considerations. The patent of some biological agents has expired, and biosimilars are available, offering the same drug at a more convenient cost. Biosimilars benefit from a less expensive tailored clinical development programs and extrapolation of indications, making them more affordable than their reference biologics. This narrative review highlights the impact of these drugs on patients access to efficacious therapy and the pharmacoeconomic benefits.

1.1. Literature search methodology

A literature search was conducted in PubMed/MEDLINE and Embase to identify relevant studies on biosimilars in psoriasis published between January 2015 and January 2026. Search terms included 'psoriasis,' 'biosimilars,' 'adalimumab,' 'infliximab,' 'etanercept,' and 'ustekinumab.' Both randomized controlled trials and real-world studies were considered.

2. Treatment of moderate-to-severe chronic plaque psoriasis

Systemic treatments for moderate-to-severe chronic plaque psoriasis are based on conventional systemic drugs, biologicals and small molecules (Table 1). According to the European Medicine Agency (EMA), some drugs have a first line label

CONTACT Giampiero Girolomoni giampiero.girolomoni@univr.it Section of Dermatology and Venereology, Department of Medicine, University of Verona, Piazzale A. Stefani 1, Verona 37126, Italy

Article highlights

- Effective treatment of moderate-to-severe plaque psoriasis is currently based on several biologics.
- Biosimilars of etanercept, adalimumab, infliximab and ustekinumab have demonstrated comparable efficacy and safety to originator biologics.
- Biosimilars can significantly reduce treatment costs in psoriasis.
- They markedly improve access to biologic therapies across healthcare systems.
- Pharmacoeconomic benefits, however, can differ significantly across countries.
- Switchback is uncommon and often driven by nocebo effect.
- Biosimilars may enable earlier intervention and reduce long-term disease burden and complications.

whereas others a second line label, meaning they should be prescribed when first line drugs are contraindicated, or had been unsuccessfully or had given side effects [12,13]. These drugs are available in different forms of administrations, including subcutaneous, intravenous or oral [12]. Biologicals and small molecules, in particular, have demonstrated effectiveness and tolerability in the long-term use and, some of them, have the potential to modify the natural course of the disease. These characteristics have markedly improved the possibility of effective treatment and the quality of life of patients [14]. Monoclonal antibodies inhibit selectively pathogenetic cytokines, such as TNF-α, IL-12/23, IL-17 or IL-23, thus blocking the inflammatory cascade and epidermal hyperplasia [1]. Small molecules have in general a broader mechanism of action by inhibiting signal transduction pathways. Recently, however, oral drugs selectively blocking cytokines such as IL-23 or IL-17 have been investigated such as icotrokinra, which is an oral small molecule drug inhibiting selectively IL-23 [15].

Given the availability of several systemic treatments, there is the possibility of tailoring the therapy according to the characteristics of the patients, the disease and the drug (Table 2) [1]. One major criterion for selecting a drug is the presence of comorbidities, such as psoriatic arthritis, other immune-mediated inflammatory diseases (IMIDs) or cardiometabolic comorbidities. Indeed, drugs can be beneficial or detrimental to certain comorbidities. Obesity is also an important element to consider [8,16,17]. Apremilast may have a beneficial effect on obese patients [18]. In contrast, TNF-α inhibitors may increase body weight. Infliximab and ustekinumab have a variable dosage weight based [17]. Other important variables to consider are sex and willingness to procreate, fear of side effects and not least patient preferences (e.g. oral vs injectable drugs) [1,19].

3. Biosimilars

Biosimilars are drugs derived from proteins developed with a recombinant DNA technology through a new process production with reverse engineering to achieve high similarity to the originator (Figure 1). A biosimilar drug is a biologic medical product highly similar to the originator; it is not an exact copy of the approved reference biologic drug, but there are no meaningful clinical differences in safety and efficacy [20,21]. In contrast, generics are exact chemical copies of

Table 1. Systemic treatments with first- and second-line label for moderate-to-severe psoriasis, according to EMA [37].

Systemic treatments	First- or second-line label
Acitretin	First-line treatment; before biologics
Cyclosporine	
Fumarate	
Methotrexate	
Adalimumab (anti-TNF-α)	First-line treatment;
Certolizumab (anti-TNF-α)	in case of inadequate response to conventional agents, or when they are contraindicated or not tolerated
Secukinumab (anti-IL-17)	
Ixekizumab (anti-IL-17)	
Brodalumab (anti-IL-17)	
Risankizumab (anti-IL-23)	
Guselkumab (anti-IL-23)	
Tildrakizumab (anti-IL-23)	
Decravacitinib (TYK2 inhibitor) - oral	
Apremilast (PDE4) - oral	Second-line treatment
Etanercept (anti-TNF-α)	
Infliximab (anti-TNF-α)	
Ustekinumab (anti-IL-12/23)	

Table 2. Principal characteristics to be considered in the choice of treatment of plaque psoriasis.

Disease-related factors	Patient-related factors	Treatment-related factors
Objective disease score	Age and gender	Short- and long-term response rates
Onset of new lesions	Body mass index	Safety
Skin areas involved	Occupation	Flexibility
Frequency of relapses	Treatment history	Cost effectiveness
Itch or other symptoms	Impact on quality of life	Long-term risk/benefit
Psoriatic arthritis	Likelihood of adherence	
Cardiometabolic diseases	Patient expectations	
	Long-term remission	
	Fear of side effects	

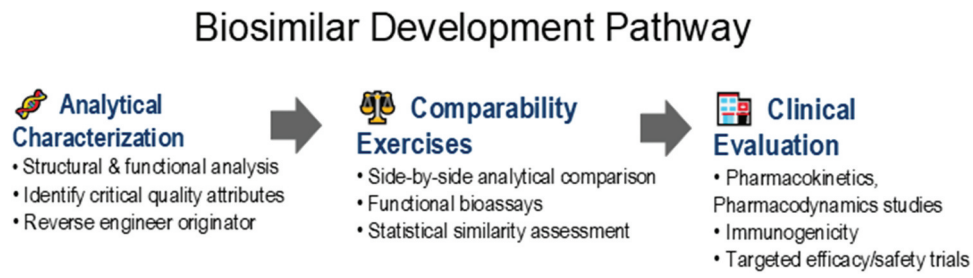

Figure 1. The biosimilar development pathway.

The stepwise development process for biosimilar medicines, beginning with extensive analytical characterization of the reference product, followed by comparative analytical and functional studies in which the proposed biosimilar is evaluated side-by-side with the originator to demonstrate a high degree of similarity. Finally, targeted clinical evaluation is conducted to verify the absence of clinically meaningful differences between the biosimilar and the reference product.

simpler, small-molecule drugs. While generics are identical, biosimilars may have minor differences, requiring rigorous clinical testing to ensure safety. Biobetters are intentionally modified, superior versions of biologics, offering enhanced efficacy or safety over the original. Biosimilar benefit of the extrapolation principle, allowing a biosimilar to be approved for indications of the reference product not directly studied in comparative clinical trials. Biological drugs have revolutionized psoriasis treatment due to their efficacy and safety profile, but their high-cost limits may limit access to patients in need. Currently, biosimilars of adalimumab, infliximab, etanercept and ustekinumab are on the market in Europe (Table 3). The overall body of evidence from randomized clinical trials and real-world studies consistently demonstrates no clinically meaningful differences in efficacy, safety, and immunogenicity between biosimilars and their reference biologic (Table 4). All biosimilars adopt the same dosing schedule as the reference product [22]. Biosimilars of secukinumab are in development [23].

The originator adalimumab was the first biologic approved for moderate-to-severe plaque psoriasis in 2008 from U.S. Food and Drug Administration (FDA). The first biosimilar of adalimumab approved in 2017 by the European Medicines Agency (EMA) was ABP 501 [22,24–26]. The adalimumab

Table 3. List of reference biologics and biosimilars approved for the treatment of moderate-to-severe plaque psoriasis [37].

Reference biologic	Biosimilar	Trade name	Approval year
Adalimumab	ABP 501	Amgevita/Solymbic	2017
	SB5	Imraldi	2017
	BI 695501	Cyltezo	2017
	FKB327	Hulio	2018
	GP2017	Hyrimoz/Hefiya/Halimatoz	2018
	MSB11022	Idacio/Kromeya	2019
	PF-06410293	Amsparity	2020
	CT-P17	Yuflyma	2021
	AVT02	Hukyndra/Libmyris	2021
Infliximab	CT-P13	Inflectra/Remsima	2013
	SB2	Flixabi	2016
	PF-06438179/GP1111	Ixifi/Zessly	2017
Etanercept	SB4	Benepali	2016
	GP2015	Erelzi	2017
	YLB113	Nepexto	2020
Ustekinumab	AVT04	Uzpruvo	2024
	SB17	Pyzchiva/Eksunbi	2024
	ABP 654	Wezenla	2024
	FYB202	Fymskina	2024
	DMB-3115	Imuldosa	2024
	CT-P43	SteQeyma	2024
	FYB202	Otulfi	2024
	DMB-3115	Absimky	2024
	KFCE	Yesintek	2025
	CT-P43	Qoyvolma	2025
	BAT2206	Usymro	2025

Table 4. Summary of efficacy and safety studies of biosimilars versus originators in plaque psoriasis.

Biosimilar	Type of evidence	Key comparative findings (biosimilar vs originator)	References
Adalimumab	Phase III, real-world	Comparable PASI75/90, similar safety and immunogenicity	[24,27,28]
Infliximab	Phase III, registries, real word	Equivalent efficacy and drug survival, no increased adverse events	[29–31]
Etanercept	Phase II, registries, observational	Similar long-term outcomes and patient-reported outcomes	[33,35]
Ustekinumab	Phase III, observational	Equivalent PASI responses and safety up to week 52	[36,38,39]
Switch studies	Real-world	No significant loss of efficacy; nocebo effect may influence discontinuation	[28,46–49]

regimen for moderate-to-severe plaque psoriasis is 40 mg subcutaneously every other week following 80 mg loading dose, and Phase III trials and real-world data have consistently shown that adalimumab biosimilars achieve psoriasis area and severity index improvement of at least 75% (PASI75) and 90% (PASI90) rates comparable to originator adalimumab, without clinically relevant differences in safety, drug survival, or immunogenicity, further supporting clinical interchangeability [24,27,28].

The originator infliximab was the first biologic approved for moderate-to-severe plaque psoriasis in 2006 from the FDA. The standard regimen consists of 5 mg/kg administered intravenously at weeks 0, 2 and 6, followed by maintenance infusions every 8 weeks. The first infliximab biosimilar approved by EMA was CT-P13 in 2013 [29,30]. Clinical evidence from both randomized-controlled trials and real-world cohorts has consistently demonstrated equivalence in efficacy, safety and immunogenicity between infliximab biosimilars and originator infliximab in psoriasis and other IMIDs, including comparable rates of PASI75 responses, supporting their interchangeability in routine care [31,32].

The originator etanercept received its marketing authorization by EMA in 2009 for moderate-to-severe plaque psoriasis. The first biosimilar authorized by EMA was SB4 in 2016, followed by GP2015 in 2017 and YLB113 in 2020 [33]. The regimen for the treatment of chronic plaque psoriasis is 50 mg subcutaneously once weekly (or 50 mg twice weekly during induction in selected protocols); etanercept originator and biosimilars are administered using identical dosing schedules [34]. Multiple comparative studies and longitudinal registry analyses have confirmed clinical similarity between biosimilars and originator etanercept with respect to efficacy, drug survival, safety, immunogenicity, and patient-reported outcomes. Importantly, no clinically relevant loss of response was observed after switching, and PASI75/90 response proportions were aligned with those observed with reference etanercept [35]. These data support both the clinical equivalence of etanercept biosimilars and their suitability for routine clinical use, including in patients already stable on the originator.

The first ustekinumab biosimilar approved by EMA was AVT04 in 2024 [36]. Subsequently, SB17 and several others have been approved by EMA in 2025 [37]. The ustekinumab regimen is weight-based; in particular, 45 mg subcutaneously at weeks 0 and 4, then every 12 weeks, with 90 mg for patients >100 kg. Ustekinumab biosimilars follow the same dosing and administration schedule [34]. Phase III psoriasis studies demonstrated that ustekinumab biosimilars achieve PASI 50/75/90/100 responses comparable to those seen with the originator (Stelara), and similar efficacy equivalence through week 52 including in switching arms [38,39]. These data support again therapeutic similarity and a high degree of functional interchangeability, particularly relevant in the context of expanding biological demand and budget-sustainability considerations.

Secukinumab patent protection for plaque psoriasis is expected to expire in Europe around 2030, with key U.S. patent expiry projected around 2032, unless potential extensions. There are currently no approved secukinumab biosimilars on the market, but several are in advanced development, with companies like Celltrion (CT-P55), BioThera (BAT2306), Mabtech (CMAB015), and Livzon (LZM012) conducting Phase 3 clinical trials [23,40].

4. Pharmacoeconomy

Biosimilars are a cost-effective alternative to off-patent biologic therapies, and there is considerable evidence to suggest they offer a valuable pharmacoeconomic strategy to lower healthcare costs in patients with psoriasis [21]. Cost-per-responder studies have confirmed the economic advantages of using biosimilars.

A real-world cost-per-responder study conducted in the Netherlands and Sweden, evaluating all classes of biologics for moderate-to-severe psoriasis, underlined the importance of biosimilars for the treatment of these patients [41]. Adalimumab showed the lowest one-year cost-per-responder compared to the other biologics. These data were based on Dutch list prices and discounts on originator of adalimumab and ustekinumab because of biosimilar availability. Swedish prices were similar to Dutch data, but more transparent. Furthermore, price transparency is essential for the dermatologist in choosing the best therapy for these patients, because price fluctuations have a great impact on cost-effectiveness [41]. Indeed, price variability in Europe is important to consider when thinking about cost-effectiveness. A study conducted in five academic centers in Denmark, France, Germany, Italy and Spain showed that Germany had the highest biosimilar prices for all TNF-α inhibitors, with costs per unit dose 15–20 times more expensive compared to the costs in Italy and Spain, which reported the lowest prices [42]. The study revealed substantial inter-country variability in biosimilar prices, related to different national and local pricing policies. Mechanisms such as mandatory discounts, progressive price reductions, and negotiated tendering all contribute to these disparities [42]. A cost-per-responder analyses conducted from the perspective of the Italian National Health System, comparing adalimumab biosimilars (MSB11022, ABP501) with the originator adalimumab and methotrexate was conducted using the efficacy data from a Phase 3 RCT trial (Champion study) [42]. Considering the PASI75 response rate at 16 weeks, the cost per responder for MSB11022 (Amgevita) and ABP 501 (Idacio) compared to the originator was 30–40% lower. A real-world study on 712 patients with moderate-to-severe psoriasis, comparing MSB11022 (Amgevita) and ABP 501 (Idacio), and subcutaneous methotrexate confirmed that biosimilars approached the cost-effectiveness of methotrexate, while avoiding several tolerability issues such as gastrointestinal tract side effects, hepatotoxicity and teratogenicity. The results of these studies strongly support first-line treatment with biosimilars as a cost-effective therapeutic strategy [43,44].

A very recent study in Denmark showed a rapid switch from ustekinumab originator to biosimilars following patent expiration, resulting in overall cost savings of almost 70% and an almost 90% reduction in price for ustekinumab [45].

The actual prices paid by healthcare systems are often not fully reflected by publicly available list prices. Confidential discounts, tender-based procurement systems, and managed

entry agreements may considerably reduce the price gap between originators and biosimilars. As a result, in some real-world settings, the net price difference may appear much smaller than expected. This variability should be considered when interpreting pharmacoeconomic analyses and comparing cost-effectiveness across different healthcare systems [42]. Reimbursement policies also play a key role in shaping access to biologic therapies. In several healthcare systems, preferential reimbursement for biosimilars is implemented through formulary restrictions or mandatory switching policies, primarily driven by cost-containment strategies [42]. Conversely, in certain contexts, originator biologics may still be preferred due to existing contractual agreements or clinician familiarity. A balanced approach integrating both cost-efficiency and individualized patient care remains essential.

5. Switchback

Therapeutic switchback refers to the return from a biosimilar to its reference biologic after an initial non-medical switch, most driven by formulary changes or cost-containment strategies [46]. Overall, reported switchback rates were relatively low (<20%), although they tended to increase with a longer follow-up [47]. The predominant reasons for switchback across studies on different IMIDS were patient-reported loss of effectiveness and subjective adverse events, consistent with a major contribution of the nocebo (negative placebo) effect [47]. Importantly, in patients with rheumatoid arthritis, axial spondyloarthritis or psoriatic arthritis, objective inflammatory parameters (C-Reactive Protein-CRP, swollen joint counts, Health Assessment Questionnaire scores) were usually unchanged at the time of switchback, despite perceived worsening [48]. Features associated with switchback included sex, with females more likely, longer prior biologic treatment duration, higher baseline disease activity, and a diagnosis of rheumatoid arthritis [49]. Data on switching back in patients with psoriasis is limited, but apparently switching back from biosimilar to originator is a rare event in patients with psoriasis [28]. Taking together, current data suggests that most patients remain on biosimilars after switching from originator and that expectation-driven subjective symptoms, rather than true pharmacological inferiority, support a large proportion of switchbacks [50].

6. Emerging treatments for psoriasis

Emerging treatments for psoriasis are shifting toward highly targeted, convenient options, including novel topicals, oral IL-23 and IL-17 inhibitors, and PDE4 and TYK2 inhibitors. Framing these innovations within the broader therapeutic landscape allows for a more comprehensive understanding of the complementary role of biosimilars in modern clinical practice.

Conventional topical drug delivery for inflammatory skin diseases, including psoriasis, is limited by poor skin penetration and the risk of long-term adverse effects. In this context, nanotechnology-based approaches have emerged as promising alternatives [51–53]. Topical metal nanoparticles have demonstrated favorable efficacy and safety profiles in the treatment of inflammatory dermatoses, offering improved drug stability, targeted delivery, and reduced systemic exposure [51]. Similarly, carrier-free nanomedicine platforms have shown significant therapeutic potential by reducing inflammatory cell infiltration and suppressing keratinocyte hyperproliferation [52]. An innovative example is represented by membrane-encapsulated nano-brakes derived from interferon-γ (IFN-γ)-stimulated macrophages. IFN-γ activation enhances the expression of pro-inflammatory cytokine receptors and PD-L1 on macrophage membranes. When applied to psoriatic lesions, these biomimetic nanoparticles interrupt the inflammatory cascade, reduce T-cell proliferation and activation, and promote the restoration of immune homeostasis [53].

Numerous monoclonal antibodies-based treatments have validated the IL-23/Th17 axis as central clinical target in psoriasis developments and persistence. These agents still suffer from limitations including a lack of oral bioavailability, high cost of production, cold conservation and immunogenicity. Oral small molecules drugs targeting IL-23 or IL-17 can address these limitations and are indeed in advanced development stage, including icotrokinra [15,54]. Oral IL-17 inhibitors under active investigation include ASC50, LY3509754, and other early-stage compounds (DC-806, DC-853) that show promise in improving skin inflammation and psoriasis [55].

Another area of interest involves the development of small-molecule modulators of intracellular signaling pathways such as phosphodiesterase 4 inhibitors (roflumilast, orismilast, mufemilast) and Janus kinase inhibitors (zasocitinib) [56]. Finally, long-acting biologics, requiring one injection every 6 to 12 months, are under active investigations, improving quality of life and convenience [57]. In addition to efficacy, safety considerations remain crucial for topical therapies. Conventional topical treatments may be associated with local adverse reactions, including skin irritation, burning sensation, contact dermatitis, and, in the case of prolonged corticosteroid use, skin atrophy and barrier impairment. Emerging delivery systems, such as nanotechnology-based formulations, may improve drug penetration and reduce systemic exposure; however, their long-term safety profile is still under investigation. Recent studies have highlighted the importance of carefully evaluating both local tolerability and potential systemic absorption when introducing novel topical approaches, particularly in chronic inflammatory dermatoses such as psoriasis [58–60]. These considerations highlight the need for long-term safety data and careful post-marketing surveillance of emerging topical therapies.

7. Conclusion

Biosimilars of TNF inhibitors and ustekinumab offer clinically equivalent efficacy, safety, and treatment durability to their originator biologics in psoriasis, and can be used in both biologic-naïve patients and those transitioning from reference products. Their lower acquisition cost and market-level price competition substantially improve the affordability and sustainability of biologic treatment strategies, supporting broader and earlier access to effective therapy. First-line treatment of psoriasis with biologics may have important benefits including early and sustained improvement in quality of life, and, more important, prevent the development of psoriatic arthritis, as well as cardiovascular diseases [50,61]. Continued optimization of communication and patient engagement strategies will be

essential to maximize long-term persistence on biosimilars and fully realize their clinical and health-economic value. Many emerging treatments will further expand the spectrum of possible therapies, but low-cost biosimilars will still offer an effective and convenient strategy for long time.

8. Expert opinion

Biosimilars are likely to become one of the most important levers for improving the long-term management of moderate-to-severe psoriasis, not because they introduce a new mechanism of action, but because they can materially change when, and in whom, effective biologic therapy is used. This distinction is crucial. In psoriasis, therapeutic innovation has often been discussed primarily in terms of higher efficacy thresholds, faster skin clearance, and convenience. However, for many patients and healthcare systems, the real unmet need is not simply access to the newest molecule, but timely access to a highly effective systemic treatment before years of undertreatment translate into avoidable cumulative burden, impaired quality of life, and possibly irreversible comorbidity. In this context, the major value of biosimilars is strategic rather than merely economic. A key message emerging from current evidence is that biosimilars should not be considered 'fallback' options reserved only for cost-constrained settings. Their clinical performance in psoriasis and in other immune-mediated inflammatory diseases strongly supports their use as genuine first-line biologic options when a TNF-inhibitor or ustekinumab is an appropriate therapeutic choice. In everyday practice, clinicians still sometimes perceive biosimilars as acceptable mainly after failure of a conventional systemic drug, or as instruments for non-medical switching in stable patients. This is too narrow a view. If biosimilars reduce treatment cost sufficiently, they can allow biologic treatment to be initiated earlier in the disease course, and that may ultimately be their greatest contribution. This issue deserves more attention in psoriasis than it has received so far. Moderate-to-severe psoriasis is often still managed with a stepwise escalation model that may delay optimal control, especially in patients with high-impact disease, recurrent flares, difficult-to-treat sites, substantial psychosocial burden, or early musculoskeletal symptoms. Yet the concept of cumulative life course impairment is now well established in psoriasis. The longer active disease remains insufficiently controlled, the greater the burden on social functioning, mental health, work productivity, and potentially systemic inflammation. In this scenario, the availability of lower-cost biologics may help close the gap between 'eligibility for biologic therapy' and 'actual access to biologic therapy.' This is where biosimilars may exert a true disease-modifying impact at the population level.

In our opinion, future psoriasis treatment algorithms should increasingly distinguish between 'high innovation' drugs and 'high access' drugs, recognizing that both categories are clinically valuable. IL-17 and IL-23 inhibitors have clearly redefined efficacy expectations and remain the preferred option for many patients, particularly when complete or near-complete skin clearance is a realistic treatment target. However, this does not diminish the importance of biosimilars. Rather, biosimilars can broaden the biologic treatment base and create a more sustainable therapeutic ecosystem, in which highly effective treatment is not restricted to a small proportion of patients. From a health-system perspective, this may be more transformative than incremental efficacy gains at the top end of available therapies. Another issue that deserves a pragmatic interpretation is switching. The accumulated evidence suggests that most switches from originator to biosimilar are clinically successful and that true pharmacologic inferiority is not the major concern. Instead, patient expectations, communication style, and trust in the therapeutic process are often more important determinants of persistence than molecular differences. This should not be underestimated. In psoriasis, where treatment satisfaction and perceived control are central to long-term adherence, the nocebo effect can become clinically relevant even in the absence of objective worsening. In our view, the success of biosimilar implementation depends as much on communication strategy as on regulatory science. A confident, transparent, and non-defensive explanation from the clinician is likely to be more effective than simply reassuring the patient that 'it is the same drug.' Future research should therefore include not only comparative effectiveness and pharmacoeconomic studies but also implementation of science and patient-centered communication interventions. There are also several important research directions that are likely to become increasingly relevant over the next few years. First, more psoriasis-specific long-term registry data are needed, especially for ustekinumab biosimilars, where real-world evidence is still less mature than for anti-TNF agents. Second, it will be important to move beyond drug survival as the only practical endpoint and examine whether earlier access to biosimilar biologics translates into measurable reductions in cumulative disease burden, healthcare utilization, psoriatic arthritis progression, or cardiometabolic outcomes. These are difficult questions, but they are clinically meaningful and align better with the real promise of biosimilars. Third, the future role of biosimilars should be interpreted within the context of a rapidly diversifying therapeutic landscape. Oral cytokine inhibitors, TYK2 inhibitors, advanced topical platforms, and potentially longer-acting biologics are all likely to reshape treatment expectations. Nevertheless, even as therapeutic innovation expands, affordability will remain a central determinant of access. For this reason, biosimilars are unlikely to become less relevant; on the contrary, they may become more important as anchor therapies that support sustainable sequencing and individualized treatment strategies.

Overall, we believe the next phase of biosimilar adoption in psoriasis should move beyond the question of equivalence, which is now largely settled, and focus instead on optimization: how to use these agents earlier, more intelligently, and in ways that maximize both patient benefit and health-system value. If this shift occurs, biosimilars will not simply represent cheaper versions of existing biologics, but an important tool for redefining what 'timely effective care' should look like in psoriasis. Future treatment strategies should integrate biosimilars not only as cost-saving alternatives but as tools to enable earlier intervention and potentially modify long-term disease burden at a population level.

Author contributions

CRediT: **Martina Maurelli:** Conceptualization, Methodology, Writing – original draft; **Andrea Danese:** Writing – original draft; **Paolo Gisondi:** Conceptualization; **Giampiero Girolomoni:** Conceptualization, Validation.

Funding

This manuscript has received funding from the European Union's H2020 Research and Innovation Program under grant agreement number 848028 (DoCTIS; Decision on optimal combinatorial therapies in IMIDs using systems approaches'), and from the European Union—Next Generation EU—PNRR- MAD-2022–12375740 M6C2—Investimento 2.1 Valorizzazione e potenziamento della ricerca biomedica del SSN, funded by Italian Ministry of Health. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Declaration of Interests

P Gisondi has received honoraria for consultancy, advisory boards, or lectures from AbbVie, Amgen, Almirall, Bristol-Myers Squibb, Boehringer-Ingelheim, Eli Lilly, Novartis, and UCB. G Girolomoni has received personal fees for consultancy or lectures from AbbVie, Almirall, Boehringer-Ingelheim, Bristol-Myers Squibb, Galderma, Eli Lilly, Incyte, Johnson & Johnson Innovative Medicine, Leo Pharma, Merck Serono, Novartis, Pfizer, Samsung Bioepis, and Sanofi. The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

Reviewer disclosures

Peer reviewers on this manuscript have no relevant financial or other relationships to disclose.

References

Papers of special note have been highlighted as either of interest (•) or of considerable interest (••) to readers.

1. Maurelli M, Gisondi P, Girolomoni G. Tailored biological treatment for patients with moderate-to-severe psoriasis. Expert Rev Clin Immunol. 2023;19(1):37–43. doi: 10.1080/1744666X.2023.2141226
2. World Health Organization. Global report on psoriasis. Geneva: World Health Organization; 2016.
3. Griffiths CEM, Armstrong AW, Gudjonsson JE, et al. Psoriasis. Lancet. 2021;397(10281):1301–1315. doi: 10.1016/S0140-6736(20)32549-6
4. Morelli M, Galluzzo M, Madonna S, et al. HLA-Cw6 and other HLA-C alleles, as well as MICB-DT, DDX58, and TYK2 genetic variants associate with optimal response to anti-IL-17A treatment in patients with psoriasis. Expert Opin Biol Ther. 2021;21(2):259–270. doi: 10.1080/14712598.2021.1862082
5. Potestio L, Lauletta G, Tommasino N, et al. Risk factors for psoriasis flares: a narrative review. Psoriasis (Auckl). 2024;14:39–50. doi: 10.2147/PTT.S323281
6. Liu S, He M, Jiang J, et al. Triggers for the onset and recurrence of psoriasis: a review and update. Cell Commun Signal. 2024;22(1):108. doi: 10.1186/s12964-023-01381-0
7. Sitton B, Walker T, Mital R, et al. Antistreptococcal treatment of psoriasis: a systematic review. Arch Dermatol Res. 2024;316(7):363. doi: 10.1007/s00403-024-03051-8
8. Wang J, Yu Y, Liu L, et al. Global prevalence of obesity in patients with psoriasis: an analysis in the past two decades. Autoimmun Rev. 2024;23(6):103577. doi: 10.1016/j.autrev.2024.103577
9. Hawkes JE, Yan BY, Chan TC, et al. Discovery of the IL-23/IL-17 signaling pathway and the treatment of psoriasis. J Immunol. 2018;201(6):1605–1613. doi: 10.4049/jimmunol.1800013
10. Bellinato F, Maurelli M, Geat D, et al. The patient with psoriasis and metabolic comorbidities. Am J Clin Dermatol. 2024;25(4):527–540. doi: 10.1007/s40257-024-00857-0
11. Albanesi C, Scarponi C, Pallotta S, et al. Chemerin expression marks early psoriatic skin lesions and correlates with plasmacytoid dendritic cell recruitment. J Exp Med. 2009;206(1):249–258. doi: 10.1084/jem.20080129
12. Nast A, Smith C, Spuls PI, et al. EuroGuiDerm guideline on the systemic treatment of Psoriasis vulgaris – part 1: treatment and monitoring recommendations. J Eur Acad Dermatol Venereol. 2020;34(11):2461–2498. doi: 10.1111/jdv.16915
13. Gisondi P, Campanati A, Ganzetti G, et al. Biologic therapy in psoriasis: safety profile. Curr Drug Saf. 2016;11(1):4–11. doi: 10.2174/1574886310666151014115532
14. De Sarro C, Bosco F, Gagliardi A, et al. Prescribing pattern and safety profile of biological agents for psoriasis in real-world practice: a four-year Calabrian pharmacovigilance analysis. Pharmaceutics. 2024;16(10):1329. doi: 10.3390/pharmaceutics16101329
15. Bissonnette R, Soung J, Hebert AA, et al. Oral icotrokinra for plaque psoriasis in adults and adolescents. N Engl J Med. 2025;393(18):1784–1795. doi: 10.1056/NEJMoa2504187
16. Wu MY, Yu CL, Yang SJ, et al. Change in body weight and body mass index in psoriasis patients receiving biologics: a systematic review and network meta-analysis. J Am Acad Dermatol. 2020;82(1):101–109. doi: 10.1016/j.jaad.2019.07.103
17. Pirro F, Caldarola G, Chiricozzi A, et al. Impact of body mass index on the efficacy of biological therapies in patients with psoriasis: a real-world study. Clin Drug Investig. 2021;41(10):917–925. doi: 10.1007/s40261-021-01080-z
18. Kaushik SB, Lebwohl MG. Psoriasis: which therapy for which patient: psoriasis comorbidities and preferred systemic agents. J Am Acad Dermatol. 2019;80(1):27–40. doi: 10.1016/j.jaad.2018.06.057
19. Jeong YD, Jo H, Cho H, et al. Biologics use for psoriasis during pregnancy and its related adverse outcomes in pregnant women and newborns: findings from who pharmacovigilance study. Int Arch Allergy Immunol. 2025;186(6):579–593. doi: 10.1159/000542217
20. Gisondi P, Kirchhoff CF, Wang XM, et al. Biosimilars: key regulatory considerations and similarity assessment tools. Biotechnol Bioeng. 2017;114(12):2696–2705. doi: 10.1002/bit.26438
21. Carrascosa JM, Jacobs I, Petersel D, et al. Biosimilar drugs for psoriasis: principles, present, and near future. Dermatol Ther (Heidelb). 2018;8(2):173–194. doi: 10.1007/s13555-018-0230-9
• **Overview of biosimilars in psoriasis and their clinical application.**
22. Menter A, Tyring SK, Gordon K, et al. Adalimumab therapy for moderate to severe psoriasis: a randomized, controlled phase iii trial. J Am Acad Dermatol. 2008;58(1):106–115. doi: 10.1016/j.jaad.2007.09.010
23. Celltrion Healthcare. Phase I results for secukinumab biosimilar CT-P55 [Internet]. 2025 [cited 2025 Dec 1]. Available from: https://www.celltrionhealthcare.com
24. Papp K, Bachelez H, Costanzo A, et al. Clinical similarity of biosimilar ABP 501 to adalimumab in the treatment of patients with moderate to severe plaque psoriasis: a randomized, double-blind, multicenter, phase III study. J Am Acad Dermatol. 2017;76(6):1093–1102. doi: 10.1016/j.jaad.2016.12.014
25. Weinblatt ME, Baranauskaite A, Niebrzydowski J, et al. Phase III randomized study of SB5, an adalimumab biosimilar, versus reference adalimumab in patients with moderate-to-severe rheumatoid

arthritis. Arthritis Rheumatol. 2018;70(1):40–48. doi: 10.1002/art.40336
26. Genovese MC, Kellner H, Arai Y, et al. Long-term safety, immunogenicity and efficacy comparing FKB327 with the adalimumab reference product in patients with active rheumatoid arthritis: data from randomised double-blind and open-label extension studies. RMD Open. 2020;6(1):e000987. doi: 10.1136/rmdopen-2019-000987
27. Blauvelt A, Lacour JP, Fowler JF, et al. Phase III randomized study of the proposed adalimumab biosimilar GP2017 in psoriasis: impact of multiple switches. Br J Dermatol. 2018;179(3):623–631. doi: 10.1111/bjd.16890
28. Loft N, Egeberg A, Rasmussen MK, et al. Outcomes following a mandatory nonmedical switch from adalimumab originator to adalimumab biosimilars in patients with psoriasis. JAMA Dermatol. 2021;157(6):676–683. doi: 10.1001/jamadermatol.2021.0221
•• **Landmark real-world study demonstrating outcomes after switching to biosimilars.**
29. Park W, Yoo DH, Jaworski J, et al. Comparable long-term efficacy, as assessed by patient-reported outcomes, safety and pharmacokinetics, of CT-P13 and reference infliximab in patients with ankylosing spondylitis: 54-week results from the randomized, parallel-group PLANETAS study. Arthritis Res Ther. 2016;18(1):25. doi: 10.1186/s13075-016-0930-4
30. Yoo DH, Hrycaj P, Miranda P, et al. A randomised, double-blind, parallel-group study to demonstrate equivalence in efficacy and safety of CT-P13 compared with innovator infliximab when coadministered with methotrexate in patients with active rheumatoid arthritis: the PLANETRA study. Ann Rheum Dis. 2013;72(10):1613–1620. doi: 10.1136/annrheumdis-2012-203090
31. Gisondi P, Bianchi L, Conti A, et al. Infliximab biosimilar CT-P13 in the treatment of chronic plaque psoriasis: data from the psobiosimilars registry. Br J Dermatol. 2017;177(6):e325–e326. doi: 10.1111/bjd.15659
32. Kameda H, Uechi E, Atsumi T, et al. A comparative study of PF-06438179/GP1111 (an infliximab biosimilar) and reference infliximab in patients with moderate to severe active rheumatoid arthritis: a subgroup analysis. Int J Rheum Dis. 2020;23(7):876–881. doi: 10.1111/1756-185X.13846
33. Schmalzing M, Askari A, Girolomoni G, et al. Clinical and real-world evidence on etanercept biosimilar switching: a narrative literature review of efficacy and safety. Adv Ther. 2025;43(1):49–75. Epub ahead of print. doi: 10.1007/s12325-025-03367-5
• **Comprehensive review of switching outcomes in clinical practice.**
34. Leonardi CL, Powers JL, Matheson RT, et al.; Etanercept psoriasis study group. Etanercept as monotherapy in patients with psoriasis. N Engl J Med. 2003;349(21):2014–2022. doi: 10.1056/NEJMoa030409
35. Giordano D, Capalbo A, Gagliostro N, et al. Biosimilar versus originator etanercept: a real-life clinical study. Ital J Dermatol Venerol. 2022;157(4):318–324. doi: 10.23736/S2784-8671.22.07224-3
36. Cheon JH, Duk Ye B, Armuzzi A, et al. The 'totality of evidence' and 'extrapolation' of SB17, a ustekinumab biosimilar. Expert Opin Biol Ther. 2025;25(6):615–632. doi: 10.1080/14712598.2025.2508838
37. European Medicines Agency. Steqeyma - EPAR product information 2025. [cited 2025 Dec 1]. Available from: https://www.ema.europa.eu/en/medicines/human/EPAR/steqeyma
38. Feldman SR, Narbutt J, Girolomoni G, et al. Biosimilar SB17 versus reference ustekinumab in moderate to severe plaque psoriasis after switching: phase 3 study results up to week 52. J Dermatolog Treat. 2024;35(1):2436607. doi: 10.1080/09546634.2024.2436607
•• **Phase III trial confirming efficacy equivalence of ustekinumab biosimilars.**
39. Papp KA, Lebwohl MG, Thaçi D, et al. Efficacy and safety of candidate biosimilar ct-p43 versus originator ustekinumab in moderate to severe plaque psoriasis: 28-week results of a randomised, active-controlled, double-blind, phase III study. BioDrugs. 2024;38(1):121–131. doi: 10.1007/s40259-023-00630-5
40. Yao F, Wang C, Ding J, et al. A randomized, double-blind, parallel-group phase I study comparing the pharmacokinetics, safety, and immunogenicity of cmab015, a candidate secukinumab biosimilar, with its reference product Cosentyx® in healthy Chinese male subjects. Drug Des Devel Ther. 2024;18:3891–3901. doi: 10.2147/DDDT.S470619
41. Godding LTH, Seyger MMB, Duvetorp A, et al. Real-world cost per responder among different classes of biologics for the treatment of psoriasis. Acta Derm Venereol. 2025;105:adv42767. doi: 10.2340/actadv.v105.42767
42. Geat D, Gisondi P, Maurelli M, et al. Price variability of TNF-α inhibitor biosimilars among European countries. J Eur Acad Dermatol Venereol. 2024;38(8):e663–e665. doi: 10.1111/jdv.19756
43. Gisondi P, Geat D, Armeni P, et al. Cost per responder of adalimumab biosimilars MSB11022 and ABP 501 versus the originator and methotrexate in chronic plaque psoriasis. Expert Opin Biol Ther. 2022;22(12):1579–1584. doi: 10.1080/14712598.2022.2070428
• **Important pharmacoeconomic analysis supporting cost-effectiveness of biosimilars.**
44. Maurelli M, Girolomoni G, Gisondi P. Cost per responder of adalimumab biosimilars versus methotrexate in patients with psoriasis: a real-life experience. J Dermatolog Treat. 2023;34(1):2218504. doi: 10.1080/09546634.2023.2218504
45. Loft N, Nguyen TT, Zachariae C, et al. Shift from ustekinumab originator to biosimilars in Denmark. J Eur Acad Dermatol Venereol. 2025. Epub ahead of print. doi: 10.1111/jdv.70224
46. Dolinar R, Kohn CG, Lavernia F, et al. The non-medical switching of prescription medications. Postgrad Med. 2019;131(5):335–341. doi: 10.1080/00325481.2019.1618195
47. Colloca L, Panaccione R, Murphy TK. The clinical implications of nocebo effects for biosimilar therapy. Front Pharmacol. 2019;10:1372. doi: 10.3389/fphar.2019.01372
48. Glintborg B, Loft AG, Omerovic E, et al. To switch or not to switch: results of a nationwide guideline of mandatory switching from originator to biosimilar etanercept. One-year treatment outcomes in 2061 patients with inflammatory arthritis from the DANBIO registry. Ann Rheum Dis. 2019;78(2):192–200. doi: 10.1136/annrheumdis-2018-213474
49. van Adrichem RCS, Voorneveld HJE, Waverijn GJ, et al. The non-medical switch from reference adalimumab to biosimilar adalimumab is highly successful in a large cohort of patients with stable inflammatory rheumatic joint diseases: a real-life observational study. Rheumatol Ther. 2022;9(4):1109–1118. doi: 10.1007/s40744-022-00465-6
50. Gisondi P, Bellinato F, Curic T, et al. Biosimilars for the treatment of moderate to severe chronic plaque psoriasis. Psoriasis (Auckl). 2025;15:27–44.
51. Zhao H, Zhao H, Tang Y, et al. Skin-permeable gold nanoparticles with modifications azelamide monoethanolamine ameliorate inflammatory skin diseases. Biomark Res. 2024;12(1):118. doi: 10.1186/s40364-024-00663-0
52. Wu F, Xia X, Han Y, et al. Breaking the psoriasis pathological signaling cycle: a novel nanomedicine strategy targeting metabolism and oxidative stress. Mater Today Bio. 2025;32:101887. doi: 10.1016/j.mtbio.2025.101887
53. Chen R, Duan B, Li S, et al. A triple-targeting "nano-brake" remodeling the impaired immune microenvironment in skin lesions for psoriasis treatment. Mater Today Bio. 2025;32:101875. doi: 10.1016/j.mtbio.2025.101875
54. Gooderham M, Lain E, Bissonnette R, et al. Targeted oral peptide icotrokinra for psoriasis involving high-impact sites. NEJM Evid. 2025;4(12):EVIDoa2500155. doi: 10.1056/EVIDoa2500155
55. Berger S, Seeger F, Yu TY, et al. Preclinical proof of principle for orally delivered Th17 antagonist miniproteins. Cell. 2024;187(16):4305–4317. doi: 10.1016/j.cell.2024.05.052
56. Armstrong AW, Gooderham M, Lynde C, et al. Tyrosine kinase 2 inhibition with zasocitinib (TAK-279) in psoriasis: a randomized

clinical trial. JAMA Dermatol. 2024;160(10):1066–1074. doi: 10.1001/jamadermatol.2024.2701

57. Gonçalves MBS, Fonseca M, Mascarenhas-Melo F, et al. Advancing insights into psoriasis: from pathogenesis to current and emerging therapies. Int Immunopharmacol. 2025;165:115429. doi: 10.1016/j.intimp.2025.115429
58. Tatu AL, Ionescu MA, Nwabudike LC. Contact allergy to topical mometasone furoate confirmed by rechallenge and patch test. Am J Ther. 2018;25(4):e497–e498. doi: 10.1097/MJT.0000000000000581
59. Tatu AL, Nwabudike LC. Metoprolol-associated onset of psoriatic arthropathy. Am J Ther. 2017;24(3):e370–e371. doi: 10.1097/MJT.0000000000000560
60. Nicolescu AC, Ionescu MA, Constantin MM, et al. Psoriasis management challenges regarding difficult-to-treat areas: therapeutic decision and effectiveness. Life (Basel). 2022;12:2050. doi: 10.3390/life12122050
61. Marzo-Ortega H, Harrison SR, Fragoulis GE, et al. EULAR points to consider and consensus definitions for difficult-to-manage and treatment-refractory psoriatic arthritis. Ann Rheum Dis. 2025;84:1319–1320. Epub ahead of print. doi: 10.1016/j.ard.2025.06.619