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Biologic Therapy in Psoriasis: A Comprehensive Review of Molecular Targets and Personalized Clinical Management

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Md Samim Shikari [1], Sanjeev Gulati [2], Prathyusha Dasari [3], Jaya Sai Sankara Vara Prasad Kommana [4], Kinjal M Vasava [5], Karthika Padmavathy [6]

1. Department of Dermatology, Venereology and Leprosy, Medical College Kolkata, Kolkata, IND 2. Department of Dermatology, Venereology and Leprosy, Dayananda Sagar University, Dr. Chandramma Dayananda Sagar Institute of Medical Education and Research, Harohalli, IND 3. Department of Dermatology, Venereology and Leprosy, Nimra Institute of Medical Sciences, Vijayawada, IND 4. Department of Internal Medicine, Andhra Medical College, Visakhapatnam, IND 5. Department of Psychiatry, Dr. N.D. Desai Faculty of Medical Science and Research, Dharmsinh Desai University, Nadiad, IND 6. Department of Pathology, Sri Lalithambigai Medical College and Hospital, Dr. M.G.R. Educational and Research Institute, Chennai, IND

Corresponding author: Md Samim Shikari, samimshikari877@gmail.com

Abstract

Psoriasis is a chronic, immune-mediated inflammatory skin disease marked by complex interactions among genetic susceptibility, dysregulated cytokine signaling, keratinocyte activation, and systemic inflammation. Biologic therapies have transformed management by selectively targeting key inflammatory pathways, particularly tumor necrosis factor-alpha (TNF-α), IL-12/23, IL-17, and IL-23. This review aims to summarize the molecular basis, clinical efficacy, safety, treatment selection, and emerging personalization strategies associated with biologic therapy in psoriasis. A structured literature search was conducted across PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for relevant publications from 2016 to 2026, with selection based on predefined inclusion and exclusion criteria. Current evidence indicates that certain IL-17 and IL-23 inhibitors have demonstrated high rates of skin clearance in clinical trials, with differences in response onset and durability depending on the specific agent, patient population, and trial comparison, whereas TNF-α inhibitors retain value in selected patients, particularly those with psoriatic arthritis and established long-term response. Treatment choice is increasingly influenced by comorbidities, prior biologic exposure, infection risk, reproductive considerations, obesity, treatment persistence, and patient preference. Biomarkers, pharmacogenomics, therapeutic drug monitoring, multi-omic profiling, and artificial intelligence-based predictive models may further support individualized treatment selection, although these approaches remain largely investigational and are not sufficiently validated for routine biologic selection. Biologic therapy has substantially improved psoriasis outcomes, although long-term comparative safety, cost, accessibility, and validation of predictive tools remain important priorities for precision clinical management.

Categories: Therapeutics, Allergy/Immunology, Dermatology
Keywords: biologic therapy, interleukin inhibitors, personalized medicine, psoriasis, tumor necrosis factor-alpha

Introduction And Background

Psoriasis is a chronic, immune-mediated inflammatory condition characterized by recurrent, well-demarcated erythematous and scaly plaques, commonly involving the scalp, extensor surfaces, and lumbosacral region [1]. It is associated with systemic inflammation contributing to psoriatic arthritis, cardiometabolic comorbidities, inflammatory bowel disease, psychiatric morbidity, and reduced quality of life [2]. The severity of the disease ranges from mild local skin lesions causing little disability to severe inflammation involving multiple parts causing physical and psychosocial morbidity [3]. It is a heterogeneous disease requiring therapies for achieving complete skin clearance, preservation of function, systemic inflammation management, and minimizing treatment toxicities [4]. The etiopathology of psoriasis is complicated, with many interactions between genetics, environment, innate immunity, adaptive immunity, and dysregulated proliferation of keratinocytes [5]. Tumor necrosis factor-alpha (TNF-α), IL-12, IL-17, and IL-23 pathways are important inflammatory mediators [6].

Dendritic cells and T-helper 17 (Th17) lymphocytes promote the production of cytokines that maintain epidermal hyperproliferation, vascular alterations, and chronic inflammation [6]. With the recognition of these molecular mechanisms, psoriasis has evolved from a condition that was treated primarily with anti-inflammatory or general immune-suppressive drugs to one in which selective immune modulation is being utilized more and more to manage the disease [7]. Inflammation due to cytokines has also been a cornerstone for the development of therapeutic agents that can specifically target parts of the pathogenic cascade [8]. However, the conventional systemic drugs such as methotrexate, cyclosporine, and acitretin are still clinically relevant in selected patients [9]. Targeted oral small-molecule therapies, including phosphodiesterase-4 inhibitors such as apremilast and tyrosine kinase 2 inhibitors such as deucravacitinib,

have also demonstrated clinical efficacy in moderate-to-severe plaque psoriasis [10]. Emerging evidence suggests that Janus kinase inhibitors may provide therapeutic benefits in pustular forms of psoriasis and related inflammatory disorders, including SAPHO syndrome, although further controlled studies are required to establish their long-term efficacy and safety [11,12]. Conventional systemic drugs are limited in use due to cumulative toxicity, organ-specific side effects, necessary laboratory monitoring, contraindications, limited long-term tolerability, and variable efficacy in severe disease [9]. Biologic therapies have significantly increased the number of treatment options for moderate-to-severe psoriasis [13]. Monoclonal antibodies and receptor-based agents directed against TNF-α, IL-12/23, IL-17, and IL-23 can result in rapid and significant decreases in disease activity, with many of the newer agents demonstrating high rates of skin clearance [13]. With the development of better dosing regimens and greater clinical experience over the long term, further evidence has emerged that supports the use of biologic therapy in modern psoriasis treatment [13].

Biologic therapy selection has also become more complex due to the increasing number of biologic agents [14]. There are variations in speed of response, magnitude of skin clearance, durability of effect, frequency of dosing, safety profile, immunogenicity, suitability for psoriatic arthritis, and applicability to patients with inflammatory bowel disease, obesity, cardiovascular risk, recurrent infections, and other comorbidities [14]. Therapeutic response can also differ by age, body weight, disease phenotype, prior exposure to biologics, treatment adherence, immunological profile, and pharmacokinetic properties [15]. A biologic agent that is effective at the population level may not provide the same benefit for every patient, highlighting the limitations of uniform treatment algorithms and the need for individualized treatment selection [15].

There are still significant knowledge gaps between the scientific advances in therapy and personalized clinical practice. There is a lack of direct head-to-head evidence for many comparisons of biologic treatments, and variability in study populations, outcome measures, duration of follow-up, and sequences of treatment make it difficult to compare studies. Long-term comparative safety data are less extensive for newer agents, especially in patients with multiple chronic conditions or prior exposure to several systemic treatments. Predictive, non-responsiveness, or adverse event biomarkers are not yet routinely used in the clinical setting. Emerging precision medicine approaches, including pharmacogenomics, therapeutic drug monitoring, molecular profiling, and artificial intelligence-based prediction models, require further clinical validation before routine use in biologic treatment selection.

The potential outcomes of personalized management also need to be taken into account, apart from measurements of skin severity. Treatment suitability may be affected by treatment burden, patient preference, quality of life, joint involvement, difficult-to-treat anatomical sites, reproductive considerations, access to therapy, and economic factors. While biosimilars may help make treatments more affordable and available, differences in healthcare systems and reimbursement remain an issue for biologic access. Increased incorporation of molecular features into clinical phenotype assessment may help inform more accurate treatment selection and minimize the need for repeated empirical switches between therapies.

This review examines the molecular targets, major biologic classes, comparative efficacy, safety considerations, and clinical determinants influencing biologic selection in psoriasis. It also evaluates current approaches to treatment personalization, including biomarkers, pharmacogenomics, therapeutic optimization, comorbidity-guided selection, and emerging strategies intended to advance precision clinical management.

Methodology

A structured literature search was conducted to identify relevant publications addressing biologic therapy in psoriasis, with emphasis on molecular targets, comparative clinical efficacy, safety, treatment selection, comorbidity-guided management, biomarkers, pharmacogenomics, and personalized therapeutic strategies. PubMed/MEDLINE, Scopus, and Web of Science were searched for studies published between January 2016 and September 2026. Google Scholar was additionally used as a supplementary search source to broaden literature coverage and identify relevant interdisciplinary studies. Search terms included combinations of “psoriasis”, “biologic therapy”, “biologics”, “TNF-alpha inhibitors”, “IL-17 inhibitors”, “IL-23 inhibitors”, “IL-12/23 inhibitors”, “personalized medicine”, “precision medicine”, “biomarkers”, “pharmacogenomics”, “treatment response”, “safety”, and “comorbidities”. The Boolean operators “AND” and “OR” were used to combine search terms and refine the literature search strategy across the selected databases. Reference lists of relevant articles were also screened to identify additional eligible publications. Selected foundational and background publications published before 2016 were cited separately from the defined literature search period to provide historical context and support established knowledge regarding psoriasis pathogenesis, psychosocial burden, biologic efficacy, treatment safety, immunogenicity, and therapeutic drug monitoring.

The eligible publications consisted of original clinical studies, randomized controlled trials, cohort studies, real-world observational studies, comparative effectiveness studies, pharmacogenomic studies, biomarker studies, and/or high-quality clinical guidance relevant to biologic treatment of psoriasis. Studies were included if the full text was available in English, the study population included patients with psoriasis, and the findings were directly relevant to the biologic mechanisms, efficacy, safety, treatment optimization, or

individualization of treatment. Non-English articles, conference abstracts with insufficient clinical information, case reports, editorials, letters, animal studies, purely experimental laboratory studies without clinical relevance, duplicate publications, and articles without adequate information on biologic therapy were excluded. Publications were initially screened for relevance based on their titles and abstracts. Potentially eligible articles were subsequently assessed through review against the predefined inclusion and exclusion criteria to determine their suitability for narrative synthesis. Eligible evidence was synthesized narratively without statistical pooling and tabulated by therapeutic target, clinical effectiveness, safety, comorbidity considerations, treatment response predictors, and new approaches to personalized management of psoriasis. Findings were compared qualitatively, considering differences in study populations, therapeutic agents, clinical outcomes, and follow-up periods.

Review

Immunopathogenesis of psoriasis and molecular basis for biologic therapy

Psoriasis is a complex interplay of genetic susceptibility, environmental triggers, abnormalities in innate immunity, dysregulation of adaptive immunity, and hyperproliferation of keratinocytes [16]. Genetic interactions with immune-regulatory genes, including antigen presentation and cytokine signaling genes, play roles in disease susceptibility and will affect clinical phenotype variation [17]. In predisposed individuals, a variety of environmental factors can trigger and/or exacerbate the inflammatory activity, including infections, psychological stress, smoking, obesity, use of certain medications, and skin trauma [17]. These triggers activate dendritic cells that produce cytokines, which trigger differentiation and proliferation of subsets of T-helper cells, including Th17 and Th1 cells [17].

The IL-23/Th17 axis plays a key role in psoriatic inflammation. IL-23 helps maintain the survival and pathogenic function of Th17 cells, which results in enhanced production of inflammatory mediators, such as IL-17A and IL-17F [17,18]. IL-17 induces keratinocyte production of chemokines, antimicrobial peptides, and other pro-inflammatory cytokines, thereby amplifying inflammation in the epidermis [18,19]. This inflammatory network is reinforced by the activation of immune cells by TNF-α and its ability to increase the expression of cytokines in psoriatic lesions [19]. Although IL-12 is involved in Th1 differentiation and interferon-mediated immune responses, current understanding of psoriasis pathogenesis places particular emphasis on the IL-23/Th17 axis as a major therapeutic pathway [17,20]. The molecular structure has allowed the manufacturing of biological products targeting specific inflammatory mediators. Blocking TNF-α, IL-12/23, IL-17, and IL-23 can block important steps in the inflammatory pathway and lead to significant clinical improvement [20]. The connection between the pathogenic pathway and its therapeutic target is summarized in Table *1*.

Immune pathway	Principal mediator	Main pathogenic effect	Biologic target	Clinical relevance	References
TNF pathway	TNF-α	Amplifies inflammatory signaling and immune-cell activation	TNF-α	Broad suppression of psoriatic inflammation	[19,20]
IL-12/Th1 pathway	IL-12	Promotes Th1 differentiation and inflammatory cytokine production	IL-12/23 p40	Combined inhibition of IL-12 and IL-23 signaling	[17,20]
IL-23/Th17 pathway	IL-23	Maintains pathogenic Th17-cell activity	IL-23 p19	Sustained control of upstream psoriatic inflammation	[17,18]
IL-17 pathway	IL-17A/IL-17F	Stimulates keratinocyte activation and neutrophil recruitment	IL-17 or IL-17R	Rapid reduction of plaque inflammation	[18,20]
Keratinocyte-immune axis	Multiple cytokines	Sustains epidermal hyperplasia and local inflammatory amplification	Downstream cytokine blockade	Improvement in epidermal and inflammatory manifestations	[16,18]

TABLE 1: Key immunological pathways and therapeutic targets in psoriasis.

IL-17R, IL-17 receptor; Th1, T-helper 1; Th17, T-helper 17; TNF-α, tumor necrosis factor-alpha

Evolution and classification of biologic therapies for psoriasis

The treatment of moderate-to-severe psoriasis has undergone a dramatic transformation with the advent of biologic agents targeting specific components of the inflammatory cascade. In the past, methotrexate, cyclosporine, retinoids, and phototherapy were the main systemic and conventional treatment options, which can achieve a reduction in disease activity but can have organ-specific effects, be associated with

monitoring requirements, have varying tolerability, and be associated with cumulative toxicity [21]. The understanding of the immune-mediated inflammatory nature of psoriasis prompted the development of targeted therapies that selectively inhibit specific cytokines rather than broadly suppressing immune activity [21]. TNF-α inhibitors were among the first major classes of biologic therapy approved for psoriasis treatment [21].

Selective cytokine inhibition has demonstrated clinical efficacy in controlling psoriasis, with agents such as etanercept, infliximab, and adalimumab providing evidence of this [22]. The subsequent introduction of ustekinumab, which inhibits the shared p40 subunit of IL-12 and IL-23, further demonstrated the therapeutic importance of cytokine-specific inhibition and the IL-23/Th17 axis in psoriasis [22]. Therapeutic efforts then shifted to more selective targeting of the IL-17 and IL-23 pathways, resulting in increased proportions of patients achieving near-complete or complete skin clearance [23].

Biologic therapies can be classified according to their primary molecular targets. TNF-α inhibitors target a broad pro-inflammatory pathway that plays a role in both cutaneous and articular disease [24]. IL-12/23 inhibition decreases signaling via the common p40, while selective IL-23 inhibitors specifically target the p19 subunit of IL-23 without directly inhibiting IL-12 signaling [22,24]. IL-17 inhibitors selectively target IL-17A, both IL-17A and IL-17F, or the IL-17 receptor A, depending on the specific agent, thereby reducing downstream inflammatory signaling and keratinocyte activation [22,23]. Such differences in mechanism of action can result in differences in response time, extent of clearance, dosing regimen, duration of action, tolerability, and appropriate use in patients with specific comorbidities [15,22].

Biologics have become an important component of treatment algorithms in patients with moderate-to-severe psoriasis when conventional systemic therapy is ineffective, contraindicated, poorly tolerated, or insufficient to achieve treatment targets [23,24]. The choice of biologic class is becoming more dependent on disease severity, psoriatic arthritis, inflammatory bowel disease, obesity, cardiovascular risk, previous treatments, infection history, patient preference, and access to therapy [22].

Certain newer IL-17 and IL-23 inhibitors have demonstrated higher rates of skin clearance than some earlier biologic agents in comparative studies, although clinical benefits vary according to the specific agents, patient characteristics, treatment durability, safety profiles, and individual therapeutic needs [22]. As a result, the use of biologic therapy has evolved from broader immunomodulation toward more selective, mechanism-based treatment approaches [23,24]. Figure *1* depicts the history of psoriasis treatment, from traditional systemic treatment to targeted biologic agents and individualized treatment selection.

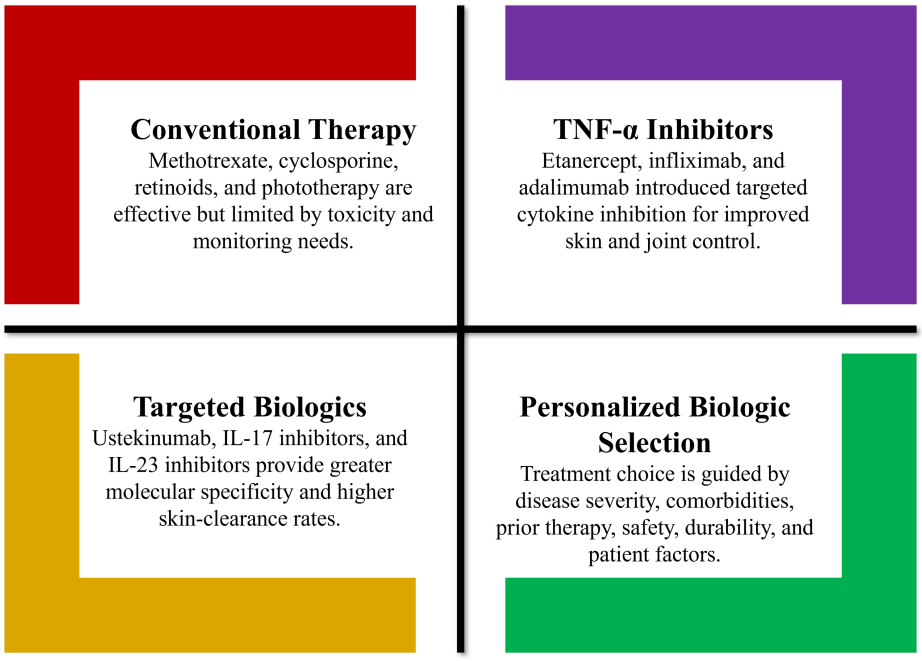


FIGURE 1: Evolution of biologic therapy in psoriasis.

Created by authors using Microsoft PowerPoint (Microsoft Corporation, Redmond, WA, USA) based on published evidence [22,24].

TNF-α, tumor necrosis factor-alpha

TNF-α Inhibitors

TNF-α plays a key role in psoriatic inflammation by promoting dendritic-cell activation, leukocyte recruitment, keratinocyte inflammatory signaling, and T-cell-mediated immune responses [19,20]. The activity of the cytokine network between innate and adaptive immunity is sustained in psoriatic lesions due to elevated levels of TNF-α. Pharmacological blockade of TNF-α abates this inflammatory amplification and decreases epidermal hyperproliferation, vascular activation, and infiltration of inflammatory cells [25].

TNF-α inhibitors were thus one of the first biologic therapies to show persistent clinical responses in moderate-to-severe plaque psoriasis. The principal agents used in psoriasis are adalimumab, infliximab, etanercept, and certolizumab pegol. Adalimumab is a fully human anti-TNF antibody, while infliximab is a chimeric antibody injected into the vein [26]. Etanercept is a soluble TNF receptor with an attached Fc component of immunoglobulin G, which forms competitive binding with circulating TNF. Certolizumab pegol is a TNF-α inhibitor used in the management of psoriasis, and its clinical efficacy can be considered within the broader comparative framework of TNF-, IL-17-, and IL-23-targeted biologic therapies [27].

There is interagent variation in clinical efficacy depending on the molecular structure, dose, treatment regimen, prior biologic treatment, and patient characteristics. Infliximab usually results in a quick improvement in the severity of plaques, while adalimumab offers long-term disease control in a large percentage of patients who are treated [26]. Etanercept can also be used for the treatment of psoriatic arthritis, as well as for cutaneous psoriasis, but clearance rates are frequently less than with newer IL-17 or IL-23 inhibitors [26,27].

Plaque psoriasis and concomitant psoriatic arthritis are clinically meaningful outcomes of certolizumab pegol therapy. Inhibition of TNF-α should be evaluated carefully for safety. Reactivation of latent tuberculosis and hepatitis B infection is an important consideration and supports appropriate pre-treatment screening [26]. Other significant risks for clinical use include severe infection, infusion or injection reactions, paradoxical inflammatory events, and uncommon neurologic complications. Moderate to severe congestive heart failure and patients with demyelinating diseases require caution [26].

Immunogenicity, adherence, body weight, comorbidity burden, treatment interruption, and secondary loss of response will all have an impact on long-term effectiveness. Antidrug antibodies can decrease the levels of the circulating drug and may affect the therapeutic persistence, especially in the case of monoclonal antibodies [28]. Although newer agents that affect a single cytokine are increasingly favored in patients who need very high levels of skin clearance, TNF-α inhibitors have demonstrated a role in the current management of psoriasis, particularly in the setting of psoriatic arthritis, extensive clinical experience, reproductive concerns, or prior durable response [26,29].

IL-12/23 and IL-23 Inhibitors

In moderate-to-severe psoriasis, the IL-23/Th17 axis is a major contributor to the inflammatory process of psoriatic disease and is one of the key therapeutic targets in moderate-to-severe disease [30]. IL-23 contributes to the survival, expansion, and pathogenic function of Th17 cells, resulting in continued production of IL-17 and other inflammatory mediators. This signaling pathway promotes activation of keratinocytes, the recruitment of neutrophils, epidermal hyperplasia, and development of chronic plaques. Disruption of this pathway can offer significant and long-lasting clinical benefits [30].

Ustekinumab blocks the common p40 subunit of the cytokines IL-12 and IL-23, which are responsible for both Th1 and Th17 signaling pathways. The introduction of the drug was a big step forward from a more general cytokine blockade and was the first to demonstrate the therapeutic relevance of the IL-23 pathway [31]. Subsequent more selective agents targeted the p19 subunit of IL-23 [31,32], enabling selective inhibition of IL-23 activity without inhibition of the immune signaling by IL-12. Key representatives of this class are guselkumab, risankizumab, and tildrakizumab, which have been found to induce a high percentage of Psoriasis Area and Severity Index (PASI) responses, including near-complete and complete skin clearance [32,33].

Selective IL-23 inhibitors have been characterized as having long-term efficacy, a relatively low drug burden for maintenance treatment, and good long-term tolerability [32,34]. Comparative trials have often demonstrated better skin clearance and treatment persistence than a number of the older biologic agents [34]. The safety profiles are generally very favorable, with mild respiratory infections, headache, injection-site reactions, and few serious treatment-related adverse events. Although biologic selection is still possible, the treatment history, the presence of psoriatic arthritis, comorbid disease, access to therapy, and treatment preference must be taken into consideration [35]. The main properties of IL-12/23 and selective IL-23 inhibitors for the treatment of psoriasis are summarized in Table *2*.

Biologic agent	Molecular target	Mechanism	Dosing characteristic	Key clinical feature	References
Ustekinumab	IL-12/23 p40	Blocks shared IL-12 and IL-23 signaling	Extended maintenance interval	Established long-term efficacy	[31]
Guselkumab	IL-23 p19	Selectively inhibits IL-23 signaling	Infrequent maintenance dosing	High skin-clearance rates	[31,32]
Risankizumab	IL-23 p19	Suppresses IL-23-mediated Th17 activation	Extended dosing interval	Durable PASI response	[31,34]
Tildrakizumab	IL-23 p19	Selectively neutralizes IL-23	Infrequent scheduled dosing	Sustained disease control	[31,34]

TABLE 2: Comparative characteristics of biologic agents targeting the IL-12/23 and IL-23 pathways.

PASI, Psoriasis Area and Severity Index; Th17, T-helper 17

IL-17 Pathway Inhibitors

IL-17 is a critical effector cytokine in the pathogenesis of psoriasis and is a critical downstream target in the IL-23/Th17 inflammatory axis. Th17 cells produce high levels of IL-17A and/or IL-17F, which directly promote keratinocyte activation, epidermal hyperplasia, and neutrophil recruitment and amplify local inflammatory signaling [36]. The upregulation of mediators associated with the IL-17 pathway in psoriatic lesions further validates the importance of this pathway in the maintenance of chronic skin inflammation. Therapeutic inhibition of IL-17 can lead to rapid inhibition of these processes and has been correlated with significant improvement in moderate to severe plaque psoriasis [37].

Secukinumab and ixekizumab are antibodies against IL-17A, and brodalumab is an antibody against the IL-17 receptor A that inhibits multiple IL-17 family cytokines. Bimekizumab blocks both IL-17A and IL-17F, offering more comprehensive suppression of key effector cytokines that contribute to psoriasis inflammation [38]. These mechanistic differences explain some of the differences in clinical response patterns, dosing schedules, and safety. Biologics that target IL-17 are some of the fastest-acting biologic treatments for psoriasis, and disease severity may improve noticeably within the first few weeks of treatment [37].

Therapy directed at IL-17 is often associated with high rates of skin clearance, such as PASI 90 and PASI 100 responses. The clinical effects are not limited to plaque psoriasis but also include management of difficult-to-treat areas like the scalp, nails, palms, soles, and genital skin [37]. These responses can make a significant contribution to patients' functional limitation and overall quality of life with a clinically significant disease in an anatomically localized patient. Inhibiting IL-17 is also therapeutic in psoriatic arthritis due to the decrease in peripheral joint inflammation, enthesitis, and musculoskeletal manifestations [38]. The side effects of IL-17 inhibitors are generally acceptable, but pathway-specific safety issues must be evaluated carefully [38].

Mucocutaneous candidiasis is more common during IL-17 blockade because of its physiological role in protection against fungal mucocutaneous infections. Other mild infectious events, including upper respiratory tract infections and injection-site reactions, are also reported [39]. Brodalumab has specific warnings regarding suicidal ideation and behavior in some regulatory regions, although a causal relationship between brodalumab treatment and these events has not been established [26]. These warnings should be considered when assessing patients before administration of the drug. However, inflammatory bowel disease is an important factor to keep in mind when considering IL-17 targeted therapy [35]. Susceptible individuals have developed new-onset disease or exacerbation of established Crohn's disease or ulcerative colitis when treated with this product, so alternative biologic classes are preferred in certain patients [40]. Despite these drawbacks, IL-17 inhibitors are nevertheless well-suited therapeutics for situations where a rapid and complete clearance of skin is a prominent therapeutic goal.

Comparative clinical efficacy and long-term treatment outcomes

The measurement of the extent and duration of skin clearance is the basis for comparative evaluation of biologic therapy in psoriasis [41]. One of the most widely used measures remains the PASI, where PASI 75, PASI 90, and PASI 100 denote at least 75%, 90%, and 100% improvement from baseline, respectively [33,41]. The new biologic therapy has moved the bar from partial disease control to almost complete or complete

skin clearance, especially with agents in the IL-17 and IL-23 pathways. Additionally, an Investigator's Global Assessment is used to assess the overall severity of the lesions and the percentage of patients who have clear and almost clear skin [42,43].

There is variability in clinical response to the different types and classes of biologic therapy, and this may affect the choice of therapy for patients with extensive disease, severe pruritus, visible lesions, and significant impairment of quality of life [44]. IL-17 inhibitors may have a quick onset of action, leading to significant improvements in plaque severity early in treatment. Selective IL-23 inhibitors can be comparatively slow to act but often achieve high rates of long-term PASI 90 and PASI 100 response [44]. Although certain newer cytokine-specific agents have demonstrated higher rates of complete skin clearance than some older biologics in comparative studies, TNF-α inhibitors remain clinically effective [41,44]. The prevention of recurrence of disease is also important, as psoriasis often needs long-term treatment [42]. Studies of long-term extension have shown that long-lasting effects can occur in patients who are treated with an effective biologic for several years [44]. Drug survival, or the number of days a patient receives the same drug, is a composite measure that incorporates efficacy, tolerability, safety, adherence, and patient satisfaction. There is real-world evidence that persistence is biologic class dependent and may be influenced by prior biologic therapy, obesity, psoriatic arthritis, treatment interruptions, and access to health services [41].

The head-to-head trials also offer important comparative information since they directly assess biologics under common clinical circumstances [45]. These studies have confirmed the greater efficacy of some IL-17 and IL-23 inhibitors in improving skin clearance vs. older classes of biologic agents [44]. Real-world data continue to play a crucial role in evaluating effectiveness across broader patient populations who have multimorbidity, prior failure of treatment, and variable adherence [45,46]. Primary nonresponse, secondary loss of efficacy, adverse events, immunogenicity, development of contraindications, treatment burden, and patient preference are common reasons for discontinuing or switching biologics. Assessment of comparative treatment efficacy should therefore consider initial efficacy, rapidity of response, persistence, drug survival, safety, and personal clinical priorities and should not be based on efficacy alone [46]. The comparative efficacy, response characteristics, and long-term treatment outcomes of the biologic therapies used for psoriasis are presented in Figure *2*.

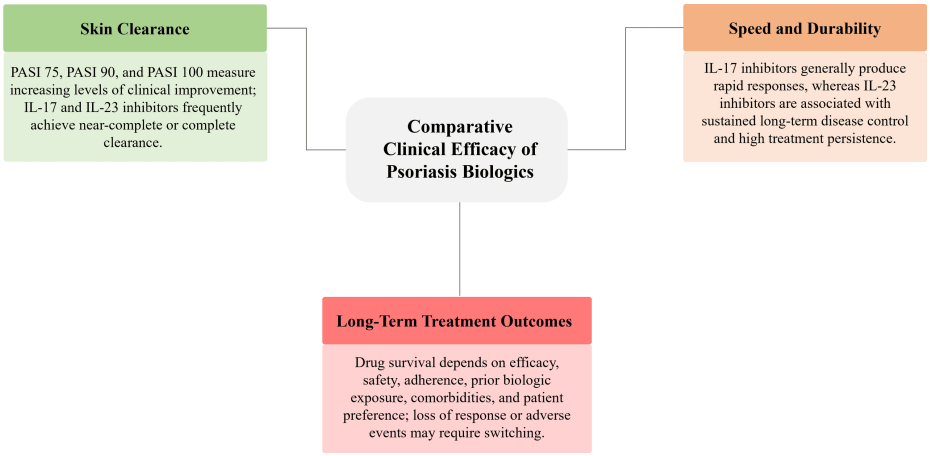


FIGURE 2: Comparative clinical efficacy and long-term outcomes of biologic therapy in psoriasis.

Created by authors using Microsoft PowerPoint (Microsoft Corporation, Redmond, WA, USA) based on published comparative evidence [27,41].

PASI: Psoriasis Area and Severity Index

Safety, immunogenicity, and monitoring of biologic therapy

Safety evaluation is a crucial aspect of the use of biologic therapies in psoriasis, especially in the long term and in patients with numerous comorbidities. Adverse reactions are dependent on the pathway that is targeted, the length of the treatment period, the immune system of the host, and other medications being used at the time [47]. To be expected are upper respiratory tract infections, reactions at the site of the injection, headache, and mild mucocutaneous infections. Although less common, serious infections also have clinical significance, particularly in those on TNF-α inhibitors or other forms of immunosuppression [48].

Because of the risk of reactivation, screening for latent tuberculosis and hepatitis B infection is

recommended before starting selected biologics, especially TNF-α inhibitors [48]. Common blood tests performed in the baseline evaluation consist of infection history, vaccination history, complete blood count, liver function testing, and assessment of pertinent chronic diseases [49]. Continuous monitoring should be tailored to the specific biologic and to clinical symptoms and should take into account comorbidities and concomitant therapy.

Long-term therapeutic efficacy may be affected by immunogenicity. Antidrug antibodies appear to decrease plasma levels, increase the clearance of the drug, and can account for secondary loss of response [47]. This effect can be more clinically relevant with some monoclonal antibodies and can depend on intermittent dosing, treatment breaks, and individual immune features. Therapeutic drug monitoring may be helpful for special cases where treatment response is not understood, but it has not been uniformly utilized clinically [50]. Risk of malignancy depends on the individual patient and should be assessed based on the patient's history of malignancy and/or strong predisposing factors. Vaccination history should also be ascertained prior to starting treatment, and live vaccines should generally be avoided while patients are receiving biologic therapy [48,49]. Table *3* provides a list of important safety issues and monitoring priorities for key biologic classes.

Biologic class	Principal safety concern	Baseline assessment	Ongoing monitoring	Additional consideration	References
TNF-α inhibitors	Serious infection, TB, or HBV reactivation	TB and HBV screening, CBC, LFTs	Infection surveillance, CBC/LFTs as indicated	Caution in demyelinating disease and CHF	[26,49]
IL-12/23 inhibitors	Infection, hypersensitivity	Infection history, CBC, LFTs	Clinical infection monitoring	Long-term safety surveillance	[26,49]
IL-17 inhibitors	Candidiasis, IBD exacerbation	IBD history, infection assessment	Mucosal infection and GI symptom monitoring	Avoid or use caution in active IBD	[39,48]
IL-23 inhibitors	Respiratory infection, injection reactions	Infection assessment, CBC, LFTs	Clinical review and laboratory testing as indicated	Favorable long-term tolerability profile	[31,49]

TABLE 3: Safety profile and monitoring considerations for major biologic classes in psoriasis.

CHF, congestive heart failure; HBV, hepatitis B virus; IBD, inflammatory bowel disease; LFTs, liver function tests; TB, tuberculosis; TNF-α, tumor necrosis factor-alpha

Personalized biologic selection in psoriasis

When considering the use of biologic therapy in psoriasis, all aspects of disease phenotype, comorbid burdens, prior treatment exposure, age, reproductive status, infection risk, and treatment preferences for the long term should be evaluated [51]. Biologic choice is especially relevant, as it should target both skin and joint symptoms of the disease (psoriatic arthritis). Both TNF-α inhibitors and some IL-23 inhibitors and IL-17 inhibitors are clinically effective in controlling joint and skin disease; the predominant clinical domain may be the basis for selecting the final therapy [52].

Pharmacokinetics may be affected, treatment adherence may be suboptimal, and response rates may be lower with certain biologic drugs in obese and metabolic syndrome patients [51]. Weight-based dosing may have some benefits with specific biologic agents, such as infliximab, while weight-stratified dosing with ustekinumab may be appropriate in selected patients, and metabolic comorbidity must be taken into account in addition to cardiovascular risk [53,54]. Control of systemic inflammation is clinically important, and psoriasis is independently associated with an increase in the cardiometabolic burden [52]. Further care needs to be taken in treatment selection in patients with inflammatory bowel disease, as there have been reports of blockade of IL-17 being detrimental to patients with Crohn's disease or ulcerative colitis [40,51]. If there is a co-existent inflammatory disease of the GI tract, then agents that target the TNF-α and IL-23 pathways might be more appropriate [51].

Liver or renal disease may affect the choice of the systemic therapy used, particularly if conventional immunosuppressive agents have organ-specific toxicities [54]. In certain patients, biologics might be an alternative, but baseline assessment and monitoring are still required [51]. Special screening is required before biologic therapy is started in cases of recurrent infections, latent TB, chronic hepatitis, or other immunocompromising disorders [51]. The choice of biologic class should be based on the type and extent of risk of infection, vaccination status, and history of infectious complications [51]. Placental transfer and neonatal exposure, maternal disease control, and available safety information need to be taken into account

during pregnancy and lactation [51]. Certolizumab pegol is of particular interest because of its low Fc-mediated placental transfer [51]. Children need age-approved agents and weight-appropriate doses, and older adults may need to be evaluated more closely for risk of infection, with polypharmacy, malignancy history, and multimorbidity [51]. Additional complexity is added by the presence of a previous or active malignancy [55].

Treatment decisions should be based on type of cancer, length of remission, risk of recurrence, and input from specialists [55]. There is no one-size-fits-all preference for any biologic class for every oncologic scenario; this is an indication for individualized risk assessment [55]. Clinical selection should consider comorbidity profile and expected efficacy, safety, dosing convenience, treatment durability, and patient priorities [51]. This stratification allows for more informed treatment decisions and minimizes unnecessary use of potentially conflicting treatment options [51].

Precision Medicine in Biologic Therapy for Psoriasis

The concept of personalized biologic therapy in psoriasis is to use the clinical, molecular, genetic, and pharmacokinetic profile of the patient to guide treatment selection as opposed to using a trial-and-error sequential approach to treatment [15,23]. There is a significant degree of variation in response, durability, side effects, and loss of effect in people who have the same class of biologic treatment [15,23]. Factors like BMI, disease duration, disease severity at baseline, psoriatic arthritis, smoking, prior biologic use, adherence, and the presence of comorbidities could also impact therapeutic outcomes [54]. These factors can be integrated to help with better and more rational treatment selection and to minimize unnecessary exposure to ineffective treatments [15,23]. One of the big areas of interest for treatment personalization is biomarkers. Circulating cytokines, inflammatory proteins, immune-cell signatures, and tissue-based molecular markers have all been explored as potential markers of response to TNF, IL-17, and IL-23 inhibitors [23]. Pathway-dominant inflammatory patterns can be detected by transcriptomic profiling of lesional skin and may relate to differential therapeutic response [23]. Alternatively, serum biomarkers could be used for a less invasive method, but there are still limitations such as reproducibility and assay standardization, as well as clinically validated thresholds [23]. To date, no specific biomarker has reached a predictive accuracy high enough to be widely used to select biologic therapy in a variety of psoriasis populations [15,56].

The use of pharmacogenomics could also enhance the precision of treatment, as properties of the genetic variants that are associated with efficacy, drug metabolism, immunogenicity, or adverse events could be identified [23]. Polymorphisms in immune-regulatory pathways and cytokine signaling have shown some potential links to response to individual biologics but have not been consistent in different populations [23]. Eventually, broader genomic and multi-omic approaches will allow the development of composite predictive models that can incorporate molecular and clinical information [23]. Another approach to optimizing therapy is to use therapeutic drug monitoring (TDM) [50]. Monitoring trough levels of a drug and antidrug antibodies may be useful to differentiate between pharmacokinetic failure and mechanistic failure [50]. Low drug levels with antidrug antibodies can facilitate dose adjustment or switch within a therapeutic class, while good drug exposure and ongoing disease activity can suggest a switch to another molecular target [47,50]. There is still a lack of full agreement about therapeutic levels and economic viability for regular use [50].

AI and machine learning models could help to integrate demographic, clinical, genetic, laboratory, and treatment-history factors into individual response predictions [56]. Future precision algorithms may help identify the biologic most likely to provide benefit with the lowest anticipated risk for an individual patient [56]. Clinical validation, external reproducibility, equitable data representation, and transparent decision frameworks are still necessary prior to widespread implementation [56]. Personalized biologic management therefore relies on integrating molecular prediction and comorbidities assessment, treatment targets, safety, adherence, accessibility, and patient preference [51].

Emerging biologic strategies, treatment gaps, and future directions

The therapeutic landscape of psoriasis is constantly changing with the availability of increasingly selective cytokine inhibitors, combination therapy, refinement of molecular engineering, and the broader application of precision medicine [23]. While current biologics targeting TNF-α, IL-17, and IL-23 achieve high disease control, the issues of incomplete response, secondary loss of efficacy, treatment burden, cost, and long-term safety are clinically relevant [22]. Next-generation agents are being developed that have the potential to provide deeper pathway suppression, longer dosing intervals, better tissue penetration, and more persistent remission [22]. With the persistence of inflammatory activity despite single-pathway blockade, dual-cytokine inhibition has become a promising strategy for such patients. The combination of IL-17A and IL-17F targeting has already proven to have the potential for more widespread suppression of downstream inflammatory signaling [35]. Other strategies, such as simultaneous blockade of complementary cytokines/immune mediators, could increase the spectrum of treatments for difficult-to-control disease. Other immune mechanisms, such as intracellular signaling, regulatory T-cell function, dendritic cell activation, and other cytokine networks, are also being tested as targets for therapy [23]. Biosimilars can potentially increase accessibility of treatment through reduced acquisition costs and increased availability of existing biologic treatments [4]. In the healthcare context, cost may be a crucial factor in access to

ongoing biologic therapy in systems that are hindered by financial barriers [4].

Drug price, drug monitoring requirements, treatment persistence, avoidance of hospitalization, productivity, quality of life, and economic impact of uncontrolled disease should all be included in cost-effectiveness assessments [4]. In order to establish the comparative effectiveness of newer agents in different populations in real-world clinical settings, long-term comparative effectiveness data are still needed [46]. Other areas of treatment interest are de-escalation and sustained remission. For selected patients with extended disease control, dose spacing or treatment reduction with careful monitoring may be of benefit, but good criteria to indicate who it is appropriate for are lacking [53]. Biomarker-guided treatment selection may help to improve this process by identifying patients that are likely to remain in remission compared to those at higher risk of relapse [15,23]. Future strategies for the management of psoriasis should continue to prioritize patient-reported outcomes, including treatment satisfaction, functional status, pruritus, psychological burden, and quality of life [3]. Shared decision-making can help clarify and align therapeutic effectiveness with treatment priorities [15]. Future advancements will require embedding molecular profiling, comparative effectiveness evidence, long-term pharmacovigilance, cost, and personalized outcomes into real-world algorithms [23]. These developments can help shift the paradigm from an empiric approach to biologic sequencing to more predictable, durable, and patient-specific disease control [56].

Limitations and future recommendations

This review has a number of limitations. The literature available is rather a diverse collection of studies with regard to study design, patient characteristics, biologic exposure, treatment duration, the definition of the outcome, and follow-up periods, making the direct comparison between therapeutic classes somewhat challenging. Evidence for safety over longer time is more limited for newer biologics, and there are no head-to-head comparisons for some clinically relevant treatment sequences. Biomarker and pharmacogenomic discoveries are also diverse, have not been externally validated, and are poorly adopted for standard clinical practice. Treatment access, regional treatment habits, adherence, and health care system variations may affect real-world evidence.

Future research should focus on large prospective cohorts, direct comparative trials, and long-term pharmacovigilance in different populations. There is a need for standardized outcome measures to assess efficacy, durability, safety, quality of life, and treatment persistence. There is a need to increase the focus on validation of predictive biomarkers, pharmacogenomic signatures, therapeutic drug monitoring thresholds, and multi-omic models. AI could potentially be used to further support personalized biologic selection if external validation, transparency, cost-effectiveness, and equitable applicability are proven.

Conclusions

The advent of biologic therapy has revolutionized the treatment of moderate-to-severe psoriasis, as it allows for targeted blockade of inflammatory pathways involved in the initiation and maintenance of psoriasis. Blocking TNF-α, IL-12/23, IL-17, and IL-23 pathways has significantly improved skin clearance, disease control, and quality of life, with additional benefits in the management of associated psoriatic arthritis. There are differences in efficacy, onset of action, durability, safety, immunogenicity, dosing frequency, and suitability for specific comorbidities; therefore, it is necessary to select treatment on an individual basis. Clinical factors such as obesity, inflammatory bowel disease, cardiovascular risk, infection history, pregnancy, age, prior biologic exposure, and patient preference should be incorporated into therapeutic decisions. New biomarkers, pharmacogenomic strategies, therapeutic drug monitoring, and multi-omic profiling could help to better predict response to treatment and minimize empirical switching between treatments. Additional AI models could further aid in precision treatment by incorporating complex clinical and molecular data. Persistent challenges include cost, unequal access, limited long-term comparative evidence, uncertainty regarding the long-term safety of newer biologic agents, and incomplete validation of predictive biomarkers. Future advances should aim at clinically validated precision instruments, harmonization of outcome assessments, long-term safety monitoring, and patient-centric treatment algorithms that can achieve sustained remission with optimal safety and therapeutic efficiency.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Md Samim Shikari, Sanjeev Gulati, Prathyusha Dasari, Jaya Sai Sankara Vara Prasad Kommana, Kinjal M Vasava, Karthika Padmavathy

Acquisition, analysis, or interpretation of data: Md Samim Shikari, Sanjeev Gulati, Prathyusha Dasari, Jaya Sai Sankara Vara Prasad Kommana, Kinjal M Vasava, Karthika Padmavathy

Drafting of the manuscript: Md Samim Shikari, Sanjeev Gulati, Prathyusha Dasari, Jaya Sai Sankara Vara

Prasad Kommana, Kinjal M Vasava, Karthika Padmavathy

Critical review of the manuscript for important intellectual content: Md Samim Shikari, Sanjeev Gulati, Prathyusha Dasari, Jaya Sai Sankara Vara Prasad Kommana, Kinjal M Vasava, Karthika Padmavathy

Supervision: Karthika Padmavathy

Disclosures

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