International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	BIOLOGICAL TREATMENT OF PSORIASIS – MECHANISMS OF ACTION, CLINICAL EFFICACY AND SAFETY OF THERAPY
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4043
RECEIVED	23 September 2025
ACCEPTED	01 December 2025
PUBLISHED	10 December 2025
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BIOLOGICAL TREATMENT OF PSORIASIS – MECHANISMS OF ACTION, CLINICAL EFFICACY AND SAFETY OF THERAPY

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ABSTRACT

Introduction and objective: Psoriasis is a chronic autoimmune skin disease that significantly reduces patients' quality of life. The coexistence of comorbidities and persistent lesions makes treatment challenging. This review summarizes current knowledge on the mechanisms, efficacy, and safety of modern therapies, focusing on TNF-α, IL-17, IL-23 inhibitors, and apremilast as a PDE4 inhibitor.
Review methods: This narrative review is based on publications from the last five years, including phase III clinical trials, meta-analyses, and updated dermatological guidelines, retrieved from PubMed and Google Scholar.
Summary of current knowledge: Psoriasis management increasingly relies on targeted therapies. TNF-α inhibitors (infliximab, adalimumab, certolizumab) are effective but may cause serious adverse effects. IL-17 inhibitors (secukinumab, ixekizumab, brodalumab) provide rapid responses with generally favorable safety, though caution is required in patients with inflammatory bowel disease. IL-23 inhibitors (guselkumab, risankizumab, tildrakizumab) combine high efficacy with good tolerability, making them among the most promising options. Apremilast, a PDE4 inhibitor, is less potent but safe and useful in patients unsuitable for immunosuppression.
Summary: Biological therapies have transformed psoriasis care, offering improved symptom control, quality of life, and reduced risk of joint involvement. These targeted agents ensure high remission rates while maintaining safety. Ongoing research and individualized approaches may further establish biological therapy as a mainstay of dermatological practice.

KEYWORDS

Psoriasis, Biological Therapy, TNF-A, Interleukin 17, Interleukin 23, PDE4 Inhibitors

CITATION

Przemysław Piskorz, Weronika Sobota, Patryk Biesaga, Olaf Jadanowski, Kamil Łebek, Alicja Bury, Daria Litworska-Sójka, Bartosz Komsta, Julia Lipiec, Wojciech Pabis. (2025). Biological Treatment of Psoriasis – Mechanisms of Action, Clinical Efficacy and Safety of Therapy. *International Journal of Innovative Technologies in Social Science*, 4(48). doi: 10.31435/ijitss.4(48).2025.4043

1. Introduction and Objective

Psoriasis is a chronic, inflammatory autoimmune skin disease characterised by the presence of erythematous-scaly plaques and frequent relapses. The condition affects approximately 2–3% of the global population and significantly impacts patients' quality of life [1]. In addition to skin changes, psoriasis is also associated with an increased risk of developing psoriatic arthritis, depressive disorders and cardiovascular diseases. The optimal therapeutic strategy requires an individualised approach that focuses not only on alleviating skin changes, but also on the early detection and treatment of comorbidities, thereby reducing the overall burden of the disease and improving patients' quality of life [2].

The pathogenesis of psoriasis is the result of complex interactions between genetic predisposition, immune system responses and environmental factors, leading to dysregulation of the immune response and excessive keratinocyte proliferation. Many years of research have significantly advanced our understanding of the mechanisms underlying this disease, enabling the development of a variety of therapeutic approaches. Contemporary treatment approaches focus on alleviating symptoms, improving patient functioning, and inhibiting disease progression [1,3]. Current therapeutic strategies for psoriasis include topical treatment, phototherapy, systemic immunomodulatory drugs, and biological therapies aimed at alleviating symptoms and improving patients' quality of life. Nevertheless, significant challenges remain, such as adverse effects, the development of resistance to therapy, high treatment costs, and varying responses among individual patients. However, there are promising directions for the future development of psoriasis therapies [4]. The latest trends and prospects in the treatment of this disease point to a potential improvement in therapeutic outcomes. These include the development of innovative biological drugs that target new molecular pathways and research into combination therapies that aim to increase efficacy while reducing adverse effects [5].

The aim of this study is to provide a comprehensive overview of the mechanisms of action, clinical efficacy and safety of biological drugs in patients with psoriasis. Particular emphasis has been placed on current

scientific knowledge, the risks associated with therapy and the future of biological treatment, including the prospects for the development of personalised therapy. The articles and reports used in this review are mostly from the last five years, which allows for the presentation of the latest achievements and trends in the treatment of psoriasis. This article aims to highlight the hopes, challenges and opportunities offered by the development of the latest therapies, as well as to indicate the directions in which the future of treatment for this chronic disease is heading.

2. Review methods

The PubMed/MEDLINE database was searched to find articles that met specific criteria. The database was searched using the phrases 'biological treatment' and 'psoriasis'. Mainly full-text materials from the last 5 years were included. The inclusion criteria considered during the database search were as follows: publications containing full text describing biological methods of treating psoriasis. The exclusion criteria were as follows: reviews of traditional forms of treatment. Ultimately, 29 publications meeting the above criteria were included. As a result, the latest data containing information on the subject described were collected.

3. The mechanism of action of biological drugs in the treatment of psoriasis.

Biological drugs used in the treatment of psoriasis act on precisely defined elements of the immune system, blocking key cytokines involved in the development and maintenance of chronic skin inflammation. There are four main classes of these drugs, differing in their molecular target, mechanism of action and therapeutic efficacy. Thanks to their high specificity and efficacy, biological therapies are now the standard of care for moderate to severe plaque psoriasis. [4,5]

Tumor necrosis factor alpha (TNF-α) plays a key role as a pro-inflammatory cytokine involved in the development of psoriasis. It participates in both the initiation and maintenance of chronic inflammation, which is the basis for the formation of characteristic skin lesions. The main sources of TNF-α are macrophages, dendritic cells, and keratinocytes. This cytokine intensifies the immune response by stimulating the production of other inflammatory mediators, such as interleukins IL-1, IL-6, and IL-23, which in turn promote the activation and proliferation of Th17 lymphocytes — key cells in the pathogenesis of psoriasis [6]. In addition, one of TNF-α's roles in psoriasis is to enhance immune cell migration by upregulating adhesion molecules like ICAM-1 and chemokines such as CCL20, which facilitates the migration of inflammatory cells to the skin and their local activation. It also directly affects keratinocytes, promoting their proliferation and disrupting the differentiation process, leading to characteristic excessive keratinisation of the epidermis. Together, these actions create a self-perpetuating inflammatory loop, resulting in chronic, recurrent skin lesions [7].

Due to the central role of TNF-α in activating the IL-23/IL-17 axis and maintaining a pro-inflammatory environment in the skin, it has become one of the first molecular targets for biological therapies used in the treatment of psoriasis. The introduction of biological drugs specifically designed to neutralise TNF-α proved to be a breakthrough in the treatment of this disease. [6,7]

3.1 The mechanism of action of anti-TNF-α drugs in the treatment of psoriasis.

TNF-α is a key mediator of inflammatory processes in the pathogenesis of psoriasis, playing an important role in both the initiation and maintenance of chronic skin inflammation. Anti-TNF-α therapy is based on the use of biological drugs that neutralise both the soluble and membrane forms of TNF-α, leading to the inhibition of TNFR1 and TNFR2 receptor activation. Blocking these receptors suppresses signalling pathways such as NF-κB and MAPK, resulting in reduced expression of pro-inflammatory genes and limited production of cytokines and chemokines [4]. Neutralisation of TNF-α also inhibits the activation and proliferation of Th1 and Th17 lymphocytes, which are fundamental to the development of psoriatic lesions through the secretion of pro-inflammatory cytokines such as IL-17, IL-23 and IFN-γ. In addition, anti-TNF-α therapy reduces the expression of adhesion molecules and chemokines, which limits the migration of inflammatory cells to the skin. As a result, keratinocyte proliferation is reduced and the normal structure of the epidermis is restored, which translates into a marked clinical improvement [2].

Currently, several anti-TNF-α preparations differing in structure and route of administration are used in the treatment of psoriasis. The most commonly used ones include: infliximab — a chimeric monoclonal antibody administered intravenously, adalimumab — a fully human monoclonal antibody administered subcutaneously, and certolizumab pegol — a pegylated Fab fragment without the Fc fragment, which reduces its ability to cross the placenta, thus limiting foetal exposure to the drug during pregnancy [8]. The efficacy of anti-TNF-α therapy has been confirmed in numerous clinical trials. At week 10 of treatment with infliximab

(5 mg/kg), the percentage of patients achieving a PASI 75 response was approximately 80%. Similar response rates, around 80%, were observed in patients treated with adalimumab (40 mg after an initial dose of 80 mg) and certolizumab pegol (400 mg) at week 16 of therapy [10]. These drugs significantly improve patients' quality of life, leading to significant control of clinical symptoms. [9]

Despite their high efficacy, treatment with TNF-α inhibitors carries a risk of serious adverse effects. The most significant include reactivation of hepatitis B and C, development of tuberculosis, drug-induced lupus, demyelinating disorders of the central nervous system, as well as paradoxical reactions, including induction or exacerbation of psoriatic lesions. Therefore, it is necessary to carefully select patients for therapy, individualise dosing, and systematically monitor clinical status during treatment [10].

TNF-α plays a key role in the pathogenesis of psoriasis by activating inflammatory pathways and stimulating Th1 and Th17 lymphocytes. Biological therapy with TNF-α inhibitors (infliximab, adalimumab, certolizumab pegol) effectively blocks both soluble and membrane forms of TNF-α, leading to inhibition of inflammatory processes in the skin, normalisation of keratinocyte function and clinical improvement. Clinical studies show high efficacy of these drugs, with a PASI 75 response rate of up to 80%. However, treatment is associated with the risk of adverse effects, such as reactivation of viral infections, tuberculosis or demyelinating symptoms, which requires careful patient selection and constant monitoring of therapy. [9,10]

3.2 Interleukin-17 (IL-17) inhibitors in the treatment of psoriasis – mechanism of action, efficacy and safety

Interleukin 17 (IL-17), and in particular its isoform IL-17A, plays a key role in the pathogenesis of psoriasis, being a central link in the Th17-dependent inflammatory response. Th17 cells, activated by IL-23, produce a number of pro-inflammatory mediators, including IL-17A, IL-17F, IL-6 and TNF-α. IL-17A acts mainly on keratinocytes and other epidermal cells, inducing the production of chemokines (such as CCL20, CXCL1, CXCL8) and antimicrobial peptides that attract neutrophils and other inflammatory cells to the skin. In this way, IL-17A enhances the local inflammatory response, promotes the persistence of chronic psoriatic lesions and disrupts the epidermal barrier, among other things by regulating filaggrin expression [11,12,13]. The IL-17 family comprises six homologous cytokines (IL-17A–F), of which IL-17A, IL-17C and IL-17F are most important in the pathogenesis of psoriasis. They exert their effects by binding to heterodimeric transmembrane receptors, mainly IL-17RA and IL-17RC, which activate signalling pathways leading to the expression of numerous pro-inflammatory genes. In addition, IL-17A acts synergistically with TNF-α, enhancing the inflammatory response in the skin. Due to these properties, IL-17A has become one of the main therapeutic targets in the treatment of plaque psoriasis. [14,15]

Currently registered IL-17 inhibitors include monoclonal antibodies that target IL-17A directly (secukinumab, ixekizumab), neutralise both IL-17A and IL-17F (bimekizumab), or block the common IL-17RA receptor subunit (brodalumab). Secukinumab is a fully human IgG1κ monoclonal antibody that selectively binds IL-17A, while ixekizumab is a humanised IgG4 antibody. Bimekizumab has the ability to simultaneously neutralise IL-17A and IL-17F, which may increase its efficacy. Brodalumab, on the other hand, as an IL-17RA antagonist, inhibits the activity not only of IL-17A and IL-17F, but also of IL-17E (IL-25), leading to broader modulation of inflammatory pathways [16,17].

These drugs are characterised by a rapid onset of action and high clinical efficacy, assessed, among others, using the PASI index. In phase III clinical trials, secukinumab at a dose of 300 mg achieved PASI 75/90/100 responses in 77.1%, 54% and 24% of patients, respectively, at week 16 of therapy. Ixekizumab (80 mg after an initial dose of 160 mg) showed PASI 75/90/100 responses of 90%, 70% and 40%, respectively, and brodalumab (210 mg) showed PASI 75/90/100 responses of 83%, 70% and 42%, respectively [16,17].

IL-17 inhibitors are also effective in the treatment of nail psoriasis and psoriatic arthritis. Their safety profile is favourable – no significant increase in the risk of malignant tumours or severe infections has been observed. The most common adverse effects include upper respiratory tract infections, injection site reactions and mucocutaneous candidiasis. In some patients, exacerbation of inflammatory bowel disease has been observed, which requires caution in people with Crohn's disease or ulcerative colitis. There have also been isolated reports of suicidal thoughts during treatment with brodalumab, although the causal relationship remains unclear [18,19].

Interleukin 17 (IL-17) plays a key role in the pathogenesis of psoriasis by inducing a Th17-dependent inflammatory response and activating keratinocytes to produce cytokines, chemokines and antimicrobial peptides. The synergistic action of IL-17A and TNF-α amplifies the inflammatory process, leading to chronic skin lesions. Monoclonal antibodies targeting IL-17A (secukinumab, ixekizumab), IL-17A/IL-17F

(bimekizumab) or the IL-17RA receptor (brodalumab) are successfully used in the treatment of plaque psoriasis. These drugs are highly effective (PASI 75–100), have a rapid onset of action and a favourable safety profile. The most common adverse effects are upper respiratory tract infections and mild candidiasis; however, caution is required in patients with inflammatory bowel disease due to possible exacerbations. IL-17 inhibitors are also an effective therapeutic option in the treatment of nail psoriasis and psoriatic arthritis. [16,18,19]

3.3 The role of interleukin-23 p19 subunit inhibitors

The most recent class of biological drugs registered for the treatment of psoriasis are selective inhibitors of the p19 subunit of interleukin 23 (IL-23), including guselkumab, risankizumab and tildrakizumab. These drugs demonstrate high clinical efficacy and a favourable safety profile, representing a significant therapeutic advance in the treatment of plaque psoriasis and psoriatic arthritis. [20] Guselkumab, the first approved IL-23p19 inhibitor (FDA, 2017), outperformed adalimumab in the VOYAGE 1 and 2 studies (73% vs. 50% and 70% vs. 47% for PASI90 at week 16, respectively). At week 48, better results were also achieved than in the secukinumab-treated group. Rizankizumab demonstrated 75% efficacy in achieving PASI90 at week 16 in phase 3 studies, compared to 45% in the ustekinumab group. Tildrakizumab achieved PASI75 in 61–64% of patients in clinical trials, which exceeded placebo and, in some cases, etanercept. [21, 22]

At the molecular level, risankizumab is a fully human IgG monoclonal antibody that binds with high affinity to the p19 subunit of IL-23, which is unique to this cytokine, unlike the p40 subunit, which is also found in IL-12. IL-23 plays a key role in the immunopathogenesis of psoriasis by stimulating the differentiation of TH17 and TH22 cells, which initiate an inflammatory cascade leading to the overproduction of IL-17. In turn, IL-17 acts on keratinocytes, causing leukocyte recruitment, IL-19/IL-36 induction and STAT3 activation, leading to epidermal hyperplasia and the formation of psoriatic plaques. [20]

Selective IL-23 p19 inhibitors, such as risankizumab, are an effective and well-tolerated therapeutic option for the treatment of psoriasis. They are highly effective in achieving PASI90 and PASI100, are well tolerated and have a favourable safety profile, with no increased risk of cancer, cardiovascular complications or tuberculosis reactivation. Unlike IL-17 inhibitors, they are not associated with candidiasis or exacerbation of inflammatory bowel disease, which may be due to the preserved production of IL-17 by TH17-independent cells. Rizankizumab, as a representative of this class, has a rapid onset of action, sustained efficacy and comprehensive clinical benefits – both skin and joint – while maintaining a high level of treatment comfort for the patient. [20,22]

3.4 Mechanism of action, efficacy of phosphodiesterase 4 (PDE4) inhibitor

Apremilast is an oral phosphodiesterase-4 inhibitor that is effective and safe in the treatment of psoriasis. It is a low molecular weight drug approved in 2014 for the treatment of moderate psoriasis and active psoriatic arthritis. It works by inhibiting phosphodiesterase 4 (PDE4), resulting in an increase in cyclic adenosine monophosphate (cAMP) levels and a decrease in AMP, which reduces cell inflammation and keratinocyte activation/proliferation. This leads to a reduction in pro-inflammatory mediators, including TNF-α and IL-23. [23,24]

It is moderately effective in the treatment of both psoriasis and psoriatic arthritis, achieving efficacy comparable to methotrexate. Its advantages include oral administration and anti-inflammatory action that does not lead to immunosuppression. In addition, it has a good safety profile, does not require regular laboratory tests and may promote weight loss. [25] The PASI75 response with apremilast 30 mg twice daily ranged from 29% to 41% after 16 weeks of treatment in clinical trials. [26]

The most commonly reported adverse reaction in clinical trials was gastrointestinal intolerance, including diarrhoea (18%) and nausea (17%), and these rates appear to be even higher in everyday clinical practice. [27,28]

In a 2016 analysis by the American Academy of Dermatology in Washington, the incidence of serious cardiac events and malignant tumours was negligible. The authors of the analysis also observed the efficacy of apremilast in psoriasis affecting the male genital organs. [29]

4. Conclusions

Biological treatment represents a true revolution in the therapy of psoriasis, a disease that for many years was difficult to control effectively, and whose chronic, recurrent nature often significantly impaired patients' quality of life. Biological therapy, thanks to its precise targeting of specific molecular inflammatory mechanisms, opens a new era in medicine, in which it is possible not only to alleviate symptoms, but also to profoundly influence the course of the disease and prevent further exacerbations. The introduction of biological

drugs blocking TNF-α, IL-17 and IL-23 p19 is a breakthrough that has changed the lives of thousands of patients with psoriasis and psoriatic arthritis around the world. Their clinical efficacy, repeatedly confirmed in studies, allows for sustained remission and significant improvement in the condition of the skin and joints, which translates into a real improvement in patients' quality of life — both physically and mentally. It is a chance for a life without constant pain, itching and discomfort, which previously limited daily functioning and social relationships.

What is more, biological therapy brings great hope for the future. Thanks to further development and the introduction of new drugs that are increasingly precise and safe, it is possible to tailor therapy more and more closely to the individual needs of the patient. This is a step towards personalised medicine, where treatment is not just a general standard, but actually takes into account the specific course of the disease and the individual immunological characteristics of the patient.

Biological therapy is also an important tool in preventing complications of psoriasis, such as psoriatic arthritis, which can lead to permanent disability. The use of biological drugs not only treats the skin, but also protects the joints, which is crucial for maintaining the full physical fitness of patients.

Despite the need to monitor the safety of the therapy and potential side effects, the benefits of biological treatment far outweigh the risks. With proper medical care and close therapeutic supervision, this treatment is not only effective but also safe, giving patients real hope for a normal, full life.

In summary, biological therapy is a real breakthrough and a source of hope for people suffering from psoriasis. It is a milestone that makes it possible to transform a chronic and burdensome disease into a condition that can be effectively controlled. As research progresses and new therapies are developed, biological treatment will become even more effective and accessible, which in the future may mean a significant reduction in the burden of disease worldwide.

Authors' contributions:

All authors contributed to the article: conceptualization: PP, methodology: WS, PB, DL-S software: WS, PP, AB, KŁ, check: BK, JL, formal analysis: OJ, WP, investigation: PP, WS, PB resources: DL-S, WP, data curation: AB, PP, OJ writing -rough preparation: KŁ, JL, writing -review and editing: PP, WS, DL-S, PB, WP, BK, KŁ visualization: WS, AB. supervision: WS; project administration: PP. All authors have read and agreed with the published version of the manuscript.

Conflict of Interest Statement: The authors report no conflict of interest.
Financial: The study did not receive any funding
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.

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