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Efficacy and safety of monoclonal antibodies used in the treatment of psoriasis

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Abstract

Biologics have revolutionized the therapeutic approach to many diseases of inflammatory etiology. In dermatology, they have been used, among others, in treating psoriasis, which affects 1-3% of the Caucasian population and is often a therapeutic challenge for physicians. Although the cause of psoriasis remains unknown, T lymphocytes and cytokines such as TNF-α, IL-17, and IL-23 play a key role in its pathogenesis. This article discusses traditional psoriasis therapies, such as phototherapy and topical drugs, and modern biological treatments, which focus on inhibiting specific inflammatory pathways. Particular attention is paid to TNF-α inhibitors (adalimumab, infliximab), IL-17 inhibitors (ixekizumab, secukinumab, bimekizumab, brodalumab), IL-12/23 inhibitors (ustekinumab) and IL-23 inhibitors (risankizumab, tildrakizumab, guselkumab), describing their efficacy, dosage and potential side effects. *(Farm Współ 2025; 18: 71-80) doi: 10.53139/FW.20251811*

Keywords: psoriasis, biologics, monoclonal antibodies

Introduction

Psoriasis is a chronic inflammatory disease affecting approximately 1–3% of the Caucasian population [1,2]. The most common clinical presentation of psoriasis is psoriasis vulgaris (plaque psoriasis), distinguished by erythematous plaques covered with silvery scales. The typical localization of plaques is on the elbows, knees, scalp, or lower back [3]. The severity and extent of psoriasis are measured using the Psoriasis Area and Severity Index (PASI score), which rates the intensity of erythema, induration, and scaling of psoriatic plaques and which helps evaluate the efficacy of the treatment [4]. The cause of psoriasis remains unknown, but the pathophysiology of the disease is complex. Several immune-related genes have been associated with psoriasis, particularly multiple human leukocyte antigen (HLA) genes and genes involved in specific inflammatory pathways [5-8]. T lymphocytes, the most prevalent inflammatory cells in the skin, play a crucial role in the immunopathology of psoriasis. Dendritic cells (DCs) are also increased in psoriatic lesions and contribute to shape the T-cell response [9,10]. Chemokines and cytokines synthesized by activated T-cells, such as TNF-α, IFN-γ, and interleukins like IL-17 (produced by Th17 cells), stimulate keratinocytes to hyperproliferate. It has been shown that natural killer T cells (NKT cells), which express several receptors for both classical and non-classical class I MHC molecules, can be activated by keratinocytes to produce large amounts of IFN-γ [11]. Increased levels of TNF-α have been observed in psoriatic skin lesions. This cytokine elevates the expression of ICAM-1 on endothelial cells and keratinocytes, a glycoprotein crucial for cellular adhesion and trafficking. TNF-α interacts with two distinct TNF receptors [TNFR1 (p55 or CD120a) and TNFR2 (p75 or CD120b)], which are expressed on a wide range of cells, including keratinocytes and dendritic cells [12]. Through these receptors, TNF-α amplifies inflammation via several pathways, activating T lymphocytes, increasing T cell infiltration, and promoting keratinocyte proliferation in psoriasis [13]. Certain external factors, such as drugs, bacterial infections, or stress, may increase the likelihood of developing psoriasis [14-16]. Many of the above mechanisms of the inflammatory process have become the target of biologics used in modern medicine. Binding antibodies to cytokines or

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their receptors inhibits signal transduction, leading to inflammation and thus alleviating disease symptoms. Monoclonal antibodies are laboratory-engineered antibodies that specifically bind to target proteins involved in inflammatory processes, such as TNF-alpha, thereby modulating the immune response and reducing disease symptoms.

Biologics have revolutionized the treatment of many diseases in dermatology and other fields of medicine, such as rheumatology, oncology, and gastroenterology [17-19].

The following article aims to discuss the efficacy and safety of selected monoclonal antibodies used to treat psoriasis. Additionally, it includes tables comparing well-known biologic drugs for psoriasis therapy and analyzing clinical trials regarding PASI score changes during treatment.

Standard therapies

The treatment depends on the affected area but generally includes phototherapy and topical medications such as calcipotriol in combination with betamethasone dipropionate, primarily due to their synergistic anti-psoriatic effects. Other treatments include vitamin D3 derivatives, topical glucocorticoids, coal tar, and calcineurin inhibitors [20]. Calcineurin inhibitors are particularly recommended for use on the face and in cases of inverse psoriasis affecting skin folds and joint creases [21]. Patients with mild psoriasis may be treated primarily with topical agents, while those with moderate to severe psoriasis should be considered for additional systemic therapies.

For systemic treatment of psoriasis, various medications are used, including cyclosporine A, methotrexate, acitretin, apremilast, and dimethyl fumarate (fumaric acid ester). Systemic treatment also includes biological drugs, encompassing a wide range of monoclonal antibodies. A meta-analysis conducted by Sbidian et al. in 2023 showed that biologic treatments anti-IL17, anti-IL12/23, anti-IL23, and anti-TNF alpha showed a higher proportion of patients reaching PASI-90 than the non-biological systemic agents [22].

Immunotherapy in the treatment of psoriasis

Tumor necrosis factor inhibitors

TNF inhibitors are the standard treatment for all patients with moderate-to-severe psoriasis as part of reimbursed therapy. Monoclonal antibodies against TNF used in psoriasis include adalimumab and infliximab. Adalimumab is the first recombinant, fully human immunoglobulin G1 monoclonal antibody that binds with high affinity and specificity to TNF-α and blocks the interaction of TNF with p55 and p75 cell surface TNF receptors [23].

Adalimumab is used to treat plaque psoriasis, Crohn's disease (CD), ulcerative colitis (CU), rheumatoid arthritis (RA), psoriatic arthritis (PsA), juvenile idiopathic arthritis, ankylosing spondylitis, and uveitis [24]. The initial dose of adalimumab for adult patients is 80 mg administered subcutaneously, followed by 40 mg every other week, starting one week after the initial dose [25]. A peak serum concentration attained 131 h after administration of a single 40 mg dose, and the estimated average absolute bioavailability was 64%, the median apparent total clearance of adalimumab was 21 mL/h, and the median apparent volume of distribution was 11.3 L. The mean terminal half-life of adalimumab was approximately 2 weeks after doses of 0.5 mg/kg (approximately 40 mg) [26, 27]. The therapeutic efficacy of adalimumab has been demonstrated in randomized, double-blind, multicenter phase III and IIIb trials. The longest study was a 52-week trial involving 1212 patients randomized to receive either adalimumab (40 mg) or a placebo. By week 16, 71% of patients receiving adalimumab achieved at least a 75% improvement in the PASI score. Most of those who experienced clinically significant improvement by week 16 could maintain this response during open-label adalimumab therapy through week 33. The efficacy of adalimumab in clinical trials, as measured by the PASI-75 response rate, was 83% at week 48 [28]. The most common side effects of adalimumab include injection site reactions and skin rashes. Adalimumab also appears to increase the risk of infections. The most frequent adverse events compared to placebo are upper respiratory infections, sinusitis, flu-like symptoms, and urinary tract infections. Rare side effects include the worsening or onset of congestive heart failure, a lupus-like syndrome, the potential promotion of lymphoma, medically significant cytopenias, and the worsening or onset of multiple sclerosis or other neurological diseases [29]. Adalimumab is an effective medication that can be safely used when its potential side effects are appropriately managed.

Infliximab is a chimeric human-mouse IgG1 monoclonal antibody developed to target TNFα specifically. It is used in treating several inflammatory

disorders, including RA, CD, and CU [24]. For adults, infliximab is dosed at 5 mg/kg body weight, administered as an intravenous infusion at weeks 0, 2, and 6, and then every 8 weeks thereafter. The peak infliximab concentrations, with medians of 158.14 mg/mL for the 5 mg/kg dose group and 298.89 mg/mL for the 10 mg/kg dose group, were observed immediately after the second dose on day 14 [30].

In the prospective, observational, open-label, multicenter study REALITY, involving 521 patients, the efficacy of infliximab at a dose of 5 mg/kg (administered at 0, 2, and 6 weeks for induction, followed by maintenance every 8 weeks) resulted in 56.8% of patients achieving PASI 75 at week 50 [29].

In a large clinical trial by Menter in 2007, 835 Patients with moderate-to-severe psoriasis were randomized to induction therapy with infliximab 3 mg/kg or 5 mg/kg or placebo. At week 10, 75.5% (5 mg/kg group) and 70.3% (3 mg/kg group) of patients in the infliximab achieved PASI 75 [31].

Barker et al. achieved an even better result in the RESTORE1 trial involving 868 patients. The study aimed to compare the efficacy and safety of infliximab versus methotrexate in adults with moderate-to-severe plaque psoriasis. The primary efficacy endpoint, PASI 75 response at week 16, was 78% in the infliximab group and 42% in the methotrexate group. By week 26, the PASI 75 response was 77% in the infliximab group and 31% in the methotrexate group [32]. A multicenter retrospective study by Kim et al. from 2015 found that 15% of patients withdrew from infliximab therapy due to adverse effects. The most common side effects were infusion-site reactions, infections, and malignancies [33].

TNF-α inhibitors may trigger autoimmune responses. Patients treated with these inhibitors often develop antinuclear antibodies (ANA), though drug-induced lupus remains rare. A study by Poulalhon et al. found that ANA positivity increased from 12% at baseline to 72% at week 22 in patients receiving infliximab, but no cases of drug-induced lupus were reported [34]. Evidence shows that long-term use of TNF alpha inhibitors may increase the risk of malignancies. This risk underscores the importance of thorough pre-treatment assessments and ongoing monitoring, especially in patients with a history of cancer [35].

Finally, hematological abnormalities, including pancytopenia, have been observed. The RESTORE2 study, which examined the long-term effects of infliximab, documented various hematological conditions, highlighting the importance of conducting regular blood counts before and during treatment [36].

Contraindications for the use of adalimumab and infliximab include hypersensitivity to TNF inhibitors and active or latent viral, bacterial, fungal, or parasitic infections, particularly HIV, HBV, HCV, and *Mycobacterium tuberculosis* infections. TNF-α plays a crucial role in both the host immune response to *Mycobacterium tuberculosis* infection and the immunopathology of tuberculosis. This underscores the necessity of screening patients for these conditions before initiating treatment. The diagnostic tests performed as part of the treatment program include a chest X-ray, a tuberculin skin test or QuantiFERON test, HBs antigen presence, anti-HCV antibodies, anti-HIV antibodies, and anti-*Borrelia burgdorferi* antibodies to rule out the conditions mentioned above [25].

Despite the exclusion of pregnant women from the treatment program, research suggests that adalimumab and other TNFα inhibitors have not shown specific safety concerns for these groups. Adalimumab appears safe for use during the first trimester of pregnancy [37].

Interleukin-17 inhibitors

The second major group of biological medicines includes interleukin-17 inhibitors, such as the monoclonal antibodies ixekizumab and secukinumab, which directly inhibit IL-17A, and brodalumab, which blocks the IL-17 receptor. Bimekizumab, the newest medication in this group, was approved by the FDA in October 2023 and inhibits both IL-17A and IL-17F [38].

By inactivating IL-17A, these monoclonal antibodies inhibit the downstream pathway of chemokine production by keratinocytes, thereby reducing the recruitment of inflammatory cells and stimulating the innate immune system [39].

Ixekizumab and secukinumab are used to treat psoriasis, PsA, ankylosing spondylitis, and non-radiographic axial spondyloarthritis. However, brodalumab is approved only for treating plaque psoriasis, with a recommended dose of 210 mg administered at weeks 0, 1, and 2, followed by 210 mg injections as needed. The recommended dosing regimen for ixekizumab involves an initial dose of 160 mg administered as subcutaneous injections at week 0, followed by 80 mg at weeks 2, 4, 6, 8, 10, and 12, and then a maintenance dose of 80 mg administered every 4 weeks.

For secukinumab, the recommended dose is 300 mg administered via subcutaneous injection. It

is initially given at weeks 0, 1, 2, 3, and 4, followed by monthly maintenance doses [20, 25].

Phase 3 trials called UNCOVER-1, UNCOVER-2, and UNCOVER-3 highlighted ixekizumab's high efficacy in treating psoriasis. In UNCOVER-1, 89.1% of participants achieved PASI 75 by week 12, compared to just 3.9% in the placebo group. Additionally, 70.9% and 35.3% of participants in the ixekizumab 2-week dosing group achieved PASI 90 and PASI 100, respectively. Similar outcomes were seen in UNCOVER-2 and UNCOVER-3. A total of 3866 participants diagnosed with moderate-to-severe plaque psoriasis were involved in these studies [40,41].

Another phase 3 clinical trial for ixekizumab was SPIRIT-P1, with patients suffering from PsA. The most frequent treatment-emergent adverse effects were upper respiratory tract infection, bronchitis, and injection-site reactions or erythema [42]. In UNCOVERS studies, infections were most frequently reported. 67.2% of patients experienced infections, mainly nasopharyngitis and upper respiratory tract infections [43].

Another IL-17 inhibitor, secukinumab, was compared to another monoclonal antibody (guselkumab) in the phase 3 randomized controlled trial ECLIPSE.

The ECLIPSE trial included 1048 patients, with 534 assigned to receive guselkumab and 514 to receive secukinumab. Although guselkumab showed better results after 48 weeks, with 84% of patients achieving PASI-90, the outcomes in the secukinumab group were still favorable. Secukinumab was administered at a dose of 300 mg at weeks 0, 1, 2, 3, and 4, followed by every 4 weeks, and achieved a PASI-75 response in 92% of patients at week 12, outperforming guselkumab at this time point. The side effects observed in the ECLIPSE trial were similar to those reported in trials for ixekizumab. The most common side effects were nasopharyngitis and upper respiratory tract infection [44].

AMAGINE-1 was a phase III study designed to evaluate the efficacy of brodalumab in adult patients with chronic plaque psoriasis. At week 12, 83% of patients achieved a PASI 75 response, with 60% of those receiving 140 mg of brodalumab and 83% of those receiving 210 mg achieving this level of improvement. The most frequent adverse events were nasopharyngitis, upper respiratory tract infection, arthralgia, and headache. There was one report of exposure-emergent carcinoma—metastatic carcinoma of the small intestine. These results are similar to those observed with other IL-17 inhibitors [45].

Bimekizumab was investigated in the BE READY study in 2021, which evaluated its efficacy and safety in patients with moderate to severe plaque psoriasis. In this study, completely clear skin (PASI100) was achieved by 68% of patients within 16 weeks of taking bimekizumab 320 mg every 4 weeks.

Bimekizumab was well tolerated, with side effects similar to other medications in this group. The most common were infections, including oral fungal infections; moreover, two cases of malignancy were reported in the study [46].

Because of its high efficacy and safety, new studies are being conducted to evaluate patients who have switched from other biologic medications. Most PASI 90 nonresponders achieved PASI 90 within 4 weeks after switching to bimekizumab from adalimumab (67%), ustekinumab (79%), and secukinumab (53%) [47].

Interleukin 12 and interleukin 23 inhibitors

So far, only ustekinumab has been used in this group of drugs [48]. Ustekinumab is a human monoclonal antibody that binds to the common p40 subunit of interleukin 12 (IL-12) and interleukin 23 (IL-23). Binding to the interleukins prevents further signal transmission to target T-cells [49]. FDA approved ustekinumab in the treatment of inflammatory diseases such as psoriasis, PsA, CU, and CD, but it is widely applied off-label in many more conditions [50].

The drug is administered subcutaneously with the first dose of 45 or 90 mg, depending on whether the patient weighs ≤100 kg or more than 100 kg. After 4 weeks, it must be administered the same dose as the first one, and then the injections are continued with the same dose (either 45 mg or 90 mg) every 12 weeks [50].

In 2021, K. Reich et al. published a study called BE VIVID in which they compared the efficacy of ustekinumab, bimekizumab, and placebo in treating moderate to severe plaque psoriasis. The study was a multicentre, randomized, double-blind, active comparator and placebo-controlled phase 3 trial conducted across 105 sites in 11 countries on four continents. The study included 567 patients over 18 years of age with moderate to severe plaque psoriasis, randomly assigned to the study groups. The group of patients treated with ustekinumab consisted of 163 people, who were given the drug according to the recommended dosage (45 mg or 90 mg) and were evaluated after 16 weeks. 81 out of 163 people (50%) had a PASI-90 index, meaning half of the patients had a 90% reduction in lesions after the

treatment. In comparison, PASI-90 was achieved in only 5% of placebo-treated patients. Through 52 weeks, serious adverse events occurred in 13 of the patients (8%) in the ustekinumab group. These serious adverse events were hepatic events (4 cases), serious infections (4 cases), basal cell carcinoma (1 case), non-melanoma skin cancer (1 case), and adjudicated suicidal ideation and behavior (1 case) [51].

Four years earlier, in 2017, Papp et al. conducted another study comparing the effects of ustekinumab with risankizbumab. This multicenter, randomized, 2-phase trial was conducted in Europe and North America and included patients aged 18 to 75 with moderate-to-severe plaque psoriasis. The study included 166 people, of whom 40 were assigned to the ustekinumab group. Ustekinumab was administered according to guidelines (as above). At week 12, PASI-90 was achieved in 40% of patients treated with ustekinumab and PASI-75 in 72% of them. Through 48 weeks, adverse events occurred in 72% of the patients in the ustekinumab group, and three serious adverse events were reported. These were Bronchitis (1 case), Diverticulitis (1 case), and Urinary tract infection (1 case) [52].

In both studies, ustekinumab's significant results were distinct from the other biological outcomes with which they were related.

Interleukin 23 inhibitors

Due to suggestions that IL-12 has a positive effect on the regulation of skin inflammation and, therefore, its blocking by drugs binding the p40 subunit of IL-12 and IL-23 does not provide additional benefits, drugs that specifically inhibit IL-23 have been introduced into the treatment [53]. These drugs bind to the p19 subunit, selectively blocking only IL-23 without affecting IL-12. Currently, the drugs used in this group in Poland are guselkumab, tildrakizumab, and risankizumab.

Risankizumab exhibits linear pharmacokinetics following subcutaneous or intravenous administration. Bioavailability is estimated at 89%, with peak serum concentrations occurring between 3 and 14 days after administration. The biological half-life is long and is approximately 28 days [54]. Risankizumab was studied in one of the studies mentioned above, conducted by Kim A. Papp et al. in 2017. In the described study, patients were divided into four groups, of which three groups were given risankizumab at different doses, and one group was given ustekinumab (as above). In one group of patients treated with risankizumab, a single dose of 18 mg was given at week 0, while in the other two groups, 90 or 180 mg, were given at weeks 0, 4, and 16, respectively. At week 12 of the study, PASI-90 was achieved in 33% of patients treated with a single dose of 18 mg of risankizumab, 73% of patients in the 90 mg risankizumab group, and 81% of patients in the 180 mg risankizumab group, respectively. In comparison, PASI-90 was achieved in 40% of patients who were given ustekinumab (see above). This study showed that risankizumab, a selective IL-23 inhibitor, had better effects in treating psoriasis than ustekinumab, which blocks IL-12 and IL-23 [52]. In the groups of 18 mg and 90 mg of risankizumab, respectively, five patients (12%) and six patients (15%) had serious adverse events. In the group of 180 mg, no serious adverse events were reported.

Tildrakizumab

In 2017, Kristian Reich et al. published results of the studies called reSURFACE 1 and reSURFACE 2 comparing the effectiveness of guselkumab and etanercept with placebo in treating psoriasis. They were two three-part, parallel-group, double-blind, randomized controlled studies run at 118 sites on four continents [55]. Patients included in the studies were 18 or older and had moderate-to-severe chronic plaque psoriasis. In the reSURFACE 1 study, patients were randomly assigned to receive tildrakizumab 200 mg, 100 mg, or placebo, respectively. In the second part of this study, patients receiving placebo were randomly assigned to receive tildralizumab 200 mg or tildralizumab 100 mg.

In the reSURFACE 2 study, participants were randomly assigned to tildrakizumab 200 mg, 100 mg, placebo, or etanercept 50 mg. In the second part of this study, patients receiving placebo were assigned to tildralizumab 200 mg or tildralizumab 100 mg. Tildrakizumab was administered subcutaneously at weeks 0 and 4 during part 1 and at week 16 during phase 2 (weeks 12 and 16 for participants re-randomized from placebo to tildrakizumab; etanercept was given twice weekly in part 1 of reSURFACE 2 and once weekly during phase 2). The effectiveness of treatment in the study groups is presented in table I and table II. In the studies, tildrakizumab at doses of 100 mg and 200 mg showed superior efficacy in treating psoriasis compared with etanercept and placebo. The percentage of serious adverse events was low and similar in all study groups, and the most common adverse event was nasopharyngitis [55].

Guselkumab

The efficacy of guselkumab in treating psoriasis was studied in one of the abovementioned studies – the ECLIPSE trial. Each of the 534 patients in the guselkumab group was given the drug at a dose of 100 mg at weeks 0 and 4, then every 8 weeks. The PASI-90 response at week 48 among patients treated with guselkumab was 84% (451 patients). The most common adverse event of guselkumab was nasopharyngitis [44].

Table I. Treatment efficacy in the reSURFACE 1 study assessed at week 12

	200 mg tildrakizumab	100 mg tildrakizumab	placebo
PASI-75	62% (192 patients)	64% (197 patients)	6% (9 patients)
PGA responses	59% (182 patients)	58% (179 patients)	7% (11 patients)

Table II. Treatment efficacy in the reSURFACE 2 study assessed at week 12

	200 mg tildrakizumab	100 mg tildrakizumab	etanercept	placebo
PASI-75	66% (206 patients)	61% (188 patients)	6% (9 patients)	48% (151 patients)
PGA responses	59% (186 patients)	55% (168 patients)	4% (7 patients)	48% (149 patients)

Table III. Biologic drugs for psoriasis – characteristics and contrindications

Medication	Group	Type of antibodies	Main Side effects	Contraindications	Approved for the treatment of psoriasis
Adalimumab	TNF inhibitor	human monoclonal antibody	injection site reactions, infections, skin rashes, worsening or initiation of congestive heart failure, a lupus-like syndrome, a promotion of lymphoma, cytopenia, worsening or initiation of multiple sclerosis/neurological disease	hypersensitivity to adalimumab or any of the excipients, severe infections, malignancy, moderate to severe heart failure (NYHA III/IV)	FDA in 2008
Infliximab	TNF inhibitor	human-mouse monoclonal antibody	infusion-site reactions, infections, malignancies, a promotion of lymphoma	hypersensitivity to infliximab or any of the excipients, active infections, severe infections (sepsis, tuberculosis), moderate to severe heart failure (NYHA III/IV)	FDA in 2006
Ixekizumab	IL-17 inhibitor	recombinant humanized monoclonal IgG4 antibody	infections, injection site reactions, sore throat, neutropenia	severe hypersensitivity to ixekizumab, active infections	FDA in 2016
Secukinumab	IL-17 inhibitor	recombinant human monoclonal IgG1 antibody	nasopharyngitis, headache, neutropenia, nausea, diarrhea, dizziness	hypersensitivity to secukinumab or any of the excipients, hypersensitivity to latex, active infections (especially tuberculosis, HIV, hepatitis B, C)	FDA in 2015

Brodalumab	IL-17 inhibitor	recombinant human monoclonal IgG2 antibody	nasopharyngitis, upper respiratory tract infection, arthralgia, headache	hypersensitivity to brodalumab or any of the excipients, inflammatory bowel disease, active infections	FDA in 2017
Bimekizumab	IL-17A and IL-17F inhibitor	humanized monoclonal IgG1 antibody	upper respiratory tract infection, headache, injection site reaction	hypersensitivity to bimekizumab or any of the excipients, active infections	FDA in 2023
Ustekinumab	IL-12 and IL-23 inhibitor	human monoclonal antibody	nasopharyngitis, headache, fatigue, vomiting	hypersensitivity to ustekinumab or any of the excipients, Active infection, Active tuberculosis	FDA in 2009
Risankizumab	IL-23A inhibitor	humanized monoclonal antibody	upper respiratory tract infections, fungal skin infections, headache, itching, fatigue	hypersensitivity to risankizumab or any of the excipients, active tuberculosis	FDA in 2019
Tildrakizumab	IL-23 inhibitor	humanized IgG1 monoclonal	upper respiratory tract infections, headaches, nausea, gastroenteritis, diarrhea, injection site pain, back pain	hypersensitivity to tildrakizumab or any of the excipients, active tuberculosis	FDA in 2018
Guselkumab	IL-23 inhibitor	human monoclonal antibody	upper respiratory tract infections, headache, diarrhea, joint pain, urticaria, fungal skin infections	hypersensitivity to guselkumab or any of the excipients, active tuberculosis	FDA in 2017

Conclusion

Monoclonal antibodies used in treating psoriasis have varying effectiveness and adverse effects, which should be considered when selecting treatment.

TNF inhibitors, infliximab, and adalimumab, have the longest history in treating psoriasis. Clinical trials show that both adalimumab and infliximab are highly effective in achieving PASI 75, with adalimumab demonstrating sustained efficacy over time and infliximab showing a rapid response. The safety profile of adalimumab appears to be more favorable than that of infliximab.

IL-17 inhibitors, including ixekizumab, secukinumab, brodalumab, and the newest addition, bimekizumab, have shown high efficacy, with PASI 75 response rates between 83% and 92% within the first 12 weeks. Bimekizumab, which targets both IL-17A and IL-17F, has demonstrated particularly strong results, achieving PASI 100 in 68% of patients at week 16. The most common side effects are upper respiratory infections.

Interleukin-12 and interleukin-23 inhibitors, such as ustekinumab, guselkumab, tildrakizumab, and risankizumab, target different subunits of IL-12 and IL-23 to inhibit inflammation. Clinical studies show that ustekinumab achieves PASI 90 in around 50% of patients. Newer drugs like risankizumab and guselku-

mab, specifically targeting IL-23, show higher efficacy, with PASI 90 responses reaching 70-80%. Common side effects include infections like nasopharyngitis.

The differences in efficacy and safety among various biological drug groups stem from their distinct mechanisms of action, with IL-17 and IL-23 inhibitors showing higher efficacy in achieving PASI 90. In contrast, TNF-α inhibitors, such as adalimumab, offer stable long-term outcomes. The choice of therapy depends on the individual patient's needs, clinical condition, and treatment tolerance, making further research on long-term safety and treatment optimization essential. A comparison of all the aforementioned drugs is presented in table III.

Conflict of interest
None

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