NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

Journal of Education, Health and Sport. eISSN 2391-8306.

Journal Home Page
https://apcz.umk.pl/JEHS/index

PACEK, Szymon, MUDA, Martyna, KALINOWSKI, Paweł, BACHURSKI, Patryk, WIŚNIEWSKI, Karol Paweł, MIĘDLAR, Maja, PIERZGA, Elisabetta, ZARAŃSKI, Bartosz, CHMIEL, Gabriela, and WITKOWSKI, Paweł. The Evolving Landscape of Psoriasis Treatment: Current Biologics and Novel Targets. Journal of Education, Health and Sport. 2026;90:69823. eISSN 2391-8306. https://doi.org/10.12775/JEHS.2026.90.69823

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 14.03.2026. Revised: 31.03.2026. Accepted: 10.04.2026. Published: 14.04.2026.

The Evolving Landscape of Psoriasis Treatment: Current Biologics and Novel Targets

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ABSTRACT

Introduction. Psoriasis is recognized as an auto- immune inflammatory disorder characterized by hyperproliferation of keratinocytes and infiltration of T- cells. Disease can occur at any age and may cause episodes of depression and psoriatic arthritis. Currently the researchers are trying to find the most valuable and specific targets in treatment.

Purpose. The primary purpose of our review is to provide comperhensive update on the most valuable and possibly best new rapidly evolving scientific approaches in treatment of chronic disease such as psoriasis.

Materials and Methods. We have reviewed PubMed data base in order to identify clinical trials, meta analyses and randomized controlled trials form the past 5 years.

Results. The review confirms the IL-23/IL-17 axis as the primary pathogenic driver. While management utilizes effective topical, systemic, phototherapy, and advanced biological agents (TNF-alpha, IL-17, and IL-23/IL-12 inhibitors), the continued therapeutic challenge, particularly for refractory patients, drives the need for innovative treatments. The emergence of the first oral, selective allosteric TYK2 inhibitor (deucravacitinib) and ongoing research into

targets like IL-21 and the microbiota highlight the rapidly evolving landscape of psoriasis therapy.

Keywords: Psoriasis (PsO),Biological Treatment,Deucravacitinib, biological treatment, TYK2 inhibitors, IL23/IL-17 axis, TNF-alpha inhibitors, Methotrexate, Phototherapy, innovative therapies

Introduction and purpose

Psoriasis(PsO) is one of the most common systemic auto-immune disorders involving skin manifestations. In the United States psoriasis prevalence is estimated at 3,0% among adults at age of 20 or more. There is no significant difference in occurrence between sexes. Study has shown that occurrence of disease was highest in white individuals. (1) Main phenotypes of PsO include plaque, inverse, guttate, pustular, and erythrodermic psoriasis. The most common symptoms include salmon pink like plaques with silvery white scales located especially on extensor surfaces of limbs along with scalp and trunk (2).

Aetiology

Genetic factors are known as the one of the most important and influential components in etiology of PsO. There is a well-established correlation between genetic predisposition and increased disease prevalence among first and second degree relatives suffering from the disease. The average age of symptoms occurrence ranges from 15 to 20 years of age. There is a well-established correlation between HLA-Cw6 positivity and early disease manifestation. (3)

Beyond the genetic background there are well known external factors triggering disease onset. Psychological stress is a well known risk factor in the incidence of disease. Stressful situations modulate immune responses, triggering the upregulation of pro-inflammatory cytokines that exacerbate psoriatic symptoms. (4)

Some of the drugs such as antimalarial drugs, interferons, lithium, beta- blockers or tetracyclines may cause onset or flare-up of PsO. These medications may dysregulate inflammatory responses, potentially precipitating a disease relaps**e.**

Pathogenesis

The pathogenesis of psoriasis is a complex autoimmune process characterized by excessive, uncontrolled keratinocyte proliferation driven by chronic inflammation. (5) The onset of

psoriatic skin inflammation may be triggered by psychological stress, mechanical trauma (the Koebner phenomenon), and environmental factors. Following the stimulation of myeloid dendritic cells, interleukin-23 is released, which promotes the activation of Th17 cells and the subsequent secretion of interleukin-17.

Interleukin-17 acts upon keratinocytes, leading to dysregulated proliferation and the production of cytokines that perpetuate the inflammatory state. With deeper insight into the genetics and pathogenesis of the condition, focus has shifted toward targeted therapies, which have recently demonstrated increasingly high rates of therapeutic success. (5,6)

Clinical Types of Psoriasis

Depending on the type of lesions, their anatomical localization, and morphology, the following clinical types of psoriasis are distinguished:

- **Plaque psoriasis** – currently the most prevalent form of psoriasis worldwide. It occurs in approximately 85–90% of diagnosed patients. Lesions are characterized by sharply demarcated, pruritic, silvery scales on an inflammatory base. Common sites of involvement include the scalp, the extensor surfaces of the extremities, and the trunk.
- **Inverse psoriasis** – the most characteristic sites of involvement are intertriginous areas. The lesions present as clearly defined, erythematous plaques. Bacterial or fungal infections are highlighted as potential contributing factors in the pathogenesis of inverse psoriasis. (7,8)
- **Guttate psoriasis** – this type is characterized by its predominance in children and young adults. The onset of lesions is typically preceded by a streptococcal tonsillar infection. Patients present with small, droplet-like papular lesions on the skin surface.
- **Pustular psoriasis** – a typical feature of this form is the presence of multiple sterile pustules. Based on lesion localization, two phenotypes can be distinguished:
    1. *Psoriasis pustulosa palmoplantaris*, which is localized exclusively on the palms and soles;
    2. *Acrodermatitis continua of Hallopeau*, which involves the nails and is localized to the distal phalanges of the hands and feet. (7,9)

Diagnosis

Currently, the cornerstone of psoriasis diagnosis is a comprehensive clinical examination of the patient combined with the histopathological evaluation of skin biopsy specimens. The characteristic morphology of the lesions and their anatomical distribution constitute indications for performing a skin biopsy. The most common sites of involvement include the extensor surfaces of the extremities, the scalp, and the intergluteal cleft.

Specialized scoring systems have been developed to assess the severity and extent of lesions; these tools also facilitate the evaluation of treatment response and patient well-being. These include the Psoriasis Area and Severity Index (PASI). The PASI score can be highly instrumental in monitoring the patient's response to the prescribed therapy. Another indicator is the Physician Global Assessment (PGA), which allows for the evaluation of the extent of lesions at a specific point in time. It is important to note that a significant limitation of the aforementioned scales is their dependence on the examiner. (10,11)

Topical Treatment

Depending on the extent and anatomical localization of the lesions, topical corticosteroids are selected as the first-line treatment for psoriasis in the majority of cases. Corticosteroids attenuate inflammation, induce vasoconstriction, and inhibit cell proliferation. Depending on the severity of the lesions, corticosteroids ranging from Class 1 (super-potent) to Class 7 (least potent) may be administered. A meta-analysis has demonstrated that the combination of vitamin D3 and corticosteroids is highly effective for lesions occurring on the scalp. In patients with active lesions, topical preparations are typically applied twice daily.

Topical preparations containing vitamin D analogs also constitute a viable option for the management of localized lesions. Their mechanism of action involves the inhibition of excessive keratinocyte proliferation and the stimulation of differentiation. Vitamin D analogs are widely considered safe, with a favorable side-effect profile.

Calcineurin inhibitors may also be utilized as an effective topical treatment alternative. These agents reduce the production of pro-inflammatory mediators, thereby achieving local symptomatic relief. The recommended formulations for tacrolimus are 0.03% or 0.1% ointments. This therapy is considered safe; however, it is not recommended for use in pregnant or lactating women due to the excretion of tacrolimus into breast milk.

Non-pharmacological topical preparations may also be employed. These substances include emollients and moisturizing creams, which facilitate the reduction of pruritus and skin fissuring, while also enhancing the absorption of topical medicinal agents.

Systemic Treatment

Cyclosporine is an agent administered orally to patients with moderate-to-severe disease activity. The primary mechanism of action of cyclosporine is the inhibition of calcineurin, which results in the suppression of T-cell activity. Although cyclosporine exhibits a rapid onset of action and effectively induces remission, its unfavorable safety profile often precludes its use in long-term therapy. (11, 12)

The action of **methotrexate** involves the competitive inhibition of folate-dependent pathways, attributable to its structural similarity to folic acid. This results in the inhibition of proper DNA and RNA synthesis. (13) While methotrexate is effective in treating and alleviating psoriatic symptoms, regular monitoring of the patient and their clinical status is required due to potential adverse effects.

Acitretin is one of the oral agents employed in the therapy of psoriasis. It belongs to the retinoid class and is a derivative of vitamin A. Notable therapeutic effects of acitretin include the inhibition of excessive keratinocyte proliferation, resulting in a reduction of psoriatic lesions. However, due to its teratogenic potential, the use of this preparation is strictly contraindicated during pregnancy.

Phototherapy

Contemporary psoriasis treatment algorithms position phototherapy as a pivotal alternative or adjunctive method to conventional topical and systemic pharmacotherapy. The introduction of narrow-band ultraviolet B (NB-UVB) radiation, with a strictly defined, therapeutically optimal wavelength of 311 nanometers, constituted a breakthrough in dermatology.

Clinical indications for this modality primarily include patients with generalized lesions covering at least 10% of the total body surface area (BSA). The molecular mechanism of action of UVB radiation is multifaceted; it has been demonstrated to induce local immunosuppression and inhibit excessive epidermal proliferation. A key process involved is the induction of apoptosis within pathogenic T-cell populations, particularly the Th17 subset, as well as the

modulation of regulatory T-cell (Treg) function. This leads to the restoration of the immune homeostasis disrupted in the pathogenesis of the disease.

Concurrently, within the spectrum of available photochemotherapy methods, PUVA is widely recognized as the "gold standard" and the most effective therapeutic option, particularly indicated for the treatment of chronic, localized psoriatic plaques. (14)

Biological Treatment

In recent years, biological treatment for psoriasis has constituted a significant breakthrough. New biological therapies have proven highly effective in a subset of patients who did not respond adequately to previous treatment stages or were refractory to them. Currently, there are 11 FDA-approved agents available on the market and in clinical practice. These registered preparations can be categorized into four main groups: TNF-alpha inhibitors, IL-17 inhibitors, IL-23 inhibitors, and IL-12/23 inhibitors.

TNF-alpha Inhibitors

The group of TNF-alpha inhibitors includes etanercept, adalimumab, certolizumab, and infliximab.

- **Etanercept** is a recombinant human TNF-α receptor protein. Currently, this agent is approved for the treatment of psoriasis at a dosage of 50 mg twice weekly for the first 12 weeks, followed by 50 mg once weekly. Studies demonstrate that nearly 50% of patients achieve PASI 75 during the initial treatment interval, which represents a desirable clinical improvement.
- **Adalimumab**, conversely, is a fully human anti-TNF-alpha antibody that competitively inhibits binding to cell-surface receptors. Treatment with this agent is recognized by researchers as one of the most effective therapies for psoriatic arthritis. The authorized dosage for adults is 80 mg subcutaneously at week 0, followed by 40 mg subcutaneously every two weeks starting from week 1.
- **Infliximab** is a chimeric monoclonal antibody approved for the treatment of psoriasis, psoriatic arthritis, and rheumatoid arthritis in adults.

Interleukin-17 Inhibitors

- **Brodalumab** is a human monoclonal antibody that functions as an antagonist of the IL-17 receptor A (IL-17RA), thereby inhibiting the signaling of IL-17A, IL-17F, IL-17A/F, and IL-17E. Treatment is initiated with 210 mg administered subcutaneously at weeks 0, 1, and 2, followed by a maintenance dose of 210 mg subcutaneously every 2 weeks. Brodalumab is characterized by high efficacy and, importantly, rapid symptom reduction; Phase 3 clinical trials demonstrated that 44% of patients achieved PASI 100 by week 12.
- **Secukinumab** is another agent in this class, which selectively binds to and blocks IL-17A. The dosing regimen consists of 300 mg administered subcutaneously at weeks 0, 1, 2, 3, and 4, followed by a maintenance dose of 300 mg subcutaneously every 4 weeks.

Interleukin-23 Inhibitors

- **Tildrakizumab** is an FDA-approved agent for the treatment of severe plaque psoriasis at a dose of 100 mg at weeks 0 and 4, followed by 100 mg every 12 weeks. This drug is noted for a relatively long time-to-relapse, which offers a distinct benefit for patients. The median time to loss of 50% of the best response is approximately 7.4 months.
- **Risankizumab** is another inhibitor in this group, defined as a humanized IgG1 monoclonal antibody that binds specifically to the p19 subunit of interleukin-23. The initial dose is 150 mg administered at weeks 0 and 4, followed by dosing every 12 weeks. The drug is currently approved by the FDA solely for the management of moderate-to-severe plaque psoriasis in adults. (15)

Innovative Therapies

Despite the extensively developed market of pharmaceutical agents and treatment modalities, psoriasis continues to present a significant therapeutic challenge, particularly in patients refractory to standard regimens and therapies. Recently, a molecule known as interleukin-21 (IL-21) has garnered scientific attention. It is postulated as a potential novel therapeutic target in psoriatic patients due to its pivotal role in mediating inflammatory responses. Studies have demonstrated upregulated expression of IL-21 and the IL-21 receptor in lesional biopsy specimens and peripheral blood obtained from patients with psoriasis. While this presents a promising avenue for novel treatment modalities, the complexity of IL-21 activity and its involvement in diverse pathomechanisms may pose significant research challenges.

Another aspect warranting investigation is the role of the microbiota in modulating the immune response in psoriasis. It has been demonstrated that in patients diagnosed with psoriasis, there is a marked enrichment of *Firmicutes* and *Actinobacteria*, accompanied by a depletion of *Bacteroidetes*. (16)

A primary objective for researchers is the identification and targeting of molecules involved in the early stages of psoriasis pathogenesis.[17,18] One such class of agents includes TYK2 inhibitors.[17,18] The inhibition of TYK2 disrupts the IL-23/IL-17 axis, effectively abrogating the inflammatory signals critical for the pathogenesis of psoriasis (PsO) and psoriatic arthritis (PsA), thereby yielding a significant therapeutic response.[18-20] Deucravacitinib represents the inaugural approved agent in the class of oral, selective allosteric TYK2 inhibitors.[17,18,20] The US FDA has authorized its use for adult patients with moderate-to-severe plaque psoriasis who are candidates for systemic therapy or phototherapy.[17,18,20]

Results

The systematic review and analysis confirm the central role of the IL-23/IL-17 axis as the primary pathogenic driver of psoriasis. While established treatments—including topical agents (corticosteroids, vitamin D analogs), conventional systemic therapies (methotrexate, cyclosporine), and phototherapy (NB-UVB, PUVA)—remain critical for foundational management, the field is rapidly progressing toward more targeted solutions.

Significant advancements are demonstrated by the effectiveness of biological agents, which offer increasingly successful therapeutic options for patients with moderate-to-severe disease. These biologics, encompassing TNF-alpha, IL-17, and IL-23/IL-12 inhibitors, have achieved high clinical success rates and improved long-term control by selectively interrupting key inflammatory pathways.

Crucially, the continued therapeutic challenge, particularly for patients who are refractory or experience sub-optimal responses, has opened up a rich and productive area for scientific inquiry. This is exemplified by the emergence of the first oral, selective allosteric **TYK2 inhibitor (deucravacitinib)**, which provides an effective new class of therapy by directly disrupting the core inflammatory cascade. Further research on novel targets like **IL-21** and the role of the **microbiota** in modulating immune response highlights the vast potential and active progress in the field. The overall picture is one of a continually improving landscape for psoriasis patients, offering great optimism for future personalized treatment strategies.

Disclosure

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All authors have read and agreed with the published version of the manuscript.

Funding Statement

The article did not receive any funding.

Institutional Review Board Statement

Not Applicable.

Informed Consent Statement

Not Applicable

Conflict Of Interest

Authors declare no conflict of interest.

ChatGPT was used during manuscript preparation to support language refinement and formatting. The authors critically reviewed and edited the text and assumed full responsibility for the content of the article.

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