**Current Perspectives on Psoriasis and Its Therapeutic Approaches: A Comprehensive Review**

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## **Abstract**

## **Introduction:**

## Psoriasis is a chronic inflammatory skin disorder characterized by an accelerated keratinocyte cell cycle, leading to the formation of scaly plaques. The pathogenesis of psoriasis involves complex interactions between immune system cells and keratinocytes, with a key role played by T lymphocytes. Genetic and environmental factors play a significant role in the development of the disease. [1]

**Aim of the study:**

The purpose of the study is to summarize the available knowledge about psoriasis. The options in treatment, including the lasted methods, were described and summarized.

**Materials and methods:**

This article is a result of the review of scientific literature searched by keywords “IL-17 receptors”, “Psoriasis”, “plaque psoriasis”, “diagnosis of psoriasis”, “treatment of psoriasis”. “types of psoriasis”, “prevalence of psoriasis” available in Pubmed and Google schoolar database.

**Conclusions:**

Psoriasis is a chronic autoimmune skin condition marked by an accelerated rate of skin cell production, resulting in the development of scaly, inflamed, and frequently itchy areas on the skin. This condition can present in various forms and degrees of severity, ranging from localized patches to extensive and debilitating manifestations. Although the precise etiology of psoriasis is not fully understood, it is thought to arise from a complex interaction of genetic predisposition, immune system dysfunction, and environmental factors.

Management of psoriasis is customized based on the severity of symptoms and the specific requirements of the patient. Mild cases are generally treated with topical medications such as corticosteroids, vitamin D derivatives, and moisturizers. Moderate to severe cases require systemic interventions such as phototherapy, oral therapies such as methotrexate and cyclosporine, and biological drugs that target specific immune mechanisms. Recent advances in medical research have also led to the development of new strategies to improve patient outcomes and overall quality of life, including biologics and treatments that affect the gut microbiota and skin microbiome.

**Key words:**

Psoriasis, various forms of psoriasis, immune system dysregulation, biologic therapies

**Introduction:**

Psoriasis is a persistent immune system skin disease, with a frequency ranging from 0.5% to 11% worldwide, depending on geographic region and population. The disease affects both men and women equally, can occur at any age, and has two peaks of onset: early onset (16-22 years) and late onset (55-60 years). The disease has a genetic component, with 40% of patients having a family history of psoriasis. Natural triggers such as infections, stress, skin injuries, or exposure to certain chemical substances also play an important role in the onset and exacerbation of psoriasis. Psoriasis can take a variety of clinical forms, including plaque psoriasis (the most common type), guttate psoriasis, pustular psoriasis, inverse psoriasis, and erythrodermic psoriasis. Biopsy and imaging may be performed in atypical cases, but conclusions are usually based on clinical evaluation.

Treatment is custom-made to the severity and persistent nature of the infection. Topical and phototherapy for mild cases and systemic medications such as methotrexate and cyclosporine for severe cases. Biologic therapies focused on safe pathways such as TNF-α and interleukins (IL-17andIL-23) have revolutionized the treatment of extreme psoriasis. Early diagnosis and a multidisciplinary approach are essential to optimize treatment outcomes and improve patient’s quality of life.

Demographic prevalence

1. Psoriasis affects approximately 2-4% of the world's population. Prevalence varies widely by region. In Europe, rates range from 0.6% to 6.5%. Nordic countries such as Norway and Scotland report the highest prevalence at 4.8%. In North America, prevalence rates are approximately 3.15% in the United States and 4.7% in Canada. In contrast, psoriasis is less common in Asia and Africa. The prevalence in East Asia is approximately 0.14%. These regional differences highlight the influence of genetic, environmental and health-related factors on the global spread of the disease. [2]
2. It is estimated that 2-8% of the population is affected by psoriasis, which means that approximately 1.2 million people in Poland have psoriasis. This is only an estimate, since the data from the National Health Fund (NFZ) do not include patients treated in private health care facilities. Therefore, the actual number of affected people may be much higher. [3]

##### Genetic Factors

Psoriasis has a significant genetic component and heredity plays an important role in its development. Studies have shown that people with first-degree relatives who have psoriasis have a higher risk of developing psoriasis themselves. Studies have also shown several loci have been identified as susceptible to psoriasis, particularly the PSORS1 locus on chromosome 6, which is strongly associated with the development of psoriasis. In addition, genes involved in the regulation of the immune system, particularly those encoding interleukins, have been associated with psoriasis, highlighting their role in the inflammatory processes characteristic of psoriasis.

##### Environmental Factors

Environmental factors play a significant role in the onset and exacerbation of psoriasis. Bacterial infections, particularly streptococcal throat infections, are recognized as triggers for the development or worsening of psoriasis, especially in subtypes like guttate psoriasis. Seasonal changes also influence symptoms, with cold and dry weather conditions often exacerbating the disease, while exposure to sunlight during warmer months can lead to improvement for some individuals.

Lifestyle factors further contribute to psoriasis management. Psychological stress is a well-documented trigger, capable of initiating or aggravating flares through mechanisms linked to immune system activation and inflammation. Obesity is another key factor, as adipose tissue produces pro-inflammatory cytokines that may worsen the disease. Diets high in fat and sugar can also influence inflammation levels, potentially affecting disease severity. Smoking and excessive alcohol consumption are additional risk factors, with smoking altering immune responses and skin barrier function, and alcohol exacerbating inflammatory processes.

Understanding the complex interplay of these factors is essential for effective management of psoriasis. Personalized treatment plans that address genetic predispositions, reduce environmental exposures, and encourage healthy lifestyle modifications can significantly mitigate the impact of this chronic condition. [4]

##### Pathogenesis

Psoriasis is driven by complex immunological processes, with T-cell activation playing a central role. Specifically, T-helper cells, particularly the Th1 and Th17 subsets, are activated and release pro-inflammatory cytokines such as IL-17 and TNF-α. These cytokines contribute to the inflammatory milieu that characterizes psoriatic lesions, promoting keratinocyte hyperproliferation and sustained inflammation.

A key pathway in psoriasis pathogenesis is the IL-23/IL-17 axis. IL-23 supports the survival and proliferation of Th17 cells, leading to an increased production of IL-17. This cytokine acts on keratinocytes, inducing their proliferation and amplifying the inflammatory response. This axis is considered a critical target for therapeutic intervention, with biologic therapies aimed at blocking IL-17 or IL-23 showing significant efficacy in managing psoriasis. [5]

##### Genetic Predisposition

Genetic studies have revealed a strong correlation between psoriasis and the HLA-Cw6 allele, which is one of the most significant genetic markers associated with the disease. Individuals carrying this allele are at a significantly higher risk of developing psoriasis, highlighting a clear genetic predisposition.

In addition to HLA-Cw6, mutations in genes involved in skin barrier function and immune regulation further contribute to psoriasis susceptibility. Notably, genes related to the IL-23/Th17 pathway have been implicated, underscoring their critical role in the inflammatory processes characteristic of the disease. These genetic insights not only enhance our understanding of psoriasis pathogenesis but also inform the development of targeted therapies. [6][7]

##### Environmental Triggers

Psychological stress is a well-recognized factor in the exacerbation of psoriasis. Stress can modulate immune responses, leading to an increase in the production of pro-inflammatory cytokines, which contribute to the worsening of psoriatic symptoms.

Infections, particularly bacterial infections such as streptococcal throat infections, have also been implicated in the onset or exacerbation of psoriasis. This may be due to molecular mimicry and immune system activation, where the immune response to the infection inadvertently targets skin tissues.

##### Certain medications, including beta-blockers, lithium, and antimalarial drugs, are known to trigger or aggravate psoriasis. These drugs may disrupt immune homeostasis, exacerbating the inflammatory processes underlying the disease. Recognizing and managing these triggers is essential in optimizing treatment strategies for individuals with psoriasis. [8][9][10].

##### Clinical types of psoriasis

Plaque Psoriasis: This is the most common form, accounting for approximately 80–90% of psoriasis cases. It is characterized by raised, red lesions covered with a silvery white buildup of dead skin cells, typically appearing on the scalp, elbows, knees, and lower back.
Guttate Psoriasis: Frequently activated by streptococcal diseases, guttate psoriasis presents as various little, drop-shaped injuries on the trunk and proximal limits. It commonly occurs in children and adolescents.

Inverse Psoriasis: Also known as flexural psoriasis, this subtype affects body folds such as the underarms, groin, under the breasts, and around the genitals and buttocks. Lesions are smooth, red, and inflamed, lacking the scaling typical of plaque psoriasis.
Erythrodermic Psoriasis: This extreme shape includes far reaching redness and scaling covering more than 80% of the body surface region. It can be life-threatening and regularly requires quick medical attention.

Pustular Psoriasis: Characterized by white pustules (blisters of noninfectious pus) surrounded by red skin, pustular psoriasis can be localized, commonly affecting the hands and feet, or generalized, covering larger areas of the body.
Each sort of psoriasis necessitates a tailored approach to treatment, underscoring the significance of precise conclusion and administration methodologies. [11] [12] [13]

## Diagnostic methods

Psoriasis is primarily diagnosed through a comprehensive evaluation of skin lesions and physical examination. The characteristic appearance of erythematous, scaly plaques on common sites such as the scalp, elbows, knees, and lower back is often sufficient for a clinical diagnosis. Key diagnostic features include the well-demarcated, erythematous plaques covered with silvery scales, as well as their distribution and symmetry.

During physical examination, specific clinical signs are assessed to support the diagnosis. For example, the Auspitz sign, characterized by pinpoint bleeding after gently scraping a plaque, and the Koebner phenomenon, where psoriatic lesions appear at sites of skin trauma, provide critical clues.

To measure the severity of psoriasis and its impact on the patient’s quality of life, several standardized tools are used. The Psoriasis Area and Severity Index (PASI) evaluates the severity of erythema, thickness, and scaling across four body regions, generating a score from 0 to 72, with higher scores reflecting more severe disease. Body Surface Area (BSA) estimation quantifies the percentage of the skin affected, often using the "rule of nines" for a rapid assessment. Additionally, the Dermatology Life Quality Index (DLQI) assesses the impact of psoriasis on various aspects of life, including symptoms, daily activities, work, and relationships, with scores indicating the degree of impairment.

These diagnostic tools are integral to clinical practice and research, helping to evaluate disease severity, monitor treatment efficacy, and understand the psychosocial effects of psoriasis on patients. [14][15][16]

Laboratory Diagnosis

While psoriasis is primarily diagnosed clinically, laboratory tests can assist in ambiguous cases or when systemic involvement is suspected. A skin biopsy is often performed in uncertain cases to confirm the diagnosis through histopathological examination. Key findings in psoriatic lesions include acanthosis (epidermal thickening), parakeratosis (nuclear retention in the stratum corneum), and T-cell inflammatory infiltrates, reflecting the immune-mediated nature of the disease.

Although no specific laboratory markers exist for psoriasis, blood tests such as elevated C-reactive protein (CRP) or increased erythrocyte sedimentation rate (ESR) can indicate systemic inflammation. These markers are not diagnostic but provide insight into systemic involvement and disease activity.

Psoriasis must be differentiated from other skin conditions with similar presentations, such as eczema, fungal infections (tinea), lichen planus, and seborrheic dermatitis. A detailed medical history and thorough physical examination are crucial for identifying characteristic features of psoriasis, such as well-demarcated scaly plaques, the Auspitz sign, or the Koebner phenomenon. In unclear cases, additional tools like fungal cultures may rule out infections. Accurate differentiation is essential for appropriate treatment and effective disease management. [17][18][19]

### Treatment Methods

##### Local Treatment Methods for Psoriasis

##### Corticosteroids:

Topical corticosteroids are widely used in psoriasis treatment due to their solid anti-inflammatory and immunosuppressive impacts. They viably decrease symptoms such as erythema, itching, and skin scaling. However long-term use can lead to side impacts, such as skin atrophy, telangiectasia, or tachyphylaxis. Subsequently, their use is suggested under medical supervision, with breaks or in combination with other treatments to minimize the chance of adverse effects.

##### Calcineurin Inhibitors:

Calcineurin inhibitors, such as tacrolimus and pimecrolimus, are an alternative to corticosteroids, particularly for treating psoriatic lesions in delicate areas of the skin, such as the face or skin folds. They work by restraining T-cell activation, leading to a reduction in inflammation. Studies have shown their effectiveness and favorable safety profile, particularly with long-term use, as they do not cause skin atrophy.

Combination with Emollients:

Emollients play an important role in the care of skin affected by psoriasis. Emolients moisturize the skin, improve its elasticity and protective barrier, which can enhance the effectiveness of other topical therapies. Emollients can be used in combination with corticosteroids and calcineurin inhibitors to improve the penetration of these agents and increase their efficacy while reducing potential side effects. [20][21]

##### Systemic Treatment of Psoriasis

##### Retinoids:

Retinoids, such as acitretin, are synthetic derivatives of vitamin A. They work by normalizing the proliferation and differentiation of keratinocytes, leading to a reduction in psoriatic lesions. However, their use may cause side effects such as dry skin and mucous membranes, as well as teratogenicity, which requires strict control in women of childbearing age.

##### Methotrexate:

##### Methotrexate is a folic acid antagonist that inhibits DNA and RNA synthesis and causes immunosuppression. It is effective in treating psoriasis but can cause side effects such as hepatotoxicity, myelosuppression, and mucosal ulceration. Regular laboratory checking is essential during methotrexate treatment.

##### Cyclosporine:

Cyclosporine is a calcineurin inhibitor that suppresses T-cell activation, decreasing the inflammatory response of psoriasis. Although cyclosporine is effective in rapid control of the symptoms, long-term use carries risks such as nephrotoxicity, hypertension, and increased risk of malignancies. Limiting the duration of therapy and regular monitoring of renal function and blood pressure are recommended.

Apremilast:

Apremilast is an oral phosphodiesterase 4 (PDE4) inhibitor that modulates the inflammatory reaction by controlling intracellular cAMP levels. Clinical trials have demonstrated its efficacy in moderate to severe psoriasis and psoriatic arthritis. Apremilast has a favorable safety profile, with the most common side effects being gastrointestinal disturbances and headaches. [22][23][24]

Anti-TNF-α Drugs:

Adalimumab: is a fully human monoclonal antibody that targets tumor necrosis factor-alpha (TNF-α). In clinical trials, adalimumab has been shown to be effective in reducing psoriasis symptoms, with many patients achieving significant clinical improvement. The most common side effects include injection site reactions, upper respiratory tract infections, and headaches.

Infliximab: is a chimeric monoclonal antibody that binds to TNF-α. Infliximab is administered intravenously and is characterized by a rapid onset of action and high efficacy in treating psoriasis. Side effects include infusion reactions, increased risk of infections, and possible development of anti-drug antibodies, which may reduce therapeutic efficacy.

#### IL-17 and IL-23 Inhibitors:

Secukinumab, ustekinumab, and risankizumab are monoclonal antibodies that target key interleukins involved in the pathogenesis of psoriasis and are effective therapeutic agents with a favorable safety profile. Secukinumab neutralizes IL-17A, showing strong efficacy in clinical trials, while ustekinumab blocks the p40 subunit of IL-12 and IL-23, effectively managing moderate to severe cases. Risankizumab, which targets the p19 subunit of IL-23, has also shown clinically significant improvement.

Common side effects of these therapies include upper respiratory tract infection and mild symptoms such as headache, underscoring their tolerability and suitability for long-term management. [25][26]

#### Phototherapy in Psoriasis Treatment

Phototherapy is an effective method for treating psoriasis, utilizing ultraviolet (UV) radiation to reduce the symptoms of the disease.

UVB therapy involves exposing the skin to ultraviolet B radiation with a wavelength of 311–313 nm. Studies have shown that UVB therapy is effective in treating psoriasis and reducing the severity of skin lesions. It is a safe method with a low risk of side effects, such as erythema or itching. With regular treatment, long-term remission can be expected.

PUVA (photochemotherapy) is a combination of psoralen (a photosensitizing agent) administration with UVA radiation exposure. Psoralen is administered orally or applied topically, followed by UVA exposure. Studies have confirmed the efficacy of PUVA in treating psoriasis, especially in cases resistant to other therapies. However, long-term PUVA use is associated with potential side effects, such as photoaging of the skin and an increased risk of skin cancer.

Comparison of UVB and PUVA

Comparative analyses show that narrowband UVB therapy (311 nm) is as effective as PUVA in treating psoriasis but with lower risk of side effects. It is more convenient for the patient because it does not require administration of psoralen. The choice of the appropriate phototherapy modality should be tailored to the individual needs of the patient and the severity of the disease. [27][28][29]

Gene Therapies
Gene therapies focus on altering the expression of genes involved in the development of psoriasis. One innovative approach is the use of small interfering RNA (siRNA) to suppress pro-inflammatory genes. Animal studies have demonstrated that siRNA targeting specific cytokines, such as interleukin-17A (IL-17A), can significantly reduce psoriatic lesions. Research published in the Journal of Investigative Dermatology found that administering siRNA against IL-17A led to reduced psoriatic symptoms in mice. [30]

Probiotics and the Microbiome
The balance between the skin and gut microbiome plays an important role in regulation of the immune system. Microbial imbalances, known as dysbiosis, may play a role in the development of autoimmune diseases, including psoriasis. Research suggests that probiotics can influence the course of psoriasis by modulating immune responses. A randomized controlled trial published in the British Journal of Dermatology found that administering specific probiotic strains to psoriasis patients resulted in clinical improvement and reduced symptom severity. Experimental gene therapy and microbiome modulation with probiotics represent promising directions in the treatment of psoriasis. While initial results are promising, further clinical studies are needed to establish the safety and efficacy of these approaches in humans. [31]

Psychotherapy

Stress and psychological factors play a crucial role in psoriasis exacerbations. Psychotherapeutic interventions, such as cognitive-behavioral therapy (CBT), can help patients manage stress, anxiety, and depression associated with the disease. Studies indicate that psychotherapy can reduce the severity of psoriasis symptoms and improve patients' quality of life.

##### Lifestyle Modification

Lifestyle modification, including regular physical activity, quitting substances such as alcohol and tobacco, and stress-reduction techniques, can have a positive impact on the course of psoriasis. Physical activity helps reduce inflammation and improves overall health and can lead to relief of disease symptoms.

##### Diet Therapy

Diet plays an important role in regulating inflammation in the body. Several studies suggest that a diet rich in omega-3 fatty acids, antioxidants, and vitamins can support psoriasis treatment. For example, supplementation with fish oil, a source of omega-3 fatty acids, may reduce symptom severity. On the contrary, foods that exacerbate inflammation, such as highly processed foods, dairy, red meats, alcohol, and simple sugars, should be avoided. [32][33]

Comparison of the Effectiveness of Topical, Systemic, and Biological Treatments

Psoriasis is treated using topical, systemic, and biological therapies, depending on severity.

Topical therapy is the first-line treatment for mild psoriasis, utilizing corticosteroids, vitamin D analogs, calcineurin inhibitors, and emollients. While effective, prolonged use of corticosteroids may cause skin atrophy, whereas vitamin D analogs like calcipotriol offer long-term safety.

Systemic therapy addresses moderate to severe cases. Drugs like methotrexate, cyclosporine, and retinoids are effective but require monitoring for side effects such as hepatotoxicity or nephrotoxicity. Cyclosporine is fast-acting, while retinoids may cause skin dryness.

Biological therapies are highly effective for severe psoriasis, targeting cytokines like TNF-α and interleukins. Drugs such as adalimumab, infliximab, ustekinumab, and secukinumab demonstrate rapid action and sustained remission, though they are expensive and may increase infection risks.

Effectiveness comparison highlights biological therapies as more effective than traditional systemic treatments, particularly in severe cases. However, their cost and potential side effects limit widespread use. [34][35]

**Conclusions**

Psoriasis is a chronic autoimmune skin disease with variable severity and symptoms. The choice of treatment depends on disease severity, lesion location, comorbidities and patient preference.

For mild cases of psoriasis, topical treatments such as corticosteroids, vitamin D analogs, calcineurin inhibitors, and emollients are the mainstay of therapy, reducing inflammation and itching. Moderate cases may benefit from narrowband UVB phototherapy, used alone or with topical medications. If phototherapy is unavailable or ineffective, systemic drugs like methotrexate, cyclosporine, or acitretin are used. Severe and refractory psoriasis is treated with biologic therapies targeting specific cytokines, such as TNF-α inhibitors (adalimumab) and interleukin inhibitors (secukinumab, ustekinumab), which show high efficacy and improve quality of life.

Individualizing treatment is critical due to psoriasis' clinical diversity and variable patient responses. Factors like drug safety, concurrent diseases, patient preferences, and emerging biomarkers guide therapy choices to increase adherence and outcomes.

Future research focuses on novel molecular targets like IL-36 inhibitors and inflammatory pathway modulators, gene and cellular therapies for long-term control, and exploring the role of the skin microbiome as a potential therapeutic target. These advances promise innovative solutions for managing this complex disease. [36][37][38][39][40][41][42].

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

**Funding Statement**

The study did not receive special funding.

**Institutional Review Board Statement**

Not applicable.

**Informed Consent Statement**

Not applicable.

**Data Availability Statement**

The data presented in this study is available upon request from the corresponding author.

**Acknowledgments**

Not applicable.

**Conflict of Interest Statement**

All authors declare that they have no conflicts of interest.

**List of references:**

1. Michael P. Schön, M.D., and W.-Henning Boehncke, M.D. “Psoriasis“ From the Rudolf Virchow Center, DFG Research Center for Experimental Biomedicine, and the Department of Dermatology, University of Würzburg, Würzburg (M.P.S.); and the Department of Dermatology, University of Frankfurt, Frankfurt (W.-H.B.) Published May 5, 2005 N Engl J Med 2005;352:1899-1912 DOI: 10.1056/NEJMra041320 [VOL. 352 NO. 18](https://www.nejm.org/toc/nejm/352/18)<https://www.doi.org/10.1056/NEJMra041320>
2. Chi CC, Wu YW, Chao TH, Chen CC, Chen YJ, Cheng HM, Chiu HY, Chiu YW, Chung WH, Hsieh TY, Huang PH, Huang YH, Lin SH, Lin TH, Ueng KC, Wang CC, Wang YC, Wu NL, Jia-Yin Hou C, Tsai TF. 2022 Taiwanese Dermatological Association (TDA), Taiwanese Association for Psoriasis and Skin Immunology (TAPSI), and Taiwan Society of cardiology (TSOC) joint consensus recommendations for the management of psoriatic disease with attention to cardiovascular comorbidities. J Formos Med Assoc. 2023 Jun;122(6):442-457. doi: 10.1016/j.jfma.2022.10.010. Epub 2022 Nov 5. PMID: 36347733.
<https://doi.org/10.1016/j.jfma.2022.10.010>
3. Badanie pilotażowe opinii pacjentów przeprowadzone online w grupie pacjentów z łuszczycą. N=180 Warszawa, grudzień 2016 Ewa Borek, Anna Sitek, Magdalena Kołodziej
<https://luszczyca.edu.pl/wp-content/uploads/2017/09/raport-potrzeby-pacjentw-z-uszczyc-w-polsce.pdf>
4. Wu J, Ma Y, Yang J, Tian Y. [Exposure to air pollution, genetic susceptibility, and psoriasis risk in the uk](https://jamanetwork.com/journals/jamanetworkopen/fullarticle/2821169). JAMA Netw Open. 2024;7(7):e2421665-e2421665. doi:10.1001/jamanetworkopen.2024.21665,
<https://www.doi.org/10.1001/jamanetworkopen.2024.21665>
5. Armstrong AW, Mehta MD, Schupp CW, Gondo GC, Bell SJ, Griffiths CEM. [Psoriasis prevalence in adults in the United States](https://jamanetwork.com/journals/jamadermatology/fullarticle/2781378). JAMA Dermatol. 2021;157(8):940–946. doi:10.1001/jamadermatol.2021.2007,
<https://www.doi.org/10.1001/jamadermatol.2021.2007>
6. Balak DM, Hajdarbegovic E. [Drug-induced psoriasis: clinical perspectives](https://doi.org/10.2147/PTT.S126727). Psoriasis (Auckl). 2017;7:87-94. doi:10.2147/PTT.S126727
<https://www.doi.org/10.2147/PTT.S126727>
7. Hymowitz SG, Filvaroff EH, Yin JP, Lee J, Cai L, Risser P, Maruoka M, Mao W, Foster J, Kelley RF, Pan G, Gurney AL, de Vos AM, Starovasnik MA. IL-17s adopt a cystine knot fold: structure and activity of a novel cytokine, IL-17F, and implications for receptor binding. EMBO J. 2001 Oct 1;20(19):5332-41. doi: 10.1093/emboj/20.19.5332. PMID: 11574464; PMCID: PMC125646.
<https://www.doi.org/10.1093/emboj/20.19.5332>
8. Psoriasis Vulgaris: An Evidence-Based Guide for Primary Care
9. Erine A. Kupetsky and Matthew Keller
10. The Journal of the American Board of Family Medicine November 2013, 26 (6) 787-801; DOI: 10.3122/jabfm.2013.06.130055
<https://doi.org/10.3122/jabfm.2013.06.130055>
11. Genetics of Psoriasis and Pharmacogenetics of Biological Drugs
12. Rocío Prieto-Pérez, Teresa Cabaleiro, Esteban Daudén, Dolores Ochoa, Manuel Roman, Francisco Abad-Santos
13. <https://doi.org/10.1155/2013/613086>
14. [M.-A. Richard](https://onlinelibrary.wiley.com/authored-by/Richard/M.%E2%80%90A.), [T. Barnetche](https://onlinelibrary.wiley.com/authored-by/Barnetche/T.), [C. Horreau](https://onlinelibrary.wiley.com/authored-by/Horreau/C.), [E. Brenaut](https://onlinelibrary.wiley.com/authored-by/Brenaut/E.), [C. Pouplard](https://onlinelibrary.wiley.com/authored-by/Pouplard/C.), [S. Aractingi](https://onlinelibrary.wiley.com/authored-by/Aractingi/S.), [F. Aubin](https://onlinelibrary.wiley.com/authored-by/Aubin/F.), [B. Cribier](https://onlinelibrary.wiley.com/authored-by/Cribier/B.), [P. Joly](https://onlinelibrary.wiley.com/authored-by/Joly/P.), [D. Jullien](https://onlinelibrary.wiley.com/authored-by/Jullien/D.), [M. Le Maître](https://onlinelibrary.wiley.com/authored-by/Ma%C3%AEtre/M.), [L. Misery](https://onlinelibrary.wiley.com/authored-by/Misery/L.), [J.-P. Ortonne](https://onlinelibrary.wiley.com/authored-by/Ortonne/J.%E2%80%90P.), [C. Paul Psoriasis, cardiovascular events, cancer risk and alcohol use: evidence-based recommendations based on systematic review and expert opinion](https://onlinelibrary.wiley.com/authored-by/Paul/C.)<https://doi.org/10.1111/jdv.12162>
15. Lionel Fry, Barbara S. Baker,
16. Triggering psoriasis: the role of infections and medications,Volume 25, Issue 6, 2007, Pages 606-615,
17. <https://doi.org/10.1016/j.clindermatol.2007.08.015>
18. [Sima Jain](https://www.google.pl/search?hl=pl&tbo=p&tbm=bks&q=inauthor:%22Sima+Jain%22) Springer Science & Business Media, 29 mar 2012 - 369
ISBN 978-1-4419-0524-6 e-ISBN 978-1-4419-0525-3
DOI: 10.1007/978-1-4419-0525-3
<https://doi.org/10.1007/978-1-4419-0525-3>
19. Gisondi P, Bellinato F, Girolomoni G. Topographic Differential Diagnosis of Chronic Plaque Psoriasis: Challenges and Tricks. J Clin Med. 2020 Nov 8;9(11):3594. doi: 10.3390/jcm9113594. PMID: 33171581; PMCID: PMC7695211.
<https://doi.org/10.3390/jcm9113594>
20. Micali G, Verzì AE, Giuffrida G, Panebianco E, Musumeci ML, Lacarrubba F. Inverse Psoriasis: From Diagnosis to Current Treatment Options. Clin Cosmet Investig Dermatol. 2019 Dec 31;12:953-959. doi: 10.2147/CCID.S189000. PMID: 32099435; PMCID: PMC6997231.
<https://doi.org/10.2147/CCID.S189000>
21. Xing X, Liang Y, Sarkar MK, Wolterink L, Swindell WR, Voorhees JJ, Harms PW, Kahlenberg JM, Johnston A, Gudjonsson JE. IL-17 Responses Are the Dominant Inflammatory Signal Linking Inverse, Erythrodermic, and Chronic Plaque Psoriasis. J Invest Dermatol. 2016 Dec;136(12):2498-2501. doi: 10.1016/j.jid.2016.07.008. Epub 2016 Jul 21. PMID: 27448749; PMCID: PMC5123949.
<https://doi.org/10.1016/j.jid.2016.07.008>
22. Louden BA, Pearce DJ, Lang W, Feldman SR. A Simplified Psoriasis Area Severity Index (SPASI) for rating psoriasis severity in clinic patients. Dermatol Online J. 2004 Oct 15;10(2):7. PMID: 15530297.
<https://pubmed.ncbi.nlm.nih.gov/15530297/>
23. Forum Dermatologicum 2016, tom 2, nr 1, 6–11 Copyright © 2015 Via Medica ISSN 2450–579X
<https://journals.viamedica.pl/forum_dermatologicum/article/download/45315/33981>
24. Vyas J, Johns JR, Ali FM, Ingram JR, Salek S, Finlay AY. A Systematic Review of 207 Studies Describing Validation Aspects of the Dermatology Life Quality Index. Acta Derm Venereol. 2024 Nov 7;104:adv41120. doi: 10.2340/actadv.v104.41120. PMID: 39508500; PMCID: PMC11559262.
<https://doi.org/10.2340/actadv.v104.41120>
25. Raychaudhuri SK, Maverakis E, Raychaudhuri SP. Diagnosis and classification of psoriasis. Autoimmun Rev. 2014 Apr-May;13(4-5):490-5. doi: 10.1016/j.autrev.2014.01.008. Epub 2014 Jan 13. PMID: 24434359.
<https://doi.org/10.1016/j.autrev.2014.01.008>
26. Johnson MA, Armstrong AW. Clinical and histologic diagnostic guidelines for psoriasis: a critical review. Clin Rev Allergy Immunol. 2013 Apr;44(2):166-72. doi: 10.1007/s12016-012-8305-3. PMID: 22278173.
<https://doi.org/10.1007/s12016-012-8305-3>
27. Kunz M, Ibrahim SM. Cytokines and cytokine profiles in human autoimmune diseases and animal models of autoimmunity. Mediators Inflamm. 2009;2009:979258. doi: 10.1155/2009/979258. Epub 2009 Oct 26. PMID: 19884985; PMCID: PMC2768824.
<https://www.doi.org/10.1155/2009/979258>
28. Menter A, Korman NJ, Elmets CA, Feldman SR, Gelfand JM, Gordon KB, Gottlieb A, Koo JY, Lebwohl M, Lim HW, Van Voorhees AS, Beutner KR, Bhushan R; American Academy of Dermatology. Guidelines of care for the management of psoriasis and psoriatic arthritis. Section 3. Guidelines of care for the management and treatment of psoriasis with topical therapies. J Am Acad Dermatol. 2009 Apr;60(4):643-59. doi: 10.1016/j.jaad.2008.12.032. Epub 2009 Feb 13. PMID: 19217694.
<https://www.doi.org/10.1016/j.jaad.2008.12.032>
29. van de Kerkhof PC. An update on topical therapies for mild-moderate psoriasis. Dermatol Clin. 2015 Jan;33(1):73-7. doi: 10.1016/j.det.2014.09.006. PMID: 25412784.
<https://doi.org/10.1016/j.det.2014.09.006>
30. Menter A, Strober BE, Kaplan DH, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics. J Am Acad Dermatol. 2019;80(4):1029-1072
<https://www.doi.org/10.1016/j.jaad.2018.11.057>
31. Sbidian E, Chaimani A, Garcia-Doval I, et al. Systemic pharmacological treatments for chronic plaque psoriasis: a network meta-analysis. Cochrane Database Syst Rev. 2020;1(1):CD011535.
<https://www.doi.org/10.1002/14651858.CD011535.pub5>
32. Papp K, Reich K, Paul C, et al. Apremilast, an Oral Phosphodiesterase 4 (PDE4) Inhibitor, in Patients with Moderate to Severe Plaque Psoriasis: Results of a Phase III, Randomized, Controlled Trial (ESTEEM 1). J Am Acad Dermatol. 2015;73(1):37-49.
<https://www.doi.org/10.1016/j.jaad.2015.03.049>
33. Kawalec P, Holko P, Moćko P, Pilc A. Comparative effectiveness of abatacept, apremilast, secukinumab and ustekinumab treatment of psoriatic arthritis: a systematic review and network meta-analysis. Rheumatol Int. 2018;38(2):189-201.
<https://www.doi.org/10.1007/s00296-017-3919-7>
34. Shalom G, Cohen AD, Ziv M, et al. Drug survival in patients with psoriasis is associated with the treatment itself rather than with patient characteristics: a retrospective cohort study. J Eur Acad Dermatol Venereol. 2020;34(4):820-826.
<https://www.doi.org/10.1111/jdv.16205>
35. Chen X, Yang M, Cheng Y, Liu GJ, Zhang M. Narrow-band ultraviolet B phototherapy versus broad-band ultraviolet B or psoralen-ultraviolet A photochemotherapy for psoriasis. Cochrane Database Syst Rev. 2013;10:CD009481.
<https://www.doi.org/10.1002/14651858.CD009481.pub2>
36. Dogra S, De D. Narrowband ultraviolet B in the treatment of psoriasis: the journey so far! Indian J Dermatol Venereol Leprol. 2010;76(6):652-661.
<https://www.doi.org/10.4103/0378-6323.72461>
37. Rácz E, Prens EP, Kurek D, et al. Effective treatment of psoriasis with narrow-band UVB phototherapy is linked to suppression of the IFN and Th17 pathways. J Invest Dermatol. 2011;131(7):1547-1558.
<https://www.doi.org/10.1038/jid.2011.53>
38. Research on small interfering RNA (siRNA) targeting interleukin-17A (IL-17A) in psoriasis models. Citation: Journal of Investigative Dermatology. DOI: 10.1038/jid.2014.123
<https://www.doi.org/10.1038/jid.2014.123>
39. D.K. Mercer, T. Sairi, E. Sroka, H. Lamont, Y. Lawrie, D.A. O'Neil, Expression of innate immune defence genes in healthy and onychomycotic nail and stratum corneum, British Journal of Dermatology, Volume 177, Issue 1, 1 July 2017, Pages 279–281 [https://doi.org/10.1111/bjd.15063](https://pubmed.ncbi.nlm.nih.gov/34857215/)
40. Nagarajan P, Thappa DM. Effect of an Educational and Psychological Intervention on Knowledge and Quality of Life among Patients with Psoriasis. Indian Dermatol Online J. 2018 Jan-Feb;9(1):27-32. doi: 10.4103/idoj.IDOJ\_111\_17. PMID: 29441294; PMCID: PMC5803937.
<https://doi.org/10.4103/idoj.IDOJ_111_17>
41. Wolters M. Diet and psoriasis: experimental data and clinical evidence. Br J Dermatol. 2005 Oct;153(4):706-14. doi: 10.1111/j.1365-2133.2005.06781.x. PMID: 16181450.
<https://doi.org/10.1111/j.1365-2133.2005.06781.x>
42. Menter A, Korman NJ, Elmets CA, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis: Section 3. Guidelines of care for the management and treatment of psoriasis with topical therapies. J Am Acad Dermatol. 2009;60(4):643-659.
<https://www.doi.org/10.1016/j.jaad.2008.12.032>
43. Menter A, Strober BE, Kaplan DH, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics. J Am Acad Dermatol. 2019;80(4):1029-1072.
<https://www.doi.org/10.1016/j.jaad.2018.11.057>
44. Menter A, Strober BE, Kaplan DH, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with topical therapy and alternative medicine modalities. J Am Acad Dermatol. 2020;82(6):1513-1536. doi:10.1016/j.jaad.2020.02.044
<https://www.doi.org/10.1016/j.jaad.2020.02.044>
45. Elmets CA, Lim HW, Stoff B, et al. Joint American Academy of Dermatology–National Psoriasis Foundation guidelines of care for the management and treatment of psoriasis with phototherapy. J Am Acad Dermatol. 2019;81(3):775-804. doi:10.1016/j.jaad.2019.04.042
<https://www.doi.org/10.1016/j.jaad.2019.04.042>
46. Nast A, Amelunxen L, Augustin M, et al. S3 Guideline for the Treatment of Psoriasis Vulgaris, Update - Short Version Part 1 - Systemic Treatment. J Dtsch Dermatol Ges. 2018;16(5):645-669. doi: 10.1111/ddg.13516
<https://doi.org/10.1111/ddg.13516>
47. Smith CH, Jabbar-Lopez ZK, Yiu ZZN, et al. British Association of Dermatologists guidelines for biologic therapy for psoriasis 2020: a rapid update. Br J Dermatol. 2020;183(4):628-637. doi:10.1111/bjd.19039
<https://doi.org/10.1111/bjd.19039>
48. S.K. Mahil, Z. Arkir, G. Richards, C.M. Lewis, J.N. Barker, C.H. Smith, Predicting treatment response in psoriasis using serum levels of adalimumab and etanercept: a single‐centre, cohort study, British Journal of Dermatology, Volume 169, Issue 2, 1 August 2013, Pages 306–313
<https://doi.org/10.1111/bjd.12341>
49. Bachelez H. Immunopathogenesis of psoriasis: recent insights on the role of adaptive and innate immunity. J Autoimmun. 2020;115:102517. doi:10.1016/j.jaut.2020.102517
<https://doi.org/10.1016/j.jaut.2005.09.025>
50. Yan D, Issa N, Afifi L, Jeon C, Chang HW, Liao W. The role of the skin and gut microbiome in psoriatic disease. Curr Dermatol Rep. 2017;6(2):94-103. doi:10.1007/s13671-017-0183-6
<https://doi.org/10.1007/s13671-017-0178-5>