QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

PERZYNSKA, Jagienka, PODKOŚCIELNA, Jaśmina, KASTERKA, Natalia, BAGIŃSKA, Weronika, RYSZKOWSKA, Kamila, PAWELEC, Natalia, PURSKA, Aleksandra and KRZYŻOWSKA, Kinga. Innovations in the Treatment of Atopic Dermatitis: Integration of Targeted Therapies and Comprehensive Care. Quality in Sport. 2026;56:72384. https://doi.org/10.12775/QS.2026.56.72384

ARTICLE TIMELINE
Received: 22.05.2026 Revised: 26.05.2026
Accepted: 26.05.2026 Published: 30.05.2026

INDEXING & EVALUATION
MEiN points: 20 Unique ID: 201398
Disciplines: Economics & Finance; Management & Quality Sciences

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Economics and Finance (Social Sciences); Management and Quality Sciences (Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Innovations in the Treatment of Atopic Dermatitis: Integration of Targeted Therapies and Comprehensive Care

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Abstract

Atopic dermatitis (AD) is a chronic inflammatory skin disease with a complex pathogenesis involving epidermal barrier dysfunction, immune dysregulation, microbiome imbalance, and environmental and psychosocial factors. Recent advances have led to the development of targeted therapies that have significantly improved disease management, particularly in moderate-to-severe AD. Biologic agents and Janus kinase (JAK) inhibitors effectively modulate key immune pathways, while topical calcineurin inhibitors remain important steroid-sparing options.

Emerging microbiome-based therapies and holistic approaches further expand therapeutic possibilities by addressing additional disease mechanisms and contributing factors. Despite these advances, challenges such as cost, accessibility, and the need for long-term safety data persist, highlighting the importance of personalized and integrated treatment strategies.

Introduction

Atopic Dermatitis (AD), also referred to as atopic eczema, is a phenotypically heterogeneous chronic inflammatory skin disease. It typically arises due to environmental triggers in individuals who are genetically predisposed to the disease.[1] This disease is characterized by relapsing inflammation of the skin accompanied by intense pruritus, which is the most prominent and burdensome symptom. Clinically, it presents with eczematous lesions, xerosis, and lichenification resulting from repeated scratching, often following an age-dependent distribution pattern. It occurs mainly in the pediatric population, with a frequency of up to ~20% in this group of patients. The disease develops in 50–60% of cases within the first year of life, and 90% of patients before the age of five. Adults also suffer from AD, mostly from childhood, and there are also new adulthood cases.[2]

The pathophysiology of the disease is complex and involves interactions between genetic, immunological, and environmental factors. A key feature is epidermal barrier dysfunction, often associated with mutations in the filaggrin gene, leading to increased transepidermal water loss. The impaired skin barrier allows irritants and allergens to penetrate the skin and cause inflammation via an overactive Th2 response in acute lesions and Th1 response in chronic lesions. Scratching of the skin also stimulates keratinocytes to release inflammatory cytokines such as TNF-alpha, IL-1, and IL-6.

Decreased anti-microbial peptides (human beta-defensins, cathelicidins) in the epidermis of atopic patients also contribute to *Staphylococcus aureus* colonization seen in more than 90% of atopic dermatitis patients.[3]

Recent advances in understanding the pathophysiology of AD have led to the development of targeted therapies. AD treatment aims to address three primary areas: restoring the skin barrier, reducing inflammation, and controlling bacterial infections.

Additionally, maintaining adequate skin hydration plays a crucial role in managing AD, as it helps alleviate dryness, itchiness, and promotes the repair of the damaged outer layer of skin (stratum corneum).[4] Historically, the management of AD relied on non-specific anti-inflammatory approaches, including topical corticosteroids, emollients, and systemic immunosuppressants such as cyclosporine. Topical drugs with moisturizing effects should be used as a basic treatment to improve dry skin in patients with elderly AD. Topical moisturizers, e.g., urea or heparinoids, are first-choice drugs.[5] Although these treatments remain important, they are often limited by variable efficacy, potential adverse effects, and lack of disease specificity. In recent years, the therapeutic landscape of AD has undergone a significant transformation with the introduction of targeted therapies, including biologic agents and small-molecule inhibitors. These treatments specifically modulate key immune pathways involved in disease pathogenesis, offering improved efficacy and safety profiles compared to traditional treatment options.

Aim

The aim of this narrative review is to provide a comprehensive overview of modern therapeutic strategies in the management of atopic dermatitis, with particular emphasis on recently developed targeted treatments. Special attention is given to biologic agents and small-molecule inhibitors that selectively modulate key immune pathways involved in the pathogenesis of the disease.

Furthermore, this review aims to discuss emerging therapeutic approaches and tohighlight the growing role of personalized medicine in optimizing treatment outcomes, improving disease control, and enhancing the quality of life of patients with atopic dermatitis.

Materials and methods

An extensive review of the literature was conducted using electronic databases (PubMed, Wiley Online Library) and textbooks. The collected information was analyzed, and the findings are presented below.

Summary

The findings of the review indicate that current management of atopic dermatitis (AD) is increasingly based on effective targeted therapies, such as biologic agents and Janus kinase (JAK) inhibitors, which significantly improve disease control. It has also been demonstrated that topical calcineurin inhibitors remain an important component of topical therapy, particularly in long-term management and in sensitive skin areas.

In addition, microbiome-targeted approaches and holistic strategies, taking into account environmental and psychosocial factors, are gaining increasing importance. At the same time, the need for further research into long-term safety, treatment accessibility, and personalization of AD therapy is emphasized.

Keywords: atopic dermatitis, dupilumab, tralokinumab, JAK inhibitors, calcineurin inhibitors, biologic treatment, holistic treatment

The state of knowledge

Biologic therapies

Biologic therapies represent a significant breakthrough in the treatment of moderate-to-severe atopic dermatitis, offering targeted action against key immunological mechanisms underlying disease pathogenesis. In general, biologic therapies target one extracellular receptor subunit or cytokine, leading to more precise targeting than immunosuppressants and oral JAKi, leading to exceptional long-term safety and no required laboratory monitoring.

Biologics are not metabolized by traditional cytochrome-based mechanisms or excreted renally, minimizing potential drug interactions.[6] The first biological medication, approved for treatment of adults and children over 12 years, diagnosed with moderate and serious type of AD is dupilumab. It is a monoclonal antibody blocking subunit alpha, a common one for the receptors of IL-14 and IL-13.[7] At the skin level, this results in an improvement of epidermal barrier function through normalization of keratinocyte differentiation, reduction of spongiosis, and partial restoration of physiological lipid synthesis in the stratum corneum. Dupilumab also increases the production of antimicrobial peptides, thereby enhancing the skin's innate immune defense and reducing colonization by *Staphylococcus aureus*.

Consequently, a clinical reduction in inflammatory activity, pruritus, and the frequency of disease flares is observed. In addition, improved skin barrier integrity leads to reduced hypersensitivity to environmental factors such as allergens and irritants. This translates into an overall improvement in patients' quality of life and better long-term disease control.

Another commonly used monoclonal antibody in the treatment of Atopic dermatitis is Tralokinumab, which selectively neutralizes interleukin 13 (IL-13), a key cytokine involved in the pathogenesis of cutaneous inflammatory processes. Tralokinumab is characterized by a more selective mechanism of action in the treatment of AD compared with other biologic therapies, as its activity is exclusively directed toward the neutralization of interleukin 13 (IL-13). Approved for the treatment of moderate-to-severe AD in adults, tralokinumab has demonstrated efficacy and safety both in clinical trials and real-life studies. Its dosing regimen involves an initial loading dose of 600 mg followed by maintenance dosing of 300 mg every two weeks.[8] Tralokinumab demonstrates the highest efficacy in the phenotype of Atopic dermatitis characterized by pronounced epidermal barrier dysfunction. This is attributable to the key role of IL-13 in impairing skin barrier integrity, including reduced lipid synthesis and abnormal keratinocyte differentiation.

Janus Kinase (JAK) Inhibitors

The most important representatives of this class include abrocitinib, upadacitinib, and baricitinib (administered orally), as well as ruxolitinib (administered topically). More recently, JAK inhibition has been hypothesized as a novel treatment for AD, given the importance of JAK-STAT signaling (particularly JAK1) for Th2 cytokines, including IL-4, IL-13, and IL-31. Experimental models have shown improved skin barrier function, suppression of pruritus, cutaneous nerve elongation, and impaired IL-4 and IL-13 dependent differentiation of Th2 cells in response to JAK inhibitors.[9] A key characteristic of this class of agents is their rapid onset of action—reduction in pruritus may be observed within a few days of treatment initiation, with clinically meaningful improvement often occurring within the first 1–2 weeks.

Cutaneous lesions improve more gradually over subsequent weeks, typically within 2–4 weeks. This rapid clinical response distinguishes JAK inhibitors from many other systemic therapies used in atopic dermatitis.

Clinical trials have demonstrated their high efficacy in the treatment of moderate-to-severe atopic dermatitis, with an acceptable safety profile. However, due to their mechanism of action, careful monitoring for adverse events is required.

The most common treatment-emergent adverse events (TEAEs) associated with abrocitinib were nausea, upper respiratory tract infection, nasopharyngitis, and headache.[10] Baricitinib is an oral inhibitor of JAK1 and JAK2 kinases used in the treatment of moderate-to-severe atopic dermatitis. By inhibiting the JAK-STAT signaling pathway, this agent reduces the activity of pro-inflammatory cytokines, leading to a decrease in cutaneous inflammation and pruritus. Clinical studies have shown that baricitinib effectively reduces the severity of AD symptoms, including pruritus and skin lesions, while reducing the numer of disease exacerbations.[11] Treating AD with baricitinib, a starting dose of 2 mg can be administered, and if the treatment response is insufficient, the dosage may be increased to 4 mg once daily.[12] Abrocitinib, as a selective JAK1 inhibitor, generally provides a quicker and more pronounced reduction in pruritus in atopic dermatitis, whereas baricitinib, which inhibits both JAK1 and JAK2, exhibits broader immunomodulatory activity with a comparable but somewhat less rapid clinical response. Monitoring of laboratory parameters during treatment with JAK inhibitors is necessary due to their effects on the immune system and haematopoiesis, which may lead to decreased neutrophil and lymphocyte counts, the development of anaemia or thrombocytopenia, as well as increased lipid levels (LDL and HDL cholesterol) and an elevated risk of infections. Overall, JAK inhibitors represent an effective and rapidly acting therapeutic option for moderate-to-severe atopic dermatitis, combining strong clinical efficacy with a manageable safety profile under appropriate medical monitoring.

Microbiome-based therapies

Disruptions in the balance of the skin and gut microbiota (dysbiosis) play a key role in the pathogenesis of the disease. In patients with atopic dermatitis, increased colonization of the skin by *Staphylococcus aureus* is frequently observed, contributing to enhanced inflammation, impairment of the epidermal barrier, and increased pruritus. In contrast to healthy skin, AD skin is permissive for *S aureus* colonization.

The antimicrobial peptides LL-37, β-defensins, and dermicidin are present at reduced levels in AD skin. One mechanism underlying this effect is the known inhibition of IL-4 and IL-13 on human β-defensin 2 and 3 gene expression.

S aureus species grow poorly in acidic conditions, as seen in healthy stratum corneum, but grow much better in higher pH conditions, which are often seen in patients with AD.[13]

More targeted therapies such as microbiome-based options are needed that aim to restore a healthy skin microbiome in AD patients, reduce overgrowth of pathogenic drivers of AD and promote the recovery of commensals. These therapies include probiotics treatment, repopulating AD lesions with beneficial commensals, phage therapies, small molecules and peptides that counteract *S. aureus* colonization, humanized monoclonal antibodies that target bacterial toxins, as well as quorum sensing inhibitors that block virulence factors.[14] In patients with atopic dermatitis, a reduced diversity of the gut microbiome is observed, along with a decrease in beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*, and a concomitant increase in *Escherichia coli*, *Clostridium difficile*, and *Staphylococcus aureus*. These alterations may occur early in life and suggest that gut dysbiosis is one of the contributing factors in the pathogenesis of the disease. The most promising probiotic strains include *Lactobacillus rhamnosus* GG, *Bifidobacterium breve*, *Bifidobacterium animalis*, *Bifidobacterium longum*, *Lactobacillus plantarum*, and *Lactobacillus paracasei*. The use of these strains may restore the Th1/Th2 balance, inhibit the overgrowth of pathogenic intestinal bacteria, increase the production of short-chain fatty acids (SCFAs), and enhance regulatory T cell (Treg) function, which ultimately leads to alleviation of atopic dermatitis symptoms and a reduction in the frequency of disease exacerbations.

Calcineurin inhibitors

TCIs are immunosuppressant agents that have also been shown to be safe and effective for the treatment of AD, as well as the prophylaxis of AD flares. Two TCIs—pimecrolimus (Elidel) and tacrolimus (Protopic)—are currently approved for the second-line, intermittent treatment of immunocompetent patients 2 years of age and older with moderate-to-severe AD.[15] Tacrolimus is naturally produced by the fungus-like bacterium *Streptomyces tsukubaensis* and pimecrolimus is a chemically modified derivative of ascomycin produced by *Streptomyces hygroscopicus.* Tacrolimus and pimecrolimus both cause inhibition of T-cell activation by binding to the cytosolic FK-506 binding protein (FKBP) to form a complex blocking the activity of the enzyme calcineurin. Pimecrolimus is more lipophilic than tacrolimus, resulting in higher affinity for the skin and therefore lower permeation through the skin.

It is a weaker immunosuppressant, requiring higher topical concentrations for treatment.[16] In addition to their anti-inflammatory and antipruritic effects, topical calcineurin inhibitors also

exhibit other beneficial properties in the treatment of atopic dermatitis, including improvement of epidermal barrier function and increased hydration of atopic skin. Also, tacrolimus has the ability to alleviate pruritus. Topically applied tacrolimus inhibits the proliferation of activated lymphocytes in a dose-dependent manner, thereby reducing skin colonization by *Staphylococcus aureus*. Tacrolimus acts more rapidly and more potently than pimecrolimus, enabling clinical improvement to be achieved within the first week of treatment. Tacrolimus ointment is recommended for moderate-to-severe atopic dermatitis. Two concentrations are available: 0.1% and 0.03%. The 0.1% ointment is used as initial therapy in patients aged ≥16 years, whereas the 0.03% formulation is indicated for children (aged ≥2 years) and for patients who do not tolerate the higher-strength treatment.

Holistic treatment

Modern therapies (e.g., JAK inhibitors, biologic agents such as IL-4/IL-13 blockers, and microbiome-based interventions) act with high specificity on distinct pathogenic pathways in atopic dermatitis, primarily targeting the Th2 immune axis and cutaneous inflammation. However, pharmacological treatments alone do not address all disease-triggering factors, such as epidermal barrier dysfunction, microbiome dysbiosis, psychological stress, or environmental exposures. A holistic approach to the treatment of atopic dermatitis (AD) involves comprehensive management that addresses both the control of cutaneous symptoms and the modification of environmental, immunological, and psychological factors influencing the course of the disease. In addition to topical and systemic pharmacotherapy, regular use of emollients, avoidance of exacerbating factors (e.g., allergens, irritant detergents, stress), and patient and family education play a key role in disease management. Psychological support is also an essential component, as chronic pruritus and visible skin lesions can significantly impair quality of life and contribute to the development of anxiety and depressive disorders. MBT practices (such as meditation, mindfulness-based stress reduction [MBSR], hypnotherapy, guided imagery, and biofeedback) are common complementary therapies that use the mind to influence physical functions and directly affect health.[17]

Interventions targeting the skin and gut microbiome, as well as lifestyle modifications, including diet and skin hygiene, are also gaining increasing importance.

Discussion

The therapeutic view of atopic dermatitis (AD) has evolved substantially over recent years, reflecting an improved understanding of its complex and multifactorial pathophysiology. The introduction of targeted therapies, including biologic agents and Janus kinase (JAK) inhibitors, has marked a significant shift from traditional non-specific immunosuppressive approaches toward precision medicine. These modern treatments specifically target key immune pathways, particularly the Th2 axis, resulting in improved clinical outcomes, rapid symptom relief, and enhanced quality of life for patients with moderate-to-severe AD.

Biologic therapies such as dupilumab and tralokinumab have demonstrated robust efficacy and favorable safety profiles, largely due to their selective mechanisms of action and minimal systemic immunosuppression. The highly adaptable and potent pathogen *S.aureus* is known to express a multitude of virulence factors, including toxins that impact on the skin barrier and the immune system.[18] Their ability to improve epidermal barrier function, reduce inflammation, and decrease *Staphylococcus aureus* colonization highlights their multifaceted benefits. However, despite their effectiveness, not all patients achieve complete remission, and variability in treatment response remains a clinical challenge. Additionally, factors such as cost, accessibility, and the need for long-term data on safety and efficacy continue to limit their widespread use.

JAK inhibitors represent another important advancement, offering rapid onset of action, particularly in the reduction of pruritus—a key and burdensome symptom of AD. Their oral administration and broad immunomodulatory effects make them an attractive option for many patients. With the introduction of dupilumab and the JAK inhibitors into the pediatric AD market, it is expected that the use of traditional systemic immunosuppressants for this indication will become further infrequent.[19] Nevertheless, their mechanism of action, which involves interference with intracellular signaling pathways, necessitates careful monitoring due to potential adverse effects, including hematological abnormalities, lipid profile changes, and increased susceptibility to infections. This underscores the importance of individualized risk–benefit assessment and ongoing laboratory surveillance during therapy.

Emerging microbiome-based therapies provide a novel and promising avenue for AD management, targeting dysbiosis as a key contributor to disease pathogenesis.

Strategies aimed at restoring microbial balance—such as probiotics, bacteriophage therapy, and the use of commensal bacteria—have shown potential in reducing inflammation and improving skin barrier function. However, these approaches are still largely experimental, and further

high-quality clinical trials are needed to establish their efficacy, safety, and long-term outcomes.

Topical calcineurin inhibitors (TCIs) remain an important component of AD management, particularly in sensitive skin areas and for long-term maintenance therapy. Their favorable safety profile compared to topical corticosteroids, especially the lack of skin atrophy, makes them a valuable steroid-sparing option. Differences between tacrolimus and pimecrolimus in terms of potency, pharmacokinetics, and clinical efficacy allow for tailored treatment selection based on disease severity and patient characteristics.

Importantly, the growing recognition of the role of psychosocial factors and the skin–brain axis in AD has reinforced the need for a holistic, multidisciplinary approach to management. While modern pharmacological therapies effectively target immunological mechanisms, they do not fully address other critical aspects of the disease, such as barrier dysfunction, environmental triggers, and psychological stress. The closest interaction between the nervous and immunological systems in the skin occurs through a complex mix of psychosocial factors and psychiatric disorders.[20] Integrating lifestyle modifications, patient education, psychological support, and complementary therapies such as mind–body interventions may enhance treatment outcomes and improve long-term disease control.

Despite these advances, several limitations remain. High treatment costs, limited accessibility to advanced therapies, and insufficient long-term safety data for newer agents pose ongoing challenges. Furthermore, the heterogeneity of AD suggests that a “one-size-fits-all” approach is inadequate, highlighting the need for continued research into biomarkers that can guide personalized treatment strategies.

Conclusion

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease with a complex pathogenesis involving epidermal barrier dysfunction, immune dysregulation, and the influence of environmental, microbial, and psychosocial factors. In recent years, significant advances in the understanding of disease mechanisms have enabled the development of modern, targeted therapeutic approaches. Current AD management is based on several complementary strategies. Biologic agents such as dupilumab and tralokinumab effectively inhibit key cytokines of the Th2 axis, thereby improving skin barrier function and reducing inflammation.

JAK inhibitors provide a rapid onset of action, particularly in alleviating pruritus and controlling symptoms in moderate-to-severe disease.

Topical calcineurin inhibitors continue to play an important role as a safe alternative to topical corticosteroids, especially in long-term management and in sensitive skin areas.

Microbiome-targeted therapies and holistic approaches are also gaining increasing importance, including skin care routines, avoidance of exacerbating factors, psychological support, and lifestyle modifications. The role of the skin–brain axis in disease pathogenesis and progression is also increasingly recognized.

Despite significant progress, several limitations remain, including treatment costs, limited accessibility of advanced therapies, and insufficient long-term safety data. The heterogeneity of AD highlights the need for further development of personalized medicine approaches. In conclusion, modern therapies have significantly improved the effectiveness of AD treatment; however, optimal disease control requires an integrated approach combining targeted therapies with comprehensive holistic care.

Disclosure

Author's contribution

Conceptualization: Jagienka Perzyńska and Kinga Krzyżowska Methodology: Aleksandra Purska Software: Jaśmina Podkościelna Validation: Weronika Bagińska and Kamila Ryszkowska Formal analysis: Natalia Kasterka and Natalia Pawelec Investigation: Natalia Pawelec and Jagienka Perzyńska Resources: Kamila Ryszkowska Data curation: Jaśmina Podkościelna Writing-original draft preparation: Jagienka Perzyńska and Aleksandra Purska Writing-review and editing: Natalia Kasterka and Kamila Ryszkowska Visualization: Weronika Bagińska Supervision: Jagienka Perzyńska Project administration: Jagienka Perzyńska and Kinga Krzyżowska Fuding acquisition: no specifing funding.

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

Funding statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data availability Statement

Not applicable.

Conflict of Interest Statement

The authors deny any conflict of interest.

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