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A Report from the 2026 American Academy of Dermatology (AAD) Annual Meeting

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SUMMARIZING SESSIONS WITH A FOCUS ON PSORIASIS

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INTRODUCTION

The 2026 American Academy of Dermatology (AAD) Annual Meeting took place in Denver, Colorado, United States, from Thursday, March 26, to Tuesday, March 31, 2026. The AAD Annual Meeting is the largest global Dermatology gathering, with more than 20,000 attendees, including leading dermatology experts, clinicians, and researchers, featuring over 275 educational sessions that highlight late-breaking research, hot topics, live demonstrations, and hands-on sessions, including the International Eczema Council (IEC) and the International Psoriasis Council (IPC) Joint Symposium: 'Psoriasis or Dermatitis? When Boundaries Blur and Treatments Flip the Script.

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Diagnostic Challenges Between Atopic Dermatitis and Psoriasis

Andrew Pink, PhD, IPC Councilor
St. John's Institute of Dermatology, Guy's & St. Thomas' NHS Foundation Trust, London, United Kingdom

Diagnostic challenges between atopic dermatitis (AD) and psoriasis are common in clinical practice. This lecture reviewed mixed phenotype presentations, diagnostic approaches, and treatment considerations.

Four cases were presented to illustrate these challenges.

Mixed-phenotype disease, particularly involving psoriasis and eczema, reflects overlapping immunological pathways (Th1/Th17 vs Th2). Presentations include coexisting conditions at separate anatomical sites, fluctuating disease patterns within the same site, and overlapping phenotypes, such as sebopsoriasis or psoriasiform eczema, in which features of both conditions are present and require careful treatment selection.

Diagnostic approaches in these cases include clinical history, symptoms and signs, investigations, and response to treatment. Key factors include the presence of atopic or psoriatic disease, disease onset and evolution, itch, rash distribution, and inflammatory arthritis. Investigations may include IgE and eosinophil levels, biopsy, and additional tests such as mycology, TCR, autoimmune screening, and immunophenotyping. Treatment response may also provide insight into the dominant inflammatory pathway and support diagnosis.

Diagnostic challenges are particularly evident at specific sites, such as the hands, where overlap among dermatitis, psoriasis, and palmoplantar pustulosis (PPP) occurs. PPP is associated with complex T cell activation patterns, including overexpression of Th2 genes in the skin and a subset of memory CD4+ T cells in the blood expressing both Th17 and Th2 markers, demonstrating Th17-to-Th2 plasticity.

Genetic overlap between psoriasis and eczema was also discussed. A meta-analysis of genome-wide association studies in AD (60,653 cases) and psoriasis (36,466 cases) identified 43 loci specific to AD, 64 specific to psoriasis, 52 loci with consistent effects shared by both conditions, and 24 loci with opposing effects. These findings highlight shared and divergent disease mechanisms with potential implications for therapeutic development.

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5. 101 Comparative Genetic Analysis of Atopic Dermatitis and Psoriasis and the Potential Therapeutic Implications. Watts K, Dand N, et al. *Journal of Investigative Dermatology*. 145S284

Eczematous Reactions in Patients Receiving Treatments for Psoriasis: Clinical Presentation

Jason Hawkes, MD, MS, FAAD, IPC Councilor
Oregon Medical Research Center, Portland, Oregon, United States

Dr. Jason Hawkes focused on eczematous reactions in patients receiving psoriasis treatments.

The objectives of the lecture were to (1) review clinical observations, the literature, and a working hypothesis to explain eczematous reactions in psoriasis patients treated with biologics, and (2) discuss challenges and confounding variables when considering a clinical approach to disease management in this subset of patients.

A case of psoriasis treated with a biologic that resulted in a change in morphologic features highlighted the potential for initial misdiagnosis as a key confounder, given the lack of diagnostic biomarkers. Dr. Hawkes also reviewed terminology used in the literature, including "paradoxical eczema," "paradoxical eczematous reaction," "biologic-induced paradoxical eczematous reaction," and "phenotype" or "class-switching."

A central clinical challenge remains differentiating psoriasis from other conditions, such as mycosis fungoides (MF), nummular eczema, or cutaneous T cell lymphoma (CTCL), as psoriasis and eczema are clinical diagnoses with overlapping features. There are currently no definitive psoriasis biomarkers or widely commercially available diagnostic tests.

In a retrospective, multicenter observational study, 44% of patients had a family history of atopy, 30% had atopic conditions, and 20% had a prior history of eczema. The main biologics implicated were ixekizumab and secukinumab, with fewer cases reported for brodalumab and risankizumab. Remission was achieved in all 54 patients after switching to an interleukin-23 (IL-23) inhibitor.

Additional cases included generalized pustular psoriasis (GPP), psoriasis, and skin infection. Treatment approaches included switching to an IL-23 inhibitor or continuing interleukin-17A (IL-17A) therapy, supporting a personalized, case-by-case approach. "Class-switching" remains weakly supported, as pre-reaction skin profiling is not routinely performed.

Prior or concomitant atopy occurs in more than 50% of cases, although de novo presentations have been described. An atopic polygenic risk score appears to be associated with paradoxical eczema in patients with psoriasis treated with biologics.

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1. Eczematous Reactions in Patients with Plaque Psoriasis Receiving Biological Therapy: An Observational Study. Burlando M, Megna M, Caldarola G, et al. *Eur Rev Med Pharmacol Sci.* 2024 Sep;28(18):4298-4301.
2. Paradoxical Eczema Associated with Interleukin-17A Inhibitor Use in a Patient with Generalized Pustular Psoriasis Accompanied by Asthma. Yatsuzuka K, Muto J, Shiraishi K, et al. *Cureus.* 2024 Jul 16;16(7):e64680.

3. Integrated Proteomics and Genomics Analysis of Paradoxical Eczema in Psoriasis Patients Treated with Biologics. Al-Janabi A, Martin P, Khan AR, et al. *J Allergy Clin Immunol.* 2023 Nov;152(5):1237-1246.

Eczematous Reactions in Patients Receiving Treatments for Psoriasis: Clinical Approach

Kenneth Gordon, MD, IPC Councilor
Medical College of Wisconsin, Milwaukee, Wisconsin, United States

In this session, Dr. Kenneth Gordon outlined a clinical approach to managing eczematous reactions in patients receiving biologic psoriasis treatments, including patient counseling, diagnostic evaluation, and therapeutic decision-making.

The incidence of paradoxical eczema is shown in Figure 1.

There appears to be an increased risk in patients with a history of atopy, although this is not universal. Biopsies typically demonstrate spongiotic psoriasiform dermatitis with eosinophils, and patients may also exhibit peripheral eosinophilia and elevated IgE levels. Diagnostic complexity remains, particularly given the possibility of an initial misdiagnosis. Skin biopsy may be helpful when considering changes in therapy, both to support features of dermatitis and to exclude cutaneous T cell lymphoma (CTCL).

From a pathophysiologic perspective, immune deviation has been proposed as a likely mechanism, although this has not been definitively demonstrated. Multiple immunologic pathways may be involved. In the context of paradoxical atopic dermatitis (AD), the role of TWEAK was discussed. TWEAK, a member of the tumor necrosis factor (TNF) superfamily, may synergize with interleukin-13 (IL-13) to promote a Th2 response. Anti-TNF therapy may induce TWEAK expression, and paradoxical reactions appear to involve both type 1 interferon signaling and Th2 cytokine activity.

Several therapeutic strategies were presented. These include continuing the biologic with the addition of topical therapy or phototherapy; switching biologics, particularly from interleukin-17 (IL-17) inhibitors to interleukin-23 (IL-23) inhibitors; discontinuing the biologic and treating with topical or phototherapy alone; or transitioning to less specific agents, such as Janus kinase (JAK) inhibitors, tyrosine kinase 2 (TYK2) inhibitors, or cyclosporine. Combination approaches, such as adding methotrexate, were also discussed.

Based on clinical experience, paradoxical eczematous reactions occur at a low rate in patients treated with biologics for psoriasis and may be difficult to distinguish from atypical baseline presentations. Assessment of atopic features is important. Initial management may include continuing the biologic with adjunctive therapy, followed by switching treatment if needed. At present, there is insufficient evidence to support a standardized treatment algorithm, and response to initial therapy should guide management decisions.

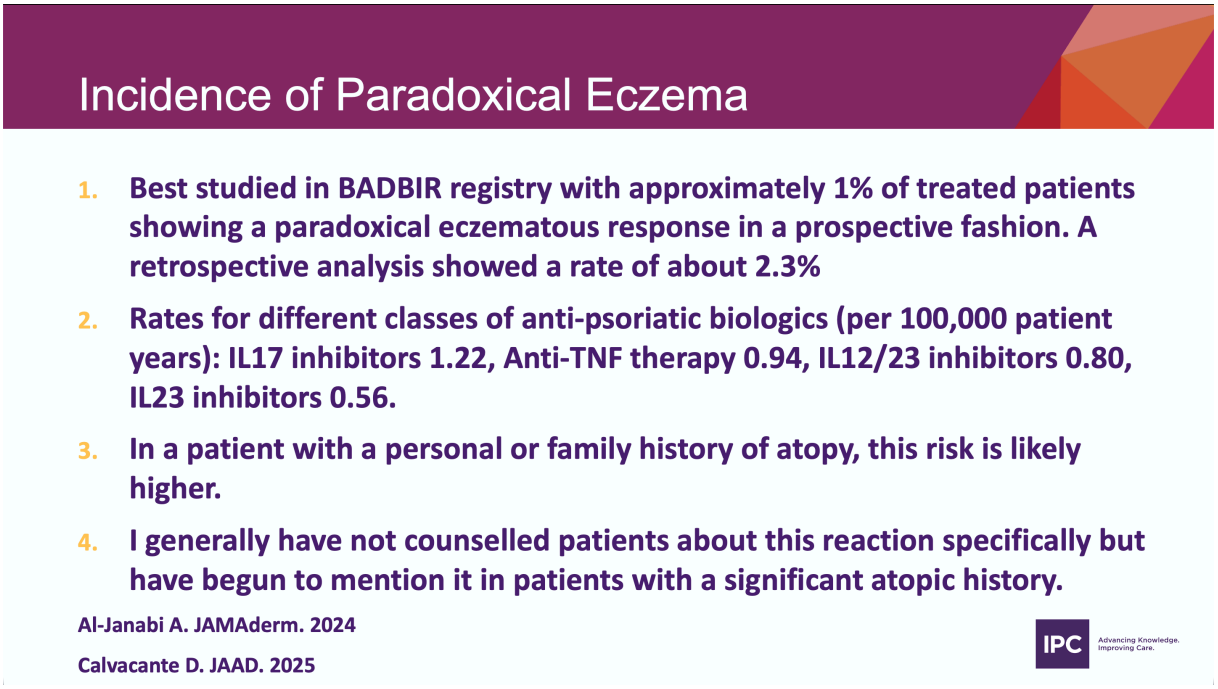

Figure 1.

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Psoriasiform Reactions in Patients Receiving Treatments for Atopic Dermatitis & Prurigo Nodularis: Clinical Presentation

Shawn G. Kwatra, MD
Johns Hopkins University School of Medicine, Baltimore, Maryland, United States

Dr. Shawn G. Kwatra presented on psoriasiform reactions in patients receiving treatments for atopic dermatitis (AD) and prurigo nodularis (PN), focusing on clinical presentation.

Psoriasiform reactions have been reported in patients treated with dupilumab, interleukin-13 (IL-13) inhibitors, including tralokinumab and lebrikizumab, and Janus kinase (JAK) inhibitors, including upadacitinib and abrocitinib. In adult patients with AD, these reactions have been reported in 1.8%–3.3% of those receiving dupilumab and in 2.1% of those receiving tralokinumab. Compared with other systemic therapies, such as corticosteroids, methotrexate, cyclosporine, and azathioprine, dupilumab has been associated with a 58% increased risk of developing psoriasis.

Plaque psoriasis is the most common presentation, accounting for 77.1% of cases, and may occur more frequently in men (54.3%). The extremities are the most commonly affected sites (47.4%). The mean time to development of psoriasiform reactions is approximately 20 weeks in adults and 32 weeks in children receiving dupilumab.

The underlying pathogenesis appears to reflect immune drift from Th2 to Th1/Th17 inflammation. As PN is a Th2-dominant condition, with involvement of Th1 and Th17/Th22 axes, inhibition of Th2 pathways may unmask Th1/Th17 responses. This immune shift appears to be more common in patients who respond to treatment. Additionally, patients with a personal or family history of psoriasis may be at increased risk.

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2. Psoriasis Risk in Patients with Atopic Dermatitis Treated with Dupilumab. Lin TL, Fan YH, Fan KS, et al. *JAMA Dermatol.* 2025 Aug 1;161(8):813-821.
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4. Development of a Clinical Algorithm to Predict Phenotypic Switches Between Atopic Dermatitis and Psoriasis (the "Flip-Flop" Phenomenon). Müller S, Welchowski T, Schmid M, et al. *Allergy.* 2024 Jan;79(1):164-173.

Psoriasiform Reactions in Patients Receiving Treatments for Atopic Dermatitis & Prurigo Nodularis: Clinical Approach

Yael Anne Leshem, MD, MCR
Rabin Medical Center, Petah Tikva and Tel Aviv University, Tel Aviv, Israel

Dr. Yael Anne Leshem presented on psoriasiform reactions in patients with atopic dermatitis (AD), focusing on pathogenesis and clinical management.

Psoriasiform reactions can occur in patients with AD receiving treatment, reflecting an imbalance between Th17 and Th2 inflammation. The pathogenesis involves a shift in immune response, contributing to the development of psoriasis-like features in this population.

Treatment approaches were outlined in four main categories: conservative management, including treating through with topical therapies or phototherapy; broader immunomodulation with agents such as methotrexate (MTX), mycophenolate mofetil (MMF), and Janus kinase inhibitors (JAKi); modification of Th2 blockade, including dose reduction, discontinuation, or intraclass switching; and targeting the Th17 pathway with therapies such as ustekinumab, interleukin-17 (IL-17), or interleukin-23 (IL-23) inhibitors.

Dr. Leshem also discussed the "flip-flop" phenomenon, defined as a phenotype switch between AD and psoriasis. This rare, immune-mediated shift is often triggered by biologic therapies, such as dupilumab, and reflects immune reprogramming from Th2-driven inflammation toward Th17/Th22-driven pathways. While AD and psoriasis are distinct conditions, they share overlapping immune mechanisms, and this switch may occur in a subset of patients.

The estimated rate of switching from AD to psoriasis under biologic therapy is approximately 1.7%. A clinical algorithm has been developed to identify patients at risk for this transformation, with an accuracy of approximately 89.7% (Figure 1).

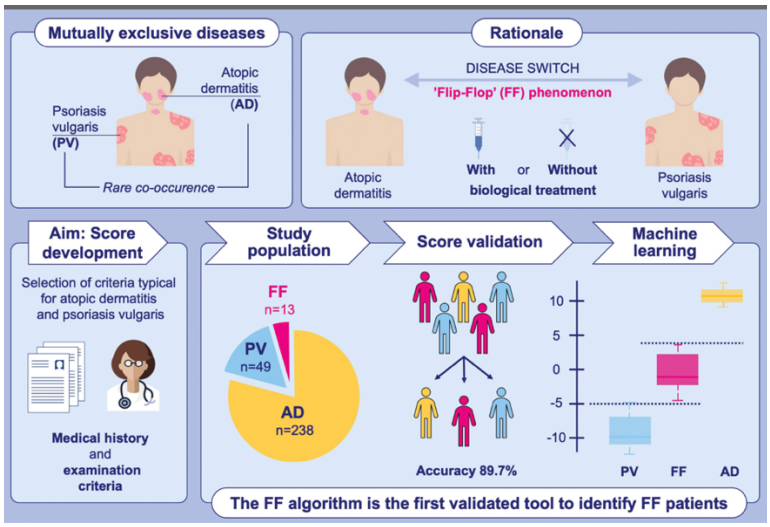

Figure 1. Müller S, Welchowski T, Schmid M, et al. Development of a clinical algorithm to predict phenotypic switches between atopic dermatitis and psoriasis (the "Flip-Flop" phenomenon). Allergy. 2024;79:164-173.

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What is Psoriasiform Spongiotic Dermatitis? Navigating Uncertainty Using Advanced Diagnostics

Kilian Eyerich, MD, PhD, IPC Councilor
University of Freiburg, Freiburg, Germany

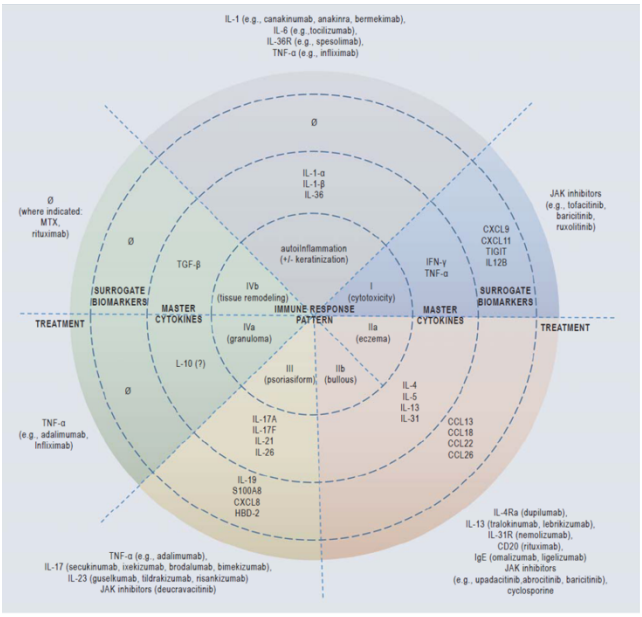
Professor Kilian Eyerich focused on the use of advanced diagnostics to guide treatment selection in patients with psoriasiform spongiotic dermatitis.

He first introduced the concept of stratified medicine in the treatment of inflammatory skin diseases. The classification of these conditions is shifting from a descriptive approach to a molecular, pathophysiology-based framework centered on immune response patterns. Four key immune response patterns were highlighted, with atopic dermatitis (AD) representing a type 2 response and psoriasis representing a type 3 (Th17) response. These patterns are amenable to targeted therapies, and identifying the dominant immune signature may help predict treatment response and support the use of specific immunotherapies.

Professor Eyerich then presented a cross-comparison of gene expression profiles across multiple inflammatory skin diseases to identify modules representing key immune pathways. Seven modules were identified: Th17, Th2, Th1, type I interferons, neutrophilic, macrophagic, and eosinophilic. These modules were used to develop a molecular map with high diagnostic utility in both well-defined and clinically indeterminate cases.

Module matching to treatment target appeared to increase response rates in both treatment-naïve patients and non-responders. This was evaluated using 80 pre-treatment biopsies from patients with AD, psoriasis (PsO), and lichen planus (LP) undergoing targeted therapy. The dominant expression module in skin samples (Th1, Th2, or Th17) was matched to the therapeutic target, including Th2 for anti–IL-4R and IL-13 therapies, Th17 for anti–IL-23 and IL-17A/F therapies, and Th1 for JAK1/2 inhibitors. All responding patients (n = 60) had matched profiles, whereas non-responders (n = 19) included both matched and unmatched profiles, suggesting that profile matching may be necessary but not sufficient for treatment response.

Despite this, overlapping phenotypes remain common and complicate diagnosis and treatment selection. These may occur through dynamic shifts in immune response patterns or simultaneous features of type 2 and type 3 inflammation. Even in conditions such as nummular eczema or psoriasiform spongiotic dermatitis, molecular diagnostics may help identify the dominant pathway. Targeting this pathway may improve inflammatory features and reduce the need for less-specific therapies, such as systemic corticosteroids or cyclosporine.



he differential treatment of inflammatory dermatoses according to their immune response patterns (signatures). The reponse pattern is shown in the center of the circle, ollowed (proceeding outward) by its master cytokines, easily measurable biomarkers serving as surrogates for it (19), and specific treatment options. In principle, all dis- uses that can be assigned to a particular immune response pattern can be treated specifically as indicated here (see *Figure 1*), but not every treatment has been approved or every disease with the corresponding response pattern.

Figure 1. Inflammatory Skin Diseases. Garsozk N. *Dtsch Arztebl.* Int 2025; 122: 277-82. DOI: 10.3238/arztebl.m2025.0045

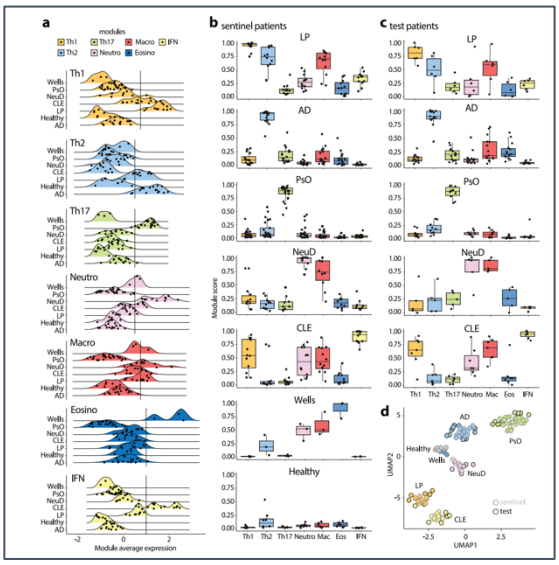


Figure 2. Immune Modules to Guide Diagnosis and Personalized Treatment of Inflammatory Skin Diseases. Seremet T, Di Domizio J, Girardin A, et al. *Nat Commun.* 2024 Dec 18;15(1):10688.

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Anti-TNF-α Induced Skin Eruptions in Patients with Inflammatory Bowel Disease: Presentation and Clinical Approach in Adults

Lev Pavlovsky, MD, PhD, IPC Councilor
Tel Aviv University, Petah-Tikva, Israel

Anti-TNF-α psoriasis-induced psoriasiform lesions are relatively common in patients with Inflammatory Bowel Disease (IBD). The overall pooled incidence was 6% in a systematic review by Xie W et al., while the mean time to onset after starting treatment was 11 months (ranging from 1 month to several years). Risk factors for the development of the reactions included being female, younger age, smoking, ileocolonic Crohn's disease (CD), and being on adalimumab or certolizumab.

In terms of clinical characteristics, a multicenter cohort study of 181 patients found that 81% had no history of psoriasis, atopy, or other immune-mediated conditions. The scalp was most commonly affected, followed by the trunk, limbs, and hands and feet. The morphology was predominantly psoriasiform.

After presenting an interesting case, a treatment approach was proposed. See Figure 1.

In general, patients with mild disease can continue anti-TNF therapy with appropriate dermatologic management. In contrast, patients with severe/refractory disease or inflammatory alopecia may require discontinuation of the responsible agent. Data suggest that ustekinumab has been associated with significant benefit on skin and prevention of IBD relapse. Key gaps remain, including a lack of standardized diagnostic criteria and personalized treatment pathways. Optimal care requires multidisciplinary management, individualized risk-benefit assessment, and close monitoring of both skin and IBD activity.

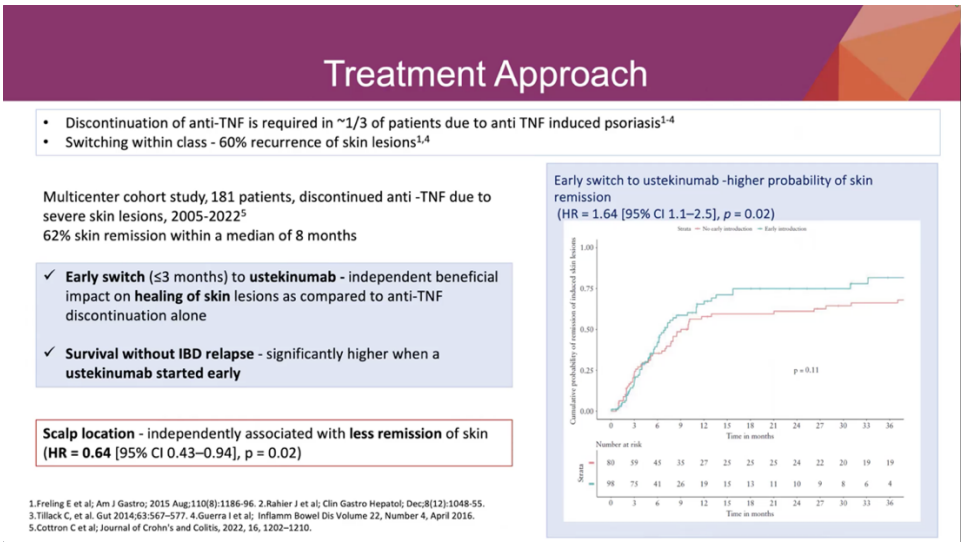

Figure 1.

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Anti-TNF-α Induced Skin Eruptions in Patients with Inflammatory Bowel Disease: Presentation and Clinical Approach in Children

Amy Paller, MS, MD, IPC Board Member
Northwestern University Feinberg Medical School, Chicago, Illinois, United States

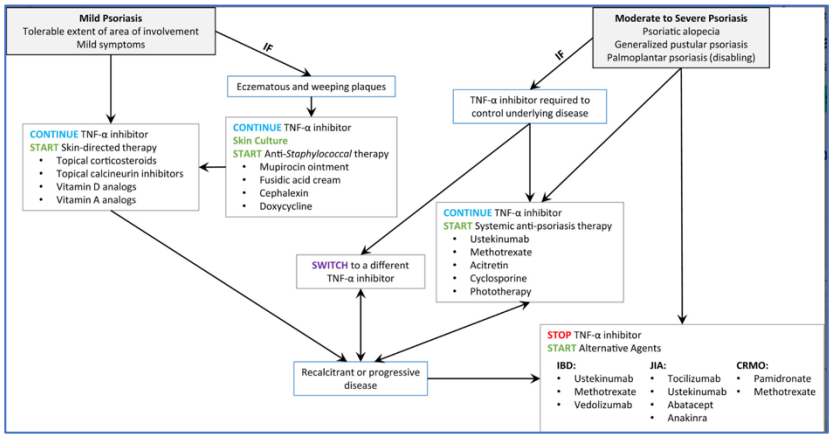
The prevalence of TNFi-induced psoriasiform dermatitis (TNFi-PsoD) in children with IBD is estimated to be 4.6%–13.6%. The median age at presentation is approximately 14 years, with a median time to onset of 14–34 months after initiation of TNFi therapy. Concurrent use of methotrexate does not appear to reduce the risk. Among TNFi agents, the risk is highest with infliximab, followed by adalimumab, while cases are rare with certolizumab pegol, golimumab, and etanercept.

Most patients present with involvement of multiple sites, although the overall body surface area is typically limited. The scalp is the most affected site (48%–73%), similar to pediatric psoriasis. Truncal lesions are often small and may appear guttate. Lesions may also involve the hands, fingers, feet, toes, and nails, including pustular features, while knees and elbows are affected in fewer than 20% of cases.

Although gastroenterology services are familiar with TNFi-induced eruptions, two key points were emphasized: the risk of classical plaque psoriasis is increased in patients with IBD, and pediatric plaque psoriasis is often underrecognized. Lesions may present as indistinct plaques with thinner scale, increased facial involvement, or isolated scalp disease. There may also be overlap between psoriasis and atopic dermatitis, including psoriasiform dermatitis presentations.

Noninvasive tape stripping and proteomic profiling may help distinguish psoriasis from TNFi-induced psoriasiform dermatitis based on biomarkers. Several genetic associations have been identified, including interleukin-23 receptor polymorphisms and TNF-α variants associated with increased risk following infliximab treatment, as well as additional genes such as FBXL19, CTLA4, TAP1, and SLC12A8.

Management strategies were outlined using a practical algorithm (Figure 1). Children may respond better than adults to topical therapies but often require discontinuation of TNFi treatment. Switching within the TNFi class is frequently associated with recurrence, while ustekinumab has demonstrated favorable efficacy when a biologic switch is needed.



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Mechanistic Aspects of Paradoxical Reactions and Iatrogenic Switches

Johann Gudjonsson, MD, PhD, IPC Board Member
University of Michigan, Ann Arbor, Michigan, United States

Professor Johann Gudjonsson discussed mechanistic aspects of paradoxical reactions and iatrogenic switches, focusing on immune polarization patterns in inflammatory skin diseases.

Four key immune response patterns were outlined: lichenoid (Th1), eczematous or blistering (Th2), psoriasiform (Th17), and fibrogenic or granulomatous (transforming growth factor-β [TGF-β]) (Figure 1). These patterns help frame the clinical and immunologic presentation of inflammatory skin conditions.

Paradoxical reactions were defined as the development of a new cutaneous inflammatory condition during immunomodulatory therapy that is either typically treated by the same class of therapy or arises through compensatory activation of alternative immune pathways. These reactions reflect immune network plasticity rather than treatment failure.

Targeted cytokine blockade, including tumor necrosis factor (TNF), interleukin-17 (IL-17), and interleukin-4 receptor (IL-4R), may unmask alternative inflammatory pathways, including Th1, Th2, Th17, interferon (IFN), and TGF-β–mediated responses. Additional pathways beyond type I and type II IFNs may also contribute to these reactions.

Paradoxical reactions highlight that inflammatory skin diseases are driven by interconnected immune networks rather than single cytokines. Psoriasis represents a dynamic immune balance centered on IL-17 but linked to multiple immune axes. Therapeutic perturbation may shift this balance, leading to compensatory activation of parallel pathways, such as IL-17 inhibition, resulting in Th2-driven eczematous responses, or IL-4R blockade, leading to psoriasiform inflammation.

These paradoxical phenotypes exhibit predictable patterns of immune polarization. Clinical manifestations, including psoriasiform, eczematous, lichenoid, and pustular presentations, align with dominant immune axes such as Th17, Th2, Th1, and innate pathways, including IL-36 and IFNs.

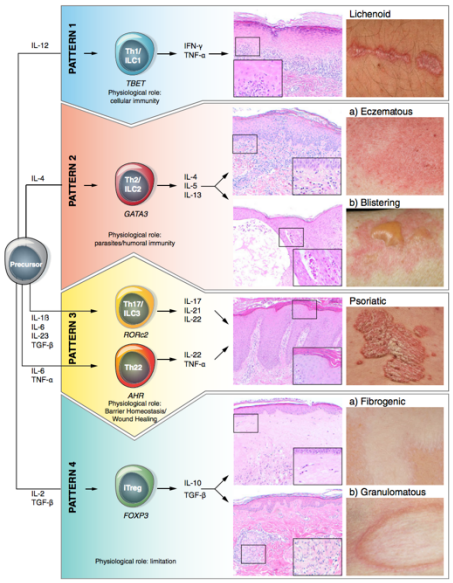

Figure 1. Immune Response Patterns in Non-Communicable Inflammatory Skin Diseases. Eyerich K, Eyerich S. J *Eur Acad Dermatol Venereol*. 2018 May;32(5):692-703.

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Therapeutic Hotline: What's New this Year in Psoriasis Biologics

Joseph Merola, MD, MMSc, IPC Councilor
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Dr. Joseph F. Merola reviewed key advances in psoriasis and psoriatic arthritis therapeutics, with a strong emphasis on agents targeting the interleukin (IL)-23 pathway, combination therapy of ixekizumab with tirzepatide, next-generation tyrosine kinase 2 (TYK2) inhibitors, and emerging long-acting biologics.

Merola reviewed the available data on icotrokinra**,** an oral IL-23 receptor antagonist.[1] Across the ICONIC program, icotrokinra demonstrated strong efficacy in moderate to severe psoriasis, including rapid improvements in PASI 90, PASI 100, investigator's global assessment (s-IGA) 0/1, and symptom scores, with durable responses extending through 52 weeks in adolescents.[2] Safety was generally favorable, with mostly common infections and gastrointestinal adverse events and low rates of serious adverse events.

Among oral TYK2 inhibitors, envudeucitinib and zasocitinib showed encouraging efficacy, with response rates exceeding placebo and apremilast, and safety profiles characterized mainly by mild to moderate adverse events, without major laboratory concerns in the data shown.

Regarding combination therapy with ixekizumab plus tirzepatide, 27.1% of participants receiving the combination reached complete skin clearance (PASI 100) and at least 10% weight loss, compared to 5.8% of patients treated with ixekizumab alone ($p<0.001$). In a key secondary endpoint, ixekizumab plus tirzepatide showed a 40% relative increase over ixekizumab monotherapy in the proportion of patients achieving PASI 100 (40.6% vs. 29.0%, respectively; $p<0.05$).[3]

Finally, the talk introduced ORKA-001**,** a novel IL-23 monoclonal antibody engineered for prolonged half-life through Fc modification, raising the possibility of less frequent dosing while maintaining potency similar to risankizumab. Overall, the session underscored a shift toward highly effective oral agents and longer-acting biologics that may reshape psoriasis treatment strategies.

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Psoriasis: Modern Therapeutic Approaches

Bruce Strober, MD, PhD, IPC President | Yale University and Central Connecticut Dermatology, United States
Richard Langley, MD, IPC Councilor | Dalhousie University, Halifax, Nova Scotia, Canada
Mona Shahriari, MD | Yale University School of Medicine, New Haven, Connecticut, United States
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Jennifer Soung, MD | Harbor University of California, Los Angeles, California, United States
Joel Gelfand, MD, MSCE, IPC Board Member | University of Pennsylvania, Philadelphia, Pennsylvania, United States
Kenneth Gordon, MD, IPC Councilor | Medical College of Wisconsin, Milwaukee, United States

This session covered current approaches to psoriasis management, including new therapies, early treatment decisions, safe prescribing, topical options, and key comorbidities.

Emerging therapies were a central focus, particularly interleukin-23 (IL-23)–targeted treatments, oral agents, and pipeline biologics in both psoriasis and psoriatic arthritis (PsA). There was also a strong emphasis on treating earlier in the disease course. Rather than relying only on body surface area or PASI scores, speakers discussed the need to consider patient burden, involvement of high-impact sites such as the scalp and nails, and the risk of disease progression.

Practical considerations were also reviewed, including treatment decisions in the setting of tuberculosis risk (including IPC and National Psoriasis Foundation recommendations), [1] pregnancy, malignancy, and surgery. Updated topical strategies were discussed, including newer steroid-sparing agents and the concept of topical failure.

Psoriasis was also addressed as a systemic condition, particularly in relation to obesity and cardiovascular disease. The need for better screening, coordination with other specialties, and a more integrated approach to care was emphasized. Finally, the possibility of delaying or preventing psoriatic arthritis in high-risk patients was discussed, and data from retrospective big-data studies were reviewed.[2] However, no causality can be inferred from these articles and requires further study.

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What is Safe to Use for Routine Dermatologic Disease if Your Patient Has Been Diagnosed with Cancer? Managing Psoriasis and Atopic Dermatitis in Patients with Cancer

Gowri Kabbur, MD
Cleveland Clinic Dermatology Residency, Cleveland, United States

Dr. Gowri Kabbur discussed the management of psoriasis and atopic dermatitis in patients with current or prior malignancy, using a case-based approach to guide treatment decisions.

The session opened with a case of an older patient with urothelial carcinoma receiving pembrolizumab and enfortumab vedotin who developed a new pruritic eruption. Biopsy findings showed overlapping spongiotic and psoriasiform features, highlighting the diagnostic and therapeutic complexity of inflammatory skin disease in the oncology setting.

A central focus was balancing effective treatment with malignancy risk, recurrence risk, and quality of life. Non-immunosuppressive therapies were presented as preferred first-line options, including topical corticosteroids, vitamin D analogs, narrowband ultraviolet B (UVB) phototherapy, and acitretin. Acitretin was emphasized as a useful option given its lack of association with increased infection or malignancy risk and its potential chemoprotective role against nonmelanoma skin cancer in select populations.

Systemic options with more reassuring data on malignancy were also reviewed. Phosphodiesterase-4 (PDE-4) inhibitors, particularly apremilast, were reported to have no clear malignancy signal in clinical trials or registry data. IL-17 and IL-23 inhibitors were also presented as reasonable options, with studies showing low malignancy incidence and no strong signal for recurrence or new cancer development, including in patients with prior malignancy.

TNF-α inhibitors were discussed more cautiously due to concerns related to nonmelanoma skin cancer, melanoma, and possible lymphoma. Methotrexate was associated primarily with increased nonmelanoma skin cancer risk rather than overall malignancy risk.

Overall, a stepwise approach was proposed, starting with non-immunosuppressive therapies, followed by IL-23, IL-17, IL-12/23 inhibitors, PDE-4 inhibitors, or acitretin, with methotrexate, cyclosporine, and TNF inhibitors generally reserved for later use in collaboration with oncology.

What is Safe to Use for Routine Dermatologic Disease if Your Patient Has Been Diagnosed with Cancer? Treatment of Hand and Foot Psoriasis

Bruce Strober, MD, PhD, IPC President
Yale University and Central Connecticut Dermatology, Cromwell, Connecticut, United States

Dr. Bruce Strober reviewed the diagnosis and management of palmoplantar psoriasis (PP) and palmoplantar pustulosis (PPP), emphasizing their effect on quality of life and the challenge of distinguishing them from other palmoplantar dermatoses. A key point was the importance of accurate diagnosis before treatment selection, particularly given that more than one condition may coexist, such as psoriasis with irritant or allergic contact dermatitis.

Palmoplantar psoriasis was described as a common and often life-altering manifestation, affecting more than 40% of patients with psoriasis. It may be triggered or worsened by smoking, stress, trauma, medications, and Koebnerization. Most plaque psoriasis treatments may be effective, although evidence supports interleukin-17 (IL-17) and interleukin-23 (IL-23) inhibitors, with specific trials supporting their use.[1-4] Dr. Strober also presented his personal treatment-ranking approach for hyperkeratotic palmoplantar plaque psoriasis, in which JAK inhibitors, particularly upadacitinib and tofacitinib, were placed first, followed by IL-23 and IL-17 inhibitors at a similar level, then methotrexate, deucravacitinib, apremilast, ustekinumab, TNF-α inhibitors, cyclosporine, acitretin, and, finally, combinations of these agents.

Palmoplantar pustulosis was presented as a distinct, less common but highly burdensome condition with a female predominance. Triggers include infection, tobacco use, stress, biologics, and contact dermatitis. In contrast to PP, no single therapy has emerged as a clear standard. Biologic therapies were described as a "double-edged sword," as they may be effective. Still, they can also trigger PPP, particularly in patients exposed to tumor necrosis factor. Classic systemic drugs such as methotrexate appear to be a good option. Dr. Strober also shared his personal therapeutic hierarchy for PPP, placing JAK inhibitors first, followed by methotrexate, apremilast, TNF-α inhibitors, IL-23 and IL-17 inhibitors at a similar level, then deucravacitinib, cyclosporine, ustekinumab, acitretin, and combinations of the above.

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Late-Breaking Research Session 1: Envudeucitinib (ESK-001) in moderate to severe plaque psoriasis: 24-week results from the randomized, double-blind, active comparator- and placebo-controlled, Phase 3 ONWARD 1 and 2 studies

Andrew Blauvelt, MD, MBA, IPC Board Member
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In this late-breaking session, Dr. Andrew Blauvelt presented 24-week results for envudeucitinib (ESK-001), a next-generation oral allosteric tyrosine kinase 2 inhibitor (TYK2i) designed to provide sustained pathway inhibition over 24 hours. The drug targets interleukin (IL)-23/IL-17, IL-12, and type I interferon pathways.

Data were presented from ONWARD1 and ONWARD2, two replicate phase 3, randomized, double-blind trials comparing envudeucitinib 40 mg twice daily with placebo and apremilast 30 mg twice daily in adults with moderate to severe plaque psoriasis. A total of 912 and 859 patients were enrolled, respectively, with baseline Psoriasis Area and Severity Index (PASI) scores of approximately 20 and body surface area involvement of approximately 25%.

Both coprimary endpoints, PASI 75 and static Physician's Global Assessment (sPGA) 0/1 at week 16 versus placebo, were met with statistical significance ($P<0.0001$). PASI 75 responses were 76.5% in ONWARD1 and 70.4% in ONWARD2. Responses continued to improve through week 24, with approximately 65% achieving PASI 90 and 40% achieving PASI 100, without evidence of plateau. Separation from placebo was observed as early as week 4.

Additional benefits included improvement in scalp psoriasis (scalp-specific PGA 0/1 of approximately 75% at week 24), rapid itch reduction, and improvements in quality of life, with Dermatology Life Quality Index (DLQI) 0/1 achieved in approximately 60% of patients. Improvements in itch and quality of life preceded PASI 90 responses.

The safety profile was favorable, with low rates of serious adverse events (2.0%) and no deaths, major adverse cardiovascular events, cytopenia signals, or tuberculosis reactivation. The most common adverse events were headache, acne, and nasopharyngitis. Phase 3 one-year data are expected in the second half of 2026.

Late-Breaking Research Session 2: Once-daily Oral Zasocitinib Demonstrates Rapid and Reproducible Skin Clearance with a Consistent Safety Profile in Moderate-to-Severe Plaque Psoriasis: Results from Two Randomized Phase 3 Trials (LATITUDE-PsO-3001 and 3002)

Melinda Gooderham, MD, IPC Councilor
SKiN Centre for Dermatology, Peterborough, Ontario, Canada

Results from the LATITUDE-PsO-3001 and 3002 trials evaluated zasocitinib, an investigational once-daily oral allosteric tyrosine kinase 2 inhibitor (TYK2i) with high selectivity for TYK2 over Janus kinase (JAK) 1, JAK2, and JAK3, designed to maintain 24-hour inhibition of interleukin-23 (IL-23) and related pathways.

These multicenter, randomized, double-blind phase 3 trials compared zasocitinib with apremilast and placebo in adults with moderate to severe plaque psoriasis. A total of 693 patients were enrolled in LATITUDE-PsO-3001 (3:1:1 randomization), and 1,108 in LATITUDE-PsO-3002 (2:1:1). Baseline disease severity was similar across groups, with a mean Psoriasis Area and Severity Index (PASI) of approximately 20 and body surface area involvement of approximately 25%.

Both coprimary endpoints at week 16 versus placebo were met ($P<0.001$). Static Physician's Global Assessment (sPGA) 0/1 responses reached 71% and 69%, and PASI 75 responses reached 76% and 71% in LATITUDE-PsO-3001 and 3002, respectively, compared with 11%–13% for placebo. Responses continued to improve through week 24, with PASI 90 rates of 69% and 63%, and PASI 100 rates of 42% and 32%. Clear skin (sPGA 0) was achieved in approximately one-third of patients by week 16. Dermatology Life Quality Index (DLQI) 0/1 responses were superior to apremilast and placebo as early as week 4, with 51% and 60% at week 24, respectively.

In LATITUDE-PsO-3002, more than 90% of patients who continued zasocitinib at week 40 maintained sPGA 0/1, PASI 75, and PASI 90 responses through week 60. In a randomized withdrawal design, 59%, 69%, and 52% of patients re-randomized to placebo maintained these responses for approximately 5 additional months.

The safety profile remained consistent through week 24, with serious adverse events reported in 3.0% of patients. The most common treatment-emergent adverse events were upper respiratory tract infection, acne, and nasopharyngitis, and were generally mild to moderate. No clinically meaningful laboratory abnormalities were observed. A 3-year long-term extension study and a head-to-head trial versus deucravacitinib are ongoing.