IPC
INTERNATIONAL PSORIASIS COUNCIL

MAY 2026

IPC Symposium Report from the 2026 Society for Investigative Dermatology (SID) Annual Meeting

Maria Julia Cura, MD, IPC Jr. Councilor
Jennifer Lavina Ngo, MD-MBA, FPDS, IPC Jr. Councilor

SUMMARIZING SESSIONS FROM THE IPC SYMPOSIUM

IPC
INTERNATIONAL PSORIASIS COUNCIL

INTRODUCTION

The International Psoriasis Council (IPC) hosted a scientific symposium, The Arc of Disease Control in Psoriasis: Early Interception - Deep Pathway Blockade - Treatment Failure, during the 2026 Society for Investigative Dermatology (SID) Annual Meeting on May 9, 2026, in Chicago, Illinois, United States. The symposium featured presentations on disease interception, immune memory, molecular profiling, treatment response, and mechanisms of therapeutic escape in psoriasis. This report summarizes the presentations and discussion from the IPC symposium.

TABLE OF CONTENTS

IPC Symposium: The Arc of Disease Control in Psoriasis: Early Interception – Deep Pathway Blockade – Treatment Failure

3	**Intercepting Psoriasis at the Earliest Stages: Lessons from STEP-IN and GUIDE** Knut Schäkel, MD
5	**Preventing Psoriatic Arthritis: Can Immune Interception Change the Natural Disease Course?** Shikha Singla, MD
8	**Implications of Controlling Psoriasis Inflammation: From Molecular Profiling to Comorbidities** Alex Tsoi, MS, PhD, IPC Councilor
10	**Oral Versus Biologics – Targeted or Broader Approaches?** Christopher Bunick, MD, PhD, IPC Councilor
11	**Why Treatment Fails: Interferons, Immune Rewiring, and Biological Escape Mechanisms** Curdin Conrad, MD, IPC Board Member
12	**Clinical Escape in Psoriasis: Paradoxical Reactions and Flip-Flop Phenomena** Wilson Liao, MD, IPC Councilor

Intercepting Psoriasis at the Earliest Stages: Lessons from STEP-IN and GUIDE

Knut Schäkel, MD
Heidelberg University Hospital, Heidelberg, Germany

This presentation focused on disease interception and modification in psoriasis, drawing on findings from the STEP-IN and GUIDE studies.

Disease modification was defined as a sustained improvement in plaque psoriasis resulting from pathophysiologic changes that minimize the need for continuous treatment, clinically reflected by maintaining a BSA <1% and PGA 0/1 for more than 12 months after treatment withdrawal.[1]

A major concept discussed was "disease memory", which may explain relapse despite clinical remission. Two complementary mechanisms were highlighted: innate immune memory driven by epigenetic reprogramming, and adaptive immune memory mediated by tissue-resident memory T cells (TRM). Experimental imiquimod models demonstrated persistent epigenetic scars in epidermal stem cells, including sustained chromatin accessibility and methylation changes involving STAT3 and AP-1 (FOS/JUN) pathways, facilitating exaggerated inflammatory responses upon re-challenge.[2]

The STEP-IN study evaluated whether early secukinumab treatment could normalize these epigenetic alterations.[3] Patients with new-onset psoriasis showed substantial normalization of differentially methylated CpGs, particularly in the epidermis. In contrast, chronic lesions retained persistent methylation abnormalities even at Week 52, supporting the concept of sustained tissue inflammatory memory. Differential methylation analyses revealed distinct transcription factor enrichment patterns according to disease duration, with ETS-family factors (SPI1/PU.1, IKZF1) predominating in new-onset psoriasis and AP-1 family factors (JUN/FOS) in chronic disease.[4,5]

Previous studies demonstrated that CD8+CD49a+ tissue-resident memory T cells capable of producing IL-17A persist in clinically resolved psoriasis lesions and may contribute to disease recurrence.[6] Building on this concept, the GUIDE study showed that patients with short disease duration achieved PASI 0 more rapidly, normalized epidermal TRM cells earlier, and maintained longer drug-free remissions after treatment withdrawal than those with longstanding disease.[7] Higher IL-17F levels during treatment withdrawal were associated with earlier relapse, whereas super-responders showed increased IL-10-producing T cells, suggesting a favorable immunoregulatory environment.[8]

References:

1. An International Delphi Consensus to Define a Clinically Appropriate Definition of Disease Modification for Plaque Psoriasis. Eyerich K, Krueger J, Stahle M, et al. *J Eur Acad Dermatol Venereol.* 2024;38(5):e424-e427. doi: 10.1111/jdv.19652.
2. Establishment, Maintenance, and Recall of Inflammatory Memory. Larsen SB, Cowley CJ, Sajjath SM, et al. *Cell Stem Cell.* 2021;28(10):1758-1774.e8. doi: 10.1016/j.stem.2021.07.001.

3. Secukinumab Treatment in New-Onset Psoriasis: Aiming to Understand the Potential for Disease Modification - Rationale and Design of the Randomized, Multicenter STEPIn Study. Iversen L, Eidsmo L, Austad J, et al. *J Eur Acad Dermatol Venereol.* 2018;32(11):1930-1939. doi: 10.1111/jdv.14979.
4. Conrad C, et al. Poster presented at ISID 2023. P828
5. Conrad C, et al. Oral presentation at EADV 2023. FCO8.1
6. Epidermal Th22 and Tc17 Cells Form a Localized Disease Memory in Clinically Healed Psoriasis. Cheuk S, Wilkén M, Blomqvist L, et al. *J Immunol.* 2014;192(7):3111-20. doi: 10.4049/jimmunol.1302313.
7. Early Disease Intervention with Guselkumab in Psoriasis Leads to a Higher Rate of Stable Complete Skin Clearance ('Clinical Super Response'): Week 28 Results from the Ongoing Phase IIIb Randomized, Double-Blind, Parallel-Group, GUIDE Study. Schäkel K, Reich K, Asadullah K, et al. *J Eur Acad Dermatol Venereol*. 2023;37(10):2016-2027. doi: 10.1111/jdv.19236.
8. Chen Y, et al. Poster presented at SID 2024. Poster number LB943.

Preventing Psoriatic Arthritis: Can Immune Interception Change the Natural Disease Course?

Shikha Singla, MD
Medical College of Wisconsin, Milwaukee, Wisconsin, United States

Psoriatic arthritis (PsA) develops in approximately 30% of patients with psoriasis and is typically preceded by a latency period of nearly seven years, creating a potential "window of opportunity" for intervention. The goal is to move beyond treating established disease toward preventing the onset of PsA.

The current model of PsA development proposes a progression from immune activation and subclinical inflammation to a symptomatic prodromal phase characterized by arthralgia, stiffness, fatigue, and imaging abnormalities before fulfillment of CASPAR criteria.[1] Ultrasound studies have demonstrated that many psoriasis patients without clinical PsA already exhibit subclinical entheseal or synovial abnormalities, supporting the concept of a preclinical disease stage.[2]

Several risk factors for PsA have been identified, particularly obesity and physical trauma.[3] Large epidemiologic studies have shown that higher BMI is associated with an increased risk of developing PsA, whereas long-term weight reduction may reduce this risk.[4] Obesity may contribute through metabolic dysfunction, insulin resistance, adipose tissue inflammation, and amplification of the IL-23/IL-17 inflammatory axis.[5]

The potential role of GLP-1 receptor agonists was also discussed. Recent real-world analyses suggest that these agents may reduce the risk of incident PsA, while weight-loss interventions in established PsA have been associated with significant improvements in disease activity in a dose-dependent manner.[6,7] However, definitive evidence supporting PsA prevention is still lacking.

The presentation further reviewed emerging evidence suggesting that biologic therapies, particularly IL-23 and IL-12/23 inhibitors, may delay or reduce the development of PsA compared with TNF inhibitors or non-biologic therapies. Several observational studies and recent data presented at the 2026 AAD Annual Meeting have consistently suggested an association between IL-23-targeted therapies and a lower risk of incident PsA.[8-12] Nevertheless, limitations inherent to electronic health record studies, including potential misclassification and residual confounding, preclude definitive causal conclusions.[13]

Finally, ongoing prospective studies such as PAMPA aim to evaluate whether early intervention in high-risk psoriasis patients can prevent progression to PsA. However, the relatively short follow-up may limit its ability to fully assess progression to clinically manifest PsA.[14] Emerging mechanistic studies have also identified the CD200–CD200R pathway as a potential regulator of skin-to-joint transition, highlighting a novel target for immune interception strategies.[15] Although current evidence is promising, no intervention has yet definitively demonstrated prevention of PsA, making disease interception one of the most important future directions in psoriasis research.

References:

1. Psoriatic Arthritis: Development, Detection and Prevention: A Scoping Review. Kimak A, Robak E, Makaowska J, et al. *J Clin Med.* 2023; 12(11):3850. doi: 10.3390/jcm12113850.
2. High Prevalence of Ultrasonographic Synovitis and Enthesopathy in Patients with Psoriasis without Psoriatic Arthritis: A Prospective Case-Control Study. Naredo E, Möller I, de Miguel E, et al. *Rheumatology (Oxford)*. 2011;50(10):1838-48. doi: 10.1093/rheumatology/ker078.
3. Modifiable Lifestyle and Environmental Factors Associated with Onset of Psoriatic Arthritis in Patients with Psoriasis: A Systematic Review and Meta-analysis of Observational Studies. Xie W, Huang H, Deng X, et al. *J Am Acad Dermatol.* 2021;84(3):701-711. doi: 10: 10.1016/j.jaad.2020.08.060.
4. Modifiable Risk Factors and the Development of Psoriatic Arthritis in People with Psoriasis. Green A, Shaddick G, Charlton R, et al. *Br J Dermatol.* 2020;182(3):714-720. doi: 10.1111/bjd.18227.
5. Glucagon-like Peptide-1 Receptor Agonists in Psoriasis and Psoriatic Arthritis: Emerging Evidence and Future Research Opportunities. Ciancio G, Maranini B, Sandri G, et al. *Front Immunol.* 2026;17:1744308. doi: 10.3389/fimmu.2026.1744308.
6. Risk of Developing Psoriatic Arthritis in Patients with Psoriasis Initiating Treatment with Glucagon-like Peptide-1 Receptor Agonists: A Population Cohort Study from the TriNetX Collaborative Network. Feng AS, Zhang J, Meydan Y, et al. *J Am Acad Dermatol.* 2026:S0190-9622(26)00399-3. doi: 10.1016/j.jaad.2026.03.023.
7. Weight Loss Improves Disease Activity in Patients with Psoriatic Arthritis and Obesity: An Interventional Study. Klingberg E, Bilberg A, Björkman, et al. *Arthritis Res Ther.* 2019;21(1):17. doi: 10.1186/s13075-019-1810-5.
8. Risk of Developing Psoriatic Arthritis in Patients with Psoriasis: Initiating Treatment with Different Biologic Classes. Gisondi P, Bellinato F, Galeone C, et al. *Ann Rheum Dis*. 2025;84(3):435-441. doi: 10.1016/j.ard.2025.01.006.
9. Association Between Biological Immunotherapy for Psoriasis and Time to Incident Inflammatory Arthritis: A Retrospective Cohort Study. Singla S, Putman M, Liew J, et al. *Lancet Rheumatol.* 2023;5(4):e200-e207. doi: 10.1016/S2665-9913(23)00034-6.
10. Treating the Skin with Biologics in Patients with Psoriasis Decreases the Incidence of Psoriatic Arthritis. Acosta Felquer ML, LoGiudice L, Galimberti ML, et al. *Ann Rheum Dis.* 2022;81(1):74-79. doi: 10.1136/annrheumdis-2021-220865.
11. Risk of Developing Inflammatory Arthritis in Patients with Psoriasis Initiating Treatment with Biologics: A Population-Based Analysis. Strober B, Soliman AM, Li C, et al. *J Am Acad Dermatol.* 2024;91(6):1143-1149. doi: 10.1016/j.jaad.2024.06.106.
12. Schmidt M, et al. AAD Annual Meeting 2026.
13. Association between biological immunotherapy for psoriasis and time to incident inflammatory arthritis: limitations and opportunities. Koehm M, Behrens F. *RMD Open.* 2023;9(3):e003166. doi: 10.1136/rmdopen-2023-003166.
14. Efficacy of Guselkumab, a Selective IL-23 Inhibitor, in Preventing Arthritis in a Multicentre Psoriasis At-Risk Cohort (PAMPA): Protocol of a Randomised, Double-Blind, Placebo Controlled Multicentre Trial. Haberman RH, MacFarlane KA, Catron S, et al. *BMJ Open.* 2022;12(12):e063650. doi: 10.1136/bmjopen-2022-063650.

15. CD200+ fibroblasts form a pro-resolving mesenchymal network in arthritis. Rauber S, Mohammadian H, Schmidkonz C, et al. *Nat Immunol.* 2024;25(4):682-692. doi: 10.1038/s41590-024-01774-4.

Implications of Controlling Psoriasis Inflammation: From Molecular Profiling to Comorbidities

Alex Tsoi, MS, PhD, IPC Councilor
University of Michigan, Ann Arbor, Michigan, United States

This presentation explored how molecular profiling can be used to define and measure "controlled inflammation" in psoriasis, and how genetic and epigenetic factors may influence treatment response and comorbidity risk.

The first section focused on transcriptomic changes during treatment. Previous studies using anti-TNF therapy demonstrated that lesional skin progressively shifts toward a non-lesional molecular profile, reflecting normalization of disease-associated gene expression programs.[1] Building on this concept, data from the IXORA-R study comparing ixekizumab and guselkumab were presented. By integrating RNA sequencing data from psoriasis lesions with cytokine-induced keratinocyte signatures, rapid downregulation of IL-17-, TNF-, and IL-36-driven inflammatory programs was observed following treatment. Normalization began as early as week 1 with ixekizumab and correlated with clinical improvement, supporting the concept that molecular normalization may serve as a surrogate measure of inflammation control.[2]

The second section addressed why some inflammatory pathways may be more difficult to normalize. Single-cell multiomic analyses of lesional, non-lesional, and resolved psoriatic skin demonstrated that gene expression abnormalities largely normalize after treatment, whereas epigenetic alterations persist to a greater extent. Many psoriasis-associated GWAS loci overlapped with differentially accessible chromatin regions, particularly in lesional skin, suggesting that genetic susceptibility may be mediated, at least in part, through disease-relevant epigenetic programs. Furthermore, analyses of allele-specific chromatin accessibility revealed context-dependent effects, especially in activated T cells, highlighting how genetic variation can influence inflammatory responses and treatment outcomes. An example involving *KLK7* illustrated how psoriasis-associated genetic variants may also contribute to differential responses to anti-TNF therapy.[3]

The final section focused on the systemic implications of psoriasis. Genetic studies have identified shared susceptibility loci and potential causal associations between psoriasis and other immune-mediated diseases, including type 2 diabetes and multiple sclerosis. Genetic studies using Mendelian randomization suggest that psoriasis may causally contribute to some comorbidities, while accumulating evidence indicates that effective control of systemic inflammation may be associated with a reduction in their incidence.[4,5] Overall, the presentation highlighted that controlled psoriasis inflammation can be measured molecularly, is influenced by genetic and epigenetic factors, and may have benefits extending beyond the skin.

References:

1. Cytokine Responses in Nonlesional Psoriatic Skin as a Clinical Predictor of Anti-TNF Agents. Tsoi LC, Patrick MT, Shuai S, et al. *J Allergy Clin Immunol.* 2022;149(2):640-649.e5. doi: 10.1016/j.jaci.2021.07.024.

2. Comparative Molecular Analysis of IL-17A and IL-23 Pathway Inhibition in Moderate-to-Severe Psoriasis: 4-Week Results from IXORA-R. Garcet S, Tsoi LC, Hur H, et al. *J Invest Dermatol.* 2026;146(3):669-677.e6. doi: 10.1016/j.jid.2025.06.1598.
3. Retrospective Pharmacogenetic Study of Psoriasis Highlights the Role of KLK7 in Tumour Necrosis Factor Signalling. Zhang H, Patrick MT, Tejasvi T, et al. *Br J Dermatol*. 2023;190(1):70-79. doi: 10.1093/bjd/ljad332.
4. Causal Relationship and Shared Genetic Loci between Psoriasis and Type 2 Diabetes through Trans-Disease Meta-Analysis. Patrick MT, Stuart PE, Zhang H, et al. *J Invest Dermatol.* 2021;141(6):1493-1502. doi: 10.1016/j.jid.2020.11.025.
5. Shared Genetic Risk Factors for Multiple Sclerosis/Psoriasis Suggest Involvement of Interleukin-17 and Janus Kinase-Signal Transducers and Activators of Transcription Signaling. Patrick MT, Nair RP, He K, et al. *Ann Neurol.* 2023;94(2):384-397. doi: 10.1002/ana.26672.

Oral Versus Biologics – Targeted or Broader Approaches?

Christopher Bunick, MD, PhD, IPC Councilor
Yale University, New Haven, Connecticut, United States

Dr. Christopher Bunick approached psoriasis treatment through structural biology, demonstrating how molecular architecture determines clinical efficacy. Using the principle that "structure dictates function", he described psoriasis as a disease largely sustained by the IL-23/IL-17 axis: myeloid antigen-presenting cells produce IL-23, which promotes TH17-derived IL-17A, IL-17F, and TNF-α, leading to keratinocyte activation, proliferation, and inflammation. He also emphasized that type I interferons from plasmacytoid dendritic cells may help initiate psoriasis before the chronic IL-23/IL-17 circuit becomes established, suggesting that durable control may require attention to earlier inflammatory triggers.[1]

Dr. Bunick organized the treatment landscape around extracellular pathway targets, including IL-23, IL-17A/F, TNF-α, and their receptors, as well as intracellular pathway targets such as TYK2, JAK1, and PDE4. Using IL-23 inhibitors as a model, he showed that drugs within the same class do not bind identically: ustekinumab targets p40, whereas p19-specific agents such as guselkumab, risankizumab, and tildrakizumab bind distinct regions of p19.[2] This structural diversity helps explain why both interclass and intraclass switching may be useful. Across comparative analyses, bimekizumab and risankizumab ranked among the strongest performers for PASI100 responses, and Dr. Bunick emphasized that epitope surface area correlates with clinical response, alongside binding kinetics such as slower dissociation from the target.[2]

He also highlighted the expanding role of oral therapies. Icotrokinra, an oral cyclic peptide targeting the IL-23 receptor, produced responses approaching some biologics, while next-generation TYK2 inhibitors such as zasocitinib and envudeucitinib showed improved selectivity, pharmacokinetics, and efficacy. In contrast, apremilast's more modest performance was linked to structural constraints that weaken its ability to mimic cyclic AMP and inhibit PDE4 effectively. Overall, Dr. Bunick showed that drug chemistry and structure are clinically consequential: they help explain why treatments excel, support switching within and across classes, and increasingly inform clinicians' decisions between oral agents and biologics.

References:

1. Psoriasis: From Pathogenesis to Targeted Therapies. Conrad C, Gilliet M. *Clin Rev Allergy Immunol.* 2018 Feb;54(1):102-113. doi: 10.1007/s12016-018-8668-1.
2. Structural Basis for p19 Targeting by Anti-IL-23 Biologics: Correlations with Short- and Long-Term Efficacy in Psoriasis. Daniele SG, Eldirany SA, Damiani G, et al. *JID Innov.* 2024 Jan 20;4(2):100261. doi: 10.1016/j.xjidi.2024.100261.

Why Treatment Fails: Interferons, Immune Rewiring, and Biological Escape Mechanisms

Curdin Conrad, MD, IPC Board Member
University Hospital of Lausanne, Lausanne, Switzerland

Dr. Curdin Conrad explained treatment failure in psoriasis as a consequence of immune circuit complexity, in which nonresponse may reflect a mismatch between therapy and the patient's active inflammatory pathways. Molecular profiling helps refine this assessment by distinguishing inflammatory diseases that may appear similar clinically but differ immunologically. A TH17/IL-23 profile characterizes plaque psoriasis, whereas atopic dermatitis may show a TH2-dominant or mixed pattern.[1] Together, these profiles illustrate why therapy should be guided by immune biology rather than clinical morphology.

Dr. Conrad described three inflammatory patterns in psoriasis: chronic plaque psoriasis driven by IL-23/IL-17 signaling, acute forms marked by type I interferon, and pustular phenotypes characterized by neutrophilic inflammation.[2-3] In generalized pustular psoriasis, neutrophilic activation becomes prominent; in palmoplantar pustular psoriasis (PPPP), TH17 activity overlaps with neutrophilic inflammation, incomplete TH1 responses, and type I interferon (Conrad C, Di Domizio J, Gilliet M. Unpublished data, 2026). This explains why apremilast, despite limited efficacy in plaque psoriasis, remains useful in PPPP: its broader effects span T-cell activity, neutrophil function, and interferon production. The same principle applies to primary failure: when several pathways remain active, blocking one may not be sufficient to control the disease.

He also discussed secondary failure through anti-drug antibodies with TNF-α inhibitors. Neutralizing antibodies block the drug's binding site, while non-neutralizing antibodies may form immune complexes that increase clearance and lower trough levels. With adalimumab, TNF-α blockade can increase type I interferon activity, activating B cells and promoting antibody formation. Together, these mechanisms lower effective drug exposure and contribute to loss of clinical response.

Dr. Conrad emphasized molecular endotyping as a bridge between immune profiling and therapeutic decision-making. His central message was to align treatment with the pathways active in each patient: as targeted as possible, but as broad as needed—focused when one axis dominates, and broader when interferon, neutrophilic, or antibody-mediated escape keeps inflammation active.

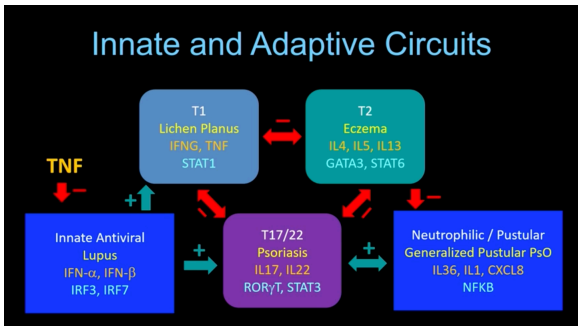
References:

1. 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. doi: 10.1038/s41467-024-54559-6.
2. Psoriasis: From Pathogenesis to Targeted Therapies. Conrad C, Gilliet M. *Clin Rev Allergy Immunol.* 2018 Feb;54(1):102-113. doi: 10.1007/s12016-018-8668-1.
3. Psoriasis: Classical vs. Paradoxical. The Yin-Yang of TNF and Type I Interferon. Mylonas A, Conrad C. *Front Immunol.* 2018 Nov 28;9:2746. doi: 10.3389/fimmu.2018.02746.

Clinical Escape in Psoriasis: Paradoxical Reactions and Flip-Flop Phenomena

Wilson Liao, MD, IPC Councilor
University of California, San Francisco, California, United States

Dr. Wilson Liao defined paradoxical reactions as immune-mediated cutaneous eruptions induced or worsened by therapies expected to treat the same or a related inflammatory phenotype. Rather than reflecting a simple drug allergy, toxicity, or nonspecific flare, these reactions usually arise from cytokine-network imbalance. He framed them as "flip-flop" phenomena among mutually regulated immune pathways, including TH1, TH2, TH17/22, and innate antiviral or neutrophilic circuits. When one pathway is blocked, inhibitory control over another may be removed, allowing a different inflammatory response to emerge.



One illustrative example is paradoxical psoriasis during TNF-α inhibitor therapy. In affected patients, lesions may show IL-17A/F levels comparable to those in classic psoriasis, but higher type I interferon activity from plasmacytoid dendritic cells.[1] TNF-α normally helps plasmacytoid dendritic cells mature beyond an interferon-producing state. When TNF-α is blocked, immature interferon-producing cells persist, amplifying type I interferon signaling and feeding the TH17 cascade that drives psoriasiform inflammation. This model also helps explain other TNF-α inhibitor-associated eruptions, including paradoxical lichen planus.

Dr. Liao also discussed paradoxical psoriasis with IL-4Rα blockade, such as dupilumab, which occurs in a small subset of patients with atopic dermatitis and often shows mixed psoriasiform and neutrophilic features, including IL-36 and IL-1 activity.[2] Conversely, paradoxical eczema may occur during psoriasis biologic therapy, especially with IL-17 inhibitors, with risk influenced by history of atopic dermatitis and genetic susceptibility.[3] Overall, paradoxical reactions occur in approximately 1–3% of biologic-treated patients and reflect antagonism among adaptive and innate immune circuits. Mild cases may be managed with adjunctive topical or oral systemic therapies, whereas severe reactions may require switching to broader agents such as JAK inhibitors.[4] Dr. Liao concluded by identifying polygenic risk scores, spatial transcriptomics, and immune profiling as tools to anticipate paradoxical reactions, identify susceptible patients, and tailor management before clinical escape occurs.

References:

1. TNF Blockade Induces a Dysregulated Type I Interferon Response without Autoimmunity in Paradoxical Psoriasis. Conrad C, Di Domizio J, Mylonas A, et al. *Nat Commun.* 2018 Jan 2;9(1):25. doi: 10.1038/s41467-017-02466-4.
2. IL-4/IL-13 Inhibitors for Atopic Dermatitis Induce Psoriatic Rash Transcriptionally Close to Pustular Psoriasis. Grolleau C, Calugareanu A, Demouche S, et al. *J Invest Dermatol.* 2023 May;143(5):711-721.e7. doi: 10.1016/j.jid.2022.10.015.
3. Risk of Paradoxical Eczema in Patients Receiving Biologics for Psoriasis. Al-Janabi A, Alabas OA, Yiu ZZN, et al. *JAMA Dermatol.* 2024 Jan 1;160(1):71-79. doi: 10.1001/jamadermatol.2023.4846.
4. Dupilumab-Associated Psoriasis and Psoriasiform Manifestations: A Scoping Review. Su Z, Zeng YP. *Dermatology.* 2023;239(4):646-657. doi: 10.1159/000530608.