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A Report from the 2026 European Academy of Dermatology and Venereology (EADV) Congress

SUMMARIZING SESSIONS
WITH A FOCUS ON
PSORIASIS

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INTRODUCTION

The 35th European Academy of Dermatology and Venereology (EADV) Congress took place at the Austria Center Vienna in Vienna, Austria, from Wednesday, Sept. 30, to Saturday, Oct. 3, 2026. The congress is the largest dermatology meeting in Europe, with 20,300 attendees from 130+ countries and more than 170 scientific sessions covering late-breaking research, clinical updates, and emerging therapies. The program included the IPC Symposium: At Last! Personalized Medicine for Psoriasis, chaired by Catherine Smith, MD, IPC Board Member, and Alexander Navarini, MD, PhD, IPC Councilor.

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Catherine Smith, MD, IPC Board Member, United Kingdom, and Alexander Navarini, MD, PhD, IPC Councilor, Switzerland, chaired the symposium. Program faculty included Curdin Conrad, MD, IPC Board Member, Switzerland; Satveer Mahil, PhD, FRCP, IPC Councilor, United Kingdom; and Tamar Nijsten, PhD, Netherlands. Peter van de Kerkhof, MD, PhD, IPC Chief Medical Officer, Netherlands, and Dina ElDisouki, MD, IPC Jr. Councilor, Egypt, joined as panelists, with Professor Smith moderating the closing panel discussion.

This session traced personalized psoriasis care across three connected dimensions: molecular profiling to characterize disease, precision dosing to tailor treatment exposure, and digital technologies to extend assessment and access.

Approaches to Personalized Medicine: From the Clinical to the Molecular

Curdin Conrad, MD, IPC Board Member

University Hospital of Lausanne, Lausanne, Switzerland

Dr. Curdin Conrad examined how integrating clinical assessment with molecular profiling could advance personalized psoriasis care. Treatment selection must balance efficacy, safety, tolerability, convenience, and persistence. It must also account for disease severity, phenotype, comorbidities, and patient expectations. Although interleukin (IL)-17 and IL-23 inhibitors broaden phenotype- and comorbidity-informed treatment selection, no therapy is universally effective. Rapid clearance, sustained control, psoriatic arthritis, body weight, inflammatory bowel disease, and other factors influence therapeutic choices.

Molecular profiling of skin biopsies can characterize innate and adaptive immune signatures.^{1,2} These findings may prompt diagnostic reassessment when inflammatory dermatoses overlap clinically or respond unexpectedly. Targeted gene-expression profiling platforms, such as NanoString, may identify discordance among the presumed diagnosis, the dominant inflammatory pathway, and the selected therapy. A type 2 signature in patients unresponsive to IL-17 or IL-23 blockade, for example, may prompt reassessment for atopic dermatitis, another type 2 inflammatory disorder, or an overlapping phenotype.

The presentation emphasized the molecular diversity of psoriasis. It described chronic inflammation involving IL-23 and IL-17, acute inflammation associated with type I interferon, and pustular inflammation involving the IL-36 and IL-1 axis. These are overlapping immunologic patterns rather than mutually exclusive clinical categories. Their overlap may help explain treatment resistance and variable responses across clinical variants, reinforcing the value of integrating clinical phenotype with molecular subtype.

Emerging artificial intelligence approaches may infer molecular signatures from digitized histopathology or other tissue-derived data, potentially improving accessibility. Integrated profiling may eventually help predict treatment response and evaluate nonresponders. It could also identify therapeutic targets, reveal opportunities for drug repurposing, and inform selection between cytokine-specific biologics and synthetic agents targeting multiple pathways. However, clinical

validation and evidence of practical utility remain essential before molecular guidance enters routine care. This approach represents a potential transition from stratified treatment selection toward personalized management of psoriasis.

Together, these molecular approaches establish a rationale for moving from disease classification to more individualized treatment selection.

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Therapeutic Drug Monitoring and Dose Optimization: Clinical and Practical Considerations

Satveer Mahil, PhD, FRCP, IPC Councilor

St John's Institute of Dermatology, London, United Kingdom

Fixed biologic dosing overlooks variability in drug exposure and response among patients with psoriasis. In her lecture, Dr. Satveer Mahil examined therapeutic drug monitoring (TDM) and dose optimization to restore efficacy and reduce treatment burden. A case of secondary adalimumab failure, accompanied by low drug concentrations and the absence of detectable anti-drug antibodies, illustrated the rationale for dose escalation after confirming adherence and interpreting the result in its clinical and assay context.

TDM uses serum concentrations to inform treatment adjustments. One multicenter observational study identified a minimally effective adalimumab concentration of 3.2 µg/mL and a response plateau near 7.0 µg/mL for a 75% improvement from baseline in the Psoriasis Area and Severity Index (PASI 75).¹ Increasing concentrations beyond this plateau may offer little additional benefit. Clinical implementation requires assays that are validated against international standards, accessible, have rapid turnaround times, and provide interpretable results.

Model-informed precision dosing (MIPD) uses Bayesian forecasting to combine population pharmacokinetic and pharmacodynamic models with individual patient characteristics and measured drug concentrations. It then predicts personalized doses and treatment intervals. This approach extends conventional TDM by supporting individualized treatment decisions rather than relying solely on population therapeutic ranges.

In silico modeling of 1,000 risankizumab-treated patients, based on the BSTOP cohort, suggested that 69% could be overtreated with standard dosing. Separately, the BeNeBio randomized trial found that stepwise dose reduction of interleukin (IL)-17 and IL-23 inhibitors was noninferior to standard dosing for the incidence of persistent flares at 18 months in patients with stable, low disease activity.²

The 12-month PLAN-psoriasis feasibility trial evaluates precision dosing, supported by patient-reported outcomes collected through the mySkin remote-monitoring system and self-collected blood samples.³ Interim data presented at the symposium indicated high clinician usability and patient acceptability; 49% received lower-than-standard dosing, with a mean interval of 16 weeks. Further simulations presented at the symposium projected 23% less drug exposure and 18% fewer injections.

Precision dosing may sustain disease control while reducing injections, costs, and cumulative treatment burden. This treatment-level personalization complements molecular stratification and leads naturally to digital tools that can support monitoring and implementation beyond the clinic.

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Digital Technologies: What's Out There for Psoriasis?

Alexander Navarini, MD, PhD, IPC Councilor | University Hospital of Basel, Basel, Switzerland

Tamar Nijsten, PhD | Erasmus MC, Rotterdam, Netherlands

The session examined digital technologies in psoriasis by asking, “How can new signals improve clinical decision-making and the patient journey?” Emerging approaches include molecular sampling, optical imaging, artificial intelligence (AI), and remote monitoring, each offering opportunities alongside implementation challenges.

Skin wash and tape stripping can detect cytokines, including interleukin (IL)-36γ, thereby potentially supporting the assessment of inflammatory pathways. However, their ability to resolve diagnostic uncertainty requires further evaluation. Line-field confocal optical coherence tomography (LC-OCT) visualizes psoriasis features that correlate with histology.^{1,2} It may eventually help avoid selected biopsies if diagnostic-performance and clinical-utility studies support its use. Histological correlation alone does not establish that imaging can replace biopsy.

AI-assisted assessment includes diagnostic support, image-based severity scoring, and multimodal models integrating clinical and dermoscopic images. These tools may improve measurement consistency and reduce interobserver variability. Nevertheless, external validation across centers, skin tones, and diverse populations remains essential. Better measurement does not establish clinical benefit from AI-selected treatment.

Teledermatology and remote monitoring offer potential improvements in access, continuity, and patient experience. Their evaluation should incorporate quality-of-life outcomes, patient preferences, and effects on clinical decisions.

Implementation in low-income countries faces several barriers. These include limited access to specialists, unreliable internet connectivity, scarce devices and imaging equipment, high costs, and limited digital literacy. Without accessible infrastructure and referral pathways, digital innovation may widen existing inequalities.

A 2026 global cross-sectional study, reported in *JAMA Dermatology* and using data from the Skin Observatory, found that only 17% of 158 countries met the estimated sufficient dermatologist workforce threshold.³ Ultimately, digital tools should complement clinical care through validated, affordable, and locally feasible approaches. Their success should be measured by meaningful patient outcomes and equitable access, not technical performance alone.

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Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA): Advancing Personalized Care in Psoriatic Disease: From Early Detection to Precision Therapeutics

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Alice Gottlieb, MD, PhD, IPC Councilor | UT Southwestern Medical Center, Dallas, Texas, United States

Leandro Leite, MD, MSc, IPC Councilor | Hospital de Clínicas de Porto Alegre, Porto Alegre, Brazil

Lone Skov, MD, PhD, IPC Board Member | Herlev and Gentofte Hospital, University of Copenhagen, Hellerup, Denmark

The session opened with Dr. Leandro Leite examining the concept of skin inflammation driving joint inflammation: a higher Psoriasis Area and Severity Index (PASI) score is associated with a higher risk of psoriatic arthritis (PsA), and early initiation of biologics for psoriasis may reduce this risk.^{1,2} Guselkumab slowed radiographic progression of PsA at week 24 and, in axial disease, showed clinical and magnetic resonance imaging (MRI) benefits at week 24.^{3,4} In a head-to-head study, bimekizumab was superior to risankizumab in American College of Rheumatology 50% response (ACR50) at weeks 16 and 24.⁵ Sonelokimab is in development for PsA and palmoplantar pustulosis (PPP).^{6,7} Combining tirzepatide with ixekizumab was superior to ixekizumab alone in patients with overweight or obesity.⁸ Next, Dr. April Armstrong discussed personalized medicine and the balance between efficacy, safety, and convenience. She described shared decision-making and a skin patch test designed to predict therapeutic response.^{9,10}

Then, Dr. Alice Gottlieb reviewed tools for measuring musculoskeletal symptoms of PsA independently of skin disease, including PEST, PsAID-12, and the IDEOM Musculoskeletal Questionnaire (IDEOM-MSK-Q).¹¹ Professor Lone Skov presented the GRAPPA expert consensus on difficult-to-treat PsA and commented on the GRAPPA-IPC joint consensus definition.¹² After that, Professor Kilian Eyerich discussed immune models guiding the treatment decision and different phenotypes. Psoriasiform diseases are stratified into group III, which overlaps with eczematous diseases (group II) and neutrophilic diseases.¹³ He presented examples of psoriasiform phenotypes where molecular and transcriptome-based stratification clarified distinct inflammatory pathways. Treatment was selected accordingly: nummular plaques responded to dupilumab, and a pustular pattern responded to an interleukin (IL)-17 inhibitor.¹⁴ His central message was that clinical patterns alone cannot reliably distinguish good responders from nonresponders to different therapies.

Closing the session, Ahmet Uğur Atılan discussed therapeutic combinations and reasons behind nonresponse. In the AFFINITY study, guselkumab plus golimumab was superior to guselkumab alone in PsA at week 24, with greater responses in patients with higher C-reactive protein (CRP) levels.¹⁵ Combinations under investigation include a tumor necrosis factor- α (TNF- α) inhibitor plus a tyrosine kinase 2 (TYK2) inhibitor (TNF- α blockade can unleash type I interferon, which TYK2 inhibition counters);^{16,17} IL-23p19 plus IL-1 blockade; and biologics and incretins in patients with obesity.^{8,18}

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Understanding Psoriasis (Updates)

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

Satveer Mahil, PhD, FRCP, IPC Councilor | St John's Institute of Dermatology, London, United Kingdom

Four lectures examined psoriasis in relation to obesity, pustular disease, special sites, and monogenic mechanisms.

Dr. José-Manuel Carrascosa presented obesity as a modifiable comorbidity affecting about 30% of patients. Mendelian randomization supports a causal role for higher body mass index, and shared Th17 pathways link psoriatic skin and adipose tissue.^{1,2} Obesity and diabetes independently predict poorer biologic response.³ Incretin-based therapies are emerging adjuncts:⁴ in TOGETHER-PsO, 27.1% of patients receiving ixekizumab plus tirzepatide achieved a 100% improvement in the Psoriasis Area and Severity Index (PASI 100) and $\geq 10\%$ weight loss at week 36, versus 5.8% with ixekizumab alone.⁵

Dr. Johann Gudjonsson described pustular psoriasis as driven by interleukin (IL)-36. Variants in IL36RN, CARD14, AP1S3, SERPINA3, and MPO converge on excessive IL-36 signaling, while neutrophil–keratinocyte loops amplify inflammation.^{6,7} Spesolimab cleared pustules at week 1 in 54% of patients with generalized pustular psoriasis versus 6% with placebo,⁸ and high-dose subcutaneous maintenance reduced 48-week flares to 10% versus 52%.⁹

Dr. Satveer Mahil highlighted that scalp, face, genital, flexural, palmoplantar, and nail psoriasis affect up to 90% of patients over their lifetime and impose a disproportionate burden despite limited body surface area. British guidance supports the use of biologics when other eligibility criteria are met for severe localized disease-causing functional impairment or distress.¹⁰ Evidence syntheses favored ixekizumab and brodalumab for scalp,¹¹ guselkumab for palmoplantar,¹² ixekizumab and apremilast for genital,¹³ and ixekizumab for nail psoriasis.¹⁴

Professor Hervé Bachelez showed that monogenic disorders reveal distinct mechanisms. CARD14 gain-of-function mutations activate the IL-23/IL-17 axis,¹⁵ whereas gain-of-function variants in IFIH1 and DDX58 link psoriasis with type I interferon signaling.^{16,17} Heterozygous ADAR1 loss-of-function variants that impair RNA editing define an interferon-dependent subtype; experimental reversal with upadacitinib and deucravacitinib supports investigating JAK1/TYK2 inhibition.¹⁸

Together, these insights support individualized care integrating metabolic health, clinical phenotype, anatomical involvement, and molecular mechanisms.

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Treating Psoriasis (Updates)

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Jo Lambert, MD, PhD, IPC Councilor | Ghent University Hospital, Ghent, Belgium

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This session reviewed current psoriasis treatment, from topical, biologic, and oral therapies to the emerging concept of disease modification. Professor Jo Lambert noted that topical therapy is still used in 80% of patients, either as monotherapy or in combination with systemic therapies or phototherapy, as needed, and that its use does not indicate mild disease.¹ The question is not whether topicals matter, but how to choose and optimize them and when to escalate. Three advances have changed the conversation around topicals: better vehicles (aerosol foams, supersaturation, and simpler application), precision signaling (AhR agonism with tapinarof, PDE4 inhibition with roflumilast, and JAK inhibition with delgocitinib), and better delivery (nanocarriers, microneedles, and hybrid systems).²⁻⁵

Turning to biologics, Dr. Julia-Tatjana Maul outlined today's treatment goals: an absolute Psoriasis Area and Severity Index (PASI) score ≤ 2 , a Dermatology Life Quality Index (DLQI) score of 0–1, and a Physician Global Assessment (PGA) score of 0–1.⁶ Tumor necrosis factor- α (TNF- α) inhibitors were the first biologics on the market and remain the first-choice biologic in many countries. They are highly effective in psoriatic arthritis, high-quality biosimilars are available to reduce costs, and certolizumab pegol is a good option during pregnancy.⁷⁻⁹ Interleukin (IL)-23 inhibitors have shown good efficacy and safety, as demonstrated in the VOYAGE trial and the UltIMMa study.^{8,10} IL-17 inhibitors offer good efficacy, rapid onset of response, and a good safety profile and are well-suited to patients with psoriatic arthritis; however, they can cause candidiasis and should be avoided in patients with inflammatory bowel disease.^{8,11-13} For patients who have maintained a PASI score of 100 for one year, interval extension can be considered.

Dr. Curdin Conrad then reviewed oral therapies, noting that conventional oral systemic agents remain the first-line systemic treatment option for most patients.^{6,14} New oral molecules in development include TYK2 inhibitors, PDE4 inhibitors, an IL-23R inhibitor, IL-17 inhibitors, an S1PR1 agonist, and an A3AR agonist. Icotrokinra is a first-in-class, targeted oral peptide that selectively binds the IL-23R with high affinity. In phase 3 trials, it showed a slower onset of action than biologics but similar efficacy at week 24, with two-thirds of patients achieving PASI 90 and 40% nearly clear.¹⁵ Oral TYK2 inhibitors block both the IFN- α and Th17 pathways. Approximately 40–50% of patients on deucravacitinib achieve PASI 90, and ~20% achieve PASI 100.^{16,17} In comparison, about two-thirds of patients on zasocitinib achieve PASI 90 and ~40% achieve PASI 100, albeit with a slower onset of action than biologics.¹⁸

Finally, Professor Kilian Eyerich addressed disease modification, whose definition is still debated because it cannot be predicted or measured.¹⁹ Insights from clinical trials and real-world evidence indicate a window of opportunity, as response depends on disease duration.²⁰⁻²² After withdrawal of effective biologic treatment (e.g., an IL-23 inhibitor),²³ several patients maintain low or no disease activity for more than a year, and early, effective treatment may reduce the

development of comorbidities.^{24,25} Potential mechanisms of disease modification include epigenetic resolution (STEP-IN)²⁶ and/or normalization of tissue-resident memory cells (GUIDE).²⁷

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Free Communication 5: Psoriasis

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Opening the session, Dr. Yuling Shi presented a new bispecific interleukin (IL)-23p19/IL-36R antibody that provides ultra-long-lasting activity for over a year with a single dose. At week 16, the 600 mg single dose produced Psoriasis Area and Severity Index (PASI) 90 responses in 90% of patients and PASI 100 responses in 65% of patients. Of the patients enrolled, 31% had prior exposure to biologics.^{1,2} Yulin Zou spoke about TDRKH-mediated pro-inflammatory M1 macrophage polarization in psoriasis, thereby identifying TDRKH as an important gene. Anastasia Fleyder presented an approach to reducing the diagnostic delay in psoriatic arthritis using the Electronic German Psoriasis-Arthritis Diagnostic questionnaire (eGEPARD) in clinical practice.³⁻⁵

After that, Luciano Ibba presented a multicenter study, highlighting an early PASI response (week 32 outperformed week 16) predicting long-term 1- and 3-year complete skin clearance with risankizumab.⁶⁻⁸ In the next lecture, Dr. April Armstrong discussed the durable efficacy of zasocitinib 30 mg once daily, a highly selective TYK2 inhibitor, with 90% of patients who used the medication for 40–60 weeks maintaining clear skin for more than five months after discontinuing use.^{8,9} Next, Professor Melinda Gooderham discussed early responses, with 33.4% of patients achieving PASI 100 at week 16 and 42.3% at week 24. Moving on, Dr. Shi presented a subgroup analysis of the efficacy of picankibart in patients with different BMIs and body weights, demonstrating no statistically significant differences in efficacy between groups at the same dose.¹⁰⁻¹⁴

Khaled Ezzedine then reported upadacitinib outcomes in nonsegmental vitiligo, with more upadacitinib-treated patients reporting reduced disease noticeability, improved health-related quality of life, better color matching, perceived severity reduction, and improved sun sensitivity at weeks 12, 24, and 48.¹⁵⁻¹⁷ Finally, Sana Gupta presented an indirect comparison of icotrokinra versus injectable IL-23 inhibitors, showing icotrokinra underperformed compared to risankizumab and guselkumab in IGA 0/1 and PASI 75/90/100 at weeks 12 or 16, but performed similarly to tildrakizumab for PASI 75.

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Psoriasis (Training and Education)

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Diamant Thaçi, MD, PhD, IPC Councilor | University of Lübeck, Comprehensive Center for Inflammation Medicine, Lübeck, Germany

This session covered the essentials of psoriasis care, from clinical subtypes and severity assessment to comorbidities and treatment. Valeria Mateeva emphasized that psoriasis has a highly heterogeneous clinical presentation rather than a single phenotype. Nonpustular forms include plaque (types I and II), guttate, inverse/flexural, erythrodermic, and linear psoriasis, while pustular forms include generalized, annular, impetigo herpetiformis, palmoplantar, and acrodermatitis continua of Hallopeau.¹⁻³ Plaque-type psoriasis is the prototype, whereas generalized pustular psoriasis is a separate disease entity.

Dr. Diamant Thaçi then addressed the evaluation of disease severity. Historically, psoriasis severity was classified as mild, moderate, or severe based solely on the percentage of body surface area covered by lesions.⁴ Today, severity is understood to be multidimensional, encompassing biological severity (Psoriasis Area and Severity Index [PASI], Physician Global Assessment [PGA], nail involvement [NAPSI], and scalp psoriasis [PSSI]), health-related quality of life (e.g., DLQI), responsiveness to treatment, and the presence of comorbidities.⁵

Turning to comorbidities, Dr. Álvaro González-Cantero explained that Mendelian randomization studies suggest genetic predictors of cardiovascular disease (CVD) are associated with increased psoriasis risk, with no reciprocal effect or association with other immune-mediated inflammatory diseases.⁶ Patients with psoriasis and nonalcoholic fatty liver disease have a higher prevalence of subclinical atherosclerosis, and those with elevated hepatic inflammation have more CVD risk factors and a greater coronary atherosclerosis burden. There also appears to be a stronger association between hepatic fat content and subclinical atherosclerosis in patients with psoriasis than in controls.⁷ A monocyte-to-high-density lipoprotein ratio >24 is associated with severe coronary risk,⁸ and early statin use, before atherosclerosis develops, can decrease the severity of psoriasis, CVD, and mortality.⁹ Obesity is an independent risk factor for the development of psoriasis and its comorbidities. It reduces treatment efficacy,¹⁰ whereas the Mediterranean diet has been shown to help lower PASI scores.¹¹

Finally, Professor Catherine Smith discussed treatment, noting that psoriasis therapeutics have evolved to include multiple highly effective and safe systemic options, including oral and injectable formulations.¹² Holistic, systematic assessment, high-quality prescribing practice with shared decision-making, and timely review are all essential to maximize patient benefit. Systemic therapy should be considered for diseases involving high-impact sites, more than 10% of the body surface area, or failure to respond to topical therapy. When treatment fails, clinicians should ask whether the

diagnosis is correct (e.g., paradoxical psoriasis, eczema, cutaneous T cell lymphoma, or other diagnoses), whether drug exposure is adequate (weight, anti-drug antibodies, adherence), whether pharmacodynamics are at play (wrong target or immune “switching”), and whether now is the right time to modify, add, or switch therapy.^{13,14} Her closing message: Be ambitious for your patients.

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Long-Term Durability and Safety of the Targeted Oral Peptide Icotrokinra for High-Impact Site Psoriasis: 2-Year Findings from the ICONIC-TOTAL Randomized Controlled Trial

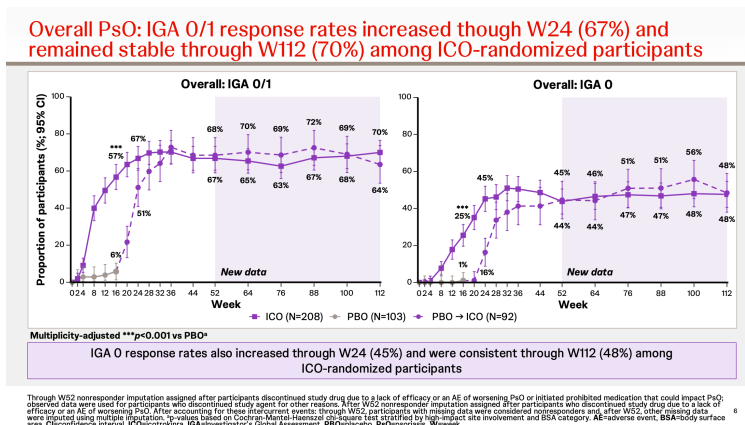
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Psoriasis involving high-impact sites such as the scalp, genitals, hands, feet, and nails significantly impairs quality of life. Icotrokinra (ICO) is the first targeted oral peptide that binds the interleukin (IL)-23 receptor and blocks IL-23 pathway signaling. It has previously shown superiority over placebo and deucravacitinib, with consistent results through one year.^{1,2}

Professor Richard B. Warren presented 2-year (week 112) data from ICONIC-TOTAL. This is an ongoing phase 3, randomized, double-blind, placebo-controlled trial in adults and adolescents (≥ 12 years) with plaque psoriasis and at least moderate involvement of one or more high-impact sites. Participants (N=311) were randomized 2:1 to ICO 200 mg once daily or placebo; placebo patients were switched to ICO at week 16.

Among ICO-randomized patients, responses achieved by week 24 were maintained through week 112:

- Overall: IGA 0/1 was 67% at week 24 and 70% at week 112 (IGA 0: 45% and 48%).
- Scalp: ss-IGA 0/1 was 78% and 74%; 60% achieved ss-IGA 0 at week 112.
- Genital: sPGA-G 0/1 was 90% and 89%; 89% achieved sPGA-G 0 at week 112.
- Hands/feet: hf-PGA 0/1 was 54% and 68%; 63% achieved hf-PGA 0 at week 112.
- Nails: mean mNAPSI improvement was 71%, and 42% achieved mNAPSI 0.



Professor Warren highlighted that exposure-adjusted adverse event rates did not increase with longer treatment, across 564 patient-years of exposure. The rate was 122 per 100 patient-years through week 112, compared with 232 per 100 patient-years in the placebo-controlled period. Serious infections, malignancies, and discontinuations due to adverse events remained infrequent, and no new safety signals were identified.

These findings support once-daily oral ICO as a durable, long-term treatment option for patients with high-impact site psoriasis.

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Zasocitinib Versus Deucravacitinib in Moderate-to-Severe Plaque Psoriasis: Results from the Phase 3 Head-to-Head LATITUDE Atlas Trial

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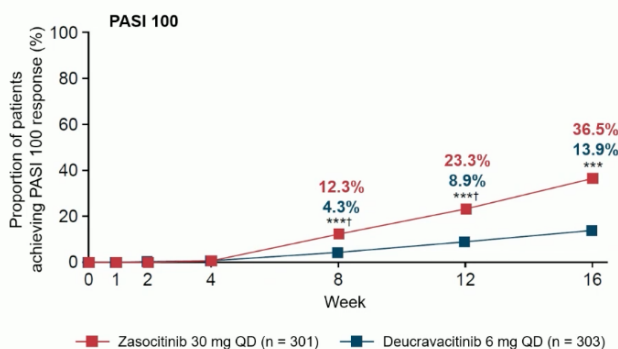
Zasocitinib is an investigational once-daily oral allosteric inhibitor of tyrosine kinase 2 (TYK2). It is more than a millionfold more selective for TYK2 than for JAK1/2/3, compared with 87-fold for deucravacitinib, and it inhibits interleukin (IL)-23 signaling for 24 hours.¹ Dr. Linda Stein Gold presented LATITUDE Atlas, the first head-to-head trial in dermatology to compare two agents with the same mechanism of action. It is also the first psoriasis trial of oral agents to use a 100% improvement in the Psoriasis Area and Severity Index (PASI 100) as the primary endpoint.

Adults with moderate to severe plaque psoriasis (PASI ≥ 12 , sPGA ≥ 3 , BSA $\geq 10\%$) were randomized 1:1 to zasocitinib 30 mg once daily (n=301) or deucravacitinib 6 mg once daily (n=303). Baseline characteristics were balanced: mean PASI was 19.7 and 19.0, and about 23% of patients in each arm had prior biologic exposure.

At week 16, zasocitinib was superior to deucravacitinib on every key endpoint (all $p < 0.001$):

- PASI 100: 36.5% vs 13.9%
- PASI 90: 62.5% vs 34.0%
- Absolute PASI ≤ 2 : 66.1% vs 38.0%
- sPGA 0: 43.2% vs 18.5%

Zasocitinib led to greater proportions of patients achieving clear skin (PASI 100) versus deucravacitinib as early as Week 8



Zasocitinib demonstrated > 2.5x greater PASI 100 response rate than deucravacitinib

Number of patients based on the full analysis set with non-responder imputation. P values for comparison versus deucravacitinib based on a stratified Mantel-Haenszel test. *** $p < 0.001$. †Nominal p value. PASI, Psoriasis Area and Severity Index; QD, once daily.

The two arms separated from week 8 onwards. Zasocitinib also improved DLQI more than deucravacitinib from week 4 (−9.51 vs −7.62 at week 16) and itch numeric rating scale (NRS) scores from week 2.

Treatment-emergent adverse events occurred in 65.9% and 58.7% of patients, respectively, and were mostly mild or moderate in severity. Rates of serious adverse events (1.3% vs 2.3%) and discontinuations due to adverse events (2.6% vs 2.3%) were similar. Acne (7.3% vs 3.0%) and acneiform dermatitis (6.0% vs 1.3%) were more common with zasocitinib. One death in the zasocitinib arm was considered unrelated to treatment.

Dr. Stein Gold concluded that zasocitinib cleared the skin better than deucravacitinib and that its safety profile was consistent with previous trials. Long-term safety and efficacy will be assessed in an ongoing open-label phase 3 study.

References:

1. Tyrosine Kinase 2 Inhibition with Zasocitinib (TAK-279) in Psoriasis: A Randomized Clinical Trial. Armstrong AW, Gooderham M, Lynde C, et al. *JAMA Dermatol.* 2024 Oct 1;160(10):1066-1074