EULAR 2025

DO NOT MISS HIGHLIGHTS IN PSORIATIC DISEASE

JUNE 2025

This special edition of the Y-GRAPPA Newsletter features a curated selection of notable abstracts in psoriatic disease, selected by our members ahead of the EULAR Congress in Barcelona. Explore what's new, what's relevant, and what deserves your attention.

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BASIC SCIENCE HIGHLIGHTS

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BASIC SCIENCE

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Basic science; Immune-stromal cell interactions; Psoriasis to PsA transition

Hannah den Braanker

TRANSITION FROM PSORIASIS TO PSORIATIC ARTHRITIS IS CHARACTERIZED BY DISTINCT ALTERATIONS IN PERIPHERAL BLOOD TC17 CELLS

H. Grasshoff et al.

Poster Tour V
Friday, 13 June, 12:42-12:48
POS0303

Full abstract here

This observational study investigated changes in peripheral blood T cell populations during the transition from psoriasis to PsA. The researchers analyzed 192 patients classified into subgroups including psoriasis with risk factors, prodromal, and established PsA of varying durations. They tracked both clinical data and used multicolor flow cytometry combined with machine learning data analysis to obtain comprehensive T cell profiling.
Results revealed that joint involvement evolved from few affected joints in the prodromal stage to polyarthritis with longer disease duration. Enthesitis, dactylitis and nail involvement were increasingly diagnosed over time. Most significantly, distinct alterations in CD4+ effector memory T cells, Th17 cells, and particularly T cytotoxic 17 (Tc17) cells were observed across patient groups, with Tc17 cells emerging as the primary differentiating feature between patients with and without enthesitis and serving as key markers of the transition phase

Why important?

Tc17 cells represent promising biomarkers and therapeutic targets for identifying patients transitioning from psoriasis to psoriatic arthritis.

Polling Question

When should we start monitoring psoriasis patients for PsA development?
A) At psoriasis diagnosis
B) After nail involvement appears
C) When imaging shows enthesopathy
D) Only with joint symptoms

BIOMARKERS OF GUT EPITHELIAL DAMAGE, MICROBIAL TRANSLOCATION, AND INNATE IMMUNE ACTIVATION CHARACTERIZE PATIENTS WITH AXIAL PSORIATIC ARTHRITIS FROM PSORIASIS PATIENTS: SOLUBLE BIOMARKER ANALYSIS INSIGHTS FROM THE ATTRACT STUDY

A. Agostinelli et al.

Poster tour II
Friday, 13 June, 14:57 - 15:03
POS0231

Full Abstract here

Previous studies in axial spondyloarthritis (axSpA) and IBD-associated spondyloarthritis have demonstrated elevated levels of gut damage markers including intestinal fatty acid binding protein (I-FABP), lipopolysaccharide-binding protein (LBP), and soluble CD14 (sCD14), suggesting that intestinal barrier dysfunction and microbial translocation drive systemic inflammation in these conditions. The authors hypothesized that similar gut-joint axis mechanisms might operate in axial psoriatic arthritis (axPsA).

This post-hoc analysis from the ATTRACT study tested plasma biomarkers of gut epithelial damage (I-FABP), microbial translocation (LBP), innate immune activation (sCD14), and bone remodeling (sclerostin/anti-sclerostin) in 30 patients with psoriasis, 20 patients with peripheral PsA, 30 patients with axPsA, and 25 healthy controls using ELISA techniques.

Patients with axPsA had significantly elevated levels of I-FABP, LBP, and sCD14 compared to psoriasis and healthy controls, indicating increased gut epithelial damage, microbial translocation, and innate immune activation similar to other spondyloarthropathies. Notably, I-FABP showed 70% sensitivity and 76.7% specificity for discriminating axPsA from psoriasis, while LBP demonstrated even better performance with 70% sensitivity and 77% specificity. These biomarkers also correlated positively with clinical disease activity measures including DAPSA and ASDAS scores.

Why important?

Gut-derived biomarkers I-FABP and LBP show promise as practical screening tools for identifying axial involvement in patients with psoriatic arthritis.

Polling Question

Which screening approach would you prefer for detecting axial PsA in patients with psoriasis?

A) MRI spine imaging
B) Blood biomarkers (I-FABP/LBP)
C) Clinical examination alone
D) HLA-B27 testing

SYNOVIAL FIBROBLASTS, MACROPHAGES AND DENDRITIC CELLS FORM AN INFLAMMATORY NICHE IN RHEUMATOID ARTHRITIS, BUT NOT IN PSORIATIC ARTHRITIS SYNOVIUM

A Khmelevskaia et al.

Session: Dendritic Cells - Orchestrators of tissue immunity
Room A3

Thursday, 12 June, 14:30 - 14:40
OP0230

Full Abstract here

While RA and PsA both cause synovial inflammation, the cellular mechanisms driving pathogenesis may differ. This study used single-cell RNA sequencing and spatial transcriptomics to investigate synovial tissue composition. Researchers analyzed synovial tissue from 12 patients with PsA, 15 patients with RA, 5 patients with undifferentiated arthritis, 5 patients with osteoarthritis, and 6 traumatic injury controls, processing 217,109 cells using advanced computational methods.

The analysis revealed striking disease-specific differences: while RA synovium showed dramatically enriched cell-cell interactions involving dendritic cells (DCs), PsA synovium resembled control samples with minimal DC interactions. In RA, FABP5+ inflammatory dendritic 3 cells formed a unique tri-cellular inflammatory niche with CHI3L1+ fibroblasts and MerTK-SPP1+ macrophages. Spatial transcriptomics confirmed physical proximity between them. This inflammatory niche architecture was completely absent in PsA synovium, suggesting fundamentally different cellular mechanisms in RA and PsA synovium. Understanding these disease-specific cellular architectures not only explains why current therapies show varying efficacy in RA and PsA, but can also help to identify distinct cellular targets for future drug discovery efforts.

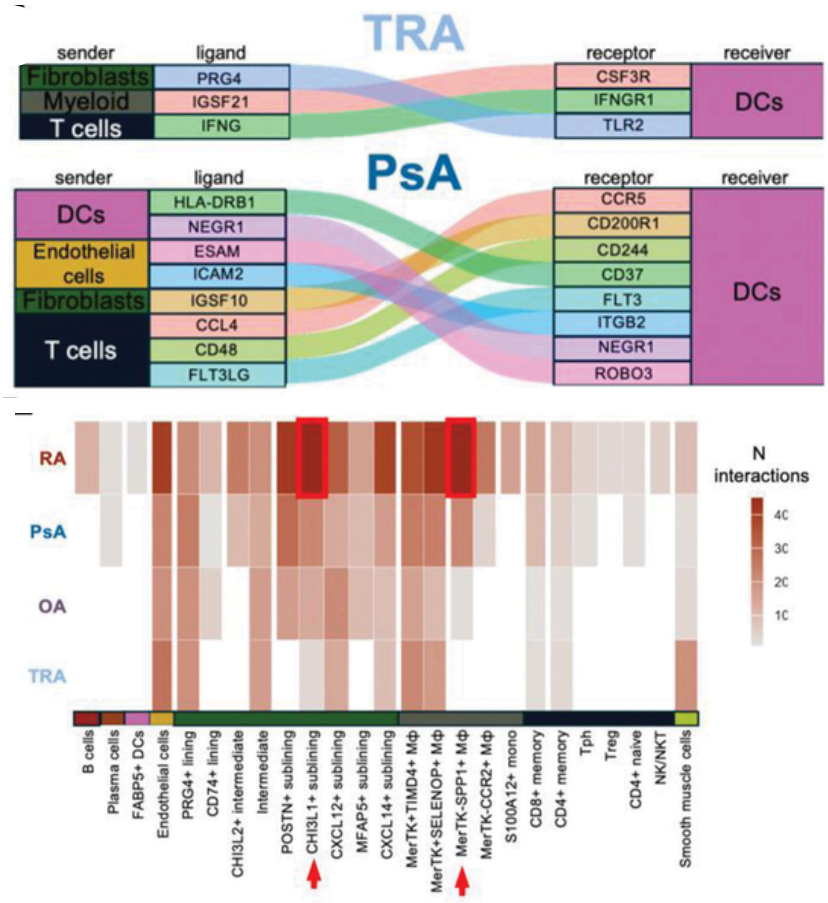
Why important?

PsA synovium lacks the inflammatory DC-fibroblast-macrophage niche characteristic of RA, providing molecular evidence for why these diseases respond differently to targeted therapies.

Polling Question

Would you consider a routine synovial biopsy analysis for differentiating PsA from RA?

A) Yes, for all unclear cases
B) Only in treatment-resistant patients
C) When clinical features overlap
D) No, too invasive/costly



BASIC SCIENCE

Viet Nguyen, PhD
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In vitro diagnosis, clinical chemistry, immunology, and macrophages in psoriasis vulgaris and IgE-mediated food

Viet Nguyen

MACHINE LEARNING AND PROTEOMICS FOR PREDICTING ANTI-TNFA RESPONSE IN PSORIATIC ARTHRITIS: IDENTIFICATION OF DRUG MODULATED PROTEINS

E. Martin-Salazar et al.

Poster Tour II
Friday, 13 June, 15:15 - 15:21
POS0234

Full Abstract here

This study aimed to identify proteomic biomarkers predictive response to TNF inhibitors (TNFi) in PsA. Peripheral blood mononuclear cells from 71 patients with PsA were analysed using the Olink 384 Explore inflammation assay. They were classified as responders or non-responders to TNFi based on a 50% reduction in DAPSA score after six months of treatment.
Machine learning algorithms identified 8 proteins that distinguished between TNFi responders and non-responders and used two of those proteins to create a discriminatory model for non-responders (AUC of 0.80 and an accuracy of 0.92).
In addition, 20 patients were followed longitudinally with proteins measured both at baseline and six months to explore the modulation of specific proteins by TNFi. 65 proteins were differentially expressed after six months. Upregulated proteins were enriched in B-cell-related pathways and the protein CD200 changed most significantly but only in responders. Downregulated proteins were associated with neutrophil pathways. CD200 plays a role in neutrophil regulation and after six months of treatment, a significant increase was observed in lymphocyte counts, accompanied by a decrease in neutrophil counts.

Why important?
The study highlights the potential of proteomics and computational tools in identifying biomarkers of anti-TNF-α response in patients with psoriatic arthritis.

Polling Question
Would you consider using a machine learning model for diagnosing conditions and predicting treatment outcomes?

SEX DIFFERENCES IN SERUM PROTEIN PROFILES OF MALES AND FEMALES WITH PSORIATIC ARTHRITIS

L. Eder et al.

Poster View
Wednesday, 11 June, 15:30 - 16:30
POS0702

Full Abstract here

This cross-sectional study collected serum samples from patients with active PsA in the University of Toronto Psoriatic Arthritis cohort. Serum proteins were analysed using an aptamer-based assay. The differentially expressed proteins (DEPs) between PsA males vs. PsA females and PsA vs. Controls were assessed using the limma package in R. Multi-protein classification models and a profile of sex-specific proteins were created to distinguish PsA from controls in males and females using logistic regression with elastic net, random forest, support vector machine, and linear discriminant analysis.

The study analysed 6402 serum proteins and determined that the number of DEPs was more than 20 times higher in PsA males (741) than in PsA females (31) when compared to controls.

The authors identified DEPs involved in 10 sex-specific pathways when comparing PsA males to PsA females. Of those DEPs, the number of sex-specific proteins expressed in males and females was 11 and 1, respectively.

The classification models distinguished PsA from controls by sex, with AUC scores ranging from 0.8 to 0.99. The variable importance analysis identified shared proteins in males and females, as well as sex-specific proteins, including leukotriene A4-hydrolase for females and IL-36A, NEK7, and PIK3CA/PIK3R1 in males.

Why important?

This study provides evidence of sex-related differences in serum proteins and biological pathways between male and female patients with PsA.

Polling Question

Would you prefer a blood test using these sex-specific proteins to an MRI for prognosis and personalised treatment of PsA?

CHAPERONE-MEDIATED AUTOPHAGY IN SYNOVITIS DISTINGUISHES PSORIATIC ARTHRITIS FROM RHEUMATOID ARTHRITIS

S. del Vescovo et al.

Poster View
Saturday, 14 June, 10:15 - 11:45
POS1429

Full Abstract here

Autophagy has been suggested to play different roles in the pathogenesis of PsA and RA. This study investigated whether the expression levels of LAMP2A, a key protein in chaperone-mediated autophagy, along with macroautophagy markers ATG7 and Beclin1, can serve as biomarkers to differentiate between PsA and RA based on their expression in synovial tissue.

Synovial biopsies from 29 patients with PsA and 11 patients with RA were stained immunohistochemically and with hematoxylin and eosin to calculate the Krenn's synovitis score.

LAMP2A and ATG7 expression in the inflammatory infiltrate was significantly higher in RA than in PsA, independent of synovitis grade. LAMP2A alone showed strong discriminatory power (AUC 0.89), as did ATG7 (AUC 0.95), and their combination further improved diagnostic accuracy (AUC 0.98).

These findings suggest that LAMP2A and ATG7 are promising biomarkers for distinguishing RA from PsA and point to a potentially greater role of chaperone-mediated autophagy in RA pathogenesis.

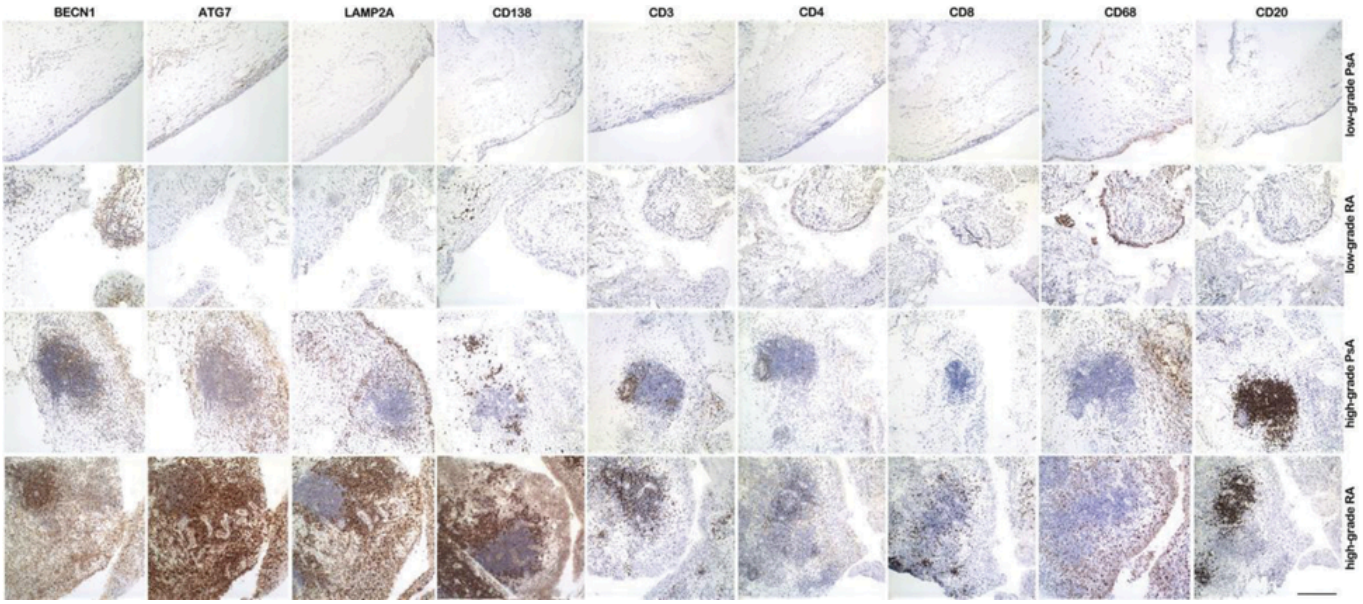
Why important?

This study identified two novel biomarkers (LAMP2A and ATG7) that can distinguish between PsA and RA.

Polling Question

Would you use synovial biopsies to differentiate between PsA and RA?

Figure 1.CD68 + sublining macrophages, plasmacells and T-lymphocytes seems to display a stronger LAMP2A and ATG7 positivity in RA patients, regardless of synovitis grading



CLINICAL HIGHLIGHTS

Sam Groothuizen, MD
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Y-GRAPPA member
Research focus: spondyloarthritis, imaging, disease development

Sam Groothuizen

DO CLINICAL MANIFESTATIONS OF PSORIATIC ARTHRITIS AT DIAGNOSIS OCCUR IN SITES PREVIOUSLY AFFECTED BY SUBCLINICAL INFLAMMATION

I. Giovannini et al.

Clinical Abstract Sessions:
From Early Detection to Complex Management - New Insights in Psoriatic Arthritis
Room B2

Thursday, 12 June, 11:20 - 11:30
OP0179

Full Abstract here

This study investigates whether joint swelling at the onset of PsA occurs in the same anatomical sites that previously showed subclinical signs, such as tenderness or ultrasound-detected inflammation.
226 patients with psoriasis from the RAPSODI and PSOART cohorts were followed longitudinally. 150 (66%) had subclinical PsA based on the EULAR criteria. After follow-up (median 12 months, IQR 4-21), 31 developed PsA, of which 78% presented with peripheral arthritis (mostly oligoarticular) and 18% with enthesitis. Interestingly, 68% of patients showed abnormalities in the anatomical sites where PsA eventually developed. 26% had tenderness, 32% ultrasound abnormalities and 42% both (see figure).
These results show that clinical PsA predominantly affects joints already affected during the subclinical phase, emphasizing the value of detailed subclinical evaluation. This supports the concept that subclinical PsA represents a very early phase of the disease. Early identification of high-risk joints can allow for earlier diagnosis and intervention.

Why important?

The findings emphasize the importance of the subclinical/very early disease phase. Future research can aid the identification of high-risk individuals to set up preventive trials.

Polling Question

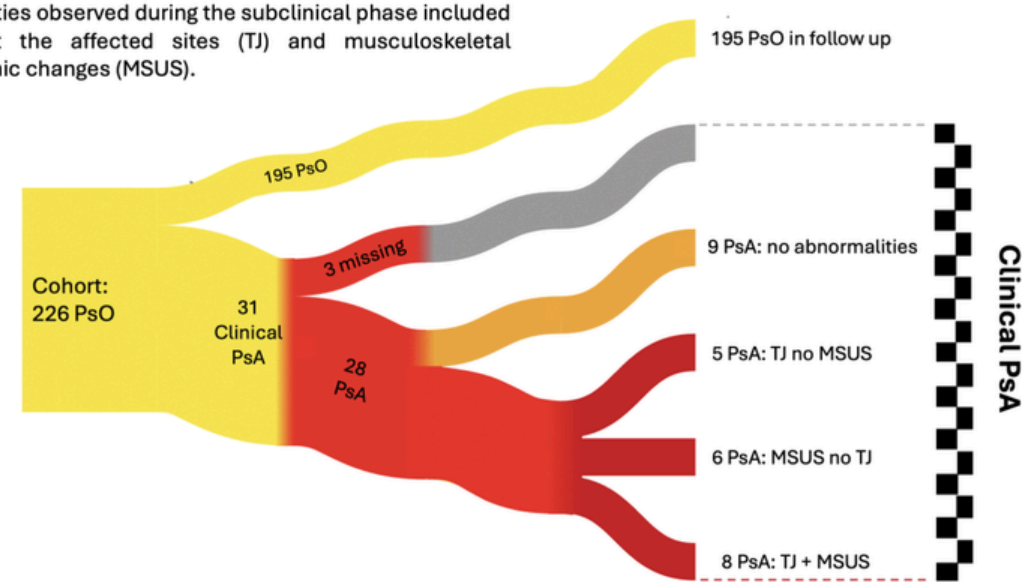
What is the next step for patients with psoriasis at risk of developing PsA? What (preventive) strategy should we focus on?

DO CLINICAL MANIFESTATIONS OF PSORIATIC ARTHRITIS AT DIAGNOSIS OCCUR IN SITES PREVIOUSLY AFFECTED BY SUBCLINICAL INFLAMMATION

I. Giovannini et al.

Clinical Abstract Sessions: From Early Detection to Complex Management - New Insights in Psoriatic Arthritis, Room B2
Thursday, 12 June, 11:20 - 11:30
OP0179

Figure 1. Diagram showing the progression of PsO patients with subclinical abnormalities at anatomical sites where PsA subsequently developed.
The abnormalities observed during the subclinical phase included tenderness at the affected sites (TJ) and musculoskeletal ultrasonographic changes (MSUS).



SACROILIAC JOINT INVOLVEMENT IN PSORIATIC ARTHRITIS – MRI, RADIOGRAPHIC AND CLINICAL FINDINGS IN 581 EUROPEAN ROUTINE CARE PATIENTS

N. Vladimirova et al.

Poster Tour V
Friday, 13 June, 12:06 - 12:12
POS0297

Full Abstract here

This study investigated the occurrence and characteristics of axial involvement in PsA using routine care imaging assessments. Patients with a clinical diagnosis of PsA (clin-PsA) or axial spondyloarthritis and psoriasis (axSpA+PsO) were included from five European registries. MRIs and radiographs were centrally evaluated.

581 patients were included (373 clin-PsA and 208 axSpA+PsO), of which 76% were biological-naïve. 31% of patients had an MRI indicative of SpA. Radiographs were available for 259 patients, of which 29% met the modified NY criteria for ankylosing spondylitis, while 38% had an MRI indicative of SpA. Patients with PsA who had MRI changes of axSpA were younger, more frequently male, HLA-B27 positive, and more likely to have nail psoriasis, uveitis, inflammatory back pain and higher CRP levels than those without axial MRI changes.

The frequency and type of MRI lesions varied by age and sex. Younger patients had more inflammatory lesions and older patients more structural lesions. The clinical presentations were different in patients with clin-PsA and axSpA+PsO with MRI changes indicative of SpA.

These findings suggest that clinical and radiographical assessments alone are insufficient for early identification of axial PsA, highlighting the importance of MRI.

Why important?

There is growing interest in axial PsA. These results highlight the importance of MRI in detecting axial involvement in PsA.

Polling Question

Where do you place MRI versus X-ray in the imaging work-up of patients with PsA and back pain?

	Overall N= 44	Positive N = 36	Negative N= 8	P-value
Age (years), median (IQR)	50 (42, 58)	51 (42, 58)	47 (42, 60)	0.9
Male/Female	26/18	21/15	5/3	> 0.9
PsO involvement, N(%) Scalp Nail	 23 (52.3%) 13 (29.5%)	 19 (52.8%) 9 (25.0%)	 4 (50.0%) 4 (50.0%)	 >0.9 0.2
BMI, median (IQR)	27.7 (24.8, 33.4)	27.8 (25.4, 34.3)	24.4 (22.7, 28.5)	0.023*
Tender Joint Count (66/68), median (IQR)	1.00 (0.0 , 4.0)	1.00 (0.0 , 4.0)	0.5 (0.0, 2.0)	0.5
VAS Pain (0 – 100), median (IQR)	35 (20, 60)	40 (30, 60)	20 (10, 30)	0.075
DAS28-CRP, median (IQR)	2.48 (1.70, 3.51)	2.73 (1.89, 3,54)	1.05 (0.77, 2.48)	0.04*
LEI > 0, N (%)	18 (40.9%)	16 (44.4%)	2 (25.0%)	0.4
MASES > 0, N (%)	10 (22.7%)	8 (22.2%)	2 (25.0%)	> 0.9
PASI, N (%)	1.65 (0.7, 3.0)	1.70 (0.80, 3.00)	1.50 (0.00, 1.90)	0.3
HAQ-DI, median (IQR)	0.38 (0.00, 0.88)	0.38 (0.00, 0.89)	0.38 (0.00, 0.63)	>0.9
CRP > 5 mg/dL, N (%) mg/dL, mean (SD)	15 (34.1%) 8.0 (6.5)	14 (38.9%) 8.9 (7.5)	1 (12.5%) 5.6 (1.6)	0.2
No therapy DMARD Therapy at Baseline, N (%) cDMARD bDMARD tsDMARD	27 (61.4%) 1 (2.3%) 13 (29.5%) 2 (4.5%)	23 (63.9%) 1 (2.8%) 10 (27.8%) 1 (2.8%)	4 (50.0%) 0 (0.0%) 3 (37.5%) 1 (12.5%)	0.7 >0.9 0.7 0.3
Final tratment status, N (%) No therapy Same therapy Start therapy/switched therapy	 13 (29.5%) 12 (27.3%) 19 (43.2%)	 11 (30.6%) 8 (22.2%) 17 (47.2%)	 2 (25.0%) 4 (50.0%) 2 (25.0%)	0.3

bDMARD, biological disease-modifying antirheumatic drugs; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BMI, body mass index; cDMARD, conventional antirheumatic drugs; CRP, C-reactive protein; DAS28-ESR, Disease Activity Score based on Erythrocyte Sedimentation Rate; HAQ, Health Assessment Questionnarie; LEI, Leeds Enthesitis Index; MASES, Maastricht Ankylosing Spondylitis Enthesitis Score; PASI, Psoriasis Area and Severity Index; VAS, Visual Analogue Scale.

CLINICAL HIGHLIGHTS

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TWO DISTINCT PATTERNS OF SACROILIAC JOINT BONE MARROW OEDEMA IN AXIAL SPONDYLOARTHRITIS ARE ASSOCIATED WITH HLA-B27 STATUS, BODY MASS INDEX AND/PSORIASIS

D. McGonagle et al.

Poster Tour IV
Thursday, 12 June, 15:03 - 15:09
POS0126

Full Abstract here

In this MRI-based study of 203 patients with axial spondyloarthritis (axSpA), researchers identified two distinct sacroiliac joint bone marrow oedema (BMO) patterns: an **upper SIJ BMO pattern** associated with older age, higher BMI, and psoriasis, and a **lower SIJ BMO pattern** strongly linked to HLA-B27 positivity, male sex, and systemic inflammation. This suggests different underlying mechanisms and phenotypes within axSpA.

Why important?

Recognizing upper vs. lower SIJ BMO patterns may help refine axSpA diagnosis and guide personalized treatment strategies based on disease phenotype and comorbidities.

Polling Question

Could identifying distinct sacroiliac MRI patterns (upper vs. lower) change how we diagnose or manage axial spondyloarthritis in clinical practice?

BONE PROPERTIES AND BIOMECHANICS IN PATIENTS WITH PSORIATIC DISEASE: A CROSS-SECTIONAL STUDY WITH HIGH-RESOLUTION PERIPHERAL QUANTITATIVE COMPUTED TOMOGRAPHY (HRPQCT)

G. Adami et al.

Poster Tour V
Friday, 13 June, 13:06 - 13:12
POS0307

Full Abstract here

This cross-sectional study used high-resolution peripheral quantitative CT (HRpQCT) to assess bone quality and mechanics in patients with psoriasis, PsA, and healthy controls.
Both patients with psoriasis and PsA showed lower cortical and total volumetric bone mineral density, thinner cortices, and impaired mechanical bone properties, especially in PsA, where stiffness and failure load were significantly reduced.
Nail involvement (onychodystrophy) was also linked to poorer bone stiffness at distal joints, independent of age, sex, and PASI.

Why important?

Psoriatic disease—especially when involving the nails—may silently impair bone quality and strength, suggesting a need for early skeletal assessment even in patients without joint symptoms.

Polling Question

Should we screen for subclinical bone fragility in patients with psoriatic disease, especially those with nail involvement?

Figure 1.
A. Total volumetric bone mineral density (vBMD) in Hounsfield Unit - conversion to mgHA/mm 3 = HU/234*2.34; B. Cortical thickness (mm) at distal radius

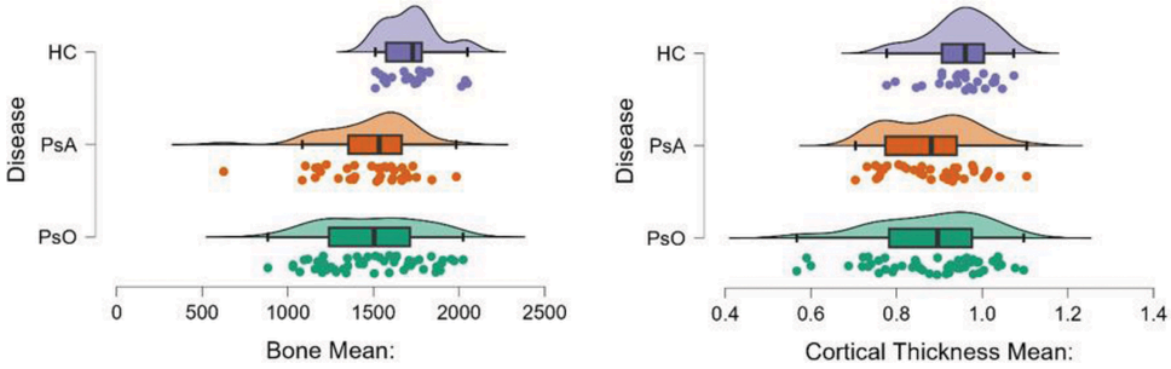
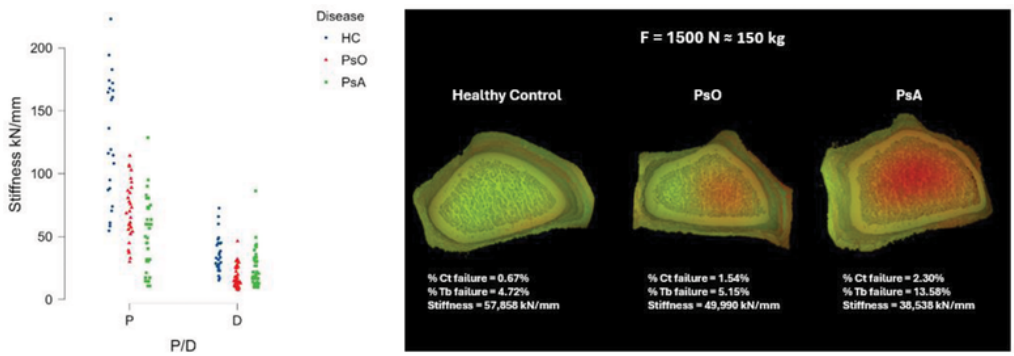


Figure 2.
Stiffness (Kn/mm) proximally (P) and distally (D) to the II, III and IV DIPs



ESTABLISHING DEFINITIONS FOR DIFFICULT-TO-TREAT PSORIATIC ARTHRITIS (D2T-PSA) AND COMPLEX-TO-MANAGE PSORIATIC ARTHRITIS (C2M-PSA): INSIGHTS FROM THE GROUP FOR RESEARCH AND ASSESSMENT OF PSORIASIS AND PSORIATIC ARTHRITIS (GRAPPA) INITIATIVE

F. Proft et al.

Clinical Abstract Sessions:
From Early Detection to Complex Management - New Insights in Psoriatic Arthritis
Room B2

Thursday, 12 June, 10:40 - 10:50
OP0175

Full Abstract here

Why important?

These new definitions offer a unified language for identifying and managing the most refractory and complex patients with PsA in both research and clinical settings.

Polling Question

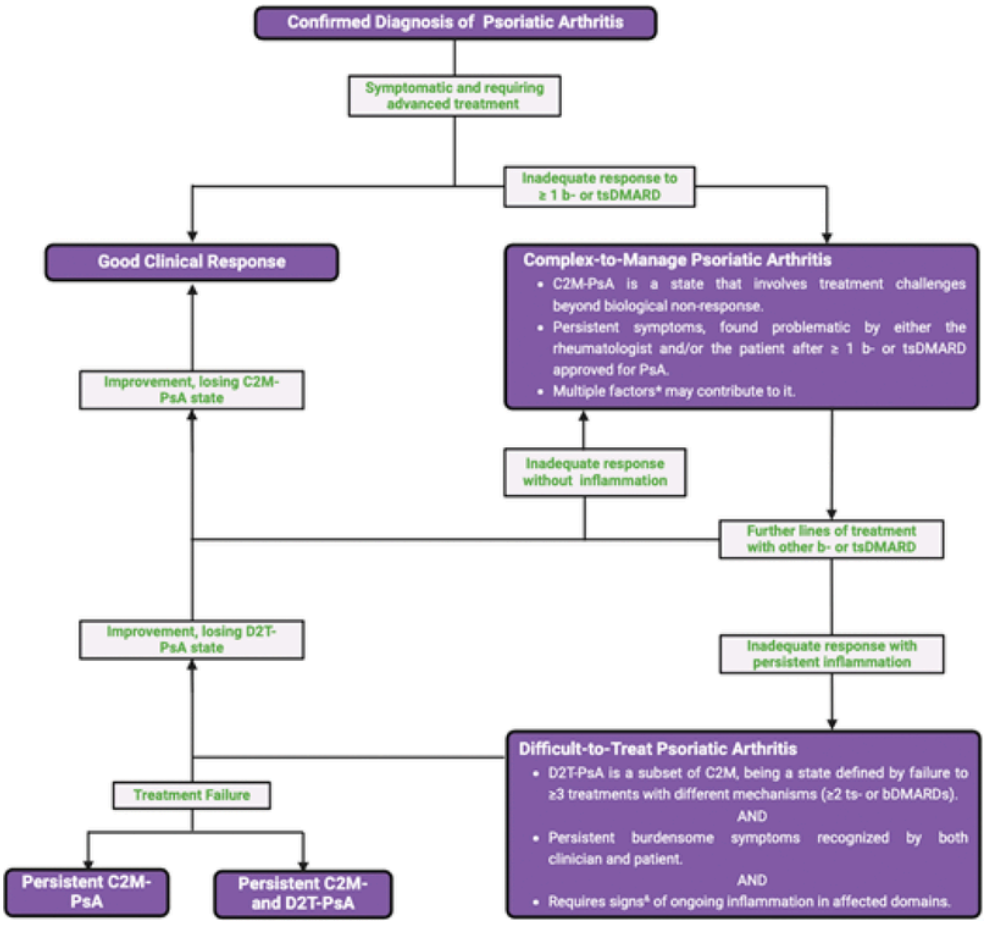
What do you think is the most critical criterion to define Difficult-to-Treat Psoriatic Arthritis?

GRAPPA has developed consensus definitions for Difficult-to-Treat Psoriatic Arthritis (D2T-PsA) and Complex-to-Manage Psoriatic Arthritis (C2M-PsA) to address unmet needs in patients who do not respond adequately to multiple therapies or face challenges beyond inflammation, such as comorbidities and chronic pain.

Using a global survey, multidisciplinary expert input, and a Delphi consensus method, the initiative produced five guiding principles and validated criteria endorsed by over 95% of GRAPPA members.

D2T-PsA is characterized by persistent inflammation despite ⩾3 treatments (including ⩾2 advanced therapies), while C2M-PsA includes broader biopsychosocial barriers to disease control.

Figure 1.
Algorithm of treatment failure in PsA with Complex-To-Manage PsA and Difficult-To-Treat PsA.



TREATMENT HIGHLIGHTS

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EARLY INTENSIVE THERAPY WITH COMBINATION CSDMARDS OR TNF INHIBITORS IS SUPERIOR TO STANDARD STEP-UP CARE IN MODERATE-TO-SEVERE PSA: THE SPEED RCT

L. C. Coates et al.

Clinical Abstract Sessions:
Shaping the Future of Psoriatic Arthritis Treatment
Room D1

Wednesday, 11 June, 16:30 - 16:40
OP0089

Full Abstract here

The SPEED trial used a pragmatic Trials Within Cohorts design to enrol newly diagnosed patients with PsA with ⩾1 poor prognostic factor (polyarthritis, elevated CRP, HAQ >1, or radiographic erosions) from the MONITOR cohort. Patients were randomized to standard step-up csDMARD therapy, combination csDMARDs, or early TNFi plus methotrexate for 24 weeks, then maintaining only methotrexate.

At week 24, both intervention arms showed superior PASDAS scores vs. standard care: adjusted mean differences of -0.69 for combination csDMARDs and -1.09 for TNFi (p=0.02 and <0.001, respectively). PASDAS good responses at week 24 were highest in the TNFi group (45.3%), followed by csDMARD combination (31.2%) and step-up care (7.5%). At week 48, the early TNFi group maintained significant benefits.

Exploratory outcomes suggested better DAPSA and PASI75 responses in both intervention arms. Only 4 serious adverse events were reported (2 in each arm), and side effects were mostly mild and manageable, with slightly more infections in the TNFi group and more liver enzyme abnormalities in the combination csDMARD arm.

Why important?

This study supports the use of early, intensive therapy in patients with PsA with poor prognostic markers. A finite course of TNFi, combined with MTX, leads to more sustained disease control than standard therapy.

Polling Question

Would you support starting combined csDMARD or TNFi therapy immediately in patients newly diagnosed with PsA with poor prognostic factors?

EARLY INTENSIVE THERAPY WITH COMBINATION CSDMARDS OR TNF INHIBITORS IS SUPERIOR TO STANDARD STEP-UP CARE IN MODERATE-TO-SEVERE PSA: THE SPEED RCT

L. C. Coates

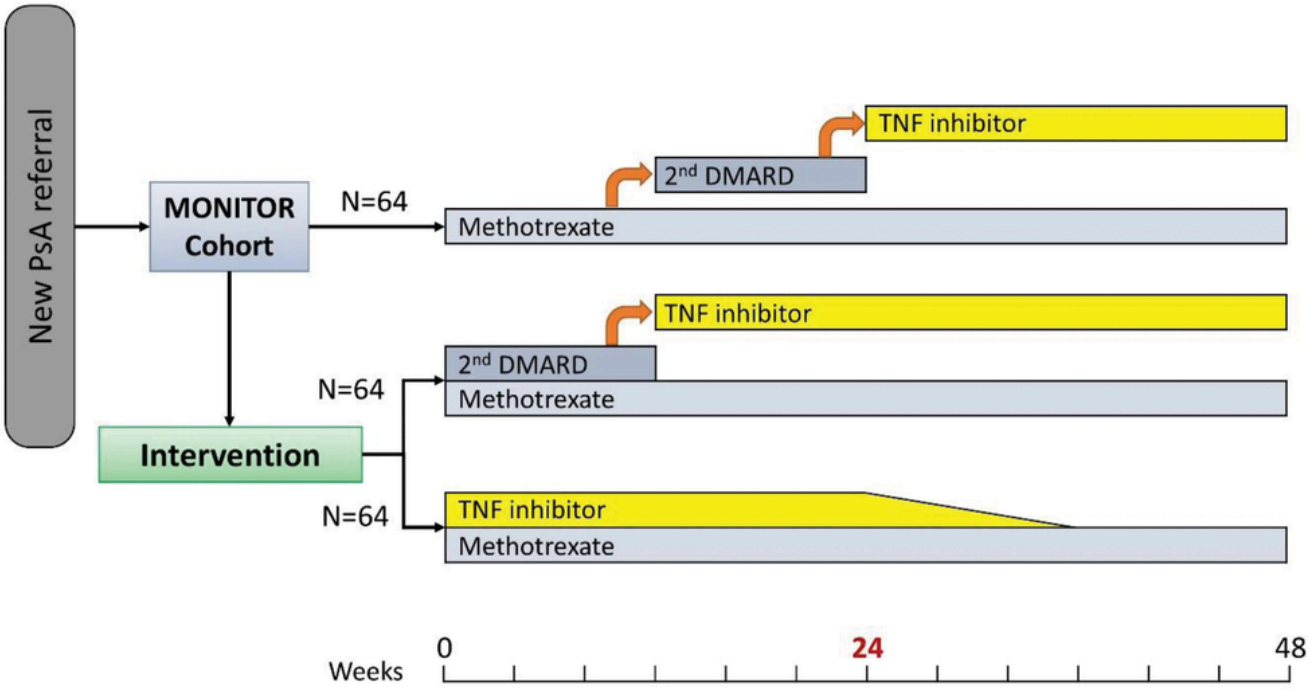
Clinical Abstract Sessions: Shaping the Future of Psoriatic Arthritis Treatment

Wednesday, 11 June, 16:30 - 16:40, Room D1

Abstract OP0089

Full Abstract here

Figure 1



REAL-WORLD COMPARATIVE EFFECTIVENESS OF UPADACITINIB IN PSORIATIC ARTHRITIS: EVALUATION OF SWITCHING TO UPADACITINIB VERSUS TUMOR NECROSIS FACTOR INHIBITORS OR INTERLEUKIN-17 INHIBITORS AFTER FIRST-LINE TUMOR NECROSIS FACTOR INHIBITORS

P.J. Mease et al.

Poster View
Friday, 13 June, 12:00 - 13:30
POS1045

Full Abstract here

This real-world, cross-sectional study evaluated the comparative effectiveness of upadacitinib (UPA) in patients with PsA who had failed a first-line TNFi. Data were collected from the Adelphi Real World Spondyloarthritis Disease Specific Programmes in Europe and the U.S., with a total of 320 patients analyzed: 101 switched from TNFi to UPA, 96 to another TNFi, and 123 to an IL-17 inhibitor (IL-17i). The key endpoint was physician-reported tender joint count (TJC) and swollen joint count (SJC) ⩽1 after ⩾3 months of second-line therapy.

Baseline characteristics were balanced using inverse-probability-weighted regression adjustment, including age, sex, Charlson Comorbidity Index, and physician-reported disease severity. At the time of treatment switch, disease severity was moderate or severe in >90% of patients across all groups.

After statistical adjustment, a significantly greater proportion of patients who switched from TNFi to UPA achieved low joint counts (TJC and SJC ⩽1) compared to those who cycled to another TNFi (94% vs 76%; P = 0.0342) or switched to an IL-17i (91% vs 72%; P = 0.0123). Most switches were due to worsened joint condition.

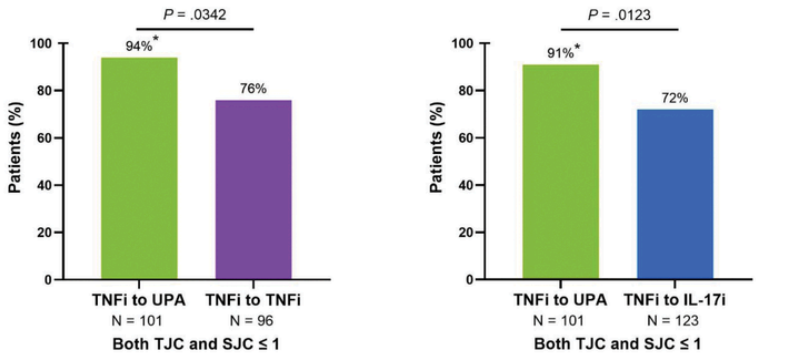
Why important?

These data suggest that switching to UPA after TNFi failure may lead to better control of joint inflammation than cycling to another TNFi or switching to an IL-17i.

Polling Question

After failure of a first TNF inhibitor in PsA, what is your preferred second-line approach?

Figure 1: Adjusted Physician-Reported Assessment of Both TJC and SJC ≤ 1 at the Time of Data Collection



IL-17i, IL-17 inhibitor; TNFi, TNF inhibitor; UPA, upadacitinib.
*P < .05.

SONELOKIMAB IMPROVES MULTIDOMAIN AND HIGH-HURDLE OUTCOMES IN PSA: 24-WEEK RESULTS FROM THE ARGO PHASE 2 TRIAL

I.B. McInnes et al.

Clinical Abstract Sessions: Shaping the Future of Psoriatic Arthritis Treatment
Room D1

Wednesday, 11 June, 17:40 - 17:50
OP0096

Full Abstract here

Why important?

Sonelokimab showed efficacy in PsA, with high rates of joint and skin remission (ACR70 + PASI100), and a favorable safety profile.

Polling Question

Would you consider switching to an IL-17A/F inhibitor in a patient with PsA who has active joint and skin disease despite IL-17Ai?

Sonelokimab is a nanobody targeting both IL-17A and IL-17F, and was superior to placebo at week 12 in the Phase 2 ARGO trial, which recruited 207 patients with active PsA. They were randomized to receive sonelokimab (with or without induction, NI), adalimumab (reference arm), or placebo (switched to sonelokimab at week 12).

At Week 24, minimal disease activity (MDA) was achieved by 61.0% of patients receiving sonelokimab 60 mg, 51.2% with 120 mg, and 46.3% with 60 mg NI, compared to 45.2% in the adalimumab reference arm. The high-hurdle composite endpoint of ACR70 plus PASI 100 was reached by 48.1% of patients receiving sonelokimab 120 mg, 34.6% with 60 mg, and 31.3% with 60 mg NI, versus 18.8% with adalimumab. ACR70 responses alone were observed in 41.9% of the 120 mg group and in 39.0% of both 60 mg groups, compared to 35.7% in the adalimumab arm. Complete skin clearance (PASI 100) was achieved in 63.0% of patients on sonelokimab 120 mg, 61.5% on 60 mg, and 59.4% on 60 mg NI, compared to 50.0% with adalimumab.

Sonelokimab was well tolerated across all treatment arms, with no unexpected safety signals. There were no reports of inflammatory bowel disease, major adverse cardiovascular events, depression, or suicidal ideation. Four cases (2%) of mild-to-moderate oral candidiasis were reported and managed without treatment discontinuation. These findings demonstrate that sonelokimab leads to consistent and high-level improvements across joint and skin domains, with favorable tolerability. The Phase 3 trials IZAR-1 and IZAR-2 are currently evaluating sonelokimab in larger patient populations.

Table 1.

Composite and high-hurdle outcomes at Week 24

n, (%)	SLK 60mg NI n=41	SLK 60mg n=41	SLK 120mg n=43	ADA ref n=42
MDA	19 (46.3)	25 (61.0)	22 (51.2)	19 (45.2)
ACR70 + PASI 100*	10 (31.3)	9 (34.6)	13 (48.1)	6 (18.8)
ACR70	16 (39.0)	16 (39.0)	18 (41.9)	15 (35.7)
PASI 100*	19 (59.4)	16 (61.5)	17 (63.0)	16 (50.0)

NRI, except for patients who switched treatment due to lack of joint response or who completed the study at Week 12 where the last observation during Part A was carried forward.
*% based on patients with ≥3% BSA at baseline in the ITT population (SLK 60mg NI, n=32; SLK 60mg, n=26; SLK 120mg, n=27; ADA ref, n=32).
ACR, American College of Rheumatology; ADA ref, adalimumab reference; BSA, body surface area; ITT, intention-to-treat; MDA, minimal disease activity; NI, no induction; NRI, non-responder imputation; PASI, Psoriasis Area and Severity Index; SLK, sonelokimab.

EFFICACY AND SAFETY OF DEUCRAVACITINIB UP TO WEEK 52 FROM POETYK PSA-2: A MULTICENTER, RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED, PHASE 3 STUDY IN PATIENTS WITH PSORIATIC ARTHRITIS

P.J. Mease et al.

Clinical Abstract Sessions:
Shaping the Future of Psoriatic Arthritis Treatment
Room D1

Wednesday, 11 June, 17:30 - 17:40
OP0095

Full Abstract here

Why important?

Deucravacitinib is the first oral TYK2 inhibitor to demonstrate efficacy in both joint and skin outcomes in PsA, with maintained responses over 52 weeks and a favorable safety profile.

Polling Question

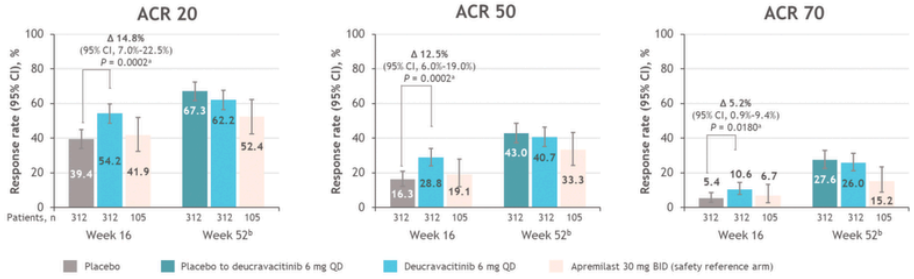
Would you consider deucravacitinib as a first oral advanced therapy in PsA after csDMARD failure?

The phase 3 POETYK PsA-2 trial evaluated the efficacy and safety of deucravacitinib — an oral, selective TYK2 inhibitor — in patients with active PsA with or without prior TNFi treatment. A total of 729 patients were randomized to deucravacitinib 6 mg once daily (n=312), placebo (n=312), or apremilast 30 mg twice daily (n=105, safety reference arm). At Week 16, significantly more patients receiving deucravacitinib achieved ACR20 (54.2% vs. 39.4%; p=0.0002), ACR50 (28.8% vs. 16.3%), and ACR70 (10.6% vs. 5.4%) compared to placebo. These responses further improved or were maintained through Week 52, with ACR20/50/70 rates reaching 67.3%, 43.0%, and 26.7%, respectively (Figure 1).

Deucravacitinib also led to greater improvements in secondary outcomes including PASI75 (40.9% vs. 15.4%; p<0.0001), MDA (25.6% vs. 14.7%; p=0.0007), and HAQ-DI (-0.32 vs. -0.21; p=0.0013). SF-36 physical component score improved by 5.8 vs. 3.8 points (p=0.0002). In pooled analyses from PsA-1 and PsA-2, resolution of dactylitis was significantly higher with deucravacitinib vs. placebo (57.6% vs. 44.1%; p=0.0100), while enthesitis resolution showed a non-significant trend (50.3% vs. 45.1%; p=0.1781). Deucravacitinib also showed superior reduction in DAS28-CRP (-1.28 vs. -0.80; p<0.0001), and numerically greater improvements in fatigue (FACIT-Fatigue +2.5 vs. +1.8; p=0.2017).

Through Week 52, deucravacitinib was well tolerated, with low rates of serious adverse events (1.9%) and treatment discontinuation due to AEs (2.2%). The most common AEs were nasopharyngitis (11.8%), COVID-19 (11.6%), and upper respiratory infections (10.9%). Rates of cardiovascular events, VTE, and malignancies were low and similar across treatment arms. No new safety signals were identified.

Figure. ACR 20/50/70 at weeks 16 and 52



No prespecified statistical comparisons were performed for the apremilast arm.

All randomized patients were assessed. Nonresponder imputation was used to handle missing data. Treatment discontinuations prior to week 16 were considered treatment failures (composite variable strategy). All rescue medication–related intercurrent events were treated with a treatment policy–estimand strategy. The Clopper-Pearson estimation method was used to estimate CI. ªA Cochran-Mantel-Haenszel test stratified by TNF inhibitor (yes/no), screening hsCRP concentration (< 10 mg/L vs ≥ 10 mg/L), and csDMARD use at baseline (yes/no) was used to compare the response rates with deucravacitinib 6 mg QD to placebo; ᵇExploratory endpoint.
ACR 20, American College of Rheumatology 20% improvement in response; ACR 50, American College of Rheumatology 50% improvement in response; ACR 70, American College of Rheumatology 70% improvement in response; BID, twice daily; csDMARD, conventional synthetic disease-modifying antirheumatic drug; hsCRP, high-sensitivity C-reactive protein; QD, once daily; TNF, tumor necrosis factor.

TREATMENT HIGHLIGHTS

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SECUKINUMAB DEMONSTRATES HIGH LONG-TERM EFFICACY, DRUG SURVIVAL, AND OSTEOPROTECTIVE EFFECTS IN PSORIATIC ARTHRITIS AND PSORIASIS: 48-MONTH FOLLOW-UP OF THE PSARTROS STUDY

L. Schuster et al.
Poster View
Saturday, 14 June, 10:15 - 11:45
POS1329

Full Abstract here

Why important?

Long-term treatment with secukinumab is effective, well-tolerated and appears to have osteoprotective effects in PsA.

Polling Question

Does the increase in studies evaluating the effects of IL-17 inhibitors in the treatment of psoriatic arthritis make you prefer these agents as the first step in the management of PsA?

This abstract presents the long-term clinical and imaging follow-up of the PSARTROS cohort was presented. This prospective, exploratory, open-label study recruited patients with PsA and patients with psoriasis with baseline subclinical changes on MRI and/or HRpQCT but no clinical evidence of PsA. Patients were treated with secukinumab 150 mg or 300 mg.

The study recruited 32 participants (19 male, 13 female) with a mean age of 55 (SD=9.5) years. Participants had an average BMI of 28.2 (SD=4.5). By the end of follow-up there was a significant decrease in DAS28 (p=0.009), LEI (p=0.023), PASI (p=0.017) and pain (p=0.030) compared to baseline.

A total of 202 HR-pQCT measurements were performed over 48 months of follow-up and no significant changes were observed in bone mineral density, microstructure, and biomechanics at either the radius or MCPs. The number and volume of erosions remained stable as well (Table 1). Moreover, secukinumab demonstrated long-term tolerability and clinical efficacy across all major disease domains over 48 or more months, with a 4-year drug survival rate of nearly 70%.

These findings demonstrate that secukinumab is a well-tolerated and effective long-term treatment for PsA and suggest that it has osteoprotective effects.

SYSTEMIC AND TISSUE-SPECIFIC MOLECULAR PROFILING OF APREMILAST IN HEPATIC STEATOSIS: A POTENTIAL THERAPEUTIC FOR MASLD IN PSA

M. Ruiz-Ponce et al.

Poster Tour II
Friday, 13 June, 14:45 - 14:51
POS0229

Full Abstract here

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is recognized as an extra-articular manifestation of PsA, suggesting a potential interaction between the pathologies. Apremilast, a phosphodiesterase (PDE4) inhibitor used in the treatment of PsA, has demonstrated positive anti-inflammatory and metabolic effects. These effects could potentially be beneficial for MASLD.
C57BL/6Jmice were fed either a standard diet (CHOW) or the GAN diet to induce MASLD. Half of the mice in each group were treated with apremilast. The impact of the treatment on body weight, fat mass and lean mass percentage was monitored and protein expression in tissues was analyzed using Olink® technology.
Apremilast slowed down the progression of MASLD in mice, including reductions in body weight, fat mass, and the weight of metabolic organs. The reduced progression of steatosis was associated with lower levels of ALT, insulin and triglycerides in MASLD mice and proteomic analysis in metabolic tissues and serum revealed significant alterations in proteins related to inflammation, apoptosis, and lipid metabolism in MASLD mice compared to control mice. Apremilast successfully restored the levels of key proteins involved in inflammation, apoptosis, fibrosis, and lipid metabolism in both serum, liver and adipose tissue of MASLD mice, highlighting its potential as a therapeutic option for MASLD, particularly in the context of PsA.

Why important?

The effects of apremilast on inflammation, fibrosis, and lipid metabolism indicate that apremilast may improve metabolic parameters in addition to controlling joint complaints in patients with PsA.

Polling Question

Do the metabolic effects of apremilast influence your treatment choice?

Exploring **Psoriatic Disease**:
Key Scientific Sessions and Discussions
at EULAR 2025

Thursday, 12 June

Dendritic Cells - orchestrators of tissue immunity. Room A3 13:30 - 14:45 CEST

Enthesitis in PsA, from disease development to specific therapeutic targets. Room F3 13:30 - 14:45 CEST

Obesity and it's impact on pathophysiology, clinic, and treatment of RMDs. Room F1 13:30 - 14:45 CEST

Friday, 13 June

What are the new treatments for spondyloarthritis and psoriatic arthritis on the horizon? Room Meet the Expert 4 09:45 - 10:15 CEST

Plenary Psoriatic Arthritis. Room Plenary 14:15 - 15:00 CEST

Saturday, 14 June

EULAR Recommendations II. Room D1 09:00 - 10:15 CEST

Your PsA Abstract Compass for EULAR

Poster Tours

Basic Poster Tours: New molecules on the horizon in Spondyloarthritis and Psoriatic Arthritis. Wednesday, 11 June, 15:30 - 16:30 CEST. Poster Tour II

Clinical Poster Tours: Advancing Psoriatic Arthritis Therapy. Thursday, 12 June, 12:00 - 13:30 CEST. Poster Tour IV

Clinical Poster Tours: Psoriatic Disease is a Journey. Friday, 13 June, 12:00 - 13:30 CEST. Poster Tour V

Basic Poster Tours: Molecular treatment signatures in Spondyloarthridities including Psoriatic Arthritis. Friday, 13 June, 14:45 - 15:45 CEST. Poster Tour II

Oral Abstracts Sessions

Clinical Abstract Sessions: Shaping the Future of Psoriatic Arthritis Treatment. Wednesday, 11 June, 16:30 - 18:00 CEST Room D1

Clinical Abstract Sessions: From Early Detection to Complex Management - New Insights in Psoriatic Arthritis. Thursday, 12 June, 10:30 - 12:00 CEST Room B2

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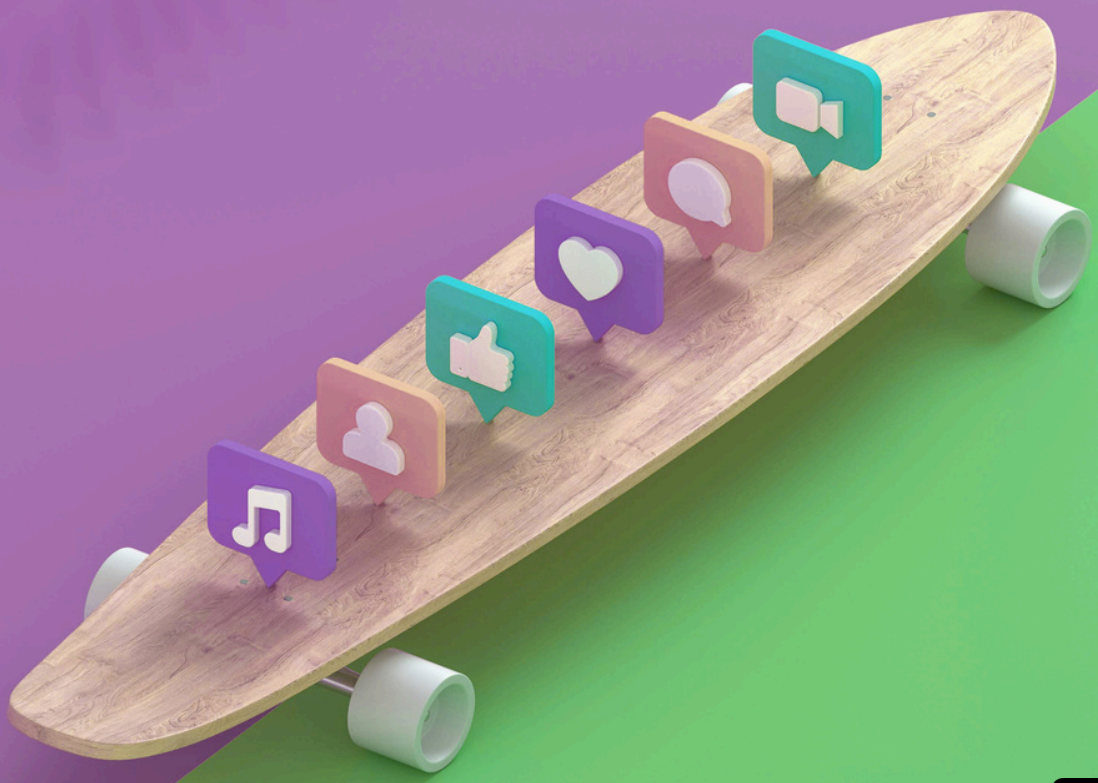

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