Congress Beyond the Joint: Weight, Ultrasound, and the Therapeutic Frontier in Psoriatic Arthritis

Author:	*André Lucas Ribeiro[1] 1. Hospital de Clínicas de Porto Alegre, Brazil *Correspondence to Lucasribeiro.andre@gmail.com

Disclosure:	The author has disclosed no conflicts of interest.
Keywords:	Combination therapy, obesity, psoriatic arthritis, treat-to-target, ultrasound, weight loss.
Citation:	EMJ Rheumatol. 2026;13[1]:24-27. https://doi.org/10.33590/emjrheumatol/V3B1LA72

PSORIATIC arthritis (PsA) at the European Alliance of Associations for Rheumatology (EULAR) 2026 Congress was defined less by a single breakthrough than by a shift in framing. Across the 'Optimising care in Psoriatic Arthritis' and late-breaking sessions, three questions dominated: what we treat, when we should intervene, and how aggressively we should combine therapies. The unifying message was that drug choice, long the centre of the PsA conversation, is now only one variable among several that determine whether a patient reaches remission.

WEIGHT IS A TREATMENT TARGET, NOT A BACKGROUND VARIABLE

The most provocative reframing came from a machine-learning reanalysis of the SPEED trial, an open-label study of early PsA with poor prognostic factors.[1] Using recursive partitioning across baseline variables in 192 patients, the investigators found that BMI (median: 28.96 kg/m²) was the single most influential predictor of 1-year PASDAS, while treatment arm ranked only sixth. The derived decision rules were clinically legible: patients with a BMI below 25 kg/m² and lower baseline disease activity (PASDAS <5.4) achieved the best outcomes irrespective of treatment, whereas the combination of obesity (BMI: ≥27 kg/m²), longer disease duration, and polyarticular disease predicted PASDAS scores as high as 6.0 at Week 48. This is a sub-analysis of a single trial and should be read as hypothesis-generating; recursive partitioning is sensitive to cohort composition and cannot disentangle adiposity from its metabolic and behavioural correlates. Even so, the signal aligns with prior observational data linking obesity to lower remission rates, reframing weight as a modifiable treatment component.

If SPEED implicated weight, the Phase IIIb TOGETHER-PsA trial tested whether acting on it changes outcomes.[2] Adults with active PsA and obesity or overweight with a weight-related comorbidity (mean BMI: 37.2–38.4 kg/m²) were randomised to ixekizumab with concomitant tirzepatide (IXE+TZP) or ixekizumab alone. The composite primary endpoint at Week 36, ACR50 (American College of Rheumatology 50% improvement response) plus at least 10% weight reduction, was achieved by 31.7% of the combination group versus 0.8% with ixekizumab alone (p<0.001). The contrast is partly mechanical, since the weight-loss component is unattainable with ixekizumab monotherapy, but the articular signal was independent and early: ACR50 separated by Week 4 (11.1% versus

3.9%; p=0.029) and minimal disease activity (MDA) favoured the combination at Week 36 (26.3% versus 15.3%; p=0.026). Even though TOGETHER-PsA does not establish whether benefits were driven by weight loss, the metabolic effects of glucagon-like peptide-1/glucose-dependent insulinotropic polypeptide agonism, or improved drug pharmacodynamics, but it provides the first randomised evidence that addressing adiposity alongside cytokine blockade is feasible and clinically active.

The DIPSA trial helped adjudicate which of those mechanisms is doing the work.[3] In 92 patients with moderately active PsA and a BMI of 25–40 kg/m², a Mediterranean diet, a low-calorie dietary approach to stop hypertension diet, and standard dietary advice were compared. All three arms produced modest weight loss and significant improvement in DAPSA by Week 24, with no differences between them, and the early MDA advantage for the dietary arms at Week 12 had dissipated by Week 24. The interpretable finding was a dose-response, with the magnitude of weight loss, not the dietary strategy, predicting improvement in DAPSA independent of arm. Read alongside SPEED and TOGETHER-PsA, DIPSA implies that weight reduction itself, however achieved, is the active ingredient and the dietary pattern secondary.

ULTRASOUND SHARPENS THE QUESTIONS OF WHO AND WHEN

If weight reframed what we treat, imaging reframed when we should act and ultrasound was the connecting thread. Zabotti et al.[4] compared clinical and ultrasound features across the psoriatic disease continuum in 183 patients spanning psoriasis, stable and progressing subclinical PsA, and early clinical disease.[4] The central finding was that clinical assessment failed where imaging succeeded: tender joint count, Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), pain, and Health Assessment Questionnaire (HAQ) increased progressively across disease states but were unable to distinguish patients with arthralgia who progressed to PsA from those who remained stable. What discriminated were ultrasound-detected active synovitis (odds ratio: 3.40; p=0.019) and active enthesitis (odds ratio: 3.45; p=0.049), alongside older age. Progression to clinically manifest PsA occurred a mean of 13.7 months after enrolment.

> **If weight reframed what we treat, imaging reframed when we should act and ultrasound was the connecting thread**

The diagnostic counterpart was the DUET study, a 17-centre effort to build and validate a PsA-specific sonographic enthesitis score.[5] Across 213 patients with early PsA, 100 with psoriasis, and 106 controls, the optimal score combined weighted inflammatory and structural lesions at the Achilles, patellar, and triceps insertions. Performance was modest in the whole cohort (area under the curve: 0.65; age-specific specificity: 73–74%; sensitivity: 47–49%) but improved in the validation cohort (specificity up to 100%) and in patients with tender entheses (sensitivity up to 63%). Two messages matter for practice. First, DUET is a specificity-oriented adjunct best deployed in symptomatic entheses rather than a standalone screen. Second, the finding that large-joint enthesitis is not a universal feature of early PsA tempers the assumption that the enthesis is uniformly the earliest target and refines where the probe should be placed.

THE THERAPEUTIC CEILING: COMPARISON AND COMBINATION

On treatment itself, EULAR 2026 moved past placebo comparisons. The late-breaking BE BOLD trial was the first head-to-head study of an IL-17A/F inhibitor against an IL-23 inhibitor in PsA.[6] Among 553 patients, bimekizumab was superior to risankizumab for the primary endpoint of ACR50 at Week 16 (49.1% versus 38.4%; p=0.0078), with early separation by Week 4 (19.9% versus 7.2%). The hierarchy stopped at the first ranked secondary endpoint, however, as MDA did not differ significantly (43.0% versus 39.9%; p=0.4408). The result narrows, but does not close, the mechanistic debate, since a joint-focused advantage at 16 weeks must be weighed against tolerability and longer-term outcomes.

The combination strategy advanced on two fronts. Exploratory MRI from the Phase IIa AFFINITY study, in TNF-inhibitor-inadequate responders, showed numerically greater reductions in PsAMRIS inflammation in hand and foot phalangeal joints with guselkumab plus golimumab than with guselkumab alone, with stable structural damage and no

separation at the heel enthesis.[7] The paired biomarker analysis found that reductions in TNF activity, C-reactive protein, and IL-6 tracked with combination therapy and that responders carried a higher baseline inflammatory burden, suggesting the dual approach may raise the efficacy ceiling selectively in the most inflamed patients.[8] Building on this logic, the COMBo trial design was presented: a Phase IV, placebo-controlled study of deucravacitinib added to ongoing TNF-inhibitor therapy in difficult-to-control PsA, powered to detect a doubling of low-disease-activity attainment.[9] With recruitment still to come, it will provide the first controlled test of dual TNF and TYK2 inhibition.

Taken together, EULAR 2026 sketched a more dimensional PsA: a disease in which the patient's metabolic state, imaging phenotype, and the combinatorial logic of immunomodulation are converging on the same goal of deeper, earlier, and more durable control. The trials that will test whether this integration improves long-term outcomes are, fittingly, still to come.

References

1. Garaïman A et al. Lighten the load: artificial intelligence reveals BMI outranks treatment in one-year PsA outcomes—findings from the SPEED trial. Abstract OP0187. EULAR Congress, 3-6 June, 2026.
2. Coates LC et al. Ixekizumab and concomitant tirzepatide achieved early disease control in adults with PsA and obesity/overweight: phase 3b TOGETHER-PsA trial. Abstract OP069. EULAR Congress, 3-6 June, 2026.
3. Eder L et al. Dietary Interventions in Psoriatic Arthritis (DIPSA): a randomised controlled clinical trial. Abstract OP070. EULAR Congress, 3-6 June, 2026.
4. Zabotti A et al. Clinical and imaging features of subclinical and early PsA reveal synovio-entheseal inflammation as a marker of psoriatic arthralgia progression. Abstract OP0183. EULAR Congress, 3-6 June, 2026.
5. Eder L et al. The development and validation of a diagnostic ultrasound enthesitis tool (duet) for psoriatic arthritis. Abstract OP0180. EULAR Congress, 3-6 June, 2026.
6. McInnes IB et al. Bimekizumab efficacy and safety versus risankizumab in active PsA: 16-week results from the head-to-head BE BOLD study. Abstract LB0001. EULAR Congress, 3-6 June, 2026.
7. Wellen J et al. Effect of guselkumab and golimumab combination therapy on MRI-detected inflammation and damage in PsA: findings from the phase 2a AFFINITY study. Abstract OP0186. EULAR Congress, 3-6 June, 2026.
8. McInnes IB et al. Biomarker-driven insights from the phase 2a AFFINITY study evaluating guselkumab + golimumab combination versus guselkumab monotherapy in PsA. Abstract POS0479. EULAR Congress, 3-6 June, 2026.
9. Merola JF et al. The COMBo study: a randomised trial of deucravacitinib–TNF inhibitor combination versus TNF inhibitor monotherapy in difficult-to-control psoriatic disease. Abstract POS0499. EULAR Congress, 3-6 June, 2026.