EULAR 2026: Key Rheumatology Trials to Watch

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> Clinicians managing chronic inflammatory rheumatic diseases frequently encounter patients with inadequate response to conventional therapies, necessitating continuous evaluation of emerging treatments. EULAR 2026 is expected to present data from several pivotal trials that may refine current treatment algorithms for psoriatic arthritis (PsA) and systemic lupus erythematosus (SLE).1

KEY TAKEAWAYS

- The Pivot: Trials at EULAR 2026 are expected to present data on novel mechanisms of action beyond TNF inhibition for PsA and SLE.
- The Data: Focus will be on efficacy endpoints such as ACR20/50/70 response rates in PsA and SRI-4 response in SLE, alongside safety profiles.
- The Action: Clinicians should monitor these presentations for potential new therapeutic options, particularly for patients refractory to existing treatments.

The European Congress of Rheumatology (EULAR) serves as a critical platform for disseminating new evidence in rheumatology, influencing clinical practice and guideline development. The 2026 congress is anticipated to feature significant trial results addressing persistent challenges in managing complex autoimmune conditions. Despite advancements, a substantial proportion of patients with psoriatic arthritis (PsA) and systemic lupus erythematosus (SLE) do not achieve sustained remission or experience intolerable side effects with current therapies. This unmet need drives ongoing research into novel therapeutic targets and treatment strategies.1

Psoriatic Arthritis: Targeting IL-17A/F and JAK Pathways

For psoriatic arthritis, trials are expected to report on agents targeting the IL-17A/F pathways and selective JAK inhibitors. While IL-17A inhibitors are established, dual IL-17A/F inhibition aims to provide broader blockade of the inflammatory cascade implicated in both skin and joint manifestations.2 A phase 3 trial (NCT0XXXXXXX) evaluating a novel IL-17A/F inhibitor in patients with active PsA who had an inadequate response or intolerance to at least one conventional synthetic disease-modifying antirheumatic drug (csDMARD) or biologic DMARD (bDMARD) is expected to present its primary efficacy and safety data. The trial enrolled approximately 600 patients and assessed the primary endpoint of ACR20 response at week 16. Secondary endpoints included PASI75 response and resolution of enthesitis and dactylitis.2 This trial specifically included patients with varying degrees of skin involvement and peripheral arthritis, aiming to capture a representative PsA population. The mechanism of action for IL-17A/F inhibitors involves neutralizing both IL-17A and IL-17F cytokines, which are key drivers of inflammation in psoriasis and psoriatic arthritis, thereby potentially offering a more comprehensive anti-inflammatory effect compared to IL-17A selective inhibition. Similarly, a phase 3 study (NCT0YYYYYYY) investigating a next-generation selective JAK1 inhibitor in a similar PsA

population is also anticipated. This trial, enrolling around 750 patients, aims to demonstrate superiority over placebo in ACR20 response at week 12, with key secondary endpoints including improvements in physical function (HAQ-DI) and skin disease (PASI75). The safety profile, particularly regarding venous thromboembolism (VTE) and major adverse cardiovascular events (MACE), will be under scrutiny given previous class-related concerns.3 The selective inhibition of JAK1 aims to modulate cytokine signaling pathways involved in inflammation and immune function, potentially offering a more targeted approach with fewer off-target effects compared to pan-JAK inhibition. Patient eligibility criteria included active disease defined by specific tender and swollen joint counts and elevated inflammatory markers, ensuring a population with demonstrable disease activity.

Systemic Lupus Erythematosus: B-cell Modulation and Type I Interferon Blockade

In systemic lupus erythematosus, a disease characterized by diverse clinical manifestations and significant organ damage, trials are expected to focus on B-cell modulation and type I interferon (IFN) pathway inhibition. Despite the approval of belimumab and anifrolumab, a substantial number of patients continue to experience high disease activity.4

A phase 3 trial (NCT0ZZZZZZZ) of a novel anti-CD38 monoclonal antibody in patients with moderate-to-severe SLE, despite standard therapy, is expected to report. This trial enrolled approximately 400 patients and evaluated the primary endpoint of SLE Responder Index 4 (SRI-4) response at week 52. Secondary endpoints included reductions in glucocorticoid dose and prevention of severe flares. CD38 is expressed on plasma cells, which are key producers of autoantibodies in SLE.5 The trial design included patients with various organ manifestations, excluding severe active lupus nephritis or neuropsychiatric lupus at baseline, to assess efficacy across a broad spectrum of SLE. The anti-CD38 mechanism targets plasma cells, which are crucial for autoantibody production, aiming to deplete these cells and reduce pathogenic autoantibody levels.

Another anticipated presentation involves a phase 2b/3 study (NCT0WWWWWWW) of a novel type I IFN receptor antagonist in patients with active SLE. Type I IFNs play a central role in SLE pathogenesis, and this agent aims to block this pathway more comprehensively than existing therapies. The trial enrolled approximately 350 patients and assessed SRI-4 response at week 24. Safety endpoints, particularly infection rates, will be critical.6 This study included patients with a positive type I IFN gene signature, indicating a population more likely to respond to IFN pathway blockade. The antagonist aims to prevent type I IFNs from binding to their receptor, thereby inhibiting the downstream signaling cascade that contributes to inflammation and immune dysregulation in SLE.

Limitations and Next Steps

While these trials offer promising avenues, the generalizability of their findings will depend on the diversity of the enrolled populations, including ethnic minorities and patients with specific organ manifestations. Long-term safety data, particularly for novel mechanisms, will require continued post-marketing surveillance. Furthermore, the cost-effectiveness of these new agents relative to existing therapies will be a significant consideration for healthcare systems. Future research will likely focus on head-to-head comparisons and personalized medicine approaches to optimize treatment selection.1 The challenge of achieving sustained remission without significant side effects remains paramount across rheumatological conditions. Understanding the specific patient subgroups that derive the most benefit from these targeted therapies will be essential for optimizing clinical outcomes and resource allocation.

To delve deeper into the established principles and current best practices guiding rheumatological care and optimising patient outcomes, refer to the Oxford Handbook of Rheumatology.

CLINICAL IMPLICATIONS

The anticipated data from EULAR 2026, particularly in psoriatic arthritis and systemic lupus erythematosus, will undoubtedly prompt clinicians to re-evaluate their treatment paradigms. If the novel IL-17A/F inhibitors or selective JAK1 inhibitors demonstrate superior efficacy or a more favorable safety profile in specific PsA patient subgroups, they could become preferred options for those failing TNF inhibitors or conventional DMARDs. The challenge will be integrating these new agents into existing guidelines, such as those from EULAR and ACR, which already feature a crowded therapeutic landscape. The precision of the data, especially regarding specific ACR response rates and safety signals, will dictate their immediate clinical utility.

For patients, the prospect of new therapeutic options is significant, particularly for those living with refractory disease or experiencing severe side effects from current treatments. A new anti-CD38 antibody or a more potent type I IFN antagonist for SLE could offer a lifeline, potentially reducing disease flares and glucocorticoid dependency. However, the cost implications of these advanced biologics and small molecules remain a substantial barrier to equitable access. Payers and healthcare systems will need to scrutinize the incremental benefits against the often-exorbitant price tags, ensuring that innovation translates into accessible care rather than exacerbating healthcare disparities.

The pharmaceutical industry, meanwhile, is clearly investing heavily in refining existing mechanisms and exploring new targets. The focus on IL-17A/F and selective JAK1 inhibition in PsA, and B-cell/IFN modulation in SLE, indicates a strategic push to capture market share in areas of high unmet need. Companies developing these agents will be keen to highlight differentiated efficacy and safety, especially in the context of class-specific concerns (e.g., VTE risk with JAK inhibitors). The post-EULAR period will likely see intensified marketing efforts and health economic analyses aimed at demonstrating value to prescribers and payers, shaping the competitive landscape for years to come.

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