



Deposited via The University of Sheffield.

White Rose Research Online URL for this paper:

<https://eprints.whiterose.ac.uk/id/eprint/244922/>

Version: Published Version

Article:

Osunbor, O., Nkeck, J.R. and Adebajo, A.O. (2026) Investigational IL-17A antagonist in the treatment of psoriatic arthritis - a review. *Expert Opinion on Investigational Drugs*, 35 (8). pp. 581-589. ISSN: 1354-3784

<https://doi.org/10.1080/13543784.2026.2710840>

© 2026 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group. This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited, and is not altered, transformed, or built upon in any way. The terms on which this article has been published allow the posting of the Accepted Manuscript in a repository by the author(s) or with their consent.

Reuse

This article is distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs (CC BY-NC-ND) licence. This licence only allows you to download this work and share it with others as long as you credit the authors, but you can't change the article in any way or use it commercially. More information and the full terms of the licence here: <https://creativecommons.org/licenses/>

Takedown

If you consider content in White Rose Research Online to be in breach of UK law, please notify us by emailing eprints@whiterose.ac.uk including the URL of the record and the reason for the withdrawal request.

Investigational IL-17A antagonist in the treatment of psoriatic arthritis - a review

Osariemen Osunbor , Jan René Nkeck & Adewale O. Adebajo

To cite this article: Osariemen Osunbor , Jan René Nkeck & Adewale O. Adebajo (2026) Investigational IL-17A antagonist in the treatment of psoriatic arthritis - a review, Expert Opinion on Investigational Drugs, 35:8, 581-589, DOI: [10.1080/13543784.2026.2710840](https://doi.org/10.1080/13543784.2026.2710840)

To link to this article: <https://doi.org/10.1080/13543784.2026.2710840>



© 2026 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group.



Published online: 29 Jul 2026.



Submit your article to this journal [↗](#)



Article views: 120



View related articles [↗](#)



View Crossmark data [↗](#)

REVIEW



Investigational IL-17A antagonist in the treatment of psoriatic arthritis - a review

Osariemen Osunbor^a, Jan René Nkeck^b and Adewale O. Adebajo ^{a,c}

^aDepartment of Rheumatology, Barnsley Hospital NHS Foundation Trust, South Yorkshire, UK; ^bFaculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon; ^cSchool of Medicine and Population Health, University of Sheffield, South Yorkshire, UK

ABSTRACT

Introduction: The global burden of psoriatic arthritis (PsA) is considerable, with major effects on both quality of life and life expectancy. Approximately 40–50% of patients do not achieve the established therapeutic targets. Challenges related to tolerance, compliance, and therapeutic escape persist.

Areas covered: This study is a comprehensive review of the current literature analyzing the central role of interleukin-17 (IL-17) in the pathogenesis of PsA and emphasizing current and investigational therapies for IL-17A blockade.

Expert opinion: The current stage of development and expansion of IL-17A antagonists encompasses the third generation of such molecules. This generation includes small, injectable molecules such as sonelokimab and izokibep; oral molecules; and bispecific monoclonal antibodies. This approach is purported to facilitate enhanced tissue diffusion, augmented efficacy in refractory forms, and simplified utilization. The future of PsA treatments is contingent upon the implementation of precision medicine. The overarching objective is no longer to treat to target, but rather to treat to intercept, while ensuring the highest rate of control and tolerance for the patient.

ARTICLE HISTORY

Received 17 December 2025
Accepted 24 July 2026

KEYWORDS

Bispecific monoclonal antibodies; interleukin 17A antagonists; psoriatic arthritis; small molecules; third generation

1. Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease characterized by musculoskeletal manifestations of spondylarthritis, which may be peripheral in the form of synovitis, enthesitis, dactylitis, and/or axial, found in patients with personal psoriasis most often, or less commonly, familial psoriasis [1–3]. PsA affects 0.05% to 0.25% of the general population as well as approximately 30% of those with cutaneous manifestation of psoriasis [4,5]. It is a significant source of healthcare expenditure, with considerable direct and indirect costs. The condition, which is sometimes referred to as psoriatic disease, can lead to premature and even sustained disability and it is often associated with therapeutic failure [6]. It is a potentially debilitating condition due to its osteoarticular damage, which causes functional disability, and its systemic consequences, particularly metabolic syndrome, cardiovascular diseases and events [7–11]. All these consequences make PsA a polymorbid condition and significantly impact the quality and life expectancy of patients who suffer from it [12–15]. Despite pharmacological advances, the management of patients with PsA remains a significant challenge due to several diagnostic and therapeutic gaps. Delayed diagnosis leads to a loss of therapeutic opportunity. Artificial Intelligence (AI) based approaches including machine learning algorithms applied to electronic health records, imaging and biomarker profiling have the potential to improve early diagnosis, risk stratification and individualized treatment selection. The integration of AI into clinical pathways may therefore enhance timely initiation of IL-17A antagonist therapy, improve long-term outcome and reduced disease burden in routine practice [16–

18]. The difficulties of clinical assessment reflect the inadequacy of current tools for evaluating patients holistically [19,20]. Finally, in terms of treatment, there are significant limitations, as nearly two out of five patients will not respond to the innovative treatments currently available, sometimes leaving them with no effective therapeutic options [21–23].

1.1. Current therapeutic arsenal for PsA

The most commonly agreed therapeutic objective is based on the ‘Treat-to-Target’ (T2T) strategy, codified by international recommendations (EULAR: European League of Associations for Rheumatology; ACR: American College of Rheumatology; GRAPPA: Group for Research and Assessment of Psoriasis and Psoriatic Arthritis), with the goal of achieving clinical remission or, failing that, a low level of activity, thereby preventing or slowing structural damage and improving quality of life [1,22,24,25]. This involves comprehensive, multidisciplinary care including non-pharmacological measures, pharmacological treatments, physical treatments, and where required, surgical management [26–28]. Pharmacological treatment is at the heart of the therapeutic strategy and is carried out according to an algorithm based on the clinical phenotype of the disease (peripheral, axial, with enthesitis, and/or with dactylitis). First-line treatments are symptomatic treatments, non-steroidal anti-inflammatory drugs (NSAIDs), and glucocorticoid injections [28,29]. For peripheral forms, conventional synthetic disease-modifying antirheumatic drugs

CONTACT Adewale O. Adebajo  a.o.adebajo@sheffield.ac.uk  Department of Rheumatology, Barnsley Hospital NHS Foundation Trust, South Yorkshire S11 9RX, UK

© 2026 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group.
This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited, and is not altered, transformed, or built upon in any way. The terms on which this article has been published allow the posting of the Accepted Manuscript in a repository by the author(s) or with their consent.

Article highlights

- PsA is a debilitating systemic disease that remains complex to treat; approximately 40% of patients do not respond to current innovative treatments or experience therapeutic escape.
- Interleukin-17A plays a pivotal role in the mediation of synovial inflammation, bone erosion, and enthesitis, which are hallmarks of the pathogenesis of PsA.
- First- and second-generation IL-17A antagonists (secukinumab, ixekizumab, brodalumab, and bimekizumab) have already transformed the functional prognosis by targeting inflammation and structural damage.
- A third generation of molecules, including nanobodies with high tissue diffusion (sonelokimab, izokibep) and oral forms (DC-806, SAR441566), aims to improve compliance and efficacy in refractory forms.
- The future of treatment is moving toward a personalized medicine approach, which relies on biomarkers such as C-reactive protein (CRP) levels and Human Leukocyte Antigen B27 (HLA-B27) status and combination therapies to achieve complete remission with halting progression and residual disease rather than controlling symptoms.

(csDMARDs) are initiated as early as possible, with methotrexate as the drug of choice, followed by other csDMARDs including most commonly, leflunomide and sulfasalazine. These treatments, however, have little effect on axial disease and enthesitis [30–32]. In cases of failure, intolerance to csDMARDs, or from the outset in severe axial and enthesitic forms, innovative advanced drug therapies are the cornerstone of treatment. Among innovative treatments, tumor necrosis factor alpha (TNF alpha) inhibitors (adalimumab, etanercept, certolizumab, golimumab, infliximab) have the longest and most robust data [33–37]. Among the most recent therapies, are IL-17 antagonists (secukinumab, ixekizumab, brodalumab, bimekizumab), which appear to be more effective and faster acting [38–42]; IL-23 antagonists (guselkumab, risankizumab, tildrakizumab) and IL-12/23 antagonists (ustekinumab) have significant benefits in cases of major skin damage or chronic inflammatory bowel disease and a favorable safety profile, particularly in terms of cardiovascular health [43–48]. Small oral molecules (tsDMARDs) that have significantly improved comfort and treatment compliance for patients who dislike injectable therapies: JAK inhibitors (tofacitinib, upadacitinib, filgotinib) with rapid action and benefits, particularly in refractory forms, and apremilast (phosphodiesterase 4 (PDE4) inhibitor) in mild to moderate forms of psoriatic arthritis [49–54]. The choice of treatment is made as part of a shared decision-making process and personalized medicine, guided by the patient's profile and preferences, taking into account the available evidence [55,56].

1.2. Limitations of current treatments for PsA

Despite the current therapeutic arsenal, the management of PsA faces significant obstacles and limitations, which justify the ongoing search for new targets and therapeutic innovations. The main limitation is the ceiling of efficacy of current innovative therapies. Clinical trials and real-world data show that approximately 40–50% of patients do not achieve the

therapeutic target using current innovative treatments [1,23,57,58]. In addition, maintaining the response remains precarious due to secondary therapeutic response escape, linked to the immunogenicity of certain molecules, which necessitates cycling between classes often with decreasing efficacy with each new line of treatment [59,60]. Several therapies also have their efficacy impacted by certain comorbidities, for example with obesity and anti-TNF agents [61]. A second limitation concerns the safety and tolerance of current innovative treatments. Each therapeutic class is associated with specific risks that complicate decision-making depending on the patient's condition, which is most often polymorbid. Anti-TNF agents, which are one of the most widely used therapeutic classes in PsA, are associated with a high risk of severe infections (included tuberculosis, opportunistic infections) and congestive heart failure [62–64]. IL-17 antagonists, although highly effective in treating cutaneous psoriasis, are associated with a high incidence of mucocutaneous candidiasis and are contraindicated in cases of active inflammatory bowel disease [65–67]. JAK antagonist increase the risk of shingles, venous thromboembolic events, and major cardiovascular events, thus limiting their use in elderly patients or those at high to very high cardiovascular risk [68,69]. A third limitation is that treatment adherence remains a significant challenge. The digestive side effects of oral treatments such as methotrexate or apremilast often lead to early discontinuation of potentially effective treatment. Similarly, fatigue associated with chronic injections and pain at the injection site affect adherence to subcutaneous biotherapies [70]. In addition, there remains the issue of patients in biological remission who continue to experience debilitating residual pain and, above all, central fatigue and sleep disorders, suggesting central sensitization mechanisms not covered by current treatments [19]. These multiple limitations raise the need for new treatments under development, with a view to diversifying the therapeutic arsenal and offering clinicians and patients more therapeutic options.

1.3. Rationale for interleukin-17 inhibition in PsA

Interleukin-17 (IL-17) is a proinflammatory cytokine of which there are currently six isoforms (A to F). Isoforms A and F are the best known in pathology. They are expressed by T helper 17 lymphocytes (Th17) and share a high degree of structural and functional homology [71]. The IL-17A isoform is a key cytokine in the pathogenesis of PsA [3,65,72]. Genetically, there is an association between polymorphism of the IL-23/IL-17 pathway genes and susceptibility to psoriasis and PsA [73]. Animal models have shown that dysfunction of the IL-17 pathway provides sufficient elements to induce inflammation as seen in psoriasis [74]. Fundamentally, this cytokine is produced mainly by Th17 lymphocytes, gamma-delta T lymphocytes, and type 3 innate lymphoid cells, sometimes under the influence of IL-23 [75–77]. Pathophysiologically, IL-17 targets synoviocytes, osteoclasts, and keratinocytes, causing local inflammation and stimulating the production of TNF-alpha, interleukin-6, and chemokines [24,78–80]. This activity drives synovial proliferation, cartilage degradation, and bone erosion, while also contributing to the characteristic skin

lesions seen in psoriatic disease. In PsA, IL-17 induces osteoclastogenesis and bone erosion by stimulating RANKL, and secondarily leads to ossification at the enthesis [81–83]. IL-17F is structurally similar to IL-17A and also contributes to inflammation. While generally less potent than IL-17A, IL-17F plays a complementary role in sustaining joint and skin inflammation in psoriatic disease. Epidemiological data corroborate this key role by highlighting elevated serum and synovial IL-17 levels in patients with PsA, positively associated with disease activity, axial involvement, and severity of skin involvement [28,73].

1.4. Interleukin-17 antagonists used in PsA

Interleukin-17 antagonists represent the most significant therapeutic innovation in PsA in recent decades. They are grouped into three classes according to their mechanism of action: selective IL-17A antagonists, IL-17 receptor (IL-17RA) antagonists, and IL-17A/ F antagonists. They have transformed the functional prognosis of PsA with superior efficacy on symptoms and inflammation, and also reduce structural damage and improve enthesitis better than conventional treatments and other innovative advanced therapies [39,41,84,85]. Consequently, they occupy a prominent place in the management of PsA.

1.5. Selective IL-17A antagonists

These are the first generation of IL-17 antagonists. They are monoclonal antibodies that bind specifically to the IL-17A homodimer, preventing it from binding to its receptor. This class includes secukinumab and ixekizumab. Secukinumab is a fully human monoclonal antibody (IgG1 κ). The data supporting its approval come mainly from the pivotal trials FUTURE 1, 2, and 5, ERASURE, and FIXTURE [38,84,86]. It is indicated for plaque psoriasis, PsA, and ankylosing spondylitis, as it has demonstrated clear efficacy in sacroiliitis and inflammatory back pain [86,87]. Ixekizumab is a humanized monoclonal antibody (IgG4). The data supporting its validation come mainly from the UNCOVER 1, 2, and 3 trials published in 2016, and the SPIRIT-P1, 2, and H2H trials. It is indicated for psoriasis and PsA, particularly in patients who have failed or are intolerant to anti-TNF agents [39,88,89].

1.6. IL-17 receptor antagonists (IL-17RA)

These inhibit the A subunit of the IL-17 receptor, preventing the binding of several IL-17 isoforms (A, F, A/F, E). The only one currently available on the market is Brodalumab. It is a fully human monoclonal antibody (IgG2). The main data on its use comes from the AMAGINE 1, 2, and 3 trials published in 2015. It has been shown to be effective in treating enthesitis and dactylitis that are refractory to conventional treatments [40,90]. However, it should be noted that there have been several reports of psychiatric events in people receiving this drug [90].

1.7. Dual IL-17A and F antagonists

These are the latest generation of IL-17 antagonists on the market. They provide bimodal inhibition targeting IL-17A and F, which gives them a superior response to Secukinumab and Ustekinumab. Bimekizumab is a humanized monoclonal antibody (IgG1). Data on its use comes mainly from the BE VIVID and BE READY trials published in 2021. However, its use is associated with a higher incidence of oral candidiasis, although this risk remains manageable in clinical practice [91].

1.8. Interleukin-17A antagonists in development

Despite the efficacy of IL-17A antagonists currently available on the market, approximately one-third of patients do not respond optimally and develop resistance mechanisms, including accelerated clearance, immunogenicity, and the exploitation of other cytokine pathways [92]. These challenges are being exploited by the pharmaceutical industry to develop a further generation of IL-17 antagonists. In order to develop what will represent the third generation of IL-17 antagonists, technological innovation is currently focusing more on the use of small molecules, oral molecules, and bispecific antibodies. These developments aim to improve the tissue specificity of the drugs used and limit long-term failures. Table 1 presents the molecules under development among IL-17 antagonists.

1.9. Small injectable molecules

These offer better tissue diffusion. The two most promising molecules at present are Sonelokimab (M1095) and Izokibep (ABY-035). Sonelokimab is a humanized trivalent Nanobody - A single-domain antibody (sdAb) with a low molecular weight (40kDa). Its structure contains three binding domains: one for IL-17A, one for IL-17F, and one for albumin. This trivalent combination provides dual inhibition of IL-17 and improves its diffusion and half-life. The most conclusive Phase 2 trials on this molecule are the MIRA and ARGO trials; these studies observed a good response using the ACR50 (66% at 12 weeks) and PASI100 criteria. It is considered the most promising molecule in development, with better activity in the phase 3 VELA study, particularly on enthesitis [93–100]. It would also be effective on residual local inflammation during PsA. Izokibep (ABY-035) is a small molecule consisting of a very small mimetic antibody (18 kDa) that binds specifically and with very high affinity to IL-17A. This affinity is in the femtomolar range. Due to its small size, it also offers rapid and impressive distribution, allowing very high tissue concentrations to be achieved at low doses. It has demonstrated its efficacy in phase 2b and 3 clinical trials on PsA, uveitis, and Verneuil's disease [101–107]. It has the most long-term data, at least 52 weeks, with rapid efficacy on dactylitis [95].

1.10. Small oral molecules

Oral therapies based on small molecules will undoubtedly help improve compliance, which is a significant step forward [108]. Currently, the most promising molecules are DC-806, DC-853, SAR441566, and LEO 153339. DC-806 and DC-853

Table 1. Current investigational IL-17 antagonists in the treatment of psoriatic arthritis.

Molecule (code)	Class (structure)	Target(s)	Laboratory / developer	Stage(phase)	Potential advantages	Potential disadvantages /Risks	References
Sonelokimab (MT095)	Nanobody® Trivalent (Anti-IL17A/F + Albumin)	IL-17A, IL-17F	MoonLake Immunotherapeutics	Phase 3	Highly effective at penetrating deep tissue, better inflammation control, prolonged half-life	High risk of candidiasis	[38,84,86]
Izokibep (ABY-035)	Affibody®, Small mimetic protein	IL-17A	Acelyrin / Affibody AB	Phase 2b/3	Highly potent, small size, minimal injection volume	Risk of immunogenicity	[95]
DC-806 / DC-853	Small oral molecule	IL-17A (Antagoniste)	Eli Lilly	Phase 2	Oral administration, lower production cost	Potentially variable bioavailability with possible gastrointestinal effects	[106,109–111]
Vunakizumab (SHR-1314)	Humanized mAb (IgG1)	IL-17A	Jiangsu Hengrui Medicine	Phase 3	Long half-life, distinct epitope	Similar to existing anti-IL17A drugs	[115–117]
Xeligekimab (GR1501)	Humanized mAb (IgG1)	IL-17A	Genrix (Innovent Biologics)	Phase 2b/3	Long half-life, high affinity	Similar to existing anti-IL17A drugs	[119]
Gumokimab (AK111)	Humanized mAb	IL-17A	Akero, Inc	Phase 2/3	Good affinity, low cost	Similar to existing anti-IL17A drugs Potential risk for exacerbation of Crohn disease or ulcerative colitis	[118]
Netakimab (BCD-085)	Humanized mAb (modified Fc)	IL-17A	Biocad	Phase 3	Better safety, cost-effective	Limited data	[120–122]
SAR441566	Small oral molecule	IL-17A	Sanofi	Phase 2	Oral alternative, macrocyclic stability	Limited data on efficacy	[112,113]
Tibulizumab	Bispecific mAb	IL-17 + BAFF	Eli Lilly	Phase 2	Combined targeting of T and B pathways	Compound complexity, risk of infections	[124]

Abbreviations: BAFF, B-cell activating factor; IL, interleukin; mAb, monoclonal antibody.

are IL-17 antagonists that prevent binding to its receptor with phase 2 efficacy similar to injectable treatments [106,109–111]. SAR441566 is an IL-17 inhibitor developed using a special stabilization technique known as macrocyclic, which gives it significant bioavailability after oral administration [112,113]. LEO 153339 is still in the preliminary stages of development [114].

1.11. Bispecific antibodies

These essentially provide a lasting effect and limit resistance. Promising molecules include Vunakizumab, Xeligekimab (GR1501), Gumokimab (AK111), Netakimab, and Tibulizumab. Vunakizumab is a humanized monoclonal antibody that binds specifically to the IL-17A homodimer on different epitopes, which improves its affinity; it is currently in phase 3 development [115–117]. Xeligekimab and Gumokimab are also humanized monoclonal antibodies targeting IL-17A in phase 2/3 development, offering good affinity and a good tolerance profile with lower development costs for Gumokimab [118,119]. Netakimab also targets IL-17A with a modified Fc fragment to reduce its immunogenicity [120–122]. It is currently in phase 3 trials. Finally, Tibulizumab has the specificity of targeting both IL-17 and BAFF (B-cell activating factor), offering efficacy in patients with PsA with autoantibodies or overlapping with lupus or Sjogren's disease. Currently in phase 2 trials [123,124].

1.12. Other innovations in development

These relate to techniques for administering these molecules using self-injection devices that do not require needles or, at most, micro-needles, which would also improve compliance, as well as other formulations, particularly inhaled ones, which are more effective in treating associated lung damage, for example [125].

2. Methods

This article provides a structured search and narrative review of literatures in PubMed for the following key terms investigational IL-17A antagonist and Psoriatic arthritis and we aimed for at least 100 research papers published from 2005 onwards to provide a comprehensive and balanced overview of the subject. In addition, ClinicalTrials.gov databases focusing search by typing Psoriatic arthritis as the condition/disease and treatment as Intervention/treatment, then clicking the search button. Specifically, a review of the literature looking at the central role of interleukin-17 (IL-17) in the pathogenesis of PsA and emphasizing current and investigational therapies for IL-17A blockade were searched. Our literature search included studies in adults with psoriatic arthritis that reported clinical or safety results of investigational drugs that block IL-17A. We excluded studies that reported laboratory animals or single-case findings.

3. Conclusion

In order to confront the challenges posed by the burden of psoriatic arthritis (PsA) and the limitations of current innovative treatments, research into new molecules targeting interleukin-17A is rapidly expanding, as this cytokine plays a central role in the pathogenesis of PsA. These therapeutic advances initially focused on the dual inhibition of interleukin-17A and F by bimekizumab, representing the second generation of IL-17 antagonists. It has been shown to be more effective in controlling certain specific phenotypes of the disease.

The third generation is underway, encompassing nanobodies and small mimetic molecules, including sonelokimab and izokibep. These molecules offer enhanced tissue diffusion due to their low molecular weight and improved control of challenging manifestations and residual disease. Current innovations in the field primarily concentrate on the development of oral therapies, with the objective of enhancing patient comfort and compliance without compromising the efficacy of the therapeutic interventions. These promising small oral molecules (DC-806, SAR441566) also offer the benefit of reducing the risk of secondary escape related to immunogenicity, a benefit that monoclonal antibodies do not offer. In this generation, there is also an expectation of an expansion of bispecific antibodies and molecules targeting multiple epitopes.

These advances have evolved beyond the initial objective of merely mitigating symptoms to a new focus on achieving sustained and significant remission. These findings offer patients suffering from PsA the prospect of stabilization of their condition, effectively transforming a potentially debilitating disease into a condition that can be managed with targeted and innovative medicine.

4. Expert opinion

Recent research into investigational IL-17A antagonists, offers encouraging developments in the treatment of psoriatic arthritis (PsA). Current IL-17A blockers like secukinumab and ixekizumab are effective, but newer treatments may bring even better outcomes, including longer-lasting relief, fewer doses, improved control of symptoms, and possibly a greater acceptability with oral formulations. New IL-17A therapies, particularly those that block both IL-17A and IL-17F (like bimekizumab and sonelokimab), show promise in reducing inflammation more effectively. Phase III trials for bimekizumab have already shown better outcomes than several existing drug treatments. Izokibep, a small molecule, could offer the added benefit of monthly dosing, making it easier for patients to adhere to the treatment plan. If these drugs prove safe in the long-term, they could significantly change treatment guidelines. Clinicians may choose to use them earlier in the disease process, or for patients who don't respond well to older biologics. There is also the potential to reduce treatment costs by improving drug effectiveness and decreasing the need for additional therapies. Another benefit in refractory disease, is the possibility of combination therapies, particularly with anti-tumor necrosis factor (TNF) alpha agents, taking advantage of a synergistic effect.

However, moving these therapies into routine clinical practice takes time. Several of these therapies are still undergoing Phase II or III trials. Regulatory approval, pricing decisions, and national health system guidelines all influence whether and when, they will become widely used. Clinicians may also hesitate to adopt new drugs, until more long-term safety data is available.

One of the biggest challenges in current research is the lack of head-to-head trials comparing new agents directly with established ones. Most studies compare new treatments to placebo, which does not reflect, and is consequently of limited benefit in, everyday practice. We also lack good biomarkers to predict which patient will respond to which therapy, and in which ways. Better tools to enable personalized medicine, would improve both patient outcomes and cost-effectiveness.

From a technical standpoint, the development of oral IL-17A pathway inhibitors (like those targeting IL-17RA or ACT1) is an exciting but early-stage area. If these therapies can be made safe and effective, they may replace existing injections for some patients. To do this, oral therapies will also need to overcome challenges with specificity and systemic side effects. Research into dual inhibitors (IL-17A and IL-17F) needs to continue to determine whether broader immune blockade leads to better control, or simply more side effects. Importantly, the end goal should not be just symptom control, but true remission. Strategies such as interleukin 22 dosing or interleukin 17F genetic signature assessment, could predict therapeutic response and guide clinicians' future decisions on the choice of these targeted therapies.

IL-17A targeting remains one of the most promising approaches in PsA. However, it may not be the only solution. Other immune pathways, like IL-23 or TNF, also play roles in PsA and might be more relevant in certain patient groups. The future may lie in combining these initiatives, possibly through dual-target drugs. This whole field is moving toward precision medicine with an understanding of each patient's disease mechanisms, so as to choose the right drug from the start in terms of likely efficacy and safety profile. This could help control disease early and prevent progression. To achieve this, we need more integrated research, linking genetic predilection, immune profiles, predictive biomarkers and clinical outcomes. Within five years, we anticipate that several of these investigational IL-17A agents, especially bimekizumab and izokibep, will be in general routine clinical use. Standard practice and care will likely include drugs with fewer injections, better skin and joint control, as well as tailored choices based on patient profiles. Routine treatment may also start to include risk assessments for fungal infections or inflammatory bowel disease issues, especially in view of the known side effects of these drugs.

By 2030, the standard of care for treating PsA is likely have shifted dramatically. Biologic drugs could be given less frequently, or even injections replaced by oral preparations altogether. Clinicians may use personalized or clinical guidelines to match each patient with the best biologic from the beginning, based on genetic or biomarker testing. This approach will not only aim to treat symptoms, but in addition, will aim to halt disease progression and achieve true remission in larger numbers of patients.

Acknowledgments

Osariemen Osunbor (OO): Conceptualization, Writing – original draft, Writing – review & editing. Jan René Nkeck (JRN): Conceptualization, Writing – review & editing. Adewale O. Adebajo (AOA): Conceptualization, Supervision, Writing – review & editing. All author reviewed and approved the final version of the manuscript.

Author contributions

CRediT: **Osariemen Osunbor**: Conceptualization, Data curation, Formal analysis, Resources; **Jan René Nkeck**: Conceptualization, Data curation, Formal analysis, Resources, Software, Supervision; **Adewale O. Adebajo**: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.

Funding

This paper was not funded.

Disclosure statement

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

The authors declare that no generative AI tools were used in the preparation of this manuscript.

Reviewer disclosures

Peer reviewers on this manuscript have no relevant financial or other relationships to disclose.

ORCID

Adewale O. Adebajo  <http://orcid.org/0000-0001-7368-7083>

References

- Coates LC, Soriano ER, Corp N, et al. Group for research and assessment of psoriasis and psoriatic arthritis (GRAPPA): updated treatment recommendations for psoriatic arthritis 2021. *Nat Rev Rheumatol*. 2022;18(8):465–479. doi: [10.1038/s41584-022-00798-0](https://doi.org/10.1038/s41584-022-00798-0)
- Ritchlin CT, Colbert RA, Gladman DD. Psoriatic arthritis. *N Engl J Med*. 2017;376(10):957–970. doi: [10.1056/NEJMr1505557](https://doi.org/10.1056/NEJMr1505557)
- Veale DJ, Fearon U. The pathogenesis of psoriatic arthritis. *Lancet*. 2018;391(10136):2273–2284. doi: [10.1016/S0140-6736\(18\)30830-4](https://doi.org/10.1016/S0140-6736(18)30830-4)
- Alinaghi F, Calov M, Kristensen LE, et al. Prevalence of psoriatic arthritis in patients with psoriasis: a systematic review and meta-analysis of observational and clinical studies. *J Am Acad Dermatol*. 2019;80(1):251–65.e19.
- Ogdie A, Weiss P. The epidemiology psoriatic arthritis. *Rheum Dis Clin N Am*. 2015;41(4):545–568.
- Tillett W, de-Vries C, McHugh NJ. Work disability in psoriatic arthritis: a systematic review. *Rheumatology (Oxford)*. 2012;51(2):275–283. doi: [10.1093/rheumatology/ker216](https://doi.org/10.1093/rheumatology/ker216)
- Atzeni F, Rodríguez-Carrio J, Alciati A, et al. Cardiovascular risk in psoriatic arthritis: how can we manage it? *Autoimmun Rev*. 2025;24(11):103889. doi: [10.1016/j.autrev.2025.103889](https://doi.org/10.1016/j.autrev.2025.103889)
- Dey M, Nikiphorou E. Cardiovascular comorbidities in psoriatic arthritis: state of the art. *Ther Adv Musculoskelet Dis*. 2024;16:1759720X241274537. doi: [10.1177/1759720X241274537](https://doi.org/10.1177/1759720X241274537)
- Masson W, Lobo M, Molinero G. Psoriasis and cardiovascular risk: a comprehensive review. *Adv Ther*. 2020;37(5):2017–2033. doi: [10.1007/s12325-020-01346-6](https://doi.org/10.1007/s12325-020-01346-6)
- Gladman DD, Antoni C, Mease P, et al. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. *Ann Rheum Dis*. 2005;64(Suppl 2):ii14–7. doi: [10.1136/ard.2004.032482](https://doi.org/10.1136/ard.2004.032482)
- Chandran V, Raychaudhuri SP. Geoepidemiology and environmental factors of psoriasis and psoriatic arthritis. *J Autoimmun*. 2010;34(3):J314–21. doi: [10.1016/j.jaut.2009.12.001](https://doi.org/10.1016/j.jaut.2009.12.001)
- Kavanaugh A, Helliwell P, Ritchlin CT. Psoriatic arthritis and burden of disease: patient perspectives from the population-based multinational assessment of psoriasis and psoriatic arthritis (MAPP) survey. *Rheumatol Ther*. 2016;3(1):91–102. doi: [10.1007/s40744-016-0029-z](https://doi.org/10.1007/s40744-016-0029-z)
- James L, Hailey LH, Suribhatla R, et al. The impact of psoriatic arthritis on quality of life: a systematic review. *Ther Adv Musculoskelet Dis*. 2024;16:1759720X241295920. doi: [10.1177/1759720X241295920](https://doi.org/10.1177/1759720X241295920)
- Ogdie A, Yu Y, Haynes K, et al. Risk of major cardiovascular events in patients with psoriatic arthritis, psoriasis and rheumatoid arthritis: a population-based cohort study. *Ann Rheum Dis*. 2015;74(2):326–332. doi: [10.1136/annrheumdis-2014-205675](https://doi.org/10.1136/annrheumdis-2014-205675)
- Polachek A, Touma Z, Anderson M, et al. Risk of cardiovascular morbidity in patients with psoriatic arthritis: a meta-analysis of observational studies. *Arthritis Care Res (Hoboken)*. 2017;69(1):67–74. doi: [10.1002/acr.22926](https://doi.org/10.1002/acr.22926)
- Karmacharya P, Ogdie A, Eder L. Psoriatic arthritis and the association with cardiometabolic disease: a narrative review. *Ther Adv Musculoskelet Dis*. 2021;13:1759720X21998279. doi: [10.1177/1759720X21998279](https://doi.org/10.1177/1759720X21998279)
- Villani C, Schoenauer M, Bonnet Y, et al. Donner un sens à l'intelligence artificielle: Pour une stratégie nationale et européenne. Paris: Mission parlementaire; 2018. [Giving Meaning to Artificial Intelligence: For a National and European Strategy. Paris: Parliamentary Mission; 2018].
- Haroon M, Gallagher P, Fitzgerald O. Diagnostic delay of more than 6 months contributes to poor radiographic and functional outcome in psoriatic arthritis. *Ann Rheum Dis*. 2015;74(6):1045–1050. doi: [10.1136/annrheumdis-2013-204858](https://doi.org/10.1136/annrheumdis-2013-204858)
- Gudu T, Gossec L. Quality of life in psoriatic arthritis. *Expert Rev Clin Immunol*. 2018;14(5):405–417. doi: [10.1080/1744666X.2018.1468252](https://doi.org/10.1080/1744666X.2018.1468252)
- Dandorfer SWH, Rech J, Manger B, et al. Differences in the patient's and the physician's perspective of disease in psoriatic arthritis. *Semin Arthritis Rheum*. 2012;42(1):32–41.
- Mease P, Walsh JA, Baraliakos X, et al. Translating improvements with ixekizumab in clinical trial outcomes into clinical practice: ASAS40, pain, fatigue, and sleep in ankylosing spondylitis. *Rheumatol Ther*. 2019;6(3):435–450. doi: [10.1007/s40744-019-0165-3](https://doi.org/10.1007/s40744-019-0165-3)
- Smolen JS, Schöls M, Braun J, et al. Treating axial spondyloarthritis and peripheral spondyloarthritis, especially psoriatic arthritis, to target: 2017 update of recommendations by an international task force. *Ann Rheum Dis*. 2018;77(1):3–17. doi: [10.1136/annrheumdis-2017-211734](https://doi.org/10.1136/annrheumdis-2017-211734)
- Mease PJ, Karki C, Palmer JB, et al. Clinical characteristics, disease activity, and patient-reported outcomes in psoriatic arthritis patients with dactylitis or enthesitis: results from the corona psoriatic arthritis/spondyloarthritis registry. *Arthritis Care Res (Hoboken)*. 2017;69(11):1692–1699. doi: [10.1002/acr.23249](https://doi.org/10.1002/acr.23249)
- Gossec L, Baraliakos X, Kerschbaumer A, et al. EULAR recommendations for the management of psoriatic arthritis with pharmacological therapies: 2019 update. *Ann Rheum Dis*. 2020;79(6):700–712. doi: [10.1136/annrheumdis-2020-217159](https://doi.org/10.1136/annrheumdis-2020-217159)
- Singh JA, Guyatt G, Ogdie A, et al. Special article: 2018 American college of rheumatology/national psoriasis foundation guideline for the treatment of psoriatic arthritis. *Arthritis Rheumatol*. 2019;71(1):5–32. doi: [10.1002/art.40726](https://doi.org/10.1002/art.40726)
- Ogdie A, Coates LC, Gladman DD. Treatment guidelines in psoriatic arthritis. *Rheumatology (Oxford)*. 2020;59(Suppl 1):i37–46. doi: [10.1093/rheumatology/kez383](https://doi.org/10.1093/rheumatology/kez383)

27. Klingberg E, Bilberg A, Björkman S, et al. Weight loss improves disease activity in patients with psoriatic arthritis and obesity: an interventional study. *Arthritis Res Ther*. 2019;21(1):17. doi: [10.1186/s13075-019-1810-5](https://doi.org/10.1186/s13075-019-1810-5)
28. Coates LC, Helliwell PS. Psoriatic arthritis: state of the art review. *Clin Med (Lond)*. 2017;17(1):65–70. doi: [10.7861/clinmedicine.17-1-65](https://doi.org/10.7861/clinmedicine.17-1-65)
29. Cather JC, Crowley JJ. Use of biologic agents in combination with other therapies for the treatment of psoriasis. *Am J Clin Dermatol*. 2014;15(6):467–478. doi: [10.1007/s40257-014-0097-1](https://doi.org/10.1007/s40257-014-0097-1)
30. Coates LC, Helliwell PS. Methotrexate efficacy in the tight control in psoriatic arthritis study. *J Rheumatol*. 2016;43(2):356–361. doi: [10.3899/jrheum.150614](https://doi.org/10.3899/jrheum.150614)
31. Wilsdon TD, Whittle SL, Thynne TR, et al. Methotrexate for psoriatic arthritis. *Cochrane Database Syst Rev*. 2019;1(1):CD012722.
32. Nash P, Lubrano E, Cauli A, et al. Updated guidelines for the management of axial disease in psoriatic arthritis. *J Rheumatol*. 2014;41(11):2286–2289. doi: [10.3899/jrheum.140877](https://doi.org/10.3899/jrheum.140877)
33. Ritchlin CT, Kavanaugh A, Merola JF, et al. Bimekizumab in patients with active psoriatic arthritis: results from a 48-week, randomised, double-blind, placebo-controlled, dose-ranging phase 2b trial. *Lancet*. 2020;395(10222):427–440. doi: [10.1016/S0140-6736\(19\)33161-7](https://doi.org/10.1016/S0140-6736(19)33161-7)
34. Mease PJ, Gladman DD, Ritchlin CT, et al. Adalimumab for the treatment of patients with moderately to severely active psoriatic arthritis: results of a double-blind, randomized, placebo-controlled trial. *Arthritis Rheum*. 2005;52(10):3279–3289. doi: [10.1002/art.21306](https://doi.org/10.1002/art.21306)
35. Kavanaugh A, Mease PJ, Gomez-Reino JJ, et al. Longterm (52-week) results of a phase III randomized, controlled trial of apremilast in patients with psoriatic arthritis. *J Rheumatol*. 2015;42(3):479–488. doi: [10.3899/jrheum.140647](https://doi.org/10.3899/jrheum.140647)
36. Gladman DD, Mease PJ, Ritchlin CT, et al. Adalimumab for long-term treatment of psoriatic arthritis: forty-eight week data from the adalimumab effectiveness in psoriatic arthritis trial. *Arthritis Rheum*. 2007;56(2):476–488. doi: [10.1002/art.22379](https://doi.org/10.1002/art.22379)
37. Furst DE, Breedveld FC, Kalden JR, et al. Updated consensus statement on biological agents for the treatment of rheumatic diseases, 2007. *Ann Rheum Dis*. 2007;66(Suppl 3):iii2–iii22. doi: [10.1136/ard.2007.081430](https://doi.org/10.1136/ard.2007.081430)
38. McInnes IB, Mease PJ, Kirkham B, et al. Secukinumab, a human anti-interleukin-17A monoclonal antibody, in patients with psoriatic arthritis (FUTURE 2): a randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet*. 2015;386(9999):1137–1146. doi: [10.1016/S0140-6736\(15\)61134-5](https://doi.org/10.1016/S0140-6736(15)61134-5)
39. Mease PJ, van der Heijde D, Ritchlin CT, et al. Ixekizumab, an interleukin-17A specific monoclonal antibody, for the treatment of biologic-naïve patients with active psoriatic arthritis: results from the 24-week randomised, double-blind, placebo-controlled and active (adalimumab)-controlled period of the phase III trial SPIRIT-P1. *Ann Rheum Dis*. 2017;76(1):79–87. doi: [10.1136/annrheumdis-2016-209709](https://doi.org/10.1136/annrheumdis-2016-209709)
40. Mease PJ, Helliwell PS, Hjuler KF, et al. Brodalumab in psoriatic arthritis: results from the randomised phase III AMVISION-1 and AMVISION-2 trials. *Ann Rheum Dis*. 2021;80(2):185–193. doi: [10.1136/annrheumdis-2019-216835](https://doi.org/10.1136/annrheumdis-2019-216835)
41. McInnes IB, Asahina A, Coates LC, et al. Bimekizumab in patients with psoriatic arthritis, naïve to biologic treatment: a randomised, double-blind, placebo-controlled, phase 3 trial (BE OPTIMAL). *Lancet*. 2023;401(10370):25–37. doi: [10.1016/S0140-6736\(22\)02302-9](https://doi.org/10.1016/S0140-6736(22)02302-9)
42. Merola JF, Landewé R, McInnes IB, et al. Bimekizumab in patients with active psoriatic arthritis and previous inadequate response or intolerance to tumour necrosis factor- α inhibitors: a randomised, double-blind, placebo-controlled, phase 3 trial (BE COMPLETE). *Lancet*. 2023;401(10370):38–48. doi: [10.1016/S0140-6736\(22\)02303-0](https://doi.org/10.1016/S0140-6736(22)02303-0)
43. Deodhar A, Helliwell PS, Boehncke W-H, et al. Guselkumab in patients with active psoriatic arthritis who were biologic-naïve or had previously received TNF α inhibitor treatment (DISCOVER-1): a double-blind, randomised, placebo-controlled phase 3 trial. *Lancet*. 2020;395(10230):1115–1125. doi: [10.1016/S0140-6736\(20\)30265-8](https://doi.org/10.1016/S0140-6736(20)30265-8)
44. Mease PJ, Rahman P, Gottlieb AB, et al. Guselkumab in biologic-naïve patients with active psoriatic arthritis (DISCOVER-2): a double-blind, randomised, placebo-controlled phase 3 trial. *Lancet*. 2020;395(10230):1126–1136. doi: [10.1016/S0140-6736\(20\)30263-4](https://doi.org/10.1016/S0140-6736(20)30263-4)
45. Kristensen LE, Keiserman M, Papp K, et al. Efficacy and safety of risankizumab for active psoriatic arthritis: 24-week results from the randomised, double-blind, phase 3 KEPPsAKE 1 trial. *Ann Rheum Dis*. 2022;81(2):225–231. doi: [10.1136/annrheumdis-2021-221019](https://doi.org/10.1136/annrheumdis-2021-221019)
46. Östör A, Van den Bosch F, Papp K, et al. Efficacy and safety of risankizumab for active psoriatic arthritis: 24-week results from the randomised, double-blind, phase 3 KEPPsAKE 2 trial. *Ann Rheum Dis*. 2022;81(3):351–358. doi: [10.1136/annrheumdis-2021-221048](https://doi.org/10.1136/annrheumdis-2021-221048)
47. McInnes IB, Chakravarty SD, Apaolaza I, et al. Efficacy of ustekinumab in biologic-naïve patients with psoriatic arthritis by prior treatment exposure and disease duration: data from PSUMMIT 1 and PSUMMIT 2. *RMD Open*. 2019;5(2):e001006. doi: [10.1136/rmdopen-2019-000990](https://doi.org/10.1136/rmdopen-2019-000990)
48. Mease PJ, Chohan S, Fructuoso FJG, et al. Efficacy and safety of tildrakizumab in patients with active psoriatic arthritis: results of a randomised, double-blind, placebo-controlled, multiple-dose, 52-week phase IIb study. *Ann Rheum Dis*. 2021;80(9):1147–1157. doi: [10.1136/annrheumdis-2020-219014](https://doi.org/10.1136/annrheumdis-2020-219014)
49. Mease P, Hall S, FitzGerald O, et al. Tofacitinib or adalimumab versus placebo for psoriatic arthritis. *N Engl J Med*. 2017;377(16):1537–1550. doi: [10.1056/NEJMoa1615975](https://doi.org/10.1056/NEJMoa1615975)
50. Gladman D, Rigby W, Azevedo VF, et al. Tofacitinib for psoriatic arthritis in patients with an inadequate response to TNF inhibitors. *N Engl J Med*. 2017;377(16):1525–1536. doi: [10.1056/NEJMoa1615977](https://doi.org/10.1056/NEJMoa1615977)
51. McInnes IB, Anderson JK, Magrey M, et al. Trial of upadacitinib and adalimumab for psoriatic arthritis. *N Engl J Med*. 2021;384(13):1227–1239. doi: [10.1056/NEJMoa2022516](https://doi.org/10.1056/NEJMoa2022516)
52. Mease PJ, Lertratanakul A, Anderson JK, et al. Upadacitinib for psoriatic arthritis refractory to biologics: SELECT-PsA 2. *Ann Rheum Dis*. 2021;80(3):312–320. doi: [10.1136/annrheumdis-2020-218870](https://doi.org/10.1136/annrheumdis-2020-218870)
53. Mease P, Coates LC, Helliwell PS, et al. Efficacy and safety of filgotinib, a selective Janus kinase 1 inhibitor, in patients with active psoriatic arthritis (EQUATOR): results from a randomised, placebo-controlled, phase 2 trial. *Lancet*. 2018;392(10162):2367–2377. doi: [10.1016/S0140-6736\(18\)32483-8](https://doi.org/10.1016/S0140-6736(18)32483-8)
54. Edwards CJ, Blanco FJ, Crowley J, et al. Apremilast, an oral phosphodiesterase 4 inhibitor, in patients with psoriatic arthritis and current skin involvement: a phase III, randomised, controlled trial (PALACE 3). *Ann Rheum Dis*. 2016;75(6):1065–1073. doi: [10.1136/annrheumdis-2015-207963](https://doi.org/10.1136/annrheumdis-2015-207963)
55. Gladman DD. Toward treating to target in psoriatic arthritis. *J Rheumatol Suppl*. 2015;93:14–16.
56. El Miedany Y, El Gaafary M, GadAllah N, et al. Psoriatic arthritis treatment to the target: a consensus, evidence-based clinical practice recommendations for the management of psoriatic arthritis and its concomitant clinical manifestations. *Egypt Rheumatol Rehabil*. 2022;49(1):32. doi: [10.1186/s43166-022-00128-y](https://doi.org/10.1186/s43166-022-00128-y)
57. Joven B, Manteca CF, Rubio E, et al. Real-world persistence and treatment patterns in patients with psoriatic arthritis treated with anti-IL17 therapy in Spain: the PerflL-17 study. *Adv Ther*. 2023;40(12):5415–5431. doi: [10.1007/s12325-023-02693-w](https://doi.org/10.1007/s12325-023-02693-w)
58. Walsh JA, Cai Q, Lin I, et al. Treatment persistence and adherence among patients with psoriatic arthritis who initiated targeted immune modulators in the US: a retrospective cohort study. *Adv Ther*. 2021;38(5):2353–2364. doi: [10.1007/s12325-021-01687-w](https://doi.org/10.1007/s12325-021-01687-w)
59. Kerschbaumer A, Smolen JS, Ferreira RUJ, et al. Efficacy and safety of pharmacological treatment of psoriatic arthritis: a systematic literature research informing the 2023 update of the EULAR recommendations for the management of psoriatic arthritis. *Ann Rheum Dis*. 2024;83(6):760–774. doi: [10.1136/ard-2024-225534](https://doi.org/10.1136/ard-2024-225534)

60. Glintborg B, Ostergaard M, Krogh NS, et al. Clinical response, drug survival, and predictors thereof among 548 patients with psoriatic arthritis who switched tumor necrosis factor α inhibitor therapy: results from the Danish nationwide DANBIO registry. *Arthritis Rheum.* **2013**;65(5):1213–1223. doi: [10.1002/art.37876](#)
61. Singh S, Facciorusso A, Singh AG, et al. Obesity and response to anti-tumor necrosis factor- α agents in patients with select immune-mediated inflammatory diseases: a systematic review and meta-analysis. *PLOS ONE.* **2018**;13(5):e0195123. doi: [10.1371/journal.pone.0195123](#)
62. Curtis JR, Xie F, Yun H, et al. Real-world comparative risks of herpes virus infections in tofacitinib and biologic-treated patients with rheumatoid arthritis. *Ann Rheum Dis.* **2016**;75(10):1843–1847. doi: [10.1136/annrheumdis-2016-209131](#)
63. Rutherford AI, Subesinghe S, Hyrich KL, et al. Serious infection across biologic-treated patients with rheumatoid arthritis: results from the British society for rheumatology biologics register for rheumatoid arthritis. *Ann Rheum Dis.* **2018**;77(6):905–910. doi: [10.1136/annrheumdis-2017-212825](#)
64. Ekici M. Safety profiles of biologic drugs in psoriasis and psoriatic arthritis. **2025**.
65. Blauvelt A, Chiricozzi A. The immunologic role of IL-17 in psoriasis and psoriatic arthritis pathogenesis. *Clin Rev Allergy Immunol.* **2018**;55(3):379–390. doi: [10.1007/s12016-018-8702-3](#)
66. Yamasaki K, Nakagawa H, Kubo Y, et al. Japanese Brodalumab Study Group. Efficacy and safety of brodalumab in patients with generalized pustular psoriasis and psoriatic erythroderma: results from a 52-week, open-label study. *Br J Dermatol.* **2017**;176(3):741–751. doi: [10.1111/bjd.14702](#)
67. Iannone LF, Bennardo L, Palleria C, et al. Safety profile of biologic drugs for psoriasis in clinical practice: an Italian prospective pharmacovigilance study. *PLOS ONE.* **2020**;15(11):e0241575. doi: [10.1371/journal.pone.0241575](#)
68. Winthrop KL, Tanaka Y, Lee EB, et al. Prevention and management of herpes zoster in patients with rheumatoid arthritis and psoriatic arthritis: a clinical review. *Clin Exp Rheumatol.* **2022**;40(1):162–172. doi: [10.55563/clinexpheumatol/cpu6r9](#)
69. Bechman K, Subesinghe S, Norton S, et al. A systematic review and meta-analysis of infection risk with small molecule JAK inhibitors in rheumatoid arthritis. *Rheumatol Oxf Engl.* **2019**;58(10):1755–1766. doi: [10.1093/rheumatology/kez087](#)
70. Haddad A, Gazitt T, Feldhamer I, et al. Treatment persistence of biologics among patients with psoriatic arthritis. *Arthritis Res Ther.* **2021**;23(1):44. doi: [10.1186/s13075-021-02417-x](#)
71. Zenobia C, Hajishengallis G. Basic biology and role of interleukin-17 in immunity and inflammation. *Periodontol 2000.* **2015**;69(1):142–159. doi: [10.1111/prd.12083](#)
72. McInnes IB, Schett G. Pathogenetic insights from the treatment of rheumatoid arthritis. *Lancet Lond Engl.* **2017**;389(10086):2328–2337. doi: [10.1016/S0140-6736\(17\)31472-1](#)
73. Vecellio M, Hake VX, Davidson C, et al. The IL-17/IL-23 axis and its genetic contribution to psoriatic arthritis. *Front Immunol.* **2021**;11:596086. doi: [10.3389/fimmu.2020.596086](#)
74. Moos S, Mohebiany AN, Waisman A, et al. Imiquimod-induced psoriasis in mice depends on the IL-17 signaling of keratinocytes. *J Invest Dermatol.* **2019**;139(5):1110–1117. doi: [10.1016/j.jid.2019.01.006](#)
75. Taams LS. Interleukin-17 in rheumatoid arthritis: trials and tribulations. *J Exp Med.* **2020**;217(3):e20192048. doi: [10.1084/jem.20192048](#)
76. Bridgwood C, Watad A, Russell T, et al. Identification of myeloid cells in the human enthesis as the main source of local IL-23 production. *Ann Rheum Dis.* **2019**;78(7):929–933. doi: [10.1136/annrheumdis-2018-214944](#)
77. Scher JU, Ogdie A, Merola JF, et al. Preventing psoriatic arthritis: focusing on patients with psoriasis at increased risk of transition. *Nat Rev Rheumatol.* **2019**;15(3):153–166. doi: [10.1038/s41584-019-0175-0](#)
78. Gravallese EM, Schett G. Effects of the IL-23-IL-17 pathway on bone in spondyloarthritis. *Nat Rev Rheumatol.* **2018**;14(11):631–640. doi: [10.1038/s41584-018-0091-8](#)
79. Koenders MI, van den Berg WB. Secukinumab for rheumatology: development and its potential place in therapy. *Drug Des Devel Ther.* **2016**;10:2069–2080. doi: [10.2147/DDDT.S105263](#)
80. Menon B, Gullick NJ, Walter GJ, et al. Interleukin-17+CD8+ T cells are enriched in the joints of patients with psoriatic arthritis and correlate with disease activity and joint damage progression. *Arthritis Rheumatol.* **2014**;66(5):1272–1281. doi: [10.1002/art.38376](#)
81. Simon D, Tascilar K, Kleyer A, et al. Association of structural enthesal lesions with an increased risk of progression from psoriasis to psoriatic arthritis. *Arthritis Rheumatol.* **2022**;74(2):253–262. doi: [10.1002/art.41239](#)
82. Schett G, Lories RJ, D'Agostino M-A, et al. Enthesitis: from pathophysiology to treatment. *Nat Rev Rheumatol.* **2017**;13(12):731–741. doi: [10.1038/nrrheum.2017.188](#)
83. Raychaudhuri SP, Raychaudhuri SK, Genovese MC. IL-17 receptor and its functional significance in psoriatic arthritis. *Mol Cell Biochem.* **2012**;359(1–2):419–429. doi: [10.1007/s11010-011-1036-6](#)
84. McInnes IB, Mease PJ, Ritchlin CT, et al. Secukinumab sustains improvement in signs and symptoms of psoriatic arthritis: 2 year results from the phase 3 FUTURE 2 study. *Rheumatol Oxf Engl.* **2017**;56(11):1993–2003. doi: [10.1093/rheumatology/kex301](#)
85. Kuwabara T, Ishikawa F, Kondo M, et al. The role of IL-17 and related cytokines in inflammatory autoimmune diseases. *Mediators Inflamm.* **2017**;2017:1–11. doi: [10.1155/2017/3908061](#)
86. Langley RG, Elewski BE, Lebwohl M, et al. Secukinumab in plaque psoriasis—results of two phase 3 trials. *N Engl J Med.* **2014**;371(4):326–338. doi: [10.1056/NEJMoa1314258](#)
87. Gottlieb AB, Langley RG, Philipp S, et al. Secukinumab improves physical function in subjects with plaque psoriasis and psoriatic arthritis: results from two randomized, phase 3 trials. *J Drugs Dermatol.* **2015**;14(8):821–833.
88. Gordon KB, Blauvelt A, Papp KA, et al. Phase 3 trials of ixekizumab in moderate-to-severe plaque psoriasis. *N Engl J Med.* **2016**;375(4):345–356. doi: [10.1056/NEJMoa1512711](#)
89. Nash P, Kirkham B, Okada M, et al. Ixekizumab for the treatment of patients with active psoriatic arthritis and an inadequate response to tumour necrosis factor inhibitors: results from the 24-week randomised, double-blind, placebo-controlled period of the SPIRIT-P2 phase 3 trial. *Lancet Lond Engl.* **2017**;389(10086):2317–2327. doi: [10.1016/S0140-6736\(17\)31429-0](#)
90. Lebwohl M, Strober B, Menter A, et al. Phase 3 Studies Comparing Brodalumab with Ustekinumab in Psoriasis. *N Engl J Med.* **2015**;373(14):1318–1328. doi: [10.1056/NEJMoa1503824](#)
91. Reich K, Papp KA, Blauvelt A, et al. Bimekizumab versus ustekinumab for the treatment of moderate to severe plaque psoriasis (BE VIVID): efficacy and safety from a 52-week, multicentre, double-blind, active comparator and placebo controlled phase 3 trial. *Lancet.* **2021**;397(10273):487–498. doi: [10.1016/S0140-6736\(21\)00125-2](#)
92. Huangfu L, Li R, Huang Y, et al. The IL-17 family in diseases: from bench to bedside. *Signal Transduct Target Ther.* **2023**;8(1):402. doi: [10.1038/s41392-023-01620-3](#)
93. Papp KA, Weinberg MA, Morris A, et al. IL17A/F nanobody sonelokimab in patients with plaque psoriasis: a multicentre, randomised, placebo-controlled, phase 2b study. *Lancet Lond Engl.* **2021**;397(10284):1564–1575. doi: [10.1016/S0140-6736\(21\)00440-2](#)
94. Childs BA, Merola JF. Novel therapeutic targets in the management of psoriatic disease. *Rheum Dis Clin.* **2025**;51(3):447–468. doi: [10.1016/j.rdc.2025.05.005](#)
95. Mease PJ, Merola JF, Kristensen LE, et al. Sonelokimab demonstrates superior tissue penetration and enthesitis resolution in active PsA: primary results from the phase 3 VELA program. *Arthritis Rheumatol.* **2025**;77(Suppl 10):1450.
96. Madej B, Tomaszewski F, Szmajda-Krygier D, et al. Monoclonal antibodies: historical perspective and current trends in biological drug development. *Int J Mol Sci.* **2025**;26(18):8794. doi: [10.3390/ijms26188794](#)
97. Ramiro S, Schett G, Marzo-Ortega H, et al. The impact of IL-17A inhibition in rheumatic and musculoskeletal diseases: current

- insights and future prospects. *Rheumatol Ther.* **2025**;12(3):435–451. doi: [10.1007/s40744-025-00754-w](https://doi.org/10.1007/s40744-025-00754-w)
98. Dogra S, Singh S. Assessing the clinical progress of the bispecific nanobody sonelokimab. *Expert Opin Investig Drugs.* **2025**;34(4):253–258. doi: [10.1080/13543784.2025.2500292](https://doi.org/10.1080/13543784.2025.2500292)
 99. McInnes IB, Coates LC, Mease PJ, et al. OP0195 efficacy and safety of sonelokimab, a novel IL-17A- and IL-17F-inhibiting nanobody, in patients with active psoriatic arthritis (PsA): results from the global, randomized, double-blind, placebo-controlled phase 2 ARGO trial. *Ann Rheum Dis.* **2024**;83(Suppl 1):154–155.
 100. McInnes IB, Coates LC, Mease PJ, et al. Sonelokimab, a novel IL-17A/F-inhibiting nanobody, in patients with active psoriatic arthritis: a randomized, placebo-controlled, phase 2 trial. *Nat Med.* **2024**;30(5):1374–1382.
 101. Taylor PC, Mease PJ, de Vlam K, et al. Efficacy and safety of izokibep in patients with active psoriatic arthritis: a randomised, double-blind, placebo-controlled, phase 2 study. *Ann Rheum Dis.* **2025**;S0003-4967(25):00815–5.
 102. Behrens F, Taylor PC, Wetzel D, et al. OP0258 izokibep (ABY-035) in patients with active psoriatic arthritis: 16-week results from a phase 2 study. *Ann Rheum Dis.* **2022**;81(Suppl 1):170–171.
 103. ACELYRIN Inc. A phase 2b pivotal study to evaluate the efficacy and safety of izokibep in subjects with non-infectious, intermediate-, posterior- or pan-uveitis [Internet]. Bethesda (MD): National Library of Medicine (US); **2025** Feb [cited 2025 Dec 9]. Identifier: NCT05384249.
 104. Ståhl S, Gräslund T, Eriksson Karlström A, et al. Affibody molecules in biotechnological and medical applications. *Trends Biotechnol.* **2017**;35(8):691–712. doi: [10.1016/j.tibtech.2017.04.007](https://doi.org/10.1016/j.tibtech.2017.04.007)
 105. Lo A, Greenzaid JD, Gantz HY, et al. Clinical pharmacokinetics and pharmacodynamics of topical non-biological therapies for psoriasis patients. *Expert Opin Drug Metab Toxicol.* **2024**;20(4):235–248. doi: [10.1080/17425255.2024.2337749](https://doi.org/10.1080/17425255.2024.2337749)
 106. Kim D, Yang S, Gill M, et al. Next-generation anti-IL-17 agents for psoriatic disease: a pipeline review. *Am J Clin Dermatol.* **2025**;26(3):307–320. doi: [10.1007/s40257-025-00928-w](https://doi.org/10.1007/s40257-025-00928-w)
 107. Mease PJ, Behrens F, Kivitz A, et al. LBA0005 efficacy and safety of izokibep, a novel IL-17A inhibitor, in patients with active psoriatic arthritis: week 16 results from a randomized, double-blind, placebo-controlled, multicenter phase 2b/3 study. *Ann Rheum Dis.* **2024**;83(Suppl 1):235–236. doi: [10.1136/annrheumdis-2024-eular.LBA85](https://doi.org/10.1136/annrheumdis-2024-eular.LBA85)
 108. Blauvelt A, Warren RB, Ghoreschi K, et al. Efficacy and safety of DC-853, a novel oral IL-17 antagonist, in moderate-to-severe psoriasis and psoriatic arthritis: phase 2b results. *Arthritis Rheumatol.* **2025**;77(Suppl 10):L05.
 109. Andrews MD, Dack KN, de Groot MJ, et al. Discovery of an oral, rule of 5 compliant, interleukin 17A protein-protein interaction modulator for the potential treatment of psoriasis and other inflammatory diseases. *J Med Chem.* **2022**;65(13):8828–8842.
 110. Raavi, Koehler AN, Vegas AJ. At the interface: small-molecule inhibitors of soluble cytokines. *Chem Rev.* **2025**;125(9):4528–4568.
 111. Drakos A, Torres T, Vender R. Emerging oral therapies for the treatment of psoriasis: a review of pipeline agents. *Pharmaceutics.* **2024**;16(1):111. doi: [10.3390/pharmaceutics16010111](https://doi.org/10.3390/pharmaceutics16010111)
 112. Pharmaceutical Technology. SAR-441566 by sanofi for rheumatoid arthritis: likelihood of approval [Internet]. London: Verdict Media; **2023** [cited 2025 Dec 9]. Available from: <https://www.pharmaceutical-technology.com/data-insights/sar-441566-sanofi-rheumatoid-arthritis-likelihood-of-approval/>
 113. Sanofi. A phase 2, international, multicenter, randomized, double-blind, placebo-controlled, dose-ranging study of efficacy and safety of SAR441566 in adults with moderate to severe plaque psoriasis [Internet]. Bethesda (MD): National Library of Medicine (US); **2025** Oct [cited 2025 Dec 9]. Identifier: NCT06073119. Available from: <https://clinicaltrials.gov/study/NCT06073119>
 114. Yilmaz O, Pinto JP, Torres T. New and emerging oral therapies for psoriasis. *Drugs Context.* **2024**;13:1–24. doi: [10.7573/dic.2024-5-6](https://doi.org/10.7573/dic.2024-5-6)
 115. Zhu K, Zheng Q, Zhu Y, et al. Efficacy and safety of vunakizumab in patients with moderate-to-severe plaque psoriasis across different regions of China: a post-hoc exploratory analysis of a phase III, randomized controlled trial. *J Dermatol Treat.* **2025**;36(1):2560505. doi: [10.1080/09546634.2025.2560505](https://doi.org/10.1080/09546634.2025.2560505)
 116. Zhang C, Yan K, Diao Q, et al. A multicenter, randomized, double-blinded, placebo-controlled, dose-ranging study evaluating the efficacy and safety of vunakizumab in patients with moderate-to-severe plaque psoriasis. *J Am Acad Dermatol.* **2022**;87(1):95–102. doi: [10.1016/j.jaad.2022.01.005](https://doi.org/10.1016/j.jaad.2022.01.005)
 117. Dong Y, Gao X, Feng S, et al. Safety, pharmacokinetics, and bioequivalence of vunakizumab injection, a specific dermatitis drug, in healthy Chinese volunteers: an open-label, randomized, two-stage, two-sequence cross-over study. *Expert Opin Drug Metab Toxicol.* **2025**;21(9):1117–1123. doi: [10.1080/17425255.2025.2558829](https://doi.org/10.1080/17425255.2025.2558829)
 118. Li Z, Xia Y, Wang M, et al. Efficacy and safety of AK111 in patients with active ankylosing spondylitis: results from a randomized, double-blind, placebo-controlled, phase II clinical study. *J Immunol.* **2023**;210(1 Suppl):165.18.
 119. Cai L, Li C, Li S, et al. Investigation of the efficacy and safety of xeligekimab (GR1501) in patients with moderate-to-severe plaque psoriasis: a multicenter, randomized, double-blind phase II clinical trial. *Dermatol Ther.* **2025**;15(10):2803–2816. doi: [10.1007/s13555-025-01450-x](https://doi.org/10.1007/s13555-025-01450-x)
 120. Korotaeva TV, Mazurov VI, Lila AM, et al. Netakimab for the treatment of psoriatic arthritis: 3-year results of the phase III BCD-085-8/PATERA study. *Mod Rheumatol J.* **2024**;18(4):33–42. doi: [10.14412/1996-7012-2024-4-33-42](https://doi.org/10.14412/1996-7012-2024-4-33-42)
 121. Mazurov VI, Erdes SF, Gaydukova IZ, et al. Long-term efficacy and safety of netakimab in patients with active ankylosing spondylitis: results of three years of use in the international multicentre, randomized, double-blind, phase III clinical trial BCD-085-5/ASTERA. *Mod Rheumatol J.* **2024**;18(1):35–46. doi: [10.14412/1996-7012-2024-1-35-46](https://doi.org/10.14412/1996-7012-2024-1-35-46)
 122. Puig L, Bakulev AL, Kokhan MM, et al. Efficacy and safety of neta-kimab, a novel anti-IL-17 monoclonal antibody, in patients with moderate to severe plaque psoriasis. Results of a 54-week randomized double-blind placebo-controlled PLANETA clinical trial. *Dermatol Ther.* **2021**;11(4):1319–1332. doi: [10.1007/s13555-021-00554-4](https://doi.org/10.1007/s13555-021-00554-4)
 123. Firdous S, Amjad H, Choudhary MU, et al. Emerging biological therapies for psoriatic arthritis: a systematic review. *Medicine (Baltimore).* **2025**;104(42):e45259. doi: [10.1097/MD.00000000000045259](https://doi.org/10.1097/MD.00000000000045259)
 124. Benschop RJ, Chow C-K, Tian Y, et al. Development of tibilizumab, a tetravalent bispecific antibody targeting BAFF and IL-17A for the treatment of autoimmune disease. *MAbs.* **2019**;11(6):1175–1190. doi: [10.1080/19420862.2019.1624463](https://doi.org/10.1080/19420862.2019.1624463)
 125. Nazary Abrbekoh F, Salimi L, Saghati S, et al. Application of micro-needle patches for drug delivery; doorstep to novel therapies. *J Tissue Eng.* **2022**;13:20417314221085390. doi: [10.1177/20417314221085390](https://doi.org/10.1177/20417314221085390)