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Investigational IL-17A antagonist in the treatment of Psoriatic Arthritis - A review

Osariemen Osunbor[1], Jan René Nkeck[2], Adewale O. Adebajo[1,3]

[1]Barnsley Hospital NHS Foundation Trust, South Yorkshire, United Kingdom;

[2]Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon;

[3]University of Sheffield, South Yorkshire, United Kingdom.

***CORRESPONDING AUTHOR**

Prof Adewale O. Adebajo (AOA), MBE, MBBS, MSc, FMCP, FWACP, FRCP, FACP, FAcMedMD

Emil address: a.o.adebajo@sheffield.ac.uk;

Phone:+447963671131

ORCID: https://orcid.org/0000-0001-7368-7083.

ABSTRACT

Introduction

The global burden of psoriatic arthritis (PsA) is considerable, with major effects on both quality of life and life expectancy. Despite the existence of a diverse array of therapeutic interventions, 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. A structured search was conducted in PubMed for the following terms investigational IL-17A antagonist and Psoriatic arthritis. We aimed for at least 100 research papers published within the last 20years. In addition, clinical trials databases focusing on phase II – III IL-17A antagonists treatment in Psoriatic Arthritis were also reviewed.

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, which leverages predictive biomarkers to personalize treatment from the moment of diagnosis. This could help control disease early and prevent progression, beyond the current Treat-to-Target strategy which mainly focuses on managing

existing symptoms. 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.

Keywords: Bispecific monoclonal antibodies, Interleukin 17A antagonists, Psoriatic arthritis, Small molecules, Third generation.

Abbreviations: IL-17A: interleukin 17A; PsA: psoriatic arthritis.

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 towards 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.

1.0 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 (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 significant 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 (IgG1κ). 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 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].

2.0 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.

3.0 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.

4.0 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 personalised 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 towards 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 personalised 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.

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Author contributions

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Declaration of Generative AI in Scientific Writing

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

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