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REVIEW

# Psoriatic Arthritis – An Overview

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## Abstract

Psoriatic arthritis (PsA) is a multifaceted immune-mediated inflammatory disease affecting approximately 30% of patients with psoriasis. Its clinical spectrum is highly heterogeneous, spanning peripheral arthritis, axial involvement, enthesitis, dactylitis, and skin/nail disease. Current management emphasizes a "treat-to-target" (T2T) strategy, utilizing validated instruments like DAPSA and MDA to achieve clinical remission or low disease activity. Despite an expanding therapeutic armamentarium, managing complex phenotypes and treatment-resistant cases remains a significant challenge.

While traditional therapies have focused on IL-17A, recent evidence highlights the pathogenic role of IL-17F, which is highly expressed in the synovial tissue and skin of PsA patients. This review highlights the clinical significance of dual IL-17A and IL-17F inhibition, specifically via bimekizumab, a monoclonal antibody designed to simultaneously neutralize both isoforms and their heterodimers. Phase 3 clinical data (BE OPTIMAL and BE COMPLETE) demonstrate that dual inhibition provides superior efficacy over placebo in both biologic-naïve and bio-experienced populations.

Advances in our understanding of PsA pathogenesis, particularly the introduction of dual IL-17A/F inhibition, have significantly expanded the therapeutic armamentarium and created new benchmarks for disease control. However, critical gaps remain, including the absence of predictive biomarkers and the challenge of managing treatment-resistant cases. To address these unmet needs, the modern standard of care must prioritize a personalized approach—tailoring interventions to the patient's specific phenotype, comorbidities, and preferences through a framework of shared decision-making.

***Keywords:***. psoriatic arthritis, IL-17A/F pathway, treat-to-target strategy, bimekizumab, cytokine signaling.

## Introduction

Psoriatic arthritis (PsA) is a systemic complex immune mediated disease, that is characterized by inflammation of enthesis and articular synovium [1]. It can be considered part of the broader psoriatic disease, which is comprised of many possible involvements of different organ systems (skin, musculoskeletal system, eyes, gut, cardiovascular system, etc). It is usually preceded by skin psoriasis (PsO), even though in about 15% of cases, PsA precedes PsO [1,2]. PsA affects around 30% of PsO patients. Besides its own disease spectrum (peripheral and axial skeleton arthritis, tenosynovitis, enthesitis, dactylitis), PsA is associated with an array of possible comorbidities: diabetes mellitus, depression, cardiovascular disease, anxiety, metabolic syndrome, etc [2]. The global prevalence of PsA is around 112 per 100 000 adults, being more prevalent in Europe and Noth America than South America and Asia [3]. The disease affects equally men and women [1,2].

The clinical spectrum of the disease is heterogenous and can be grouped in several domains: peripheral arthritis (oligo- and poli-articular), axial (psoriatic spondylitis), enthesitis (inflammation of the insertion point of ligaments, tendons, articular capsulae), dactylitis ("sausage" fingers), cutaneous and nail psoriasis [1,4]. As mentioned, there may be extraarticular manifestations: uveitis, inflammatory bowel disease. It is worth mentioning that nail psoriasis (in the form of pitting and onycholysis), are present in 80 to 90% of PsA patients; they are an important clinical marker, in that they represent a marker of risk of PsA in PsO patients [4,5].

## Pathogenesis

The mainstay of PsA pathogenesis is represented by interleukin (IL)-23/IL-17 axis. Th-17 cells have the most important role, as they secrete IL-17. Interleukin 17 is a family of cytokines, including 6 structurally relates isoforms, named from A to F. Interleukin 17A is the prototypic member and was first documented in Th17 cells, in 1993. Along with IL-17F, they are the most studied and have a well-established role in some of the inflammatory arthritides (PsA, spondyloarthritis) [6]. Furthermore, IL-17F shares the highest homology to IL-17A (around 50%) [6]. IL-

17A and IL-17F evolved to protect against infection via the regulation of protective responses against infections at mucosal and epithelial surfaces: maintenance of gut epithelium, skin and wound healing. They are involved in host defense against bacterial and fungal infections, through an epithelial barrier protection function [7,8]. All IL-17 isoforms bind to receptors on the surface of target cells, thus inducing several intracellular signaling pathways [9]. IL-17A and F form homo- and heterodimers with each other and then they bind to the receptor. Thus, they promote inflammation. [9]. Interestingly enough, IL-17A can bind in an autocrine manner to the receptor, activating nuclear factor kB (NF-kB), thus inducing the secretion of IL-24. This interleukin acts as a suppressor of the expression of other Th17 cell's cytokines. Therefore, blocking IL-17A alone no longer allows for the repressive role of IL-24, offering an unexpected pro-inflammatory "escape route" for the production of IL-17F and other cytokines. Thus, appear the limitations of targeting IL-17A alone, shedding light on some of the ineffectiveness of IL-17A inhibitors in some inflammatory arthritides [10]. Morerover, there is an IL-23 independent IL-17A and IL-17F production, by innate cells (gamma-delta T cells and mucosal-associated invariant T cells), thus evading IL-23 inhibition and contributing to IL-17 mediated inflammation [11].

## Elevate standard of care and treat-to-target strategy

Modern therapeutic approach in PsA is based upon treat-to-target (T2T) strategy, ever since the TICOPA trial demonstrated that a systematic therapeutic escalation leads to superior clinical outcomes (higher rates of achieving ACR20, ACR50, ACR70 and PASI75 responses) than the standard approach [12]. This approach presumes establishing a clear target to be achieved, as well as regular assessments (at predefined times and anytime they are felt needed by the patient or the treating physician), with therapeutic adjustments on the way every time they are warranted, until the target is reached [13]. As for other inflammatory musculoskeletal diseases, the target in treating PsA is represented by attaining a state of remission or, alternatively, low disease activity [13]. Whenever you want to reach a target, there must be an instrument to measure it. In PsA the proposed instruments to be used in order to evaluate the attainment of the proposed target, are the DAPSA (Disease Activity in PSoriatic Arthritis) and the MDA (Minimal Disease Activity) or it's more stringent variant VLDA (Very Low Disease Activity) [14]. DPASA is calculated by summing tender joint count (68 joints), swollen joint count (66 joints), patient reported pain (0 to 10 on a numeric scale), patient global assessment (also 0 to 10 on a numeric rating scale) and C-reactive protein (in mg/dl). The activity states it shows correspond to remission (<4), low disease activity (>4 to <14), moderate disease activity >14 to <28) and high disease activity (>28) [15]. While it allows a definition of all activity states as well as different levels of response to therapy [16] in PsA, DAPSA has the possible downside that it focuses only on the articular domain of the disease [14]. In contrast, MDA is a binary measure of the actual disease state of a patient, and it includes other important domains of the disease [16]. When a patient meets five of the following seven criteria, then they achieved MDA.

**Table 1.** *Minimal disease activity criteria for PsA [17]*

| |
|---|
| Tender joint count less or equal to 1 |
| Swollen joint count less or equal to 1 |
| PASI less or equal to 1 or body surface area less or equal to 3 |
| Patient pain visual analogue scale (VAS) score less or equal to 15 |
| Patient global disease activity VAS less or equal to 20 |
| Health assessment questionnaire less or equal to 0.5 |
| Tender entheseal points less or equal to 1 |
| When all the above seven criteria are met by a patient, they are in very low disease activity (VLDA) state of their disease. |

## EULAR guidelines – what do they bring?

Most recent guidelines (2023 EULAR recommendations [13]) have updated the pharmacological approach to PsA, integrating all available therapeutical classes in a practical and progressive manner. From recognizing that PsA is a multifaceted disease which may need multidisciplinary approach, passing through the need that treatment should aim at best care and must be the result of an informed shared decision with the patient, through acknowledging that rheumatologists are those who should primary care for PsA patients, through setting the primary general goal of treatment, through giving consideration to each musculoskeletal manifestation, to the need to take into account all the extra-musculoskeletal manifestations and comorbidities, the seven overarching principles, of whom 3 are unchanged from the previous version, 3 are reformulated and the last one is newly added, set the frame within which therapy of PsA patients should evolve [13].

Of the previous 12 recommendations, 3 remained unchanged, 2 were merged and modified, another 3 were only modified, 1 was split into 2, 1 was reformulated and one is new. To summarize them:

-the target is remission or low disease activity/MDA;

-NSAID's are only symptomatic therapy, while glucocorticoids used only as local adjunctive therapy;

-csDMARDs (conventional synthetic disease modifying anti rheumatic drugs) are to be used as early as possible in patients with polyarthritis or mono/oligoarthritis with poor prognostic factors, with methotrexate being the preferred one;

-bDMARDs (biological DMARDs): if the target is not attained with cs DMARDs, and when NSAIDs or corticoid injection do not resolve enthesitis, a bDMARD is recommended, with no preference between classes (anti IL-17A, anti IL-17A/F, anti TNF, anti IL-23, anti IL-12/23;

-JAKi (Janus kinase inhibitor) are recommended mostly after bDMARDs failure or inappropriateness of use;

-PDE4 (phosphodiesterase 4) inhibitors may be recommended when there is failure to the previous classes; nonetheless, this is a situation in which changing within classes or between classes is also permitted.

The most interesting thing with the 2023 update is the new recommendation (recommendation number 9) that pertains to the choice of mode of action (therefore of the drug), with respect to the non-musculoskeletal manifestations: if the relevant manifestation is the skin involvement, then, preference should be given to an anti-IL-17A/F or anti IL-A, or anti IL-23, or anti IL-12/23; with uveitis being the prominent feature of the disease, an anti TNF monoclonal antibody is recommended, and so on [13]. This is a recognition of the fact that different manifestations are driven by different cytokines, so if we aim for best care, we should adapt the treatment to the pathogenesis of the manifestations.treatment.

## Unmet needs in PsA

Even though nowadays the therapeutic armamentarium has extended significantly, some patients do not reach optimal or sustained response; furthermore, loss of efficacy and adverse events often lead to discontinuation of treatment [18].

One of the most important unmet needs in PsA pertains to choosing the most efficacious treatment: we do not yet, have predictive response biomarkers, that is biological markers that could predict which treatment for which patient (the best personalized treatment) [19]. This would allow for the best choice for the initial therapy and would prevent unnecessary expenses with inefficacious treatments.

Another unmet need is the fact that some domains of the disease are insufficiently recognized by patients and doctors as well; a good example is fatigue, a totally non-specific symptom that doctor and patients alike do not always take into account when assessing the efficacy of the treatment that the patients uses [20].

Probably the most important unmet need and complex situation in PsA treatment is difficult to manage PsA and treatment refractory PsA [21]. These concepts are very important in day-to-day practice, as they can individualize for every treating physician the particular patient that he/she is not treating inadequately but who is just a difficult to manage or refractory to treatment patient. Also, they open the way for researching those features that characterize, as well as the strategy that should be used for such patients.

Last but not least, exploring the possibility of intercepting the transition of a patient from PsO to PsA is a very important hot topic, because it would prevent the occurrence of articular manifestations of psoriatic disease by treating the cutaneous and nail disease with the appropriate strategy [19].

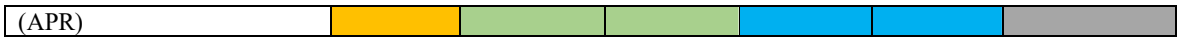
Obviously, to treat early thus efficaciously, means to diagnose early and this is also an unmet need in PsA: there is the subclinical period of disease which is still very difficult to ascertain in a PsO patient having non specific articular symptoms, let alone in one having no articular symptoms at all [22].

## Current therapeutical options

Therapeutic armamentarium in PsA includes multiple drug classes, each with their indications and efficacy, sometimes differentiated by disease domain (see next table).

**Table 2.** *Efficacy results of randomized controlled trials stratifies by mode of action and disease domain (adapted from 23).*

| Target | Disease Domain | | | | | |
|---|---|---|---|---|---|---|
| | **Arthritis** | **Physical function** | **Skin** | **Enthesitis** | **Dactylitis** | **Radiographic damage** |
| TNF (ADA,CZP,ETN,IFX,GOL) | | | | | | |
| IL-17A (IXE, SEC) | | | | | | |
| IL-17A/F (BKZ) | | | | | | |
| IL12/23 (UST) | | | | | | |
| IL-23-p19 (GUS, RIS)# | | | | | | |
| JAK (TOFA, UPA)* | | | | | | |
| CD80/86 (ABA) | | | | | | |
| PDE-4 | | | | | | |




*LEGEND: Green – statistically superior to placebo; dark blue – no difference to placebo; yellow – numerical (but not statistically) different to placebo; light blue – superior to placebo (pre-specified post-hoc analysis); grey – not evaluated/reported; # - the dark blue square concerns RIS; * - all dark blue squares concern TOFA.*

The following table summarizes indications of main current therapeutical options.

**Table 3.** *Main therapeutical classes and their main indications in PsA (adapted from 13, 23, 24).*

| Therapeutic class | Examples | Main indications |
|---|---|---|
| csDMARD | Methotrexate, Leflunomide, Sulfasalazine | Peripheral arthtitis, first line |
| Anti-TNF | Adalimumab, Etanercept, Golimumab, Certolizumab, Infliximab | Axial and peripheral arthritis, enthesitis, dactilitis, uveitis |
| Anti-IL-17A | Secukinumab, Ixekizumab | Axial snad peripheral arthritis, skin, enthesitis |
| Anti-IL-17A/F | Bimekizumab | Axial and peripheral arthritis, skin, enthesitis, dactilitis |
| Anti-IL-12/23 | Ustekinumab | Peripheral arthritis, skin |
| Anti-IL-23 | Guselkumab, Risankizumab | Peripheral arthritis, skin enthesitis |
| JAK inhibitors | Tofacitinib, Upadacitinib | After bDMARD failure, after cardiovascular risk evaluation |

## Dual inhibition IL-17A/F – a new therapeutic step

From a historical point of view, therapeutic blockade of IL-17 has been focused on IL-17A. Nevertheless, IL-17F, even though less potent than IL-17A, is often expressed at higher levels in the synovial tissue of PsA patients, as well as in their skin [25]. Preclinical data showed that while IL-17F alone inhibition had no significant effect on stimulated synoviocytes of PsA patients, dual inhibition of IL-17A and IL-17F resulted in a greater suppression of production of IL-6, IL-8 and MMP3 (all proinflammatory cytokines) than suppression of IL-17A alone [26]. Hence, the concept that IL-17A/F dual inhibition might lead to a more intense suppression of the IL-17 pathogenic pathway in psoriatic disease.

Bimekizumab is a humanized immunoglobulin G1 (IgG1) monoclonal antibody that has two identical antigen binding fragments (Fab). Since IL-17 A and F realize homo- as well as heterodimers, this particular structure of bimekizumab allows for a single molecule to simultaneously bind to IL-17A and F homodimers, as well as to the IL-17A/F heterodimer. Consequently, bimekizumab prevents any interaction of these isoforms of IL-17A and F to their complex receptor and, therefore, it's downstream intracellular signaling [25,26]. The affinity of this binding is high, irrespective of the concentration of the IL-17 isoforms [27]. Bimekizumab has a half-life of about 24 days and has a linear and dose-proportional pharmacokinetics [25,26].

Phase 3 randomised controlled trials BE OPTIMAL (in bDMARD naïve patients) and BE COMPLETE (in bDMARD insufficient responders patients) have demonstrated superiority over placebo, in every primary and secondary endpoints [28,29]. In BE OPTIMAL 44% of bimekizumab treated patients achieved ACR50 at Week 16, compared to 10% in the placebo arm ($p<0.0001$) and 65% of bimekizumab treated patients with more than 3% of body surface area involvement, achieved PASI100 (that is complete clear skin) versus 11 in the placebo arm ($p<0.0001$). In BE COMPLETE, a strikingly 43% of bimekizumab treated patients achieved ACR50 at Week 16, versus 7% in the placebo arm, highlighting it's high effectiveness in patients prior exposed to biologics [25,28,29]. Moreover, these good results extended to 52 weeks and up to 3 years: 55% at Week 52 and 53,2% at Week 160 of sustained ACR50 response in biologic naïve patients and 52% at Week 52 and 55% at Week 156 for ACR50 response maintenance in bio-experienced patients. Similarly, high rates of PASI100 response were maintained through year three of follow-up [30-33], bimekizumab establishing a new benchmark for efficacy in patients with concurrent skin disease. The safety profile of bimekizumab in these studies showed no new alarm signals, with the most notable finding being an increased incidence of fungal infections, particularly oropharyngeal candidiasis, which is a predictable consequence of the mechanism of action of this drug; however, these were mild to moderate in severity, localized, not

generalized, resolved with standard antifungal therapy and lead to discontinuation of therapy in a minority of cases (<1%) [25,28-33].

## Personalizing PsA treatment

With all the knowledge accumulated in the last 10-15 years and the technological progress, we now have, as stated before, a large array of available therapies for PsA patients' treatment. Ideally, the therapeutic approach should be adapted to the patient's dominant phenotypic expression of the disease, as well as to the comorbidities and preference of the patients. It is sometimes quite difficult to harmonize all these factors, but nevertheless the treating physician should bear in mind the following: if significant skin disease, think about inhibition of IL-17A/F, IL-17A, IL-23; if significant articular (axial or peripheral), entheseal or dactilits disease, think about IL-17A/F, IL-17A or TNF inhibition; remember, IL-23 inhibitors did not prove efficacy in axial disease; if significant ocular disease, think about TNF inhibition with a monoclonal antibody. The presence of inflammatory bowel disease should make the treating physician avoid IL-17 inhibition and the presence of cardiovascular risk factors or thromboembolic events history should caution for JAK inhibition [13,23,24]. The robust response in the articular, enthesitis, dactylitis and particularly skin domains, coupled with the efficacy on inhibiting radiographic progression of the disease, make dual IL-17A/F inhibition a meaningful therapeutic option for PsA patients. It is noteworthy that there is a lot more research to be done, particularly concerning the predictive (diagnostic and treatment response) markers, the interception of transition from PsO to PsA and the difficult to manage cases.

## PsA patient's perspective in 2026

The impact of psoriatic disease on the patient is multidimensional and significant. For instance, fatigue is reported by more than half of the patients and, unfortunately is frequently underrecognized by doctors. PsA impacts daily living activities, social implication, quality of life [34]. Psoriatic Arthritis Impact of Disease (PsAID) questionnaire is a validated instrument for assessing the patients' perspective on the impact the disease has upon them [20]. Health related quality of life improves with efficacious therapy, but changing multiple drugs or multiple modes of action are associated with a lower quality of life [35]. One important issue is that sometimes the expectations of the patient do not match those of his treating physician, hence the need for a shared decision-making process in every step of the therapeutic avenue [36]. Multidisciplinary approach to the PsA patient along with the shared decision are essential for optimizing results.

## Conclusion

Psoriatic arthritis is a complex disease, with a significant impact on quality of life of patients. Recent progress in pathogenesis understanding along with the expansion of the therapeutic armamentarium (in which dual IL-17A/F is a very important component) provides new opportunities for disease control. EULAR guidelines integrate therapeutic options in a progressive approach based on treat-to-target strategy. However, there are important unmet needs, like the lack of predictive treatment response biomarkers and difficult to manage and treatment resistant disease. Personalizing treatment according to disease's predominant phenotype, comorbidities and patient preference should be the current standard of care, with the patient being involved in decision making on every step of the way.

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