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Targeted Therapy in Psoriatic Arthritis, Including Juvenile Psoriatic Arthritis: Efficacy and Safety of TNF-alpha, IL-17 and JAK Inhibitors - A Literature Review

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Abstract

Background: Psoriatic arthritis (PsA) and juvenile psoriatic arthritis (JPsA) are chronic inflammatory diseases with heterogeneous, multi-domain presentation; targeted therapies against TNF-alpha, IL-17 and the JAK-STAT pathway have expanded treatment options. **Aim**: To evaluate efficacy and safety of three classes of targeted therapies in PsA and JPsA: TNF-alpha inhibitors (TNF-i), IL-17 inhibitors (IL-17-i) and Janus kinase inhibitors (JAK-i). **Material and methods**: A narrative PubMed (MEDLINE) review of phase I-III randomized trials and open-label extensions, real-world studies, and selected reviews assessing efficacy and safety of TNF-i, IL-17-i and JAK-i in adults and children with PsA/JPsA. **Results**: Anti-TNF therapies show high ACR response rates, favourable skin effects, and inhibition of radiographic progression, with acceptable long-term safety. IL-17-i provide sustained improvement across major PsA domains, particularly in TNF-i-naive patients, with a stable safety profile. JAK-i are effective after failure of conventional and biologic treatment, improving joint activity, function and quality of life, but carry a characteristic adverse-event profile requiring assessment. Real-world analyses in PsA and axial spondyloarthritis show no significantly increased cardiovascular or malignancy risk with JAK-i versus anti-TNF/anti-IL-17 therapies, though rheumatoid arthritis data warrant caution in high-risk patients. In JPsA, TNF-i remain the principal biologic class, while anti-IL-17 trials confirm efficacy and safety in children.

Conclusions: Targeted therapy of PsA and JPsA requires individualization based on phenotype, prior treatment, and cardiovascular, thromboembolic and oncologic risk. Further paediatric studies are needed to define each class's role in JPsA management.

Keywords: psoriatic arthritis; juvenile psoriatic arthritis; targeted therapy; TNF-alpha inhibitors, Janus kinase inhibitors, IL-17 inhibitors

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease of the musculoskeletal system with an autoimmune background and is classified among spondyloarthropathies. It affects around 2% of the general population [2], with prevalence showing significant geographic variation - for example, the disease is diagnosed more frequently in Europe than in Asian countries [31]. It develops in nearly 30% of individuals with psoriasis [2], presenting a wide variety of clinical manifestations ranging from synovitis of peripheral joints, through enthesitis, to involvement of the sacroiliac joints and the spine. The disease occurs with almost equal frequency in women and men aged 40-50 years [2], with the axial form involving the spine being more often observed in men, and the peripheral form in women. PsA is frequently associated with uveitis, inflammatory bowel disease, cardiovascular disorders and osteoporosis [1]. Juvenile psoriatic arthritis (JPsA) is one of the subtypes of juvenile idiopathic arthritis (JIA) and accounts for approximately 1-7% of all JIA cases [29]. In the ILAR classification, its diagnosis is based on the presence of chronic arthritis coexisting with skin psoriasis or - in its absence - at least two of the following features: dactylitis, typical nail changes (pitting, onycholysis) or psoriasis in a first-degree relative [29, 30]. This entity is characterized by a heterogeneous clinical phenotype, and two subgroups can be distinguished: one with early onset and presence of ANA antibodies and another with features of spondyloarthropathy [30]. The heterogeneity of the clinical picture and the ambiguity of pathogenetic data mean that JPsA remains the subject of ongoing classification debate [29, 30].

The pathogenesis of PsA has not been fully elucidated, but its development is associated with the interplay of immunological, genetic and environmental mechanisms. Genetic studies have shown that specific HLA alleles (including HLA-B08:01, HLA-B38, HLA-B39:01 and HLA-C07:01) increase susceptibility to both peripheral and axial forms of the disease, characterized by asymmetric sacroiliitis and discontinuous spinal involvement with non-marginal syndesmophytes. In contrast, the HLA-B27 allele is mainly associated with axial PsA, presenting as symmetric sacroiliitis [1, 3].

Activation of immune system cells, especially dendritic cells and macrophages, plays a key role in PsA pathogenesis. Antigen presentation to T lymphocytes by these cells initiates the immune response and leads to increased production of pro-inflammatory cytokines such as TNF-alpha, IL-17, IL-23, IL-1 and IL-6, which are responsible for maintaining chronic inflammation and progressive joint damage. T helper 1 (Th1), T helper 17 (Th17) and CD8+ lymphocytes are particularly important, accumulating in the synovial membrane and synovial

fluid, where they secrete numerous inflammatory mediators that promote the development of inflammation and destruction of joint tissues [5]. Macrophages, mast cells, NK cells and innate lymphoid cells (ILC) also participate in the disease process, further sustaining the inflammatory response through cytokine secretion [3, 4]. The IL-23/IL-17 axis is currently regarded as one of the main mechanisms driving the development and progression of PsA [4]. The combined involvement of TNF-alpha, IL-23 and IL-17A in maintaining chronic inflammation has led to the development and implementation of targeted therapies directed against these key mediators [5]. As a result, it has become possible to use TNF-alpha-blocking drugs (TNF-i) and IL-17A inhibitors (IL-17A-i), such as secukinumab, ixekizumab and bimekizumab [1, 5], as well as a new class of oral agents - Janus kinase inhibitors (JAK-i), including tofacitinib and upadacitinib [2].

TNF-alpha is one of the key mediators of the inflammatory response in PsA. By binding to TNFR1 and TNFR2 receptors, it initiates an intracellular signalling cascade leading to activation of NF-κB and caspases and increased expression of numerous pro-inflammatory genes [6]. Consequently, production of secondary cytokines (including IL-1, IL-6), expression of adhesion molecules and chemokines, and recruitment of inflammatory cells to the synovial membrane are enhanced. This sequence of events contributes to persistence of chronic inflammation and gradual destruction of joint structures [1]. TNF-i, by specifically binding TNF-alpha or its receptors, lead to functional neutralization of this cytokine. These drugs thereby interrupt signalling to effector cells and suppress the pro-inflammatory cascade described above [14].

Within this drug class, several molecules with distinct structural and pharmacological profiles can be distinguished. Infliximab is a recombinant chimeric IgG1 monoclonal antibody that binds all forms of TNF-alpha, blocks its interaction with soluble and transmembrane receptors, induces lysis of cells expressing TNF-alpha and reduces IFN-gamma production [6]. Etanercept, a fusion protein composed of a dimer of extracellular fragments of TNFR2 linked to the Fc fragment of IgG1, functions as a “decoy receptor” for soluble and membrane TNF-alpha, preferentially binding its trimeric, biologically active form. Additionally, it influences the expression of pro-inflammatory genes (NF-κB) and induces apoptosis of dendritic cells, disrupting the positive feedback loop dependent on TNF-alpha. Adalimumab, a fully human IgG1 antibody, is characterized by a longer half-life and lower immunogenicity. It reduces TNF-alpha and IL-6 levels and acute-phase reactants and modulates the Th17/Treg axis by inhibiting IL-17 secretion and increasing the proportion of Treg cells. Certolizumab pegol, a PEGylated Fab fragment lacking an Fc region, does not induce complement- or antibody-

dependent cytotoxicity; due to PEGylation, it penetrates inflamed tissues more effectively and persists longer in these sites. Golimumab, a fully humanized IgG1 antibody with high affinity for TNF-alpha, efficiently neutralizes the soluble and membrane form of the cytokine and limits leukocyte infiltration by reducing the expression of adhesion molecules and secretion of pro-inflammatory cytokines [6].

IL-17 inhibitors, such as secukinumab, ixekizumab and bimekizumab, are monoclonal antibodies directed against IL-17A or elements of its pathway and block the key IL-23/IL-17 axis responsible for maintaining skin and joint lesions [5]. In contrast, Janus kinase inhibitors (JAK), represented among others by tofacitinib and upadacitinib, act at the level of the JAK-STAT pathway. By binding to JAK kinases, they inhibit their activity, blocking phosphorylation of STAT proteins and limiting the expression of genes regulated by numerous cytokines [9, 11, 12, 13]. Tofacitinib is an orally administered inhibitor primarily of JAK1/JAK3, with minor effects on JAK2, and its pharmacokinetics are closely linked to metabolism via CYP3A4/CYP2C19 [7, 8]. Upadacitinib, a selective JAK1 inhibitor, preferentially suppresses JAK-dependent signalling, modulating several cytokines (IL-6, IL-23, interferons) and systemically reducing inflammatory activity [10].

In this review article, the focus is on comparing the effects of three key drug classes used in psoriatic arthritis: TNF-alpha inhibitors, IL-17 inhibitors and JAK kinase inhibitors. Their mechanisms of action, clinical efficacy in the main disease domains (peripheral joints, axial involvement, skin) and available safety data are discussed, with particular emphasis on cardiovascular risk, infections and malignancies, in order to better understand the role of each of these therapies in modern targeted treatment of PsA and JPsA.

Research materials and methods

This work is a narrative literature review aimed at assessing the efficacy and safety of three main classes of targeted therapies used in psoriatic arthritis: TNF-alpha inhibitors, IL-17 inhibitors and JAK kinase inhibitors, with a separate discussion of available data on juvenile psoriatic arthritis. The literature search was carried out in the PubMed (MEDLINE) database and restricted to publications in English. Combinations of keywords and MeSH terms were used: “psoriatic arthritis”, “juvenile psoriatic arthritis”, “TNF inhibitors”, “tumor necrosis factor”, “IL-17 inhibitors”, “interleukin-17”, “secukinumab”, “JAK inhibitors”, “Janus kinase inhibitors”, “tofacitinib”, “upadacitinib”, “efficacy”, “safety”, combined with AND/OR operators.

Research results

For analysis, randomized, controlled phase II-III clinical trials (RCTs) and their open-label extensions evaluating the efficacy and/or safety of TNF-alpha inhibitors, IL-17 inhibitors or JAK inhibitors in adult patients with active PsA were primarily included. In addition, larger observational studies with real-world data and selected review articles and systematic literature analyses were taken into account if they synthesized the results of key primary studies relevant to the aim of the work. Studies of purely descriptive character (single case reports, small case series), conference abstracts without full publication and papers outside the scope of interest (e.g. dealing solely with psoriasis without joint involvement) were excluded from further analysis. Due to the narrative character of the review, literature selection was partly subjective and was not conducted according to formal systematic review guidelines (e.g. PRISMA).

Effectiveness of TNF-alpha inhibitors using etanercept and infliximab as examples

In one of the key phase III studies conducted by Mease et al., a total of 205 adult patients with active PsA were randomized to treatment with etanercept or placebo. Patients received etanercept 25 mg subcutaneously twice weekly for 24 weeks or placebo. After three months, an ACR20 response was achieved in approximately six out of ten patients treated with etanercept, whereas in the placebo group this proportion was several times lower and amounted to about 15%. An ACR50 response was observed in nearly 40% of patients receiving etanercept and only in a small proportion of those on placebo, reflecting a clear advantage of the active drug [16, 35]. Similarly marked differences were noted for PsARC criteria - after 24 weeks, about 70% of patients on etanercept and 23% in the placebo group met the response definition, and treatment was also associated with significant improvement in quality of life and function as assessed by the HAQ (Health Assessment Questionnaire) [35]. In patients with skin involvement, etanercept significantly improved the severity of psoriatic lesions: after 24 weeks, PASI75, i.e. $\geq$ 75% improvement in the Psoriasis Area and Severity Index, was achieved in 23% of patients in the etanercept group compared with 3% in the placebo group ($p = 0.001$) [16]. In summary, the results of the Mease et al. trial confirm that etanercept rapidly and clinically significantly reduces joint activity and the severity of skin lesions in patients with PsA. From a practical perspective, this strengthens the position of etanercept as one of the main first-line biologic options in patients with simultaneous joint and skin involvement [16].

Long-term efficacy and effects on radiographic progression have been demonstrated both in the open-label extension of the Mease et al. study and in the large, non-interventional, prospective PRERA trial (Etanercept is Effective and Halts Radiographic Progression in

Rheumatoid Arthritis and Psoriatic Arthritis) conducted in Germany. In PsA patients treated with etanercept, the annual median change in the modified total Sharp score (mTSS) was 0, indicating no meaningful radiographic progression in the typical case, and annualized mTSS progression was clearly greater in the period before initiation of etanercept than during therapy, with a higher proportion of patients without radiographic progression after starting the drug [15].

The efficacy of infliximab in psoriatic arthritis was assessed, among others, in two randomized, double-blind phase III trials, IMPACT [17] and IMPACT 2 [18, 19]. In IMPACT 2, which included 200 patients with active PsA, infliximab at a dose of 5 mg/kg was compared with placebo. After about three months of treatment, an ACR20 response was seen in nearly 60% of patients receiving infliximab, whereas in the placebo arm only one in ten patients achieved such a response. Differences in favour of infliximab were also evident for the more stringent ACR50/70 criteria and for improvement in skin lesions assessed by PASI75. Notably, in a subset of patients a very deep response (ACR70) could be maintained for a prolonged period, defined as major clinical response - maintenance of ACR70 for at least 24 consecutive weeks - which was demonstrated for the first time in a PsA population [18, 19].

Data from a separate radiographic analysis of the IMPACT 2 population also indicate a beneficial effect of infliximab on radiographic progression [36]. After 54 weeks of treatment, mTSS remained practically stable in patients who had received infliximab from the beginning of the study, whereas those initially treated with placebo (with later switch to infliximab) showed a greater increase in mTSS. These findings confirm that infliximab not only rapidly reduces joint inflammation and skin lesions, but also inhibits structural damage progression in PsA while maintaining an acceptable safety profile [36]. Radiographic data suggest that early initiation of infliximab may limit structural joint destruction.

Results from RCTs with etanercept and infliximab indicate that TNF inhibitors are highly effective in treating psoriatic arthritis, providing both reduction of joint inflammation and improvement in skin manifestations. In the Mease et al. trial, etanercept yielded statistically significant ACR20/50 response rates and skin improvement (PASI75) [16], and importantly, long-term data from extension studies and the PRERA observational program confirmed inhibition of radiographic progression with a favourable safety profile [15]. In the IMPACT and IMPACT 2 trials, infliximab showed very high ACR20/50/70 and PASI75 response rates and significant inhibition of structural damage assessed by the modified Sharp/van der Heijde score, confirming that different TNF inhibitors effectively modify the course of PsA across various disease phenotypes [18, 19, 36].

Effectiveness of IL-17 inhibitors (IL-17-i) using secukinumab as an example

In the FUTURE 2 trial, several doses of secukinumab were evaluated, demonstrating a clear clinical effect in all active-treatment arms. Regardless of dose, the majority of patients met ACR20 criteria, with the highest response rates observed in the 300 mg and 150 mg groups. The lowest dose, 75 mg, was also effective, though with smaller response rates, confirming a dose-dependent relationship between drug exposure and clinical effect. Clinically meaningful improvements were also maintained in other key PsA domains, including function (HAQ-DI), enthesitis, dactylitis and severity of skin lesions, and the therapeutic effect was observed both in patients naive to TNF-alpha inhibitors and in those previously exposed to TNF-i [32, 33]. Secukinumab provides long-term improvement across all PsA domains, particularly when used as the first biologic therapy.

In a separate subgroup analysis of FUTURE 2, Kavanaugh et al. showed that responses were clearly higher in patients not previously treated with TNF-i than in those who had received this drug class [32, 33]. At week 24, the ACR20 response rate for secukinumab 300 mg was 58.2% in TNF-i-naive patients versus 45.5% in those previously treated with TNF-i, and for the 150 mg dose, 63.5% and 29.7%, respectively. A similar trend was observed for other efficacy endpoints, suggesting that the greatest clinical benefit from secukinumab is achieved when it is used as first-line biologic therapy. The safety profile of secukinumab in FUTURE 2 remained stable over time - the frequency and type of adverse events were similar to earlier reports for this molecule, and long-term follow-up revealed no new safety signals or disproportionate increases in serious adverse events [32, 33].

Final 5-year results from the FUTURE 1 trial confirm that secukinumab provides durable improvement in PsA symptoms across the main disease domains. After five years of treatment, 71.0% of patients achieved an ACR20 response, 51.8% ACR50 and 36.3% ACR70, with sustained functional and quality-of-life benefits. In more than one-third of patients, dose escalation from 150 to 300 mg was required, which was associated with further efficacy improvement and outcomes comparable to those who remained on 150 mg from the start. The safety profile of secukinumab remained favourable and stable, with exposure-adjusted rates of serious infections, candidiasis, Crohn's disease and major cardiovascular events being low and no new safety signals emerging during long-term therapy [34]. From a clinical perspective, long-term FUTURE 1 data indicate that secukinumab can be used long-term with maintained efficacy and a stable safety profile, making it suitable as a chronic therapy rather than only a short-term intervention during disease flares [32-34].

Effectiveness of JAK inhibitors (JAK-i) using upadacitinib and tofacitinib and comparison with TNF-i

In SELECT-PsA 2, a randomized, double-blind phase III trial involving 642 patients with active psoriatic arthritis and inadequate response or intolerance to at least one biologic disease-modifying drug, the efficacy and safety of upadacitinib 15 mg and 30 mg once daily were assessed versus placebo [20]. In this trial, which included patients after failure of prior biologic therapies, both upadacitinib doses were superior to placebo in terms of ACR20 response rates as early as week 12. Approximately half of patients receiving 15 mg and slightly over 60% of those on 30 mg met ACR20 criteria, while the response rate in the placebo group was several times lower. At week 24, minimal disease activity (MDA) was also achieved more frequently in the active-treatment arms than in the control group, confirming a strong anti-inflammatory effect of upadacitinib in this difficult-to-treat population [20].

The most commonly reported adverse events included upper respiratory tract infections, elevations in liver enzymes, increased creatine kinase levels and nausea; their overall frequency was similar in the placebo and 15 mg upadacitinib groups, whereas the 30 mg dose was associated with a higher rate of adverse events, including infections. The incidence of serious infections was 0.5% in the placebo group, 0.5% in the 15 mg group and 2.8% in the 30 mg group, reflecting a dose-dependent increase in infection risk at the higher dose. Nevertheless, the overall safety profile of upadacitinib was consistent with previous observations for this drug class, and the 15 mg dose is considered preferred due to its more favourable efficacy-to-risk ratio [20]. The SELECT-PsA 2 results show that upadacitinib is an effective therapeutic option for PsA patients after failure of prior biologic therapies, providing high response rates despite the challenging patient population [20].

In two randomized, double-blind phase III trials, OPAL Broaden and OPAL Beyond, the efficacy and safety of oral tofacitinib were evaluated in patients with active psoriatic arthritis. OPAL Broaden included patients with inadequate response to conventional synthetic disease-modifying drugs (csDMARDs) [21], whereas OPAL Beyond enrolled patients with insufficient response to prior TNF-i therapy [22]. In OPAL Broaden, patients were randomized to tofacitinib 5 mg or 10 mg twice daily, adalimumab 40 mg every 2 weeks, or placebo; the primary endpoint was the proportion of patients achieving ACR20 at month 3. ACR20 was achieved in 50% of patients treated with tofacitinib 5 mg BID and 61% with 10 mg BID, compared with 33% in the placebo group ($p = 0.01$ for 5 mg vs placebo; $p < 0.001$ for 10 mg vs placebo), with efficacy comparable to adalimumab (ACR20: 52%) [21].

Tofacitinib also significantly improved function and quality of life: mean change in HAQ-DI (Health Assessment Questionnaire - Disability Index) was -0.35 and -0.40 for the 5 mg and 10 mg doses, respectively, versus -0.18 for placebo ($p = 0.006$ and $p < 0.001$), and beneficial effects were observed in skin lesions and other disease domains assessed in PRO analyses [22]. The safety profile was consistent with prior experience for JAK-I - adverse events more frequent than with placebo mainly included upper respiratory tract infections, elevated liver enzyme activity and lipid abnormalities; during the 12-month follow-up, isolated cases of malignancies, serious infections and herpes zoster were reported, which must be considered in individual risk assessment. OPAL Beyond, in patients after TNF-i failure, similarly showed significantly higher ACR20 response rates and HAQ-DI improvements with tofacitinib versus placebo [22].

Taken together, the SELECT-PsA 2 and OPAL Broaden/Beyond results indicate that both upadacitinib and tofacitinib are effective therapeutic options for patients with active PsA, including those with inadequate response to prior biologic treatment [20-22]. Upadacitinib, particularly at 15 mg, shows very high and rapidly emerging ACR20 and MDA response rates with an acceptable safety profile [20]. Tofacitinib demonstrates significantly better efficacy than placebo in patients after csDMARDs as well as after TNF-i [21], although its use is associated with typical JAK-i adverse events such as infections and biochemical abnormalities [22]. In clinical practice, the choice of a specific JAK inhibitor should take into account not only anti-inflammatory potency and RCT data, but also the patient's individual risk profile (age, comorbidities, cardiovascular and thrombotic risk) and preferences regarding oral therapy.

Safety profile

In the study by Zhao et al., the safety of JAK inhibitors was compared with that of TNF-alpha inhibitors and IL-17 inhibitors in patients with psoriatic arthritis and axial spondyloarthropathy (axSpA) in real-world clinical practice [23]. Data from electronic medical records of a North American population were used to identify adult patients diagnosed with PsA or axSpA who initiated treatment with tofacitinib or upadacitinib (JAK-i), TNF-i or IL-17-i. Patients starting JAK-i therapy were matched 1:1 using propensity-score methods to those starting TNF-i and separately to those starting IL-17 inhibitors, and Cox models were then applied to assess the risk of major cardiovascular events (myocardial infarction, stroke, revascularization procedures) and common solid malignancies (breast, colorectal, lung, prostate) over 3 years of follow-up [23].

The analysis included 2,200 matched patients in each of the JAK-i and TNF-i groups (3,092 and 4,618 patient-years, respectively) and 2,287 patients in each of the JAK-i and IL-17-i groups. Compared with TNF-i, the use of JAK-i was not associated with an increased risk of cardiovascular events (HR 0.98; 95% CI 0.63-1.51) or solid malignancies (HR 0.71; 95% CI 0.46-1.09), and similar results were obtained in comparison with IL-17-i (for cardiovascular events HR 1.11; 95% CI 0.72-1.72; for malignancies HR 0.74; 95% CI 0.48-1.12) [23]. The direction and magnitude of effects remained similar across multiple sensitivity analyses (1- and 5-year follow-up, patients ≥65 years, treatment initiation before 2021), allowing the authors to conclude that in a large PsA and axSpA population, JAK-i are not associated with a significantly higher risk of major cardiovascular events or the most common solid tumours compared with TNF-i or IL-17-i, although continued long‑term safety monitoring is required [23].

In available real-world analyses involving patients with PsA and axial spondyloarthropathy, JAK inhibitors were not associated with a significantly higher risk of major cardiovascular events or the most frequent solid malignancies compared with TNF-alpha or IL-17 inhibitors (HR values close to 1, 95% CIs including 1) [23]. On the other hand, results from the ORAL Surveillance trial in rheumatoid arthritis (RA) showed a significantly increased risk of serious cardiovascular events, malignancies, venous thromboembolism and severe infections with tofacitinib versus TNF-i in high-risk patients [24]. Although this study involved an RA population, the safety signals observed for tofacitinib are relevant when assessing the risk-benefit profile of the entire JAK-i class and should be taken into account in therapeutic decision-making.

Therapeutic management in juvenile PsA

In juvenile psoriatic arthritis and other forms of juvenile spondyloarthropathy, biologic treatment is based primarily on TNF-i and newer targeted agents, IL-17-i [27, 28]. Etanercept, evaluated in the long-term CLIPPER/CLIPPER2 programme in children and young adults with extended juvenile idiopathic arthritis (JIA), enthesitis-related arthritis (ERA) and juvenile PsA, showed sustained clinical benefit: over 10 years of follow-up, more than 45% of patients achieved a Juvenile Idiopathic Arthritis - American College of Rheumatology 30% improvement (JIA-ACR30) from month 2 of treatment, and 33% and 27% of patients met remission criteria according to the Juvenile Arthritis Disease Activity Score (JADAS) and

ACR, respectively, with no new safety signals and decreasing rates of adverse events over time [26].

In the randomized, double-blind phase 3 JUNIPERA trial (NCT03031782), Brunner et al. evaluated secukinumab in patients with active ERA and JPsA refractory to conventional therapy. After 12 weeks of treatment in the open-label phase, most patients achieved a JIA-ACR30 response, and after randomization, a significantly longer time to disease flare was demonstrated in the secukinumab arm compared with placebo (HR 0.28; 95% CI 0.13-0.63; approximately 72% reduction in flare risk), with sustained high JIA-ACR response rates and a favourable safety profile up to week 104 [25]. In paediatric practice, data from the CLIPPER programme indicate that etanercept is a well-documented and long-term safe biologic option in children with extended JIA, ERA and JPsA. This reinforces its role as a first-choice drug in this patient group, particularly in cases with polyarticular involvement [25].

Current reviews of juvenile spondyloarthropathy therapy emphasize that biologic disease-modifying drugs provide clinically meaningful improvement in children with ERA and JPsA, with TNF inhibitors remaining first-line therapy, while IL-17 inhibitors represent an important option, especially when skin involvement is prominent. At the same time, due to the relatively small number of paediatric studies, many therapeutic decisions continue to rely partly on extrapolation from adult PsA and from other juvenile idiopathic arthritis subtypes [27, 28].

Discussion

The presented data confirm that the efficacy of targeted therapies in PsA results from their direct impact on key immunopathogenetic mechanisms of the disease. TNF-alpha inhibitors, IL-17 inhibitors and JAK inhibitors act on distinct but interrelated elements of the inflammatory response, which translates into their usefulness across different clinical domains of PsA. TNF-i remain the reference point for modern PsA treatment because they have the longest clinical experience and a broad evidence base confirming their efficacy. Their importance derives not only from effects on joint and skin symptoms, but also from documented ability to limit structural progression, strengthening their position as therapies of established clinical value [16].

IL-17 inhibitors, represented in the analysed studies mainly by secukinumab, expand therapeutic possibilities particularly in patients with a predominant skin-joint phenotype. Long-term observations suggest that this drug class combines durable clinical effects with a favourable safety profile, and efficacy may be especially marked in patients receiving them at an earlier stage of biologic treatment [32-34].

JAK inhibitors occupy an important place in the therapy of more difficult-to-treat patients, especially after failure of previous strategies. Their advantages include oral administration and broad influence on cytokine pathways; however, this mechanism of action justifies more cautious patient selection, particularly with regard to infection, cardiovascular and thrombotic risks. In relation to JAK-i, available data therefore argue for slightly more cautious qualification of patients, in light of safety signals observed in other populations [2, 20-22]. A key aspect in interpreting JAK-i data is the comparison of randomized trial results with real-world observations. Although available analyses in PsA and axSpA have not shown a significant increase in cardiovascular or oncologic risk versus TNF-i and IL-17-i, ORAL Surveillance indicates that safety assessment of this class should also take into account experience from other inflammatory diseases. In practice, this means that therapeutic decisions must be individualized rather than automatically transferring results between populations [23,24].

With regard to juvenile PsA, the available data are clearly more limited than in adults. Results of studies on etanercept and secukinumab are promising and support the use of biologic therapies in children, but the small number of trials and clinical heterogeneity of JPsA mean that a substantial evidence gap persists in this group. Consequently, treatment of JPsA requires particular caution, and further paediatric studies are necessary to more precisely define optimal therapeutic strategies [25, 27-28]. Because of the narrative character of this review, the conclusions are primarily illustrative and do not replace formal society guidelines. Interpretation of results should take into account the lack of full standardization of search and selection methods.

Conclusions

Psoriatic arthritis, both in adults and in its juvenile form, is a multidomain disease whose treatment should account for clinical heterogeneity and complex immunologic background. The development of targeted therapies against TNF-alpha, IL-17 and the JAK-STAT pathway has significantly expanded options for effective control of disease activity and limitation of structural consequences [2,5]. Based on the analysed studies, TNF-alpha inhibitors remain one of the best-documented drug classes in PsA, with established clinical efficacy and data confirming their importance in long-term disease control [16]. IL-17 inhibitors are a valuable alternative, particularly in patients with significant skin involvement and in cases of inadequate response to TNF-i [32-34]. JAK inhibitors are effective therapeutic options after failure of previous treatment, but their use requires careful safety assessment and an individualized risk

profile [20-22]. Available clinical-practice data do not clearly indicate higher cardiovascular or oncologic risk in PsA, but results from other patient populations justify caution when qualifying patients for this class [23, 24].

In juvenile PsA, biologic treatment is mainly based on TNF inhibitors, while IL-17 inhibitors represent a promising and increasingly well-documented therapeutic option. However, the limited number of paediatric studies means that some therapeutic decisions still rely on extrapolation from adult PsA and other JIA subtypes. In clinical practice, therapy choice should be individualized, taking into account predominant disease domains, prior treatment, comorbidities and the safety profile of each drug class. Further studies, especially in the paediatric population, are needed to better define the place of individual therapies in the treatment of JPsA [25, 27-28].

Disclosure

Author's Contribution

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References

1. Azuaga, A. B., Ramírez, J., & Cañete, J. D. (2023). Psoriatic Arthritis: Pathogenesis and Targeted Therapies. International journal of molecular sciences, *24*(5), 4901. https://doi.org/10.3390/ijms24054901
2. Ocampo D, V., & Gladman, D. (2019). Psoriatic arthritis. F1000Research, 8, F1000 Faculty Rev-1665. https://doi.org/10.12688/f1000research.19144.1
3. Fitzgerald, O., & Winchester, R. (2009). Psoriatic arthritis: from pathogenesis to therapy. Arthritis research & therapy, 11(1), 214. https://doi.org/10.1186/ar2580
4. Saeed, F., & Adamopoulos, I. E. (2025). Pathogenesis of psoriatic arthritis: new insights from a bone marrow perspective. Current opinion in rheumatology, 37(2), 136–141. https://doi.org/10.1097/BOR.0000000000001064
5. Kannappan, R., Kim, S., Lau, A., & Brent, L. H. (2025). Psoriatic Arthritis: From Diagnosis to Treatment. Journal of clinical medicine, 14(22), 8151. https://doi.org/10.3390/jcm14228151
6. Jang, D. I., Lee, A. H., Shin, H. Y., Song, H. R., Park, J. H., Kang, T. B., Lee, S. R., & Yang, S. H. (2021). The Role of Tumor Necrosis Factor Alpha (TNF-α) in Autoimmune Disease and Current TNF-α Inhibitors in Therapeutics. International journal of molecular sciences, 22(5), 2719. https://doi.org/10.3390/ijms22052719
7. Berekmeri, A., Mahmood, F., Wittmann, M., & Helliwell, P. (2018). Tofacitinib for the treatment of psoriasis and psoriatic arthritis. Expert review of clinical immunology, 14(9), 719–730. https://doi.org/10.1080/1744666X.2018.1512404
8. Wang, T., Wu, W., Zhang, X., Gan, B., Zhou, Y., & Cheng, X. (2025). Tofacitinib treatment for plaque psoriasis and psoriatic arthritis: A meta-analysis of randomised controlled trials. Indian journal of dermatology, venereology and leprology, 91(2), 172–179. https://doi.org/10.25259/IJDVL_14_2024
9. Banerjee, S., Biehl, A., Gadina, M., Hasni, S., & Schwartz, D. M. (2017). JAK-STAT Signaling as a Target for Inflammatory and Autoimmune Diseases: Current and Future Prospects. *Drugs*, *77*(5), 521–546. https://doi.org/10.1007/s40265-017-0701-9
10. Mohamed, M. F., Bhatnagar, S., Parmentier, J. M., Nakasato, P., & Wung, P. (2024). Upadacitinib: Mechanism of action, clinical, and translational science. Clinical and translational science, 17(1), e13688. https://doi.org/10.1111/cts.13688
11. Xin, P., Xu, X., Deng, C., Liu, S., Wang, Y., Zhou, X., Ma, H., Wei, D., & Sun, S. (2020). The role of JAK/STAT signaling pathway and its inhibitors in diseases.

International immunopharmacology, 80, 106210. https://doi.org/10.1016/j.intimp.2020.106210

12. O'Shea, J. J., Schwartz, D. M., Villarino, A. V., Gadina, M., McInnes, I. B., & Laurence, A. (2015). The JAK-STAT pathway: impact on human disease and therapeutic intervention. Annual review of medicine, 66, 311–328. https://doi.org/10.1146/annurev-med-051113-024537
13. Lv, Y., Qi, J., Babon, J. J., Cao, L., Fan, G., Lang, J., Zhang, J., Mi, P., Kobe, B., & Wang, F. (2024). The JAK-STAT pathway: from structural biology to cytokine engineering. Signal transduction and targeted therapy, 9(1), 221. https://doi.org/10.1038/s41392-024-01934-w
14. Esposito, E., & Cuzzocrea, S. (2009). TNF-alpha as a therapeutic target in inflammatory diseases, ischemia-reperfusion injury and trauma. *Current medicinal chemistry*, *16*(24), 3152–3167. https://doi.org/10.2174/092986709788803024
15. Wassenberg, S., Rau, R., Klopsch, T., Plenske, A., Jobst, J., Klaus, P., Meng, T., & Löschmann, P. A. (2023). Etanercept is Effective and Halts Radiographic Progression in Rheumatoid Arthritis and Psoriatic Arthritis: Final Results from a German Non-interventional Study (PRERA). Rheumatology and therapy, 10(1), 117–133. https://doi.org/10.1007/s40744-022-00491-4
16. Mease, P. J., Kivitz, A. J., Burch, F. X., Siegel, E. L., Cohen, S. B., Ory, P., Salonen, D., Rubenstein, J., Sharp, J. T., & Tsuji, W. (2004). Etanercept treatment of psoriatic arthritis: safety, efficacy, and effect on disease progression. Arthritis and rheumatism, 50(7), 2264–2272. https://doi.org/10.1002/art.20335
17. Antoni, C. E., Kavanaugh, A., van der Heijde, D., Beutler, A., Keenan, G., Zhou, B., Kirkham, B., Tutuncu, Z., Burmester, G. R., Schneider, U., Furst, D. E., Molitor, J., Keystone, E., Gladman, D. D., Manger, B., Wassenberg, S., Weier, R., Wallace, D. J., Weisman, M. H., Kalden, J. R., … Smolen, J. S. (2008). Two-year efficacy and safety of infliximab treatment in patients with active psoriatic arthritis: findings of the Infliximab Multinational Psoriatic Arthritis Controlled Trial (IMPACT). The Journal of rheumatology, 35(5), 869–876. https://www.jrheum.org/content/35/5/869.long
18. Antoni, C., Krueger, G. G., de Vlam, K., Birbara, C., Beutler, A., Guzzo, C., Zhou, B., Dooley, L. T., Kavanaugh, A., & IMPACT 2 Trial Investigators (2005). Infliximab improves signs and symptoms of psoriatic arthritis: results of the IMPACT 2 trial. Annals of the rheumatic diseases, 64(8), 1150–1157. https://doi.org/10.1136/ard.2004.032268

19. Kavanaugh, A., Krueger, G. G., Beutler, A., Guzzo, C., Zhou, B., Dooley, L. T., Mease, P. J., Gladman, D. D., de Vlam, K., Geusens, P. P., Birbara, C., Halter, D. G., Antoni, C., & IMPACT 2 Study Group (2007). Infliximab maintains a high degree of clinical response in patients with active psoriatic arthritis through 1 year of treatment: results from the IMPACT 2 trial. *Annals of the rheumatic diseases*, *66*(4), 498–505. https://doi.org/10.1136/ard.2006.058339
20. Mease, P. J., Lertratanakul, A., Anderson, J. K., Papp, K., Van den Bosch, F., Tsuji, S., Dokoupilova, E., Keiserman, M., Wang, X., Zhong, S., McCaskill, R. M., Zueger, P., Pangan, A. L., & Tillett, W. (2021). Upadacitinib for psoriatic arthritis refractory to biologics: SELECT-PsA 2. *Annals of the rheumatic diseases*, *80*(3), 312–320. https://doi.org/10.1136/annrheumdis-2020-218870
21. Mease, P., Hall, S., FitzGerald, O., van der Heijde, D., Merola, J. F., Avila-Zapata, F., Cieślak, D., Graham, D., Wang, C., Menon, S., Hendrikx, T., & Kanik, K. S. (2017). Tofacitinib or Adalimumab versus Placebo for Psoriatic Arthritis. *The New England journal of medicine*, *377*(16), 1537–1550. https://doi.org/10.1056/NEJMoa1615975
22. Gladman, D., Rigby, W., Azevedo, V. F., Behrens, F., Blanco, R., Kaszuba, A., Kudlacz, E., Wang, C., Menon, S., Hendrikx, T., & Kanik, K. S. (2017). Tofacitinib for Psoriatic Arthritis in Patients with an Inadequate Response to TNF Inhibitors. The New England journal of medicine, 377(16), 1525–1536. https://doi.org/10.1056/NEJMoa1615977
23. Zhao, S. S., Riley, D., Hernandez, G., & Alam, U. (2025). Comparative safety of JAK inhibitors versus TNF or IL-17 inhibitors for cardiovascular disease and cancer in psoriatic arthritis and axial spondyloarthritis. Clinical therapeutics, 47(4), 293–297. https://doi.org/10.1016/j.clinthera.2025.01.005
24. Ytterberg, S. R., Bhatt, D. L., Mikuls, T. R., Koch, G. G., Fleischmann, R., Rivas, J. L., Germino, R., Menon, S., Sun, Y., Wang, C., Shapiro, A. B., Kanik, K. S., Connell, C. A., & ORAL Surveillance Investigators (2022). Cardiovascular and Cancer Risk with Tofacitinib in Rheumatoid Arthritis. The New England journal of medicine, 386(4), 316–326. https://doi.org/10.1056/NEJMoa2109927
25. Brunner, H. I., Foeldvari, I., Alexeeva, E., Ayaz, N. A., Calvo Penades, I., Kasapcopur, O., Chasnyk, V. G., Hufnagel, M., Żuber, Z., Schulert, G., Ozen, S., Rakhimyanova, A., Ramanan, A., Scott, C., Sozeri, B., Zholobova, E., Martin, R., Zhu, X., Whelan, S., Pricop, L., … Paediatric Rheumatology INternational Trials Organization (PRINTO) and Pediatric Rheumatology Collaborative Study Group (PRCSG) (2023).

Secukinumab in enthesitis-related arthritis and juvenile psoriatic arthritis: a randomised, double-blind, placebo-controlled, treatment withdrawal, phase 3 trial. Annals of the rheumatic diseases, 82(1), 154–160. https://doi.org/10.1136/ard-2022-222849

26. Vojinović, J., Foeldvari, I., Dehoorne, J., Panaviene, V., Susic, G., Horneff, G., Stanevicha, V., Kobusinska, K., Zuber, Z., Dobrzyniecka, B., Akikusa, J., Avcin, T., Borlenghi, C., Arthur, E., Tatulych, S. Y., Zang, C., Tsekouras, V., Vlahos, B., Martini, A., & Ruperto, N. (2024). Ten-year safety and clinical benefit from open-label etanercept treatment in children and young adults with juvenile idiopathic arthritis. Rheumatology (Oxford, England), 63(1), 140–148. https://doi.org/10.1093/rheumatology/kead183
27. Srinivasalu, H., Simpson, J., & Stoll, M. L. (2024). Drug therapy in juvenile spondyloarthritis. Current opinion in rheumatology, 36(4), 295–301. https://doi.org/10.1097/BOR.0000000000001016
28. Katsicas, M. M., & Russo, R. (2016). Biologic agents in juvenile spondyloarthropathies. Pediatric rheumatology online journal, 14(1), 17. https://doi.org/10.1186/s12969-016-0076-6
29. Brunello, F., Tirelli, F., Pegoraro, L., Dell'Apa, F., Alfisi, A., Calzamatta, G., Folisi, C., & Zulian, F. (2022). New Insights on Juvenile Psoriatic Arthritis. Frontiers in pediatrics, 10, 884727. https://doi.org/10.3389/fped.2022.884727
30. Naddei, R., Rebollo-Giménez, A., Burrone, M., Natoli, V., Rosina, S., Consolaro, A., & Ravelli, A. (2023). Juvenile Psoriatic Arthritis: Myth or Reality? An Unending Debate. Journal of clinical medicine, 12(1), 367. https://doi.org/10.3390/jcm12010367
31. Mikołajczak, K. (2025). The Role of Physical Activity in the Therapy of Psoriatic Arthritis - Review. *Quality in Sport*, *40*, 59823. https://doi.org/10.12775/QS.2025.40.59823
32. McInnes, I. B., Mease, P. J., Ritchlin, C. T., Rahman, P., Gottlieb, A. B., Kirkham, B., Kajekar, R., Delicha, E. M., Pricop, L., & Mpofu, S. (2017). Secukinumab sustains improvement in signs and symptoms of psoriatic arthritis: 2 year results from the phase 3 FUTURE 2 study. Rheumatology (Oxford, England), 56(11), 1993–2003. https://doi.org/10.1093/rheumatology/kex301
33. Kavanaugh, A., McInnes, I. B., Mease, P. J., Hall, S., Chinoy, H., Kivitz, A. J., Wang, Z., & Mpofu, S. (2016). Efficacy of Subcutaneous Secukinumab in Patients with Active Psoriatic Arthritis Stratified by Prior Tumor Necrosis Factor Inhibitor Use: Results

from the Randomized Placebo-controlled FUTURE 2 Study. *The Journal of rheumatology*, *43*(9), 1713–1717. https://doi.org/10.3899/jrheum.160275

34. Mease, P. J., Kavanaugh, A., Reimold, A., Tahir, H., Rech, J., Hall, S., Geusens, P., Pellet, P., Delicha, E. M., Pricop, L., Mpofu, S., & FUTURE 1 study group (2020). Secukinumab Provides Sustained Improvements in the Signs and Symptoms of Psoriatic Arthritis: Final 5-year Results from the Phase 3 FUTURE 1 Study. *ACR open rheumatology*, *2*(1), 18–25. https://doi.org/10.1002/acr2.11097
35. Culy, C. R., & Keating, G. M. (2002). Etanercept: an updated review of its use in rheumatoid arthritis, psoriatic arthritis and juvenile rheumatoid arthritis. *Drugs*, *62*(17), 2493–2537. https://doi.org/10.2165/00003495-200262170-00013
36. van der Heijde, D., Kavanaugh, A., Gladman, D. D., Antoni, C., Krueger, G. G., Guzzo, C., Zhou, B., Dooley, L. T., de Vlam, K., Geusens, P., Birbara, C., Halter, D., & Beutler, A. (2007). Infliximab inhibits progression of radiographic damage in patients with active psoriatic arthritis through one year of treatment: Results from the induction and maintenance psoriatic arthritis clinical trial 2. *Arthritis and rheumatism*, *56*(8), 2698–2707. https://doi.org/10.1002/art.22805