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EFFICACY AND SAFETY OF IL-23 INHIBITORS IN PSORIATIC ARTHRITIS: A LITERATURE REVIEW

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ABSTRACT

Introduction: Psoriatic arthritis (PsA) is a chronic, heterogeneous immune-mediated inflammatory disease affecting multiple clinical domains, including peripheral arthritis, skin lesions, enthesitis, and dactylitis. Given the key role of the IL-23/IL-17 axis in PsA pathogenesis, IL-23 inhibitors have emerged as an important therapeutic option.
Methodology: A structured literature search of the PubMed database was performed to identify studies evaluating the efficacy and safety of IL-23 inhibitors in PsA. The available evidence was based mainly on randomized trials of guselkumab, risankizumab, and tildrakizumab. Phase III randomized controlled trials were prioritized, while relevant meta-analyses, network meta-analyses, systematic reviews, and selected post hoc or pooled analyses were included to complement the assessment of multidomain outcomes, structural progression, and safety.
Results: IL-23 inhibitors showed significant efficacy in peripheral arthritis and provided clinically meaningful benefits in other important disease domains, including skin lesions, physical function, minimal disease activity, enthesitis, and dactylitis. The most robust evidence supported guselkumab and risankizumab, whereas the evidence base for tildrakizumab remained more limited. Guselkumab also showed the clearest evidence for inhibition of structural progression. Across studies, IL-23 inhibitors demonstrated a generally favorable and stable safety profile, with infections being the most commonly reported adverse events and no clear emerging long-term safety concerns.
Conclusions: IL-23 inhibitors represent an effective and clinically relevant therapeutic strategy in PsA, with benefits across multiple disease domains and a generally favorable safety profile. Current evidence most strongly supports guselkumab and risankizumab, while further data are needed to better define the role of tildrakizumab.

KEYWORDS

Psoriatic Arthritis, IL-23 Inhibitors, Guselkumab, Risankizumab, Tildrakizumab

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Introduction

Psoriatic arthritis (PsA) is a chronic, heterogeneous, immune-mediated inflammatory disease classified within the spondyloarthritis group. Its clinical manifestations include involvement of peripheral joints and the axial skeleton, as well as enthesitis, dactylitis, and skin and nail lesions, underscoring its multisystemic and clinically diverse nature. PsA is further associated with many comorbidities [1,2]. If left untreated or inadequately controlled, PsA can result in progressive joint damage, disability, and substantial reductions in quality of life [2,3]. This diminished quality of life arises not only from pain and restricted physical function, but also from fatigue, sleep disturbances, impaired mental health, and social and occupational challenges [3,4].

Psoriatic arthritis management is guided by strategies that prioritize achieving remission or, at a minimum, low disease activity. These strategies consider concomitant extra-articular manifestations and comorbidities, with the primary objective of maximizing health-related quality of life. For patients presenting with polyarthritis or poor prognostic factors, early initiation of conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) is recommended. In cases of peripheral arthritis where response to csDMARDs is inadequate, biologic therapy should be considered. Treatment selection should be informed by the presence of extra-articular symptoms and the safety profile of the therapy to optimize the balance of benefits and risks [5].

The pathogenesis of psoriatic arthritis involves a complex network of immunological, genetic, and environmental interactions. The IL-23/IL-17 axis is particularly significant, as it contributes to the activation and maintenance of chronic inflammation in joints, tendon insertions, skin, and other tissues. IL-23 facilitates the differentiation and proliferation of Th17 cells, which increases the production of pro-inflammatory cytokines and promotes tissue damage and sustained inflammation. Consequently, IL-23 is recognized as a critical component in the immunopathogenesis of psoriatic arthritis and serves as a key therapeutic target. The increasing recognition of IL-23's role in disease pathogenesis has led to the development of targeted therapies focused on this immune axis. In contrast to earlier approaches that inhibited the shared p40 subunit of IL-12 and IL-23, newer agents selectively target the IL-23 p19 subunit, enabling more precise modulation of the inflammatory response. Selective IL-23 inhibitors include monoclonal antibodies such as guselkumab, risankizumab, and tildrakizumab [2,6].

The aim of this paper is to present current data on the efficacy and safety of IL-23 inhibitors in the treatment of psoriatic arthritis, with a particular focus on randomized trial results and their impact on key clinical aspects of the disease.

Methodology

A structured literature search of the PubMed database was executed to assess the efficacy and safety of IL-23 inhibitors in psoriatic arthritis. Publications from 2021 to 2026 were included to focus on the most recent clinical evidence. The search strategy incorporated the keywords psoriatic arthritis, IL-23 inhibitors, guselkumab, risankizumab, tildrakizumab, and biologic therapy. Boolean operators AND and OR refined the search results. Phase III randomized controlled trials evaluating IL-23 inhibitors in patients with active psoriatic arthritis were prioritized. Relevant meta-analyses, network meta-analyses, systematic reviews, and selected post-hoc or pooled analyses were included to supplement the evaluation of peripheral arthritis outcomes, skin and multidomain manifestations, structural progression, and safety. Only full-text articles published in English were included. Studies that did not specifically address psoriatic arthritis or lacked clinically relevant outcome data were excluded. The selected publications underwent qualitative analysis, focusing on study design, patient characteristics, primary and secondary endpoints, and reported safety outcomes.

Results

Characteristics of Randomized Controlled Trials

Clinical evidence from the past five years on IL-23 inhibitors in psoriatic arthritis comes mainly from six randomized, double-blind, placebo-controlled trials evaluating guselkumab, risankizumab, and tildrakizumab in adults with active disease. Guselkumab was assessed in Phase III trials, including COSMOS, APEX, and DISCOVER-2 [7–9]. This review references the 2022 publication on DISCOVER-2, which reports long-term efficacy and safety outcomes beyond the initial double-blind period [9]. Risankizumab was studied in two Phase III trials, KEEPsAKE-1 and KEEPsAKE-2 [10,11]. Tildrakizumab was evaluated in a Phase IIb placebo-controlled trial. Although less evidence is available for tildrakizumab than for guselkumab and risankizumab, this study still provided valuable data on IL-23 inhibition in psoriatic arthritis [12].

The patient populations in these studies were generally comparable and consisted of adults meeting the classification criteria for psoriatic arthritis, with active peripheral arthritis and varying severity of other disease manifestations, including skin lesions, nail lesions, enthesitis, or dactylitis. However, there was a key distinction: prior exposure to treatment. The COSMOS trial included patients with an inadequate response to TNF inhibitors [7]. The APEX study enrolled biologic-naive patients [8]. Long-term efficacy and safety of guselkumab were based on the DISCOVER-2 trial, which included patients who received standard non-biologic therapies and had not previously been treated with biologics or JAK inhibitors [9]. In KEEPsAKE-1, patients had an inadequate response, intolerance, or contraindications to csDMARD therapy and were biologic-naïve [10]. In contrast, KEEPsAKE-2 included patients with an inadequate response to biologic therapy and/or csDMARDs [11]. The Phase IIb tildrakizumab trial included both patients without prior anti-TNF therapy and those with previous anti-TNF exposure [12].

Across these trials, the primary efficacy endpoint was typically the proportion of patients achieving at least 20% improvement according to the American College of Rheumatology criteria (ACR20) at week 24. Secondary endpoints included ACR50 and ACR70 responses, improvement in skin lesions assessed by Psoriasis Area and Severity Index (PASI), change from baseline in the Health Assessment Questionnaire–Disability Index (HAQ-DI), achievement of minimal disease activity (MDA), and resolution of enthesitis and dactylitis. Safety was assessed by monitoring adverse events (AEs) and serious adverse events (SAEs), as well as laboratory and physical examination findings and vital signs. Additional long-term safety data were provided in extension analyses and follow-up publications [7–12].

Peripheral Arthritis Outcomes

Outcomes related to peripheral arthritis in the included studies were primarily assessed based on the percentage of patients who achieved ACR20, ACR50, and ACR70 responses, most commonly at week 24. In randomized trials, IL-23 inhibitors showed significant efficacy in alleviating joint symptoms in patients with active psoriatic arthritis.

Guselkumab demonstrated consistent efficacy in improving peripheral joint symptoms across diverse patient populations with psoriatic arthritis. In the COSMOS study, which included patients with inadequate response to TNF inhibitors, 44.4% of patients treated with guselkumab achieved an ACR20 response at week 24, compared with 19.8% of patients receiving placebo. A beneficial treatment effect was observed as early as week 4. In the same analysis, the rates of ACR50 and ACR70 responses were also higher, at 19.6% versus 5.2% and 7.9% versus 1.0%, respectively, in favor of guselkumab. Importantly, the clinical response was sustained during follow-up, and among patients who achieved an ACR20, ACR50, or ACR70 response at week 24, 83.3%, 81.1%, and 86.7%, respectively, maintained it at week 48 [7]. An additional post hoc analysis of the COSMOS study showed that guselkumab's superiority over placebo in ACR20 and ACR50 responses was observed regardless of age, sex, BMI, or prior or current therapy [13]. Similar results were obtained in the APEX study, conducted in patients who had not previously received biologic therapy. At week 24, an ACR20 response was achieved by 66.6% of patients receiving guselkumab every 4 weeks and 68.3% of patients receiving the drug every 8 weeks, compared with 47.0% in the placebo group. More stringent response criteria also favored guselkumab treatment: ACR50 was achieved by 41.4% and 42.2% of patients in the guselkumab groups, respectively, compared with 20.5% in the placebo group, while ACR70 was achieved by 22.0% and 22.4% of patients treated with guselkumab and by 11.2% of patients receiving placebo. Differences compared with placebo were already evident by week 8 [8]. The long-term efficacy of guselkumab was confirmed by a publication on the DISCOVER-2 study. At week 100, an ACR20 response was achieved by 76% of patients receiving guselkumab every 4 weeks and 74% of patients treated every 8 weeks. ACR50 responses were observed in 56% and 55% of patients, respectively, and ACR70 responses in 35% and 36%. Furthermore, among patients who achieved an ACR20, ACR50, or ACR70 response at week 52, a high proportion

maintained it through week 100 [9]. These data indicate that guselkumab improves symptoms of peripheral arthritis and allows for the maintenance of clinical response during long-term follow-up.

Risankizumab also demonstrated significant efficacy in improving symptoms of peripheral arthritis among patients with psoriatic arthritis. In the KEEPsAKE-1 study, which included patients with inadequate response, intolerance, or contraindications to csDMARD treatment, 57.3% of patients treated with risankizumab achieved an ACR20 response at week 24, compared with 33.5% of patients receiving placebo. The difference in favor of active treatment was already evident at week 4 and persisted through week 24. Higher ACR20 response rates were also observed across various patient subgroups, regardless of age, sex, BMI, disease duration, presence of enthesitis or dactylitis, or concomitant csDMARD use. A similar pattern was observed with more stringent response criteria: a higher proportion of patients treated with risankizumab achieved ACR50 and ACR70 than those receiving placebo [10]. In the KEEPsAKE-2 study, which included a more difficult-to-treat patient population with an inadequate response to biologic therapy and/or csDMARDs, an ACR20 response at week 24 was achieved in 51.3% of patients treated with risankizumab and in 26.5% of patients in the placebo group. In this study, the superiority of risankizumab was also evident early on, after a single dose at week 4, and at week 16, an ACR20 response was achieved by 48.3% of patients in the active treatment group versus 25.3% in the placebo group. Favorable results were also observed for ACR50 and ACR70, which were achieved at week 24 in 26.3% of patients versus 9.3% and 12.0% versus 5.9%, respectively. Importantly, higher ACR20 response rates were observed in patients receiving risankizumab monotherapy as well as those receiving concomitant csDMARDs, and in the csDMARD-IR and Bio-IR subgroups [11]. Data from long-term follow-up indicate that the response to risankizumab was sustained. In the 52-week analysis of the KEEPsAKE-2 study, the percentage of patients achieving ACR20 increased from 51.3% at week 24 to 58.5% at week 52, while the ACR50 and ACR70 response rates increased from 26.3% to 32.1% and from 12.0% to 16.5%, respectively. In patients with a prior inadequate response to biologic therapy, efficacy was also maintained through week 52 [14]. A pooled analysis of the KEEPsAKE-1 and KEEPsAKE-2 studies demonstrated the durability of the effect over an even longer follow-up period: at week 100, 64.3% of patients receiving continuous risankizumab met the ACR20 criteria. Among patients who achieved an ACR20 response at week 24, 89.1% maintained it through week 52, and 80.3% through week 100. Similar response durability was also observed for ACR50 and ACR70 [15]. These results indicate that risankizumab not only provides significant improvement in joint symptoms but also maintains long-term clinical benefit.

Evidence regarding the efficacy of tildrakizumab in peripheral arthritis is more limited since it comes from a single Phase IIb trial. However, the results indicate a beneficial effect of this drug on joint response in patients with psoriatic arthritis. At week 24, a significantly higher proportion of patients receiving any dose of tildrakizumab achieved an ACR20 response compared with those receiving placebo. Response rates ranged from 71.4% to 79.5%, compared with 50.6% in the placebo group. The most pronounced effect was observed with the 200 mg dose, administered every 4 or 12 weeks. For this dose, higher ACR50 and ACR70 response rates were also reported compared with placebo. The 100 mg dose administered every 12 weeks was also associated with higher response rates, except for ACR70. In contrast, the 20 mg dose every 12 weeks was associated with a higher ACR50 response rate than placebo. Extended follow-up demonstrated sustained clinical response through week 52. Furthermore, patients who switched from placebo or the 20 mg dose to tildrakizumab 200 mg every 12 weeks at week 24 achieved a response comparable to patients who were actively treated from the start of the study. Response rates at week 24 were numerically lower in patients previously treated with anti-TNFα therapy than in those without such exposure. However, the overall pattern of ACR20, ACR50, and ACR70 responses remained similar regardless of prior anti-TNFα therapy [12]. These results suggest that tildrakizumab may improve symptoms of peripheral arthritis. However, the available data are significantly more limited than those for guselkumab and risankizumab.

The efficacy of IL-23 inhibitors in treating peripheral arthritis has been further confirmed in meta-analyses and network meta-analyses. In a meta-analysis of 6 randomized trials, treatment with IL-23 inhibitors was associated with a significantly higher likelihood of achieving an ACR20 response compared with placebo (RR=1.74), as well as ACR50 (RR=2.49) and ACR70 (RR=2.89) responses [16]. Similar results were obtained in a more recent systematic review with a meta-analysis, in which the pooled relative risks for ACR20, ACR50, and ACR70 were 1.86, 2.75, and 3.06, respectively, with no significant heterogeneity observed in most analyses [17]. The network meta-analyses confirmed that IL-23 inhibitors are more effective than placebo for ACR20, ACR50, and ACR70 responses. In most indirect comparisons, the efficacy of this class of drugs was comparable to that of other biologic therapeutic options used in psoriatic arthritis [18]. No significant differences were observed between IL-23 inhibitors and TNF or IL-17 inhibitors in some analyses, nor was

there a clear advantage of combination therapy with methotrexate over biologic monotherapy [19]. However, it should be noted that in some indirect comparisons, selected drugs, particularly bimekizumab, demonstrated greater efficacy than guselkumab or risankizumab for ACR20, ACR50, and ACR70 responses [20]. Therefore, data from network meta-analyses suggest that IL-23 inhibitors are an effective treatment option for peripheral arthritis. However, their position relative to other biologic classes remains uncertain.

In summary, the available evidence demonstrates that IL-23 inhibitors provide clinically significant improvement in peripheral arthritis in psoriatic arthritis. The strongest evidence supports the efficacy of guselkumab and risankizumab, whereas the data on tildrakizumab remain limited.

Skin and Multidomain Outcomes

In randomized trials, the efficacy of IL-23 inhibitors was assessed not only in peripheral arthritis but also in other clinically relevant domains, including skin lesions, physical function, minimal disease activity, enthesitis, and dactylitis.

Guselkumab demonstrated a beneficial effect not only on joint symptoms but also on other clinically relevant domains of psoriatic arthritis. In the COSMOS study, at week 24, patients treated with guselkumab achieved a significantly greater improvement in physical functioning as assessed by the HAQ-DI than those receiving placebo (mean LS change: −0.18 vs −0.01), and were also more likely to achieve a PASI 100 response (30.8% vs 3.8%), PASI 90 (51.1% vs 7.5%), and PASI 75 (59.4% vs 9.4%). The proportion of patients achieving minimal disease activity was also higher (14.8% vs 3.1%), as was very low disease activity, which was achieved by 3.7% of patients treated with guselkumab, while no such cases were reported in the placebo group. A beneficial effect of treatment was also observed in terms of resolution of enthesitis and dactylitis, with resolution at week 24 in 39.7% of patients versus 18.8% and 44.8% versus 25.0%, respectively [7]. A similar multi-domain efficacy profile was observed in the APEX study. At week 24, a higher proportion of patients treated with guselkumab every 4 and every 8 weeks, compared with placebo, achieved PASI 90 (69.4% and 60.0%, respectively, versus 22.0%) and PASI 100 (37.6% and 39.5%, respectively, versus 11.6%). Improvements were also observed in physical functioning, as the change in mean LS on the HAQ-DI was greater in both active treatment groups than in the placebo group (Q4W: −0.41; Q8W: −0.42 vs. −0.27). At the same time, a higher proportion of patients achieved minimal disease activity (29.6% and 27.7% in the guselkumab groups vs. 13.7% in the placebo group). For dactylitis, greater improvement in the Dactylitis Severity Score (DSS) was observed in the guselkumab-treated groups, whereas for enthesitis, the trend in changes also favored active treatment, though without statistical significance [8]. The long-term efficacy of guselkumab across multiple disease domains was confirmed by the publication of the DISCOVER-2 study. At week 100, complete resolution of enthesitis was achieved by 62% of patients receiving guselkumab every 4 weeks and by 70% of patients receiving it every 8 weeks, while complete resolution of dactylitis was observed in 72% and 83% of patients, respectively. Sustained improvement was also observed in physical function, as 63–64% of patients achieved clinically meaningful improvement in HAQ-DI, and 35–40% achieved normalization of physical function (HAQ-DI ≤0.5) by week 100. At the same time, 82–83% of patients achieved PASI 75, 70–74% achieved PASI 90, and 53–59% achieved PASI 100. Minimal disease activity at week 100 was achieved by 38% (Q4W) and 40% (Q8W) of patients, and very low disease activity by 14% and 17%, respectively [9]. An additional post hoc analysis of the COSMOS study confirmed that the benefits of guselkumab treatment, including PASI 90, PASI 100, HAQ-DI, MDA, enthesitis, and dactylitis, were observed regardless of baseline patient characteristics and persisted or increased through week 48 [13]. A post hoc analysis of the COSMOS study, focusing on the impact of resolution of dactylitis and enthesitis on disease control, showed that patients treated with guselkumab who experienced resolution of dactylitis or enthesitis at week 24 were more likely ($P < 0.05$) to achieve disease control at week 48, as assessed using stringent joint criteria, than patients in whom dactylitis or enthesitis had not resolved [21]. These results indicate that guselkumab exerts a broad and sustained therapeutic effect, encompassing both skin symptoms and physical function, as well as other extra-articular manifestations of psoriatic arthritis.

Risankizumab also demonstrated a beneficial effect on several clinically relevant domains of psoriatic arthritis. In the KEEPsAKE-1 study, patients treated with risankizumab were more likely than those in the placebo group to achieve resolution of enthesitis (51.2% vs. 37.2%) and dactylitis (66.9% vs. 54.4%) at week 24. At the same time, among patients with skin involvement ≥3% BSA, a higher proportion achieved PASI 90 (52.3% vs. 9.9%) and showed improvement as early as week 8. Treatment with risankizumab was also associated with improved physical function, as evidenced by a greater reduction in HAQ-DI scores from baseline ($p<0.001$), and a higher rate of achieving a minimally clinically significant improvement in the HAQ-DI (≥0.35 from baseline) compared with placebo (50.3% vs 27.9%). A higher proportion of patients treated

with risankizumab also achieved minimal disease activity (25.0% vs 10.2%) [10]. A similar efficacy profile was observed in the KEEPsAKE-2 study, which included a more challenging patient population with an inadequate response to biologic therapy and/or csDMARDs. At week 24, resolution of enthesitis was observed in 42.9% of patients treated with risankizumab, compared with 30.4% in the placebo group, and resolution of dactylitis was observed in 72.5% and 42.1%, respectively. Compared with the placebo group, more patients achieved PASI 90 (55.0% vs 10.2%), experienced greater HAQ-DI improvement (−0.22 vs −0.05), achieved clinically meaningful improvement in HAQ-DI more frequently (39.9% vs 23.6%), and attained minimal disease activity more often (25.6% vs 11.4%) [11]. Data from long-term follow-up indicate that this effect was sustained. In the 52-week analysis of the KEEPsAKE-2 study, the proportion of patients with resolution of enthesitis remained stable (43.5%), and resolution of dactylitis persisted in 67.5% of patients. At the same time, the percentage of patients achieving PASI 90 increased (from 55.0% to 64.2%), as did the percentages of patients with clinically meaningful improvements in HAQ-DI (from 39.9% to 42.9%) and MDA (from 25.6% to 27.2%) [14]. The 100-week results from the KEEPsAKE-1 and KEEPsAKE-2 studies and their pooled analyses confirmed the durability of response across various disease domains. At week 100 in the KEEPsAKE-1 study, resolution of enthesitis was observed in 57.7% of patients treated continuously with risankizumab, and resolution of dactylitis in 76.2% of patients. A PASI 90 response was achieved by 71.3% of patients, a clinically meaningful improvement in HAQ-DI was achieved by 54.9% of patients, and minimal disease activity was achieved by 38.2% of patients [22]. In the KEEPsAKE-2 study, after 100 weeks, resolution of enthesitis was observed in 51.7% of patients treated continuously with risankizumab, and resolution of dactylitis in 77.5% of patients. PASI 90 was achieved by 67.5% of patients, clinically meaningful improvement in HAQ-DI by 39.8%, and minimal disease activity by 33.0% [23]. A pooled analysis of the KEEPsAKE-1 and KEEPsAKE-2 studies further confirmed the durability of the response, both in terms of resolution of enthesitis and dactylitis, as well as skin response assessed by PASI 90 [15]. These results indicate that risankizumab exerts a broad and sustained therapeutic effect, encompassing skin lesions, physical function, minimal disease activity, and the resolution of enthesitis and dactylitis.

For tildrakizumab, the available data on other domains of psoriatic arthritis are more limited and less conclusive than those for guselkumab and risankizumab. In a Phase IIb study, improvement in physical function, as assessed by the HAQ-DI, was observed in patients receiving tildrakizumab 200 mg every 12 weeks, whereas no similar effect was observed with the every-4-week dosing regimen. At week 24, no significant improvement in Leeds Enthesitis Index (LEI) or Leeds Dactylitis Index (LDI) scores was observed with any tildrakizumab dose compared with placebo, and treatment did not increase the overall rate of enthesitis and dactylitis remission. At the same time, in exploratory and post hoc analyses, a higher proportion of patients with measurable skin psoriasis at baseline achieved PASI 75, PASI 90, and PASI 100 responses after treatment with tildrakizumab than after placebo, and this effect persisted through week 52. The proportion of patients achieving very low disease activity was also numerically higher with the 200 mg dose administered every 4 or 12 weeks. Furthermore, by week 52, at least half of patients with baseline LEI ≥1 achieved enthesitis remission [12]. These results suggest that tildrakizumab may have a beneficial effect on selected skin and functional domains. However, the available evidence base remains limited, and its multidomain effect is less well documented than with other IL-23 inhibitors.

The results of randomized trials examining the effect of IL-23 inhibitors on skin lesions and other domains of psoriatic arthritis have been further confirmed in meta-analyses and network meta-analyses. In a meta-analysis based on randomized controlled trials, treatment with IL-23 inhibitors was associated with a significantly higher likelihood of achieving a PASI 90 response compared with placebo (RR = 6.11), as well as with more frequent achievement of minimal disease activity (RR = 3.19), resolution of enthesitis (RR = 1.50), and resolution of dactylitis (RR = 1.40) [16]. Similar results were obtained in a more recent systematic review with a meta-analysis, which demonstrated a significant advantage of IL-23 inhibitors over placebo for PASI 90 (RR = 5.98), MDA (RR = 2.85), enthesitis remission (RR = 1.46), and resolution of dactylitis (RR = 1.39) [17]. These data confirm that IL-23 inhibitors have a beneficial effect not only on the joint response but also on other clinically important disease domains. Furthermore, findings from network meta-analyses indicate that the efficacy of IL-23 inhibitors for skin and multi-domain responses is at least comparable to many other biologic options, and in some analyses, guselkumab achieved some of the highest PASI 90 results [18]. At the same time, some indirect comparisons suggested greater efficacy of selected IL-17 inhibitors in achieving MDA than risankizumab [20]. Overall, the pooled data support the conclusion that IL-23 inhibitors represent an effective therapeutic option for the treatment of cutaneous and extra-articular manifestations of psoriatic arthritis, with the strongest clinical evidence currently supporting guselkumab and risankizumab.

Structural Progression

Structural progression in the included studies was assessed radiographically using modified Sharp-based scoring methods, reported as either the PsA-modified van der Heijde-Sharp score (vdH-S) or the PsA-modified Total Sharp Score (PsA-mTSS). The strongest evidence for the effect of IL-23 inhibitors on radiographic progression comes from the APEX study evaluating guselkumab. At week 24, patients treated with guselkumab showed significantly less radiographic progression than those receiving placebo, and the LS mean change in the total vdH-S score was 0.55 in the Q4W group and 0.54 in the Q8W group, compared with 1.35 in the placebo group. Treatment was associated with less progression of both erosions (LS mean change: Q4W 0.35 and Q8W 0.32 vs. placebo 0.87) and joint space narrowing (Q4W 0.22 and Q8W 0.24 vs. placebo 0.50). At the same time, a higher proportion of patients treated with guselkumab showed no progression of structural joint damage at week 24, defined as a change in the total vdH-S score of ≤0 or ≤0.5, compared with patients receiving placebo [8]. Long-term data from the DISCOVER-2 study confirmed the sustained low rate of radiographic progression during guselkumab treatment through week 100. Low rates of radiographic progression were observed with both guselkumab dosing regimens from week 0 to week 100, and radiographic progression was reduced in patients initially receiving placebo after switching to guselkumab [9]. Data from a network meta-analysis further suggested a favorable position for guselkumab in inhibiting structural progression, particularly with the Q4W regimen, although the advantage over most other therapies was not clear-cut [18].

For risankizumab, short-term results from the KEEPsAKE-1 study showed no difference in the change in PsA-mTSS compared with placebo [10]. However, the 100-week follow-up showed minimal radiographic progression from baseline, with a high proportion of patients without radiographic progression in both the continuous risankizumab group (90.6%) and the placebo/risankizumab group (86.9%) at week 100 [22]. Overall, the available data suggest that IL-23 inhibitors, particularly guselkumab, may help limit structural progression in psoriatic arthritis.

Safety Profile

In randomized trials, the safety profile of IL-23 inhibitors was primarily assessed based on the incidence of adverse events, serious adverse events, and treatment discontinuation due to adverse events.

In the COSMOS study, by week 24, 42% of patients treated with guselkumab and 48% of those receiving placebo reported at least one adverse event. The most common events were nasopharyngitis (5%) and upper respiratory tract infection (4%), with similar frequency in both groups. By week 56, among 279 guselkumab-treated patients, the adverse event rate was 144.9 per 100 patient-years (PY), compared with 369.8 per 100 PY in the placebo group. Infections remained the most frequent adverse events, occurring at 37.2 per 100 PY in the guselkumab group and 99.6 per 100 PY in the placebo group. Rates of serious adverse events and events leading to treatment discontinuation were 6.3 and 2.7 per 100 PY, respectively. Two serious infections were reported during the study, both of which resolved following antibiotic treatment. No opportunistic infections, active tuberculosis, or deaths occurred. Injection-site reactions, all mild, appeared in 1.8% of guselkumab patients. No anaphylactic or serum sickness-like reactions were reported. There were isolated cases of serious cardiovascular events, malignant neoplasms, psychiatric-related events, and hematologic abnormalities (decreased neutrophil and white blood cell counts). Most ALT and AST elevations were at most Grade 1 according to the NCI-CTCAE, and two cases of elevated ALT were classified as serious adverse events [7]. In the APEX study, by week 24, at least one adverse event had occurred in 38.2% of participants in the Q4W group, 42.5% in the Q8W group, and 37.3% in the placebo group. The most commonly reported types of adverse events were infections, occurring in 18.6%, 23.5%, and 21.0% of participants, respectively, with upper respiratory tract infections and COVID-19 being the most commonly observed. Serious adverse events occurred in 1.8% of participants in the Q4W group, 3.1% in the Q8W group, and 2.6% in the placebo group. Serious infections were rare and mainly included COVID-19, pneumonia, appendicitis, and diverticulitis. One COVID-19-related death was reported in an unvaccinated elderly patient in the Q4W group. No opportunistic infections or active tuberculosis were identified in the study, and no new cases of inflammatory bowel disease were observed. Isolated cases of major adverse cardiac events (MACE) and malignancies, including prostate cancer and kidney cancer, were reported. Injection-site reactions, which were mild in all cases, occurred in 2.9% of patients in the Q4W group and 0.5% in the Q8W group. No anaphylaxis or serum sickness-like reactions were reported. Elevations in ALT and AST were generally mild, insufficient to meet the criteria for serious adverse events, and did not result in discontinuation of the study drug [8]. Long-term data from the DISCOVER-2 study showed that, through week 112, infections remained the most commonly reported adverse event type, primarily upper respiratory tract infections (8.5%) and nasopharyngitis (7.5%). A total of 21

guselkumab-treated patients reported a serious infection, with infection and serious infection rates of 37.3 and 1.9 per 100 PY, respectively, compared with 50.5 and 0.9 per 100 PY in the placebo group. Opportunistic infections occurred in three patients after week 52 and included fungal esophagitis, disseminated herpes zoster, and Listeria-induced meningitis. No active tuberculosis was observed. During the study, two malignancies, three serious cardiovascular events, and a single case of iridocyclitis were reported. No cases of inflammatory bowel disease were observed in patients treated with guselkumab. Injection-site reactions occurred in 2.7% of patients and were mostly mild. Decreases in neutrophil count and elevations in ALT and AST were most often transient and usually did not require discontinuation of treatment [9]. An integrated analysis of the long-term safety of guselkumab in psoriasis and psoriatic arthritis studies confirmed a low, stable rate of adverse events during follow-up. During the placebo-controlled period, the incidence of all adverse events was similar in the guselkumab and placebo groups (281 vs. 272 per 100 PY), and the incidence of events leading to treatment discontinuation was low during both the placebo-controlled period and long-term exposure. Infections were the most commonly reported adverse events, while the incidence of serious infections, malignancies, MACE, uveitis-related events, and hypersensitivity reactions remained low. By the end of the observation period, no cases of active tuberculosis, anaphylaxis, or serum sickness-like reactions had been reported [24].

In the KEEPsAKE-1 study, treatment-emergent adverse events were reported with similar frequency in the risankizumab and placebo groups (40.4% and 38.7%, respectively), and most were mild or moderate in severity. The incidence of serious adverse events was comparable in both groups, and adverse events leading to study discontinuation were rare (0.8% in both groups). The most commonly reported adverse events included nasopharyngitis, upper respiratory tract infection, elevated ALT and AST levels, and headaches, with similar frequencies in both groups. Injection-site reactions occurred more frequently in patients receiving risankizumab, but none were serious, and no anaphylactic reactions were reported. Serious infections were reported in five patients treated with risankizumab and six patients in the placebo group. A few cases of herpes zoster were observed, all mild and resolving after treatment. No active tuberculosis or other opportunistic infections were observed. No malignancies were reported in the risankizumab group, whereas isolated cases of breast cancer and non-small cell lung cancer were reported in the placebo group. One death occurred in an 81-year-old patient with dementia treated with risankizumab, who developed pneumonia followed by urinary sepsis. Grade 3 elevations in transaminases occurred in less than 2% of patients and were transient in patients receiving risankizumab, without concomitant increases in bilirubin [10]. In the KEEPsAKE-2 study, treatment-emergent adverse events were reported in 55.4% of patients treated with risankizumab and in 54.8% of patients receiving placebo, with most events being mild or moderate in severity**.** The most commonly reported adverse event was upper respiratory tract infection (7.6% vs. 5.5%). The incidence of serious and severe adverse events was similar in both groups, while adverse events leading to treatment discontinuation occurred less frequently in the risankizumab group than in the placebo group (0.9% vs 2.3%). No deaths were reported during the 24-week double-blind period. Serious infections occurred in 0.9% of patients receiving risankizumab and in 2.3% of patients in the placebo group. No active tuberculosis or other opportunistic infections were observed, and one case of herpes zoster was reported exclusively in the placebo group. One case of uveitis was reported in a patient treated with risankizumab, and one non-fatal stroke was reported in a patient with a history of hypertension. Injection-site reactions were more common in the risankizumab group, but none were serious or led to discontinuation of treatment. No Grade 3 elevations in transaminases were observed in either group [11]. Long-term results from the KEEPsAKE-1 and KEEPsAKE-2 studies confirmed that risankizumab maintained a stable safety profile through week 100. In the KEEPsAKE-1 analysis, which included 1,708.4 PY of exposure, the incidence of all treatment-emergent adverse events was 130.1 per 100 PY, and the incidence of events leading to treatment discontinuation was 2.1 per 100 PY. Serious adverse events occurred at a rate of 7.6 per 100 PY, and serious infections at a rate of 2.3 per 100 PY; a significant proportion of these were COVID-19 infections or COVID-19-related pneumonia. No active tuberculosis or new opportunistic infections were reported between weeks 52 and 100. Nine malignancies and two serious cardiovascular events were reported, along with 43 cases of hypersensitivity; none of the hypersensitivity reactions were severe [22]. In the KEEPsAKE-2 analysis, covering 810.1 PY of exposure, the incidence of all adverse events was 180.5 per 100 PY, and that of serious adverse events was 9.9 per 100 PY. Serious infections occurred at a rate of 1.6 per 100 PY, opportunistic infections at 0.2 per 100 PY, and events leading to treatment discontinuation at 1.2 per 100 PY. By week 100, four confirmed serious cardiovascular events and 15 malignancies were reported. No new grade ≥3 elevations in ALT, AST, or bilirubin were observed between weeks 52 and 100 [23].

Safety data for tildrakizumab are more limited and come from a single Phase IIb trial. No deaths were reported during the 52-week study, and of the 391 patients analyzed, only one (0.3%) discontinued the study

due to a treatment-emergent adverse event. Overall, treatment-emergent adverse events occurred in 64.5% of patients, with nasopharyngitis (8.4%) and upper respiratory tract infection (6.4%) being the most commonly reported. Most events were mild in nature and comparable across treatment groups. Serious treatment-emergent adverse events were observed in 3.3% of patients. During the first 24 weeks, one serious infection (chronic tonsillitis) was reported, and, in addition, pyelonephritis and a urinary tract infection were recorded in one patient as adverse events of special interest. Fungal skin infections caused by *Candida* occurred in 0.5% of patients. One malignancy was diagnosed during weeks 25–52. However, no cases of systemic candidiasis, uveitis, inflammatory bowel disease, MACE, suicidal ideation, or clinically significant laboratory abnormalities classified as serious adverse events were observed. These results suggest that tildrakizumab was generally well tolerated, although the available data remain more limited than for other IL-23 inhibitors [12].

A systematic review and meta-analysis that includes both short- and long-term studies provides additional context for assessing the safety of IL-23 inhibitors. The short-term analysis showed no increased risk of serious infections or malignancies with IL-23 inhibitors, either in the overall population or in subgroups for individual drugs. No increased risk of nasopharyngitis, upper respiratory tract infections, or herpes zoster was observed. No cases of tuberculosis were reported. In the long-term analysis, incidence rates for serious infections, overall infections, non-melanoma skin cancers (NMSC), malignancies excluding NMSC, nasopharyngitis, and upper respiratory tract infections remained low and did not show an upward trend with increasing duration of exposure. The overall EAIR for tuberculosis was estimated at 0/100 PY. Cases of viral hepatitis and herpes zoster occurred sporadically. Although some of the data in this analysis came from studies in patients with psoriasis, the results are consistent with observations from clinical trials in psoriatic arthritis and support the conclusion that IL-23 inhibitors have a stable and predictable safety profile, with no clear new risk signals observed during long-term follow-up [25].

Discussion

Available data indicate that IL-23 inhibitors are an effective therapeutic option for the treatment of psoriatic arthritis, particularly for peripheral arthritis and certain other aspects of the disease. The most mature and consistent clinical data currently pertain to guselkumab and risankizumab, for which improvements in joint response, skin lesions, physical function, minimal disease activity, and resolution of enthesitis and dactylitis have been demonstrated. These findings are consistent with meta-analyses and network meta-analyses, which confirm the superiority of IL-23 inhibitors over placebo and indicate that the efficacy of this class of drugs is at least comparable to that of many other biologic options used in PsA [16–18,20].

Among the drugs analyzed, guselkumab appears to have the strongest evidence base. Its efficacy has been demonstrated in both biologic-naive patients and those with an inadequate response to TNF inhibitors, and it also has the most compelling data regarding the inhibition of structural progression. In contrast, risankizumab demonstrates a consistent efficacy profile, including in patients with a more complex treatment history. However, data regarding its impact on radiographic progression remain less conclusive than those for guselkumab.

The evidence base for tildrakizumab remains considerably limited. Published Phase IIb results indicate a beneficial effect on joint symptoms and selected skin and functional domains. However, the available data are not as comprehensive as those for guselkumab and risankizumab [12]. The Phase III INSPIRE-1 and INSPIRE-2 trials are central to the ongoing evaluation of tildrakizumab, yet no full peer-reviewed publications currently report detailed results from these trials, which restricts broader interpretation [26,27]. Additional data suggest that tildrakizumab may enhance the number and function of regulatory T cells in older adults and potentially inhibit psoriatic arthritis progression by modulating disease-progression factors. While these findings do not constitute direct evidence of tildrakizumab's clinical efficacy in PsA, they support further investigation into its potential therapeutic role [28].

The safety profile of IL-23 inhibitors in the studies analyzed was generally favorable and consistent with current knowledge about this class of drugs. Mild or moderate infections were most commonly observed, primarily upper respiratory tract infections and nasopharyngitis, whereas serious infections, malignancies, MACE, and adverse events leading to treatment discontinuation were rare. Importantly, the available long-term data do not suggest an increasing risk with longer exposure. These findings are consistent with the results of safety meta-analyses, which did not show an increased risk of severe infections or malignancies and confirmed the stable safety profile of IL-23 inhibitors in long-term follow-up [17,24,25].

When interpreting the results of this review, several limitations should be considered. The available studies differed in terms of patient populations, prior treatment exposure, and the domains assessed, among

other factors, making direct comparisons between individual drugs difficult. Some of the data also came from post hoc analyses, pooled analyses, and long-term publications that extended beyond the original double-blind period. Additionally, the lack of head-to-head studies prevents a definitive assessment of the position of IL-23 inhibitors relative to other therapeutic classes. Despite these limitations, the available data indicate that IL-23 inhibitors, particularly guselkumab and risankizumab, represent a well-established treatment option for PsA, especially when the goal of therapy is to improve not only joint symptoms but also other clinically relevant disease domains.

Conclusions

The available evidence supports IL-23 inhibitors as an effective therapeutic strategy in psoriatic arthritis, providing clinically meaningful improvement in peripheral arthritis and other important disease domains, including skin lesions, physical function, minimal disease activity, enthesitis, and dactylitis. Their safety profile appears generally favorable and stable, with no clear indication of emerging long-term safety concerns. At present, the strongest evidence supports guselkumab and risankizumab, whereas the evidence base for tildrakizumab remains more limited. Overall, IL-23 inhibitors represent an important treatment option in psoriatic arthritis, particularly in patients requiring multidomain disease control.

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