

Targeting IL-23 in psoriasis: current perspectives

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Abstract: The recent advances in the understanding of psoriasis pathogenesis have clarified the pivotal role of interleukin (IL)-23. It is a heterodimeric cytokine consisting of two subunits, the unique p19 and the p40, which are shared with IL-12. The basic role of IL-23 in psoriasis is the activation and maintenance of the T-helper 17 pathway. New research findings indicate that IL-23 is more important than IL-12 in the pathogenesis of psoriasis. Based on that background, the selective targeting of the IL-23p19 subunit emerged as an attractive therapeutic option and led to the development of a new category of biologic agents. Three monoclonal antibodies that selectively inhibit the IL-23p19 subunit, guselkumab, tildrakizumab, and risankizumab, are in the pipeline for the treatment of moderate-to-severe psoriasis. In this article, we review the most recent efficacy and safety data regarding these IL-23p19 inhibitors.

Keywords: psoriasis, IL-23, treatment, Th17 axis, anti-IL-23p19 monoclonal antibodies

Introduction

Psoriasis is a chronic inflammatory, immune-mediated skin disorder that is characterized by a complex pathophysiology. The synergistic influence of genetic and environmental factors along with the interplay of innate and adaptive immunity eventually leads to the abnormal keratinocyte proliferation and formation of the psoriatic lesions.¹ The recent advances in the understanding of this mechanism have further enlightened the important role of a specific cytokine, interleukin (IL)-23 (IL-23).² This cytokine has become the therapeutic target of a new category of biologic drugs for psoriasis. In this article, we reviewed the updated information regarding the role of IL-23 in psoriasis and the most recent efficacy and safety data on the emerging IL-23 inhibitors.

IL-23 and its receptors: structure and function

IL-23 is a heterodimeric cytokine comprising a unique p19 subunit linked with a p40 subunit which is shared with IL-12.^{3,4} The main sources of IL-23 are tissue-resident or recruited dendritic cells and macrophages.⁴⁻⁶ The biologic action of IL-23 is achieved through a receptor complex which is composed of the following two parts: (1) IL-12R β 1, a part common with IL-12, and (2) IL-23R, a part specific for IL-23.^{6,7} The receptor of IL-23 is expressed by a great range of cells (natural killer cells, macrophages, dendritic cells, memory T cells, keratinocytes).^{6,8,9} Naïve T-helper cells also express the IL-23R receptor in the presence of transforming growth factor beta (TGF- β) and IL-6.^{6,8} The IL-23 signal transmission requires the phosphorylation of Signal Transducer and Activator of Transcription (STAT) 1–4, but especially STAT-3, which

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allows the activation of the T-helper 17 (Th17) inflammatory pathway in several autoimmune diseases.^{3,5,10}

IL-23 and psoriasis pathogenesis

Early after its identification in the year 2000, IL-23 was recognized as a crucial player in the pathogenesis of chronic autoimmune diseases in general and of psoriasis in particular.⁶ Until then, the proinflammatory role of the shared IL-12/23p40 subunit was attributed chiefly to IL-12. IL-12 was considered “responsible” for the development of the T-helper 1 (Th1) cell lineage and the production of characteristic Th1 cytokines such as interferon gamma (IFN- γ) and IL-2, while IL-23 activated the Th17 lineage and its archetypic cytokines IL-17 and IL-22.⁴ However, several researchers reported that the levels of p40 and p19 mRNA were profoundly increased in the psoriatic lesional skin compared with the non-lesional skin, but this was not the case for the p35 mRNA.^{2,11,12} These findings indicate that, probably, IL-23 is the cytokine with the most important role in the inflammation process of psoriasis.⁹ Also, large-scale genetic studies have associated only the genetic loci of IL-23p19 and IL-12/23p40, and not the IL-12p35, with the presence of psoriasis.^{13,14} In addition, intradermal injections of IL-23 in murine skin models have provoked clinically and histologically confirmed psoriatic lesions, in contrast with IL-12 which did not induce such lesions.^{15,16} Moreover, in certain studies, clinical improvement of psoriasis patients under conventional and/or biologic therapies was followed with subsequent decrease of the IL-23 levels in these patients.^{17,18}

The basic role of IL-23 in the pathogenesis of psoriasis has been clarified, and it is associated with the biology of the Th17 lineage. The initial differentiation of naïve T lymphocytes to Th17 requires the presence of TGF- β , IL-6, and IL-1 β , while IL-23 is necessary for the activation and maintenance of Th17 in order to secrete the pro-inflammatory cytokines IL-17, IL-22, IL-21, and tumor necrosis factor alpha, which eventually contribute to the formation of the psoriatic plaques.^{3,19}

Therapeutic rationale of the IL-23 inhibitors

The first approved biologic agent targeting IL-23 was ustekinumab (Janssen Biotech, Inc., 2009), a fully human monoclonal antibody against the shared IL-12/23p40 subunit. This agent, based on data from clinical trials and everyday practice, proved to be safe and efficacious in the treatment of patients suffering from moderate-to-severe plaque psoriasis.^{20–22} Briakinumab (Abbott Laboratories, Abbott

Park, IL, USA), another fully human monoclonal antibody against the shared IL-12/23p40 subunit, was withdrawn before approval, despite its excellent efficacy results in the treatment of psoriasis, due to a safety issue concerning a possibly increased risk for major cardiovascular events.^{9,23,24}

The rationale of targeting IL-23 selectively was based partly on the recently upgraded role of the IL-23p19 subunit in psoriasis pathogenesis and partly on an effort to increase safety by preserving the IL-12-mediated Th1 response against human pathogens, while having analogous efficacy results as by inhibiting the IL-12/23p40.^{9,25} Indeed, studies in mouse models have confirmed these hypotheses and have shown that the selective inhibition of IL-23p19 has great efficacy results in the treatment of psoriasis.^{15,26} Moreover, other animal studies found that the neutralization of IL-23p19 did not influence the host defense against mycobacteria, provided that remaining cytokines such as IL-12p70 and IFN- γ were intact.²⁷

Based on that background, three monoclonal antibodies that selectively inhibit the IL-23p19 subunit have been developed, although not yet officially approved, for the treatment of plaque psoriasis.^{25,28} For guselkumab and tildrakizumab each, results have been reported from two ongoing Phase III trials, while for risankizumab no results from Phase III trials have been reported yet.

Guselkumab

Guselkumab (Janssen Biotech, Inc.) is a fully human monoclonal antibody against the IL-23p19 subunit of IL-23.²⁹ Several Phase III trials are ongoing, and preliminary results have been reported for VOYAGE 1, VOYAGE 2, and NAVI-GATE trials.^{30–32}

The VOYAGE 1 was a 48-week randomized, double-blind Phase III trial which included 870 patients suffering from moderate-to-severe psoriasis who were randomly allocated to one of the three treatment arms: guselkumab 100 mg at weeks 0 and 4 followed by every 8-week dosing thereafter; adalimumab 80 mg at week 0 and 40 mg at week 1, followed by every 2-week dosing; and placebo at weeks 0, 4, and 12, and crossed over to receive guselkumab at weeks 16 and 20, and every 8 weeks thereafter.³⁰ The primary end points were the percentage of patients under guselkumab who reached Psoriasis Area and Severity Index (PASI) 90 (ie, 90% reduction from the baseline PASI score) and Physician Global Assessment (PGA) 0 (clear) or 1 (minimal) at week 16 compared with placebo. At week 16, a greater proportion of patients receiving guselkumab achieved PASI90 and PGA 0–1 compared with both patients receiving placebo

and adalimumab. Specifically, PASI90 was exhibited by 73.3% of guselkumab-treated vs 2.9% of placebo-treated vs 49.7% adalimumab-treated patients ($P<0.001$), while PGA 0–1 was achieved by 85.1% of guselkumab-treated vs 6.9% of placebo-treated vs 65.9% of adalimumab-treated patients ($P<0.001$).³⁰ At week 48, patients in the guselkumab arm, compared with those in the adalimumab arm, were significantly more likely to achieve PASI90 (76.3% vs 47.9%, $P<0.001$).³⁰ As far as safety issues were concerned, at week 16 the rates of at least one adverse event between the three treatment groups were comparable (49.4% guselkumab, 51.1% placebo, 51.7% adalimumab; $P<0.001$).³⁰ The percentage of serious adverse events (SAEs) between guselkumab and adalimumab was comparable through week 48 (4.9% guselkumab vs 4.85% adalimumab).³⁰

The VOYAGE 2, a double-blind Phase III trial, included 992 patients with moderate-to-severe plaque psoriasis who were randomized to receive either guselkumab 100 mg at weeks 0, 4, 12, and 20, or placebo at weeks 0, 4, and 12, and then guselkumab at weeks 16 and 20 or adalimumab 80 mg at week 0, 40 mg at week 1, and 40 mg every 2 weeks through week 23.³¹ At week 28, patients under guselkumab who achieved PASI90 were re-randomized to receive placebo or guselkumab. Patients under guselkumab who did not respond adequately continued with the drug, while guselkumab responders, initially on placebo, switched back to placebo at week 28 but were retreated with guselkumab when they lost 50% of their 28-week PASI response.³¹ At week 28, adalimumab nonresponders received guselkumab, while adalimumab responders were switched to placebo and they were also administered guselkumab if they lost 50% of their PASI response at week 28.³¹ At week 16, significantly more patients on guselkumab reached PASI90 compared with the placebo and adalimumab group (70% guselkumab vs 2.4% placebo, $P<0.001$). At week 24, significantly more patients on guselkumab achieved PASI90 and PASI100 compared with adalimumab arm (PASI90: 75.2% guselkumab vs 54.8% adalimumab, $P<0.001$; PASI100: 44.2% guselkumab vs 26.6% adalimumab, $P<0.001$). At week 48, the percentage of patients with PASI90 was significantly higher in the maintenance group (ie, the guselkumab responders who remained on the drug after week 28) compared with the withdrawal group (ie, the patients who were re-randomized to receive placebo at week 28).³¹ Specifically, 88.6% of patients from the maintenance group achieved PASI90 at week 48 compared with 36.8% of patients from the withdrawal group.³¹ Adalimumab nonresponders who switched to guselkumab showed an increase in PASI90 (66.1%) and PASI100 (26.8%) at week 48.³¹ At week 16, the rates of at least one adverse event were comparable

between all treatment groups and placebo. In the active comparator group, two myocardial infarctions (one guselkumab, one adalimumab), one prostate cancer (guselkumab), and two non-melanoma skin cancers (one guselkumab, one placebo to guselkumab) were observed.³¹

The NAVIGATE was a randomized, double-blind study which evaluated the efficacy and safety of guselkumab in patients with moderate-to-severe psoriasis who responded inadequately to ustekinumab.³² In total, 871 psoriasis patients received open-label ustekinumab (45 or 90 mg) at weeks 0 and 4. At week 16, 261 patients who did not respond adequately to ustekinumab were randomized (double blind) to either receive guselkumab 100 mg or continue ustekinumab, while those who responded adequately to ustekinumab (585) continued with the drug. The primary end point was the number of visits at which randomized patients achieved an Investigator Global Assessment (IGA) 0/1 and a >2-grade improvement from week 16.³² The mean number of visits at which patients had an IGA 0/1 and ≥ 2 -grade improvement (weeks 28–40) was significantly greater in the guselkumab group vs the randomized ustekinumab group (1.5 vs 0.7, $P<0.001$).³³ At week 52, PASI90 was reached by 51% of guselkumab-treated patients vs 24.1% of randomized ustekinumab-treated patients ($P<0.001$), while this difference for PASI100 was 20.0% vs 7.5% ($P=0.003$). The percentage of patients who exhibited at least one adverse event after week 16 was comparable between treatment groups (64.4% vs 55.6%), while the percentage of patients showing at least one SAE was 6.7% in the guselkumab arm and 4.5% in the ustekinumab arm.³²

Tildrakizumab

Tildrakizumab (MK-3222) is a humanized monoclonal antibody inhibiting selectively the p19 subunit of IL-23. Early data from Phase II trials have shown a considerable efficacy in the treatment of moderate-to-severe psoriasis.³³ The efficacy and safety results from two randomized double-blind Phase III trials (reSURFACE 1 and reSURFACE 2) were published recently.³⁴

The reSURFACE 1 included 1772 patients with moderate-to-severe psoriasis who were randomized to three treatment groups to receive either tildrakizumab 100 or 200 mg or placebo at weeks 0, 4, and 16. At week 12, the placebo patients were crossed over to receive either 100 or 200 mg tildrakizumab at weeks 12 and 16. Primary end points were the percentage of patients in each group who achieved PASI75 and PGA 0/1 with >2 reduction from baseline at 12 weeks. At 12 weeks, a significantly greater percentage of

patients in both tildrakizumab groups achieved PASI75 and PGA 0/1 compared with placebo (PASI75: 100 mg 64%, 200 mg 62%, placebo 6%; $P<0.001$ and PGA 0/1: 100 mg 59%, 200 mg 58%, placebo 7%; $P<0.001$). The rate of at least one adverse event was comparable among treatment groups (100 mg 42.2%, 200 mg 47.2%, placebo 48.1%; $P<0.001$) at 12 weeks.³³ The SAEs were not common but were slightly higher in the 200 mg treatment group (100 mg 1.6%, 200 mg 2.6%, placebo 0.6%).³³

The reSURFACE 2 included 1090 patients with moderate-to-severe psoriasis who were randomized to one of the following treatment groups: placebo, tildrakizumab 100 mg or 200 mg at weeks 0, 4, and 16, or etanercept 50 mg twice a week for 12 weeks followed by once-weekly dose until week 28.³⁴ At week 12, PASI75 was reached by a significantly greater percentage of patients in the tildrakizumab groups compared with placebo and etanercept groups (100 mg 61%, 200 mg 66%, etanercept 48%, placebo 6%).³⁴ This was also the case for the PGA 0/1 at week 12 (100 mg 55%, 200 mg 59%, placebo 4%; $P<0.001$) and at week 28 (100 mg 66%, 200 mg 71%, etanercept 48%; $P<0.001$).³⁴ The percentage of patients with at least one adverse event in each treatment group at week 12 was as follows: 100 mg 44.3%, 200 mg 49.4%, placebo 55.1%, etanercept 54.0%.³⁴ One death occurred in a patient in the 100 mg group with alcoholic cardiomyopathy and hepatic steatosis, and the cause of death was undetermined.³⁴

Risankizumab

Risankizumab (BI655066) is a humanized monoclonal antibody targeting selectively the p19 subunit of IL-23. There are some encouraging data regarding the efficacy of risankizumab in psoriasis from one Phase I and one Phase II study that have been recently announced.^{35,36} However, several Phase III trials are still ongoing.^{35,36} Risankizumab is under evaluation, at the moment, for several other inflammatory diseases such as Crohn's disease and ankylosing spondylitis.³⁷

In the Phase II randomized double-blind trial, 166 patients were randomly assigned to receive either one of the following schemes: (1) a single subcutaneous dose of risankizumab 180 mg, (2) 90 mg or 180 mg of risankizumab at weeks 0, 4, and 16, or (3) ustekinumab (45 or 90 mg) at weeks 0, 4, and 16.³⁶ Primary end point results at week 12 showed PASI90 in 32.6%, 73.2%, 81.0%, and 40% of the above treatment groups, respectively.³⁶ A very interesting finding of this study is that the group of patients who were treated with risankizumab 180 mg achieved PASI90 score 8 weeks earlier compared with the ustekinumab group, and the result lasted

2 months longer. The rate of adverse events was comparable among all treatment groups, and no SAE was related to the study medications.³⁶

Conclusion

The recently acknowledged the critical role of IL-23 in the pathogenesis of psoriasis was the basis for the development of a new category of biologic agents for the treatment of the disease, namely the selective inhibitors of the p19 subunit of IL-23. Preliminary data from Phase II and III studies show great promise regarding their efficacy and short-term safety. In that light, their possible official approval for the treatment of moderate-to-severe psoriasis will enrich the therapeutic armamentarium of the disease and will give patients even more treatment options. However, larger, long-term studies and evidence from everyday practice are needed in order to confirm these assumptions.

Disclosure

The authors report no conflicts of interest in this work.

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