Review Article

Immune-Inflammatory Axis in Psoriasis: From TNF-α to IL-23

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Abstract

Psoriasis is a chronic inflammatory disease with an immunomediated basis, characterized by a complex interaction between the innate and adaptive immune systems. Its pathophysiology is driven by dendritic cells, Th17 lymphocytes, and keratinocytes, which form a fundamental pathogenic axis mediated by the cytokines TNF-α, IL-23, and IL-17. This immuno-inflammatory axis not only initiates but also perpetuates cutaneous inflammation, generating a feedback loop that leads to epidermal hyperplasia and the clinical expression of the disease. The involvement of γδ T cells, innate lymphoid cells, and neutrophils, along with epidermal barrier dysfunction, further contributes to the complexity of the condition. The relevance of this axis has been confirmed by experimental and clinical evidence through murine models, immunohistochemical studies, transcriptomic analyses, and serum cytokine measurements, all of which correlate with disease activity. This understanding has enabled the development of targeted biological therapies that selectively block TNF-α, IL-23, or IL-17 and have demonstrated clinical efficacy in psoriasis and psoriatic arthritis. Despite these advances, some patients do not achieve complete remission, prompting the search for new therapeutic targets. In

this context, personalized medicine, supported by the development of biomarkers and multi-omic technologies, represents a promising strategy. Finally, therapeutic repositioning and the investigation of new molecules such as IL-36 inhibitors or JAK-STAT pathway blockers open innovative perspectives for the comprehensive and personalized management of psoriasis and its comorbidities.

Key words

Psoriasis, Tumor necrosis factor alpha, Interleukin-23, Interleukin-17, Biological therapy, Precision medicine.

Introduction

Psoriasis is a chronic multisystem inflammatory disease characterized by cutaneous plaques and, in some cases, arthritis. It affects approximately 2% of the global population and is associated with important comorbidities such as cardiovascular disease, metabolic syndrome, and psychological disorders [1, 2]. The clinical and epidemiological relevance of psoriasis is underscored not only by its prevalence but also by the substantial burden it places on patients' quality of life [3]. In this context, the immunological approach to understanding psoriasis is justified by the central role of immune dysregulation in its pathogenesis, particularly regarding the cytokine pathways of tumor necrosis factor alpha (TNF-α), interleukin-23 (IL-23), and interleukin-17 (IL-17) [4, 5].

From a pathogenic perspective, the immunoinflammatory axis represents the core of psoriasis pathophysiology. TNF-α, IL-23, and IL-17 play key roles in initiating and sustaining the inflammatory process, leading to chronic immune activation and abnormal keratinocyte proliferation [3, 4]. This inflammatory response is driven by a complex interaction among various cell types, where dendritic cells, T helper 17 (Th17) lymphocytes, and keratinocytes form a pathogenic triad. Within this circuit, activated dendritic cells produce TNF-α and IL-23, promoting the differentiation and expansion of Th17 cells which, in turn, release IL-17 and other pro-inflammatory cytokines that stimulate keratinocytes and perpetuate inflammation [3].

This immune network is modulated by a combination of genetic and environmental factors that contribute to the chronicity and recurrence of the inflammatory process. Genetic predisposition influences individual susceptibility, while environmental triggers such as infections, stress, or skin trauma act as precipitating or exacerbating factors [4].

In recent decades, advances in understanding these immunological pathways have had a major impact on the therapeutic management of psoriasis. Biological therapies, especially monoclonal antibodies targeting TNF-α, IL-23, and IL-17, have transformed clinical care by offering more specific suppression of inflammation along with improved efficacy and safety profiles compared with conventional treatments [3, 4]. Within this same framework, the JAK-STAT signaling pathway has gained interest due to its essential role in cytokine-mediated immune responses. Selective inhibitors such as deucravacitinib have shown promising results by interfering with key components of the IL-23/IL-17 axis, providing new therapeutic opportunities [4].

Beyond the treatment of cutaneous lesions, it is essential to consider the systemic impact of psoriasis. The strong association with cardiovascular and metabolic diseases highlights the need for comprehensive therapeutic strategies that address both cutaneous inflammation and associated comorbidities [1]. This holistic perspective is reinforced by advances in precision medicine, which enable therapeutic decisions to be tailored to the patient's immunologic profile. The accumulated

understanding of the immunoinflammatory axis has paved the way for individualized approaches capable of optimizing clinical outcomes and improving quality of life [1, 3].

The objective of this review is to provide an integrated analysis of the immunoinflammatory axis formed by TNF-α, IL-23, and IL-17 in the pathophysiology of psoriasis, emphasizing its relevance as a molecular foundation for the development of targeted biological therapies.

Methodology

For this investigation on the immuno-inflammatory axis in psoriasis, a literature review was conducted to analyze the role of the cytokines TNF-α, IL-23, and IL-17 in the pathophysiology of the disease and their therapeutic relevance. The search was performed in well-established scientific databases, including PubMed, Scopus, and Web of Science, chosen for their broad coverage of dermatology, clinical immunology, and evidence-based medicine.

Articles published between 2020 and 2025 in English or Spanish were included, provided they addressed clinical, molecular, or therapeutic aspects related to these cytokines in the context of psoriasis. Publications that were duplicated, not peer-reviewed, or incomplete were excluded. The keywords used were: Psoriasis, Tumor necrosis factor alpha, Interleukin-23, Interleukin-17, Biological therapy, Precision medicine.

A total of 32 relevant sources were selected, including original studies, systematic reviews, and clinical guidelines. The information was organized into thematic categories covering immunological mechanisms, targeted therapeutic interventions, and clinical applications. To ensure the formal and methodological quality of the manuscript, the criteria of the SANRA Scale (Scale for the Assessment of Narrative Review Articles) were followed, which allowed the evaluation and optimization of key aspects such as justification of the topic, bibliographic coverage, logical structure, and critical interpretation of the literature. Artificial intelligence tools were used exclusively to support text editing and linguistic refinement, without contributing to scientific content, data interpretation, or the generation of original ideas.

General Pathophysiology of Psoriasis

Psoriasis involves a complex pathological network of interactions among multiple components of the innate and adaptive immune systems. In this context, dendritic cells, Th17 cells, and keratinocytes stand out as a central pathogenic triad in the disease's pathophysiology. Activated dendritic cells secrete cytokines such as TNF-α and IL-23, which induce the differentiation of Th17 cells. These cells, in turn, produce interleukin-17, interferon gamma (IFN-γ), and interleukin-22 (IL-22), contributing to chronic skin inflammation [5].

In addition to the role of adaptive immune cells, components of the innate immune system also actively participate in the pathogenesis of psoriasis. These include innate lymphoid cells and γδ T cells, both of which contribute to the inflammatory process through mechanisms that are either dependent or independent of IL-17 [6].

Moreover, dysfunction of the epidermal barrier represents an essential component of this pathological process. Activated keratinocytes release antimicrobial peptides and inflammatory cytokines, compromising the integrity of the epidermal barrier. This alteration increases exposure to immunological stimuli and facilitates sustained immune activation, thereby perpetuating the characteristic inflammatory cycle of psoriasis [2].

Within this immunological framework, the activation of dendritic cells, T lymphocytes, and keratinocytes assumes a central role. Dendritic cells are crucial for initiating the immune response by activating T cells, particularly Th17 cells, whose expansion and cytokine production are key to the development and maintenance of

psoriatic lesions [5]. Keratinocytes, for their part, are not merely passive targets of immune attack but actively participate in amplifying the inflammatory response by secreting mediators that stimulate other immune cells [2].

Beyond immunological and cellular factors, several external and systemic elements have been identified as relevant modulators in the physiopathology of psoriasis. Notably, alterations in the gut microbiome - similar to profiles observed in patients with diabetes - have been linked to systemic inflammatory mechanisms that may influence the skin manifestations of the disease [2]. Likewise, environmental factors such as trauma, clinically described through the Koebner phenomenon, and psychological stress have been recognized as triggers or exacerbating factors of psoriasis due to their ability to activate various immune pathways involved in chronic inflammation [7].

The Immune-Inflammatory Axis in Psoriasis

In the immunopathological context of psoriasis, several cell types play key roles in the initiation, maintenance, and amplification of the inflammatory process. Dendritic cells, both myeloid and plasmacytoid, constitute a fundamental component in the early phases of the disease. These cells produce cytokines such as TNF-α, IL-12, and IL-23, which are essential for the activation and differentiation of T lymphocytes, particularly toward the Th17 profile, thereby promoting a sustained inflammatory response [5, 8].

Among the T-cell subpopulations, Th17 cells stand out for their relevance in the physiopathogenesis of psoriasis. Their differentiation is driven by IL-23, and their activation leads to the production of IL-17 and IL-22, cytokines that induce keratinocyte proliferation and amplify cutaneous inflammation [9, 10]. In parallel, Th1 cells also contribute to the inflammatory microenvironment through the secretion of interferon gamma (IFN-γ), further reinforcing immune activation [5].

Keratinocytes, traditionally considered passive targets of the immune response, are now recognized as active participants in the inflammatory process. They respond to IL-17 and IL-22 with exaggerated proliferation and the production of additional inflammatory mediators, generating a feedback loop that perpetuates epidermal inflammation [11, 12].

In severe forms of psoriasis and pustular variants, neutrophils also play an important role. These cells release neutrophil extracellular traps, structures that intensify the inflammatory process by activating keratinocytes and other immune cells, thereby contributing to tissue damage and clinical exacerbation [13, 14].

The involvement of these key cell types is mediated by a cytokine cascade that orchestrates the immunoinflammatory response. TNF-α functions as both an initiating and amplifying cytokine, promoting the production of additional pro-inflammatory molecules as well as the activation and survival of various immune cells [5, 8]. In parallel, IL-12 and IL-23 regulate T-cell differentiation: IL-12 drives the response toward a Th1 phenotype, whereas IL-23 is essential for the expansion and maintenance of Th17 cells, which are central to the development and persistence of psoriatic lesions [8, 9].

Once differentiated, Th17 cells release IL-17, an effector cytokine that acts directly on keratinocytes to induce the production of chemokines, antimicrobial peptides, and other cytokines, thereby perpetuating the inflammatory cycle and driving the epidermal hyperplasia characteristic of the disease [10, 15]. Additionally, IL-22 secreted by these cells promotes keratinocyte proliferation and alters their differentiation, contributing to the skin thickening that clinically defines psoriasis [5, 9].

It is important to emphasize that these cytokines do not act in isolation; instead, they exhibit

synergistic interactions that enhance their biological effects. For example, the combination of IL-17 and TNF-α has been shown to exert a significantly stronger effect on keratinocytes than either cytokine alone, thereby reinforcing epidermal inflammation and worsening the clinical expression of the disease [11, 12].

Experimental and Clinical Evidence of the Role of TNF-α, IL-17 and IL-23

Several experimental strategies have contributed to elucidating the role of the cytokines TNF-α, IL-17, and IL-23 in the pathophysiology of psoriasis, including animal models, histological and immunohistochemical analyses, and molecular and clinical studies. Murine models, particularly those involving gene inactivation or overexpression, have been essential for clarifying the specific functions of these cytokines in the context of cutaneous inflammation. These experiments have demonstrated that the absence or increased expression of TNF-α, IL-17, or IL-23 can significantly alter the course of the disease, highlighting their essential participation in the pathogenesis of psoriasis [9, 16].

These experimental findings have been corroborated by evidence obtained from human samples. Biopsies of psoriatic lesions reveal increased expression of TNF-α, IL-17, and IL-23, an observation that correlates directly with clinical severity. Immunohistochemical studies have confirmed the presence of these cytokines in affected skin tissue, supporting their central role in the inflammatory cascade characteristic of psoriasis [5, 17].

Complementary transcriptomic and proteomic studies have enabled a deeper characterization of the molecular mechanisms involved. Comprehensive analyses of psoriatic skin have identified upregulation of both genes and proteins associated with TNF-α, IL-17, and IL-23 pathways. These findings have not only expanded our understanding of the underlying biology of the disease but also facilitated the identification of potential biomarkers useful for assessing pathological activity [5, 18].

Finally, serum levels of these cytokines have also shown a significant correlation with clinical activity in both cutaneous psoriasis and psoriatic arthritis. Elevated levels of TNF-α, IL-17, and IL-23 in the blood have been associated with greater disease severity, reinforcing their utility as biomarkers. Moreover, these same molecules have become key therapeutic targets for biological treatments, which have demonstrated efficacy in reducing symptoms and improving clinical outcomes [16, 17].

Targeted Therapies; From Biology to the Clinic

The development of biological therapies has transformed the clinical management of psoriasis and psoriatic arthritis by enabling targeted intervention on specific immunological pathways. Among the most established options are TNF-α inhibitors, such as etanercept, infliximab, and adalimumab, which are primarily used in patients with moderate to severe disease. These drugs have demonstrated efficacy in reducing inflammatory activity and significantly improving patients' quality of life [19]. However, despite their benefits, a considerable proportion of patients do not achieve complete remission, and resistance may develop over time, diminishing therapeutic efficacy [20]. Additionally, associated adverse effects such as an increased risk of infections and the formation of neutralizing antibodies against the drugs constitute important limitations of this therapeutic group [19].

In the search for more specific alternatives, IL-12/23 inhibitors emerged, with ustekinumab as the leading representative. This biologic targets the p40 subunit shared by both interleukins, offering a dual mechanism that has demonstrated efficacy in the treatment of psoriasis and psoriatic arthritis. Moreover, it presents a more favorable safety profile compared with TNF-α inhibitors [21, 22]. Nevertheless, its effectiveness

is not uniform across all patients, particularly those with high initial disease activity (Smolen, et al. 2021). Long-term studies have shown persistence comparable to that observed with anti-TNF agents but with a lower incidence of adverse events [23].

With an even more selective approach, IL-23 inhibitors that specifically target the p19 subunit - such as guselkumab, risankizumab, and tildrakizumab have emerged as an advanced therapeutic alternative. These agents offer significant advantages over IL-12/23 inhibitors by focusing on a more specific pathogenic target, resulting in higher drug survival rates and a lower incidence of adverse effects [21, 24]. Their efficacy has been particularly notable in patients who have failed prior biologic therapies, maintaining high persistence and sustained clinical response in real-world practice settings [24, 25].

IL-17 inhibitors, including secukinumab, ixekizumab, and brodalumab, are characterized by a rapid onset of action, allowing early symptomatic relief in patients with active psoriasis [21]. Despite their efficacy, they present specific limitations, such as the potential exacerbation of inflammatory bowel diseases, which necessitates careful patient selection and monitoring [26]. In the context of refractory disease, particularly in cases unresponsive to ustekinumab, secukinumab has demonstrated both clinical and molecular superiority over guselkumab. This finding suggests that, in certain patients, the IL-17–mediated pathway may operate independently of IL-23, reinforcing the importance of therapeutic selection based on the individual immunological profile [27].

Clinical Implications and New Perspectives

Advances in the understanding of immunological mechanisms have driven the development of more personalized treatment strategies for immune-mediated inflammatory diseases (IMIDs), including psoriasis. In this context, the ImmuniVerse Consortium has actively promoted personalized medicine through the identification of novel biomarkers using a multi-omic approach. This strategy is crucial for patient stratification and prediction of treatment response not only in psoriasis but also in related conditions such as ulcerative colitis and atopic dermatitis. The biomarkers under development aim to provide clinicians with valuable information regarding disease severity and progression, thereby facilitating clinical decision-making and the implementation of treatment plans tailored to the individual characteristics of each patient [28].

In parallel with biomarker development, therapeutic combinations and drug repurposing strategies are being explored to address psoriasis and its associated comorbidities. Guselkumab, a monoclonal antibody directed against the IL-23 p19 subunit, has shown sustained improvements in pain and disease activity in patients with psoriatic arthritis. These findings suggest potential utility within combined therapeutic regimens aimed at controlling the systemic manifestations of the disease [29, 30]. Additionally, agents such as JAK inhibitors and SOCS mimetics are being investigated due to their ability to modulate immune responses in various autoimmune diseases, including psoriasis and uveitis. This approach raises the possibility of repurposing these molecules for the management of related comorbidities [31].

Within the spectrum of emerging therapies, a group of IL-36–targeted drugs is under development, with particular application in generalized pustular psoriasis. These investigations focus on the role of cytokines in the pathogenesis of this specific clinical subtype, opening new avenues for the treatment of patients with severe or refractory disease [32]. Complementarily, JAK inhibitors continue to be evaluated for their therapeutic potential in autoimmune disorders, given their direct involvement in the regulation of immune responses. Ongoing studies aim to more precisely

determine their safety and efficacy profiles across different patient populations [31].

Conclusion

Psoriasis results from a complex interaction between innate and adaptive immune components, in which dendritic cells, Th17 lymphocytes, and keratinocytes form a key pathogenic axis mediated by the cytokines TNF-α, IL-23, and IL-17. The sustained activation of this axis perpetuates the cutaneous inflammatory process characteristic of the disease.

Experimental and clinical evidence supports the central role of the TNF-α/IL-23/IL-17 axis in the pathophysiology of psoriasis, as demonstrated by animal models, molecular studies, and serum biomarkers. This knowledge has enabled the development of targeted biological therapies that have transformed disease management, although challenges remain regarding incomplete treatment responses and the need for individualized therapeutic selection.

Current approaches are increasingly oriented toward precision medicine based on biomarkers, selective therapies, and personalized combinations. Advances in omics technologies and the exploration of new therapeutic targets, such as IL-36 and the JAK/STAT pathways, open new perspectives for the comprehensive management of psoriasis and its comorbidities.

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