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Role of Il-17 and Novel Monoclonal Antibody Treatment in Psoriasis

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Abstract

Psoriasis is a chronic skin condition that affects about 2-3% of the global population, characterized by red, scaly plaques. Its development involves a mix of genetic factors, environmental triggers, and immune system dysregulation, particularly mediated through interleukin-17 (IL-17). IL-17, primarily produced by T helper 17 (Th17) cells, is crucial for initiating and maintaining skin inflammation. Over the years, treatments for psoriasis have evolved from traditional options to targeted biologics. Monoclonal antibodies, such as Secukinumab, Ixekizumab, and Brodalumab, have emerged as powerful IL-17 inhibitors, effectively blocking IL-17 signaling and addressing the root causes of the disease. This review explores IL-17's role in psoriasis, the mechanisms behind IL-17 inhibitors, and the clinical evidence that supports their effectiveness and safety. Furthermore, how these inhibitors offer advantages over conventional therapies, significantly reducing skin lesions and improving patients' quality of life while lowering the risks associated with broad immunosuppression, is emphasized in the review. In the future, optimizing IL-17 targeted therapies through combination treatments and personalized medicine will be vital. By tailoring approaches to individual needs and incorporating genetic and environmental insights, we can enhance treatment effectiveness and patient satisfaction. Overall, ongoing research into IL-17 targeted therapies holds promise for achieving sustained control of psoriasis and significantly improving patient outcomes in the long run.

1. Introduction

Psoriasis is a common chronic skin condition characterized by red, scaly plaques affecting approximately 2-3% of the global population. Its development involves genetic predisposition, environmental factors, and immune system dysregulation, notably interleukin-17 (IL-17). IL-17, primarily produced by T helper 17 (Th17) cells, plays a crucial role in initiating and perpetuating skin inflammation through various biological pathways.

Over time, treatment strategies for psoriasis have progressed from traditional therapies to targeted biologics, driven by the need to specifically address immune dysregulation. Monoclonal antibodies that target IL-17, such as Secukinumab, Ixekizumab, and Brodalumab, have emerged as highly effective treatments by directly blocking IL-17 signaling pathways. IL-17 inhibitors represent a groundbreaking advancement in the treatment of psoriasis, offering targeted therapy that specifically addresses the underlying immune mechanisms of the disease. Understanding their therapeutic potential underscores ongoing efforts in precision medicine to effectively manage psoriasis and enhance the quality of life for affected individuals. This review paper delves into IL-17's role in the pathophysiology of psoriasis, explores the mechanisms of IL-17 inhibitors, and evaluates the clinical evidence supporting their efficacy and safety. It also discusses current challenges in psoriasis management, proposes strategies to optimize IL-17 targeted therapies, and discusses future research directions aimed at improving treatment outcomes.

2. IL-17 in Psoriasis and Autoimmune Diseases

Interleukin-17 (IL-17) is a pro-inflammatory cytokine that plays a crucial role in the immune system, particularly in mediating immune

responses and inflammation. It is predominantly produced by a subset of T helper cells known as Th17 cells, as well as other immune cells such as γδ T cells, natural killer cells, and neutrophils [1]. IL-17 has several isoforms, with IL-17A being the most studied and best understood. Biologically, IL-17 acts by binding to its receptor complex, IL-17R, which is expressed on a variety of cell types, including epithelial cells, endothelial cells, and fibroblasts [2]. This binding triggers the activation of several signaling pathways, notably the NF-κB, MAPK, and C/EBP pathways, leading to the production of other pro-inflammatory cytokines, chemokines, and metalloproteinases [3]. These molecules collectively contribute to the recruitment and activation of neutrophils and other immune cells to sites of infection or tissue damage, thereby amplifying the inflammatory response. In the context of autoimmune diseases such as psoriasis, IL-17 is particularly significant.

Psoriasis is characterized by hyperproliferation of keratinocytes and chronic skin inflammation, where IL-17 contributes to the pathogenesis by promoting the proliferation and activation of keratinocytes, leading to the formation of psoriatic plaques [4]. IL-17 signaling pathways involved in psoriasis pathogenesis include the activation of STAT3, which induces the expression of additional pro-inflammatory cytokines and sustains Th17 cell responses. Thus, the activation of IL-17 signaling and the subsequent chronic inflammation underscores the pivotal role of IL-17 in the development and perpetuation of psoriasis, making it a key target for therapeutic interventions aimed at mitigating autoimmune and inflammatory diseases [5].

3. Pathogenesis of Psoriasis

The pathogenesis of psoriasis is a complex process involving intricate interactions between immune cells and keratinocytes, leading to chronic skin inflammation and characteristic epidermal hyperproliferation [6]. Central to this dysregulation are T cells, particularly Th17 cells, dendritic cells, and keratinocytes, which interact in a pathogenic feedback loop [7]. Dendritic cells, upon activation by environmental triggers or genetic factors, produce cytokines, such as IL-23, which are crucial for the differentiation and maintenance of Th17 cells. Th17 cells, in turn, secrete high levels of IL-17 which plays a pivotal role in psoriasis pathophysiology. The IL-23/IL-17 axis is thus central to the disease, with IL-23 stabilizing and promoting Th17 cells, which produce IL-17A and IL-17F. These cytokines act on keratinocytes, inducing them to proliferate abnormally and produce antimicrobial peptides and additional pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6. This inflammatory cascade not only leads to the thickened, scaly plaques characteristic of psoriasis but also recruits other immune cells to the skin, perpetuating inflammation [8]. The contribution of IL-17 to epidermal hyperproliferation involves the direct stimulation of keratinocyte proliferation and the inhibition of their further differentiation, which disrupts the normal skin barrier function. Furthermore, IL-17 amplifies the inflammatory milieu by enhancing the expression of chemokines that attract neutrophils and other inflammatory cells to the skin, thereby sustaining the inflammatory cascade [9]. These interactions between immune cells and keratinocytes create a vicious cycle of inflammation and skin cell turnover, highlighting the critical role of IL-17 in both initiating and perpetuating psoriasis lesions. The understanding of IL-17 signaling in the context of psoriasis pathogenesis has led to the development of targeted therapies that aim to interrupt the IL-23/IL-17 axis, offering a new avenue for effective management of psoriasis [10].

4. Current Treatment Landscape of Psoriasis

Topical agents are pivotal in the management of psoriasis, especially for patients presenting with mild to moderate forms of the disease. These therapies are directly applied to the affected skin areas, targeting localized plaques to mitigate inflammation, reduce hyperproliferation of keratinocytes, and alleviate symptoms, including itching and scaling [11]. Corticosteroids, the most commonly prescribed topical agents, exert their effects by binding to glucocorticoid receptors, leading to the suppression of pro-inflammatory gene expression and reduction of cytokine production [12]. They are available in various potencies and formulations, tailored to different body areas and severity levels, to maximize efficacy while minimizing potential side effects like skin atrophy [13]. Vitamin D analogs, such as calcipotriene and calcitriol, work by modulating the immune response and inhibiting keratinocyte proliferation [14]. These agents bind to vitamin D receptors, influencing gene transcription to normalize skin cell growth and differentiation [15, 16]. Calcineurin inhibitors, including tacrolimus and pimecrolimus, inhibit the activity of calcineurin, a key enzyme in the activation of T-cells [17, 18].

By blocking T-cell activation, these agents reduce the inflammatory response, making them particularly useful for sensitive skin areas like the face and intertriginous regions, where corticosteroids might cause thinning of the skin[19]. Coal tar preparations, including crude coal tar and tar distillates, are also long-standing treatments primarily used for mild to moderate psoriasis [20]. They work by slowing down the rapid growth of skin cells and have anti-inflammatory properties, though their exact mechanism of action is not fully understood. These agents can reduce scaling, itching, and inflammation [21]. Similarly, salicylic acid, often used in conjunction with other treatments, acts as a keratolytic agent by promoting the shedding of the outer layer of the skin and softening plaques, which helps other medications penetrate more effectively [22, 23]. It is typically used for mild to moderate psoriasis.

Transitioning to another class of topical treatments, retinoids like tazarotene and anthralin (dithranol) are also employed in the management of mild to moderate psoriasis [24]. Tazarotene, a vitamin A derivative, works by modulating skin cell growth and reducing inflammation [25]. It normalizes abnormal keratinocyte differentiation and proliferation [26]. On the other hand, anthralin, though less commonly used today due to its potential for skin irritation, inhibits DNA synthesis, which slows down the proliferation of skin cells and reduces plaque formation [27, 28]. Furthermore, emollients and moisturizers, such as petrolatum-based ointments, urea creams, and lactic acid lotions, play a supportive role in psoriasis treatment and are used for all severities of the disease, from mild to severe [29-32]. They help maintain skin hydration, which is crucial for managing psoriasis symptoms and improving the skin barrier function. Petrolatum-based ointments provide a protective barrier on the skin, preventing moisture loss [33]. Lactic acid, an alpha-hydroxy acid, breaks down the bonds between dead skin cells, facilitating their removal (34). Urea, besides being a humectant, also has keratolytic properties, making it effective in softening and removing the thickened skin characteristic of psoriatic plaques (35). These agents, by promoting exfoliation and hydration, help reduce scaling and improve the overall appearance of the skin. The array of topical agents available for psoriasis treatment addresses various aspects of the disease's pathology, from reducing inflammation and normalizing skin cell proliferation to maintaining hydration and facilitating the removal of scales.

Phototherapy, or light therapy, serves as a pivotal treatment modality for psoriasis, particularly for patients with moderate to severe forms of the disease or those resistant to topical treatments [34]. This therapy involves exposing the skin to ultraviolet (UV) light, which helps to slow down the rapid turnover of skin cells and reduce inflammation [11]. The two primary types of UV light used in phototherapy for psoriasis are ultraviolet B (UVB) and psoralen plus ultraviolet A (PUVA) [35]. Narrowband UVB (NB-UVB) is the most commonly utilized form of phototherapy for psoriasis due to its effectiveness and reduced risk of side effects compared to broad-spectrum UVB [36]. It works by penetrating the skin and slowing down the excessive growth of skin cells, thereby reducing scaling, redness, and thickness of plaques. PUVA therapy combines the ingestion or topical application of a photosensitizing agent called psoralen with UVA exposure [37]. Psoralen makes the skin more sensitive to UVA light, enhancing its therapeutic effects [38]. UVA penetrates deeper into the skin than UVB and is particularly effective for thick plaques and psoriasis affecting the palms, soles, and nails. PUVA therapy is typically reserved for patients who have not responded adequately to other treatments or have widespread diseases, such as HIV or an internal malignancy [39]. Indications for phototherapy in psoriasis include cases where topical treatments alone are insufficient or impractical, such as when lesions are widespread or resistant to conventional therapies [34]. It is also considered for patients who prefer non-systemic treatments or are unable to tolerate systemic medications due to side effects [40]. Phototherapy provides a targeted approach to managing psoriasis by modulating immune responses and reducing the excessive proliferation of keratinocytes [41]. However, like all treatments, it requires careful monitoring to minimize potential side effects, such as skin burning, premature skin aging, and an increased risk of skin cancer with long-term use [42]. Adjustments in treatment parameters and frequency are often necessary to balance therapeutic benefits with safety concerns, ensuring optimal outcomes for patients with psoriasis undergoing phototherapy. Systemic drugs play a critical role in the treatment paradigm for moderate to severe psoriasis, particularly when topical therapies and phototherapy yield inadequate results [43]. These medications target specific immune pathways central to the pathogenesis of psoriasis. Conventional systemic agents like methotrexate

inhibit dihydrofolate reductase, disrupting DNA synthesis and thereby reducing keratinocyte proliferation [44]. Cyclosporine suppresses T-cell activation and cytokine production, pivotal processes in the inflammatory cascade underlying psoriasis [45]. Acitretin, a retinoid, modulates cell differentiation and decreases keratinocyte hyperproliferation, contributing to improved skin appearance [46]. Systemic drugs are indicated for patients with moderate to severe psoriasis who have shown inadequate responses to other therapies or have extensive skin involvement impacting their quality of life. They are also employed in cases of psoriatic arthritis, a condition characterized by joint inflammation concurrent with skin manifestations. Treatment selection hinges on considerations including disease severity, patient preferences, comorbidities, and potential side effects. Vigilant monitoring is essential during treatment to manage risks such as infections, liver toxicity, and other immune-related complications associated with systemic immunosuppression.

Despite their efficacy, conventional treatments for moderate-to-severe psoriasis present several limitations and challenges that necessitate consideration in clinical practice and research. Topical therapies, such as corticosteroids and vitamin D analogs, though effective for localized disease, often struggle to manage widespread or recalcitrant plaques due to limitations in skin penetration and patient adherence to long-term treatment regimens [47, 48]. Phototherapy, while beneficial for broader areas of involvement, requires consistent clinic visits and may pose long-term risks of skin aging and carcinogenesis with extended use [49]. Furthermore, conventional systemic agents like methotrexate and cyclosporine, while effective in suppressing immune responses and reducing inflammation, carry risks of systemic side effects including hepatotoxicity, nephrotoxicity, and increased susceptibility to infections [50]. The cumulative toxicities associated with prolonged use necessitate regular monitoring and careful management by healthcare providers.

Additionally, these treatments may not adequately address the underlying immunopathology of psoriasis, particularly in patients with severe or refractory disease, highlighting the need for more targeted therapies that offer improved efficacy and safety profiles [51]. While conventional treatments remain foundational in psoriasis management, their limitations underscore the ongoing pursuit of novel therapeutic strategies. Addressing these challenges through continued research and clinical innovation is essential to optimize outcomes and enhance the quality of life for patients with moderate-to-severe psoriasis.

Monoclonal antibodies represent a newer class of systemic therapies that offer targeted treatment by blocking specific cytokines or immune pathways implicated in psoriasis. Tumor necrosis factor (TNF) inhibitors such as etanercept, adalimumab, and infliximab neutralize TNF-α, a pro-inflammatory cytokine central to psoriatic inflammation cascade [52]. These inhibitors effectively alleviate symptoms by mitigating inflammation and slowing skin cell turnover [53]. Additionally, IL-23 inhibitors such as guselkumab and risankizumab target IL-23, a cytokine critical for activating and maintaining Th17 cells involved in psoriasis pathogenesis. By inhibiting IL-23, which is essential for Th17 cell differentiation and activation, these agents disrupt the immune signaling cascade that leads to skin inflammation and plaque formation [54]. Unlike conventional therapies like methotrexate or cyclosporine, which broadly suppress the immune system and may cause systemic side effects, monoclonal antibodies target specific molecules involved in psoriatic inflammation. Monoclonal antibodies generally exhibit a better safety profile in terms of long-term use, as they are designed to selectively modulate the specific immune response without suppressing it entirely [55]. This targeted approach not only improves efficacy but also reduces the risk of systemic adverse effects commonly associated with traditional systemic treatments. As a result, immunotherapies have become a preferred choice for managing moderate to severe psoriasis, offering patients improved symptom control, better quality of life, and long-term disease management. These biologic agents have demonstrated substantial clinical efficacy in patients with moderate to severe psoriasis, offering a valuable therapeutic alternative to traditional systemic treatments.

5. Clinical Trials of IL-17 Inhibitors in Psoriasis Treatment

The development of IL-17 inhibitors—Secukinumab, Ixekizumab, and Brodalumab—marks a significant advancement in the treatment of psoriasis. The monoclonal antibodies specifically target IL-17, a pro-inflammatory cytokine implicated in the pathogenesis of psoriasis [56]. By selectively neutralizing IL-17A or its receptor, these therapies suppress key inflammatory pathways involved in psoriasis

without compromising systemic immunity to the same extent as traditional systemic therapies like methotrexate or cyclosporine. Clinical studies have demonstrated the efficacy of IL-17 inhibitors in achieving rapid and sustained improvements in psoriatic skin lesions, as well as in quality-of-life measures for patients [57]. The favorable safety profiles of these treatments, characterized by a lower risk of hepatotoxicity and nephrotoxicity compared to conventional treatments, have positioned IL-17 inhibitors as preferred options, particularly for patients with comorbidities or contraindications to other therapies, such as chronic kidney disease or alcohol use disorder.

The mechanisms of action of Secukinumab, Ixekizumab, and Brodalumab involve blocking IL-17 signaling pathways, thereby reducing cytokine-mediated inflammation and preventing keratinocyte proliferation. Pharmacokinetic studies have elucidated their absorption, distribution, metabolism, and excretion profiles, contributing to our understanding of optimal dosing regimens and therapeutic monitoring strategies [58]. Key clinical trials, such as the pivotal Phase III ERASURE and FIXTURE studies for Secukinumab [59] and the UNCOVER trials for Ixekizumab [60], have provided crucial insights into their efficacy and safety profiles. These trials demonstrated that both Secukinumab and Ixekizumab achieved significantly higher rates of skin clearance and improvement in quality-of-life measures compared to placebo and traditional systemic therapies. For instance, Secukinumab showed superiority over etanercept in clearing skin with moderate to severe psoriasis in the CLEAR trial [61]. Comparative studies have highlighted the favorable efficacy of IL-17 inhibitors versus other biologics targeting different cytokines, with IL-17 inhibitors often showing superior or comparable efficacy in achieving better scores on the Psoriasis Area Severity Index (PASI) and the Dermatology Life Quality Index (DLQI) [62].

Moreover, IL-17 inhibitors generally exhibit a faster onset of action, with many patients achieving significant skin clearance within weeks of starting treatment, contrasting with the slower response times observed with TNF-α inhibitors and IL-12/23 inhibitors [63]. Real-world evidence, including the PROSE study [64], further supports the long-term safety and sustainability of IL-17 inhibitor therapy. Data from large-scale registries and observational studies have consistently shown that IL-17 inhibitors maintain their efficacy over extended treatment periods, with a manageable safety profile comparable to or better than other biologics. The incidence of serious adverse events, including infections and malignancies, appears low and is generally consistent with the safety profiles established in clinical trials. Importantly, the lower risk of hepatotoxicity and nephrotoxicity associated with IL-17 inhibitors compared to traditional systemic therapies has contributed to their favorable risk-benefit profile in clinical practice [65]. These findings underscore IL-17 inhibitors, such as Secukinumab, Ixekizumab, and Brodalumab, as highly effective and well-tolerated options for the long-term management of moderate to severe psoriasis, offering patients sustained disease control and improved quality of life.

6. Challenges and Future Directions

Despite the significant advancements brought by IL-17 inhibitors in psoriasis treatment, several challenges remain. One key issue is the persistence and loss of response to these therapies. Some patients may experience a decrease in efficacy over time, which can be attributed to mechanisms, such as the development of neutralizing antibodies against the biologic, alterations in IL-17 receptor expression, or changes in the underlying disease pathology [66]. Managing loss of response involves strategies, such as dose adjustment, switching to alternative IL-17 inhibitors, or combining therapies to enhance efficacy. Further research is needed to elucidate the precise mechanisms behind diminished response and to develop targeted interventions to maintain long-term efficacy [67]. Additionally, there are unmet needs in psoriasis treatment, particularly concerning patient subpopulations and comorbidities. Many patients with psoriasis also suffer from comorbid conditions, such as metabolic syndrome, cardiovascular disease, or mental health disorders, which can complicate treatment and affect outcomes [68]. Addressing these needs requires a comprehensive approach that includes not only effective psoriasis management but also integrated care for associated health issues. Furthermore, exploring the potential of combination therapies and personalized medicine approaches could offer more tailored and effective treatment strategies.

Combining IL-17 inhibitors with other therapeutic modalities or developing personalized treatment plans based on individual patient profiles and comorbidity considerations

could enhance overall treatment success and patient satisfaction [69]. As research progresses, these strategies will be crucial in addressing the diverse needs of psoriasis patients and optimizing treatment outcomes.

7. Conclusion

Interleukin-17 (IL-17) plays a crucial role in the development of psoriasis, driving chronic inflammation and abnormal growth of skin cells, which are characteristic features of the disease. This cytokine is mainly produced by Th17 cells and other immune cells, triggering a series of inflammatory responses that worsen immune reactions in the skin, leading to typical psoriasis symptoms. Recognizing IL-17's central role has prompted the development of targeted treatments, IL-17 inhibitors, which have significantly changed how psoriasis is managed. IL-17 inhibitors, including Secukinumab, Ixekizumab, and Brodalumab, represent a new approach to treating psoriasis. These biologics work by specifically blocking IL-17 or its receptor, directly interfering with the inflammatory processes of the disease. This results in notable reductions in skin lesions and improvements in the quality of life for patients. Compared to traditional systemic therapies, IL-17 inhibitors offer better effectiveness with a safer profile, reducing the risks associated with broad immunosuppression. Looking forward, the future of psoriasis treatment shows promise with IL-17 targeted therapies. Fine-tuning treatment strategies through combination therapies, personalized treatment plans tailored to individual patient needs, and continuous monitoring of long-term outcomes will be crucial. Combining IL-17 inhibitors with other biologics or systemic agents could potentially enhance treatment effectiveness and durability, meeting the diverse needs of patients with varying disease severities and accompanying conditions. Furthermore, recognizing psoriasis as a systemic inflammatory condition underscores the importance of personalized medicine. By integrating genetic, environmental, and clinical data, personalized treatment plans can optimize outcomes while minimizing adverse effects.

Ongoing research and clinical innovation will be the key to uncovering new insights into psoriasis mechanisms and therapeutic targets, paving the way for innovative therapies that further improve disease management. In conclusion, IL-17 inhibitors have transformed psoriasis treatment by targeting the underlying causes of inflammation and excessive cell growth. With ongoing advancements in IL-17 targeted therapies, the goal is to achieve sustained disease control and improve quality of life for patients, ultimately aiming for transformative outcomes in psoriasis care.

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