SKIN

IN-DEPTH REVIEW

Expanding Therapeutic Applications of Tofacitinib in Immune-Mediated Skin Disorders: A Comprehensive Review

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ABSTRACT

Tofacitinib, a Janus kinase (JAK) inhibitor targeting JAK1 and JAK3, has gained attention for its immunomodulatory effects, particularly in autoimmune and inflammatory conditions. While initially approved for rheumatoid arthritis, its off label uses in dermatology are expanding, with promising results in conditions such as vitiligo, alopecia areata, atopic dermatitis, psoriasis, and plaque psoriasis. By inhibiting the JAK-STAT signaling pathway, tofacitinib reduces cytokine-mediated inflammation and immune cell activation, offering a novel therapeutic option for dermatological disorders where traditional treatments have failed. This review explores the pharmacology of tofacitinib, its current off-label dermatological uses, and future research opportunities. It also addresses challenges related to long-term safety, accessibility, and treatment resistance. Ongoing clinical trials and potential new indications, as well as combination therapies, are discussed, highlighting tofacitinib's evolving role in dermatology.

INTRODUCTION

Tofacitinib is an orally administered selective inhibitor of Janus kinase (JAK) enzymes, particularly JAK1 and JAK3.[1,2] The JAK-STAT signaling pathway is crucial for the mediation of cytokines that regulate immune responses, cell growth, and differentiation. Initially developed and approved for rheumatoid arthritis, tofacitinib has shown efficacy in various inflammatory conditions, including those related to dermatology.[3]

Immune dysregulation plays a significant role in numerous dermatologic conditions including vitiligo, alopecia areata, and atopic dermatitis. Changes to the immunological landscape in vitiligo are characterized by autoimmune destruction of melanocytes from oxidative stress caused by IFN-gamma and TNF-alpha release from cytotoxic T-cells. Alopecia areata results from immune overreactivity to the body's hair follicles leading to an inflammatory environment and loss of hair due to T helper 1 (Th1) cell and cytokine-mediated activity. Additionally, increased levels of T helper 2 (Th2) inflammatory cytokines including IL-4 and IL-13 contribute to the skin barrier dysregulation associated with atopic dermatitis. Overall, these processes are mediated by the overproduction of cytokines through various inflammatory pathways which can result in cellular changes that precipitate disease.[1,2]

Tofacitinib particularly disrupts the JAK-STAT signaling pathway by preventing the phosphorylation of STAT proteins and reducing the production of pro-inflammatory

cytokines like interferon-gamma (IFN-γ) (**Figure 1**).[4] This immunosuppression helps halt disease progression and allows for tissue recovery, such as re-pigmentation in vitiligo or hair regrowth in alopecia areata.[5]

The JAK-STAT pathway mediates cytokines. When a cytokine binds to a receptor on the cell surface, associated JAK enzymes phosphorylate the receptor, triggering the phosphorylation and dimerization of STAT proteins. The STAT dimers translocate to the nucleus and bind DNA, leading to the initiation of gene transcription to allow for cell growth, differentiation, and immune response. Through inhibition of JAK1 and JAK3, tofacitinib disrupts this cytokine-mediated pathway needed for lymphocyte activation and proliferation. This suppresses the immune system and reduces inflammation which has historically shown benefit in treating rheumatoid arthritis.[6] Through similar anti-inflammatory pathways, JAK inhibitors have shown utility in the treatment of multiple skin conditions. We reviewed studies exploring the pharmacology of tofacitinib, its current off-label dermatological uses, and future research opportunities (**Figure 2**). Some of the indications for the use of Tofacitinib in Dermatology, which include:

Alopecia Areata:

Alopecia Areata (AA) is an autoimmune condition where immune dysregulation targets hair follicles, resulting in hair loss.[7] It is categorized into patchy AA, alopecia totalis (complete scalp hair loss), and alopecia universalis (complete loss of hair on the scalp and body). [8] Affecting up to 2% of the global population, AA shows no clear preference for race, ethnicity, or gender.[9,10]

While the pathophysiology of AA is not fully understood, research from mouse models and human studies suggests that a breakdown of immune privilege, activation of interferon-γ (IFN-γ) signaling, and cytotoxic CD8+ T cell pathways are involved. These findings highlight the immune system's complex role in driving the disease.[11]

Recent studies have shown that JAK inhibitors can be effective in treating severe forms of AA, such as alopecia universalis (AU) and alopecia totalis (AT), which are often resistant to traditional therapies like corticosteroids. A selective JAK3 inhibitor, such as tofacitinib, has been particularly promising in these cases. In a study involving six patients with AU/AT, all showed significant hair regrowth after treatment with tofacitinib, with some patients experiencing complete regrowth of scalp and body hair within 12 weeks.[12] This response highlights the drug's potential to reverse the immune attack on hair follicles by interrupting the JAK-STAT signaling pathway, which plays a critical role in the pathogenesis of AA. Additionally, while most patients tolerated the treatment well, mild adverse effects, such as acneiform eruptions, were managed with topical treatments, and the relapse was seen in one patient after discontinuing the drug, indicating the need for ongoing monitoring and potential maintenance therapy.[12]

In contrast, a study using ruxolitinib, (a JAK1/2 inhibitor), showed similar promising results, with three patients experiencing significant scalp hair regrowth over 5 months.[13] However, like tofacitinib, discontinuation of therapy may lead to relapse, indicating the chronic nature of the condition and the need for long-term management strategies.

Further research highlights additional findings related to the use of tofacitinib in AA. A cross-sectional study among dermatologists in Saudi Arabia reported effectiveness for 72.4% of those prescribing

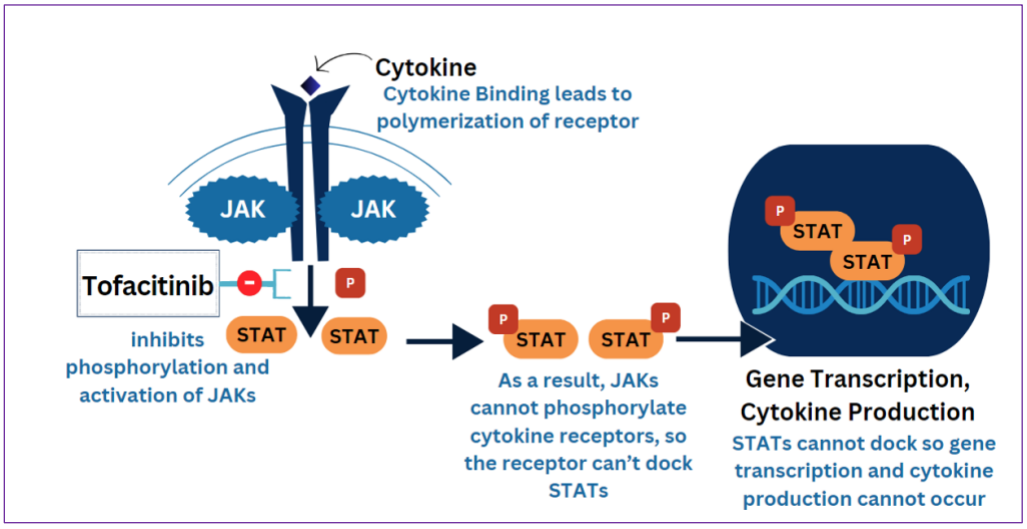

Figure 1. Tofacitinib Mechanism of Action

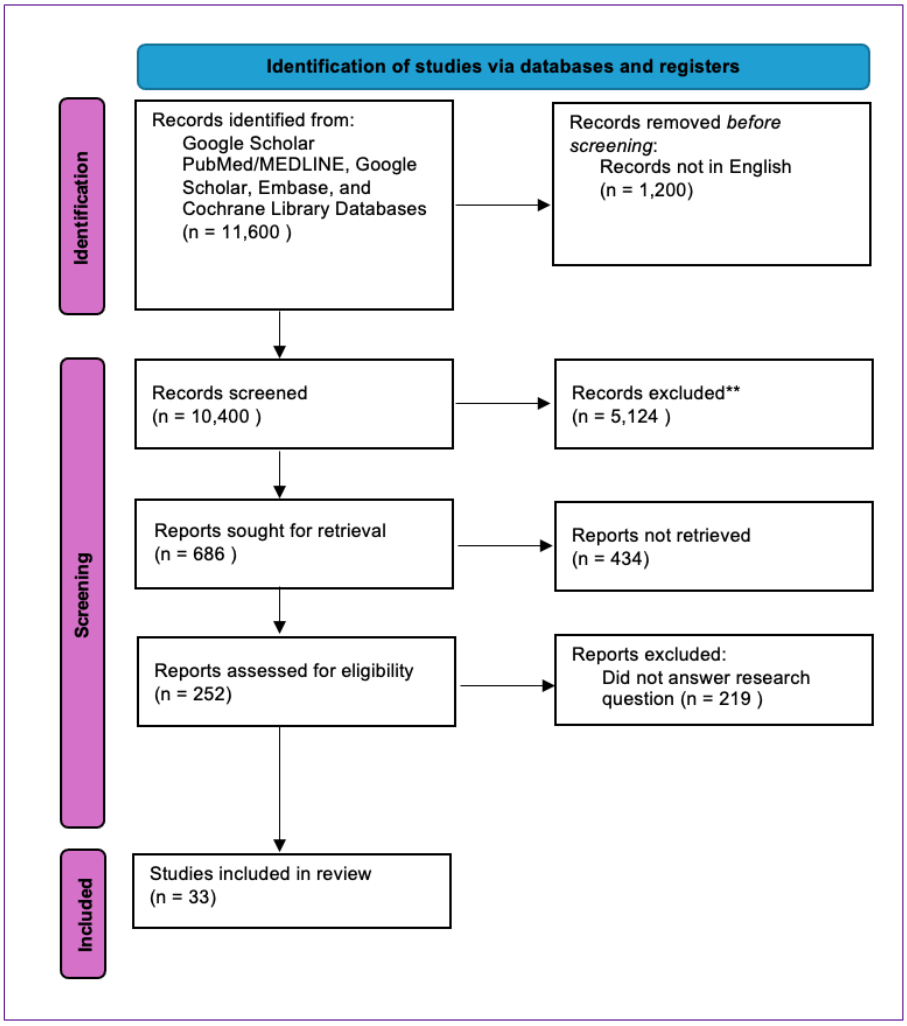

Figure 2. PRISMA flow diagram

tofacitinib.[14] The drug was most prescribed for alopecia totalis, and dermatologists noted enhanced results when used in combination with treatments such as topical steroids or minoxidil. Despite its potential, only 23.2% of dermatologists surveyed prescribed tofacitinib, with barriers like high costs, lack of availability, and insurance coverage limitations being key challenges. Minor side effects such as headaches, nausea, and respiratory infections were reported.[14] These findings underscore the promise of tofacitinib while highlighting ongoing challenges in its widespread application.

Atopic Dermatitis:

Atopic dermatitis (AD) is a chronic, inflammatory skin condition marked by pruritus, which significantly impacts patients' quality of life.[15] Pruritus is a hallmark symptom of AD, often exacerbating the cycle of skin damage and inflammation due to scratching.[16] The pathophysiology of atopic dermatitis (AD) is complex, involving skin barrier dysfunction and immune dysregulation. Loss-of-function mutations in filaggrin weaken the barrier, increasing water loss and susceptibility to irritants. Immune imbalances, particularly a dominant Th2 response, drive inflammation and IgE-mediated hypersensitivity. Environmental triggers further exacerbate these mechanisms by activating innate and adaptive immune pathways, perpetuating inflammation and barrier disruption.[17]

One study investigated the use of topical tofacitinib 2% for the treatment of atopic dermatitis (AD) and found significant improvements in EASI-50, EASI-75, and EASI-90 scores in the treatment group compared to placebo. Additionally, 73% of patients treated with topical tofacitinib achieved clear or almost clear skin based on Physician's Global Assessment (PGA) after four weeks. Another study further highlighted the rapid onset of action of topical tofacitinib, reporting itch and pain relief within 2–3 days in two patients. One patient achieved full remission that was sustained for 12 weeks, while another reached near-complete remission within one week of starting treatment.[18]

In the context of systemic treatment, tofacitinib has been shown to significantly improve both disease severity and patient-reported outcomes. In a multicenter study involving 16 patients with moderate to severe AD, the mean Eczema Area and Severity Index (EASI) score improved from 23.38 ± 9.56 to 8.50 ± 7.57 after 6 months of treatment, and the SCORAD score decreased from 41.25 ± 8.69 to 14.93 ± 7.82. Additionally, patients reported marked improvements in quality of life, with the Dermatology Life Quality Index (DLQI) score decreasing from 15.18 ± 2.73 to 5.31 ± 4.11.[19] These results suggest that tofacitinib not only provides rapid relief from pruritus but also leads to substantial improvements in disease severity and quality of life. However, further research is necessary to fully establish its long-term efficacy and safety.

Furthermore, combination therapies involving tofacitinib have shown impressive results in refractory cases. In one case, a 6-year-old child with severe AD who did not respond to conventional therapies including cyclosporine, was treated with a combination of tofacitinib and narrow-band ultraviolet B (NB-UVB) phototherapy. This combination significantly reduced pruritus and improved the lichenification of the lesions, with the EASI score halved after just four weeks of tofacitinib. The addition of NB-UVB therapy further enhanced lesion resolution, showcasing the potential of tofacitinib when used alongside phototherapy in severe cases.[20] Such combination approaches may provide promising options for patients with

refractory AD, particularly in pediatric populations where treatment options can be more limited.

Vitiligo:

Vitiligo is an autoimmune condition characterized by the progressive loss of melanocytes, leading to depigmented patches of skin. The JAK signaling pathway has been identified as a key contributor to this depigmentation, primarily mediated through the production of interferon-gamma (IFN-γ) by CD8+ T cells. IFN-γ activates JAK1/2 pathways, which subsequently lead to melanocyte destruction through various inflammatory processes, including the upregulation of CXCL9 and CXCL10 chemokines that recruit cytotoxic T cells to the skin.[21,22]

Tofacitinib, a JAK1/3 inhibitor, has emerged as a promising off-label therapy for vitiligo by blocking the JAK-STAT pathway, thus preventing the inflammatory destruction of melanocytes.[22,23] Multiple studies have demonstrated its effectiveness in halting the progression of vitiligo and promoting repigmentation, particularly when combined with phototherapy. In a retrospective case series, 64% of patients treated with oral tofacitinib experienced a halt in disease progression, while 40% showed varying degrees of repigmentation, often enhanced by narrowband ultraviolet B (NB-UVB) phototherapy. [21]

Further studies have compared tofacitinib with other systemic treatments, such as corticosteroids. A comparative study found that patients treated with tofacitinib exhibited greater reductions in the Vitiligo Area Scoring Index (VASI) and Body Surface Area (BSA) scores compared to those treated with betamethasone pulse therapy.[23] In addition, tofacitinib has shown significant efficacy in pediatric populations, where it led to near-complete repigmentation in several cases within 6 months of therapy.[24]

Overall, the growing body of evidence supports tofacitinib as a potentially effective off-label treatment for vitiligo, especially in patients resistant to traditional therapies like corticosteroids and calcineurin inhibitors. While further large-scale randomized controlled trials are necessary, current data indicate a favorable risk-benefit profile, particularly when used in combination with sun exposure or phototherapy. [23]

Psoriasis:

Psoriasis is a chronic inflammatory skin disease driven by genetic predisposition, environmental factors, and immune dysregulation. Key drivers include tumor necrosis factor-α, the IL-23/IL-17 axis, and innate lymphoid cells (ILC3), which produce IL-17 and IL-22 in response to keratinocyte-derived signals. These processes underlie the characteristic skin lesions and systemic associations of psoriasis, including plaque psoriasis, psoriatic arthritis, cardiovascular and metabolic conditions.[25]

Tofacitinib represents an innovative and promising option for patients dealing with moderate-to-severe psoriasis. This medication targets JAK1 and JAK3 [26], which play crucial roles in the JAK-STAT signaling pathway that regulates pro-inflammatory cytokines such as IL-17 and IL-23. These same cytokines are responsible for the mechanism behind both plaque psoriasis and psoriatic arthritis.[27] By inhibiting this JAK-STAT pathway, tofacitinib effectively reduces inflammation and leads to significant improvements in psoriatic symptoms. Clinical trials have showcased impressive efficacy, with PASI75 response rates ranging from approximately 39.5% to 81.1% at doses between 5 mg and 10 mg twice daily. Notably, one phase 2 trial demonstrated a

remarkable 66.7% PASI75 response at higher doses.[28]

Plaque psoriasis:

Plaque psoriasis is a common manifestation of psoriasis, being present in about 80% of cases.[29] It commonly presents on the scalp and torso causing patients to experience itchiness, dry skin, punctate bleeding, and scaling.[29] Although there is no cure for plaque psoriasis, choosing an effective treatment can dramatically improve a patient's outcomes and well-being. Being that plaque psoriasis is frequently diagnosed and a burden to patients, it is worth mentioning how tofacitinib targets plaque psoriasis specifically and can be used as an effective treatment choice.[29] This study emphasizes how tofacitinib directly treats human psoriasis by directly inhibiting the JAK-STAT pathway, and blocking the signaling of cytokines and thereby directly decreasing inflammatory processes involved in plaque psoriasis.[29] A mmeta-analysis found that 5 mg BID of tofacitinib treatment for 12 weeks is the best treatment for psoriatic disease, to optimize dosing schedule and limit adverse effects.[29] In conducting subgroup analysis, this study also found that tofacitinib exhibited significant efficacy in treating both plaque psoriasis and psoriatic arthirtis when compared to the placebo.[29]

Psoriatic arthritis:

Not only does tofacitinib target plaque psoriasis, but psoriatic arthritis as well. Psoriatic arthritis is a chronic inflammatory arthritis that can be associated with enthesitis, dactylitis and spondylitis that can occur with or without nail/skin changes.[30] Tofacitinib is approved for the treatment of psoriatic arthritis and is indicated in those who have failed treatment response to DMARDS or methotrexate.[30] Fortunately, tofacitinib has been proven to help patients with treatment resistant psoriatic arthritis.[30] The recommended dosing is 5 mg BID.[30] One study demonstrates that tofacitinib helps alleviate joint degeneration and decreases proliferation of inflammatory cells in patients with psoriatic arthritis.[27] Specifically for psoriatic arthritis and plaque psoriasis, it was found that tofacitinib significantly improved all metrics of PSAI/PGA response.[27] This drug was found to improve joint health and overall quality of life in patients who are affected by psoriatic arthritis. It was also noted that using higher doses of tofacitinib lead to better patient outcomes for both psoriatic arthritis and plaque psoriasis.[27] There was also research that tofacitinib significantly ameliorate symptoms of psoriatic arthritis for those living in Mainland China and highlighted both its efficacy and safety. It was also highlighted that tofacitinib has been well tolerated in patients with psoriatic arthritis, when compared to other biologics, with only a slight increased risk of herpes zoster.[30]

Additionally, the oral administration of tofacitinib offers a significant advantage over most biologics, which typically require subcutaneous injections, enhancing patient convenience.[28] Though it may not match the highest efficacy rates of biologics, tofacitinib remains useful for patients requiring non-biologic options. Its long-term safety profile is consistent with prior findings, underscoring its role in patient-specific treatment strategies. Continuous comparison with biologics ensures optimized management of this chronic condition.[31] The future of psoriasis management likely lies in a tailored approach and comprehensive treatment profile that can surpass the efficacy of current treatment combinations, integrating biologics and innovative treatments like tofacitinib to provide the greatest benefit to patients.

Challenges and Limitations:

In terms of safety, tofacitinib is generally well-tolerated, although it is important to monitor

potential side effects such as increased cholesterol levels and the risk of infections, particularly herpes zoster.[28] Recent black box warnings regarding cardiovascular risks associated with higher doses in rheumatoid arthritis patients further underscore a need for careful long-term monitoring.[28] Tofacitinib has shown efficacy in dermatological conditions, but challenges related to its long-term safety and accessibility persist. It has been shown that limited availability and high costs are significant barriers to prescribing the drug, with nearly half citing these factors as reasons for non-prescription.[14] Insurance coverage also remains a hurdle in making the medication widely accessible.[14]

Tofacitinib has demonstrated efficacy in treating various dermatological conditions; however, concerns persist regarding its long-term safety and effectiveness. Extended use of JAK inhibitors, including tofacitinib, has been associated with an increased risk of infections, such as herpes zoster, and potential malignancies.[32] The FDA has issued warnings about heightened risks of cardiovascular events and thromboembolism, particularly at higher doses, underscoring the necessity for close monitoring of adverse events.[32,33]

Future Directions and Research Opportunities:

The expanding use of JAK inhibitors in dermatology presents opportunities to explore new indications for tofacitinib, such as lichen planus and cutaneous lupus. Combination therapies with biologics may enhance efficacy while reducing individual drug doses, potentially minimizing adverse effects with future research aiming to address areas like pediatric applications, disease relapse prevention, and the development of new topical formulations. These advances could further refine tofacitinib's role in managing complex dermatological conditions.

CONCLUSION

Tofacitinib has emerged as a promising treatment option for a range of dermatological conditions, from alopecia areata and atopic dermatitis to vitiligo and psoriasis. Its mechanism as a JAK inhibitor allows it to modulate immune responses effectively, providing clinical benefits such as reduced inflammation, hair regrowth, re-pigmentation, and improvement in skin conditions. Despite its efficacy, challenges such as long-term safety, potential adverse effects, and high costs highlight the need for continued monitoring and judicious patient selection. Moreover, existing studies often have limitations, including small sample sizes, lack of diverse patient populations, and limited long-term follow-up data. Future research with larger, more diverse control groups and robust randomized trials will be essential to better understand its safety profile and refine its clinical applications. The role of tofacitinib may be further enhanced through combination therapies with biologics, which could improve outcomes while reducing individual drug dosages. Research focusing on pediatric applications, relapse prevention, and new formulations will also play a critical role in optimizing its use and positioning it within a comprehensive treatment framework for complex dermatological conditions.

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