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*Review Article*

# JAK inhibitors in dermatology

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## ABSTRACT

The four Janus Kinase (JAK) proteins and seven signal transducer and activator of transcription (STAT) factors mediate the intracellular signaling pathway of cytokine receptors, which are described in the pathogenesis of many autoimmune, allergic, and inflammatory dermatoses. The development of targeted small-molecule therapies like JAK inhibitors has enabled a paradigm shift in the treatment of various cutaneous disorders. JAK inhibitors are effective in the treatment of atopic dermatitis, vitiligo, alopecia areata, psoriasis, lupus erythematosus, dermatomyositis, mastocytosis, etc. Various blood parameters include complete blood count, liver, and renal function, the viral marker to be checked and tuberculosis to be ruled out before starting the therapy. There is a risk of acquiring serious infections such as upper respiratory tract infections, urinary tract infections, reactivation of latent tuberculosis, opportunistic infections, hepatitis B virus reactivation, and alteration of various blood parameters; so regular monitoring is required. The use of JAK inhibitors will open a new horizon by reducing the burden of systemic steroids and other non-specific immunosuppressants in the treatment of immune-mediated inflammatory dermatoses.

**Keywords:** Tofacitinib, Ruxolitinib, Baricitinib, Janus Kinase-signal transducer and activator of transcription, Janus Kinase inhibitor, Alopecia areata, Atopic dermatitis, psoriasis, Vitiligo

## INTRODUCTION

Janus Kinase (JAK) inhibitors are a recent addition to the therapeutic armamentarium of various inflammatory cutaneous diseases. These drugs target intracellular transcription through JAK-signal transducer and activator of transcription (STAT) signaling pathways. Many cutaneous autoimmune diseases are mediated by cytokines that depend on the JAK-STAT signaling pathway. Thus, inhibition of this pathway using JAK inhibitors could be an important management strategy for various inflammatory dermatoses.

There are two generations of JAK inhibitors presently known. The first generations are non-specific and target two or more different types of JAKs, whereas second-generation JAK inhibitors are specific and target only a single type of JAK.

Tofacitinib, Ruxolitinib, Baricitinib, and oclacitinib are examples of first-generation JAK inhibitors. Decernotinib, Peficitinib (ASP015K), Filgotinib (GLPG0634), Fedratinib, Momelotinib, Apeficitinib (ASP015K), Upadacitinib (ABT-494), and Lestaurtinib are considered as second-generation JAK inhibitors. Although the first-generation JAK inhibitors are already being used in the treatment of different autoimmune dermatoses, the second-generation JAK inhibitors are yet to clear their phase III trials but promise to have fewer adverse effects.

## METHODS

A literature review of MEDLINE, PubMed, Embase, and the Cochrane Controlled Register of Trials database for studies evaluating the efficacy of JAK inhibitors in dermatological disorders was done. All the studies between 2013 and 2021 describing the mechanism of action, pharmacokinetics, efficacy, or side-effects of JAK inhibitors were identified. The therapeutic guidelines are proposed to provide an evidence-based approach to combining JAK inhibitors with conventional agents to treat various autoimmune dermatoses.

## MECHANISM OF ACTION

JAK s are a group of cytoplasmic enzymes that work on the intracellular domains of several cytokine receptors. The mammalian JAK family has four members, namely, JAK1, JAK2, JAK3, and TYK2 (tyrosine kinase 2). Each cytokine receptor is associated with a specific JAK enzyme, but one JAK can be associated with multiple receptors.

JAK Type 1 is associated with cytokine receptors such as INF, IL-6, and IL-10. Similarly, JAK2 is associated with hematopoietic receptors. Cytokines for lymphocyte function like IL-2, IL-4, IL-7, IL-9, IL-15, and IL-21 work through

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JAK3. TYK2 in conjunction with JAK2 works on INF, IL-12, and IL-23 receptors.

The STAT family has seven members, namely STAT1, STAT2, STAT3, STAT4, STAT5a, STAT5b, and STAT6. These proteins, in conjunction with JAKs, are involved in signal transduction and activation of transcription.[1]

Cytokines such as interleukins and IFNs utilize the JAK-STAT pathway to transmit signals from the cell membrane to the nucleus.

Extracellular ligands bind the cytokine receptors (Type I/II) to activate the intracellular JAK proteins. It then phosphorylates the STAT proteins, resulting in the dimerization of the STAT proteins. Dimerized STAT proteins reach the nucleus and regulate the transcription of cytokines and the maturation of dendritic cells.[2] [Figure 1].

Inflammatory mediators related to different dermatoses associated with various JAK-STATs are listed in [Table 1].[3]

## PHARMACOKINETICS

Tofacitinib is orally absorbed with an average bioavailability of about 74%. The peak plasma level is about 30 min and the plasma half-life is about 3 h. It is metabolized in the liver through the CYP3A4 pathway, though the elimination of the drug is through both the hepatic (70%) and renal (30%) routes.[4]

Similarly, Baricitinib also gets metabolized in the liver by the CYP3A4 pathway, but 90% of the molecule gets excreted unchanged in urine and stool.

## CONTRAINDICATIONS[5]

Absolute contraindications of Tofacitinib use are hypersensitivity to the drug, active infections (e.g., active tuberculosis, sepsis, hepatitis B, hepatitis C, serious localized cellulitis, herpes zoster), malignancy and lymphoproliferative disorders, hematological abnormalities-lymphocyte count (absolute) <500/mm$^3$, neutrophil count (absolute) <1000/mm$^3$ or hemoglobin < 9 g/dL, severe hepatic disorder, gastrointestinal perforations, etc.

Relative contraindications include a history of severe opportunistic infection, exposure to tuberculosis, low-to-moderate hepatic dysfunction, chronic kidney disease, and chronic lung disease are the relative contraindications.

## SPECIAL PRECAUTIONS

### Pregnancy

Tofacitinib pregnancy category "C." For Baricitinib, the US FDA pregnancy category has not been assigned till now.

### Lactation

Tofacitinib and Baricitinib can both be secreted in breast milk and should be avoided in lactating mothers.[5]

### Children and adolescents

Tofacitinib has got U.S. FDA approval for children 2 years and older with juvenile idiopathic arthritis, though the data related to dermatological disorders is not adequate and best to be avoided below 18 years of age.

### Hepatic and renal disorders

Dose adjustment is required to 5 mg once a day for Tofacitinib in moderate hepatic dysfunction (Child-Pugh score Grade B, 7–9 points) and in moderate-to-severe renal dysfunction (creatinine clearance <49 mL/min).

### Active infection

Treatment with tofacitinib should not be started if a localized active infection is present, for example, nasopharyngitis and cellulitis.

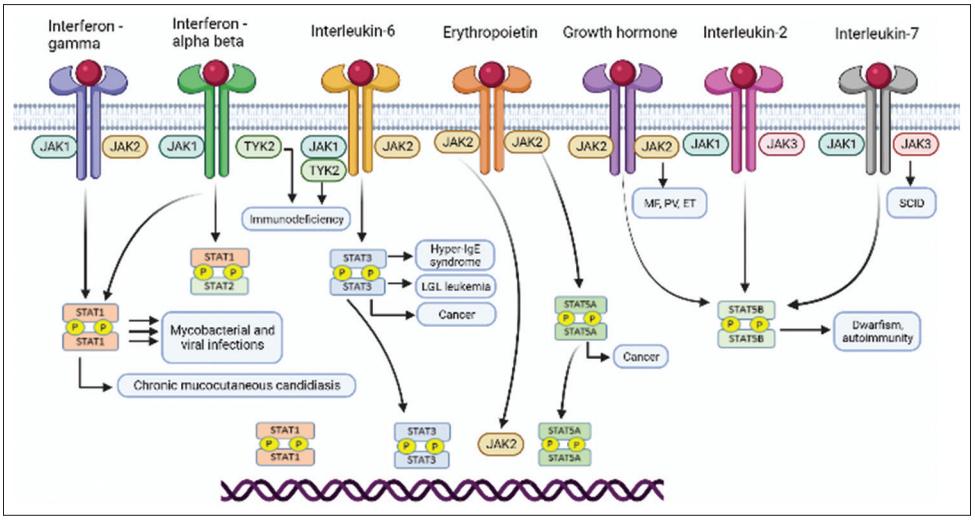


**Figure 1:** Four Janus kinase signal transducer and activator of transcription pathways in health and disease. MF: Primary myelofibrosis, PV: Polycythemia vera, ET: Essential thrombocythemia.

**Table 1:** Inflammatory dermatoses related with JAK-STAT pathways.

| Disease | Inflammatory mediators | JAK utilized | STAT utilized |
|---|---|---|---|
| Alopecia areata | IL-15 | JAK1/3 | STAT3/5 |
| | IFN-γ | JAK1, TYK2 | STAT1 |
| Atopic dermatitis | IL-4 | JAK1, JAK3 | STAT6 |
| | IL-5 | JAK2 | STAT3/5/6 |
| | IL-13 | JAK1/2/3, TYK2 | STAT6 |
| Psoriasis | IL-12 | JAK2, TYK2 | STAT4 |
| | IL-23, TNF-α, IL-17 | JAK2, TYK2 | STAT3/4 |
| Vitiligo | IFN-γ | JAK1, TYK2 | STAT1 |
| Lupus Erythematosus | IFN-α/β | JAK1, TYK2 | STAT1/2/4 |
| | IFN-γ | JAK1, TYK2 | STAT1 |
| | IL-6 | JAK1/2, TYK2 | STAT1/3 |
| Dermatomyositis | IFN-α/β | JAK1, TYK2 | STAT1/2/4 |
| | IL-6 | JAK1/2, TYK2 | STAT1/3 |
| | IL-15 | JAK1/3 | STAT3/5 |
| Mastocytosis/mast cell disease | IL-4 | JAK1, JAK3 | STAT6 |
| | IL-5 | JAK2 | STAT3/5/6 |
| | IL-13 | JAK1/2/3, TYK2 | STAT6 |
| Cutaneous T cell lymphoma | JAK1/3, STAT3/5b mutation | JAK1/3 | STAT3/5b |
| Chronic mucocutaneous candidiasis | STAT1 mutation | unclear | STAT1 |

STAT: Signal transducer and activator of transcription, JAK: Janus kinase

## ADVERSE EFFECTS

There is a risk of acquiring serious infections like upper respiratory tract infections including nasopharyngitis, urinary tract infections, reactivation of latent tuberculosis, opportunistic infections, hepatitis B virus reactivation, and reactivation of Varicella-Zoster virus.[6] The use of live vaccines concurrently with JAK inhibitors should be avoided as it can affect the response to vaccination. Immunizations should be performed at least 2 weeks before initiating therapy with JAK inhibitors.[7] Lymphocytopenia, neutropenia, thrombocytopenia, anemia, leucopenia, liver enzyme elevation, and altered lipid profile are also known side effects. An increased risk of malignancies was observed, which include melanoma, non-melanoma skin cancers, solid organ tumors, and lymphoproliferative disorders.[8] Cutaneous adverse effects such as urticaria, angioedema, and rash can also occur.

## MONITORING GUIDELINES FOR TOFACITINIB

A baseline complete hemogram, renal function tests, liver function tests, fasting lipid profile, HBsAg, anti-HCV, Mantoux, or IFN-γ release assay to exclude tuberculosis and HIV should be done.

Complete hemogram, renal function tests, liver function tests, and fasting lipid profile should be repeated after 1 month, then every 3 months.

## DRUG INTERACTIONS

Concomitant use of ketoconazole should be avoided as it is metabolized through CYP3A4, and it reduces its metabolism and increases its serum level. Similarly, CYP3A4 inducers like Rifampicin decrease its serum level by increasing the metabolism of the drug. Immunosuppressives such as azathioprine and cyclosporine should also not be coadministered.

## CLINICAL USE OF ORAL JAK INHIBITORS IN SKIN DISORDERS

FDA-approved uses of JAK inhibitors and their dosing are listed in [Table 2].[3] However, different clinical trials have established efficacy and safety of the JAK inhibitors in various dermatological condition beyond its FDA-approved indications.

### Psoriasis and psoriatic arthritis

Multiple clinical trials have been published relating to the use of JAK inhibitors in the treatment of psoriasis and psoriatic arthritis. PASI 75 response (63.6%) was better with 10 mg BD dose than 5 mg BD dose (39.5%) at 12 weeks was shown in a study done by Bachelez *et al.*[9] Tofacitinib 10 mg BD was as efficacious as Etanercept 50 mg subcutaneous twice weekly.[9] Improvement in DLQI was also significantly improved by 47.3% with Tofacitinib 10 mg BD and 43.6% with Etanercept. Pruritus was much less common among the Tofacitinib group.[10] It is also very effective in treating psoriatic arthritis. ACR20 response was 50%, 61%, 52%, and 33% respectively in Tofacitinib 5 mg BD versus Tofacitinib 10 mg BD versus Adalimumab 40 mg sc fortnightly versus placebo.[11] Topical Tofacitinib is also proven to be efficacious in localized plaque psoriasis.[12] Baricitinib can also be tried in psoriasis, which

**Table 2:** FDA approved indications of JAK inhibitors.

| Drug | Works on | FDA-approved indications | FDA-approved dosing |
|---|---|---|---|
| Tofactinib | JAK1/3>2 | Rheumatoid arthritis (2012), psoriatic arthritis (2017), Ulcerative colitis (2018) | 5 mg twice daily |
| Ruxolitiniib | JAK1/2 | Myelofibrosis (2011), polycythemia vera (2014) | 5–25 mg twice daily |
| Baricitinib | JAK1/2 | Rheumatoid arthritis (2018) | 2 mg once daily |

JAK: Janus kinase

has proven reduction of PASI 75, 43%, and 54%, respectively, in 8 mg OD versus 10 mg OD dose.[13]

### Atopic dermatitis (AD)

Many clinical trials examining the efficacy of oral and topical JAK inhibitors in AD have already been established. A 66.6% reduction in the Severity Scoring of AD Index (SCORAD) and a 69.9% reduction in pruritus and sleep loss scores were noticed with Tofacitinib 5 mg BD in a study conducted by Levy *et al.*[14] Topical 2% Tofacitinib also causes an 81.7% of reduction in the Eczema Area and Severity Index score, compared to 29.9% in the placebo group.[15] Baricitinib is also very effective in treating recalcitrant AD. Patients achieved the primary endpoint of Validated Investigator's Global Assessment of AD 4.8%, 11.85%, 11.4%, and 16.8% in placebo, Baricitinib 1 mg, 2 mg, or 4 mg group, respectively.[16]

### Alopecia areata (AA)

Initially, Tofacitinib showed promising results when a patient with alopecia universalis developed complete regrowth of hair within 8 months.[17] Since then, multiple clinical trials have been published relating to the use of JAK inhibitors in patients with AA.[18] Although recurrent relapses and remissions are known to occur in patients with AA who are treated with JAK inhibitors; it demands proper counseling at the beginning of the course.[19]

Tofacitinib and oral ruxolitinib have shown similar efficacy in the management of AA, but they have a better therapeutic outcome than contact immunotherapy and a less adverse event profile than conventional immunosuppressive treatments such as corticosteroids and cyclosporine in AA.[20] The molecule also improves the nail symptoms of AA.[21] In a study by Liu *et al*., it is seen that 77% achieved clinical response, 58% achieved >50% change in SALT score with Tofacitinib 5 mg BD at 4–18 months of treatment.[22] Ruxolitinib has shown better results as >92% hair regrowth occurred in 75% of patients with a 20 mg BD dose at 12–24 weeks.[23]

### Vitiligo

At first, tofacitinib showed complete re-pigmentation in a patient with generalized vitiligo treated with tofacitinib. However, there was a relapse after the cessation of therapy.[24] Similarly, a patient having both vitiligo and AA who was treated with systemic Ruxolitinib experienced significant improvement in both conditions, but again they relapsed after discontinuation of therapy.[25] Combination therapy with tofacitinib and low-dose narrowband UVB has shown noteworthy improvement in vitiligo that was documented by Kim *et al.*[26]

The topical application of JAK inhibitors has also proven efficacious. Significant repigmentation was noticed with Ruxolitinib 1.5% of cream in a small group of 12 patients with vitiligo.[27] Similar results were established in a study with 16 patients applying 2% of tofacitinib cream, and facial lesions responded better than non-facial lesions.[28]

### Dermatomyositis (DM)

Significant clinical efficacy was observed in an open-label pilot study of tofacitinib in refractory DM in 10 patients as measured by validated myositis response criteria.[29] A literature review in 2021 of 53 DM patients treated with JAK-inhibitors reported no serious adverse events. Tofacitinib was the most frequently used JAK-inhibitor, followed by Ruxolitinib. The review revealed that most studies reported significant improvement in clinical parameters such as CDASI score, muscle strength, body weight, and skin lesions.[30]

### Lupus erythematosus

Wenzel *et al*. used Ruxolitinib in an elderly patient suffering from chilblain lupus, resulting in complete remission of the condition in 4 months.[31] However, the use of JAK inhibitors in LE demands further research.

### Lichen planopilaris (LPP)

JAK inhibitors can have a promising result in LPP as a recent study has demonstrated the upregulation of JAK1 and JAK3 in dermal inflammatory cells in the pathogenesis of LPP.[32] Eight out of 10 patients showed significant clinical improvement in LPP with oral tofacitinib (10–15 mg/day) for 2–9 months in a study without significant adverse effects.[33]

## CONCLUSION

There is growing evidence that the JAK/STAT pathway has a pathogenic role in many inflammatory dermatological conditions. Their capability to induce faster results and limited toxicity has added a greater dimension to their use and will be more widely used shortly. Although the sustained efficacy of this molecule is still a question. Large, multicentric, randomized, and controlled trials are required

to establish the efficacy and safety of JAK inhibitors in a variety of cutaneous conditions.

### Key message

The JAK-STAT pathway is linked with many auto-immune dermatoses. It has got significant efficacy in treating various cutaneous conditions such as psoriasis, psoriatic arthritis, AA, and vitiligo. It is relatively safer than other systemic immunosuppressives such as steroids and cyclosporine. It should be given with proper follow-up and monitoring.

### Declaration of patient consent

Patient's consent not required as there are no patients in this study.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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