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Tofacitinib: The selected selective JAK inhibitor in paediatric dermatology

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Abstract
Tofacitinib is a strong, selective JAK inhibitor that favorably inhibits *JAK1 and JAK3* in addition to the inhibition of the phosphorylation of STAT5, STAT3 and STAT1. It is renowned and accepted for the therapeutic purposes of several conditions like psoriatic arthritis, ulcerative colitis, and rheumatoid arthritis. It is of great value for several dermatological related conditions of various age groups. Nonetheless, the use of tofacitinib for pediatric age group is still under research. A literature review was done which showed that tofacitinib orally can be a safe and effective alternative for several other dermatological conditions like refractory juvenile dermatomyositis, atopic dermatitis, *alopecia Areata*, and several types of psoriasis. Topically it is used for conditions like vitiligo vulgaris, *alopecia Areata* and halo naevus. The pros and cons must be weighed out prior to considering this drug. In this article, we have compiled a brief synopsis of the corroboration of utilizing tofacitinib (both topical and oral preparations) in paediatric age groups.

Keywords: Dermatology, Tofacitinib, Janus Kinase inhibitors, Paediatric age group, Atopic dermatitis, Vitiligo vulgaris, *Alopecia Areata*, Halo nevus and Psoriasis

Introduction
Tofacitinib citrate is an oral Janus kinase 1/3 inhibitor. Approval of Tofacitinib for various types of Arthritis, Polyarticular Juvenile Idiopathic Arthritis and Ulcerative Colitis have been given by the FDA. But in pediatric age group, tofacitinib is permitted only for the therapeutics of polyarticular juvenile idiopathic arthritis in children above 2 years by the FDA. Vitiligo, atopic dermatitis (AD). *alopecia Areata* (AA) and dermatomyositis are few of the dermatoses for which it is used off-label [1, 2]. But there is still inadequate data for children on its effectiveness and safety. This study aims to compile all available information on the use and safety outline of Tofacitinib in children with different dermatoses.

Materials and Methods
We searched the PubMed database and Google Scholar for studies on the use of tofacitinib using keywords such as Dermatology, Tofacitinib, Paediatric age group, Atopic dermatitis, Vitiligo vulgaris, *Alopecia Areata* and Psoriasis.
Original articles, reviews, and meta-analyses on the themes addressed were preferred. Our review included more than 32 articles that were extracted from publications between 2010 and 2023. The findings of these studies were synthesized into a narrative review.

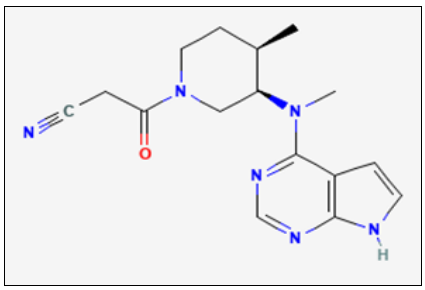

Fig 1: Structure of Tofacitinibl

Discussion

Structure: Tofacitinib is a pyridopyrimidine with an N-methyl, N-(1-cyanoacetyl-4-methylpiperidin-3-yl) amino moiety at position 4 substituting for pyrrolo[2,3-d]pyrimidine [3].

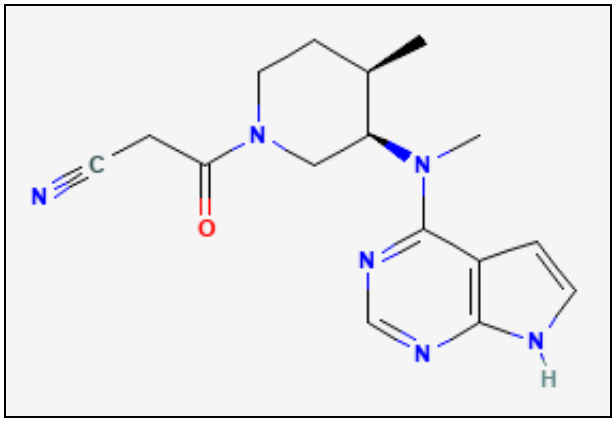

Fig 1: Structure of Tofacitinib

Dosage of tofacitinib

Dosage of tofacitinib approves for juvenile dermatomyositis is given in TABLE I

Tofacitinib can also be used topically as a 2% ointment.

Pharmacokinetics

Tofacitinib is highly absorbable after oral administration, with a 74 % bioavailability in healthy individuals. When given with a fatty meal, plasma concentrations are reduced by 32%. Tofacitinib binds to 40 % of proteins, prominently albumin, and distributes evenly between RBCs and plasma [4].

Mechanisam of action of tofacitinib

Various cytokines like IFN-a, IFN-b, IFN-c and Interleukin (IL)-2 receptors employ Janus Kinase–signal transduce [5, 6]. The tyrosine kinase family comprises of JAK1, JAK2, JAK3, and TYK2. Every JAK contains four structural domains and seven types of homologous regions (JH1–7) within them. JH1 is a catalytic phosphotransferase that contains an adenosine triphosphate binding site, which triggers autophosphorylation of the JAK and STATs. JAK inhibitors block the adenosine triphosphate (ATP) binding site on JH1 in a competitive manner [7].

Tofacitinib mainly inhibits JAK 1 and JAK3 which are located on T cells, B cells, NK cells and mast cells. Tofacitinib is predominantly metabolized by CYP3A4 and to a lesser extent by CYP2C19 [7].

Adverse Effects of Tofacitinib [8]

The adverse effects of tofacitinb are mentioned in Table-II Tofacitinib can lead to a risk of pulmonary embolism and also deep vein thrombosis at higher doses [6].

Relation between vaccination and tofacitinib:

Prior to treatment, the patient should be up to date on their influenza vaccine and pneumococcal vaccine. Specifically, patient should consider receiving the varicella vaccine. The recombinant shingles vaccine is preferred as live vaccines are not preferred while on Tofacitinib therapy or within 3 months of stopping or starting the drug.

Drug interactions

Interaction with ketoconazole, increases the systemic exposure of tofacitinib [4].

Administration of fluconazole produced an increased efficacy in tofacitinib.

Immunosuppressive drugs, like azathioprine, tacrolimus, and cyclosporine, can cause increase in the risk of immunosuppression when administered with tofacitinib hence should be avoided [4]. Administration of rifampin, a potent CYP3A4 inducer, substantially decreased the mean AUC by 84% and decreased C_{max} by 74%.Concomitant potent immunosuppressive drugs, such as azathioprine, tacrolimus, and cyclosporine, increase the risk of immunosuppression when taken with tofacitinib. Although the combined use of multiple-dosed tofacitinib with immunosuppressive agents has not been studied in RA, the theoretical risk exists. Coadministration of tofacitinib with stable methotrexate therapy produces a 10% decrease in methotrexate AUC and a 13% decrease in methotrexate C_{max}. However, this is not considered to be clinically significant.

Investigations [2]

The pretreatment and post treatment investigations for tofacitinb are mentioned in Table-3.

The tests are advised 4 weeks after completion of treatment and every 3 months thereafter.

Hb – fall of Hb less than or equal to 2g/dl and Hb greater or equal to 9gm/dl can be on maintenance dose.

Fall of Hb more than 2g/dl and Hb lesser than to 8gm/dl, should be advised interruption of tofacitinib till hemoglobin level is normalized.

Monitoring

It is recommended that lymphocyte counts are to be evaluated at baseline and every 3 months throughout tofacitinib therapy. Additionally, neutrophil counts and haemoglobin laboratory values should be evaluated at baseline, after 4 to 8 weeks of treatment and every 3 months throughout therapy. Routine monitoring of liver function is recommended to identify liver enzyme elevations indicative of tofacitinib-induced liver injury [1]. A complete lipid panel should be taken at baseline and after approximately 4 to 8 weeks of tofacitinib therapy. Additional monitoring parameters may include clinical markers and symptoms of disease progression. The absolute neutrophil count should be regularly monitored. The dosage and duration of drug exposure were linked to the decrease in level. Six to eight weeks of treatment at doses of 5 and 10 mg orally resulted in the low neutrophil levels within six weeks of stopping the medication, the neutrophil count returned to normal.

Juvenile dermatoses

JDM is a rare autoimmune vasculopathy in children that causes proximal muscle weakness and pathognomonic skin rashes. Organs such as the lungs, intestines, and heart are also susceptible. Refractory JDM occurs when a patient does not respond to two disease-modifying anti-rheumatic drugs (DMARDS) or steroids of high dosage [8]. Tofacitinib is a suitable choice because it promptly improves signs and symptoms and helps with steroid tapering [10].

Following JAK inhibitor treatment, *Papadopoulou et al.,* [11] observed a decrease in IFN gene response and an increase in endothelial damage biomarkers. In addition, AK inhibitors

also have an anti-inflammatory property [12].

Infections, such as infections of the upper respiratory tract and opportunistic infections, are common adverse effects reported. Herpes virus infection and BK viraemia are rare side effects [9] coexisting use with DMARDs with immuno-suppressants and tofacitinib should be approached with precaution, since fatality has been reported in two elderly patients on combination therapy, due to the result of severe infection [11].

Evidence of use of tofacitinib for juvenile dermatoses are mentioned in Table-IV

Alopecia Areata

Alopecia Areata (AA) is a common autoimmune condition that Results in non-scarring type of hair loss. [14] In individuals with severe AA who have not responded adequately to standard medication, tofacitinib may be considered a treatment option [1]. IFN-c, IL-2, 7, 9, I5, and 21 signaling are all inhibited. IL-15 is released from the hair follicle's outer root sheath, causing CD8+ T cells to be recruited; tofacitinib inhibits CD8+ T cell activation. Treatment duration is usually over 6 months.

Minor adverse events (AEs) recorded during a 12-month period include URTIs, urinary tract infections and tonsillitis. [14] Ruxolitinib (Janus Kinase 1/2 inhibitor), tofacitinib (Janus Kinase 1/3 >2 inhibitor), and baricitinib (Janus Kinase 1/2 inhibitor) have all been proved to be extremely effective with good tolerability in individuals with different types of psoriasis, vitiligo, atopic dermatitis, and *Alopecia Areata* [15]

Evidence of use of tofacitinib for *Alopecia Areata* are mentioned in Table-5

Vitiligo

Vitiligo is a chronic autoimmune condition in which melanocytes gradually disappear, resulting in depigmentation [18]. Case reports were done on JAK inhibitors for the treatment of vitiligo [19], as well as an open-label research using topical 1.5 percent ruxolitinib cream, which all demonstrated endearing results in the treatment of vitiligo [20].

Inhibition of IFN-c occurs that leads to lowering of CXCL10 expression in the basal keratinocytes. JAK–STAT inhibitors combined with narrowband ultraviolet B (NB-UVB) produced a fast and satisfactory response in a case of segmental vitiligo, according to olamigu and Craiglow [20]. However, it is unclear whether the improvement was due to the NB- UVB alone or due to the combination with tofacitinib.

Safety. Given the existing lack of compelling evidence, topical JAK inhibitors may be chosen for localized vitiligo, particularly in children [20].

Evidence of use of tofacitinib for Vitiligo are mentioned in Table 6

Psoriasis

Psoriasis is an inflammatory, immune-mediated systemic disease that has a physical and psychological impact on the patients, resulting in significant impairment of the quality of life [21]. Clinical heterogeneity may lead to diverse therapy courses, making management difficult.

Since the majority of current anti psoriatic medicines have not been licensed for use in children, treating psoriasis in children can be difficult. Toxicity, ineffectiveness, and teratogenicity are just some of the concerns that limit their usage un-children. Tofacitinib is a safe and efficacious treatment choice for psoriasis.

Tofacitinib can cause reduction in keratin 16 expression and in number of CD3+, CD8+, CD11c+ and CD25+ cells in psoriatic plaques [22] tofacitinib also suppresses IL-31, a mediator for pruritus [6].

With 12 weeks of tofacitinib medication, Almutairi and Nour observed minimal acceptable AEs as URTIs, headaches, and nausea. Neutropenia, lymphocytosis, and an elevation in liver enzymes were recorded for a short period of time, then gradually recovered to baseline by Week 36. [23]. Over the course of 36 weeks, mild elevations in LDL and HDL were observed. All of these adverse effects have only been reported in adult research.

Evidence of use of tofacitinib for refractory Psoriasis are mentioned in Table 7.

Halo nevus

It is a benign melanocytic naevus with a rim of depigmentation around it, like a halo [24].

Usage and safety as reported. After a treatment period of 2 months with tofacitinib 1.5 % cream twice a day, Hu *et al*. [24] observed a notable improvement. The two patients were observed with a dermoscope for a one and a half years, respectively, and neither had recurring symptoms and signs of halo naevus, nor was there any evidence of any malignant change. However, it is unclear why the treatment was not extended beyond the 8-week mark.

Atopic dermatitis

Atopic dermatitis is a chronic, pruritic inflammatory autoimmune illness marked by xerosis, eczematous lesions, redness, and intense pruritus [25].

Inhibition of JAK- signaling, which medicates for intense pruritus resulting in decreased itching and Improvement of the skin barrier function. A 63-year-old man who had failed various systemic therapies and had a prolonged history of severe case of AD had presented with atopic dermatitis since infancy, which had progressively worsened in the last 8 years, escalating to the point of total body involvement.

Tofacitinib citrate 5 mg was started on a daily basis. He was maintained on prednisone 20 mg/daily for the first month, then it was gradually tapered from 20 mg to 10 mg, then 5 mg, until it was completely stopped. Only moisturizers were utilized during this time instead of topical corticosteroids. He obtained virtually total clearance of his face, torso, upper and lower extremities after three months of treatment 19. Multiple major trials have been unsuccessful to show any significant risk of cancer related with tofacitinib medication.

HIV

Tofacitinib and other JAK inhibitors like ruxolitinib have both been shown to reduce various cytokine which inhibit HIV-1 viral replication in macrophages and lymphocytes. [26].

COVID

The acetylcholinesterase receptor, IFN is stimulated by the SARS-CoV-2 virus whose location is expressed in the human respiratory epithelium.

JAK2 inhibitor such as fedratinib or tofacitinib may be beneficial since they would not hinder with JAK1-mediated or IFN-mediated antiviral and antibacterial properties [27, 28].

Pyoderma gangrenosum
JAK1, JAK2, and JAK3 activation was detected in immunohistochemistry staining of tissues from patients in retrospective studies [29].

Generalised deep morphea
The growth factors IL-4 and TGF-b cause fibroblasts to produce more collagen formation and extracellular protein. Janus Kinase inhibitors have been found to decrease IL-4 signaling and TGF-b-induced fibrosis in JAK-2-dependent cells [30].

Eosinophilic fascitis
Eosinophil proliferation is triggered by IL-5 release by clonal cells. JAK inhibitors reduce the proliferation of IL-5 by blocking its secretion and activity [30].

Lichen planopilaris
The hair follicle bulge responds to IFN by producing more chemokines, which attract CD8+ T cells. In the lichen planopilaris, JAK inhibitors reduce IFN-mediated inflammation, reducing hair follicle loss [31].

Palmoplantar pustulosis
After 3 months of therapy with tofacitinib of dose 5 mg twice a day, the Nail Psoriasis Severity Index and Palmoplantar Pustulosis Area and Severity Index improved significantly in a prospective study Acute tonsillitis was reported in one patient and a temporary elevation in LDL and HDL cholesterol levels were also noted as adverse effects [32].

Table 1. Shows dosage of tofacitinib.

Table 1: Dosage of Tofacitinib

Body Weight (KG)	Dose (mg)	Dose (ml)
5 -11	1.1	1
12-18	1.1.5	1.5
19-24	2	2
25-31	2.5	2.5
32-39	3	3
>40	5	5

Table 2: Side Effects of Tofacitinib

Common Side Effects	Rare Side Effects
Upper respiratory tract infections	Reactivation of Herpes zoster. Tuberculosis
Headache	Histoplasmosis
Nasopharyngitis	Candidiasis
Diarrhoea	Reactivation of Herpes zoster. Tuberculosis
Hypertension	Lymphoma[9]
Urinary tract infection	Epstein–Barr virus-associated post-transplant lymphoproliferative conditions
Dyslipidemia	
Anemia	

Table 2 shows side effects of tofacitinib.

Table 3: The pre-treatment and post treatment investigations for Tofacitinib

Pre-treatment investigations	Post treatment investigations
Complete blood investigation	Complete blood investigation
Absolute neutrophilic count (Do Not Start IF ANC < 1000 cells/mm^3)	Serum creatinine
Absolute lymphocytic count (Do Not Start IF ALC < 500 cells/mm^3)	Fasting lipid profile
Serum creatinine	Tuberculosis screening yearly
Liver function test	
Fasting lipid profile	
HIV serology	
HBsAg and core antibody and Hep C Serology	
Mantoux test	

Table 3. Shows the pretreatment and post treatment investigations

Table 4: Evidence of use of tofacitinib for juvenile dermatoses

Refrence	Number of patients per study	Dose	Duration	Result	Side Effects
Ding *et al.*, [11]	25	2.5 mg twice a day - weight < 25 kg 5 mg twice a day - weight > 25 kg	3-18 months	66% showed complete response, while the remaining had visible clinical improvement post therapy.	URTIs
Sozeri *et al*	2	5 mg twice daily	3 months	Marked improvement	None
Sabbagh *et al.*, 2019 [13]	2	5 mg twice daily	6 months	Improved pulmonary function and improvement in disease activity measured by ACR/EULAR myositis response criteria.	None

Table 4. Shows evidence of use of tofacitinib for juvenile dermatoses.

Table 5: Evidence of use of tofacitinib for *alopecia Areata*

Refrence	Number of patients per study	Status of patient	Dosage	Result	Adverse effect
Bayart *et al*., 2017 [16]	6	tofacitinib cream 1%;	1 year	Marked improvement	None
Dai & Chen, 2019 [17]	4	2.5 mg once a day	1 year	More than 90% improvement with 1 year of therapy in 20% in one fourth of patients. >50% with 6 and 21 months of therapy in the remaining patients	GI disturbance like diarrhea and URTI
Almutairi N, *et al*., 2019 [14]	75	The first group- ruxolitinib (20 mg) twice daily second group-oral tofacitinib (5 mg) twice a day	Treatment period - 6 months. Follow up - 3 months.	Tofacitinib and ruxolitinib showed immense regrowth of hair	None

Table 5. Shows evidence of tofacitinob for *alopecia Areata*

Table 6: Evidence of use of tofacitinib for vitiligo

Refrence	Number of patients per study	Dose	Duration	Results	Adverse effect
Olamiju & Craiglow, 2020 [20]	1	Tofacitinib 2% two times a day, application, with NB-UVB therapy	6 months	Near-complete response at 12 weeks; complete response at 6 months. Synergistic effect between NB-UVB and JAK inhibitor	None

Table 6. Shows evidence of tofacitinib for vitiligo

Table 7: Evidence of use of tofacitinib for refractory psoriasis

Refrence	Number of patients per study	Dose	Duration	Result	Adverse effect
Almutairi & Nour, 2020 [23]	47	Tofacitinib 5 mg BD for	9 months	PASI75 and PASI90 reached by 55% and 29% of patients, respectively, by Week 12, and by 70.21% and 42.55% of patients, respectively, Week 36	Nasopharyngitis, URTI, headache, paronychia, and nausea

Table 7. Shows evidence of use of tofacitinib for refractory Psoriasis

Conclusion

The important outcomes of the study should be mentioned in this section.

Note: Results, Discussion and Conclusion can be combined if seems appropriate.

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Conflict of Interest

Not available

Financial Support

Not available

References

1. Agarwal I, Panda M, Das A. Tofacitinib in paediatric dermatoses: A narrative review. Clin Exp Dermatol. 2022 Jul 1;47(7):1256-64.
2. Sonthalia S, Aggarwal P. Oral tofacitinib: Contemporary appraisal of its role in dermatology. Indian Dermatol Online J. 2019 Sep 1;10(5):503-18.
3. PubChem [Internet]. Bethesda (MD): National Library of Medicine (US), National Center for Biotechnology Information; 2004-. PubChem Compound Summary for CID 9926791, Tofacitinib; [Cited 2022 May 29].
4. Cada DJ, Demaris K, Levien TL, Baker DE. Tofacitinib. Hosp Pharm. 2013 May;48:413-24.
5. Ruperto N, Brunner HI, Zuber Z, *et al*. Pharmacokinetic and safety profile of tofacitinib in children with polyarticular course juvenile idiopathic arthritis: results of a phase 1, open-label, multicenter study. Pediatr Rheumatol Online J. 2017;15:86.
6. Samadi A, Ahmad Nasrollahi S, Hashemi A, *et al*. Janus kinase (JAK) inhibitors for the treatment of skin and hair disorders: a review of literature. J Dermatolog Treat. 2017;28:476-83.
7. Kerrigan SA, McInnes IB. JAK inhibitors in rheumatology: implications for paediatric syndromes? Curr Rheumatol Rep. 2018;20:83.
8. Tegtmeyer K, Zhao J, Maloney NJ, *et al*. Off-label studies on tofacitinib in dermatology: a review. J Dermatolog Treat. 2021;32:399-409.
9. Damsky W, King BA. JAK inhibitors in dermatology: the promise of a new drug class. J Am Acad. Dermatol. 2017;76:736-44.
10. Yu Z, Wang L, Quan M, *et al*. Successful management with Janus kinase inhibitor tofacitinib in refractory juvenile dermatomyositis: A pilot study and literature review. Rheumatology (Oxford). 2021;60:1700-7.
11. Papadopoulou C, Hong Y, Omoyinmi E, *et al*. Janus kinase 1/2 inhibition with baricitinib in the treatment of juvenile dermatomyositis. Brain, 2019, 142.
12. Ding Y, Huang B, Wang Y, *et al*. Janus kinase inhibitor significantly improved rash and muscle strength in

juvenile dermatomyositis. Ann Rheum Dis. 2021;80:543-545.
13. Sabbagh S, De Jesus AA, Hwang SJ, *et al*. Treatment of anti-MDA5 autoantibody-positive juvenile dermatomyositis using tofacitinib. Brain, 2019, 142.
14. Almutairi N, Nour TM, Hussain NH. Janus kinase inhibitors for the treatment of severe *alopecia Areata*: An open-label comparative study. Dermatology. 2019;235(2):130-136.
15. Liu LY, Craiglow BG, Dai F, King BA. Tofacitinib for the treatment of severe *alopecia Areata* and variants: A study of 90 patients. J Am Acad. Dermatol. 2017;76:22-28.
16. Bayart CB, DeNiro KL, Brichta L, *et al*. Topical Janus kinase inhibitors for the treatment of pediatric *alopecia Areata*. J Am Acad Dermatol. 2017;77:167-170.
17. Dai YX, Chen CC. Tofacitinib therapy for children with severe *alopecia Areata*. J Am Acad. Dermatol. 2019;80:1164-1166.
18. Berbert Ferreira S, Berbert Ferreira R, Neves Neto AC, Assef SMC, Scheinberg M. Topical Tofacitinib: A Janus Kinase Inhibitor for the Treatment of Vitiligo in an Adolescent Patient. Case Rep. Dermatol. 2021;13(1):190-194.
19. Rothstein B, Joshipura D, Saraiya A, Abdat R, Ashkar H, Turkowski Y, *et al*. Treatment of vitiligo with the topical Janus kinase inhibitor ruxolitinib. J Am Acad Dermatol. 2017;76(6):1054-1060.
20. Olamiju B, Craiglow BG. Tofacitinib cream plus narrowband ultraviolet B phototherapy for segmental vitiligo in a child. Pediatr Dermatol. 2020;37:754-755.
21. Tian F, Chen Z, Xu T. Efficacy and safety of tofacitinib for the treatment of chronic plaque psoriasis: A systematic review and meta-analysis. J Int. Med Res. 2019 Jun;47(6):2342-23450.
22. Ortiz-Ibáñez K, Alsina MM, Muñoz-Santos C. Tofacitinib and other kinase inhibitors in the treatment of psoriasis. Actas Dermosifiliogr. 2013;104:304-10.
23. Almutairi N, Nour T. Tofacitinib in pediatric psoriasis: an open-label trial to study its safety and efficacy in children. Dermatology. 2020;236:191-8.
24. Hu WT, Lin FQ, Xu AE. Treatment of halo naevus with the topical Janus kinase inhibitor tofacitinib 1.5%. Clin Exp Dermatol. 2018;43:935-7.
25. Berbert Ferreira S, Berbert Ferreira R, Scheinberg MA. Atopic dermatitis: Tofacitinib, an option for refractory disease. Clin Case Rep. 2020 Dec;8(12):3243-6.
26. Gavegnano C, Detorio M, Montero C, *et al*. Ruxolitinib and tofacitinib are potent and selective inhibitors of HIV-1 replication and virus reactivation in vitro. Antimicrob Agents Chemother. 2014;58:1977-86.
27. Ziegler CGK, Allon SJ, Nyquist SK, *et al*. SARS-CoV-2 receptor ace2 is an interferon-stimulated gene in human airway epithelial cells and is detected in specific cell subsets across tissues. Cell. 2020;181:1016-35.e19.
28. Pillaiyar T, Laufer S. Kinases as potential therapeutic targets for anti-coronaviral therapy. J Med Chem. 2022;65:955-82.
29. Kochar B, Herfarth N, Mamie C, *et al*. Tofacitinib for the treatment of pyoderma gangrenosum. Clin Gastroenterol Hepatol. 2019;17:991-3.
30. Kim SR, Charos A, Damsky W, *et al*. Treatment of generalized deep morphea and eosinophilic fasciitis with the Janus kinase inhibitor tofacitinib. JAAD Case Rep. 2018;4:443-5.
31. Yang CC, Khanna T, Sallee B, *et al*. Tofacitinib for the treatment of lichen planopilaris: a case series. Dermatol Ther. 2018;31:e12656.
32. 32. Li C, Li Z, Cao Y, *et al*. Tofacitinib for the treatment of nail lesions and palmoplantar pustulosis in synovitis, acne, pustulosis, hyperostosis, and osteitis syndrome. JAMA Dermatol. 2021;157:74-8.

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