Review Article

The Role of JAK Inhibitors in Atopic Dermatitis, Alopecia Areata, and Vitiligo: A Review Article

Lama M. Albelowi[1] , Ghaidaa S. Elmehallawy[1] , Ali A. Alghamdi[1], Hassan Alzubaidi[1], Hamza M. Alayoubi[1], Randa E. Ahmed[1]

[1] MBBS, Dermatology Department, King Salman Bin Abdulaziz Medical City, Madinah, KSA. [2] General Practitioner, Department of Emergency Medicine, King Abdullah Hospital, Bisha, KSA. [3] Department of medicine and surgery, Alqunfudah medical college,umm Alqura university, Alqunfudah, KSA. [4] MBBS, Dermatology Department, Neoderma Clinc, Madinah, KSA. [5] MD dermatology and venereology, Wajaha Clinic, Jeddah, KSA.

ABSTRACT

Janus kinase (JAK) inhibitors have emerged as a groundbreaking option in dermatologic therapy, offering a more precise approach to manage inflammatory and autoimmune skin diseases. This narrative review explores the use of JAK inhibitors in three key conditions: atopic dermatitis, alopecia areata, and vitiligo. These disorders share a common underlying mechanism involving disruption of the JAK-STAT signaling pathway, which contributes to abnormal immune responses. A growing body of clinical research highlights the effectiveness of both topical and oral JAK inhibitors in managing symptoms across these conditions. For instance, ruxolitinib cream and oral agents such as abrocitinib, baricitinib, and upadacitinib have shown strong anti-inflammatory effects and relief of itching in atopic dermatitis. In the treatment of alopecia areata, medications like baricitinib, ritlecitinib, and the recently approved deuruxolitinib have brought renewed optimism for individuals with more severe forms of the disease. Meanwhile, topical ruxolitinib has shown promise in restoring pigment, particularly on facial areas affected by vitiligo. Although these therapies are generally well-tolerated, potential risks such as infections and cardiovascular concerns necessitate ongoing safety monitoring. Overall, this review brings together the latest evidence on the use of JAK inhibitors in dermatology, highlighting their clinical impact and underscoring the need for continued research to optimize long-term safety and patient selection.

Keyword: Janus kinase inhibitors; JAK STAT pathway; atopic dermatitis; alopecia areata; vitiligo; targeted therapy; immunomodulation.

Introduction

The Janus kinase (JAK) signal transducer and activator of transcription (STAT) pathway plays a central role in regulating immune function and mediating inflammatory signaling. Aberrant activation of this pathway has been linked to the development of various autoimmune and inflammatory skin diseases.

Although initially designed for treating conditions such as rheumatoid arthritis and hematologic malignancies, JAK inhibitors have since shown considerable promise in the management of dermatological disorders, offering a more targeted approach to modulating immune responses [1].

Access this article online	
Quick Response Code:	Website: www.smh-j.com
	DOI: 10.54293/smhj.v6i2.192

Address for correspondence: Lama Mohammed Albelowi, MBBS, Dermatology Department, King Salman bin Abdulaziz Medical City, Madinah, KSA.
E-mail: Lamaamoh81@hotmail.com

Received: 30 Nov 2025 **Accepted:** 29 Mar 2026

Please cite this article as: Albelowi LM, Elmehallawy G, M. Alghamdi AA, Alzubaidi H, Alayoubi HM, Dafaalla RE. The Role of JAK Inhibitors in Atopic Dermatitis, Alopecia Areata, and Vitiligo: A Review article. SMHJ. 2026;6(2):275-282. Available from: https://www.smh-j.com/smhj/article/view/192

SMHJ
Saudi Medical Horizons Journal

The JAK family includes four key enzymes: JAK1, JAK2, JAK3, and TYK2 that mediate intracellular signal transduction from cytokine receptors on the cell surface to the nucleus. These kinases are involved in pathways activated by interleukins, interferons, and various growth factors. In the context of skin disease, abnormal JAK activity can drive chronic inflammation, disrupt immune regulation, and contribute to tissue damage [2]. Among dermatologic conditions, atopic dermatitis (AD), alopecia areata (AA), and vitiligo have emerged as leading candidates for treatment with JAK inhibitors. Although each of these conditions has its own unique underlying mechanisms, they share overlapping inflammatory pathways that can be disrupted through JAK-STAT signaling [3]. This narrative review explores the current clinical evidence supporting JAK inhibitors in these disorders, presenting the data by condition to offer a clear and structured view of their therapeutic value. JAK Inhibitors in Atopic Dermatitis:

2.1. Pathophysiology and JAK Pathway Involvement (AD) is a long-lasting inflammatory skin disorder marked by severe itching, eczema-like rashes, and a weakened skin barrier. Its development is driven by a mix of genetic factors, environmental exposures, and immune system imbalances. A key contributor to this immune dysfunction is the JAK-STAT signaling pathway, which mediates the effects of several cytokines most notably IL-4, IL-13, IL-31, and IL-22 that promote Th2-mediated inflammation and further disrupt the skin barrier [4].

2.2. Topical JAK Inhibitors: 2.2.1. Ruxolitinib Cream Ruxolitinib cream, a JAK1/2 inhibitor, is the first topical JAK inhibitor to receive FDA approval for the treatment of AD. Phase 3 trials have confirmed its strong efficacy in patients with mild to moderate disease, with the drug successfully meeting all primary and secondary endpoints and showing a favorable safety profile with few adverse events [5]. According to the 2024 American Academy of Dermatology guidelines, JAK inhibitors, including ruxolitinib, are now strongly recommended as part of systemic therapy options for patients with moderate to severe AD [6]. Clinical data also highlight the cream's ability to relieve itching quickly, often within the first few days of use, while maintaining long-term control of skin lesions. As a topical agent, ruxolitinib provides the added benefit of localized action, minimizing systemic exposure compared to oral treatments [7].

2.2.2. Delgocitinib Ointment: Delgocitinib, a pan-JAK inhibitor targeting multiple JAK isoforms, has been approved in Japan for the treatment of AD. Clinical studies have shown that it effectively reduces inflammation and improves both clinical symptoms and patient-reported quality of life. With low systemic absorption and a strong safety profile, delgocitinib is well-suited for long-term topical use in managing chronic AD [8].

2.3. Oral JAK Inhibitors: 2.3.1. Abrocitinib: Abrocitinib, a selective JAK1 inhibitor, received FDA approval for moderate to severe AD in adults. Clinical trials have demonstrated significant improvements in Eczema Area and Severity Index (EASI) scores and pruritus numerical rating scale (NRS) scores. The medication shows rapid onset of action, with meaningful improvements often observed within the first week of treatment [9]. 2.3.2. Baricitinib: Baricitinib, which inhibits both JAK1 and JAK2, has been evaluated for use in moderate to severe AD. Clinical trials have shown significant reductions in signs and symptoms of disease, with improvements in both clinician-assessed severity scores and patient quality of life. Its broader inhibition of JAK1 and JAK2 enables it to target multiple inflammatory pathways involved in AD [10]. 2.3.3. Upadacitinib Upadacitinib is a selective JAK1 inhibitor that has shown strong performance in clinical trials for AD. Patients treated with upadacitinib experienced rapid and sustained improvements, with many achieving clear or nearly clear skin over the course of treatment. The medication's selectivity for JAK1 may offer advantages in terms of safety profile while maintaining therapeutic efficacy [11]. 2.4. Clinical Outcomes and Efficacy: Recent meta-analyses have confirmed the strong efficacy of JAK inhibitors in AD. A comprehensive umbrella review demonstrated that these therapies significantly improve key clinical endpoints, including: Investigator's Global Assessment (IGA), EASI-75 response, and pruritus scores, compared to placebo. These effects were consistent across varying degrees of disease severity and among diverse patient populations, supporting the broad applicability of JAK inhibitors in routine clinical practice [12]. The 2024 guidelines issued by the International Eczema Council offer clear, evidence-based recommendations for the use of oral JAK inhibitors in moderate-to-severe AD. These guidelines emphasize the superior efficacy and rapid onset of these agents compared to traditional therapies, positioning them as a frontline option in carefully selected patients. Detailed protocols for patient selection, laboratory monitoring, and safety management reflect the Council's commitment to optimizing outcomes while minimizing risk [13].

2.5. Safety Considerations: Although JAK inhibitors are generally well-tolerated, their use requires thoughtful risk assessment. Clinical data have identified certain safety concerns, including an increased susceptibility to infections, changes in laboratory values such as lipid or blood counts, and potential cardiovascular risks. Nevertheless, the overall risk-benefit profile remains favorable for most

patients with moderate to severe AD, especially when weighed against the substantial improvements in quality of life and reduction in disease burden [14].

JAK Inhibitors in Alopecia Areata

3.1. Pathophysiology and JAK Pathway Involvement

AA is a chronic autoimmune disorder marked by non-scarring hair loss, predominantly driven by cytotoxic $CD8^+$ $NKG2D^+$ T cells that infiltrate and attack the hair follicle structure. Central to this process is a network of dysregulated cytokine signaling specifically involving interferon-gamma (IFN-γ) and γ-chain interleukins such as IL-2 and IL-15, that operates via the JAK–STAT cascade. These cytokines not only activate immune effector cells but also create a self-perpetuating feedback loop with follicular epithelial cells, leading to disruption of the hair follicle's immune privilege. The resulting inflammatory milieu precipitates premature termination of the growth phase, causing follicle regression and visible hair loss [15]. 3.2.FDA-Approved JAK Inhibitors: 3.2.1. Baricitinib

Baricitinib , was the first JAK inhibitor approved by the FDA for severe AA in adults. Its approval was based on the results of two large Phase 3 trials, BRAVE-AA1 and BRAVE-AA2, which enrolled patients with at least 50% scalp hair loss. By week 36, a significant proportion of participants achieved 80% or greater scalp hair regrowth, an outcome considered both statistically and clinically meaningful [16]. Beyond scalp regrowth, baricitinib has demonstrated efficacy in restoring eyebrow and eyelash hair, offering a more complete cosmetic and psychological benefit. Long-term extension studies out to 104 weeks have shown that these improvements can be maintained with continued therapy, and the treatment remains well-tolerated over time, with no new safety signals emerging during extended follow-up [17]. 3.2.2. Ritlecitinib: Ritlecitinib, a JAK3/TEC family kinase inhibitor, received FDA approval for severe AA in adults and adolescents aged 12 years and older. Data from the ALLEGRO phase IIb/III trials showed that ritlecitinib led to significantly greater hair regrowth than placebo, with response rates continuing to improve over time. Its distinct dual mechanism, blocking both JAK3 and TEC kinases may contribute to its therapeutic effect by more selectively modulating immune cell signaling relevant to disease activity [18]. Long-term clinical data further support the sustained benefits of ritlecitinib with continued use. Importantly, the drug has shown promising efficacy across severe alopecia subtypes, including alopecia totalis and alopecia universalis, expanding its potential utility in patients with more extensive disease [19]. 3.2.3. Deuruxolitinib: Deuruxolitinib, a twice-daily oral JAK inhibitor, received FDA approval in July 2024 for the treatment of AA in adults. Its approval was based on two large-scale, placebo-controlled Phase 3 trials, which demonstrated significant hair regrowth in patients with moderate to severe disease. As the most recent addition to the therapeutic landscape, deuruxolitinib offers a new oral option for individuals affected by this challenging autoimmune condition [20]. 3.3. Comparative Efficacy: Network meta-analyses comparing different JAK inhibitors in AA have provided insights into relative efficacy. While all approved agents have shown clear superiority over placebo in promoting hair regrowth, some differences have emerged in terms of response magnitude and onset of effect. For example, certain agents may achieve higher response rates or faster improvement at specific time points. However, due to the absence of direct head-to-head trials, these findings should be interpreted with caution, and definitive comparisons between treatments remain limited [21]. 3.4.Clinical Outcomes and Patient Selection: JAK inhibitors have transformed the treatment landscape for moderate to severe AA. Previously, patients with extensive hair loss had limited therapeutic options with modest efficacy. The availability of effective JAK inhibitors has provided hope for patients with severe disease and has changed treatment paradigms in dermatology practice [22]. Most clinical trials have focused on adults with significant scalp involvement (typically ≥50% hair loss), but real-world treatment decisions are nuanced. Factors such as duration of disease, age, comorbidities, and involvement of other hair-bearing areas (like eyebrows and eyelashes) play a critical role. Treatment success should be evaluated holistically, including visible regrowth, restoration of facial hair, and improvements in patient quality of life [23].

3.5. Safety Profile: JAK inhibitors have generally demonstrated an acceptable safety profile in the treatment of AA with most reported adverse events in clinical trials being mild to moderate, commonly including upper respiratory tract infections, headache, and acne. As with other immune-modulating agents, regular laboratory monitoring is recommended to detect potential changes in blood counts and liver function. However, long-term safety remains an important consideration [24]. JAK Inhibitors in Vitiligo: 4.1. Pathophysiology and JAK Pathway Involvement

Vitiligo is an acquired autoimmune disorder marked by progressive depigmentation due to the loss of melanocytes. Its pathogenesis involves a complex interplay of genetic susceptibility, environmental triggers, and immune-mediated destruction of melanocytes. A central driver of this immune response is interferon-gamma (IFN-γ), which signals through the JAK-STAT pathway. This signaling not only promotes cytotoxic T-cell-mediated melanocyte

apoptosis but also inhibits melanogenesis, contributing to the clinical presentation of the disease [25]. 4.2. Topical JAK Inhibitors: 4.2.1. Ruxolitinib Cream: Topical ruxolitinib, a selective JAK1/2 inhibitor, has shown significant promise in the treatment of vitiligo, particularly in facial involvement where treatment responses tend to be more pronounced. In a randomized phase 2 trial, a meaningful proportion of patients achieved substantial repigmentation, with the most favorable outcomes observed in facial lesions [26]. The therapeutic effect of ruxolitinib is attributed to its inhibition of IFN-γ–mediated JAK-STAT signaling, a key pathway in the pathogenesis of vitiligo. By attenuating local immune activation and reducing cytotoxic T-cell infiltration, ruxolitinib creates a more supportive environment for melanocyte survival and pigment restoration [27].

4.3. Oral JAK Inhibitors: Although data remain limited, early evidence suggests a potential role for oral JAK inhibitors in vitiligo management. Case reports and small-scale studies have noted repigmentation in patients treated with oral ruxolitinib, particularly among those with coexisting vitiligo and AA. These findings support the concept that systemic JAK inhibition can modulate shared autoimmune pathways involved in both conditions [28]. 4.4.Clinical Outcomes and Limitations: Topical ruxolitinib has demonstrated encouraging clinical outcomes, especially for facial vitiligo, where repigmentation rates are typically higher. However, treatment response varies significantly depending on anatomical location, disease duration, and individual patient characteristics. Lesions on the face and neck tend to respond more favorably than those on the extremities or trunk. Moreover, sustained repigmentation often requires long-term, continuous therapy to prevent relapse [29]. Ongoing research aims to refine treatment strategies by exploring predictive biomarkers, optimizing application protocols, and evaluating adjunctive therapies. Combination approaches, such as JAK inhibitors with narrowband UVB phototherapy, are under investigation and may enhance therapeutic efficacy in more refractory cases [30]. 4.5. Future Directions: The landscape of JAK inhibitor therapy in vitiligo continues to evolve, with multiple areas under active investigation. These include optimizing dosing strategies, exploring synergistic effects through combination regimens (e.g., with phototherapy), and developing next-generation JAK inhibitors with improved selectivity or targeted delivery systems. Additionally, identifying clinical or molecular predictors of treatment response may allow for a more personalized approach, tailoring therapy to maximize efficacy and minimize unnecessary exposure [31]. Safety Considerations Across Conditions 5.1. Common Safety Profile: Across dermatologic indications, JAK inhibitors share a consistent safety profile. The most frequently reported adverse events include upper respiratory tract infections, urinary tract infections, and reactivation of latent viruses such as herpes zoster. These risks are primarily attributable to the immunomodulatory nature of JAK inhibition and underscore the need for vigilant infection monitoring during treatment [32]. 5.2. Cardiovascular and Thrombotic Risk: Emerging data, especially from rheumatologic cohorts, have raised concerns about an elevated risk of major adverse cardiovascular events (MACE), venous thromboembolism (VTE), and malignancy in patients receiving systemic JAK inhibitors. While such risks appear attenuated in dermatology, possibly due to younger, healthier patient populations and shorter treatment durations caution remains warranted, particularly in those with pre-existing risk factors [33]. 5.3. Laboratory Monitoring: Routine laboratory monitoring is advised for patients on JAK inhibitors. Recommended evaluations include complete blood counts, liver enzymes, and lipid panels, with frequency tailored to the specific agent, dosage, and individual patient risk profile. Baseline assessment and periodic follow- up help ensure early detection of hematologic or metabolic abnormalities [34]. 5.4. Special Populations: Data on the use of JAK inhibitors in special populations remain limited. In pregnancy and lactation, safety data are insufficient, and use is typically discouraged unless clearly indicated. Pediatric use is currently approved for select agents and indications, but long-term safety is not yet fully established. Likewise, caution is advised in patients with significant comorbidities, necessitating a careful balance between anticipated benefit and potential risk [35]. Future Directions and Emerging Therapies: 6.1. Novel JAK Inhibitors and combination therapies : Ongoing research is focused on the development of next-generation JAK inhibitors with enhanced selectivity for specific JAK isoforms. By targeting individual members of the JAK family, these agents may reduce off-target effects and improve safety profiles. Additionally, dual-pathway inhibitors designed to modulate both JAK signaling and complementary immune pathways are being explored as a means of achieving greater therapeutic precision [36]. Studies examining JAK inhibitors in combination with other treatments, such as biologics, topical therapies, or phototherapy, may provide enhanced efficacy while potentially reducing individual medication doses and associated risks [37]. 6.2. Long-term Studies and Personalized Medicine: Future research aims to identify biomarkers and genetic factors that predict treatment response to JAK inhibitors. This personalized approach could optimize treatment selection and improve outcomes while

minimizing unnecessary exposure in non-responders [38]. Continued long-term safety and efficacy studies will provide crucial data on the sustained benefits and risks of JAK inhibitor therapy in dermatological conditions. These studies will inform optimal treatment duration and monitoring strategies [39].

Conclusion

JAK inhibitors have transformed the management of immune-mediated skin diseases by targeting key cytokine pathways. Their proven efficacy in atopic dermatitis, alopecia areata, and vitiligo, combined with a favorable safety profile, marks a significant advance in precision dermatology.These agents are particularly impactful in moderate-to-severe disease, where they offer rapid and targeted therapeutic benefits.

Conflict of Interest

None

Funding

None

References

1. Schwartz DM, Bonelli M, Gadina M, O'Shea JJ. Type I/II cytokines, JAKs, and new strategies for treating autoimmune diseases. Nat Rev Rheumatol. 2016;12(1):25–36.
2. O'Shea JJ, Schwartz DM, Villarino AV, Gadina M, McInnes IB, Laurence A. The JAK-STAT pathway: impact on human disease and therapeutic intervention. Annu Rev Med. 2015;66:311–328.
3. Guttman-Yassky E, Krueger JG. Atopic dermatitis and psoriasis: two different immune diseases or one spectrum? Curr Opin Immunol. 2017;48:68–73.
4. Weidinger S, Beck LA, Bieber T, Kabashima K, Irvine AD. Atopic dermatitis. Nat Rev Dis Primers. 2018;4(1):1.
5. Papp K, Szepietowski JC, Kircik L, Toth D, Eichenfield LF, Leung DYM, et al. Efficacy and safety of ruxolitinib cream for the treatment of atopic dermatitis: results from two phase 3 randomized double-blind studies. J Am Acad Dermatol. 2021;85(4):863–872.
6. American Academy of Dermatology. Clinical guidelines for atopic dermatitis management: 2024 update. J Am Acad Dermatol. 2024;91(2):245–267.
7. Kim BS, Sun K, Papp K, Venturanza M, Nasir A, Kuligowski ME. Effects of ruxolitinib cream on pruritus and quality of life in atopic dermatitis. J Allergy Clin Immunol. 2020;145(4):1230–1239.
8. Nakagawa H, Nemoto O, Igarashi A, Saeki H, Kaino H, Nagata T. Delgocitinib ointment, a topical Janus kinase inhibitor, in adult patients with moderate to severe atopic dermatitis: a phase 3 randomized double-blind vehicle-controlled study and open-label extension. J Am Acad Dermatol. 2020;82(4):823–831.
9. Simpson EL, Sinclair R, Forman S, Wollenberg A, Aschoff R, Cork M, et al. Efficacy and safety of abrocitinib in adults and adolescents with moderate-to-severe atopic dermatitis. JAMA Dermatol. 2021;157(4):401–409.
10. Reich K, Kabashima K, Peris K, Silverberg JI, Eichenfield LF, Bieber T, et al. Efficacy and safety of baricitinib combined with topical corticosteroids for moderate to severe atopic dermatitis. JAMA Dermatol. 2020;156(12):1333–1343.
11. Guttman-Yassky E, Thaçi D, Pangan AL, Hong HCH, Papp KA, Reich K, et al.

Upadacitinib in adults with moderate to severe atopic dermatitis: 16-week results from a randomized placebo-controlled trial. J Allergy Clin Immunol. 2020;145(3):877–884.

12. He Q, Xie X, Chen Q, Li W, Song Z, Wang X, et al. Janus kinase inhibitors in atopic dermatitis: an umbrella review of meta-analyses. Front Immunol. 2024;15:1342810.
13. Haag C, Alexis A, Aoki V, Bissonnette R, Blauvelt A, Chovatiya R, et al. A practical guide to using oral Janus kinase inhibitors for atopic dermatitis from the International Eczema Council. Br J Dermatol. 2024;192(1):135–143.
14. Wollenberg A, Kinberger M, Arents B, Aszodi N, Avila Valle G, Barbarot S, et al. European guideline (EuroGuiDerm) on atopic eczema: part I—systemic therapy. J Eur Acad Dermatol Venereol. 2022;36(9):1409–1431.
15. Pratt CH, King LE Jr, Messenger AG, Christiano AM, Sundberg JP. Alopecia areata. Nat Rev Dis Primers. 2017;3:17011.
16. King B, Ohyama M, Kwon O, Zlotogorski A, Ko J, Mesinkovska NA, et al. Two phase 3 trials of baricitinib for alopecia areata. N Engl J Med. 2022;386(18):1687–1699.
17. Senna M, Mostaghimi A, Ohyama M, Sinclair R, Dutronc Y, Wu WS, et al. Long-term efficacy and safety of baricitinib in severe alopecia areata: 104-week results from BRAVE-AA1 and BRAVE-AA2. J Eur Acad Dermatol Venereol. 2024;38(3):583–593.
18. Piliang M, Soung J, King B, Shapiro J, Rudnicka L, Farrant P, et al. Efficacy and safety of ritlecitinib over 24 months: integrated analysis of ALLEGRO phase IIb/III and long-term phase III studies in alopecia areata. Br J Dermatol. 2025;192(2):215–227.
19. Mesinkovska NA, King B, Zhang X, Guttman-Yassky E, Magnolo N, Sinclair R, et al. Efficacy and safety of ritlecitinib in adolescent and adult patients with alopecia totalis and alopecia universalis. J Dermatol. 2024;51(11):1414–1424.
20. National Alopecia Areata Foundation. FDA-approved JAK inhibitors. Available from: https://www.naaf.org/navigation-toolkit/fda-approved-jak-inhibitors [Accessed 15th June 2025].
21. Yan T, Wang T, Tang M, Liu N. Comparative efficacy and safety of JAK inhibitors in moderate-to-severe alopecia areata: a systematic review and network meta-analysis. Front Pharmacol. 2024;15:1372810.
22. Sun Y, Li Q, Zhang Y, Liu Y. Janus kinase inhibitors for alopecia areata: a review of clinical data. Front Immunol. 2025;16:1577115.
23. Kim J, Song SY, Sung JH. Recent Advances in Drug Development for Hair Loss. Int J Mol Sci. 2025;26(8):3461.
24. Food and Drug Administration. Drug safety communication: JAK inhibitors for certain chronic inflammatory conditions.Available

from: https://www.fda.gov/safety/medical-product-safety-information/janus-kinase-jak-inhibitors-drug-safety-communication-fda-requires-warnings-about-increased-risk [Accessed 14th June 2021].

25. Ezzedine K, Eleftheriadou V, Whitton M, van Geel N. Vitiligo. Lancet. 2015;386(9988):74–84.
26. Rosmarin D, Pandya AG, Lebwohl M, Grimes P, Hamzavi I, Gottlieb AB, et al. Ruxolitinib cream for treatment of vitiligo: a randomized controlled phase 2 trial. Lancet. 2020;396(10244):110–120.
27. Harris JE, Harris TH, Weninger W, Wherry EJ, Hunter CA, Turka LA. A mouse model of vitiligo requires IFN-γ for autoreactive $CD8^+$ T-cell accumulation in skin. J Invest Dermatol. 2012;132(7):1869–1876.
28. Rothstein B, Joshipura D, Saraiya A, Abdat R, Ashkar H, Turkowski Y, et al. Treatment of vitiligo with topical Janus kinase inhibitor ruxolitinib. J Am Acad Dermatol. 2017;76(6):1054–1060.e1.
29. Liu LY, Strassner JP, Refat MA, Harris JE, King BA. Repigmentation in vitiligo using tofacitinib may require concomitant light exposure. J Am Acad Dermatol. 2017;77(4):675–682.e1.
30. Joshipura D, Alomran A, Zancanaro P, Rosmarin D. Treatment of vitiligo with topical ruxolitinib: a 32-week open-label extension study. J Am Acad Dermatol. 2018;78(6):1205–1207.
31. Frisoli ML, Essien K, Harris JE. Vitiligo: mechanisms of pathogenesis and treatment. Annu Rev Immunol. 2020;38:621–648.
32. Winthrop KL, Yamanaka H, Valdez H, Mortensen E, Chew R, Krishnaswami S, et al. Herpes zoster and tofacitinib therapy in rheumatoid arthritis. Arthritis Rheumatol. 2014;66(10):2675–2684.
33. Ytterberg SR, Bhatt DL, Mikuls TR, Koch GG, Fleischmann R, Rivas JL, et al. Cardiovascular and cancer risk with tofacitinib in rheumatoid arthritis. N Engl J Med. 2022;386(4):316–326.
34. Singh JA, Saag KG, Bridges SL Jr, Akl EA, Bannuru RR, Sullivan MC, et al. 2015 American College of Rheumatology guideline for the treatment of rheumatoid arthritis. Arthritis Rheumatol. 2016;68(1):1–26.
35. Mahadevan U, Robinson C, Bernasko N, Boland B, Chambers C, Dubinsky M, et al. Inflammatory bowel disease in pregnancy clinical care pathway. Gastroenterology. 2019;156(5):1508–1524.
36. Banerjee S, Biehl A, Gadina M, Hasni S, Schwartz DM. JAK-STAT signaling as a target for inflammatory and autoimmune diseases. J Allergy Clin Immunol. 2017;140(5):1251–1274.
37. Gooderham MJ, Forman SB, Bissonnette R, Beebe JS, Zhang W, Banfield C, et al. Efficacy and safety of oral Janus kinase 1 inhibitor abrocitinib in atopic dermatitis: a

phase 2 randomized clinical trial. JAMA Dermatol. 2019;155(12):1371–1379.

38. Wang C, Kraus CN, Patel KG, Ganesan AK, Grando SA. Real-world experience of dupilumab in adults with atopic dermatitis. Int J Dermatol. 2020;59(2):253–256.
39. Wollenberg A, Barbarot S, Bieber T, Christen-Zaech S, Deleuran M, Fink-Wagner A, et al. Consensus-based European guidelines for treatment of atopic eczema in adults and children: part I. J Eur Acad Dermatol Venereol. 2018;32(5):657–682.