Review

Janus Kinase (JAK) Inhibitors in Dermatology: An Update and Critical Review

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Abstract: **Background:** The integration of Janus kinase inhibitors into dermatologic practice has fundamentally transformed the management of complex, immune-mediated cutaneous diseases. Originally pioneered for rheumatologic and hematologic conditions, these targeted therapies have rapidly expanded into dermatology, offering unprecedented efficacy for conditions such as atopic dermatitis, alopecia areata, and vitiligo. However, the rapid proliferation of both topical and systemic Janus kinase inhibitors requires a careful synthesis of their evolving efficacy profiles, long-term safety data, and novel applications beyond their initial regulatory approvals. **Methods:** A comprehensive literature review was performed to capture the most recent advancements and critical evaluations in this domain. Studies published between 2024 and 2026 within MEDLINE, PubMed, and Ovid databases were systematically reviewed. The gathered literature was analyzed to extract data on drug efficacy, post-marketing safety, and emerging indications. All included studies were rigorously classified according to the Oxford Centre for Evidence-Based Medicine Levels of Evidence (March 2009) to appropriately stratify the current clinical landscape. **Results:** Fifty-eight contemporary articles met the inclusion criteria, encompassing a diverse array of systemic reviews, pharmacovigilance reports, and clinical observations. The strongest evidence supports JAK inhibitors in atopic dermatitis and alopecia areata, where randomized trials, meta-analyses, and real-world studies show rapid symptom control, high clinical response rates, and acceptable short- to medium-term safety. Evidence is also substantial for vitiligo, particularly topical therapy for facial disease, and for chronic hand eczema with topical delgocitinib as an effective steroid-sparing option. Moderate-to-lower level evidence suggests benefit in psoriatic arthritis, inflammatory nail disorders, granuloma annulare, lichen sclerosus, autoimmune bullous diseases, scarring alopecia, and other refractory inflammatory dermatoses. Safety signals across studies include acne, herpes zoster, mild infections, laboratory abnormalities, and the need for individualized cardiovascular, malignant, and reproductive risk assessment before treatment initiation and during longitudinal dermatologic monitoring in practice. **Conclusions:** Janus kinase inhibitors represent a paradigm shift in dermatologic therapeutics, offering highly effective, targeted interventions for refractory skin conditions. Nevertheless, their optimal utilization demands meticulous clinical judgment, balancing potent immunological blockade against comprehensive, long-term safety surveillance.

Keywords: janus kinase inhibitors; dermatology; alopecia areata; dermatitis; atopic; vitiligo; pharmacovigilance

1. Introduction

The therapeutic landscape of dermatology has undergone a profound revolution over the past decade, driven primarily by the elucidation of targeted molecular pathways and the subsequent development of highly specific pharmacologic interventions [1]. Among these breakthrough therapies, Janus kinase (JAK) inhibitors have emerged as a cornerstone in the management of complex, immune-mediated skin disorders [2]. By reversibly binding to the intracellular JAK family of tyrosine kinases, specifically JAK1, JAK2, JAK3, and tyrosine kinase 2 (TYK2), these agents disrupt the downstream signal transducer and activator of transcription (STAT) signaling cascade [3]. This interruption effectively downregulates the transcription of multiple pro-inflammatory cytokines integral to the pathogenesis of various dermatologic conditions, offering a distinct advantage over broad-spectrum immunosuppressants [4].

Initially approved for rheumatologic and hematologic indications, the application of JAK inhibitors within dermatology has expanded at an unprecedented rate. Conditions that previously relied on generalized systemic therapies with significant toxicity profiles are now being managed with precision [5]. The most prominent clinical successes have been observed in atopic dermatitis, where systemic and topical JAK inhibitors have demonstrated rapid pruritus relief and profound skin clearance, fundamentally altering patient expectations and standard-of-care guidelines [6]. Similarly, the management of adult vitiligo has been advanced by the introduction of approved topical JAK inhibitors, providing a targeted mechanism to halt depigmentation and promote repigmentation [7]. Furthermore, conditions characterized by intense localized inflammation, such as chronic hand eczema, have shown marked improvement with the advent of topical pan-JAK inhibitors like delgocitinib, which offer a therapeutic alternative where traditional corticosteroids have proven inadequate or deleterious over long-term use [8].

Despite these remarkable clinical benefits, the rapid integration of JAK inhibitors into routine dermatologic practice has necessitated a rigorous and ongoing evaluation of their safety profiles [9]. The immunosuppressive nature of these agents inherently carries risks, and real-world pharmacovigilance has become critical in identifying adverse events that may not be fully apparent during controlled clinical trials. Recent analyses of adverse event reporting systems have highlighted concerns ranging from local application site reactions with topical formulations [10] to more systemic issues such as infection risks, hematologic abnormalities, and paradoxical dermatologic reactions [11]. Additionally, the exploration of JAK inhibitors as a novel therapeutic approach for autoimmune bullous diseases indicates their potent immunomodulatory capacity, yet underscores the need for careful risk-benefit stratification in vulnerable patient populations [12]. Considerations extending to the impact of JAK inhibitors on diagnostic procedures, such as patch testing, further illustrate the complex clinical nuances these drugs introduce [13].

Alopecia areata represents another major frontier where JAK inhibition has provided life-altering therapeutic options for severe, recalcitrant cases. Narrative reviews and real-world experiences consistently report significant hair regrowth with oral therapies; however, these outcomes are accompanied by necessary discussions regarding long-term maintenance, relapse upon discontinuation, and systemic tolerability [14]. The post-marketing safety signals associated with treatments for alopecia areata demand continual scrutiny to ensure patient safety remains paramount [15]. Rare but severe adverse events, including the potential association with cutaneous lymphomas, require diligent pharmacovigilance and a high index of suspicion among prescribing physicians [16].

As the clinical applications of JAK inhibitors continue to diversify, encompassing off-label uses for generalized granuloma annulare, hidradenitis suppurativa, and other challenging dermatoses [5], the need for a comprehensive, critically appraised update becomes increasingly vital. The objective of this review is to systematically evaluate the literature concerning the application of JAK inhibitors in dermatology. By synthesizing current evidence regarding their efficacy, safety, and emerging therapeutic roles, this manuscript aims to provide clinicians with a robust framework for optimizing the use of these powerful immunomodulators in clinical practice, ensuring therapeutic efficacy while meticulously managing associated risks.

2. Methods

A comprehensive review of the current medical literature was conducted to evaluate the evolving role, efficacy, and safety of Janus kinase inhibitors in dermatology. Studies published between the years 2024 and 2026 within the MEDLINE, PubMed, and Ovid databases were systematically retrieved and reviewed. The inclusion criteria focused on articles addressing the dermatologic applications of topical and systemic JAK inhibitors, including clinical trials, real-world pharmacovigilance data, systematic reviews, and meta-analyses. Exclusion criteria involved studies lacking direct relevance to cutaneous diseases or those entirely focused on non-dermatologic primary endpoints without skin-related outcomes. A total of 58 articles were identified and selected for detailed analysis. To objectively assess the quality and reliability of the synthesized literature, all included studies were rigorously classified according to the Oxford Centre for Evidence-Based Medicine (CEBM) Levels of Evidence (March 2009) [17]. Data regarding study design, dermatologic indication, efficacy outcomes, reported adverse events, and primary limitations were extracted from each article to form the basis of the critical synthesis.

3. Results

The literature published in 2026 demonstrated a significant shift toward post-marketing safety surveillance, the exploration of broad off-label applications, and real-world efficacy studies across diverse dermatologic conditions.

Foggi et al. [18] reviewed the emerging applications of JAK inhibitors beyond their current FDA-approved labels in dermatology. The article systematically addressed the mechanistic rationale for employing these agents in recalcitrant conditions such as hidradenitis suppurativa, dermatomyositis, and rare inflammatory dermatoses. The authors concluded that while off-label use is expanding rapidly due to potent broad-spectrum cytokine blockade, clinical implementation remains heavily dependent on physician discretion and small-scale reports. This matters to dermatologists as it highlights

the versatility of JAK inhibitors while underscoring the pressing need for larger, controlled trials to formally establish efficacy and standardized dosing protocols in these orphan indications (Level 5).

Jalili et al. [19] provided a practical dermatologist's guide to mastering the use of systemic JAK inhibitors for moderate-to-severe atopic dermatitis. The authors detailed patient selection criteria, pre-treatment laboratory screening, and nuanced dose-titration strategies based on real-world clinical experience. The study concluded that optimal outcomes rely on individualized therapy, balancing rapid symptom control against the necessity for vigilant safety monitoring. This is highly relevant for clinical practice, offering a pragmatic framework for transitioning patients from standard systemic agents to targeted therapies. The primary limitation remains the narrative nature of the guidelines, which rely heavily on expert consensus rather than head-to-head randomized comparative data. (Level 5).

Słomianny et al. [20] conducted a comprehensive literature review focusing on the long-term safety profile of JAK inhibitors utilized in dermatology from 2020 to 2025. The synthesis evaluated extended clinical trial extensions and observational registries, concluding that the overall safety profile remains consistent over time, with infections and laboratory anomalies being the most frequent, yet generally manageable, adverse events. This matters profoundly as long-term safety data are crucial for chronic dermatologic management. However, the study is limited by the inherent heterogeneity of the underlying source data and the variability in reporting standards across different multinational registries and clinical trials (Level 2a).

Greco et al. [21] evaluated the management of adult vitiligo, comparing the use of the approved topical JAK inhibitor with traditional standard therapies. The review concluded that topical JAK inhibition provides superior and more targeted repigmentation, particularly on the face and neck, compared to conventional corticosteroids or calcineurin inhibitors. This finding validates the shift toward targeted topical therapy in vitiligo management, offering a safer long-term profile. The main limitation is that achieving significant repigmentation on acral surfaces remains a substantial challenge, indicating that topical JAK inhibitors, while revolutionary, are not a universal panacea for all anatomical areas affected by vitiligo (Level 5).

Termini et al. [22] updated the clinical evidence regarding pan-JAK inhibition via topical delgocitinib across chronic hand eczema and other cutaneous diseases. The authors analyzed phase III trial data demonstrating robust efficacy in reducing eczema severity and improving patient-reported quality of life metrics. The conclusion strongly supports delgocitinib as a highly effective, steroid-sparing alternative for chronic hand eczema. This is clinically vital given the frequent treatment resistance and skin atrophy associated with long-term topical steroid use. A notable limitation is the current lack of extensive post-marketing real-world data specifically regarding the durability of response upon discontinuation of the topical agent (Level 1a).

Zhou et al. [23] performed a real-world analysis of adverse events associated with ruxolitinib cream based on the FDA Adverse Event Reporting System (FAERS) from 2021 to 2024. The study identified application site reactions and mild systemic absorption concerns as the primary safety signals, concluding that the cream maintains a highly favorable safety profile consistent with its clinical trial data. This matters to dermatologists by reassuring them of the drug's safety in widespread daily practice. The limitation inherent to this study is the reliance on a spontaneous reporting database, which is subject to underreporting, missing clinical context, and the inability to establish definitive causation (Level 2c).

Olisova et al. [24] explored JAK inhibitors as a novel therapeutic approach for autoimmune bullous diseases. The review examined preliminary evidence suggesting that JAK/STAT pathway blockade can effectively reduce autoantibody production and limit inflammatory cell recruitment in pemphigus and pemphigoid. The authors concluded that JAK inhibitors hold significant promise as corticosteroid-sparing agents in these highly morbid conditions. This is therapeutically important for managing fragile patient populations where conventional immunosuppression carries immense risk. The primary limitation is the current reliance on case reports and small open-label series, necessitating robust randomized controlled trials to confirm these promising early observations (Level 4).

Gratz et al. [25] conducted a systematic review to determine the effect of Janus kinase inhibitors on patch testing outcomes. The analysis concluded that systemic JAK inhibitors possess the immunomodulatory potential to suppress delayed-type hypersensitivity reactions, thereby potentially yielding false-negative patch test results in patients with suspected allergic contact dermatitis. This finding is of critical importance to procedural dermatologists and allergists, dictating that these medications should ideally be paused prior to testing. The study's main limitation is the relatively small sample sizes of the included reports and a lack of standardized timing guidelines for drug cessation before diagnostic testing (Level 2a).

Ohyama et al. [26] presented a narrative review and real-world experience detailing the use of oral Janus kinase inhibitors for the treatment of severe alopecia areata. The authors reported substantial hair regrowth in a significant proportion of treated patients, concluding that these agents represent the most effective systemic therapy currently available for the condition. This deeply impacts dermatology by formalizing a viable treatment pathway for a historically intractable disease. The major limitation highlighted is the high rate of disease relapse following treatment cessation, indicating that indefinite maintenance therapy may be required to sustain clinical benefits (Level 5).

Zhan et al. [27] investigated post-marketing safety signals of four distinct JAK inhibitors specifically prescribed for alopecia areata using an indication-restricted FAERS pharmacovigilance approach. The study concluded that while major adverse cardiovascular events (MACE) and severe infections are rare, specific signals for acne, weight gain, and minor upper respiratory tract infections are statistically prominent. This is crucial for pre-treatment patient counseling and expectation management. The main limitation is the inability of the FAERS database to provide incidence rates due to the lack of an exact patient denominator, limiting the quantification of absolute risk (Level 2c).

Lu et al. [28] conducted a pharmacovigilance analysis of the FAERS database paired with a literature review to assess cutaneous lymphoma associated with JAK inhibitors. The authors found a weak but notable reporting signal linking long-term systemic JAK inhibition with primary cutaneous lymphomas, concluding that vigilant dermatologic screening is warranted in this patient cohort. This matters as it highlights a potential rare, severe complication requiring long-term monitoring. The limitation is the confounding factor of underlying severe immune dysregulation in these patients, making it difficult to firmly distinguish drug-induced malignancy from disease-driven malignant transformation (Level 2c).

Jaguan et al. [29] systematically reviewed the treatment of generalized granuloma annulare with JAK inhibitors. The synthesis of case reports and small series demonstrated rapid clinical clearance and symptomatic relief in patients resistant to conventional therapies. The authors concluded that targeted JAK blockade represents a highly effective, emerging therapeutic option for disseminated forms of the disease. This is important for offering hope in a notoriously stubborn dermatosis. However, the evidence remains limited by the absence of controlled clinical trials, high potential for publication bias favoring positive outcomes, and a lack of long-term follow-up data (Level 4).

Zhang et al. [30] reviewed the genetic pharmacoepidemiology of JAK inhibitors in chronic immune-mediated skin diseases. The article concluded that specific single nucleotide polymorphisms in the JAK/STAT pathway and cytochrome P450 enzymes can significantly alter drug metabolism, efficacy, and toxicity profiles. This matters greatly for the future of precision dermatology, suggesting that pharmacogenomic testing could optimize patient selection and dosing. The primary limitation is that such genetic markers are not yet validated for routine clinical use, and the current evidence remains largely theoretical and restricted to specialized research settings (Level 5).

Cui et al. [31] discussed the immunologic intricacies of targeting the JAK/STAT pathway in atopic dermatitis. The authors concluded that broad JAK inhibition efficiently suppresses the Th2 inflammatory axis, but also modulates Th1 and Th22 pathways, explaining both the robust clinical efficacy and the specific side effect profile seen in patients. This mechanistic understanding is essential for dermatologists when selecting agents based on specific patient endotypes. The main limitation is the complexity of translating broad immunologic theories into predictive clinical biomarkers for individual patient treatment responses (Level 5).

Tan et al. [32] performed a scoping review on the evidence for using Janus kinase inhibitors in the management of Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN). The study concluded that experimental and limited clinical data suggest JAK inhibitors can rapidly halt keratinocyte apoptosis by blocking key cytokine storm mediators. This represents a potentially groundbreaking, life-saving application in dermatology. The critical limitation is that the current evidence is exceedingly sparse, relying heavily on in vitro data and isolated clinical cases, precluding immediate widespread adoption in acute care settings (Level 4).

Solimani et al. [33] investigated how inhibition of the JAK/STAT signaling pathway provides a protective effect against acantholysis in pemphigus. The authors concluded that aside from suppressing systemic autoantibodies, JAK inhibitors exert a direct stabilizing effect on keratinocyte adhesion molecules. This dual mechanism matters significantly, offering a novel rationale for using these drugs to achieve rapid blistering control in pemphigus patients. The primary limitation is that much of the mechanistic data was derived from ex vivo tissue models, necessitating extensive in vivo clinical validation to confirm these localized epidermal effects (Level 5).

Burlando et al. [34] reviewed the evolution of chronic hand eczema management in the era of targeted topical JAK inhibition. The authors concluded that understanding the diverse inflammatory pathways involved in hand eczema has aligned perfectly with the pan-JAK blockade provided by delgocitinib, moving the condition from nosological ambiguity to a distinct therapeutic identity. This matters as it provides a clear, evidence-based treatment pathway for a highly disabling condition. The limitation is the narrative structure of the review, summarizing existing trial data without offering new quantitative meta-analytical insights (Level 5).

Zou et al. [35] presented preliminary observations from a single-arm clinical trial evaluating tofacitinib for the treatment of primary cutaneous amyloidosis. The study reported significant reductions in intractable pruritus and measurable flattening of amyloid papules in treated patients. The conclusion suggests that JAK inhibitors may disrupt the neuroimmune signaling driving the disease's vicious itch-scratch cycle. This is a vital finding for an orphan disease with virtually no effective therapies. The main limitation is the single-arm, unblinded design with a small patient cohort, requiring larger, placebo-controlled trials for validation (Level 4).

Quraishi et al. [36] conducted a systematic review and meta-analysis on the incidence of acne in patients treated with Janus kinase inhibitors. The analysis concluded that there is a statistically significant, dose-dependent increase in the incidence of new-onset or worsened acne across multiple JAK inhibitors, though typically mild and manageable. This is

important for dermatologists as it necessitates proactive acne management protocols when initiating therapy. The limitation lies in the subjective grading of acne severity across different trials, leading to potential inconsistencies in reporting the true burden of the adverse event (Level 1a).

Huang et al. [37] evaluated postmarketing adverse events comparing biologics and Janus kinase inhibitors in patients with atopic dermatitis using real-world data. The study concluded that while biologics primarily exhibit injection-site reactions and conjunctivitis, JAK inhibitors present higher relative risks for acne, herpes zoster, and laboratory abnormalities. This comparative data is crucial for shared clinical decision-making, allowing physicians to tailor treatments based on patient-specific risk tolerance. The limitation is the inherent bias in retrospective database queries, which cannot account for varying disease severities and patient baseline comorbidities (Level 2c).

Chang et al. [38] conducted a multicenter study assessing the efficacy of oral JAK inhibitors combined with a 308-nm excimer laser in pediatric progressive vitiligo. The authors concluded that the combination therapy synergistically halted disease progression and stimulated rapid repigmentation significantly faster than monotherapy. This is a major advancement in pediatric dermatology, offering a robust protocol for a psychologically distressing disease. The limitation is the lack of a standardized control group receiving only laser therapy, making it difficult to precisely isolate the independent effect size of the JAK inhibitor (Level 2b).

Ghafoor et al. [39] systematically reviewed the incidence of keratinocyte carcinomas reported in association with tyrosine kinase and Janus kinase inhibitors. The study concluded that while long-term immunosuppression theoretically increases skin cancer risk, the current data do not show a statistically significant spike in non-melanoma skin cancers linked specifically to JAK inhibitors over short-to-medium follow-up periods. This provides reassuring safety data for dermatologists prescribing these agents to older adults. The limitation is the relatively short duration of available follow-up, as cutaneous malignancies typically require decades to clinically manifest (Level 2a).

Altınöz Güney et al. [40] investigated the infection risk associated with Janus kinase inhibitors via a pharmacovigilance analysis using the FDA's FAERS database. The authors identified a clear signal for herpes zoster and mild respiratory tract infections, concluding that prophylactic measures, such as recombinant zoster vaccination, should be strongly considered prior to initiating therapy. This is highly relevant for establishing standard of care safety protocols in dermatology clinics. The limitation is the lack of clinical context in FAERS data, making it impossible to determine whether the infections were opportunistic or community-acquired (Level 2c).

Dai et al. [41] reviewed the developmental trends of Janus kinase inhibitors over three decades, charting their transition from common rheumatologic diseases to rare dermatologic conditions. The authors concluded that the refinement of molecule selectivity from first-generation pan-JAKs to second-generation highly specific inhibitors has maximized efficacy while minimizing off-target hematologic toxicities. This historical perspective is vital for understanding current therapeutic rationale. The limitation is that it functions as a broad historical review rather than providing new, actionable quantitative data on specific drug performance (Level 5).

Lin et al. [42] analyzed real-world safety concerning boxed warning outcomes for JAK inhibitors in a multinational cohort of patients with skin immune-mediated inflammatory diseases. The study concluded that the absolute incidence of MACE, venous thromboembolism (VTE), and severe malignancies is exceedingly low in standard dermatologic populations, compared to the older, higher-risk rheumatoid arthritis cohorts from which the warnings originated. This is practice-changing, as it alleviates undue hesitation in prescribing to young, healthy patients. The limitation is the retrospective cohort design, which may still suffer from unmeasured confounding variables (Level 2b).

Fang et al. [43] performed a systematic review and meta-analysis on the effectiveness and safety of oral JAK inhibitors in patients aged 12 and under with alopecia areata. The authors concluded that oral therapy is highly effective for severe pediatric alopecia, demonstrating significant hair regrowth with an acceptable short-term safety profile. This matters immensely, as severe alopecia in children causes profound psychological distress and standard therapies often fail. The primary limitation is the small pooled sample size and the critical lack of long-term safety data concerning pediatric growth and development (Level 1a).

The literature published in 2025 focused heavily on consolidating clinical guidelines, elucidating mechanisms of toxicity, and expanding the targeted use of topical formulations across various inflammatory states.

Rajput Khokhar et al. [44] reviewed the adverse effects of Janus kinase inhibitors with specific relevance for daily practice in dermatology. The authors provided a practical grading system for common side effects such as acne, hyperlipidemia, and mild infections, concluding that proactive monitoring and early intervention allow the majority of patients to continue therapy safely. This is an essential guide for clinician confidence and patient adherence. The study's limitation is its narrative reliance on aggregate trial data rather than providing a new independent statistical analysis of adverse event incidence (Level 5).

Shen et al. [45] conducted a systematic review exploring Janus kinase inhibitors for the treatment of lichen sclerosus. The review concluded that both topical and oral JAK inhibitors show emerging efficacy in reducing genital pruritus, induration, and disease progression in cases refractory to ultrapotent topical corticosteroids. This is highly relevant for dermatology and gynecology, providing a novel therapeutic target for a scarring, precancerous dermatosis. The primary

limitation is the very low quality of the primary evidence, consisting exclusively of isolated case reports and small, uncontrolled observational series (Level 4).

Lamba et al. [46] provided a comprehensive overview of delgocitinib as a newer topical Janus kinase inhibitor tailored for dermatologists. The authors highlighted its broad mechanism of action inhibiting all JAK family members, concluding that it holds unique potential for localized inflammatory diseases without the systemic absorption risks associated with oral counterparts. This matters for expanding the topical armamentarium beyond steroids and calcineurin inhibitors. The limitation is that the paper serves primarily as an educational overview rather than presenting novel clinical trial data or head-to-head comparisons (Level 5).

Haag et al. [47] published a practical guide from the International Eczema Council regarding the use of oral Janus kinase inhibitors for atopic dermatitis. The consensus concluded that while these drugs offer unparalleled speed of onset and itch relief, careful patient screening, particularly regarding cardiovascular and thrombotic risk factors, is mandatory. This document is a critical cornerstone for standardizing global dermatologic practice. The main limitation is that the guidelines are primarily based on expert consensus opinion and extrapolations from existing trial data, rather than prospective registry outcomes (Level 5).

Martin et al. [48] reviewed the use of Janus kinase inhibitors in the management of scarring alopecias, including lichen planopilaris and frontal fibrosing alopecia. The authors concluded that JAK inhibitors can successfully arrest disease progression and reduce perifollicular inflammation in recalcitrant cases, though regrowth of permanently destroyed follicles remains impossible. This provides a crucial targeted option for halting irreversible hair loss. The primary limitation is the lack of standardized dosing regimens and the reliance on retrospective chart reviews to ascertain clinical efficacy (Level 4).

Malekan et al. [49] evaluated JAK inhibitors as a promising therapy for immune-mediated photodermatoses such as polymorphic light eruption and actinic prurigo. The review concluded that inhibition of specific cytokine pathways can effectively prevent ultraviolet-induced inflammatory cascades, offering a new systemic alternative to antimalarials and phototherapy. This is a significant conceptual leap for managing severe light-sensitive dermatoses. The major limitation is that the evidence is currently theoretical and restricted to early-phase clinical observations, requiring large-scale trials to confirm both efficacy and safety (Level 5).

Sivadas et al. [50] published a succinct analysis aimed at understanding the underlying mechanisms of JAK inhibitor toxicity. The authors concluded that adverse events are not random but are directly linked to the specific downstream pathways blocked, such as erythropoietin signaling (anemia) or antiviral interferon pathways (herpes zoster). This mechanistic insight is vital for dermatologists to anticipate and logically manage specific side effects based on the prescribed molecule's selectivity. The limitation is the brevity of the commentary, which limits an in-depth statistical analysis of clinical toxicity rates (Level 5).

Honap et al. [51] conducted an international, multicenter, retrospective cohort study detailing JAK inhibitor-induced acne in patients with inflammatory bowel disease, which carries crossover relevance to dermatology. The authors concluded that acne is a frequent, mostly mild, class-effect complication that rarely requires drug discontinuation when managed promptly with standard topical acne therapies. This is highly relevant for dermatologists who often co-manage these cutaneous adverse events. The limitation is the retrospective nature of the study, which may underestimate the incidence of mild acne not formally documented in gastroenterology charts (Level 2b).

Haider et al. [52] reviewed the role of delgocitinib in dermatology, focusing specifically on its utility as a topical pan-JAK inhibitor for chronic hand eczema and atopic dermatitis. The authors concluded that the formulation provides highly effective, localized immune suppression with minimal systemic absorption, making it an ideal long-term maintenance therapy. This is practically significant for managing chronic barrier-defect dermatoses safely. The limitation is that it functions as a short narrative summary of existing data without providing new comparative insights against emerging biologic therapies (Level 5).

Su et al. [53] performed a meta-analysis of randomized clinical trials assessing the risk of serious infection with JAK inhibitors in immune-mediated inflammatory skin diseases. The study rigorously concluded that while the overall risk of opportunistic and serious systemic infections is slightly elevated compared to placebo, the absolute risk in standard dermatologic cohorts remains reassuringly low. This matters immensely for informed consent and risk stratification in clinical practice. The primary limitation is the relatively short duration of the included trials, which may not adequately capture long-term infectious complications (Level 1a).

Li et al. [54] described the development of biomimetic polydopamine loaded with a Janus kinase inhibitor for synergistic vitiligo therapy delivered via hydrogel microneedles. The authors concluded that this novel delivery system dramatically enhanced drug skin penetration and local retention, resulting in superior repigmentation in animal models compared to traditional topical creams. This is a crucial technological advancement for overcoming the thick stratum corneum barrier in dermatology. The limitation is that the data are strictly preclinical, requiring extensive human trials before clinical availability (Level 5).

Al-Mamoori et al. [55] presented a case series detailing Janus kinase inhibitors as a breakthrough treatment for refractory dissecting cellulitis of the scalp. The authors reported rapid reduction in suppuration, pain, and nodule formation in patients who had failed biologics and systemic antibiotics. The conclusion supports JAK inhibition as a highly effective salvage therapy for this scarring, debilitating condition. This is highly impactful for treating severe hidradenitis suppurativa-spectrum disorders. The limitation is the small number of cases and lack of a control group, preventing definitive efficacy conclusions (Level 4).

Heidari et al. [56] provided a clinical, narrative review of the emerging role of Janus kinase inhibitors in treating granuloma annulare. The authors concluded that systemic and topical JAK inhibitors efficiently clear extensive lesions by disrupting the macrophage-mediated inflammatory cascades central to the disease's pathogenesis. This matters because it offers a targeted mechanism for a disease historically reliant on broad, often ineffective therapies. The limitation is the reliance on aggregated case reports, leaving questions regarding optimal duration of therapy and long-term relapse rates unanswered (Level 4).

Wang et al. [57] analyzed the efficacy and safety of Janus kinase inhibitors in the treatment of psoriasis and psoriatic arthritis utilizing evidence from 2014 to 2022. The authors concluded that while highly effective for joint disease, systemic JAK inhibitors offer moderate, though generally less robust, skin clearance compared to advanced anti-IL-17 and anti-IL-23 biologics. This is vital for therapeutic positioning, suggesting JAKs are best reserved for patients with severe concomitant arthritis or those who fail biologics. The limitation is the historical data cutoff, missing the latest head-to-head comparative trials (Level 2a).

Iorizzo et al. [58] systematically reviewed clinical outcomes and therapeutic potential for JAK inhibitors in inflammatory nail disorders. The authors concluded that systemic and topical JAK therapy yields significant clinical improvement in isolated nail psoriasis, nail lichen planus, and severe alopecia areata-associated nail dystrophy. This represents a major breakthrough for nail unit conditions, which are notoriously difficult to penetrate and treat. The primary limitation is the scarcity of large-scale, dedicated randomized controlled trials utilizing validated nail severity scoring systems (Level 2a).

Packer et al. [59] explored the role of Janus kinase inhibitors for the treatment of cutaneous T-cell lymphomas (CTCL). The review concluded that targeted inhibition of the overactive JAK3/STAT3 pathway in malignant T-cells shows potent anti-tumor activity and rapid relief of intractable tumor-associated pruritus in early-phase studies. This is a highly significant emerging application in dermato-oncology. The critical limitation is that current evidence is restricted to preclinical models and very small phase I/II trials, requiring substantial further investigation regarding long-term survival outcomes (Level 5).

Stratman et al. [60] reported on the management of granuloma annulare using systemic Janus kinase inhibitors in a clinical case series. The authors concluded that oral tofacitinib and upadacitinib induce rapid remission of generalized lesions with high patient satisfaction and acceptable short-term safety. This is relevant as it adds valuable clinical weight to the growing consensus that JAK inhibitors are highly effective for this condition. The limitation lies in the retrospective nature of the report, the lack of standardized dosing regimens, and the absence of a control group (Level 4).

Tsiogkas et al. [61] conducted a systematic review and network meta-analysis of Janus kinase inhibitors for psoriatic arthritis. The authors concluded that upadacitinib and tofacitinib offer excellent joint efficacy and significant skin improvement, positioning them competitively against standard biologic therapies, particularly in patients preferring oral medications. This is important for dermatologists managing complex, multi-domain psoriatic disease. The limitation is the indirect nature of network meta-analyses, which mathematically compare trials with differing baseline patient characteristics and outcome measures (Level 1a).

Bieber et al. [62] reviewed the systemic therapy of atopic dermatitis utilizing Janus kinase inhibitors. The author concluded that these agents represent a paradigm shift due to their unprecedented speed of onset and profound impact on severe pruritus, effectively transforming the management of acute disease flares. This matters profoundly as it establishes JAK inhibitors as a first-line option for patients requiring rapid relief. The limitation is that it functions primarily as an expert narrative review, reiterating known trial results rather than providing new, independent epidemiological data (Level 5).

Sun et al. [63] provided a comprehensive review of clinical data regarding Janus kinase inhibitors for alopecia areata. The authors concluded that oral baricitinib, ritlecitinib, and upadacitinib have revolutionized care, demonstrating high rates of clinically meaningful hair regrowth, though emphasizing that continuous therapy is required to maintain results. This matters for managing patient expectations regarding the chronic nature of treatment. The major limitation remains the lack of clear predictive biomarkers to determine which specific patients are most likely to respond optimally to therapy (Level 5).

Gordon et al. [64] reviewed current evidence on the safety of Janus kinase inhibitors during pregnancy and lactation. The authors concluded that based on animal teratogenicity data and a lack of human safety trials, all systemic JAK inhibitors are strictly contraindicated during pregnancy and breastfeeding, requiring robust contraception counseling. This is a critical safety directive for dermatologists managing women of childbearing age. The limitation is the absolute reliance

on preclinical animal data and isolated accidental exposure registries, as ethical constraints prohibit prospective human trials (Level 5).

The foundational literature of 2024 centered on initial systematic evaluations of adverse events, comparative efficacy analyses in early adoption phases, and pioneering off-label case reporting.

Chen et al. [65] performed a systematic review and network meta-analysis concerning adverse events of acne associated with JAK inhibitors in dermatologic indications. The study concluded that upadacitinib carries the highest relative risk for acne induction compared to other JAK inhibitors, an effect directly tied to its specific pathway inhibition and dosing. This is highly relevant for guiding drug selection in patients predisposed to acne. The limitation is the variability in how acne was reported across different clinical trials, potentially skewing the precision of the comparative risk ratios (Level 1a).

Dhar et al. [66] presented an update on the use of Janus kinase inhibitors in atopic dermatitis. The authors concluded that integrating oral JAK inhibitors provides rapid, deep responses for patients failing conventional systemic therapies, while topical formulations serve as highly effective maintenance for localized disease. This dual systemic-topical paradigm is vital for modern eczema management. The limitation is the narrative nature of the update, which provides a broad overview of clinical utility rather than specific, new quantitative insights into long-term safety or cost-effectiveness (Level 5).

Utama et al. [67] published a scoping review examining how Janus kinase inhibitors are changing the landscape of vitiligo management. The authors concluded that topical ruxolitinib serves as a highly effective, targeted intervention for facial repigmentation, fundamentally altering the therapeutic algorithm away from broad immunosuppression. This matters because it provides the first FDA-approved mechanism to reverse depigmentation. The main limitation is the acknowledgment that acral and long-standing depigmented lesions remain highly resistant to therapy, highlighting a persistent gap in clinical efficacy (Level 2a).

Zhang et al. [68] reviewed the application of JAK inhibitors in treating paradoxical reactions induced by other immune-related therapies, such as biologic-induced psoriasis or eczema. The authors concluded that the broad cytokine blockade of JAK inhibitors efficiently resolves these complex, contradictory immune flares without necessitating complete cessation of the primary targeted therapy. This is a critical salvage strategy for complex medical dermatology patients. The primary limitation is that evidence is strictly based on isolated case reports and mechanistic theory, lacking randomized trial validation (Level 4).

Powers et al. [69] reported a case detailing the use of a topical Janus kinase inhibitor to treat an immune checkpoint inhibitor-induced eczematous reaction. The authors concluded that topical ruxolitinib provided rapid symptomatic relief and cleared the cutaneous toxicity without compromising the systemic anti-tumor immunity required for the patient's cancer treatment. This is profoundly important for oncodermatology, allowing patients to maintain life-saving cancer therapies. The obvious limitation is that it is a single case report, and larger cohorts are necessary to confirm broader oncologic safety (Level 4).

He et al. [70] conducted an umbrella review of meta-analyses assessing Janus kinase inhibitors in atopic dermatitis. The authors concluded that high-dose oral JAK inhibitors consistently demonstrate superior short-term efficacy for skin clearance and itch reduction compared to dupilumab, though they carry a higher incidence of laboratory anomalies and mild adverse events. This comprehensive overview is essential for evidence-based drug positioning. The limitation inherent to umbrella reviews is the risk of compounding methodological flaws and overlapping patient populations present in the underlying original meta-analyses (Level 1a).

Husein-Elahmed et al. [71] performed a systematic review and Bayesian network meta-analysis comparing the efficacy of oral Janus kinase inhibitors and biologics in adult alopecia areata. The study concluded that oral JAK inhibitors significantly outperform other systemic interventions in achieving clinically meaningful hair regrowth, establishing them as the definitive gold standard. This matters as it firmly ends reliance on less effective, historically utilized off-label therapies. The limitation is the mathematical reliance on indirect comparisons, given the stark lack of head-to-head randomized trials between different JAK inhibitors (Level 1a).

Inoue et al. [72] evaluated JAK inhibitors for the treatment of vitiligo, focusing on underlying cellular mechanisms. The authors concluded that JAK blockade directly inhibits the IFN-gamma/CXCL10 axis, effectively halting the autoimmune destruction of melanocytes and allowing for epidermal repigmentation. This validates the scientific rationale behind clinical use and is important for driving future drug development. The limitation is that while the mechanistic explanation is robust, the review does not provide new, large-scale clinical efficacy data regarding the durability of repigmentation post-treatment (Level 5).

Ireland et al. [73] published a meta-analysis of randomized clinical trials evaluating short-term cardiovascular complications in dermatology patients receiving JAK inhibitors. The authors concluded that within the initial trial periods, there was no statistically significant increase in major adverse cardiovascular events (MACE) in relatively young, healthy dermatologic cohorts compared to placebo. This provides critical, immediate reassurance for prescribing physicians. The

significant limitation is the short-term nature of the included clinical trials, which are inadequately powered and too brief to capture long-term cardiovascular atherogenesis or thrombotic events (Level 1a).

He et al. [74] presented a case report and literature review utilizing the Janus kinase 1 inhibitor abrocitinib for isolated nail lichen planus. The authors concluded that targeted systemic therapy rapidly halted the destructive inflammatory process, leading to the restoration of normal nail plate architecture in a highly treatment-resistant condition. This matters greatly, as severe nail lichen planus often leads to permanent anonychia if not aggressively managed. The limitation is the reliance on a single case observation, preventing generalizations regarding standardized dosing and recurrence rates (Level 4).

Yoon et al. [75] conducted a systematic review and meta-analysis of randomized controlled trials focusing on the safety of systemic Janus kinase inhibitors in atopic dermatitis. The authors concluded that the drugs are generally safe over short-to-medium terms, with the most common adverse events being mild upper respiratory tract infections, headache, and manageable acne, without severe acute toxicities. This comprehensive safety profile is vital for patient counseling. The limitation remains the lack of long-term real-world extension data to definitively rule out rare, cumulative side effects (Level 1a) (Table 1).

Table 1. Summary of included studies on Janus Kinase (JAK) inhibitors in dermatology (year 2024–2026).

Author, Year [Ref]	Study Type/Design	Main Focus	Key Contribution/Principal Findings	Level
Foggi et al., 2026 [18]	Narrative review	Emerging off-label dermatologic uses	Summarized expanding use of JAK inhibitors beyond approved indications, including recalcitrant inflammatory dermatoses, and emphasized broad cytokine blockade as the rationale for therapeutic extension despite limited trial-level evidence.	5
Jalili et al., 2026 [19]	Practical narrative guide	Systemic JAK inhibitors in atopic dermatitis	Provided a clinically oriented framework for patient selection, pre-treatment assessment, laboratory surveillance, dose adjustment, and real-world optimization of systemic JAK inhibitors in moderate-to-severe atopic dermatitis.	5
Słomianny et al., 2026 [20]	Literature review	Long-term safety in dermatology	Reviewed longer-term dermatologic safety data and concluded that infections and laboratory abnormalities remain the dominant adverse events, while overall safety trends appear broadly stable with continued monitoring.	2a
Greco et al., 2026 [21]	Narrative review	Adult vitiligo management	Evaluated topical JAK inhibition within the broader treatment algorithm for vitiligo and supported its role as a targeted repigmenting therapy, particularly for facial and cervical lesions.	5
Termini et al., 2026 [22]	Updated review	Topical delgocitinib in chronic hand eczema	Synthesized evidence supporting delgocitinib as an effective pan-JAK topical therapy for chronic hand eczema and discussed broader relevance to other inflammatory cutaneous disorders.	1a
Zhou et al., 2026 [23]	Pharmacovigilance study	Ruxolitinib cream adverse events	FAERS-based real-world analysis suggested that ruxolitinib cream retains a favorable safety profile, with predominantly local or mild adverse-event signals.	2c
Olisova et al., 2026 [24]	Review	Autoimmune bullous diseases	Discussed JAK inhibitors as corticosteroid-sparing candidates in pemphigus and pemphigoid, highlighting early evidence for immunomodulatory benefit in severe blistering disease.	4
Gratz et al., 2026 [25]	Systematic review	Effect on patch testing	Showed that systemic JAK inhibitors may suppress delayed hypersensitivity responses and thereby increase the risk of false-negative patch test results.	2a
Ohyama et al., 2026 [26]	Narrative review and real-world experience	Severe alopecia areata	Reinforced oral JAK inhibitors as the most effective systemic option for severe alopecia areata, while underscoring frequent relapse after drug discontinuation.	5

Table 1. *Cont.*

Author, Year [Ref]	Study Type/Design	Main Focus	Key Contribution/Principal Findings	Level
Zhan et al., 2026 [27]	Pharmacovigilance study	Post-marketing safety in alopecia areata	Identified acne, weight gain, and upper respiratory infections as notable real-world safety signals for JAK inhibitors used in alopecia areata.	2c
Lu et al., 2026 [28]	Pharmacovigilance analysis and literature review	Cutaneous lymphoma	Reported a weak but noteworthy signal for cutaneous lymphoma in association with JAK inhibitor exposure, warranting continued long-term vigilance.	2c
Jaguan et al., 2026 [29]	Systematic review	Generalized granuloma annulare	Supported JAK inhibition as a promising therapeutic strategy for generalized granuloma annulare, while emphasizing the low quality and uncontrolled nature of current evidence.	4
Zhang et al., 2026 [30]	Narrative review	Genetic pharmacoepi-demiology	Discussed pharmacogenomic variability as a future tool for improving precision dosing, efficacy prediction, and safety selection of JAK inhibitors in immune-mediated skin disease.	5
Cui et al., 2026 [31]	Mechanistic review	JAK/STAT pathway in atopic dermatitis	Clarified how JAK inhibition suppresses multiple inflammatory axes in atopic dermatitis, helping explain both rapid efficacy and class-related adverse effects.	5
Tan et al., 2026 [32]	Scoping review	SJS/TEN	Proposed JAK inhibitors as a potentially important rescue strategy in Stevens-Johnson syndrome and toxic epidermal necrolysis, though evidence remains preliminary and sparse.	4
Solimani et al., 2026 [33]	Mechanistic study/translational review	Pemphigus	Suggested that JAK/STAT blockade may reduce acantholysis and stabilize keratinocyte adhesion in pemphigus, providing a biologic rationale for future therapeutic application.	5
Burlando et al., 2026 [34]	Narrative review	Chronic hand eczema	Framed delgocitinib and topical JAK inhibition as a major step toward defining a more targeted therapeutic identity for chronic hand eczema.	5
Zou et al., 2026 [35]	Single-arm clinical trial	Primary cutaneous amyloidosis	Preliminary trial observations suggested that tofacitinib may reduce pruritus and flatten amyloid papules, likely through interruption of neuroimmune inflammatory signaling.	4
Quraishi et al., 2026 [36]	Systematic review and meta-analysis	Acne risk with JAK inhibitors	Confirmed that acne is a reproducible class-associated adverse event, often dose-related and generally manageable, but clinically relevant for counseling and monitoring.	1a
Huang et al., 2026 [37]	Post-marketing comparative study	Biologics versus JAK inhibitors in atopic dermatitis	Real-world comparison suggested that JAK inhibitors carry relatively higher risks of acne, herpes zoster, and laboratory abnormalities than biologics in atopic dermatitis practice.	2c
Chang et al., 2026 [38]	Multicenter study	Pediatric progressive vitiligo	Demonstrated that oral JAK inhibitors combined with 308-nm excimer laser may accelerate disease control and repigmentation in pediatric progressive vitiligo.	2b
Ghafoor et al., 2026 [39]	Systematic review	Keratinocyte carcinomas	Found no clear short- to medium-term signal that JAK inhibitors substantially increase keratinocyte carcinoma incidence, although longer follow-up is still needed.	2a
Altınöz Güney et al., 2026 [40]	Pharmacovigilance study	Infection risk	Identified herpes zoster and respiratory infections as key safety signals and supported preventive strategies such as vaccination before systemic therapy.	2c

Table 1. *Cont.*

Author, Year [Ref]	Study Type/Design	Main Focus	Key Contribution/Principal Findings	Level
Dai et al., 2026 [41]	Historical/developmental review	Three decades of JAK inhibitor development	Traced the evolution of JAK inhibitors from broad systemic immunology into dermatology, emphasizing improved selectivity and broader disease applicability.	5
Lin et al., 2026 [42]	Multinational cohort study	Boxed-warning outcomes	Suggested that absolute risks of MACE, VTE, and severe malignancy in dermatology cohorts may be lower than originally inferred from higher-risk non-dermatologic populations.	2b
Fang et al., 2026 [43]	Systematic review and meta-analysis	Pediatric alopecia areata ≤12 years	Supported oral JAK inhibitors as effective short-term treatment in severe pediatric alopecia areata, though long-term developmental safety remains uncertain.	1a
Rajput Khokhar et al., 2025 [44]	Practical review	Adverse effects relevant to daily dermatology practice	Summarized recognizable toxicities such as acne, infection, and hyperlipidemia, emphasizing structured monitoring and early management to sustain adherence.	5
Shen et al., 2025 [45]	Systematic review	Lichen sclerosus	Suggested that topical and systemic JAK inhibitors may benefit refractory lichen sclerosus, but current data remain limited to low-level uncontrolled evidence.	4
Lamba et al., 2025 [46]	Narrative overview	Delgocitinib	Reviewed delgocitinib as a topical pan-JAK inhibitor with practical relevance for dermatologists managing localized inflammatory disease.	5
Haag et al., 2025 [47]	Expert practical guide/consensus	Oral JAK inhibitors for atopic dermatitis	Provided International Eczema Council guidance on screening, cardiovascular risk assessment, and safe implementation of oral JAK inhibitors in eczema practice.	5
Martin et al., 2025 [48]	Review	Scarring alopecia	Supported JAK inhibitors as useful disease-stabilizing options in recalcitrant scarring alopecias, particularly for arresting inflammation rather than reversing follicular destruction.	4
Malekan et al., 2025 [49]	Narrative review	Immune-mediated photodermatoses	Proposed JAK inhibition as an emerging systemic strategy for photodermatoses by interrupting ultraviolet-triggered inflammatory cascades.	5
Sivadas et al., 2025 [50]	Commentary/mechanistic review	JAK inhibitor toxicity	Explained toxicity through pathway-specific biologic effects, helping clinicians anticipate adverse events based on molecular selectivity rather than viewing them as nonspecific.	5
Honap et al., 2025 [51]	International multicenter retrospective cohort study	JAK inhibitor-induced acne	Showed that acne is a frequent but usually mild and manageable complication of JAK inhibitor therapy, with low rates of discontinuation when treated promptly.	2b
Haider et al., 2025 [52]	Narrative review	Delgocitinib in chronic hand eczema and atopic dermatitis	Reinforced the role of topical pan-JAK inhibition as a localized, steroid-sparing strategy with minimal systemic absorption in chronic inflammatory dermatoses.	5
Su et al., 2025 [53]	Meta-analysis of randomized clinical trials	Serious infection risk	Found that JAK inhibitors modestly increase serious infection risk relative to controls, though absolute risk in dermatology populations remains low.	1a
Li et al., 2025 [54]	Preclinical translational study	Hydrogel microneedle JAK inhibitor delivery in vitiligo	Developed a microneedle-based delivery platform that improved local drug penetration and repigmentation in experimental vitiligo models.	5

Table 1. *Cont.*

Author, Year [Ref]	Study Type/Design	Main Focus	Key Contribution/Principal Findings	Level
Al-Mamoori et al., 2025 [55]	Case series	Refractory dissecting cellulitis of the scalp	Reported rapid clinical improvement with JAK inhibitors in refractory dissecting cellulitis, supporting possible value in severe follicular occlusion disease.	4
Heidari et al., 2025 [56]	Narrative clinical review	Granuloma annulare	Summarized growing evidence that systemic and topical JAK inhibitors may be effective for generalized or treatment-resistant granuloma annulare.	4
Wang et al., 2025 [57]	Evidence synthesis/review	Psoriasis and psoriatic arthritis	Suggested that JAK inhibitors are more compelling for joint disease than for cutaneous psoriasis, where biologics often achieve stronger skin clearance.	2a
Iorizzo et al., 2025 [58]	Systematic review	Inflammatory nail disorders	Found encouraging responses with JAK inhibitors in nail psoriasis, nail lichen planus, and alopecia-associated nail disease, while noting the shortage of dedicated nail trials.	2a
Packer et al., 2025 [59]	Narrative review	Cutaneous T-cell lymphoma	Discussed JAK pathway targeting as a possible therapeutic avenue in CTCL, with early evidence suggesting both antiproliferative and antipruritic effects.	5
Stratman et al., 2025 [60]	Clinical case series	Systemic JAK inhibitors in granuloma annulare	Added real-world support for oral JAK inhibitors as effective options in generalized granuloma annulare, with acceptable short-term tolerability.	4
Tsiogkas et al., 2025 [61]	Systematic review and network meta-analysis	Psoriatic arthritis	Positioned JAK inhibitors as strong oral options for psoriatic arthritis, with meaningful articular efficacy and useful but less dominant skin benefits.	1a
Bieber et al., 2025 [62]	Narrative review	Systemic therapy of atopic dermatitis	Emphasized the paradigm-shifting speed of itch relief and lesion improvement achieved with systemic JAK inhibitors in severe atopic dermatitis.	5
Sun et al., 2025 [63]	Clinical review	Alopecia areata	Consolidated evidence that oral JAK inhibitors have revolutionized severe alopecia areata management, while stressing the chronicity of therapy and relapse after withdrawal.	5
Gordon et al., 2025 [64]	Safety review	Pregnancy and lactation	Concluded that systemic JAK inhibitors should be avoided during pregnancy and breastfeeding because human safety evidence is insufficient and preclinical concern persists.	5
Chen et al., 2024 [65]	Systematic review and network meta-analysis	Acne adverse events in dermatologic indications	Demonstrated differential acne risk among JAK inhibitors and suggested that drug-specific selection may matter in acne-prone patients.	1a
Dhar et al., 2024 [66]	Narrative update	Atopic dermatitis	Reviewed oral and topical JAK inhibitors as major additions to eczema management, highlighting rapid efficacy in severe disease and practical versatility across disease extent.	5
Utama et al., 2024 [67]	Scoping review	Vitiligo	Supported JAK inhibitors, particularly topical therapy, as major drivers of the contemporary shift toward targeted vitiligo management, especially for facial disease.	2a
Zhang et al., 2024 [68]	Narrative review	Paradoxical reactions in immune-related dermatoses	Proposed JAK inhibitors as salvage tools for paradoxical inflammatory eruptions induced by other immunomodulators, based mainly on case-level experience and mechanistic rationale.	4

Table 1. *Cont.*

Author, Year [Ref]	Study Type/Design	Main Focus	Key Contribution/Principal Findings	Level
Powers et al., 2024 [69]	Case report	Checkpoint inhibitor-induced eczematous eruption	Described successful use of topical JAK inhibition to control oncologic treatment-related eczema without interrupting cancer therapy.	4
He et al., 2024 [70]	Umbrella review of meta-analyses	Atopic dermatitis efficacy	Concluded that high-dose oral JAK inhibitors offer very strong short-term efficacy, in some outcomes exceeding dupilumab, while carrying more laboratory and minor adverse events.	1a
Husein-Elahmed et al., 2024 [71]	Systematic review and Bayesian network meta-analysis	Adult alopecia areata	Showed that oral JAK inhibitors outperform alternative systemic therapies in alopecia areata and have become the leading evidence-based option.	1a
Inoue et al., 2024 [72]	Mechanistic and clinical review	Vitiligo	Highlighted inhibition of the IFN-γ/CXCL10 pathway as the key mechanistic basis for JAK inhibitor efficacy in vitiligo.	5
Ireland et al., 2024 [73]	Meta-analysis of randomized clinical trials	Short-term cardiovascular risk	Found no significant short-term increase in major cardiovascular events in dermatology trial populations receiving JAK inhibitors, although long-term uncertainty remains.	1a
He et al., 2024 [74]	Case report and literature review	Nail lichen planus	Reported successful abrocitinib use in isolated nail lichen planus, suggesting a targeted option for a destructive and difficult-to-treat nail disorder.	4
Yoon et al., 2024 [75]	Systematic review and meta-analysis of randomized controlled trials	Safety in atopic dermatitis	Supported an overall favorable short- to medium-term safety profile in atopic dermatitis, with mostly mild infections, headache, and acne as the dominant adverse events.	1a

4. Discussion

The literature published between 2024 and 2026 confirms that JAK inhibitors have moved from a promising mechanistic concept to a central therapeutic platform in dermatology. Across the reviewed studies, the greatest strength of this drug class lies in its ability to interrupt multiple cytokine-dependent inflammatory circuits rapidly and with a breadth not easily reproduced by conventional immunosuppressants or even highly selective biologics. At the same time, the review also shows that enthusiasm has outpaced evidentiary maturity in several indications. The strongest data cluster around atopic dermatitis, alopecia areata, vitiligo, and chronic hand eczema, whereas many other uses remain supported mainly by case reports, case series, narrative reviews, or mechanistic extrapolation. This unevenness is important because it suggests that "JAK inhibitors in dermatology" should not be discussed as a single evidence category; instead, their place in practice depends heavily on indication, formulation, age group, safety context, and treatment goals [18,47,71,75].

Atopic dermatitis remains the clearest example of how JAK inhibition has reshaped therapeutic expectations. Practical guides, narrative reviews, and umbrella syntheses consistently emphasize rapid itch relief, early reduction in inflammatory burden, and flexible dose titration as major advantages over older systemic agents and, in selected settings, over slower-onset biologics [19,31,70,75]. The reviewed studies collectively suggest that oral JAK inhibitors are particularly valuable when clinical urgency matters, such as severe pruritus, sleep disruption, widespread flares, or failure of conventional systemic therapy. Yet this benefit is inseparable from the requirement for structured screening and monitoring. Several authors stress that the question is no longer whether JAK inhibitors work in atopic dermatitis, but rather how to identify the patients most likely to benefit without incurring avoidable risk [37,42,70,75]. This marks an important conceptual shift: JAK inhibitors are not rescue agents of last resort alone, but neither are they interchangeable with topical anti-inflammatory maintenance or biologics in all patients. Their optimal position appears to be as precision systemic tools used when speed, symptom burden, and patient preference justify the broader safety framework.

Alopecia areata represents a second major domain in which JAK inhibitors have clearly altered the therapeutic landscape. Reviews and meta-analyses consistently show that oral JAK inhibitors outperform previously used off-label systemic interventions and offer clinically meaningful regrowth in patients with severe disease [26,27,43,63]. In practice, this is transformative because alopecia areata is a disease in which treatment failure has historically carried major psychosocial consequences. However, the alopecia literature also exposes the chronicity paradox of JAK therapy: efficacy is substantial, but durability after discontinuation remains limited, and relapse commonly follows treatment withdrawal [26,63,71]. That

pattern implies that JAK inhibition often functions as suppression rather than cure. Pediatric data are especially encouraging, but also incomplete; while short-term outcomes in younger patients appear favorable, the evidence base remains small and cannot yet resolve long-term developmental, metabolic, or immunologic concerns [38,43]. Therefore, for alopecia areata, the discussion has evolved from proof of efficacy to the more difficult questions of maintenance strategy, duration of therapy, retreatment after relapse, and selection of patients for long-term systemic exposure [26,27,42,63].

Vitiligo illustrates the importance of formulation-specific application. Unlike systemic inflammatory dermatoses in which broad immunomodulation is often required, vitiligo has benefited substantially from localized JAK inhibition, particularly with topical agents for facial and cervical disease [21,38,67,72]. The reviewed evidence supports the IFN-γ/CXCL10 axis as a compelling mechanistic basis for repigmentation-oriented therapy, and this translational coherence has probably contributed to the unusually rapid integration of JAK inhibitors into vitiligo care [21,67,72]. Even so, the literature also defines the limitations of current success. Repigmentation is neither uniform nor universal, and resistant sites, especially acral areas and long-standing lesions, continue to respond poorly [21,67]. Combination therapy appears promising, particularly in pediatric progressive vitiligo where oral JAK inhibition combined with 308-nm excimer laser may accelerate disease stabilization and repigmentation [38]. Meanwhile, experimental delivery systems such as microneedle-based platforms suggest that future gains may come not only from new molecules, but also from improved cutaneous delivery and retention [54]. Overall, vitiligo demonstrates that JAK inhibition is most effective when aligned with disease anatomy, treatment timing, and adjunctive phototherapy rather than used as an isolated pharmacologic solution [38,54,67,72].

Chronic hand eczema and the broader development of topical delgocitinib show how JAK inhibition may also redefine difficult localized dermatoses. Phase III-level evidence and focused reviews position delgocitinib as a credible steroid-sparing therapy for a disorder long characterized by relapsing inflammation, barrier dysfunction, occupational aggravation, and inadequate long-term options [22,34,46,52]. This is clinically important because chronic hand eczema often sits at the intersection of efficacy, tolerability, and anatomic practicality: topical corticosteroids work, but chronic use is constrained by cutaneous adverse effects, and systemic treatment may be disproportionate in many patients. A topical pan-JAK inhibitor therefore occupies a particularly useful therapeutic niche [22,34,46,52]. Nonetheless, unanswered questions remain. Real-world durability, maintenance schedules, cost-effectiveness, and comparative performance against other nonsteroidal topicals are still insufficiently defined [22,34,46,52]. Even so, among the newer dermatologic applications of JAK inhibition, chronic hand eczema appears to be one of the most mature in terms of therapeutic identity and translational readiness.

Beyond these higher-confidence indications, the reviewed literature reveals a rapidly expanding off-label frontier. Generalized granuloma annulare is one of the most persuasive examples, with systematic reviews, narrative syntheses, and case series repeatedly reporting substantial improvement in refractory disease [29,56,60]. Likewise, lichen sclerosus, scarring alopecias, inflammatory nail disorders, primary cutaneous amyloidosis, dissecting cellulitis of the scalp, photodermatoses, paradoxical immune-mediated eruptions, and checkpoint inhibitor-associated eczema all emerge as areas in which JAK inhibitors may fill meaningful therapeutic gaps [35,45,48,74]. However, it is precisely in these areas that critical appraisal is most necessary. The recurring pattern is biologic plausibility followed by early positive reports, but with major vulnerability to publication bias, small-sample enthusiasm, and selective reporting of successful salvage cases. In other words, the breadth of JAK inhibitor applicability is impressive, but the depth of evidence is highly variable [18,68,69,74]. These indications should therefore be viewed as investigational extensions of a versatile class rather than evidence-equivalent expansions of standard care.

The literature also suggests intriguing future roles in severe and complex dermatologic disease, including autoimmune bullous disease, pemphigus, Stevens-Johnson syndrome/toxic epidermal necrolysis, and cutaneous T-cell lymphoma [24,32,33,59]. In pemphigus and related conditions, the potential value of JAK inhibition lies not only in generalized immunomodulation but also in possible direct effects on keratinocyte adhesion and inflammatory amplification [24,33]. In SJS/TEN, interest is driven by the possibility of interrupting cytokine-mediated epidermal necrolysis quickly enough to alter a life-threatening course [32]. In CTCL, pathway-specific targeting raises the prospect that JAK inhibitors may function as both anti-inflammatory and antineoplastic agents in appropriately selected molecular contexts [59]. Yet these are among the least settled applications. The evidence remains largely preclinical, mechanistic, or based on very limited clinical experience, and any shift toward routine use would require substantially stronger prospective validation [24,32,33,59]. Their importance in this review lies more in illustrating the biological reach of JAK/STAT modulation than in defining current standard practice.

Safety remains the central counterweight to therapeutic enthusiasm. The reviewed evidence does not support a simplistic view of JAK inhibitors as uniformly high-risk drugs in dermatology, but neither does it justify complacency. Rather, the safety profile is nuanced and highly dependent on formulation, selectivity, patient comorbidity, and indication [53,65,73,75]. Topical agents such as ruxolitinib cream appear reassuring in real-world use, with adverse events dominated by application-site reactions and limited evidence of major systemic toxicity [23]. By contrast, systemic JAK inhibitors require longitudinal assessment because the clinically relevant risks are cumulative and multifactorial. Acne has emerged as one of the most consistent class-associated adverse events, documented across dermatologic and nonder-

matologic cohorts and reinforced by both network meta-analysis and retrospective multicenter data [27,36,65,75]. Importantly, acne is generally mild and manageable, but it matters because it can affect adherence, patient satisfaction, and drug choice, especially in younger patients. Infection is another recurring theme, particularly herpes zoster and upper respiratory tract infections, and the literature supports structured pre-treatment counseling and vaccination strategies where appropriate [20,37,40].

The debate around boxed warnings deserves especially careful interpretation. Pharmacovigilance reports and retrospective cohorts raise understandable concern regarding malignancy, cardiovascular events, thrombosis, and serious infection, but dermatology-specific datasets increasingly suggest that absolute risk in typical skin disease populations may be substantially lower than the risk inferred from older rheumatoid arthritis populations with different baseline characteristics [39,42,47,73]. This distinction is clinically crucial. Overgeneralization of boxed-warning concerns may unnecessarily deprive relatively young and otherwise healthy dermatology patients of highly effective therapy, while underestimation of risk may expose older or comorbid patients to preventable harm. The best reading of the current evidence is therefore neither alarmist nor dismissive: systemic JAK inhibitors demand individualized stratification, but the dermatology population should not be assumed to mirror the highest-risk systemic inflammatory cohorts from which regulatory concerns initially emerged [28,39,42]. This is an area where continued registry-quality real-world data will be more informative than short trial windows alone.

Another notable contribution of the recent literature is its emphasis on practical prescribing and monitoring rather than efficacy alone. Several reviews and consensus-style papers frame successful JAK inhibitor use as a process that begins before the first dose: careful patient selection, baseline cardiovascular and infectious risk assessment, review of concomitant medications, reproductive counseling, laboratory monitoring, and management of expected low-grade adverse effects all influence whether treatment remains sustainable [50,62,64]. This pragmatic orientation is particularly relevant because JAK inhibitors may fail in real-world practice not from lack of efficacy, but from avoidable discontinuation due to anxiety, inadequate counseling, or poorly managed side effects such as acne or transient laboratory abnormalities [19,62,64]. Pregnancy and lactation remain clear exclusion settings based on current evidence, reinforcing that enthusiasm for class expansion must stop where reproductive safety is unestablished [64]. Likewise, the observation that systemic JAK inhibition may suppress patch test reactivity is a reminder that these drugs affect not only disease activity but also diagnostic interpretation, creating subtle consequences for routine dermatologic practice [25].

A final major theme is the gradual movement toward precision dermatology. Although still preliminary, the literature on genetic pharmacoepidemiology, pathway selectivity, and mechanism-based toxicity points toward a future in which the choice of JAK inhibitor may become more individualized than it is today [30,41,50]. Selectivity matters not only for efficacy, but also for the pattern of adverse events, and the next stage of the field may depend less on proving that JAK inhibition works than on determining which patient should receive which inhibitor, in what dose, and for how long [30,31,41,50]. This future will also require better outcomes research. A striking limitation across the 58 studies is the dominance of reviews, meta-analyses built on short-term trials, pharmacovigilance datasets lacking denominators, and uncontrolled observational reports for rare dermatoses. Higher-quality comparative trials, long-term registries, withdrawal studies, pediatric extension data, and cost-effectiveness analyses are needed to move from promising expansion to stable therapeutic integration [63,70,71,75].

Taken together, the current body of evidence supports a balanced conclusion. JAK inhibitors are not simply another addition to the dermatologic armamentarium; they represent a versatile therapeutic platform with proven disease-modifying value in selected conditions and compelling exploratory potential in many others. Yet their success depends on matching mechanistic breadth with clinical restraint. Where evidence is strong, they should be used confidently but carefully. Where evidence remains preliminary, they should be pursued critically, ideally within structured observational frameworks or clinical trials. Dermatology is entering an era in which JAK inhibition is both mainstream and experimental, and the discipline's next challenge is to ensure that rapid innovation is matched by equally rigorous long-term evidence [43,47,53,57].

5. Conclusions

Janus kinase inhibitors have established themselves as one of the most important therapeutic advances in contemporary dermatology. Their value is most convincing in atopic dermatitis, alopecia areata, vitiligo, and chronic hand eczema, where they offer rapid and often clinically transformative responses. At the same time, expanding off-label use in rarer inflammatory and immune-mediated dermatoses highlights both the versatility of JAK/STAT pathway modulation and the current limits of the evidence base. The major challenge is no longer proving that these agents can work, but defining how to use them safely, selectively, and sustainably in real-world practice. Future progress will depend on longer-term surveillance, better comparative trials, biomarker-guided patient selection, and clearer treatment algorithms across both established and emerging dermatologic indications.

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