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The Role of Janus Kinase Inhibitors in Dermatology

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ABSTRACT

Janus kinase (JAK) is an intracellular protein of non-receptor tyrosine kinase that functions in cellular regulation and cytokine signaling, thereby producing inflammation. Inhibition of JAK with small molecules that modulate cytokines by blocking the release of Adenosine Triphosphate (ATP) for JAK phosphorylation can prevent inflammation. These molecules are known as Janus Kinase Inhibitors (JAKi). Several related studies have demonstrated that JAKi can be used in the treatment of autoimmune and neoplastic conditions both topically and systemically. Currently, JAKi has been utilized in dermatology and has obtained approval from the Food and Drug Administration (FDA), primarily for treating Atopic Dermatitis (AD), Alopecia Areata (AA), psoriasis, and vitiligo. This review examines the molecular mechanisms of JAK/STAT pathway dysregulation in inflammatory skin diseases, evaluates the efficacy and safety profiles of both topical and systemic JAKi formulations across multiple dermatological conditions, and compares their therapeutic effectiveness against conventional immunosuppressants and biologic agents. The findings indicate that JAKi demonstrates superior rapid onset of action, multi-target cytokine suppression, and favorable safety profiles in short-to-medium term use, positioning them as innovative therapeutic alternatives for patients with treatment-refractory inflammatory skin diseases.

KEYWORDS *alopecia areata, atopic dermatitis, Janus Kinase Inhibitor, psoriasis, vitiligo*

INTRODUCTION

The process of cellular regulation in the human body results from a complex interaction of various enzymes and signaling molecules that ensure physiological balance. One enzyme that plays an important role in this mechanism is tyrosine kinase, which mediates tyrosine phosphorylation in target proteins. The activity of tyrosine kinase regulates various biological processes such as proliferation, differentiation, apoptosis, and immune responses to infection and inflammation. Disruption of tyrosine kinase activity can trigger a variety of pathological conditions, including cancer, autoimmune diseases, and viral infections, making this enzyme an important target in the development of modern therapies (K. Bhanumathy et al., 2021).

Globally, inflammatory skin diseases represent a substantial public health burden with significant socioeconomic implications. According to the Global Burden of Disease Study 2019 conducted by the Institute for Health Metrics and Evaluation (IHME), dermatological conditions affect over 1.9 billion people worldwide, with inflammatory skin diseases accounting for a substantial proportion of this burden. Atopic dermatitis (AD) affects approximately 15–20% of children and 7–10% of adults globally, with prevalence rates continuing to rise in industrialized nations (Huang & Armstrong, 2023). Psoriasis affects 2–3% of the global population, translating to over 125 million individuals worldwide, and is associated with substantial disability-adjusted life years (DALYs) due to its chronic, relapsing nature and comorbidities including psoriatic arthritis, cardiovascular disease, and metabolic syndrome (World Health Organization, 2016). Alopecia areata, though affecting a smaller

proportion (0.1–0.2% of the population), carries profound psychological and social impacts, particularly given its unpredictable course and visible manifestations. Vitiligo affects 0.5–2% of the global population, with higher prevalence rates observed in certain ethnic groups, and is associated with significant psychosocial morbidity and reduced quality of life.

The economic costs of these conditions are substantial. A 2021 systematic review estimated that the annual direct healthcare costs of atopic dermatitis in the United States alone exceed $5.3 billion, with indirect costs (including productivity losses) adding another $8.7 billion annually (Drucker et al., 2021). Similarly, psoriasis imposes annual direct costs exceeding $112 billion globally, with patients experiencing significant work productivity losses and social stigmatization. These conditions collectively represent not only clinical challenges but also major economic burdens on healthcare systems worldwide, underscoring the urgent need for effective, safe, and accessible therapeutic interventions.

One group of tyrosine kinases that does not bind to receptors is Janus kinase (JAK). This protein plays a role in intracellular signal transduction through the phosphorylation cascade mechanism, where activation begins when cytokines bind to their receptors, followed by receptor dimerization and JAK activation. This process then recruits Signal Transducer and Activator of Transcription (STAT), which undergoes phosphorylation, dimerization, and translocation into the nucleus to regulate gene expression (Awasthi et al., 2021; McLornan et al., 2021). Thus, the JAK–STAT pathway represents the main axis of communication between immune cells and inflammatory responses. Since its discovery in 1989, dysregulation of the JAK–STAT pathway has been associated with a variety of autoimmune and inflammatory diseases in dermatology, such as atopic dermatitis (AD), alopecia areata (AA), psoriasis, and vitiligo (Huang & Armstrong, 2023). Overactivation of this pathway leads to increased production of proinflammatory cytokines, which then trigger chronic inflammation. Therefore, inhibition of JAK represents one of the most promising new therapeutic approaches.

Despite the availability of existing treatment modalities for inflammatory skin diseases—including topical corticosteroids, systemic immunosuppressants (cyclosporine, methotrexate, azathioprine), and biologic agents (dupilumab, ustekinumab, secukinumab)—a substantial unmet medical need remains for therapies that combine rapid efficacy, favorable safety profiles, and convenient administration routes. Topical corticosteroids, while effective for mild-to-moderate disease, are associated with skin atrophy, telangiectasia, and hypothalamic–pituitary–adrenal axis suppression with prolonged use. Conventional systemic immunosuppressants carry risks of hepatotoxicity, nephrotoxicity, bone marrow suppression, and increased susceptibility to infections, limiting their long-term utility (Samuel et al., 2023). Biologic agents, though highly effective and generally well-tolerated, are expensive (annual costs often exceeding $30,000–$60,000 per patient), require subcutaneous or intravenous administration, target single cytokine pathways (limiting efficacy in patients with complex immune dysregulation), and may take weeks to months to achieve optimal therapeutic effects (Reich et al., 2022). Furthermore, approximately 30–40% of patients with moderate-to-severe atopic dermatitis fail to achieve adequate disease control with currently available biologics, highlighting the need for alternative therapeutic approaches.

This therapeutic gap has prompted intensive investigation into JAK inhibitors (JAKi) as a novel class of small-molecule drugs capable of simultaneously targeting multiple pro-inflammatory cytokine pathways. By inhibiting intracellular JAK enzymes rather than individual extracellular cytokines, JAKi offer the theoretical advantages of broader immunomodulation, oral or topical administration, rapid onset of action, and potentially lower costs compared to biologic therapies. The exploration of JAKi represents a paradigm shift from traditional immunosuppression and single-target biologics toward multipathway molecular intervention in inflammatory skin diseases.

JAK inhibitors, or Janus kinase inhibitors (JAKi), are small molecules that function by blocking the release of adenosine triphosphate (ATP) required for the phosphorylation process of JAK. As a result, inflammatory signals can be halted, and excessive immune reactions can be suppressed. This mechanism makes JAKi a promising therapeutic candidate for various autoimmune and inflammatory diseases. Since the Food and Drug Administration (FDA) approved the first JAKi for the treatment of myelofibrosis in 2011, the development of this molecule has progressed rapidly and is now expanding into the field of dermatology (Richter et al., 2022).

Preclinical and clinical research demonstrates the effectiveness of JAKi in both topical and systemic forms. Nakagawa et al. (2011) showed that JAKi can decrease the activity of T helper 2 (Th2) and Th17 cells in a model of atopic dermatitis in experimental animals. Clinically, (Kalil et al., 2025) research showed the success of oral JAKi in reducing the Severity of Alopecia Tool (SALT) score in patients with moderate to severe alopecia areata. Furthermore, the study by (Estevinho et al., 2023) demonstrated that JAKi use effectively reduces the Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI) in psoriasis patients. (Reich et al., 2022) study also showed that, in atopic dermatitis, therapy with JAKi produces a greater reduction in the Eczema Area and Severity Index (EASI) compared to biological agents. In addition, (Rosmarin et al., 2022) research on vitiligo found that the use of topical JAKi resulted in an increase in the facial Vitiligo Area Scoring Index (F-VASI) of up to 75%, higher than placebo. Overall, this body of scientific evidence highlights the great potential of JAKi as a new therapy that is more selective and rapid in suppressing chronic inflammation across various inflammatory skin diseases.

However, the safety of JAKi use remains a concern. The FDA issued a boxed warning against the long-term use of JAKi in patients with systemic autoimmune diseases, such as rheumatoid arthritis, due to an increased risk of venous thromboembolism (VTE) (Samuel et al., 2023). Nevertheless, Schneeweiss's (2021) (Tsai et al., 2024) study found that the use of JAKi in dermatological diseases such as atopic dermatitis (AD), alopecia areata (AA), psoriasis, and vitiligo did not show a significant increase in VTE risk. The most common side effects are upper respiratory tract infections (ARIs), acne, headaches, and nausea, which are generally mild and manageable with careful clinical monitoring (Deng et al., 2025; Huang & Armstrong, 2023; Samuel et al., 2023; Truong et al., 2024; Yi et al., 2025).

Research on Janus kinase inhibitors (JAKi) in dermatology demonstrates their effectiveness across multiple inflammatory skin diseases, organized thematically into mechanistic studies, clinical trials, and comparative effectiveness research. Mechanistic studies, such as those by Nakagawa et al., established the foundational proof of concept by showing that JAK inhibition suppresses key inflammatory pathways. Clinically, efficacy has been robustly demonstrated: baricitinib gained FDA approval for alopecia areata, abrocitinib showed superiority to dupilumab in atopic dermatitis within weeks, deucravacitinib outperformed apremilast in psoriasis, and ruxolitinib cream became the first approved therapy for vitiligo.

However, significant evidence gaps remain despite these advances. The existing literature is limited by a lack of long-term safety data beyond one to two years, a scarcity of head-to-head comparisons with the newest biologics, and an absence of predictive biomarkers or economic analyses. These limitations underscore the urgent need for a comprehensive synthesis, especially as FDA approvals for dermatological JAKi have rapidly expanded since 2019, marking a new era of targeted therapy. Furthermore, emerging data suggest that the risk profile for dermatology patients may differ from that of rheumatologic populations, and the COVID-19 pandemic has increased interest in oral therapies, adding layers of clinical and practical relevance.

This review aims to address these needs through a novel, integrative approach. Its uniqueness lies in providing a cross-disease comparison encompassing atopic dermatitis, alopecia areata, psoriasis, and vitiligo, while simultaneously linking mechanistic insights to clinical outcomes. It will contextualize JAKi efficacy alongside both conventional and biologic therapies, incorporate evidence for topical and systemic formulations, and include the latest 2024 data to present the most current synthesis available.

The primary objectives are threefold: to explain the disease-specific dysregulation of the JAK–STAT pathway in each condition, to critically evaluate the latest evidence on JAKi effectiveness and safety, and to provide a practical, evidence-based framework for clinicians. This framework will guide therapeutic decision-making, including patient selection and monitoring, thereby positioning JAKi within the modern dermatological treatment landscape for diseases refractory to conventional therapy.

METHOD

Design and Research Approach

This study is a qualitative research study with a descriptive approach that aims to review and describe the role of Janus kinase inhibitors (JAKi) in the field of dermatology. The study was conducted by analyzing various relevant and current scientific sources, including clinical research findings, scientific publications, and internationally accredited literature reviews. A descriptive approach was employed to provide a comprehensive overview of the mechanism of action of JAK, its role in immune regulation, and the effectiveness of JAKi against various inflammatory skin diseases such as atopic dermatitis (AD), alopecia areata (AA), psoriasis, and vitiligo.

This methodology was chosen because it aligns with the objectives of exploratory research, which seek to describe existing phenomena based on empirical data from previous studies rather than through direct laboratory experiments. This study emphasizes the analysis of the theoretical and clinical relationship between the JAK/STAT pathway and the pathogenesis of dermatological diseases, as well as therapeutic strategies based on JAK inhibition.

Research Location and Time

This research was carried out at the Dermatology, Venereology, and Aesthetics Study Program, Faculty of Medicine, Andalas University / Dr. M. Djamil Padang Hospital in 2024. The selection of this location was based on the availability of academic facilities that support literature research activities and its relevance to the field of dermatology. The research was conducted from July to December 2023, through stages including the collection of scientific references, literature analysis, and the preparation of integrated study results in the form of scientific documentation.

The research implementation process was conducted systematically by utilizing access to scientific databases such as PubMed, ScienceDirect, and Google Scholar to obtain valid and up-to-date scientific literature on JAK and JAK inhibitors in the context of dermatology.

This study focuses on the molecular mechanisms of Janus kinase and Signal Transducer and Activator of Transcription (STAT), as well as their effects on immune system regulation in various inflammatory skin diseases. The scope of discussion includes a basic understanding of tyrosine kinase, the structure and function of JAK, the role of STAT in signal transduction, and an overview of the JAK/STAT pathway in pathological conditions.

In addition, this study discusses the effectiveness and safety of JAK inhibitors in the treatment of dermatological diseases such as atopic dermatitis, alopecia areata, psoriasis, and vitiligo. The safety aspects of therapy were also reviewed based on reports of side effects documented in various clinical studies and randomized controlled trials (RCTs).

Thus, this study presents a comprehensive review that not only examines the fundamental molecular aspects but also translates them into clinical context to clarify the potential application of JAKi as an innovative therapy in the field of dermatology.

Data Sources and Study Populations

The data sources in this study were derived from secondary scientific literature, including journal articles, textbooks, clinical trial reports, and the results of systematic reviews and meta-analyses published between 2019 and 2024. The selection of this time frame aimed to ensure that the data used remained relevant to the latest developments regarding the use of JAK inhibitors in dermatology.

The research population encompassed all studies and publications discussing the role of the JAK/STAT pathway and the clinical application of JAKi in inflammatory skin diseases. From the collected literature, a selection process was carried out based on methodological rigor, clinical relevance, and the validity of research findings. This study did not involve direct patient sampling but relied entirely on a population of scientific data that met established academic eligibility criteria.

The unit of analysis in this study consisted of data from published clinical and preclinical research with high scientific validity. The analysis was conducted thematically to identify patterns of similarity and difference among previous research findings.

RESULTS AND DISCUSSION

In the last decade, therapies for chronic inflammatory dermatological diseases have progressed significantly due to advances in understanding the molecular mechanisms of the immune system. However, diseases such as atopic dermatitis (AD), alopecia areata (AA), psoriasis, and vitiligo remain major challenges because they are chronic, recurrent, and often fail to respond optimally to conventional therapies. Various studies have shown that the underlying mechanisms of these diseases are strongly associated with activation of the Janus kinase/Signal Transducer and Activator of Transcription (JAK/STAT) pathway, which plays an important role in proinflammatory cytokine signaling (Hu et al., 2021); (Huang & Armstrong, 2023).

Globally, the prevalence of inflammatory skin diseases is increasing in line with lifestyle changes, environmental pollution, and immunogenetic factors. Atopic dermatitis, for example, affects about 20% of children and 10% of adults worldwide, while psoriasis affects 2–3% of the population and can significantly reduce quality of life (Huang & Armstrong, 2023). These conditions impose substantial socioeconomic burdens and have profound psychological impacts on affected individuals.

The urgency of this research arises from the need for therapies that are more specific, rapid, and safe than classic immunosuppressive drugs such as cyclosporine, methotrexate, or corticosteroids, which have the potential to cause long-term toxic effects (Huang & Armstrong, 2023). Therefore, the JAKi-based approach represents a new breakthrough in modern dermatology.

The main cause of chronic inflammatory skin diseases is immune system dysregulation, which leads to overactivation of proinflammatory cytokines such as IL-4, IL-5, IL-13, IL-17, IL-22, and IFN-γ. This overactivation triggers repetitive inflammatory processes and disrupts normal skin tissue regeneration (Xin et al., 2020). The JAK/STAT pathway serves as a critical link between cytokine signaling and gene expression that regulates immune responses, cell proliferation, and differentiation (Philips et al., 2022).

Dysfunction in the JAK pathway leads to abnormal activation of STAT, resulting in increased transcription of proinflammatory genes. Consequently, hyperproliferation of keratinocytes occurs in psoriasis, increased Th2 cell activity in atopic dermatitis, and

melanocyte destruction in vitiligo (Miot et al., 2023). The JAK pathway also contributes to the activation of $CD8^+$ T cells that attack hair follicles in alopecia areata, leading to localized inflammation and hair loss (Zhou et al., 2021).

Understanding that the root of these conditions lies in cytokine activation through the JAK/STAT pathway makes JAK suppression a logical and promising therapeutic target. JAK inhibitors function by preventing the release of adenosine triphosphate (ATP), which is required for JAK phosphorylation, thereby blocking STAT activation. This process inhibits the transduction of inflammatory signals and decreases the expression of proinflammatory genes (Hu et al., 2021).

The drug is divided into two generations:

1. First-generation (non-selective) such as *baricitinib* and *delgocitinib*, which act on multiple isoforms of JAK at once (JAK1, JAK2, JAK3, TYK2).
2. Second-generation (selective) such as *upadacitinib*, *abrocitinib*, and *deucravacitinib*, which target specific isoforms with milder side effects (Shawky et al., 2022); (Dhillon, 2020).

This specific mechanism of action makes JAKi superior to biologic therapies because it works on multiple cytokine pathways at once (Th1, Th2, Th17, and Th22), not just on a single mediator (Huang & Armstrong, 2023).

The anti-inflammatory effects of JAKi can be felt in a short time: in a matter of hours on topical preparations and a few days on oral preparations (Samuel et al., 2023). This indicates that JAKi not only suppresses clinical symptoms, but also targets the molecular root of the disease.

Analysis of Discussion by Disease

Atopik Dermatitis (AD)

Atopic dermatitis is a multifactorial chronic disease with a predominance of the Th2 and Th22 immune responses, which stimulate the release of IL-4, IL-5, IL-13, and IL-31 (Huang & Armstrong, 2023). This pathway is directly activated by JAK1 and JAK2, so that the inhibition of both can break the inflammatory cycle.

Research by Reich et al. (2022) showed that the use of abrocitinib 200 mg/day lowered the *Eczema Area and Severity Index (EASI)* by up to 90% within 4 weeks, higher than dupilumab (an anti-IL-4Rα biologic agent). The meta-analysis of Chen et al. (2023) also confirmed that *upadacitinib 30 mg* had a *Relative Risk (RR) of 5.14* in achieving EASI-75, making it one of the most effective therapies for DA.

In addition to the oral form, JAKi has also been shown to be effective in a topical form. The study of Papp et al. (2021) reported that *ruxolitinib 1.5% cream* achieved EASI-90 in 44.3% of patients, much higher than placebo (9.5%). This data shows the advantages of JAKi that works quickly and safely in reducing itching and inflammation in DA.

Alopesia Areata (AA)

AA is an autoimmune disease that attacks hair follicles through the activation of cytotoxic CD8+ T cells that produce IFN-γ. This pathway involves JAK1 and JAK3 in the transduction of IL-15 signals (King et al., 2022). Inhibition of these two JAKs will suppress the IL-15 signal, break the positive feedback loop, and return the hair follicle to the anagen phase.

Research by King et al. (2022) showed that *baricitinib 4 mg/day* lowered *the Severity of Alopecia Tool (SALT)* score by 38.8% compared to placebo (6.2%) at week 36. Meta-analysis of Husein et al. (2024) also proved that *baricitinib 4 mg* is the most effective therapy compared to dupilumab and apremilast.

Thus, JAKi provides solutions to problems that have been difficult to overcome with conventional therapies such as corticosteroids or topical immunotherapy which have limited effects and a high risk of relapse.

Psoriasis

Psoriasis is characterized by a hyperproliferation of keratinocytes due to activation of Th1, Th17, and Th22 cells via the IL-12, IL-23, and IFN-α pathways, all of which are mediated by JAK1, JAK2, and TYK2 (Słuczanowska-Głąbowska et al., 2021). The main target in psoriasis therapy is TYK2 because it plays an important role in IL-23 signaling.

A study by (Estevinho et al., 2023) showed that *deucravacitinib (TYK2 inhibitor)* increased PASI-75 by 75% at a dose of 12 mg per day, compared to placebo (7%). These results are consistent with the findings of Krueger et al. (2022) who reported *a 0/1 DLQI* in 64% of patients with deucravacitinib compared to 4% at placebo.

Compared to *apremilast*, deucravacitinib gave better results with *PASI-75* by 58.7% compared to 35.1% (Ben et al., 2021). This confirms that TYK2 inhibitors are a new, safe, effective, and selective therapy in controlling chronic inflammation of psoriasis.

Vitiligo

Vitiligo is caused by the destruction of melanocytes due to activation of CD8+ T cells that produce IFN-γ and CXCL10, mediated by JAK1 and JAK2 (Qi et al., 2021). Inhibition of both JAKs can suppress CXCL10 expression and inhibit lymphocyte infiltration into the skin.

In a randomized controlled clinical trial by Rosmarin et al. (2022), the use *of ruxolitinib 1.5% cream* twice daily for 24 weeks showed *a Facial Vitiligo Area Scoring Index (F-VASI-75)* result of 29.8%, compared to placebo of 7.4%. This effect illustrates the significant role of JAKi in repigmenting the skin through IFN-γ inhibition as well as stimulation of new melanocyte differentiation.

To date, *ruxolitinib* is the only FDA-approved topical JAKi for the treatment of non-segmental vitiligo with a good safety profile (Chapman et al., 2022).

Clinical Implications and Impacts of JAKi Implementation

The application of JAK inhibitors in dermatology has a broad clinical impact. The use of these drugs not only accelerates the healing of skin lesions but also improves patients' quality of life, as evidenced by significantly improved Dermatology Life Quality Index (DLQI) scores in various studies (Estevinho et al., 2023); Reich, 2022).

From a pharmacological perspective, JAKi offers both rapid efficacy and flexible routes of administration (oral or topical), advantages not shared by biologic therapies. The socioeconomic impact is also favorable, as patients are less dependent on long-term treatment with expensive biologic injections.

Nevertheless, ongoing clinical monitoring remains essential due to the risk of venous thromboembolism (VTE), major adverse cardiovascular events (MACE), and mild infections in patients with certain comorbidities (Samuel et al., 2023). Therefore, evaluating liver, kidney, and lipid profile function is an important part of pre-treatment assessment and ongoing therapy.

The novelty of this study compared with previous research lies in its integrative approach, which examines the direct relationship between clinical interest in JAKi and empirical findings on their effectiveness across dermatological diseases. Whereas previous studies—such as Imarotun et al. (2022), which reviewed the effectiveness of Scratch in the field of education, or Hu et al. (2021), which focused on the basic mechanisms of the JAK/STAT pathway—addressed isolated aspects, this study presents an integration of molecular, clinical, and therapeutic safety dimensions.

In addition, comparative findings show that JAKi demonstrates greater effectiveness than conventional biologic agents. For example, upadacitinib 30 mg induces more rapid reductions in Eczema Area and Severity Index (EASI) scores than dupilumab in atopic dermatitis (Reich,

2022), and deucravacitinib has shown superiority over apremilast in psoriasis (Estevinho et al., 2023).

Thus, the novelty of this research strengthens the position of JAKi as a selective, multitarget, and efficient molecular therapy with the potential to replace biologic agents in modern dermatological medicine.

CONCLUSION

Janus kinase plays an important role in regulating cell survival, proliferation, differentiation, hematopoiesis, immune response, and apoptosis-induced cell death. Dysregulation of the JAK/STAT pathway has implications for many autoimmune and inflammatory disorders in the field of dermatology, such as atopic dermatitis (AD), alopecia areata (AA), psoriasis, and vitiligo. Janus kinase inhibitors suppress the JAK/STAT pathway and inhibit the activity of multiple proinflammatory cytokines. Understanding the mechanism of action of JAK inhibitors, along with their benefits and potential side effects, can help optimize therapeutic outcomes for patients.

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