

VITILIGO: NEW THERAPEUTIC APPROACHES BASED ON IMMUNE PATHOPHYSIOLOGY

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ABSTRACT

Vitiligo is a chronic autoimmune skin disease characterized by the progressive destruction of melanocytes, resulting in a profound psychosocial impact. Historically challenging, therapeutic management has been revolutionized by recent discoveries in the field of immune pathophysiology, as the disease is mediated by autoreactive cytotoxic CD8+ T cells and the Janus Kinase/Signal Transducer and Activator of Transcription (JAK/STAT) signaling pathway, activated by interferon-gamma (IFN- γ). Therefore, the present literature review aimed to analyze new therapeutic approaches based on this immune pathophysiology, encompassing studies from 2016 to 2026 selected from the PubMed/MEDLINE, Embase, Cochrane, and Web of Science databases. The findings demonstrate that the recognition of the JAK/STAT pathway has enabled the development of targeted immunomodulators with highly promising results. Topical ruxolitinib stands out as the first JAK inhibitor drug approved for the treatment of vitiligo, showing consistent and clinically significant repigmentation, particularly in facial lesions. Concurrently, systemic JAK inhibitors, such as baricitinib, ritlecitinib, and upadacitinib, show promising clinical results, with even more notable synergistic effects when combined with phototherapy. Biological therapies and mesenchymal stem cells are also emerging as viable alternatives. It is concluded that the transition from empirical treatments to interventions targeted at the pathophysiology of the disease represents a transformative advancement in the treatment of vitiligo. However, gaps in clinical evidence persist, especially regarding long-term outcomes and pediatric populations, demanding the need for further and deeper research, primarily focused on identifying biomarkers and eliminating resident memory T cells, in order to prevent long-term relapses.

Keywords: Dermatology, Immunological factors, Immunomodulation, Janus kinase inhibitors, Vitiligo.

INTRODUCTION

Vitiligo is a chronic autoimmune skin disease characterized by the progressive destruction of melanocytes, resulting in visible depigmented patches on the skin. It affects approximately 0.5% to 2.0% of the global population, with a significant impact on patients' quality of life due to the associated psychosocial burden.¹

Although it does not affect life expectancy, vitiligo imposes a profound psychosocial impact, with more than half of patients presenting moderate to severe depressive symptoms and high rates of diagnosed anxiety.² The impairment in quality of life is particularly pronounced in patients with facial or

hand lesions, greater extent of affected body surface area, and darker skin phototypes.^{2,3}

Historically, the therapeutic management of vitiligo has been challenging. Conventional options include topical and systemic corticosteroids, calcineurin inhibitors, and narrowband ultraviolet B phototherapy (NB-UVB).⁴⁻⁶ However, many patients, particularly those with extensive or refractory disease, are unable to achieve satisfactory or lasting repigmentation.⁴ The clinical course is further complicated by high relapse rates and heterogeneous treatment responses at different anatomical sites.⁴

Over the past two decades, significant advances in the understanding of the immunological pathophysiology of vitiligo have transformed the therapeutic landscape.⁷⁻⁹

Studies have demonstrated that the disease is mediated by autoreactive cytotoxic CD8+ T lymphocytes that destroy melanocytes through the local production of interferon-gamma (IFN- γ), a central cytokine that activates the Janus kinase/signal transducer and activator of transcription (JAK/STAT) signaling pathway.⁸⁻¹⁰ In this regard, this pathway promotes the secretion of chemokines that recruit T cells to the skin, perpetuating a self-sustaining inflammatory cycle.^{8,10} Additionally, epidermal resident memory T cells are responsible for disease relapse after treatment discontinuation.⁸

In this context, recognition of the JAK/STAT pathway as a central mechanism in melanocyte destruction paved the way for the development of targeted therapies.^{4,5,9} JAK inhibitors have emerged as a promising therapeutic strategy, with topical ruxolitinib (a JAK1/JAK2 inhibitor) becoming the first medication approved specifically for repigmentation in vitiligo.^{4,10,11} Phase III clinical trials demonstrated clinically significant repigmentation, particularly in facial lesions.¹⁰ In turn, oral JAK inhibitors, including ritlecitinib, upadacitinib, and povorcitinib, are currently in phase II and III clinical trials, with promising results as monotherapy or in combination with phototherapy.^{1,4,5}

Given the recent advances in the understanding of the immunological pathophysiology of vitiligo, particularly the role of the JAK/STAT pathway, pro-inflammatory cytokines, and autoreactive T cells in melanocyte destruction, new targeted therapeutic strategies have been developed and incorporated into clinical practice. Despite the growing number of studies on these approaches, there is still a need to critically integrate the evidence related to the immunological mechanisms of the disease and the clinical outcomes of emerging therapies. In this context, the present review brings together and analyzes the most recent scientific evidence on new therapeutic approaches for vitiligo, with an emphasis on JAK inhibitors and other promising immunomodulatory strategies, discussing their perspectives, limitations, and potential applications in the contemporary management of the disease.

OBJECTIVES

• General objective:

Analyze the available scientific evidence on new therapeutic approaches for vitiligo based on the understanding of the immunological pathophysiology of the disease, with emphasis on Janus kinase (JAK) inhibitors and other emerging immunomodulatory strategies, based on studies published between 2016 and 2026.

• Specific objectives:

1. Describe the immunological mechanisms involved in the pathogenesis of vitiligo, with emphasis on the JAK/STAT pathway and the role of cytotoxic CD8+ T cells and IFN- γ .
2. Evaluate the efficacy and safety of topical and systemic JAK inhibitors in the treatment of vitiligo,

based on randomized controlled clinical trials.

3. Identify other emerging immunomodulatory therapies and their therapeutic potential in the management of vitiligo.

4. Discuss future perspectives and challenges in the development of targeted treatments for vitiligo.

METHODOLOGY

OR "tofacitinib" OR "upadacitinib" OR "ritlecitinib" OR "povorcitinib") AND ("treatment" OR "therapy" OR "repigmentation") AND ("immunopathogenesis" OR "interferon-gamma" OR "IFN-gamma" OR "JAK-STAT pathway" OR "CD8 T cells" OR "CXCL10" OR "autoimmune"). Additionally, the reference lists of the selected articles were consulted to identify relevant complementary studies.

The inclusion criteria were:

(a) randomized clinical trials, systematic reviews, meta-analyses, clinical guidelines, and phase II and III studies published between 2016 and 2026;

(b) publications in English and Portuguese;

(c) studies addressing the immunological pathophysiology of vitiligo, with a focus on the JAK/STAT pathway and T-cell-mediated mechanisms;

(d) studies on emerging immunomodulatory therapies;

(e) systematic reviews and meta-analyses on vitiligo treatment;

(f) international consensus guidelines for the management of vitiligo;

(g) studies on biomarkers and mechanisms of relapse.

The exclusion criteria, in turn, included:

(a) articles published before 2016;

(b) isolated case reports without relevance to the understanding of therapeutic mechanisms;

(c) studies in animal models without clinical correlation;

(d) articles that did not specifically address the relationship between immunological pathophysiology and therapeutic approaches; (e) duplicate or retracted studies, or studies with inadequate methodology.

The initial selection was based on the reading of titles and abstracts. Potentially relevant articles were retrieved in full and assessed for eligibility. Phase II and III clinical trials, systematic reviews, meta-analyses, and international consensus guidelines were prioritized. Data extraction focused on: pathophysiological mechanisms, therapeutic agents investigated, study design, study population, efficacy outcomes (including repigmentation indices such as F-VASI, from the English Facial Vitiligo Area Scoring Index, or Facial Vitiligo Area Scoring Index, and T-VASI, from the English Total Body Vitiligo Area Scoring Index, or Total Vitiligo Area Scoring Index), safety and tolerability profile, and main conclusions.

RESULTS AND DISCUSSION

Immunological Pathophysiology of Vitiligo and the Jak/Stat Pathway

The modern understanding of the pathophysiology of vitiligo establishes the disease as an autoimmune disorder mediated by cytotoxic CD8⁺ T cells that destroy melanocytes.^{7,8,12} Genetic predisposition and increased melanocyte vulnerability to stressors lead to a specific immune response against these melanocytes, associated with an interferon-gamma-mediated immune signature.¹³

IFN- γ plays a central role in the pathogenesis of vitiligo, signaling through the JAK-STAT pathway. 10 This pathway is activated by key cytokines, including IFN- γ and interleukin 15 (IL-15), which sustain cytotoxic T-cell infiltration and melanocyte apoptosis.⁴ Keratinocytes detect IFN- γ in vitiligo lesions and are the main producers of Monokine Induced by Gamma Interferon (CXCL9) and Interferon Gamma-Induced Protein 10 (CXCL10), chemokines that promote T-cell recruitment. 14 This process creates a positive feedback loop: autoreactive CD8⁺ T cells engage melanocytes and promote disease progression through the local production of IFN- γ , and IFN- γ -induced chemokines are then secreted by surrounding keratinocytes to recruit even more T cells to the skin.⁸

In addition, JAK inhibitors target the pathway through which resident memory T cells become dependent on IL-15 for their survival as they establish residence in the epidermis. 1 These autoreactive cells are precisely those responsible for disease relapse after treatment discontinuation. In view of this, new therapeutic strategies focus specifically on eliminating these cells to promote lasting benefit.⁸

Furthermore, new studies suggest that the immune response in vitiligo is not restricted to IFN- γ , but may be associated with a type 2 immune response, evidenced by increased IL-13 production. 1 This immunological complexity highlights the need for therapeutic approaches capable of modulating multiple inflammatory pathways.

Topical Jak Inhibitors: Ruxolitinib

Topical ruxolitinib represents the first JAK inhibitor approved for the treatment of vitiligo. 4,10,13 As a JAK1 and JAK2 inhibitor, ruxolitinib cream has demonstrated consistent and clinically significant efficacy in phase 2 and 3 clinical trials.^{10,14}

Specifically, in the randomized controlled phase II study, ruxolitinib cream resulted in repigmentation over 52 weeks in adults with vitiligo. 14 Among patients who applied 1.5% ruxolitinib cream twice daily, 45% achieved at least a 50% improvement from baseline in the Facial Vitiligo Area Scoring Index (F-VASI) at week 24 (primary endpoint), and 30% achieved a reduction of at least 75%, with continued improvement through week 52.¹⁰

Additionally, 45% of patients with a baseline total body surface area of 20% or less (and who therefore could treat all depigmented skin) who received 1.5% ruxolitinib cream twice daily achieved a 50% or greater improvement from baseline in the Total Vitiligo Area Scoring Index (T-VASI50; total body assessment, including the face) at week 52.¹⁴

Two phase III clinical trials (Topical Ruxolitinib Evaluation in Vitiligo Study 1 and Study 2 - TRuE-V1 and TRuE-V2) were conducted in adolescents and adults with nonsegmental vitiligo. 10 These studies confirmed the efficacy of topical ruxolitinib, demonstrating consistent and clinically significant repigmentation, particularly in facial lesions.^{4,10}

The safety profile of topical ruxolitinib is favorable, with application-site reactions being rare during treatment.¹⁴ Topical agents are generally well tolerated with minimal systemic absorption, making them an attractive option for patients with localized disease, especially involving the face and less than 10% of body surface area.^{1,4}

In addition, topical ruxolitinib is approved in Europe through the European Medicines Agency (EMA) and in the United States through the Food and Drug Administration (FDA), for use in adolescents aged 12 years and older and adults with nonsegmental vitiligo and facial involvement.¹³ To date, the medication has not yet been approved by the Brazilian Health Regulatory Agency (ANVISA) for use in Brazil.

Systemic Jak Inhibitors

As systemic therapy, oral JAK inhibitors may have advantages over topical treatments for rapidly stabilizing the disease in patients with very active disease and when larger areas are involved. The following stand out:

- **Baricitinib:** a recent randomized clinical trial evaluated the combination of baricitinib and phototherapy in adults with active vitiligo. Baricitinib, a JAK1/JAK2 inhibitor, demonstrated clinical benefit in the management of vitiligo, particularly when combined with NB-UVB phototherapy. This study highlights the potential of systemic therapies for patients with extensive or rapidly progressive disease.

- **Ritlecitinib:** ritlecitinib: a JAK3/TEC inhibitor (tyrosine kinase expressed in hepatocellular carcinoma), showed promising efficacy, either as monotherapy or in combination with NB-UVB phototherapy. Ongoing phase III clinical trials should further define its role in clinical practice.

- **Upadacitinib and Povorcitinib:** these JAK1 inhibitors have also demonstrated promising efficacy in clinical studies. Upadacitinib has been described in previous reports as providing clinical benefit in the management of vitiligo, and povorcitinib is being evaluated in phase II and III clinical trials.

- **Other agents:** tofacitinib, abrocitinib, and other JAK inhibitors are currently under investigation or being used off-label in clinical practice. Although JAK inhibitors demonstrate high rates of repigmentation, optimal results may take several months to years.

JAK inhibitors exhibit variable safety profiles depending on selectivity, formulation, and dose. While topical agents are generally well tolerated with minimal systemic absorption, oral JAK inhibitors require monitoring due to potential risks of infection, hematological abnormalities, and cardiovascular events.

Combination of Jak Inhibitors With Phototherapy

The influence of environmental ultraviolet (UV) exposure on repigmentation in patients receiving immunomodulatory medications remains largely unexplored. However, the combined effect of phototherapy with JAK inhibitors is striking and suggests that targeted immune treatment may still require additional melanocyte stimulation for repigmentation.

Several studies have demonstrated that the combination of JAK inhibitors with NB-UVB phototherapy may produce superior results compared with monotherapy. This combined approach allows both suppression of the autoimmune response and stimulation of melanocyte proliferation and migration from follicular reservoirs.

Other Emerging Immunomodulatory Therapies

Considering recent advances in the understanding of the pathophysiology of vitiligo, a number of other immunomodulatory therapies have been developed and studied, including:

- **Biological Therapies:** monoclonal antibodies targeting specific cytokines are being investigated for the treatment of vitiligo. Anifrolumab, an anti-type I interferon monoclonal antibody, is being evaluated in phase II and III clinical trials. However, treatment results with biologics have been more variable compared with JAK inhibitors.

- **Mesenchymal Stem Cells (MSCs):** MSCs regulate cytokine secretion and the balance of T-cell subsets, making them a promising cell-based treatment option for autoimmune diseases. The induction of MSC-mediated immunomodulation is complex and occurs through contact-dependent mechanisms and soluble extracellular vesicle (EV) mediators containing various growth factors and cytokines with anti-inflammatory effects on the cutaneous immune response. Although promising, these therapies

still require additional in vitro studies and extensive clinical research involving a larger population.⁵

- **Other Approaches:** minimally invasive procedures, such as fractional lasers or microneedling, may help achieve optimal treatment outcomes when appropriately used. In addition, identifying alternative melanocyte stimulants beyond UV light remains crucial for the future management of vitiligo.⁹

International Consensus Guidelines

The International Vitiligo Task Force, composed of 42 international vitiligo experts and four patient representatives, developed the most comprehensive and up-to-date consensus recommendations for the diagnosis and management of vitiligo in 2023.^{6,17} These guidelines represent a milestone in the standardization of global disease management, emphasizing the importance of shared decision-making between physician and patient, with detailed explanations regarding prognosis and expected therapeutic outcomes based on clinical examination.¹⁷

The guidelines establish that assessment of disease activity remains a cornerstone of the personalized approach to patients with vitiligo.¹⁷ Treatment should be discussed considering different goals: disease stabilization versus repigmentation, recognizing that vitiligo generally requires a long-term approach.¹⁷ Treatment choice is individualized based on disease extent, stability, cost, accessibility, and response to previous therapy.¹⁸

The recommendations summarize the most recent evidence on the use of topical corticosteroids (TCS), topical calcineurin inhibitors (TCI), and topical JAK inhibitors (tJAKi).⁶ Topical calcineurin inhibitors, particularly tacrolimus, are recommended as first-line therapy for vitiligo in children, adolescents, and young adults, with a robust level of evidence (A1-A2, level 1-2).¹⁹ Pimecrolimus is also supported as first-line therapy, although with a slightly lower level of evidence than tacrolimus (A2, level 2).¹⁹ In turn, 1.5% ruxolitinib cream, as previously discussed, emerges as an important tool for achieving consistent and clinically significant repigmentation, particularly in facial lesions.⁴

Guidelines from the European Dermatology Forum and the Vitiligo Japanese Task Force recommend combination therapies with phototherapy as first-line treatment for vitiligo.²⁰ The European Dermatology Forum recommends NB-UVB, excimer light combined with topical corticosteroids or topical immunomodulators, while the Japanese task force recommends NB-UVB, UV-A photochemotherapy combined with topical corticosteroids or topical vitamin D analogues. 20

Along these lines, the guidelines recommend, during the unstable phase of the disease, aggressive therapy aimed at halting progression, using low-dose oral minipulse therapy together with standard topical therapy with steroids, topical calcineurin inhibitors, or vitamin D3 analogues.¹⁸ Stabilization therapy in some patients may include other immunosuppressive agents such as low-dose methotrexate, azathioprine, cyclosporine, or mycophenolate mofetil. 18 JAK inhibitors may be used as stabilization therapy.¹⁸

Additionally, interventional approaches, including surgical therapies, are presented for specific indications, particularly for stable vitiligo lesions in which medical therapy fails to induce repigmentation.^{6,18}

Challenges and Future Perspectives

Despite significant advances, gaps in the clinical evidence remain, especially regarding long-term outcomes and pediatric populations.⁷ Bibliometric analysis of research on vitiligo treatment from 2005 to 2025 revealed a gradual transition from traditional therapies, such as corticosteroids and phototherapy, to targeted immune strategies.⁷ Annual scientific output increased at a compound growth rate of 7.27%, reflecting the growing interest in this area.⁷

Disease relapse after treatment discontinuation remains a significant challenge.^{4,8} New therapeutic strategies focus on eliminating autoreactive resident memory T cells to promote lasting benefit.⁸ In this context, identifying predictive biomarkers of patients who are responsive to treatment with JAK inhibitors would be of considerable value and warrants continued investigation.¹⁴

CONCLUSION

Advances in the understanding of the immunological pathophysiology of vitiligo, especially the role of the JAK/STAT pathway and IFN- γ -mediated cytotoxic CD8⁺ T cells, have transformed the understanding of the disease and enabled the development of targeted therapies. This progress represents a paradigm shift in the management of vitiligo, with the transition from empirical approaches to more specific immunomodulatory strategies.

JAK inhibitors, particularly topical ruxolitinib, stand out as the main recent therapeutic innovation, demonstrating consistent efficacy in repigmentation, especially in facial areas. Systemic formulations and therapies combined with NB-UVB phototherapy have also shown promising results, although they still depend on long-term evidence to consolidate their efficacy and safety.

Despite these advances, some important challenges remain, including the need for long-term data, the identification of predictive biomarkers of response, and strategies for relapse prevention. Further investigation of these issues will be essential to optimize therapeutic outcomes and consolidate the new approaches in the treatment of vitiligo.

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