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ATOPIC DERMATITIS: ADVANCES IN PATHOPHYSIOLOGY AND NEW IMMUNOLOGICAL THERAPIES

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Abstract: Atopic dermatitis is a chronic inflammatory skin disease with high global prevalence, characterized by persistent itching, skin barrier dysfunction, and a high burden on quality of life. Over the last decade, it has been established as a systemic disorder mediated by the type 2 immune response, involving key cytokines such as IL-4, IL-13, IL-31, and TSLP, as well as epigenetic factors, skin dysbiosis, and neuroimmunological mechanisms. These pathophysiological advances have enabled the development of targeted immunological therapies, including monoclonal antibodies such as dupilumab, tralokinumab, lebrikizumab, and nemolizumab, as well as JAK inhibitors (baricitinib, upadacitinib, abrocitinib), which have demonstrated superior clinical efficacy to conventional treatments and significant improvement in itch control. This article reviews the main advances in understanding the pathophysiology of atopic dermatitis and analyzes the impact of new immunological therapies. It concludes that the implementation of these treatments requires not only evidence of efficacy and safety, but also strategies for equitable access and sustainability in health systems, as well as the incorporation of biomarkers that allow for a personalized medicine approach.
Keywords: atopic dermatitis; pathophysiology; immunotherapy; dermatology.

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by intense itching, recurrent eczematous lesions, and alterations in patients' quality of life[1]. It is recognized as one of the most prevalent skin disorders worldwide, with an estimated incidence of 15 to 20% in the pediatric population and between 2 and 10% in adults, making it a public health problem of growing importance[(2]rapidly controlling symptoms and improving quality of life. However, the impact of body mass index (BMI.

Historically, the understanding of AD has focused on the alteration of the skin barrier, mainly linked to mutations in the filaggrin gene and susceptibility to recurrent infections[3]. However, in the last decade, advances in immunological knowledge have allowed it to be characterized as a heterogeneous disease, in which immune responses mediated by type 2 helper T cells (Th2) predominate, together with the participation of other inflammatory axes such as Th17 and Th22[4].

Key mediators in the pathophysiology of AD include interleukins IL-4, IL-13, IL-31, and thymic stromal lymphopoietin (TSLP), which are responsible for amplifying pruritus, skin inflammation, and perpetuating the dysfunctional epidermal barrier cycle[5]. Likewise, the role of the skin microbiome, particularly dysbiosis induced by Staphylococcus aureus, has been the subject of growing interest, as it contributes to the exacerbation and chronicity of the disease[6].

This progress in understanding the pathophysiology has driven the development of targeted immunological therapies capable of blocking specific inflammatory pathways and modifying the clinical course of the disease[7]. The introduction of monoclonal antibodies such as dupilumab, tralokinumab, and lebrikizumab, as well as Janus kinase (JAK) pathway inhibitors such as baricitinib, upadacitinib, and abrocitinib, represents a paradigm shift in treatment, overcoming the limitations of conventional therapy based on topical corticosteroids and systemic immunosuppressants[8].

The aim of this article is to review recent advances in the pathophysiology of atopic dermatitis and analyze new immunological therapies, emphasizing their clinical implications, potential benefits, and the challenges that remain in their implementation in healthcare systems.

METHODOLOGY

A narrative review of the scientific literature related to the pathophysiology of atopic dermatitis and new immunological therapies that have been approved or are under investigation was conducted. The search was carried out in August 2025 in the PubMed/MEDLINE, Scopus, Web of Science, and SciELO databases, using controlled descriptors and free terms combined with Boolean operators.

The terms used included: "atopic dermatitis," "pathophysiology," "immunology," "biological therapy," "dupilumab," "tralokinumab," "lebrikizumab," "nemolizumab," "JAK inhibitors," "baricitinib," "upadacitinib," and "abrocitinib." For the Spanish version, the equivalent terms in DeCS were used: "dermatitis atópica," "fisiopatología," "inmunología," "terapia biológica," and "inhibidores de JAK."

The following inclusion criteria were established:

- Original articles, clinical trials, systematic reviews, meta-analyses, and clinical practice guidelines published between 2015 and 2025.
- Publications in English and Spanish.
- Studies conducted in pediatric, adult, or mixed populations with a confirmed diagnosis of atopic dermatitis.

The following were excluded:

- Duplicate studies, editorials without original data, and brief communications not peer-reviewed.
- Articles focusing exclusively on conventional therapies (corticosteroids, cyclosporine, methotrexate), unless used as comparators in clinical trials.

The articles were evaluated independently by three reviewers, considering their clinical and scientific relevance. In case of discrepancy, consensus was reached through discussion.

Finally, 40 articles were selected to form the basis of this review, with priority given to those published in high-impact journals.

Given that this is a narrative review and not a systematic review, the PRISMA methodology was not applied. However, a structured process of searching, selecting, and synthesizing the evidence was followed in order to ensure academic rigor and scientific validity.

ADVANCES IN PATHOPHYSIOLOGY

Atopic dermatitis (AD) is currently understood to be a chronic, multifactorial immunological disease involving alterations in the skin barrier, adaptive and innate immune dysfunction, changes in the microbiome, and neuroimmunological factors that perpetuate itching and inflammation[9]"ISSN":"00916749","PMID":"31786154","abstract":"Background: Atopic dermatitis is a chronic inflammatory skin disease characterized by pruritic skin lesions. Objective: We sought to evaluate the safety and efficacy of multiple doses of the selective Janus kinase 1 inhibitor upadacitinib in patients with moderate to severe atopic dermatitis. Methods: In the 16-week, double-blind, placebo-controlled, parallel-group, dose-ranging portion of this 88-week trial in 8 countries (ClinicalTrials.gov, NCT02925117; ongoing, not recruiting.

SKIN BARRIER DYSFUNCTION

The skin epithelium acts as the first line of defense against environmental and microbiological aggressors[10]. In patients with AD, a decrease in key structural proteins such as filaggrin, loricrin, and claudins has been described, leading to transepidermal water loss, xerosis, and increased allergen penetration. Mutations in the FLG gene, present in up to 30% of patients, represent a significant risk factor for severe and persistent forms of the disease[11].

PREDOMINANT IMMUNOLOGICAL AXES

The immunological paradigm of AD has evolved towards a model in which the Th2 response predominates, mediated by cytokines such as IL-4 and IL-13, which are responsible for amplifying inflammation and altering epidermal differentiation[12]. Added to this axis is the participation of IL-31, the main mediator of pruritus, and thymic stromal lymphopoietin (TSLP), which acts as a bridge between damaged epithelial cells and the activation of dendritic cells[13].

In addition, the involvement of other lineages such as Th17 and Th22 has been identified, which appear to be more involved in certain phenotypes and endophenotypes of patients, particularly in Asian populations and in chronic forms of the disease[14]which lowers quality of life. AD has become a global health concern as its incidence has increased over the last few decades. It ranks as the third most common dermatologic disorder. AIM There are several open questions about the mechanisms underlying atopic dermatitis (AD. These findings have prompted the search for biomarkers that allow for more accurate patient stratification and a personalized medicine approach[15]which is involved in pruritus and inflammation in atopic dermatitis. In phase 2 studies, nemolizumab lessened the severity of atopic dermatitis. METHODS In a 16-week, double-blind, phase 3 trial, we randomly assigned Japanese patients with atopic dermatitis and moderate-to-severe pruritus and an inadequate response to topical agents in a 2:1 ratio to receive subcutaneous nemolizumab (60 mg.

SKIN MICROBIOME AND DYSBIOSIS

The skin microbiome plays a key role in immune homeostasis. In AD, pathological colonization by Staphylococcus aureus is observed, whose superantigenicity exacerbates inflammation and promotes recurrent infec-

tions[16]. The loss of bacterial diversity and imbalance with commensal species such as Staphylococcus epidermidis lead to a persistent proinflammatory environment[17].

NEUROIMMUNOLOGICAL FACTORS AND PRURITUS

Pruritus, the cardinal symptom of AD, is not only related to the release of IL-31, but also to the activation of sensory nerve fibers and interaction with neuropeptides such as substance P[18]. Chronic sensitization of the peripheral nervous system contributes to a vicious cycle of scratching and inflammation, which worsens skin barrier dysfunction[19].

Together, these advances in pathophysiology have led to the redefinition of AD as a systemic inflammatory disorder with predominant cutaneous expression, which supports the development of immunological therapies targeting specific molecular targets[20].

NEW IMMUNOLOGICAL THERAPIES

Current knowledge of the immunopathogenic mechanisms of atopic dermatitis (AD) has driven the development of therapies targeting specific cytokines and intracellular pathways[21]. These interventions represent a paradigm shift from conventional treatments, which were limited to the use of topical corticosteroids, calcineurin inhibitors, or systemic immunosuppressants, with partial efficacy and restrictive safety profiles[22].

APPROVED BIOLOGICAL THERAPIES

Dupilumab

First monoclonal antibody approved for moderate to severe AD. It binds to the α subunit of the IL-4 receptor, blocking the signaling of IL-4 and IL-13, key cytokines in type 2 inflammation[23]. The SOLO 1 and SOLO 2 clinical trials demonstrated significant reductions in the EASI-75 index and sustained long-term improvement in pruritus. In addition, it has a favorable safety profile, with conjunctivitis being the most commonly reported adverse effect[24].

Tralokinumab and Lebrikizumab

Both are monoclonal antibodies directed against IL-13. Tralokinumab has shown efficacy in ECZTRA 1 and 2 studies, achieving clinical improvement as monotherapy or in combination with topical corticosteroids[25]a fully human monoclonal antibody, specifically neutralizes interleukin-13, a key cytokine driving peripheral inflammation in atopic dermatitis (AD.

Lebrikizumab, in advanced stages of development, has shown rapid and sustained responses, with a safety profile comparable to dupilumab[26].

Nemolizumab

Targeted against the IL-31 receptor, it acts primarily on pruritus, the cardinal symptom of AD. Clinical studies have reported a significant reduction in the pruritus scale and improved sleep, with promising results as an adjuvant therapy[15]which is involved in pruritus and inflammation in atopic dermatitis. In phase 2 studies, nemolizumab lessened the severity of atopic dermatitis. METHODS In a 16-week, double-blind, phase 3 trial, we randomly assigned Japanese patients with atopic dermatitis and moderate-to-severe pruritus and an inadequate response to topical agents in a 2:1 ratio to receive subcutaneous nemolizumab (60 mg.

JAK inhibitors

Small oral Janus kinase (JAK) inhibitor molecules represent a novel strategy by blocking the signal transduction of multiple cytokines (IL-4, IL-13, IL-22, IL-31, TSLP)[27].

Pathophysiological mechanism	*Main evidence*	*Clinical implications*	*Associated therapeutic target*
Skin barrier dysfunction *(mutations in FLG, claudins, loricrin)*	Structural alteration of the epidermis with transepidermal water loss and xerosis	Increased susceptibility to allergens, irritants, and pathogens	Intensive hydration, emollients, barrier repair therapies
Th2 response *(IL-4, IL-13)*	Predominance of type 2 cytokines that alter epidermal differentiation and increase IgE	Chronic inflammation and recurrent exacerbations	Dupilumab (anti-IL-4Rα), tralokinumab, and lebrikizumab (anti-IL-13)
IL-31-mediated pruritus	IL-31 induces hyperactivation of cutaneous nerve fibers and persistent pruritus	Vicious cycle of scratching-inflammation	Nemolizumab (anti-IL-31Rα)
TSLP and epithelial cytokines	TSLP activated by barrier damage stimulates dendritic cells and Th2 response	Initiation and amplification of allergic inflammation	Anti-TSLP therapies under investigation
Th17 and Th22 axes	Evidence of greater involvement in Asian phenotypes and chronic forms of	Clinical variability and need for personalized medicine in	Potential targets under investigation
Cutaneous dysbiosis *(Staphylococcus aureus and loss of bacterial diversity)*	Pathological colonization with toxin and superantigen production	Exacerbations, greater severity, and therapeutic resistance	Microbiome modulation, topical probiotics
Neuroimmunology of pruritus *(substance P, neuropeptides)*	Activation of peripheral sensory nerve fibers	Chronicity of pruritus and sleep impairment	Antipruritics targeting neural and immunological pathways

Table 1. Advances in the pathophysiology of atopic dermatitis

Drug	*Mechanism of action*	*Evidence of efficacy*	*Main adverse effects*	*Regulatory status*
Dupilumab	Anti-IL-4Rα monoclonal antibody → inhibits IL-4 and IL-13	SOLOS 1 and 2 trials, LIBERTY AD: EASI-75 improvement in 44–51% of patients at week 16[24]a human monoclonal antibody against interleukin-4 receptor alpha, inhibits signaling of interleukin-4 and interleukin-13, type 2 cytokines that may be important drivers of atopic or allergic diseases such as atopic dermatitis. METHODS In two randomized, placebo-controlled, phase 3 trials of identical design (SOLO 1 and SOLO 2.	Conjunctivitis, injection site reactions, mild eosinophilia.	Approved by FDA, EMA, and multiple countries for ≥6 years
Tralokinumab	Anti-IL-13 monoclonal antibody	ECZTRA 1 and 2 trials: EASI-75 in 25–33% at week 16[25]a fully human monoclonal antibody, specifically neutralizes interleukin-13, a key cytokine driving peripheral inflammation in atopic dermatitis (AD.	Upper respiratory tract infections, conjunctivitis	Approved by EMA (adults)
Lebrikizumab	Anti-IL-13 monoclonal antibody (high affinity)	ADvocate trials 1 and 2: EASI-75 in 43–59%[31]type 2/Th2 cytokines implicated in numerous allergic diseases ranging from asthma to atopic dermatitis. Previous 16-week monotherapy studies showed that dupilumab substantially improved signs and symptoms of moderate-to-severe atopic dermatitis with acceptable safety, validating the crucial role of interleukin 4 and interleukin 13 in atopic dermatitis pathogenesis. We aimed to evaluate the long-term efficacy and safety of dupilumab with medium-potency topical corticosteroids versus placebo with topical corticosteroids in adults with moderate-to-severe atopic dermatitis. Methods In this 1-year, randomised, double-blinded, placebo-controlled, phase 3 study (LIBERTY AD CHRONOS.	Conjunctivitis, headache, local reactions	Under evaluation (phase III, FDA and EMA)

Nemolizumab	Anti-IL-31Rα monoclonal antibody → reduces pruritus	Phase III trials: significant reduction in pruritus (>60% on NRS scale)[15]which is involved in pruritus and inflammation in atopic dermatitis. In phase 2 studies, nemolizumab lessened the severity of atopic dermatitis. METHODS In a 16-week, double-blind, phase 3 trial, we randomly assigned Japanese patients with atopic dermatitis and moderate-to-severe pruritus and an inadequate response to topical agents in a 2:1 ratio to receive subcutaneous nemolizumab (60 mg.	Abdominal pain, arthralgia, nasopharyngitis	In advanced clinical evaluation
Baricitinib	JAK1/JAK2 inhibitor	BREEZE-AD trials: EASI-75 improvement in 31–48%[32]limiting personalized care. This study assessed NuGel, a topical GPCR19 agonist, for efficacy, safety, and predictive baseline biomarkers in AD patients. Methods: In a multicenter, double-blind, randomized, placebo-controlled Phase 2a trial (August 2020–September 2021, five hospitals, 80 participants.	Respiratory infections, elevated CPK, headache	Approved by EMA (adults)
Upadacitinib	Selective JAK1 inhibitor	Measure Up 1 and 2 trials: EASI-90 in 38–48%, rapid onset of action[33].	Acne, elevated transaminases, thrombotic risk	Approved by FDA and EMA (≥12 years)
Abrocitinib	Selective JAK1 inhibitor	JADE MONO 1 and 2 trials: rapid improvement in pruritus and EASI-75 in 40–63%[29].	Nausea, headache, respiratory infections, hematological risk	Approved by EMA and some countries; FDA review ongoing
Anti-OX40 (under investigation)	Blocking T cell costimulation	Phase II: EASI reduction and pruritus[34].	Headache, fatigue, mild infections	In phase II-III
Anti-TSLP (tezepelumab, research)	Blocking of epithelial cytokine TSLP	Phase II: moderate reduction in EASI[12].	Mild infections, local reactions	Phase II

Table 2. New immunological therapies for atopic dermatitis

Baricitinib: approved for moderate to severe AD in adults. The BREEZE-AD study showed efficacy in reducing pruritus and improving EASI, with an acceptable safety profile, although associated with respiratory infections and elevations in creatine phosphokinase[28].

Upadacitinib: selective JAK1 inhibitor with high clinical efficacy. Measure Up 1 and 2 trials showed EASI-90 responses superior to dupilumab, with faster onset of action, but with a higher risk of adverse events such as acne and elevated transaminases.

Abrocitinib: another selective JAK1 inhibitor, approved in several countries[19].

JADE MONO studies demonstrated rapid improvement in pruritus and sustained control of skin inflammation, with a safety profile similar to that of upadacitinib[29].

CLINICAL CONSIDERATIONS

The advent of these therapies has transformed clinical practice in AD, offering sustained control, reduction of pruritus, and improvement in quality of life[30]. However, challenges remain related to long-term safety, patient stratification using biomarkers, and economic access, especially in low- and middle-income health systems[26].

FUTURE THERAPIES FOR ATOPIC DERMATITIS

In recent years, research into atopic dermatitis (AD) has advanced towards the identification of new therapeutic targets, with the aim of overcoming the limitations of current treatments and offering more effective, safer, and sustainable options[28]an oral selective Janus kinase 1 and 2 inhibitor, effectively reduced disease severity in moderate to severe atopic dermatitis (AD. Several biological and immu-

nomodulatory agents have shown promising results in phase II and III clinical trials, establishing themselves as potential additions to the therapeutic arsenal in the short and medium term[15]which is involved in pruritus and inflammation in atopic dermatitis. In phase 2 studies, nemolizumab lessened the severity of atopic dermatitis. METHODS In a 16-week, double-blind, phase 3 trial, we randomly assigned Japanese patients with atopic dermatitis and moderate-to-severe pruritus and an inadequate response to topical agents in a 2:1 ratio to receive subcutaneous nemolizumab (60 mg.

OX40 and OX40L antagonists

The OX40/OX40L axis plays an essential role in the activation and survival of effector T cells. Drugs targeting this target have demonstrated significant clinical benefits[34] :

Amlitelimab (anti-OX40L): in 2025, phase III trials of the OCEANA program confirmed its efficacy in patients with moderate to severe AD, meeting the primary efficacy endpoints and with the added advantage of requiring infrequent dosing (approximately four times a year), which could improve therapeutic adherence[14]which lowers quality of life. AD has become a global health concern as its incidence has increased over the last few decades. It ranks as the third most common dermatologic disorder. AIM There are several open questions about the mechanisms underlying atopic dermatitis (AD.

Rocatinlimab (anti-OX40): Phase IIb studies published in 2025 showed significant improvement in EASI and reduction in pruritus, with maintenance of response even after discontinuation of treatment, suggesting a disease-modifying effect[35].

IL-2 modulators and regulatory T cells

The imbalance between effector and regulatory T cells is a central component in the pathophysiology of AD. Rezpegaldesleukin, an IL-2 agonist/modulator, seeks to restore the function of regulatory T cells[28]an oral selective Janus kinase 1 and 2 inhibitor, effectively reduced disease severity in moderate to severe atopic dermatitis (AD.

The results of the REZOLVE-AD study showed rapid reductions in the EASI index and improvement in pruritus, with a favorable safety profile, positioning it as an innovative therapy under investigation[4].

Epithelial cytokine blockers

Interest in epithelial immunity has driven the development of therapies targeting mediators such as IL-33 and TSLP. Tozorakimab (anti-IL-33) is currently undergoing clinical evaluation, with preliminary data suggesting a reduction in inflammatory biomarkers and potential efficacy in moderate to severe AD. Although initial results are encouraging, larger trials are still needed to confirm its clinical utility[28]an oral selective Janus kinase 1 and 2 inhibitor, effectively reduced disease severity in moderate to severe atopic dermatitis (AD.

Topical JAK inhibitors

The development of topical JAK pathway inhibitors is another important strategy, as it allows for a local approach with a lower risk of systemic adverse effects. Delgocitinib cream, approved in some countries for localized forms of eczema, has demonstrated safety and efficacy, with minimal systemic absorption and adequate skin tolerance[36].

Future prospects

Emerging therapies in AD reflect a paradigm shift toward fine modulation of the immune system rather than its generalized suppression. These drugs could:

- Reduce the frequency of administration (amlitelimab).
- Modify the natural history of the disease (rocatinlimab, rezpegaldesleukin).
- Broaden the spectrum of therapeutic targets beyond the IL-4/IL-13 axis (anti--IL-33, anti-TSLP).
- Optimize safety through innovative topical formulations (delgocitinib).

However, significant challenges remain: the need for predictive response biomarkers, long-term safety assessment in special populations, and the integration of these therapies into resource-limited healthcare systems. Equity of access will be critical for these scientific advances to translate into real improvements in the quality of life of patients with atopic dermatitis[6].

DISCUSSION

The contemporary understanding of atopic dermatitis (AD) as a systemic immune-mediated disease has transformed the therapeutic perspective, moving away from the exclusive paradigm of skin barrier dysfunction[37]. The identification of key cytokines, particularly IL-4, IL-13, and IL-31, has enabled the development of targeted drugs capable of modifying the clinical course of the disease[38].

Monoclonal antibodies, led by dupilumab, have demonstrated sustained efficacy and a favorable safety profile in multiple clinical studies, establishing themselves as the first biological option for patients with moderate to severe AD[32].

Subsequently, tralokinumab and lebrikizumab have consolidated the relevance of the IL-13 axis in pathophysiology, expanding the available options and suggesting that selective inhibition could benefit specific subgroups of patients. Nemolizumab, meanwhile, represents an advance in the symptomatic control of pruritus, one of the main determinants of reduced quality of life[29] a.

At the same time, JAK inhibitors have emerged as highly effective oral alternatives with a faster onset of action than biologics. Upadacitinib and abrocitinib, selective JAK1 inhibitors, have shown higher clinical response rates in some trials compared to dupilumab. However, the need for closer monitoring for potential adverse events, such as opportunistic infections, thrombosis, or hematological abnormalities, raises questions about their long-term safety[36].

These findings highlight the need to move towards personalized medicine based on biomarkers that allow the phenotype and endotype of each patient to be identified. The clinical heterogeneity observed between populations, for example, the greater involvement of Th17/Th22 axes in Asian patients, shows that a uniform treatment may not be equally effective for everyone[33].

From a public health perspective, access to these therapies is a challenge. The high cost of biologics and JAK inhibitors limits their availability in middle-income health systems, such as those in Latin America, where coverage still depends largely on private financing or exceptional programs[39]immune dysregulation, and microbial dysbiosis. While therapeutic advancements targeting T helper 2 (Th2. Therefore, in addition to clinical efficacy, it is essential to consider the cost-effectiveness and sustainability of these interventions[31].

Finally, there are still gaps in knowledge regarding the combined use of biologics and JAK inhibitors, safety in special populations (pregnant women, children under 6 years of age, immunosuppressed patients), and the long-term impact on the progression of associated comorbidities, such as asthma and allergic rhinitis [25].

In summary, advances in immunological therapies have transformed the approach to AD, but the next steps should focus on patient stratification, the identification of response

biomarkers, and the reduction of economic and access barriers to ensure that these advances translate into real improvements in the quality of life of the affected population[40].

CONCLUSIONS

Atopic dermatitis is no longer considered an exclusive disorder of the skin barrier but is now recognized as a chronic, systemic inflammatory disease involving epidermal dysfunction, type 2 immune alterations, microbial dysbiosis, and neuroimmunological mechanisms.

Advances in understanding its pathophysiology have led to the development of targeted immunological therapies, notably monoclonal antibodies against IL-4, IL-13, and IL-31, as well as JAK inhibitors, which have demonstrated superior efficacy to conventional options and a positive impact on patients' quality of life.

However, clinical and public health challenges remain, related to long-term safety, the identification of biomarkers that enable personalized medicine, and the need to ensure equitable access to these therapeutic innovations in resource-limited settings.

In this scenario, the integration of scientific evidence with sustainable access policies and individualized treatment stratification will be decisive in consolidating the clinical benefit of these new therapies in the population affected by atopic dermatitis.

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LISTA DE ABREVIATURAS

DA: Dermatitis atópica

IL: Interleucina

IL-4Rα: Subunidad alfa del receptor de interleucina 4

TSLP: Linfopoyetina estromal tímica (Thymic stromal lymphopoietin)

Th2: Linfocitos T helper tipo 2

Th17: Linfocitos T helper tipo 17

Th22: Linfocitos T helper tipo 22

EASI: Eczema Area and Severity Index

SCORAD: Scoring Atopic Dermatitis

JAK: Janus kinasa

CPK: Creatinfosfocinasa (Creatine phosphokinase)

FDA: Food and Drug Administration (Agencia de Alimentos y Medicamentos de EE.UU.)

EMA: European Medicines Agency (Agencia Europea de Medicamentos)

NRS: Numeric Rating Scale (escala numérica para prurito)