DOI: https://doi.org/10.17816/KMJ680075 EDN: KFHMJP

Atopic Dermatitis: Pathogenetic Mechanisms and Role of Biomarkers in Diagnosis

Irina Kh. Borukaeva, Farida Kh. Temirzhanova, Kazbek Y. Shkhagumov, Zalina Kh. Abazova, Amina Z. Getigezheva

Kabardino-Balkarian State University, Nalchik, Russia

ABSTRACT

Atopic dermatitis is a chronic inflammatory skin disorder that typically develops in childhood and often persists into adulthood. Its multifactorial pathogenesis involves genetic predisposition, epidermal barrier dysfunction, immune dysregulation with a predominance of the Th2 response, and environmental and microbiome-related influences. One of its key genetic contributors is filaggrin deficiency due to gene mutations, which leads to decreased of natural moisturizing factor synthesis and increased stratum corneum permeability. Other significant mechanisms include impaired tight junction integrity and epidermal protease–antiprotease activity imbalance. The immune component of atopic dermatitis is characterized by increased levels of cytokines such as interleukin (IL)-4, IL-13, and IL-31, which contribute to inflammation and further skin barrier impairment. Cutaneous microbiota dysbiosis, particularly overgrowth of *Staphylococcus aureus*, also plays a crucial role in disease exacerbation. Despite advances in understanding the molecular and cellular mechanisms of atopic dermatitis, its diagnosis remains clinical, with limited use of laboratory biomarkers owing to the lack of universal, sensitive, and specific indicators. This review addresses key aspects of epidermal barrier function, genetic mutations, immune responses, and the role of the skin microbiome. Special attention is given to filaggrin gene mutations and the potential of cytokines and other serological markers as diagnostic and prognostic biomarkers. Analysis identified potential targets for diagnosis and disease severity assessment. However, large-scale studies are required to validate their clinical utility. This is especially relevant in personalized medicine and treatment optimization for patients with atopic dermatitis.

Keywords: atopic dermatitis; skin barrier; filaggrin; immune cytokines; keratinocytes; skin microbiome; immunoglobulin E.

To cite this article:
Borukaeva IKh, Temirzhanova FKh, Shkhagumov KYu, Abazova ZKh, Getigezheva AZ. Atopic dermatitis: pathogenetic mechanisms and role of biomarkers in diagnosis. *Kazan Medical Journal.* 2025;106(4):609–618. DOI: 10.17816/KMJ680075 EDN: KFHMJP

Submitted: 20.05.2025 **Accepted**: 03.06.2025 **Published online**: 23.07.2025

DOI: https://doi.org/10.17816/KMJ680075 EDN: KFHMJP

Атопический дерматит: патогенетические механизмы и роль биомаркеров в диагностике

И.Х. Борукаева, Ф.Х. Темиржанова, К.Ю. Шхагумов, З.Х. Абазова, А.З. Гетигежева

Кабардино-Балкарский государственный университет им. Х.М. Бербекова, г. Нальчик, Россия

АННОТАЦИЯ

Атопический дерматит — это хроническое воспалительное заболевание кожи, возникающее преимущественно в детском возрасте, однако часто сохраняющееся и у взрослых. Патогенез атопического дерматита является многофакторным и включает генетическую предрасположенность, нарушение функции эпидермального барьера, иммунную дисрегуляцию с преобладанием Th2-ответа, а также влияние факторов окружающей среды и микробиоты кожи. Одним из ключевых генетических факторов считается дефицит филаггрина, обусловленный мутациями в соответствующем гене, что ведёт к снижению синтеза естественного увлажняющего фактора и повышенной проницаемости рогового слоя кожи. Также значимыми являются нарушения в системе плотных межклеточных контактов и дисбаланс активности эпидермальных протеаз и антипротеаз. Иммунная составляющая заболевания характеризуется активностью цитокинов интерлейкин-4 (IL-4), IL-13, IL-31, которые способствуют как воспалению, так и дополнительному нарушению барьерной функции кожи. Нарушение кожного микробиома, в частности избыточный рост *Staphylococcus aureus,* также играет важную роль в обострении заболевания. Несмотря на активное изучение молекулярных и клеточных механизмов атопического дерматита, диагностика по-прежнему остаётся клинической, а использование лабораторных биомаркеров ограничено отсутствием универсальных, чувствительных и специфичных индикаторов. В обзоре литературы рассмотрены особенности эпидермального барьера, генетические мутации, иммунные механизмы и влияние микробиоты. Особое внимание уделяется роли гена филаггрина, а также возможности использования цитокинов и других серологических маркеров как потенциальных диагностических и прогностических биомаркеров. В результате анализа выявлены потенциальные мишени для диагностики и оценки тяжести заболевания, однако их клиническое применение требует дальнейших масштабных исследований. Это особенно актуально в контексте развития персонализированной медицины и оптимизации терапии пациентов с атопическим дерматитом.

Ключевые слова: атопический дерматит; кожный барьер; филаггрин; иммунные цитокины; кератиноциты; микробиом кожи; иммуноглобулин Е.

Как цитировать:

Борукаева И.Х., Темиржанова Ф.Х., Шхагумов К.Ю., Абазова З.Х., Гетигежева А.З. Атопический дерматит: патогенетические механизмы и роль биомаркеров в диагностике // Казанский медицинский журнал. 2025. Т. 106, № 4. С. 609–618. DOI: 10.17816/KMJ680075 EDN: KFHMJP

Рукопись получена: 20.05.2025 **Рукопись одобрена:** 03.06.2025 **Опубликована online:** 23.07.2025

BACKGROUND

Atopic dermatitis (AD) is a multifactorial disease of significant medical and social importance, affecting up to 20% of children and up to 10% of adults [1]. Recent studies demonstrated that the pathogenesis of AD is allergic in nature and includes genetic defects, innate and adaptive immune response disorders, microbiological imbalances, and neuroimmune dysregulation [2, 3].

AD is frequently accompanied by increased serum immunoglobulin E (IgE) levels and is associated with a family history of atopy, which is a group of conditions that includes eczema, bronchial asthma, and allergic rhinitis [4]. Although sensitization to environmental or food allergens is associated with the AD phenotype, it does not appear to be a causative factor; however, it may contribute to the development of severe disease in some patients [5].

As molecular medicine advances, the need for biomarkers that function as objective indicators for diagnosis, assessment of disease severity, prognosis, and prediction of therapeutic efficacy increases. Integrating genetic, immunological, and microbiological data in diagnostic practices is a promising personalized approach to managing patients with AD.

This study analyzed and reviewed 88 data sources found in the public domain using the Yandex and Google search engines and the databases PubMed, eLibrary.Ru, Scopus, and Google Scholar from 2019 to 2025. The search queries used were *атопический дерматит* (*atopic dermatitis*), *патогенетические механизмы атопического дерматита* (*pathogenetic mechanisms of atopic dermatitis*), *биомаркеры атопического дерматита* (*biomarkers of atopic dermatitis*), and *новые методы лечения атопического дерматита* (*new methods of treatment of atopic dermatitis*). Publications with high methodological quality were selected. Theses, abstracts, and repetitive data were excluded.

GENETIC FACTORS AND MOLECULAR BIOMARKERS OF PREDISPOSITION TO ATOPIC DERMATITIS

The pathogenesis of AD involves a complex interaction of various mechanisms, including epidermal barrier disruption, immune dysregulation, genetic predisposition, altered skin microbiome, and exposure to external factors [6–8]. Whether the primary cause is skin barrier damage (the "outside–in" hypothesis) or immune disorders (the "inside–out" hypothesis) remains debatable [9]. These processes interact to form different AD endotypes and phenotypes, each of which may have a unique biomarker profile [10].

Genetic predisposition to AD is confirmed by high concordance rates in monozygotic twins and indicated by the results of large-scale genome-wide association studies (GWAS) [11–15]. A key genetic biomarker is a mutation in the *FLG* gene (chromosome 1q21.3), which encodes profilaggrin, which is a filaggrin precursor [16, 17]. These loss-of-function mutations increase the risk of AD by three- to fourfold and are often associated with severe clinical phenotypes, including early onset, chronic progression, and concomitant allergies [18–21].

FLG allelic variants (e.g., R501X and 2282del4) have been linked to increased skin permeability, transepidermal water loss, and decreased efficacy of therapy [22, 23]. The prevalence of these variants varies across populations: in up to 50% of Europeans and approximately 27% of Asian patients [24]. Furthermore, these cells are associated with systemic allergic sensitization indicators, including a predisposition to asthma, allergic rhinitis, and food allergies [25, 26].

The *FLG* genotype may be regarded as a pharmacogenetic biomarker, influencing an individual's response to therapy. Children with homozygous loss-of-function variants in *FLG* exhibited a decreased likelihood of achieving long-term remission and an increased propensity for regular corticosteroid use [27].

Other genetic biomarkers of AD susceptibility have also been identified, including those associated with innate immunity and T-cell function. A comprehensive meta-analysis of 26 GWAS involving >21,000 patients determined 31 loci, including the cytokine cluster on 5q31.1 (interleukin [IL]-4 and IL-13), the epidermal differentiation complex on 1q21.3 (*FLG*, *LCE*, and *SPINK5*), and a site on 11q13.5 with the candidate genes *EMSY* and *LRRC32* that are associated with AD [28]. These genes regulate immune activation, skin barrier function, and inflammatory response, making them promising targets for personalized therapies and diagnostics.

PATHOGENETIC MECHANISMS AND BIOMARKERS OF EPIDERMAL DYSFUNCTION IN ATOPIC DERMATITIS

The stratum corneum is the main structural component of the skin's barrier. It consists of corneocytes, which are nuclear-free cells, enclosed in a lipid matrix rich in ceramides, filaggrin breakdown products, cholesterol, and fatty acids [29–31]. In AD, there are changes in lipid composition and decreased natural moisturizing factor levels, which may be reflected in the levels of biomarkers, such as filaggrin, loricrin, and involucrin [32–34]. These changes lead to increased transepidermal water loss (TWL) and skin permeability, as confirmed by clinical and laboratory methods [35].

Decreased filaggrin levels is a key biomarker of skin barrier disruption in AD. Filaggrin is a structural protein involved in keratinocyte differentiation and stratum corneum formation [36, 37]. Mutations in the *FLG* gene, which encodes profilaggrin, lead to decreased filaggrin synthesis. This is accompanied by impaired corneocyte integrity, altered lipid composition, and increased TWL [38]. At the molecular level, the process involves the phosphorylation of profilaggrin

and its storage in keratohyalin granules and cleavage into filaggrin monomers, which promote keratin filament aggregation [39].

Further degradation products of filaggrin, such as pyrrolidone carboxylic acid and trans-urocanic acid, include the natural moisturizing factor that maintains skin hydration. Decreased levels of these metabolites in the stratum corneum are clinically relevant metabolic indicators of AD [40].

Moreover, a disturbance in the balance between the activity of epidermal proteases and their inhibitors is considered a biomarker of epidermal differentiation disorders [41]. Other molecular indicators include abnormalities in tight junction proteins, such as claudin-1. A decrease in these proteins indicates impaired skin barrier function [42].

Disturbances in the proteolytic balance of the epidermis manifest as overactivity of enzymes such as kallikreins (KLK5 and KLK7) and decreased levels of their physiological inhibitors, including the lymphoepithelial Kazal-type inhibitor (LEKTI) [43]. These changes contribute to the degradation of intercellular connections and inflammation, establishing kallikreins and LEKTI as additional barrier dysfunction markers [44].

Another crucial class of biomarkers is the tight junction proteins that provide epidermal tightness. Claudin-1 is especially significant, and decreased claudin-1 expression has been found in patients with AD [45]. Claudin-1 deficiency leads to increased skin permeability, activation of immune receptors, and increased allergic inflammation [46]. Claudin-1 and other tight junction components, such as occludin, tricellulin, and JAM-A, are potential biomarkers for diagnosing and predicting disease severity [47].

Additionally, decreased claudin-1 expression is correlated with increased TWL, severe skin dryness, and increased sensitivity to allergens (e.g., food allergens). This emphasizes its significance in systemic allergic responses in AD [48].

IMMUNE DYSREGULATION IN ATOPIC DERMATITIS

Increased production of pro-inflammatory cytokines is a key mechanism of chronic inflammation in AD. Among these cytokines, IL-4, IL-13, IL-17A, IL-22, IL-25, and IL-31 are particularly critical [49]. These cytokines directly affect filaggrin expression in keratinocytes, decreasing its synthesis and contributing to the progressive loss of the skin barrier [50]. IL-4 and IL-13 are key T-helper 2 (Th2)-type cytokines that inhibit keratinocyte differentiation and filaggrin, loricrin, and involucrin synthesis [51]. Their levels in the skin and serum correlate with AD severity and are often used as biomarkers of Th2-dependent inflammation. IL-31 is associated with the intensity of pruritus characteristic of AD and is a potential indicator of pruritus symptoms and severity [52]. IL-17A and IL-22 play a role in epidermal hyperplasia and impaired skin barrier function [53]. Their expression in the skin or plasma may be used as biomarkers for assessing AD endotype, inflammation severity, and response to therapy.

Immune inflammation in AD is caused by a complex interaction of different T-helper subpopulations. The dynamics of this interaction may be reflected through corresponding immune indices. During the acute phase of the disease, the predominant response is Th2-mediated, characterized by increased secretion of IL-4, IL-5, and IL-13 [54]. These cytokines contribute to IgE-mediated sensitization and mast cell activation, making them key biomarkers of allergic inflammation and potential therapeutic targets [55].

As AD progresses to a chronic condition, the involvement of other T-helper cell subpopulations (Th1, Th17, and Th22) is observed. IL-22 promotes keratinocyte proliferation and epidermal hyperplasia and is a hyperplastic inflammation marker [56]. IL-17A increases inflammation and attracts neutrophils, playing a role in resistant AD. IFN-γ, associated with the Th1 response, is involved in maintaining long-term immune system activation and is a potential marker of the transition to chronic inflammation [57].

Keratinocytes and antigen-presenting cells of the epidermis express Toll-like receptors (TLR). The activation of these cells is initiated by tissue damage or exposure to microbial components, which triggers the production of alarmins, which are early mediators of inflammation and stress signaling. Alarmins with increased expression in AD include thymic stromal lymphopoietin (TSLP), IL-25, IL-33, IL-1α, proteases (e.g., kallikreins and cathepsins), and extracellular matrix proteins (e.g., periostin) [58].

Alarmins activate epidermal dendritic cells, type 2 innate lymphoid cells, mast cells, and basophils. This induces secretion of Th2-associated cytokines, primarily IL-4 and IL-13 [59]. These cytokines support the inflammatory cascade and activate the signal transducer and activator of transcription (STAT) signaling pathway, particularly STAT6. This then stimulates B lymphocytes to produce immunoglobulin of the IgE class, a classic serological AD biomarker [60].

Additionally, IL-4, IL-13, IL-31, and IL-22 directly affect the epidermal barrier by decreasing the expressions of the *FLG*, loricrin, and involucrin genes. This results in impaired terminal differentiation of keratinocytes and weakening of the skin barrier function. Concurrently, suppression of antimicrobial peptide synthesis is observed; thereby, skin susceptibility to bacterial and viral colonization increases [61].

Thus, the profile of inflammatory and epithelial cytokines, alarmins, immunoglobulins, and epidermal differentiation factors constitutes a comprehensive panel of biomarkers that reflect the patient's immune status, disease severity, and endotype characteristics.

DOI: https://doi.org/10.17816/KMJ680075

NEUROIMMUNE MECHANISMS AND CHRONIC PRURITUS IN ATOPIC DERMATITIS

Chronic pruritus is a characteristic and distressing symptom of AD that significantly decreases patients' quality of life [62]. At the molecular level, pruritus is caused by activation of unmyelinated C fibers in the peripheral nervous system, which are subdivided into histamine-sensitive and histamine-insensitive types. These fibers are located in the posterior medullary ganglia of the spinal cord and extend to the epidermis, dermal papillae, and skin appendages. In this setting, various pruritogenic mediators is identified [63].

In patients with AD, histamine-independent mechanisms play a critical role in the development of pruritus, including neuroimmune interaction between sensory neurons, keratinocytes, and Th2 cells. Th2-type cytokines, such as IL-4, IL-13, IL-31, and TSLP, act as key mediators. Increased expression of these cytokines is considered a molecular biomarker of chronic pruritus in AD [64].

Experimental studies in animal models have demonstrated that sensory neurons innervating the skin express IL-4Rα, IL-13Rα1, and IL-31Ra receptors, indicating a direct modulation of pruritus by cytokines [65]. IL-31 can directly induce pruritus, whereas IL-4 and IL-13 sensitize neurons to other pruritogens by lowering the sensitivity threshold and enhancing pruritus perception [66].

The clinical significance of these interactions is supported by the efficacy of neuroimmune target inhibitors. Dupilumab, for example, blocks the IL-4Rα receptor and decreases the intensity of pruritus and inflammation. JAK kinase inhibitors suppress IL-4/IL-13 signaling pathways and provide marked antipruritic effects [67].

SKIN MICROBIOTA AND ITS INFLUENCE ON ATOPIC DERMATITIS

One of the main characteristics of AD is skin microbiota imbalance accompanied by a significant increase in colonization by opportunistic microorganisms, primarily *Staphylococcus aureus (S. aureus).* This bacterium is present in >90% of patients with AD, compared with 5%–30% of healthy individuals [68].

S. aureus colonization is a crucial microbiological biomarker of AD exacerbations. This condition is accompanied by the production of various toxins, superantigens, proteases, and adhesive proteins, including adhesion factor B, fibronectin-binding proteins, and enterotoxins [69]. These molecules have several functions:

- Enhancing Th2-mediated inflammation
- Damage to epithelial intercellular contacts and lipid matrix
- Impaired expression of antimicrobial peptides
- Activation of innate immunity receptors, including TLR2 and TLR4, which causes cascade production of pro-inflammatory cytokines (IL-4, IL-13, and IL-31) [70]

The microbiota of normal skin comprises commensal microorganisms, such as *Staphylococcus epidermidis*, *Cutibacterium acnes*, and *Corynebacterium spp*., which maintain barrier integrity and immune homeostasis. However, patients with AD demonstrate decreased microbial diversity and dysbiosis, which contributes to *S. aureus* proliferation and disease progression [71].

Studies of the skin microbiome using 16S rRNA gene sequencing showed a significant decrease in bacterial diversity during AD exacerbations, including a decrease in *Streptococcus*, *Corynebacterium*, and *Propionibacterium* and a marked increase in *S. aureus* density [72].

Interestingly, the recovery of microbial diversity has been observed following anti-inflammatory or antimicrobial therapy. This confirms the diagnostic and prognostic significance of microbiota as biomarkers of active AD and remission [73].

A key microbial factor that increases inflammation in AD is superantigen production by *S. aureus* [74]. These superantigens include toxic shock syndrome toxin-1 (TSST-1) and staphylococcal enterotoxins of serotypes SEA, SEB, SEC, SED, SEE, and SEG. These protein toxins may cause nonspecific (polyclonal) activation of T lymphocytes by simultaneously binding to MHC II molecules on antigen-presenting cells and TCRs on T cells [75].

This mechanism leads to massive production of pro-inflammatory cytokines, including IL-2, TNF-α, and IFN-γ, which trigger and sustain systemic inflammation [76]. The increased presence of superantigens and cytokines induced in the skin and blood of patients with AD may be a biomarker of severe and refractory cases, particularly those involving frequent infectious exacerbations.

In addition to their pro-inflammatory effects, superantigens have allergic potential and act as allergens. Superantigens may induce IgE synthesis, activate mast cells, and cause degranulation, increasing pruritus, swelling, and skin rashes [77].

The bacterial composition of the skin, expression of virulence factors, and immune response activity to microbial products may be considered diagnostically significant biomarkers that indicate the disease's phase and degree of inflammation.

HUMORAL AND CELLULAR BIOMARKERS IN CLINICAL PRACTICE

Although the clinical picture remains the primary basis for diagnosing AD, humoral and cellular biomarkers that can objectively measure the inflammatory process and facilitate disease phenotype stratification are being actively investigated. One of the most commonly reported laboratory findings is an increase in total and/or allergen-specific IgE levels

in the blood, which is a potential biomarker of Th2-mediated inflammation [78].

However, the significance of IgE as a diagnostic and prognostic marker is limited because IgE hyperproduction occurs in the late stages of the disease and may result from skin barrier disruption and epicutaneous sensitization [79].

Furthermore, the total IgE level does not always correlate with disease severity. Allergen-specific IgE has low specificity and may be detected in patients without significant symptoms [80]. Additionally, increased IgE levels are found in non-atopic conditions such as helminthic diseases, malignant tumors, and autoimmune diseases, which decreases its diagnostic specificity [81]. Although serum IgE may be a biomarker of the Th2 response, it lacks the sensitivity and specificity required for use as a universal diagnostic criterion.

Similar limitations apply to other hematologic biomarkers, such as eosinophil levels and the number of mast cell. Despite their involvement in the pathogenesis of AD, these peripheral blood parameters exhibit high variability and no stable correlations with the clinical manifestations of the disease [82].

Serum and cellular biomarkers, including IgE, eosinophils, and mast cells, may be used as auxiliary parameters to complement the clinical and immunological picture. However, these biomarkers require careful interpretation and cannot be used as independent diagnostic criteria.

The development of immunological methods and in-depth study of the pathogenesis of AD have led to the identification of new subpopulations of T lymphocytes and discovery of cytokines and chemokines that were not previously directly associated with this disease. The most significant serum markers currently being studied are as follows:

• CD30: a marker of T-cell activation associated with the Th2 response

• TARC (thymus-activated regulatory chemokine CCL17): regulates the migration of Th2 cells into the skin

• MDC (macrophage-derived chemokine, CCL22): a chemoattractant for activated Th2 lymphocytes

• IL-12, IL-16, and IL-18: cytokines that play a role in the inflammatory microenvironment [83]

Studies have shown that levels of these molecules in serum correlate with the clinical severity of AD, as measured by the Scoring Atopic Dermatitis scale [84]. For example, increased levels of TARC and MDC are consistently associated with an active disease state and decrease when remission is achieved. IL-31 shows a high correlation with subjective pruritus intensity, and CD30 and IL-18 may be biomarkers of systemic inflammation in severe AD cases [85].

Despite active study of molecular and cellular biomarkers of AD, none have shown sufficient sensitivity and specificity to serve as reliable, universal diagnostic or prognostic tools [86]. Most of the presented biomarkers reflect individual links in the pathogenesis, such as barrier dysfunction, immune activation, microbial exposure, and neuroimmune pruritus. However, none of them cover the entire spectrum of clinical and pathogenetic manifestations of the disease. The main limitations of published studies were small sample size and the fact that the patients were predominantly from specialized medical institutions with severe forms of the disease. Moreover, there was a lack of comparison with similar parameters in patients with other eczematous or atopic pathologies [87]. Additionally, prognostic markers show mixed results; however, increased levels of total IgE and null mutations of the *FLG* gene are more often associated with a more severe and prolonged clinical course of the disease [88].

CONCLUSION

AD is a complex, multifactorial disease based on the interaction of genetic, immunological, microbiological, and neuroimmune mechanisms. A comprehensive understanding of the pathogenesis of this condition enables the identification of increasingly accurate and informative biomarkers. These biomarkers may reflect the clinical severity and phase of the disease and predict response to therapy.

Genetic markers, such as *FLG* gene mutations, are considered promising biomarkers that reflect key pathogenetic links and determine vulnerability to early-onset and severe disease courses. Additionally, Th2-type cytokines (IL-4, IL-13, and IL-31), alarmins (TSLP and IL-33), and chemokines (TARC and MDC) are sensitive indicators of inflammation and pruritus in AD. TWL values and decreased filaggrin, claudin-1, and involucrin levels are crucial markers of skin barrier insufficiency that objectively indicate disorders of epidermal homeostasis. The presence of *S. aureus* and the production of superantigens (SEA, SEB, and TSST-1) indicate an increased risk of exacerbation, a refractory course, and systemic inflammation. Methods of 16S rRNA sequencing revealed a decrease in skin microbial diversity. Therefore, the microbiome structure is a potential marker for monitoring therapeutic efficacy and prognosis in AD.

ADDITIONAL INFORMATION

Author contributions: B.I.Kh.: conceptualization, supervision, writing—review & editing; T.F.Kh.: methodology, investigation, writing—original draft; Sh.K.Yu.: investigation, writing—original draft; A.Z.Kh.: investigation, writing—original draft, writing—review & editing; G.A.Z.: investigation, writing—original draft. All the authors approved the version of the manuscript to be published and agreed to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ethics approval: Ethics committee approval was not required because the article is a review.

Funding sources: This study was conducted at Kabardino-Balkarian State University named after Kh.M. Berbekov with support from the Priority 2030 Strategic Academic Leadership Program. The authors received no financial support from pharmaceutical or medical device companies. The authors had full access to all data obtained through the scientific articles search and analysis.

Disclosure of interests: The authors have no relationships, activities, or interests for the last three years related to for-profit or not-for-profit third parties whose interests may be affected by the content of the article.

Statement of originality: No previously published material (text, images, or data) was used in this work.
Data availability statement: The editorial policy regarding data sharing does not apply to this work, as no new data was collected or created.
Generative AI: No generative artificial intelligence technologies were used to prepare this article.
Provenance and peer review: This paper was submitted unsolicited and reviewed following the fast-track procedure. The peer review process involved two external reviewers, a member of the editorial board, and the in-house scientific editor.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Б.И.Х. — разработка концепции, написание рукописи — рецензирование и редактирование, научное руководство; Т.Ф.Х. — разработка методологии, проведение исследования, написание черновика рукописи; Ш.К.Ю. — проведение исследования, написание черновика рукописи; А.З.Х. — проведение исследования, написание черновика рукописи, написание рукописи — рецензирование и редактирование; Г.А.З. — проведение исследования, написание черновика рукописи. Все авторы одобрили рукопись, а также согласились нести ответственность за все аспекты работы, гарантируя надлежащее рассмотрение и решение вопросов, связанных с точностью и добросовестностью любой её части.
Этическая экспертиза. Одобрение этического комитета на проведение исследования не получали. Причина — обзорный характер статьи.
Источники финансирования. Работа выполнена в Кабардино-Балкарском государственном университете им. Х.М. Бербекова при поддержке программы стратегического академического лидерства «ПРИОРИТЕТ-2030». Финансовой поддержки со стороны компаний-производителей лекарственных препаратов и медицинского оборудования авторы не получали. У авторов есть полный доступ ко всем данным, полученным в результате поиска и анализа литературы.
Раскрытие интересов. Авторы заявляют об отсутствии отношений, деятельности и интересов за последние три года, связанных с третьими лицами (коммерческими и некоммерческими), интересы которых могут быть затронуты содержанием статьи.
Оригинальность. При создании настоящей работы авторы не использовали ранее опубликованные сведения (текст, иллюстрации, данные).
Доступ к данным. Редакционная политика в отношении совместного использования данных к настоящей работе не применима, новые данные не собирали и не создавали.
Генеративный искусственный интеллект. При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.
Рассмотрение и рецензирование. Настоящая работа подана в журнал в инициативном порядке и рассмотрена в соответствии с процедурой fast-track. В рецензировании участвовали два внешних рецензента, член редакционной коллегии и научный редактор издания.

REFERENCES

1. Fishbein AB, Silverberg JI, Wilson EJ, Ong PY. Update on Atopic Dermatitis: Diagnosis, Severity Assessment, and Treatment Selection. *J Allergy Clin Immunol Pract*. 2020;8(1):91–101. doi: 10.1016/j.jaip.2019.06.044 EDN: DTYCUJ
2. Silverberg JI, Barbarot S, Gadkari A, et al. Atopic dermatitis in the pediatric population: A cross-sectional, international epidemiologic study. *Ann Allergy Asthma Immunol*. 2021;126(4):417–428.e2. doi: 10.1016/j.anai.2020.12.020 EDN: HZILPQ
3. Davis DMR, Drucker AM, Alikhan A, et al. American Academy of Dermatology Guidelines: Awareness of comorbidities associated with atopic dermatitis in adults. *J Am Acad Dermatol*. 2022;86(6):1335–1336.e18. doi: 10.1016/j.jaad.2022.01.009 EDN: VGTWUA
4. Chen Y, Peng C, Zhu L, et al. Atopic Dermatitis and Psoriasis: Similarities and Differences in Metabolism and Microbiome. *Clin Rev Allergy Immunol*. 2024;66(3):294-315. doi: 10.1007/s12016-024-08995-3 EDN: LEBCHO
5. Tsuge M, Ikeda M, Matsumoto N, et al. Current Insights into Atopic March. *Children*. 2021;8(11):1067. doi: 10.3390/children8111067 EDN: EKKXTX
6. Tsakok T, Woolf R, Smith CH, et al. Atopic dermatitis: the skin barrier and beyond. *Br J Dermatol*. 2019;180:464. doi: 10.1111/bjd.16934 EDN: FEOWKZ
7. Ständer S. Atopic Dermatitis. *N Engl J Med*. 2021;384:1136. doi: 10.1056/NEJMra2023911 EDN: WYOBVJ
8. Czarnowicki T, He H, Krueger JG, Guttman-Yassky E. Atopic dermatitis endotypes and implications for targeted therapeutics. *J Allergy Clin Immunol*. 2019;143(1):1–11. doi: 10.1016/j.jaci.2018.10.032
9. Sroka-Tomaszewska J, Trzeciak M. Molecular Mechanisms of Atopic Dermatitis Pathogenesis. *Int J Mol Sci*. 2021;22(8):4130. doi: 10.3390/ijms22084130 EDN: APIXFP
10. Potekaev NN, Serov DN, Mikhailova IA, et al. Current aspects of pathogenesis and treatment of atopic dermatitis. *Russian Journal of Clinical Dermatology and Venereology*. 2019;18(3):259–263. doi: 10.17116/klinderma201918031259 EDN: RKDTTU
11. Kalashnikova IG, Nekrasova AI, Korobeynikova AV, et al. The Association between Gut Microbiota and Serum Biomarkers in Children with Atopic Dermatitis. *Biomedicines*. 2024;12(10):2351. doi: 10.3390/biomedicines12102351 EDN: PPIJYN
12. Marques-Mejias A, Bartha I, Ciaccio CE, et al. Skin as the target for allergy prevention and treatment. *Ann Allergy Asthma Immunol*. 2024;133(2):133–143. doi: 10.1016/j.anai.2023.12.030 EDN: DDPIUU
13. Criado PR, Miot HA, Bueno-Filho R, et al. Update on the pathogenesis of atopic dermatitis. *An Bras Dermatol*. 2024;99(6):895–915. doi: 10.1016/j.abd.2024.06.001 EDN: XICPLH
14. Gao H, Kosins AE, Cook-Mills JM. Mechanisms for initiation of food allergy by skin pre-disposed to atopic dermatitis. *Immunol Rev*. 2024;326(1):151–161. doi: 10.1111/imr.13367 EDN: HCGWGU
15. Niehues T, von Hardenberg S, Velleuer E. Rapid identification of primary atopic disorders (PAD) by a clinical landmark-guided, upfront use of genomic sequencing. *Allergol Select*. 2024;8:304–323. doi: 10.5414/ALX02250E EDN: ENLVRD
16. Huffaker MF, Kanchan K, Bahnson HT, et al. Epidermal differentiation complex genetic variation in atopic dermatitis and peanut allergy. *J Allergy Clin Immunol*. 2023;151(4):1137–1142.e4. doi: 10.1016/j.jaci.2022.11.008 EDN: TFCUUZ
17. Stamatas GN, Sato T, Chaoimh CN, et al. Early skin inflammatory biomarker is predictive of development and persistence of atopic dermatitis in infants. *J Allergy Clin Immunol*. 2024;153(6):1597–1603.e4. doi: 10.1016/j.jaci.2024.02.018 EDN: NMNLXU
18. Khatib CM, Klein-Petersen AW, Rønnstad ATM, et al. Increased loss-of-function filaggrin gene mutation prevalence in atopic dermatitis patients across northern latitudes indicates genetic fitness: A systematic review and meta-analysis. *Exp Dermatol*. 2024;33(7):e15130. doi: 10.1111/exd.15130 EDN: RXBETE
19. Vaseghi-Shanjani M, Snow AL, Margolis DJ, et al. Atopy as Immune Dysregulation: Offender Genes and Targets. *J Allergy Clin Immunol Pract*. 2022;10(7):1737–1756. doi: 10.1016/j.jaip.2022.04.001 EDN: JWCFAH
20. González-Tarancón R, Sanmartín, R, Lorente F, et al. Prevalence of FLG loss-of-function mutations R501X, 2282del4, and R2447X in Spanish children with atopic dermatitis. *Pediatr Dermatol*. 2020;37:98–102. doi: 10.1111/pde.14025
21. Martin MJ, Estravís M, García-Sánchez A, et al. Genetics and Epigenetics of Atopic Dermatitis: An Updated Systematic Review. *Genes*. 2020;11(4):442. doi: 10.3390/genes11040442 EDN: AKPALK

22. Murashkin NN, Opryatin LA, Epishev RV, et al. Filaggrin Defect at Atopic Dermatitis: Modern Treatment Options. *Current Pediatrics*. 2022;21(5):347–351. doi: 10.15690/vsp.v21i5.2452 EDN: PCZGSM
23. Berezina AS, Karacheva YuV, Vinnik YuYu, Tartarakova SS. Atopic dermatitis. Features of pathogenesis, clinical image and diagnosis. *Vestnik SurGU. Meditsina*. 2023;16(2):8–13. doi: 10.35266/2304-9448-2023-2-8-13 EDN: ZLLJEY
24. Kozlova IB, Chikin VB, Karamova AE, Kubanov AA. Prevalence of the filaggrin gene loss-of-function variants in different countries and the effect of their carriage on the course of atopic dermatitis. *Medical genetics*. 2023;22(9):3–18. doi: 10.25557/2073-7998.2023.09.3-18 EDN: VDFEHU
25. Mulick AR, Mansfield KE, Silverwood RJ, et al. Four childhood atopic dermatitis subtypes identified from trajectory and severity of disease and internally validated in a large UK birth cohort. *Br J Dermatol*. 2021;185:526. doi: 10.1111/bjd.19885 EDN: JBKCAQ
26. Biazus Soares G, Hashimoto T, Yosipovitch G. Atopic Dermatitis Itch: Scratching for an Explanation. *J Invest Dermatol*. 2024;144(5):978–988. doi: 10.1016/j.jid.2023.10.048 EDN: DRDFMA
27. Astolfi A, Cipriani F, Messelodi D, et al. Filaggrin Loss-of-Function Mutations Are Risk Factors for Severe Food Allergy in Children with Atopic Dermatitis. *J Clin Med*. 2021;10. doi: 10.3390/jcm10020233 EDN: KJKPHF
28. Farmer WS, Marathe KS. Atopic Dermatitis: Managing the Itch. *Adv Exp Med Biol*. 2024;1447:191–207. doi: 10.1007/978-3-031-54513-9_16
29. Ivert LU, Wahlgren CF, Lindelöf B, et al. Association between atopic dermatitis and autoimmune diseases: a population-based case-control study. *Br J Dermatol*. 2021;185(2):335–342. doi: 10.1111/bjd.19624 EDN: XGXQZC
30. Jabbar-Lopez ZK, Ung CY, Alexander H, et al. The effect of water hardness on atopic eczema, skin barrier function: A systematic review, meta-analysis. *Clin Exp Allergy*. 2021;51:430. doi: 10.1111/cea.13797 EDN: DRBZBK
31. Schmuth M, Eckmann S, Moosbrugger-Martinz V, et al. Skin Barrier in Atopic Dermatitis. *J Invest Dermatol*. 2024;144(5):989–1000.e1. doi: 10.1016/j.jid.2024.03.006 EDN: CBNESW
32. Silverberg JI. Comorbidities and the impact of atopic dermatitis. *Ann Allergy Asthma Immunol*. 2019;123(2):144–151. doi: 10.1016/j.anai.2019.04.020 EDN: GQWHKP
33. de Boer FL, van der Molen HF, Kezic S. Epidermal biomarkers of the skin barrier in atopic and contact dermatitis. *Contact Dermatitis*. 2023;89(4):221–229. doi: 10.1111/cod.14391 EDN: VHKXAW
34. Schuler CF 4th, Tsoi LC, Billi AC, et al. Genetic and Immunological Pathogenesis of Atopic Dermatitis. *J Invest Dermatol*. 2024;144(5):954–968. doi: 10.1016/j.jid.2023.10.019 EDN: MQKHUA
35. Mandlik DS, Mandlik SK. Atopic dermatitis: new insight into the etiology, pathogenesis, diagnosis and novel treatment strategies. *Immunopharmacol Immunotoxicol*. 2021;43(2):105–125. doi: 10.1080/08923973.2021.1889583 EDN: ZUJKHE
36. Moosbrugger-Martinz V, Leprince C, Méchin MC, et al. Revisiting the Roles of Filaggrin in Atopic Dermatitis. *Int J Mol Sci*. 2022;23(10):5318. doi: 10.3390/ijms23105318 EDN: JMHBKB
37. Boothe WD, Tarbox JA, Tarbox MB. Atopic Dermatitis: Pathophysiology. *Adv Exp Med Biol*. 2024;1447:21–35. doi: 10.1007/978-3-031-54513-9_3
38. Virolainen SJ, Satish L, Biagini JM, et al. Filaggrin loss-of-function variants are associated with atopic dermatitis phenotypes in a diverse, early-life prospective cohort. *JCI Insight*. 2024;9(9):e178258. doi: 10.1172/jci.insight.178258 EDN: FHDLSS
39. Luger T, Amagai M, Dreno B, et al. Atopic dermatitis: Role of the skin barrier, environment, microbiome, and therapeutic agents. *J Dermatol Sci*. 2021;102(3):142–157. doi: 10.1016/j.jdermsci.2021.04.007 EDN: AOJVXA
40. Kim BE, Kim J, Goleva E, et al. Particulate matter causes skin barrier dysfunction. *JCI Insight*. 2021;6(5):e145185. doi: 10.1172/jci.insight.145185 EDN: GPGPDU
41. Stefanovic N, Irvine AD. Filaggrin and beyond: New insights into the skin barrier in atopic dermatitis and allergic diseases, from genetics to therapeutic perspectives. *Ann Allergy Asthma Immunol*. 2024;132(2):187–195. doi: 10.1016/j.anai.2023.09.009 EDN: WGOIIX
42. Drislane C, Irvine AD. The role of filaggrin in atopic dermatitis and allergic disease. *Ann Allergy Asthma Immunol*. 2020;124:36. doi: 10.1016/j.anai.2019.10.008 EDN: KRVUKD
43. Tamrazova OB, Glukhova EA. Unique molecule filaggrin in epidermal structure and its role in the xerosis development and atopic dermatitis pathogenesis. *Russian Journal of Clinical Dermatology and Venereology*. 2021;20(6):102–110. doi: 10.17116/klinderma202120061102 EDN: BRNNIZ
44. O'Regan Stamatas GN, Sato T, Chaoimh CN, et al. Early skin inflammatory biomarker is predictive of development and persistence of atopic dermatitis in infants. *J Allergy Clin Immunol*. 2024;153(6):1597–1603.e4. doi: 10.1016/j.jaci.2024.02.018 EDN: NMNLXU
45. Bellinato F, Gisondi P, Medori MC, et al. Novel loss-of-function variants in filaggrin exon 3 in patients with severe atopic dermatitis. *Arch Dermatol Res*. 2024;316(8):606. doi: 10.1007/s00403-024-03273-w EDN: RZHCJN
46. Thibault Greugny E, Bensaci J, Fages F, Stamatas GN. Computational modelling predicts impaired barrier function and higher sensitivity to skin inflammation following pH elevation. *Exp Dermatol*. 2023;32(2):177–185. doi: 10.1111/exd.14698 EDN: MLBZTG
47. Gwak YS, Kim SY, Woo CE, et al. Association between Atopic Dermatitis and Dementia: Evidence from Systematic Review, Meta-analysis, and Mendelian Randomization. *Acta Derm Venereol*. 2025;105:adv41321. doi: 10.2340/actadv.v105.41321 EDN: IGZYPJ
48. Hu XQ, Tang Y, Ju Y, et al. Scratching damages tight junctions through the Akt-claudin 1 axis in atopic dermatitis. *Clin Exp Dermatol*. 2021;46(1):74–81. doi: 10.1111/ced.14380 EDN: JBXZFZ
49. Xia Y, Cao H, Zheng J, Chen L. Claudin-1 Mediated Tight Junction Dysfunction as a Contributor to Atopic March. *Front Immunol*. 2022;13:927465. doi: 10.3389/fimmu.2022.927465 EDN: RKOQLK
50. Carr S, Pratt R, White F, Watson W. Atopic dermatitis. *Allergy Asthma Clin Immunol*. 2024;20(Suppl 3):63. doi: 10.1186/s13223-024-00927-2 EDN: DZENKM
51. Dubin C, Del Duca E, Guttman-Yassky E. The IL-4, IL-13 and IL-31 pathways in atopic dermatitis. *Expert Rev Clin Immunol*. 2021;17(8):835–852. doi: 10.1080/1744666X.2021.1940962 EDN: QHBBYA
52. Hashimoto T, Yokozeki H, Karasuyama H, Satoh T. IL-31-generating network in atopic dermatitis comprising macrophages, basophils, thymic stromal lymphopoietin, and periostin. *J Allergy Clin Immunol*. 2023;151(3):737–746.e6. doi: 10.1016/j.jaci.2022.11.009 EDN: OJHCOC
53. Shiomitsu S, Gillen J, Frasca SJr, Santoro D. Evaluation of the cutaneous expression of IL-17, IL-22, IL-31, and their receptors in canine atopic dermatitis. *Res Vet Sci*. 2021;136:74–80. doi: 10.1016/j.rvsc.2020.12.015 EDN: DVQMUW
54. García-Reyes MM, Zumaya-Pérez LC, Pastelin-Palacios R, Moreno-Eutimio MA. Serum thymic stromal lymphopoietin (TSLP) levels in atopic dermatitis patients: a systematic review and meta-analysis. *Clin Exp Med*. 2023;23(8):4129–4139. doi: 10.1007/s10238-023-01147-5 EDN: ACNUMQ
55. Lawson LP, Parameswaran S, Panganiban RA, et al. Update on the genetics of allergic diseases. *J Allergy Clin Immunol*. 2025;155(6):1738–1752. doi: 10.1016/j.jaci.2025.03.012
56. Gallo RL, Horswill AR. Staphylococcus aureus: The Bug Behind the Itch in Atopic Dermatitis. *J Invest Dermatol*. 2024;144(5):950–953. doi: 10.1016/j.jid.2024.01.001 EDN: JFSFVE
57. Paternoster L. Genetic landscape of atopic dermatitis. *Curr Opin Allergy Clin Immunol*. 2024;24(5):409–415. doi: 10.1097/ACI.0000000000001005 EDN: HSFKBI
58. Elhage KG, Kranyak A, Jin JQ, et al. Mendelian Randomization Studies in Atopic Dermatitis: A Systematic Review. *J Invest Dermatol*. 2024;144(5):1022–1037. doi: 10.1016/j.jid.2023.10.016 EDN: QIMGXC
59. Eggel A, Pennington LF, Jardetzky TS. Therapeutic monoclonal antibodies in allergy: Targeting IgE, cytokine, and alarmin pathways. *Immunol Rev*. 2024;328(1):387–411. doi: 10.1111/imr.13380 EDN: RDWJTZ
60. Kim Y, Lim KM. Skin barrier dysfunction and filaggrin. *Arch Pharm Res*. 2021;44(1):36–48. doi: 10.1007/s12272-021-01305-x EDN: HZEWFU
61. Honda T, Kabashima K. Reconciling innate and acquired immunity in atopic dermatitis. *J Allergy Clin Immunol*. 2020;145:1136. doi: 10.1016/j.jaci.2020.02.008 EDN: RJDSLM
62. Elizalde-Jiménez IG, Ruiz-Hernández FG, Carmona-Cruz SA, et al. Global Antimicrobial Susceptibility Patterns of Staphylococcus aureus in Atopic Dermatitis: A Systematic Review and Meta-Analysis. *JAMA Der-*

matol. 2024;160(11):1171–1181. doi: 10.1001/jamadermatol.2024.3360 EDN: CRDSKF

63. Garcovich S, Maurelli M, Gisondi P, et al. Pruritus as a Distinctive Feature of Type 2 Inflammation. *Vaccines*. 2021;9(3):303. doi: 10.3390/vaccines9030303 EDN: AVNKJP

64. Burger E, Gallo RL. Host-microbiome interactions in the holobiome of atopic dermatitis. *J Allergy Clin Immunol*. 2023;151(5):1236–1238. doi: 10.1016/j.jaci.2022.11.019 EDN: FWODPE

65. Tokura Y, Hayano S. Subtypes of atopic dermatitis: From phenotype to endotype. *Allergol Int*. 2022;71(1):14–24. doi: 10.1016/j.alit.2021.07.003 EDN: ERBQKQ

66. Scala E, Madonna S, Abeni D, et al. A microarray-based IgE-molecular mimicry index (IgE-MMI): A biomarker for disease severity, clinical phenotypes, and therapeutic response in atopic dermatitis? *Allergy*. 2024;79(12):3415–3429. doi: 10.1111/all.16377 EDN: UFQJWF

67. Bangert C, Loesche C, Skvara H, et al. IgE Depletion with Ligelizumab Does Not Significantly Improve Clinical Symptoms in Patients with Moderate-to-Severe Atopic Dermatitis. *J Invest Dermatol*. 2023;143(10):1896–1905.e8. doi: 10.1016/j.jid.2023.01.040 EDN: LOGCLN

68. Beck LA, Cork MJ, Amagai M, et al. Type 2 Inflammation Contributes to Skin Barrier Dysfunction in Atopic Dermatitis. *JID Innov*. 2022;2(5):100131. doi: 10.1016/j.xjidi.2022.100131 EDN: IODLTF

69. Wollenberg A, Christen-Zäch S, Taieb A, et al. European Task Force on Atopic Dermatitis/EADV Eczema Task Force. ETFAD/EADV Eczema task force 2020 position paper on diagnosis and treatment of atopic dermatitis in adults and children. *J Eur Acad Dermatol Venereol*. 2020;34(12):2717–2744. doi: 10.1111/jdv.16892 EDN: CWTNIS

70. Bakker D, de Bruin-Weller M, Drylewicz J, et al. Biomarkers in atopic dermatitis. *J Allergy Clin Immunol*. 2023;151(5):1163–1168. doi: 10.1016/j.jaci.2023.01.019 EDN: ZZDBUQ

71. Yosipovitch G, Berger T, Fassett MS. Neuroimmune interactions in chronic itch of atopic dermatitis. *J Eur Acad Dermatol Venereol*. 2020;34:239. doi: 10.1111/jdv.15973 EDN: KZKKDK

72. Brooks SG, Yosipovitch G. Adjunctive Management of Itch in Atopic Dermatitis. *Dermatol Clin*. 2024;42(4):577–589. doi: 10.1016/j.det.2024.04.008 EDN: CLIUPT

73. Yu L, Li L. Potential biomarkers of atopic dermatitis. *Front Med*. 2022;9:1028694. doi: 10.3389/fmed.2022.1028694 EDN: RZAJIT

74. Mastraftsi S, Vrioni G, Bakakis M, et al. Atopic Dermatitis: Striving for Reliable Biomarkers. *J Clin Med*. 2022;11(16):4639. doi: 10.3390/jcm11164639 EDN: JLBJMW

75. Asahina R, Ueda K, Oshima Y, et al. Serum canine thymus and activation-regulated chemokine (TARC/CCL17) concentrations correlate with disease severity and therapeutic responses in dogs with atopic dermatitis. *Vet Dermatol*. 2020;31(6):446–455. doi: 10.1111/vde.12894 EDN: JXTXAR

76. Ogulur I, Mitamura Y, Yazici D, et al. Type 2 immunity in allergic diseases. *Cell Mol Immunol*. 2025;22(3):211–242. doi: 10.1038/s41423-025-01261-2 EDN: OQXERE

77. Misery L, Belloni Fortina A, El Hachem M, et al. A position paper on the management of itch and pain in atopic dermatitis from the International Society of Atopic Dermatitis (ISAD)/Oriented Patient-Education Network in Dermatology (OPENED) task force. *J Eur Acad Dermatol Venereol*. 2021;35(4):787–796. doi: 10.1111/jdv.16916 EDN: YLZQMM

78. Torres T, Cruz MJ, Gonçalo M, et al. Dupilumab in Patients with Atopic Dermatitis: A Multicentric, Long-Term, Real-World Portuguese Study. *Dermatol Ther*. 2024;14(8):2209–2221. doi: 10.1007/s13555-024-01235-8 EDN: VHYMID

79. Liao Q, Pan H, Guo Y, et al. Comparative efficacy and safety of dupilumab versus newly approved biologics and JAKi in pediatric atopic dermatitis: A systematic review and network meta-analysis. *PLoS One*. 2025;20(2):e0319400. doi: 10.1371/journal.pone.0319400 EDN: VYBAEN

80. Schachner LA, Andriessen A, Gonzalez ME, et al. Consensus on Staphylococcus aureus Exacerbated Atopic Dermatitis and the Need for a Novel Treatment. *J Drugs Dermatol*. 2024;23(10):825–832. doi: 10.36849/JDD.2024.8240 EDN: CFJYOI

81. Wang Z, Hülpüsch C, Traidl-Hoffmann C, et al. Understanding the role of Staphylococcus aureus in atopic dermatitis: strain diversity, microevolution, and prophage influences. *Front Med*. 2024;11:1480257. doi: 10.3389/fmed.2024.1480257 EDN: NTOZND

82. Hülpüsch C, Rohayem R, Reiger M, Traidl-Hoffmann C. Exploring the skin microbiome in atopic dermatitis pathogenesis and disease modification. *J Allergy Clin Immunol*. 2024;154(1):31–41. doi: 10.1016/j.jaci.2024.04.029 EDN: KSHVKU

83. Özdemir E, Öksüz L. Effect of Staphylococcus aureus colonization and immune defects on the pathogenesis of atopic dermatitis. *Arch Microbiol*. 2024;206(10):410. doi: 10.1007/s00203-024-04134-w EDN: WWAJRN

84. Leung DYM, Berdyshev E, Goleva E. Cutaneous barrier dysfunction in allergic diseases. *J Allergy Clin Immunol*. 2021;148(3):905. doi: 10.1016/j.jaci.2021.06

85. Tian T, Li Y, Yuan G, Jiang W. Efficacy and safety of dupilumab in patients with moderate-to-severe atopic dermatitis and comorbid allergic rhinitis. *Front Med*. 2025;12:1556769. doi: 10.3389/fmed.2025.1556769

86. Yu X, Li L. A Multi-centre Analysis of Serum IgE Levels in Atopic Dermatitis. *Indian J Dermatol*. 2024;69(6):486. doi: 10.4103/ijd.ijd_151_24 EDN: XDWYEM

87. D'Erme AM, Fidanzi C, Bevilacqua M, et al. Cord Blood Serum Levels of IL-31 and CCL17, Cutaneous Markers, and Development of Atopic Dermatitis. *JAMA Dermatol*. 2024;160(10):1112–1115. doi: 10.1001/jamadermatol.2024.3178 EDN: THWLOI

88. Parisi GF, Leonardi S, Ciprandi G, et al. Antihistamines in children and adolescents: A practical update. *Allergol Immunopathol*. 2020;48(6):753–762. doi: 10.1016/j.aller.2020.02.005 EDN: EKOODB

AUTHORS INFO

*** Irina Kh. Borukaeva**, MD, Dr. Sci. (Medicine), Head, Depart. of Normal and Pathological Human Physiology;
address: 5 Gorky st, Nalchik, Russia, 360051;
ORCID: 0000-0003-1180-228X;
eLibrary SPIN: 9102-2336;
e-mail: irborukaeva@yandex.ru

Farida Kh. Temirzhanova, postgraduate student, Depart. of Normal and Pathological Human Physiology;
ORCID: 0009-0007-0997-1099;
eLibrary SPIN: 7279-9097;
e-mail: temirzhanova.farida@yandex.ru

Kazbek Yu. Shkhagumov, MD, Cand. Sci. (Medicine), Assistant Professor, Depart. of Normal and Pathological Human Physiology;
ORCID: 0000-0002-3671-481X;
eLibrary SPIN: 3214-4894;
e-mail: kazbek07_07@mail.ru

Zalina Kh. Abazova, MD, Cand. Sci. (Medicine), Assistant Professor, Depart. of Normal and Pathological Human Physiology;
ORCID: 0000-0003-2827-5068;
eLibrary SPIN: 5442-5253;
e-mail: zalina.abazova@mail.ru

Amina Z. Getigezheva, MD, Cand. Sci. (Medicine), Assistant Professor, Depart. of General Medical Training and Medical Rehabilitation;
ORCID: 0000-0001-8498-1165;
eLibrary SPIN: 1308-6694;
e-mail: amina.geti@yandex.ru

ОБ АВТОРАХ

*** Борукаева Ирина Хасанбиевна**, д-р мед. наук, заведующая, каф. нормальной и патологической физиологии человека;
адрес: Россия, 360051, Нальчик, ул. Горького, д. 5;
ORCID: 0000-0003-1180-228X;
eLibrary SPIN: 9102-2336;
e-mail: irborukaeva@yandex.ru

Темиржанова Фарида Хасановна, аспирант, каф. нормальной и патологической физиологии человека;
ORCID: 0009-0007-0997-1099;
eLibrary SPIN: 7279-9097;
e-mail: temirzhanova.farida@yandex.ru

Шхагумов Казбек Юрьевич, канд. мед. наук, доцент, каф. нормальной и патологической физиологии человека;
ORCID: 0000-0002-3671-481X;
eLibrary SPIN: 3214-4894;
e-mail: kazbek07_07@mail.ru

Абазова Залина Хасановна, канд. мед. наук, доцент, каф. нормальной и патологической физиологии человека;
ORCID: 0000-0003-2827-5068;
eLibrary SPIN: 5442-5253;
e-mail: zalina.abazova@mail.ru

Гетигежева Амина Заурбиевна, канд. мед. наук, доцент, каф. общей врачебной подготовки и медицинской реабилитации;
ORCID: 0000-0001-8498-1165;
eLibrary SPIN: 1308-6694;
e-mail: amina.geti@yandex.ru

* Corresponding author / Автор, ответственный за переписку