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Atopic Dermatitis: Pathogenetic Mechanisms and Role of Biomarkers in Diagnosis

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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.

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Атопический дерматит: патогенетические механизмы и роль биомаркеров в диагностике

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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 филагрина, 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 филагрина, 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 E.

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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-1 α , 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.

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-4R α , IL-13R α 1, and IL-31R α 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-4R α 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 non-specific (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 cells. 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.

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ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

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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.

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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.

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