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Review Article

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Epigenetic and Immunological Changes in the Occurrence of Atopic Dermatitis

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ABSTRACT

Changes in the genetic background and the impact of a deteriorating ecosystem lead to the development of a large number of allergic diseases, which also became a prerequisite for the study of the etiological theory of the origin of atopic dermatitis. In this aspect, epigenetic changes are in the first place. Genetic changes cannot explain such a rapid increase in the incidence of atopic dermatitis. Changing environmental factors such as Western lifestyles, industrialization, air pollution, changing diets, obesity, increased use of antibiotics, and smoking are coming to the fore. Epigenetic changes are a probable mechanism of environmental influence on the cells of the body. When describing the etiological factors of the development of atopic dermatitis, it is impossible not to note the role and place of immunological regulation and reaction. This review article is devoted to the study of literature information regarding epigenetic and immunological aspects in the development of atopic dermatitis.

Keywords: Atopic dermatitis, epigenetics, immunology

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Epigenetics is the study of the regulation of gene expression that is not related to the modification of DNA sequences. Modifications result in the activation or inhibition of the transcription of certain genes, resulting in the translation of the new mRNA into a polypeptide chain. In general, they affect the functioning, activation and polarization of cells, and the ability to secrete cytokines. Epigenetic modifications mainly consist of DNA methylation via microRNA and histone acetylation. It is important to note that changes in the epigenome can become permanent in the next generations, and environmental changes affect not only the postnatal, but also the prenatal period.

A systematic review study of the genetics and epigenetics of atopic dermatitis was conducted by a team of scientists led by M.J. Martin et al. [1]. Epigenetic regulation has been confirmed with a high degree of probability as one of the determinants of the development of atopic dermatitis, along with polymorphisms of the filaggrin gene and genes associated with the immune system and the skin barrier.

Studies by Z. Mu et al. [2] confirm the differences in the epigenome between skin lesions in patients with atopic dermatitis and healthy subjects. In atopic dermatitis, epigenetic changes involve genes known to affect the regulation of the immune response, genes for innate immunity, and genes encoding structural proteins in the epidermis. DNA methylation is one of the most common epigenetic mechanisms that regulate gene expression. The methylation process targets the cytosine-phosphate-guanose-rich promoter sequences that indicate the direction and enable the transcription process. The addition of other methyl groups reduces gene expression.

In an epigenomic association study in adult patients with atopic dermatitis, significant differences in DNA methylation were observed at a total of 19 cytosine phosphate-guanosine sites and correlation with altered gene transcript levels between epidermal lesions in patients with atopic dermatitis and healthy control epidermis. These genes are mainly involved in the differentiation, proliferation and innate immune response of keratinocytes, including the S100A genes.

Activation of the transcription factor GATA3 in Th2 lymphocytes induces the production of IL-4, IL-5, and IL-13 by demethylation of the promoters of the IL-13 and IL-4 genes, as well as methylation of H3 histones in this region. It is accompanied by an increase in methylation of the filaggrin gene promoter and a decrease in H3 histone acetylation in this region of the gene.

Epigenetic changes in pregnant women have been extensively studied. For example, does exposure to smoking in utero cause methylation of cord blood DNA? Studies have confirmed that high smoke exposure can lead to hypomethylation of TSLP 5'CpG island, which is positively correlated with atopic dermatitis.

Other prenatal environmental factors, such as maternal allergies, maternal cytokine production, and exposure to tobacco smoke, can modify the DNA methylation of the FOXP3 locus in umbilical cord blood. It causes low levels of Treg in babies at birth and thus contributes to the development of atopic dermatitis or allergy to food allergens in the first years of life.

In addition to the transcriptional regulation of gene expression by chromatin modification, there is another mechanism, microRNA-mediated post-transcriptional regulation. MicroRNAs are a class of small, evolutionarily conserved, non-coding molecules, single-stranded RNAs. Specific sequences allow them to bind to specific mRNAs, resulting in mRNA degradation or translation inhibition.

The effects of the action are expressed in the regulation of apoptosis, morphogenesis, proliferation, regulation of cell metabolism, signaling and cell differentiation.

In atopic dermatitis, they are involved in the regulation of gene expression that determines Th2 polarization, the function of regulatory T-lymphocytes, inflammatory processes, tight junctions, proliferation and apoptosis of epidermal keratinocytes, and the synthesis of cytokines and chemokines.

Not only healthy skin and those affected by atopic dermatitis were compared with the identification of 44 microRNAs, which differed significantly between patients with atopic dermatitis and healthy control groups with 34 suppressed and 10 elevated microRNAs. The authors also confirmed that microRNA-155 is significantly overexpressed in infiltrating T lymphocytes in skin lesions in atopic dermatitis.

Infiltrating skin cells have been found to express microRNA-155, and CD4+ T cells are the main cell type responsible for increasing microRNA-155 levels in skin lesions. Environmental factors such as mites and staphylococcal superantigens can induce the expression of microRNA-155 in atopic skin. MicroRNA-155 can promote T cell activation and inhibit CTLA-4 expression, leading to the maintenance of chronic inflammation [3].

Another study found that microRNA-223 levels were elevated in blood whole cells in patients with atopic dermatitis, and histamine-N-methyltransferase (HNMT),

the main histamine-degrading enzyme, was elevated in patients with atopic dermatitis and in mouse atopic dermatitis models [4].

Thus, to summarize, epigenetic regulation is the link between the changing environment and genetic changes that collectively affect other pathogenic pathways, such as dysregulation of the immune system and disruption of the epidermal barrier.

When describing the etiological factors of the development of atopic dermatitis, it is impossible not to note the role and place of immunological regulation and reaction. According to N.B. Silverberg and J.I Silverberg [5], there are two main oppositely directed hypotheses of such disorders, which were conventionally designated as "from inside out" and "from outside in".

Proponents of the "inside-out" hypothesis believe that the initial event in the development of atopic dermatitis is the process of immunological aberration led by allergen stimulation, which leads to a weakening of the epidermal barrier. In contrast to these judgments, supporters of the "outside-in" hypothesis believe that disruption of the skin barrier is the first step in the pathogenesis of atopic eczema and is necessary for the onset of immune dysregulation.

In any case, at the first stage of the origin of atopic dermatitis, the immunological reaction occurs due to the body's first line of defense from the innate immunity, which is responsible for rapid and non-specific protection against external factors that may be pathogenic. According to T. Hato and P.C. Dagher [6], this system consists of the epidermal barrier, immune system cells, cytokines, pattern recognition receptors, antimicrobial peptides, and skin microflora.

Pattern recognition receptors are responsible for distinguishing pathogen-associated molecular patterns. These include toll-like receptors, proteins containing a nucleotide-binding domain of oligomerization (NOD-like receptors or NLRs), a retinoic acid-induced gene, C-type lectin receptors, and peptidoglycan recognition proteins. It has been noted that toll-like receptor polymorphisms, such as toll-like receptors-1 (rs5743571 and rs5743604), toll-like receptors-6 (rs5743794), and toll-like receptors-10 (rs11466617), as well as genetic mutation variants of toll-like receptors-2, may increase susceptibility to atopic dermatitis by increasing colonization by Staphylococcus aureus [7].

The relationship between R753Q in the toll-like receptor-2 gene and severe atopic dermatitis has also been described [8].

On the other hand, NOD1 and NOD2, which belong to the NLPR family, depending on different genetic variants or mutations, can lead to improper immunomodulation in allergic diseases. A significant association of NOD1 SNPs rs2907748, rs2907749, and rs2075822 with IgE levels was also observed. The rs2736726 and rs2075817 polymorphisms showed a weak association with atopic eczema. The analysis revealed a significant association between the NOD1 haplotype G-A-C-C-G-C-G-G-T-G and IgE, as well as the A-G-T-A-C-C-C-G-G-T-A-C-G haplotype and atopic dermatitis [9].

The meta-analysis included a total of nine case-control studies. It has been shown that the heterogeneous "GA" genotype of toll-like receptors-2 rs5743708 and the "AG" genotype of toll-like receptors-4 rs4986790 can be associated with increased susceptibility to atopic dermatitis in Caucasoids [10].

Heterozygous carriers of toll-like receptor-2 R753Q with atopic dermatitis, compared to healthy carriers, showed altered CD36 expression after stimulation and increased production of IL-6 and IL-12 by monocytes after stimulation of toll-like receptor-2 [11]. An association has also been observed between the expression of toll-like receptors-2 and the levels of high-affinity IgE receptors (FcεRI) [12]. FcεRI is found on various immune cells, binds to immunoglobulin E (IgE), and plays an important role in allergic diseases [13]. Expression patterns of FcεRI and toll-like receptors-2 were found to correlate with overall IgE levels. FcεRI-mediated signals can prolong the survival of monocytes, thereby contributing to the development of chronic allergies. Bacterial infections induce pro-inflammatory cytokines by activating toll-like receptors-2, which exacerbates allergies. They may also increase the regulation of FcεRI, which further enhances the current allergic reaction.

This assumption is supported by the study of mast cell reactivity under IgE-mediated ligand activation of toll-like receptors. Long-term exposure of mast cells to toll-like receptor ligands modulates effector responses, prompting them to increase the release of several inflammatory mediators in combination with the river. IgE [14].

In their research, D.P. Potaczek et al. [15] evaluated the co-effect of the rs4696480 gene of toll-like receptors-2, rs2252226 FcεRI (FCER1A) and rs2251746 α chain gene polymorphism on the severity of atopic dermatitis. A higher SCORAD was observed in the homozygotes of the main toll-like receptor-2 rs4696480 and simultaneously carried the smaller rs2252226 FCER1A allele.

Stimulation of human basophils by parallel activation of toll-like receptors and FcεRI directs the response towards a Th2-dependent response [16].

Not all studies confirm a positive correlation between toll-like receptors and FcεRI. In one study, FcεRI levels were reduced at protein and mRNA levels after stimulation with toll-like receptors-1/2 or toll-like receptors-2/6 [17].

The expression of FcεRI is likely to depend on the stage of maturation and the type of cells and tissues in which they are located, the total serum IgE levels, and various cofactors.

The skin produces antimicrobial peptides to kill or inhibit the growth of microbes. They contain more than 20 peptides with antibacterial activity, including cathelicidin, defensins and psoriazines. Altered expression and secretion of antimicrobial peptides may contribute to increased susceptibility to skin infections caused by viruses, bacteria, and fungi in patients with atopic dermatitis [18].

Innate lymphoid cells are a unique family of immune effector cells that functionally resemble T cells, but they lack clonal antigen receptors. Innate lymphoid cells stimulate the production of cytokines and act on immune and non-immune cells in the local tissue environment. Type 2 innate lymphoid cells are known for their ability to secrete proallergenic cytokines, including IL-4, IL-5, IL-9, and IL-13. This fact indicates that type 2 lymphoid cells may be involved in various allergic diseases, initiating the Th2 response [19].

The exact mechanism of activation of type 2 lymphoid cells in atopic dermatitis remains debatable. In patients with atopic dermatitis, infiltration of type 2 lymphoid cells in damaged skin was observed [20].

Low levels of type 2 lymphoid cells in mice with atopic dermatitis-like skin inflammation alleviated inflammation [21]. Increased expression of various receptors, such as the IL-25 receptor, the IL-33 ST2 receptor, the thymic stromal lymphopoietin receptor, and the PGD2 receptor CRTH2, on innate type 2 lymphoid cells has been observed in the skin of patients with atopic dermatitis. This suggests that type 2 innate lymphoid cells respond to non-specific factors of cellular origin, such as IL-33, IL-25, and thymic stromal lymphopoietin, or eicosanoids [22].

Some studies suggest the influence of innate type 2 lymphoid cells in acute inflammation and explain the increase in the number of innate type 2 lymphoid cells in tissues due to an overall increase in infiltrating immune cell populations.

One of the characteristic phenomena is a preference for differentiation of CD4 lymphocytes towards the Th2 line. Excess production of Th2 lymphocytes leads to an increase in the production of cytokines IL-4, IL-5 and IL-13. Cytokines stimulate IgE antibodies and eosinophils in peripheral blood and tissues [23].

Inflammation damages the epidermal barrier, which overlaps with the primary defects of the barrier. Factors that affect the destruction of the epidermis, such as damage, infections, or ongoing inflammation, stimulate keratinocytes to produce pro-inflammatory cytokines, such as thymic stromal lymphopoietin, IL-25, and IL-33. They also activate a Th2-mediated immune response [24].

Thymus stromal lymphopoietin, through its receptor, activates immature dendritic cells, enhances the maturation of antigen-presenting cells. In addition, thymic stromal lymphopoietin promotes eosinophilic activity and chemotaxis and enhances the expression of IL-4, IL-5, and IL-13 [25].

IL-25 induces the expression of various chemokines such as eotaxin, TARC (CCL17, thymus, and activation-regulated chemokine), and chemokine derived from macrophages, which are essential for the recruitment of eosinophilia and Th2 cells. IL-33 activates activated B cell nuclear factor and mitogen-activated protein kinases through the receptor, which stimulates the production of cytokines associated with the Th2 response, such as IL-4, IL-5, and IL-13. Continuous stimulation of IL-4 and IL-13 leads to a decrease in the expression of filaggrin in the epidermis [26].

Acute inflammation interferes with the synthesis of other proteins involved in the differentiation of keratinocytes, which leads to impaired barrier reconstruction. Chemotaxis of Th1 lymphocytes and increased production of cytokines IL-2, IL-12, TNFα and INF occur during the development of the disease in the chronic phase. It is also important to note that there is much discussion about an additional activation pathway through the cytokines Th22 and Th17, which release IL-17, IL-19, and IL-22, as well as the role of regulatory lymphocytes as another mechanism of atopic eczema [27].

The pro-inflammatory cytokines IL-36α, IL-36β and IL-36γ and their receptor antagonists IL-36Ra and IL-38 are signaling proteins belonging to the IL-1 family. A change in the regulation of newly discovered representatives was observed in atopic diseases. Recent studies suggest that they may be involved in the pathogenesis of atopic dermatitis [28].

Atopic dermatitis is such a complex and diverse disease that different groups of patients have different immune reactions. Thus, these specific molecular mechanisms underlying disease have been defined as disease endotypes that establish and alter constellations, giving rise to a specific phenotype. In acute lesions, there is an increase in cytokines along the Th2 and Th22 axes, and to a lesser extent Th17. With the development of the pathological process, an increase in biomarkers of the Th1 response, Th2 and Th22, the responses are enhanced. In addition to the typical Th2-dependent response in the pediatric population, there is a targeted increase in Th17 and Th22 cytokines and low levels of Th1 cytokines [29].

There are increasing discussions about different endotypes depending on ethnic origin. Responses of Th2 and Th22 lineages with lower Th1 and Th17 dominate European and American populations [30].

Evidence suggests that Asian patients with atopic dermatitis exhibit unique immune dysregulation compared to European and American patients. Thus, in the Japanese population, an increased frequency of the Th17 axis (and its relatives IL-17A, IL-19, IL-22 and S100A12) and suppression of the Th1 axis are indicated. In the Chinese population, in addition to Th2 activation and associated activation of IL-4, IL-13, IL-5, IL-10, IL-31 chemokines, increased Th17/IL-23 (e.g., IL-17F/IL-19/IL-21/CCL20) and increased expression of Th22-induced markers are noticeable. African Americans with atopic dermatitis have a targeted response to Th2 and Th22 with concomitant attenuation of Th1/Th17. Elevated plasma IgE levels and specific IgE have been observed in patients with atopic dermatitis. Higher expression of FcεRI is noticeable in the affected skin [31].

These disorders are associated with higher eosinophil counts and a family history of atopic disease. External stimuli and damage to the epidermal barrier lead to the stimulation of the Th2 response. The B lymphocytes then produce IgE antibodies specific to their proteins. Immunoglobulins bind to FcεRI on skin dendritic cells, enhancing the immune response and inflammatory response of the skin. Currently, there is still debate about whether autoreactivity transforms into a clinically significant autoimmune process. There are three arguments against this hypothesis: the lack of correlation between IgE levels and disease severity, the ambiguous effect of eliminating allergens associated with air and food allergens, and the cases of patients with atopic dermatitis with an unfavorable history of atopic disease.

Conflict of interest – the authors declare the absence of a conflict of interest

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ATOPIK DERMATIT PAYDO BO'LISHIDA EPIGENETIK VA IMMUNOLOGIK O'ZGARISHLAR

Gulmira Razikova

Allergologiya va klinik immunologiya Respublika ixtisoslashtirilgan ilmiy-amaliy tibbiyot markazi

ABSTRAKT

Genetik fondagi o'zgarishlar va yomonlashib borayotgan ekotizimning ta'siri ko'plab allergik kasalliklarning rivojlanishiga olib keladi, bu atopik dermatitning kelib chiqishi etiologik nazariyasini o'rganish uchun zaruriy shart bo'ldi. Bu jihatdan epigenetik o'zgarishlar birinchi o'rinda turadi. Genetik o'zgarishlar atopik dermatit bilan kasallanishning bunday tez o'sishini tushuntira olmaydi. G'arb turmush tarzi, sanoatlashtirish, havoning ifloslanishi, ovqatlanishning o'zgarishi, semizlik, antibiotiklardan ko'proq foydalanish va chekish kabi o'zgaruvchan atrof-muhit omillari oldinga chiqmoqda. Epigenetik o'zgarishlar organizm hujayralariga atrof-muhitning ta'sirining ehtimoliy mexanizmidir. Atopik dermatit rivojlanishining etiologik omillarini tavsiflashda immunologik tartibga solish va reaktsiyaning o'rnini va o'rnini qayd etmaslik mumkin emas. Ushbu ko'rib chiqish maqolasi atopik dermatit rivojlanishida epigenetik va immunologik jihatlarga oid adabiy ma'lumotlarni o'rganishga bag'ishlangan.

Kalit so'zlar: Atopik dermatit, epigenetika, immunologiya

ЭПИГЕНЕТИЧЕСКИЕ И ИММУНОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В ВОЗНИКНОВЕНИИ АТОПИЧЕСКОГО ДЕРМАТИТА

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АБСТРАКТ

Изменения генетического фона и влияние ухудшения состояния экосистемы приводят к развитию большого числа аллергических заболеваний, что также стало предпосылкой для изучения этиологической теории происхождения атопического дерматита. В этом аспекте на первом месте стоят эпигенетические изменения. Генетические изменения не могут объяснить столь стремительный рост заболеваемости атопическим дерматитом. Меняющиеся факторы окружающей среды, такие как западный образ жизни, индустриализация, загрязнение воздуха, изменение рациона питания, ожирение, более широкое использование антибиотиков и курение, выходят на первый план. Эпигенетические изменения являются вероятным механизмом влияния окружающей среды на клетки организма. При описании этиологических факторов развития атопического дерматита нельзя не отметить роль и место иммунологической регуляции и реакции. Данная обзорная статья посвящена изучению литературных сведений, касающихся эпигенетических и иммунологических аспектов в развитии атопического дерматита.

Ключевые слова: атопический дерматит, эпигенетика, иммунология