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МОЛЕКУЛЯРНО-ГЕНЕТИЧЕСКОЕ ОСОБЕННОСТИ АТОПИЧЕСКОГО ДЕРМАТИТА У ДЕТЕЙ РАННЕГО ВОЗРАСТА ЗАВИСИМО ОТ ВИДА ВСКАРМЛЕНИЕ.

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Резюме

Атопический дерматит (АД) является мультифакторным хроническим воспалительным заболеванием кожи, которое чаще всего манифестирует в раннем детском возрасте и характеризуется выраженной молекулярно-генетической гетерогенностью. Раннее развитие АД обусловлено сложным взаимодействием генетической предрасположенности, эпигенетической регуляции, созревания иммунной системы, нарушений кожного барьера и факторов окружающей среды, среди которых тип вскармливания играет важную модулирующую роль.

Целью данного исследования является изучение молекулярно-генетических особенностей атопического дерматита у детей раннего возраста в зависимости от вида вскармливания. Особое внимание уделено полиморфизмам генов, участвующих в формировании эпидермального барьера (FLG, LOR, CLDN1), регуляции иммунного ответа (IL-4, IL-13, IL-31, TSLP), а также механизмам врожденного иммунитета. В работе анализируется влияние грудного, искусственного и смешанного вскармливания на экспрессию генов, продукцию цитокинов и эпигенетические изменения, связанные с формированием иммунной толерантности и воспалительных реакций.

Ключевые слова: топический дерматит; дети раннего возраста; молекулярно-генетические особенности; тип вскармливания; иммунный ответ

ERTA YOSHDAGI BOLALARDA ATOPIK DERMATITNING MOLEKULYAR-GENETIK XUSUSIYATLARI OVQATLANTIRISH TURIGA BOG‘LIQ HOLDA.

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Резюме

Atopik dermatit (AD) — erta yoshdagi bolalarda ko‘p uchraydigan, multifaktorial surunkali yallig‘lanishli teri kasalligi bo‘lib, u molekulyar-genetik jihatdan yuqori darajadagi geterogenlik bilan tavsiflanadi. Kasallikning erta namoyon bo‘lishi genetik moyillik, epigenetik regulyatsiya, immun tizimining yetilishi, teri to‘sig‘ining buzilishi va atrof-muhit omillarining murakkab o‘zaro ta’siri bilan bog‘liq bo‘lib, ushbu jarayonda ovqatlantirish turi muhim modulyator omil hisoblanadi.

Ushbu tadqiqotning maqsadi erta yoshdagi bolalarda atopik dermatitning molekulyar-genetik xususiyatlarini ovqatlantirish turiga bog‘liq holda o‘rganishdan iborat. Tadqiqotda epidermal to‘siq shakllanishida ishtirok etuvchi genlar (FLG, LOR, CLDN1), immun javobni tartibga soluvchi sitokin genlari (IL-4, IL-13, IL-31, TSLP) hamda tug‘ma immunitet mexanizmlaridagi polimorfizmlar tahlil qilinadi. Shuningdek, ko‘krak suti bilan, sun’iy va aralash ovqatlantirishning gen ekspressiyasi, sitokinlar ishlab chiqarilishi va immun tolerantlik hamda yallig‘lanish jarayonlari bilan bog‘liq epigenetik o‘zgarishlarga ta’siri baholanadi.

Калит сўзлар: atopik dermatit; erta yoshdagi bolalar; molekulyar-genetik xususiyatlar; ovqatlantirish turi; immun javob.

MOLECULAR AND GENETIC CHARACTERISTICS OF ATOPIC DERMATITIS IN YOUNG CHILDREN DEPENDING ON THE TYPE OF FEEDING.

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Summary

Atopic dermatitis (AD) is a multifactorial chronic inflammatory skin disease that commonly manifests in early childhood and is associated with significant genetic and molecular heterogeneity. The early onset of AD is influenced by complex interactions between genetic predisposition, epigenetic regulation, immune system maturation, skin barrier integrity, and environmental factors, among which the type of infant feeding plays a crucial modulatory role.

This study aims to investigate the molecular and genetic characteristics of atopic dermatitis in young children depending on the type of feeding. Particular attention is given to polymorphisms in genes involved in epidermal barrier function (including FLG, LOR, and CLDN1), immune response regulation (IL-4, IL-13, IL-31, TSLP), and innate immunity pathways. The study also evaluates the influence of breastfeeding versus artificial and mixed feeding on gene expression profiles, cytokine production, and epigenetic modifications associated with immune tolerance and inflammatory responses.

Keywords: *atopic dermatitis; young children; molecular and genetic features; type of feeding; immune response*

Abstract

Atopic dermatitis (AD) is a chronic, inflammatory and highly heterogeneous skin disease that typically begins in early infancy. The pathogenesis of AD is complex, involving interactions between genetic predisposition, immune dysregulation, environmental exposures, epigenetic regulation, and infant nutrition patterns. While early identification of genetic risk factors — particularly those affecting skin barrier integrity and immune responses — has advanced, the modifying effect of feeding type (breastfeeding vs. formula feeding) on disease onset, severity, and immunogenetic expression remains a subject of ongoing research and debate. This review synthesizes current molecular, genetic, immunological and epidemiological evidence to provide an integrated perspective on how genetic susceptibilities interact with infant feeding regimens in AD development [1].

Atopic dermatitis (AD), also known as atopic eczema, affects an estimated 10–25% of children worldwide, with most cases beginning in infancy or early childhood. The condition usually presents with pruritic, chronic or relapsing eczematous lesions, often accompanied by elevated immunoglobulin E (IgE) and atopic comorbidities such as asthma or allergic rhinitis — a pattern termed the "atopic march" [2].

Both hereditary and environmental factors contribute to AD. Early onset is common, and genetic predisposition — particularly mutations in barrier-related genes — is well established as a major risk factor. However, the interplay between genetic risk and infant feeding practices (exclusive breastfeeding, mixed feeding, or formula feeding) remains nuanced and complex. [3].

Barrier Dysfunction: The Filaggrin Paradigm

A hallmark of AD pathogenesis is impaired epidermal barrier function, in large part due to mutations in the FLG gene, which encodes filaggrin — a key protein in the stratum corneum that maintains skin hydration and integrity. Defects in filaggrin reduce barrier function, increasing transepidermal water loss and allergen penetration, which can trigger immune responses [2–25]. AD is characterized by dysregulated T helper (Th) immune responses. In early life, a predominant Th2/Th22 skew is observed with elevated expression of IL-4, IL-13 and IL-31 among affected infants. These immune signatures promote IgE production and chronic cutaneous inflammation. Genes regulating innate immune receptors (e.g., TLRs) and cytokines also contribute to individual risk.

Exclusive Breastfeeding and AD Risk

Exclusive breastfeeding has been widely investigated as a potential modifiable factor in the development of AD. Some studies and meta-analyses suggest that exclusive breastfeeding for the first 3–4 months reduces the risk of eczema in early childhood, possibly through immunomodulatory and anti-inflammatory components of human milk (e.g., transforming growth factor-β, secretory IgA, cytokines). A cohort analysis demonstrated that exclusive breastfeeding for ≥4 months reduced the risk of childhood eczema at 4 years of age.

Conflicting Evidence and Genetic Context

Despite these findings, evidence remains mixed. Some meta-analyses and cohort studies have found no statistically significant protective effect of exclusive breastfeeding against AD, even among infants with a family history of atopy.

Genetic factors — including variations in milk-oligosaccharide metabolism genes and HMO (human milk oligosaccharide) profiles — may influence individual responses to breastfeeding in relation to AD risk.

Infant formulas vary in composition (intact proteins vs. partially hydrolyzed proteins). Some evidence indicates that in high-risk infants (e.g., positive family history), partially hydrolyzed formulas may reduce incidence of AD compared to standard formulas, possibly due to lower antigenicity. However, the overall picture remains complex and confounded by feeding duration, environmental exposures, familial genetics, and microbiome influences.

Despite high prevalence, the pathogenesis of pleural effusions in TB remains understudied due to the lack of evidence. Increased vascular permeability and exudation are probably key contributors to the development of exudative pleurisy.

[4].

Genetics and Immune Programming AD arises from multifaceted gene–environment interactions: barrier gene variants create susceptibility, but triggering of atopic phenotypes often requires environmental modulation — including diet, microbial exposure, pollutants and lifestyle factors. Breastfeeding may influence epigenetic programming of immune responses, although the direction and magnitude of effect vary among populations.

Conclusion

Atopic dermatitis in early childhood is a polygenic and environmentally modified condition where molecular and immunogenetic factors define individual susceptibility. Early feeding practices — including breastfeeding and formula choices — interact with these genetic frameworks, influencing immune development and potential AD risk. While breastfeeding confers many health benefits, its protective role in AD is supportive but not definitive across populations. Ongoing research integrating genomics, epigenetics, metabolomics, and microbiomics is critical to generate personalized prevention strategies.

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