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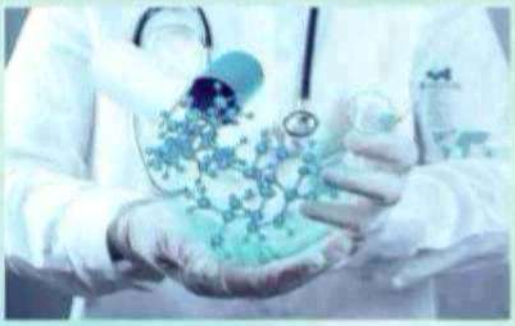

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E: ndmuz@mail.ru
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THE ROLE OF SKIN MICROBIOTA IN ATOPIC DERMATITIS IN CHILDREN

Sunyoung Woo https://orcid.org/0000-0002-9461-2432 e-mail: woosunyounganna@gmail.com
Mun Andrey Vitalevich https://orcid.org/0000-0002-9461-2432 e-mail: andrey_mun@yahoo.com

Tashkent State Medical University, 100109 Tashkent, Uzbekistan, 2 Farobiy Street, Tel: +998781507825 E-mail: info@tdmu.uz

✓ ***Resume***

Pediatric atopic dermatitis (AD) is a chronic, relapsing, and pruritic inflammatory skin disorder with high prevalence worldwide. It is characterized by epidermal barrier defects, immune dysregulation, and heightened sensitivity to environmental stimuli. AD in children often initiates the "atopic march," predisposing them to food allergies, asthma, and allergic rhinitis. The hallmark symptom, pruritus, triggers an itch-scratch cycle that exacerbates barrier dysfunction and facilitates microbial colonization. The skin microbiota plays a critical role in disease pathogenesis; dysbiosis, marked by reduced microbial diversity and overgrowth of Staphylococcus aureus, correlates with disease severity. Commensal organisms such as Staphylococcus epidermidis contribute to barrier protection but may act variably depending on strain and disease stage. Bacteriophages targeting pathogenic bacteria are emerging as potential therapeutics. Lesion morphology differs by disease stage, with acute lesions showing erythema, edema, and vesiculation, whereas chronic lesions demonstrate lichenification and xerosis. Age-specific lesion distribution informs sampling and treatment strategies. Secondary infections, barrier dysfunction, and flare triggers further complicate management. Topical corticosteroids, calcineurin inhibitors, and emollients are central to therapy, reducing inflammation, restoring barrier integrity, and normalizing microbiota. Standardized scoring systems, such as SCORAD and EASI, correlate disease severity with microbial imbalance. Understanding the interplay between skin microbiota, host immunity, and barrier function is essential for improving therapeutic outcomes and developing targeted interventions in pediatric AD.

Keywords: Atopic dermatitis, Children, Bacteriophages, Dysbiosis, Staphylococcus aureus

РОЛЬ МИКРОБИОТЫ КОЖИ ПРИ АТОПИЧЕСКОМ ДЕРМАТИТЕ У ДЕТЕЙ

Сонгенг Ву https://orcid.org/0000-0002-9461-2432 e-mail: woosunyounganna@gmail.com
Мун Андрей Витальевич https://orcid.org/0000-0002-9461-2432 e-mail: andrey_mun@yahoo.com

Ташкентский государственный медицинский университет, 100109 Ташкент, Узбекистан, ул. Фаробия, 2, Тел: +998781507825 E-mail: info@tdmu.uz

✓ ***Резюме***

Педиатрический атопический дерматит (АД) — это хроническое, рецидивирующее и зудящее воспалительное заболевание кожи с высокой распространённостью в мире. Он характеризуется нарушением эпидермального барьера, дисрегуляцией иммунной системы и повышенной чувствительностью к внешним раздражителям. У детей АД часто инициирует «атопический марш», предрасполагая к пищевым аллергиям, астме и аллергическому риниту. Основной симптом, зуд, вызывает цикл «зуд–воспаление», который усугубляет нарушение барьера и способствует колонизации микробов. Кожная микробиота играет критическую роль в патогенезе заболевания; дисбиоз, характеризующийся снижением разнообразия микробов и ростом Staphylococcus aureus, коррелирует с тяжестью заболевания. Комменсальные микроорганизмы, такие как Staphylococcus epidermidis, способствуют защите барьера, но их влияние зависит от штамма и стадии болезни. Бактериофаги, нацеленные на патогенные бактерии, рассматриваются как

перспективная терапия. Морфология поражений различается в зависимости от стадии болезни: острые поражения характеризуются эритемой, отёком и везикуляцией, тогда как хронические поражения проявляются лихенификацией и ксерозом. Возраст-зависимое распределение поражений помогает планировать забор образцов и стратегии лечения. Вторичные инфекции, нарушение барьера и провоцирующие факторы усугубляют течение болезни. Местные кортикостероиды, ингибиторы кальциневрина и эмоленты являются основой терапии, снижая воспаление, восстанавливая целостность барьера и нормализуя микробиоту. Стандартизированные шкалы оценки тяжести, такие как SCORAD и EASI, коррелируют с микробиальным дисбалансом. Понимание взаимодействия кожной микробиоты, иммунитета хозяина и целостности барьера имеет ключевое значение для улучшения терапевтических исходов и разработки целенаправленных вмешательств при педиатрическом АД.

Ключевые слова: Атопический дерматит, Дети, Бактериофаги, Дисбиоз, Золотистый Стафилококк

BOLALARDA ATOPIK DERMATITDA TERI MIKROBIOTASINING ROLI

Sunyoung Woo https://orcid.org/0000-0002-9461-2432 e-mail: woosunyounganna@gmail.com
Mun Andrey Vitalyevich https://orcid.org/0000-0002-9461-2432 e-mail: andrey_mun@yahoo.com

Toshkent Davlat Tibbiyot Universiteti, 100109 Toshkent, O‘zbekiston Farobiy ko‘chasi 2,
Tel: +998781507825 E-mail: info@tdmu.uz

✓ ***Rezyume***

Pediatrik atopik dermatit (AD) - bu surunkali, takrorlanuvchi va qichishuvchi yallig‘lanishli teri kasalligi bo‘lib, butun dunyoda keng tarqalgan. U epidermis to‘siqining buzilishi, immun tizimining disregulyatsiyasi va atrof-muhitga nisbatan sezgirlikning oshishi bilan tavsiflanadi. Bolalarda AD ko‘pincha «atopik marsh»ni boshlaydi, bu esa oziq-ovqat allergiyalari, astma va allergik rinitsga moyillikni oshiradi. Asosiy simptom - qichishish - “qichishish-tirnash” siklini keltirib chiqaradi, bu esa to‘siq buzilishini kuchaytiradi va mikroblarning kolonizatsiyasini osonlashtiradi. Teri mikrobiotasi kasallik patogenezida muhim rol o‘ynaydi; disbioz, mikroblar xilma-xilligining kamayishi va Staphylococcus aureusning ko‘payishi bilan tavsiflanadi, bu esa kasallik og‘irligi bilan bog‘liq. Staphylococcus epidermidis kabi kommensal mikroorganizmlar to‘siqni himoya qiladi, ammo ularning ta’siri shtam va kasallik bosqichiga qarab farq qiladi. Patogen bakteriyalarga yo‘naltirilgan bakteriofaglar istiqbolli terapiya sifatida ko‘rilmoqda. Lezyonlarning morfologiyasi kasallik bosqichiga qarab farqlanadi: o'tkir lezyonlarda eritema, shish va pufaklanish kuzatiladi, surunkali lezyonlarda esa likenifikatsiya va quruqlik ko‘rinadi. Yoshga bog‘liq lezyonlarning tarqalishi namunalarni olish va davolash strategiyalarini belgilashda yordam beradi. Ikkinchi darajali infektsiyalar, to‘siq buzilishi va tetiklovchi omillar kasallik kechishini kuchaytiradi. Mahalliy kortikosteroidlar, kalsineurin ingibitorlari va emolientlar terapiyaning asosiy qismini tashkil etadi, yallig‘lanishni kamaytiradi, to‘siq yaxlitligini tiklaydi va mikrobiotani normallashtiradi. SCORAD va EASI kabi standartlashtirilgan og‘irlik baholash tizimlari mikrobiota disbalansi bilan bog‘liq. Teri mikrobiotasi, mezbon immuniteti va to‘siq yaxlitligi o‘rtasidagi o‘zaro ta’sirni tushunish pediatrik AD-da terapevtik natijalarni yaxshilash va maqsadli aralashuvlarni ishlab chiqishda muhimdir.

Kalit so‘zlar: Atopik dermatit, Bolalar, Bakteriofaglar, Disbioz, Stafilokokk tillarang

Relevance

Pediatric Atopic Dermatitis (AD) is a chronic, relapsing, and intensely pruritic inflammatory skin disorder that represents one of the most common dermatological conditions in children worldwide [1]. From a dermatological perspective, AD is characterized by a primary defect in the epidermal barrier and increased sensitivity to environmental stimuli, resulting in the development of characteristic eczematous lesions [3].

In children, AD extends beyond a localized skin condition and is considered a systemic disorder that often initiates the "atopic march," a progression of allergic diseases that may include food allergies, asthma, and allergic rhinitis [3]. Diagnosis remains primarily clinical, based on established criteria such as those proposed by Hanifin and Rajka, which emphasize pruritus and typical lesion morphology [8, 9]. Notably, pediatric AD is associated with a substantial non-fatal disease burden, significantly affecting the physical, psychosocial, and economic well-being of both patients and their families [1].

The term eczema is frequently used interchangeably with atopic dermatitis but more precisely refers to the morphological pattern of cutaneous inflammation [3, 5]. Its presentation evolves depending on the stage of the disease and varies significantly across pediatric age groups [3, 5].

Acute eczema is characterized by erythema, edema, vesiculation, and serous exudation, often occurring during disease flares [3]. In contrast, chronic eczema is marked by lichenification, in which the skin becomes thickened and leathery with exaggerated markings due to persistent inflammation and repeated mechanical trauma from scratching [3].

These manifestations reflect an underlying disruption of the skin's "brick-and-mortar" structure, where deficiencies in structural proteins and lipids lead to increased transepidermal water loss and enhanced penetration of irritants and allergens [3].

Pruritus is the hallmark and most burdensome symptom of pediatric AD [3]. It often precedes the appearance of visible skin lesions, giving rise to the description of AD as "the itch that rashes" [5].

The resulting scratching behavior initiates an "itch-scratch cycle," in which mechanical injury exacerbates inflammation and further disrupts the epidermal barrier [1]. This cycle promotes microbial colonization and facilitates the penetration of environmental antigens [1].

In children, persistent pruritus leads to significant sleep disturbances, irritability, and impaired daily functioning, thereby exerting a substantial psychosocial impact on both the patient and caregivers [1]. Clinically, pruritus severity is a key indicator of disease activity and is incorporated into standardized scoring systems such as SCORAD [1].

Skin Microbiota. The human skin functions as a complex ecosystem inhabited by a diverse community of microorganisms, including bacteria, fungi, and viruses, collectively referred to as the skin microbiota [3]. In healthy individuals, this ecosystem exists in a balanced state known as eubiosis, contributing to host defense, maintenance of the skin barrier, and immune regulation [5].

In pediatric AD, this balance is disrupted, resulting in microbial dysbiosis characterized by reduced species diversity and overrepresentation of opportunistic pathogens [1]. Importantly, dysbiosis is not merely a consequence of the disease but actively contributes to its pathogenesis [1] (Table 1). Microbial diversity tends to decrease during disease flares and may recover with effective treatment, highlighting its dynamic relationship with disease activity [19, 20].

Table 1

Microbiota in Pediatric AD

Microorganism	Role in AD	Clinical Relevance
Staphylococcus aureus	Opportunistic pathogen; disrupts barrier; promotes Th2 inflammation	High colonization correlates with disease severity; target for therapy
Staphylococcus epidermidis	Commensal; inhibits *S. aureus*; strain-dependent effects	Protective in early life; may contribute to flares depending on strain
Bacteriophages	Infect and regulate bacterial populations	Potential targeted therapy against pathogenic bacteria
Other commensals	Maintain microbial diversity and barrier function	Dysbiosis associated with flare risk and chronicity

Staphylococcus aureus is the most significant opportunistic pathogen associated with pediatric AD [1]. Colonization rates reach up to 90% in affected children, compared to approximately 20% in healthy individuals [1].

The density of S. aureus colonization correlates positively with disease severity and plays a central role in exacerbating inflammation [5]. The bacterium produces multiple virulence factors, including toxins and proteases, which disrupt the epidermal barrier, damage keratinocytes, and promote Th2-mediated immune responses [5].

Additionally, S. aureus forms biofilms that protect it from topical treatments and host immune defenses, contributing to chronic persistence and recurrent disease flares [7].

Staphylococcus epidermidis is a predominant commensal organism on healthy skin and contributes to maintaining skin homeostasis [4]. It exerts protective effects by inhibiting the growth of S. aureus through antimicrobial peptide production and competition for ecological niches [5].

Early colonization with S. epidermidis in infancy has been associated with a reduced risk of developing AD, suggesting its role in immune and barrier maturation [6]. However, in established AD, its role becomes more complex [6]. Its abundance may increase during flares, and certain strains may contribute to barrier damage, indicating that its effects are strain-dependent and context-specific [6].

Bacteriophages are viruses that selectively infect bacteria and play a critical role in regulating the composition of the skin microbiota [13]. The skin "phageome" represents an important but often overlooked component of this ecosystem [24, 26].

In children with AD, a reduction in phage abundance targeting pathogenic bacteria such as S. aureus has been observed, potentially contributing to bacterial overgrowth [6]. Due to their high specificity, bacteriophages represent a promising therapeutic alternative to broad-spectrum antibiotics [24, 25].

Emerging strategies, including lytic phages and phage-derived enzymes, aim to selectively reduce pathogenic bacteria while preserving beneficial commensal microorganisms [13].

Lesion Types (Acute vs. Chronic) Pediatric AD presents with distinct morphological lesion types corresponding to different stages of inflammation [1]. Acute lesions are characterized by erythema, edema, vesiculation, and oozing, reflecting active inflammation [7].

Chronic lesions, in contrast, exhibit xerosis, scaling, and lichenification due to prolonged inflammation and repeated scratching [1]. Although these lesion types may coexist, their differentiation is essential for clinical staging and for selecting appropriate sites for microbiological sampling [1].

Acute lesions are particularly associated with higher levels of S. aureus colonization compared to chronic or non-lesional skin [3].

Affected Body Regions. The anatomical distribution of AD lesions changes predictably with age [1, 6]. In infancy, lesions commonly affect the cheeks, scalp, neck, and extensor surfaces of the limbs [1, 6].

During childhood, the distribution shifts to flexural areas such as the antecubital and popliteal fossae, as well as the wrists and ankles [1]. In adolescence, lesions tend to persist in flexural regions but often involve the face, neck, and hands in a more chronic form [1].

These age-dependent patterns are essential for accurate diagnosis and for standardizing sampling strategies in microbiota studies [1].

Secondary Infection. Children with AD are highly susceptible to secondary skin infections due to barrier dysfunction and reduced antimicrobial defense [1]. The most common pathogen is Staphylococcus aureus, which frequently causes impetiginization characterized by erythema and honey-colored crusts [1].

Streptococcus pyogenes is also commonly involved, either alone or in combination with S. aureus [1, 10]. These infections exacerbate inflammation, trigger disease flares, and complicate treatment outcomes [1, 10]. Prompt recognition and management are therefore critical [1, 10].

Barrier Dysfunction is the fundamental pathological feature of AD [3]. It involves structural and functional abnormalities of the stratum corneum, resulting in xerosis, increased transepidermal water loss, and elevated skin pH [5].

These changes are often associated with deficiencies in essential lipids, such as ceramides, and structural proteins like filaggrin [5]. The impaired barrier facilitates the penetration of allergens and the colonization of pathogenic microorganisms, thereby perpetuating the inflammatory cycle [5].

Topical Therapies are the cornerstone of AD management [2, 5]. Topical corticosteroids remain the first-line treatment for controlling inflammation during acute flares, while topical calcineurin inhibitors are used for maintenance therapy and for sensitive areas [2].

Effective topical treatment not only improves clinical symptoms but also restores microbial diversity and reduces the burden of S. aureus, highlighting the interplay between inflammation control and microbiota normalization [2].

Moisturizers / Emollients. Regular use of moisturizers and emollients is essential for maintaining skin hydration and repairing the epidermal barrier [2]. These agents reduce transepidermal water loss and improve skin integrity [3].

Consistent application has been associated with a steroid-sparing effect and contributes to the prevention of disease relapses [2]. Additionally, emollients positively influence the skin microbiota by increasing microbial diversity and reducing pathogenic colonization [2].

Flare Triggers. AD is characterized by recurrent flares triggered by environmental, mechanical, and immunological factors [6]. Common triggers include temperature changes, low humidity, irritants, allergens, and excessive washing [6].

Microbial factors, particularly overgrowth of S. aureus, often act as a final amplifier of inflammation, converting subclinical irritation into a full disease flare [6].

Disease Severity Scores (SCORAD, EASI)

Standardized scoring systems are essential for assessing disease severity in both clinical practice and research [1]. The SCORAD index integrates lesion extent, intensity, and subjective symptoms, while the EASI provides an objective assessment based on lesion area and severity [1].

Higher scores on these indices are consistently associated with reduced microbial diversity and increased S. aureus colonization, reinforcing the link between microbiota imbalance and disease severity [1].

Colonization Dynamics

The colonization dynamics of the skin microbiota in children with AD are influenced by age, anatomical site, and environmental factors [4]. Early-life microbial composition plays a critical role, with certain commensal bacteria offering protective effects against disease development [4].

As children grow, physiological changes alter the microbial landscape [4]. In AD, S. aureus colonization is dynamic and involves adaptation of specific strains to the host environment, contributing to persistent and recurrent disease activity [4].

Comorbidity Relevance. Pediatric AD is frequently associated with multiple comorbidities, including asthma, allergic rhinitis, and food allergies, reflecting its role in systemic allergic disease [2].

Additionally, patients are at increased risk of viral infections such as eczema herpeticum and molluscum contagiosum [2]. The chronic nature of the disease and its visible manifestations also contribute to psychological comorbidities, including anxiety, depression, and sleep disorders [2, 5].

These factors emphasize the need for a comprehensive management approach that addresses both dermatological and systemic aspects of the disease [2, 5].

Conclusion

Pediatric atopic dermatitis is a multifactorial disease involving genetic, immunological, environmental, and microbial factors. The integrity of the epidermal barrier is central to disease prevention and management, with barrier dysfunction driving pruritus, microbial colonization, and inflammation. Skin microbiota dysbiosis, particularly *S. aureus* overgrowth, contributes significantly to disease exacerbation and correlates with severity. Protective commensals, such as *S. epidermidis*, and bacteriophages offer promising avenues for microbiome-targeted therapies. Lesion type and distribution vary by age and disease stage, guiding clinical assessment and microbiological sampling. Topical therapies, moisturizers, and emollients improve skin hydration, reduce inflammation, and help restore microbial balance. Early-life microbial composition may influence AD risk, suggesting preventive strategies could be microbiota-informed. Secondary infections and environmental triggers act as critical amplifiers of disease flares, necessitating vigilant management. Standardized severity scoring (SCORAD, EASI) provides objective measures correlating microbial imbalance with clinical outcomes. Overall, integrating skin microbiota considerations into pediatric AD management may enhance treatment efficacy and reduce disease burden. Future research should focus on phage therapy, commensal modulation, and personalized microbiome-based interventions to achieve long-term disease control.

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