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INTESTINAL MICROBIOTA IN CHILDREN AND ITS ROLE IN THE DEVELOPMENT OF ATOPIC DERMATITIS.

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Annotation
Representatives of the normal microflora of intestinal biota perform a physiologically important function of maintaining the constancy of the internal environment of the body, take part in the formation of the immunobiological reactivity of the macroorganism. Violation of normocenosis contributes to the chronicity of pathological processes (in particular, atopic dermatitis).

Keywords: atopic dermatitis, children, local corticosteroids, topical calcineurin inhibitors, proactive therapy.

Аннотация
Представители нормальной микрофлоры биоты кишечника выполняют физиологически важную функцию поддержания постоянства внутренней среды организма, принимают участие в формировании иммунобиологической реактивности макроорганизма. Нарушение нормоценоза способствует хронизации патологических процессов (в частности, атопического дерматита).

Ключевые слова: атопический дерматит, дети, местные глюкокортикостероиды, топические ингибиторы кальциневрина, проактивная терапия.

Introduction

The second half of the 20th century will be remembered in the history of mankind as the time of the "new global epidemic" - the widespread occurrence of allergic diseases. Already by the 1980s, more than 20% of the European and North American population had at least episodic manifestations of allergic reactivity [1]. Over the past 30 years, according to the World Health Organization, the frequency of chronic allergic diseases has doubled: worldwide, about 5% of the adult population and 15% of children have a verified diagnosis of bronchial asthma, atopic dermatitis, food allergies and/or allergic rhinitis [2]. Of particular concern is the so-called atopic march — a natural increase in the severity of an allergic disease with age: from minimal manifestations of allergic dermatosis to mature bronchial asthma [3]. dermatitis. It is generally accepted that this is a skin disease that develops in individuals with a hereditary predisposition to allergies under the influence of specific factors.

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Discussion

Currently, genome-wide studies have identified more than 500 candidate genes associated with the implementation of atopy [4]. It is believed that the progressive increase in the prevalence of allergic diseases is largely associated with the growing environmental and climatic problems, leading to an imbalance of epigenetic factors that regulate the penetrance and expression of candidate genes [5]. It is possible that a significant “rejuvenation” of atopic diseases is also associated with unfavorable environmental factors — their manifestation at an earlier age than 20–30 years ago [6]. In this regard, the search for fundamentally new approaches to the prevention of atopic diseases, in particular, the assessment of the risks of atopy formation (at the stage of progenesis, intrauterine and perinatal ontogenesis), as well as the development of directions for "advanced elimination" of triggers of atopic conditions, is of particular relevance. To date, dysfunction of the skin barrier and altered immune responses appear to be major factors in the pathogenesis of AD [2–5]. At the heart of AD, according to modern concepts, is a violation of the skin barrier caused by a mutation in the FLG gene encoding the filaggrin protein. Filaggrin plays an important role in maintaining epidermal homeostasis by retaining water and maintaining the barrier function of the skin. A mutation in the FLG gene leads to an increase in transdermal water loss, as well as an increase in skin permeability, which facilitates the penetration of antigens and various microorganisms into it from the environment [6, 7]. In the acute stage of the disease, antigens from the environment penetrate the damaged skin barrier and cause the release of pro-inflammatory cytokines by keratinocytes, such as interleukin 33 and interleukin 25. The latter, in turn, activate type 2 T-lymphocytes, which leads to an increase in the inflammatory reaction in the skin, which is even more enhanced when Langerhans cells are involved in the process. Tissue remodeling observed in the chronic stage of AD may be secondary to interleukin-17-mediated release of cytokines with profibrotic activity, such as interleukin 11 and tumor growth factor beta (TGF-β) [8]. When studying atopic dermatitis (AD), it was noted that most patients are diagnosed with various pathologies of the gastrointestinal tract (GIT), while during the period of exacerbation, the severity of these changes significantly increases. During the period of exacerbation in patients with AD, characteristic dysbiotic changes in the intestinal microflora, metabolic and immune disorders were revealed [10,11]. The intestinal microflora plays a key role in the formation and functioning of the immune system, influencing the course of immunopathological processes in all organs and tissues.

It is known that numerous bacteria that inhabit the intestines, in particular Bacteroidesfragilis, Faecalibacteriumprausnitzii and bacteria belonging to the Clostridium IV and XI clusters, through various mechanisms, have a modulating effect on immunocompetent cells that produce pro- and anti-inflammatory cytokines in various organs and tissues, including the skin. [nine]. A number of studies have demonstrated a relationship between a violation of the composition of the intestinal microflora (intestinal dysbiosis) and blood pressure [16]. In particular, the use of metagenomic analysis of stool samples in patients with AD demonstrated a significant decrease in the abundance of F. prausnitzii in these patients compared with the control group [12,13]. In parallel, there was a decrease in the production of SCFA by the intestinal microflora in patients with AD. The authors emphasize the connection between the decrease in the number of F. prausnitzii, on the one hand, and the disruption

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of the epithelial barrier and secondary inflammation, on the other [17, 18]. At the same time, disruption of the intestinal barrier against the background of dysbiosis facilitates the penetration of antigens, microbes, and their toxins into the bloodstream and their interaction with target tissues, including the skin, where they trigger or enhance immune responses, leading to further tissue damage [16, 18–20] . Various variants of the composition of the microbiota are described in relation to the severity of atopic reactions. Thus, in children who developed a polyvalent allergy by the age of two, at the age of 1 month, the lowest Rhodotorula were detected [14,15]. A stable decrease in the same type of indigenous microorganisms by the age of 3 months (in combination with a violation of their metabolic profile) is proposed to be considered a predictor of bronchial asthma [19]. Significant increase in Staphylococcusaureus, Candidaspp. in the intestines of patients with AD during the period of exacerbation, it probably causes damage to the integrity of the intestinal wall, which is confirmed by the data of microscopic examination of colon biopsy specimens, thereby causing violations of the MTKK, and further stimulates the activation of all parts of the immune system, including allergic reactions [20]. At the same time, a decrease in the level of Lactobacillus spp. in the intestines of patients with AD reduces the antiallergic effect of the microflora. Lactobacillus spp. stimulate the formation of IgA, which, especially in early childhood, neutralize food allergens and reduce their absorption in the intestine [9–11]. In addition to the direct influence of Lactobacillus spp. on sensitization in AD, an indirect effect of intestinal dysbiosis on the composition of the skin microflora in AD has now been proven: a decrease in the content of lactobacilli in the intestine leads to an increase in the level of Staphylococcus epidermidis on the skin, which are an additional source of allergization of the body [12]. The unique composition of breast milk provides not only its high nutritional qualities, but also an irreplaceable immunoprotective and immunomodulatory effect, since it contains many biologically active components and its own microbiota, i.e. a complex of substrates that ensure the formation of food tolerance through the modulation of the functions of the intestinal lymphoid tissue [17,18].

According to many foreign researchers, deciphering the subtle mechanisms of the preventive action of breastfeeding in relation to the formation of allergies is difficult due to the vagueness and inconsistency of definitions: on the one hand, the nature of infant feeding, on the other hand, allergic diseases, especially in their initial manifestations [20]. Limited data indicate an association of exclusive breastfeeding and its varying duration with the degree of allergy prevention [10]. Perhaps this is why some researchers confirm the protective effect of adequate breastfeeding in children at risk for allergies [11–13], while others reject such a relationship [14, 15]. Nevertheless, no one denies the preventive role of breastfeeding in relation to infectious (including respiratory) diseases, the high frequency of which is associated with a risk of the asthmatic process [19,20]. Meta-analyses confirm the association of the duration of breastfeeding with a reduced risk of developing asthma [11]. It is noted that the minimum duration of breastfeeding to achieve a preventive effect should be 16 weeks [17]. Conclusion. Thus, the available data indicate a close functional relationship of the intestinal microflora with both physiological and pathological processes occurring in the skin, which allows us to speak about the existence of a functional axis "intestine - skin". A contribution to the development of AD can be made both by an imbalance in the intestinal microbiocenosis and a violation of the skin barrier. Promising

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strategies for the prevention of atopy include the development of methods for targeted correction of the intestinal microbiota in children at risk for atopy. Exploratory studies in this direction are currently being carried out.

List of used Literature

1. Булатова, Е.М. Кишечная микрофлора – один из факторов формирования здоровья человека / Е.М. Булатова, Н.М. Богданова // Медицинский совет. – 2013. – № 1. – С.30-33.
2. Макарова, С.Г. Кишечная микробиота и использование пробиотиков в практике педиатра. Что нового?/ С.Г.Макарова, Л.С.Намазова-Баранова // Педиатрическая фармакология. – 2015. – Т.12, № 1. – С. 38-45.
3. Хавкин, А.И. Нарушение микробиоценоза кишечника у детей: пособие для врачей/ А.И. Хавкин, О.Н. Комарова. – М.: ФГБОУ ВО РНИМУ им. Н.И. Пирогова. – 2016. – 48 с.
4. Blaser, M.J. The microbiome revolution / M. J. Blaser // J. Clin. Invest. – 2014. – Vol. 124, № 10. – P. 4162-4165.
5. Decreased gutmicrobiota diversity, delayed Bacteroidetes colonisation and reduced Th1 responses in infants delivered by Caesarean section / H.E. Jakobsson [et al.] // Gut – 2013. – Vol. 63, № 4. – P. 559-566.
6. Faecalibacterium prausnitzii subspecies-level dysbiosis in the human gut microbiome underlying atopic dermatitis / H. Song [et al.] // J. Allergy Clin. Immunol. – 2015. – Vol. 137. – P. 852-860.
7. Francino, M.P. Early development of the gut microbiota and immune health / M.P. Francino // Pathog. (Basel, Switzerland). – 2014. – Vol. 3, № 3. – P. 769-790.