Allergic diseases and childhood obesity: is there a relationship?

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Resume. Over the past decades, the proportion of obese children has increased 4-fold. At the same time, there is an increase in allergic pathology in the children's population.
The aim is to present modern data on the relationship between childhood obesity and allergic diseases.
Materials and methods. A search was conducted for domestic and foreign literature on the relationship between various links in the pathogenesis of allergic pathology and obesity using the databases Scopus, Web of Science, PubMed, Google Scholar, eLibrary, Cyberleninka. The review includes studies published from January 2016 to January 2025.
Results. Data on the mechanical and inflammatory effects of obesity in relation to atopy in children are described. In addition, obesity is associated with increased production of inflammatory cytokines and adipokines, which supports low-activity systemic inflammation and increases the risk of exacerbations of allergic diseases. Allergic rhinitis, atopic dermatitis, food allergies, and chronic urticaria also appear to be associated with the chronic systemic low-activity inflammation characteristic of obesity. Vitamin D deficiency, characteristic of obesity, appears to play a role in the development of bronchial asthma and allergic rhinitis, while dyslipidemia and skin barrier defects may explain the link between obesity and atopic dermatitis.
Conclusion. Further research on the relationship between obesity and atopy is needed, confirming the role of adipose tissue in the development of allergic diseases, in order to develop new therapeutic strategies.

Keywords: obesity, allergy, atopic dermatitis, asthma, children

Conflict of interest:
The authors declare that there is no conflict of interest.

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Обзор / Review

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Резюме. В течение последних десятилетий удельный вес детей, страдающих ожирением, увеличился в 4 раза. Параллельно в детской популяции отмечается рост аллергической патологии.
Цель — представить современные данные о взаимосвязи между детским ожирением и аллергическими заболеваниями.
Материалы и методы. Проведен поиск отечественной и зарубежной литературы о взаимосвязи различных звеньев патогенеза аллергической патологии и ожирения с использованием баз данных Scopus, Web of Science, PubMed, Google Scholar, eLibrary, Cyberleninka. *В обзор включены исследования, опубликованные с января 2016 года по январь 2025 года.*
Результаты. Описаны данные о механических и воспалительных эффектах ожирения в отношении атопии у детей. Кроме того, ожирение связано с повышенной выработкой воспалительных цитокинов и адипокинов, что поддерживает системное воспаление низкой активности и повышает риск обострений аллергических заболеваний. Аллергический ринит, атопический дерматит, пищевая аллергия и хроническая крапивница также, по-видимому, связаны с хроническим системным низкоактивным воспалением, характерным для ожирения. Дефицит витамина D, характерный для ожирения, по-видимому, играет определенную роль в развитии бронхиальной астмы и аллергического ринита, в то время как дислипидемия и дефекты кожного барьера могут объяснить связь между ожирением и атопическим дерматитом.
Заключение. Необходимы дальнейшие исследования взаимосвязи между ожирением и атопией, подтверждающие роль жировой ткани в развитии аллергических заболеваний, для разработки новых терапевтических стратегий.

Ключевые слова: ожирение, аллергия, атопический дерматит, бронхиальная астма, дети

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INTRODUCTION

Obesity and allergic diseases are a major public health burden today [1]. The World Obesity Federation predicts that by 2035, more than 1.5 billion adults and nearly 400 million children will be overweight and obese, and the global prevalence of childhood obesity will more than double between 2020 and 2035, from 10% to 20% among boys and 8% to 18% among girls [2].

At the same time, the prevalence of allergic diseases is also increasing significantly. Allergic rhinitis (AR) is the most common allergic disease, affecting 20 to 30% of adults and up to 40% of children in industrialized countries [3]. Atopic dermatitis (AtD) affects up to 12% of children and 7.2% of adults worldwide, and in Russia, according to the results of studies, the prevalence of AtD among children 7-8 years old varied from 5.5% to 49.2% depending on the region, and among children 13-14 years old - from 3.3% to 45.3% [4, 5]. The prevalence of bronchial asthma (BA) in childhood is registered with a frequency from 6 to 9% in different countries of the world and has a steady tendency to increase. In Russia, according to a recent epidemiologic study, the prevalence of BA among adults is 6-8% in children and up to 1.5% in adults [6]. Also in recent decades, there has been a significant increase in the frequency of food allergy (FA) - up to 6.7% [5]. Up to 1% of the population in the USA and Europe suffer from chronic urticaria (CU) [7]. At the same time, there is increasing evidence that obesity raises the risk of developing allergic diseases [8].

OBJECTIVE of this review is to present current data on the association between childhood obesity and allergic pathology.

MATERIALS AND METHODS. A search of Russian and foreign literature on the relationship between various links in the pathogenesis of allergic pathology and obesity was conducted using Scopus, Web of Science, PubMed, Google Scholar, eLibrary, and Cyberleninka databases. *The review included studies published from January 1, 2015 to January 1, 2025 in English and Russian languages.*

RESULTS. Allergic diseases arise not only from the interaction of genetic factors, but also from external causes that may increase susceptibility to allergic diseases through epigenetic changes. In particular, obesity represents an external factor involved in immunologic changes leading to a switch of the immune system to a Th2 cytokine profile and increasing the risk of developing atopy [9]. However, studies on the association between obesity and atopy have conflicting results. Some data indicate a correlation between BMI and the prevalence of atopy, while other results demonstrate a lack of association between atopy biomarkers such as serum IgE levels and blood eosinophil counts with obesity [10].

Inflammation caused by an imbalance of immune cells in adipose tissue may be associated with decreased tolerance to allergens and damage to the intestinal barrier. Adipose tissue contains many immune cells: mast cells, Th cells, cytotoxic T cells (Tc), regulatory T cells (Treg), regulatory B cells (Breg), invariant natural killer cells (iNKT) and M2 macrophages, which maintain immune balance. Obesity increases the number of pro-inflammatory immune cells, which secrete large amounts of pro-inflammatory cytokines (TNF-α, IL-6, etc.) and decreases the number of anti-inflammatory Treg, while anti-inflammatory M2 macrophages turn into pro-inflammatory (Ml) macrophages. Large amounts of pro-inflammatory cytokines can activate the nuclear factor kappa B (NF-κB) signaling pathway, induce the production of pro-inflammatory cytokines, chemokines, etc. and exacerbate the pro-inflammatory immunological effect. TNFα also activates NF-κB, which can increase the permeability of the epithelial barrier, disrupting the tight junctions of intestinal epithelial cells [11].

Treg dysfunction is one of the important links in the development of allergic diseases, since Treg are able to inhibit the proliferation of Th cells, reduce allergen-specific IgE secretion and migration of T cells into tissues, limit the interaction of eosinophils, mast cells and neutrophils with resident tissue cells [11]. This leads to decreased tolerance to allergens, promotes differentiation of native CD4+ T cells (Th0) into Th2 cells, causing allergic reactions. Elevated circulating levels of adipose tissue-derived pro-inflammatory cytokines may impair the survival and/or maturation of circulating Treg cells.

Mast cells are effector cells, secrete inflammatory mediators, and in allergic reactions cause symptoms in the skin, respiratory tract, and digestive tract. Mast cells are abundant in adipose tissue and can interact with adipocytes to recruit inflammatory cells.

Adipose tissue secretes a variety of adipocytokines, including leptin, adiponectin, IL-6 and TNF-α etc., which are actively involved in metabolic homeostasis. In adipose tissue accumulation, adipocyte dysfunction and dysregulation of adipocytokines are noted, which can cause local or systemic effects, leading to pathological processes associated with inflammation.

Leptin activates CD4+ T cells, stimulates the secretion of proinflammatory cytokines (TNF-α, IL-6, IL-12), which, in turn, suppresses Treg activity. Adiponectin enhances fatty acid degradation, reduces blood glucose levels and increases insulin sensitivity, has anti-inflammatory, antioxidant activity, and counteracts TNF-α expression in adipocytes and macrophages. In addition, adiponectin, the level of which is reduced in obesity, suppresses the synthesis of IL-10 secreted by macrophages and adipocytes. All these changes impair the immunoregulatory function of Treg, resulting in decreased antigen tolerance.

PPARγ DYSREGULATION AND ALLERGIC REACTION. Peroxysome proliferator-activated receptors (PPAR's) are a class of ligand-dependent nuclear receptors expressed in various organs and tissues, including adipose tissue. PPARγ is expressed in various immune cells, regulates genes involved in lipid metabolism, immune and inflammation-related genes, and exerts anti-inflammatory effects by inhibiting the expression of pro-inflammatory genes. The pro-inflammatory effects of adipose tissue may attenuate the anti-inflammatory effects of PPARγ by reducing its expression.

There is evidence that supplementation with ω-3 and ω-6 polyunsaturated fatty acids (PUFAs) can alleviate inflammation by upregulating PPARγ expression [12]. The effect of PUFAs on PPARγ also mediates changes in adipocytokine levels. The reduction of PPARγ in adipose tissue in obesity may affect Treg activity. Because PPARγ is a key regulator of mast cell maturation, decreased PPARγ expression in obesity accelerates mast cell differentiation, and increased mast cell activation may lead to intestinal barrier damage and increased risk of food allergy through the secretion of more tryptase.

OBESITY AND BRONCHIAL ASTHMA. Epidemiologic studies in recent years have demonstrated an association between BA and obesity. Although the mechanism of this relationship from the pathophysiologic point of view is not yet fully understood, obesity has been shown to be a risk factor for BA [9]. According to some authors, obesity increases the risk of BA development by 2.7 times, and overweight - by 2 times compared to patients with normal body weight [5]. J. E. Lang et al. (2019) showed that the highest risk of obesity-associated asthma is observed in prepubertal children in the absence of AR. The authors hypothesized that at this age, the likelihood of developing obesity-associated asthma would increase, especially in girls, while in boys this pattern is observed after 12 years of age [10]. But in a study conducted by R. Parlar-Chun et al. (2020), with the participation of 995 children, there was no association between childhood obesity and the severity of asthma course, resulting in prolonged hospitalization and intensive care [13].

L. B. Orriëns et al. (2020) suggested that overweight/obese children with BA have a high risk of intentional discontinuation of inhaled corticosteroid therapy, which, in turn, may lead to an increased frequency of asthma exacerbations [14]. An analysis of 16 European cohorts involving 21,130 children aged 3 to 8 years demonstrated that persistent wheezing and early onset of BA are associated with an increased risk of obesity [8].

The association between BA and obesity can be explained partly by mechanical factors and partly by the presence of chronic low-grade inflammation due to obesity. In some patients, obesity precedes asthma; in others, BA precedes obesity. This suggests that BA or its therapy may be a risk factor for obesity [15]. Bronchial asthma associated with obesity is more difficult to control. Patients tend to have more frequent exacerbations and have a lower quality of life [16]. Various studies show that obesity-associated BA differs from the classical phenotype, demonstrating a non-Th2-mediated response. BA exacerbations in obese patients are often characterized by a reduced response to baseline therapy [5]. This phenotype of BA is characterized predominantly by women and is accompanied by the absence of eosinophilic airway inflammation [5]. A significant negative correlation between the percentage of eosinophils in induced sputum and BMI and waist circumference was found, which may indicate the role of visceral fat in the maintenance of non-eosinophilic airway inflammation, typical of obese individuals.

Several studies have focused on the association between BMI and exhaled nitric oxide (FeNO) concentration with opposite results. Some authors describe a positive relationship between FeNO concentration and BMI, concluding that FeNO may be a systemic link between airway inflammation and obesity. Separate studies demonstrate a negative correlation between BMI and FeNO in patients with obesity-associated asthma [5].

A diet rich in carbohydrates and saturated fatty acids and low in fiber and antioxidants increases the risk of obesity and respiratory symptoms [17]. Vitamin D deficiency may also increase the risk of BA in obese patients [18]. Dyslipidemia and insulin resistance (IR) are associated with impaired forced vital capacity of the lungs (FVC) and the severity of BA. Insulin is a trophic stimulus for lower airway smooth muscle cells; it promotes laminin production, which increases airway hyperresponsiveness by stimulating parasympathetic innervation, thereby contributing to airway obstruction. High levels of total cholesterol and low-density lipoprotein are more common in children with obesity-associated BA and are associated with impaired lung function [19]. However, the exact mechanism by which dyslipidemia affects lung function is not yet known.

Maternal obesity before and during pregnancy also appears to play a role in the development of BA in children later in life. In a study of the effect of maternal diet on bronchial hyperresponsiveness in adult offspring in a mouse model, it was found that maternal diet during pregnancy, rich in saturated fatty acids, plays a key role in the development of airway hyperresponsiveness in the offspring [5]. The increased risk of allergic reactions in children born to obese mothers appears to be due to excessive synthesis of pro-inflammatory cytokines caused by excess adipose tissue. Such changes are probably the result of long-term changes in the expression of miR-155 and miR-133b microRNAs [10]. In addition, environmental factors play a role: various studies show that air pollution and passive smoking are independent risk factors for the development of both asthma and obesity in children [20]. Impaired lung development and growth also play a role: obese children have increased lung volume relative to airway diameter ("dysanapsia"), as reflected by a lower ratio of forced expiratory volume in 1 second to forced vital capacity (FEV1/FVC), despite normal FEV1 and FVC values. Dysanapsia is associated with decreased airway patency, more frequent exacerbations of BA, and use of systemic glucocorticoids in obese children [21].

Mechanical factors also play a role: obesity causes significant changes in lung and chest mechanics that cause asthma-like symptoms such as dyspnea, wheezing and airway hyperresponsiveness. Excess fat mass in the chest and abdomen reduces functional residual lung capacity (FRLC) [17]. It is also associated with decreased FVC and FEV1 [19-22]. Breathing with reduced respiratory volume due to excess fat in the thorax and abdomen leads to decreased lung volume, causing alveolar hypoventilation and increased airway resistance. These phenomena, in turn, cause airway hyperresponsiveness, leads to increased respiratory rate and increased chest elasticity. Collectively, all these changes lead to stiffness of airway smooth muscle in obese individuals, which leads to a decrease in bronchodilator effect [5].

In addition, the function of immune cells is also altered: traditionally, BA and other atopic diseases are thought to be associated with type 2 Th and increased levels of IgE and eosinophils. In obesity, the functions of adaptive and innate immune cells are impaired. Some studies show suppression of Th2 function in obese patients, i.e., the immune response shifts toward Th1 type [23]. Visceral inflammation with increased numbers of pro-inflammatory macrophages (M1) is also observed in patients with obesity-associated asthma and may determine systemic inflammation and the severity of BA [5]. In obese patients, oxidative stress, cell necrosis products, and excess free fatty acids lead to polarization toward the M1 phenotype, while the number of anti-inflammatory M2 macrophages decreases [24].

Eosinophil function is also altered in obesity. While the number of submucosal eosinophils is increased in obese patients with BA, the number of eosinophils in peripheral blood and sputum is not increased in obesity. All these aspects may explain why current drugs used in the therapy of BA, including corticosteroids, leukotriene inhibitors, and biologics that affect Th2 response and eosinophils, are less effective in patients with obesity-associated BA [19].

Adipose tissue mediators also play a role in the pathogenesis of BA. Adipose tissue is an active endocrine organ that can influence the functions of other organs, as well as a source of proinflammatory cytokines, chemokines, and growth factors [5]. Obesity is associated with increased production of inflammatory cytokines (IL-6, IL-1β and TNF-alpha), leading to low activity systemic inflammation and increased risk of frequent exacerbations of BA [8, 19]. High circulating IL-6 levels are associated with poor control of BA, and inflammatory biomarkers such as C-reactive protein (CRP) and fibrinogen are elevated in patients with obesity-associated asthma compared to patients with non-obese BA [5, 19].

In recent years, several cytokines produced by adipose tissue, the so-called adipokines, have been identified. Adipokines play a crucial role in energy homeostasis as well as in inflammatory and immune responses, supporting inflammation [24]. Leptin and resistin are pro-inflammatory cytokines, while adiponectin has anti-inflammatory properties. Leptin levels are positively correlated with adipose tissue mass, so it is considered one of the factors explaining the association between obesity and BA. The most important role of leptin is to suppress appetite by inhibiting the hypothalamic nuclei that stimulate hunger and activating the nuclei that induce the feeling of satiety. However, in obesity, patients develop leptin resistance with decreased sensitivity to anorexinergic stimuli. Leptin dysfunction leads to hyperphagia, rapid weight gain, and IR. Leptin stimulates neutrophil activation and chemotaxis, oxygen radical release and survival of macrophages, eosinophils, basophils and natural killer cells. Leptin and IL-6 decrease the activity of regulatory T lymphocytes (Treg), reducing immunologic tolerance to antigens, thereby increasing the risk of developing allergies and other immune-mediated diseases [25]. Adiponectin reduces the risk of AD, independent of BMI [25]. In macrophages, adiponectin promotes polarization of the M2 phenotype, decreases TNF-alpha secretion and enhances phagocytic activity. In addition, adiponectin stimulates the release of IL-10, one of the major anti-inflammatory cytokines that plays a key role in regulating the immune response and increasing insulin sensitivity. The role of resistin in the development of BA in obesity remains unclear. Some studies have found higher levels of resistin in patients with BA, and correlation of its levels with worsening disease control, while other authors have suggested that resistin prevents the development of BA [25]. Thus, adipokines play a key role in realizing the association between obesity and changes in the immune response, resulting in poorly expressed systemic inflammation and decreased immune tolerance. Body weight reduction combined with lifestyle modification effectively suppresses the level of serum markers of inflammation and IR in obese children and adolescents, resulting in improved BA control, lung function parameters and quality of life of patients [5, 25].

OBESITY AND ALLERGIC RHINOCONJUNCTIVITIS . Several studies have investigated the relationship between obesity and AR and rhinoconjunctivitis. A meta-analysis of 30 observational studies by Zhou J. et al. (2020) found a statistically significant association between obesity and the risk of AR in children [26]. The authors suggested that this association may be due to a common inflammatory etiology. Immunologic changes caused by obesity may lead to decreased immunologic antigen tolerance, thereby increasing the risk of developing AR. However, other factors, such as vitamin D deficiency, may also contribute to the association between obesity and AR: obese patients with vitamin D deficiency appear to have an increased risk of developing atopic diseases [5]. A positive correlation was found between obesity and atopic dermatitis and AR in adults, as well as a positive correlation between increasing BMI and the prevalence of AR and conjunctivitis, especially among young women and children [5]. M. W. Han et al. (2021) investigated the mechanisms by which obesity can influence the severity of AR. The authors analyzed the levels of leptin and inflammatory biomarkers in serum and found that the level of IL-1β, a biomarker of active inflammation, was significantly higher in patients with AR than without AR, as well as in obese individuals than in the group of people with normal body weight. Data analysis showed that leptin levels were associated with increased IL-1β expression in children with AR. In multivariate analysis, elevated leptin level and high IL-1β expression were found to be significant risk factors for the development of moderate to severe persistent AR [27].

Also, recent studies suggest a role for leptin in the development of AR in obese children. Leptin appears to increase the expression of innate lymphoid cell type 2 (ILC2), which may be involved in the development of AR [28]. These results suggest that obesity is a significant risk factor for exacerbations and severity of AR course. Allergic rhinitis is often a prerequisite for the subsequent development of AD (the so-called "atopic march"). Therefore, it becomes extremely important to control body weight to prevent the development of AR and subsequent BA [28].

OBESITY AND CHRONIC POLYPOUS RHINOSINUSITIS (CPRS). In recent years, several studies have been published to evaluate the association between obesity and CRPS. In a recent prospective population-based study conducted in Norway (2013-2018) involving 5769 patients, the authors found that the likelihood of developing CPRS was 53% higher in the obese group compared with the normal weight group [29]. Thus, BMI seems to be an important risk factor for the development of CPRS and should be taken into account in therapy. In a cross-sectional study by S. Nam et al. analyzed data from 32,384 patients aged 19 to 86 years and found that the prevalence of CPRS was higher in obese patients than in those with normal body weight [30].

In patients with asthma, CPRS is associated with increased eosinophilic airway inflammation and worsening lung function as measured by spirometry. The shared pathophysiology of CPRS and BA has important implications for the therapy of these conditions and provides a rationale for systemic therapy with new biologic agents targeting common Th2 inflammatory processes, such as dupilumab and omalizumab [31].

OBESITY AND ATD. A significant correlation was found between high BMI and AtD in children. In addition, obese children are significantly more likely to have a severe course of the disease. There is also evidence that prolonged obesity (more than 2.5 years) in early childhood is a modifiable risk factor for childhood AtD, while short-term obesity does not increase the risk of developing ATD. This suggests that rapid reduction of body weight is an important non-pharmacologic intervention for the prevention and treatment of AtD in children [32]. Chronic sluggish inflammation caused by excess adipose tissue is probably involved in the pathogenesis of AtD, and increased leptin expression and decreased adiponectin expression in obese patients may contribute to the pathogenetic link between obesity and AtD [33, 34]. A. K. Jaworek et al. (2020), while evaluating the levels of various adipokines in the blood of adult patients with normal body weight, suffering from chronic AtD since childhood, found increased levels of leptin and decreased levels of adiponectin in patients with AtD compared to healthy people. The level of adiponectin was negatively correlated with the severity of the disease [35]. However, the direct role of adipokines in the pathogenesis of AtD is still not fully understood. S. P. Bapat et al (2022) studied 2 mouse models of AtD and found marked differences in the immune response in lean and obese mice. Obesity transformed the classic Th2-dependent inflammation, into a more severe form of the disease with a predominance of Th17 inflammation. The authors also observed a different response to biological therapies targeting Th2 cytokines, which protected lean mice but worsened the condition in obese mice [36]. This may be important for the development of effective treatments for children with obesity and allergic diseases.

Another mechanism that may explain the association between obesity and AtD is related to changes in lipid profile. A recent study by J. H. Kim et al. (2022) showed that children with AtD had significantly higher levels of total cholesterol and triglycerides, and this is associated with the AtD index (SCORAD) indicating the involvement of dyslipidemia in the pathogenesis of pediatric AtD [37]. It is also believed that mechanical factors such as skin maceration and stretch marks, often observed in obese patients, may also contribute to the development of AtD. Excess subcutaneous adipose tissue may adversely affect epidermal barrier function, as obese children have significantly higher rates of transepidermal water loss through the skin than normal body weight [5]. This suggests that obesity may exacerbate the course of AtD by compromising the integrity of the skin barrier, thereby facilitating transdermal penetration of allergens, leading to AtD. Further prospective cohort studies are needed to confirm the association between AtD and obesity.

OBESITY AND FOOD ALLERGY. Various studies show that obesity may contribute to the increase in the prevalence of FA in children. In a study conducted by K. Hayashi et al. (2021) in Japan, an analysis of data from 1,772 children revealed that in girls, excess weight was statistically significantly associated with FA (OR 1.99, $p = 0.046$), while in boys, no such association was found [38].

Dietary lipids can affect innate immune function and antigen presentation to adaptive immune cells. Lipids appear to alter the immunostimulatory properties of proteins and affect their absorption in the intestine, thereby altering the bioavailability of allergens [39]. A correlation has been found between CRP and total IgE levels, atopy, and food hypersensitivity [5]. This result suggests that the presence of low-grade inflammation in obese children may contribute to the development of FA. Some authors hypothesize that damage to the gastrointestinal barrier caused by a high-fat diet, hyperglycemia, and chronic systemic low-grade inflammation may explain the link between obesity and FA [40]. However, the pathophysiological mechanisms underlying this phenomenon have not yet been fully studied. L. Torres et al. (2023), in a study of changes in the intestinal mucosa in mice with alimentary obesity, found that they had increased permeability and reduced numbers of intraepithelial Tregs, and after oral administration of ovalbumin, tolerance did not develop, but a stronger reaction to ovalbumin was observed [41]. In addition, changes in the intestinal microbiota also play an important role in the development of obesity and FA. Further prospective studies are needed to identify the causal link between obesity and FA.

OBESITY AND CHRONIC URTICARIA. An Italian study analyzed the influence of various risk factors associated with CU and showed that the risk of developing CU was statistically significantly higher in obese patients. Chronic low-grade systemic inflammation in obesity may reduce immunological tolerance to antigens, thereby increasing the risk of CU [42]. There is evidence that central obesity is more common in patients with CU and correlates significantly with levels of TNF-alpha, total serum IgE, and eosinophil cationic protein [5]. However, further research is needed to confirm these findings and the possible link between obesity and the onset of CU.

CONCLUSIONS. Obesity is one of the risk factors for developing allergic diseases and, at least in part, is responsible for their recent increase. Disorders of innate and acquired immunity in obese patients also affect the development and severity of asthma. Polarization of the immune response towards Th1 and Th17 and an increase in the number of pro-inflammatory macrophages (M1) lead to an increase in the severity of asthma and explain the low effectiveness of basic therapy in patients with obesity-associated asthma. Excess body weight is associated with increased production of inflammatory cytokines (IL-6, IL-1β, and TNF-alpha) and adipokines (leptin and resistin), which leads to the formation of chronic systemic inflammation and increases the risk of BA exacerbations. However, the exact mechanism linking obesity and other atopic diseases remains unclear. Vitamin D deficiency appears to play a role in the development of

AR, while dyslipidemia and skin barrier defects may explain the link between obesity and AtD. Therefore, further research is needed on the association between obesity and atopic diseases to clarify the role of adipose tissue in the development of allergic diseases for the development of new therapeutic strategies.

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Nataliya A. Belykh — conceptualization, writing — review & editing — preparation, creation and/or presentation of the published work by those from the original research group, specifically critical review, commentary or revision — including pre- or post-publication stages.

Inna V. Piznyur, Polina O. Kotova — data curation.

Inna N. Lebedeva, Yulia V. Deeva — writing — review & editing.

ВКЛАД АВТОРОВ В РАБОТУ

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Пизнюр И. В., Котова П. О. — проведение исследования.

Лебедева И. Н., Деева Ю. В. — подготовка текста.